MINISTRY OF HEALTH OF THE REPUBLIC OF UZBEKISTAN
BUKHARA STATE MEDICAL INSTITUTE
NAMED AFTER ABU ALI IBN SIN
DEPARTMENT OF THERAPEUTIC DENTISTRY

Training and metodology complex
for 5th year students of the Faculty of Dentistry
on subject
DISEASES OF THE MUCOSA OF THE ORAL CAVITY

Bukhara – 2021

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MINISTRY OF HIGHER AND SECONDARY SPECIALIZED EDUCATION OF THE REPUBLIC OF UZBEKISTAN
BUKHARA MEDICAL INSTITUTE NAMED AFTER ABU ALI IBN SINO
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DISEASES OF THE MUCOSA OF THE ORAL CAVITY

Area of expertise - 500,000 "Health and Social Security"
Education area - 510000 "Healthcare"
Direction of education - 5510400 "Dentistry"

Bukhara – 2021

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Lecture text 1

Diseases of the oral mucosa are an important section of therapeutic dentistry, and not only for dentists, but also for doctors of other specialties. The mucous membrane of the mouth reflects the state of many organs and systems of the body. It is under the constant influence of environmental factors, often pathogenic, exceeding the capabilities of the body's defense reactions.

For a long time, diseases of the oral mucosa were considered as a local pathological process caused by insufficient oral hygiene and exposure to local traumatic factors. The merit in a fundamentally new approach to the study of diseases of the oral mucosa belongs to Russian scientists I. G. Lukomsky, E. E. Platonov, A. I. Evdokimov, B. M. Pashkov, A. L. Mashkillyson and others. This approach consists in the correct assessment of the role of local and general, external and internal, the relationship and interdependence of a number of factors in the study and approval of the cause-and-effect relationships of common diseases and manifestations of pathology in the oral cavity.

Currently, diseases of the oral mucosa are considered from the standpoint of the whole organism, since no one doubts the presence of a connection between most pathological processes on the oral mucosa and the red border of the lips with changes in organs and body systems, the nature of metabolic processes, immune status, etc. e. Changes in the oral mucosa are often the first clinical symptoms of metabolic disorders, diseases of the hematopoietic, cardiovascular systems, gastrointestinal tract, skin and venereal diseases, etc. Therefore, in order to learn how to diagnose diseases of the oral mucosa, the dentist must have a high professional level, have good knowledge of related specialties. At the same time, the principles of deontological

gia and medical ethics in combination with an individual approach to each patient, with knowledge of his psychological and social characteristics, the ability to see the personality of the patient as a whole with his experiences, and not just individual manifestations, symptoms of the disease.

When diagnosing diseases of the oral mucosa, it is important to correctly identify the elements of the lesion, understanding the features of the clinical course, localization of pathological changes, knowledge of the morphological and functional features of the oral mucosa, saliva, detailed study of pathomorphological changes in the oral mucosa in various diseases. A comprehensive and deep study of individual diseases that manifest themselves on the oral mucosa made it possible to create scientifically based classifications of diseases of the lips (G.D.Savkina), pemphigus (N.D.Sheklakov), precancerous lesions of the lips (A.L. systematics of diseases of the oral mucosa (E.E. Platonov) and their classification (T.F. Vinogradova, E.V. Borovsky, A.L. Mashkillyson, etc.).

For the correct recognition and treatment of diseases of the oral mucosa, it is often necessary to contact the dentist with other specialists in order to select the methods of additional examination, draw up a comprehensive therapy plan, taking into account the recommendations of doctors of other specialties and the nature of the manifestation of diseases in the oral cavity.

CLASSIFICATION OF DISEASES OF THE ORAL MUCOSA

Difficulties in diagnosing and choosing a method of treatment for pathology of the oral mucosa are due to the variety of manifestations of diseases localized in this area and often having similar clinical manifestations with different etiology or pathogenesis. The classification of diseases of the oral mucosa is very important for the practitioner, since it helps to navigate their existing variety, thereby contributing to the correct diagnosis, the choice of justified treatment methods and the implementation of preventive measures.

Previous classifications of diseases of the oral mucosa were based on different principles: anatomical and clinical (stomatitis, cheilitis, glossitis), the nature of the course (acute and chronic), clinical and morphological

their signs (catarrhal, ulcerative, etc.), the depth of the lesion (superficial and deep), the nature of the rashes (bullous, ulcerative, vesiculate, etc.) - However, each of them had its drawbacks and did not fully meet the above classification requirements.

The World Health Organization (WHO) has adopted the following nomenclature of diseases:

I. Infectious and parasitic diseases II. Neoplasms

III. Diseases of the blood and hematopoietic organs and lesions caused by the immune mechanism

IV. Endocrine, metabolic and nutritional diseases

V. Mental and character disorders VI. Diseases of the nervous system IX. Diseases of the circulatory system X. Diseases of the respiratory system XI. Diseases of the digestive system XII. Diseases of the skin and subcutaneous tissue

XIII. Diseases of the musculoskeletal system and connective tissue

XIV. Diseases of the genitourinary system

XV. Pregnancy, childbirth and the puerperium

XVII. Birth defects, deformities and chromosomal abnormalities

XVIII. Symptoms, signs, abnormalities, not elsewhere classified

XIX. Injury, poisoning and other consequences of external factors

XX. External factors leading to morbidity and mortality

For each of the listed sections of the classifications, there is a list of dental diseases and syndromes manifested in the oral cavity, characteristic of a given systemic pathology or condition. Systematization of dental diseases within the group of

It is based on clinical and morphological, pathomorphological characteristics, the nature of microflora, etc.

w The main goal of creating an international nomenclature of dental diseases and syndromes in the oral cavity is to standardize approaches to identifying dental pathology with subsequent registration and determination of the level of dental morbidity in different countries. In addition, the information obtained allowed us to determine the correlations of the general state of the body with the level of dental health, as well as to identify systemic diseases associated with pathology in the oral cavity.

Thus, at present, the etiological and pathogenetic relationship between diseases of various organs and systems and pathology in the oral cavity has been proven, and primarily diseases of the oral mucosa.

To systematize the known pathological conditions (diseases) of the oral mucosa, E.V. Borovsky and A.L.

I. Traumatic injuries due to mechanical factors, high and low temperatures, radiation, adverse meteorological factors (meteorological cheilitis, cracked lips), chemicals, etc. Form of manifestation: hyperemia, erosion, ulcers, hyperkeratosis (leukoplakia).

II. Infectious diseases:

1. Lesions of the oral mucosa in acute and chronic infectious diseases (measles, scarlet fever, chickenpox, tuberculosis, syphilis, leprosy, etc.);

2. Actually infectious and parasitic diseases of the mucous membrane of the mouth and lips;

^ viral (herpes, HIV infection, warts, etc.);

A fusospirochetosis;

^ bacterial (streptococcal and staphylococcal, gonorrheal, etc.);

And fungal (candidiasis, actinomycosis, etc.).

III. Allergic and toxic-allergic diseases:

^ contact allergic stomatitis, gingivitis, glossitis, cheilitis (from medicines, plastics and

other materials used in dentistry, dyes, toothpastes, elixirs and other chemicals in contact with the mucous membrane or red border of the lips, ultraviolet rays);

^ fixed and widespread toxic-allergic lesions (from medicines, food substances and other allergens entering the body in various ways);

^ dermatoses with lesions of the oral mucosa of toxic-allergic genesis (exudative erythema multiforme, Stevens-Johnson syndrome, Lyell's syndrome, primary systemic vasculitis, including Wegener's syndrome).

IV. Diseases with an autoimmune component of the pathogenesis:

A recurrent aphthous stomatitis, including cicatricial aphthae;

A. Behcet's syndrome, including Touraine's aphthous disease;

A Sjogren's syndrome;

And dermatoses with lesions of the oral mucosa (pemphigus, pemphigoid, Duhring's disease, systemic lupus erythematosus, systemic scleroderma).

V. Mucocutaneous reaction - lichen planus.

Vi. Changes in the oral mucosa with exogenous intoxication.

Vii. Changes in the mucous membrane of the mouth and the red border of the lips in the pathology of various organs and systems of the body and metabolic disorders:

And with visceral and endocrine pathology;

And with hypo- and avitaminosis;

And with diseases of the blood and hematopoietic organs;

And with the pathology of the nervous system;

And during pregnancy.

VIII. Congenital and genetically determined diseases

nevi and epithelial dysplasias: vascular nevi, including Sturge-Weber syndrome, bo-

birth and pigment nevi, epidermoid cyst, Fordyce's disease, white spongy nevus (mild leukoplakia, “buccal biting”, etc.), hereditary benign intraepithelial dyskeratosis;

^ folded and rhomboid glossitis; A glandular cheilitis;

^ dermatoses with lesions of the mucous membrane of the mouth and lips, epidermolysis bullosa, atopic dermatitis (cheilitis), psoriasis, ichthyosis, Darier's

disease, Peitz-Jägers-Touraine syndrome, congenital paraonychia, anhydrotic epithelial dysplasia.

IX. Precancerous diseases, benign and malignant neoplasms:

And obligate precancer: Bowen's disease, warty precancer, limited hyperkeratosis of the red border of the lips, abrasive precancerous cheilitis of Manganotti;

And optional precancer: leukoplakia, keratinizing papilloma and papillomatosis, keratoacanthoma, cutaneous horn, erosive-ulcerative and hyperkeratotic forms of lupus erythematosus and lichen planus erythematosus, postradiation cheilitis;

A benign neoplasms;

And cancer.

At the Moscow Medical Dental Institute, the following classification of diseases of the oral mucosa is used in the educational process and medical work.

Classification of diseases of the oral mucosa

I. Traumatic injuries (>; chemical, physical! Kaya erythema, erosion, ulcer, leukoplakia, nicotinic leukeratosis, actinic cheilitis, radiation, chemical damage, etc.

II. Infectious diseases:

And viral (herpetic stomatitis, shingles

lichen, foot and mouth disease, viral warts, influenza, HIV infection);

And Vincent's ulcerative necrotizing stomatitis;

And bacterial infections (streptocal stomatitis, pyogenic granuloma, chancriform pyoderma, tuberculosis, etc.);

and sexually transmitted diseases (syphilis, gonorrheal stomatitis);

And mycoses (candidiasis, actinomycosis, etc.);

III. Allergic diseases (Quincke's edema, allergic stomatitis, cheilitis and glossitis, drug-induced stomatitis, glossitis, cheilitis, erythema multiforme exudative, recurrent aphthous stomatitis, etc.),

IV. Changes in the oral mucosa with exogenous intoxication.

V. Changes in the oral mucosa in some systemic diseases and metabolic diseases (hypo- and avitaminosis; diseases of the endocrine, gastrointestinal tract, cardiovascular system, blood system, nervous system; rheumatic diseases, or collagenoses).

Vi. Changes in the oral mucosa with dermatoses (pemphigus, Duhring's dermatitis herpetiformis, lichen planus, lupus erythematosus).

Vii. Anomaly and independent diseases of the tongue

(folded tongue; black "hairy" tongue; diamond-shaped, desquamative).

VIII. Independent cheilitis (glandular, exfoliative, actinic, meteorological, atopic, eczematous, contact, macrocheilit).

IX. Precancerous diseases (obligate and facultative) and tumors (benign and malignant). 11.2. TRAUMATIC INJURIES

The mucous membrane of the mouth is in direct contact with the external environment and is constantly exposed to various mechanical, thermal, chemical, microbiological, physical and other factors. If these stimuli do not exceed the irritability threshold of the oral mucosa, then it, due to its protective function, does not change. However, when exposed to stronger suprathreshold stimuli, changes occur on the oral mucosa, the nature of which depends on the type, intensity and duration of the traumatic factor. The severity of these changes is also determined by the localization

of the effect of an external factor, the state of the body's reactivity, individual features of the structure of the mucous membrane, etc. Due to the fact that the main role in the clinical picture of traumatic lesions of the oral mucosa belongs to the type of irritant, it is necessary to separately consider changes in the mucosa shells resulting from exposure to mechanical, temperature, radiation and chemical factors.

Mechanical trauma

This injury can be acute when the mucous membrane is damaged by a short-term but significant factor, and chronic - with prolonged exposure to a weak stimulus.

Acute mechanical trauma

Acute mechanical trauma (trauma mechanicum acutum) in the form of biting or wounding with instruments is rare. It manifests itself as a hematoma (interstitial hemorrhage without disturbing the integrity of the epithelium), erosion or ulcer. With interstitial hemorrhage at the site of injury, pain is noted, which quickly (after 1-3 days) disappears, but if the integrity of the epithelium is damaged, painful erosion or an ulcer with infiltration is formed. Secondary infection, the wound can turn into long-term non-healing ulcers. Diagnosis, as a rule, is not difficult - the cause is revealed from the anamnesis.

Treatment. The amount of medical assistance depends on the depth and magnitude of the damage. With hematomas, erosions and chalk

such wounds are sufficiently thorough antiseptic treatment of the damaged area and the entire oral cavity. To do this, rinse with antiseptic solutions (potassium permanganate 1: 5000; 0.5% hydrogen peroxide solution and other drugs), applications of agents that stimulate epithelization (oil solution of vitamins A, E, carotolin, rosehip oil, jelly, solcoseryl ointment and etc.). For deep wounds, stitches are applied.

Chronic mechanical trauma

Chronic mechanical trauma (trauma mechanicum chronicum) is common. A traumatic factor can be sharp edges of teeth, bridges and removable dentures, tartar, bad habits (biting the mucous membrane), etc. , the strength of the irritating factor. Damage to the mucous membrane under the influence of chronic irritants is more common in older people. This is facilitated by a decrease in the turgor of the mucous membrane, a decrease in the height of the bite due to abrasion of hard tissues of the teeth, loss, displacement of teeth. In older people, the regeneration process is slowed down, which is the reason for the slow healing of the damaged mucous membrane.

Changes in the mucous membrane during chronic mechanical injury may not bother the patient for a long time, but sometimes a feeling of awkwardness, discomfort, slight soreness, and swelling may occur. When examining the mucous membrane, catarrhal inflammation (edema, hyperemia), violation of its integrity (erosion, ulcers), proliferative changes (hypertrophy of the gingival papillae, gingival margin), hypertrophy of the papillae of the tongue such as papillomatosis, increased keratinization (leukoplakia) can be detected. These changes can occur in combination with each other. It should be remembered that simultaneously with mechanical injury, the mucous membrane is exposed to the microflora of the oral cavity, which often affects the clinical picture of the disease.

CATARRH. It occurs most often in chronic trauma and is manifested by hyperemia, edema with infiltration. The severity of these changes depends on the strength and

duration of the stimulus. In this case, the process can be accompanied by the expression

female exudation (at first the secret is serous, and then purulent).

Of the factors that can cause irritation and damage to the oral mucosa, dentures should be singled out. They are inappropriate stimuli. The removable prosthesis transfers the chewing pressure to the mucous membrane, delays the self-cleaning of the oral cavity, which leads to a violation of the established balance between different types of microorganisms, changes the analytic function of the mucous membrane receptors. The onset of inflammation of the mucous membrane under the prosthesis is due to the influence of not one, but several factors (traumatic, allergic, infectious).

Inflammation of the mucous membrane of the prosthetic bed can be focal in the form of pinpoint hyperemia or large hyperemic spots and diffuse, often occupying the entire surface of the prosthetic bed. Against the background of an inflamed and edematous mucous membrane, punctate hemorrhages, erosion, as well as hyperplasia of the mucous membrane in the form of its granularity and lobularity may occur. Changes in the mucous membrane can also be observed when using fixed prostheses. Even with a perfectly made crown, the edges of which are immersed in the gingival pocket, the condition of the gums is disturbed - self-cleaning becomes difficult, conditions are created for the retention of food residues.

The course of catarrhal inflammation can be acute or chronic. Acute inflammation lasts 7-14 days and quickly disappears when the irritant is removed. With purulent exudate, the mucous membrane undergoes superficial destruction and erodes. Elimination of the irritant and the treatment carried out lead to a rapid elimination of the lesion. In the absence of treatment, chronic focal purulent inflammation occurs with the formation of a limited focus, consisting of a purulent-molten mass. As a result, a traumatic ulcer is formed.

TRAUMATIC ULTRA. Traumatic ulcers (Fig. 11.1), which are also called decubital, are localized at the sites of injuries, more often on the tongue, lips and cheeks along the line of closing of the teeth. Ulcers are usually painful, especially when eating, talking. More often it is a single ulcer. The clinical manifestation of a traumatic ulcer is diverse and depends on the irritant, the condition of the ulcer, the reactivity of the body, the influence of the oral microflora. With the prolonged existence of the ulcer, its edges and base are compacted (due to the predominance of the phenomenon of proliferation). However, the edges of the ulcer

Traumatic ulcer of the lower lip. Ulcer with infiltrated edges, the bottom is covered with fibrinous bloom.

hyperemic, painful on palpation, the bottom is covered with necrotic plaque. The depth is different - up to the muscle layer. Regional lymph nodes are enlarged, mobile, painful on palpation.

Traumatic ulcers can be complicated by fusospirochetosis or candidiasis, with prolonged course (2-3 months) they can become malignant.

Differential diagnosis. It is necessary to distinguish a traumatic ulcer from A cancer, A tuberculous ulcers, A syphilitic ulcers, A chronic ulcerative necrotizing Vincent's gingivostomatitis, A trophic ulcers.

Removal of the traumatic factor also serves differential diagnostic purposes. Fast, in several days, ulcer healing testifies to its traumatic origin.

A cancerous ulcer is distinguished by a greater density of edges and bases, often keratinization of the edges. After removal of the irritant, it does not heal. With cytological or histological examination, it is possible to detect the atypicality of epithelial cells.

A tuberculous ulcer has more undermined edges, a granular bottom with a yellowish coating, does not epithelize after removal of the stimulus. In the scraping from the ulcer,

epithelioid cells and giant cells of Langhans, and staining according to Ziehl-Nielsen often reveals mycobacterium tuberculosis. The general condition of the patient, as a rule, deteriorates. To clarify the diagnosis, the patient is referred to a phthisiatrician.

A chancre in typical cases differs from a traumatic ulcer by the presence of a dense infiltrate surrounding the ulcer, smooth edges, a smooth bottom, enlargement and compaction of regional lymph nodes (scleradenitis). The diagnosis is confirmed when a pale spirochete is found in the discharge of the ulcer. The Wasserman reaction and other reactions to syphilis become positive 3-4 weeks after the onset of the ulcer. Elimination of the injury does not significantly affect the course of the hard chancre.

A secondary complication of a traumatic ulcer by fusospirochetosis leads to the appearance of a greenish-gray fetid plaque; in scrapings, spirochetes and fusiform bacilli are found in large numbers (see section 11.3.2).

A trophic ulcer differs from a * traumatic ulcer in a more sluggish course, mild symptoms of inflammation (areactive ulcers), a sharp violation of the general condition (most often a disease of the cardiovascular system). The traumatic factor can be very mild, and after its elimination, such ulcers do not tend to heal unless general therapy is carried out.

Treatment. With traumatic ulcers, it comes down to eliminating the irritant, antiseptic treatment of the ulcer and rinsing the mouth. With a sharp soreness of the ulcer, the application of painkillers is indicated. Necrotic tissue from the bottom of the ulcer is carefully removed mechanically under anesthesia or using proteolytic enzymes. Prescribe applications of keratoplastic agents (vitamins A and E; rosehip oil; jelly, ointment solcoseryl, actovegin, etc.). Moxibustion is categorically contraindicated. Perform a thorough sanitation of the oral cavity. Damage with removable dentures, manifested by hyperemia, erosion or ulcers, quickly disappear after the correction of the denture.

HYPERKERATOSIS. Long-term slight irritation from a poorly fixed removable prosthesis can lead to changes in the type of leukoplakia - hyperkeratosis.

LARGE FIBROMA. Sometimes prolonged irritation with the edge of the prosthesis causes long-term

Lobular fibroma in the area of the transitional fold.

chatting fibroids. Erosion or superficial ulceration occurs first. If the injury is not eliminated, then gradually fibrous tissue develops at the base of the ulcer, lobular fibroma grows - a dense, slightly painful tumor-like formation into which the edge of the prosthesis is embedded. A fold with a superficial linear ulcer or erosion is formed at the place of adhesion of the edge of the prosthesis. The ulcer becomes epithelialized if the prosthesis is not used; when wearing a prosthesis, it recurs again. Chronic trauma from the edge of the prosthesis can result in multiple folds parallel to the edge of the prosthesis. Lobular fibroma can exist for several years.

Treatment. It consists in correcting the prosthesis or stopping wearing it; after 2-3 weeks, the lobular fibroma is excised and a new prosthesis is made.

PAPILLOMATOUS HYPERPLASIA. Irritation of the oral mucosa under a removable denture (more often on the upper jaw) is usually called prosthetic stomatitis (a combination of mechanical, chemical and infectious factors). It manifests itself in the form of hyperemia - diffuse or only around the openings of the ducts of the small salivary glands in the back of the palate. With prolonged wearing of a low-quality removable prosthesis against the background of diffuse hyperemia, papillomatous hyperplasia develops - a soft granular bright red surface. The bone tissue of the alveolar process is resorbed in places, the alveolar edge becomes soft, mobile ("dangling").

Rice. 11.3. Papillary hyperplasia of the mucous membrane of the palate.

Angular cheilitis often develops at the same time. In the occurrence of such conditions, in addition to chronic trauma, a certain role is played by the effect of the fungus of the genus Candida, which, as a rule, is found in large quantities on prostheses and less on the mucous membrane of the prosthetic bed. It should also be remembered about the possibility of an allergic reaction of the oral mucosa to the material of the base of the prosthesis (usually acrylic plastic).

Treatment. It consists in stopping wearing a defective prosthesis and making a better one; if multiple Candida is found, antifungal treatment is indicated. Careful care of dentures, brushing them with a toothbrush with powder or paste is of great importance. At night, it is recommended to put dentures in weak alkaline solutions (2% borax or sodium bicarbonate solution).

HABITATIC BITTING OF THE MUCOSA. Habitual biting or sucking on the cheeks, lips, and less often on the tongue is a common occurrence among neuropathies, mainly of young age (senior schoolchildren and students). It may not be noticed by the sick or be conscious. The habit of constantly biting the mucous membrane leads to chronic damage of a peculiar kind, localized along the line of closing of the teeth and adjacent areas accessible to biting. The mucous membrane swells, has a whitish, macerated, flaky surface in the form of spilled spots or large, indistinctly limited areas. The epithelium is unevenly desquamated, has a fringed appearance due to multiple small patches of epithelium, which give the affected surface an unevenness (the surface is, as it were, eaten away by moths). This altered epithelium is removed by scraping. Usually there is no pain, in severe cases painful erosion may occur. Pathohistologically, the phenomena of parakeratosis are found.

Differential diagnosis. This kind of change 482

mucous membrane should be differentiated from A leukoplakia, A chronic candidiasis, A white spongy nevus, A leukoedema.

The cessation of biting leads to the disappearance of all pathological changes in a short time. In scrapings from lesions, fungi of the genus Candida are not found.

White spongy nevus differs in that it manifests itself in early childhood and then progresses, often has a family character; the mucous membrane of the cheeks looks thickened, deeply folded and spongy.

Treatment. It consists in stopping cheek biting.

Chemical damage

Chemical damage (trauma chymicum) can be both acute and chronic. Acute injuries occur as a result of exposure to the mucous membrane of chemicals in a highly damaging concentration. Most often this happens with an accidental injury at home, at work, or attempting to commit suicide, as well as during an appointment with the dentist. Recently, dentists have abandoned many cauterizing agents, but there are still some potent chemicals in practice that require careful handling. Burns of the oral mucosa can occur upon contact with acids, bases (alkalis), the use of arsenous paste, phenol, formalin, silver nitrate, formalin-resorcinol mixture, when wearing prostheses made of insufficiently polymerized plastic.

A distinction should be made between burns resulting from exposure to high concentrations of chemicals and allergic reactions such as plastics, amalgam, low concentrations of chemicals. A burn and an allergic reaction can be combined. Sometimes patients, trying to get rid of toothache on their own, apply alcohol, acetylsalicylic acid and other substances to the tooth, and along the way to the mucous membrane, which leads to burns.

The clinical picture. In acute lesions, sharp pain occurs, as a rule, immediately and is localized in the places where the chemical has entered. The clinical picture depends on the nature and amount of the damaging substance, the time of action.

Acid burns lead to the appearance of coagulation necrosis on the mucous membrane - a dense film of brown (from sulfuric acid), yellow (from nitric acid) or white-gray (from other acids) color. Films are located

gnawed against the background of pronounced inflammation with edema and hyperemia, are tightly connected to the underlying tissues.

A burn with bases (alkalis) leads to colliquation necrosis of the mucous membrane, while a dense film does not form, the necrotic tissues have a gelatinous consistency. The lesion is deeper than with an acid burn. Alkaline burn necrosis can involve all layers of soft tissue, especially on the gums and hard palate. Burns, especially extensive ones, cause severe suffering to the patient. A few days after the rejection of necrotic tissue, slowly healing erosive or ulcerative surfaces are exposed. In mild cases, alkali burns can only cause a catarrhal inflammatory process.

Treatment. After contact of a chemical substance on the mucous membrane, it is necessary to remove it as quickly as possible. If such a burn occurs in front of a doctor, you should immediately start rinsing the oral cavity (rinsing, irrigation) with a weak solution of a neutralizing substance. If the doctor does not have it, then they rinse with water, and then quickly prepare the necessary neutralizing solution. For acid burns, soapy water, 1% lime water, burnt magnesia with water, 0.1% ammonia solution (15 drops per glass of water) are used. Alkalis are neutralized with 0.5% solutions of citric, acetic acids (1/4 teaspoon of acid per glass of water), 0.1% solution of hydrochloric acid (10 drops per glass). Thus, it is possible to stop the further penetration of the chemical into the tissues. For arsenous acid burns, see section 7.6.

To reduce the absorption of concentrated solutions of silver nitrate, a 2-3% solution of sodium chloride or Lugol's solution is used, while insoluble silver compounds are formed. In case of a phenol burn, the mucous membrane is treated with 50% ethyl alcohol or castor oil.

Further treatment of patients with chemical burns is carried out according to the type of therapy for an acute nonspecific inflammatory process: pain relievers are used (1%

trimecaine solution, trimecaine with hexamethylenetetramine, 1-2% lidocaine solution, etc.), weak solutions of antiseptic agents in the form of oral baths and rinses, preparations accelerating epithelization (1% citral solution in peach oil, vitamins A and E, aevit, rosehip oil, sea buckthorn, etc.). Recommended

There is a non-irritating high-calorie mashed food, cold (ice cream), inside multivitamins.

With extensive scars, surgical intervention is indicated.

For chronic exposure to chemicals see section 11.2.4.

Physical damage

Acute injuries arise from burns of the mucous membrane with hot water, hot steam, fire (thermal injury), electric current, with short-term exposure to large doses of ionizing radiation. Burns with hot steam or water initially cause a sharp pain, the intensity of which then quickly decreases, a feeling of roughness of the mucous membrane appears. Acute catarrhal stomatitis develops. The epithelium of the oral mucosa is partially or completely macerated; after a severe burn, it sloughs off in layers or bubbles appear that quickly burst, erosion or superficial ulcers are found under them.

Treatment. It is carried out in the same way as for nonspecific acute inflammatory processes.

Prevention. Prevention of such lesions consists in carrying out sanitary and educational work, observing safety measures.

Lecture number 2

Topic: Changes in OSR in infectious diseases. Viral diseases. Necrotizing ulcerative stomatitis of Vincent. Tuberculosis, candidiasis, syphilis, AIDS.

Purpose of examination methods

1.1. Technological models for education

Lesson time 80 minutes	Number of students
Activity type	Introduce lecture news
Lecture plan 1. Viral disease: A) herpes simplex B) shingles 2. Vincent's ulcerative necrotizing stomatitis 3.Tuberculosis 4.syphilis 5.speed 6.candidiasis	Familiarization with viral diseases .. Herpes simplex. There are 2 types: A) acute herpetic stomatitis B) chronic recurrent herpes A) acute herpetic stomatitis: Herpes simplex virus is visiting. The incubation period is 3-4 days. At the beginning of the disease, there is an increase in temperature, malaise, the appearance of blisters, and ends with the appearance of erosion. Cytology: Multinucleated giant cells, neutrophils, histocytes, epithelial cells appear.

	Histological examination: the bladder is inside the epithelium, Acantholysis is noted. Dif. diagnostics: with foot and mouth disease, herpangina, MEE, allergies.
Objective of the lesson	The basis of the disease is necrotizing ulcerative stomatitis of Vincent. Tuberculosis, candidiasis, syphilis, AIDS.
Teaching methods	Conversation, lecture visuals
Activity type	common-collective
Visual aids on the topic	Study guide, lecture material, projector, computer
The setting for the lesson	Methodical equipped classroom
Monitoring and evaluation criteria	Oral survey

1.2. Technological map of lectures

Stages of work	**Teacher**	**Student**
Preparation stages (10 minutes)	1.Objective of the lesson 2. Preparation of slides for the lecture material 3.Literature on the topic 1. Borovskiy E.V. Danilevsky N.F. Diseases of the oral mucosa. Atlas - M., 1991. - 320 p. 2. Borovskiy E.V. Mashkillayson A.L. Diseases of the mucous membrane of the mouth and lips. M .: Medpress, 2001. 3. Diseases of the oral mucosa. Ed. L.M. Lukinykh. - Novgorod, 2001.	**Listens and records**
1. Introduction (15 minutes)	***1. Purpose and objectives of the lecture material:*** ***Purpose: Familiarization of students with changes in OSR in infectious diseases.*** ***preliminary diagnosis and early preparation of the patient*** ***Task:*** • ***Familiarization of students with etiology, pathogenesis, differential diagnosis of diseases of oral mucosa,*** • ***Explain the morphological elements.*** • ***Dif. diagnosis of infectious disease*** • ***Development of a treatment and prevention plan.***	**Listen** **Answers students' questions**

	2.Questions on the topic ***• Viral disease of oral mucosa*** ***• Differential diagnosis and clinic of herpes simplex.*** ***• Differential diagnosis and clinic of herpes zoster.*** ***• Treatment of viral disease.*** ***• Clinic of ulcerative necrotizing stomatitis.*** ***• Etiopathogenesis of tuberculosis.*** ***• The appearance of syphilis in the OAS.*** ***• Clinic and differential. diagnosis of AIDS*** ***• Clinic and differential. diagnosis of candidiasis.***	
2 main stage (55 minutes)	1. Introduction to the topic with slide show	Listen and record Listen
Final step 10 minutes	1. Conclusion. 2. Independent work. 3. Homework.	Listens and records Writes down

Text

HIV infection in the oral cavity

The human immunodeficiency virus (HIV) is the causative agent of an infectious disease called HIV infection and manifested by the acquired immunodeficiency syndrome (AIDS), which is expressed in a decrease in the general resistance of patients to opportunistic microorganisms and an increased tendency to cancer.

Human Immunodeficiency Virus (HIV) belongs to a group of retroviruses, so named because they possess an enzyme, reverse transcriptase, that allows information to be transmitted from RNA to DNA. HIV infects T4 lymphocytes and causes them to die. As a result, the system of cellular immunity is disrupted, and the organism affected by the virus loses its resistance to opportunistic microflora, becomes susceptible to a number of nonspecific infections and tumors, i.e. AIDS develops.

The source of infection is an HIV-infected person, or a virus carrier. HIV-infected persons include everyone (regardless of their clinical condition) infected with the virus, as evidenced by positive serological tests: immunosorbent assay (ELISA), confirmed by immunoblotting (weight-

turn blot), an immunofluorescence reaction or radioimmunoassay and virus isolation.

After HIV infection, 20% of infected people develop AIDS during the first 5 years, and approximately 50% within 10 years.

In people infected with HIV, the pathogen is found in various biological fluids (blood, semen, vaginal secretions, breast milk, saliva, lacrimal fluid, sweat, etc.). However, infection is transmitted only through blood, semen, vaginal secretions and breast milk.

There are 3 known transmission routes of HIV infection: sexual, parenteral and perinatal.

Most often, HIV is transmitted sexually. The infection can be passed from an infected person to their sexual partner. Parenteral infections are transmitted through transfusions of infected blood or blood products, or through the use of needles, syringes, dental or other instruments to pierce the skin or oral mucosa if these items are contaminated with blood. Transmission of HIV from a woman to a fetus or breastfeeding baby can occur before, during, or shortly after birth (through breast milk).

Despite the fact that HIV was also isolated from other biological fluids (saliva, lacrimal fluid, etc.), there is currently no data indicating other routes of infection transmission (respiratory, contact, food, etc.).

Persons infected with HIV are infected for life. Most of them, at least for several years, may not develop any symptoms or signs of illness, and therefore, they will not be aware of the infection. During this time, they are the source of transmission of the virus to other people.

The clinical signs and symptoms of HIV infection are many and varied. The course of HIV infection can be divided into 4 stages, although not all of them are necessarily observed in every infected person.

The acute period of HIV infection can develop as early as one or several weeks after infection, when there is still no seropositive response to HIV, which is usually noted 6-12 weeks after infection. Typical clinical manifestations of this period are fever, lymphadenopathy, night sweats, skin rashes, headaches, and cough.

In / stage, the disease is either asymptomatic,

or persistent generalized lymphadenopathy (PGL) develops, characterized by an increase in lymph nodes in diameter by more than 1 cm in two or more places outside the groin. This state lasts for about 3 months. As the disease progresses, persistent generalized lymphadenopathy slowly reverses.

Stage II of the disease is referred to as the AIDS-related complex (AIDS-related complex). This complex is characterized by the following signs and symptoms: weight loss, diarrhea, malaise, fatigue, drowsiness, loss of appetite, night sweats, neurological changes leading to memory loss, and peripheral neuropathies. These symptoms and signs often appear intermittently. This stage is also characterized by the presence of typical lesions of the skin and oral mucosa, such as hairy leukoplakia, shingles, exacerbation of chronic recurrent herpes on the oral mucosa and genitals.

Stage III is called the "intermediate stage of the disease." Clinical manifestations in stage III often occur some time after the onset of early symptoms, but before the full expanded picture of AIDS. Candidosis, hairy leukoplakia, acute herpetic stomatitis or exacerbation of chronic herpes appear in the oral cavity at this stage of the disease. Pulmonary tuberculosis, alveolar pneumonia may develop.

At the IV stage of the disease (late stage of the disease), the severe clinical picture of AIDS fully unfolds. It is characterized by the development of inflammatory processes caused by opportunistic microflora and the formation of tumors such as Kaposi's sarcoma, the appearance of which is associated with a deficiency of cellular immunity caused by HIV. The most typical AIDS infections include protozoal invasions (pneumocystis pneumonia, toxoplasmosis), as well as fungal (systemic candidiasis, cryptococcosis), bacterial (atypical mycobacteriosis, salmonella septicemia) or viral (cytomegaly, papovavirus, etc.). At this stage, especially severe forms of lymphoma,

cachexia and neurological disorders (neuropathy, memory loss, dementia) may also appear.

The first manifestations - "signs" indicating the introduction of HIV, which has a destructive effect on the immunological protection in the body, may be lesions of the oral mucosa.

Diseases of the oral mucosa associated with HIV infection include: A. Various clinical forms of candidiasis. A Viral infections. A Hairy (villous) leukoplakia. A. Necrotizing ulcerative gingivostomatitis. A Progressive form of periodontitis (HIV-periodontitis). And Kaposi's Sarcoma.

Diseases that should alert the dentist regarding HIV infection of the patient include squamous cell carcinoma of the oral mucosa, lymphoma (non-Hodgkin's).

Candidiasis. It is the most common intraoral sign of HIV infection. The most characteristic is acute pseudomembranous candidiasis. In HIV-infected people, it lasts for a long time (months), so the term "acute" loses its meaning. Clinical manifestations are fully consistent with the usual candidiasis. On the mucous membrane of the mouth there is a grayish-white coating, reminiscent of curdled masses. The plaque is easily removed with a spatula; under it, a hyperemic mucous membrane of the mouth is found. It is localized mainly on the mucous membrane of the cheeks, tongue, floor of the mouth, palate, gums. Candidiasis should be distinguished visually from similar changes in leukoplakia, lichen planus.

Acute atrophic candidiasis resembles lesions of the oral mucosa with allergies, hypovitaminosis C, B, B2, B6. The defeat is usually localized in the tongue in the form of an area of hyperemia located along its midline; filiform papillae are atrophied.

Less common is chronic hyperplastic candidiasis, which is usually localized on both sides on the mucous membrane of the cheeks, hard and soft palate. When the lesion is localized in the corners of the mouth, candidal angular cheilitis and cracks with pronounced manifestations of epithelial hyperplasia often occur. Cracks grow in size over time, become hard, and crust over. Painful sensations in patients may be absent or minor painful sensations, a burning sensation are possible.

The differential diagnosis of candidiasis should take into account the symptoms of similar diseases: A leukoplakia; And lichen planus; And allergic stomatitis; And traumatic injuries.

Diagnosis of candidiasis is based on typical clinical signs and the results of a bacterioscopic examination of scrapings from the affected oral mucosa. The presence of multiple fungus of the genus Candida in the form of spores and hyphae always confirms the diagnosis of candidiasis.

Viral infections. HIV-infected patients are characterized by lesions of the oral mucosa with herpes simplex, which occurs in the form of frequent and painful exacerbations of recurrent herpetic stomatitis. Along with intraoral lesions of the genital organs are often noted. The lesions are atypical in localization, long-lasting and painful. Vesicles appear on the tongue, soft palate, floor of the mouth, on the lips, very quickly transform into erosion, which often turn into ulcers of large sizes (0.5-3 cm in diameter). Ulcers can take the form of a crater with raised irregularly shaped edges and a bright hyperemic bottom, which can be covered with a grayish-white coating. In the absence of timely treatment, these lesions progress, their size increases, which can

subsequently lead to dissemination of the viral herpes infection into the visceral organs and increase the severity of the clinical course, and sometimes even cause death.

The mucous membrane of the mouth of HIV-infected patients is also quite often affected by herpes zoster and viral warts (papillomas). They appear as nodular lesions covered with multiple papillary projections. Localized most often on the mucous membrane of the gums, hard palate.

Hairy leukoplakia. Hairy leukoplakia (villous leukoplakia, flat candidoma, oral viral leukoplakia) is observed in HIV-seropositive patients and is a lesion associated with immunodeficiency and HIV infection. The presence of hairy leukoplakia is an indicator for HIV testing. According to the latest literature data, 98% of the examined patients with hairy leukoplakia were HIV seropositive.

Manifestations of hairy leukoplakia can vary in size and localization, they are one- or two-sided. Clinically, hairy leukoplakia appears as white folds or villi that rise above the surface of the oral mucosa, resembling hair in shape. These villi are tightly fixed on

surfaces of the mucous membrane and it is impossible to scrape them off. Hairy leukoplakia is localized more often on the lateral surfaces of the tongue, but can spread to its entire posterior surface; less often it can manifest itself on the mucous membrane of the lips, cheeks, floor of the mouth and palate.

Differential diagnosis of hairy leukoplakia is carried out with contact allergic reactions to dental materials, true leukoplakia and smokers' leukoplakia, lichen planus, hyperplastic candidiasis, lesions of the oral mucosa in the event of galvanism.

Necrotizing ulcerative gingivostomatitis. A sign of HIV infection in the oral cavity is necrotizing ulcerative gingivostomatitis. The disease begins acutely, suddenly or imperceptibly with bleeding gums when brushing teeth. The acute course of necrotizing ulcerative gingivostomatitis can last 3-4 weeks, after which the disease in many patients becomes chronic and often recurs. For HIV-associated necrotizing ulcerative gingivostomatitis, a progressive course is characteristic, leading to the loss of gum tissue and bone structures, often with sequestration.

In HIV-infected patients, an aggressive form of periodontitis with generalization of the process in the periodontal tissues can be observed. Severe bleeding and hyperplasia of the gums are noted, the mobility of the teeth increases, diffuse destruction of the bone tissue of the alveolar process occurs, which is often accompanied by sequestration of the interdental septa.

Kaposi's sarcoma. It is one of the most characteristic clinical manifestations of AIDS. The tumor forms from the lymphatic vessels. Most often it affects the skin of the extremities. Kaposi's sarcoma in HIV infection as an initial symptom is described in 30% of cases. It is more common in whites and homosexuals than in blacks and drug addicts.

In the oral cavity, in most of the described cases, Kaposi's sarcoma is localized in the palate, less often on the mucous membrane of the gums. It has the appearance of bluish, reddish or black spots, flat in the initial stages. Subsequently, they darken, increase in size, often divide into lobes and ulcerate. Ulceration on the oral mucosa is much more common than on the skin. The lesions in the oral cavity are painful to the stage of ulceration. Kaposi's sarcoma, localized on the gums, resembles epulis in appearance.

When diagnosing HIV infection, along with the described symptoms in the oral cavity, it is necessary to take into account other signs of the disease that complement the clinical signs of immunodeficiency: lymphadenopathy, pneumocystis pneumonia, unreasonable weight loss, damage to internal organs and systems, including the nervous system.

When collecting anamnesis, attention should be paid to past and concomitant diseases, the use of corticosteroids, cytostatics and other drugs that could cause disorders of the immune system. It is very important to take into account the professional characteristics of the patient, the nature of sexual intercourse, especially among persons on business trips abroad.

The diagnosis of HIV infection can be made in the laboratory by detecting specific antibodies to the virus. The most commonly used immunosorbent assay (ELISA), as well as Western blot or indirect immunofluorescence. A certain role in the diagnosis of HIV infection is assigned to the study of the immunological status, since in HIV-infected patients the ratio of T-helpers to T-suppressors decreases, the number of leukocytes and lymphocytes decreases, the content of immunoglobulins, especially A and G. However, increases. also change with secondary immunodeficiencies of various etiologies, which reduces their importance in the diagnosis of HIV infection.

Prevention of HIV infection. An essential part of effective day-to-day HIV prevention is the application of traditional sterilization and disinfection principles. To prevent the transmission of HIV, it is more than enough to comply with the recommendations for the prevention of infection with infectious agents transmitted with blood, such as the hepatitis B virus.

The fight against HIV infection involves:

precautions for blood and other body fluids;

precautions for injections and procedures involving skin piercing;

effective sterilization and disinfection.

Because most dental procedures are invasive, they often lead to bleeding, and HIV is a blood infection, which puts the dentist at risk for HIV infection. Therefore, dentists, middle and junior 528

medical personnel must comply with precautions with regard to blood and other body fluids (pus, saliva). Hands and other body parts contaminated with blood or other body fluids should be thoroughly washed with soap and water. After removing protective gloves, hands should also be washed immediately. Gloves should be worn for all direct contact with blood and other body fluids. During procedures in which blood splashes or suspensions may form (for example, dispersion of aerosols from high-speed dental units and ultrasound machines) - eyes, nose and mouth should be protected with a mask and glasses or a plastic shield. Extreme care should be taken when handling needles, scalpels, discs, endodontic and other sharp instruments to prevent punctures, cuts and other injuries. Disposable instruments must be destroyed after use. If injury from sharp instruments is possible (for example, when cleaning them), it is recommended to wear additional heavy gloves and handle the instruments with extreme care. It is advisable to use special ultrasonic installations for mechanical cleaning of small instruments from contamination. Physicians with exudative skin lesions should not perform invasive procedures or treat infected patients.

Reliable sterilization is an important condition for the prevention of HIV infection by means of instruments used for piercing the skin, mucous membranes and other invasive procedures. HIV is very sensitive to conventional high-intensity sterilization and disinfection methods. It is inactivated by methods designed to suppress other pathogens (eg, hepatitis B virus). The most effective method of inactivating HIV is high temperature, therefore one of the most reliable methods for sterilizing instruments (including dental ones) is high-intensity sterilization and disinfection based on heating (boiling, autoclaving, sterilization with dry hot air).

High-intensity disinfection can also be carried out with the help of disinfectants: 2% glutaral (glutaraldehyde) solution, 30% hydrogen peroxide solution. Soaking the cleaned instruments in these solutions for 30 minutes provides a high disinfection rate.

For treatment of medical tables, drills, handpieces and other surrounding surfaces, it is enough to rinse them with a solution of a disinfectant of medium or low intensity, such as chlorine-releasing compounds, such as sodium hypochloride, calcium hypochloride, 4% chloramine solution, sodium dichloroisocyanurate, 4% chlorhexidine solution. The disinfecting ability of these chlorine-releasing compounds is measured by the presence of "active" chlorine in them (in percent). For example, sodium hypochloride contains 5% "active" chlorine, calcium hypochloride - 70%, chloramine - 25%.

For the treatment of body tissues, bactericidal agents are used - antiseptics (70% ethyl alcohol, 70% isopropyl alcohol, 1% iodine solution, 10% iodoform solution).

Disinfection of dental materials is carried out by cleaning them from saliva and blood (impression material, material for determining the bite, etc.). Impressions and intraoral orthodontic and orthopedic structures should be cleaned and disinfected before being sent to the laboratory.

After use, the handpieces for drills are rinsed with a strong jet of water, detergent to remove adhering material. Then they are wiped with a material soaked in a disinfecting detergent. The chemical is washed off with distilled water.

Necrotizing ulcerative stomatitis of Vincent

Necrotizing stomatitis of Vincent (stomatitis ulceronecroticans Vincenti) is an infectious disease caused by fusiform sticks (Bacillus fusiformis) and Borellia vincentii.

Described under various names: ulcerative gingivitis and ulcerative stomatitis, ulcerative membranous stomatitis, fusospirochetous stomatitis, Plaut-Vincent stomatitis, "trench mouth", ulcerative membranous stomatitis, etc. According to the modern classification, the disease is called "ulcerative necrotizing stomatitis of Vincent" , or "Vincent's stomatitis". In the case of gum disease, the disease is defined as Vincent's gingivitis; with simultaneous damage to the gums and other areas of the mucous membrane - Vincent's stomatitis, and with localization of the process in the area of the palatine tonsils - Simanovsky-Plaut-Vincent's angina.

Etiology. Vincent's ulcerative necrotizing stomatitis is caused by the symbiosis of the fusiform bacillus and Vincent's spirochete. Under normal conditions, these microorganisms are representatives of the resident microflora of the oral cavity and are detected in small numbers in all people with teeth. They are found mainly in the gingival groove, periodontal pockets, carious cavities, crypts of the tonsils. In the unsanded oral cavity, with its poor hygienic condition, as well as with periodontitis,

the number of fusobacteria and spirochetes increases sharply. The development of Vincent's ulcerative necrotizing stomatitis is associated with a sharp decrease in the body's resistance to infection, which can occur as a result of past viral diseases (acute respiratory infections, herpetic stomatitis, pneumonia, etc.), vitamin deficiencies, stress, overwork, malnutrition. Necrotizing ulcerative gingivitis often complicates the course of severe general diseases (leukemia, agranulocytosis, pneumonia, infectious mononucleosis). It can occur as a complication of erythema multiforme exudative, erosive allergic stomatitis. In case of violation of specific and nonspecific defense mechanisms in the body, the virulence of fusobacteria and spirochetes increases. Their number increases to such an extent that they become dominant in comparison with other microflora. A decrease in the general resistance of the body negatively affects the resistance of the oral mucosa - it cannot play the role of a reliable barrier on the path of infection, and the violation of its integrity, which always occurs in the unsanded oral cavity, due to the presence of local traumatic factors (sharp edges of the teeth, dentures, tartar deposits, etc.) creates conditions for the introduction of fusobacteria and spirochetes. Therefore, Vincent's ulcerative necrotizing stomatitis often develops in people with an unsanitary oral cavity and poor hygiene.

Despite the fact that there are known cases of group morbidity with Vincent's stomatitis (in military units, schools, kindergartens), this disease is considered non-contagious, and such cases are explained by similar unfavorable living conditions (malnutrition, lack of vitamins, lack of hygienic measures for caring for the oral cavity and etc.).

The clinical picture. Mostly young people (17-30 years old), mostly men, are ill.

Hypothermia is often a provoking factor in the development of Vincent's ulcerative necrotizing stomatitis, which explains the highest frequency of its occurrence in the autumn-spring period; the maximum incidence occurs in the period from October to December.

Clinically distinguish between acute and chronic course of necrotizing ulcerative stomatitis of Vincent, and in terms of severity - its mild, moderate and severe form.

The disease begins acutely with an increase in body temperature to 37.5-38. Regional lymph nodes increase, thicken, become painful on palpation, retain mobility. Patients are worried about headache, general weakness, soreness of the oral mucosa, aggravated by eating, talking, bleeding gums, hypersalivation, putrid breath.

Catarrhal phenomena on the oral mucosa quickly turn into ulcerative. The process most often begins on the gums, and then moves on to other parts of the mucous membrane. The gums become swollen, hyperemic, sharply painful and bleed when touched. The epithelium of the gum edge and interdental papillae becomes cloudy and then necrotic. As a result, the gingival margin looks like a cut, with uneven notches, its surface is covered with an easily removable grayish-yellow coating. Subsequently, the affected gum edge is not completely restored and remains deformed. Most often and to a greater extent, the area near the lower eighth teeth is affected, where necrosis from the alveolar process quickly spreads to the mucous membrane of the cheek and the retromolar region, causing in some cases trismus and pain when swallowing. Ulcers on the mucous membrane of the cheeks can reach large sizes (up to 5-6 cm in diameter) and depth (Fig. 11.15). Their edges are uneven, soft. The bottom is covered with a thick grayish-greenish necrotic plaque with a putrid fetid odor, after removal of which

the bleeding bottom of the ulcer is exposed. There are no seals in the area of the base and edges of the ulcer. The mucous membrane around the ulcer is edematous, hyperemic. In the presence of local traumatic factors in the oral cavity (roots of decayed teeth, sharp edges of teeth, prostheses), other areas of the oral mucosa (hard, soft palate, arches, tonsils, tongue) may be affected. Oral ulcers can be single or multiple, of various sizes and depths. When the ulcer is localized on the hard palate, necrosis of all layers develops rather quickly.

Vincent's acute necrotizing stomatitis. An extensive ulcer of the buccal mucosa along the line of closing of the teeth in the area of the large molars, covered with necrotic plaque.

mucous membrane and bone is exposed. An isolated lesion of the pharynx (angina Simanovsky-Plaut-Vincent), as a rule, is unilateral, in the practice of a dentist is rare. 2-3 weeks after the onset of necrotizing ulcerative stomatitis usually occurs resolution of the process with complete epithelization of ulcerative surfaces.

In rare cases, when treatment is not carried out or it is ineffective, a chronic form of necrotizing ulcerative stomatitis of Vincent develops, in which there are no general symptoms. Patients are worried about constant bleeding and soreness of the gums, as well as bad breath. The clinical picture is erased, the gum is congestive-hyperemic, edematous, its ulcerated edge is often compacted, necrotic areas are located mainly in the interdental spaces and can be seen at a cursory examination. On careful examination and probing of the gingival margin, exposed bone tissue is determined. Only some teeth have affected areas. Lymph nodes (submandibular, submental) in patients with chronic ulcerative necrotizing stomatitis of Vincent are compacted, slightly painful. With a disease duration of 4-8 months, the lymph nodes acquire a cartilage-like consistency.

The histological picture of Vincent's ulcerative necrotizing stomatitis. A site of tissue necrosis in the surface layers (1), maturing granulation tissue (2), edema and small cell infiltration (3).

In the pathological examination of the areas of the ulcerated edges of the gums, two zones are revealed: superficial - necrotic and deep - inflammatory (Fig. 11.16). In the surface layers of the necrotic gum tissue, an abundant variety of microflora (cocci, sticks, fusobacteria, spirochetes, etc.) is revealed. In deeper layers, fusobacteria and spirochetes predominate sharply (Fig. 11.17). The deeper layers of connective tissue are inflamed: they are edematous, the vessels are dilated. Blood corpuscles are found in the perivascular inflammatory infiltrate. In the same zone of inflammation, only spirochetes that have penetrated between the epithelial cells are found inside the intact tissue.

The cytological picture of scrapings from the ulcerative surfaces of the mucous membrane in patients with necrotizing ulcerative stomatitis of Vincent corresponds to a nonspecific inflammatory process. At the beginning of the disease, an abundance of structureless masses is determined, a sharp predominance of neutrophils, mainly in a state of decay, and erythrocytes (due to severe bleeding). In the second period of the disease, when healing begins, along with disintegrated neutrophils, complete

Syphilis (syphilis, lues) is a chronic infectious disease caused by Treponema pallida. The mucous rim of the mouth is the site of localization of syphilitic rashes in all periods of syphilis.

Distinguish between congenital and acquired syphilis. With congenital syphilis, pale treponema enters the fetus through the placenta from the sick mother. Acquired syphilis develops through infection through the skin and mucous membranes by direct contact (mainly sexual) or through various objects contaminated with secretions containing the pathogen. The source of infection can be medical instruments, including

and dental, in case of violation of the regime of their sterilization. A prerequisite for infection with syphilis is even the smallest damage to the stratum corneum or epithelium of the oral mucosa (cracks, abrasions, etc.), although some authors believe that the pathogen can also penetrate through the intact mucous membrane. Infection with syphilis can also occur when pale treponema gets into the blood directly, for example, during blood transfusions or other medical procedures (injection, during operations, etc.). Medical workers (more often gynecologists and dentists) can become infected when treating patients, conducting gynecological and dental examinations, operations, opening abscesses, periodontal interventions, as a result of careless handling of endodontic and other instruments. The most dangerous source of infection are patients with active manifestations of syphilis in the primary and secondary periods with localization of lesions on the genitals and oral mucosa.

In connection with the undulating course of syphilis, characterized by a change in periods of active manifestations and latent stages of the disease, a variety of clinical and morphological signs that arise at different stages of the disease, four periods are distinguished: incubation, primary, secondary and tertiary.

The incubation period for syphilis lasts on average 3-4 weeks.

PRIMARY SYPHILIS (syphilis primaria Lues I) lasts an average of 6-7 weeks, sometimes 2-3 months without treatment. It is characterized by the appearance at the site of the introduction of pale treponema of a painless inflammatory infiltrate - a hard chancre (primary syphiloma). 5-7 days after the formation of a hard chancre, the second mandatory symptom of the primary period appears - an increase in the lymph nodes closest to the hard chancre, or regional scleradenitis. The lymph nodes are cartilaginous-dense, up to 2-3 cm in diameter, painless, not adhered to the skin and to each other, the skin above them is not changed. By the end of the primary period, the lymph nodes of all groups are enlarged (polyadenitis). In these nodes, an intensive reproduction of treponema occurs. Hard chancre can be single or in the amount of 2-3. Most often it is located on the red border of the lips, the mucous membrane of the tongue, palatine tonsils, less often on the mucous membrane

... Chancre. Saucer-shaped ulcer of the tongue with raised, even edges.

cheeks, soft and hard palate or other areas. The development of a hard chancre on the oral mucosa in typical cases begins with limited hyperemia and induration due to an inflammatory infiltrate that increases in size and usually reaches 1–2 cm in diameter. Then its central part is necrotic and a meat-red color is formed, a painless erosion or a superficial saucer-shaped ulcer of a round or oval shape with raised smooth edges from 3 mm to 1.5 cm in diameter At the base of the erosion or ulcer, a dense cartilaginous infiltrate is palpable. The bottom of the

ulcer is smooth, shiny, meat-red in color without plaque, or covered with a grayish-yellow "greasy" coating.

Often there are atypical forms of hard chancre that differ from the classic described above. In this case, a hard chancre can look like a crack, abrasion, erosion, seizure with a slight infiltration at the base. Its form depends on the localization. On the tongue, a hard chancre due to a pronounced inflammatory infiltrate at the base protrudes sharply above the level of the surrounding tissues and has the shape of a saucer turned upside down. In persons with a folded tongue, the chancre is localized along the folds and has a slit-like shape.

When the hard chancre is located in the corners of the mouth, it clinically resembles seizures with infiltration at the base.

On the gum, a hard chancre has the appearance of a bright red erosion, which adjoins several teeth in the form of a crescent.

When localized on the amygdala, a hard chancre can have one of three forms: ulcerative, angina-like (amygdalitis) and combined (ulcerative against a background of angina-like). The amygdala is affected on one side only. It acquires a copper-red color, is enlarged, dense,

painless. With an ulcerative form, a meat-red ulcer appears on this background, oval in shape with gentle, even edges. The mucous membrane around the ulcer is hyperemic. The process can be accompanied by painful sensations, sometimes significant.

In an unsanded oral cavity with poor hygiene, a hard chancre can be complicated by fusospirochetosis, staphylococcal infection. Due to the development of an acute inflammatory process, the typical clinical symptoms of syphilis are obscured, soreness appears, at the bottom of the ulcer there is a purulent or necrotic plaque, the depth of the ulcer increases.

Diagnosis. The diagnosis of hard chancre should be confirmed by the detection of pale treponema from the surface of the primary syphiloma or in the punctate of regional lymph nodes. If there is a negative result, repeated studies of the hard chancre that is separated from the surface for the presence of pale treponema should be carried out, since its detection is decisive in the diagnosis of primary syphilis. Specific serological reactions (Wasserman reaction, sedimentary reactions) become positive a little later, only 3 weeks after the appearance of a hard chancre. Starting from the 4th week, classical serological reactions (complement fixation reaction, Wasserman reaction, Kahn reaction, Sachs-Vitebsky reaction) and the immunofluorescence reaction become persistently positive. In this regard, the primary period of syphilis is divided into primary seronegative, when the indicated serological reactions are negative, and primary seropositive, which occurs on average 3-4 weeks after the appearance of a hard chancre, when serological reactions become positive and remain so for a long time.

By the end of the primary period, patients may experience general (prodromal) phenomena: malaise, bone pain, fever, headache, increased ESR, leukocytosis.

Differential diagnosis. Primary chancre should be differentiated from traumatic ulcers, in which there is no compaction at the base characteristic of syphilis. In addition, the traumatic ulcer is quickly eliminated after the cause is eliminated.

Chancre, localized on the amygdala, differs from angina in one-sided lesion, absence of pain, acute inflammatory hyperemia and general phenomena.

When the chancre is localized on the lip, differential diagnosis should be carried out with erosion that occurs with recurrent herpes. In the case of herpes, unlike syphilis, erosion is located on a slightly edematous, hyperemic base, is painful and has scalloped outlines. Herpetic erosion is also characterized by a rapid course (rapid onset and rapid epithelialization). In addition, with recurrent herpes, erosion is preceded by the formation of bubbles, which never occurs with syphilis.

Chancre may resemble chancriform pyoderma, which is characterized by the presence of inflammation, purulent discharge from the surface, pain and duration of existence.

Differential diagnosis of hard chancre and severe recurrent aphthous stomatitis (Setton's aft) is carried out. The differences lie in the sharp soreness of the ulcer or aphthae in recurrent aphthous stomatitis, a pronounced inflammatory reaction of the mucous membrane, as well as the history of the recurrent nature of the disease.

Chancre may resemble a decaying cancer. When carrying out differential diagnosis, it should be borne in mind that the infiltration of a cancerous tumor is located much deeper than the primary syphiloma. The edges of the cancer ulcer are very dense, uneven, the bottom bleeds easily. Decisive for the diagnosis are the results of laboratory tests - the detection of atypical cells in cancer and treponema pale in syphilis.

SECONDARY SYPHILIS (syphilis secundaria, Lues II) lasts an average of 3-4 years. There is a generalization of the process with damage to the mucous membranes in the form of a characteristic rash (syphilis), disturbances in the general condition of the body (malaise, weakness, subfebrile body temperature, headaches, etc.), possible damage to internal organs, bone and nervous systems. The mucous membrane of the mouth is one of the favorite places of localization in this stage of syphilis, both secondary fresh and recurrent. Manifestations on the oral mucosa occur simultaneously with skin manifestations, but they can also be isolated. The secondary period of syphilis is characterized by high infectivity, focus of the location of the elements, frequent absence of subjective sensations, true or false polymorphism. At this stage of the disease in the oral cavity, there are mainly two types -

lesions: macular (macular) and papular syphilides.

Spotted syphilis is most often localized on the palatine arches, soft palate and tonsils. It is characterized by the appearance of red, sharply limited spots, which tend to merge into a copper-red erythema, clearly delimited from the unchanged mucous membrane. Without treatment, these changes last for about 1 month.

Erythematous syphilitic sore throat should be distinguished from the banal catarrhal sore throat, in which there is pain, fever, swelling, soreness. Differential diagnosis is also carried out with allergic medicinal lesions of the oral mucosa, which differ from erythematous syphilitic sore throat in the prevalence of lesions - not only the arches and tonsils are involved, but also the cheeks, tongue and other areas. Medicinal catarrhal stomatitis is accompanied by burning, soreness of the oral mucosa. All symptoms quickly disappear after stopping the intake of the medicinal substances that caused them.

Papular syphilis is the most common form of manifestation of secondary syphilis in the oral cavity, especially its recurrent stage. Papular rashes can form anywhere in the mucous membrane, but most often occur on the tonsils, soft palate and arch, where they often merge into large plaques. Papules are dense, rounded, elements 1 - 1.5 cm in diameter. They are painful, delimited from the unchanged mucous membrane. Due to constant maceration in the oral cavity, the papules acquire a grayish-white color and have a tendency to peripheral proliferation. Merging into plaques, they sometimes rise above the level of the oral mucosa. When scraping with a spatula, the plaque covering the papule is removed and meat-red erosion is exposed under it. There is an inflammatory rim around the papule. Papules and discharge from the erosion surface contain a large number of treponemas.

On the back of the tongue, in places where papules appear, filamentous and mushroom papillae disappear and elements resembling foci of desquamation appear, smooth, shiny, sinking below the level of the mucous membrane ("sloped meadow plaques") In the unsanded oral cavity, with poor hygienic care, papules can ulcerate or become complicated fusospirochetosis. In this case, the clinical picture becomes similar to stomatitis.

Syphilitic papules. Smooth shiny plaques on the tongue due to atrophy of filiform and mushroom papillae.

and Vincent's angina, there is significant soreness and an area of hyperemia around the papules. When papules are localized in the corners of the mouth, due to constant injury, they can grow and spread to the skin. When the mouth is opened, the infiltrated skin and mucous membrane are injured, forming deep, bleeding, painful cracks covered with crusts against the background of a dense infiltrated base.

Syphilitic papules on the oral mucosa should be differentiated from leukoplakia, papules of lichen planus and lupus erythematosus, the surface of which is also grayish-white. However, if a whitish plaque is quite easily removed from the surface of a syphilitic papule by scraping, then with leukoplakia, lichen planus and lupus erythematosus, it cannot be removed, since it is a consequence of hyperkeratosis. In addition, with leukoplakia, papules with lichen planus and foci of hyperkeratosis with lupus erythematosus, there is no infiltration at the base, and there is no inflammatory rim along the edges of the focus.

Syphilitic papules differ from candidiasis in that after removing a grayish-white plaque from their surface, erosion is exposed, in the discharge of which pale treponemas are found. Scraping off the same plaque in candidiasis exposes the hyperemic surface of the mucous membrane, sometimes eroded.

In some cases, papular syphilis resembles exudative erythema multiforme and allergic stomatitis. In these diseases, there is no infiltration at the base of erosion, the oral mucosa is hyperemic, diseases are often accompanied by a violation of the general condition and pronounced subjective sensations.

Syphilitic papules on the dorsum of the tongue should be distinguished from desquamative glossitis. The bright red color of the desquamation sites, the presence of a white border along the periphery, the absence of compaction at the base, the migration of desquamation foci and the chronic course of the disease distinguish desquamative glossitis from syphilitic lesions.

Sometimes it is necessary to carry out differential diagnosis of syphilitic papules with recurrent aphthous stomatitis. The presence of a yellowish-gray plaque on the surface of the aphtha, surrounded by a corolla of hyperemia, severe pain and recurrent nature of the disease make it possible to distinguish aphthae from syphilitic papules.

Diagnosis. Along with the clinical picture, the diagnosis of the secondary period of syphilis should be confirmed by the detection of pale treponemas in scraping from the surface of the papules and positive serological reactions (Wasserman reaction, sedimentary reactions, the reaction of immobilization of pale treponemas).

TERTIARY SYPHILIS (syphilis tertiaria, Lues III) begins 4-6 years after the onset of the disease. It can last for decades. At this stage, gum and lumpy eruptions may appear on the mucous membrane of the mouth and on the skin.

Gummy syphilide can be localized anywhere in the oral mucosa, but more often on the soft and hard palate, as well as on the tongue. Usually a single gum appears in the thickness of the mucous membrane of the mouth, a painless node is formed, which gradually increases, reaching large sizes (3 cm or more in diameter). Then the center of the gum is opened, a deep crater-shaped ulcer with a necrotic core in the center arises. The ulcer is painful, surrounded by a dense infiltrate, dense, even edges, the bottom is bright red, covered with small granulations. The healing of the ulcer leads to the formation of a retracted stellate scar. This process lasts 3-4 months and is almost not accompanied by subjective sensations. Less often, gum dissolves or turns into a dense fibrous node. Depending on the localization of the gum, its clinical picture has certain features. Gummy lesions of the tongue can occur either in the form of the formation of separate gummas (nodular glossitis), or in the form of diffuse sclerosing glossitis, which is the most severe form of lesions of the oral mucosa in syphilis. Initially, there is a diffuse infiltration of the thickness of the tongue, mainly by plasma cells, as a result of which the tongue increases, and no nodes are formed. Tongue tight, papillae

... Syphilitic gumma. Large knot in the thickness of the tongue.

smoothed. Subsequently, the infiltrate is replaced by scar tissue, gradually tightening the tongue, which leads to its reduction. On the back of the tongue, very painful grooves, cracks, trophic ulcers form, the edges of which can become malignant. The tongue becomes very dense, its mobility is limited. There are also combinations of sclerosing glossitis with gum.

When localized on the hard palate, bone tissue and periosteum are very quickly involved in the process, bone necrosis, its sequestration, extensive defects, perforation of the hard palate occur.

Diagnostics. It is rather difficult to detect pale treponema in the discharge of tubercles and gummas in the tertiary period of syphilis. Therefore, the immunofluorescence reaction (RIF) and the treponema pallidum immobilization reaction (PALT), which are positive in 100% of cases, are of decisive importance in the diagnosis of tertiary syphilis. Wasserman's reaction and sedimentary reactions in the tertiary period of syphilis are positive in 50-80% of patients.

Differential diagnosis. Syphilitic gummas

should be differentiated from tumors and miliary ulcerative tuberculosis.

Tuberous syphilis on the oral mucosa is less common than gummy. Bumps, as well as gum, can appear anywhere in the oral mucosa, but are more often localized on the lips,

alveolar ridge and in the palate. Tuberous syphilis on the oral mucosa is painless, dense tubercles of a bluish-red color. They tend to be grouped, then disintegrate rather quickly, leading to the formation of ulcers. After the ulcers heal, rough scars form. For tubercular syphilis on the oral mucosa, a relatively rapid course is characteristic - usually several months from the onset to the formation of a scar.

Regional lymph nodes in tertiary syphilis may not respond to the process in the oral cavity and give a nonspecific response to the addition of a secondary infection.

The differential diagnosis of tubercular syphilis is carried out with tuberculous lupus, which is characterized by a much slower development, calculated in years; soft consistency lupom. Ulcers that form during the disintegration of tubercles in tuberculous lupus have uneven, soft edges.

Treatment. Treatment of patients with syphilis is carried out in specialized venereal medical institutions.

Tuberculosis is a chronic infectious disease caused by Mycobacterium tuberculosis (Koch's bacillus).

Tuberculous lesions of the mucous membrane of the mouth and the red border of the lips are usually secondary and occurs when there is a tuberculous process in the lungs, lymph nodes or bones. Mycobacterium tuberculosis gets to the mucous membrane of the mouth most often by hematogenous or lymphogenous route from the primary tuberculous focus. The mucous membrane of the mouth, due to its pronounced resistance, is an unfavorable environment for the reproduction of mycobacterium tuberculosis. As a rule, they quickly die on its surface. Primary tuberculosis in the oral cavity of adults practically does not occur, it is extremely rare in young children.

On the mucous membrane of the mouth and the red border of the lips, the second

Typical tuberculosis infection usually manifests itself in the form of tuberculous lupus and miliary ulcerative tuberculosis. Scrofuloderma (colonic tuberculosis) is extremely rare.

Lupus erythematosus (lupus vulgaris, tuberculous lupus). This is the most common disease among tuberculous lesions of the oral mucosa and the red border of the lips. In tuberculous lupus erythematosus, lesions of the oral mucosa are usually combined with skin lesions. The favorite localization of tuberculous lupus is the skin of the face, which is affected in about 75% of patients. Very often, the red border of the upper lip is affected as a result of the spread of the process from the skin of the nose. Subsequently, the mucous membrane of the mouth is often involved in the process. Sometimes there is an isolated lesion of only the red border of the upper lip. Selective lesion only of the oral mucosa is rare. The lupus process can affect any part of the oral mucosa or the red border of the lips, but its most typical localization is on the mucous membrane of the upper lip, the alveolar process of the upper jaw in the region of the front teeth, hard and soft palate. The primary element of this form of tuberculosis is a tubercle or lupoma, which is a limited, initially flat, painless formation the size of a pinhead (diameter 1-3 mm), soft, red or yellowish-red in color.

The tubercles are prone to peripheral growth and fusion with neighboring elements, which leads to the formation of plaques of different sizes and outlines, in which individual tubercles are distinguished. The center of the plaque breaks down rather easily, turning into an ulcer with soft, pitted edges. The ulcer is superficial, slightly painful, the bottom is covered with a yellowish or yellowish-red bloom, in the

presence of growing granulations - resembling raspberries. Characteristic of tuberculous lupus are the apple jelly symptom and the probe test. When pressed on any microscope slide, the red color due to perifocal vasodilation disappears, and the primary elements of the lupa become visible - yellowish-brown or waxy nodules resembling apple jelly in color (a symptom of apple jelly). If you press on anybody with a bulbous probe, then it easily falls through (Pospelov's symptom), which is mainly due to the destruction of elastic and collagen fibers in the tubercle.

When the process is localized on the mucous membrane of the alveolar process, the bone tissue of the interdental septa is destroyed, the teeth become mobile and fall out. On the red border of the lips, ulcers are covered with bloody-purulent crusts. In this case, the affected lip swells greatly, increases in size, and painful cracks appear on it. After the resolution of the process, a scar remains. If the process proceeded without ulceration, a smooth atrophic scar is formed. In places of ulceration, the scars are dense, rough, solder the mucous membrane with the underlying tissues. Recurrent formation of lupus on the scar is characteristic. Localization of the focus of lupus on the lip subsequently leads to its deformation, as a result of which it becomes difficult to eat, speech is distorted. If both lips are affected, a microstomy may develop.

The course of tuberculous lupus erythematosus is chronic. Without treatment, the process can continue indefinitely; fresh tubercles appear on the scars. Regional lymph nodes enlarge and thicken. Lupus tuberculosis can be complicated by erysipelas, candidiasis. Malignancy of lupus ulcers on the oral mucosa occurs in 1-10% of cases (lupus carcinoma).

Diagnostics. In typical cases, the diagnosis of tuberculous lupus is not difficult. The Mantoux reaction is in most cases positive. In the discharge from ulcers, as a rule, Mycobacterium tuberculosis is not found.

Histologically, in the reticular layer of connective tissue, a tubercle is determined, consisting of giant Pirogov-Langhans cells, plasma, epithelioid cells surrounded by a shaft of lymphocytes. Caseous necrosis is absent or mild. Elastic and collagen fibers in the specific infiltrate area are destroyed. In the area of infiltration and around it, there are a large number of dilated blood vessels, including newly formed ones.

Differential diagnosis. Lupus tuberculosis is differentiated from the tubercles that occur with tertiary syphilis. Syphilitic tubercles are larger than with tuberculous lupus, they are more dense. The edges of the ulcers in syphilis are even, dense, and in tuberculous lupus, they are soft, corroded. Unlike lupus, syphilitic eruptions do not recur on scars. There is no symptom of a failing probe and apple jelly for syphilis.

Tuberculous ulcer. Ulcer with uneven, edematous edges.

MILIAR-ULCER TUBERCULOSIS (tuberculosis miliaris ulcerosa). Miliary ulcerative tuberculosis of the oral mucosa is rare. It occurs in patients suffering from severe forms of pulmonary or laryngeal tuberculosis, accompanied by the release of a large number of tuberculosis mycobacteria with sputum. Due to a decrease in the body's resistance in such patients, the causative agent of tuberculosis is easily introduced into the mucous membrane and multiplies unhindered, causing ulcerative processes. Mainly affected are permanently traumatized areas of the mucous membrane of the cheeks, along the line of closing of the teeth, the back and lateral surfaces of the tongue, the soft palate, the bottom of the oral cavity. Usually 1-3 ulcers form. At the site of the introduction of mycobacterium tuberculosis, tubercles the size

of a pinhead of a grayish-red color appear, which very quickly turn into microabscesses and open up with the formation of small, very painful, superficial ulcers. Gradually increasing along the periphery, the ulcer often merges with similar, adjacent ulcers, sometimes reaching 2-3 cm in diameter (Fig. 11.21). Such ulcers are usually shallow, irregular in shape; undermined, pitted edges, sharply painful. The bottom of the ulcer is covered with a yellowish-gray bloom with a slight purulent discharge, bleeds easily, has a granular appearance due to non-disintegrated tubercles. At the bottom and around the ulcers, small yellowish spots (small abscesses) are found - the so-called Treel grain. Inflammation around the ulcers is insignificant. Regional lymph nodes may not be palpable at first, later they become enlarged, indurated, painful. With the prolonged existence of an ulcer and its secondary infection, compaction of the edges and bottom can be observed. On the tongue or along the transitional fold, ulcers can take the form of deep, painful cracks. With the localization of ulcers on the gums, in addition to damage to soft tissues, destruction may occur

Scraping from a tuberculous ulcer.

1 - neutrophilic leukocytes in the stage of deep decay; 2 - lymphocytes;

3 - giant Pirogov-Langhansa cage. Micrograph, x 500.

periodontal and bone, which can lead to mobility and loss of teeth.

The general condition of patients suffers sharply: emaciation, increased sweating, shortness of breath, increased body temperature, hypersalivation are observed. There are changes in the leukocyte formula, increased ESR. In scrapings from ulcers during cytological examination, giant cells of Pirogov-Langhansa (Fig. 11.22) and epithelioid cells are revealed. At a bacterioscopic examination, Koch's bacilli are found (staining according to Ziehl-Nielsen).

Differential diagnosis. It is performed with a gummy ulcer, which, in contrast to miliary-ulcerative tuberculosis, is painless, has a crater-like shape with dense ridge-like edges.

Tuberculous ulcer should also be differentiated from necrotizing ulcerative stomatitis of Vincent, traumatic, trophic and cancerous ulcers. Decisive in the diagnosis are the results of cytological and bacterioscopic studies.

Treatment. Since tuberculous lesion of the oral mucosa is a manifestation of general tuberculosis infection, the general treatment of patients is carried out in specialized anti-tuberculosis dispensaries. Local

treatment is aimed at eliminating traumatic factors, treatment of hard tissues of teeth and periodontium. Treatment of the oral mucosa with antiseptic and anesthetic drugs in the form of oral baths and applications is also used.

Lecture number 3

Topic 3: Changes in the oral cavity and red border of the lips in case of allergies and dermatoses. Cheilitis. Anatomy of the language. Pathological processes of the OSS. Purpose of examination methods

1.1. Technological models for education

Lesson time 80 minutes	Number of students 20-40
Activity type	Introduce lecture news
Lecture plan	The problem of complications of pharmaco-therapy is

1.treatment and practice of allergic reactions (angioedema anaphylactic shock) dental practice 2.treatment and clinic of slow current allergic reactions (allergic stomatitis cheilitis glossitis) 3. Treatment and clinic of bacterial allergies 4 treatment and clinic of cheilitis 5 treatment and clinic of lingual anomalies	especially urgent at the present time. This is due, on the one hand, to a significant increase in the arsenal of synthesized drugs that are allergens for the human body. In the pathogenesis of drug allergy, any type of allergic reaction or, more often, their combination may lie, which may be due to the individual reactivity of the organism. The clinical manifestations of drug allergy in the oral cavity are varied. Depending on the localization of US Pat. changes in mucus. obol. The mouth is distinguished by medicinal stomatitis, cheilitis, glossitis. In the treatment of these diseases, antihistamines and CA drugs are prescribed. It is recommended to eat non-irritating food and drink plenty of fluids.
Objective of the lesson	The basis for the prevention of diseases of oral mucosa and red border of the lips with allergies and dermatoses.
Teaching methods	Conversation, lecture visuals
Activity type	common-collective
Visual aids on the topic	Study guide, lecture material, projector, computer
The setting for the lesson	Methodical equipped classroom
Monitoring and evaluation criteria	Oral survey

1.2. Technological map of lectures

Stages of work	**Teacher**	**Teacher**
Preparation stages (10 minutes)	1.Objective of the lesson 2. Preparation of slides for the lecture material 3.Literature on the topic Banchenko G.V., Maksimov Yu.M. Language is the “mirror” of the body. M .: Business center "Dentistry" 2000 - 407 p. Borovskiy E.V. et al. Therapeutic dentistry: Textbook - M., 2004 .-- 840 p. Borovskiy E.V. Danilevsky N.F. Diseases of the oral mucosa. Atlas - M., 1991. - 320 p. Borovskiy E.V. Mashkillayson A.L. Diseases of the mucous membrane of the mouth and lips. M .: Medpress, 2001. Diseases of the oral mucosa. Ed. L.M. Lukinykh. - Novgorod, 2001.	**Listens and records**

1. Introduction (15 minutes)	***1. Purpose and objectives of the lecture material:*** ***Target:*** ***preliminary diagnosis and early preparation of the patient*** ***Task:*** ***• Features of the course of allergies and dermatoses. Cheilitis. Anatomy of the language. Pathological processes of the OSS.*** ***• Examination of the patient and develop a treatment plan;*** ***• Make a plan for treatment and prevention of stomas. diseases.*** ***2.Questions on the topic*** ***• Slow running allergic reactions?*** ***• Bacterial allergy?*** ***• Clinic of allergic reactions?*** ***• Treatment, clinic, differential. diagnosis of exfoliative cheilitis?*** ***• Treatment, clinic, differential. diagnosis of glandular cheilitis?*** ***• Treatment, clinic, differential. diagnosis of symptomatic cheilitis?*** • •	**Listen** **Answers students' questions**
2 main stage (55 minutes)	1. Introduction to the topic with slide show	Listen and record Listen
Final step 10 minutes	1. Conclusion. 2. Independent work. 3. Homework.	Listens and records Writes down

Lecture text 3

ANOMALIES AND INDEPENDENT DISEASES OF THE LANGUAGE

This section provides information about anomalies and diseases of the tongue, which are usually called independent glossitis - developing only in the tongue, without affecting other parts of the oral mucosa. These include desquamative glossitis, black (hairy) tongue, rhomboid glossitis, folded tongue.

Folded tongue

This is a congenital anomaly in the shape and size of the tongue. The folded tongue (lingua plicata) is equally common in children and adults, but children have a shallower depth of folds. The folded tongue is characterized by the presence of deep grooves (folds) on its surface, which are located mainly symmetrically in the longitudinal and transverse directions. The deepest longitudinal groove usually runs along the midline of the tongue. It originates from the tip of the tongue and often reaches the level of the grooved papillae. The transverse folds are less deep and

usually directed towards the main and deeper longitudinal fold, which unites them into a single system and creates a picture of the division of the tongue into many lobules. The bottom and lateral surfaces of the folds are covered with papillae characteristic of the normal mucous membrane of the tongue. The papillae are well expressed, sometimes hypertrophied (Fig. 11.49). The folding is clearly visible when the tongue protrudes. In most cases, it is accompanied by a moderate increase in the size of the tongue - macroglossia,

and its thickening is often noted. Folding and enlargement of the tongue as a developmental anomaly can capture only the anterior third of the tongue.

A folded tongue can be one of the symptoms of Melkerson-Rosenthal syndrome. In addition, the folded tongue in 30-50% of cases is combined with desquamative glossitis. The enlargement and folding of the tongue in combination with the folding of the cheeks is accompanied by acromegaly. In elderly people, due to a decrease in the turgor of the mucous membrane, slight folding of the tongue and cheeks may occur, which should not be equated with a congenital folded tongue.

With a folded tongue, complaints are usually absent, but once they discover an unusual appearance of the tongue and take the folds for cracks, patients turn to a doctor. With poor oral care, decreased salivation, food debris, cells of desquamated epithelium, microflora accumulate in the folds, which can lead to inflammation and complaints of burning and pain. The presence of deep folds favors the growing season of the fungal flora, especially in persons with reduced body reactivity.

Treatment. Local treatment of the folded tongue is not required. Oral hygiene and timely sanitation are essential.

Black ("hairy") tongue (lingua villosa nigra) is a disease characterized by keratinization and hyperplasia of the filiform papillae of the tongue of varying severity.

Quite a rare disease, it occurs mainly in men, more often in middle and old age.

Etiology. Unknown. Quite often, this disease occurs in people who abuse smoking and drinking alcohol.

The clinical picture. There may be no complaints. Most often, patients are worried about an unusual appearance of the tongue, sometimes a feeling of a foreign body on it, a gag reflex when talking, itching in the palate, a decrease in taste sensitivity. On the dorsum of the tongue, in front of the grooved papillae, the filiform papillae lengthen, thicken and acquire a color from light brown to black (Fig. 11.50). The length of the filiform papillae sometimes reaches 2 cm, and the diameter is up to 2 mm, externally, the papillae resemble hair. At the base, the papillae are thicker, but less pigmented than at the apex. A characteristic feature of a black ("hairy") tongue is

its color is determined, which is determined by the activity of chromatogenic bacteria. More often, hyperplastic papillae have a color from slightly brown to black, but sometimes they can be straw yellow, brown, dark gray. The altered area is usually oval or triangular in shape and is located in the midline. The front of the tongue and its lateral surfaces are not involved in the process and have a normal appearance. This focus without treatment can persist indefinitely, sometimes it can spontaneously disappear and after a while relapse again. In bacterioscopic examination, an increased number of leptotrichia is often determined.

Differential diagnosis. Black tongue should be distinguished from clinically similar conditions, the so-called false black tongue, which is a pronounced pigmentation of the papillae of the tongue and plaque. The reason for such pigmentation can be deterioration of oral hygiene, exacerbation of diseases of the gastrointestinal tract, the use of drugs (rinsing with solutions of potassium permanganate, chloramine, chlorhexidine, ingestion of antibiotics, metronidazole, etc.), food staining. Differential diagnosis is facilitated by anamnesis. In the case of staining with drugs or other exogenous staining, there is no pronounced hyperplasia of the filiform papillae; in addition, the entire back of the tongue is stained and after the withdrawal of the drug or other agent that caused the staining, the color normalizes within a few days. The black tongue is also differentiated from changes in the tongue with pigment-papillary dystrophy.

Treatment. Reorganization of the oral cavity is carried out (oral hygiene, removal of local traumatic factors). For local treatment, it is recommended to use keratolytic agents - lubricating the back of the tongue with 3-5% resorcinol solution, 5-10% salicylic alcohol solution (2-3 times a day). Used for the same purpose injections under the lesion of a 0.25% solution of calcium chloride. Introduce 0.5-1 ml of 0.25% calcium chloride with 0.5 ml of 1-2% novocaine under the lesion twice a week (for a course of 3-5 injections). Smoking, abuse of irritating food is prohibited. In severe hyperplasia, filiform papillae can be removed using cryodestruction.

Desquamative glossitis

Etiology and pathogenesis. Not fully clarified. Most often, desquamative glossitis (glossitis desquamativa, geographical language, exfoliative, or migratory, glossitis) occurs in diseases of the gastrointestinal tract, autonomic-endocrine disorders, rheumatic diseases (collagenoses). It is also assumed that a viral infection, a hyperergic state of the body, and hereditary factors play a certain role in the occurrence of desquamative glossitis. The disease occurs equally often in different age groups.

The clinical picture. The process begins with the appearance of a whitish-gray area of epithelial opacity with a diameter of several millimeters. Then it swells and in the center its filiform papillae slough off, exposing a bright pink or red rounded area that stands out against the background of the slightly raised area of epithelial opacity surrounding it (Fig. 11.51). The area of desquamation increases rapidly, maintaining an even circular shape, but the intensity of desquamation decreases. The zone of desquamation of the epithelium can be of different shapes and sizes and is reddish spots. Sometimes desquamation sites are in the form of rings or half rings. In the area of desquamation, mushroom papillae are clearly visible, which have the appearance of bright red dots. When the focus of desquamation reaches a significant size, its boundaries blur in the surrounding mucous membrane, and in its center, following desquamation, normal keratinization of the filiform papillae begins to recover, while in the areas of keratinization, on the contrary, desquamation occurs. Foci of desquamation can be single, but more often multiple and as a result of constantly changing processes of keratinization

and desquamation, they are layered on top of each other. Against the background of old foci, new ones are formed, as a result of which the shape of the desquamation sites and the color of the tongue are constantly changing, which gives the surface of the

tongue a look resembling a geographical map. This was the rationale for the names "geographical language", "migratory glossitis". A rapid change in the outlines of foci of desquamation is characteristic, the picture changes even when viewed the next day. Desquamation foci are localized on the back and lateral surfaces of the tongue, usually not spreading to the lower surface.

In most patients, especially in children, changes in the language occur without any subjective sensations and are detected by chance, during examination of the oral cavity. Only some patients complain of burning, tingling, paresthesia, pain from irritating food. The patients are also disturbed by the strange appearance of the tongue; carcinophobia may develop. Emotional stressful states contribute to a more severe course of the process. Desquamative glossitis, which occurs against the background of gastrointestinal tract pathology and other systemic diseases, can periodically worsen, which is often due to exacerbation of somatic diseases. Exacerbation of desquamative glossitis is accompanied by an increase in the intensity of desquamation of the epithelium of the mucous membrane of the tongue. Desquamative glossitis is combined with a folded tongue in about 50% of cases.

The disease lasts indefinitely, without causing concern to patients, sometimes disappears for a long time, then reappears in the same or other places.

There are cases when desquamations occur mainly in the same place.

Diagnostics. Recognition of the disease does not present any particular difficulties, since its clinical symptoms are very characteristic. Desquamative glossitis should be differentiated from:

And lichen planus;

And leukoplakia;

And plaques in secondary syphilis;

A hypovitaminosis B2, B6, B12;

And allergic stomatitis.

Histological changes are characterized by thinning of the epithelium and flattening of the filamentous papillae at the desquamation site, parakeratosis and moderate hyperkeratosis in the epithelium of the areas surrounding the lesion. In the actual mucous layer - mild edema and inflammatory infiltration.

Treatment. In the absence of complaints and discomfort, treatment is not carried out. With the appearance of a burning sensation, pain, it is recommended to sanitize the oral cavity, eliminate various irritants, and rational oral hygiene. Hygienic recommendations are especially relevant in the case of a combination of desquamative glossitis with a folded tongue, in which the anatomical features of the structure create favorable conditions for the reproduction of microflora in the folds, which can cause inflammation that brings pain. In the presence of a burning sensation, pain, light antiseptic rinsing, irrigation and oral baths with citral solution (25-30 drops of 1% citral solution per half a glass of water), applications of 5-10% suspension of anesthesin in an oil solution of vitamin E, applications of ke- ratoplastic agents (oil solution of vitamin A, rosehip oil, carotolin, etc.). Good results are obtained by treatment with calcium pantothenate (0.1-0.2 g 3 times a day orally for a month). In some patients, a positive effect is noted from the use of novocaine blockades in the lingual nerve region (for a course of 10 injections). In case of severe pain, it is advisable to prescribe local anesthetic drugs. It is imperative to identify and treat

concomitant diseases. This treatment is symptomatic, it is aimed at eliminating or reducing pain, reducing the frequency of relapses. However, there is still no means of completely eliminating relapses of the disease,

Diamond-shaped glossitis.

On the back of the tongue there is a lumpy formation of pink, oval shape,

protruding above the surface of the mucous membrane.

especially in old age. Carcinophobia often develops. Prevention of such conditions can be individual conversations with patients and correct deontological tactics. The prognosis of the disease for life is favorable, the possibility of malignancy of desquamative glossitis is excluded.

Diamond-shaped glossitis

Rhomboid (rhombic) glossitis (glossitis mediana rhombica) is a chronic inflammatory disease of the tongue of a characteristic type and localization.

Etiology and pathogenesis. Not fully clarified. Currently, most researchers assume that rhomboid glossitis is a congenital disease and its development is due to impaired embryogenesis. Other authors consider rhomboid glossitis to be chronic candidiasis, since the fungus of the genus Candida is almost always detected on the surface of the lesion. The disease occurs mainly in men - smokers and alcohol abusers.

The clinical picture. Clinically, rhomboid glossitis is manifested by a rhomboid or oval-shaped lesion, slightly compacted to the touch, located strictly along the midline in front of the grooved papillae. The dimensions of this lesion in different patients range from 1.5 to 5 cm in length and from 0.5 to 2.5 cm in width. In shape, this formation resembles a rhombus, in connection with which it received the name "rhomboid glossitis" (Fig. 11.52). Hearth

lesions are single, very rarely there are 2-3, located along the midline of the tongue. The surface of the focus is devoid of papillae.

Diamond glossitis is a chronic condition that lasts for years and usually does not progress. The variety of clinical course of rhomboid glossitis made it possible to distinguish three forms of the disease: flat (or smooth), tubercular (or tuberous) and papillomatous (or hyperplastic).

With a flat form, the surface of the tongue in the affected area is smooth, polished, does not protrude above the surrounding mucous membrane, pink or red, sometimes (mainly in the elderly) with a cyanotic tint, the papillae in the affected area are absent. On palpation, it can be revealed that the affected area is slightly compacted, painless. Submandibular lymph nodes with flat and other forms of rhomboid glossitis are not palpable.

In the case of the tubercle form, the diamond-shaped, round or oval lesion consists of tubercles of various sizes, separated from each other by pronounced folds. This arrangement of the bumps resembles a pavement lined with granite. The surface of the tubercles and folds is red with a cyanotic tint, devoid of papillae.

The most pronounced changes are observed in the papillomatous form of rhomboid glossitis. The lesion is a lumpy tumor-like growth protruding above the surface of the tongue. The focus often reaches 3-5 cm in diameter. On palpation, the seal is not detected or it is insignificant. With tubercle and papillomatous form, the surface of the center of the focus may become keratinized, then it acquires a milky white color. The

affected area does not increase throughout the patient's life, it constantly retains its shape and size.

All three clinical forms of rhomboid glossitis can occur without any subjective sensations. Most patients, if you do not pay their attention, usually do not know about the existence of such changes. Complaints of burning, roughness of the back of the tongue, and sometimes pain appear in cases where inflammation develops.

The course of rhomboid glossitis is usually benign. The clinical and morphological picture remains unchanged for many years. Only when not-

favorable conditions (constant chronic irritation, a decrease in the body's defenses) tuberous and papillomatous forms of glossitis show a tendency to the progression of the process. Isolated cases of malignancy of rhomboid glossitis are described.

Differential diagnosis. The flat form is differentiated from: A desquamative glossitis; a. avitaminosis;

tubercle and papillomatous from: A tumors; and candidiasis.

Treatment. The oral cavity is sanitized to exclude local traumatic factors, it is recommended to stop smoking. If a fungus of the genus Candida is found, anti-candidiasis treatment is performed. As a rule, the listed therapeutic measures are enough to eliminate inflammation in flat, tuberous and mild papillomatous forms. If a tendency to proliferation of tubercular and papillomatous forms is detected, they are subject to excision within the lesion focus. The use of cryotherapy is effective.

Heilith

Cheilitis is a benign inflammatory disease of the lips. There are two groups of cheilitis: cheilitis proper and symptomatic cheilitis.

The group of cheilitis proper unites independent diseases of the lips of various etiologies. These include exfoliative cheilitis, glandular cheilitis, meteorological and actinic cheilitis. The second group - symptomatic cheilitis - includes lip lesions, which are a symptom of diseases of the oral mucosa, skin, and general somatic diseases. The group of symptomatic cheilitis includes atopic cheilitis, eczematous cheilitis, contact cheilitis, macrocheilitis with Melkerson-Rosenthal syndrome.

Exfoliative cheilitis

Exfoliative cheilitis (cheilitis exfoliativa) is a chronic disease in which only the red border of the lips is affected. This disease was first described by Stelwagon in 1900 under the name "persistent desc-

Exfoliative cheilitis (dry form). Transparent scales with lagging edges on the dry and almost unchanged red border of the lips in color.

lip vation ". Later Miculicz and Kypte1 proposed the name "exfoliative cheilitis".

Exfoliative cheilitis is more common in women between the ages of 20 and 40.

Etiology and pathogenesis. Not fully studied. Most researchers believe that the disease is based on neurogenic factors. In patients with exfoliative cheilitis, various variants of psychoemotional disorders were revealed. There is evidence of a connection between thyroid diseases and exfoliative cheilitis. The role of genetic predisposition in the occurrence of exfoliative cheilitis is noted. Data on the possible role of immunological factors in the pathogenesis of the disease have been obtained.

The clinical picture. There are two clinical forms of exfoliative cheilitis - dry and exudative. Exfoliative cheilitis has a very characteristic localization. Pathological changes are localized on the red border of both lips or one of them, and only part of the

red border of the lips is affected from the Klein line to its middle. The affected area looks like a tape extending from one corner of the mouth to another. The part of the red border of the lips adjacent to the skin, as well as the corners of the mouth, remain unaffected.

Exfoliative cheilitis (exudative form).

Abundant deposition of scales with lagging edges on the hyperemic and edematous red border of the lips.

The process never spreads to the skin and mucous membrane of the mouth.

The dry form is characterized by the appearance of stagnant hyperemia on the surface of the red border of the lips, on which dry translucent mica-like scales of gray or grayish-brown color are formed in the affected area, attached by their center to the red border and somewhat lagging along the edges (Fig. 11.53). The lips are dry, constantly peeling. The scales are quite easily removed and a stagnant red surface is exposed under them, but without the formation of erosion. After 5-7 days, mica-like scales are formed again. Patients note dry lips and the habit of constantly biting scales.

The course of the disease is long, without a tendency to remission or self-healing. The dry form of exfoliative cheilitis can be transformed into exudative.

The exudative form of exfoliative cheilitis is characterized by severe pain, lip swelling, hyperemia. In the Klein zone, an abundance of scales and crusts of a grayish-yellow or yellow-brown color is formed, which cover the affected area from the corner to the corner of the mouth with a layer (Fig. 11.54). Sometimes the crusts reach significant sizes

ditch and hang from the lip in the form of an apron, creating the appearance of the defeat of the entire red border. However, the strip of red border of the lips adjacent to the skin, as well as the corners of the mouth, remain unaffected. After removing the crusts, a brightly hyperemic lip surface is exposed without erosion, which is a distinctive feature of exfoliative cheilitis. Patients with exudative form of exfoliative cheilitis are worried about a burning sensation, soreness of the lips, especially when closing, which makes it difficult to eat, speak, therefore, the mouth of these patients is often slightly open.

The reason for the pronounced exudative phenomena is a sharp increase in capillary permeability. Under the influence of treatment, the exudative form of exfoliative cheilitis can turn into dry.

Pathologically, acanthosis, para- and hyperkeratosis are noted. The presence of a large number of light cells in the spiny layer of the epithelium is characteristic; these are cells with reduced metabolic activity, in contrast to other cells that are actively functioning. Loosening of the epithelial layer and the formation of extensive gaps in it due to intracellular lysis are noted, which contributes to an increase in exudation.

Differential diagnosis. The dry form of exfoliative cheilitis should be differentiated from: ^ meteorological cheilitis; Atopic cheilitis; And contact allergic cheilitis.

With meteorological cheilitis, the entire surface of the red border of the lips is affected with more pronounced symptoms of inflammation. In addition, with meteorological cheilitis, a direct dependence of its course on the action of various meteorological factors is revealed.

With atopic cheilitis, a part of the red border adjacent to the skin, the corners of the mouth, is affected. The area of the red border of the lips adjacent to the mucous

membrane is not affected. Atopic cheilitis is characterized by lichenization and a change in periods of exacerbations with remissions, often of a seasonal nature.

For contact allergic cheilitis, in contrast to the dry form of exfoliative cheilitis, the presence of erythema in the area of contact with the allergen is characteristic, as well as the rapid elimination of inflammation after the elimination of the allergen.

The exudative form of exfoliative cheilitis must be differentiated from:

A exudative form of actinic cheilitis;

^ eczematous cheilitis;

A erosive and ulcerative form of lupus erythematosus.

With the exudative form of actinic cheilitis, there is a connection with the season; the defeat covers the entire surface of the red border, its swelling and the presence of erosions are noted.

With eczematous cheilitis, polymorphism of rashes (erosion, crusts, vesicles, cracks) is expressed with frequent changes in lesion elements, periods of exacerbations and remission. Localization of the lesion in eczematous cheilitis captures the entire surface of the red border of the lips with the transition to the skin of the face.

The erosive and ulcerative form of lupus erythematosus, in contrast to the exudative form of exfoliative cheilitis, proceeds with significantly pronounced erythema, hyperkeratosis, the formation of erosions and ulcers, and cicatricial atrophy.

Treatment. Therapy should be comprehensive and have differences in dry and exudative forms of exfoliative cheilitis.

An important role in treatment is given to the means of influencing the psychoemotional sphere. For this purpose, patients are prescribed tranquilizers: phenazepam 0.0005 g 3 times a day, sibazone (seduxen, relanium) 0.005 g 3 times a day, or antipsychotics: thioridazine (melleril, sonapax) 0.01 g 3 times a day. Given the predominance of depressive reactions in patients with dry form of exfoliative cheilitis, they should be prescribed antidepressants (amitriptyline, melipramine 0.025 g 2 times a day) and tranquilizers: trioxazine 0.03 g 3 times a day, chlosepide (elenium) 0 , 01 g 3 times a day. Locally, patients with a dry form for lubricating lips are recommended indifferent creams "Delight", "Spermaceti", hygienic lipstick.

If thyrotoxicosis is detected, it is necessary to undergo treatment with an endocrinologist.

For the treatment of patients with exudative form of exfoliative cheilitis, the use of complex therapy is effective, including exposure to borderline rays (Bucca radiation) - 2 Gy 2 times a week, the total dose is from 16 to 30 Gy. Before each session, it is necessary to remove the crusts from the red border, it is preferable to soak them with a 2% boric acid solution. It is good to combine this treatment with taking drugs that increase the reactivity of the body, for example, pyrogenal, which is administered

they are given intramuscularly (starting from 50 MTD and adding 100 MTD every other day, bring a single dose to 1000–1500 MTD). Patients with exudative form of exfoliative cheilitis need 3-4 courses of complex treatment with a break of 6-8 months.

To eliminate edema, soreness, burning sensation in the exudative form of exfoliative cheilitis, the method of reflexology is successfully used.

It should also be remembered about the role of psychotherapeutic methods that are effective in the treatment of exfoliative cheilitis.

Glandular cheilitis

Glandular cheilitis (cheilitis glandularis) is a disease of the lip, mainly the lower one, develops as a result of hyperplasia, hyperfunction, and sometimes heterotopia of the salivary glands in the area of the red border of the lips and the transition zone (Klein zone).

There are primary and secondary glandular cheilitis. The development of primary glandular cheilitis is associated with a congenital anomaly of the salivary glands. Secondary glandular cheilitis develops, as a rule, against the background of various diseases with localization on the lips (lichen planus, leukoplakia, etc.).

Etiology. In the etiology of primary glandular cheilitis, the leading role is assigned to hereditary anomalies of the lower lip with hyperplasia, heterotopia and excessive secretion of small salivary glands, which, under the influence of unfavorable irritating factors (tartar, decayed teeth, sharp edges of teeth, periodontal disease, etc.) begin intensively produce saliva.

Secondary glandular cheilitis is a consequence of chronic inflammatory processes on the red border of the lip. As a result, inflammatory infiltration in leukoplakia, lupus erythematosus and other diseases with localization on the lips causes irritation, hyperplasia and hyperfunction of the glandular tissue. Increased secretion, lip maceration and infection are noted. However, only a few people develop these phenomena.

The clinical picture. Glandular cheilitis develops mainly in people over the age of 50. In men, primary glandular cheilitis is found 2 times more often than in women. The lower lip is twice as likely to be affected as the upper lip.

Simple glandular cheilitis.

On the slightly hyperemic mucous membrane of the lower lip are visible

droplets of saliva (a symptom of "dew drops").

Glandular cheilitis has a typical clinical presentation. In the area of transition of the mucous membrane to the red border of the lip, and sometimes on the red border, dilated orifices of the salivary glands are visible in the form of red dots, from which drops of saliva stand out - a symptom of "dew drops" (Fig. 11.55). Sometimes around the mouths of the salivary glands, leukoplakia develops in the form of rings, and in some cases, the red border of the lip becomes keratinized over a large extent.

Due to the frequent moistening of the lips with saliva and evaporation of the latter, dryness of the red border of the lips develops, maceration, cracks and erosion appear. Subsequently, the mucous membrane and red border may gradually become keratinized. Initially, this occurs around the mouths of the excretory ducts of the salivary glands, and then diffusely. With a prolonged course of glandular cheilitis due to constant irritation of the lip with the appearance of cracks, foci of hyperkeratosis, its unfavorable course with the development of precancerous lesions is possible.

As a result of penetration into the dilated ducts of the salivary glands of a pyogenic infection, glandular cheilitis can turn into a purulent form. It is characterized by soreness, swelling of the lip, which is covered with thick yellow-green or brown-black crusts. Purulent discharge from the dilated ducts of the salivary glands, cracks and erosion are localized around them. Sometimes the mouths of the excretory ducts of the glands can be clogged with purulent exudate, which leads to multiple or single abscess formation.

Diagnostics. Clinical manifestations of glandular

cheilitis is so characteristic that diagnosis, as a rule, is not difficult. Histopathological examination reveals hypertrophy of the salivary glands with a slight inflammatory infiltration, mainly around their excretory ducts. In some patients, acanthosis and parakeratosis are observed in the epithelium.

Treatment. For the treatment of primary glandular cheilitis, anti-inflammatory ointments (prednisolone, hydrocortisone, naphthalan, etc.) are used. Electrocoagulation or excision of hypertrophied salivary glands is used.

Treatment of secondary glandular cheilitis involves, first of all, the treatment of the underlying disease that caused it, anti-inflammatory therapy is performed locally.

Forecast. With glandular cheilitis, the prognosis is favorable, but in the absence of timely and effective treatment against its background, the development of precancerous diseases is possible.

Actinic and meteorological cheilitis

Actinic and meteorological cheilitis (cheilitis actinica, cheilitis meteorologica) are inflammatory diseases of the lips, caused by a variety of meteorological factors (solar radiation, high or low humidity, wind, dustiness, cold).

Unlike meteorological cheilitis, with actinic cheilitis, the leading is the presence of increased sensitivity to ultraviolet rays.

The diseases are more common in men between the ages of 20 and 60.

ACTINIC CHEILIT (cheilitis actinica). The main reason for the occurrence of actinic cheilitis is the development of a delayed-type reaction to UV rays.

The clinical picture. There are dry and exudative forms of actinic cheilitis.

The dry form of actinic cheilitis is manifested by dryness of the lower lip, the upper lip and facial skin are rarely affected. The defeat covers the entire surface of the red border, it becomes bright red, covered with small, dry silvery-white scales. In the future, the appearance of abrasions, erosion is possible. In some patients, areas of hyperkeratosis are formed on the red border, and sometimes verrucous growths.

With the prolonged existence of actinic cheilitis and the addition of the action of irritating factors (smoking, humidity, insolation), its malignancy is possible.

The clinical picture of the exudative form of actinic cheilitis is characterized by the appearance on the edematous hyperemic surface of the red border of the lower lip of bright red erythema, small vesicles, weeping erosions covered with crusts. Patients are worried about itching, burning, soreness of the lips. Characterized by the seasonality of the course of actinic cheilitis: exacerbation of the disease in the spring-summer period and remission in the autumn-winter period.

Differential diagnosis. The dry form of actinic cheilitis should be distinguished from:

A dry form of exfoliative cheilitis;

^ lupus erythematosus.

The exudative form is differentiated from: A contact allergic cheilitis;

^ atopic cheilitis.

Treatment. First of all, it is necessary to avoid insolation if possible, change the profession if it is associated with a long stay in the open air. Prescribe nicotinic acid, vitamins of group B (B2, B6, B12), in some cases they are combined with antimalarial drugs (Delagil 0.25 g 2 times a day for 2-3 weeks) and small doses of corticosteroids (for example, prednisolone according to 10 mg daily). Topically use ointments with corticosteroids (0.5% prednisolone, "Flucinar", "Lokakorten", "Lorinden", etc.).

In order to prevent actinic cheilitis, use photoprotective creams "Ray", "Shield", "Antilux", 10% salol ointment.

Forecast. Favorable, however, with a prolonged course of the dry form, malignancy of the process can occur.

METEOROLOGICAL CHEILIT (cheilitis meteorologic). It is an inflammatory disease of the lips, the cause of which is a variety of meteorological factors (high or low humidity, solar radiation, dustiness, wind, cold, etc.). An important role in the development of this disease is played by the duration of exposure to these factors and the constitutional features of the skin. Meteorological cheilitis often occurs either in persons with white and delicate skin,

or in people with skin diseases accompanied by its increased dryness (seborrhea, seborrheic eczema, diffuse neurodermatitis, etc.).

The clinical picture. With meteorological cheilitis, the red border of the lip is affected, usually the lower one along its entire length. The red border of the lip is slightly hyperemic and infiltrated, dry, often covered with small scales. Patients are worried about dry lips, a feeling of tightness of the lips, peeling. Long-term exposure to unfavorable meteorological factors contributes to the appearance of erosion or cracks.

Histologically, hyperplasia is determined in the epithelium, in places with slight keratinization, and stromal infiltration.

Differential diagnosis. Differentiate meteorological cheilitis from:

A dry form of actinic cheilitis; And allergic contact cheilitis; A dry form of exfoliative cheilitis.

Treatment. It is carried out according to the same principles as for actinic cheilitis.

Forecast. Favorable, but with a long course, although rarely, obligate forms of precancer are possible against its background (abrasive precancerous cheilitis of Manganotti, limited hyperkeratosis).

Contact allergic cheilitis

Contact allergic cheilitis (cheilitis allergica contactilis) develops as a result of sensitization of the red border of the lips upon contact with various substances. The development of this disease is based on a delayed-type allergic reaction to various chemicals.

Contact allergic stomatitis occurs mainly in women over the age of 20.

Etiology and pathogenesis. Contact allergic cheilitis is a clinical manifestation of a delayed-type allergic reaction with substances in contact with the red border of the lips. The most common cause of allergic contact cheilitis are lipsticks, toothpastes, and plastic dentures. It is possible to develop cheilitis from contact with metal objects (mouthpieces of wind instruments, pens, pencils, etc.). Sometimes in the case

Allergic contact cheilitis. Small erosions and cracks on a hyperemic and edematous red border

In teas, the disease can be of a professional nature.

The clinical picture. The process is usually localized on the red border of the lips, sometimes it slightly captures the skin. Less common is a combined lesion of the red border of the lips and the mucous membrane of the mouth. At the site of contact with the allergen, erythema of the red border of the lips develops. With pronounced inflammatory phenomena against the background of a hyperemic red border of the

lips, small bubbles appear, quickly opening and forming erosion, cracks (Fig. 11.56). Patients complain of severe itching, burning, swelling of the lips. Contact allergic cheilitis can occur without a significant inflammatory reaction. In these cases, there is only limited erythema and slight peeling on the red border of the lips. With a prolonged course of the disease, the red border of the lips becomes dry, small grooves and cracks appear on it.

Differential 'diagnostics. The disease should be differentiated from:

^ exfoliative cheilitis;

A dry form of actinic cheilitis;

^ atopic cheilitis.

Treatment. When treating allergic contact heilitis, first of all, it is necessary to identify and eliminate the factors that caused the disease. For local treatment, ointments containing corticosteroids (0.5% prednisolone, "Flucinar", "Lorinden") are used, which must be applied to the surface of the red border of the lips 5-6 times a day. Inside, desensitizing drugs are prescribed (suprastin, tavegil, fenkarol, etc.).

Prevention. Prevention of recurrence of contact allergic cheilitis is to exclude contact with the allergen.

Atopic cheilitis

Atopic cheilitis (cheilitis atopicalis) is one of the symptoms of atopic dermatitis or neurodermatitis, that is, it belongs to the group of symptomatic cheilitis.

The disease is more common in children and adolescents of both sexes between the ages of 7 and 17 years.

Etiology. In the etiology of atopic cheilitis, an important role belongs to genetic factors that create a predisposition to the so-called atopic allergy. Allergens can be medicines, food products, flower and household dust, microorganisms, cosmetics, etc.

The clinical picture. With atopic cheilitis, the red border of the lips is affected with the involvement of adjacent skin areas in the pathological process, and in the area of the corners of the mouth, the lesion manifests itself most intensely. The red border of the lips is infiltrated, flakes off with small scales (Fig. 11.57). Infiltration and dryness of the lips over time leads to the formation of cracks and furrows. The process never spreads to the mucous membrane of the mouth. Lip changes are often combined with dry, flaky skin.

Patients with atopic haylight complain of itching, burning, peeling of the red border of the lips.

Atopic cheilitis lasts a long time, exacerbations of the disease occur mainly in the autumn-winter period, remission usually occurs in summer.

Differential diagnosis. When carrying out the differential diagnosis of atopic cheilitis, concomitant skin lesions that are present in most cases should be taken into account. Typical rashes are found on the skin of the face, elbows, popliteal hollows, less often on the skin of the trunk.

Atopic cheilitis. On the infiltrated red border of the lips there are small scales and cracks.

Atopic cheilitis must be differentiated from those clinically similar to it: ^ actinic cheilitis; And allergic cheilitis; And contact cheilitis; And streptococcal seizures.

Treatment. In the treatment of atopic cheilitis, an important place is given to the conduct of desensitizing therapy. Inside, antihistamines are prescribed (suprastin, diazolin, claritin, fencarol). With prolonged persistent course of the disease, corticosteroid drugs can be administered orally for a period of 2-3 weeks (prednisone for children 8-14 years old at 10-15 mg / day, for adults at 15-20 mg / day or dexamethasone). Also used intravenous infusion of 30% sodium thiosulfate solution (5-10 ml daily, for a course of up to 10 injections). In some patients, good results have been obtained from the use of histaglobulin, which is prescribed in courses of 6-8 injections; the drug is administered subcutaneously 2 times a week in increasing doses, starting from 0.2 ml to 1 ml. Inside, vitamins of group B (B „ B6, B | 2) are also prescribed. Topically applied corticosteroid ointments (0.5% prednisolone, "Lorinden", "Flucinar", etc.), which must be applied to the affected surface 4-5 times a day. With a small effect 701 ---------- page 703 ----------

from conservative therapy and the stubborn course of the disease, the boundary rays of Bucca are used. Substances that can cause sensitization of the body should be excluded from the diet: caviar, chocolate, coffee, strawberries, citrus fruits, etc., spicy, salty, spicy foods, alcohol; drastically limit carbohydrate intake.

Eczematous cheilitis

Eczematous cheilitis (cheilitis eczematosa) also refers to symptomatic diseases of the lips, since it manifests itself as a symptom of a general eczematous process, which is based on inflammation of the surface layers of the skin of a neuroallergic nature.

Allergens in this case can be various factors: microorganisms, food substances, drugs, materials for the manufacture of prostheses, metals such as nickel and chromium, amalgam, filling materials, etc.

The clinical picture. Eczematous cheilitis, like any eczema, can be acute, subacute or chronic. The lesion of the red border of the lips can be combined with lesions of the skin of the face or be isolated.

The acute stage of the disease is characterized by polymorphism: vesicles, oozing, crusts, scales. The process is accompanied by significant swelling of the lips. At this stage of the disease, patients complain of itching, burning in the lips, edema and hyperemia. The spread of the process to the skin adjacent to the red border is very characteristic.

The transition of the disease into a chronic form is characterized by a decrease in inflammatory phenomena (edema, hyperemia). The red border of the lips and areas of the affected skin around the mouth become denser due to inflammatory infiltration, nodules and scales appear. The rash of vesicles stops, and desquamation occurs. In this state, the process can continue for a long time. All the described clinical symptoms of eczematous cheilitis develop on unchanged lips. Sometimes the occurrence of an eczematous process is preceded by long-standing microbial seizures, cracks. In these cases, developing eczema of the lips is usually considered as microbial sensitization of the red border of the lips or skin. The clinical manifestations of microbial eczema on the red border of the lips are completely identical to the described clinical picture of eczematous cheilitis. Feature of microbial eczema of the lips

is a more pronounced eczematous reaction near the microbial focus.

Differential diagnosis. Eczematous cheilitis is differentiated from: A atopic cheilitis; ^ allergic contact cheilitis; A exudative form of actinic cheilitis.

Treatment. Desensitizing and sedative medications are prescribed. Topical treatment consists in prescribing ointments containing corticosteroids, and in the presence of oozing, aerosols with corticosteroids are used. For microbial eczema, ointments and aerosols containing corticosteroids and antimicrobial drugs (Lorinden S, Sinalar-N, aerosols Dekson, Dexocort, etc.) are prescribed.

Macroheilit

Macrocheilitis (macrocheilitis) is the leading symptom of the disease first described in 1901 by GI Rossolimo; then the Swedish physician Melkersson also described a disease characterized by unilateral paralysis of the facial nerve and edema of the lips. Later in 1931, in the description of the disease, Rosental added the symptom of a folded tongue to these two signs. Since then, the disease manifested by this triad of symptoms has been called Melkerson-Rosenthal syndrome.

Etiology. In the etiology of this disease, a number of authors assign an important role to the constitutional and hereditary characteristics of a person. Others tend to consider Melkerson-Rosenthal syndrome as angioneurosis. The infectious-allergic theory of the onset of the disease also has supporters.

The clinical picture. Melkerson-Rosenthal syndrome is characterized by three symptoms: macrocheilitis, folded tongue, and facial nerve palsy. This disease is somewhat more common in women.

The illness usually begins suddenly. As a rule, swelling of one or both lips is the first symptom of the disease. Sometimes the first sign may be neuralgia-type facial pain, often followed by facial nerve palsy. Within a few hours, swelling of one or both lips appears. Patients note itchy lips, an increase in its size, sometimes the appearance of edema in other parts of the face. There is a formless swelling of the lip, it shines, it is not changed in color, in some cases it acquires

Macrocheilitis in Melkerson-Rosenthal syndrome. The lower lip is swollen, enlarged, its color is not changed.

bluish tint (Fig. 11.58). Sometimes the swelling is so great that the lips are 3-4 times thicker than normal. Lip thickening is uneven, with one side of the lip usually more swollen than the other. On palpation, a uniformly soft or densely elastic consistency of the lip tissue is felt. There is no pressure after palpation. Such edema in a number of patients exists constantly, sometimes increasing, then weakening. In some patients, edema spontaneously disappears, but later recurs with varying frequency, and then becomes permanent. The intensity of the swelling can vary throughout the day. Edema is localized most often on the lips, cheeks, eyelids and other parts of the face.

The second symptom of the disease is paralysis of the facial nerve. It consists in unilateral vasomotor rhinopathy, unpleasant sensations in the mouth, changes in saliva secretion. Due to the loss of tone of the affected half of the face, the corners of the mouth descend, the palpebral fissure expands.

The third symptom of the disease is a folded tongue, which is observed in 2/3 of patients and, obviously, is not a symptom, but a developmental anomaly. Swelling of the tongue is clinically noted, leading to its uneven increase.

It is far from always that in patients with Melkerson-Rosenthal syndrome, a combination of all three symptoms is observed, sometimes the disease can be clinically manifested only by macrocheilitis.

Diagnostics. If the patient has all three symptoms of the disease, the diagnosis of Melkerson-Rosenthal syndrome is not difficult. Difficulties arise when there is only one symptom - macrocheilitis. In this case, differential diagnosis is carried out with: A Quincke's edema ^ erysipelas; A lymphangioma; A hemangioma.

Treatment. It is a rather difficult task. Each patient should be carefully examined in order to identify his pathology, contributing to the onset of this disease. Particular attention is paid to identifying foci of chronic infection, including the maxillofacial area.

When treating patients with Melkerson-Rosenthal syndrome, corticosteroid drugs (20-30 mg prednisolone, or 2-3 mg dexazone per day) are prescribed along with broad-spectrum antibiotics (olettrin 750,000-1,000,000 IU per day) and synthetic antimalarial drugs (Delagil 0.25 g 2 times a day, for a course of 20-25 g). Antihistamines, vitamins C and B are also used. The combination of drugs used for treatment is determined by the general condition, the patient's age, clinical symptoms, and the stage of the disease. The specified course of treatment is carried out within 30-40 days.

When a microbial allergy is detected in a patient, specific hyposensitizing therapy is performed.

Treatment of persistent, persistently ongoing forms of the disease during the period of remission is carried out with pyrogenic drugs (pyrogenal, prodigiosan, etc.).

For the treatment of macrocheilitis, electrophoresis of heparin and Dimexide is used.

If there is no effect of conservative treatment, surgical methods are used. For cosmetic purposes, part of the lip tissue is excised. However, surgical treatment does not prevent recurrence of the disease.

In the treatment of Melkerson-Rosenthal syndrome, the best results were obtained in the early stages of the disease. It is advisable to repeat the indicated courses of treatment after 2-3 months.

PRECANCERAL DISEASES OF THE MUCOSA OF THE ORAL AND RED BORDER OF THE LIPS

Timely diagnosis of precancerous diseases of the oral mucosa and the red border of the lips is the main link in the prevention of cancer. Cancer of the red border of the lips and oral mucosa is quite common, accounting for about 5% of all malignant tumors.

Most often, the red border of the lower lip (in the lateral region) is affected, in the oral cavity - the tongue (lateral-surface) and the floor of the mouth.

Among patients with cancer of the red border of the lips and the mucous membrane of the mouth, men over the age of 40 prevail.

Most cancer patients are preceded by certain diseases of the oral mucosa and the red border of the lips, which are called precancerous. Their occurrence is facilitated primarily by injuries, especially chronic ones, including smoking and chewing tobacco, betel nut, the use of nasa, and alcohol. Injuries are considered as external factors of carcinogenesis.

Cancer is often preceded by proliferative processes, benign tumors, chronic inflammatory diseases, accompanied by erosions and ulcers.

A precancerous disease exists for a long time (from several months to tens of years), then it can (but not necessarily) turn into cancer.

Early detection and treatment of precancerous diseases eliminates the threat of cancer or allows for timely, more effective treatment.

Depending on the likelihood of malignancy, there are obligate and optional precancerous diseases of the oral mucosa and the red border of the lips. Obligate precancers have a high likelihood of malignancy; without timely treatment, they usually transform into cancer. Many of them represent cancer in situ from the very beginning of their development. Facultative precancers do not always lead to cancer. In 1970, the classification of precancerous conditions of the mucous membrane of the mouth and the red border of the lips, proposed by AL Mashkilleison, was adopted and subsequently (in 1976) approved.

Classification of precancerous processes of the oral mucosa and the red border of the lips

Classification of precancerous processes of the oral mucosa

I. With a high incidence of malignancy (obligate):

Bowen's disease.

II. With a low incidence of malignancy (optional):

leukoplakia verrucous and erosive;

papillomatosis;

erosive-ulcerative and hyperkeratotic forms of lupus erythematosus;

erosive-ulcerative and hyperkeratotic forms of lichen planus;

postradiation stomatitis.

Classification of precancerous processes of the red border of the lips

I. With a high incidence of malignancy (obligate):

warty precancer;

limited precancerous hyperkeratosis;

abrasive pre-cancerous cheilitis of Manganotti.

II. With a low incidence of malignancy (optional):

leukoplakia;

keratoacanthoma;

cutaneous horn;

papilloma with keratinization;

erosive-ulcerative and hyperkeratotic forms of lupus erythematosus;

erosive-ulcerative and hyperkeratotic forms of lichen planus;

post-radiation cheilitis.

Bowen's disease. An area of hyperkeratosis with a small tuberous surface on the buccal mucosa.

The following is information about obligate and some optional precancerous conditions of the mucous membrane and the red border of the lips. Descriptions of other optional precancerous conditions are given in the corresponding sections of this textbook.

Bowen's disease

Bowen's disease (Bowen morbus) was first described by Bowen in 1912. It is originally a cancer in situ.

The clinical picture. On the mucous membrane of the mouth, one is more often found, less often two or more lesions. The disease manifests itself as a maculo-nodular lesion. Initially, a bright hyperemic spot with a smooth or velvety surface, due to papillary growths, is formed on the mucous membrane. With the long-term existence of the disease, a slight atrophy of the mucous membrane occurs, as a result of which the lesion focus slightly sinks in comparison with the surrounding areas, in places it forms easily bleeding erosion. The central part of the lesion resembles leukoplakia with a small tuberous surface on hyperemia

against a background (Fig. 11.59). When several foci merge, irregular plaques are formed. The diameter of the lesion can be from 1-2 mm to 5-6 cm, its outlines are clear, uneven, the compaction at the base is not determined. The favorite localization of Bowen's disease on the oral mucosa is the soft palate, uvula, retromolar region, tongue. When the elements are localized on the tongue, the papillae disappear at the site of the lesion. Regional lymph nodes are usually not palpable. Subjective sensations, as a rule, are absent, only with erosions can soreness be expressed.

Not all cases of Bowen's disease have a typical clinical picture. The disease may be characterized by only a small area of hyperemia or resemble leukoplakia without pronounced inflammation. Sometimes there is a clear grayish-white pattern, reminiscent of lichen planus.

The disease continues indefinitely, in some cases invasive growth occurs rapidly, and trauma accelerates this process. In other patients, the disease remains in the stage of cancer in situ for years.

Histologically, in Bowen's disease, a picture of intraepithelial cancer is found: polymorphism of the cells of the spiny layer, an increase in the number and irregularity of mitoses. In the spinous layer, giant cells are found, sometimes with multiple nuclei. Weakly expressed hyper- and parakeratosis are noted, there is always acanthosis, the basement membrane and the basal layer are preserved. In the upper part of the stroma there is a small infiltrate, consisting mainly of lymphocytes and plasma cells.

Differential diagnosis is carried out with: A leukoplakia;

^ lichen planus;

^ chronic traumatic injuries.

Treatment. Surgical - removal of the affected area within healthy tissues.

Warty precancer

Warty precancer (praecancer verrucosus) manifests itself as a hemispherical nodule with a bumpy surface, towering above the level of the mucous membrane, 4-10 mm in diameter (Fig. 11.60). The knot is located, as a rule, on an unchanged surface,

Verrucous precancer. A nodule, covered with gray scales, on the red border of the lower lip.

mainly the lower lip, its palpation is painless. The color of the nodule can vary from the normal color of the red border to stagnant red. The color of the nodule can be grayish-red if the surface of the nodule is covered with a layer of hard-to-remove, tight-fitting gray scales.

Histologically, a pronounced limited proliferation of the epithelium due to the expansion of the prickly layer is revealed. There are acanthosis, hyperkeratosis, interspersed with zones of parakeratosis, polymorphism of cells of the prickly layer of varying severity, up to a sharp one. The basement membrane is preserved.

The course of warty precancer is quite fast. The transition to the invasive form of cancer can occur within 1-2 months after the onset of the disease.

Differential diagnosis. Warty precancer should be differentiated from: A papilloma; And warts; And keratoacanthomas.

The final diagnosis is confirmed by the results of histological examination.

Treatment. Removal of the affected area followed by histological examination.

Limited precancerous hyperkeratosis. The focus of hyperkeratosis below the level of the red border of the lower lip, is covered with thin scales.

Limited precancerous hyperkeratosis of the red border of the lips

Limited precancerous hyperkeratosis of the red border of the lips (hyperkeratosis precancerosacircumscripta) is a lesion that looks like a limited area from 0.2 to 1 cm in diameter. The surface of the focus is flat, covered with thin, densely-seated scales, and has a grayish-white color (Fig. 11.61). Scraping cannot remove them. The lesion focus in most patients sinks slightly in relation to the surface of the lip, but sometimes it can rise somewhat above it. On palpation, the seal is not detected.

Histological examination reveals limited proliferation of the epithelium deep into the tissues, often accompanied by discomplexation of spiny cells with hyperkeratosis on the surface. In the connective tissue under the focus of proliferation, polymorphic-cellular infiltration is determined with a large number of plasma and mast cells, which in places destroy the basement membrane.

Differential diagnosis. The disease is distinguished from: A leukoplakia; And lupus erythematosus; And lichen planus.

Haylit Manganotti.

Red erosion with smooth surface on unaltered red

border of the lower lip.

Malignancy occurs 6 months after the onset of the disease.

Treatment. Surgical - removal of the lesion within healthy tissues with histological examination.

Abrasive pre-cancerous cheilitis Manganotti

Abrasive pre-cancerous cheilitis Manganotti (cheilitis abrasiva praecancerosa Manganotti) is a disease characterized by the appearance on the red border of the lower lip of one, less often several erosions. Usually, erosion is located on the lateral areas of the lip, less often in the center or at the corner of the mouth. It has an oval or irregular shape with a smooth, as if polished surface, deep red in color (Fig. 11.62). Erosion is superficial, sometimes covered with a dense bloody or serous crust, which can be removed with difficulty, with slight bleeding. Uncrusted erosions do not tend to bleed. There is usually no compaction of tissue at the base and around the erosion. Erosions in Manganotti cheilitis are usually painless or slightly painful. Sometimes they occur against the background of a slight inflammation, which is characterized by instability. Erosion is different

Pathohistology of Manganotti cheilitis. Violation of the integrity of the epithelium, in the underlying mucous membrane itself, inflammatory infiltration. The integrity of the basement membrane is not traced. The epithelium along the edges of erosion in a state of acanthosis.

a sluggish and persistent course, do not respond well to drug therapy. They can exist for a long time, sometimes spontaneously epithelize, but after a while they reappear in

the same or different places. The timing of epithelialization of erosions in Manganotti cheilitis can vary from 3 months to 2 years.

The duration of the course of abrasive pre-cancerous cheilitis Manganotti before its transformation into cancer is individual. A rapid malignancy of the process may occur (after 4-6 months); in some patients it occurs 5-7 years after the onset of the disease. Clinical signs indicating a possible beginning of transformation into cancer are the appearance of compaction at the base and around erosion, papillary growths on its surface, bleeding after mild trauma, keratinization around erosion.

During a histological examination, an epithelial defect is determined (Fig. 11.63). The epithelium along the edges of erosion is usually in a state of acanthosis, wide epithelial outgrowths are deeply embedded in the underlying stroma. The cells of the prickly layer are in a state of varying degrees of discomplexation and atypia. In the stroma there is a diffuse infiltrate consisting of lymphocytes, plasma cells, histiocytes.

Cytological examination can reveal the phenomena of dyskaryosis of epithelial cells, elements of inflammation, but more often only inflammation is detected.

Differential diagnosis. Abrasive precancerous

Manganotti's cheilitis should be distinguished from:

A erosive form of leukoplakia;

^ erosive form of lichen planus;

A erosive form of lupus erythematosus;

A pemphigus;

And actinic cheilitis;

And exudative erythema multiforme;

A herpetic erosion;

And lip cancer.

Treatment. With Manganotti cheilitis, treatment consists primarily in eliminating local traumatic factors. Reorganization of the oral cavity is carried out, including rational prosthetics. It is strictly forbidden to smoke, eat irritating food, avoid sun exposure. An examination is necessary to identify and treat concomitant general somatic diseases. If the histological examination does not reveal signs of malignancy of Manganotti's cheilitis, then general and local conservative treatment can be carried out for no more than 1-2 months. Inside appoint vitamin A (3.44% solution of retinol acetate in oil or 5.5% solution of retinol palmitate in oil) 10 drops 3 times a day, multivitamins. Locally, applications are carried out with an oil solution of vitamin A, in the presence of background inflammation, ointments with corticosteroid drugs and antibiotics, solcoseryl, etc. In the absence of the effect of conservative therapy, surgical treatment is performed - removal of the focus within healthy tissues with subsequent histological examination.

Lecture number 4.

Topic: Changes in the red border of the lips and oral mucosa in metabolic disorders and pathology of other organ systems.

Knowledge transfer technology models

Class time	Number of students: 20-40

80 minutes	
Activity type	Introduction of informational lectures
Lecture plan 1Changing the red border of the lips 2 Oral mucosa in metabolic disorders 3 Pathologies of other organ systems.	With the disease of gastritis, the secretory state of the stomach is disturbed, in connection with this, various changes also occur in the mucous membrane of the oral cavity. The patient complains of a change in the color of the mucous membrane of the red border of the lips, white crusts and the appearance of superficial cracks. There may be a smoothing of the filiform papillae of the tongue, an increase in the mushroom papillae. Sometimes atrophy of the filiform papillae occurs, only during dental examinations is the appearance of small erosions determined. Patients may feel burns and pains only after eating. This feeling of desquamation can last from 3-5 days sometimes from 2-3 weeks.
Objective of the lesson	Demonstration lecture conversation
Teaching methods	Collective, public
Activity type	Educational development, educational lecture texts, computer projector.
Visual aids on the topic	Methodically equipped auditorium
The setting for the lesson	Устный контроль ,ответы-вопросы технологическая карта лекционных занятий.
Monitoring and evaluation criteria	Learning knowledge.
Objective of the lesson	1. Prepare educational content of the topic 2. Prepare slides for the introduction of the lecture 3. Develop a list of literatures for teaching the subject.

1.2. Technological map of lectures

Stages of work	**Teacher**	**Student**
Preparation stages (10 minutes)	1.Objective of the lesson 2. Preparation of slides for the lecture material 3.Literature on the topic Banchenko G.V., Maksimov Yu.M. Language is the “mirror” of the body. M .: Business center "Dentistry" 2000 - 407 p. Borovskiy E.V. et al. Therapeutic dentistry: Textbook - M., 2004 .-- 840 p. Borovskiy E.V. Danilevsky N.F. Diseases of the oral mucosa. Atlas - M., 1991. - 320 p.	**Listens and records**

	Borovskiy E.V. Mashkillayson A.L. Diseases of the mucous membrane of the mouth and lips. M .: Medpress, 2001. Diseases of the oral mucosa. Ed. L.M. Lukinykh. - Novgorod, 2001	
1. Introduction (15 minutes)	***1. Purpose and objectives of the lecture material:*** ***Target:*** ***preliminary diagnosis and early preparation of the patient*** ***Task:*** ***• Features of the course of caries in childhood and treatment.*** ***• Examination of the patient and develop a treatment plan;*** ***• Develop the main syndromes of dental diseases*** ***• Make a plan for treatment and prevention of stomas. diseases.*** ***2.Questions on the topic*** ***• Classification of dental caries diseases in children?*** ***• Complications of inappropriate caries treatment in children?*** ***• What is the value of fluoridated water in caries prevention?***	**Listen** **Answers students' questions**
2 main stage (55 minutes)	1. Introduction to the topic with slide show	Listen and record Listen
Final step 10 minutes	1. Conclusion. 2. Independent work. 3. Homework.	Listens and records Writes down

Lecture text

CHANGES IN THE MUCOSA OF THE ORAL IN CERTAIN SYSTEMIC DISEASES

The oral cavity is in close connection with various internal organs and systems. Modern clinical and experimental research methods have made it possible to establish a close anatomical and physiological connection with different systems of the body: the receptors of the oral mucosa have extensive connections with the central nervous system, as well as the gastrointestinal tract, endocrine organs.

rinous, cardiovascular and other systems. This led the researchers to the conclusion that most diseases of the oral mucosa are caused by dysfunctions of certain systems, primarily the gastrointestinal tract.

A significant contribution to the study of this problem was made by Russian scientists EE Platonov, BM Pashkov, GV Banchenko, and others.

The first symptoms of some systemic diseases often appear on the oral mucosa, which makes patients first seek help from a dentist. Moreover, changes in the oral mucosa do not have specific manifestations characteristic of a particular somatic disease, which creates difficulties in their correct recognition. Therefore, in some

cases, the participation of doctors of other specialties is necessary for the correct diagnosis.

The mucous membrane of the mouth is involved in the pathological process in various somatic diseases to varying degrees. In this section, we will focus only on those diseases that are accompanied by pronounced changes in the oral mucosa, the knowledge of which is necessary for the early diagnosis of the underlying disease and the choice of the correct treatment tactics for its manifestations in the oral cavity.

11.7.1. Changes in the oral mucosa in diseases of the gastrointestinal tract

Diseases of the gastrointestinal tract are often accompanied by changes in the oral cavity. This is largely due to the morphofunctional unity of the oral mucosa and the entire digestive tract. Studies of many authors have proven that in chronic gastritis, gastric ulcer and duodenal ulcer, chronic colitis and enterocolitis, various changes in the oral mucosa are found, the severity of which depends on the form, severity and duration of the underlying disease. Treatment of the underlying disease leads to a decrease or disappearance of pathological changes in the oral cavity.

The relationship between the violation of the acid-forming function of the stomach and pathological changes in the oral mucosa has been established. The secretion of the stomach, as shown by the studies of S.P. Kolomiets (1970), is closely related to

secretion of the salivary glands. Exacerbation of peptic ulcer disease is accompanied by a decrease in the reactivity of the oral mucosa.

In the works of V.V. Khazanova it is shown that recurrent aphthous stomatitis occurs against the background of intestinal dysbiosis. Many authors have described that in patients with recurrent aphthous stomatitis, changes in the small intestine occur similar to lesions of the oral mucosa.

The data obtained by V.E.Rudneva (1971) indicate that in patients with gastric ulcer, the concentration of histamine in the blood serum increases and at the same time the activity of histaminase decreases, and the activity of hyaluronidase also increases.

One of the important factors causing changes in the oral mucosa in the pathology of the gastrointestinal tract is a deficiency of vitamins, especially of group B.

Thus, as follows from the data obtained by numerous authors, all the above links in the pathogenesis of gastrointestinal diseases (increased vascular tissue permeability, hypovitaminosis, intestinal dysbiosis, imbalance of the immune system, etc.) form a causal relationship with changes in the cavity mouth.

Change of language. In diseases of the gastrointestinal tract, the state of the tongue is best studied. The type of tongue, according to many authors, can have an important diagnostic value and indicate the existing pathology of the digestive tract.

The lingering of the tongue is found most often. Plaque consists mainly of keratinized epithelial cells, bacteria, fungi, food debris. The severity of plaque depends on various reasons. The amount of plaque on the tongue increases with a decrease in its self-cleaning, mainly when chewing. In this case, the composition, consistency of food intake, as well as the regularity of individual hygiene measures and other factors are important. The amount of plaque on the tongue varies during the day: in the morning it is more than in the daytime and evening, since there is a decrease in the amount of plaque after eating. Violation of the process of normal (physiological) keratinization and desquamation of the epithelium also determines the amount and density of plaque.

So, with atrophy of the filiform papillae of the tongue, there is little or no plaque at all. With hypertrophy of these papillae on the surface

The tongue has a hard-to-remove thick layer of plaque, consisting mainly of stuck together keratinized filiform papillae.

The condition of the tongue may indicate disorders of the digestive system. Tongue obstruction is one of the characteristic symptoms of various diseases of the gastrointestinal tract. So, with an exacerbation of gastritis, peptic ulcer, enterocolitis and colitis, the amount of plaque increases, it covers the entire back of the tongue, localizing mainly in its posterior parts. Plaque formation, as a rule, is not accompanied by subjective sensations in patients. Only in the presence of a thick, dense layer of plaque on the tongue can there be a feeling of discomfort and a slight decrease in taste sensitivity. Usually, the coating on the tongue has a grayish-white color, but it can acquire various shades (yellow, brown). The color of plaque is mainly due to dyes of food products, drugs or exacerbations of gastrointestinal diseases: peptic ulcer, chronic hepatitis, cholecystitis (yellow, brown).

The edematous condition of the tongue is an important sign of diseases of the gastrointestinal tract. As a rule, tongue swelling is detected by a doctor during examination, since it does not cause painful sensations, except in cases of significant edema, when the tongue is bitten while eating or talking. The edematous state is determined upon examination by the pronounced imprints of the teeth on its lateral surfaces, as well as an increase in its size. The McClure-Aldrich blister test is used as an objective research method to determine the presence of edema (see Chapter 4 for a detailed description). With the help of a blister test, it is possible to determine the state of latent edema, which will make it possible to diagnose early (preclinical) changes. V.A.Epishev (1970) established a violation of the hydrophilia of the oral mucosa in chronic gastritis. With anacid gastritis, a decrease was found, and with hyperacid gastritis - an increase in the time of resorption of the blister test. It should be borne in mind that swelling of the tongue is a condition associated with chronic intestinal diseases (enterocolitis, colitis), in which a violation of water metabolism is detected.

Changes in the papillae of the tongue are often found in patients with pathology of the digestive tract. The pathogenesis of these changes is mainly trophic disorders.

state, as well as a violation of the vitamin balance due to insufficient assimilation and synthesis of vitamins B ,, B ,,

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Depending on the severity and color of the papillae of the tongue, some authors distinguish between hyperplastic and atrophic glossitis. Hyperplastic glossitis is observed more often in patients with gastritis with high acidity, with exacerbation of peptic ulcer disease. Glossitis is characterized by hypertrophy of the papillae of the tongue, the presence of dense plaque, an increase in the size of the tongue due to pronounced swelling. Atrophic glossitis is found in patients with gastritis with secretory insufficiency, with hepatitis, gastroenteritis, colitis. With this form of glossitis, atrophy and flattening of the papillae of the tongue, the absence of plaque are observed. Sometimes the atrophy of the papillae is pronounced, the tongue is smooth, shiny. The tongue may be hyperaemic (erythematous) or pale pink. In some cases, the tongue looks "lacquered" with bright red spots and stripes, reminiscent of Meller's

glossitis. Atrophy of the papillae of the tongue can cause a burning sensation, soreness, tingling sensation when taking spicy, spicy food.

Desquamation of the epithelium of the tongue is quite often found in diseases of the gastrointestinal tract and can have varying degrees of severity. Desquamation occurs more often in patients with chronic gastritis with secretory insufficiency, chronic colitis, and liver diseases. It is characterized by the appearance on the back of the tongue of foci of desquamation of the epithelium of filiform papillae. In patients with secretory insufficiency, infectious liver lesions, a combination of desquamative glossitis with atrophy and flattening of the papillae of the tongue is often observed. These changes, as a rule, do not cause painful sensations and patients often do not assume their existence, only sometimes they complain of burning sensation, pain when eating irritating food. Characterized by the appearance of foci of desquamation in the period of exacerbation of chronic diseases of the gastrointestinal tract and their disappearance during remission.

Paresthesia of the tongue is often associated with diseases of the digestive system. There is a burning sensation, tingling, tingling sensation of the tongue. These sensations often accompany desquamative glossitis, but they can also appear without visible changes in language (see section 11.7.6 for details).

Violation of taste sensitivity. Taste sense

The vigor of the language is determined by the method of functional mobility of its receptors. It is known that the number of functioning receptors depends on the age and functional state of the digestive tract. Normally, the maximum activity of taste buds is observed on an empty stomach. After eating, there is a decrease in the level of their mobility. The reaction of the taste buds of the tongue occurs in response to incoming excitation impulses from the receptors of the gastric mucosa in a centrifugal way. With a peptic ulcer, tumors of the stomach due to a disorder of its secretory and motor function, the reflex connection of the receptors of the tongue and the stomach is disrupted. This is manifested by various types of violations of the functional mobility of taste buds (an increase in their activity and the absence of demobilization after a meal, etc.). Disturbances in taste sensitivity can also occur with changes in the papillary apparatus of the tongue (heavily coated tongue, phenomena of atrophy or desquamation), which is associated with disturbances in the taste buds of the tongue.

Disorders of the oral mucosa. Erosive and ulcerative lesions of the oral mucosa arising from diseases of the gastrointestinal tract are mainly the result of trophic disorders. There are a large number of reports on the presence of erosions and ulcers on the oral mucosa in case of gastric ulcer, liver disease, colitis, enterocolitis, etc. The relationship between recurrent aphthous stomatitis and gastrointestinal pathology is known. About 50% of patients with diseases of the gastrointestinal tract (chronic gastritis, gastric ulcer and duodenal ulcer) suffer from recurrent aphthous stomatitis of varying severity.

A change in the color of the oral mucosa can manifest itself against the background of gastrointestinal pathology. During the period of exacerbation of peptic ulcer, enterocolitis, colitis, catarrhal gingivitis, glossitis or stomatitis often develop, the severity of which depends on the duration and frequency of exacerbations of the underlying disease. The mucous membrane of the mouth in the affected area is characterized by hyperemia with symptoms of cyanosis due to the chronic course of

the process. In this case, patients complain of a burning sensation in the mouth, discoloration of the mucous membrane, and sometimes soreness when eating irritating food. The most pronounced phenomena of catarrhal gingivitis and stomatitis with exacerbation of chronic colitis. During the period

remission of gastrointestinal diseases, symptoms of catarrhal gingivitis or stomatitis become mild or completely disappear.

It should be remembered that such changes in the oral mucosa can be manifestations of other diseases and conditions of the body (infectious, including fungal, allergic, cardiovascular, hypovitaminosis). In this regard, for a successful diagnosis and choice of treatment methods, a thorough examination of the patient is necessary.

Salivation disorders. May manifest as hyper- or hyposalivation. In the studies of a number of authors it has been proved that morphological and functional changes in the small salivary glands are observed in patients with gastric ulcer and duodenal ulcer. With peptic ulcer disease in the initial stage (up to a year), as well as its exacerbation, an increase in salivation is noted, followed by the development of hyposalivation. Patients begin to complain of dry mouth. Clinically, impaired salivation is often combined with other previously described changes in the oral cavity characteristic of gastrointestinal pathology.

Treatment. As a rule, for the treatment of catarrhal stomatitis, gingivitis or glossitis against the background of gastrointestinal diseases, there is no need for special local treatment. Recommendations are given on the care of the oral cavity and on rational hygiene, if necessary, rinse with antiseptic or anti-inflammatory drugs (chlorhexidine, etonium, tincture of calendula, chamomile, etc.) is prescribed. It is mandatory to treat the underlying disease of the gastrointestinal tract. In addition, given the presence of signs of group B hypovitaminosis and dysbiosis in the oral cavity and intestines in most patients with chronic diseases of the gastrointestinal tract, vitamins of group B should be prescribed internally (multivitamin complexes "Dekamevit", "Pangexavit", "Glutamevit", "Unikap" and others) or, preferably, parenterally (thiamine, riboflavin, pyridoxine, cyanocobalamin).

11.7.2. Changes in the oral mucosa in cardiovascular diseases

With violations of the cardiovascular system, changes in the oral cavity are determined by the degree of circulatory disorders and damage to the vascular wall. Compen-

severe forms of cardiovascular failure, as a rule, are not accompanied by any significant changes in the oral cavity.

Swelling and cyanosis of the oral mucosa, blueness of the lips. Usually observed in de- compensated forms of cardiovascular insufficiency: hypertension, rheumatic heart disease or other diseases. Teeth prints appear on the lateral surfaces of the tongue, cheeks. This condition is often combined with the cyanosis of the red border of the lips. Swelling of the tongue can be expressed to a large extent, as a result of which it increases in size; speech becomes difficult. With myocardial infarction, swelling of the tongue can be combined with changes in its color, the appearance of cracks, erosions and ulcers. The severity of these changes is determined by the severity of the underlying disease. With a large-focal infarction, lesions of the oral mucosa are more pronounced. As the patient's condition improves after general treatment, the situation in the oral cavity improves - the swelling of the tongue decreases, epithelialization of

erosions and ulcers occurs. Puffiness and ulceration of the oral mucosa often appear in areas adjacent to dentures: the marginal edge of the gums adjacent to metal crowns; the mucous membrane under the intermediate part of the bridge; under the prosthetic bed of removable plates, etc.

Despite significant changes in the mucous membrane, most patients do not experience subjective sensations, however, if swelling is detected, the doctor must determine the cause of its occurrence. It is necessary to carry out differential diagnostics with similar changes in the mucous membrane in gastrointestinal pathology, infectious and other diseases.

VASCULAR VASCULAR SYNDROME IN THE ORAL CAVITY. Described by A.L. Mashkillyson et al. (1972). It is characterized by the appearance of dense blisters with transparent or hemorrhagic contents of various sizes (Fig. 11.36). The authors explain the mechanism of blistering in patients with cardiovascular pathology by rupture of small vessels of the oral mucosa as a result of increased
their permeability and fragility of the vascular wall. Along with this, a weakening of the connection between the epithelium and the connective tissue layer of the oral mucosa was noted, apparently associated with the destruction of the basement membrane. T. I. Lemetskoy us-
Rice. 11.36. Vesicovascular syndrome. Bladder with hemorrhagic content on the tongue.

A decrease in the resistance of the capillaries of the oral mucosa to vacuum (Kulazhenko's test) was found in patients with vesicovascular syndrome.

Vesicovascular syndrome occurs more often in women aged 40-75 years. Blisters come on suddenly, often with meals. The appearance of blisters is usually associated with an increase in blood pressure, which patients are often unaware of. Favorite localization - on the mucous membrane of the soft palate, lateral surfaces of the tongue, cheeks. Bubbles can exist unchanged from several hours to several days. Sometimes they disappear without opening, but more often they open up with the formation of erosion, epithelializing within 3-7 days, depending on the size. In smears-prints from the area of erosion, elements of peripheral blood are found, there are no acantholytic cells. Nikolsky's symptom is negative.

Differential diagnosis. Vesicovascular syndrome is differentiated from:

A pemphigus;

And angiomas;

And exudative erythema multiforme.

Treatment. First of all, it is aimed at treating background cardiovascular diseases, without which it is impossible to prevent the reappearance of blisters. Prescribe drugs that normalize the state of the vascular wall (askorutin, compliance).

Local treatment consists of oral sanitation, including rational prosthetics. The mucous membrane of the mouth is treated with antiseptic agents (solution of chlorhexidine, chloramine, etc.), followed by the use of keratoplastic agents (oil solution of vitamins A, E, rosehip oil, sea buckthorn, Actovegin ointment, solcoseryl, etc.). In cases of rapid spontaneous epithelialization of erosions, sometimes within 2-3 days after their formation, special local treatment is not required.

ULTRA-NECROTIC LESIONS OF THE ORAL MUCOSA WITH THE FORMATION OF TROPHIC ULTRASONS. They develop in a number of cases in

patients with I-III degree circulatory disorders. Against the background of a violation of the general condition of patients (weakness, shortness of breath, swelling of the extremities), soreness occurs in the oral cavity, and food intake is difficult.

One, rarely several ulcers appear on the mucous membrane of the mouth. The emergence of trophic ulcers, as a rule, is facilitated by trauma by the sharp edges of decayed teeth, poor-quality prostheses or other traumatic factors. Most often, ulcers form on the lateral surfaces of the tongue, the mucous membrane of the cheeks, the floor of the mouth, palate, etc. The edges of the ulcers are uneven, the bottom is covered with a grayish-white necrotic plaque. If necrotic tissues are not rejected for a long time, they acquire a dark color. A characteristic feature of a trophic ulcer is the absence of a pronounced inflammatory reaction in the surrounding tissue. The necrotic process can spread to adjacent areas of the face, nasopharynx. Bad breath appears, saliva becomes viscous. Cases of ulcerative-necrotic lesions of the oral mucosa against the background of circulatory disorders with necrosis and sequestration of the jaw bone tissue, the formation of defects in the cheek tissues are described. Necrotic tissue breakdown can lead to severe bleeding.

Prolonged existence of trophic ulcers can lead to their malignancy.

Diagnostics. A cytological examination of the papilla from the surface of a trophic ulcer reveals a small number of epithelial cells with signs of degeneration, which is expressed in a decrease in cell size; lack of clear contours in some of the cells, pycnosis. Neutrophils are found in varying degrees of decay. There is a significant decrease in the cells of histiocytic

series, which characterizes the areactive nature of the course of the inflammatory process.

Pathohistologically, a chronic inflammatory process with extensive necrosis and proliferation of interstitial tissue is determined in the area of a trophic ulcer; sclerotic changes in blood vessels and damage to nerve fibers.

Differential diagnosis. A trophic ulcer is differentiated from:

A traumatic ulcer;

^ cancerous ulcer;

^ tuberculous ulcer;

And Vincent's ulcerative necrotizing stomatitis;

^ necrotic lesions of the oral mucosa in blood diseases.

Treatment. Conducted in contact with a therapist. General treatment should be directed to the elimination of cardiovascular failure. In some cases, it is advisable to carry out general treatment in a hospital setting. Symptomatic therapy is carried out locally according to indications. First of all, all local traumatic factors are eliminated. Remove necrotic tissue using proteolytic enzymes. The oral mucosa is treated with antiseptic and epithelial stimulating drugs (oil solution of vitamins A, E, sea buckthorn oil, rose hips, propolis, tsigerol, etc.). A gentle, high-calorie, vitamin-rich diet is prescribed.

11.7.3. Changes in the oral mucosa with endocrine diseases

The endocrine system is a regulator of metabolic processes in the human body. It ensures the consistency of the functions of individual organs and systems, and also controls the balance of a number of body functions (glucose and calcium in the blood, temperature, etc.). In some endocrine disorders, changes in the mucous membrane of

the mouth and the red border of the lips are observed. Clinical manifestations in the oral cavity may necessitate the study of individual functions of the endocrine system. For example, the progressive development of periodontal disease may be associated with diabetes mellitus or other diseases of the endocrine system.

Changes in the oral cavity are most often detected in diabetes mellitus, dysfunction of the gonads, thyroid and parathyroid glands, pituitary gland, adrenal cortex. Therefore, a timely and correct assessment by the dentist of the local manifestations of these diseases in the oral cavity will contribute to their early diagnosis.

DIABETES. It is characterized by a violation of carbohydrate metabolism, against which changes occur in the oral cavity. Their severity depends on the severity and duration of diabetes mellitus. Oral manifestations are often the first sign of illness.

Xerostomia - dryness in the mouth caused by decreased salivation, the severity of which varies from patient to patient. Dryness is often combined with increased thirst and appetite, burning and tingling in the area of the tongue, lips, gums; sometimes accompanied by itching and burning in the genitals or other parts of the body. On examination, the mucous membrane of the mouth is dry, shiny, edematous and hyperemic, especially on the tongue. Along with this, paresthesias may occur.

Differentiate xerostomia against the background of diabetes mellitus from salivation disorders in disorders of the nervous system, Sjogren's syndrome, sialoadenitis and sialodochitis, Mikulich's disease.

Catarrhal, erosive and ulcerative stomatitis, glossitis. Inflammation of the oral mucosa or its individual areas in diabetes mellitus occurs as a result of a decrease in its resistance due to violations of redox processes, dysbiosis in the oral cavity, imbalance of local immunity and other reasons. All this leads to a change in the reactivity of the oral mucosa to local stimuli, a decrease in resistance to infection; there is a predisposition to inflammation, the healing processes of erosions and ulcers slow down. Even a slight effect of a traumatic factor leads to inflammation of the oral mucosa and in some cases to a violation of its integrity with the formation of cracks, erosions and ulcers. In this case, patients complain of pain that occurs when eating hot, spicy or dry food. Erosions and ulcers are characterized by a long course due to a decrease in the regenerative properties of the mucous membrane.

In diabetes mellitus, periodontal tissues are also quite often affected. First, catarrhal gingivitis develops with severe swelling and bleeding of the gums.

Subsequently, it rather quickly transforms into periodontitis, characterized by a progressive course with pronounced inflammatory and destructive changes in all periodontal tissues. Periodontal abscesses are common. Moreover, the more severe the course of diabetes mellitus and the longer its duration, the stronger the damage to the periodontal tissues. Especially rapid destructive processes in the periodontium are characteristic of young patients with diabetes mellitus.

Fungal stomatitis, mycotic seizure. In diabetes mellitus, candidiasis is a fairly common form of oral pathology. Favorable conditions for its development are dysbacteriosis in the oral cavity in patients with diabetes mellitus, a violation of the acid-base balance with the accumulation of under-oxidized metabolic products (lactic and pyruvic acids) in the tissues. Candidiasis of the oral mucosa in diabetes mellitus is characterized by a persistent and persistent course. Mycotic seizure is common, in

which cracks form in the corners of the mouth, covered with small grayish-white crusts.

For the diagnosis of candidiasis, the presence of general symptoms of diabetes mellitus, laboratory data (an increase in glucose in the blood and urine), as well as the results of a bacterioscopic examination of scrapings from the surface of the oral mucosa are important.

Antifungal therapy is carried out in consultation with an endocrinologist treating diabetes mellitus.

Paresthesia, taste disturbance. In patients with diabetes mellitus, along with dryness and burning of the oral mucosa, paresthesia sometimes occurs. With the decompensated form of diabetes mellitus, some patients have a decrease in taste sensitivity to sweet, salty, sour, and in some cases to bitter. Sometimes there is a disorder of the nervous system (neuritis, neuralgia of the branches of the trigeminal nerve).

In diagnostics, data from anamnesis, including family history, are important; a picture of changes in the oral cavity and the results of laboratory tests (blood, urine).

Treatment is carried out in contact with an endocrinologist. Along with the treatment of diabetes mellitus, a thorough debridement of the oral cavity and symptomatic local therapy are carried out.

ITSENKO-CUSHING'S DISEASE. It develops after-

the result of hyperfunction of the adrenal cortex with damage to the hypothalamic-pituitary system. Increased production of steroids by the adrenal cortex leads to osteoporosis of bone tissue, including the jaws, and predisposes to periodontal disease. Some patients develop macrocheilitis. Candidiasis is common. Trophic ulcers can form on the mucous membrane of the tongue, cheeks and other parts of the oral cavity.

Treatment. The underlying disease is treated by an endocrinologist. Local treatment includes oral debridement. Symptomatic therapy is carried out according to indications.

ADDISON'S DISEASE. It occurs as a result of insufficient function of the adrenal cortex. One of the first signs of the disease may be pigmentation of the skin and mucous membranes, caused by excessive deposition of melanin in the connective tissue and epithelial cells. Skin pigmentation appears in areas exposed to light (face, back of the hands). As the disease progresses, the skin becomes light brown or bronze in color. On the mucous membrane of the mouth in the cheeks, along the edges of the tongue, on the palate, gums, small (from one to several square millimeters) spots or stripes of grayish-black, dark brown or bluish-gray appear without signs of inflammation (Fig. 11.37). They do not stand out above the level of the mucous membrane. At the same time, patients have no subjective sensations. The general well-being of patients may suffer. Asthenia, gastrointestinal disturbances (nausea, vomiting, diarrhea, abdominal pain), headaches, memory loss, weight loss are noted.

Differential diagnosis. Addison's disease is differentiated from:

^ congenital pigmentation of the oral mucosa, observed in some peoples;

^ intoxication with salts of heavy metals.

Treatment. The main disease is treated by an endocrinologist. No special local treatment is required.

MIXEDEM. A disease resulting from severe hypothyroidism. Mostly women are ill. The main symptom of myxedema is a widespread mucous edema of the skin and subcutaneous tissue. Edema is formed as a result of a violation of lymph outflow and the accumulation of specific mucus in the organs and tissues

Rice. 11.37. Pigmentation of the mucous membrane of the tongue with addison-

that substance, consisting of glycosaminoglycans, which retain water and form edema. In a pronounced stage of the disease, the patient's face has a characteristic appearance: puffy, pale, lips, nose are thickened, upper eyelids are sharply swollen, almost close the eye slit. The tongue is significantly enlarged in size (macroglossia), teeth imprints are visible on its lateral surfaces. There is anemicity, swelling and dryness of the oral mucosa. Due to the swelling of the mucous membrane of the larynx and vocal cords, the timbre of the voice decreases; speech slows down. The general condition of patients is characterized by weakness, drowsiness, lethargy, sluggishness, loss of memory and hearing. With hypothyroidism in childhood, enamel hypoplasia, hyposalivation, high intensity of caries lesions are observed, the timing of teething is delayed; cretinism develops.

Differentiate swelling of the tongue with myxedema from its swelling with other general somatic diseases described in this section.

Treatment. General treatment is carried out by an endocrinologist and consists of thyroid replacement therapy. No specific topical treatment is indicated. The oral cavity is sanitized.

DIFFUSION TOXIC GOITER (thyrotoxicosis, Graves' disease). It develops as a result of hyperplasia and hyperfunction of the thyroid gland. Patients complain of a burning sensation of the oral mucosa, a decrease in taste sensitivity, a deterioration in general well-being

(shortness of breath, increased fatigue, irritability, sweating, etc.). On examination of the oral cavity, the mucous membrane is pale, edematous. Desquamative glossitis is sometimes detected. Accelerated teething, an anomaly in the development of enamel are characteristic.

Treatment. The general treatment is carried out by an endocrinologist. Local treatment is carried out when indicated.

JUVENILE (YOUNG) GINGIVITIS. It occurs in boys and girls during puberty, with menstrual irregularities. The change in the oral mucosa occurs as a result of the action of the gonadotropic hormones of the pituitary gland. Local factors contribute to the development of juvenile gingivitis: poor oral hygiene, malocclusion, etc. The mucous membrane of the gums is swollen, hyperemic, bleeds easily. Hypertrophy of the gingival papillae, the marginal edge of the gums, develops, mainly in the area of the anterior teeth of the lower jaw.

Treatment. Systematic hygiene procedures, dental calculus removal, and oral cavity sanitation are required. Orthodontic treatment is carried out according to indications. Drug treatment of hypertrophic gingivitis is carried out according to the generally accepted method described in Chapter 9.

GINGIVITIS OF PREGNANT WOMEN. It develops in connection with pregnancy or worsens during pregnancy. It is manifested by hypertrophy of the interdental papillae, and sometimes the marginal edge of the gums. "False" pockets are formed, bleeding of the gums occurs. The severity of its course can vary. Sometimes

hypertrophied papillae reach the incisal edge of the crowns of the teeth or their chewing surface, which causes soreness and bleeding when eating. The symptoms of pre-pregnancy gingivitis are in most cases exacerbated. After childbirth, the gum is often transformed into its previous (normal state), but sometimes (with poor oral hygiene, tartar, poor-quality prostheses, occlusion anomalies and other local traumatic factors), the phenomena of gingival hypertrophy persist.

Treatment. -With gingivitis of pregnant women, first of all, careful systematic care of the oral cavity, the use of anti-inflammatory toothpastes ("Forest", "Chamomile", "Zodiac", etc.) is required. If after childbirth the phenomena of hypertrophic gingivitis persist, its traditional treatment is carried out according to the generally accepted method.

11.7.4. Changes in the oral mucosa in diffuse connective tissue diseases

These include a group of diseases, the common feature of which is a diffuse inflammatory lesion of the connective tissue, characterized by fibrinous degeneration and sclerosis. The term used earlier to designate this group of diseases "collagenosis" is not used in the modern international classification of diseases, since it does not reflect the essence of their pathogenesis. It has been established that in these diseases, not only collagen is affected, but also other components of the connective tissue.

The group of connective tissue diseases includes systemic lupus erythematosus, systemic scleroderma, dermatomyositis, Sjogren's disease, rheumatoid arthritis. Their main clinical feature is polysyndromism, that is, the presence of changes in almost all organs and systems (skin, mucous membrane, muscles, joints, internal organs) due to a single pathogenesis - diffuse lesions of connective tissue.

Changes in the oral mucosa in diseases of the connective tissue, as well as in other diseases of internal organs, is a local manifestation of general pathology.

Desquamative glossitis is a fairly common lesion of the mucous membrane of the tongue in patients suffering from connective tissue diseases (systemic lupus erythematosus, systemic scleroderma, rheumatoid arthritis).

Xerostomia has a different degree of severity in various diseases of the connective tissue. To the greatest extent, salivation is reduced in Sjogren's syndrome. In a number of patients, it reaches a significant degree, which reduces the resistance of the oral mucosa to the effects of traumatic and infectious factors, and also causes difficulties in patients with eating and talking.

Ltrophine stomatitis, glossitis, gingivitis. Damage to the oral mucosa is a common symptom of scleroderma. In this disease, the mucous membrane of the mouth is pale, atrophic, thinned, as a result of which its vascular pattern is clearly visible. Telangiectasias often appear. The red border of the lips is also atrophic, thinned. There is a pronounced atrophy of the papillae of the tongue, it takes on the appearance of a varnished. On defeat

muscles of the tongue, it can decrease in size. The mucous membrane of the gums is compacted, anemic; atrophy of the marginal part of the gums is often noted. Due to the severity of atrophic processes, the resistance of the oral mucosa decreases. It becomes easily traumatized, long-term non-epithelializing erosion or ulcers appear.

Generalized cutaneous lesions are typical of scleroderma. Their nature is determined by the clinical form of scleroderma. So, with a plaque form, lesions on the skin look like spots or stripes of bluish-pink color, somewhat rising above the skin level. Their

central part is gradually sclerosed, acquiring an ivory color, and a purple ring is preserved along the edges, indicating the further progression of the process. With a superficial form of scleroderma, skin lesions look like multiple white spots, with the resolution of which foci of atrophy remain. The skin is cold to the touch. On the face, the skin is also thinned, anemic, with multiple telangiectasias. Sometimes there is pain in the temporomandibular joint, as a result of which the opening of the mouth may be limited. The defeat of the skin and mucous membrane of the mouth goes through the stages of edema, induration and atrophy. Lesions are localized mainly in the face (mask-like) and hands (sclerodactyly). The skin becomes yellowish, thickens, telangiectasias occur.

Treatment. Patients with lesions of the connective tissue are treated by rheumatologists, therapists, etc. Dental care consists primarily in a thorough sanitation of the oral cavity. It is very important to educate patients in rational oral hygiene, given the reduced resistance of the oral mucosa to the effects of microflora. In some cases, due to the limitation of opening the mouth, the performance of a number of dental procedures may have some peculiarities. You should also take into account the ongoing sclerotic processes in the oral mucosa in these diseases in connection with the injection of anesthetics or other drugs. To prevent injury and detachment of the periosteum, drugs should be administered slowly and in small quantities. Taking into account the pathogenesis of connective tissue diseases, it is necessary to examine patients to identify and eliminate foci of odontogenic infection.

11.7.5. Changes in the oral mucosa in diseases of the hematopoietic system

Diseases of the blood and hematopoietic organs are often accompanied by changes in the oral mucosa, which in some cases appear earlier than the clinical signs of the underlying disease. This forces patients to seek help from the dentist first. The dentist is often the first to encounter such patients, so he must be well versed in the manifestations of the pathology of the hematopoietic system in the oral cavity. This will make it possible to diagnose the disease in a timely manner and carry out its treatment. In addition, the inability to recognize a disease of the hematopoietic system leads to an erroneous diagnosis and, as a consequence, the appointment of incorrect treatment, which can lead to a significant deterioration in the patient's condition. The dentist must know the scope of necessary and acceptable interventions in the treatment of patients with diseases of the hematopoietic system. Unjustified dental intervention can lead to profuse bleeding from the socket of the extracted tooth or during curettage, as well as to the progression of necrotic processes in the oral cavity and even death.

The psychological and deontological aspects of the first contact of a dentist with a hematological patient are very important. It is necessary to be able to carefully and convincingly explain to the patient the need for some special examination methods or consultation with a hematologist in connection with the specific picture of lesions of the oral mucosa. When communicating with a patient, it is very important not to frighten him or cause psychological trauma, since fear of a malignant neoplasm (acute and chronic leukemia) is a natural condition for most people ..

Treatment of patients with diseases of the hematopoietic system is associated with the use of special means and methods. As a rule, it is carried out in specialized hematological institutions. The volume of dental treatment procedures is determined

by the severity and stage of the clinical course of the underlying disease and is carried out by a dentist in some cases in a dental or specialized hematology department.

ACUTE LEUKEMIA. It is characterized by an increase in the number of blast, "young" cells in the bone marrow, with

lesenka, lymph nodes and other organs and tissues. Depending on the morphological and cytochemical characteristics of blast cells, several forms of acute leukemia are distinguished: myeloblastic, lymphoblastic, plasmablastic, etc. The lesion of the oral mucosa is observed in 90.9% of patients with acute leukemia. It occurs mainly at a young age (up to 30 years).

In the diagnosis of acute leukemia, an important role belongs to general symptoms (general weakness, malaise, fatigue, pallor of the skin). Body temperature can be high with large differences, but sometimes it is subfebrile. The patient gives the impression of being seriously ill. Due to a sharp decrease in the body's defenses, patients with leukemia sometimes develop candidiasis, and herpes infection in the oral cavity manifests itself. Blast cells are present in the blood, and the number of platelets and red blood cells is often reduced.

Oral symptoms are of great importance in recognizing acute leukemia. They are very characteristic and easy to spot. On examination, pallor of the skin, pallor, pastiness, slight vulnerability and bleeding of the oral mucosa, hemorrhages on the gums, cheeks (especially along the line of teeth closing), palate, tongue are noted. Hematomas and hemorrhages can be observed both on the mucous membrane and on the skin.

Bleeding of the gums is very often the first clinical sign of acute leukemia. At the same time, the gums become loose, bleed, ulcerate. Bleeding occurs at the slightest touch, and sometimes spontaneously. Bleeding may occur not only from the gums, but also from the tongue, cheeks along the line of closing the teeth and other areas of the oral mucosa. Sometimes, extensive hemorrhages and hematomas of the oral mucosa are found. The clinical manifestations of hemorrhagic syndrome can be different: from small-point hemorrhages on the oral mucosa and skin to extensive hematomas and profuse bleeding. Hemorrhagic syndrome is detected in 50-60% of patients and is based on pronounced thrombocytopenia, which develops as a result of suppression of normal hematopoiesis due to leukemic hyperplasia and bone marrow infiltration.

Differentiation of hemorrhagic syndrome in acute leukemia follows from hypovitaminosis C, traumatic injuries (especially on the mucous membrane of the cheeks along the line of closing the teeth). The final diagnosis is established on the basis of the clinical picture of the disease,

Rice. 11.38. Acute leukemia.

Necrotizing ulcerative gingivitis with gingival hyperplasia and bleeding.

general blood test and bone marrow punctate examination.

Gingival hyperplasia is a common symptom of acute leukemia, especially in its severe course, and is assessed by hematologists as an unfavorable prognostic sign. Hyperplasia and infiltration of the gums with leukemic cells can be so significant that the crowns of the teeth are almost completely closed by a loose, bleeding, ulcerated shaft in places that prevents the patient from eating and talking. It is characteristic that in a significant part of cases, already at the very beginning of the disease, hyperplasia is more pronounced on the inner (palatal or lingual) surface than on the buccal. This

clinical symptom sometimes helps to differentiate gingival hyperplasia in leukemia from the banal hypertrophic gingivitis.

According to histological data, gingival hyperplasia is caused by infiltration of the connective tissue layer of the mucous membrane by myeloid cells, which leads to disruption of trophism, followed by tissue necrosis and ulceration.

Necrotic ulcerative lesions of the oral mucosa often develop in acute leukemia. It is characteristic that the apex of the gingival papilla undergoes necrosis. Around the focus of necrosis, the gums have a bluish color, while the entire mucous membrane is pale, anemic (Fig. 11.38). Necrosis spreads quickly and soon around the tooth

an ulcer develops with a dirty gray fetid bloom. Often, necrosis is detected on the tonsils, in the retromolar region and other parts of the oral cavity. A feature of the necrotic process in acute leukemia is its tendency to spread to adjacent areas of the mucous membrane. As a result, there may be extensive ulcers with irregular contours, covered with a gray necrotic plaque. Reactive changes around the ulcer are absent or weak. In the presence of necrotic changes in the oral cavity, patients complain of sharp soreness when eating, difficulty swallowing, putrid fetid odor from the mouth, general weakness, dizziness, headache. At the beginning of the development of ulcerative necrotic changes, hypersalivation may occur, and then the amount of saliva decreases, which is associated with dystrophic processes in the salivary glands.

The causes of necrotic processes on the oral mucosa in acute leukemia have not been completely clarified. It is believed that necrosis can occur as a result of the disintegration of leukemic infiltrates, in areas of extensive hemorrhage, as well as due to neuro-trophic disorders in tissues and a violation of the body's defenses. An important point that determines the occurrence of necrosis is the action of external factors, especially microorganisms.

Lymph nodes in acute leukemia increase slightly - up to 0.5-1 cm, they are soft, painless. In some cases, acute leukemia can occur with the formation of pronounced specific leukemic infiltrates in the skin and oral mucosa. As a result of the formation of infiltrates in the gums, a sharp deformation of the gingival margin occurs. This condition is often diagnosed as hypertrophic gingivitis. Leukemic infiltrates can ulcerate, which often leads to bleeding.

Differential diagnosis. Differentiating the manifestations of acute leukemia on the mucous membrane follows from:

^ hypertrophic gingivitis of other etiology;

^ ulcerative necrotizing stomatitis of Vincent;

^ hypovitaminosis C;

And intoxication with salts of heavy metals.

The results of blood tests are decisive in the diagnosis of leukemia.

Treatment. The task of the dentist is the correct and timely recognition of acute leukemia by clinical

some symptoms in the oral cavity and other manifestations, analysis of peripheral blood. Treatment of acute leukemia is carried out in a specialized hematology department. Local treatment is carried out in consultation with the hematologist. Oral hygiene is very important. Treatment and extraction of teeth, removal of tartar is carried out under the supervision of a hematologist in a hospital. In the presence of ulcerative necrotic lesions of the oral mucosa, it is anesthetized, treated with antiseptic

solutions (hydrogen peroxide, chloramine, etonium, romazulan, furacilin, etc.), proteolytic enzymes and agents that stimulate epithelization (rosehip oil, sea buckthorn oil, propolis preparations, oil solution of vitamins A, E, etc.). When detecting fungal or herpetic lesions of the oral mucosa, conventional antifungal or antiviral therapy is performed, described in detail in the relevant sections of the textbook.

CHRONIC LEUKEMIA. Chronic leukemia, depending on the nature of the lesion of the hematopoietic organs, is divided into myeloproliferative (myeloid leukemia) and lymphoproliferative (lymphocytic leukemia) forms. Chronic myeloid leukemia goes through two stages: benign, lasting several years, and malignant (terminal), which lasts 3-6 months. The first stage often begins without pronounced clinical symptoms, then neutrophilic leukocytosis appears with a shift to promyelocytes and single blast cells. In the second stage, as in the first during blast crises, there are many blast forms in the blood (myeloblasts, hemocytoblasts), there is a rapid increase in the number of leukocytes in the blood. As leukemia progresses, anemia and thrombocytopenia, accompanied by hemorrhages, develop.

Chronic myeloid leukemia is asymptomatic for a long time and can be accidentally diagnosed during a clinical blood test. The blood picture is characterized by high leukocytosis (50-60 * 109 / l). In a blood smear, various intermediate forms of the myeloid series are found: myeloblasts, promyelocytes, myelocytes.

Hemorrhagic manifestations are characteristic of chronic myeloid leukemia, although their intensity is much less than in acute leukemia. Bleeding of the gums does not occur spontaneously, but when injured. The mucous membrane of the mouth is pale. The gums are swollen, cyanotic, bleed at the slightest touch, there is no inflammation. Absence

inflammation and severe bleeding are characteristic symptoms for leukemia.

Erosive and ulcerative lesions of the oral mucosa, according to V.M. Uvarov (1975), are observed in patients with myeloid leukemia. The appearance of severe necrotic lesions is considered as an unfavorable sign indicating an exacerbation of the process. In the severe stage of the disease, candidiasis often develops.

Chronic lymphocytic leukemia usually develops in middle-aged and elderly people, is characterized by a long-term benign course and a significant increase in the number of leukocytes in the blood (lymphocytes prevail among them). The number of leukocytes can reach 500 109 / l.

The initial period of chronic lymphocytic leukemia often goes unnoticed by the patient. The only complaint may be an increase in lymph nodes (on palpation, they are not very dense, mobile, painless). Leukemic lymphocytic infiltrates or tumor-like nodes are formed in the oral cavity in chronic lymphocytic leukemia. They are more often located on the mucous membrane of the gums, cheeks, tongue, palatine arches, tonsils. The resulting infiltrates (nodes or nodules) have a soft (pasty) consistency, bluish color, mobile, rise above the level of the mucous membrane. Ulceration is rare. Bleeding occurs with minor injury. Leukemic lymphocytic infiltrates are also localized in the bone marrow, lymph nodes, spleen, and liver.

Treatment. General and local treatment is carried out in the same way as for acute leukemia.

AGRANULOCYTOSIS. A syndrome characterized by a decrease in the number or complete disappearance of granulocytes (granular leukocytes) from the peripheral

blood. By the mechanism of occurrence, myelotoxic and immune agranulocytosis are distinguished. The cause of myelotoxic agranulocytosis is a violation of the formation of granulocytes in the bone marrow as a result of exposure to the body of ionizing radiation, cytotoxic drugs, benzene vapors, etc. drugs (amidopyrine, butadione, analgin, phenacetin, sulfonamides, some antibiotics, etc.).

Agranulocytosis often begins with the manifestation of changes

niy in the oral cavity, which forces patients to go to the dentist at the onset of the disease. Against the background of a deterioration in the general condition of patients (fever, lethargy, headache, pain when swallowing, odor from the mouth), ulcerative-necrotic processes develop on the oral mucosa, which tend to spread rapidly. The destruction of the oral mucosa proceeds without a pronounced leukocyte reaction of the surrounding tissues and insignificant pain. Fetid ulcers that bleed easily. Sometimes the jaw bone tissue is also involved in the ulcerative-necrotic process. The gums, lips, tongue, cheeks, tonsils are more often affected. In case of damage to the tonsils, pain appears when swallowing. In some patients, the appearance of ulcers along the digestive tract is noted. The lymph nodes are enlarged. The number of lymphocytes in the blood is significantly reduced (from 1 to 3 109 / l, and sometimes less than 1 109 / l). In the leukocyte formula, the number of granulocytes is sharply reduced or they are completely absent, only lymphocytes and monocytes are found.

Diagnostics. The diagnosis is made on the basis of anamnesis, clinical presentation, results of a study of peripheral blood and bone marrow punctate.

Differential diagnosis. Agranulocytosis should be differentiated from necrotizing ulcerative stomatitis of Vincent and other blood diseases.

Treatment. General treatment is carried out under the supervision of a therapist and hematologist, first of all, blood transfusion, elimination of the etiological factor, etc.

Local treatment includes the use of analgesics, application of proteolytic enzymes to necrotic areas of the oral mucosa, antiseptic treatment and the use of drugs that stimulate epithelialization.

THROMBOCYTOPENIC PURPLE. A syndrome characterized by increased bleeding due to a decrease in the number of platelets in the blood. Thrombocytopenic purpura can be an independent disease - Verlhof's disease, as well as develop as a result of other diseases (malignant neoplasms, radiation sickness, hepatitis, infectious diseases, drug intoxication, etc.) - a symptomatic form.

Idiopathic thrombocytopenic purpura, 'or Werlhof's disease, can occur in acute or chronic

form. The main clinical symptom of the disease is hemorrhage in the skin, mucous membranes, as well as bleeding from the nose, gums, etc., arising spontaneously or. under the influence of minor trauma. Hemorrhages are observed in the serous membranes, retina and other parts of the eye. Hemorrhages in the heart and brain are life-threatening. Patients have a slight increase in body temperature and a pronounced thrombocyte singing (up to 20 109 / l or less), as a result of which the retraction of the blood clot is impaired and the bleeding time is lengthened up to 10 minutes or more (normally 3-4 minutes). As a result of massive bleeding, iron deficiency anemia can develop.

Diagnostics. The diagnosis of thrombocytopenic purpura is established on the basis of anamnesis data (detection of contact with various chemicals, uncontrolled intake of analgesics, hypnotics, etc.), clinical picture and blood test.

Differential diagnosis. Differentiate the disease from:

^ hemophilia;

^ grief;

^ hemorrhagic vasculitis.

Treatment. General treatment is carried out by a hematologist, as a rule, in a hospital setting. Dental interventions should be performed with extreme caution after consulting a hematologist.

POLYCYTHEMIA (erythremia, or Vakez's disease). Chronic disease of the hematopoietic system, characterized by a persistent increase in the number of erythrocytes and hemoglobin per unit volume of blood, along with an increase in the volume of circulating blood. The disease is associated with increased production of erythrocytes, leukocytes and platelets in the bone marrow. It develops gradually and has a long chronic course. It occurs more often in men aged 40-60 years. There is a change in the color of the skin and mucous membrane of the mouth. They acquire a dark cherry color with a cyanotic hue, which is due to the increased content of reduced hemoglobin in the capillaries. Bleeding of the gums is common. Bleeding from the stomach, intestines, uterus is possible. Characterized by a dark cherry color with a cyanotic shade of lips and cheeks. In the oral cavity, Kuperman's symptom is typical - pronounced cyanosis of the mucous membrane of the soft palate and pale coloration of the hard palate. Many patients have

pruritus, paresthesias of the oral mucosa, associated, according to many authors, with increased blood vessels and neurovascular disorders. The picture of peripheral blood is characterized by an increase in the content of erythrocytes to 6-8-10 | 2 / l), hemoglobin (180-200 g / l), a decrease in ESR to 1-3 mm / h.

Treatment. Carried out by a hematologist. Special local therapy is not indicated.

ANEMIA. This is a group of various pathological conditions characterized by a decrease in the hemoglobin content and (or) the number of erythrocytes per unit of blood volume. Anemias are hereditary and acquired. According to the modern classification, anemias can develop as a result of blood loss (posthemorrhagic), increased destruction of red blood cells (hemolytic) and due to a violation of the formation of red blood cells.

Some forms of anemia, along with general manifestations, are characterized by typical symptoms in the oral cavity. These are primarily anemias that develop as a result of a violation of the formation of erythrocytes - deficiency anemias associated with insufficiency of such hematopoietic factors as iron (iron deficiency anemia), as well as vitamin B | 2 and folic acid (megaloblastic anemia).

Iron deficiency anemia, hypochromic. The causes of the disease are associated with insufficient intake of iron in the body or its increased consumption (during growth, pregnancy, lactation, with increased sweating), as well as with endogenous disorders leading to insufficient absorption of iron (with resection of the stomach, intestines, achilic gastritis, gastroenteritis , chronic colitis, endocrine disorders, etc.).

The clinical picture. It is characterized by a violation of the general condition of the patient: pallor of the skin, weakness, dizziness, tinnitus, cold clammy sweat, a decrease in body temperature and blood pressure, an increased tendency to fainting.

Along with general symptoms, changes in the oral cavity are noted, which have a certain diagnostic value. Thus, paresthesias and disturbances in taste appear long before the decrease in the level of serum iron and the development of obvious anemia. On examination, the mucous membrane of the mouth is pale, atrophic, insufficiently moisturized. Patients are worried about dry mouth, difficulty swallowing food, burning and pain in the tongue, lips,

eaten while eating, taste perversion. Patients eat chalk, tooth powder, raw cereals. Cracks often appear in the corners of the mouth and on the lips (especially in patients with anacid gastritis and after resection of the stomach). The back of the tongue is hyperemic, shiny. The papillae of the tongue are atrophic. The tongue is very sensitive to all mechanical and chemical stimuli. There is soreness when taking spicy, sour, hot food. Some swelling of the tongue is noted, which is determined by the imprints of the teeth on its lateral surfaces. In a number of patients, there is a loss of the natural gloss of the enamel, increased abrasion of the teeth.

Diagnostics. The diagnosis is made on the basis of anamnesis data, clinical manifestations, but the decisive moment is the blood picture. It is characterized by hypochromic anemia - a decrease in the hemoglobin content and a low color index (0.5-0.6 and below), anisocytosis and poikilocytosis, as well as a decrease in the content of iron and ferritin in the blood serum.

Treatment. General treatment is aimed at eliminating the causes of the anemia and iron deficiency. Prescribe iron supplements, vitamins. They recommend a balanced diet.

Local treatment is aimed primarily at eliminating traumatic factors. The oral cavity is sanitized. According to indications, symptomatic therapy is prescribed, aimed at eliminating cracks in the red border of the lips, normalizing salivation, eliminating burning sensation, paresthesia and others.

Anemia B (2-deficiency and (or) folate deficiency (malignant, or pernicious, Addison-Birmer anemia). Characterized by impaired erythropoiesis. Vitamin B | 2 and folic acid are necessary factors for normal hematopoiesis. Deficiency of vitamin B | 2 violates the process of maturation of erythroblasts in the bone marrow, resulting in the formation of immature cells - megaloblasts, megalocytes, which quickly die. Deficiency of vitamin B | 2 in the body is a consequence of the absence of Castle's internal factor - a gastromucoprotein produced by the gastric mucosa, without which it does not occur assimilation of vitamin B | 2 (external factor) Vitamin B12 deficiency can be caused by atrophic processes in the gastric mucosa, gastric resection, inflammatory bowel disease or resection of certain areas of the intestine, vitamin B | 2 deficiency in food.

Folic acid deficiency anemia occurs when folic acid absorption is impaired (in patients with alcoholism, sprue, etc.), with insufficient intake of folic acid from food, with the appointment of certain antiepileptic drugs.

Vitamin B12-deficiency anemia is more common in people over 40 years of age. The appearance of patients is characteristic: a pale face, skin with a yellowish tinge, thin, atrophic, hair turns gray early. The general condition suffers, weakness and fatigue

develop. The clinical picture is characterized by damage to the hematopoietic tissue, nervous and digestive systems.

Complaints of patients about pain and burning of the tip or edges of the tongue, paresthesia, in most cases are the first, ahead of the further development of symptoms of anemia.

On examination, a pale, atrophic with an icteric tinge of the mucous membrane is revealed, sometimes there are punctate hemorrhages on the cheeks, bleeding of the gums.

Changes in language are characteristic. It becomes smooth, shiny, polished due to atrophy of the papillae, thinning of the epithelium and muscle atrophy. Bright red spots may appear on the tongue, when they merge, the entire tongue becomes bright red (Gunther's glossitis, Genter-Möller). It is in stark contrast to the pale mucous membrane in the rest of the mouth.

Diagnostics. The diagnosis is made on the basis of the history data, the clinical picture of the disease and the results of a blood test. Hyperchromic anemia is observed in the blood - a decrease in the number of erythrocytes saturated with hemoglobin; the color indicator is 1.1-1.3; erythrocytes are large (megalocytes). There are erythrocytes with nuclear remnants and giant hypersegmented neutrophils. Examination of bone marrow punctate reveals a large number of megaloblasts.

Treatment. Prescribe vitamin B12 at 100-500 mcg and folic acid at 0.005 g per day. In severe cases, the administration of vitamins is combined with blood and red blood cell transfusions. Changes in the oral cavity go away quickly after general treatment.

Local treatment is not required. The oral cavity is sanitized, according to indications, agents are prescribed for local anesthesia of the oral mucosa (2% trimecaine solution, 1-2% pyromecaine solution, anesthesin in glycerin, etc.).

11.7.6. Changes in the oral mucosa in diseases of the nervous system

The mucous membrane of the mouth and tongue is a zone in which a powerful flow of afferentation to the central nervous system (CNS) is formed. At the same time, a number of diseases of the nervous system significantly alter the formation of the flow of afferentation and the processing of information from the periphery by the central nervous structures. Compensatory changes in the oral mucosa may occur. More often, diseases of the nervous system manifest themselves in the form of unpleasant sensations in the oral cavity. Only in rare cases are morphological changes in the mucous membrane observed (erosion, ulcers in patients with organic lesions and CNS injuries). With stomalgia (glossalgia) and taste disturbance, the mucous membrane of the mouth usually does not change, but these diseases are quite relevant for dentistry and neurostomatology.

11.7.6.1. Stomalgia (glossalgia)

Stomalgia (syn: glossalgia, glossodynia, paresthesia of the tongue) is a chronic disease with persistent orofacial pain, usually not accompanied by damage to the mucous membrane of the mouth and tongue.

In 1838, Weisse gave the first scientific description of stomalgia. Many works have been published on various aspects of this suffering, but to date, the issues of etiology, pathogenesis and treatment of stomalgia have not been finally resolved.

Due to the unclearness of the mechanism of the development of the disease, there is no single designation for stomalgia. Many terms have been proposed: burnt tongue

syndrome, glossalgia, or dysesthesia of the oral cavity, glossodynia, vegetative tongue, sensory neurosis of the tongue, glossoperosis. R. M. Basker, D. M. Main (1991) propose a new term for this suffering - "burning mouth syndrome", considering that it more accurately reflects the leading complaint of patients.

Etiology and pathogenesis. A number of authors suggest an increased level of monomers in the acrylic bases of prostheses as the cause of stomalgia, but not all confirm an allergic reaction to acrylates of prostheses as an important cause in the development of the disease. More likely causes are mechanical irritation with prostheses and parafunctional habits. It was believed that in

galvanic currents are of great importance for the development of stomalgia, but the results of recent studies have shown that this is not the case. Stomalgia patients have hypersensitivity to mercury, nickel, cobalt, gold, palladium, copper, zinc and food additives. The hypersensitivity reaction to mercury was the most frequent, however, replacing the amalgam fillings relieved the burning sensation in the oral cavity in less than half of the patients.

Stomalgia may develop after the use of enalopril, captopril and isinopril (angiotensin-converting enzyme inhibitors). A large number of chemical agents and drugs have been described that can cause pathological changes in orofascial localization. With this pathology, approximately 20% of the prevalence of xerostomia is observed. Complaints of dry mouth may not always indicate the hypofunction of the salivary glands. Attempts have been made to determine the existence of a link between stomalgia and salivary gland dysfunction. There is evidence of a decrease in the function of the salivary glands in stomalgia.

Given that the majority of patients with stomalgia are menopausal women, many authors suggest that dry mouth may be due to a decrease in the functional activity of the salivary glands during menopause. However, most researchers have shown that there is no significant decrease in the function of the salivary glands during menopause. The cause of xerostomia can be concomitant somatic pathology and the use of certain drugs for its treatment.

During the histological examination of the salivary glands, some authors indicate changes in morphology, while others reject these data.

The clinical picture. With stomalgia, pain can often be classified as dysesthesia of hard-to-determine localization. The pain is superficial and deep, mainly at the tip and along the edges of the tongue, can spread to the mouth, pharynx, lips and be combined with a burning sensation in the mouth, dry mouth, thirst, taste disturbances, anxiety, irritability, and a reduced desire for social interaction. The level of pain in stomalgia, although different in quality, is similar in intensity to toothache.

In the case of dysesthesia and paresthesia, the burning sensation appears in the morning, intensifies during the day and reaches a maximum in the evening, weakening or completely disappearing with food intake. The daily rhythm of occurrence is clearly traced

pain. It is repeated for months with periodic exacerbations. This condition sometimes appears after an affective trauma, death or cancer of a loved one, with personal and professional difficulties.

Most often, women suffer from stomalgia, mainly during menopause. They feel irritation in the mucous membrane of the tongue and mouth, which is usually dry and

similar to that of the elderly. The excitement of the tongue joins. Patients examine their language for a long time, trying to detect changes in it. All these symptoms are combined with short-term general excitement, aggravated by hot flashes. There is a fear of the possible development of cancer.

Many patients with stomalgia complain of a taste disorder called dysgeusia. Dysgeusia was detected in 68% of patients with stomalgia, especially for bitter food.

The response to local anesthesia differs in patients in the groups with dysgeusia, burning sensation in the oral cavity and in the group with a combination of dysgeusia and burning sensation.

It was found that dysgeusia is of peripheral origin, since its decrease after the application of local anesthesia is noted. Apparently, local anesthesia can relieve peripheral depression of the central sensory structures in these patients. The lack of influence on the burning sensation of local anesthetics justifies the use of medications acting on the central structures.

Stomalgia symptoms subside with exposure to hot and spicy foods. This effect is associated with the capsaicin found in spicy food. A good effect was revealed when using capsaicin (a preparation of capsicum for external use) in the treatment of orofacial pain. Intraoral triggered trigeminal neuralgia was less responsive to topical capsaicin therapy than stomalgia.

In patients with stomalgia, a correlation was found between the level of beta-endorphin and the severity of stomalgia.

The activation of the sympathoadrenal system was revealed (the level of vanillin mandelic acid, which is a metabolite of adrenaline, was determined in the urine of patients, the content of which was significantly higher in patients than in healthy people). At the same time, a correlation was found between the level of vanillin mandelic acid and the severity of paresthesia of the tongue. Based on this, the authors concluded that the pathogenetic role of sympathetic activation

adrenal system in the development of stomalgia - an increase in the content of adrenergic substances affects peripheral receptors and central mechanisms of perception of sensations.

Electroencephalographic studies in stomalgia gave conflicting results: in some studies, a shift with a predominance of rhythm activation phenomena was revealed, which is typical for any pain phenomenon, in others, an increased functional activity of synchronizing structures was described.

Stomalgia should be considered along with other pathological manifestations of chronic emotional stress.

Many authors point to the frequent combination of stomalgia with diseases of the gastrointestinal tract, cardiovascular pathology, osteochondrosis, endocrine and autoimmune pathology, and diseases of the nasopharynx. Others do not find a connection between stomalgia and these pathological conditions.

It is assumed that hormonal changes during menopause may be a factor provoking the development of discomfort in the oral mucosa. A number of researchers reported a decrease in the intensity of oral symptoms and psychopathological manifestations (depression, anxiety) in most postmenopausal women who were treated with estrogens. Researchers associate the success of estrogen therapy with the presence of estrogen receptors in the oral mucosa in some women.

Emotional changes (especially anxiety and depression) are detected in stomalgia to such an extent that many authors consider this pathology as psychogenic. In recent years, psychological testing has been included in the examination plan for all patients with stomalgia. The most commonly used are the Swedish and Karolinska personality scales, the scale of mental functioning, the scale of the quality of life, etc.

It was found that, in comparison with the control group, patients with stomalgia have significantly lower indices in the socialization scale, higher in somatic anxiety in muscle tension and in psychoasthenic scales. According to a number of authors, up to 95% of patients with stomalgia note the presence of anxiety and depression. Patients had significantly higher characteristics of depression (an indicator of the severity of chronic symptoms) than in the sample of patients with somatic pathology, but lower than patients with mental illness with clinical depression. The same correlation can be seen in the level of anxiety.

The most characteristic feature of stomalgia is the presence of depression with sleep disturbance, pronounced neurovegetative reactivity and somatic response to stress. However, latent depression is also revealed. While numerous studies point to a link between depression and stomalgia, not all support this conclusion unequivocally.

Nerve damage or dysfunction that occurs with oral or facial trauma, as well as after medical interventions, can be a source of pain in stomalgia. Burning pain is characteristic of pain conditions associated with nerve damage. Chronic pain can be associated with changes in the sympathetic nervous system as well as with impaired blood flow.

While peripheral lesions can be provoking factors, the leading factors in the pathogenesis of stomalgia are changes in the central nociceptive structures.

Recent studies have shown that damage or inflammation of peripheral tissues or nerves can create neuroplastic changes in the central nociceptive systems, which are given an important role in stomalgia. Given the possibility of such changes, methods of treating stomalgia associated with damage to facial tissues and nerves (tooth extraction, etc.) should be abandoned: they are usually ineffective and can aggravate neuroplastic changes and the progression of symptoms.

Differential diagnosis. Neuralgia and neuropathy of the lingual nerve have a clinical picture similar to stomalgia, in which sensory disturbances in the form of pain or hypesthesia are more pronounced. However, the main difference between stomalgia is the disappearance of all painful manifestations during a meal.

Treatment. In the treatment of stomalgia, a complex application of therapeutic measures is necessary: drug therapy, physiotherapy, acupuncture, laser therapy, hyperbaric oxygenation and psychotherapy. Drug therapy includes the use of antidepressants, benzodiazepines, antihistamines, sex hormones, vitamins and sialic supplements, antifungal drugs, analgesics, vasoactive drugs, and vegetotropics.

Antidepressants are widely used for the treatment of painful conditions for more than 30 years. The usual daily doses of antidepressants are 25-100 mg. Doses of antidepressants should be limited if anticholinergic side effects occur (dry mouth, constipation, poor accommodation, urinary retention). Patients, especially those with concomitant cardiovascular pathology, may experience complications (orthostatic hypotension, arrhythmia).

Benzodiazepines are often given to patients with chronic pain, although long-term use can stimulate or worsen depression. The most commonly used is phenazepam at a dose of 0.25-0.5 mg 2-3 times a day, tazepam (daily dose 30-90 mg), mezapam (daily dose 10-30 mg). During the period of exacerbation of discomfort with stomalgia, a course of Relanium is prescribed intramuscularly, and in severe cases of the disease, antipsychotics are used, for example, haloperidol at a dose of 3.6-15 mg per day.

To improve microcirculation, especially in people with vascular diseases and degenerative changes in the cervical spine, use pentoxylline at a dose of 300-600 mg per day, nicotinic acid, nikoshpan, no-shpu, stugeron, cavinton.

It is necessary to use vitamins (parenteral administration of thiamine, riboflavin, pyridoxine, cyanocobalamin, ascorbic acid; oral administration of tocopherol acetate and retinol). It is recommended to take vegetative preparations: anticholinergics (bellaspon, belloid, platifillin); anticholinesterase drugs (galantamine); ganglion blockers (gangleron, benzohexonium). Antihistamines are used - pipolfen, diphenhydramine, suprastin.

In order to inhibit peripheral mechanisms, local anesthetics are used (a suspension of anesthesin on glycerin, solutions of trimecaine and lidocaine) in the form of applications, oral baths, aerosol irrigation.

To reduce the phenomena of xerostomia in order to stimulate the function of the salivary glands, pilocarpine is used, 5 mg 1 time per day sublingually, vitamin A, potassium iodide. Bromhexin is used to change the consistency of saliva. Siala substitutes of artificial and natural origin are used as substitution therapy, for example salenum, a water-soluble flaxseed extract.

In the complex treatment of stomalgia, acupuncture is being used more and more widely and with high efficiency. The effect of auriculoelectro-

puncture. There is a high therapeutic efficacy from the use of percutaneous electroneurostimulation [Borisova EG, 1993].

Physiotherapeutic procedures are widely used: massage of the collar zone and head, galvanization of the upper cervical sympathetic nodes, endonasal electrophoresis of novocaine, etc. Many authors point to the positive effect of laser therapy, which plays the role of a biostimulator and has an analgesic effect.

A method of treating glossalgias with medical leeches is used, which is especially effective in patients with diseases of the cardiovascular system.

There are indications of the positive effect of the use of hyperbaric oxygenation and the use of oxygen when it is injected with a syringe into the mucous membrane of the mouth, cheeks and on the lateral surfaces of the tongue.

According to a number of authors, an effective course of psychotherapeutic treatment, consisting of hypnotherapy and autogenous training.

Reorganization of the oral cavity, grinding of the sharp edges of the teeth, restoration of the occlusal height are required. Despite the availability of a large number of drugs and techniques used to treat stomalgia, their effectiveness is low, and therefore requires joint efforts of doctors of different specialties - dentists, neurologists and psychiatrists - to achieve success in the study and treatment of stomalgia.

11.7.6.2. Taste disturbance

Disturbance of taste (dysgeusia) occurs with pathology of the facial nerve in the event that the lesion is localized in the canal of the facial nerve (fallopian canal). This is due

to the fact that on this segment of the facial nerve, a drum string passes next to it, which contains taste conductors that carry information from the taste buds of the anterior 2/3 of the tongue to the cerebral cortex. Accordingly, in this case, a one-sided loss of gustatory sensitivity in the front 73 of the tongue is revealed.

Outside the canal of the facial nerve, taste fibers are located in the lingual nerve. Consequently, even with its defeat, there will also be a loss of taste sensitivity in the front 2/3 of the tongue, but in combination with other symptoms. It should be noted that one hundred

Early loss of gustatory sensitivity in the posterior tongue is associated with damage to the glossopharyngeal nerve.

Complete bilateral, less often unilateral loss of gustatory sensitivity occurs with organic lesions of the central nervous system (brain tumors, neurosyphilis, etc.).

Disturbance of taste can manifest itself in its complete loss, decrease and perversion. This applies to all types of sensitivities (sweet, salty, sour, bitter) or only some of them.

Taste bulbs are located mainly in the mucous membrane of the tongue, therefore the etiological factors affecting them, and, therefore, causing taste disturbances, are diverse: viral infections, allergic glossitis, radiation damage, injuries, vitamin deficiencies A and B, Sjogren's syndrome, gastrointestinal diseases tract, etc. In this case, there is increased sloughing or temporary loss of the epithelium in the tongue, in connection with which the taste buds are exposed and become available for excessive irritation. In such cases, gustatory sensitivity is restored as the papillae regenerate.

With a strong overlapping and extensive lesions of the tongue with leukoplakia, the taste often decreases, which is associated with the difficult access of flavoring substances to the taste buds. A sour feeling can occur in patients with galvanic phenomena. Gingivitis, periodontitis, chronic tonsillitis, periodontitis can become a source of unpleasant taste in the oral cavity. A feeling of bitter, sour can appear with hepatocholecystitis, gastritis. Perversion or loss of taste is possible with hysteria.

Treatment. Due to the fact that taste disturbances often occur with a lesion localized in the canal of the facial nerve, where taste fibers pass in the -composition of the facial nerve, with pathology of the lingual nerve (which also includes taste fibers), in the medulla oblongata (the nucleus of the glossopharyngeal nerve), as well as in hysteria, treatment is most often carried out by a neuropathologist according to the principles of treatment of the corresponding diseases and taking into account their etiology.

11.7.6.3. Salivation disorder

Pathology of the innervation of the salivary glands and damage to the salivary glands themselves and the mucous membrane of the oral cavity often cause impaired salivation.

There are two types of impaired salivation: hypersalivation and hyposalivation (with its extreme degree - xerostomia).

HYPERSALIVATION. The etiological factors causing hypersalivation are acute inflammatory processes of the oral mucosa, accompanied by an unconditioned reflex increase in saliva secretion due to sharp irritation of the mucous membrane, which is a positive protective reaction of the body. In some cases, hypersalivation occurs as a result of direct irritation of the secretory cells of the salivary glands with mercury, iodine when these substances enter the body.

Prolonged hypersalivation is observed in some organic diseases of the central nervous system - parkinsonism, bulbar and pseudobulbar paralysis, in some cases after a stroke, etc. Swallowing disorders in these and other diseases enhances hypersalivation. The cause of hypersalivation may be helminthic invasion.

With neurosis, obsessive states, false hypersalivation is possible, when patients complain of profuse salivation, and this is not confirmed by an objective examination.

Patients with hypersalivation, in addition to a dentist, should be examined by a neurologist and a therapist if indicated.

Treatment. With hypersalivation, treatment in all cases should be aimed at eliminating the underlying disease that caused this condition. A temporary decrease in salivary secretion can be achieved with the use of atropine preparations.

HYPOSALIVATION. The reasons for hyposalivation are diverse: avitaminosis A, B, E, hyposiderosis; salivation decreases in diabetes mellitus, systemic connective tissue diseases, radiation injury, with bilateral chronic parotitis, after surgical removal of the large salivary glands. Dryness of the oral cavity is most pronounced in patients with Sjogren's syndrome. An increase in the tone of the sympathetic nervous system in thyrotoxicosis, in the climacteric period, in neurotic conditions leads to hyposalivation. As a temporary phenomenon, xerostomia can occur in acute infectious diseases, with botulism, after taking certain medications (for example, the atropine group). In people with impaired nasal breathing (polyps, curvature of the nasal septum), constantly forced

breathe through the mouth, dryness of the oral cavity is caused by increased evaporation of fluid from the oral cavity (false hyposalivation). Dry mouth only at night can be the result of sleeping with an open mouth, which is more common in old age. It should be noted that in old and senile age, salivation decreases, therefore, factors that reduce it are stronger in these age groups.

Hyposalivation and especially xerostomia cause in patients a feeling of dry mouth, difficulty in eating and speaking, pain from spicy and solid food, burning sensation, roughness of the mucous membrane. Hyposalivation is often associated with dryness of other mucous membranes.

On physical examination, the mucous membrane is poorly moistened or dry, dull. There is little saliva, it is frothy or completely absent. The active growth of microflora (due to the shutdown of the protective effect of saliva) leads to inflammation of the mucous membrane, the occurrence of erosions, ulcers, injuries when eating. In patients with prolonged hyposalivation, the amount of dental plaque increases, and multiple dental caries occurs.

Treatment. It consists in eliminating the cause of hyposalivation. Reorganization of the oral cavity is mandatory, including prosthetics. Locally, it is recommended to use anti-inflammatory drugs and agents that protect the mucous membrane from irritation (borax on glycerin, lysozyme, peach, sunflower oil, etc.).

As a rule, vitamin therapy is prescribed (vitamins A, Bp B6, B12, C, E), according to indications - sex hormones; iodine preparations inside. A good effect is provided by galvanizing the area of the large salivary glands. Anticholinesterase drugs are successfully used as symptomatic agents, in particular, a 0.5% galantamine solution, 1.0 ml subcutaneously, by electrophoresis, or by mouth daily for 1 month. Prescribe 1% solution of pilocarpine hydrochloride, 4 drops 1-2 times a day

Lecture number 5

Topic number 5. Introduction to physiotherapy. The role of physiotherapy in the prevention and rehabilitation of dental patients.

1.1. Technological models for education

Lesson time 80 minutes	Number of students 20-40
Activity type	Introduce lecture news
Lecture plan 1. An introduction to physiotherapy. 2 The role of physiotherapy in prevention. 3 And rehabilitation of dental patients.	Introduction to Physical Therapy. The role of physiotherapy in the prevention and rehabilitation of dental patients.
Objective of the lesson	Background The role of physiotherapy in the prevention and rehabilitation of dental patients
Teaching methods	Conversation, lecture visuals
Activity type	common-collective
Visual aids on the topic	Study guide, lecture material, projector, computer
The setting for the lesson	Methodical equipped classroom
Monitoring and evaluation criteria	Oral survey

1.2. Technological map of lectures

Stages of work	**Teacher**	**Student**
Preparation stages (10 minutes)	1.Objective of the lesson 2. Preparation of slides for the lecture material 3.Literature on the topic Banchenko G.V., Maksimov Yu.M. Language is the “mirror” of the body. M .: Business center "Dentistry" 2000 - 407 p. Borovskiy E.V. et al. Therapeutic dentistry: Textbook - M., 2004 .-- 840 p. Borovskiy E.V. Danilevsky N.F. Diseases of the oral mucosa. Atlas - M., 1991. - 320 p. Borovskiy E.V. Mashkillayson A.L. Diseases of the mucous membrane of the mouth and lips. M .:	**Listens and records**

	Medpress, 2001. Diseases of the oral mucosa. Ed. L.M. Lukinykh. - Novgorod, 2001	
1. Introduction (15 minutes)	***1. Purpose and objectives of the lecture material:*** ***Target:*** ***preliminary diagnosis and early preparation of the patient*** ***Task:*** ***Introduction to Physical Therapy. The role of physiotherapy in the prevention and rehabilitation of dental patients.*** ***• Develop the main syndromes of dental diseases*** ***• Make a plan for treatment and prevention of stomas. diseases.***	**Listen** **Answers students' questions**
2 main stage (55 minutes)	1. Introduction to the topic with slide show	Listen and record Listen
Final step 10 minutes	1. Conclusion. 2. Independent work. 3. Homework.	Listens and records Writes down

Lecture 5

Alternating electric currents of high frequency

Darsonvalization is a treatment with high-frequency (100-300 Hz) pulsed alternating sinusoidal

lump of high voltage (20 kV) and low power (0.02 mA). In this case, the intervals between their individual

current pulses are many times longer than their duration

pulse rate. These currents were first obtained by Fran

Zuz physiologist, physicist d'Arsonval in 1892.

Physiological action of d'Arsonval currents.

With local darsonvalization, when contact between

the skin and electrode are loose, a stream of small sparks jumps from it onto the skin. Patient testing

at the same time, there is a slight tingling sensation, there is an expansion

vascular rhenium, acceleration of microcirculation, the skin turns red. Due to the low current strength, the feeling of warmth

la is poorly expressed. The currents of d'Arsonval affect the tro

physical properties of tissues, increase tissue metabolism, have an analgesic, anti-spastic effect on blood vessels and sphin

cters. In the skin and oral mucosa, you

are round cell infiltration and foci of mic

ronecrosis, which causes nonspecific activation

tion of protective tissue processes. If the electrode is in direct contact with tissue, it forms

A quiet electrical discharge that has less physiological effect than a spark discharge.

With local darsonvalization, segments appear
tary and general reflex reactions, since afferent impulses of the act from the zone of influence
into the central nervous system, and from there efferen
in this way, a regulating reflex effect occurs, which leads to the normalization of muscle tone
latura and blood vessels, improving trophism.
Indications for the use of local darsonvalization.
Assign for:
wounds and ulcers;
fracture of the jaws;
periodontal disease, periodontitis;
catarrhal, hypertrophic gingivitis;
acute arthritis of the temporomandibular joint;
periodontitis;
simple form of lichen planus;
glossalgia;
trigeminal neuralgia. Contraindications to the use of darsonvalization:
malignant neoplasms;
insufficiency of the cardiovascular system;
a tendency to bleeding;
active tuberculous process.
Apparatus
Technique and methodology of darsonvalization
1. Prepare the device for operation:
install the mains voltage switch, races
placed on the back wall, so that the indicator of its insertion exactly matches the mains voltage (127 or 220 V);
connect the resonator to the connector on the front panel of the device; ground the machine and turn on the plug
ku the cord into the network;
required vacuum electrode and
twist into the resonator until it stops;
turn the mains switch 1 to position "1", while the signal lamp comes on;
check the supply high voltage using the measuring device 3. To conduct a pro
The indicator hand should be located within the shaded part of the scale. If in position
the switch of the network "I", it is not installed on the color sector, then this is achieved by moving it further to position "2", "3", etc .;
Used electrodes are washed with soap, treated
are stored and stored immersed to the base in a jar with
side in alcohol, it is impossible to boil (they are sterilized by the block method in alcohol for 20 minutes).
Local procedures are carried out on an area of up to 600 cm2, lasting from 2 minutes to 20 minutes with low, medium
and high discharge intensity, which determines

It is the patient's subjective sensations as well as the location of the power knob. The course of treatment is from 3 to 30 procedures.
Private methods
Darsonvalization of the gums is contact (Fig. 28). Vacuum gingival, ear or nasal electrode iso
lined with a rubber tube so that it remains from
covered only the tip is long
noah 1-2 cm. The electrode is inserted in the vestibule of the oral cavity and placed along the transition fold of the lower or upper jaw. Turning the "Power" knob, supply voltage
living until fiole appears
glow, crackling and slowly, without touching the teeth, move the electrode along the alveolar process
lusty. At the end of the impact
effects reduce stress
to zero, and then remove the electrode.
Spark darsonvalization of the gums. Gingival, ush
nasal or nasal vacuum electrode is insulated by
with a zine tube so that only the tip 1–2 cm long remains open.
voltage before sparking. The exposure time is 3-5 minutes. For a course of treatment up to 5-6 impacts, hedgehog
day or every other day.
8. Darsonvalization of the wound on the face through the
knitting. Mushroom vac
smart electrode overlays
put on the bandage, if its thickness does not exceed 2 cm, increase the voltage until crackling appears, use
cover and move elect
kind by bandage. Time is
action 8-10 minutes. The course of treatment has 6-10 effects, which are carried out daily or every other day. There are other techniques (Fig. 30).
Diathermy, diathermocoagulation
Diathermy is the application of a high frequency (1-2 Mgu) electric current of low voltage (150-200 V) and a high power of 2 A for therapeutic purposes.
Diathermocoa is widely used in dentistry.
Walking, The method of diathermotomy finds limited application in surgical dentistry.
With direct contact of the active electrode, heat is generated in the tissues, proteins and blood are coagulated, and absorption of tissue products is prevented.
pada, the ingress of microorganisms into the vascular bed (the vascular membrane is tucked inward, the blood is
bleeding is prevented, nerve receptors are destroyed, which reduces pain).
Indications for the use of diathermocoagulation.
Applied for:
coagulation of the contents of root canals with pulpitis and periodontitis;
removal of benign neoplasms if
zystoma of the oral cavity (hemangiomas, papillosis

we, fibroids);
removal of granulation tissue from pathological
kih gingival pockets.
Contraindications to the use of diathermocoag
rations:
lack of cardiovascular system;
individual intolerance.
There are several types of devices. In stomato
logic, a special model of the DKS-2M dental diathermocoagulator is used (Fig. 31). On the front panel of the device there are: The monoactive electrode is placed in a holder with a collet clamp. To insert the electrode into the holder, unscrew the clamp cap
ma, insert the electrode into the collet and, screwing the cap, clamp it.
Technique and technique of diathermocoagulation
1. Prepare the device for operation (connect to the mains, ground, turn on the mains switch,
heat for 3-5 minutes, insert the electrode into the hole for
load, close it, set the required power
ness).
Prepare the surgical site. Perform di-athermocoagulation.
Diathermocoagulation with pulpitis:
conduct anesthesia (conduction, infiltration);
Treat the root canal with hydrogen peroxide, alcohol.
Dry the root canal, fill it
on the tooth.
R.I. Mikhailova proposes to warm it up for 8 seconds at a current of 35-40 mA before filling the tooth canal.
Each canal of a multi-rooted tooth is treated separately.
Private methods
Diathermocoagulation neoplasm mucus
that shell of the oral cavity. DKS-2M apparatus
press to work. When adjusting the power through the "equivalent", it is set at the 12-15th division of the measuring instrument scale. It is better to use a "wire loop" or "ma
a lazy little knife ”included in the set for the device.
During coagulation, the operating field is isolated from saliva and dried. In the koa process
gulation periodically remove the coagulant film formed on the electrode.
Diathermogingivotomy. Anesthesia is performed and the DKS-2M apparatus and the operating field are prepared for work. As an electrode, a "small
cue knife ", which horizontally simultaneously dissect the gum in the area of 4-5 teeth.
Diathermocoagulation of granulations in pathologists
gum pocket. Prepare apparatus DKS-2M for work. Power through the "equivalent" set are at the 12-15th division of the scale of the measuring device
ra. It is better to use a thick root needle with a blunt or bent end in the form of a small loop as an electrode.
with previously produced devices for diathermy UDL-200M, UDL-350M.

Ultra-high frequency electric field
UHF therapy is the use of alternating electro
richesky field of ultra-high frequency (40 MHz EPUHF) for therapeutic purposes.
Physiological actions. Under the influence of electric
the magnetic field in the tissues, the ions oscillate and the charged particles are oriented along the lines of force
holes. This causes the conversion of electrical energy into heat. In tissues with high resistance (not
ditches, bones, tendons), heat generation is most intense. Oscillatory effect leads to change
natural rhythms of biophysical and chemical
chemical processes. At the same time, new conditions are created for the course of physiological reactions.
UHF electric field:
causes vasodilation, especially deep
tissues, activates blood and lymph circulation (while tissue nutrition improves, they can
she to resist various influences);
increases the permeability of the walls of blood vessels, thus
leukocyte formation, phagocytosis, which contributes to a faster resorption of exudates and a decrease in edema
ness of tissues;
activates metabolism, enzymes
tative activity;
activates cellular, tissue, defense mechanisms.
The physiological response is more dependent
sieves on the intensity of the applied electric field. So, a low-intensity field is exerting you
strong anti-inflammatory effect. Electri
medium intensity field well stimulated
stimulates metabolic processes, of high intensity - stimulates
increases inflammation due to the breakdown of large protein molecules with the formation of amino acids
lot. Therefore, when appointing an UHF electric field, it is necessary to take into account the severity and stage of the pathological
whom process.
UHF electric field acts anti-inflammatory
spasmodic, antispasmodic, stimulates regeneration
tion of damaged tissues.
Indications for use:
acute, purulent inflammatory processes;
trauma to soft tissues and jaws;
• erosive and ulcerative lesions of the skin, oral mucosa;
diseases of the nerves of the face. Contraindications for use.
malignant formations;
blood diseases;

insufficiency of the cardiovascular system;
hypotonic illness;
pregnancy. Apparatus
At present, the models of devices UHCh-66 (Fig. 32), UHCh-30 and "Miniterm" are used.
All devices can be operated for 6 hours a day with a 5-minute break every 30 minutes of operation.
What is the technique and method of using electrically
of the UHF field?
What kind of dental diseases is the appointment
What is the UHF EP?
What are the physiotherapy apparatus
you but uhf therapy?
When is an ultra high frequency alternating magnetic field (UHF Pe MP) applied?
What is the technique and technique of microwave treatment
mi?
What is Franklinization?
What is the Franklinization technique and technique?
Target setting.
Have an idea of:
phototherapy;
mechanism of action on body tissues
high light, infrared, ultraviolet, laser
radiation.
Know:
technique and technique of physiotherapy for light therapy
nia;
indications and contraindications for the use of light therapy in dentistry.
Phototherapy is a method of physical therapy in which
rum on the body of a sick person is dosed
bathroom infrared, visible, ultraviolet, laser radiation.
Visible light can be obtained if a beam of sunlight
light through a trihedral quartz prism. At the same time, stripes of all colors of the rainbow (red, orange
chewy, yellow, green, blue, blue, purple).
According to its physical properties, light is a stream of electromagnetic oscillations in the optical range with a wavelength of 400 microns to 2 nm (nanometer ~ 10 9 or 1 billionth of a meter.) The visible part of the spectrum consists of radiation with a wavelength of 760 - 400 nm. On both sides of the relatively narrow frequency band of the electric
irritating multiple recipes
moat embedded in the skin. In this case, the corresponding impulses are sent to the central nervous system
the topic of what causes functional shifts in the organ
nism, morphological changes occur and

biologically active substances are developed (vitamin D2, protein breakdown products, etc.). Biologically active substances enter the blood and lymph and eye
They have an effect on the entire body. The permeability of tissues for rays of different lengths is different: the longer the wavelength of the visible rays, the deeper they penetrate
cabins in leather and vice versa.
The biological effect is only absorbed
naya energy. Sources from
radiation can be calories
thermal (thermal) and luminescent, when the candle
is caused not by heating, but by the phi
zico-chemical processes
yourself. The source of visible radiation is a Sollux lamp, which is a powerful incandescent lamp.
livinia placed in a special
special reflector on a tripod (fig. 40). The lamp also emits infrared
light.
Physiological action
infrared radiation
This is based on its heat effect. Penetrating into the light-thermal depth of fabric up to 14 mm and movable
absorbed by these tissues, infrared rays cause
increase in temperature, which leads to the acceleration of physicochemical and biological processes. In this case, there is an expansion of blood vessels, an acceleration of blood
current, hyperemia occurs, tissue pro
loss and metabolism, phagocytosis, oxidant
processes, muscle tone decreases, spasm with
ships. Infrared rays help to improve tissue nutrition, accelerate cell proliferation and re generation of tissues, accelerate the healing of wounds, ulcers,
pains. Skin irritation leads to reflex vasodilation and hyperemia.
Indications for the use of infrared radiation
cheniya.
Infrared radiation is shown when:
• chronic inflammatory processes of the maxillofacial area;
to accelerate suppuration;
post-traumatic period;
burn;
frostbite.

Apparatus
The source of infrared radiation - irradiators, where the source is a metal spiral, there is a reflector and a tripod, light-thermal irradiators "Sol
deluxe ", which can be stationary, so
ny and portable. Great spread in a hundred

matology received a table lamp "Sollux" (LSN-1, OSN-70), portable - LSN-4 and stationary - LSS-6 can be used.
The radiation source is a table lamp with a power of 200 to 1000 W, it can also be used Xia emitter type Lik-5, in which cylinders from fire
stubborn clay with a filament, placed in the center of a chrome reflector. In Sollux lamps, the light source is a tungsten filament supplied by the city current, which is placed in a glass cylinder filled with an inert gas. Filament temperature
yarn reaches 2300 ° -2700 ° C. As infrared
emitter, a Minin lamp is used, blue light
that, with a power of 200 W, while excluding from the oblast
the rest of the segments of the light spectrum.
Technique and technique of infrared irradiation
1. Prepare the device for the procedure:
place the lamp on the side of the patient at the level of the pathological focus at a distance of 50-100 cm;
insert the plug of the power cord a
the parate into a wall outlet (if there is a switch, turn it to the appropriate position);
warm up the device for 2-3 minutes;
turn on the procedural clock.
2. Prepare the patient for the procedure:
sit down (lay down), put on goggles for him - with cardboard instead of glasses;
to familiarize the patient with possible sensations
mi (light pleasant warmth)
Carry out the procedure.
Turn off the illuminator.
Note. After the end of the procedure, the patient should rest for 20-30 minutes. The duration of the procedure is 20-40 minutes, the course of treatment is up to 20 procedures daily or every other day.
When using a Minin lamp, the distance is
gully usually according to the patient's sensation, it is 15-25 cm from the irradiated area of the body, the procedure is carried out 1-2 times a day for 10-15 minutes.
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For the prevention of latent (latent) infections before surgery, 8-10 high-frequency electrotherapy procedures are prescribed for 10 days
rapia - UHF and CMW therapy.
Physiotherapy treatment of certain diseases of the oral mucosa, diseases of the tongue and lips
Decubital ulcer
Of the physical factors for the treatment of decubital ulcers, you can apply:
irradiation with short UV rays (irradiation on
repaired from 1 biodose, increasing in each subsequent
visit for 1 biodose. The course of treatment is 4-5 pro
daily).
local darsonvalization of the ulcerative surface within 3-5 minutes, 4-5 impacts;
aerosol therapy;
aeroinotherapy (20-30 minutes, course - 15 procedures);

franklinization.
Chronic recurrent aphthous stomatitis
Apply:
general UV irradiation according to the generally accepted method, 2 times a year, in the period November - April to improve the body's resistance during the remission period
levania;
UV irradiation of the aorta in the oral cavity, 5-6 exposures daily or every other day, start with 2 biodoses and uve
each subsequent irradiation is increased by 1 biodose;
Giaava 9. Physical treatments ..
• local darsonvalization, with a short spark not
mediocre for each aorta 1-3 minutes;
• franklinization (impact on collar
zone);
aerosol therapy (with novocaine - from 2 to 15 minutes
chickpeas 2 times a day up to 30 procedures, with medicinal substances up to 10-15 impacts daily);
hydrotherapy - irrigation with antiseptics and mi
neral waters.
Exudative erythema multiforme
For the treatment of this disease, the following is prescribed
other physiotherapy procedures:
• total UV exposure (November - April) in the period
remission;
• aeronization (with antiseptics, novocaine, chemo-
trypsin);
general franklinization;
electrophoresis of magnesium on the collar zone;
• UV irradiation with short or integral spectrum
thrombus lesions on the mucous membranes, the course of treatment - 8-12 procedures;
• aeroionotherapy;
• UHF electric field in athermal dose or microwave therapy for submandibular lymphatic
kie nodes (with a pronounced reaction). Power 1-3 W, duration 5-6 minutes, 3-5 procedures daily.
Lichen planus
From physical methods for this disease
you can assign:
• local darsonvalization by contact or dis
by the tantalizing method for 3-5 minutes on the focus
excitement. The course of treatment - 10-12 impacts daily
but or every other day;
• electrophoresis of nicotinic acid alternately with vitamin Bj. Two courses of drug electrophoresis are recommended. The first course is electropho

cut of nicotinic and ascorbic acids. The second course is electrophoresis of vitamin Bg with trimecaine. About
lead 20-30 impacts;
UV irradiation - local for the erosive and ulcerative form of the disease;
act first with 1 biodose, bringing up to 3-4 biodoses (5-6 procedures in total);
aerosol therapy with an antiseptic (novocaine, tri-mecaine), yegalipt, enzymes;
• aeroionotherapy;
• galvanized collar, galvanized seats
ny sympathetic nodes.
Viral diseases of the mucous membrane (influenza, foot and mouth disease, shingles)
With these diseases from the first day of appointment
yut:
• UV irradiation with short rays, starting with 1 biodose and increasing the subsequent exposure to
1 biodose, the duration is increased to 4 biodoses by the end of the course. 5-6 procedures are prescribed for the course of treatment.
daytime;
• diadynamophoresis of trimecaine, novocaine for an
pain in addition to UV radiation. The impact of the beginning
nyut with a push-pull continuous current for 20 s, and then a "short period" of 2 minutes in one polarity and
2 min in reverse. The current strength is brought to the appearance of pronounced vibration. The course of treatment is prescribed 5-6 procedures.
Phonophoresis with 10% analginic ointment or hydrocortisone using a mobile technique, continuously
nominal operating mode of intensity 0.05-0.2 W / cm2. The course of treatment is up to 8-12 impacts.
Diseases of the tongue. Glossalgia
Glossalgia needs to be treated in conjunction with a stoma
tologist and neuropathologist. Assign:
electrosleep (affect 60-120 Hz, with a current strength of 7-15 mA. The course of treatment - up to 20-30 procedures daily
but or every other day, with the predominance of the processes
excitement);
electrophoresis of bromine, magnesium, platifillin, euphilin and iodine on the collar zone;
galvanic collar, calcium electrophoresis on the collar zone;
electrophoresis of vitamins B, B6, B12, C, PP, novoka
ina, trimecaine on the paresthesia zone. The course of treatment is prescribed up to 20-30 impacts daily or every other day;

diadynamic therapy is carried out with modulated currents - type of work III, IV mode alternating, hour
modulation tone 100 Hz, burst duration - pau
s 2-3 s, modulation depth - 75%, action time - 6-8 min. A course of 5-10 impacts daily;

fluctuating currents of the first form, medium dose for relieving tongue edema. Prescribed daily, course
5-10 impacts;
• local darsonvalization (prescribed for stagnation
phenomena in the tongue with a short spark for 5 min. The course of treatment - up to 12 impacts, every other day);
• phonophoresis - 10% analgin ointment in the mucous membrane of the tongue in combination with ultrasound
com on the cervical spine and cervical sim
pathic nodes with severe pain.
Glossitis chronic
For physiotherapeutic effects when given
the disease is used:
• electrophoresis of antiseptics (trimecaine, novoka
in) for pain relief - segmental effect on the tongue. The procedure lasts 20 minutes, the course of treatment is 10-12 times;
amplipulse phoresis is carried out in a rectified mode, type I work, modulation depth - 0%, frequency 150 Hz, duration - 2-3 s, exposure time - 6-8 minutes, course of treatment - up to 6-8 procedures daily;
ultraphonophoresis with 10% analgin ointment or 10% analgin solution;
darsonvalization of the tongue with a short spark in the flow
5-8 min. The course of treatment is 6-10 procedures, hedgehog
daytime.
Cheilitis Angular cheilitis (zayed)
From physiotherapy for angular chelitis, you can apply:
• UV irradiation with a short spectrum, start with 1 biodose and increase to 4-5 biodoses, increasing the length
the rate of each subsequent irradiation per 1 bio
dose. Treatment is carried out in 1-2 days, a course of treatment
up to 5-6 impacts;
ultraphonophoresis of nystatin and galascorbin izlu
cells with an area of 1 cm2 in continuous mode at an intensity of 0.02 W / cm2, the duration of the procedure
ry - 5-6 minutes;
Integrated spectrum UV irradiation in subery-dark doses according to the scheme:

Productive form. With a productive form of the process, the courses of treatment are repeated after 1-2 months.
tsa, combining various factors from the above
them.

Salivary stone disease
In the presence of inflammation, appoint:
UHF electric field on the gland area in an athermal dose with an output power of up to 30 W and a cart
stuffy gap 0.5-2 cm or

microwave therapy for 5-6 minutes at an intensity of 1-3 W.
In the presence of small stones, smooth
what musculature of the outlet duct:
• sinusoidal modulated currents with parameters: mode alternating frequency 100 Hz, mu
modulation bin 100%, III-IV type of work, duration
parcels for 3 s. The exposure time is 8-10 minutes. The course of treatment is 15 procedures daily.
After surgery, apply fi
ziotherapy according to the general principles of physiotherapy pos
leoperative period.
Fistulas of the salivary glands
Physiotherapy treatment of this pathology consists of the following procedures:
• electrophoresis of iodine 5-10% alcohol tincture, electrophoresis of silver or copper;
• diathermocoagulation of the fistulous tract.
Hyposalivation, xerotomy
With decreased salivation, physical
The methods are quite effective.
To normalize the function of the central and vegetation
the active nervous system is prescribed:
electrosleep (with the predominance of processes of excitation
denia);
galvanization of the collar zone or cervical sympathetic nodes;
• franklinization;
aeroionotherapy. Salivation is stimulated by:
anoid-galvanized;
• pulse currents (sinusoidal, modules
drowned;
diadynamic, fluctuating);
paraffin, azocerite and mud therapy.

1.Practical lesson

Topic: Features of the structure of the SOPR. SOPR functions.

Technological map of the practical lesson

Stages of work	**Teacher**	**Student**
1. Stages of preparation (10 minutes)	1. Controls the purity of the audience; 2. Checks students' readiness for classes; 3. Controls attendance;	Listen
2.The introductory stage to classes (15 minutes)	1.Preparation on the topic of content; 2. Preparation of slides for the introductory report; 3. To develop the literature used for the study of the subject;	Write down the topic and listen
3. The main stage (195 minutes)	1. Dividing students into 2 small subgroups, asking questions on the topic; 2. Use of slides and multimedia; 3. Carries out medical work; 4. Combines all information on a given topic, actively the participating students are encouraged and appreciated;	Divided into small groups watching, participating, listening. The student expresses his opinion, complements and asks questions
4. Final stage (10 minutes)	1. Conclusion. 2. Independent work. 3. Homework.	Listen Record Record

<u>Questions on the topic</u>

1.Features and structure of SOPR?
2. Classification of the disease of oral mucosa?
3. The histological structure of the OSS?
4.What is the location of the greasy and mixed glands of the cheek?

Tests

Who is the author of the classification of diseases of the oral mucosa?
* E.V. Borovsky, A.A. Mashkillyson
T.F. Vinogradova, A.A. Kolesov
O. P. Maksimova
B.N.Davydov, R.D. Novoselov
In what year was the classification of diseases of the oral mucosa compiled?
* 1984 year
1972 year
1978 year
1965 year
The gums are ...
stratified squamous epithelium, usually non-keratinizing epithelium;
dense fibrous connective tissue, consisting of inelastic fibers;
yellowish tissue with a thickness of 0.02 to 0.03 mm covering the dentin of the root in the area of the tooth neck;
from cancellous bone, the plates of which are located, the horizontal space between them is filled with yellow bone marrow;
In the gum (gingiva) we distinguish: the papillary part; free gingival margin; cement; gum fused with bone; non-adherent gums with bone;
A, B, D;
A, B, D;
A, B, D;
C, D, D;
What diseases are fungal diseases?
acute and chronic candidiasis, candidiasis;
Vincent's stomatitis, acute herpetic stomatitis;
tuberculous stomatitis, gonorrheal stomatitis, syphilis;
glossitis, gingivitis, papillitis;
According to the clinical course of the disease of the oral mucosa
stomatitis, papillitis, gingivitis;
acute, chronic, recurrent and parchment;
primary, secondary;
serous and purulent;
Which of the following papillae are not found in the tongue
diamond-shaped
leafy
mushroom

filiform
What is more important to base the classification of diseases of the oral mucosa in order to make a diagnosis of the disease?
course, type and localization of pathological changes, etiological factors;
the nature of the elements of the lesion and the severity of the clinical development of the disease;
localization of pathological changes;
causes of pathology;
What concept characterizes a pathological condition in which there is a violation of the integrity of the oral mucosa,
impaired function?
damage to the oral mucosa
disease
changes in the oral mucosa
any of the named
What concept characterizes a pathological condition in which a violation of the body's vital activity develops under the influence of extreme stimuli of the external and internal environment, a decrease in adaptability while simultaneously mobilizing the body's defenses?
disease
damage
the change
not named
What concept characterizes a pathological condition in which diseases of the body appear in the form of separate symptoms on the oral mucosa?
changes in the oral mucosa
disease of the oral mucosa
damage to the oral mucosa
any of the named
In the stratified squamous epithelium of the cheek, layers
basal, spiny
basal
basal, spiny, granular
basal, spinous, granular, horny
In the stratified squamous epithelium of the hard palate, layers
basal, spinous, granular, horny
basal
basal, spiny
basal, spiny, granular
In the stratified squamous epithelium of the gums, layers are distinguished
basal, spinous, granular, horny
basal
basal, spiny
basal, spiny, granular
Petechiae, purpura, ecchymosis, roseola are spots
inflammatory

hemorrhagic
pigmented
erosive
Limited discoloration of the mucous membrane is
spot
crust
aphtha
Interactive method
USING THE HANDLE IN THE MIDDLE TABLE METHOD
All students of the group are divided by lot into 3 subgroups of 3 students each. Each subgroup sits down at a separate table, prepares a blank sheet of paper and a pen. The date, group number, last name and first name of the student are written on the sheet. The task is offered, to answer one question for the whole subgroup. Each student writes down his last name and one answer option on a sheet and passes the sheet to a neighbor, and moves his pen to the middle of the table. The teacher supervises the work of the group and everyone's participation in it. The general correct version is written down in a notebook. Students who gave the correct answer options receive the maximum score - 100% of the theoretical part rating - 0.8b. Second place students - 85.9% of the rating. Third place - 70.9% of the rating. Those who did not answer or answered incorrectly 30% of the rating. The score received is taken into account when assigning marks for the current lesson. Student works are saved by the teacher
Text

Diseases of the oral mucosa are an important section of therapeutic dentistry, and not only for dentists, but also for doctors of other specialties. The mucous membrane of the mouth reflects the state of many organs and systems of the body. It is under the constant influence of environmental factors, often pathogenic, exceeding the capabilities of the body's defense reactions.

For a long time, diseases of the oral mucosa were considered as a local pathological process caused by insufficient oral hygiene and exposure to local traumatic factors. The merit in a fundamentally new approach to the study of diseases of the oral mucosa belongs to Russian scientists I. G. Lukomsky, E. E. Platonov, A. I. Evdokimov, B. M. Pashkov, A. L. Mashkillyson and others. This approach consists in the correct assessment of the role of local and general, external and internal, the relationship and interdependence of a number of factors in the study and approval of the cause-and-effect relationships of common diseases and manifestations of pathology in the oral cavity.

Currently, diseases of the oral mucosa are considered from the standpoint of the whole organism, since no one doubts the presence of a connection between most pathological processes on the oral mucosa and the red border of the lips with changes in organs and body systems, the nature of metabolic processes, immune status, etc. e. Changes in the oral mucosa are often the first clinical symptoms of metabolic disorders, diseases of the hematopoietic, cardiovascular systems, gastrointestinal tract, skin and venereal diseases, etc. Therefore, in order to learn how to diagnose diseases of the oral mucosa, the dentist must have a high professional level, have good knowledge of related specialties. At the same time, the principles of deontological

gia and medical ethics in combination with an individual approach to each patient, with knowledge of his psychological and social characteristics, the ability to see the personality of the patient as a whole with his experiences, and not just individual manifestations, symptoms of the disease.

When diagnosing diseases of the oral mucosa, it is important to correctly identify the elements of the lesion, understanding the features of the clinical course, localization of pathological changes, knowledge of the morphological and functional features of the oral mucosa, saliva, detailed study of pathomorphological changes in the oral mucosa in various diseases. A comprehensive and deep study of individual diseases that manifest themselves on the oral mucosa made it possible to create scientifically based classifications of diseases of the lips (G.D.Savkina), pemphigus (N.D.Sheklakov), precancerous lesions of the lips (A.L. systematics of diseases of the oral mucosa (E.E. Platonov) and their classification (T.F. Vinogradova, E.V. Borovsky, A.L. Mashkillyson, etc.).

For the correct recognition and treatment of diseases of the oral mucosa, it is often necessary to contact the dentist with other specialists in order to select the methods of additional examination, draw up a comprehensive therapy plan, taking into account the recommendations of doctors of other specialties and the nature of the manifestation of diseases in the oral cavity.

CLASSIFICATION OF DISEASES OF THE ORAL MUCOSA

Difficulties in diagnosing and choosing a method of treatment for pathology of the oral mucosa are due to the variety of manifestations of diseases localized in this area and often having similar clinical manifestations with different etiology or pathogenesis. The classification of diseases of the oral mucosa is very important for the practitioner, since it helps to navigate their existing variety, thereby contributing to the correct diagnosis, the choice of justified treatment methods and the implementation of preventive measures.

Previous classifications of diseases of the oral mucosa were based on different principles: anatomical and clinical (stomatitis, cheilitis, glossitis), the nature of the course (acute and chronic), clinical and morphological

their signs (catarrhal, ulcerative, etc.), the depth of the lesion (superficial and deep), the nature of the rashes (bullous, ulcerative, vesiculate, etc.) - However, each of them had its drawbacks and did not fully meet the above classification requirements.

The World Health Organization (WHO) has adopted the following nomenclature of diseases:

I. Infectious and parasitic diseases II. Neoplasms

III. Diseases of the blood and hematopoietic organs and lesions caused by the immune mechanism

IV. Endocrine, metabolic and nutritional diseases

V. Mental and character disorders VI. Diseases of the nervous system IX. Diseases of the circulatory system X. Diseases of the respiratory system XI. Diseases of the digestive system XII. Diseases of the skin and subcutaneous tissue

XIII. Diseases of the musculoskeletal system and connective tissue

XIV. Diseases of the genitourinary system

XV. Pregnancy, childbirth and the puerperium

XVII. Birth defects, deformities and chromosomal abnormalities

XVIII. Symptoms, signs, abnormalities, not elsewhere classified

XIX. Injury, poisoning and other consequences of external factors

XX. External factors leading to morbidity and mortality

For each of the listed sections of the classifications, there is a list of dental diseases and syndromes manifested in the oral cavity, characteristic of a given systemic pathology or condition. Systematization of dental diseases within the group of

It is based on clinical and morphological, pathomorphological characteristics, the nature of microflora, etc.

w The main goal of creating an international nomenclature of dental diseases and syndromes in the oral cavity is to standardize approaches to identifying dental pathology with subsequent registration and determination of the level of dental morbidity in different countries. In addition, the information obtained allowed us to determine the correlations of the general state of the body with the level of dental health, as well as to identify systemic diseases associated with pathology in the oral cavity.

Thus, at present, the etiological and pathogenetic relationship between diseases of various organs and systems and pathology in the oral cavity has been proven, and primarily diseases of the oral mucosa.

To systematize the known pathological conditions (diseases) of the oral mucosa, E.V. Borovsky and A.L.

I. Traumatic injuries due to mechanical factors, high and low temperatures, radiation, adverse meteorological factors (meteorological cheilitis, cracked lips), chemicals, etc. Form of manifestation: hyperemia, erosion, ulcers, hyperkeratosis (leukoplakia).

II. Infectious diseases:

1. Lesions of the oral mucosa in acute and chronic infectious diseases (measles, scarlet fever, chickenpox, tuberculosis, syphilis, leprosy, etc.);

2. Actually infectious and parasitic diseases of the mucous membrane of the mouth and lips;

^ viral (herpes, HIV infection, warts, etc.);

A fusospirochetosis;

^ bacterial (streptococcal and staphylococcal, gonorrheal, etc.);

And fungal (candidiasis, actinomycosis, etc.).

III. Allergic and toxic-allergic diseases:

^ contact allergic stomatitis, gingivitis, glossitis, cheilitis (from medicines, plastics and

other materials used in dentistry, dyes, toothpastes, elixirs and other chemicals in contact with the mucous membrane or red border of the lips, ultraviolet rays);

^ fixed and widespread toxic-allergic lesions (from medicines, food substances and other allergens entering the body in various ways);

^ dermatoses with lesions of the oral mucosa of toxic-allergic genesis (exudative erythema multiforme, Stevens-Johnson syndrome, Lyell's syndrome, primary systemic vasculitis, including Wegener's syndrome).

IV. Diseases with an autoimmune component of the pathogenesis:

A recurrent aphthous stomatitis, including cicatricial aphthae;

A. Behcet's syndrome, including Touraine's aphthous disease;

A Sjogren's syndrome;

And dermatoses with lesions of the oral mucosa (pemphigus, pemphigoid, Duhring's disease, systemic lupus erythematosus, systemic scleroderma).

V. Mucocutaneous reaction - lichen planus.

Vi. Changes in the oral mucosa with exogenous intoxication.

Vii. Changes in the mucous membrane of the mouth and the red border of the lips in the pathology of various organs and systems of the body and metabolic disorders:

And with visceral and endocrine pathology;

And with hypo- and avitaminosis;

And with diseases of the blood and hematopoietic organs;

And with the pathology of the nervous system;

And during pregnancy.

VIII. Congenital and genetically determined diseases

nevi and epithelial dysplasias: vascular nevi, including Sturge-Weber syndrome, bo-

birth and pigment nevi, epidermoid cyst, Fordyce's disease, white spongy nevus (mild leukoplakia, “buccal biting”, etc.), hereditary benign intraepithelial dyskeratosis;

^ folded and rhomboid glossitis; A glandular cheilitis;

^ dermatoses with lesions of the mucous membrane of the mouth and lips, epidermolysis bullosa, atopic dermatitis (cheilitis), psoriasis, ichthyosis, Darier's disease, Peitz-Jägers-Touraine syndrome, congenital paraonychia, anhydrotic epithelial dysplasia.

IX. Precancerous diseases, benign and malignant neoplasms:

And obligate precancer: Bowen's disease, warty precancer, limited hyperkeratosis of the red border of the lips, abrasive precancerous cheilitis of Manganotti;

And optional precancer: leukoplakia, keratinizing papilloma and papillomatosis, keratoacanthoma, cutaneous horn, erosive-ulcerative and hyperkeratotic forms of lupus erythematosus and lichen planus erythematosus, postradiation cheilitis;

A benign neoplasms;

And cancer.

At the Moscow Medical Dental Institute, the following classification of diseases of the oral mucosa is used in the educational process and medical work.

Classification of diseases of the oral mucosa

I. Traumatic injuries (>; chemical, physical! Kaya erythema, erosion, ulcer, leukoplakia, nicotinic leukeratosis, actinic cheilitis, radiation, chemical damage, etc.

II. Infectious diseases:

And viral (herpetic stomatitis, shingles

lichen, foot and mouth disease, viral warts, influenza, HIV infection);

And Vincent's ulcerative necrotizing stomatitis;

And bacterial infections (streptocal stomatitis, pyogenic granuloma, chancriform pyoderma, tuberculosis, etc.);

and sexually transmitted diseases (syphilis, gonorrheal stomatitis);

And mycoses (candidiasis, actinomycosis, etc.);

III. Allergic diseases (Quincke's edema, allergic stomatitis, cheilitis and glossitis, drug-induced stomatitis, glossitis, cheilitis, erythema multiforme exudative, recurrent aphthous stomatitis, etc.),

IV. Changes in the oral mucosa with exogenous intoxication.

V. Changes in the oral mucosa in some systemic diseases and metabolic diseases (hypo- and avitaminosis; diseases of the endocrine, gastrointestinal tract, cardiovascular system, blood system, nervous system; rheumatic diseases, or collagenoses).

Vi. Changes in the oral mucosa with dermatoses (pemphigus, Duhring's dermatitis herpetiformis, lichen planus, lupus erythematosus).

Vii. Anomaly and independent diseases of the tongue

(folded tongue; black "hairy" tongue; diamond-shaped, desquamative).

VIII. Independent cheilitis (glandular, exfoliative, actinic, meteorological, atopic, eczematous, contact, macrocheilit).

IX. Precancerous diseases (obligate and facultative) and tumors (benign and malignant).

Evaluation Criteria for Monitoring

№	Progress in% and points	Grade	Student knowledge level
1.	96-100	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Shows high activity, creative approach when conducting interactive games. Correctly solves situational problems and test questions. Analyzes independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
2.	91-95	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Analyzes research results independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
3.	86-90	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
4.	81-85	Good "4"	The correct answer to questions about the clinic, diagnosis and treatment of TBI, but there are 2-3 inaccuracies, errors. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
5.	76-80	Good "4"	Correct, but incomplete coverage of the issue. The student knows questions about the clinic and

			diagnosis of TBI, but is not fully versed in determining the tactics of treatment. Correctly solves situational problems, but the rationale for the answer is not complete. Understands the essence of the issue. Knows, speaks confidently
6.	71-75	Good "4"	Correct, but incomplete coverage of the issue. Correctly solves situational problems, but the rationale for the answer is incomplete. Has 2-3 errors. Knows, tells confidently. Has accurate ideas.
7.	66-70	Satisfactorily "3"	Understands the essence of the issue. Correctly solves situational problems, but cannot substantiate the answer. Has accurate ideas on specific issues.
8.	61-65	Satisfactorily "3"	Has an incomplete understanding of the topic. Makes mistakes when solving situational tasks. Tells not confidently.
9.	55-60	Satisfactorily "3"	Knows, tells not confidently. Has a partial view.
10.	54 и ниже	Unsatisfactory «2»	Has no exact idea. Does not know.

2- Practical lesson

Topic: Methods of examination of patients with injuries of the oral mucosa.

Technological map of the practical lesson

Stages of work	**Teacher**	**Student**
1. Stages of preparation (10 minutes)	1. Controls the purity of the audience; 2. Checks students' readiness for classes; 3. Controls attendance;	Listen
2.The introductory stage to classes (15 minutes)	1.Preparation on the topic of content; 2. Preparation of slides for the introductory report; 3. To develop the literature used for the study of the subject;	Write down the topic and listen
3. The main stage (195 minutes)	1. Dividing students into 2 small subgroups, asking questions on the topic; 2. Use of slides and multimedia; 3. Carries out medical work; 4. Combines all information on a given topic, actively the participating students are encouraged and appreciated;	Divided into small groups watching, participating, listening. The student expresses his opinion, complements and asks questions
4. Final stage (50	1. Conclusion.	Listen

minutes)	2. Independent work. 3. Homework.	Record

Questions on the topic

1. Examination of patients with clinical and laboratory methods.
2. Examination of patients with functional methods.
3. Examination of patients with cytological methods.
4. Examination of patients with bacteriological methods.

Tests

Decubital ulcer is caused by a factor
1) physical
2) chemical
3) mechanical

... Traumatic ulcer results from trauma
1) acute
2) chronic
3) subacute

... Chronic traumatic factors include
1) long-term smoking
2) accidental biting
3) prolonged irritation by sharp edges of the teeth
4) wound with a sharp object
5) microcurrents
6) acid exposure
7) balancing removable dentures

... Additional examination methods for traumatic ulcers
1) Kulazhenko test
2) allergic
3) bacterioscopic
4) cytological
5) Yasinovsky test
6) histological
7) measurement of microcurrents

Cytological picture in traumatic ulcer characterized by the presence
1) atypical epithelial cells
2) elements of inflammation
3) acantholytic cells
4) Langhans cells

Signs of malignancy of a traumatic ulcer
1) change in the size of the ulcer
2) the disappearance of pain
3) saucer-shaped ulcer
4) eosinophilia
5) compaction of the edges and bottom of the ulcer
6) keratinization of the edges of the ulcer

Traumatic ulcer is differentiated
1) lichen planus, erosive and ulcerative form
2) with syphilis (chancre)
3) with tuberculosis ulcer
4) with glossalgia
5) with a cancerous ulcer
6) with a trophic ulcer
7) with exudative erythema multiforme
8) with allergic stomatitis

... Treatments for traumatic ulcers include
1) elimination of traumatic factors
2) the use of steroid ointments
3) removal of steel crowns
4) the use of epithelizing agents
5) surgical excision of lesions
6) antiseptic treatment
7) rational prosthetics
8) replacement of metal seals with plastic ones

After eliminating the traumatic factor ulcers have a pronounced tendency to heal

1) cancerous
2) beam
3) traumatic
4) tuberculous

Prevention of chronic mechanical injury includes
1) diet
2) correction of removable dentures
3) exclusion of smoking and alcohol intake
4) timely prosthetics
5) sanding off sharp edges

Acute traumatic factors include
1) long-term smoking
2) accidental biting
3) prolonged irritation by sharp edges of the teeth
4) wound with a sharp object
5) microcurrents
6) acid exposure
7) balancing removable dentures

... Acute trauma is exposure to a traumatic agent
1) single
2) multiple

With drug treatment of erosive and ulcerative lesions
oral mucosa cauterizing agents
1) use
2) do not use

Possible complication after application arsenic paste
1) decubital ulcer
2) arsenous necrosis
3) trophic ulcer
4) arsenic periodontitis

Arsenic necrosis refers to
1) bacterial infections
2) to specific infections
3) to traumatic injuries

Arsenic necrosis caused by trauma
1) physical
2) chemical
3) mechanical

Differential diagnosis of arsenous necrosis is carried out
1) with alkaline necrosis
2) with acute herpetic stomatitis
3) with necrotizing ulcerative gingivitis of Vincent
4) with chronic recurrent aphthous stomatitis

The sequence of manipulations during treatment
arsenic gum necrosis
1) application of proteolytic enzymes
2) anesthesia
3) application of epithelizing agents
4) antiseptic treatment
5) removal of necrotic tissue

Arsenic antidotes are
1) sodium bicarbonate solution
2) an alcohol solution of iodine
3) burnt magnesia
4) a weak solution of acetic acid
5) unitiol

Thermal burn of the oral mucosa refers to trauma
1) chemical
2) mechanical
3) physical

Conditional rate of galvanic current in the oral cavity
1) up to 6 μA
2) up to 10 μA
3) up to 20 μA

The effect of galvanic current on the oral mucosa
attributed to injury
1) chemical
2) mechanical
3) physical

The presence of galvanic current in the oral cavity can lead to
to the emergence
1) burning of the oral mucosa
2) herpetic erosion
3) papillomatous growths
4) metal taste

5) blisters on the oral mucosa
Galvanic current increases when present in the oral cavity
1) dissimilar metals
2) homogeneous metal and plastic
3) homogeneous metal and composite materials
Arsenic paste in molars is applied
1) for a day
2) for two days
3) for three days
Regional lymph nodes in traumatic ulcers
in the oral cavity:
1) enlarged, painless, tightly elastic, mobile
2) enlarged, painless, dense, welded
3) enlarged, painful, soft, mobile
... Teeth with chronic foci of inflammation at the root apex in front of radiation therapy
1) remove
2) treat conservatively
3) covered with plastic mouth guards
... Teeth with cavities before radiation therapy:
1) delete
2) seal
3) covered with plastic trays 9. Penetration depth of secondary radiation from metal
structures during irradiation
1) 1-2 mm
2) 3-4 mm
3) 5-6 mm
... Metal structures in the oral cavity before irradiation are advisable:
1) remove
2) replace with ceramic or plastic
3) cover with plastic mouthguards

Interactive method

USING THE HANDLE IN THE MIDDLE TABLE METHOD
All students of the group are divided by lot into 3 subgroups of 3 students each. Each subgroup sits down at a separate table, prepares a blank sheet of paper and a pen. The date, group number, last name and first name of the student are written on the sheet. The task is offered, to answer one question for the whole subgroup. Each student writes down his last name and one answer option on a sheet and passes the sheet to a neighbor, and moves his pen to the middle of the table. The teacher supervises the work of the group and everyone's participation in it. The general correct version is written down in a notebook. Students who gave the correct answer options receive the maximum score - 100% of the theoretical part rating - 0.8b. Second place students - 85.9% of the rating. Third place - 70.9% of the rating. Those who did not answer or answered incorrectly 30% of the rating. The score received is taken into account when assigning marks for the current lesson. Student works are saved by the teacher

Text

The mucous membrane of the mouth is in direct contact with the external environment and is constantly exposed to various mechanical, thermal, chemical, microbiological, physical and other factors. If these stimuli do not exceed the irritability threshold of the oral mucosa, then it, due to its protective function, does not change. However, when exposed to stronger suprathreshold stimuli, changes occur on the oral mucosa, the nature of which depends on the type, intensity and duration of the

traumatic factor. The severity of these changes is also determined by the localization of the effect of an external factor, the state of the body's reactivity, individual features of the structure of the mucous membrane, etc. Due to the fact that the main role in the clinical picture of traumatic lesions of the oral mucosa belongs to the type of irritant, it is necessary to separately consider changes in the mucosa shells resulting from exposure to mechanical, temperature, radiation and chemical factors.

ramma

This injury can be acute when the mucous membrane is damaged by a short-term but significant factor, and chronic - with prolonged exposure to a weak stimulus.

Acute mechanical trauma

Acute mechanical trauma (trauma mechanicum acutum) in the form of biting or wounding with instruments is rare. It manifests itself as a hematoma (interstitial hemorrhage without disturbing the integrity of the epithelium), erosion or ulcer. With interstitial hemorrhage at the site of injury, pain is noted, which quickly (after 1-3 days) disappears, but if the integrity of the epithelium is damaged, painful erosion or an ulcer with infiltration is formed. Secondary infection, the wound can turn into long-term non-healing ulcers. Diagnosis, as a rule, is not difficult - the cause is revealed from the anamnesis.

Treatment. The amount of medical assistance depends on the depth and magnitude of the damage. With hematomas, erosions and chalk

such wounds are sufficiently thorough antiseptic treatment of the damaged area and the entire oral cavity. To do this, rinse with antiseptic solutions (potassium permanganate 1: 5000; 0.5% hydrogen peroxide solution and other drugs), applications of agents that stimulate epithelization (oil solution of vitamins A, E, carotolin, rosehip oil, jelly, solcoseryl ointment and etc.). For deep wounds, stitches are applied.

Chronic mechanical trauma

Chronic mechanical trauma (trauma mechanicum chronicum) is common. A traumatic factor can be sharp edges of teeth, bridges and removable dentures, tartar, bad habits (biting the mucous membrane), etc. , the strength of the irritating factor. Damage to the mucous membrane under the influence of chronic irritants is more common in older people. This is facilitated by a decrease in the turgor of the mucous membrane, a decrease in the height of the bite due to abrasion of hard tissues of the teeth, loss, displacement of teeth. In older people, the regeneration process is slowed down, which is the reason for the slow healing of the damaged mucous membrane.

Changes in the mucous membrane during chronic mechanical injury may not bother the patient for a long time, but sometimes a feeling of awkwardness, discomfort, slight soreness, and swelling may occur. When examining the mucous membrane, catarrhal inflammation (edema, hyperemia), violation of its integrity (erosion, ulcers), proliferative changes (hypertrophy of the gingival papillae, gingival margin), hypertrophy of the papillae of the tongue such as papillomatosis, increased keratinization (leukoplakia) can be detected. These changes can occur in combination with each other. It should be remembered that simultaneously with mechanical injury, the mucous membrane is exposed to the microflora of the oral cavity, which often affects the clinical picture of the disease. Correct, but incomplete coverage of the issue. The student knows questions about the clinic and diagnosis of TBI, but is not fully versed in determining the tactics of treatment. Correctly solves situational

problems, but the rationale for the answer is not complete. Understands the essence of the issue. Knows, speaks confidently
Correct, but incomplete coverage of the issue. Correctly solves situational problems, but the rationale for the answer is incomplete. Has 2-3 errors. Knows, tells confidently. Has accurate ideas.

Критерии оценки текущего контроля

№	Progress in% and points	Grade	Student knowledge level
1.	96-100	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Shows high activity, creative approach when conducting interactive games. Correctly solves situational problems and test questions. Analyzes independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
2.	91-95	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Analyzes research results independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
3.	86-90	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
4.	81-85	Good "4"	The correct answer to questions about the clinic, diagnosis and treatment of TBI, but there are 2-3 inaccuracies, errors. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.

5.	76-80	Good "4"	Correct, but incomplete coverage of the issue. The student knows questions about the clinic and diagnosis of TBI, but is not fully versed in determining the tactics of treatment. Correctly solves situational problems, but the rationale for the answer is not complete. Understands the essence of the issue. Knows, speaks confidently
6.	71-75	Good "4"	Correct, but incomplete coverage of the issue. Correctly solves situational problems, but the rationale for the answer is incomplete. Has 2-3 errors. Knows, tells confidently. Has accurate ideas.
7.	66-70	Satisfactorily "3"	Understands the essence of the issue. Correctly solves situational problems, but cannot substantiate the answer. Has accurate ideas on specific issues.
8.	61-65	Satisfactorily "3"	Has an incomplete understanding of the topic. Makes mistakes when solving situational tasks. Tells not confidently.
9.	55-60	Satisfactorily "3"	Knows, tells not confidently. Has a partial view.
10.	54 и ниже	Unsatisfactory "2"	Has no exact idea. Does not know.

3- Practical lesson

Topic: Pathological changes in the oral mucosa. Morphological elements of the lesion.

practical lesson flowchart

Stages of work	**Teacher**	**Student**
1. Stages of preparation (10 minutes)	1. Controls the purity of the audience; 2. Checks students' readiness for classes; 3. Controls attendance;	Listen
2.The introductory stage to classes (15 minutes)	1.Preparation on the topic of content; 2. Preparation of slides for the introductory report; 3. To develop the literature used for the study of the subject;	Write down the topic and listen
3. The main stage (195	1. Dividing students into 2 small subgroups, asking questions on the topic; 2. Use of slides and multimedia;	Divided into small groups watching,

minutes)	3. Carries out medical work; 4. Combines all information on a given topic, actively the participating students are encouraged and appreciated;	participating, listening. The student expresses his opinion, complements and asks questions
4. Final stage (50 minutes)	1. Conclusion. 2. Independent work. 3. Homework.	Listen Write down

Questions on the topic
1. Explained infiltrative morphological elements.
2. Count the Primary morphological elements.
3. Count the secondary morphological elements.
4. What is Acantolysis.
5.What is a stain

Tests

What is acanthosis.
A) thickening of the epithelial layer of the mucosa due to proliferation
B) thickening of the basal layer due to inflammation
C) reduction of the thorny layer
D) reduction of the epithelial layer due to proliferation

In the stratified squamous epithelium of the cheek, layers are distinguished.
A) basal, spiny, granular
B) basal, spiny
B) basal
D) basal, spiny, horny

Normally, the epithelium is not keratinized.
A) papillary gums
B) alveolar gums
C) the gingival sulcus
D) tooth, gum

What is cheilitis.
A) inflammation of the gums
B) inflammation of the red border of the lips
C) pulp inflammation
D) inflammation of the oral mucosa

Change in the oral cavity with measles.

A) preceded by a skin rash
B) does not appear
B) appear simultaneously with a skin rash
D) not simultaneously with a skin rash

How many layers does the epidermis of the skin consist of?
A) 7
B) 8
AT 5
D) 4

The gums are ...
A) the supporting and holding apparatus of the tooth
B) the mucous membrane covering the alveolar process
B) osteoid tissue covering the tooth root
D) connective tissue formation

In the stratified squamous epithelium of the gums, layers are distinguished.
A) basal, spiny, granular
B) basal
B) basal, spiny, granular, horny
D) basal, spiny

Change in the oral cavity with measles.
A) bleeding gums
B) gum atrophy
C) a rash around the lip
D) Filatov-Koplik spots

What is xerostomia.
A) dry mouth
B) increased salivation
C) dryness of all mucous membranes
D) everything is correct

Interactive method
USING THE HANDLE IN THE MIDDLE TABLE METHOD
All students of the group are divided by lot into 3 subgroups of 3 students each. Each subgroup sits down at a separate table, prepares a blank sheet of paper and a pen. The date, group number, last name and first name of the student are written on the sheet. The task is offered, to answer one question for the whole subgroup. Each student writes down his last name and one answer option on a sheet and passes the sheet to a neighbor, and moves his pen to the middle of the table. The teacher supervises the work of the group and everyone's participation in it. The general correct version is written down in a notebook. Students who gave the correct answer options receive the

maximum score - 100% of the theoretical part rating - 0.8b. Second place students - 85.9% of the rating. Third place - 70.9% of the rating. Those who did not answer or answered incorrectly 30% of the rating. The score received is taken into account when assigning marks for the current lesson. The works of students are saved by the teacher.

Text

Examination of the oral cavity itself

Then proceed to the study of the actual oral cavity. First of all, a general examination is performed, paying attention to the color and moisture content of the mucous membrane. Normally, it is pale pink, but it can become hyperaemic, edematous, and sometimes acquires a whitish hue, which indicates the phenomenon of para- or hyperkeratosis.

Examination of the tongue begins with determining the condition of the papillae, especially if there are complaints of changes in sensitivity or burning and soreness in any areas. There may be a lining of the tongue due to a slowdown in the rejection of the outer layers of the epithelium. Such

the phenomenon may be a consequence of a disruption in the activity of the gastrointestinal tract, and possibly pathological changes in the oral cavity with candidiasis. Sometimes there is an increased desquamation of the papillae of the tongue in some area (more often at the tip and lateral surface). This condition may not bother the patient, but pain from irritants, especially chemical ones, may occur. With atrophy of the papillae of the tongue, its surface becomes smooth, as if polished, and due to hyposalivation, it becomes sticky. Individual areas, and sometimes the entire mucous membrane, can be bright red or crimson. This state of the tongue is observed in malignant anemia and is called Gunther's glossitis (after the name of the author who described it for the first time). Hypertrophy of the papillae may also be noted, which, as a rule, does not cause concern to the patient. Hypertrophy of the papillae of the tongue is often combined with hyperacid gastritis.

When examining the tongue, it should be remembered that at the root of the tongue on the right and left there is a pink or bluish-pink lymphoid tissue. Often this formation is sick, and sometimes even doctors take it for pathological. In the same place, a pattern of veins is sometimes clearly visible due to their varicose enlargement, but this symptom has no clinical manifestation.

When examining the tongue, pay attention to its size, relief. With an increase in size, the time of manifestation of this symptom (congenital or acquired) should be determined. It is necessary to distinguish macroglossia from edema. The tongue can be folded in the presence of a significant number of longitudinal folds, but patients may not be aware of this, since in most cases it does not bother them. Folding appears when the tongue is spread. Patients mistake them for cracks. The difference is that with a crack, the integrity of the epithelial layer is broken, and with a fold, the epithelium is not damaged.

When examining the floor of the mouth, pay attention to the mucous membrane. Its peculiarity is compliance, the presence of folds, frenulum of the tongue and excretory ducts of the salivary glands, and sometimes droplets of accumulated secretion. In smokers, the mucous membrane may turn dull.

In the presence of keratinization, which manifests itself in areas of grayish-white color, their density, size, adhesion with the underlying tissues, the level of elevation of the focus above the mucous membrane, and soreness are determined.

Rice. 4.4. Noncavity infiltrative elements of the lesion, a - spot; b - a knot; в - node; d - tubercle; e - blister.

Rice. 4.4. Continuation.

The importance of identifying these signs is that sometimes they serve as the basis for active intervention, since foci of hyperkeratosis of the oral mucosa are considered precancerous conditions.

If any changes are detected on the oral mucosa (ulcer, erosion, hyperkeratosis, etc.), it is necessary to exclude or confirm the possibility of a traumatic factor. This is necessary for the diagnosis, and if the cause is identified, it is important for the treatment. However, it should be remembered that the detection of trauma to the mucous membrane by teeth or a prosthesis is possible in the state of the physiological position of the lower jaw and tongue, i.e. with closed jaws. Otherwise, when opening the mouth, especially full, there is a significant displacement of the tissues of the cheeks, tongue, and in this position the injured area may not be in contact.

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Rice. 4.5. Cavity elements of the lesion, a - vesicle; b - abscess; c - intraepithelial bladder; d - sub-epithelial bladder; e - cyst.

touch the edge of the tooth or denture that is actually causing these changes.

When making a diagnosis, it is important to know the elements of the lesion of the oral mucosa and the red border of the lips. Correct determination of the element of the lesion, to a large extent, ensures the correct diagnosis.

Among the elements of the lesion, there are primary and secondary, arising in place of the primary, as well as infiltrative, vesicular and other groups of elements.

The primary elements of the lesion include a spot, a nodule, a tubercle, a knot, a vesicle, an abscess, a bladder, a blister, a cyst. Secondary elements are erosion, ulcer, crack, crust, scale, scar, pigmentation.

Spot (macula). A spot is called a limited area of the discolored oral mucosa (Fig. 4.4, a). Distinguish between spots of inflammatory and non-inflammatory origin. An inflammatory spot with a diameter of up to 1.5 cm is defined as roseola, more than 1.5 cm as erythema. Spots appear during burns, trauma, or as manifestations of common diseases - measles, scarlet fever, B1G hypovitaminosis. Pigmented spots as a result of melanin deposition (congenital staining of mucous membrane areas), taking medications containing bismuth and lead are referred to the group of spots of non-inflammatory origin.

Knot (papula). This is a noncavity element of inflammatory origin with a diameter of up to 5 mm, rising above the level of the mucous membrane, capturing the epithelium and the surface layers of the mucous membrane itself (Fig. 4.4, b). Small cell infiltration, hyperkeratosis and acanthosis are morphologically determined. A typical example of papules on the oral mucosa is lichen planus. Merged papules, if their diameter reaches 5 mm or more, form a plaque.

Node (nodus). The node differs from the nodule in its large size and involvement in the inflammatory process of all layers of the mucous membrane (Fig. 4.4, c). On palpation, a slightly painful infiltrate is determined.

Tubercle (tuberculum). The tubercle, as an element of inflammatory origin, captures all layers of the mucous membrane. Its diameter is 5-7 mm. It is dense on palpation, painful, the mucous membrane is hyperemic, edematous (Fig. 4.4, d). The tubercle is prone to decay with the formation of an ulcer. When healing, a scar is formed. Bumps are formed with tuberculosis.

Blister (urtica). This pronounced limited edema of the mucous membrane itself (Fig.4.4, e) is observed with an allergic reaction (Quincke's edema), etc.

Vesicle (vesicula). This is a rounded cavity formation (up to 5 mm in diameter), protruding above the level of the mucous membrane and filled with serous or hemorrhagic contents (Fig. 4.5, a). The vesicle is located intraepithelially, it is easily opened. Bubbles occur with viral lesions: shingles, foot and mouth disease, herpes.

Pustule (pustula). This element is similar to a bubble, but

Rice. 4.6. Secondary elements of defeat with violation of integrity

enamel.

a - erosion; b - ulcer; c - crack.

with purulent contents (Fig. 4.5, b). It is observed on the skin and the red border of the lips.

Bubble (bulla). It differs from the bubble in its large size. It can be located intraepithelially (Fig. 4.5, c) as a result of stratification of epithelial cells (for example, with acantholytic pemphigus) and subepithelially (Fig. 4.5, d), when the epithelial layer is detached (with exudative erythema multiforme, allergies and other diseases). In the oral cavity, bubbles are very rare (practically invisible), since they open up and erosion forms in their place. Often, a bubble cap is noted along the edges of erosion. The contents of the bladder are usually serous, less often hemorrhagic.

Cyst (cysta). A cyst is a cavity formation that has an epithelial lining and a connective tissue membrane (Fig. 4.5, e).

Erosion (erosio). This is damage to the mucous membrane within the epithelium (Fig. 4.6, a), which occurs after the opening of the vesicle, bladder or develops at the site of a papule, plaque, as well as as a result of trauma. Heals without scar formation.

Afta (aphta). Aphtha is an oval erosion, covered with fibrinous plaque and surrounded by a hyperemic rim.

Ulcer (ulcus). A defect that covers all layers of the oral mucosa is called an ulcer (Fig. 4.6, b). In contrast to erosion, ulcers distinguish between the bottom and the walls. Ulcers occur with trauma, tuberculosis, syphilis, with the disintegration of a neoplasm. After healing, a scar forms.

Crack (rhagades). This is a linear defect that occurs when tissue elasticity is lost (Fig. 4.6, c).

Scale (squama). Scales are defined as the formation of layers of epithelium due to a violation of the process of its desquamation (Fig. 4.7, a).

Crust (Crysta). The shriveled exudate forms a crust usually at the site of cracks, erosion (Figure 4.7, b).

Scar (cicatrix). It is formed if a defect in the mucous membrane is replaced by connective tissue (Figure 4.7, c).

Pigmentation (pigmentatio). Changes in the color of the mucous membrane or skin at the site of the pathological process due to the deposition of melanin or other pigment are called pigmentation. Pigmentation should be distinguished as a physiological

phenomenon, when the mucous membrane of the mouth acquires a dark shade. This is observed among residents of the south. Pathological pigmentation is observed upon admission

Rice. 4.7. Secondary elements of the lesion, a - scale; b - crust; c - scar.

into the body of salts of heavy metals (lead, bismuth). The beginning of the manifestation of melanoma is also the appearance of a site of pigmentation of the mucous membrane.

It is necessary to distinguish between general changes in the epidermis, which, as a rule, develop as a result of the emergence

Rice. 4.8. Accumulation of fluid (spongiosis) between the cells of the spinous layer (a) and ballooning degeneration with acantholysis (b).

pathological process in the body, from the processes occurring in the oral mucosa.

Spongiosis (spongiosis). This is the accumulation of fluid between the cells of the prickly layer (Fig. 4.8, a).

Ballooning degeneration consists in a violation of the prickly layer (Fig. 4.8, b), which leads to the free arrangement of individual cells or their groups in the exudate of the resulting bubbles (in the form of balloons-balloons).

Figure 4.9. Acanthosis with hyperkeratosis.

Acantholysis. These are degenerative changes in the cells of the spiny layer, expressed in the melting of intercellular cytoplasmic connections (see Fig. 4.8, b).

Acanthosis (acanthosis). This is a thickening of the cells of the prickly layer, characteristic of inflammation (Fig. 4.9).

Hyperkeratosis (hyperkeratosis). Excessive keratinization due to impaired desquamation or increased production of keratinized cells forms the basis of hyperkeratosis (see Fig. 4.9).

Parakeratosis (parakeratosis). This is a violation of the keratinization process, which is expressed in incomplete keratinization of the superficial cells of the spinous layer (Figure 4.10).

Rice. 4.10. Incomplete keratinization of the superficial cells of the spinous layer - parakeratosis.

Rice. 4.11. Overgrowth of the papillary layer of the epithelium - papillomatosis.

Papillomatosis (papillomatosis). The growth towards the epithelium of the papillary layer of the mucous membrane is called papillomatosis.

Evaluation Criteria for Monitoring

№	Progress in% and points	Grade	Student knowledge level
1.	96-100	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Shows high activity, creative approach when conducting interactive games. Correctly solves situational problems and test

			questions. Analyzes independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
2.	91-95	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Analyzes research results independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
3.	86-90	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
4.	81-85	Good "4"	The correct answer to questions about the clinic, diagnosis and treatment of TBI, but there are 2-3 inaccuracies, errors. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
5.	76-80	Good "4"	Correct, but incomplete coverage of the issue. The student knows questions about the clinic and diagnosis of TBI, but is not fully versed in determining the tactics of treatment. Correctly solves situational problems, but the rationale for the answer is not complete. Understands the essence of the issue. Knows, speaks confidently
6.	71-75	Good "4"	Correct, but incomplete coverage of the issue. Correctly solves situational problems, but the rationale for the answer is incomplete. Has 2-3 errors. Knows, tells confidently. Has accurate ideas.
.	66-70	Satisfactorily "3"	Understands the essence of the issue. Correctly solves situational problems, but cannot substantiate the answer. Has accurate ideas on specific issues.
8.	61-65	Satisfactorily "3"	Has an incomplete understanding of the topic. Makes mistakes when solving

			situational tasks. Tells not confidently.
9.	55-60	Satisfactorily "3"	Knows, tells not confidently. Has a partial view.
10.	54 and below	Unsatisfactory "2"	Has no exact idea.

4- Практическое занятие

Тема: Механические, физические и химические поражения слизистой оболочки полости рта Клиника, диф.диагностика и лечение.

Технологическая карта практического занятия

Stages of work	Teacher	Student
1. Stages of preparation (10 minutes)	1. Controls the purity of the audience; 2. Checks students' readiness for classes; 3. Controls attendance;	Listen
2.The introductory stage to classes (15 minutes)	1.Preparation on the topic of content; 2. Preparation of slides for the introductory report; 3. To develop the literature used for the study of the subject;	Write down the topic and listen
3. The main stage (195 minutes)	1. Dividing students into 2 small subgroups, asking questions on the topic; 2. Use of slides and multimedia; 3. Carries out medical work; 4. Combines all information on a given topic, actively the participating students are encouraged and appreciated;	Divided into small groups watching, participating, listening. The student expresses his opinion, complements and asks questions
4. Final stage (50 minutes)	1. Conclusion. 2. Independent work. 3. Homework.	Listen Record

Questions on the topic
1.What is hyperkeratosis
2.what is papillomatous hyperplasia
3.what is hematoma

Tests

Decubital ulcer is caused by a factor
1) physical
2) chemical

3) mechanical

... Traumatic ulcer results from trauma
1) acute
2) chronic
3) subacute

... Chronic traumatic factors include
1) long-term smoking
2) accidental biting
3) prolonged irritation by sharp edges of the teeth
4) wound with a sharp object
5) microcurrents
6) acid exposure
7) balancing removable dentures

Additional examination methods for traumatic ulcers
1) Kulazhenko test
2) allergic
3) bacterioscopic
4) cytological
5) Yasinovsky test
6) histological
7) measurement of microcurrents

... Cytological picture in traumatic ulcer
characterized by the presence
1) atypical epithelial cells
2) elements of inflammation
3) acantholytic cells
4) Langhans cells

Signs of malignancy of a traumatic ulcer
1) change in the size of the ulcer
2) the disappearance of pain
3) saucer-shaped ulcer
4) eosinophilia
5) compaction of the edges and bottom of the ulcer
6) keratinization of the edges of the ulcer
Traumatic ulcer is differentiated
1) lichen planus, erosive and ulcerative form
2) with syphilis (chancre)
3) with tuberculosis ulcer
4) with glossalgia
5) with a cancerous ulcer
6) with a trophic ulcer

7) with exudative erythema multiforme
8) with allergic stomatitis
Treatments for traumatic ulcers include
1) elimination of traumatic factors
2) the use of steroid ointments
3) removal of steel crowns
4) the use of epithelizing agents
5) surgical excision of lesions
6) antiseptic treatment
7) rational prosthetics
8) replacement of metal seals with plastic ones
After eliminating the traumatic factor
ulcers have a pronounced tendency to heal
1) cancerous
2) beam
3) traumatic
4) tuberculous

Prevention of chronic mechanical injury includes
1) diet
2) correction of removable dentures
3) exclusion of smoking and alcohol intake
4) timely prosthetics
5) sanding off sharp edges
Acute traumatic factors include
1) long-term smoking
2) accidental biting
3) prolonged irritation by sharp edges of the teeth
4) wound with a sharp object
5) microcurrents
6) acid exposure
7) balancing removable dentures
Acute trauma is exposure to a traumatic agent
1) single
2) multiple With medical treatment of erosive and ulcerative lesions
oral mucosa cauterizing agents
1) use
2) do not use
Possible complication after application
arsenic paste
1) decubital ulcer
2) arsenous necrosis
3) trophic ulcer
4) arsenic periodontitis
Arsenic necrosis refers to
1) bacterial infections

2) to specific infections
3) to traumatic injuries
Arsenic necrosis caused by trauma
1) physical
2) chemical
3) mechanical
Differential diagnosis of arsenous necrosis is carried out
1) with alkaline necrosis
2) with acute herpetic stomatitis
3) with necrotizing ulcerative gingivitis of Vincent
4) with chronic recurrent aphthous stomatitis

The sequence of manipulations during treatment
arsenic gum necrosis
1) application of proteolytic enzymes
2) anesthesia
3) application of epithelizing agents
4) antiseptic treatment
5) removal of necrotic tissue

Arsenic antidotes are
1) sodium bicarbonate solution
2) an alcohol solution of iodine
3) burnt magnesia
4) a weak solution of acetic acid
5) unitiol

Interactive method
USING THE HANDLE IN THE MIDDLE TABLE METHOD
All students of the group are divided by lot into 3 subgroups of 3 students each. Each subgroup sits down at a separate table, prepares a blank sheet of paper and a pen. The date, group number, last name and first name of the student are written on the sheet. The task is offered, to answer one question for the whole subgroup. Each student writes down his last name and one answer option on a sheet and passes the sheet to a neighbor, and moves his pen to the middle of the table. The teacher supervises the work of the group and everyone's participation in it. The general correct version is written down in a notebook. Students who gave the correct answer options receive the maximum score - 100% of the theoretical part rating - 0.8b. Second place students - 85.9% of the rating. Third place - 70.9% of the rating. Those who did not answer or answered incorrectly 30% of the rating. The score received is taken into account when assigning marks for the current lesson. The works of students are saved by the teacher.

Text
Chronic mechanical trauma

Chronic mechanical trauma (trauma mechanicum chronicum) is common. A traumatic factor can be sharp edges of teeth, bridges and removable dentures, tartar, bad habits (biting the mucous membrane), etc. , the strength of the irritating factor. Damage to the mucous membrane under the influence of chronic irritants is more common in older people. This is facilitated by a decrease in the turgor of the mucous membrane, a decrease in the height of the bite due to abrasion of hard tissues of the teeth, loss, displacement of teeth. In older people, the regeneration process is slowed down, which is the reason for the slow healing of the damaged mucous membrane.

Changes in the mucous membrane during chronic mechanical injury may not bother the patient for a long time, but sometimes a feeling of awkwardness, discomfort, slight soreness, and swelling may occur. When examining the mucous membrane, catarrhal inflammation (edema, hyperemia), violation of its integrity (erosion, ulcers), proliferative changes (hypertrophy of the gingival papillae, gingival margin), hypertrophy of the papillae of the tongue such as papillomatosis, increased keratinization (leukoplakia) can be detected. These changes can occur in combination with each other. It should be remembered that simultaneously with mechanical injury, the mucous membrane is exposed to the microflora of the oral cavity, which often affects the clinical picture of the disease.

CATARRH. It occurs most often in chronic trauma and is manifested by hyperemia, edema with infiltration. The severity of these changes depends on the strength and duration of the stimulus. In this case, the process can be accompanied by the expression

female exudation (at first the secret is serous, and then purulent).

Of the factors that can cause irritation and damage to the oral mucosa, dentures should be singled out. They are inappropriate stimuli. The removable prosthesis transfers the chewing pressure to the mucous membrane, delays the self-cleaning of the oral cavity, which leads to a violation of the established balance between different types of microorganisms, changes the analytic function of the mucous membrane receptors. The onset of inflammation of the mucous membrane under the prosthesis is due to the influence of not one, but several factors (traumatic, allergic, infectious).

Inflammation of the mucous membrane of the prosthetic bed can be focal in the form of pinpoint hyperemia or large hyperemic spots and diffuse, often occupying the entire surface of the prosthetic bed. Against the background of an inflamed and edematous mucous membrane, punctate hemorrhages, erosion, as well as hyperplasia of the mucous membrane in the form of its granularity and lobularity may occur. Changes in the mucous membrane can also be observed when using fixed prostheses. Even with a perfectly made crown, the edges of which are immersed in the gingival pocket, the condition of the gums is disturbed - self-cleaning becomes difficult, conditions are created for the retention of food residues.

The course of catarrhal inflammation can be acute or chronic. Acute inflammation lasts 7-14 days and quickly disappears when the irritant is removed. With purulent exudate, the mucous membrane undergoes superficial destruction and erodes. Elimination of the irritant and the treatment carried out lead to a rapid elimination of the lesion. In the absence of treatment, chronic focal purulent inflammation occurs with the formation of a limited focus, consisting of a purulent-molten mass. As a result, a traumatic ulcer is formed.

TRAUMATIC ULTRA. Traumatic ulcers (Fig. 11.1), which are also called decubital, are localized at the sites of injuries, more often on the tongue, lips and cheeks along the line of closing of the teeth. Ulcers are usually painful, especially when eating, talking. More often it is a single ulcer. The clinical manifestation of a traumatic ulcer is diverse and depends on the irritant, the condition of the ulcer, the reactivity of the body, the influence of the oral microflora. With the prolonged existence of the ulcer, its edges and base are compacted (due to the predominance of the phenomenon of proliferation). However, the edges of the ulcer

Traumatic ulcer of the lower lip. Ulcer with infiltrated edges, the bottom is covered with fibrinous bloom.

hyperemic, painful on palpation, the bottom is covered with necrotic plaque. The depth is different - up to the muscle layer. Regional lymph nodes are enlarged, mobile, painful on palpation.

Traumatic ulcers can be complicated by fusospirochetosis or candidiasis, with prolonged course (2-3 months) they can become malignant.

Differential diagnosis. It is necessary to distinguish a traumatic ulcer from A cancer, A tuberculous ulcers, A syphilitic ulcers, A chronic ulcerative necrotizing Vincent's gingivostomatitis, A trophic ulcers.

Removal of the traumatic factor also serves differential diagnostic purposes. Fast, in several days, ulcer healing testifies to its traumatic origin.

A cancerous ulcer is distinguished by a greater density of edges and bases, often keratinization of the edges. After removal of the irritant, it does not heal. With cytological or histological examination, it is possible to detect the atypicality of epithelial cells.

A tuberculous ulcer has more undermined edges, a granular bottom with a yellowish coating, does not epithelize after removal of the stimulus. In the scraping from the ulcer,

epithelioid cells and giant cells of Langhans, and staining according to Ziehl-Nielsen often reveals mycobacterium tuberculosis. The general condition of the patient, as a rule, deteriorates. To clarify the diagnosis, the patient is referred to a phthisiatrician.

A chancre in typical cases differs from a traumatic ulcer by the presence of a dense infiltrate surrounding the ulcer, smooth edges, a smooth bottom, enlargement and compaction of regional lymph nodes (scleradenitis). The diagnosis is confirmed when a pale spirochete is found in the discharge of the ulcer. The Wasserman reaction and other reactions to syphilis become positive 3-4 weeks after the onset of the ulcer. Elimination of the injury does not significantly affect the course of the hard chancre.

A secondary complication of a traumatic ulcer by fusospirochetosis leads to the appearance of a greenish-gray fetid plaque; in scrapings, spirochetes and fusiform bacilli are found in large numbers (see section 11.3.2).

A trophic ulcer differs from a * traumatic ulcer in a more sluggish course, mild symptoms of inflammation (areactive ulcers), a sharp violation of the general condition (most often a disease of the cardiovascular system). The traumatic factor can be very mild, and after its elimination, such ulcers do not tend to heal unless general therapy is carried out.

Treatment. With traumatic ulcers, it comes down to eliminating the irritant, antiseptic treatment of the ulcer and rinsing the mouth. With a sharp soreness of the ulcer, the

application of painkillers is indicated. Necrotic tissue from the bottom of the ulcer is carefully removed mechanically under anesthesia or using proteolytic enzymes. Prescribe applications of keratoplastic agents (vitamins A and E; rosehip oil; jelly, ointment solcoseryl, actovegin, etc.). Moxibustion is categorically contraindicated. Perform a thorough sanitation of the oral cavity. Damage with removable dentures, manifested by hyperemia, erosion or ulcers, quickly disappear after correction of the denture

Physical damage

Acute injuries arise from burns of the mucous membrane with hot water, hot steam, fire (thermal injury), electric current, with short-term exposure to large doses of ionizing radiation. Burns with hot steam or water initially cause a sharp pain, the intensity of which then quickly decreases, a feeling of roughness of the mucous membrane appears. Acute catarrhal stomatitis develops. The epithelium of the oral mucosa is partially or completely macerated; after a severe burn, it sloughs off in layers or bubbles appear that quickly burst, erosion or superficial ulcers are found under them.

Treatment. It is carried out in the same way as for nonspecific acute inflammatory processes.

Prevention. Prevention of such lesions consists in carrying out sanitary and educational work, observing safety measures.

Galvanic current in the oral cavity is increased in the presence of different metals (prostheses and fillings). The presence of various metals and microcurrents of large magnitude do not always cause pathological changes, however, in some individuals, even small currents can cause them. Conventionally, microcurrents in the oral cavity up to 10 μA are considered the norm. Their increase is caused by a significant (in comparison with the norm) amount of solder formed when the technology of manufacturing prostheses is violated. The highest value of microcurrent is formed by the following pbram: gold - amalgam, steel - solder, amalgam - solder and amalgam - steel.

Galvanic current is assigned a certain role in the appearance of inflammatory processes of the oral mucosa, especially leukoplakia and lichen planus, as well as in the aggravation of existing pathological processes. In many patients with leukoplakia and lichen planus, the resolution of the process or a more favorable course of the disease occurs as a result of replacing some prostheses with others, leading to a decrease in the magnitude of microcurrents. The effect occurs in 1-4 months. We should also not forget about the possibility of increased sensitivity of the mucous membrane to mercury, silver and other substances.

In some patients, galvanic current causes unpleasant subjective sensations, a burning sensation in the tongue or other areas of the mucous membrane without pronounced objective changes.

The appearance of complaints in time coincides with the setting of prostheses or metal fillings. Galvanic current in the oral cavity exceeds 10 μA. In these cases, replacing amalgam fillings with cement or plastic ones, and removing metal prostheses help to heal.

Radiation sickness (morbus radialis) develops as a result of exposure to ionizing radiation on the entire body or its large parts: chest, abdomen, pelvis. Radiation injury

can cause any type of ionizing radiation: X-rays, gamma rays, neutron fluxes, etc. In the irradiated tissues, the morphological structure of the vessel walls changes, the barrier function of connective tissue decreases, and regeneration is suppressed. Distinguish between acute and chronic forms of radiation sickness.

Acute radiation sickness (morbus radialis acutus) develops after a single exposure of the body to massive doses (1-10 Gy, or 100-1000 rad). In the first period - the period of primary reactions, which begins soon after irradiation (after 1-2 hours) and lasts up to 2 days, dryness (or salivation) appears in the oral cavity, the taste and sensitivity of the mucous membrane decrease. The mucous membrane of the mouth and lips swells, hyperemia appears, and punctate hemorrhages may occur. In the second, latent period, which lasts from several hours to 2 weeks, all these phenomena disappear. The period of pronounced clinical phenomena (third period) is the height of the disease. Against the background of a sharp deterioration in the general condition, changes in the oral cavity reach a maximum. A burning sensation appears, the mucous membrane becomes anemic, dry. There is a picture of radiation stomatitis.

Changes in blood vessels and blood composition in this

period cause the phenomenon of hemorrhagic diathesis: increased bleeding, hemorrhage. Due to a sharp decrease in tissue resistance, a rapidly developing autoinfection, especially putrefactive, joins. Thus, the clinical picture of radiation stomatitis consists mainly of hemorrhagic syndrome and ulcerative necrotic process. The latter is most pronounced in places of injuries (overhanging fillings, tartar) and where there were accumulations of microflora before the disease, that is, the edge of the gums and tonsils first suffer, and then the lateral surfaces of the tongue, the palate. In places of contact with the mucous membrane of metal prostheses and fillings, the lesion may be more pronounced due to secondary radiation. The mucous membrane of the mouth, gebs and face swell. The gingival papillae are loosened, bleed, then the gum edge is necrotic. The bone tissue of the alveolar process is resorbed, the teeth become loose and fall out. Multiple necrosis of the mucous membrane does not have sharp boundaries, the inflammatory response of the surrounding tissues is weak. The bottom of the ulcers is covered with a dirty gray necrotic plaque with a putrid odor. In severe cases, necrosis can spread from the mucous membrane to the underlying soft tissues and bone, radial bone necrosis with sequestration occurs, and jaw fractures are possible. This is facilitated by the spread of infection from foci of chronic periodontitis and periodontitis.

The tongue swells, becomes covered with abundant plaque, cracks, hemorrhages and necrosis appear, more often in the area of the tongue root. The taste and sensitivity of the tongue are lost. Severe necrotizing sore throats develop. If the patient does not die, then the fourth period begins - recovery, when there is a slow reverse development of the symptoms of the disease. In the oral cavity, everything also returns to a relative norm. During this period, relapses of the disease are possible.

Chronic radiation sickness (morbus radialis chronicus) develops as a result of prolonged exposure to low doses of radiation on the entire body or a significant part of it. The oral cavity is sensitive to the effects of ionizing radiation, therefore, at the onset of the disease, changes in the oral cavity can be especially pronounced. Dryness gradually increases due to damage to the salivary glands. There is a persistent catarrhal gingivitis, which can later turn into ulcerative. Erosions, and then ulcers of the aft type,

can appear on the transitional folds from the vestibular side, and later cover the gums and lips. Glossalgia may develop, and then glossitis with swelling of the tongue,

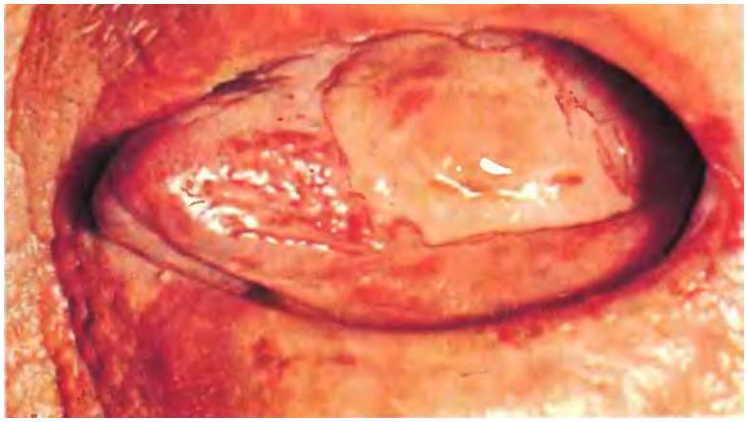
cracks, bloom on the tongue. Long-term course of chronic radiation sickness leads, as a rule, to a picture similar to periodontitis - the so-called radiation periodontitis.

Treatment. With radiation injuries, both general and local therapy are performed. The scope of therapeutic measures differs depending on the degree and stage of the disease, the predominant localization of lesions, the state of various organs and systems. General treatment includes the use of agents that slow down radiochemical reactions - radioprotectors (cystamine, bathylol, etc., which must be administered before irradiation), which reduce the body's radiosensitivity, the use of corticosteroid drugs, antihistamines (suprastin, tavegil, fenkarol), antibacterial drugs (broad antibiotics spectrum of action). They also use substances that restore the functions of the blood system (vitamins B6 and B12, folic acid, sodium nucleinate, pentoxil, coamide), prevent hemorrhagic diathesis (rutin, vicasol, calcium gluconate), multivitamins, produce blood transfusions.

Changes in the oral mucosa during radiation therapy of neoplasms of the maxillofacial region

In the treatment of neoplasms of the maxillofacial region, methods of radiation therapy are used: remote, short-focus and interstitial. In this case, in addition to the affected tissues, normal tissues, including the mucous membrane of the mouth, fall into the sphere of irradiation. The severity of its response to radiation varies widely and depends on the dose rate, single and total absorbed radiation dose, duration of exposure, biological characteristics of the organism, and the state of the oral cavity before exposure.

The clinical picture of the reaction of the mucous membrane to radiation develops gradually - from hyperemia and swelling to the appearance of erosion. This reaction has its own characteristics in different parts of the mucous membrane. The first clinical signs on the mucous membrane, which does not have a stratum corneum in the epithelium (cheeks, floor of the mouth, soft palate), are manifested by mild hyperemia and edema, which gradually increase as the absorbed dose increases. Then the mucous membrane becomes cloudy, loses its luster, thickens, appears wrinkled the surface layer is not removed when scraping. This is due to increased keratinization of the epithelium. Some of the affected areas resemble leukoplakia or lichen planus. If the radiation dose increases, then the keratinized epithelium is first rejected in some areas, desquamating, erosions appear covered with a sticky necrotic plaque - focal membranous radiomucositis, then the epithelium is rejected in large areas, the erosion merges and focal radiomucositis is transformed into a confluent fig. ... The mucous membrane of the soft palate has a high radiosensitivity, the stage of keratinization during irradiation is absent, the reaction develops faster than in other parts of the oral cavity. In areas of the mucous membrane, which normally undergo keratinization, the radiation reaction proceeds more favorably and leads only to focal desquamation of the epithelium or single erosions.



Rice. 11.4. Drained membranous radioactive mucositis. On the lateral surface of the tongue, against the background of pronounced hyperemia, extensive erosion, covered with necrotic plaque.

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The course of pathological processes in the oral mucosa is complicated by damage to the salivary glands (with remote methods of irradiation). Initially, there may be increased salivation (3-5 days), which is quickly replaced by dryness of the oral cavity up to complete xerostomia, which is practically not amenable to stimulation therapy.

The consequence of the death of the taste buds of the tongue is
violation of taste. At first, sensations in the tongue can manifest itself as glossalgia, then a perversion of taste appears, and in the future it is lost.

Radiation changes in the oral cavity are largely reversible. After the termination of irradiation or during a break in treatment, the mucous membrane quickly returns to a relative norm. This period lasts 2-3 weeks. With a large absorbed dose (more than 50-60 Gy, or 5000-6000 rad), irreversible changes may occur in the salivary glands and mucous membrane (edema, hyperemia, telangiectasia, atrophy, radiation ulcers).

Radiation therapy should be preceded by sanitation of the oral cavity, since the reaction of the mucous membrane to radiation exposure is more difficult in the non-sanitized oral cavity.

Due to trophic disorders, the reactivity of the mucous membrane to mechanical injury and infection is sharply reduced. The edematous mucous membrane with defective epithelial cover is easily injured by the sharp edges of the teeth and prostheses, which can lead to the appearance of sharply painful ulcers that do not heal for a long time.

Treatment. Preparation of the oral cavity for radiation therapy is necessary to mitigate the radiation response during treatment and to prevent its complications. Therapeutic measures in the oral cavity are recommended to be carried out in the following sequence:

■ removal under anesthesia of all roots and teeth with periodontitis, as well as sharply mobile teeth no later than 3-5 days before the start of radiation therapy. Removal should be low-traumatic, the wound is sutured after it for faster healing;

■ removal of supra- and subgingival calculus, curettage of gingival and periodontal pockets;

■ treatment of carious teeth, removal of metal prostheses and amalgam fillings to eliminate secondary radiation. This goal can be achieved by imposing on the dentition during irradiation rubber or plastic aligners with a thickness of 2-3 mm (the path length of electrons during secondary radiation at the time of radiation therapy does not exceed 1-2 mm). Instead of mouth guards, you can use gauze or cotton swabs moistened with vaseline oil or novocaine.

Frequent oral irrigations are recommended before irradiation to reduce mucosal response

a solution of adrenaline in isotonic sodium chloride solution (2: 100) or the introduction of adrenaline under the skin, treatment of the mucous membrane with the emulsion of tezan, 5% dieton ointment.

During radiation therapy, smoking, drinking alcohol, irritating food, and wearing removable dentures are prohibited. Food should be crushed, non-irritating, high in calories and fortified. At the initial manifestations of a radiation reaction, it is recommended to rinse your mouth with warm boiled water, a solution of potassium permanganate (1: 5000). The oral cavity, especially the teeth and gingival papillae, are treated 4-5 times a day with tampons moistened with weak antiseptic solutions (0.5% hydrogen peroxide solution, furacilin solutions 1: 5000, 0.25% chloramine solution, 2% boric solution acids, applications of 10% solution of Dimexide), aerosol "Lyoxazol".

At the height of the radiation reaction, the oral cavity and periodontal pockets are washed from a syringe with solutions of weak antiseptics, the mucous membrane is lubricated with 1% citral solution in peach or other vegetable oil, applications are made with rosehip, sea buckthorn oils, Leosin ointments, methyluracilic, olazole , liniment of dibunol.

For local anesthesia, 1-2% solutions of trimecaine, lidocaine or 10% oil solution of anesthesin are used.

Surgical intervention, including tooth extraction, curettage of periodontal pockets, is contraindicated at this time. You should refrain from them for several months or years. Relative normalization of the mucous membrane occurs in 1-2.5 months.

In case of radiation ulcers, in addition to general (vitamin therapy), local treatment is necessary. A thorough antiseptic treatment of the oral cavity and radiation ulcer is performed daily under anesthesia. Necrotic tissues are removed using solutions of proteolytic enzymes (lysoamidase, ronidase, DNase, etc.). With some success over a long period of time (up to 1.5-2 months), applications of ointments ("Iruksol", "Levosin", "Propolis", "Solcoseryl", "Actovegin", etc.), fortified oils (sea buckthorn , wild rose), galascorbin.

Long-term non-healing radiation ulcers that are not amenable to conservative therapy can be radically excised within healthy tissues with subsequent plasty. The surgical method is indicated in the case of local irradiation (short-focus X-ray therapy, interstitial gamma therapy). With external methods of radiation therapy, the effectiveness of surgical treatment will be less.

If necessary, treatment of radiation injuries of the teeth and radiation osteomyelitis should be carried out.

Chemical damage (trauma chymicum) can be both acute and chronic. Acute injuries occur as a result of exposure to the mucous membrane of chemicals in a highly damaging concentration. Most often this happens with an accidental injury at home, at work, or attempting to commit suicide, as well as during an appointment with the dentist. Recently, dentists have abandoned many cauterizing agents, but there are still some potent chemicals in practice that require careful handling. Burns of the oral mucosa can occur upon contact with acids, bases (alkalis), the use of arsenous paste, phenol, formalin, silver nitrate, formalin-resorcinol mixture, when wearing prostheses made of insufficiently polymerized plastic.

A distinction should be made between burns resulting from exposure to high concentrations of chemicals and allergic reactions such as plastics, amalgam, low concentrations of chemicals. A burn and an allergic reaction can be combined. Sometimes patients, trying to get rid of toothache on their own, apply alcohol, acetylsalicylic acid and other substances to the tooth, and along the way to the mucous membrane, which leads to burns.

The clinical picture. In acute lesions, sharp pain occurs, as a rule, immediately and is localized in the places where the chemical has entered. The clinical picture depends on the nature and amount of the damaging substance, the time of action.

Acid burns lead to the appearance of coagulation necrosis on the mucous membrane - a dense film of brown (from sulfuric acid), yellow (from nitric acid) or white-gray (from other acids) color. Films are located

gnawed against the background of pronounced inflammation with edema and hyperemia, are tightly connected to the underlying tissues.

A burn with bases (alkalis) leads to colliquation necrosis of the mucous membrane, while a dense film does not form, the necrotic tissues have a gelatinous consistency. The lesion is deeper than with an acid burn. Alkaline burn necrosis can involve all layers of soft tissue, especially on the gums and hard palate. Burns, especially extensive ones, cause severe suffering to the patient. A few days after the rejection of necrotic tissue, slowly healing erosive or ulcerative surfaces are exposed. In mild cases, alkali burns can only cause a catarrhal inflammatory process.

Treatment. After contact of a chemical substance on the mucous membrane, it is necessary to remove it as quickly as possible. If such a burn occurs in front of a doctor,

you should immediately start rinsing the oral cavity (rinsing, irrigation) with a weak solution of a neutralizing substance. If the doctor does not have it, then they rinse with water, and then quickly prepare the necessary neutralizing solution. For acid burns, soapy water, 1% lime water, burnt magnesia with water, 0.1% ammonia solution (15 drops per glass of water) are used. Alkalis are neutralized with 0.5% solutions of citric, acetic acids (1/4 teaspoon of acid per glass of water), 0.1% solution of hydrochloric acid (10 drops per glass). Thus, it is possible to stop the further penetration of the chemical into the tissues. About arsenous acid burns.

To reduce the absorption of concentrated solutions of silver nitrate, a 2-3% solution of sodium chloride or Lugol's solution is used, while insoluble silver compounds are formed. In case of a phenol burn, the mucous membrane is treated with 50% ethyl alcohol or castor oil.

Further treatment of patients with chemical burns is carried out according to the type of therapy for an acute nonspecific inflammatory process: pain relievers are used (1% trimecaine solution, trimecaine with hexamethylenetetramine, 1-2% lidocaine solution, etc.), weak solutions of antiseptic agents in the form of oral baths and rinses, preparations accelerating epithelization (1% citral solution in peach oil, vitamins A and E, aevit, rosehip oil, sea buckthorn, etc.). Recommended

There is a non-irritating high-calorie mashed food, cold (ice cream), inside multivitamins.

With extensive scars, surgical intervention is indicated.

Критерии оценки текущего контроля

№	Progress in% and points	Grade	Student knowledge level
1.	96-100	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Shows high activity, creative approach when conducting interactive games. Correctly solves situational problems and test questions. Analyzes independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
2.	91-95	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Analyzes research results independently. Understands the essence of the issue.

			Knows, tells confidently. Has an accurate idea.
3.	86-90	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
4.	81-85	Good "4"	The correct answer to questions about the clinic, diagnosis and treatment of TBI, but there are 2-3 inaccuracies, errors. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
5.	76-80	Good "4"	Correct, but incomplete coverage of the issue. The student knows questions about the clinic and diagnosis of TBI, but is not fully versed in determining the tactics of treatment. Correctly solves situational problems, but the rationale for the answer is not complete. Understands the essence of the issue. Knows, speaks confidently
6.	71-75	Good "4"	Correct, but incomplete coverage of the issue. Correctly solves situational problems, but the rationale for the answer is incomplete. Has 2-3 errors. Knows, tells confidently. Has accurate ideas.
7.	66-70	Satisfactorily "3"	Understands the essence of the issue. Correctly solves situational problems, but cannot substantiate the answer. Has accurate ideas on specific issues.
8.	61-65	Satisfactorily "3"	Has an incomplete understanding of the topic. Makes mistakes when solving situational tasks. Tells not confidently.
9.	55-60	Satisfactorily "3"	Knows, tells not confidently. Has a partial view.
10.	54 and below	Unsatisfactory "2"	Has no exact idea. Does not know.

5- Practical lesson
Topic: Leukoplakia, clinic, diagnosis, treatment

Technological map of the practical lesson

Stages of work	Teacher	Student
1. Stages of preparation (10 minutes)	1. Controls the purity of the audience; 2. Checks students' readiness for classes; 3. Controls attendance;	Listen
2.The introductory stage to classes (15 minutes)	1.Preparation on the topic of content; 2. Preparation of slides for the introductory report; 3. To develop the literature used for the study of the subject;	Write down the topic and listen
3. The main stage (195 minutes)	1. Dividing students into 2 small subgroups, asking questions on the topic; 2. Use of slides and multimedia; 3. Carries out medical work; 4. Combines all information on a given topic, actively the participating students are encouraged and appreciated;	Divided into small groups watching, participating, listening. The student expresses his opinion, complements and asks questions
4. Final stage (50 minutes)	1. Conclusion. 2. Independent work. 3. Homework.	Listen Write down

Questions on the topic
1.What is Leukoplakia.
2.Etiology of Leukoplakia
3.Leukoplakia treatment
4.Dif - diagnosis of leukoplakia
5.Prevention of leukoplakia.
6.Dispanserization

Tests

The element of defeat in a simple form of leukoplakia
1) bubble
2) stain
3) papule
4) atrophy
5) crack

Localization of leukoplakia elements
1) leather
2) mucous membrane
3) skin and mucous membrane

The verrucous form of leukoplakia includes
1) to dermatosis
2) to cheilit
3) to obligate precancer
4) to an optional precancer
Pathological changes in the epithelium in leukoplakia are based on
1) spongiosis
2) hyperkeratosis
3) acantholysis
4) dyskeratosis
Tuppainer's leukoplakia occurs
1) with allergic stomatitis
2) when smoking
3) in case of intoxication with salts of heavy metals

Differential diagnosis of verrucous leukoplakia
spend
1) with glossalgia
2) with desquamative glossitis
3) lichen planus
4) with exudative erythema multiforme

The most significant causes of leukoplakia
1) stress
2) smoking
3) microcurrents
4) hypothermia
5) trauma from sharp edges of teeth

Clinical signs of premalignant malignancy
1) lip swelling
2) compaction at the base of the lesion
3) strengthening of keratinization processes
4) the appearance of fibrinous plaque
5) a rapid increase in the size of the focus
When treating leukoplakia, it is recommended
1) novocaine blockade
2) quitting smoking
3) sanitation of the oral cavity
4) replacement of amalgam fillings
5) the use of photoprotective agents

In case of ineffectiveness of conservative treatment of leukoplakia
use
1) physiotherapy
2) radiation therapy

3) surgical excision
4) chemotherapy

For differential diagnosis of leukoplakia and candidiasis
apply research method
1) serological
2) cytological
3) bacterioscopic
4) immunological

Duration of conservative therapeutic measures
with an erosive form of leukoplakia
1) one year
2) one month
3) two weeks

With an erosive form of leukoplakia, it is recommended
1) pain relievers
2) cauterizing agents
3) antiseptics
4) epithelizing ointments
5) sanitation of the oral cavity

Interactive method
USING THE WEB METHOD

Steps:
In advance, students are given time to prepare questions for the lesson they have completed.
Participants sit in a circle.
One of the participants is given a skein of thread, and he asks his prepared question (to which he himself must know the full answer), holding the end of the thread and throwing the skein to any student.
The student who received the skein answers the question (at the same time, the person who asked him comments on the answer) and passes the question relay on. Participants continue to ask and answer questions until everyone is on the web.
As soon as all students finish asking questions, the student holding the skein returns it to the participant from whom he received the question, while asking his own question, etc., until the coil is completely "unwound"

Text

Leukoplakia (leucoplakia) is a chronic disease of the mucous membrane of the mouth or the red border of the lips, accompanied by increased keratinization of the epithelium and inflammation of the mucous membrane itself.

Leukoplakia is observed not only in the oral cavity, it occurs, although rarely, on other mucous membranes (bladder, genitals, rectum, nose, esophagus, trachea). In children, leukoplakia is extremely rare. The disease is observed mainly in middle-aged and older people, more often in men, which is probably due to their exposure to the oral mucosa of a large number of strong irritants (smoking, alcohol, mechanical trauma). Leukoplakia is an optional precancer.

Etiology. Local irritants (mechanical, thermal, chemical) play the main role in the occurrence of leukoplakia. The most common cause of leukoplakia is smoking, in which a combined effect of thermal and chemical irritants (ammonia and phenolic compounds, nicotine, tar derivatives) is exerted on the oral mucosa. In the occurrence of leukoplakia, the effects of weak, but long-term irritants are important: the use of very hot or spicy food, strong alcoholic beverages, chewing tobacco, betel nut, the use of nasa (nasa contains ash, tobacco, slaked lime, cottonseed oil or water) , adverse effects of meteorological factors (strong insolation, cold, wind). Mechanical irritation can be caused by sharp edges of teeth, roots of decayed teeth, a large amount of tartar, poor-quality dentures, defects in the dentition, anomalies in the position of individual teeth. Galvanic current that occurs between various metals of crowns and metal fillings in the mouth can also be one of the etiological factors of leuko-

placia. In some people, increased keratinization occurs in the places where the amalgam fillings adhere to the oral mucosa. Replacing amalgam fillings with plastic or cement fillings leads to the rapid disappearance of the area of hyperkeratosis.

Leukoplakia can occur as a result of exposure to unfavorable occupational factors (aniline paints and varnishes, pitch, coal tar, phenol, some benzene compounds, formaldehyde, products of dry distillation of coal, gasoline vapor, bromine, etc.).

In the occurrence of leukoplakia, it is also of great importance to reduce the resistance of the oral mucosa to external stimuli, which is primarily associated with the state of the gastrointestinal tract, stress, hypovitaminosis A, hormonal disorders, and genetic factors.

The clinical picture. The initial manifestations of leukoplakia usually go unnoticed, since there are no subjective sensations. Leukoplakia usually begins with the appearance of areas of opacity of the epithelium, grayish in color with elements of keratinization on the surface. The lesion occurs against the background of an unchanged mucous membrane. Typical localization of leukoplakia foci is the mucous membrane of the cheeks along the line of closing of the teeth in the anterior region, the corners of the mouth and the red border of the lower lip without skin damage. The back and lateral surfaces of the tongue are somewhat less frequently affected. Kurilikirv is characterized by a lesion of the palate, described under the name "leukoplakia of Tuppainer's smokers", as well as triangular foci of hyperkeratosis in the corners of the mouth, with the apex facing the molars.

There are the following forms of leukoplakia: flat, or simple, verrucous and erosive. These forms can transform one into another. A combination of different forms of leukoplakia in different parts of the oral mucosa in the same patient is possible. The disease begins with the appearance of a flat form.

Flat, or simple, leukoplakia (leucoplakia plana) occurs most often. This form usually does not cause subjective sensations and is discovered by chance. Sometimes patients complain of a feeling of tightness, burning, and an unusual appearance of the mucous

membrane. In the presence of extensive foci on the tongue, a decrease in taste sensitivity is possible. The main morphological element of the lesion in flat leukoplakia is a hyperkeratic spot, which is a cloudy area.

epithelium with clear contours. The examination reveals foci of hyperkeratosis of various shapes and sizes, which do not rise above the level of the mucous membrane of the surrounding areas, but with clear boundaries of the lesion. Elements of flat leukoplakia resemble a burn of the mucous membrane with lapis, glued on thin tissue paper, or a white coating that does not come off even with intense scraping. Keratinization is of varying intensity, so the color of the affected areas varies from pale grayish to intense white. The surface of the plot is flat
Leukoplakia is usually slightly rough and dry. There is no compaction at the base of the lesion, just as there is no visible inflammatory reaction along its periphery.

There is a direct relationship between the shape, color, size of the areas of keratinization and their localization. So, with hyperkeratosis of the mucous membrane in the area of the corners of the mouth in 85% of cases, there is a symmetry of the lesion. The shape of the focus has the form of a triangle, the base of which is directed towards the corner of the mouth, and the apex towards the retromolar space. With the localization of the leukoplakia focus on the mucous membrane of the cheeks along the line of closing the teeth, the elements of the lesion represent an elongated line, the continuity of which may be disrupted in certain areas. Leukoplakia on the red border of the lips looks like a sticky tissue paper of irregular shape, grayish-white in color (Fig. 11.5). On the mucous membrane of the tongue, hard palate, floor of the mouth, limited areas of lesion are formed, sometimes resembling wide stripes or solid spots. More extensive lesions are also noted.

The relief of the mucous membrane, its turgor, is also reflected in the appearance of the lesion. So, if leukoplakia develops against the background of the folding of the tongue, the protruding areas become more keratinized, and the surface of the tongue resembles a cobblestone pavement. A similar picture can be seen with a decrease in turgor and folding on the cheeks. If there is no pronounced folding on the tongue, flat leukoplakia looks like grayish-white, slightly sunken spots, where the papillae of the tongue are smoothed.

Under favorable conditions, flat leukoplakia can exist for years, decades and not cause concern, not progress and not transform into more pronounced forms of the disease.

Leukoplakia of smokers Tappeiner (Ieucoplakia nicotinica Tappeiner, nicotinic leukokeratosis of the palate) occurs in
Rice. 11.5. Flat leukoplakia. A focus of hyperkeratosis on the lower lip with clear boundaries.

Rice. 11.6. Tuppainer's leukoplakia. Hyperkeratosis of the mucous membrane of the hard and soft palate with small red nodules - hyperplastic salivary glands and a point of the dilated excretory duct.

hard-core smokers (especially pipes). It is mainly the mucous membrane of the hard palate and the adjacent part of the soft palate that is affected. Sometimes the gum edge is involved. The mucous membrane in the affected area is grayish-white, often folded. Against this background, mainly in the posterior half of the hard palate, red dots stand out - gaping orifices of the excretory ducts of the acid-like dilated salivary glands,

which look like small nodules (Fig. 11.6). They are formed due to blockage of the excretory ducts with hyperkeratic masses. The defeat of the palate with leukoplakia of a smoker can combine -

Rice. 11.7. Verrucous leukoplakia.

Hyperkeratic plaques with an uneven surface protruding above the buccal mucosa.

Xia with the arrangement of elements on the mucous membrane of the cheeks, corners of the mouth, lower lip, etc. This form of leukoplakia refers to easily reversible processes: the cessation of smoking (irritating factor) leads to the disappearance of the disease.

Verrucous leukoplakia (leucoplaria verrucosa) is the next stage in the development of flat leukoplakia. This is facilitated by local irritants: trauma by sharp edges of teeth and dentures, biting of leukoplakia areas, smoking, eating hot and spicy food, microcurrents, etc.

The main sign that distinguishes this form of leukoplakia from flat is a more pronounced keratinization, in which a significant thickening of the stratum corneum is found. The area of leukoplakia rises significantly above the level of the mucous membrane and sharply differs in color (Fig. 11.7). On palpation, superficial compaction can be determined. Patients usually complain of a feeling of roughness and tightness of the mucous membrane, burning and soreness during meals, especially acute.

Distinguish between plaque and warty forms of leukoplakia. The plaque form of leukoplakia has the appearance of limited milky-white, sometimes straw-yellowish plaques, towering over the surrounding mucous membrane and having clear edges. The warty variety of verru-
goat leukoplakia is characterized by dense, lumpy or warty formations that protrude sharply above the level of the mucous membrane. The warty form of leukoplakia has a greater potential for malignancy in comparison with the plaque form. On palpation, the elements of the lesion are dense, painless, not soldered to the underlying areas of the mucous membrane. The thickness of the raised areas of hyperkeratosis is not the same and ranges from significant to barely detectable during normal examination. With a slight elevation of the elements, the area of the lesion is almost undetectable by palpation. With a pronounced thickness of the leukoplakic focus, it becomes dense to the touch and it is not possible to take it into the fold.

Erosive leukoplakia (Ieucoplakia erosiva) is actually a complication of simple or verrucous leukoplakia due to trauma. With this form of leukoplakia

patients have complaints of soreness, aggravated by all types of stimuli (eating, talking, etc.). Usually, against the background of foci of simple or verrucous leukoplakia, erosions, cracks, and less often ulcers occur (Fig. 11.8). Erosions are difficult to epithelialize and often recur. Patients are especially worried about erosion on the keratinized area of the red border of the lips. Under the influence of sun exposure and other irritating factors, they increase in size, showing no tendency to heal. The painful sensations are intensified.

Pathohistological changes in leukoplakia are characterized by thickening of the epithelium due to the growth of the stratum corneum and granular layers. The stratum

corneum reaches a considerable thickness, especially in the verrucous form of leukoplakia. In it, foci of hyperkeratosis often alternate with foci of parakeratosis (Fig. 11.9). The granular layer of the epithelium in the affected area has varying degrees of severity. The verrucous and erosive form of leukoplakia is characterized by a pronounced thickening of the epithelium due to hyperkeratosis and acanthosis. In the connective tissue stroma of the affected areas of the mucous membrane, diffuse chronic inflammation is determined with pronounced infiltration of its surface layers by lymphocytes and plasma cells, as well as with the manifestation of sclerosis. The latter explains poor healing and frequent relapses with the appearance of cracks and erosions, which is most characteristic of the plaque form of leukoplakia.

Leukoplakia refers to precancerous conditions, since all of its forms are capable of malignancy with varying degrees of probability, transforming into spinocellular cancer. Flat leukoplakia is malignant in 3-5% of patients, and in some the process of malignancy is rapid (1-1.5 years), in others the disease can exist for decades without transforming into cancer. Most often, malignancy of the verrucous and erosive forms of leukoplakia occurs (in 20-30% of cases). Clinical signs of malignancy are increased keratinization processes; a rapid increase in the size and density of the focus; the appearance of compaction at the base of the plaque, erosion; papillary growths on the surface of erosion; bleeding with injury; the appearance of non-healing cracks.

The diagnosis is clarified with an urgent cytological or histological examination.

Differential diagnosis. Leukoplakia should be differentiated from:

And lichen planus;

And lupus erythematosus;

A secondary syphilis;

A chronic hyperplastic candidiasis;

A clouding of the epithelium during its regeneration;

Bowen's disease;

A mild leukoplakia;

And keratinizing squamous cell carcinoma.

Leukoplakia differs from lichen planus by the nature of the lesion elements: with leukoplakia, it is a hyperkeratotic spot or plaque; with lichen planus - multiple papules on the mucous membrane of the mouth, merging, as a rule, into a patterned pattern, and in some cases also multiple papules on the skin. With leukoplakia, the skin is never affected. Signs of difference also include the absence of inflammation or their insignificant severity in leukoplakia and the presence of inflammation of the oral mucosa with lichen planus (except for the typical form).

Leukoplakia differs from lupus erythematosus in the absence of bright erythema with typical outlines, atrophy and characteristic skin lesions.

In the differential diagnosis of leukoplakia with syphilitic papules, which have an external similarity with it, the following differences are taken into account: the horny masses in leukoplakia are not removed by scraping, while the grayish-white plaque covering the syphilitic papules is easily removed, exposing the erosion on the surface of which it is found many pale trepans. At the base of leukoplakia, there is no infiltrate characteristic of a syphilitic papule. The Wassermann reaction and sedimentary reactions in secondary syphilis are usually positive.

Unlike leukoplakia, a whitish plaque in chronic hyperplastic candidiasis is removed by scraping and a large number of Candida fungi are found in it in the form of spores or pseudomycelium.

In some cases, leukoplakia is mistaken for clouding of the epithelium in the process of epithelialization of erosions or ulcers of various etiologies, which spontaneously disappears after the completion of epithelialization.

In Bowen's disease, in contrast to leukoplakia, in addition to a grayish-white plaque, when removed, a velvety stagnant red surface is exposed,

there is an inflammatory reaction around the lesion. In addition, the lesion area in Bowen's disease is most often located on the soft palate, arches, uvula, where leukoplakia foci are extremely rare.

Leukoplakia is differentiated from soft leukoplakia, which looks like a whitish-gray, as if scaly surface. Unlike true leukoplakia, with mild leukoplakia, this whitish-gray plaque is removed by scraping, since it is a consequence of parakeratosis.

Keratinizing squamous cell carcinoma, in contrast to leukoplakia, has a compaction at the base and a characteristic cytological and histological picture - the presence of atypical cells.

When leukoplakia is localized on the red border of the lip, differential diagnosis with limited precancerous hyperkeratosis is performed. It is distinguished from leukoplakia by the presence of hyperkeratic scales on the surface, the retardation of the lesion focus and its small size.

Treatment. The volume of therapeutic measures for leukoplakia is determined primarily by the form of its course, as well as the size of the lesion and the rate of development of the process. Flat leukoplakia after elimination of irritating factors, smoking cessation can regress with the complete disappearance of foci of hyperkeratosis. However, when smoking resumes, leukoplakia develops again. Treatment for a flat form of leukoplakia consists of the following stages: elimination of local irritating factors; normalization of metabolic processes in the epithelium with the use of multivitamins and primarily vitamin A.

Patients undergo a thorough sanitation of the oral cavity with grinding the sharp edges of the teeth; rational prosthetics; amalgam fillings, if indicated, are changed to plastic or cement. When leukoplakia is localized on the red border of the lips, patients should be exposed to insolation as little as possible; when going outside, the lips should be lubricated with a photoprotective cream "Ray" or hygienic photoprotective lipstick. It is very important to completely stop smoking, taking hot, spicy foods, alcoholic beverages. The dangers of smoking should be explained to the patient and, if necessary, referred to special treatment. The use of cauterizing and irritating agents is categorically contraindicated due to the possible malignancy of the process. Along with the elimination of local irritants for

to increase the resistance of the oral mucosa, treatment of concomitant diseases of the gastrointestinal tract is important.

Inside appoint vitamin A (3.4% solution of retinol acetate in oil or 5.5% solution of retinol palmite in oil) 10 drops 2-3 times a day for 1.5-2 months. Locally, applications are carried out with the indicated oil solutions of vitamin A 3-4 times a day. Treatment continues for 1.5-2 months. The course of treatment is repeated 2-3 times a year. These

therapeutic measures usually lead to the disappearance of leukoplakia or its stabilization.

If candidiasis is detected with leukoplakia, then at the same time, treatment with antifungal agents is carried out.

Treatment for verrucous and erosive forms of leukoplakia is initially carried out in the same way as for flat. If it does not lead to positive dynamics (the transition of the verrucous form to the flat one) within 1 month, it is necessary to apply surgical methods of treatment: excision of the lesion within the healthy tissues, followed by histological examination. Removal of leukoplakia areas can be performed using electrocautery or cryodestruction.

Particular attention should be paid to the treatment of erosions and ulcers on the surface of leukoplakia foci in order to prevent malignancy of the process. Treatment of the erosive form of leukoplakia, in addition to the above, includes the local use of agents that stimulate the epithelialization of the mucous membrane (application of oil solutions of vitamins A, E, rosehip oil, sea buckthorn, etc.), and in the presence of pain, local anesthetic drugs (lidocaine, trimecaine, pyromecaine). If erosion or ulcers under the influence of drug treatment do not epithelize within 2 weeks, then it is necessary to excise the focus with an urgent histological examination.

Patients with leukoplakia should be under constant dispensary supervision of the dentist, being for control every 3-4 months. At the slightest suspicion of the possibility of malignancy, it is urgently necessary to perform a biopsy followed by a histological examination, and then decide on the tactics of further treatment.

Prevention. The basis for the prevention of leukoplakia is the timely prevention of trauma to the oral mucosa: smoking, eating hot and spicy food, poor quality dentures, sharp edges of teeth, galvanic currents and other irritants.

11.2.5. Pashkov's mild leukoplakia

BM Pashkov in 1964 described mild leukoplakia - a disease that other authors also call "leukoedema" (the surface of the mucous membrane is smooth) and "cheek biting" (the surface is rough).

Mild leukoplakia (leucoplakia mollis) is a benign lesion of the oral mucosa. It is characterized by periodically advancing desquamation of the surface layers of the epithelium. Mild leukoplakia is observed mainly at a young age, mainly in children and persons under 30 years of age, somewhat more often in women and usually proceeds without pronounced subjective sensations. It is found mainly during dental treatment or during preventive examinations of the oral cavity. Sometimes patients complain of roughness, peeling of the mucous membrane, a feeling of thickened interfering tissue. Often, patients try to get rid of these sensations by biting on elevated areas, which often becomes a habit.

Etiology. The disease is hereditary and is a type of epithelial dysplasia.

The clinical picture. It is characterized by the presence of areas of desquamation of a slightly edematous, pasty mucous membrane with indistinct boundaries without inflammation and induration (Fig. 11.10). The mucous membrane is grayish-white in color. Most often, mild leukoplakia is localized on the mucous membrane of the cheeks, lips, less often on the tongue and gums. The lesion can be limited, localized on the mucous membrane along the line of teeth closing, or diffuse, when almost the

entire mucous membrane is involved in the process, and sometimes the red border of the lips. With the habitual biting or sucking of the cheeks, lips, tongue, which is often observed in patients with neuropathy, predominantly of a young age, the epithelium along the line of closing the teeth is unevenly desquamated, has a fringed appearance due to multiple small patches of epithelium, which causes the unevenness of the affected surface (it is, as it were, eaten away by moths). This altered epithelium is partially removed by scraping. In severe cases, painful erosions from gnawing of the epithelium may occur.

Pathological anatomy. Histologically, pronounced parakeratosis, acanthosis are noted, at all levels of the styloid layer there are so-called light cells, the cytoplasm of which is almost not stained, and the nuclei are deformed (pyknotized). In the connective tissue layer

Rice. 11.10. Mild leukoplakia. The mucous membrane of the cheek is pasty, whitish in color.

expansion of small blood vessels, thickening of collagen, thinning of elastic fibers.

Diagnostics. It does not cause any difficulties. The typical appearance of the mucous membrane and the habit of constantly biting the cheeks, often pressing the mucous membrane between the teeth with a finger, allows a correct diagnosis to be made.

Differential diagnosis. The disease should be differentiated from:

^ flat leukoplakia;

A typical form of lichen planus;

^ candidiasis;

^ drug-induced stomatitis.

Treatment. An oily solution of vitamin A, vitamins of group B, C is prescribed, and the oral cavity is sanitized. Treatment sometimes helps to eliminate the process, but more often only temporary improvement is noted.

Критерии оценки текущего контроля

№	Progress in% and points	Grade	Student knowledge level
1.	96-100	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Shows high activity, creative approach when conducting interactive games. Correctly solves situational problems and test questions. Analyzes independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
2.	91-95	Fine	Complete correct answer to questions

		"5"	about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Analyzes research results independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
3.	86-90	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
4.	81-85	Good "4"	The correct answer to questions about the clinic, diagnosis and treatment of TBI, but there are 2-3 inaccuracies, errors. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
5.	76-80	Good "4"	Correct, but incomplete coverage of the issue. The student knows questions about the clinic and diagnosis of TBI, but is not fully versed in determining the tactics of treatment. Correctly solves situational problems, but the rationale for the answer is not complete. Understands the essence of the issue. Knows, speaks confidently
6.	71-75	Good "4"	Correct, but incomplete coverage of the issue. Correctly solves situational problems, but the rationale for the answer is incomplete. Has 2-3 errors. Knows, tells confidently. Has accurate ideas.
7.	66-70	Satisfactorily "3"	Understands the essence of the issue. Correctly solves situational problems, but cannot substantiate the answer. Has accurate ideas on specific issues.
8.	61-65	Satisfactorily "3"	Has an incomplete understanding of the topic. Makes mistakes when solving situational tasks. Tells not confidently.
9.	55-60	Satisfactorily "3"	Knows, tells not confidently. Has a partial view.
10.	54 and below	Unsatisfactory "2"	Has no exact idea. Does not know.

6- Practical lesson

Topic: Manifestation of viral diseases of the oral cavity (Herpes zoster and herpangina, herpes simplex) Clinic, Dif-diagnosis, treatment

Technological map of the practical lesson

Stages of work	Teacher	Student
1. Stages of preparation (10 minutes)	1. Controls the purity of the audience; 2. Checks students' readiness for classes; 3. Controls attendance;	Listen
2.The introductory stage to classes (15 minutes)	1.Preparation on the topic of content; 2. Preparation of slides for the introductory report; 3. To develop the literature used for the study of the subject;	Write down the topic and listen
3. The main stage (195 minutes)	1. Dividing students into 2 small subgroups, asking questions on the topic; 2. Use of slides and multimedia; 3. Carries out medical work; 4. Combines all information on a given topic, actively the participating students are encouraged and appreciated;	Divided into small groups watching, participating, listening. The student expresses his opinion, complements and asks questions
4. Final stage (50 minutes)	1. Conclusion. 2. Independent work. 3. Homework.	Listen Write down

Questions on the topic

What viral diseases can be found in SOPR

Dif - diagnosis and clinical picture of herpes simplex.

Clinic and Dif - diagnosis of herpes zoster.

Herpangia clinic.

Treatment of viral diseases.

Yashura clinic

Treatment and differential diagnostics of Yanshur.

Tests

A viral disease that affects the red border of the lips is called
1) atopic cheilitis
2) glandular cheilitis
3) chronic recurrent herpes
4) chronic recurrent aphthous stomatitis

To clarify the diagnosis, you should carry out
additional research methods
1) cytological
2) sialography
3) bacterioscopy
4) skin allergy tests

The study of smear-prints with herpes simplex shows the presence
1) polymorphonuclear neutrophils at various stages of necrobiosis
2) lymphocytes
3) giant multinucleated cells
4) acantholytic cells

The primary element of defeat
with chronic recurrent herpes is
1) afta
2) crust
3) papule
4) erosion
5) bubble
6) flake

Pathological process as a result of fluid accumulation
between the cells of the prickly layer is called
1) acanthosis
2) spongiosis
3) parakeratosis
4) papillomatosis
5) ballooning degeneration

Recurrence of the disease is possible as a result
1) stress
2) dysbiosis
3) hypothermia
4) increased insolation
5) activation of latent infection

6) activation of opportunistic microflora
... Chronic recurrent herpes needs to be differentiated
1) with syphilis
2) with leukoplakia
3) with Vincent's stomatitis
4) with contact allergic cheilitis
5) with acantholytic pemphigus
6) with exudative erythema multiforme

Disease Clinical Symptoms
1) shingles a) the general condition does not suffer
2) chronic b) neuralgic pain
recurrent herpes c) the elements of the lesion are grouped
to the local focus
d) elements of defeat are located
along the nerve trunk

Processing of elements of defeat
with chronic recurrent herpes in the first days of the disease
1) antiseptic treatment
2) antiviral drugs
3) pain relief
4) treatment with enzymes
Erosion treatment for chronic recurrent herpes
on the 5-7th day of the disease
1) antiseptic treatment
2) epithelial drugs
3) pain relief
4) enzymes
For general treatment, use
1) aevit 1 tablet once a day
2) acyclovir 0.2, 1 tab. 5 times a day
3) diazepam 0.005, 1 tab. before bedtime
4) Bonafton 0.1, 1 tab. 5 times a day
5) nystatin 500,000 U - 5tab. 3 times a day
For local treatment, use
1) oil solution of vitamin A
2) acyclovir - ointment
3) injections of vitamin PP under the lesions
4) solcoseryl - ointment
5) nystatin ointment
6) a suspension of anesthesin in peach oil
In order to lengthen the period of remission, it is recommended
1) sedative effect on the central nervous system

2) blockade of H-cholinergic receptors
3) stimulation of the production of endogenous interferon
4) desensitization
5) stimulation of salivation

For the purpose of non-specific desensitization, it is used
1) leukocyte interferon
2) cultured inactivated herpes vaccine
3) ascorbic acid
4) histaglobulin

A secondary element of the lesion in chronic recurrent
herpetic stomatitis is
1) afta
2) bubble
3) stain
4) erosion
5) bubble
6) plaque

The causative agent of herpetic stomatitis remains latent
state in:
1) implicit endings of the mucous membrane
2) sensitive ganglia of the central nervous system
3) suture ends of the skin

The elements of the lesion on the mucous membrane are preceded by:
1) neuralgic pain
2) hyperemia of the skin
3) feeling of tension and itching
4) headache, malaise

Interactive method

USING THE WEB METHOD
Steps:
In advance, students are given time to prepare questions for the lesson they have completed.
Participants sit in a circle.
One of the participants is given a skein of thread, and he asks his prepared question (to which he himself must know the full answer), holding the end of the thread and throwing the skein to any student.
The student who received the skein answers the question (at the same time, the person who asked him comments on the answer) and passes the question relay on. Participants continue to ask and answer questions until everyone is on the web.

As soon as all students finish asking questions, the student holding the skein returns it to the participant from whom he received the question, while asking his own question, etc., until the coil is completely "unwound"

Text

Viral diseases of the oral mucosa occupy a significant place in terms of prevalence in the group of diseases of the mucous membrane. Several types of viruses are present in the oral cavity of an adult. Most often, they are hidden in the body, without causing disease. With a decrease in the body's resistance, viruses actively multiply and cause disease. In healthy people, the oral cavity may contain herpes simplex virus, cytomegalovirus, adenoviruses, some enteroviruses, reoviruses and some others (asymptomatic carrier of viruses).

11.3.1.1. Simple (common) herpes

Herpes simplex is the most common viral disease caused by the herpes simplex virus, or herpesvirus. According to antigenic properties, herpes simplex viruses are divided into two main groups - 1 and 2. Type 1 viruses cause herpetic lesions on the oral mucosa, and type 2 - on the genitals. The herpes simplex virus is DNA-containing. In the human body, it multiplies mainly in the epithelial cells. Having penetrated into the human body and causing the manifestation of a primary herpes infection, it remains, basically, throughout a person's life in a latent state or causes relapses of the disease. Herpes simplex virus is found in 75-90% of the adult population. Primary infection usually occurs at the age of 1 to 3 years, when the amount of antibodies received from the mother disappears or sharply decreases in the child's blood, and the body becomes

susceptible to infection with the herpes virus. Very often, the primary infection of a child passes imperceptibly, but in some cases a pronounced manifestation of the disease is observed in children - the so-called primary herpes. This disease is especially difficult in the first months of a child's life, when generalized lesions of the mucous membranes, skin, eyes and other organs occur. In rare cases, primary herpes develops in adults who have not had previous contact with the herpes virus. After recovery from primary herpes, the infection may not recur, but may go into a latent state and recur under the influence of various adverse factors (hypothermia, stress situations, tonsillitis, flu, pneumonia, diseases of the gastrointestinal tract, etc.).

Infection with herpes simplex occurs through direct contact with a patient with herpes or a virus carrier, since the herpes virus is isolated from saliva in individuals without visible manifestations of infection; as well as by airborne droplets.

Clinically, herpetic infection in the oral cavity manifests itself in two forms: acute herpetic stomatitis (primary herpes); chronic recurrent herpes (recurrent herpetic stomatitis).

ACUTE HERPETIC STOMATITIS (stomatitis herpetica acuta). The disease used to be called "acute aphthous stomatitis". Depending on the severity of disorders of the general condition and the prevalence of lesions on the oral mucosa, mild, moderate and severe forms of acute herpetic stomatitis are distinguished. In the pathogenesis of the disease, there are periods of incubation, prodromal (catarrhal), a period of rashes (development of the disease), extinction and clinical recovery (convalescence).

The disease is contagious for people who have not previously been infected with the virus. Primary herpes in newborns is extremely difficult.

The clinical picture. The incubation period lasts 1 to 4 days. In the incubation and especially in the prodromal period, there is an increase in the submandibular, in severe cases of the cervical lymph nodes. The disease begins acutely. Depending on the severity, the body temperature rises to 37-41, general malaise, weakness, headache, skin and muscle hyperesthesia, pallor of the skin, nausea and vomiting of central origin are noted, since the herpes simplex virus

Rice. 11.11. Acute herpetic stomatitis. Multiple aphthae and erosions with polycyclic outlines against the background of the hyperemic mucous membrane of the lower lip.

encephalotropic. At first, diffuse hyperemia, swelling of the mucous membrane appear in the oral cavity, and then bubbles (vesicles) containing a transparent exudate appear, single or more often in groups. The rash of vesicles is accompanied by a tingling sensation, burning, in some cases, swelling of the oral mucosa. The number of vesicles varies from 2-3 to several dozen closely spaced to each other. The bubbles quickly open up and in their place are formed round erosion, covered with a grayish-white bloom, very reminiscent of aphthae. Erosions formed from the confluence of many bubbles have irregular, polycyclic outlines (Fig. 11.11). Vesicles are localized mainly on the hard palate, the back of the tongue, gums, cheeks, lips. The red border of the lips and adjacent skin is often affected. Keratoconjunctivitis, vesicular rashes on the face, vulvovaginitis are somewhat less common. The process is accompanied by profuse salivation and severe pain. Due to the fact that the elements of the lesion can appear within several days, when examining the oral cavity, you can observe them at different stages of development. Characterized by diffuse damage to the gums - acute catarrhal gingivitis, more pronounced from the palate and tongue.

If oral care is inadequate, catarrhal gingivitis can develop into ulcerative gingivitis. Gingivitis in acute herpetic lesions persists for some time after the disappearance of clinical signs of the disease and epithelialization of erosions. 508

Rice. 11.12. Giant multinucleated cells (1) for recurrent herpes. Cytogram. Micrograph, x 500.

Regional lymphadenitis, which appears even in the prodromal period, persists throughout the disease and even after the epithelialization of erosions for another 5-10 days.

Nonspecific changes characteristic of an acute inflammatory process are found in the blood. The reaction of saliva at the beginning of the disease is acidic, then a shift to the alkaline side (pH 5.8-6.4) is determined. The content of lysozyme is reduced, interferon is absent. Histological changes in the mucous membrane of the mouth and skin in herpes simplex are characterized by the presence of intraepithelial vesicles formed as a result of ballooning and lenticular degeneration of the cells of the thorny layer, the phenomenon of acantholysis. In the degenerated epithelial cells, inclusions containing DNA are found. In the underlying mucosa itself, there is an edema of the papillary layer, expansion of lymphatic and blood vessels, and an infiltrate of neutrophils and lymphocytes.

To confirm the diagnosis of acute herpetic stomatitis, a cytological method is used. The study is carried out in the first 2-3 days of the disease, after the appearance of bubbles. The material is scraped off the erosion area or the contents of the bubbles. When staining the preparation according to

Romanovsky-Giemsa reveals giant multinucleated cells. They are distinguished by sharp polymorphism in shape, color and size (30-120 microns in diameter). The center of the cell is occupied by a dense conglomerate of nuclei (from 2-3 to several dozen). The nucleoli (nucleoli) are not detected (Fig. 11.12).

Cytological examination also determines polymorphonuclear neutrophils at various stages of necrobiosis, layers of epithelial cells with polymorphism and in the form of syncytia, macrophages.

In the first days of the disease with acute herpetic stomatitis, as well as during the period of relapse, the herpes virus can be isolated from the contents of the vesicles. Antibodies to the herpes virus are not detected at the onset of the disease, but as it develops, the antibody titer increases.

For the diagnosis of acute herpetic stomatitis, methods of immunofluorescence, skin tests with a specific antigen, and serological reactions are used.

Differential diagnosis. It is carried out with other viral lesions (herpangina, foot and mouth disease,

vesicular stomatitis); ^ exudative erythema multiforme; And allergic lesions.

The difference from herpangina lies primarily in the localization of the lesion - with herpangina, this is the area of the oropharynx, as a result of which myalgia and dysphagia are often observed. The final diagnosis is confirmed by the data of the virological study.

Differential diagnosis with foot and mouth disease is carried out taking into account the epidemiological situation and typical skin lesions in this disease. Objective confirmation of the presence of foot and mouth disease are the results of laboratory diagnostic methods: biological samples, serological and virological studies.

In differential diagnosis with vesicular stomatitis, the results of virological research methods are decisive in the diagnosis.

Erythema multiforme, in contrast to herpes simplex, is characterized by the presence of seasonality of exacerbations (spring and autumn). There is also a variety of lesion elements: large subepithelial blisters, erythema on the skin and oral mucosa, extensive erosive surfaces. Differential diagnosis is confirmed by the results of allergy tests and virological studies.

Allergic bullosa-erosive lesions differ from herpes simplex by history and data of allergic studies.

Treatment. Acute herpetic stomatitis, like a number of other infectious diseases, can heal spontaneously. It lasts on average 2-3 weeks. Treatment tactics are determined by the severity and the period of development of the disease. Treatment for acute herpetic stomatitis includes the use of antiviral drugs from the first days of the disease and symptomatic therapy to alleviate the patient's condition.

General therapy. In severe cases of the disease, antiviral drugs are used from the first days: Bonafton, 0.1 g, 3-5 times a day, in cycles of 5 days, with breaks of 1-2 days; acyclovir 0.2 g 5 times a day for 5 days, use for this purpose deoxyribonuclease (DNase), which is injected intramuscularly at 10-25 mg, after dissolving the powder in distilled water or isotonic sodium chloride solution. Injections are done every other day; for a course of 6-10 injections.

Desensitizing and anti-inflammatory therapy is carried out. What are antihistamines used for (suprastin, tavegil, fenkarol, etc.); sodium salicylate or acetylsalicylic acid

(0.25 g 3 times a day, after meals), calcium gluconate (0.5 g 3 times a day). To increase the body's resistance, vitamins are prescribed, especially C and P, multivitamins. In a hospital setting, lysozyme is used for this purpose (150 mg 2-3 times a day for 7 days), prodigiosan is injected intramuscularly, 25-50 μg per day with an interval of 4-7 days; the course of treatment is 3-6 injections. In case of complications by fusospirochetosis, metronidazole is administered orally. According to indications, cardiovascular agents are used. Prescribe high-calorie fortified food, drinking plenty of fluids.

Local therapy. In the first days or even hours of the disease, the use of antiviral drugs is effective: leukocyte interferon, a solution of which is applied to the oral mucosa in the form of applications 6-7 times a day. Antiviral ointments are successfully used: 2% tebrofen, 1-2% florenal, 1-5% helepin, 3% liniment of gossypol. These drugs are applied to the entire surface of the oral mucosa after pretreatment with proteolytic enzymes and antiseptics. Antiviral drugs must be applied to the entire surface of the mucous membrane, and not just to the

affected areas, since they have, in addition to therapeutic, and prophylactic effect.

Anesthesia of the oral mucosa precedes medication or food intake. It is carried out with 1% trimecaine solution, 1-2% pyromecaine solution, 1-2% lidocaine solution, aerosols "Lidestin", "Xylostesin", 5-10% solution of anesthesin in peach oil, etc.

It is advisable to use 0.2% deoxyribonuclease solution or 1% lysoamidase solution, since these enzymes have proteolytic and antiviral effects.

For antiseptic treatment of the oral cavity, warm solutions are used in the form of rinsing or oral baths: 0.25-0.5% hydrogen peroxide solution, 0.25% chloramine solution, furacilin solution (1: 5000), 0.5% etonium solution, 0 , 1% gossypol solution, etc. In the first days of the disease, it is recommended to treat the oral cavity every 3-4 hours.

To stimulate the processes of regeneration of the oral mucosa, applications of oil solutions of vitamins A, E, carotolin, rosehip oil, solcoseryl ointment and jelly, 0.5-1% Actovegin ointment and jelly, aerosols "Livian", "Spedian", "Hyposol" are used.

The use of physiotherapeutic treatment is effective from the first days of the disease to complete epithelialization: KUV irradiation, helium-neon, infrared laser therapy.

The elements of the lesion on the skin are treated with solutions of aniline dyes, including fucarcinum, antiviral ointments.

Forecast. In acute herpetic stomatitis it is very favorable, recovery occurs in 1-3 weeks, erosion epithelializes without scarring, the gum retains its shape. In the unsanded oral cavity and with untimely treatment due to the addition of fusospirochetosis, ulcerative-necrotizing Vincent's gingivostomatitis may develop.

Prevention. Since acute herpetic stomatitis is a contagious disease, it is necessary to isolate the patient. In children's institutions, exclude contact of healthy children with patients with acute herpetic stomatitis, regardless of the severity of its course and localization.

CHRONIC RAPID HERPES (herpes chronicus recidivans) is the most common form of endogenous herpes simplex infection.

Rice. 11.13. Chronic recurrent herpes. Groups of vesicles on the hyperemic red border of the lips.

It appears at any age in people who have previously been infected with the herpes simplex virus and have antiviral antibodies. In the pathogenesis of recurrent herpes, the main role belongs to factors that reduce cellular immunity, such as hypothermia, viral infections, pneumonia, chronic stress, severe general diseases (leukemia, cancer, etc.). In women, exacerbation of chronic herpes is often associated with the menstrual cycle. Local factors contributing to the occurrence of an exacerbation include trauma, increased insolation, drying of the red border of the lips. Relapses can occur with different frequencies, at different times of the year and do not have a seasonality. In some patients, relapses occur 1-2 times a year; in others 3-4 times a month - the disease becomes permanent.

The clinical picture. Recurrence of chronic herpes infection is characterized by rashes of single or groups of vesicles 1-2 mm in diameter on the hyperemic mucosa. The process usually begins with a burning sensation, itching, and sometimes mild soreness at the site of future rashes. Then slight hyperemia and swelling of the mucous membrane appear, against the background of which small grouped vesicles appear (Fig. 11.13). They open very quickly, as a result of which

There are bright red painful erosions of irregular shape with finely festooned outlines due to the merging of bubbles and separate small erosions around. Erosions are then covered with a white-yellow fibrinous bloom, on the red border of the lips and skin - hemorrhagic crusts. Healing occurs on the 8-10th day without scarring. Most often, bubbles appear on the red border of the lips in the area of the border with the skin (herpes labialis), the anterior parts of the hard palate, the back of the tongue, cheeks, wings of the nose (herpes nasalis), less often on the skin of the buttocks, in the region of the sacrum, on the thighs. Recurrent herpes on the oral mucosa is localized mainly in places that are normally keratinized. With repeated localization of herpes in the same place, they speak of fixed herpes. Sometimes the appearance of bubbles is accompanied by an increase in the submandibular and sublingual lymph nodes.

Differential diagnosis. Chronic recurrent herpes must be differentiated from: A recurrent aphthous stomatitis; ^ allergic stomatitis; And streptococcal impetigo.

Differential diagnosis is based on the peculiarities of the clinical picture of chronic recurrent herpes, as well as on the data of cytological studies of the contents of the vesicles and scrapings from the area of erosion formed after the opening of the vesicles in the first 2-3 days of the disease.

The preparations contain giant multinucleated cells. A virological research method is also used.

Treatment. It is aimed at increasing the level of specific and nonspecific protection of the patient's body so that he can suppress the persistent herpes simplex virus.

To achieve a significant lengthening of the periods of remission and relief of clinical manifestations of subsequent relapses, you can use levamisole (decaris) orally, 150 mg 2 times a week for 1-3 months, depending on the effectiveness of treatment.

It is necessary to conduct a thorough examination of patients with chronic recurrent herpes in order to identify and eliminate foci of chronic infection c. the body, including in the oral cavity (periodontitis, periodontitis, tonsillitis, sinusitis, etc.). Eliminated local

factors contributing to the occurrence of relapses (chronic trauma, dry lips, chronic lip cracks).

Good results are obtained with the use of a specific herpes polio vaccine. The drug is administered in the inter-relapse period at a dose of 0.1-0.2 mg intradermally 2 times a week. The course of treatment consists of 5 injections. The interval between the first two courses is 2 weeks, between the next - 3-4 months.

Deoxyribonuclease (DNase), which is administered intramuscularly in a dose of 10-25 mg, has a slightly less therapeutic effect, after dissolving the powder in distilled water or isotonic sodium chloride solution. Injections are done every other day; for a course of 6-10 injections.

In the inter-relapse period, intramuscular injections of gamma globulin are used, 3 ml daily, with an interval of 3-4 days, for a course of 6 injections; the interval between courses is 2 months.

Dibazol has a good interferonogenic effect, which is prescribed at 0.01 g 2 times a day for a month.

Usually large doses of ascorbic acid are prescribed.

Antiviral ointments are used locally from the first days of relapses, leukocyte interferon, a solution of which in the form of applications is applied to the affected area 5-6 times a day.

Effective use of laser therapy (helium-neon, infrared)

Критерии оценки текущего контроля

№	Progress in% and points	Grade	Student knowledge level
1.	96-100	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Shows high activity, creative approach when conducting interactive games. Correctly solves situational problems and test questions. Analyzes independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
2.	91-95	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Analyzes research results independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
3.	86-90	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment

			of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
4.	81-85	Good "4"	The correct answer to questions about the clinic, diagnosis and treatment of TBI, but there are 2-3 inaccuracies, errors. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
5.	76-80	Good "4"	Correct, but incomplete coverage of the issue. The student knows questions about the clinic and diagnosis of TBI, but is not fully versed in determining the tactics of treatment. Correctly solves situational problems, but the rationale for the answer is not complete. Understands the essence of the issue. Knows, speaks confidently
6.	71-75	Good "4"	Correct, but incomplete coverage of the issue. Correctly solves situational problems, but the rationale for the answer is incomplete. Has 2-3 errors. Knows, tells confidently. Has accurate ideas.
7.	66-70	Satisfactorily "3"	Understands the essence of the issue. Correctly solves situational problems, but cannot substantiate the answer. Has accurate ideas on specific issues.
8.	61-65	Satisfactorily "3"	Has an incomplete understanding of the topic. Makes mistakes when solving situational tasks. Tells not confidently.
9.	55-60	Satisfactorily "3"	Knows, tells not confidently. Has a partial view.
10.	54 and below	Unsatisfactory "2"	Has no exact idea. Does not know.

7- Practical lesson

Topic: Manifestation of AIDS in the oral cavity.

Technological map of the practical lesson

Stages of work	Teacher	Student
1. Stages of preparation (10 minutes)	1. Controls the purity of the audience; 2. Checks students' readiness for classes; 3. Controls attendance;	Listen
2.The introductory stage to classes (15 minutes)	1.Preparation on the topic of content; 2. Preparation of slides for the introductory report; 3. To develop the literature used for the study of the subject;	Write down the topic and listen
3. The main stage (195 minutes)	1. Dividing students into 2 small subgroups, asking questions on the topic; 2. Use of slides and multimedia; 3. Carries out medical work; 4. Combines all information on a given topic, actively the participating students are encouraged and appreciated;	Divided into small groups watching, participating, listening. The student expresses his opinion, complements and asks questions
4. Final stage (50 minutes)	1. Conclusion. 2. Independent work. 3. Homework.	Listen Write down

Questions on the topic

The source of HIV infection.

The clinical picture of candidiasis in HIV infection.

Clinic for Caponia's sarcoma.

Ulcerative necrotizing stomatitis in HIV infection.

5. Clinic of periodontitis in HIV infection

Tests

HIV infection

001. Ways of transmission of human immunodeficiency virus
1) transplacental
2) parenteral
3) airborne
4) sexual

002. Clinical manifestations of HIV infection
1) opportunistic infections
2) collagenosis
3) oncology

003. Marker diseases accompanying HIV infection in the oral cavity
1) herpes
2) candidiasis
3) pemphigoid
4) Hodgkin's lymphoma
5) non-Hodgkin's lymphoma
6) Kaposi's sarcoma

004. Vaccination against HIV infection
1) it is mandatory

2) carried out at will
3) not carried out

005. Acquired immunity to HIV
1) workable, resistant
2) generated, unstable
3) not produced

006. When working with HIV-infected, the dentist uses
1) high-speed turbine
2) mechanical drill
3) ultrasonic scaler
4) manual scaler

007. Ways to mask
4) protective shield
5) rubber dam
6) headwear

008. Disease HIV infection is caused by
1) RNA virus
2) yeast-like mushrooms
3) fusobacteria

009. The incubation period of HIV disease is
1) 10 to 14 days
2) from 3 to 12 months
3) from 2 to 3 years

010. The lesions of the oral cavity in people infected with HIV are manifested
1) always
2) in some cases
3) never

Interactive method

USE OF THE "DARK LANDSCAPE" METHOD:
USING THE WEB METHOD

Steps: Pre-students are given time to prepare questions for the lesson.
Participants sit in a circle.
One of the participants is given a skein of thread, and he asks his prepared question (to which he himself must know the full answer), holding the end of the thread and throwing the skein to any student.
The student who received the skein answers the question (at the same time, the person who asked him comments on the answer) and passes the question relay on. Participants continue to ask and answer questions until everyone is on the web.
As soon as all students finish asking questions, the student holding the skein returns it to the participant from whom he received the question, while asking his own question, etc., until the coil is completely "unwound"

Text

HIV infection in the oral cavity

The human immunodeficiency virus (HIV) is the causative agent of an infectious disease called HIV infection and manifested by the acquired immunodeficiency

syndrome (AIDS), which is expressed in a decrease in the general resistance of patients to opportunistic microorganisms and an increased tendency to cancer.

Human Immunodeficiency Virus (HIV) belongs to a group of retroviruses, so named because they possess an enzyme, reverse transcriptase, that allows information to be transmitted from RNA to DNA. HIV infects T4 lymphocytes and causes them to die. As a result, the system of cellular immunity is disrupted, and the organism affected by the virus loses its resistance to opportunistic microflora, becomes susceptible to a number of nonspecific infections and tumors, i.e. AIDS develops.

The source of infection is an HIV-infected person, or a virus carrier. HIV-infected persons include everyone (regardless of their clinical condition) infected with the virus, as evidenced by positive serological tests: immunosorbent assay (ELISA), confirmed by immunoblotting (weight-

turn blot), an immunofluorescence reaction or radioimmunoassay and virus isolation.

After HIV infection, 20% of infected people develop AIDS during the first 5 years, and approximately 50% within 10 years.

In people infected with HIV, the pathogen is found in various biological fluids (blood, semen, vaginal secretions, breast milk, saliva, lacrimal fluid, sweat, etc.). However, infection is transmitted only through blood, semen, vaginal secretions and breast milk.

There are 3 known transmission routes of HIV infection: sexual, parenteral and perinatal.

Most often, HIV is transmitted sexually. The infection can be passed from an infected person to their sexual partner. Parenteral infections are transmitted through transfusions of infected blood or blood products, or through the use of needles, syringes, dental or other instruments to pierce the skin or oral mucosa if these items are contaminated with blood. Transmission of HIV from a woman to a fetus or breastfeeding baby can occur before, during, or shortly after birth (through breast milk).

Despite the fact that HIV was also isolated from other biological fluids (saliva, lacrimal fluid, etc.), there is currently no data indicating other routes of infection transmission (respiratory, contact, food, etc.).

Persons infected with HIV are infected for life. Most of them, at least for several years, may not develop any symptoms or signs of illness, and therefore, they will not be aware of the infection. During this time, they are the source of transmission of the virus to other people.

The clinical signs and symptoms of HIV infection are many and varied. The course of HIV infection can be divided into 4 stages, although not all of them are necessarily observed in every infected person.

The acute period of HIV infection can develop as early as one or several weeks after infection, when there is still no seropositive response to HIV, which is usually noted 6-12 weeks after infection. Typical clinical manifestations of this period are fever, lymphadenopathy, night sweats, skin rashes, headaches, and cough.

In / stage, the disease is either asymptomatic,

or persistent generalized lymphadenopathy (PGL) develops, characterized by an increase in lymph nodes in diameter by more than 1 cm in two or more places outside

the groin. This state lasts for about 3 months. As the disease progresses, persistent generalized lymphadenopathy slowly reverses.

Stage II of the disease is referred to as the AIDS-related complex (AIDS-related complex). This complex is characterized by the following signs and symptoms: weight loss, diarrhea, malaise, fatigue, drowsiness, loss of appetite, night sweats, neurological changes leading to memory loss, and peripheral neuropathies. These symptoms and signs often appear intermittently. This stage is also characterized by the presence of typical lesions of the skin and oral mucosa, such as hairy leukoplakia, shingles, exacerbation of chronic recurrent herpes on the oral mucosa and genitals.

Stage III is called the "intermediate stage of the disease." Clinical manifestations in stage III often occur some time after the onset of early symptoms, but before the full expanded picture of AIDS. Candidosis, hairy leukoplakia, acute herpetic stomatitis or exacerbation of chronic herpes appear in the oral cavity at this stage of the disease. Pulmonary tuberculosis, alveolar pneumonia may develop.

At the IV stage of the disease (late stage of the disease), the severe clinical picture of AIDS fully unfolds. It is characterized by the development of inflammatory processes caused by opportunistic microflora and the formation of tumors such as Kaposi's sarcoma, the appearance of which is associated with a deficiency of cellular immunity caused by HIV. The most typical AIDS infections include protozoal invasions (pneumocystis pneumonia, toxoplasmosis), as well as fungal (systemic candidiasis, cryptococcosis), bacterial (atypical mycobacteriosis, salmonella septicemia) or viral (cytomegaly, papovavirus, etc.). At this stage, especially severe forms of lymphoma, cachexia and neurological disorders (neuropathy, memory loss, dementia) may also appear.

The first manifestations - "signs" indicating the introduction of HIV, which has a destructive effect on the immunological protection in the body, may be lesions of the oral mucosa.

Diseases of the oral mucosa associated with HIV infection include: A. Various clinical forms of candidiasis. A Viral infections. A Hairy (villous) leukoplakia. A. Necrotizing ulcerative gingivostomatitis. A Progressive form of periodontitis (HIV-periodontitis). And Kaposi's Sarcoma.

Diseases that should alert the dentist regarding HIV infection of the patient include squamous cell carcinoma of the oral mucosa, lymphoma (non-Hodgkin's).

Candidiasis. It is the most common intraoral sign of HIV infection. The most characteristic is acute pseudomembranous candidiasis. In HIV-infected people, it lasts for a long time (months), so the term "acute" loses its meaning. Clinical manifestations are fully consistent with the usual candidiasis. On the mucous membrane of the mouth there is a grayish-white coating, reminiscent of curdled masses. The plaque is easily removed with a spatula; under it, a hyperemic mucous membrane of the mouth is found. It is localized mainly on the mucous membrane of the cheeks, tongue, floor of the mouth, palate, gums. Candidiasis should be distinguished visually from similar changes in leukoplakia, lichen planus.

Acute atrophic candidiasis resembles lesions of the oral mucosa with allergies, hypovitaminosis C, B, B2, B6. The defeat is usually localized in the tongue in the form of an area of hyperemia located along its midline; filiform papillae are atrophied.

Less common is chronic hyperplastic candidiasis, which is usually localized on both sides on the mucous membrane of the cheeks, hard and soft palate. When the lesion is localized in the corners of the mouth, candidal angular cheilitis and cracks with pronounced manifestations of epithelial hyperplasia often occur. Cracks grow in size over time, become hard, and crust over. Painful sensations in patients may be absent or minor painful sensations, a burning sensation are possible.

The differential diagnosis of candidiasis should take into account the symptoms of similar diseases: A leukoplakia; And lichen planus; And allergic stomatitis; And traumatic injuries.

Diagnosis of candidiasis is based on typical clinical signs and the results of a bacterioscopic examination of scrapings from the affected oral mucosa. The presence of multiple fungus of the genus Candida in the form of spores and hyphae always confirms the diagnosis of candidiasis.

Viral infections. HIV-infected patients are characterized by lesions of the oral mucosa with herpes simplex, which occurs in the form of frequent and painful exacerbations of recurrent herpetic stomatitis. Along with intraoral lesions of the genital organs are often noted. The lesions are atypical in localization, long-lasting and painful. Vesicles appear on the tongue, soft palate, floor of the mouth, on the lips, very quickly transform into erosion, which often turn into ulcers of large sizes (0.5-3 cm in diameter). Ulcers can take the form of a crater with raised irregularly shaped edges and a bright hyperemic bottom, which can be covered with a grayish-white coating. In the absence of timely treatment, these lesions progress, their size increases, which can subsequently lead to dissemination of the viral herpes infection into the visceral organs and increase the severity of the clinical course, and sometimes even cause death.

The mucous membrane of the mouth of HIV-infected patients is also quite often affected by herpes zoster and viral warts (papillomas). They appear as nodular lesions covered with multiple papillary projections. Localized most often on the mucous membrane of the gums, hard palate.

Hairy leukoplakia. Hairy leukoplakia (villous leukoplakia, flat candidoma, oral viral leukoplakia) is observed in HIV-seropositive patients and is a lesion associated with immunodeficiency and HIV infection. The presence of hairy leukoplakia is an indicator for HIV testing. According to the latest literature data, 98% of the examined patients with hairy leukoplakia were HIV seropositive.

Manifestations of hairy leukoplakia can vary in size and localization, they are one- or two-sided. Clinically, hairy leukoplakia appears as white folds or villi that rise above the surface of the oral mucosa, resembling hair in shape. These villi are tightly fixed on

surfaces of the mucous membrane and it is impossible to scrape them off. Hairy leukoplakia is localized more often on the lateral surfaces of the tongue, but can spread to its entire posterior surface; less often it can manifest itself on the mucous membrane of the lips, cheeks, floor of the mouth and palate.

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The fight against HIV infection involves:

precautions for blood and other body fluids;

precautions for injections and procedures involving skin piercing;

effective sterilization and disinfection.

Because most dental procedures are invasive, they often lead to bleeding, and HIV is a blood infection, which puts the dentist at risk for HIV infection. Therefore, dentists, middle and junior 528

medical personnel must comply with precautions with regard to blood and other body fluids (pus, saliva). Hands and other body parts contaminated with blood or other body fluids should be thoroughly washed with soap and water. After removing protective gloves, hands should also be washed immediately. Gloves should be worn for all direct contact with blood and other body fluids. During procedures in which blood splashes or suspensions may form (for example, dispersion of aerosols from high-speed dental units and ultrasound machines) - eyes, nose and mouth should be protected with a mask and glasses or a plastic shield. Extreme care should be taken when handling needles, scalpels, discs, endodontic and other sharp instruments to prevent punctures, cuts and other injuries. Disposable instruments must be destroyed after use. If injury from sharp instruments is possible (for example, when cleaning them), it is recommended to wear additional heavy gloves and handle the instruments with extreme care. It is advisable to use special ultrasonic installations for mechanical cleaning of small instruments from contamination. Physicians with exudative skin lesions should not perform invasive procedures or treat infected patients.

Reliable sterilization is an important condition for the prevention of HIV infection by means of instruments used for piercing the skin, mucous membranes and other invasive procedures. HIV is very sensitive to conventional high-intensity sterilization and disinfection methods. It is inactivated by methods designed to suppress other pathogens (eg, hepatitis B virus). The most effective method of inactivating HIV is high temperature, therefore one of the most reliable methods for sterilizing instruments (including dental ones) is high-intensity sterilization and disinfection based on heating (boiling, autoclaving, sterilization with dry hot air).

High-intensity disinfection can also be carried out with the help of disinfectants: 2% glutaral (glutaraldehyde) solution, 30% hydrogen peroxide solution. Soaking the cleaned instruments in these solutions for 30 minutes provides a high disinfection rate.

For treatment of medical tables, drills, handpieces and other surrounding surfaces, it is enough to rinse them with a solution of a disinfectant of medium or low intensity, such as chlorine-releasing compounds, such as sodium hypochloride, calcium hypochloride, 4% chloramine solution, sodium dichloroisocyanurate, 4% chlorhexidine solution. The disinfecting ability of these chlorine-releasing compounds is measured by the presence of "active" chlorine in them (in percent). For example, sodium hypochloride contains 5% "active" chlorine, calcium hypochloride - 70%, chloramine - 25%.

For the treatment of body tissues, bactericidal agents are used - antiseptics (70% ethyl alcohol, 70% isopropyl alcohol, 1% iodine solution, 10% iodoform solution).

Disinfection of dental materials is carried out by cleaning them from saliva and blood (impression material, material for determining the bite, etc.). Impressions and intraoral orthodontic and orthopedic structures should be cleaned and disinfected before being sent to the laboratory.

After use, the handpieces for drills are rinsed with a strong jet of water, detergent to remove adhering material. Then they are wiped with a material soaked in a disinfecting detergent. The chemical is washed off with distilled water.

Evaluation Criteria for Monitoring

№	Progress in% and points	Grade	Student knowledge level
1.	96-100	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Shows high activity, creative approach when conducting interactive games. Correctly solves situational problems and test questions. Analyzes independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
2.	91-95	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Analyzes research results independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
3.	86-90	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
4.	81-85	Good "4"	The correct answer to questions about the clinic, diagnosis and treatment of TBI, but there are 2-3 inaccuracies, errors. Thinks creatively. Solves situational tasks correctly. Understands the essence

			of the issue. Knows, tells confidently. Has an accurate idea.
5.	76-80	Good "4"	Correct, but incomplete coverage of the issue. The student knows questions about the clinic and diagnosis of TBI, but is not fully versed in determining the tactics of treatment. Correctly solves situational problems, but the rationale for the answer is not complete. Understands the essence of the issue. Knows, speaks confidently
6.	71-75	Good "4"	Correct, but incomplete coverage of the issue. Correctly solves situational problems, but the rationale for the answer is incomplete. Has 2-3 errors. Knows, tells confidently. Has accurate ideas.
7.	66-70	Satisfactorily "3"	Understands the essence of the issue. Correctly solves situational problems, but cannot substantiate the answer. Has accurate ideas on specific issues.
8.	61-65	Satisfactorily "3"	Has an incomplete understanding of the topic. Makes mistakes when solving situational tasks. Tells not confidently.
9.	55-60	Satisfactorily "3"	Knows, tells not confidently. Has a partial view.
10.	54 and below	Unsatisfactory "2"	Has no exact idea. Does not know.

8- Practical lesson
Topic: Vincent's ulcerative necrotizing stomatitis. Manifestation in the oral cavity, clinic, diagnosis and treatment

Technological map of the practical lesson

Stages of work	Teacher	Student
1. Stages of preparation (10 minutes)	1. Controls the purity of the audience; 2. Checks students' readiness for classes; 3. Controls attendance;	Listen
2.The introductory stage to classes (15 minutes)	1.Preparation on the topic of content; 2. Preparation of slides for the introductory report; 3. To develop the literature used for the study of the subject;	Write down the topic and listen
3. The main stage	1. Dividing students into 2 small	Divided into

(195 minutes)	subgroups, asking questions on the topic; 2. Use of slides and multimedia; 3. Carries out medical work; 4. Combines all information on a given topic, actively the participating students are encouraged and appreciated;	small groups watching, participating, listening. The student expresses his opinion, complements and asks questions
4. Final stage (50 minutes)	1. Conclusion. 2. Independent work. 3. Homework.	Listen Write down

Questions on the topic

Etiology of Wenson's ulcerative necrotizing stomatitis.
Localization of Wenson's necrotizing stomatitis.
Patient research methods.

Tests

1.Necrotizing ulcerative stomatitis of Vincent belongs to
1) viral diseases
2) allergic diseases
3) infectious diseases

Vincent's ulcerative necrotizing stomatitis is a disease caused by symbiosis
1) staphylococci and streptococci
2) fusobacteria and spirochetes
3) streptococci and fusobacteria
4) streptococci and lactobacilli

Common factors contributing to the occurrence
ulcerative necrotizing stomatitis
1) vitamin deficiency
2) lipid metabolism disorders
3) hypothermia
4) stress

Elements of defeat in necrotizing ulcerative stomatitis of Vincent
1) stain
2) afta
3) plaque
4) erosion
5) ulcer

... Additional examination methods for necrotic ulcers
stomatitis of Vincent
1) general clinical blood test
2) blood test for glucose content
3) blood test for HIV infection
4) bacterioscopic examination

Pathological processes in the area of necrotic ulcers
oral mucosa
1) necrosis
2) akatolysis
3) inflammatory infiltration
4) vacuolar dystrophy
5) acanthosis
6) proliferation

Differential diagnosis of necrotizing ulcerative stomatitis
Vincent is being held
1) with acute herpetic stomatitis

2) with acute pseudomembranous candidiasis
3) with HIV infection
4) with acute leukemia
5) with diabetes
6) with syphilis
7) with leukoplakia

Vincent's angina is differentiated
1) with diphtheria
2) with syphilis
3) with agrolyslocytosis
4) with candidiasis

Local factors provoking a relapse of necrotizing
stomatitis of Vincent
1) chronic inflammatory diseases of periodontal tissues
2) unsanded oral cavity
3) language anomalies
4) difficult eruption of a wisdom tooth
5) galvalose
6) hyposalivation
7) poor quality dentures

Vincent's topical remedies for necrotizing ulcerative stomatitis
1) solution 0.05% chlorhexine
2) Metrogyl Denta gel
3) 5% butadione ointment
4) solution of 1% hydrogen peroxide
5) trypsin, chymopsin
6) 20% sodium borate solution in glycerin
7) indomethacin ointment

In the acute stage of necrotizing ulcerative stomatitis, Vincent is not indicated
1) the use of pain medications
2) tooth extraction
3) the use of cauterizing agents
4) the use of keratoplasty
5) carrying out professional oral hygiene

For the general treatment of Vincent's ulcerative necrotizing stomatitis
apply
1) multivitamins
2) antifungal drugs
3) desensitizing drugs
4) antibiotics
5) hormonal drugs
6) sedatives
7) antimicrobial drugs

Prevention of relapses of necrotizing ulcerative stomatitis by Vincent includes
1) vaccination
2) professional oral hygiene
3) desensitization
4) immunization
5) sanitation of the oral cavity

Specify the correct sequence

Stages of treatment of ulcers with necrotizing ulcerative stomatitis of Vincent
1) application with proteolytic enzymes
2) pain relief
3) removal of necrotic masses
4) antiseptic treatment
5) application of antimicrobial agents

Interactive Method
USING THE DARK HORSE METHOD:
USING THE WEB METHOD

Steps:
In advance, students are given time to prepare questions for the lesson they have completed.

Participants sit in a circle.
One of the participants is given a skein of thread, and he asks his prepared question (to which he himself must know the full answer), holding the end of the thread and throwing the skein to any student.
The student who received the skein answers the question (at the same time, the person who asked him comments on the answer) and passes the question relay on. Participants continue to ask and answer questions until everyone is on the web.
As soon as all students finish asking questions, the student holding the skein returns it to the participant from whom he received the question, while asking his own question, etc., until the coil is completely "unwound"

Text

Necrotizing ulcerative stomatitis of Vincent

Necrotizing stomatitis of Vincent (stomatitis ulceronecroticans Vincenti) is an infectious disease caused by fusiform sticks (Bacillus fusiformis) and Borellia vincentii.

Described under various names: ulcerative gingivitis and ulcerative stomatitis, ulcerative membranous stomatitis, fusospirochetous stomatitis, Plaut-Vincent stomatitis, "trench mouth", ulcerative membranous stomatitis, etc. According to the modern classification, the disease is called "ulcerative necrotizing stomatitis of Vincent" , or "Vincent's stomatitis". In the case of gum disease, the disease is defined as Vincent's gingivitis; with simultaneous damage to the gums and other areas of the mucous membrane - Vincent's stomatitis, and with localization of the process in the area of the palatine tonsils - Simanovsky-Plaut-Vincent's angina.

Etiology. Vincent's ulcerative necrotizing stomatitis is caused by the symbiosis of the fusiform bacillus and Vincent's spirochete. Under normal conditions, these microorganisms are representatives of the resident microflora of the oral cavity and are detected in small numbers in all people with teeth. They are found mainly in the gingival groove, periodontal pockets, carious cavities, crypts of the tonsils. In the unsanded oral cavity, with its poor hygienic condition, as well as with periodontitis, the number of fusobacteria and spirochetes increases sharply. The development of Vincent's ulcerative necrotizing stomatitis is associated with a sharp decrease in the body's resistance to infection, which can occur as a result of past viral diseases (acute respiratory infections, herpetic stomatitis, pneumonia, etc.), vitamin deficiencies, stress, overwork, malnutrition. Necrotizing ulcerative gingivitis often complicates the course of severe general diseases (leukemia, agranulocytosis, pneumonia, infectious mononucleosis). It can occur as a complication of erythema multiforme exudative, erosive allergic stomatitis. In case of violation of specific and nonspecific defense mechanisms in the body, the virulence of fusobacteria and spirochetes increases. Their number increases to such an extent that they become dominant in comparison with other microflora. A decrease in the general resistance of the body negatively affects the resistance of the oral mucosa - it cannot play the role of a reliable barrier on the path of infection, and the violation of its integrity, which always occurs in the unsanded oral cavity, due to the presence of local traumatic factors (sharp edges of the teeth, dentures, tartar deposits, etc.) creates conditions for the introduction of fusobacteria and spirochetes. Therefore, Vincent's ulcerative necrotizing stomatitis often develops in people with an unsanitary oral cavity and poor hygiene.

Despite the fact that there are known cases of group morbidity with Vincent's stomatitis (in military units, schools, kindergartens), this disease is considered non-contagious, and such cases are explained by similar unfavorable living conditions (malnutrition, lack of vitamins, lack of hygienic measures for caring for the oral cavity and etc.).

The clinical picture. Mostly young people (17-30 years old), mostly men, are ill.

Hypothermia is often a provoking factor in the development of Vincent's ulcerative necrotizing stomatitis, which explains the highest frequency of its occurrence in the autumn-spring period; the maximum incidence occurs in the period from October to December.

Clinically distinguish between acute and chronic course of necrotizing ulcerative stomatitis of Vincent, and in terms of severity - its mild, moderate and severe form.

The disease begins acutely with an increase in body temperature to 37.5-38. Regional lymph nodes increase, thicken, become painful on palpation, retain mobility. Patients are worried about headache, general weakness, soreness of the oral mucosa, aggravated by eating, talking, bleeding gums, hypersalivation, putrid breath.

Catarrhal phenomena on the oral mucosa quickly turn into ulcerative. The process most often begins on the gums, and then moves on to other parts of the mucous membrane. The gums become swollen, hyperemic, sharply painful and bleed when touched. The epithelium of the gum edge and interdental papillae becomes cloudy and then necrotic. As a result, the gingival margin looks like a cut, with uneven notches, its surface is covered with an easily removable grayish-yellow coating. Subsequently, the affected gum edge is not completely restored and remains deformed. Most often and to a greater extent, the area near the lower eighth teeth is affected, where necrosis from the alveolar process quickly spreads to the mucous membrane of the cheek and the retromolar region, causing in some cases trismus and pain when swallowing. Ulcers on the mucous membrane of the cheeks can reach large sizes (up to 5-6 cm in diameter) and depth (Fig. 11.15). Their edges are uneven, soft. The bottom is covered with a thick grayish-greenish necrotic plaque with a putrid fetid odor, after removal of which the bleeding bottom of the ulcer is exposed. There are no seals in the area of the base and edges of the ulcer. The mucous membrane around the ulcer is edematous, hyperemic. In the presence of local traumatic factors in the oral cavity (roots of decayed teeth, sharp edges of teeth, prostheses), other areas of the oral mucosa (hard, soft palate, arches, tonsils, tongue) may be affected. Oral ulcers can be single or multiple, of various sizes and depths. When the ulcer is localized on the hard palate, necrosis of all layers develops rather quickly.

Rice. 11.15. Vincent's acute necrotizing stomatitis. An extensive ulcer of the buccal mucosa along the line of closing of the teeth in the area of the large molars, covered with necrotic plaque.

mucous membrane and bone is exposed. An isolated lesion of the pharynx (angina Simanovsky-Plaut-Vincent), as a rule, is unilateral, in the practice of a dentist is rare. 2-3 weeks after the onset of necrotizing ulcerative stomatitis usually occurs resolution of the process with complete epithelization of ulcerative surfaces.

In rare cases, when treatment is not carried out or it is ineffective, a chronic form of necrotizing ulcerative stomatitis of Vincent develops, in which there are no general symptoms. Patients are worried about constant bleeding and soreness of the gums, as

well as bad breath. The clinical picture is erased, the gum is congestive-hyperemic, edematous, its ulcerated edge is often compacted, necrotic areas are located mainly in the interdental spaces and can be seen at a cursory examination. On careful examination and probing of the gingival margin, exposed bone tissue is determined. Only some teeth have affected areas. Lymph nodes (submandibular, submental) in patients with chronic ulcerative necrotizing stomatitis of Vincent are compacted, slightly painful. With a disease duration of 4-8 months, the lymph nodes acquire a cartilage-like consistency.

Rice. 11.16. The histological picture of Vincent's ulcerative necrotizing stomatitis. A site of tissue necrosis in the surface layers (1), maturing granulation tissue (2), edema and small cell infiltration (3).

In the pathological examination of the areas of the ulcerated edges of the gums, two zones are revealed: superficial - necrotic and deep - inflammatory (Fig. 11.16). In the surface layers of the necrotic gum tissue, an abundant variety of microflora (cocci, sticks, fusobacteria, spirochetes, etc.) is revealed. In deeper layers, fusobacteria and spirochetes predominate sharply (Fig. 11.17). The deeper layers of connective tissue are inflamed: they are edematous, the vessels are dilated. Blood corpuscles are found in the perivascular inflammatory infiltrate. In the same zone of inflammation, only spirochetes that have penetrated between the epithelial cells are found inside the intact tissue.

The cytological picture of scrapings from the ulcerative surfaces of the mucous membrane in patients with necrotizing ulcerative stomatitis of Vincent corresponds to a nonspecific inflammatory process. At the beginning of the disease, an abundance of structureless masses is determined, a sharp predominance of neutrophils, mainly in a state of decay, and erythrocytes (due to severe bleeding). In the second period of the disease, when healing begins, along with disintegrated neutrophils, full-534

Rice. 11.17. Acute ulcerative necrotizing stomatitis. Fusospirochetosis. Scraping from the ulcer. Fusobacteria and spirochetes (1), neutrophils in the stage of deep decay (2). Cytogram. x 700.

phagocytic cells, many macrophages. During the period of epithelialization that has begun, layers of young epithelial cells appear, the number of fusobacteria and spirochetes decreases.

In the chronic course of Vincent's ulcerative necrotizing stomatitis, the relative amount of fusobacteria and spirochetes decreases and the number of cocci increases, but fusospirochetes still prevail.

The diagnosis of ulcerative necrotizing stomatitis of Vincent is made on the basis of a characteristic clinical picture and the detection of an abundance of fusobacteria and spirochetes in the scraping from the surface of ulcers.

Differential diagnosis. Vincent's ulcerative necrotizing stomatitis must be differentiated primarily from ulcerative lesions in blood diseases (leukemia, agranulocytosis, infectious mononucleosis). Along with clinical differences (pallor of the oral mucosa, the presence of hemorrhages, leukemic infiltrates, severe and prolonged bleeding of the gums in blood diseases), the detected changes in peripheral

blood in leukemia, agranulocytosis, and infectious mononucleosis are of decisive importance in this case.

Wensan's ulcerative-necrotizing stomatitis is differentiated from allergic stomatitis on the basis of anamnesis data, peculiarities of clinical manifestations and results of bacterioscopic examination.

An ulcerative-necrotic process in the oral cavity, similar to Vincent's stomatitis, can occur with mercury intoxication. If contact with mercury is detected, urine is analyzed for mercury content.

It should be remembered that ulcerative necrotic lesions of the oral mucosa can complicate the course of specific infections (syphilis, HIV infection) or malignant tumors (cancer, sarcoma). To avoid diagnostic errors in these cases, you should carefully collect anamnesis, take into account only local, but also general clinical manifestations of the disease and conduct the necessary laboratory tests (clinical blood test, Wasserman serological tests or to identify HIV infection, cytological, bacterioscopic, pathohistological and etc.).

Treatment. The scope of treatment for necrotizing ulcerative stomatitis of Vincent is determined mainly by the severity of its course. Since the disease occurs more often in young people, most of them practically healthy, local treatment is of decisive importance. The earlier and more thoroughly the treatment of the oral cavity is carried out, the faster the patient's condition improves. High-quality treatment of the oral mucosa, removal of traumatic factors are decisive in the further course and outcome of the disease.

Local treatment consists in removing local traumatic factors, necrotic tissues, influencing the microflora and stimulating the processes of regeneration of the oral mucosa. Treatment of the oral cavity should begin with anesthesia. For this purpose, application and injection anesthesia with solutions of pyromecaine, trimecaine, lidocaine is used. After that, all mechanical irritants are removed: the sharp edges of the teeth and prostheses are grinded, tartar and plaque are removed. Carious cavities should be treated with antiseptic solutions. The removal of decayed teeth should be postponed until the epithelialization of ulcers, since the extraction of teeth in the infected oral cavity of a patient with ulcerative necrotizing stomatitis is fraught with serious complications (alveolitis, periostitis, abscess, phlegmon). Cleansing ulcerative surfaces from necrotic tissues is carried out using proteolytic enzymes: trypsin, chymotrypsin, lysoamidase, deoxyribonuclease.

The entire oral cavity is treated with warm antiseptic solutions (0.5% hydrogen peroxide solution, 1% chloramine solution, 0.06% chlorhexidine solution, 0.5% etonium solution, potassium permanganate solution 1: 5000, etc.). Gingival pockets, ulcer surfaces, interdental spaces and sub-hood spaces are best rinsed with a pressure jet. On the first visit, the entire oral cavity should be treated. In the future, daily processing is carried out. At home, the patient is prescribed warm antiseptic rinses: 3% hydrogen peroxide solution (2 tablespoons per glass of water), 0.25% chloramine solution, potassium permanganate solution (1: 5000), 0.06% chlorhexidine solution or other antiseptic drugs every 3-4 hours

With a mild course of necrotizing ulcerative stomatitis of Vincent, local treatment is sufficient. In more severe cases, general therapy is necessary. For the purpose of antimicrobial action, metronidazole (trichopolum, flagil, clion) is prescribed inside

0.25 g 2 times a day for 7-10 days. Metronidazole is also used for local treatment (liquid aqueous gruel is applied to ulcerative surfaces after removing necrotic tissue). In severe cases, broad-spectrum antibiotics are sometimes used (bicillin-3, 300,000 IU once every 3 days or 600,000 IU once every 6 days; erythromycin, oletetrin, oxytetracycline in a daily dose of 800,000-1,000,000 IU per within 5-10 days). For any severity, it is recommended to prescribe B vitamins, askorutin (0.1 g orally 2 times a day, for 10-14 days).

With proper treatment, the improvement of the condition of patients with necrotizing ulcerative stomatitis of Vincent occurs within 24-48 hours, the pain decreases or disappears, the patients can eat and sleep. Edema and hyperemia of the oral mucosa decrease, epithelialization of ulcers begins, which, with a mild degree of the disease and a satisfactory state of the oral cavity, ends by the 3rd-6th day. In the unsanded oral cavity, epithelialization of ulcerative surfaces proceeds more slowly. After the improvement of the general condition of the patient and the disappearance of acute inflammatory phenomena, it is necessary to carry out a thorough sanitation of the oral cavity with the removal of tartar, tooth roots, to treat carious teeth, periodontal diseases.

Recurrences of necrotizing stomatitis can occur if there are foci of chronic

infections (periodontal pockets, hoods over the incompletely erupted third large molars), or traumatic factors (overhanging fillings, carious cavities, roots of decayed teeth, tartar, low-quality prostheses, etc.). Poor oral hygiene may be the cause of relapse.

Treatment of symptomatic ulcerative necrotizing stomatitis in blood diseases, allergic conditions, mercury intoxication consists mainly in carrying out general treatment of the underlying disease causing these changes.

Forecast. With timely and correct treatment, the prognosis is favorable. Epithelialization of ulcerative surfaces in an acute process occurs after 3-6 days, in a chronic one - somewhat slower. In the unsanded oral cavity, in the presence of many traumatic factors and untimely or incorrect treatment, omission (retraction) or deformation of the gums, resorption of the bone tissue of the alveolar process may occur. These changes contribute to the further progression of periodontitis.

Patients who have suffered Vincent's stomatitis are subject to active observation throughout the year. The first examination is carried out after 1-2 months, the subsequent ones - after 6 months.

Prevention. It consists in observing oral hygiene, regular sanitation, complete and timely treatment of infectious and other diseases that lead to a decrease in immunity.

Evaluation Criteria for Monitoring

№	Progress in% and points	Grade	Student knowledge level
1.	96-100	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Shows high

			activity, creative approach when conducting interactive games. Correctly solves situational problems and test questions. Analyzes independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
2.	91-95	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Analyzes research results independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
3.	86-90	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
4.	81-85	Good "4"	The correct answer to questions about the clinic, diagnosis and treatment of TBI, but there are 2-3 inaccuracies, errors. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
5.	76-80	Good "4"	Correct, but incomplete coverage of the issue. The student knows questions about the clinic and diagnosis of TBI, but is not fully versed in determining the tactics of treatment. Correctly solves situational problems, but the rationale for the answer is not complete. Understands the essence of the issue. Knows, speaks confidently
6.	71-75	Good "4"	Correct, but incomplete coverage of the issue. Correctly solves situational problems, but the rationale for the answer is incomplete. Has 2-3 errors. Knows, tells confidently. Has accurate ideas.
7.	66-70	Satisfactorily "3"	Understands the essence of the issue. Correctly solves situational problems, but cannot substantiate the answer. Has

			accurate ideas on specific issues.
8.	61-65	Satisfactorily "3"	Has an incomplete understanding of the topic. Makes mistakes when solving situational tasks. Tells not confidently.
9.	55-60	Satisfactorily "3"	Knows, tells not confidently. Has a partial view.
10.	54 and below	Unsatisfactory "2"	Has no exact idea. Does not know.

9- Practical lesson
Topic: Changing the OAS in tuberculosis.

Technological map of the practical lesson

Stages of work	Teacher	Student
1. Stages of preparation (10 minutes)	1. Controls the purity of the audience; 2. Checks students' readiness for classes; 3. Controls attendance;	Listen
2.The introductory stage to classes (15 minutes)	1.Preparation on the topic of content; 2. Preparation of slides for the introductory report; 3. To develop the literature used for the study of the subject;	Write down the topic and listen
3. The main stage (195 minutes)	1. Dividing students into 2 small subgroups, asking questions on the topic; 2. Use of slides and multimedia; 3. Carries out medical work; 4. Combines all information on a given topic, actively the participating students are encouraged and appreciated;	Divided into small groups watching, participating, listening. The student expresses his opinion, complements and asks questions
4. Final stage (50 minutes)	1. Conclusion. 2. Independent work. 3. Homework.	Listen Write down

Questions on the topic
What is Tuberculosis.
What group does Tuberculosis belong to?
The source of infection is Tuberculosis.
Clinical picture of Tuberculosis
Dif - diagnosis and treatment of Tuberculosis

Tests

The causative agent of mycobacterium tuberculosis is.
A) Koch's wand
B) Streptococci
C) Staphylococci
D) Polyinfection

Ways of spreading infection in primary tuberculosis.
A) hematogenous
B) odontogenic
C) lymphogenous
D) answers a and c

On the mucous membrane of the mouth and the red border of the lips, a secondary tuberculosis infection manifests itself in the form.
A) tuberculous lupus
B) miliary ulcerative tuberculosis
C) answers a and b
D) there is no correct answer

Mantoux reaction in most cases with lupus tuberculosis.
A) Negative
B) Neutral
B) Positive
D) all answers are correct

The course of tuberculous lupus erythematosus is mainly.
A) Chronic
B) Acute
C) Subacute
D) all answers are correct

Miliary ulcerative tuberculosis - translate into Latin.
A) tuberculosis miliares cerosa
B) tuberculosis miliares ulcerosa
B) tuberculosis ulcerosa
D) tuberculosis miliares

What areas are most often affected by miliary ulcerative tuberculosis?
A) The mucous membrane of the cheeks, the line of closing of the teeth, the tongue
B) red border of lips
C) hard palate
D) all answers are correct

In miliary-ulcerative tuberculosis, ulcers usually form.
A) 1-4 ulcers
B) 2-4 ulcers
B) 1-3 ulcers
D) 10-15 ulcers

The diameter of the ulcer (with miliary ulcerative tuberculosis).
A) 10-15 cm
B) 2-3 cm
B) 1-3cm
D) 1-3 mm

10. With miliary ulcerative tuberculosis ESR.
A) Rising
B) Decreases
C) Does not change

Interactive method
USING THE WEB METHOD
Steps:

1. In advance, students are given time to prepare questions for the lesson.
2. Participants sit in a circle.

3. One of the participants is given a skein of thread, and he asks his prepared question (to which he himself must know the full answer), holding the end of the thread and throwing the skein to any student.
4. The student, who received the skein, answers the question (at the same time, the person who asked him comments on the answer) and passes the baton on the question further. Participants continue to ask and answer questions until everyone is on the web.
5. As soon as all students finish asking questions, the student holding the skein returns it to the participant from whom he received the question, at the same time asking his own question, etc., until the ball is completely "unwound".

Text

Tuberculosis is a chronic infectious disease caused by Mycobacterium tuberculosis (Koch's bacillus).

Tuberculous lesion of the mucous membrane of the mouth and the red border of the lips is usually secondary and occurs when there is a tuberculous process in the lungs, lymph nodes or bones. Mycobacterium tuberculosis gets to the mucous membrane of the mouth most often by hematogenous or lymphogenous route from the primary tuberculous focus. The mucous membrane of the mouth, due to its pronounced resistance, is an unfavorable environment for the reproduction of mycobacterium tuberculosis. As a rule, they quickly die on its surface. Primary tuberculosis in the oral cavity of adults practically does not occur, it is extremely rare in young children.

On the mucous membrane of the mouth and the red border of the lips, the second

Typical tuberculosis infection usually manifests itself in the form of tuberculous lupus and miliary ulcerative tuberculosis. Scrofuloderma (colonic tuberculosis) is extremely rare.

Lupus erythematosus (lupus vulgaris, tuberculous lupus). This is the most common disease among tuberculous lesions of the oral mucosa and the red border of the lips. In tuberculous lupus erythematosus, lesions of the oral mucosa are usually combined with skin lesions. The favorite localization of tuberculous lupus is the skin of the face, which is affected in about 75% of patients. Very often, the red border of the upper lip is affected as a result of the spread of the process from the skin of the nose. Subsequently, the mucous membrane of the mouth is often involved in the process. Sometimes there is an isolated lesion of only the red border of the upper lip. Selective lesion only of the oral mucosa is rare. The lupus process can affect any part of the oral mucosa or the red border of the lips, but its most typical localization is on the mucous membrane of the upper lip, the alveolar process of the upper jaw in the region of the frontal teeth, hard and soft palate. The primary element of this form of tuberculosis is a tubercle or lupoma, which is a limited, initially flat, painless formation the size of a pinhead (diameter 1-3 mm), soft texture, red or yellowish-red in color.

The tubercles are prone to peripheral growth and fusion with neighboring elements, which leads to the formation of plaques of different sizes and outlines, in which individual tubercles differ. The center of the plaque breaks down rather easily, turning into an ulcer with soft, pitted edges. The ulcer is superficial, slightly painful, the bottom is covered with a yellowish or yellowish-red bloom, in the presence of growing granulations - resembling raspberries. Characteristic of tuberculous lupus are the apple jelly symptom and the probe test. When pressed on any microscope slide, the red color

due to perifocal vasodilation disappears, and the primary elements of the lupa become visible - yellowish-brown or waxy nodules resembling apple jelly in color (a symptom of apple jelly). If you press on anybody with a bulbous probe, then it easily falls through (Pospelov's symptom), which is mainly due to the destruction of elastic and collagen fibers in the tubercle.

When the process is localized on the mucous membrane of the alveolar process, the bone tissue of the interdental septa is destroyed, the teeth become mobile and fall out. On the red border of the lips, ulcers are covered with bloody-purulent crusts. In this case, the affected lip swells greatly, increases in size, and painful cracks appear on it. After the resolution of the process, a scar remains. If the process proceeded without ulceration, a smooth atrophic scar is formed. In places of ulceration, the scars are dense, rough, solder the mucous membrane with the underlying tissues. Recurrent formation of lupus on the scar is characteristic. Localization of the focus of lupus on the lip subsequently leads to its deformation, as a result of which it becomes difficult to eat, speech is distorted. If both lips are affected, a microstomy may develop.

The course of tuberculous lupus erythematosus is chronic. Without treatment, the process can continue indefinitely; fresh tubercles appear on the scars. Regional lymph nodes enlarge and thicken. Lupus tuberculosis can be complicated by erysipelas, candidiasis. Malignancy of lupus ulcers on the oral mucosa occurs in 1-10% of cases (lupus carcinoma).

Diagnostics. In typical cases, the diagnosis of tuberculous lupus is not difficult. The Mantoux reaction is in most cases positive. In the discharge from ulcers, as a rule, Mycobacterium tuberculosis is not found.

Histologically, in the reticular layer of connective tissue, a tubercle is determined, consisting of giant Pirogov-Langhans cells, plasma, epithelioid cells surrounded by a shaft of lymphocytes. Caseous necrosis is absent or mild. Elastic and collagen fibers in the specific infiltrate area are destroyed. In the area of infiltration and around it, there are a large number of dilated blood vessels, including newly formed ones.

Differential diagnosis. Tuberculous lupus erythematosus is differentiated from the tubercles that occur with tertiary syphilis. Syphilitic tubercles are larger than with tuberculous lupus, they are more dense. The edges of the ulcers in syphilis are even, dense, and in tuberculous lupus, they are soft, corroded. Unlike lupus, syphilitic eruptions do not recur on scars. There is no symptom of a failing probe and apple jelly for syphilis.

Rice. 11.21. Tuberculous ulcer. Ulcer with uneven, edematous edges.

MILIAR-ULCER TUBERCULOSIS (tuberculosis miliaris ulcerosa). Miliary ulcerative tuberculosis of the oral mucosa is rare. It occurs in patients suffering from severe forms of pulmonary or laryngeal tuberculosis, accompanied by the release of a large number of tuberculosis mycobacteria with sputum. Due to a decrease in the body's resistance in such patients, the causative agent of tuberculosis is easily introduced into the mucous membrane and multiplies unhindered, causing ulcerative processes. Mainly affected are permanently injured areas of the mucous membrane of the cheeks, along the line of closing of the teeth, the back and lateral surfaces of the tongue, the soft palate, the bottom of the oral cavity. Usually 1-3 ulcers form. At the site of the introduction of mycobacterium tuberculosis, tubercles the size of a pinhead of a grayish-red color appear, which very quickly turn into microabscesses and open

up with the formation of small, very painful, superficial ulcers. Gradually increasing along the periphery, the ulcer often merges with similar, adjacent ulcers, sometimes reaching 2-3 cm in diameter (Fig. 11.21). Such ulcers are usually shallow and irregular in shape; undermined, pitted edges, sharply painful. The bottom of the ulcer is covered with a yellowish-gray bloom with a slight purulent discharge, bleeds easily, has a granular appearance due to unresolved tubercles. At the bottom and around the ulcers, small yellowish spots (small abscesses) are found - the so-called Treel grain. Inflammation around the ulcers is insignificant. Regional lymph nodes may not be palpable at first, later they become enlarged, indurated, painful. With the prolonged existence of an ulcer and its secondary infection, compaction of the edges and bottom can be observed. On the tongue or along the transitional fold, ulcers can take the form of deep, painful cracks. With the localization of ulcers on the gums, in addition to damage to soft tissues, destruction may occur

Rice. 11.22. Scraping from a tuberculous ulcer.

1 - neutrophilic leukocytes in the stage of deep decay; 2 - lymphocytes;

3 - giant Pirogov-Langhansa cage. Micrograph, x 500.

periodontal and bone, which can lead to mobility and loss of teeth.

The general condition of patients suffers sharply: emaciation, increased sweating, shortness of breath, increased body temperature, hypersalivation are observed. There are changes in the leukocyte formula, increased ESR. In scrapings from ulcers during cytological examination, giant cells of Pirogov-Langhansa (Fig. 11.22) and epithelioid cells are revealed. At a bacterioscopic examination, Koch's bacilli are found (staining according to Ziehl-Nielsen).

Differential diagnosis. It is performed with a gummy ulcer, which, in contrast to miliary-ulcerative tuberculosis, is painless, has a crater-like shape with dense ridge-like edges.

Tuberculous ulcer should also be differentiated from necrotizing ulcerative stomatitis of Vincent, traumatic, trophic and cancerous ulcers. The results of cytological and bacterioscopic studies are decisive in the diagnosis.

Treatment. Since tuberculous lesion of the oral mucosa is a manifestation of general tuberculosis infection, the general treatment of patients is carried out in specialized anti-tuberculosis dispensaries. Local

treatment is aimed at eliminating traumatic factors, treatment of hard tissues of teeth and periodontium. Treatment of the oral mucosa with antiseptic and anesthetic drugs in the form of oral baths and applications is also used.

Evaluation Criteria for Monitoring

№	Progress in% and points	Grade	Student knowledge level
1.	96-100	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Shows high activity, creative approach when

			conducting interactive games. Correctly solves situational problems and test questions. Analyzes independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
2.	91-95	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Analyzes research results independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
3.	86-90	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
4.	81-85	Good "4"	The correct answer to questions about the clinic, diagnosis and treatment of TBI, but there are 2-3 inaccuracies, errors. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
5.	76-80	Good "4"	Correct, but incomplete coverage of the issue. The student knows questions about the clinic and diagnosis of TBI, but is not fully versed in determining the tactics of treatment. Correctly solves situational problems, but the rationale for the answer is not complete. Understands the essence of the issue. Knows, speaks confidently
6.	71-75	Good "4"	Correct, but incomplete coverage of the issue. Correctly solves situational problems, but the rationale for the answer is incomplete. Has 2-3 errors. Knows, tells confidently. Has accurate ideas.
7.	66-70	Satisfactorily "3"	Understands the essence of the issue. Correctly solves situational problems, but cannot substantiate the answer. Has accurate ideas on specific issues.

8.	61-65	Satisfactorily "3"	Has an incomplete understanding of the topic. Makes mistakes when solving situational tasks. Tells not confidently.
9.	55-60	Satisfactorily "3"	Knows, tells not confidently. Has a partial view.
10.	54 and below	Unsatisfactory "2"	Has no exact idea. Does not know.

10- Practical lesson

Topic: Manifestation of skin and venereal diseases in the OAS. Syphilis

Technological map of the practical lesson

Stages of work	Teacher	Student
1. Stages of preparation (10 minutes)	1. Controls the purity of the audience; 2. Checks students' readiness for classes; 3. Controls attendance;	Listen
2.The introductory stage to classes (15 minutes)	1.Preparation on the topic of content; 2. Preparation of slides for the introductory report; 3. To develop the literature used for the study of the subject;	Write down the topic and listen
3. The main stage (195 minutes)	1. Dividing students into 2 small subgroups, asking questions on the topic; 2. Use of slides and multimedia; 3. Carries out medical work; 4. Combines all information on a given topic, actively the participating students are encouraged and appreciated;	Divided into small groups watching, participating, listening. The student expresses his opinion, complements and asks questions
4. Final stage (50 minutes)	1. Conclusion. 2. Independent work. 3. Homework.	Listen Write down

Questions on the topic
What group of diseases does syphilis include?
Sources of syphilis infection.
The clinical picture of syphilis.
Dif - diagnostics.
Treatment and prevention

Tests

The causative agent of chickenpox
1) varicella zoster virus
2) Leffler's wand
3) Coxsackie virus
4) herpes simplex virus
5) Epstein-Barr virus

Elements of lesion on the oral mucosa
with chickenpox
1) are grouped into a local focus
2) are located separately from each other
3) are located along the nerve trunk

The elements of the lesion in chickenpox are called
1) crust
2) papule
3) erosion
4) bubble
5) flake

Oral changes with measles
1) preceded by skin rashes
2) appear simultaneously with a skin rash
3) do not appear
A symptom pathognomonic for measles
1) crimson tongue
2) Belsky-Filatov-Koplik spots
3) polymorphic rash
Belsky-Filatov-Koplik spots are
1) papules
2) erosion
3) aphthae
4) vesicles

With scarlet fever, a typical symptom of the disease
1) pallor of the oral mucosa
2) crimson tongue
3) folded tongue

The characteristic appearance of the tongue with scarlet fever is due to
1) hyperplasia of the epithelium
2) desquamation of the epithelium
3) necrotic plaque

Changes in the oral cavity with scarlet fever appear during the period
1) incubation
2) prodromal
3) the height of the disease

Interactive method
USING THE HANDLE IN THE MIDDLE TABLE METHOD

All students of the group are divided by lot into 3 subgroups of 3 students each. Each subgroup sits down at a separate table, prepares a blank sheet of paper and a pen. The date, group number, last name and first name of the student are written on the sheet. The task is offered, to answer one question for the whole subgroup. Each student writes down his last name and one answer option on a sheet and passes the sheet to a neighbor, and moves his pen to the middle of the table. The teacher supervises the work of the group and everyone's participation in it. The general correct version is written down in a notebook. Students who gave the correct answer options receive the maximum score - 100% of the theoretical part rating - 0.8b. Second place students - 85.9% of the rating. Third place - 70.9% of the rating. Those who did not answer or answered incorrectly 30% of the rating. The resulting score is taken into account when assigning marks for the current lesson. The works of students are saved by the teacher.

Text

Syphilis (syphilis, lues) is a chronic infectious disease caused by Treponema pallida. The mucous rim of the mouth is the site of localization of syphilitic rashes in all periods of syphilis.

Distinguish between congenital and acquired syphilis. With congenital syphilis, pale treponema enters the fetus through the placenta from the sick mother. Acquired syphilis develops through infection through the skin and mucous membranes by direct contact (mainly sexual) or through various objects contaminated with secretions containing the pathogen. The source of infection can be medical instruments, including and dental, in case of violation of the regime of their sterilization. A prerequisite for infection with syphilis is even the smallest damage to the stratum corneum or epithelium of the oral mucosa (cracks, abrasions, etc.), although some authors believe that the pathogen can also penetrate through the intact mucous membrane. Infection with syphilis can also occur when pale treponema gets into the blood directly, for example, during blood transfusions or other medical procedures (injection, during operations, etc.). Medical workers (more often gynecologists and dentists) can become infected when treating patients, conducting gynecological and dental examinations, operations, opening abscesses, periodontal interventions, as a result of careless handling of endodontic and other instruments. The most dangerous source of infection is patients with active manifestations of syphilis in the primary and secondary periods with localization of lesions on the genitals and oral mucosa.

In connection with the undulating course of syphilis, characterized by a change in periods of active manifestations and latent stages of the disease, a variety of clinical

and morphological signs that arise at different stages of the disease, four periods are distinguished: incubation, primary, secondary and tertiary.

The incubation period for syphilis lasts on average 3-4 weeks.

PRIMARY SYPHILIS (syphilis primaria Lues I) lasts an average of 6-7 weeks, sometimes 2-3 months without treatment. It is characterized by the appearance at the site of the introduction of pale treponema of a painless inflammatory infiltrate - a hard chancre (primary syphiloma). 5-7 days after the formation of a hard chancre, the second mandatory symptom of the primary period appears - an increase in the lymph nodes closest to the hard chancre, or regional scleradenitis. The lymph nodes are cartilaginous-dense, up to 2-3 cm in diameter, painless, not adhered to the skin and to each other, the skin above them is not changed. By the end of the primary period, the lymph nodes of all groups are enlarged (polyadenitis). In these nodes, an intensive reproduction of treponema occurs. Hard chancre can be single or in the amount of 2-3. Most often it is located on the red border of the lips, the mucous membrane of the tongue, palatine tonsils, less often on the mucous membrane

Rice. 11.18. Chancre. Saucer-shaped ulcer of the tongue with raised, even edges.

cheeks, soft and hard palate, or other areas. The development of a hard chancre on the oral mucosa in typical cases begins with limited hyperemia and induration due to an inflammatory infiltrate that increases in size and usually reaches 1–2 cm in diameter. Then its central part necrotizes and forms a meat-red, painless erosion or superficial saucer-shaped ulcer of a round or oval shape with raised smooth edges from 3 mm to 1.5 cm in diameter (Fig. 11.18). At the base of the erosion or ulcer, a dense cartilage-like infiltrate is palpable. The bottom of the ulcer is even, shiny, meat-red in color without plaque, or covered with a grayish-yellow "greasy" coating.

Often there are atypical forms of hard chancre that differ from the classic described above. In this case, a hard chancre can look like a crack, abrasion, erosion, seizure with a slight infiltration at the base. Its form depends on the localization. In the tongue, a hard chancre due to a pronounced inflammatory infiltrate at the base protrudes sharply above the level of the surrounding tissues and has the shape of a saucer turned upside down. In persons with a folded tongue, the chancre is localized along the folds and has a slit-like shape.

When a hard chancre is located in the corners of the mouth, it clinically resembles seizures with an infiltration at the base.

On the gum, a hard chancre has the appearance of a bright red erosion, which adjoins several teeth in the form of a crescent.

When localized on the amygdala, a hard chancre can have one of three forms: ulcerative, angina-like (amygdalitis) and combined (ulcerative against a background of angina-like). The amygdala is affected on one side only. It acquires a copper-red color, is enlarged, dense,

painless. With an ulcerative form, a meat-red ulcer appears on this background, oval in shape with gentle, even edges. The mucous membrane around the ulcer is hyperemic. The process can be accompanied by painful sensations, sometimes significant.

In an unsanded oral cavity with poor hygiene, a hard chancre can be complicated by fusospirochetosis, staphylococcal infection. Due to the development of an acute inflammatory process, the typical clinical symptoms of syphilis are obscured, soreness

appears, at the bottom of the ulcer there is a purulent or necrotic plaque, the depth of the ulcer increases.

Diagnosis. The diagnosis of hard chancre should be confirmed by the detection of pale treponema from the surface of the primary syphiloma or in the punctate of regional lymph nodes. If there is a negative result, repeated studies of the hard chancre that is separated from the surface should be carried out for the presence of pale treponema, since its detection is decisive in the diagnosis of primary syphilis. Specific serological reactions (Wasserman reaction, sedimentary reactions) become positive a little later, only 3 weeks after the appearance of a hard chancre. Starting from the 4th week, classical serological reactions (complement fixation reaction, Wasserman reaction, Kahn reaction, Sachs-Vitebsky reaction) and the immunofluorescence reaction become persistently positive. In this regard, the primary period of syphilis is divided into primary seronegative, when the indicated serological reactions are negative, and primary seropositive, which occurs on average 3-4 weeks after the appearance of a solid chancre, when serological reactions become positive and remain so for a long time.

By the end of the primary period, patients may experience general (prodromal) phenomena: malaise, bone pain, fever, headache, increased ESR, leukocytosis.

Differential diagnosis. Primary chancre should be differentiated from traumatic ulcers, in which there is no compaction at the base characteristic of syphilis. In addition, the traumatic ulcer is quickly eliminated after the cause is eliminated.

Chancre, localized on the amygdala, differs from angina in one-sided lesion, absence of pain, acute inflammatory hyperemia and general phenomena.

When the chancre is localized on the lip, differential diagnosis with erosion that occurs with recurrent herpes should be carried out. In the case of herpes, unlike syphilis, erosion is located on a slightly edematous, hyperemic base, is painful and has scalloped outlines. Herpetic erosion is also characterized by a rapid course (rapid onset and rapid epithelialization). In addition, with recurrent herpes, erosion is preceded by the formation of bubbles, which never occurs with syphilis.

Chancre may resemble chancriform pyoderma, which is characterized by the presence of inflammation, purulent discharge from the surface, pain and duration of existence.

Differential diagnosis of hard chancre and severe recurrent aphthous stomatitis (Setton's aft) is carried out. The differences lie in the sharp soreness of the ulcer or aphthae with recurrent aphthous stomatitis, a pronounced inflammatory reaction of the mucous membrane, as well as anamnesis data on the recurrent nature of the disease.

Chancre may resemble a decaying cancer. When carrying out differential diagnosis, it should be borne in mind that the infiltration of a cancerous tumor is located much deeper than the primary syphiloma. The edges of the cancer ulcer are very dense, uneven, the bottom bleeds easily. Decisive for the diagnosis are the results of laboratory tests - the detection of atypical cells in cancer and treponema pale in syphilis.

SECONDARY SYPHILIS (syphilis secundaria, Lues II) lasts an average of 3-4 years. There is a generalization of the process with damage to the mucous membranes in the form of a characteristic rash (syphilis), disturbances in the general condition of the body (malaise, weakness, subfebrile body temperature, headaches, etc.), possible

damage to internal organs, bone and nervous systems. The mucous membrane of the mouth is one of the favorite places of localization in this stage of syphilis, both secondary fresh and recurrent. Manifestations on the oral mucosa occur simultaneously with skin manifestations, but they can also be isolated. The secondary period of syphilis is characterized by high infectivity, focus of the location of the elements, frequent absence of subjective sensations, true or false polymorphism. At this stage of the disease in the oral cavity, there are mainly two types -

lesions: macular (macular) and papular syphilides.

Spotted syphilis is most often localized on the palatine arches, soft palate and tonsils. It is characterized by the appearance of red, sharply limited spots, which tend to merge into a copper-red erythema, clearly delimited from the unchanged mucous membrane. Without treatment, these changes last for about 1 month.

Erythematous syphilitic sore throat should be distinguished from the banal catarrhal sore throat, in which there is pain, fever, swelling, soreness. Differential diagnosis is also carried out with allergic drug lesions of the oral mucosa, which differ from erythematous syphilitic sore throat in the prevalence of lesions - not only the arches and tonsils are involved, but also the cheeks, tongue and other areas. Medicinal catarrhal stomatitis is accompanied by burning, soreness of the oral mucosa. All symptoms quickly disappear after stopping the intake of the medicinal substances that caused them.

Papular syphilis is the most common form of manifestation of secondary syphilis in the oral cavity, especially its recurrent stage. Papular rashes can form anywhere in the mucous membrane, but most often occur on the tonsils, soft palate and arch, where they often merge into large plaques. Papules are dense, rounded, elements 1 - 1.5 cm in diameter. They are painful, delimited from the unchanged mucous membrane. Due to constant maceration in the oral cavity, the papules acquire a grayish-white color and have a tendency to peripheral proliferation. Merging into plaques, they sometimes rise above the level of the oral mucosa. When scraping with a spatula, the plaque covering the papule is removed and meat-red erosion is exposed under it. There is an inflammatory rim around the papule. Papules and discharge from the erosion surface contain a large number of treponemas.

On the back of the tongue, in places where papules appear, filamentous and mushroom papillae disappear and elements resembling foci of desquamation appear, smooth, shiny, sinking below the level of the mucous membrane ("plaques of a mown meadow") (Fig. 11.19). In an unsanded oral cavity, with poor hygienic care, papules can ulcerate or be complicated by fusospirochetosis. In this case, the clinical picture becomes similar to stomatitis.

Rice. 11.19. Syphilitic papules. Smooth shiny plaques on the tongue due to atrophy of filiform and mushroom papillae.

and Vincent's angina, significant soreness and an area of hyperemia around the papules appear. When papules are localized in the corners of the mouth, due to constant injury, they can grow and spread to the skin. When the mouth is opened, the infiltrated skin and mucous membrane are injured, forming deep, bleeding, painful cracks covered with crusts against the background of a dense infiltrated base.

Syphilitic papules on the oral mucosa should be differentiated from leukoplakia, papules of lichen planus and lupus erythematosus, the surface of which is also grayish-

white. However, if a whitish plaque is quite easily removed from the surface of a syphilitic papule by scraping, then with leukoplakia, lichen planus and lupus erythematosus, it cannot be removed, since it is a consequence of hyperkeratosis. In addition, with leukoplakia, papules with lichen planus and foci of hyperkeratosis with lupus erythematosus, there is no infiltration at the base, and there is no inflammatory rim along the edges of the lesion.

Syphilitic papules differ from candidiasis in that after removing a grayish-white plaque from their surface, erosion is exposed, in the discharge of which pale treponemas are found. Scraping off the same plaque in candidiasis exposes the hyperemic surface of the mucous membrane, sometimes eroded.

In some cases, papular syphilis resembles exudative erythema multiforme and allergic stomatitis. In these diseases, there is no infiltration at the base of erosion, the oral mucosa is hyperemic, diseases are often accompanied by a violation of the general condition and pronounced subjective sensations.

Syphilitic papules on the dorsum of the tongue should be distinguished from desquamative glossitis. The bright red color of the

desquamation stacks, the presence of a white border on the periphery, the absence of compaction at the base, the migration of desquamation foci and the chronic course of the disease distinguish desquamative glossitis from syphilitic lesions.

Sometimes it is necessary to carry out differential diagnosis of syphilitic papules with recurrent aphthous stomatitis. The presence of a yellowish-gray plaque on the surface of aphtha, surrounded by a corolla of hyperemia, severe pain and recurrent nature of the disease make it possible to distinguish aphthae from syphilitic papules.

Diagnosis. Along with the clinical picture, the diagnosis of the secondary period of syphilis should be confirmed by the detection of pale treponemas in scraping from the surface of the papules and positive serological reactions (Wasserman reaction, sedimentary reactions, the reaction of immobilization of pale treponemas).

TERTIARY SYPHILIS (syphilis tertiaria, Lues III) begins 4-6 years after the onset of the disease. It can last for decades. At this stage, gum and lumpy eruptions may appear on the mucous membrane of the mouth and on the skin.

Gummy syphilide can be localized anywhere in the oral mucosa, but more often on the soft and hard palate, as well as the tongue. Usually a single gum appears (Figure 11.20). In the thickness of the mucous membrane of the mouth, a painless node forms, which gradually increases, reaching large sizes (3 cm or more in diameter). Then the center of the gum is opened, a deep crater-shaped ulcer with a necrotic core in the center arises. The ulcer is painful, surrounded by a dense infiltrate, dense, even edges, the bottom is bright red, covered with small granulations. The healing of the ulcer leads to the formation of a retracted stellate scar. This process lasts 3-4 months and is almost not accompanied by subjective sensations. Less often, gum dissolves or turns into a dense fibrous node. Depending on the localization of the gum, its clinical picture has certain features. Gummy lesions of the tongue can occur either in the form of the formation of individual gums (nodular glossitis), or in the form of diffuse sclerosing glossitis, which is the most severe form of lesions of the oral mucosa in syphilis. Initially, there is a diffuse infiltration of the thickness of the tongue, mainly by plasma cells, as a result of which the tongue increases, and no nodes are formed. Tongue tight, papillae

Rice. 11.20. Syphilitic gumma. Large knot in the thickness of the tongue.

smoothed. Subsequently, the infiltrate is replaced by scar tissue, gradually tightening the tongue, which leads to its reduction. On the back of the tongue, very painful grooves, cracks, trophic ulcers form, the edges of which can become malignant. The tongue becomes very dense, its mobility is limited. There are also combinations of sclerosing glossitis with gum.

When localized on the hard palate, bone tissue and periosteum are very quickly involved in the process, bone necrosis, its sequestration, extensive defects, perforation of the hard palate occur.

Diagnostics. It is rather difficult to detect pale treponema in the discharge of tubercles and gum in the tertiary period of syphilis. Therefore, the immunofluorescence reaction (RIF) and the treponema pallidum immobilization reaction (PALT), which are positive in 100% of cases, are of decisive importance in the diagnosis of tertiary syphilis. Wasserman's reaction and sedimentary reactions in the tertiary period of syphilis are positive in 50-80% of patients.

Differential diagnosis. Syphilitic gummas

should be differentiated from tumors and miliary ulcerative tuberculosis.

Tuberous syphilis on the oral mucosa is less common than gummy. Bumps, as well as gum, can appear anywhere in the oral mucosa, but are more often localized on the lips, alveolar ridge and in the palate. Tuberous syphilis on the oral mucosa is painless, dense tubercles of a bluish-red color. They tend to be grouped, then disintegrate rather quickly, leading to the formation of ulcers. After the ulcers heal, rough scars form. For tubercular syphilis on the oral mucosa, a relatively rapid course is characteristic - usually several months from the onset to the formation of a scar.

Regional lymph nodes in tertiary syphilis may not respond to the process in the oral cavity and give a nonspecific response to the addition of a secondary infection.

The differential diagnosis of tubercular syphilis is carried out with tuberculous lupus, which is characterized by a much slower development, calculated in years; soft consistency lupom. Ulcers that form during the disintegration of tubercles in tuberculous lupus have uneven, soft edges.

Treatment. Treatment of patients with syphilis is carried out in specialized venereal medical institutions.

Evaluation Criteria for Monitoring

№	Progress in% and points	Grade	Student knowledge level
1.	96-100	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Shows high activity, creative approach when conducting interactive games. Correctly solves situational problems and test questions. Analyzes independently.

			Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
2.	91-95	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Analyzes research results independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
3.	86-90	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
4.	81-85	Good "4"	The correct answer to questions about the clinic, diagnosis and treatment of TBI, but there are 2-3 inaccuracies, errors. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
5.	76-80	Good "4"	Correct, but incomplete coverage of the issue. The student knows questions about the clinic and diagnosis of TBI, but is not fully versed in determining the tactics of treatment. Correctly solves situational problems, but the rationale for the answer is not complete. Understands the essence of the issue. Knows, speaks confidently
6.	71-75	Good "4"	Correct, but incomplete coverage of the issue. Correctly solves situational problems, but the rationale for the answer is incomplete. Has 2-3 errors. Knows, tells confidently. Has accurate ideas.
7.	66-70	Satisfactorily "3"	Understands the essence of the issue. Correctly solves situational problems, but cannot substantiate the answer. Has accurate ideas on specific issues.
8.	61-65	Satisfactorily "3"	Has an incomplete understanding of the topic. Makes mistakes when solving situational tasks. Tells not confidently.

9.	55-60	Satisfactorily "3"	Knows, tells not confidently. Has a partial view.
10.	54 and below	Unsatisfactory "2"	Has no exact idea. Does not know.

11- Practical lesson
Topic: Candidiasis. Its manifestation in the oral cavity. Clinic diagnosis and treatment

Technological map of the practical lesson

Stages of work	Teacher	Student
1. Stages of preparation (10 minutes)	1. Controls the purity of the audience; 2. Checks students' readiness for classes; 3. Controls attendance;	Listen
2.The introductory stage to classes (15 minutes)	1.Preparation on the topic of content; 2. Preparation of slides for the introductory report; 3. To develop the literature used for the study of the subject;	Write down the topic and listen
3. The main stage (195 minutes)	1. Dividing students into 2 small subgroups, asking questions on the topic; 2. Use of slides and multimedia; 3. Carries out medical work; 4. Combines all information on a given topic, actively the participating students are encouraged and appreciated;	Divided into small groups watching, participating, listening. The student expresses his opinion, complements and asks questions
4. Final stage (50 minutes)	1. Conclusion. 2. Independent work. 3. Homework.	Listen Write down

Questions on the topic

What causes candidiasis.
What types of candidiasis do you know?
Differential diagnosis of candidiasis.
Tactics Doctor - Dentist
Prevention of candidiasis.

Tests

The cause of candidiasis is
1) poor oral hygiene
2) stress
3) dysbiosis
4) antibiotic allergy
5) hypothermia

The nature of candidiasis
1) allergic
2) viral
3) traumatic
4) bacterial
5) fungal

Element lesion in candidiasis
1) papule
2) erosion
3) plaque
4) bubble
5) tubercle

Factors contributing to the development of candidiasis
1) diabetes mellitus
2) hypovitaminosis B12
3) hypothermia
4) professional harm
5) wearing removable dentures
6) trauma to the oral mucosa
7) long-term use of antibiotics
The causative agent of candidiasis is
1) fusobacteria
2) Candida mushrooms
3) gonococci
4) actinomycetes
5) spirochetes
The diagnosis of candidiasis is confirmed by research
1) allergic
2) bacterioscopic

3) cytological
4) immunographic
Material for bacterioscopic examination with candidiasis is taken
1) before meals
2) on an empty stomach
3) after eating
Keratinous candidiasis
1) accompanied by
2) not accompanied
Optimal environment for the development of the fungus Candida
1) alkaline
2) neutral
3) sour
Plaque for candidiasis contains
1) lactobacilli and cocci
2) neutrophils and desquamated epithelial cells
3) single Candida cells and epithelial cells
4) budding forms of blastospores and yeast pseudomycelium
Course of candidiasis Clinical form
1) acute a) hypertrophic
2) chronic b) atrophic
c) pseudomembranous
Workers may have occupational candidiasis
1) antibiotic production
2) vegetable stores
3) petrochemical production
4) confectionery factories
According to the localization of the lesion by the Candida fungus,
1) stomatitis
2) palatinitis
3) laryngitis
4) cheilitis
5) glossitis
6) sinusitis
7) I'll stop by
... Candidiasis develops against the background of long-term use
1) corticosteroids
2) cytostatics
3) keratoplasty
4) antibiotics
5) multivitamins
6) enzymes
... Acute pseudomembranous candidiasis is differentiated
1) with leukoplakia
2) lichen planus
3) with acute herpetic stomatitis

4) with glossalgia
5) with neuritis of the lingual nerve
6) with HIV infection
7) with desquamative glossitis
... Chronic atrophic candidiasis is differentiated
1) with chronic recurrent herpes
2) lichen planus
3) with plastic allergy
4) with medical stomatitis
5) with desquamative glossitis
6) with stomalgia

Yeast jam is differentiated
1) with tuberculous
2) with syphilitic
3) with streptococcal
4) with vitamin deficiency
5) with eczematous
With bacterioscopic confirmation of candidiasis,
1) observation and re-scraping
2) repeated scraping
3) treatment and re-scraping
Drug therapy for candidiasis is directed
1) for pain relief
2) on the desensitization of the body
3) for epithelialization
4) to inhibit the growth of the fungus Candida
Effective dosage form for yeast jam
1) solution
2) ointment
3) pill Effects of antifungal therapy
1) symptomatic
2) pathogenetic
3) etiotropic
... The goal of candidiasis treatment is
1) desensitize the body
2) restore normal microflora
3) eliminate the focus of infection
4) normalize the function of hematopoiesis
... Antifungal drugs include
1) metronidazole and diazolin
2) gentamicin and nystatin
3) amphotericin B and nystatin
4) levorin and fluconazole (diflucan)

Prophylactic daily dose of fluconazole (diflucan, diflazone)

1) 50-100 mg
2) 5-15 mg
3) 25-50 mg

025. Daily therapeutic dose of fluconazole (diflucan, diflazone) with oral candidiasis
1) 50-100 mg
2) 150-200 mg
3) 200-400 mg
Prophylactic daily dose of nystatin
1) 1-2 million
2) 2-3 million
3) 3-4 million
General remedies for candidiasis
1) multivitamins (groups B, C, PP)
2) histaglobulin
3) nystatin
4) levorin
5) fluconazole (diflucan)
6) orungal
7) nizoral
8) lamisil
9) 3% potassium iodide solution
Local remedies for candidiasis
1) 20% sodium borate solution in glycerin
2) Castellani liquid
3) 0.5% oxolinic ointment
4) Canesten ointment
5) nystatin ointment
6) Solcoseryl ointment
7) amphotericin B ointment
8) alkaline rinses

Restriction in the diet for candidiasis
1) proteins
2) fat
3) mineral salts
4) carbohydrates
5) water

Factors provoking relapse of candidiasis
1) hypothermia
2) a long course of antibiotic therapy for systemic diseases
3) the presence of low-quality removable dentures
4) hypovitaminosis C and B
5) violation of carbohydrate metabolism
6) hyposalivation

7) galvanosis

Acute atrophic candidiasis is differentiated
1) with acute herpetic stomatitis
2) with leukoplakia
3) with B12-deficiency anemia
4) with plastic allergy
5) with stomalgia

Candidal glossitis differentiate
1) with folded tongue
2) with allergic glossitis
3) with glossalgia
4) with Gunther's glossitis
5) with desquamative glossitis
6) with diamond-shaped glossitis

Risk groups for developing candidiasis
1) breastfeeding malnourished children
2) older children
3) men 30-60 years old, heavy smokers
4) elderly people weakened by chronic diseases
5) persons taking long-term cystostatic drugs, corticosteroids
6) persons suffering from epilepsy

Interactive method
USING THE HANDLE IN THE MIDDLE TABLE METHOD

All students of the group are divided by lot into 3 subgroups of 3 students each. Each subgroup sits down at a separate table, prepares a blank sheet of paper and a pen. The date, group number, last name and first name of the student are written on the sheet. The task is offered, to answer one question for the whole subgroup. Each student writes down his last name and one answer option on a sheet and passes the sheet to a neighbor, and moves his pen to the middle of the table. The teacher supervises the work of the group and everyone's participation in it. The general correct version is written down in a notebook. Students who gave the correct answer options receive the maximum score - 100% of the theoretical part rating - 0.8b. Second place students - 85.9% of the rating. Third place - 70.9% of the rating. Those who did not answer or answered incorrectly 30% of the rating. The resulting score is taken into account when assigning marks for the current lesson. The works of students are saved by the teacher.

Text

Candidiasis (candidosis) - a disease that is caused by opportunistic yeast-like fungi of the genus Candida - often Candida albicans, sometimes Candida tropicalis or other species.

Various types of yeast-like fungi live on the mucous membrane of the mouth. Pathological changes are most often caused by Candida albicans, Candida tropicalis, Candida pseudotropicalis, Candida krusei, Candida guilliermondi. The localization of

Candida in the oral cavity is diverse: various parts of the mucous membrane, carious cavities, root canals. Candida albicans is a representative of the resident microflora of the human oral cavity and is found in a small amount and in an inactive state in 50-70% of individuals who do not have clinical signs of candidiasis. It is also present on the surface of healthy skin. Candida albicans is a yeast or budding fungus. It consists of oval budding cells 3-5 μm in size. The fungus loves an "acidic" environment (pH 5.8-6.5) and produces numerous enzymes that break down proteins, lipids and carbohydrates.

Etiology and pathogenesis. The manifestation of the pathogenic properties of fungi of the genus Candida mainly depends on the state of the macroorganism. As shown by the results of recent studies, the weakening of the immune system plays a major role in the development of candidiasis. Candidiasis occurs, as a rule, against the background of a more or less pronounced immunodeficiency or imbalance of the immune system. The development of candidiasis can be promoted by severe concomitant diseases: malignant neoplasms, HIV infection, tuberculosis, diseases of the endocrine system (diabetes mellitus, hypothyroidism, hypoparathyroidism, hypo- and hyperfunction of the adrenal glands). Diseases of the gastrointestinal tract, especially low acidity of gastric juice and achilia, often cause the development of candidiasis of the oral mucosa. Violation of carbohydrate metabolism in diabetes mellitus is a favorable background for the development of candidiasis. Therefore, candidiasis of the oral mucosa often becomes the first clinical sign of an

symptomatic diabetes mellitus. In all cases of chronic candidiasis, especially recurrent ones, it is necessary to conduct a blood test for glucose content to exclude diabetes mellitus.

The development of oral candidiasis is facilitated by long-term treatment with corticosteroid drugs, which suppress the body's immune system and increase the virulence of yeast-like fungi.

Due to the widespread use of antibiotics in recent years, the number of candidiasis of the oral mucosa has increased significantly. With prolonged use of antibiotics, the composition of the resident microflora of the oral cavity is disturbed - dysbiosis is formed. There is a suppression of the resident flora and a sharp increase in the virulence of opportunistic Candida fungi that cause diseases of the oral mucosa (superinfection, autoinfection). A similar effect is exerted by the long-term use of various antimicrobial drugs (trichopolum, chlorhexidine, sanguirithrin, etc.). Along with dysbiosis in the oral cavity, antibiotics also cause dysbiosis in the intestine, which results in hypo- and avitaminosis of groups B, C, PP, which in turn negatively affects the functional state of the oral mucosa (it becomes susceptible to candidal infection).

Great importance in the development of candidiasis is attached to the state of the oral mucosa and its immunity. The emergence of candidiasis is facilitated by acute and chronic injuries of the oral mucosa (sharp edges of the teeth, low-quality prostheses, destroyed crowns of teeth, etc.). The allergenic effect of acrylic plastic prostheses was established during their prolonged contact with the oral mucosa. The decreased resistance of the oral mucosa due to chronic trauma allows Candida to invade easily and cause disease. In addition, Candida grows well on the surface of removable acrylic dentures, supporting chronic inflammation of the mucous membrane under the denture.

Candidiasis of the oral mucosa is more often affected by infants and the elderly, especially weakened by chronic, severe diseases. It is possible for a newborn to become infected with yeast-like fungi from the mother when passing through the birth canal.

According to the classification of N.D.Sheklakov, there are:

superficial candidiasis of mucous membranes, skin and nails;

chronic generalized (granulomatous) candidiasis of children;

visceral (systemic) candidiasis.

The most common forms of candidiasis are superficial. The dentist treats patients with candidiasis of the oral mucosa included in this group. Candidiasis of the oral mucosa can occur in isolation, or with damage to other mucous membranes and skin. Sometimes in the presence of unfavorable factors, primarily cellular immunodeficiency, as well as untimely and insufficient treatment, mucosal candidiasis can transform into generalized with damage to internal organs, the prognosis of which is very serious.

The clinical picture. The clinical manifestations of oral mucosa candidiasis are diverse and depend on age, the state of the immune system, the presence of concomitant diseases, taking medications (antibiotics, corticosteroids) and other factors. According to the clinical course, acute and chronic forms of oral mucosa candidiasis are distinguished. Acute candidiasis can occur in the form of thrush (acute pseudomembranous candidiasis) or acute atrophic candidiasis. Chronic candidiasis also exists in two clinical forms: chronic hyperplastic and chronic atrophic.

Acute pseudomembranous candidiasis, or thrush (candidosis.acuta, s.soor). One of the most common forms of oral candidiasis. In infants, thrush is common and relatively easy. In adults, acute pseudomembranous candidiasis is often accompanied by any general somatic serious diseases: diabetes mellitus, blood diseases, hypovitaminosis, malignant neoplasms, etc.

Most often, the mucous membrane of the back of the tongue, cheeks, palate, lips is affected. The mucous membrane of the mouth is brightly hyperemic and dry. Against the background of hyperemia, there is a white coating, reminiscent of curdled milk or cottage cheese. Plaque rises above the level of the mucous membrane and at the onset of the disease is easily removed by scraping with a spatula, after which a smooth, slightly edematous hyperemic surface is exposed (Fig. 11.23). In heavy running

Rice. 11.23. Acute pseudomembranous candidiasis. White curdled plaque on the hyperemic mucous membrane

In advanced cases, plaque becomes denser and is difficult to remove.

Patients complain of a burning sensation in the mouth, pain when eating, especially acute, the presence of plaque.

Acute pseudomembranous glossitis should be differentiated from desquamative glossitis, in which areas of epithelial desquamation appear on the dorsum of the tongue, surrounded by a corolla of exfoliating epithelium, constantly migrating along the dorsum of the tongue. Acute candidal stomatitis must be differentiated from leukoplakia and lichen planus, in which whitish films and nodules on the surface of the mucous membrane are formed due to hyperkeratosis, and therefore it is impossible to remove them by scraping. Candidiasis also needs to be differentiated from mild leukoplakia, or white spongy nevus, in which the localization of the lesion is

predominantly along the line of closing of the teeth and on the mucous membrane of the lips. The color of the mucous membrane with soft leukoplakia in the affected area is whitish-gray, its surface is rough, uneven, there are multiple small superficial erosions (abrasions). The final diagnosis is made on the basis of bacteriological data.

Acute atrophic candidiasis (candidosis acuta atrophica) is characterized by significant pain, burning and dryness in the mouth. The mucous membrane is fiery red, dry. When localized in the tongue, its back becomes crimson-red, dry, shiny, filiform papillae are atrophied. Plaque is absent or persists in deep folds, is removed with difficulty and is a conglomerate of desquamated epithelium and a large number of Candida fungi in the stage of active budding (mycelium, pseudomycelium).

Acute atrophic candidiasis should be differentiated from an allergic reaction to plastic of removable dentures. An important role in this case is played by clinical observation of the dynamics of changes in the oral mucosa after elimination of the prosthesis and conducting bacterioscopic research.

The general condition of patients with acute candidiasis does not suffer: the body temperature is not changed, the lymph nodes are not palpable.

Chronic hyperplastic candidiasis (candidosis chronica hyperplastica) is characterized by the formation on the hyperemic mucous membrane of the mouth of a thick layer of dense plaque in the form of nodules or plaques, tightly adhered to an uneven surface. Plaque is usually located on the back of the tongue, on the palate. When localized in the tongue, the area typical of rhomboid glossitis is more often affected. Chronic hyperplastic candidiasis in the palate looks like papillary hyperplasia. In cases of long-term persistent disease, the plaque is impregnated with fibrin, yellowish-gray films are formed, tightly adhered to the underlying mucous membrane. When scraping with a spatula, the plaque is removed with difficulty, after which a hyperemic bleeding erosive surface is exposed. Patients complain of dryness in the mouth, burning, and in the presence of erosions - pain. This form of candidiasis should be differentiated from leukoplakia and lichen planus.

Chronic atrophic candidiasis (candidosis chronica atrophica) is also manifested by dryness in the oral cavity, burning sensation, pain when wearing a removable denture. The area of the mucous membrane corresponding to the boundaries of the prosthetic bed is hyperemic, edematous, painful.

Chronic atrophic candidiasis in persons who use removable plate prostheses for a long time is characterized most often by lesions of the oral mucosa

Rice. 11.24. Mycotic jam. Scales and crusts at the corners of the mouth covering cracks.

under prostheses (hyperemia, erosion, papillomatosis) in combination with mycotic (yeast) seizure and candidal atrophic glossitis, in which the back of the tongue is crimson-red, dry, shiny, filiform papillae are atrophic. A small amount of whitish-gray plaque is present only in deep folds and on the lateral surfaces of the tongue. The plaque is removed with difficulty. Under the microscope, spores and mycelium of the fungus of the genus Candida are found in it. This triad (inflammation of the palate, tongue and corners of the mouth) is so characteristic of atrophic candidal stomatitis that its diagnosis is not difficult.

Differential diagnosis is carried out with lichen planus; allergic stomatitis caused by acrylic plastics, various forms of drug stomatitis, syphilitic papules.

Mycotic (yeast) seizure is observed mainly in the elderly with low bite height due to improper prosthetics, pronounced erasure of hard tooth tissues or edentulous (Fig. 11.24). The presence of deep folds in the corners of the mouth and the constant maceration of these areas of the skin with saliva create favorable conditions for the formation of candidal seizure. Patients complain of burning, soreness in the corners of the mouth. The disease is characterized by the appearance in the corners of the mouth of gray, transparent scales, delicate crusts or plaque, easily removable, after removal of which dry and weakly wet erosion or cracks are exposed. The process is more often

It is bilateral and localized within the skin fold. Along with the defeat of the corners of the mouth, the process can go to the mucous membrane of the red border of the lips, as a result, candidal cheilitis develops. It is characterized by hyperemia, edema, the presence of grayish scales and small transverse cracks. When the red border of the lips is stretched, soreness occurs.

Mycotic seizure should be differentiated from streptococcal seizure, which is characterized by abundant exudation, hyperemia extending beyond the skin fold. Slit-like erosion is covered with honey-yellow crusts. Differential diagnostics should also be carried out with a hard chancre and syphilitic papules localized in the corners of the mouth, having a seal at the base. The final diagnosis is made on the basis of the results of microscopic examination of scraping from the affected area, as well as Wasserman's reaction. Candidal seizures are also differentiated from hypo- and avitaminosis B2.

Diagnosis. Diagnosis of candidiasis is based on the typical complaints of patients, the clinical picture and laboratory data (microscopic examination of scraping from the surface of the oral mucosa). CBC and serum glucose tests are also required. The skin and nails are examined. According to indications - consultations of a mycologist, endocrinologist, gynecologist.

Candidiasis is diagnosed based on the detection of a fungus of the genus Candida in a scraping from the surface of the affected oral mucosa. Microscopic examination of scrapings from the surface of the oral mucosa and removable dentures is carried out. The sampling of material for research should be carried out on an empty stomach before brushing teeth and rinsing the mouth, or 4-5 hours after eating or rinsing.

In the oral cavity, the conditionally pathogenic fungus of the genus Candida is present in small quantities in the form of round (young) or elongated (mature) cells. The diameter of single cells ranges from 2 to 5 microns, the diameter of budding cells can reach 12-16 microns. Normally, fungi of the genus Candida in the scraping preparation are found in the form of single yeast-like cells, which does not give the right to diagnose candidiasis. In case of candidiasis in the scraping preparation, an accumulation of budding yeast-like cells and thin branching filaments are found

Rice. 11.25. Candidiasis. Scraping from the oral mucosa in acute

candidiasis.

1 - disputes; 2 - pseudomycelium. Micrograph, x 1200.

pseudomycelium (Fig. 11.25). Filaments of pseudomycelium are formed due to the elongation of cells and their arrangement in long chains, which are called pseudomycelium. Yeast-like fungi have almost no real mycelium. The acute course of the disease is accompanied by a predominance of cellular forms, rounded, partially budding. In the chronic course, mainly pseudomycelium threads and chains of rounded elongated budding cells are detected.

Microscopic examinations of the material should be repeated after the end of the course of treatment and the disappearance of clinical signs of the disease.

The identification of the obtained cultures of yeast-like fungi is carried out on the basis of the morphological characteristics of bacterial cells and the appearance of the grown colonies. To obtain cultures of fungi of the genus Candida, solid and liquid nutrient media with carbohydrates are used. In some cases, serological tests are performed for diagnosis.

Treatment. Treatment of patients with candidiasis includes exposure to the pathogen and treatment of concomitant diseases, an increase in specific and nonspecific factors.

Coy protection, sanitation of the oral cavity, rational nutrition. Patients with persistent chronic forms of candidiasis should be examined. Treatment of generalized and visceral forms of candidiasis is carried out by physicians-mycologists.

An important condition for the successful treatment of candidiasis is a thorough examination of the patient and the treatment of concomitant diseases, especially gastrointestinal pathology, diabetes mellitus, leukemia. In case of persistently flowing candidal seizure, prosthetics are necessary, in which, first of all, the height of the bite should be restored.

General treatment. To influence the causative agent of the disease, it is necessary to prescribe internally such antifungal drugs as nystatin or levorin, 500,000 IU 6-8 times a day after meals, the daily dose should be at least 3,000,000 IU.

The drugs are taken within 10 days. It is recommended to crush the tablets and, putting them under the tongue, suck, since they are poorly absorbed in the gastrointestinal tract. It is better to prescribe Levorin in the form of buccal (cheek) tablets. Each tablet contains 500,000 IU of levorin.

A good antifungal effect is obtained from sucking decamine, which is prescribed in the form of caramel: 1-2 caramel 6-8 times a day (every 3-4 hours). One or two caramels are placed under the tongue or by the cheek and held until completely absorbed, without making swallowing movements, if possible, so that the drug is in contact with the oral mucosa for as long as possible. One caramel contains 0.00015 g of decamine. Amphoglucamine is prescribed inside 200,000-500,000 IU 2 times a day after meals.

For the treatment of chronic persistent forms of oral mucosa candidiasis, amphotericin B is used, which is prescribed at the rate of 250 IU per 1 kg of body weight and locally in the form of an ointment. Diflucan has a more pronounced antifungal effect. It is prescribed in capsules of 50-100 mg (depending on the severity of the disease) 1 time per day. Diflucan has a long half-life.

To reduce dryness in the mouth and affect the fungal flora, a 2-3% solution of potassium iodide is prescribed, 1 tablespoon inside, 2-3 times a day after meals. Moreover, iodine exhibits its fungistatic properties during the period of its excretion through the skin, oral mucosa

and mucous glands. In addition, iodine stimulates salivation well. Patients with candidiasis need high-grade, high-quality nutrition with a decrease in the amount of easily digestible carbohydrates. Inside, vitamins of group B (B, B2, B6), PP, C are prescribed.

Local treatment. Includes the use of antifungal drugs. For applications and lubrication of the oral mucosa, 0.5% decamine ointment, amphotericin B ointment (30,000 U / g),

1% ointment and 1% clotrimazole solution (kanesten) are used. To treat the mucous membrane of the mouth and the red border of the lips, solutions of aniline dyes are used, primarily violet (1-2% solution of gentian violet, 2% methylene blue, fucorcin solution). Means are effective that alkalize the environment in the oral cavity, which has a detrimental effect on the fungal flora. For this purpose, rinse with 2-5% borax solution (sodium tetroborate), 2% sodium bicarbonate solution, 2% boric acid solution. In this case, at least 1 glass is rinsed out at a time, repeating the procedure 5-6 times a day. For applications and lubrication, a 20% solution of borax in glycerin is used, Lugol's solution in glycerin, etc.

With yeast jam and cheilitis, a good effect is obtained by nystatin ointment (100,000 units per 1 g of base), 5% levorin ointment, 0.5% decamine ointment. For local treatment, it is better to prescribe several different drugs and change them throughout the day or every other day.

Careful sanitation of the oral cavity, exclusion of any kind of injury to the mucous membrane, is of great importance in the treatment. The oral cavity can be sanitized 2 to 3 days after the start of the antifungal treatment. In acute and chronic candidiasis, careful treatment of prostheses with the same means (with the exception of dyes) that are used to treat the oral mucosa is necessary.

Prevention. It consists primarily in the correct and regular care of the oral cavity and dentures. With long-term treatment with antimicrobial drugs, antibiotics, corticosteroids, it is necessary to prescribe nystatin or levorin, 1,500,000 units per day for prophylactic purposes, vitamins of group B (B, B2, B6), C, alkaline mouth rinses. To clean your teeth, you should recommend toothpastes "Borglycerinovaya", "Berry" containing a solution of borax in glycerin. Removable dentures should be treated with special cleaning agents.

Evaluation Criteria for Monitoring

№	Progress in% and points	Grade	Student knowledge level
1.	96-100	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Shows high activity, creative approach when conducting interactive games. Correctly solves situational problems and test questions. Analyzes independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
2.	91-95	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment

			of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Analyzes research results independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
3.	86-90	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
4.	81-85	Good "4"	The correct answer to questions about the clinic, diagnosis and treatment of TBI, but there are 2-3 inaccuracies, errors. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
5.	76-80	Good "4"	Correct, but incomplete coverage of the issue. The student knows questions about the clinic and diagnosis of TBI, but is not fully versed in determining the tactics of treatment. Correctly solves situational problems, but the rationale for the answer is not complete. Understands the essence of the issue. Knows, speaks confidently
6.	71-75	Good "4"	Correct, but incomplete coverage of the issue. Correctly solves situational problems, but the rationale for the answer is incomplete. Has 2-3 errors. Knows, tells confidently. Has accurate ideas.
7.	66-70	Satisfactorily "3"	Understands the essence of the issue. Correctly solves situational problems, but cannot substantiate the answer. Has accurate ideas on specific issues.
8.	61-65	Satisfactorily "3"	Has an incomplete understanding of the topic. Makes mistakes when solving situational tasks. Tells not confidently.
9.	55-60	Satisfactorily "3"	Knows, tells not confidently. Has a partial view.
10.	54 and below	Unsatisfactory "2"	Has no exact idea. Does not know.

12- Practical lesson
Topic: Allergic diseases. Drug allergy. Clinic, diagnosis and treatment.

Technological map of the practical lesson

Stages of work	Teacher	Student
1. Stages of preparation (10 minutes)	1. Controls the purity of the audience; 2. Checks students' readiness for classes; 3. Controls attendance;	Listen
2.The introductory stage to classes (15 minutes)	1.Preparation on the topic of content; 2. Preparation of slides for the introductory report; 3. To develop the literature used for the study of the subject;	Write down the topic and listen
3. The main stage (195 minutes)	1. Dividing students into 2 small subgroups, asking questions on the topic; 2. Use of slides and multimedia; 3. Carries out medical work; 4. Combines all information on a given topic, actively the participating students are encouraged and appreciated;	Divided into small groups watching, participating, listening. The student expresses his opinion, complements and asks questions
4. Final stage (50 minutes)	1. Conclusion. 2. Independent work. 3. Homework.	Listen Write down

Questions on the topic
Give concepts about microbial allergy.
The located place is angioedema.
Clinic of catarrhal hemorrhagic stomatitis.
What can be the cause of angioedema?
Dif-diagnosis of angioedema.

Tests

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Allergic reaction of an immediate anaphylactic type
manifests itself in the form
1) urticaria
2) bronchial asthma
3) IEE
4) drug allergy
5) anaphylactic shock
Differential diagnosis is carried out
1) with Lyell's disease

2) with myocardial infarction
3) with acute heart failure
4) with hay fever
5) with epilepsy

Emergency measures
1) suspension of the receipt of the medicinal product
2) providing the patient with a sitting position
3) removal of removable dentures
4) removing tight clothes
5) measurement of body temperature
6) oxygen therapy
7) prevention of asphyxia
Medicinal substances used for the provision of emergency care
with anaphylactic shock
1) sympathomimetics
2) antihistamines
3) water-soluble glucocorticoids
4) cytostatics
5) bronchodilators
6) enzymes
7) antibiotics
8) tranquilizers and antipsychotics

In order to increase blood pressure, use
1) 0.1% adrenaline solution
2) 0.1% solution of nicotinic acid
3) 1% solution of mesaton
4) 1% solution of novocaine
5) 0.2% norepinephrine solution
Time of development of anaphylactic shock
1) a few minutes
2) a few hours
3) day

For the prevention of anaphylactic shock,
1) premedication
2) careful collection of anamnesis
3) pain relief
Quincke's edema has a nature
1) toxic-allergic
2) infectious-allergic
3) infectious toxic

Swelling can lead to the rapid development of asphyxia.
1) lower lip
2) lower lip and tongue
3) lower lip, tongue and larynx

Differential diagnosis of Quincke's edema with localization
on the lips spend
1) with Melkerson-Rosenthal syndrome
2) with erysipelas
3) with mechanical injury
4) with lip abscess

General treatment of angioedema
1) hyposensitization
2) antiviral drugs
3) detoxification therapy
4) restorative treatment
5) physiotherapy

Delayed allergic reactions include
1) hives
2) drug allergy
3) contact allergic stomatitis
4) bronchial asthma

Factors causing drug allergies
1) stress
2) smoking
3) taking the medicinal product
4) application of the medication to the mucous membrane of the mouth and skin

Elements of the lesion on the oral mucosa
with drug allergies
1) papule
2) stain
3) blister
4) bubble
5) bubble

6) erosion

1) general clinical blood test
2) immunological
3) serological
4) cytological

Clinical forms of drug allergy

1) catarrhal
2) catarrhal-hemorrhagic
3) hyperkeratosis
4) bullous
5) atypical
6) erosive
7) ulcerative necrotic

Necrotic ulcerative form of drug allergy differentiate from

1) ulcerative necrotizing stomatitis
2) lesions in AIDS
3) lupus erythematosus
4) leukemia
5) candidiasis

Non-specific hyposensitizing therapy includes prescribing

1) antihistamines
2) histaglobulin
3) cytostatics
4) calcium preparations
5) corticosteroids
6) sulfonamides

1) pain relievers
2) anti-inflammatory
3) antifungal
4) antihistamines
5) keroplastic
6) antiviral

Specific signs of drug allergy in the mouth

1) are detected
2) not detected

Interactive method

USING THE HANDLE IN THE MIDDLE TABLE METHOD

All students of the group are divided by lot into 3 subgroups of 3 students each. Each subgroup sits down at a separate table, prepares a blank sheet of paper and a pen. The date, group number, last name and first name of the student are written on the sheet. The task is offered, to answer one question for the whole subgroup. Each student writes down his last name and one answer option on a sheet and passes the sheet to a neighbor, and moves his pen to the middle of the table. The teacher supervises the work of the group and everyone's participation in it. The general correct version is written down in a notebook. Students who gave the correct answer options receive the maximum score - 100% of the theoretical part rating - 0.8b. Second place students - 85.9% of the rating. Third place - 70.9% of the rating. Those who did not answer or answered incorrectly 30% of the rating. The resulting score is taken into account when assigning marks for the current lesson. The works of students are saved by the teacher.

Text

Allergic diseases are now widespread, and their number is constantly growing and, which is especially important, the severity of the course; this turns the given problem into a social one.

The reasons for such a widespread occurrence of allergic diseases are different. First of all, an important role in this is played by environmental pollution by emissions of industrial waste, exhaust gases, the use of pesticides, herbicides, etc. in agriculture. The rapid development of the chemical industry and the associated appearance in

everyday life and in production of many synthetic materials, dyes, washing powders, cosmetics and other substances, many of which are allergens, also contribute to the spread of allergic diseases.

The widespread and often uncontrolled use of drugs also leads to an increase in the number of allergic reactions. Hypersensitivity to drugs often occurs due to the unjustified use of several drugs at the same time (polypharmacy). Along with this, a common cause of allergic reactions is the unreasonable and uncontrolled intake of drugs, especially antibiotics; insufficient knowledge by doctors of the pharmacokinetics of the prescribed drug, etc.

The influence of climatic factors (increased insolation, humidity), heredity, general somatic pathology, nutritional status, etc. also play a role in the occurrence of allergic diseases.

Allergy is an increased, and therefore altered, sensitivity of the body to certain substances of an antigenic nature, which do not cause painful phenomena in normal individuals. An important role in the development of allergies is assigned to the state of the nervous, as well as endocrine systems, pathology of the gastrointestinal tract.

The cause of allergies can be various substances - from simple chemical compounds (iodine, bromine) to the most complex (proteins, polysaccharides, as well as their combinations), which, when entering the body, cause an immune response of the humoral or cellular type. Substances that can cause an allergic reaction are called allergens. The number of allergens in nature is large, they are diverse in composition and properties. Some of them fall into the organ-

from the outside, they are called exoallergens, others are formed in the body and represent the body's own, but modified proteins - endoallergens, or autoallergens. Exoallergens are of non-infectious origin (plant pollen, household dust, animal hair, medicines, food products, detergents, etc.) and infectious (bacteria, viruses, fungi and their waste products). Exoallergens enter the body in various ways: through the respiratory tract, digestive tract, skin and mucous membranes, causing damage to various organs and systems.

Endoallergens are formed in the body from its own proteins under the influence of various damaging factors, which can be bacterial antigens and their toxins, viruses, thermal effects (burns, cooling), ionizing radiation, etc.

Allergens can be complete antigens and incomplete - haptens. Haptens can cause an allergic reaction by combining with macromolecules in the body that induce the production of antibodies, while the specificity of the immunological response will be directed against the hapten, and not against its carrier; the formation of antigenic complexes with body molecules, while antibodies are formed only to the complexes, and not to its components.

Due to the multitude of naturally occurring and formed in the body, allergens are manifold and manifestations of allergic reactions. However, allergic reactions of different clinical manifestations have common pathogenetic mechanisms. There are three stages of allergic reactions: immunological, pathochemical (biochemical) and pathophysiological, or the stage of functional and structural disorders.

The immunological stage begins with the contact of the allergen with the body, which results in the sensitization of the latter, that is, the formation of antibodies or sensitized lymphocytes capable of acting with this allergen. If the allergen is removed from the

body by the time the antibodies are formed, no painful manifestations occur. The first introduction of an allergen into the body has a sensitizing effect. With repeated exposure to an allergen, an allergen-antibody or allergen-sensitized lymphocyte complex is formed in an organism already sensitized to it. From this moment, the pathochemical stage of an allergic reaction begins, characterized by the release of biologically

active substances, mediators of allergy: histamine, serotonin, bradykinin, etc.

The pathophysiological stage of an allergic reaction, or the stage of clinical manifestation of damage, is the result of the action of the isolated biologically active substances on tissues, organs and the body as a whole. This stage is characterized by circulatory disorders, spasm of smooth muscles of the bronchi, intestines, changes in the composition of blood serum, impaired coagulability, cell cytolysis, etc.

According to the mechanism of development, 4 types of allergic reactions are distinguished: I - immediate type reaction (reagin type); II - cytotoxic type; III - tissue damage by immune complexes (Artyus type); IV - delayed-type reaction (cellular hypersensitivity). Each of these types has a special immune mechanism and a set of mediators inherent in it, which determines the features of the clinical picture of the disease.

An allergic reaction of type I, or a reaction of an immediate type, is also called a reaginic, anaphylactic or atopic type of reaction. It develops with the formation of antibodies, called reagins, belonging mainly to the IgE and IgG4 class. Reagins are fixed on mast cells and basophilic leukocytes. When reagins are combined with the corresponding allergen, mediators are released from these cells: histamine, heparin, serotonin, platelet-activating factor, prostaglandins, leicotrienes, etc., which determine the clinical picture of an immediate allergic reaction. After contact with a specific allergen, clinical manifestations of the reaction occur in 15-20 minutes; hence its name "immediate reaction."

Allergic reaction type II, or a cytotoxic type reaction. This type is characterized by the fact that antibodies are formed against tissue cells and are mainly represented by IgG and IgM. This type of response is only triggered by antibodies capable of activating complement. The antibodies bind to the modified cells of the body, which leads to the activation of complement, which also causes damage and even destruction of cells. As a result of the cytotoxic type of allergic reaction, cell destruction occurs, followed by phagocytosis and the removal of destroyed cells and tissues. The cytotoxic type of reactions includes drug allergy, characterized by leukopenia, thrombocytopenia, hemolytic anemia.

Allergic reaction of type III, or tissue damage by immune complexes (Arthus type, immunocomplex type). This type of reaction occurs as a result of the formation of circulating immune complexes, which include antibodies of the IgG and IgM class. Antibodies of this class are called precipitating antibodies, since they form a precipitate when combined with the corresponding antigen. Allergens with this type of reaction can be bacterial, food.

This type of reaction is leading in the development of serum sickness, allergic alveolitis, in some cases, drug and food allergies, in a number of auto-allergic diseases (systemic lupus erythematosus, rheumatoid arthritis, etc.).

Type IV allergic reaction, or delayed-type allergic reaction (delayed-type hypersensitivity, cellular hypersensitivity). In this type of reaction, the role of antibodies is played by sensitized T-lymphocytes, which have receptors on their membranes that can specifically interact with the sensitizing antigen. When such a lymphocyte combines with an allergen, which can be dissolved or be on cells, mediators of cellular immunity - lymphokines - are released. More than 30 lymphokines are known, which show their action in various combinations and concentrations, depending on the characteristics of the allergen, the genotype of lymphocytes and other conditions. Lymphokines cause macrophages and other lymphocytes to accumulate, resulting in inflammation. One of the main functions of mediators is their involvement in the process of destruction of antigen (microorganisms or foreign cells), to which lymphocytes are sensitized. If a foreign tissue graft acts as antigenic substances that stimulated delayed-type hypersensitivity, then it is destroyed and rejected. A delayed-type reaction develops in a sensitized organism, usually 24-48 hours after contact with an allergen. The cellular type of reaction underlies the development of most viral and some bacterial infections (tuberculosis, syphilis, leprosy, brucellosis, tularemia), some forms of infectious-allergic bronchial asthma, rhinitis, transplant and antitumor immunity.

The type of development of an allergic reaction is determined by the nature and properties of antigens, as well as the state of the body's reactivity.

Specific diagnostics of allergic diseases.

It consists of collecting an allergic anamnesis, carrying out diagnostic tests and laboratory tests.

When collecting an allergic anamnesis, it is necessary to focus on identifying the entire set of household and industrial contacts with various substances that can act as allergens. Along with this, the anamnesis allows you to establish the presence of an allergic predisposition (hereditary or acquired), as well as possible exo- and endogenous factors affecting the course of the disease (climatic, endocrine, mental, etc.). When collecting an anamnesis, it is necessary to find out how the patient reacts to the introduction of vaccines, serums, medication and the circumstances of the onset of an exacerbation, as well as housing and. working conditions.

Establishing professional contacts with various substances is very important. It is known that contact with simple chemicals more often causes delayed-type allergic reactions (contact dermatitis). Complex organic substances can cause immediate allergic reactions with the development of diseases such as Quincke's edema, urticaria, allergic rhinitis, bronchial asthma, etc.

A carefully collected medical history suggests a possible type of allergic reaction and a probable allergen. The specific allergen, which is the cause of the development of the disease, is established using special diagnostic tests and laboratory tests.

Allergic diagnostic tests are a method for detecting specific sensitization of the body.

Allergic diagnostic tests are performed outside the phase of exacerbation of the disease 2-3 weeks after the acute allergic reaction, during the period when there is a decrease in the body's sensitivity to the allergen.

Distinguish between skin and provocative allergic diagnostic tests.

Skin tests are based on identifying specific sensitization of the body by introducing an allergen through the skin and assessing the nature of the developing inflammatory

reaction. There are the following methods of performing skin tests: application, scarification and intradermal. The choice of skin testing method is determined by the nature of the disease, the type of allergic reaction and the group belonging of the tested allergen.

So, for the diagnosis of drug allergy, application tests are the most convenient. Determination of hypersensitivity to allergens of bacterial and fungal origin is carried out by the method of intradermal samples.

Provocative tests are carried out in cases where the data of the allergic history does not correspond to the results of skin tests. Provocative tests are based on the reproduction of an allergic reaction by introducing an allergen into an organ or tissue, the defeat of which is the leading one in the clinical picture of the disease. Distinguish between nasal, conjunctival and inhalation provocative tests. To provocative tests also include cold and heat tests, used for cold and heat urticaria.

Specific diagnostics of allergic reactions is also carried out using laboratory research methods: the reaction of degranulation of basophilic leukocytes (Shelley test), the reaction of blast transformation of leukocytes, the reaction of damage to neutrophils, the reaction of leukocytolysis, etc. The advantage of diagnostic methods for allergic reactions carried out in vitro is the complete absence of danger the occurrence of anaphylactic shock.

11.4.1. Clinical manifestations of some allergic diseases

11.4.1.1. Anaphylactic shock

Anaphylactic shock (choc anaphylactics) has no specific manifestations on the oral mucosa, but is the most serious prognostic allergic disease that occurs in the dentist's practice and is often fatal. In the arsenal of remedies for the dentist, there are many drugs that can cause the development of anaphylactic shock. As you know, many materials and medicines are used in dental practice: anesthetics, analgesics, antiseptics, antibiotics, vitamins, acrylic plastics, amalgam, paste for filling root canals (based on formalin, eugenol), etc. Most of these substances have pronounced antigenic properties, especially acrylic plastics, mercury, novocaine.

Most often, anaphylactic shock develops after

the result of parenteral, especially intravenous, administration of drugs. However, there are known cases of anaphylactic shock with oral and local (applications to the mucous membrane of the mouth, periodontal dressings, the introduction of the drug into the socket of the extracted tooth, etc.), the use of medicinal substances.

Under certain conditions associated with the individual reactivity of the organism, as well as the type and method of application of these substances, they can act as allergens and cause an allergic reaction.

Anaphylactic shock refers to an immediate-type allergic reaction, which is based on the formation of antibodies-reagins. Their subsequent contact with the antigen (allergen) leads to the formation of biologically active substances (histamine, leukotrienes, prostaglandins, etc.), which forms a clinical picture of anaphylactic shock caused by the following mechanisms: increased vascular permeability of the microcirculatory bed, spasm of bronchial smooth muscles , intestines and other organs, a drop in vascular tone.

The clinical picture. The clinical manifestations of anaphylactic shock are diverse and can have several clinical variants.

Hemodynamic variant with a predominance of symptoms of acute cardiovascular failure: a weak rapid pulse, hyperemia of the skin, alternating with pallor, profuse sweating, an increasing drop in blood pressure to immeasurable values. The patient is pale and in extremely severe cases loses consciousness.

Anaphylactic shock can occur with a predominance of disorders of the central nervous system. Patients become restless, there is a feeling of fear, convulsions, symptoms of cerebral edema (headache, vomiting, epileptiform seizures, hemiplegia, aphasia, etc.).

In the clinical picture of anaphylactic shock, disorders of the respiratory system (bronchospasm, symptoms of edema of the larynx, lungs) or the gastrointestinal tract (nausea, vomiting, diarrhea, pain in the stomach and intestines) may dominate.

The time of development of anaphylactic shock from the moment of administration of the antigen to the appearance of clinical signs ranges from several minutes to half an hour. The shorter the latent period of anaphylactic shock, the more severe it is. And if the patient is not provided with timely assistance, a lethal outcome is possible. Highly sensitive

In bilized patients, neither the dose nor the method of administration of the drug have a decisive effect on the severity of the course of anaphylactic shock.

There are three degrees of severity of anaphylactic shock: mild, moderate and severe.

Typical manifestations of anaphylactic shock, observed in most patients, are characterized by the following clinical symptoms: there is a state of discomfort, general anxiety with vague painful feelings of fear of death. There is a feeling of "heat", "the whole body seems to have been burned with nettles." Patients complain of itching and tingling of the skin of the face, hands, sudden weakness, headaches, dizziness, a feeling of rush of blood to the head, face, tongue, heaviness in the chest or chest compression. Disturbed by pain in the region of the heart, difficulty breathing, sometimes - pain in the abdominal cavity. In severe anaphylactic shock, the patient does not have time to make complaints and immediately loses consciousness.

Objective symptoms of anaphylactic shock are hyperemia of the skin of the face and body, alternating with pallor and cyanosis, edema of the eyelids, red border of the lips, and oral mucosa. Often there are clonic convulsions of the extremities, and sometimes widespread convulsive seizures, motor restlessness. Pupils dilate and do not respond to light.

A detailed clinical picture of a severe form of anaphylactic shock is characterized by serious disorders of vital organs and systems. Cardiovascular and hemodynamic disorders develop: profuse sweating, weakening of cardiac activity. Deaf heart sounds, frequent threadlike pulse, tachycardia. Blood pressure decreases rapidly; in severe cases, diastolic pressure is not determined. Cardiovascular failure is often fatal.

Usually, simultaneously with the formation of cardiovascular insufficiency, shortness of breath, shortness of breath with wheezing and foaming at the mouth appear. In the future, a picture of pulmonary edema develops, which indicates a severe course of anaphylactic shock.

Gastrointestinal disorders are common. There are spastic abdominal pains, vomiting, often diarrhea mixed with blood.

Spasms of smooth muscles and other organs occur. This is accompanied by neuropsychiatric disorders

yami in the form of strong excitement, replaced by complete indifference, headache, visual impairment, hearing and balance. A coma develops, sometimes convulsions, urinary and fecal incontinence are observed. The causes of death, as a rule, are vascular insufficiency or asphyxia due to bronchospasm or laryngeal edema. An increase in body temperature is uncharacteristic, for the most part it even goes down.

The outcome of anaphylactic shock depends not only on the severity of the course and the severity of the clinical picture, but to a large extent on the timeliness and usefulness of the therapy.

In the diagnosis of anaphylactic shock, the degree of alertness of the doctor in this regard is of great importance. Usually, the diagnosis is not difficult, since it is quite easy to establish a connection between the violent reaction of the body and exposure to the allergen.

Differential diagnosis. Anaphylactic shock is differentiated from

^ acute heart failure,

^ myocardial infarction,

^ epilepsy (in the presence of seizures).

Treatment. The fight against anaphylactic shock should begin immediately when the first signs of anaphylaxis appear and should be aimed primarily at stopping the further entry of the allergen into the body or reducing its absorption (if the drug has already been administered). To do this, a tourniquet is applied above the injection site (if possible) or the injection site is injected with 0.3-0.5 ml of 0.1% adrenaline solution. These measures help to reduce the "absorption of the allergen. The patient is given a horizontal position on his back with his head slightly lowered, the lower jaw is extended anteriorly to prevent asphyxia due to the retraction of the tongue or aspiration of vomit, removable dentures are removed from the mouth. They release the neck, chest and abdomen from compression, provide oxygen flow Oxygen therapy is carried out by supplying oxygen through a mask or nasal catheter.

In order to increase blood pressure, sympathomimetics are used: subcutaneously or intramuscularly, 0.5 ml of a 0.1% solution of adrenaline, or 0.3-1.0 ml of a 1% solution of mezaton, or intravenously (drip) 2-4 ml 0, 2% norepinephrine solution diluted in 1 liter of 5% glucose solution or isotonic sodium chloride solution. For faster absorption, it is better to administer the drugs fractionally in different parts of the body (for example, epinephrine is injected in fractional doses of 0.5 ml every 15-30 minutes until the patient is taken out of shock). In severe cases, intravenous administration of 0.1-0.2 ml of 0.1% epinephrine solution for 3-5 minutes is indicated. Long-term therapy is carried out in the absence of an effect. Add 1 ml of 0.1% adrenaline solution to 250.0 ml of 5% glucose solution. Start infusion at a rate of 50-60 drops per minute.

Antihistamines are administered after blood pressure is normalized. Intramuscular injections of 1% diphenhydramine solution, 2.5% diprazine solution, 2% suprastin solution, 2 ml of tavegil solution or other antihistamines are used.

In moderate and severe forms of anaphylactic shock, water-soluble glucocorticosteroid preparations are used, which have a pronounced desensitizing and anti-inflammatory effect. They are administered intravenously (jet or drip) in 5% glucose solution or isotonic sodium chloride solution. More often used 50-150 mg of hydrocortisone hemisuccinate, in severe cases the dose is increased to 300 mg, 60-120 mg of prednisolone hemisuccinate.

To stop bronchospasm, a 2.4% solution of aminophylline is used, which is administered intravenously in 5-10 ml doses, diluted in 10 ml of isotonic sodium chloride solution, or in 10 ml of 10% or 40% glucose solution.

In the presence of seizures and increased excitement of the patient, antipsychotics and tranquilizers are used (seduxen, relanium, elenium, droperidol, etc.).

If anaphylactic shock develops from penicillin, 1 000 000 U of penicillinase in 2 ml of isotonic sodium chloride solution should be injected intramuscularly once.

Due to the lightning speed of the development of anaphylactic shock, the time for emergency care is limited to minutes, therefore, the doctor should have a set of prepared means for providing emergency care to such patients. It includes: sympathomimetics: ampoules with adrenaline solutions (0.1%),

norepinephrine (0.2%), mezatone (1%); i antihistamines: ampoules with solutions

suprastin (2%), diphenhydramine (1%), tavegil (0.001 g in 2 ml);

corticosteroids: ampoules with solutions of prednisolone hemisuccinate (in ampoules of 25 mg), tablets of prednisolone 5 mg, hydrocortisone hemisuccinate in ampoules of 25 and 100 mg, hydrocortisone Solu-Cortef for intravenous administration in vials of 300 mg;

bronchodilators: ampoules with solutions of ephedrine hydrochloride (5%), aminophylline (2.4%, 10.0 ml);

anticonvulsants: ampoules with calcium pantothenate (20%, 2 ml);

solutions: 5% glucose solution in 10 ml ampoules, 0.9% sodium chloride solution in 5-10 ml ampoules and in 400 ml vials, hemodez in 200 and 400 ml vials;

penicillinase, 1 million units in ampoules;

equipment: disposable systems for intravenous administration of drugs, disposable syringes from 1 to 20 ml; harnesses, mouth dilators.

Prevention of anaphylactic shock consists in a thorough history taking. Before the introduction of a drug, it is necessary to find out whether its use or drugs of a related group was accompanied by any reactions. For the prevention of anaphylactic shock in patients with a history of allergic reactions, it is necessary to prescribe antihistamines before introducing a new drug.

Evaluation Criteria for Monitoring

№	Progress in% and points	Grade	Student knowledge level
1.	96-100	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Shows high activity, creative approach when conducting interactive games. Correctly solves situational problems and test

			questions. Analyzes independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
2.	91-95	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Analyzes research results independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
3.	86-90	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
4.	81-85	Good "4"	The correct answer to questions about the clinic, diagnosis and treatment of TBI, but there are 2-3 inaccuracies, errors. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
5.	76-80	Good "4"	Correct, but incomplete coverage of the issue. The student knows questions about the clinic and diagnosis of TBI, but is not fully versed in determining the tactics of treatment. Correctly solves situational problems, but the rationale for the answer is not complete. Understands the essence of the issue. Knows, speaks confidently
6.	71-75	Good "4"	Correct, but incomplete coverage of the issue. Correctly solves situational problems, but the rationale for the answer is incomplete. Has 2-3 errors. Knows, tells confidently. Has accurate ideas.
7.	66-70	Satisfactorily	Understands the essence of the issue.

		"3"	Correctly solves situational problems, but cannot substantiate the answer. Has accurate ideas on specific issues.
8.	61-65	Satisfactorily "3"	Has an incomplete understanding of the topic. Makes mistakes when solving situational tasks. Tells not confidently.
9.	55-60	Satisfactorily "3"	Knows, tells not confidently. Has a partial view.
10.	54 and below	Unsatisfactory "2"	Has no exact idea. Does not know.

13- Practical lesson

Topic: Erythema multiforme exudative, Clinic, diagnostics and treatment

Technological map of the practical lesson

Stages of work	Teacher	Student
1. Stages of preparation (10 minutes)	1. Controls the purity of the audience; 2. Checks students' readiness for classes; 3. Controls attendance;	Listen
2.The introductory stage to classes (15 minutes)	1.Preparation on the topic of content; 2. Preparation of slides for the introductory report; 3. To develop the literature used for the study of the subject;	Write down the topic and listen
3. The main stage (195 minutes)	1. Dividing students into 2 small subgroups, asking questions on the topic; 2. Use of slides and multimedia; 3. Carries out medical work; 4. Combines all information on a given topic, actively the participating students are encouraged and appreciated;	Divided into small groups watching, participating, listening. The student expresses his opinion, complements and asks questions

Questions on the topic

Etiology of erythema multiforme exudative.

Classification of erythema multiforme exudative.

The clinical picture is erythema multiforme exudative.

Differential diagnosis of erythema multiforme exudative.

Local treatment of erythema multiforme

Tests

Erythema multiforme (EEE) is referred to as
1) to precancerous
2) to cystic dermatoses
3) to infectious diseases
4) to viral diseases
5) to traumatic injuries

Factors provoking IEE
1) hypothermia
2) contact with infectious patients
3) insolation
4) taking the medicinal product

MEE is more common
1) in children
2) in young and middle-aged people
3) in the elderly

Indicate the numbers of all correct answers

Known forms of the disease in PEE
1) infectious-allergic
2) infectious toxic
3) toxic-allergic
4) exudative hyperemic

Seasonality of MEE disease
1) expressed
2) not expressed
3) is persistent

With MEE, may be affected
1) leather
2) mucous membrane
3) skin and mucous membrane

A lesion located on the skin
1) stain
2) pustule
3) cockade
4) ecchymosis
Primary elements of lesion on the mucous membrane with PEE
1) stain
2) bubble
3) bubble

4) abscess
5) knot

Secondary elements of the lesion on the mucous membrane
and red border of lips with MEE
1) erosion
2) ulcer
3) flake
4) crust
5) scar

The cytological picture with MEE corresponds to
pathological state of the epithelium
1) edema
2) infiltration
3) acantholysis
4) hyperkeratosis
5) parakeratosis

The severe form of MEE is called
1) Behcet's syndrome
2) Sjogren's syndrome
3) Rosenthal syndrome
4) Stevens-Johnson syndrome

Set correspondence

Period of PEE disease Research method
1) acute course a) histamine test
2) remission b) general clinical blood test
c) skin-allergic tests
d) leukocytolysis reaction
e) the reaction of inhibition of migration
leukocytes

Additional examination methods for MEE
1) general clinical blood test
2) allergy tests
3) blood test for RW
4) scraping for Candida mushroom
Differential diagnosis of PEE is carried out
1) with acute herpetic stomatitis
2) with pemphigus vulgaris
3) with flat leukoplakia
4) with candidiasis
5) with medical stomatitis

6) lichen planus of an erosive form
Elements of lesion on the lips with MEE
1) crust
2) flake
3) crack
4) erosion
Set correspondence

Period of illness PEE Methods of treatment PEE
1) remission a) desensitizing
2) exacerbation b) vitamin therapy
c) tonic
d) sanitation of the oral cavity
e) detoxification

Specify the correct sequence

Local treatment of lesions with MEE is carried out
1) keratoplasty
2) antiseptics
3) local anesthetics
Local treatment during exacerbation of MEE includes
1) pain relief
2) antiseptic treatment
3) antifungal drugs
4) antiviral drugs
5) epithelizing drugs
6) proteolytic enzymes
General treatment for MEE
1) detoxification
2) hyposensitizing
3) antifungal
4) antiviral
5) vitamin therapy
Drugs used for the general treatment of MEE
1) sodium thiosulfate
2) sodium salicylate
3) delagil
4) bonafton

Interactive method
USING THE HANDLE IN THE MIDDLE TABLE METHOD

All students of the group are divided by lot into 3 subgroups of 3 students each. Each subgroup sits down at a separate table, prepares a blank sheet of paper and a pen. The date, group number, last name and first name of the student are written on the sheet. The task is offered, to answer one question for the whole subgroup. Each student writes down his last name and one answer option on a sheet and passes the sheet to a neighbor, and moves his pen to the middle of the table. The teacher supervises the work of the group and everyone's participation in it. The general correct version is written down in a notebook. Students who gave the correct answer options receive the maximum score - 100% of the theoretical part rating - 0.8b. Second place students - 85.9% of the rating. Third place - 70.9% of the rating. Those who did not answer or answered incorrectly 30% of the rating. The resulting score is taken into account when assigning marks for the current lesson. The works of students are saved by the teacher.

Text

Exudative erythema multiforme (erythema exudativum multiforme) is an inflammatory disease of the mucous membranes and skin, characterized by polymorphism of lesion elements (blisters, spots, blisters).

The mucous membrane of the mouth or skin can be affected in isolation, but their combined lesion is common. Erythema multiforme is characterized by an acute onset and lasts for years. Exacerbations are different

mainly in the autumn-spring period. Mostly young people (20-40 years old) are ill, more often men.

Etiology and pathogenesis. Not fully clarified. According to the etiological principle, there are two types of exudative erythema multiforme. The true, or idiopathic, form is of an infectious-allergic nature. In most patients with this form of the disease, skin tests reveal sensitization to bacterial allergens (streptococcus, staphylococcus, Escherichia coli). The source of sensitization is the foci of chronic infection. Decreased body reactivity due to hypovitaminosis, hypothermia, viral infections, stress provoke exacerbation of exudative erythema multiforme. The toxic-allergic, or symptomatic, form has a similar clinical picture with the true infectious-allergic exudative erythema multiforme, but in essence it is a hyperergic reaction of the body to drugs (antibiotics, salicylates, amidopyrine, etc.).

The clinical picture. The disease begins suddenly. Malaise, chills, weakness appear, body temperature rises (in severe cases, up to 38 ° C and above). Patients complain of headache, aching pains throughout the body, pain in muscles and joints, in the throat. After 1-2 days, bluish-red spots appear on the skin of the hands, forearm, lower leg, sometimes the face and neck, slightly rising above the surrounding skin (Fig. 11.29). The central part of them sinks slightly and takes on a bluish tint, while the peripheral part retains a pinkish-red color (cockades). Subsequently, a subepidermal bladder filled with serous or hemorrhagic contents may appear in the central part. The appearance of skin elements is sometimes accompanied by itching and burning, or even disappears without pain.

The most commonly affected mucous membrane of the lips, cheeks, floor of the mouth, tongue, soft palate. The first manifestations of exudative erythema multiforme in the oral cavity are diffuse or limited erythema and edema of the mucous membrane,

against which subepithelial blisters of various sizes appear. The defeat of the oral mucosa is accompanied by sharp pains even at rest. With the movements of the tongue and lips, the pain increases sharply, as a result of which it becomes difficult to eat. Patients are starving, which further aggravates their condition.

Rice. 11.29. Erythema multiforme exudative. Hemorrhagic crust at the site of erosion on the skin of the eyelid.

The bubbles quickly open up, forming painful erosions on the mucous membrane of the mouth, covered with fibrinous plaque (Fig. 11.30). On the red border of the lips, erosion becomes covered with bloody crusts, which make it difficult to eat and open the mouth (Fig. 11.31). In the first days after opening the blisters, along the edge of the erosion, you can see grayish-white remnants of the blister cover, when you pull on which it is not possible to stratify the epithelium (negative symptom Nikolsky). The unsatisfactory state of oral hygiene, the presence of carious teeth aggravate the course of exudative erythema multiforme. Erosive surfaces are infected by the microflora of the oral cavity. In some cases, the course of exudative erythema multiforme is complicated by the addition of fusospirochetosis. Erosion on the mucous membrane of the mouth is covered with a thick layer of yellowish-gray plaque, plaque appears on the teeth and tongue, and bad breath. Salivation increases. Regional lymph nodes are enlarged, painful. The period of exacerbation lasts 2-4 weeks. Erosions are epithelized after 7-12 days. After their healing, no scars remain.

The picture of peripheral blood during exacerbation of exudative erythema multiforme corresponds to an acute inflammatory process.

The severity of the clinical course of exudate multiforme

Rice. 11.30. Erythema multiforme exudative.

Erosions covered with a fibrinous film on the mucous membrane of the upper lip and gums.

Rice. 11.31. Erythema multiforme exudative.

Hemorrhagic crusts on the surface of extensive erosions on the red border of the lips.

tive erythema is mainly due to the nature of the lesion of the oral mucosa. The severe form is characterized by a pronounced hyperergic reaction of the body, as well as generalized damage to the mucous membrane of the mouth and skin. In the case of a mild course of exudative erythema multiforme, the general condition of the patients does not change significantly;

single elements of defeat. However, with an increase in the duration of the disease, the degree of its severity is aggravated.

For exudative erythema multiforme of an infectious-allergic nature, a long recurrent course is typical. Exacerbations of the disease are observed mainly in the autumn-spring period (1-2 times a year), although there are cases of more frequent exacerbations of the disease. Sometimes relapses can be triggered by hypothermia, past infectious diseases and other factors that weaken the body's resistance. Erythema multiforme lasts for years. In the periods between exacerbations, there are no changes in the oral mucosa and skin.

Symptomatic (toxic-allergic) multiforme exudative erythema recurs only if the patient comes into contact with the etiological factor (drug-allergen).

Cytological examination of scrapings from the area of erosion reveals a picture of acute nonspecific inflammation.

Histologically, the subepithelial location of the blisters is determined in exudative erythema multiforme. There are no acantholysis phenomena. The rejected epithelium undergoes necrosis, edema in the underlying connective tissue, inflammatory infiltration.

Stevens-Johnson syndrome (acute mucocutaneous ocular syndrome, ectodermosis erosiva pluriorificialis) is a severe variant of exudative erythema multiforme with characteristic specific symptoms. The disease begins suddenly, with high fever and joint pain. Against the background of a serious general condition, bubbles appear on the lips, mucous membrane of the cheeks, tongue, soft palate, back wall of the pharynx, arches, larynx and on the skin, after opening which bleeding erosions form. Merging, they turn into a continuous bleeding sharply painful surface. Part of the erosion is covered with fibrinous plaque. The mucous membrane of the eyes (bilateral conjunctivitis and keratitis), nose (rhinitis, nosebleeds), genitals is also affected. Due to the generalized lesion of the oral cavity and lips, patients cannot talk, eat, which leads to their exhaustion. Cases of death in this disease have been described.

Differential diagnosis. Erythema multiforme is differentiated from:

And acantholytic pemphigus;

A non-acantholytic pemphigus;

And acute herpetic stomatitis;

And secondary syphilis.

Unlike pemphigus, exudative erythema multiforme is characterized by an acute course with rapid dynamics of lesion elements, pronounced inflammatory phenomena, a negative Nikolsky symptom, the absence of erosions in smears-prints from the surface of erosions and in exudate of acantholytic cell bubbles.

From acute herpetic stomatitis, exudative erythema multiforme is distinguished by larger erosions that do not have polycyclic outlines, the absence of lesions in the areas of the oral mucosa typical for herpetic stomatitis, and the absence of multinucleated cells in the scrapings from erosions.

The acute course, the severity of the inflammatory reaction of the oral mucosa, the seasonal recurrent nature of the course, in the intervals between which there are no signs of the disease, distinguishes exudative erythema multiforme from benign non-acantholytic pemphigus.

A mild course of exudative erythema multiforme may resemble eroded papules in secondary syphilis, at the base of which there is always infiltration. Hyperemia around syphilitic papules, including eroded ones, has the form of a narrow rim, sharply delimited from the healthy mucous membrane, while with exudative erythema multiforme there is extensive, diffuse hyperemia. Soreness of syphilitic papules is not very pronounced, pale treponemas are found in scrapings from their surface, serological reactions to syphilis are positive.

The toxic-allergic form is diagnosed on the basis of the medical history data, as well as the results of in vitro immunological studies (Shelley's basophil degranulation test, lymphocyte blast transformation test, cytopathological test) and the termination of the exacerbation after the withdrawal of the allergen drug. In the presence of skin rashes, the diagnosis is not difficult.

Treatment. In the acute period of the disease, symptomatic treatment is carried out aimed at reducing the intoxication of the body, desensitization, relieving inflammation and accelerating the epithelialization of the affected oral mucosa.

General treatment is carried out using desensitizing drugs: diphenhydramine, suprastin, tavegil, fenkarol, claritin, etc. For anti-inflammatory therapy, salicylates (acetylsalicylic acid, sodium salicylate), calcium gluconate, calcium glycerophosphate, etc. are used. For the same purpose, intravenous thiosulfate is prescribed for the same purpose. sodium (10 ml of 30% solution daily, for a course of 8-10 injections).

B vitamins (B, B2, B6), ascorutin are prescribed.

The use of ethacridine lactate (0.05 g three times a day for 10-20 days) in combination with levamisole (150 g a day, 2 consecutive days a week, with five-day breaks during 2 months).

General treatment of patients with severe exudative erythema multiforme should be carried out in a hospital setting, where they are prescribed complex therapy: detoxifying; desensitizing; anti-inflammatory.

To do this, use corticosteroid drugs - prednisolone (20-30 mg per day, depending on the severity of the process). The drug in the indicated dose is taken for 5-7 days, then every 2-3 days the dose of prednisolone is reduced by 5 mg until the drug is completely discontinued. The initial dose of dexamethasone is 3-5 mg.

In Stevens-Johnson syndrome, corticosteroid drugs are prescribed in higher dosages (60-80 mg of prednisone per day). Detoxifying and desensitizing therapy is carried out. Intravenous rheopolyglucin, hemodez, sodium thiosulfate, sodium hyposulfite, etc. are administered.

With the toxic-allergic form of exudative erythema multiforme, it is necessary to identify the causative allergen drug and stop taking it.

Local treatment is aimed at eliminating inflammation, swelling and accelerating the epithelialization of the affected oral mucosa. Before carrying out drug treatment of the oral mucosa, it must be anesthetized with 1-2% trimecaine solution, 1-2% pyromecaine solution, 1-2% lidocaine solution. For application of anesthesia, anesthetics in aerosols Xylostesin, Lidocain-spray, Anesthesie-spray, etc. are successfully used. In order to reduce pain before meals, oral baths with 1-2% trimecaine solution are prescribed. Antiseptic treatment of the oral mucosa is carried out with 0.25% - 0.5% hydrogen peroxide solution,

0.25% chloramine solution, 0.02% furacilin solution, 0.5% etonium solution, etc.

In the presence of necrotic plaque on the surface of erosion, applications of proteolytic enzymes (trypsin, chymotrypsin, lysoamidase) are used, after which keratoplastic agents (carotolin, rosehip and sea buckthorn oil, oil solution of vitamins A, E, etc.) are used to accelerate the epithelialization of the damaged mucous membrane.

Treatment of the oral mucosa during the period of exacerbation must be carried out daily, and in the case of treatment in a hospital 2-3 times a day.

Skin lesions, as a rule, do not require special treatment. In the presence of itching and burning in the area of erythema on the skin, it is recommended to lubricate them with Castellani liquid or 2% salicylic alcohol.

In case of eye damage, use 0.5% hydrocortisone eye ointment, 0.1% dexamethasone solution.

An indispensable condition for the successful treatment of patients with exudative erythema multiforme is the identification and elimination of foci of chronic infection. During the period of remission of the disease, patients should be thoroughly examined and sanitized. In the presence of microbial sensitization, a specific hyposensitizing therapy is carried out with allergens to which hypersensitivity has been established. In some cases, repeated courses of subcutaneous administration of histaglobin are effective (1-2 ml 2-3 times a week, for a course of only 8-10 injections), as well as measles and antistaphylococcal gamma globulin (for a course of treatment 5-7 injections) ...

Forecast. With exudative erythema multiforme, the prognosis for life is favorable and very serious in Stevens-Johnson syndrome

Evaluation Criteria for Monitoring

№	Progress in% and points	Grade	Student knowledge level
1.	96-100	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Shows high activity, creative approach when conducting interactive games. Correctly solves situational problems and test questions. Analyzes independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
2.	91-95	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Analyzes research results independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
3.	86-90	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently.

			Has an accurate idea.
4.	81-85	Good "4"	The correct answer to questions about the clinic, diagnosis and treatment of TBI, but there are 2-3 inaccuracies, errors. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
5.	76-80	Good "4"	Correct, but incomplete coverage of the issue. The student knows questions about the clinic and diagnosis of TBI, but is not fully versed in determining the tactics of treatment. Correctly solves situational problems, but the rationale for the answer is not complete. Understands the essence of the issue. Knows, speaks confidently
6.	71-75	Good "4"	Correct, but incomplete coverage of the issue. Correctly solves situational problems, but the rationale for the answer is incomplete. Has 2-3 errors. Knows, tells confidently. Has accurate ideas.
7.	66-70	Satisfactorily "3"	Understands the essence of the issue. Correctly solves situational problems, but cannot substantiate the answer. Has accurate ideas on specific issues.
8.	61-65	Satisfactorily "3"	Has an incomplete understanding of the topic. Makes mistakes when solving situational tasks. Tells not confidently.
9.	55-60	Satisfactorily "3"	Knows, tells not confidently. Has a partial view.
10.	54 and below	Unsatisfactory "2"	Has no exact idea. Does not know.

14- Practical lesson
Topic: Chronic recurrent aphthous stomatitis. Clinic, differential diagnosis and treatment

<u>Technological map of the practical lesson</u>

Stages of work	Teacher	Student
1. Stages of preparation (10 minutes)	1. Controls the purity of the audience; 2. Checks students' readiness for classes; 3. Controls attendance;	Listen
2.The introductory stage to classes (15 minutes)	1.Preparation on the topic of content; 2. Preparation of slides for the introductory report; 3. To develop the literature used for the study of the subject;	Write down the topic and listen
3. The main stage (195 minutes)	1. Dividing students into 2 small subgroups, asking questions on the topic; 2. Use of slides and multimedia; 3. Carries out medical work; 4. Combines all information on a given topic, actively the participating students are encouraged and appreciated;	Divided into small groups watching, participating, listening. The student expresses his opinion, complements and asks questions

Questions on the topic
What group of disease does recurrent aphthous stomatitis belong to?
Etiology of phthous stomatitis
Clinic.clinic of aphthous stomatitis
Dif - diagnosis of phthous stomatitis
Treatment and prevention of aphthous stomatitis

Tests

Severe forms of chronic recurrent aphthous stomatitis
1) Behcet's syndrome
2) Setton's aphthosis
3) Sjogren's syndrome
4) Rosenthal syndrome
5) Stevens-Johnson syndrome

Element of defeat
with chronic recurrent aphthous stomatitis
1) stain
2) papule
3) blister
4) bubble
5) crack
6) ulcer
7) afta

Affected areas in chronic recurrent aphthous stomatitis is treated with a solution
1) anesthetic
2) anesthetic and enzymes
3) anesthetic, enzymes, antiseptics
4) anesthetic, enzymes, antiseptics, keratoplasty

Causes of occurrence
chronic recurrent aphthous stomatitis
1) hypothermia
2) diseases of the gastrointestinal tract
3) transferred ARVI

4) bacterial infection
5) viral infection

Activities carried out during the period of remission
chronic recurrent aphthous stomatitis
1) antibiotic therapy
2) sanitation of the oral cavity
3) training in oral hygiene
4) rinse with chlorhexidine
5) examination by a gastroenterologist
6) identification and elimination of foci of chronic infection
7) examination by an allergist
Specify the correct sequence

Treatment of chronic recurrent aphthous stomatitis
1) keratoplasty
2) antiseptics
3) proteolytic enzymes
4) trimecaine solution

Chronic recurrent aphthous stomatitis is differentiated
1) with acute herpes
2) with chronic herpes
3) with secondary syphilis
4) with Behcet's syndrome
5) with ulcerative necrotizing stomatitis
6) with exudative erythema multiforme
7) with traumatic erosion
General management of chronic recurrent aphthous stomatitis
1) antibiotics
2) antihistamines
3) hormonal drugs
4) sulfonamides
5) multivitamins
6) agents that increase the body's resistance
7) sedatives

Medicinal substances used in treatment chronic recurrent aphthous stomatitis
1) prednisone tablets
2) sodium thiosulfate
3) tetracycline tablets
4) undevite
5) magnesium sulfate tablets
6) suprastin tablets
7) histaglobulin
8) acetylsalicylic acid
9) sulfadimethoxine

Local remedies
chronic recurrent aphthous stomatitis
1) aerosol of lidocaine
2) bonafton ointment
3) hydrogen peroxide
4) potassium permanganate solution
5) keratoplasty
6) streptocide ointment

Disease Clinical picture
1) chronic a) on the mucous membrane, multiple
recurrent erosion, aphthae prone to fusion
aphthous stomatitis b) erosion with scalloped edges,
2) acute herpetic often on the hard palate, gums,
stomatitis of the red border of the lips
c) on the oral mucosa
1-3 aft
d) long-term non-healing single
ulcers prone to scarring

Disease Clinical picture
1) chronic a) on the mucous membrane, multiple
recurrent herpes erosion, aphthae prone to fusion
2) Setton's aphthous disease b) erosion with scalloped edges,
more often on the hard palate, gums
red border of lips
c) on the oral mucosa
1-3 aft
d) long-term non-healing single

ulcers prone to scarring

Local factors provoking a relapse of aphthous stomatitis
1) injection
2) deep incisal overlap
3) trauma
4) galvanosis
5) amalgam fillings

Interactive method
USING THE HANDLE IN THE MIDDLE TABLE METHOD

All students of the group are divided by lot into 3 subgroups of 3 students each. Each subgroup sits down at a separate table, prepares a blank sheet of paper and a pen. The date, group number, last name and first name of the student are written on the sheet. The task is offered, to answer one question for the whole subgroup. Each student writes down his last name and one answer option on a sheet and passes the sheet to a neighbor, and moves his pen to the middle of the table. The teacher supervises the work of the group and everyone's participation in it. The general correct version is written down in a notebook. Students who gave the correct answer options receive the maximum score - 100% of the theoretical part rating - 0.8b. Second place students - 85.9% of the rating. Third place - 70.9% of the rating. Those who did not answer or answered incorrectly 30% of the rating. The resulting score is taken into account when assigning marks for the current lesson. The works of students are saved by the teacher.

Text

Recurrent aphthous stomatitis (stomatitis aphtosa recidiva) is a chronic inflammatory disease of the oral mucosa, characterized by a recurrent rash of aphthous stomatitis and ulcers, a prolonged course with recurrent exacerbations.

Recurrent aphthous stomatitis is one of the most common diseases of the oral mucosa.

The prevalence of recurrent aphthous stomatitis, according to different authors, ranges from 10 to 40% in different age groups of the population. Over the past 10 years, there has been a pronounced tendency towards an increase in the number of patients with recurrent aphthous stomatitis, and with its severe form.

Etiology and pathogenesis. Most researchers studying the etiology and pathogenesis of recurrent aphthous stomatitis are inclined to the leading role of the immune system in the pathogenesis of this disease.

Recurrent aphthous stomatitis is a disease characterized by decreased immunological reactivity and impaired nonspecific defense. The reason for the development of which are foci of chronic infection in the body (angina, chronic tonsillitis, pharyngitis, diseases of the gastrointestinal tract, etc.), as well as the influence of a number of adverse factors (chronic stressful situations, frequent changes in climatic zones, occupational hazards, etc.).

In patients with recurrent aphthous stomatitis, disorders of the immune status and nonspecific protection were revealed: depression of the T-system of the immune system was established, which manifests itself in a decrease in the number and functional activity of T-lymphocytes. Disorders of the T-lymphocyte subpopulation, a marked decrease in the number of T-helpers and an increase in T-suppressors were

revealed. Changes in the B-system of immunity were noted, which is manifested by an increase in the number of B-lymphocytes, an increase in the level of serum immunoglobulin G, circulating immune complexes, against the background of a decrease in the content of immunoglobulin M.

With recurrent aphthous stomatitis, the indices of nonspecific humoral and cellular defense change (a decrease in the concentration of lysozyme and an increase in beta-lysines in the blood serum, a decrease in the content of complement fractions C3, C4 and an increase in C5 fractions). Against the background of the established weakening of the phagocytic activity of i leukocytes to the majority of the studied microbial allergens, its enhancement to S. salivarius and C. albicans was noted.

Violation of local factors of protection of the oral cavity in patients with recurrent aphthous stomatitis is characterized by a decrease in the concentration of lysozyme, an increase in beta-lysines, as well as a decrease in the content of secretory and serum immunoglobulin A in the oral cavity.

liquids. As a result, the protection of the oral mucosa from the effects of microorganisms is impaired, and the number and species composition of the resident microflora also changes. As a result, the number of microbial associations in the oral cavity increases and their virulence increases.

Microbial associations of the oral mucosa in patients with recurrent aphthous stomatitis are mainly represented by coccal flora, in which coagulase-negative staphylococcus and anaerobic cocci (peptococci, peptostreptococci) occupy a significant place, and the number of anaerobic cocci increases with an increase in the number of microbial associations. The microbial associations contain a significant number of bacteroids, the content of which increases with the growth of associations. With an increase in the virulence of microorganisms, the bacterial sensitization of the body of patients with recurrent aphthous stomatitis increases - a chain of immunological reactions of an immediate and delayed type is turned on, causing frequent relapses of the disease.

In the pathogenesis of recurrent aphthous stomatitis, an essential role is played by the so-called cross-immune reaction, which acts according to the following principle. On the surface of the oral mucosa of patients with recurrent aphthous stomatitis, there is a large number of streptococci (52.9%), among them Streptococcus mutans, sanquis, salivarius, mitis, which have antigenic similarity to the cells of the oral mucosa. It has been established [Knolle, 1970] that the oral mucosa is capable of largely depositing antigen. In patients with recurrent aphthous stomatitis, a violation of the recognition of target cells by T-lymphocytes is partly genetically determined, on the one hand, and along with this there is a diverse antigenic spectrum on the surface of the oral mucosa, on the other. As a result, the mechanism of antibody-dependent cytotoxicity is activated, which, according to some authors, is the cause of this disease. This mechanism may well explain the formation of aft as a result of the manifestation of the Arthus phenomenon, as well as the importance of gastrointestinal diseases and foci of chronic infection, accompanied by an imbalance between the macroorganism and bacterial flora in the origin of recurrent aphthous stomatitis.

The clinical picture. There are two clinical forms of recurrent aphthous lesions of the oral mucosa: mild and severe (recurrent deep

Rice. 11.32. Recurrent aphthous stomatitis. Lightweight form. Aphthae on the tip of the tongue with a corolla of hyperemia around.

cicatricial aphthae), which have their own specific clinical characteristics. The symptom complex, which determines the severity of the course of recurrent aphthous stomatitis, includes the frequency of relapses of the disease and, accordingly, the duration of its remission, the nature and number of lesions on the oral mucosa and the period of their epithelization.

Recurrent aphthous stomatitis can be one of the symptoms of generalized aphthous disease, in which the genital and intestinal areas are affected. In addition to recurrent aphthous rashes on the oral mucosa, eye lesions and sometimes pyoderma occur.

RECURRENT AFTHOUS STOMATITIS (LIGHT FORM). Common aphthae on the oral mucosa are very characteristic. The process of the formation of aphthae on the oral mucosa begins with the appearance of a small spot up to 1 cm in diameter, hyperemic, sharply delimited, round or oval, painful, which after a few hours slightly rises above the surrounding mucous membrane. After a few more hours, the element erodes and becomes covered with a fibrinous grayish-white, densely seated plaque. Such a fibro-necrotic focus is often surrounded by a thin hyperemic rim (Fig. 11.32). Aphtha is very painful when touched, soft

kaya to the touch. At the base of the aphtha, infiltration occurs, as a result of which the aphthae slightly rises above the surrounding tissues, the necrotic masses on its surface form a grayish fibrinous film. Aphtha is surrounded by a sharply demarcated, brightly hyperemic, slightly edematous rim. It is sharply painful and is often accompanied by lymphadenitis. After 2-4 days the necrotic masses are rejected, and after 2-3 days the aphthae is usually resolved; for some time, congestive hyperemia persists in its place. Often a few days before the onset of aphthia, patients feel a burning sensation or pain at the site of future changes. With a mild form of recurrent aphthous stomatitis, one or two aphthae occur simultaneously, rarely more. A feature of the disease is the recurrent nature of the rash. The frequency of the appearance of aphthous stomatitis in recurrent aphthous stomatitis varies from several days to months. Rashes are more often localized on the mucous membrane of the cheeks, lips, tip and lateral surfaces of the tongue, but they can occur anywhere on the mucous membrane of the mouth.

A mild form of recurrent aphthous stomatitis, as a rule, is invisible to the patient, since at first it proceeds with rather poorly expressed clinical symptoms. In 50% of patients with this form of recurrent aphthous stomatitis, exacerbations occur 1-2 times a year, as a result of which they rarely go to the doctor. The other half of patients with recurrent aphthous stomatitis of a mild form seek help from a doctor more often, since an exacerbation of the disease occurs 5-6 times a year and the severity of its course is aggravated.

Factors provoking an exacerbation are trauma to the oral mucosa, stress, overwork, previous viral infection, premenstrual period, etc. In some cases, patients cannot associate the occurrence of an exacerbation with any specific factor.

Clinical observations have shown that during the first three years of existence, recurrent aphthous stomatitis proceeds mainly in a mild form. Sometimes there are cases of rapid transformation of the disease into a severe form. This is mainly typical for young people (17-20 years old). In most cases, the excess of the duration of the

existence of recurrent aphthous stomatitis for more than three years is accompanied by an aggravation of the severity of its clinical course and the transformation of a mild form into a severe one. To factors that accelerate the transition of a mild form of recurrent aphthous

severe stomatitis, includes occupational hazards, frequent changes in climatic zones, the presence of chronic diseases (angina, diseases of the gastrointestinal tract - chronic gastritis, colitis, gastric ulcer and duodenal ulcer), in some cases young age (up to 25 years).

Histological examination of normal aphthae reveals deep fibrinous-necrotic inflammation of the mucous membrane. The process begins with changes in the lamina propria of the mucous membrane and submucosa. Following the vasodilatation, small perivascular infiltration, edema of the prickly layer of the epithelium occurs, then spongiosis and the formation of microcavities. Alterative changes end with epithelial necrosis and erosion of the mucous membrane. The defect in the epithelium is filled with fibrin, which is firmly soldered to the underlying tissues.

Differential diagnosis. In appearance, aphthae are similar to:

A traumatic erosion;

And herpetic erosion;

And syphilitic papules, on the surface of which, some time after their appearance, a necrotic grayish-white plaque forms.

Herpetic erosion differs from aphthae in polycyclic outlines, not so pronounced soreness, more diffuse inflammatory reaction around; erosion in herpes is preceded by grouped vesicles. Syphilitic papules are characterized by low soreness, the presence of an infiltrate at the base, the stagnant nature of the inflammatory rim along the periphery and the presence of pale treponemas in the discharge of erosion.

RECURRENT AFTHOUS STOMATITIS (SEVERE), or recurrent deep cicatricial aphthae, or Setton aphthae. A severe form of recurrent aphthous stomatitis can have the following types of clinical course:

an element of the lesion on the mucous membrane of the mouth is aphtha, the period of its epithelialization is 14-20 days. The course of the disease is characterized by the occurrence of monthly exacerbations;

deep crater-like, sharply painful ulcers with a long period of epithelialization (25-35 days) are formed on the oral mucosa. Exacerbations of the disease occur 5-6 times a year;

on the mucous membrane of the mouth, aphthae and ulcers are found at the same time. The term of their epithelialization is 25-35 days.

During the year, the severe form of recurrent aphthous stomatitis is exacerbated 5-6 times or monthly. The course of the disease is chronic. In a number of patients, aphthae appear paroxysmal within several weeks, replacing each other or occurring simultaneously in large numbers. In other patients, single aphthae appear at different times. The course of the disease in the same patient may change over time.

In patients with a severe form of recurrent aphthous stomatitis, the general condition suffers: there is increased irritability, poor sleep, loss of appetite (in 70% of patients), in 22% of patients a neurotic status is formed due to constant pain in the oral cavity, there is often regional lymphadenitis ... The influence of seasonal factors on the occurrence of exacerbations of the disease in severe recurrent aphthous stomatitis is

very insignificant. As a rule, exacerbations of a severe form of recurrent aphthous stomatitis occur monthly and the disease becomes permanent, and with an increase in the duration of the disease, the severity of its course is aggravated.

An exacerbation of the disease usually begins with the appearance of a limited painful compaction of the mucous membrane, on which first a superficial, fibrinous plaque is formed, and then a deep crater-shaped ulcer with slight hyperemia around (Fig. 11.33). The ulcer usually increases in size. Sometimes in the beginning a superficial aphtha is formed, at the base of which an infiltrate is formed after 6-7 days, and the aphtha itself is transformed into a deep ulcer. Ulcers in severe recurrent aphthous stomatitis epithelialize extremely slowly (up to 1.5-2 months). After their healing, coarse connective tissue scars remain, leading to deformation of the oral mucosa. When such ulcers are located in the corners of the mouth, deformities can occur, sometimes leading to a microstoma. The duration of the existence of scarring aft varies from 2 weeks to 2 months. Rashes are more often located on the lateral surfaces of the tongue, the mucous membrane of the lips and cheeks, accompanied by severe pain.

Diagnostics. Histologically, with deep recurrent aphthiasis, a site of necrosis is determined with complete destruction of the epithelium and basement membrane, as well as

Rice. 11.33. Recurrent aphthous stomatitis. Severe form.

Ulcer with raised infiltrated edges, the bottom is covered

fibrinous plaque.

palpation in the lamina propria and submucosa. Often the lesions have salivary glands with pronounced periglandular infiltration, which gave Sutton reason to call this disease recurrent necrotizing periadenitis of the mucous membrane. However, A. L. Mashkillayson observed deep cicatricial aphthae and without the phenomena of periadenitis. Differential diagnosis. The severe form of recurrent aphthous stomatitis is differentiated from:

^ traumatic erosion;

^ traumatic ulcers;

A recurrent herpes;

^ Behcet's disease;

And Vincent's ulcerative necrotizing stomatitis;

And ulcers with specific infections (syphilis, tuberculosis);

And malignant ulcers.

With Behcet's disease, combined aphthous and ulcerative lesions of the mucous membrane of the mouth, eyes and genitals are observed.

With ulcerative necrotizing stomatitis of Vincent, an abundance of fusobacteria and spirochetes is found in the scraping of ulcers.

In a malignant ulcer, the edges are dense, slightly painful, often there is a chronic injury. Cytological examination reveals atypical cells.

Treatment. Treatment for recurrent aphthous stomatitis is effective only in the case of profound clinical

immunological examination of patients, which allows, on the basis of the data obtained, to select the appropriate individual complex pathogenetic therapy. Before starting treatment, the patient should be examined by a therapist, otorhinolaryngologist

and other specialists in order to identify concomitant diseases, primarily the gastrointestinal tract, foci of chronic infection, and the state of the immune system. Particular attention should be paid to the detection and treatment of dental and periodontal diseases.

All these measures make it possible to individualize the process of complex pathogenetic treatment of patients with recurrent aphthous stomatitis.

Complex pathogenetic treatment of patients with recurrent aphthous stomatitis includes the use of immunocorrective agents, metabolic correction drugs with obligatory sanitation of foci of chronic infection.

For immunocorrection, thymogen is used, which has a regulating effect on the reactions of cellular and humoral immunity, as well as on the factors of nonspecific resistance of the organism. Thymogen is injected intramuscularly at 100 mcg daily for 10 days. Necessarily under the control of the immunogram before and after treatment.

Levamisole (decaris) is also used to treat recurrent aphthous stomatitis. The drug is prescribed 2 days a week (in a row or at intervals of 3-4 days, 150 mg at a time or 50 mg 3 times a day). Treatment is carried out for 1.5-2 months under the control of the clinical formula of peripheral blood and the general condition of the patient.

In order to normalize the cellular metabolism of lymphocytes, patients with recurrent aphthous stomatitis are prescribed metabolic drugs that stimulate metabolic processes at the mitochondrial level. The selection of drugs and the duration of metabolic therapy is determined by cytochemical indicators of the enzyme status of blood lymphocytes (activity of mitochondrial and succinate dehydrogenase, alpha-glycerophosphate dehydrogenase).

Patients are prescribed two complexes of metabolic drugs. The first complex is aimed at improving the energy processes in lymphocytes. It is prescribed for 10 days: calcium pantothenate (2 ml of a 20% solution intramuscularly, or orally, 0.1 g 4 times a

day), riboflavin mononucleotide (1 ml of 1% solution intramuscularly); lipamide (0.025 g 3 times a day after meals); cocarboxylase (0.05 g intramuscularly); potassium orotate (0.5 g 3 times a day 1 hour before meals).

Over the next 10 days, a second complex of metabolic drugs is prescribed: vitamin B | 2 (1 ml of a 0.01% solution intramuscularly); folic acid (0.005 g 3 times a day); pyridoxal phosphate (0.02 g 3 times a day after meals); methylmethionine sulfonium chloride (0.1 g 3 times a day after meals); calcium pangamate (0.05 g 3-4 times a day); potassium orotate (0.5 g 3 times a day 1 hour before meals).

The sequence of the introduction of the complexes is determined by the indicators of cytochemical analysis, but, as a rule, the first complex is introduced first, which optimizes the energetics of the cells, then the second, which requires the energetic readiness of tissues for its utilization.

To achieve a state of stable clinical remission of recurrent aphthous stomatitis, characterized by the normalization of indicators of the cytochemical status of blood lymphocytes, it is necessary to carry out 4-6 courses of metabolic correction at intervals of 6 months, regardless of the characteristics of the clinical course of the disease. It should be especially noted the need for metabolic therapy in the spring, when pronounced phenomena of hypovitaminosis in the body are characteristic.

Hypovitaminosis, as a rule, leads to severe exacerbations of recurrent aphthous stomatitis.

In some cases, the complex of therapeutic measures includes sedatives (valerian root, "small" tranquilizers).

Special attention should be paid to measures to eliminate foci of chronic infection in the body of patients with recurrent aphthous stomatitis, the treatment of which must necessarily be carried out along with metabolic correction (treatment of chronic diseases of the ENT organs, gastrointestinal tract, etc.).

The presence of foci of chroniosepsis in patients with recurrent aphthous stomatitis causes constant bacterial sensitization, including lymphocyte sensitization, which leads to frequent exacerbations of the disease.

In order to stimulate the factors of specific and nonspecific protection in patients with recurrent aphthous stomatitis, modern methods of physical influence on the body (percutaneous electric

troneurostimulation, laser therapy for reflexogenic zones, aeroionic massage for the affected oral mucosa).

The complex of therapeutic measures, including immunocorrective, metabolic and reflexotherapy, contributes to the rapid elimination of exacerbation of the disease, significantly lengthens the periods of its remission, eliminates cellular and tissue hypoxia and normalizes immunological parameters in patients with recurrent aphthous stomatitis. A positive therapeutic effect can also be obtained from the use of each of the previously mentioned methods of treatment separately, but its effectiveness is less pronounced. Therefore, it is recommended to apply these methods of treatment of recurrent aphthous stomatitis in combination.

Diet plays an important role in the successful treatment of recurrent aphthous stomatitis. Patients are prohibited from eating spicy, spicy foods, alcoholic beverages, and smoking.

Local treatment is reduced to the sanitation of the oral cavity, the elimination of traumatic factors and foci of chronic infection. During the period of exacerbation of the disease, pain relievers are used, since aphthae and especially ulcers cause pain. For the purpose of anesthesia, anesthetics are used (1-2% lidocaine solution, 1-2% trimecaine solution, 1-2% pyromecaine solution or 5% pyromecaine ointment). For anesthesia, a 5 or 10% suspension of anesthesin in glycerin or liquid oils (peach, apricot, sunflower) is also used.

Effectively combined effect of medicinal and physiotherapeutic methods of anesthesia (microelectrophoresis on the area of lesion of the oral mucosa with a 2% solution of novocaine followed by aeroionic massage; laser irradiation).

Fibrinous and necrotic plaque from the surface of aphtha and ulcers is removed using proteolytic enzymes (trypsin, chymotrypsin, chymopsin, lysoamidase). Antiseptic treatment is carried out with antiseptic solutions (1% etonium solution, 0.02-0.06% chlorhexidine solution, 0.02% furacilin solution, etc.).

To stimulate the epithelialization of the affected oral mucosa in recurrent aphthous stomatitis, it is advisable to prescribe an oil solution of vitamins A, E, carotolin, 5% linethol ointment, solcoseryl ointment and jelly, 5% ointment and 20% actovegin jelly, etc.

Applications and irrigation of the oral mucosa are carried out 3-4 times a day after meals.

A good therapeutic effect is provided by the use of biopolymer soluble films containing various drugs. To stimulate the regeneration of the oral mucosa, an oblekol film containing sea buckthorn oil is successfully used.

Forecast. With recurrent aphthous stomatitis, the prognosis is favorable, especially in the case of early diagnosis and treatment of its mild form.

Prevention. It consists mainly in the timely identification and elimination of foci of chronic infection, including in the oral cavity, early diagnosis and treatment of chronic diseases of the gastrointestinal tract, nervous and endocrine systems, etc. Systematic oral care and regular sanitation are important. It is necessary to strictly adhere to the regime of work and rest, active physical education, hardening, rational, balanced nutrition.

Evaluation Criteria for Monitoring

№	Progress in% and points	Grade	Student knowledge level
1.	96-100	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Shows high activity, creative approach when conducting interactive games. Correctly solves situational problems and test questions. Analyzes independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
2.	91-95	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Analyzes research results independently. Understands the

			essence of the issue. Knows, tells confidently. Has an accurate idea.
3.	86-90	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
4.	81-85	Good "4"	The correct answer to questions about the clinic, diagnosis and treatment of TBI, but there are 2-3 inaccuracies, errors. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
5.	76-80	Good "4"	Correct, but incomplete coverage of the issue. The student knows questions about the clinic and diagnosis of TBI, but is not fully versed in determining the tactics of treatment. Correctly solves situational problems, but the rationale for the answer is not complete. Understands the essence of the issue. Knows, speaks confidently
6.	71-75	Good "4"	Correct, but incomplete coverage of the issue. Correctly solves situational problems, but the rationale for the answer is incomplete. Has 2-3 errors. Knows, tells confidently. Has accurate ideas.
7.	66-70	Satisfactorily "3"	Understands the essence of the issue. Correctly solves situational problems, but cannot substantiate the answer. Has accurate ideas on specific issues.
8.	61-65	Satisfactorily "3"	Has an incomplete understanding of the topic. Makes mistakes when solving situational tasks. Tells not confidently.

9.	55-60	Satisfactorily "3"	Knows, tells not confidently. Has a partial view.
10.	54 and below	Unsatisfactory "2"	Has no exact idea. Does not know.

15- Practical lesson
Topic: Changes in the oral mucosa with exogenous intoxication.

Technological map of the practical lesson

Stages of work	Teacher	Student
1. Stages of preparation (10 minutes)	1. Controls the purity of the audience; 2. Checks students' readiness for classes; 3. Controls attendance;	Listen
2.The introductory stage to classes (15 minutes)	1.Preparation on the topic of content; 2. Preparation of slides for the introductory report; 3. To develop the literature used for the study of the subject;	Write down the topic and listen
3. The main stage (195 minutes)	1. Dividing students into 2 small subgroups, asking questions on the topic; 2. Use of slides and multimedia; 3. Carries out medical work; 4. Combines all information on a given topic, actively the participating students are encouraged and appreciated;	Divided into small groups watching, participating, listening. The student expresses his opinion, complements and asks questions

Questions on the topic
What group does exogenous intoxication of SOPRA belong to?
Etiology of exogenous intoxication of SOPRA
Clinic exogenous intoxication SOPRA
Dif - diagnostics of exogenous intoxication SOPRA
Treatment and prevention of exogenous intoxication SOPRA

Interactive method
USING THE HANDLE IN THE MIDDLE TABLE METHOD

All students of the group are divided by lot into 3 subgroups of 3 students each. Each subgroup sits down at a separate table, prepares a blank sheet of paper and a pen. The date, group number, last name and first name of the student are written on the sheet. The task is offered, to answer one question for the whole subgroup. Each student writes down his last name and one answer option on a sheet and passes the sheet to a

neighbor, and moves his pen to the middle of the table. The teacher supervises the work of the group and everyone's participation in it. The general correct version is written down in a notebook. Students who gave the correct answer options receive the maximum score - 100% of the theoretical part rating - 0.8b. Second place students - 85.9% of the rating. Third place - 70.9% of the rating. Those who did not answer or answered incorrectly 30% of the rating. The resulting score is taken into account when assigning marks for the current lesson. The works of students are saved by the teacher.

Text

CHANGES IN THE MUCOSA OF THE ORAL WITH EXOGENIC INTOXICATIONS

Lesions of the oral mucosa can be caused by unfavorable factors of a person's professional activity. The influence of relatively low, but long-acting concentrations of harmful chemicals can cause the development of chronic intoxication, often with specific symptoms on the oral mucosa. In the development of chronic intoxication, the property of a substance to accumulate in the body with the formation of depots in bones, parenchymal organs, subcutaneous tissue, etc. is very essential, which is especially characteristic of heavy metals. Workers in contact with metals such as lead, mercury, bismuth, etc., over time, may develop chronic intoxication, which is manifested by a clinical picture characteristic of each metal in the oral cavity.

Similar changes in the oral cavity can also occur as a result of long-term use of drugs for the treatment of chronic diseases: bismuth (bioquinol, pentabismol, bismoverol), mercury, gold (crisanol, auranofin). The severity of the toxic effect of a drug is determined by its dose and concentration in the body. Heavy metal salts are partially secreted by the salivary glands, so symptoms of intoxication often appear in the mouth. Each metal causes specific clinical symptoms of intoxication.

BISMUTE INTOXICATION. It develops mainly in persons who have been using bismuth preparations for a long time for therapeutic purposes. They are used to treat syphilis, non-syphilitic lesions of the central nervous system (meningomyelitis, arachnoencephalitis).

The clinical picture. Bismuth intoxication is manifested by general disorders in the body (nephropathy, lesions of the gastrointestinal tract, nervous system: weakness, apathy) and lesions of the oral mucosa. A mild degree of intoxication is accompanied by catarrhal inflammation of the oral mucosa. In severe cases, erosive-ulcerative gingivostomatitis develops with hypersalivation and fetid odor from the mouth.

As a result of long-term use of bismuth preparations for the treatment of chronic diseases, bismuth particles accumulate in the mucous membrane of the gums, as a result of which a characteristic bluish-gray border is formed along the gingival margin. In some cases, bismuth intoxication is also accompanied by the formation of dark, almost black pigmentation in other areas of the oral mucosa (tongue, cheeks, lips, palate, tonsils). Sometimes pigmentation looks like black circles around the openings of the excretory ducts of the salivary glands. Bismuth fringe is a consequence of the formation of bismuth sulfide when it is excreted with saliva. With severe intoxication, deep ulceration appears, surrounded by a bluish-gray border. Ulcerative

gingivostomatitis, accompanied by painful sensations, rapidly develops in the unsanded oral cavity in the presence of dental plaque and decayed teeth.

Treatment. First of all, it is necessary to stop the further intake of bismuth preparations into the body. A complex of active detoxifying and symptomatic general therapy is carried out. Locally, the affected oral mucosa is treated with proteolytic enzymes, antiseptics and agents that stimulate epithelialization.

LEAD INTOXICATION. It develops as a result of exposure to harmful occupational factors in people working in metallurgy, battery production, typographers, workers in the chemical and mining industries, etc. Intoxication is caused by inhalation of lead vapors and dust, which accumulates in the body, mainly in bone tissue. The degree of toxic effect depends on the concentration of lead and the duration of contact with it.

The clinical picture. An early symptom of lead intoxication is a pale gray complexion (lead coloration) caused by lead damage to the walls of blood vessels and their spasm. One of the clinical manifestations of severe lead intoxication is "lead-

howling colic ". Perhaps the development of peripheral paralysis, blindness associated with damage to the central nervous system. Weakness, apathy, indigestion, kidney damage (nephropathy) are noted. Anemia, thrombocytopenia, monocytosis are detected in the blood.

Characteristic symptoms in the mouth are a sweet metallic taste and odor from the mouth - "leaden breath", as well as a "lead border" (halo saturninus), which is often the first sign of saturnism in the mouth. The border is located around the necks of the anterior teeth along the gingival margin, mainly from the vestibular surface and has a grayish-black color. A close examination shows that it is not uniform, like a bismuth border, but is composed of small grains. Lead deposits in the form of black and blue spots can simultaneously appear on the gums, cheeks, tongue, lips, palate. In severe cases, ulcerative stomatitis may develop.

The defeat of the oral mucosa in some cases is combined with skin lesions. Erythematous or bullous dermatitis occurs.

Treatment. Stop further contact with lead. According to indications, general detoxification, oxygen and symptomatic therapy is carried out. Prescribe vitamin preparations (C, B, B2, B6, B12), as well as food containing a lot of calcium and phosphorus. Local treatment includes the removal of dental plaque, necrotic areas of the gums using proteolytic enzymes, antiseptics and keratoplastic preparations (oil solutions of vitamins A, E, rosehip oil, sea buckthorn, etc.).

Prevention. The prevention of lead intoxication mainly envisages compliance with the maximum permissible concentrations (MPC) of lead in the air of working rooms that are safe for humans. To prevent the toxic effects of lead on the tissues and organs of the oral cavity, it is necessary to use respirators, rinse with a 1% solution of acetic acid, irrigate the oral cavity with solutions saturated with oxygen, vitamins using special installations.

MERCURY INTOXICATIONS. May occur as a result of exposure to harmful occupational factors among workers of mercury production. In this case, mercury and its compounds enter the human body mainly through the respiratory tract in the form of vapors and dust, and partly through the gastrointestinal tract and skin.

Along with this, chronic mercury intoxication can occur with prolonged use of mercury-containing drugs (merkuzal, novurit, mercuric chloride, calomel, mercury-

containing ointments). When mercury-containing preparations are used locally, mercury interacts with tissue proteins with the formation of albuminate. In this case, the astringent or irritating effect of the drugs is manifested. In the presence of sodium chloride and salts of other halogens, the dissolution and penetration depth of mercury compounds increase.

The clinical picture. In the case of intoxication with mercury vapor, the most pronounced symptoms of respiratory tract damage (runny nose, tracheobronchitis, toxic pneumonia). At the same time, damage to the gastrointestinal tract and the central nervous system occurs. Drowsiness appears, alternating with periods of excitement, hand tremors ("mercury tremor"). Oral and topical administration of mercury preparations affects the gastrointestinal tract to a greater extent (sharp abdominal pain, vomiting, diarrhea with blood).

Clinical manifestations of mercury intoxication in the oral cavity begin with catarrhal lesions of the gums. Mercury gingivitis develops, characterized by swelling, soreness, bleeding of the gums and the appearance of an ulcerative-necrotic process. As a result of the reaction of mercury with hydrogen sulfide with the formation of mercury sulfide, a characteristic black border appears along the edge of the gums. In severe cases, the process can spread to the mucous membrane of the cheeks, tongue and tonsils, where areas of necrosis are formed, surrounded by a hyperemic border. Fetid odor from the mouth appears, salivation increases. Due to the swelling of the tongue and the mucous membrane of the cheeks, teeth imprints are clearly visible on them. Cervical lymph nodes are enlarged, mobile, painful. Patients complain of a metallic taste in the mouth, throbbing pain in the gums, headache, general malaise, fever. Difficulty speaking and swallowing, painful chewing.

Treatment. First of all, it is necessary to stop the further intake of mercury into the body and carry out therapeutic measures aimed at its elimination. For this, methods of active detoxification of the body are used (gastric lavage, forced diuresis, hemodialysis). 5% unithiol solution, 30% sodium thiosulfate solution, vitamins (B ,, B6, B12), etc. are introduced.

Topical treatment includes antiseptic treatment

oral cavity, removal of dental plaque. In the presence of ulcerative lesions, applications of proteolytic enzymes and keratoplasty are used.

Prevention. The prevention of mercury intoxication consists in strict adherence to the rules for the use and storage of mercury at work and especially at home. The rooms in which work with mercury is carried out must have powerful ventilation, fume hoods. A sanitary and hygienic control of the room air for the content of mercury and its compounds is required. A very important factor in the prevention of mercury intoxication is health education.

GOLD PREPARATIONS. These drugs (chrysanol, auronofin) are used in the treatment of rheumatoid arthritis and lupus erythematosus as a means of stimulating cellular immune responses and inhibiting humoral immunity. Gold preparations accumulate mainly in the lungs and kidneys. It is fairly well excreted in the urine. With an overdose of gold preparations, the development of nephropathy, dermatitis, stomatitis, anemia is possible.

Changes in the oral mucosa with an overdose of gold preparations are characterized by hypersalivation, the appearance of erythematous edema (more often on the tongue).

A dense whitish plaque is formed in the form of mother-of-pearl plaques, reminiscent of the clinical picture of lichen planus.

Evaluation Criteria for Monitoring

№	Progress in% and points	Grade	Student knowledge level
1.	96-100	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Shows high activity, creative approach when conducting interactive games. Correctly solves situational problems and test questions. Analyzes independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
2.	91-95	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Analyzes research results independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
3.	86-90	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
4.	81-85	Good "4"	The correct answer to questions about the clinic, diagnosis and treatment of TBI, but there are 2-3 inaccuracies, errors. Thinks creatively. Solves situational tasks correctly. Understands the

			essence of the issue. Knows, tells confidently. Has an accurate idea.
5.	76-80	Good "4"	Correct, but incomplete coverage of the issue. The student knows questions about the clinic and diagnosis of TBI, but is not fully versed in determining the tactics of treatment. Correctly solves situational problems, but the rationale for the answer is not complete. Understands the essence of the issue. Knows, speaks confidently
6.	71-75	Good "4"	Correct, but incomplete coverage of the issue. Correctly solves situational problems, but the rationale for the answer is incomplete. Has 2-3 errors. Knows, tells confidently. Has accurate ideas.
7.	66-70	Satisfactorily "3"	Understands the essence of the issue. Correctly solves situational problems, but cannot substantiate the answer. Has accurate ideas on specific issues.
8.	61-65	Satisfactorily "3"	Has an incomplete understanding of the topic. Makes mistakes when solving situational tasks. Tells not confidently.
9.	55-60	Satisfactorily "3"	Knows, tells not confidently. Has a partial view.
10.	54 and below	Unsatisfactory "2"	Has no exact idea. Does not know.

16- Practical lesson
Topic: Change in SPR in hypo and avitaminosis
Technological map of the practical lesson

Stages of work	Teacher	Student
1. Stages of preparation (10 minutes)	1. Controls the purity of the audience; 2. Checks students' readiness for classes; 3. Controls attendance;	Listen
2.The introductory stage to classes (15 minutes)	1.Preparation on the topic of content; 2. Preparation of slides for the introductory report; 3. To develop the literature used for the	Write down the topic and listen

	study of the subject;	
3. The main stage (195 minutes)	1. Dividing students into 2 small subgroups, asking questions on the topic; 2. Use of slides and multimedia; 3. Carries out medical work; 4. Combines all information on a given topic, actively the participating students are encouraged and appreciated;	Divided into small groups watching, participating, listening. The student expresses his opinion, complements and asks questions

Questions on the topic
The concept of vitamin B1
The clinical picture with avitaminosis B1
Give the concept of vitamin B2
Clinical picture with vitamin B2 deficiency

Tests

51. What vitamin causes regulation of differentiation of epithelial cells and the process of keratinization.
A) vit A
B) vit B
B) vit PP
D) vit D

52. When what vitamin is deficient, the epithelium changes - atrophy and keratinization
A) vit B
B) vit C
B) vit A
D) vit PP

53. At what hypovitaminosis is there dryness of the mucous membrane and skin?
A) vit PP
B) vit B2
B) vit A
D) vit C

54. Daily dose in the treatment of hypovitaminosis A
A) 50,000-100,000 IU
B) 600000-700000 IU
B) 10-100 IU
D) 20-40 IU

55. At what hypovitaminosis the tongue is enlarged due to edema
A) vit PP
B) vit B2
C) vit B1
D) vit C

56. In case of hypovitaminosis of which vitamin, symptoms of a disorder of the nervous system are observed
A) ascorbic acid
B) pyridoxine
C) nicotinic acid

D) riboflavin

57. What vitamin is used in dentistry as a means of accelerating the epithelialization of ulcer erosions reduces dryness and normalizes processes in the epithelium
A) retinol
B) ascorbic acid
C) pyridoxine
D) nicotinic acid

58. With a lack of which vitamin develops Addison-Birmer anemia
A) B12
B) A
B) PP
D) E

Interactive method

USING THE WEB METHOD
Steps:
1. In advance, students are given time to prepare questions for the lesson.
2. Participants sit in a circle.
3. One of the participants is given a skein of thread, and he asks his prepared question (to which he himself must know the full answer), holding the end of the thread and throwing the skein to any student.
4. The student who received the skein answers the question (at the same time, the person who asked him comments on the answer) and passes the question relay on. Participants continue to ask and answer questions until everyone is on the web.
5. As soon as all students finish asking questions, the student holding the skein returns it to the participant from whom he received the question, at the same time asking his own question, etc., until the ball is completely "unwound".

Text
Vitamins are biologically active organic substances, which, along with proteins, lipids and carbohydrates, are necessary for the normal functioning of the human body. Vitamins are biological catalysts that interact with enzymes and hormones. They are part of many enzymes as cofactors or are involved in their synthesis. The absence or deficiency of any vitamin leads to a disruption in the formation of enzymes and hormones that regulate metabolism in the body.

The mucous membrane of the mouth is very sensitive to vitamin deficiencies. With vitamin deficiency, the resistance of the oral mucosa to injury, the effects of microorganisms decreases, and the processes of its regeneration slow down.

The main source of vitamins is food

products. Moreover, not only the amount of incoming vitamins is important, but also the violation of the ratio between the qualitative composition of food and the quantitative content of vitamins in it. So, when eating food rich in carbohydrates, the need for vitamin B increases, and with a lack of protein, the absorption of vitamin B2, nicotinic and ascorbic acid, etc., decreases.

Hypovitaminosis can be primary (exogenous), due to a low content of vitamins in food, as well as with a monotonous unbalanced or insufficient nutrition. Secondary (endogenous) vitamin deficiency occurs in diseases of the gastrointestinal tract, when the absorption of nutrients, including vitamins, decreases; in violation of the assimilation of vitamins after their absorption, for example, with liver diseases,

endocrine disorders, etc. It should be especially noted cases when vitamin deficiency occurs as a result of increased consumption of vitamins by the body. This can occur under the influence of low or high ambient temperatures, with increased physical exertion, mental stress, oxygen starvation, etc. During pregnancy and breastfeeding, the need for ascorbic acid, pyridoxine, folic acid, calciferol and tocopherol increases.

Most vitamins in the process of exchange are closely related to each other. It is believed that there is practically no deficiency of one type of vitamin. In this regard, the possibility of synergism and antagonism of the action of vitamins should be pointed out. It was found that with an increase in the intake of ascorbic acid, the need for vitamin B2 decreases, and with a lack of vitamin B2 in the tissues, the level of vitamins C and B decreases. There is a clear relationship between ascorbic acid and thiamine; folic acid and vitamin B12; thiamine with riboflavin and pyridoxine; riboflavin with retinol and pantothenic acid; nicotinic acid and riboflavin. The interaction of vitamins A and D is antagonistic. Vitamin D reduces the toxic effect of vitamin A. The antagonism of niacin with thiamine, choline, pantothenic acid has been established; vitamin A with vitamin K.

INSUFFICIENCY OF RETINOL (VITAMIN A). One of the important functions of vitamin A is to regulate the differentiation of epithelial cells and keratinization processes, including the oral mucosa. The participation of vitamin A in the normalization of metabolic processes in the epithelium

Alial tissue is considered its main physiological function in the body. Moreover, retinol has the ability to restore the structural integrity of not only the epithelium, but also the connective tissue, causing thickening of collagen and elastic fibers.

The clinical picture. Deficiency of vitamin A leads to significant changes in the epithelium - atrophy and keratinization, as a result of which the protective properties of the skin and oral mucosa are significantly reduced. With hypovitaminosis A, there is dryness of the oral mucosa and skin, increased keratinization processes (hyperkeratosis), a significant violation of the activity of the sebaceous glands. Salivation decreases. On the mucous membrane, the process can be complicated by inflammation, followed by erosion and ulceration.

Histologically, with hypovitaminosis A, metaplasia of the epithelium is determined with the replacement of part of the rows of cells of the spinous layer with keratinizing cells.

Treatment. Retinol is prescribed up to 50,000-100,000 IU per day. It is necessary to use large doses of vitamin A under the supervision of a physician, since hypervitaminosis may develop, accompanied by drowsiness, lethargy, headache, facial flushing, followed by peeling of the skin. Vitamin A is prescribed in the form of a concentrate or dragee.

In dental practice, vitamin A is widely used as a means of accelerating the epithelialization of erosions and ulcers on the oral mucosa; for the treatment of hyperkeratosis (leukoplakia, lichen planus, etc.); to reduce dryness of the oral mucosa. Vitamin A is prescribed orally after meals in the form of pills or tablets, 3300 IU 1-2 times a day; an oil solution of retinol acetate 3.44% or retinol palmitate 5.5%, 8-10 drops 2 times a day, for a course of treatment 1.5-2 months. Treatment. Riboflavin is prescribed orally at 0.01 g 3 times a day for 1 - 1.5 months.

LACK OF NICOTINIC ACID (VITAMIN PP). With a lack of nicotinic acid, a person develops pellagra. Nicotinic acid is a specific antipellagric agent, and therefore it is designated as "vitamin PP", that is, it prevents pellagra. Nicotinic acid takes part in the regulation of metabolism, primarily carbohydrate. Hypo- and avitaminosis PP develops mainly with insufficient intake of it with food. In this case, a certain role is played by the insufficient content of tryptophan in food, as well as alcoholism.

The clinical picture. With hypovitaminosis PP, appetite worsens, nausea and diarrhea appear. The tongue increases, becomes edematous, its papillae at first hypertrophy. The dorsum of the tongue is usually covered with a thick layer of plaque, separated by grooves. Then the plaque is gradually rejected, the tongue in these areas becomes bright red, shiny, sharply painful. Develops hyper-

myia of the oral mucosa and erythema of the skin. At the same time, patients complain of weakness, apathy, rapid fatigability, memory impairment.

With pellagra, there are typical changes in the form of a triad: dementia, diarrhea, dermatitis. Violation of the nervous system (dementia) is expressed by a mental disorder in the form of oppression, depression. Diarrhea is characterized by persistent, debilitating diarrhea. Dermatitis is manifested by the following symptoms: the skin becomes pigmented, especially in areas exposed to sunlight (the back of the hands, neck, face), slightly hyperemic, dry, peeling, thinning. The papillae of the tongue completely atrophy, the tongue becomes varnished, shiny, bright red, painful. The mucous membrane of the mouth is edematous, hyperemic, hypertrophied, blisters may appear on it, in place of which painful erosions then form. Patients note dry mouth, taste disturbance. It has now been established that insufficient intake of vitamins B, B2 and B6 also plays a role in the development of pellagra. The lack of vitamin PP is confirmed by the results of laboratory tests. Treatment. It consists in the appointment of nicotinic acid inside (after meals) 0.1 g 2-3 times a day or parenterally for 15-20 days. At the same time they give thiamine, riboflavin and pyridoxine, as well as fortifying agents.

INSUFFICIENCY OF ASCORBIC ACID (VITAMIN C). In the absence or insufficient amount of ascorbic acid in food or its incomplete assimilation, scurvy develops (synonym: scorching, Möller-Barlow's disease). In Russia, vitamin C deficiency practically does not occur, but cases of hypovitaminosis are observed. With a lack of vitamin C, the system of redox enzymes is disrupted, significant changes occur in carbohydrate and protein metabolism, the reactivity of the body decreases (phagocytic activity of cells and the formation of antibodies decrease), collagen synthesis and maturation of connective tissue are disrupted, which causes increased vascular and tissue permeability.

In the occurrence of vitamin deficiency, along with a lack of ascorbic acid in food, a number of factors play an important role: excessive physical exertion, infectious diseases, hypothermia, tension of the nervous system, etc.

digestion. So, in the absence of free hydrochloric acid in the stomach, ascorbic acid is destroyed faster, and in the inflammatory process in the intestine, it is poorly absorbed.

The clinical picture. Changes caused by a lack of vitamin C consist of the manifestation of hemorrhagic syndrome and complications caused by a secondary infection. Lack of ascorbic acid and especially vitamin C deficiency are accompanied

by malaise, weakness, fatigue, lack of appetite, weight loss, pain in the calf muscles. The skin becomes dark due to the accumulation of melanin in it, dry, easily peels off.

As a result of a sharp increase in the permeability of capillaries and small vessels, hemorrhagic syndrome occurs: petechial hemorrhages in various parts of the oral mucosa, subcutaneous tissue (fiber), joints. Permeability increases especially sharply with a combination of ascorbic acid and rutin deficiency (vitamin P).

Deficiency of vitamin C is always manifested by changes in the oral cavity. Along with petechiae in places of mucosal injury, there are extensive hemorrhages. At the very beginning of the disease, swelling of the gingival papillae occurs, they become bluish-red, gradually increase in size, covering the crowns of the teeth, and bleed heavily. Mobility and loss of teeth are noted. In the future, in the areas of hemorrhage, an ulcerative-necrotic process develops, which is due to the addition of a secondary infection. Necrosis is more often localized in the area of the tonsils and the gingival margin.

Differential diagnosis. The enlargement of the gingival papillae in the initial stage of hypovitaminosis C must be differentiated from hypertrophic gingivitis. In the presence of punctate hemorrhages on the oral mucosa, blood diseases should be excluded, in which a similar picture can be observed. Necrotic ulcerative changes during scorbut are differentiated from necrotizing ulcerative stomatitis of Vincent, changes in the oral mucosa with leukemia. The history, the presence of other symptoms of scurvy, and the results of laboratory tests are of decisive importance in the diagnosis. Hypo- or vitamin C deficiency is confirmed by the determination of a low level of ascorbic acid in the blood.

Treatment. Assign ascorbic acid 0.1 g 4-5 times a day (after meals) or sodium ascorbate paren-

terally, 1-3 ml of a 5% solution. At the same time, it is advisable to take rutin (50-100 mg 2-3 times a day). With a pronounced symptom of bleeding, calcium chloride, vitamin K are also prescribed.

LACK OF PYRIDOXIN (VITAMIN B6). Pyridoxine is actively involved in the exchange of amino acids (tryptophan, methionine, cysteine, serine, glutamic acid, etc.).

Vitamin B6 takes part in the processes of lipid metabolism, in particular, it normalizes it in atherosclerosis.

The clinical picture. With vitamin B6 deficiency, there is a dysfunction of the central and peripheral nervous system. Lethargy, drowsiness, increased excitability, peripheral neuritis, neurodermatitis, stomalgia, glossitis, glossalgia, eczematous cheilitis, seborrheic dermatitis develop.

Treatment. In dental practice, pyridoxine is used in the complex treatment of eczematous and atopic cheilitis, herpes zoster, and lupus erythematosus.

Assign pyridoxine inside (after meals) in tablets of 0.002; 0.005 and 0.01 g 1-2 times a day. Parenterally (under the skin, intramuscularly or intravenously) is administered to adults at 0.05-0.1 g per day (in 1-2 doses). The course of treatment is 1 month.

INSUFFICIENCY OF CYANOCOBALAMIN (VITAMIN B12). Cyanocobalamin is essential for normal blood formation and maturation of red blood cells. It is a catalyst in the exchange reaction of the synthesis of nucleic acids and nucleoproteins necessary for normal hematopoiesis. The effect of vitamin B12 on hematopoiesis is carried out in pr in the absence of folic acid, which, under its influence, turns into an active form and

contributes to normal hematopoiesis. Vitamin B | 2 and folic acid are involved in the synthesis of hemoglobin.

The clinical picture. In case of B deficiency, malignant megaloblastic anemia of Addison-Birmer develops. In addition to dysfunction of hematopoiesis, hypovitaminosis B | 2 is characterized by lesions of peripheral nerves (neuritis, neuralgia, paresthesia of the tongue and oral mucosa). Early signs of developing B ,, - deficiency anemia are lesions of the tongue: burning, tingling, paresthesia of the tongue, there may be hyperemia of the tip and lateral surfaces of the tongue against the background of atrophy of its papillae - "polished" tongue (Gunther's glossitis, or Meller-Gunther's glossitis).

Treatment. For the treatment of Addison's anemia-Birmer Vita-

min B | 2 is injected intramuscularly or subcutaneously at 100-200 μg 1 time in 2 days. At the same time, folic acid is prescribed, which enhances its effect.

Cyanocobalamin is also used in the treatment of candidiasis, lichen planus, Dühring's dermatitis, lupus erythematosus, etc.

Cyanocobalamin is available in ampoules containing 30, 100, 200 and 500 mcg of the vitamin.

Joint administration (in one syringe) of solutions of vitamins B {and B6 is not recommended, since the cobalt ion contained in the cyanocooalamine molecule causes the destruction of other vitamins. INSUFFICIENCY OF TOCOPHEROL (VITAMIN E). Tocopherol is a natural antioxidant. It inhibits the process of lipid peroxidation and, accordingly, oxidation products in organs and tissues. Participates in the processes of cellular metabolism, as well as the synthesis of heme and proteins. Vitamin E normalizes the permeability and strength of the vascular wall and, first of all, the gingival capillaries. With hypovitaminosis E, the experiment revealed an increase in capillary permeability and fragility, degenerative changes in nerve cells, skeletal muscles and heart muscle, fatty liver infiltration.

In dentistry, vitamin E is used to treat periodontitis. The drug is administered intramuscularly, as well as in the form of periodontal dressings. In the treatment of diseases of the oral mucosa, vitamin E is used to accelerate its regeneration. Tocopherol acetate is combined with vitamin A for application to the area of erosions and ulcers. When administered intramuscularly tocopherol acetate is prescribed at 50-100 mg per day for 20-40 days. To do this, use a 5%, 10% and 30% solution of tocopherol acetate in oil. When taken orally, the doses are the same. Capsules of 0.1 and 0.2 ml of a 50% solution containing, respectively, 0.05 and 0.1 g of tocopherol acetate.

Evaluation Criteria for Monitoring

№	Progress in% and points	Grade	Student knowledge level
1.	96-100	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a

			decision. Thinks creatively. Shows high activity, creative approach when conducting interactive games. Correctly solves situational problems and test questions. Analyzes independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
2.	91-95	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Analyzes research results independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
3.	86-90	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
4.	81-85	Good "4"	The correct answer to questions about the clinic, diagnosis and treatment of TBI, but there are 2-3 inaccuracies, errors. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
5.	76-80	Good "4"	Correct, but incomplete coverage of the issue. The student knows questions about the clinic and diagnosis of TBI, but is not fully versed in determining the tactics of treatment. Correctly solves situational problems, but the rationale for the answer is not complete. Understands the essence of the issue. Knows,

			speaks confidently
6.	71-75	Good "4"	Correct, but incomplete coverage of the issue. Correctly solves situational problems, but the rationale for the answer is incomplete. Has 2-3 errors. Knows, tells confidently. Has accurate ideas.
7.	66-70	Satisfactorily "3"	Understands the essence of the issue. Correctly solves situational problems, but cannot substantiate the answer. Has accurate ideas on specific issues.
8.	61-65	Satisfactorily "3"	Has an incomplete understanding of the topic. Makes mistakes when solving situational tasks. Tells not confidently.
9.	55-60	Satisfactorily "3"	Knows, tells not confidently. Has a partial view.
10.	54 and below	Unsatisfactory "2"	Has no exact idea. Does not know.

17- Practical lesson
Topic: Changes in OSR in endocrine diseases. Changes in OSR in diabetes mellitus. Clinic, diagnosis and treatment

Technological map of the practical lesson

Stages of work	Teacher	Student
1. Stages of preparation (10 minutes)	1. Controls the purity of the audience; 2. Checks students' readiness for classes; 3. Controls attendance;	Listen
2.The introductory stage to classes (15 minutes)	1.Preparation on the topic of content; 2. Preparation of slides for the introductory report; 3. To develop the literature used for the study of the subject;	Write down the topic and listen
3. The main stage (195 minutes)	1. Dividing students into 2 small subgroups, asking questions on the topic; 2. Use of slides and multimedia; 3. Carries out medical work; 4. Combines all information on a given topic, actively the participating students are encouraged and appreciated;	Divided into small groups watching, participating, listening. The student expresses his opinion, complements

		and asks questions

Questions on the topic
What group does the disease include diabetes mellitus?
Etiology of endocrine diseases.
Clinic of diabetes mellitus.
Dif-diagnosis of diabetes mellitus.
Diabetes mellitus treatment.
Prevention of diabetes mellitus

Tests

11. What is the main reason for the development of fungal stomatitis in diabetes mellitus.
A) metabolic disorders in general
B) violation of the balance of acid-base balance
C) impaired absorption of proteins, carbohydrates
D) there is no right answer

12. What is paresthesia.
A) paralysis of facial muscles
B) taste disturbance
C) dry mouth
D) all answers are correct

13. As a result, Addison's disease develops.
A) hyperfunction of the adrenal cortex
B) insufficient function of the adrenal cortex
B) due to hypothyroidism
D) hyperfunction of the thyroid gland

14. What disease are we talking about:
- the patient has developed macrocheilitis, candidiasis of the oral mucosa, there are atrophic ulcers on the mucous membrane of the tongue.
A) Addison's disease
B) diabetes mellitus
C) Itsenko-Cushingo's disease
D) Mikulich's disease

15. One of the first signs of Addison's disease.
A) pigmentation of the skin and mucous membranes
B) stomatitis, seizures
C) atrophic ulcers
D) bleeding gums

16. What is paresthesia.
A) paralysis of facial muscles
B) taste disturbance
C) dry mouth
D) all answers are correct

Interactive method

USING THE HANDLE IN THE MIDDLE TABLE METHOD

All students of the group are divided by lot into 3 subgroups of 3 students each. Each subgroup sits down at a separate table, prepares a blank sheet of paper and a pen. The date, group number, last

name and first name of the student are written on the sheet. The task is offered, to answer one question for the whole subgroup. Each student writes down his last name and one answer option on a sheet and passes the sheet to a neighbor, and moves his pen to the middle of the table. The teacher supervises the work of the group and everyone's participation in it. The general correct version is written down in a notebook. Students who gave the correct answer options receive the maximum score - 100% of the theoretical part rating - 0.8b. Second place students - 85.9% of the rating. Third place - 70.9% of the rating. Those who did not answer or answered incorrectly 30% of the rating. The score received is taken into account when assigning marks for the current lesson. The works of students are saved by the teacher.

Text

Changes in the oral mucosa with endocrine diseases

The endocrine system is a regulator of metabolic processes in the human body. It ensures the consistency of the functions of individual organs and systems, and also controls the balance of a number of body functions (glucose and calcium in the blood, temperature, etc.). In some endocrine disorders, changes in the mucous membrane of the mouth and the red border of the lips are observed. Clinical manifestations in the oral cavity may necessitate the study of individual functions of the endocrine system. For example, the progressive development of periodontal disease may be associated with diabetes mellitus or other diseases of the endocrine system.

Changes in the oral cavity are most often detected in diabetes mellitus, dysfunction of the gonads, thyroid and parathyroid glands, pituitary gland, adrenal cortex. Therefore, a timely and correct assessment by the dentist of the local manifestations of these diseases in the oral cavity will contribute to their early diagnosis.

DIABETES. It is characterized by a violation of carbohydrate metabolism, against which changes occur in the oral cavity. Their severity depends on the severity and duration of diabetes mellitus. Oral manifestations are often the first sign of illness.

Xerostomia is dryness in the mouth caused by decreased salivation, the severity of which varies from patient to patient. Dryness is often combined with increased thirst and appetite, burning and tingling in the area of the tongue, lips, gums; sometimes accompanied by itching and burning in the genitals or other parts of the body. On examination, the mucous membrane of the mouth is dry, shiny, edematous and hyperemic, especially on the tongue. Along with this, paresthesias may occur.

Differentiate xerostomia against the background of diabetes mellitus from salivation disorders in disorders of the nervous system, Sjogren's syndrome, sialoadenitis and sialodochitis, Mikulich's disease. Catarrhal, erosive and ulcerative stomatitis, glossitis. Inflammation of the oral mucosa or its individual areas in diabetes mellitus occurs as a result of a decrease in its resistance due to violations of redox processes, dysbiosis in the oral cavity, imbalance of local immunity and other reasons. All this leads to a change in the reactivity of the

oral mucosa to local stimuli, a decrease in resistance to infection; there is a predisposition to inflammation, the healing processes of erosions and ulcers slow down. Even a slight effect of a traumatic factor leads to inflammation of the oral mucosa and in some cases to a violation of its integrity with the formation of cracks, erosions and ulcers. In this case, patients complain of pain that occurs when eating hot, spicy or dry food. Erosions and ulcers are characterized by a long course due to a decrease in the regenerative properties of the mucous membrane.

In diabetes mellitus, periodontal tissues are also quite often affected. First, catarrhal gingivitis develops with severe swelling and bleeding of the gums.

Subsequently, it rather quickly transforms into periodontitis, characterized by a progressive course with pronounced inflammatory and destructive changes in all periodontal tissues. Periodontal abscesses are common. Moreover, the more severe the course of diabetes mellitus and the longer its duration, the stronger the damage to the periodontal tissues. Especially fast destructive processes in the periodontium are characteristic of young patients with diabetes mellitus.

Fungal stomatitis, mycotic seizure. With diabetes mellitus, candidiasis is a fairly common form of oral pathology. Favorable conditions for its development are dysbacteriosis in the oral cavity in patients with diabetes mellitus, violation of acid-base balance with the accumulation of under-oxidized metabolic products (lactic and pyruvic acids) in the tissues. Candidiasis of the oral mucosa in diabetes mellitus is characterized by a persistent and persistent course. Mycotic seizure is common, in which cracks form in the corners of the mouth, covered with small grayish-white crusts. For the diagnosis of candidiasis, the presence of general symptoms of diabetes mellitus, laboratory data (an increase in glucose in the blood and urine), as well as the results of a bacterioscopic examination of scrapings from the surface of the oral mucosa are important.

Antifungal therapy is carried out in consultation with an endocrinologist treating diabetes mellitus.

Paresthesia, taste disturbance. In patients with diabetes mellitus, along with dryness and burning of the oral mucosa, paresthesia sometimes occurs. With the decompensated form of diabetes mellitus, some patients have a decrease in taste sensitivity to sweet, salty, sour, and in some cases to bitter. Sometimes there is a disorder of the nervous system (neuritis, neuralgia of the branches of the trigeminal nerve).

In diagnostics, data from anamnesis, including family history, are important; a picture of changes in the oral cavity and the results of laboratory tests (blood, urine).

Treatment is carried out in contact with an endocrinologist. Along with the treatment of diabetes mellitus, a thorough debridement of the oral cavity and symptomatic local therapy are carried out.

ITSENKO-CUSHING'S DISEASE. It develops after-

the result of hyperfunction of the adrenal cortex with damage to the hypothalamic-pituitary system. Increased production of steroids by the adrenal cortex leads to osteoporosis of bone tissue, including the jaws, and predisposes to periodontal disease. Some patients develop macrocheilitis.

Candidiasis is common. Trophic ulcers can form on the mucous membrane of the tongue, cheeks and other parts of the oral cavity.

Treatment. The underlying disease is treated by an endocrinologist. Local treatment includes oral debridement. Symptomatic therapy is carried out according to indications.

ADDISON'S DISEASE. It occurs as a result of insufficient function of the adrenal cortex. One of the first signs of the disease may be pigmentation of the skin and mucous membranes, caused by excessive deposition of melanin in the connective tissue and epithelial cells. Skin pigmentation appears in areas exposed to light (face, back of the hands). As the disease progresses, the skin becomes light brown or bronze in color. On the mucous membrane of the mouth in the cheeks, along the edges of the tongue, on the palate, gums, small (from one to several square millimeters) spots or stripes of grayish-black, dark brown or bluish-gray appear without signs of inflammation (Fig. 11.37). They do not stand out above the level of the mucous membrane. At the same time, patients have no subjective sensations. The general well-being of patients may suffer. Asthenia, gastrointestinal disturbances (nausea, vomiting, diarrhea, abdominal pain), headaches, memory loss, weight loss are noted.

Differential diagnosis. Addison's disease is differentiated from:

▲ congenital pigmentation of the oral mucosa, observed in some ethnic groups;

▲ intoxication with salts of heavy metals.

Treatment. The main disease is treated by an endocrinologist. No special local treatment is required.

MIXEDEM. A disease resulting from severe hypothyroidism. Mostly women are ill. The main symptom of myxedema is a widespread mucous edema of the skin and subcutaneous tissue. Edema is formed as a result of a violation of lymph outflow and the accumulation in organs and tissues of a specific mucous substance, consisting of glycosaminoglycans, which retain water and form edema.

Rice. 11.37. Pigmentation of the mucous membrane of the tongue

In a pronounced stage of the disease, the patient's face has a characteristic appearance: puffy, pale, lips, nose are thickened, upper eyelids are sharply swollen, almost close the eye slit. The tongue is significantly enlarged in size (macroglossia), teeth imprints are visible on its lateral surfaces. There is anemicity, swelling and dryness of the oral mucosa. Due to the swelling of the mucous membrane of the larynx and vocal cords, the timbre of the voice decreases; speech slows down. The general condition of patients is characterized by weakness, drowsiness, lethargy, sluggishness, loss of memory and hearing. With hypothyroidism in childhood, enamel hypoplasia, hyposalivation, high intensity of caries lesions are observed, the timing of teething is delayed; cretinism develops.

Differentiate swelling of the tongue with myxedema from its swelling with other general somatic diseases described in this section.

Treatment. General treatment is carried out by an endocrinologist and consists of thyroid replacement therapy. No specific topical treatment is indicated. The oral cavity is sanitized.

DIFFUSION TOXIC GOITER (thyrotoxicosis, Graves' disease). It develops as a result of hyperplasia and hyperfunction of the thyroid gland. Patients complain of a burning sensation of the oral mucosa, a decrease in taste sensitivity, a deterioration in general well-being

(shortness of breath, increased fatigue, irritability, sweating, etc.). On examination of the oral cavity, the mucous membrane is pale, edematous. Desquamative glossitis is sometimes detected. Characterized by accelerated teething, an anomaly in the development of enamel.

Treatment. The general treatment is carried out by an endocrinologist. Local treatment is carried out when indicated.

JUVENILE (YOUTH) GINGIVITIS. It occurs in boys and girls during puberty, with menstrual irregularities. The change in the oral mucosa occurs as a result of the action of the gonadotropic hormones of the pituitary gland. Local factors contribute to the development of juvenile gingivitis: poor oral hygiene, malocclusion, etc. The mucous membrane of the gums is edematous, hyperemic, bleeds easily. Hypertrophy of the gingival papillae, the marginal edge of the gums, develops, mainly in the area of the anterior teeth of the lower jaw.

Treatment. Systematic hygiene procedures, dental calculus removal, and oral cavity sanitation are required. Orthodontic treatment is carried out according to indications. Drug treatment of hypertrophic gingivitis is carried out according to the generally accepted method.

DIABETES. It is characterized by a violation of carbohydrate metabolism, against which changes occur in the oral cavity. Their severity depends on the severity and duration of diabetes mellitus. Oral manifestations are often the first sign of illness.

Xerostomia is dryness in the mouth caused by decreased salivation, the severity of which varies from patient to patient. Dryness is often combined with increased thirst and appetite, burning and tingling in the area of the tongue, lips, gums; sometimes accompanied by itching and burning in the genitals or other parts of the body. On examination, the mucous membrane of the mouth is dry, shiny, edematous and hyperemic, especially on the tongue. Along with this, paresthesias may occur.

Differentiate xerostomia against the background of diabetes mellitus from salivation disorders in disorders of the nervous system, Sjogren's syndrome, sialoadenitis and sialodochitis, Mikulich's disease.

Catarrhal, erosive and ulcerative stomatitis, glossitis. Inflammation of the oral mucosa or its individual areas in diabetes mellitus occurs as a result of a decrease in its resistance due to violations of redox processes, dysbiosis in the oral cavity, imbalance of local immunity and other reasons. All this leads to a change in the reactivity of the oral mucosa to local stimuli, a decrease in resistance to infection; there is a predisposition to inflammation, the healing processes of erosions and ulcers slow down. Even a slight effect of a

traumatic factor leads to inflammation of the oral mucosa and in some cases to a violation of its integrity with the formation of cracks, erosions and ulcers. In this case, patients complain of pain that occurs when eating hot, spicy or dry food. Erosions and ulcers are characterized by a long course due to a decrease in the regenerative properties of the mucous membrane.

In diabetes mellitus, periodontal tissues are also quite often affected. First, catarrhal gingivitis develops with severe swelling and bleeding of the gums.

Subsequently, it rather quickly transforms into periodontitis, characterized by a progressive course with pronounced inflammatory and destructive changes in all periodontal tissues. Periodontal abscesses are common. Moreover, the more severe the course of diabetes mellitus and the longer its duration, the stronger the damage to the periodontal tissues. Especially fast destructive processes in the periodontium are characteristic of young patients with diabetes mellitus.

Fungal stomatitis, mycotic seizure. With diabetes mellitus, candidiasis is a fairly common form of oral pathology. Favorable conditions for its development are dysbacteriosis in the oral cavity in patients with diabetes mellitus, violation of acid-base balance with the accumulation of under-oxidized metabolic products (lactic and pyruvic acids) in the tissues. Candidiasis of the oral mucosa in diabetes mellitus is characterized by a persistent and persistent course. Mycotic seizure is common, in which cracks form in the corners of the mouth, covered with small grayish-white crusts.

For the diagnosis of candidiasis, the presence of general symptoms of diabetes mellitus, laboratory data (an increase in glucose in the blood and urine), as well as the results of a bacterioscopic examination of scrapings from the surface of the oral mucosa are important. Antifungal therapy is carried out in consultation with an endocrinologist treating diabetes mellitus.

Paresthesia, taste disturbance. In patients with diabetes mellitus, along with dryness and burning of the oral mucosa, paresthesia sometimes occurs. With the decompensated form of diabetes mellitus, some patients have a decrease in taste sensitivity to sweet, salty, sour, and in some cases to bitter. Sometimes there is a disorder of the nervous system (neuritis, neuralgia of the branches of the trigeminal nerve).

In diagnostics, data from anamnesis, including family history, are important; a picture of changes in the oral cavity and the results of laboratory tests (blood, urine).

Treatment is carried out in contact with an endocrinologist. Along with the treatment of diabetes mellitus, a thorough debridement of the oral cavity and symptomatic local therapy are carried out.

Evaluation Criteria for Monitoring

№	Progress in% and points	Grade
1.	96-100	Fine "5"

					interactive games. Correctly solves situational problems and test questions. Analyzes independently. Understands the essence of the issue. Knows, tells	
			7.	66-70	confidently. Has an accurate idea.	Satisfactorily "3"
2.	91-95	Fine "5"			Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Analyzes research results independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.	
			8.	61-65		Satisfactorily "3"
			9.	55-60		Satisfactorily "3"
3.	86-90	Fine "5"	10.	54 and below		Unsatisfactory "2"
4.	81-85	Good "4"			The correct answer to questions about the clinic, diagnosis and treatment of TBI, but there are 2-3 inaccuracies. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.	
5.	76-80	Good "4"			Correct, but incomplete coverage of the issue. The student knows questions about the clinic and diagnosis of TBI, but is not fully versed in determining the tactics of treatment. Correctly solves situational problems, but the rationale for the answer is not complete. Understands the essence of the issue. Knows, speaks confidently.	
6.	71-75	Good "4"			Correct, but incomplete coverage of the issue. Correctly solves	

18. Practical lesson

Topic: Change in OAS in diseases of the gastrointestinal tract

Technological map of the practical lesson

Stages of work	Teacher
1. Stages of preparation (10 minutes)	1. Controls the purity of th 2. Checks students' readine 3. Controls attendance; 1. Preparation on the topic 2. Preparation of slides for report; 3. To develop the literatu study of the subject;
2.The introductory stage to classes (15 minutes)	
3. The main stage (195 minutes)	1. Dividing students subgroups, asking question 2. Use of slides and multim 3. Carries out medical wor 4. Combines all informa topic, actively the participating students and appreciated;

		questions

Questions on the topic
1Change of language in diseases of the g / c tract
2.Changes in the papillae of the tongue in diseases of the glandular tract
3. Violation of taste sensitivity in diseases of the g / c tract

Interactive method

USING THE HANDLE IN THE MIDDLE TABLE METHOD

All students of the group are divided by lot into 3 subgroups of 3 students each. Each subgroup sits down at a separate table, prepares a blank sheet of paper and a pen. The date, group number, last name and first name of the student are written on the sheet. The task is offered, to answer one question for the whole subgroup. Each student writes down his last name and one answer option on a sheet and passes the sheet to a neighbor, and moves his pen to the middle of the table. The teacher supervises the work of the group and everyone's participation in it. The general correct version is written down in a notebook. Students who gave the correct answer options receive the maximum score - 100% of the theoretical part rating - 0.8b. Second place students - 85.9% of the rating. Third place - 70.9% of the rating. Those who did not answer or answered incorrectly 30% of the rating. The resulting score is taken into account when placing

Text

Changes in the oral mucosa in diseases of the gastrointestinal tract

Diseases of the gastrointestinal tract are often accompanied by changes in the oral cavity. This is largely due to the morphofunctional unity of the oral mucosa and the entire digestive tract. Studies of many authors have proven that in chronic gastritis, gastric ulcer and duodenal ulcer, chronic colitis and enterocolitis, various changes in the oral mucosa are found, the severity of which depends on the form, severity and duration of the underlying disease. Treatment of the underlying disease leads to a decrease or disappearance of pathological changes in the oral cavity.

The relationship between the violation of the acid-forming function of the stomach and pathological changes in the oral mucosa has been established. The secretion of the stomach, as shown by the studies of S.P. Kolomiets (1970), is closely related to

secretion of the salivary glands. Exacerbation of peptic ulcer disease is accompanied by a decrease in the reactivity of the oral mucosa.

In the works of V.V. Khazanova it is shown that recurrent aphthous stomatitis occurs against the background of intestinal dysbiosis. Many authors have described that in patients with recurrent aphthous stomatitis, changes in the small intestine occur similar to lesions of the oral mucosa.

The data obtained by V.E. Rudneva (1971) indicate that in patients with gastric ulcer, the concentration of histamine in the blood serum increases and at the same time the activity of histaminase decreases, and the activity of hyaluronidase also increases.

One of the important factors causing changes in the oral mucosa in the pathology of the gastrointestinal tract is a deficiency of vitamins, especially of group B.

Thus, as follows from the data obtained by numerous authors, all the above links in the pathogenesis of gastrointestinal diseases (increased vascular tissue permeability, hypovitaminosis, intestinal dysbiosis, imbalance of the immune system, etc.) form a causal relationship with changes in the cavity mouth.

Change of language. In diseases of the gastrointestinal tract, the state of the tongue is best studied. The type of tongue, according to many authors, can have an important diagnostic value and indicate the existing pathology of the digestive tract.

The lingering of the tongue is found most often. Plaque consists mainly of keratinized epithelial cells, bacteria, fungi, food debris. The severity of plaque depends on various reasons. The amount of plaque on the tongue increases with a decrease in its self-cleaning, mainly when chewing. In this case, the composition, consistency of food intake, as well as the regularity of individual hygiene measures and other factors are important. The amount of plaque on the tongue varies during the day: it is more in the morning than in the daytime and evening, since there is a decrease in the amount of plaque after eating. Violation of the process of normal (physiological) keratinization and desquamation of the epithelium also determines the amount and density of plaque. So, with atrophy of the filiform papillae of the tongue, there is little or no plaque at all. With hypertrophy of these papillae, on the surface of the tongue there is a hard-to-remove thick layer of plaque, consisting mainly of stuck together keratinized filiform papillae. The condition of the tongue may indicate disorders of the digestive system. The obstruction of the tongue is one of the characteristic symptoms of various diseases of the gastrointestinal tract. So, with an exacerbation of gastritis, peptic ulcer, enterocolitis and colitis, the amount of plaque increases, it covers the entire back of the tongue, localizing mainly in its posterior parts. Plaque formation, as a rule, is not accompanied by subjective sensations in patients. Only in the presence of a thick, dense layer of plaque on the tongue can there be a feeling of discomfort and a slight decrease in taste sensitivity. Usually, the coating on the tongue has a grayish-white color, but it can acquire various shades (yellow, brown). The color of plaque is mainly due to dyes of food products, drugs or exacerbations of gastrointestinal diseases: peptic ulcer, chronic hepatitis, cholecystitis (yellow, brown).

The edematous condition of the tongue is an important sign of diseases of the gastrointestinal tract. As a rule, tongue swelling is detected by a doctor during examination, since it does not cause painful sensations, except in cases of significant edema, when the tongue is bitten while eating or talking. The edematous state is determined upon examination by the pronounced imprints of the teeth on its lateral surfaces, as well as an increase in its size. The McClure-Aldrich blister test is used as an objective research method to determine the presence of edema (see Chapter 4 for a detailed description). With the help of a blister test, it is possible to determine the state of latent edema, which will make it possible to diagnose early (preclinical) changes. V.A.Epishev (1970) established a violation of the hydrophilia of the oral

mucosa in chronic gastritis. With anacid gastritis, a decrease was found, and with hyperacid gastritis, an increase in the time of resorption of the blister test was established. It should be borne in mind that swelling of the tongue is a condition associated with chronic intestinal diseases (enterocolitis, colitis), in which a violation of water metabolism is detected.

Changes in the papillae of the tongue are often found in patients with pathology of the digestive tract. In the pathogenesis of these changes lie mainly trophic disorders, as well as a violation of the vitamin balance due to insufficient absorption and synthesis of B vitamins,

Depending on the severity and color of the papillae of the tongue, some authors distinguish between hyperplastic and atrophic glossitis. Hyperplastic glossitis is observed more often in patients with gastritis with high acidity, with exacerbation of peptic ulcer disease. Glossitis is characterized by hypertrophy of the papillae of the tongue, the presence of dense plaque, an increase in the size of the tongue due to pronounced swelling. Atrophic glossitis is found in patients with gastritis with secretory insufficiency, with hepatitis, gastroenteritis, colitis. With this form of glossitis, atrophy and flattening of the papillae of the tongue, the absence of plaque are observed. Sometimes the atrophy of the papillae is pronounced, the tongue is smooth, shiny. The tongue may be hyperaemic (erythematous) or pale pink. In some cases, the tongue looks "lacquered" with bright red spots and stripes, reminiscent of Meller's glossitis. Atrophy of the papillae of the tongue can cause a burning sensation, soreness, tingling sensation when taking spicy, spicy food.

Desquamation of the epithelium of the tongue is quite often found in diseases of the gastrointestinal tract and can have varying degrees of severity. Desquamation occurs more often in patients with chronic gastritis with secretory insufficiency, chronic colitis, and liver diseases. It is characterized by the appearance on the back of the tongue of foci of desquamation of the epithelium of filiform papillae. In patients with secretory insufficiency, infectious liver lesions, a combination of desquamative glossitis with atrophy and flattening of the papillae of the tongue is often observed. These changes, as a rule, do not cause painful sensations, and patients often do not assume their existence, only sometimes they complain of burning sensation, pain when eating irritating food. Characterized by the appearance of foci of desquamation in the period of exacerbation of chronic diseases of the gastrointestinal tract and their disappearance during remission.

Paresthesia of the tongue is often associated with diseases of the digestive system. There is a burning sensation, tingling, tingling sensation of the tongue. These sensations often accompany desquamative glossitis, but they can also appear without visible changes in language (see section 11.7.6 for details). Нарушение вкусовой чувствительности. Taste sensitivity of the tongue is determined by the method of functional mobility of its receptors. It is known that the number of functioning receptors depends on the age and functional state of the digestive tract. Normally, the maximum activity of taste buds is observed on an empty stomach. After eating, there is a decrease in the level of their mobility. The reaction of the taste buds of the

tongue occurs in response to incoming excitation impulses from the receptors of the gastric mucosa in a centrifugal way. With a peptic ulcer, tumors of the stomach due to a disorder of its secretory and motor function, the reflex connection of the receptors of the tongue and the stomach is disrupted. This is manifested by various types of violations of the functional mobility of taste buds (an increase in their activity and the absence of demobilization after a meal, etc.). Disturbances in taste sensitivity can also occur with changes in the papillary apparatus of the tongue (heavily coated tongue, phenomena of atrophy or desquamation), which is associated with disturbances in the taste buds of the tongue.

Disorders of the oral mucosa. Erosive and ulcerative lesions of the oral mucosa arising from diseases of the gastrointestinal tract are mainly the result of trophic disorders. There are a large number of reports on the presence of erosions and ulcers on the oral mucosa in case of gastric ulcer, liver disease, colitis, enterocolitis, etc. The relationship between recurrent aphthous stomatitis and gastrointestinal pathology is known. About 50% of patients with diseases of the gastrointestinal tract (chronic gastritis, gastric ulcer and duodenal ulcer) suffer from recurrent aphthous stomatitis of varying severity.

A change in the color of the oral mucosa can manifest itself against the background of gastrointestinal pathology. During the period of exacerbation of peptic ulcer, enterocolitis, colitis, catarrhal gingivitis, glossitis or stomatitis often develop, the severity of which depends on the duration of the course and the frequency of exacerbations of the underlying disease. The mucous membrane of the mouth in the affected area is characterized by hyperemia with symptoms of cyanosis due to the chronic course of the process. In this case, patients complain of a burning sensation in the mouth, discoloration of the mucous membrane, and sometimes soreness when eating irritating food. The most pronounced phenomena of catarrhal gingivitis and stomatitis with exacerbation of chronic colitis. During the period

remission of gastrointestinal diseases, symptoms of catarrhal gingivitis or stomatitis become mild or completely disappear.

It should be remembered that such changes in the oral mucosa can be manifestations of other diseases and conditions of the body (infectious, including fungal, allergic, cardiovascular, hypovitaminosis). In this regard, for a successful diagnosis and choice of treatment methods, a thorough examination of the patient is necessary.

Salivation disorders. May manifest as hyper- or hyposalivation. In the studies of a number of authors, it has been proved that morphological and functional changes in the small salivary glands are observed in patients with gastric ulcer and duodenal ulcer. With a peptic ulcer in the initial stage (up to a year), as well as its exacerbation, an increase in salivation is noted, followed by the development of hyposalivation. Patients begin to complain of dry mouth. Clinically, impaired salivation is often combined with other previously described changes in the oral cavity, characteristic of gastrointestinal pathology.

Treatment. As a rule, for the treatment of catarrhal stomatitis, gingivitis or glossitis against the background of

gastrointestinal diseases, there is no need for special local treatment. Recommendations are given on the care of the oral cavity and on rational hygiene, if necessary, rinse with antiseptic or anti-inflammatory drugs (chlorhexidine, etonium, tincture of calendula, chamomile, etc.) is prescribed. Treatment of the underlying disease of the gastrointestinal tract is mandatory. In addition, given the presence of signs of group B hypovitaminosis and dysbiosis in the oral cavity and intestines in most patients with chronic diseases of the gastrointestinal tract, vitamins of group B should be prescribed internally (multivitamin complexes "Dekamevit", "Pangexavit", "Glutamevit", "Unikap", etc.) or, preferably, parenterally (thiamine, riboflavin, pyridoxine, cyanocobalamin).

Evaluation Criteria for Monitoring

№	Progress in% and points	Grade		Student knowledge level
1.	96-100	Fine "5"		Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively.
2.	91-95	Fine "5"	6.	71-75 Good "4" Shows high activity, creative approach when conducting interactive games. Correctly solves situational problems and test questions. Analyzes independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
			7.	66-70 Satisfactorily "3" Complete correct answer to questions about the clinic, diagnosis and treatment of TBI.
			3.	86-90 Fine "5"
			4.	81-85 Good "4"
			5.	76-80 Good "4"

			speech tissues.
8.	61-65	Satisfactorily "3"	Has an incomplete understanding of the topic. Makes mistakes when solving situational tasks. Tells not confidently.
9.	55-60	Satisfactorily "3"	Knows, tells not confidently. Has a partial view.
10.	54 and below	Unsatisfactory "2"	Has no exact idea. Does not know.

19- Practical lesson
Topic: Changes in OSR in cardiovascular diseases.

Technological map of the practical lesson

Stages of work	Teacher	Student
1. Stages of preparation (10 minutes)	1. Controls the purity of the audience; 2. Checks students' readiness for classes; 3. Controls attendance;	Listen
2.The introductory stage to classes (15 minutes)	1.Preparation on the topic of content; 2. Preparation of slides for the introductory report; 3. To develop the literature used for the study of the subject;	Write down the topic and listen
3. The main stage (195 minutes)	1. Dividing students into 2 small subgroups, asking questions on the topic; 2. Use of slides and multimedia; 3. Carries out medical work; 4. Combines all information on a given topic, actively the participating students are encouraged and appreciated;	Divided into small groups watching, participating, listening. The student expresses his opinion, complements and asks questions

Questions on the topic
1. Hemorrhages in the oral mucosa are possible
2. Nikolsky's symptom is positive when

Tests

Diabetes mellitus in the oral cavity is manifested in the form
1) dryness
2) necrotic processes
3) catarrhal gingivitis
4) petechial rash
5) Gunther's glossitis

Characteristic changes in the oral cavity with leukemia
1) ulcerative necrotic processes
2) "lacquer" tongue
3) enamel erosion
4) leukemic infiltrates

Lacquered "tongue is characteristic of the disease
1) stomalgia
2) multiforme exudative erythema
3) Gunther's glossitis

The cause of Gunther's glossitis is
1) influenza virus
2) fusospirochetosis
3) vitamin B12 deficiency

The Castle factor is not produced when
1) diabetes mellitus
2) Addison-Birmer anemia
3) avitaminosis "C"

The hemogram of a patient with Addison-Birmer anemia reveals
1) color index is higher than 1
2) color index below 1
3) the color index is normal

Clinical blood test for Addison-Birmer anemia reveals
1) a decrease in the number of red blood cells
2) a decrease in the number of leukocytes
3) decrease in hemoglobin

The synergist of vitamin B12 is
1) folic acid
2) vitamin C
3) vitamin PP

The primary element of the lesion in lichen planus is
1) stain
2) node
3) knot
4) bubble
Latin name of the primary element for lichen planus
1) nodus
2) ulcus
3) papula
4) macula
One of the secondary elements of the lesion with lichen planus is
1) afta
2) erosion
3) crack
4) crust
... Papula is education
1) cavity
2) cavityless

A papule is formed
1) from the epithelium
2) from the surface layer of the mucous membrane
3) from the epithelium and the superficial layer of the mucous membrane

Pathological changes in the epithelium with lichen planus
1) acanthosis
2) parakeratosis
3) spongiosis
4) hyperkeratosis
5) acantholysis
6) ballooning degeneration

Set correspondence

Types of keratinization disorders Pathological processes
in the epithelium in the epithelium
1) hyperkeratosis a) thickening of the epithelial layer
2) parakeratosis of the mucous membrane due to
3) acanthosis of basal proliferation and spiny cells
b) incomplete keratinization of superficial
cells of the spiny layer
c) excessive thickening of the stratum corneum

Lichen planus include
1) to keratoses
2) to allergic conditions
3) to infectious diseases

The most significant causes of lichen planus
1) stress
2) insolation
3) diseases of the gastrointestinal tract
4) lowering the height of the lower part of the face
5) hypothermia
6) diseases of the cardiovascular system
7) chronic trauma to the oral mucosa
The appearance of the lesion with lichen planus is affected
1) hyposalivation
2) hypersalivation
3) relief of the mucous membrane
4) turgor of the mucous membrane
spicy
2) chronic
3) chronic with exacerbations

In cytological material with erosion with lichen planus, they reveal
1) fusospirochetosis
2) acantholytic cells
3) a picture of nonspecific inflammation

The combination of the erosive and ulcerative form of lichen planus

with diabetes mellitus and hypertension
called a syndrome
1) Grinshpana
2) Melkerson-Rosenthal
3) Stevens-Johnson

The typical form of lichen planus is differentiated
1) with flat leukoplakia
2) with secondary syphilis
3) with pemphigus
4) with hyperplastic candidiasis
5) with chronic recurrent herpes
6) with acute mechanical injury
7) with medical stomatitis

The erosive and ulcerative form of lichen planus is differentiated
1) with flat leukoplakia
2) with chronic recurrent aphthous stomatitis
3) with erosive leukoplakia
4) with pemphigus
5) with exudative erythema multiforme
6) with ulcerative necrotizing stomatitis (sluggish)
7) with a traumatic ulcer with signs of hyperkeratosis around
8) with atrophic candidiasis

The hyperkeratotic form of lichen planus is differentiated
1) with pseudomembranous candidiasis
2) with flat leukoplakia
3) with verrucous leukoplakia
4) with chronic recurrent herpes
5) with mild leukoplakia
Set correspondence

017. The form of lichen planus Diseases with which differential diagnosis
1) typical a) flat leukoplakia
2) erosive and ulcerative b) erosive leukoplakia
3) hyperkeratotic c) verrucous leukoplakia
d) secondary syphilis
e) medical stomatitis
f) pemphigus
g) hyperplastic candidiasis
h) chronic recurrent herpes
i) traumatic erosion

diagnostic signs
1) lichen planus a) lesion element - spot or plaque
2) leukoplakia b) lesion element - papule
c) favorite localization - front cheeks, lower lip
d) favorite location - cheeks and lateral surfaces of the tongue in the middle and rear third
e) skin lesions are possible
f) absence of skin lesions

Lichen planus form Local remedies and methods
1) typical a) hirudotherapy
2) exudative-hyperemic b) laser therapy
3) erosive and ulcerative c) vitamin A and its analogues
d) steroid ointments
e) pain relievers
f) epithelizing agents
g) cauterizing agents
h) antiseptic treatment
i) nicotinic acid injections under the lesion
j) application of enzymes

020. The form of lichen planus Means and methods of general treatment
1) typical a) treatment of pathology
2) exudative-hyperemic gastrointestinal tract

3) erosive and ulcerative b) vitamin A and its analogues
c) nicotinic acid
d) solcoseryl injections
e) sedatives
f) pressure chamber
g) hormonal drugs
h) Zovirax
i) multivitamins

021. To remove the focus of hyperkeratosis in hyperkeratotic form lichen planus are shown
1) excision using a laser scalpel
2) cryodestruction
3) chemotherapy
4) radiotherapy
5) surgical excision
6) hirudotherapy

022. For the general treatment of patients with a typical form of lichen planus is prescribed
1) multivitamins
2) sedatives
3) antibiotics
4) antiviral drugs
5) cytostatics
6) antihistamines

For local treatment of patients with erosive and ulcerative form lichen planus is prescribed
1) hirudotherapy
2) UHF therapy
3) laser therapy
4) sanitation of the oral cavity
5) pain relievers
6) epithelizing agents
7) steroid ointments
8) antiseptic treatment
9) cauterizing agents
10) agents affecting differentiation and proliferation epithelial cells

For regulation of proliferation and differentiation of epithelial cells use
1) trypsin
2) solcoseryl
3) vitamin A
4) retinoids
5) chlorhexidine

The doctor's tactics in the presence of a focus of hyperkeratosis, towering over the surrounding tissues
1) observation
2) conservative treatment
3) excision

Keratoplastic properties are
1) vitamin A
2) solcoseryl
3) urotropine
4) rosehip oil
5) methyluracil
6) carotoline
7) chymotrypsin

Stages of local treatment of erosive lichen planus
1) keratoplasty
2) enzymes
3) antiseptics
4) pain relievers

Dispensary observation of patients with lichen planus
1) shown
2) not shown

When examining and treating patients with lichen planus need to be alert
1) immunological
2) venereal
3) cancer
4)hematological

Interactive method
USING THE HANDLE IN THE MIDDLE TABLE METHOD

All students of the group are divided by lot into 3 subgroups of 3 students each. Each subgroup sits down at a separate table, prepares a blank sheet of paper and a pen. The date, group number, last name and first name of the student are written on the sheet. The task is offered, to answer one question for the whole subgroup. Each student writes down his last name and one answer option on a sheet and passes the sheet to a neighbor, and moves his pen to the middle of the table. The teacher supervises the work of the group and everyone's participation in it. The general correct version is written down in a notebook. Students who gave the correct answer options receive the maximum score - 100% of the theoretical part rating - 0.8b. Second place students - 85.9% of the rating. Third place - 70.9% of the rating. Those who did not answer or answered incorrectly 30% of the rating. The resulting score is taken into account when placing

Text

With violations of the cardiovascular system, changes in the oral cavity are determined by the degree of circulatory disorders and damage to the vascular wall. Compen-

severe forms of cardiovascular failure, as a rule, are not accompanied by any significant changes in the oral cavity.

Swelling and cyanosis of the oral mucosa, blueness of the lips. Usually observed in de- compensated forms of cardiovascular insufficiency: hypertension, rheumatic heart disease or other diseases. Teeth prints appear on the lateral surfaces of the tongue, cheeks. This condition is often combined with the cyanosis of the red border of the lips. Swelling of the tongue can be expressed to a large extent, as a result of which it increases in size; speech becomes difficult. With myocardial infarction, swelling of the tongue can be combined with changes in its color, the appearance of cracks, erosions and ulcers. The severity of these changes is determined by the severity of the underlying disease. With a large-focal infarction, lesions of the oral mucosa are more pronounced. As the patient's condition improves after general treatment, the situation in the oral cavity improves - the swelling of the tongue decreases, epithelialization of erosions and ulcers occurs. Puffiness and ulceration of the oral mucosa often appear in areas adjacent to dentures: the marginal edge of the gums adjacent to metal crowns; the mucous membrane under the intermediate part of the bridge; under the prosthetic bed of removable plates, etc.

Despite significant changes in the mucous membrane, most patients do not experience subjective sensations, however, if swelling is detected, the doctor must determine the cause of its occurrence. It is necessary to carry out differential diagnostics with similar changes in the mucous membrane in gastrointestinal pathology, infectious and other diseases.

VASCULAR VASCULAR SYNDROME IN THE ORAL CAVITY. Described by A.L. Mashkillyson et al. (1972). It is characterized by the appearance of dense blisters with transparent or hemorrhagic contents of various sizes (Fig. 11.36). The authors explain the mechanism of blistering in patients with cardiovascular pathology by the rupture of small vessels of the oral mucosa as a result of increased permeability and fragility of the vascular wall. Along with this, a weakening of the connection between the epithelium and the connective tissue layer of the oral mucosa was noted,

apparently associated with the destruction of the basement membrane. T. I. Lemetskoyus-

Rice. 11.36. Vesicovascular syndrome. Bladder with hemorrhagic contents on the tongue.

Vesicovascular syndrome occurs more often in women aged 40-75 years. Blisters come on suddenly, often with meals. The appearance of blisters is usually associated with an increase in blood pressure, which patients are often unaware of. Favorite localization - on the mucous membrane of the soft palate, lateral surfaces of the tongue, cheeks. Bubbles can exist unchanged from several hours to several days. Sometimes they disappear without opening, but more often they open up with the formation of erosion, epithelizing within 3-7 days, depending on the size. In smears-prints from the area of erosion, elements of peripheral blood are found, there are no acantholytic cells. Nikolsky's symptom is negative.

Differential diagnosis. Vesicovascular syndrome is differentiated from:

A pemphigus;

And angiomas;

And exudative erythema multiforme.

Treatment. First of all, it is aimed at treating background cardiovascular diseases, without which it is impossible to prevent the reappearance of blisters. Prescribe drugs that normalize the state of the vascular wall (askorutin, compliance).

Local treatment consists of oral sanitation, including rational prosthetics. The mucous membrane of the mouth is treated with antiseptic agents (solution of chlorhexidine, chloramine, etc.), followed by the use of keratoplastic agents (oil solution of vitamins A, E, rosehip oil, sea buckthorn, Actovegin ointment, solcoseryl, etc.). In cases of rapid spontaneous epithelialization of erosions, sometimes within 2-3 days after their formation, special local treatment is not required.

ULTRA-NECROTIC LESIONS OF THE MUCOSA OF THE ORAL WITH THE FORMATION OF TROPHIC ULTRASONS. They develop in a number of cases in patients with I-III degree circulatory disorders. Against the background of a violation of the general condition of patients (weakness, shortness of breath, swelling of the extremities), soreness occurs in the oral cavity, and food intake is difficult.

One, rarely several ulcers appear on the mucous membrane of the mouth. The emergence of trophic ulcers, as a rule, is facilitated by trauma by the sharp edges of decayed teeth, poor-quality prostheses or other traumatic factors. Most often, ulcers

form on the lateral surfaces of the tongue, the mucous membrane of the cheeks, the floor of the mouth, palate, etc. The edges of the ulcers are uneven, the bottom is covered with a grayish-white necrotic plaque. If necrotic tissues are not rejected for a long time, they acquire a dark color. A characteristic feature of a trophic ulcer is the absence of a pronounced inflammatory reaction in the surrounding tissue. The necrotic process can spread to adjacent areas of the face, nasopharynx. Bad breath appears, saliva becomes viscous. Cases of ulcerative-necrotic lesions of the oral mucosa against the background of circulatory disorders with necrosis and sequestration of the jaw bone tissue, the formation of defects in the cheek tissues are described. Necrotic tissue breakdown can lead to severe bleeding. Prolonged existence of trophic ulcers can lead to their malignancy.

Diagnostics. A cytological examination of the papilla from the surface of a trophic ulcer reveals a small number of epithelial cells with signs of degeneration, which is expressed in a decrease in cell size; lack of clear contours in some of the cells, pycnosis. Neutrophils are found in varying degrees of decay. There is a significant decrease in the cells of histiocytic

series, which characterizes the areactive nature of the course of the inflammatory process.

Pathohistologically, a chronic inflammatory process with extensive necrosis and proliferation of interstitial tissue is determined in the area of a trophic ulcer; sclerotic changes in blood vessels and damage to nerve fibers.

Differential diagnosis. A trophic ulcer is differentiated from:

A traumatic ulcer;

▲ cancerous ulcer;

▲ tuberculous ulcer;

And Vincent's ulcerative necrotizing stomatitis;

▲ necrotic lesions of the oral mucosa in blood diseases.

Treatment. Conducted in contact with a therapist. General treatment should be directed to the elimination of cardiovascular failure. In some cases, it is advisable to carry out general treatment in a hospital setting. Symptomatic therapy is carried out locally according to indications. First of all, all local traumatic factors are eliminated. Remove necrotic tissue using proteolytic enzymes. The oral mucosa is treated with antiseptic and epithelial stimulating drugs (oil solution of vitamins A, E, sea buckthorn oil, rose hips, propolis, tsigerol, etc.). A gentle, high-calorie, vitamin-rich diet is prescribed.

Evaluation Criteria for Monitoring

№	Progress in% and points	Grade	Student knowledge level
1.	96-100	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Shows high activity, creative

			approach when conducting interactive games. Correctly solves situational problems and test questions. Analyzes independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
2.	91-95	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Analyzes research results independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
3.	86-90	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
4.	81-85	Good "4"	The correct answer to questions about the clinic, diagnosis and treatment of TBI, but there are 2-3 inaccuracies, errors. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
5.	76-80	Good "4"	Correct, but incomplete coverage of the issue. The student knows questions about the clinic and diagnosis of TBI, but is not fully versed in determining the tactics of treatment. Correctly solves situational problems, but the rationale for the answer is not complete. Understands the essence of the issue. Knows, speaks confidently
6.	71-75	Good	Correct, but incomplete coverage

		"4"	of the issue. Correctly solves situational problems, but the rationale for the answer is incomplete. Has 2-3 errors. Knows, tells confidently. Has accurate ideas.
7.	66-70	Satisfactorily "3"	Understands the essence of the issue. Correctly solves situational problems, but cannot substantiate the answer. Has accurate ideas on specific issues.
8.	61-65	Satisfactorily "3"	Has an incomplete understanding of the topic. Makes mistakes when solving situational tasks. Tells not confidently.
9.	55-60	Satisfactorily "3"	Knows, tells not confidently. Has a partial view.
10.	54 and below	Unsatisfactory "2"	Has no exact idea. Does not know.

20- Practical lesson
Topic: Change in OAS in diseases of the blood system

Technological map of the practical lesson

Stages of work	Teacher	Student
1. Stages of preparation (10 minutes)	1. Controls the purity of the audience; 2. Checks students' readiness for classes; 3. Controls attendance;	Listen
2.The introductory stage to classes (15 minutes)	1.Preparation on the topic of content; 2. Preparation of slides for the introductory report; 3. To develop the literature used for the study of the subject;	Write down the topic and listen
3. The main stage (195 minutes)	1. Dividing students into 2 small subgroups, asking questions on the topic; 2. Use of slides and multimedia; 3. Carries out medical work; 4. Combines all information on a given topic, actively the participating students are encouraged and appreciated;	Divided into small groups watching, participating, listening. The student expresses his opinion, complements and asks questions

Questions on the topic
1.etiology of leukemia
2 clinic of leukemia
3 differential diagnosis of leukemia

Tests

... Changes in the OAS in a disease of the hematopoietic system
A) myeloblastic
B) lymphoblastic
C) plasmablastic
D) all answers are correct

60. The defeat of the OAS in acute leukemia, what is the percentage.
A) 90.9%
B) 80.9%
B) 70.9%
D) 71.9%

61. Up to what age does acute leukemia occur?
A) 30 years
B) 40 years old
C) 50 years old
D) 60 years

62. A common symptom in the diagnosis of acute leukemia is.
A) general weakness, pallor of the skin
B) headache, muscle pain
C) bone pain, fever
D) all answers are correct

63. With leukemia, the number of platelets and leukocytes
A) often reduced
B) normal
B) increased
D) there is no right answer

64. Bleeding of the gums is very often ... in acute leukemia
A) the first clinical sign
B) the last clinical sign
B) with chronic leukemia
D) there is no right answer

65. Hemorrhagic syndrome is detected in acute leukemia
A) in 50-60%

B) 60-70%
B) in 70-80%
D) in 80-90%

66. Differentiating hemorrhagic syndrome in acute leukemia follows from ...
A) hypovitaminosis C
B) traumatic injuries
C) when teeth sticking to the gum
D) all answers are correct

3 students each. Each subgroup sits down at a separate table, prepares a blank sheet of paper and a pen. The date, group number, last name and first name of the student are written on the sheet. The task is offered, to answer one question for the whole subgroup. Each student writes down his last name and one answer option on a sheet and passes the sheet to a neighbor, and moves his pen to the middle of the table. The teacher supervises the work of the group and everyone's participation in it. The general correct version is written down in a notebook. Students who gave the correct answer options receive the maximum score - 100% of the theoretical part rating - 0.8b. Second place students - 85.9% of the rating. Third place - 70.9% of the rating. Those who did not answer or answered incorrectly 30% of the rating. The resulting score is taken into account when placing

Текст

Diseases of the blood and hematopoietic organs are often accompanied by changes in the oral mucosa, which in some cases appear earlier than the clinical signs of the underlying disease. This forces patients to seek help from the dentist first. The dentist is often the first to encounter such patients, so he must be well versed in the manifestations of the pathology of the hematopoietic system in the oral cavity. This will make it possible to diagnose the disease in a timely manner and carry out its treatment. In addition, the inability to recognize a disease of the hematopoietic system leads to an erroneous diagnosis and, as a consequence, the appointment of incorrect treatment, which can lead to a significant deterioration in the patient's condition. The dentist must know the scope of necessary and acceptable interventions in the treatment of patients with diseases of the hematopoietic system. Unjustified dental intervention can lead to profuse bleeding from the socket of the extracted tooth or during curettage, as well as to the progression of necrotic processes in the oral cavity and even death.

The psychological and deontological aspects of the first contact of a dentist with a hematological patient are very important. It is necessary to be able to carefully and convincingly explain to the patient the need for some special examination methods or consultation with a hematologist in connection with the specific picture of lesions of the oral mucosa. When communicating with a patient, it is very important not to frighten him or cause psychological trauma, since fear of a malignant neoplasm (acute and chronic leukemia) is a natural condition for most people .. Treatment of patients with diseases of the hematopoietic system is associated with the use of special means and methods. As a rule, it is carried out in specialized hematological institutions. The

volume of dental treatment procedures is determined by the severity and stage of the clinical course of the underlying disease and is carried out by a dentist in some cases in a dental or specialized hematology department.

ACUTE LEUKEMIA. It is characterized by an increase in the number of blast, "young" cells in the bone marrow, spleen, lymph nodes and other organs and tissues. Depending on the morphological and cytochemical characteristics of blast cells, several forms of acute leukemia are distinguished: myeloblastic, lymphoblastic, plasmablastic, etc. The lesion of the oral mucosa is observed in 90.9% of patients with acute leukemia. It occurs mainly at a young age (up to 30 years).

In the diagnosis of acute leukemia, an important role belongs to general symptoms (general weakness, malaise, fatigue, pallor of the skin). Body temperature can be high with large differences, but sometimes it is subfebrile. The patient gives the impression of being seriously ill. Due to a sharp decrease in the body's defenses, patients with leukemia sometimes develop candidiasis, and herpes infection in the oral cavity manifests itself. Blast cells are present in the blood, and the number of platelets and red blood cells is often reduced.

Oral symptoms are of great importance in recognizing acute leukemia. They are very characteristic and easy to spot. On examination, pallor of the skin, pallor, pastiness, slight vulnerability and bleeding of the oral mucosa, hemorrhages on the gums, cheeks (especially along the line of teeth closing), palate, tongue are noted. Hematomas and hemorrhages can be observed both on the mucous membrane and on the skin.

Gum bleeding is very often the first clinical sign of acute leukemia. At the same time, the gums become loose, bleed, ulcerate. Bleeding occurs at the slightest touch, and sometimes spontaneously. Bleeding may occur not only from the gums, but also from the tongue, cheeks along the line of closing the teeth and other areas of the oral mucosa. Sometimes, extensive hemorrhages and hematomas of the oral mucosa are found. The clinical manifestations of hemorrhagic syndrome can be different: from small-point hemorrhages on the oral mucosa and skin to extensive hematomas and profuse bleeding. Hemorrhagic syndrome is detected in 50-60% of patients and is based on pronounced thrombocytopenia, which develops as a result of suppression of normal hematopoiesis due to leukemic hyperplasia and bone marrow infiltration.

Differentiation of hemorrhagic syndrome in acute leukemia follows from hypovitaminosis C, traumatic injuries (especially on the mucous membrane of the cheeks along the line of teeth closing). The final diagnosis is established on the basis of the clinical picture of the disease,

Rice. 11.38. Acute leukemia.

Necrotizing ulcerative gingivitis with gingival hyperplasia and bleeding.

general blood test and bone marrow punctate examination.

Gingival hyperplasia is a common symptom of acute leukemia, especially in its severe course, and is assessed by hematologists as an unfavorable prognostic sign. Hyperplasia and infiltration of the gums with leukemic cells can be so significant that the crowns of the teeth are almost completely closed by a loose, bleeding, ulcerated shaft in places that prevents the patient from eating and talking. It is characteristic that in a significant part of cases, already at the very beginning of the disease, hyperplasia is more pronounced on the inner (palatal or lingual) surface than on the buccal. This

clinical symptom sometimes helps to differentiate gingival hyperplasia in leukemia from the banal hypertrophic gingivitis.

According to histological data, gingival hyperplasia is caused by infiltration of the connective tissue layer of the mucous membrane by myeloid cells, which leads to disruption of trophism, followed by tissue necrosis and ulceration.

Necrotic ulcerative lesions of the oral mucosa often develop in acute leukemia. It is characteristic that the apex of the gingival papilla undergoes necrosis. Around the focus of necrosis, the gums have a bluish color, while the entire mucous membrane is pale, anemic (Fig. 11.38). Necrosis spreads quickly and soon around the tooth

an ulcer develops with a dirty gray fetid bloom. Often, necrosis is detected on the tonsils, in the retromolar region and other parts of the oral cavity. A feature of the necrotic process in acute leukemia is its tendency to spread to adjacent areas of the mucous membrane. As a result, there may be extensive ulcers with irregular contours, covered with a gray necrotic plaque. Reactive changes around the ulcer are absent or weak. In the presence of necrotic changes in the oral cavity, patients complain of sharp soreness when eating, difficulty swallowing, putrid fetid breath, general weakness, dizziness, headache. At the beginning of the development of ulcerative necrotic changes, hypersalivation may occur, and then the amount of saliva decreases, which is associated with dystrophic processes in the salivary glands.

The causes of necrotic processes on the oral mucosa in acute leukemia have not been completely clarified. It is believed that necrosis can result from the breakdown of leukemic their infiltrates, in areas of extensive hemorrhage, as well as due to neurotrophic disorders in tissues and violations of the body's defenses. An important point that determines the occurrence of necrosis is the action of external factors, especially microorganisms.

Lymph nodes in acute leukemia increase slightly - up to 0.5-1 cm, they are soft, painless. In some cases, acute leukemia can occur with the formation of pronounced specific leukemic infiltrates in the skin and oral mucosa. As a result of the formation of infiltrates in the gums, a sharp deformation of the gingival margin occurs. This condition is often diagnosed as hypertrophic gingivitis. Leukemic infiltrates can ulcerate, which often leads to bleeding.

Differential diagnosis. Differentiating the manifestations of acute leukemia on the mucous membrane follows from:

^ hypertrophic gingivitis of other etiology;

^ ulcerative necrotizing stomatitis of Vincent;

^ hypovitaminosis C;

And intoxication with salts of heavy metals.

The results of blood tests are decisive in the diagnosis of leukemia.

Treatment. The task of the dentist is the correct and timely recognition of acute leukemia by clinical

some symptoms in the oral cavity and other manifestations, analysis of peripheral blood. Treatment of acute leukemia is carried out in a specialized hematology department. Local treatment is carried out in consultation with the hematologist. Oral hygiene is very important. Treatment and extraction of teeth, removal of tartar is carried out under the supervision of a hematologist in a hospital setting. In the presence of ulcerative-necrotic lesions of the oral mucosa, it is anesthetized, treated

with antiseptic solutions (hydrogen peroxide, chloramine, etonium, romazulan, furacilin, etc.), proteolytic enzymes and agents that stimulate epithelization (rosehip oil, sea buckthorn, propolis preparations, oil solution of vitamins A, E, etc.). When detecting fungal or herpetic lesions of the oral mucosa, conventional antifungal or antiviral therapy is performed, described in detail in the relevant sections of the textbook.

CHRONIC LEUKEMIA. Chronic leukemia, depending on the nature of the lesion of the hematopoietic organs, is divided into myeloproliferative (myeloid leukemia) and lymphoproliferative (lymphocytic leukemia) forms. Chronic myeloid leukemia goes through two stages: benign, lasting several years, and malignant (terminal), which lasts 3-6 months. The first stage often begins without pronounced clinical symptoms, then neutrophilic leukocytosis appears with a shift to promyelocytes and single blast cells. In the second stage, as in the first during blast crises, there are many blast forms in the blood (myeloblasts, hemocytoblasts), there is a rapid increase in the number of leukocytes in the blood. As leukemia progresses, anemia, thrombocytopenia, accompanied by hemorrhages develop.

Chronic myeloid leukemia has been asymptomatic for a long time and can be accidentally diagnosed during a clinical blood test. The blood picture is characterized by high leukocytosis (50-60 * 109 / l). In a blood smear, various intermediate forms of the myeloid series are found: myeloblasts, promyelocytes, myelocytes.

Hemorrhagic manifestations are characteristic of chronic myeloid leukemia, although their intensity is much less than in acute leukemia. Bleeding of the gums does not occur spontaneously, but when injured. The mucous membrane of the mouth is pale. The gums are swollen, cyanotic, bleed at the slightest touch, there is no inflammation. Absence

inflammation and severe bleeding are characteristic symptoms for leukemia.

Erosive and ulcerative lesions of the oral mucosa, according to V.M. Uvarov (1975), are observed in patients with myeloid leukemia. The appearance of severe necrotic lesions is considered as an unfavorable sign indicating an exacerbation of the process. In the severe stage of the disease, candidiasis often develops.

Chronic lymphocytic leukemia usually develops in middle-aged and elderly people, is characterized by a long-term benign course and a significant increase in the number of leukocytes in the blood (lymphocytes prevail among them). The number of leukocytes can reach 500 109 / l.

The initial period of chronic lymphocytic leukemia often goes unnoticed by the patient. The only complaint may be an increase in lymph nodes (on palpation, they are not very dense, mobile, painless). Leukemic lymphocytic infiltrates or tumor-like nodes are formed in the oral cavity in chronic lymphocytic leukemia. They are more often located on the mucous membrane of the gums, cheeks, tongue, palatine arches, tonsils. The resulting infiltrates (nodes or nodules) have a soft (pasty) consistency, bluish color, mobile, rise above the level of the mucous membrane. Ulceration is rare. Bleeding occurs with minor injury. Leukemic lymphocytic infiltrates are also localized in the bone marrow, lymph nodes, spleen, and liver. Лечение. Общее и местное лечение проводится так же, как при остром лейкозе.

AGRANULOCYTOSIS. A syndrome characterized by a decrease in the number or complete disappearance of granulocytes (granular leukocytes) from the peripheral

blood. By the mechanism of occurrence, myelotoxic and immune agranulocytosis are distinguished. The cause of myelotoxic agranulocytosis is a violation of the formation of granulocytes in the bone marrow as a result of exposure to the body of ionizing radiation, cytotoxic drugs, benzene vapors, etc. drugs (amidopyrine, butadione, analgin, phenacetin, sulfonamides, some antibiotics, etc.).

Agranulocytosis often begins with the manifestation of changes

niy in the oral cavity, which forces patients to go to the dentist at the onset of the disease. Against the background of a deterioration in the general condition of patients (fever, lethargy, headache, pain when swallowing, odor from the mouth), ulcerative-necrotic processes develop on the oral mucosa, which tend to spread rapidly. The destruction of the oral mucosa proceeds without a pronounced leukocyte reaction of the surrounding tissues and insignificant pain. Fetid ulcers bleed easily. Sometimes the jaw bone tissue is also involved in the ulcerative-necrotic process. The gums, lips, tongue, cheeks, tonsils are more often affected. In case of damage to the tonsils, pain appears when swallowing. In some patients, the appearance of ulcers along the digestive tract is noted. The lymph nodes are enlarged. The number of lymphocytes in the blood is significantly reduced (from 1 to 3 109 / l, and sometimes less than 1 109 / l). In the leukocyte formula, the number of granulocytes is sharply reduced or they are completely absent, only lymphocytes and monocytes are found.

Diagnostics. The diagnosis is made on the basis of anamnesis, clinical presentation, results of a study of peripheral blood and bone marrow punctate.

Differential diagnosis. Agranulocytosis should be differentiated from necrotizing ulcerative stomatitis of Vincent and other blood diseases.

Treatment. General treatment is carried out under the supervision of a therapist and hematologist, first of all, blood transfusion, elimination of the etiological factor, etc.

Local treatment includes the use of analgesics, application of proteolytic enzymes to necrotic areas of the oral mucosa, antiseptic treatment and the use of drugs that stimulate epithelialization. THROMBOCYTOPENIC PURPLE. A syndrome characterized by increased bleeding due to a decrease in the number of platelets in the blood. Thrombocytopenic purpura can be an independent disease - Verlhof's disease, as well as develop as a result of other diseases (malignant neoplasms, radiation sickness, hepatitis, infectious diseases, drug intoxication, etc.) - a symptomatic form.

Idiopathic thrombocytopenic purpura, or Werlhof's disease, can be acute or chronic. The main clinical symptom of the disease is hemorrhage in the skin, mucous membranes, as well as bleeding from the nose, gums, etc., arising spontaneously or. under the influence of minor trauma. Hemorrhages are observed in the serous membranes, retina and other parts of the eye. Hemorrhages in the heart and brain are life-threatening. Patients have a slight increase in body temperature and a pronounced thrombocyte singing (up to 20 109 / l or less), as a result of which the retraction of the blood clot is impaired and the bleeding time is lengthened up to 10 minutes or more (normally 3-4 minutes). As a result of massive bleeding, iron deficiency anemia can develop.

Diagnostics. The diagnosis of thrombocytopenic purpura is established on the basis of anamnesis data (identification of contact with various chemicals, uncontrolled intake of analgesics, hypnotics, etc.), clinical picture and blood test.

Differential diagnosis. Differentiate the disease from:

^ hemophilia;
^ grief;
^ hemorrhagic vasculitis.

Treatment. General treatment is carried out by a hematologist, as a rule, in a hospital setting. Dental interventions should be performed with extreme caution after consulting a hematologist. POLYCYTHEMIA (erythremia, or Vakez's disease). Chronic disease of the hematopoietic system, characterized by a persistent increase in the number of erythrocytes and hemoglobin per unit volume of blood, along with an increase in the volume of circulating blood. The disease is associated with increased production of erythrocytes, leukocytes and platelets in the bone marrow. It develops gradually and has a long chronic course. It occurs more often in men aged 40-60 years. There is a change in the color of the skin and mucous membrane of the mouth. They acquire a dark cherry color with a cyanotic hue, which is due to the increased content of reduced hemoglobin in the capillaries. Bleeding of the gums is common. Bleeding from the stomach, intestines, uterus is possible. Characterized by a dark cherry color with a cyanotic shade of lips and cheeks. In the oral cavity, Kuperman's symptom is typical - pronounced cyanosis of the mucous membrane of the soft palate and pale coloration of the hard palate. Many patients experience pruritus, paresthesias of the oral mucosa, associated, in the opinion of many authors, with increased blood vessels and neurovascular disorders. The picture of peripheral blood is characterized by an increase in the content of erythrocytes to 6-8-10 | 2 / l), hemoglobin (180-200 g / l), a decrease in ESR to 1-3 mm / h. Treatment. Carried out by a hematologist. Special local therapy is not indicated.

ANEMIA. This is a group of various pathological conditions characterized by a decrease in the hemoglobin content and (or) the number of erythrocytes per unit of blood volume. Anemias are hereditary and acquired. According to the modern classification, anemias can develop as a result of blood loss (post-hemorrhagic), increased destruction of red blood cells (hemolytic) and due to a violation of the formation of red blood cells.

Some forms of anemia, along with general manifestations, are characterized by typical symptoms in the oral cavity. These are primarily anemias that develop as a result of a violation of the formation of erythrocytes - deficiency anemias associated with insufficiency of such hematopoietic factors as iron (iron deficiency anemia), as well as vitamin B | 2 and folic acid (megaloblastic anemia).

Iron deficiency anemia, hypochromic. The causes of the disease are associated with insufficient intake of iron in the body or its increased consumption (during growth, pregnancy, lactation, with increased sweating), as well as with endogenous disorders leading to insufficient absorption of iron (with resection of the stomach, intestines, achilic gastritis, gastroenteritis , chronic colitis, endocrine disorders, etc.).

The clinical picture. It is characterized by a violation of the general condition of the patient: pallor of the skin, weakness, dizziness, tinnitus, cold clammy sweat, a decrease in body temperature and blood pressure, an increased tendency to fainting.

Along with general symptoms, changes in the oral cavity are noted, which have a certain diagnostic value. So, paresthesias and taste disturbances appear long before the decrease in the level of serum iron and the development of obvious anemia. On examination, the mucous membrane of the mouth is pale, atrophic, insufficiently

moisturized. Patients are worried about dry mouth, difficulty swallowing food, burning and pain in the tongue, lips,

eaten while eating, taste perversion. Patients eat chalk, tooth powder, raw cereals. Cracks often appear in the corners of the mouth and on the lips (especially in patients with anacid gastritis and after resection of the stomach). The back of the tongue is hyperemic, shiny. The papillae of the tongue are atrophic. The tongue is very sensitive to all mechanical and chemical stimuli. There is soreness when taking spicy, sour, hot food. Some swelling of the tongue is noted, which is determined by the imprints of the teeth on its lateral surfaces. In a number of patients, there is a loss of the natural gloss of the enamel, increased abrasion of the teeth.

Diagnostics. The diagnosis is made on the basis of anamnesis data, clinical manifestations, but the decisive moment is the blood picture. It is characterized by hypochromic anemia - a decrease in the hemoglobin content and a low color index (0.5-0.6 and below), anisocytosis and poikilocytosis, as well as a decrease in the content of iron and ferritin in the blood serum.

Treatment. General treatment is aimed at eliminating the causes of the anemia and iron deficiency. Prescribe iron supplements, vitamins. They recommend a balanced diet.

Local treatment is aimed primarily at eliminating traumatic factors. The oral cavity is sanitized. According to indications, symptomatic therapy is prescribed, aimed at eliminating cracks in the red border of the lips, normalizing salivation, eliminating burning sensation, paresthesia and others.

Anemia B (2-deficiency and (or) folic acid deficiency (malignant, or pernicious, Addison-Birmer anemia). Characterized by impaired erythropoiesis. Vitamin B | 2 and folic acid are necessary factors for normal hematopoiesis. Deficiency of vitamin B | 2 violates the process of maturation of erythroblasts in the bone marrow, resulting in the formation of immature cells - megaloblasts, megalocytes, which quickly die. Deficiency of vitamin B | 2 in the body is a consequence of the absence of Castle's internal factor - a gastromucoprotein produced by the gastric mucosa, without which it does not occur assimilation of vitamin B | 2 (external factor) Vitamin B12 deficiency can be caused by atrophic processes in the gastric mucosa, gastric resection, inflammatory bowel disease or resection of certain areas of the intestine, vitamin B | 2 deficiency in food.

Folic acid deficiency anemia occurs when folic acid absorption is impaired (in patients with alcoholism, sprue, etc.), with insufficient intake of folic acid from food, with the appointment of certain antiepileptic drugs.

Vitamin B12-deficiency anemia is more common in people over 40 years of age. The appearance of patients is characteristic: a pale face, skin with a yellowish tinge, thin, atrophic, hair turns gray early. The general condition suffers, weakness and increased fatigue develop. The clinical picture is characterized by damage to the hematopoietic tissue, nervous and digestive systems.

Complaints of patients about pain and burning of the tip or edges of the tongue, paresthesia, in most cases are the first, ahead of the further development of symptoms of anemia.

On examination, a pale, atrophic with an icteric tinge of the mucous membrane is revealed, sometimes there are punctate hemorrhages on the cheeks, bleeding of the gums.

Changes in language are characteristic. It becomes smooth, shiny, polished due to atrophy of the papillae, thinning of the epithelium and muscle atrophy. Bright red spots may appear on the tongue, when they merge, the entire tongue becomes bright red (Gunther's glossitis, Genter-Möller). It is in stark contrast to the pale mucous membrane in the rest of the mouth.

Diagnostics. The diagnosis is made on the basis of the history data, the clinical picture of the disease and the results of a blood test. Hyperchromic anemia is observed in the blood - a decrease in the number of erythrocytes saturated with hemoglobin; the color indicator is 1.1-1.3; erythrocytes are large (megalocytes). There are erythrocytes with nuclear remnants and giant hypersegmented neutrophils. Examination of bone marrow punctate reveals a large number of megaloblasts.

Treatment. Prescribe vitamin B12 at 100-500 mcg and folic acid at 0.005 g per day. In severe cases, the administration of vitamins is combined with blood and red blood cell transfusions. Changes in the oral cavity go away quickly after general treatment.

Local treatment is not required. The oral cavity is sanitized, according to indications, agents are prescribed for local anesthesia of the oral mucosa (2% trimecaine solution, 1-2% pyromecaine solution, anesthesin in glycerin, etc.).

Evaluation Criteria for Monitoring

№	Progress in% and points	Grade	Student knowledge level
1.	96-100	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Shows high activity, creative approach when conducting interactive games. Correctly solves situational problems and test questions. Analyzes independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
2.	91-95	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Analyzes research results

			independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
3.	86-90	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
4.	81-85	Good "4"	The correct answer to questions about the clinic, diagnosis and treatment of TBI, but there are 2-3 inaccuracies, errors. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
5.	76-80	Good "4"	Correct, but incomplete coverage of the issue. The student knows questions about the clinic and diagnosis of TBI, but is not fully versed in determining the tactics of treatment. Correctly solves situational problems, but the rationale for the answer is not complete. Understands the essence of the issue. Knows, speaks confidently
6.	71-75	Good "4"	Correct, but incomplete coverage of the issue. Correctly solves situational problems, but the rationale for the answer is incomplete. Has 2-3 errors. Knows, tells confidently. Has accurate ideas.
7.	66-70	Satisfactorily "3"	Understands the essence of the issue. Correctly solves situational problems, but cannot substantiate the answer. Has accurate ideas on specific issues.
8.	61-65	Satisfactorily "3"	Has an incomplete understanding of the topic. Makes mistakes when solving situational tasks.

			Tells not confidently.
9.	55-60	Satisfactorily "3"	Knows, tells not confidently. Has a partial view.
10.	54 and below	Unsatisfactory "2"	Has no exact idea. Does not know.

21- Practical lesson
Topic: Changes in OSR in diseases of the nervous system. Clinic, diagnosis and treatment

Technological map of the practical lesson

Stages of work	Teacher	Student
1. Stages of preparation (10 minutes)	1. Controls the purity of the audience; 2. Checks students' readiness for classes; 3. Controls attendance;	Listen
2.The introductory stage to classes (15 minutes)	1.Preparation on the topic of content; 2. Preparation of slides for the introductory report; 3. To develop the literature used for the study of the subject;	Write down the topic and listen
3. The main stage (195 minutes)	1. Dividing students into 2 small subgroups, asking questions on the topic; 2. Use of slides and multimedia; 3. Carries out medical work; 4. Combines all information on a given topic, actively the participating students are encouraged and appreciated;	Divided into small groups watching, participating, listening. The student expresses his opinion, complements and asks questions

Questions on the topic
What groups should glossalgia apply to.
Etiology of glossalgia.
3. Clinic of glossalgia.
4. Dif - diagnostics of glossalgia.
5. Prevention of glossalgia.
6. Treatment of glossalgia.

Tests

Forms of rhomboid glossitis
1) flat
2) convex

3) lumpy
4) papillomatous

022. Rhomboid glossitis is often complicated
1) the appearance of erosion
2) candidiasis
3) the appearance of bubbles
4) papillomatous growths
023. Strengthening of papillomatous growths in the area of rhomboid glossitis requires
1) blockade of the lingual nerve
2) cryodestruction
3) applications of keratoplasty
024. If you suspect B12-iron deficiency anemia
blood needs to be examined
1) the number of leukocytes
2) the number of erythrocytes
3) the level of hemoglobin
4) at the time of bleeding
5) on ESR
6) per color indicator
025. Bright red, painful "polished" tongue -
characteristic symptom
1) for chronic recurrent aphthous stomatitis
2) for erythema multiforme exudative
3) for B12-deficiency anemia
4) for leukemia
5) for herpes
026. Gunther's glossitis differentiate
1) with allergic glossitis
2) with acute atrophic candidiasis
3) with thrush
4) with chronic atrophic candidiasis
5) with syphilis
6) with diamond-shaped glossitis
7) with desquamative glossitis
027. Indicators of hemoglobin of blood change
1) with leukoplakia
2) with leukemia
3) with iron deficiency anemia
4) with chronic recurrent aphthous stomatitis
5) with candidiasis
028. In case of anemia of hypochromic type, the color indicator
1) 0.8-1.1
2) 0.5-0.6
3) 1.2-1.5

029. Therapeutic measures for B12-deficiency anemia
1) sanitation of the oral cavity
2) elimination of traumatic factors
3) application of vitamin A
4) parenteral administration of vitamin B12
5) enteral administration of vitamin B12
6) hirudotherapy

030. Pain in the tongue, passing during meals, is characteristic
1) for glossalgia
2) for trigeminal neuralgia
3) for catarrhal glossitis
4) for desquamative glossitis
5) for rhomboid glossitis

031. A disease characterized by burning, tingling,
a feeling of scaldedness in the tongue, disappearing when eating, is called
1) glossalgia
2) HIV infection
3) stomalgia
4) syphilis

032. Glossalgia is a disease
1) infectious
2) allergic
3) psychogenic
4) hereditary

033. Complaints of a patient with glossalgia
1) burning sensation in the tongue
2) perversion of taste
3) pain when eating irritating food
4) dry mouth
5) pain in the tongue when moving
6) burning pain in one half of the tongue, aggravated by eating
7) sudden shooting pains in the tongue
8) the feeling of "scalded" tongue, disappearing when eating
034. An objective examination of a patient with glossalgia reveals
1) regional lymphadenitis
2) no change
3) dryness of the oral mucosa
4) violation of sensitivity in one half of the tongue
5) solitary aphthae in the tongue
6) areas of increased desquamation
7) slight atrophy of the papillae of the tongue

035. The correct name of the disease with a simultaneous burning sensation in the tongue,
lower lip and hard palate
1) glossalgia
2) trigeminal neuralgia
3) neuritis
4) stomalgia

036. While eating, the burning sensation disappears
1) due to eating warm food
2) due to the impact of positive emotions
3) due to suppression of the pain dominant by a stronger food dominant

Interactive method
USING THE WEB METHOD

1. Steps:
2. 1. In advance, students are given time to prepare questions for the lesson.
3. 2. Participants sit in a circle.
4. 3. One of the participants is given a skein of thread, and he asks his prepared question (to which he himself must know the full answer), holding the end of the thread and throwing the skein to any student.
5. 4. The student who received the skein answers the question (at the same time, the person who asked him comments on the answer) and passes the question relay on. Participants continue to ask and answer questions until everyone is on the web.
6. 5. As soon as all students finish asking questions, the student holding the skein returns it to the participant from whom he received the question, at the same time asking his own question, etc., until the ball is completely "unwound".

Text
Changes in the oral mucosa in diseases of the nervous system

The mucous membrane of the mouth and tongue is the zone in which a powerful flow of afferentation to the central nervous system (CNS) is formed. At the same time, a number of diseases of the nervous system significantly alter the formation of the flow of afferentation and the processing of information from the periphery by the central nervous structures. Compensatory changes in the oral mucosa may occur. More often, diseases of the nervous system manifest themselves in the form of unpleasant sensations in the oral cavity. Only in rare cases are morphological changes in the mucous membrane observed (erosion, ulcers in patients with organic lesions and CNS injuries). With stomalgia (glossalgia) and taste

disturbances, the mucous membrane of the mouth usually does not change, but these diseases are quite relevant for dentistry and neurostomatology.

11.7.6.1. Stomalgia (glossalgia)

• Stomalgia (syn: glossalgia, glossodynia, paresthesia of the tongue) is a chronic disease with persistent orofacial pain, usually not accompanied by lesions of the mucous membrane of the mouth and tongue.

In 1838, Weisse gave the first scientific description of stomalgia. Many works have been published on various aspects of this suffering, but to date, the issues of etiology, pathogenesis and treatment of stomalgia have not been finally resolved.

Due to the unclearness of the mechanism of development of the disease, there is no single designation for stomalgia. Many terms have been proposed: burnt tongue syndrome, glossalgia, or dysesthesia of the oral cavity, glossodynia, vegetative tongue, sensory neurosis of the tongue, glossoperosis. R. M. Basker, D. M. Main (1991) propose a new term for this suffering - "burning mouth syndrome", considering that it more accurately reflects the leading complaint of patients.

Etiology and pathogenesis. A number of authors suggest an increased level of monomers in the acrylic bases of prostheses as the cause of stomalgia, but not all confirm an allergic reaction to acrylates of prostheses as an important cause in the development of the disease. More likely causes are mechanical irritation with prostheses and parafunctional habits. It was believed that in

galvanic currents are of great importance for the development of stomalgia, but the results of recent studies have shown that this is not the case. Stomalgia patients have hypersensitivity to mercury, nickel, cobalt, gold, palladium, copper, zinc and food additives. The hypersensitivity reaction to mercury was the most frequent, however, replacing the amalgam fillings relieved the burning sensation in the oral cavity in less than half of the patients.

Stomalgia may develop after the use of enalopril, captopril and isinopril (angiotensin-converting enzyme inhibitors). A large number of chemical agents and drugs have been described that can cause pathological changes in orofascial localization. With this pathology, approximately 20% of the prevalence of xerostomia is observed. Complaints about dry mouth may not always indicate hypofunction of the salivary glands. Attempts have been made to determine the existence of a link between stomalgia and salivary gland dysfunction. There is evidence of a decrease in the function of the salivary glands in stomalgia.

Given that the majority of patients with stomalgia are menopausal women, many authors suggest that dry mouth may be due to a decrease in the functional activity of the salivary glands during menopause. However, most researchers have shown that there is no significant decrease in the function of the salivary glands during menopause. The cause of xerostomia can be concomitant somatic pathology and the use of certain drugs for its treatment.

In the histological examination of the salivary glands, some authors indicate changes in morphology, while others reject these data.

The clinical picture. With stomalgia, pain can often be classified as dysesthesia of hard-to-determine localization. The pain is superficial and deep, mainly at the tip and along the edges of the tongue, can spread to the mouth, pharynx, lips and be combined

with a burning sensation in the mouth, dry mouth, thirst, taste disturbances, anxiety, irritability, and a reduced desire for social interaction. The level of pain in stomalgia, although different in quality, is similar in intensity to toothache.

In the case of dysesthesia and paresthesia, the burning sensation appears in the morning, intensifies during the day and reaches a maximum in the evening, weakening or completely disappearing with food intake. The daily rhythm of occurrence is clearly traced

pain. It is repeated for months with periodic exacerbations. This condition sometimes appears after an affective trauma, death or cancer of a loved one, with personal and professional difficulties.

Most often, women suffer from stomalgia, mainly during menopause. They feel irritation in the area of the mucous membrane of the tongue and mouth, which is usually dry and similar to that of the elderly. The excitement of the tongue joins. Patients examine their language for a long time, trying to detect changes in it. All these symptoms are combined with short-term general excitement, aggravated by hot flashes. There is a fear of the possible development of cancer.

Many patients with stomalgia complain of a taste disorder called dysgeusia. Dysgeusia was detected in 68% of patients with stomalgia, especially for bitter food.

The response to local anesthesia differs in patients in the groups with dysgeusia, burning sensation in the oral cavity and in the group with a combination of dysgeusia and burning.

It was found that dysgeusia is of peripheral origin, since its decrease after the application of local anesthesia is noted. Apparently, local anesthesia can relieve peripheral depression of the central sensory structures in these patients. The lack of influence on the burning sensation of local anesthetics justifies the use of medications acting on the central structures.

Stomalgia symptoms subside with exposure to hot and spicy foods. This effect is associated with the capsaicin found in spicy food. A good effect was revealed when using capsaicin (a preparation of capsicum for external use) in the treatment of orofacial pain. Intraoral triggered trigeminal neuralgia was less responsive to topical capsaicin therapy than stomalgia.

In patients with stomalgia, a correlation was found between the level of beta-endorphin and the severity of stomalgia.

The activation of the sympathoadrenal system was revealed (in the urine of patients, the level of vanillin mandelic acid, which is a metabolite of adrenaline, was determined, the content of which was significantly higher in patients than in healthy people). At the same time, a correlation was found between the level of vanillin mandelic acid and the severity of paresthesia of the tongue. Based on this, the authors concluded that the pathogenetic role of sympathetic activation

adrenal system in the development of stomalgia - an increase in the content of adrenergic substances affects peripheral receptors and central mechanisms of perception of sensations.

Electroencephalographic studies in stomalgia gave conflicting results: in some studies, a shift with a predominance of rhythm activation phenomena was revealed, which is typical for any pain phenomenon, in others, an increased functional activity of synchronizing structures was described.

Stomalgia should be considered along with other pathological manifestations of chronic emotional stress.

Many authors point to the frequent combination of stomalgia with diseases of the gastrointestinal tract, cardiovascular pathology, osteochondrosis, endocrine and autoimmune pathology, and diseases of the nasopharynx. Others do not find a connection between stomalgia and these pathological conditions.

It is assumed that hormonal changes during menopause may be a factor provoking the development of discomfort in the oral mucosa. A number of researchers reported a decrease in the intensity of oral symptoms and psychopathological manifestations (depression, anxiety) in most postmenopausal women who were treated with estrogens. Researchers associate the success of estrogen therapy with the presence of estrogen receptors in the oral mucosa in some women.

Emotional changes (especially anxiety and depression) are detected in stomalgia to such an extent that many authors consider this pathology as psychogenic. In recent years, psychological testing has been included in the examination plan for all patients with stomalgia. The most commonly used are the Swedish and Carolina personality scales, the scale of mental functioning, the scale of the quality of life, etc.

It was found that, in comparison with the control group, patients with stomalgia have significantly lower indices in the socialization scale, higher in somatic anxiety in muscle tension and in psychoasthenic scales. According to a number of authors, up to 95% of patients with stomalgia note the presence of anxiety and depression. Patients had significantly higher characteristics of depression (an indicator of the severity of chronic symptoms) than in the sample of patients with somatic pathology, but lower than patients with mental illness with clinical depression. The same correlation can be seen in the level of anxiety.

The most characteristic feature of stomalgia is the presence of depression with sleep disturbance, pronounced neurovegetative reactivity and somatic response to stress. However, latent depression is also revealed. While numerous studies suggest a link between depression and stomalgia, not all support this conclusion unequivocally.

Nerve damage or dysfunction that occurs with oral or facial trauma, as well as after medical interventions, can be a source of pain in stomalgia. Burning pain is characteristic of pain conditions associated with nerve damage. Chronic pain can be associated with changes in the sympathetic nervous system as well as with impaired blood flow.

While peripheral lesions can be provoking factors, the leading factors in the pathogenesis of stomalgia are changes in the central nociceptive structures.

Recent studies have shown that damage or inflammation of peripheral tissues or nerves can create neuroplastic changes in the central nociceptive systems, which are given an important role in stomalgia. Given the possibility of such changes, methods of treating stomalgia associated with damage to facial tissues and nerves (tooth extraction, etc.) should be abandoned: they are usually ineffective and can aggravate neuroplastic changes and the progression of symptoms.

Differential diagnosis. Neuralgia and neuropathy of the lingual nerve have a clinical picture similar to stomalgia, in which sensory disturbances in the form of pain or hypesthesia are more pronounced. However, the main difference between stomalgia is the disappearance of all painful manifestations during a meal.

Treatment. In the treatment of stomalgia, a complex application of therapeutic measures is necessary: drug therapy, physiotherapy, acupuncture, laser therapy, hyperbaric oxygenation and psychotherapy. Drug therapy includes the use of antidepressants, benzodiazepines, antihistamines, sex hormones, vitamins and sialic supplements, antifungal drugs, analgesics, vasoactive drugs, and vegetotropics.

Antidepressants are widely used for the treatment of painful conditions for more than 30 years. The usual daily doses of antidepressants are 25-100 mg. Doses of antidepressants should be limited if anticholinergic side effects occur (dry mouth, constipation, poor accommodation, urinary retention). Patients, especially those with concomitant cardiovascular pathology, may experience complications (orthostatic hypotension, arrhythmia).

Benzodiazepines are often given to patients with chronic pain, although long-term use can stimulate or worsen depression. The most commonly used are phenazepam at a dose of 0.25-0.5 mg 2-3 times a day, tazepam (daily dose 30-90 mg), mezapam (daily dose 10-30 mg). During the period of exacerbation of discomfort with stomalgia, a course of Relanium is prescribed intramuscularly, and in severe cases of the disease, antipsychotics are used, for example, haloperidol at a dose of 3.6-15 mg per day.

To improve microcirculation, especially in people with vascular diseases and degenerative changes in the cervical spine, use pentoxylline at a dose of 300-600 mg per day, nicotinic acid, nikoshpan, no-shpu, stugeron, cavinton.

It is necessary to use vitamins (parenteral administration of thiamine, riboflavin, pyridoxine, cyanocobalamin, ascorbic acid; oral administration of tocopherol acetate and retinol). It is recommended to take vegetative preparations: anticholinergics (bellaspon, belloid, platifillin); anticholinesterase drugs (galantamine); ganglion blockers (gangleron, benzohexonium). Antihistamines are used - pipolfen, diphenhydramine, suprastin.

In order to inhibit peripheral mechanisms, local anesthetics are used (a suspension of anesthesin on glycerin, solutions of trimecaine and lidocaine) in the form of applications, oral baths, aerosol irrigation.

To reduce the phenomena of xerostomia in order to stimulate the function of the salivary glands, pilocarpine is used at a dose of 5 mg 1 time per day sublingually, vitamin A, potassium iodide. Bromhexin is used to change the consistency of saliva. Sial substitutes of artificial and natural origin are used as replacement therapy, for example salenum, a water-soluble flaxseed extract.

In the complex treatment of stomalgia, acupuncture is being used more and more widely and with high efficiency. The effect of auriculoelectro-

puncture. There is a high therapeutic efficacy from the use of percutaneous electroneurostimulation [Borisova EG, 1993].

Physiotherapeutic procedures are widely used: massage of the collar zone and head, galvanization of the upper cervical sympathetic nodes, endonasal electrophoresis of novocaine, etc. Many authors point to the positive effect of laser therapy, which plays the role of a biostimulator and has an analgesic effect.

A method of treating glossalgias with medical leeches is used, which is especially effective in patients with diseases of the cardiovascular system.

There are indications of the positive effect of the use of hyperbaric oxygenation and the use of oxygen when it is injected with a syringe into the mucous membrane of the mouth, cheeks and on the lateral surfaces of the tongue.

According to a number of authors, an effective course of psychotherapeutic treatment, consisting of hypnotherapy and autogenous training.

Reorganization of the oral cavity, grinding of the sharp edges of the teeth, restoration of the occlusal height are required. Despite the availability of a large number of drugs and techniques used to treat stomalgia, their effectiveness is low, and therefore requires joint efforts of doctors of different specialties - dentists, neurologists and psychiatrists - to achieve success in the study and treatment of stomalgia.

Disturbance of taste (dysgeusia) occurs with pathology of the facial nerve in the event that the lesion is localized in the canal of the facial nerve (fallopian canal). This is due to the fact that on this segment of the facial nerve, a drum string passes next to it, which contains taste conductors that carry information from the taste buds of the anterior 2/3 of the tongue to the cerebral cortex. Accordingly, in this case, a one-sided loss of gustatory sensitivity in the front 73 of the tongue is revealed.

Outside the canal of the facial nerve, taste fibers are located in the lingual nerve. Consequently, even with its defeat, there will also be a loss of taste sensitivity in the front 2/3 of the tongue, but in combination with other symptoms. It should be noted that one hundred

Early loss of gustatory sensitivity on the back '/ tongue is associated with damage to the glossopharyngeal nerve.

Complete bilateral, less often unilateral loss of gustatory sensitivity occurs with organic lesions of the central nervous system (brain tumors, neurosyphilis, etc.).

Disturbance of taste can manifest itself in its complete loss, decrease and perversion. This applies to all types of sensitivities (sweet, salty, sour, bitter) or only some of them.

Taste bulbs are located mainly in the mucous membrane of the tongue, therefore, the etiological factors affecting them, and, therefore, causing taste disturbances, are diverse: viral infections, allergic glossitis, radiation damage, injuries, vitamin deficiencies A and B, Sjogren's syndrome, gastrointestinal diseases tract, etc. In this case, there is increased sloughing or temporary loss of epithelium in the tongue, in connection with which the taste buds are exposed and become available for excessive irritation. In such cases, gustatory sensitivity is restored as the papillae regenerate.

With a strong overlapping and extensive lesions of the tongue with leukoplakia, the taste often decreases, which is associated with the difficult access of flavoring substances to the taste buds. A sour feeling can occur in patients with galvanic phenomena. Gingivitis, periodontitis, chronic tonsillitis, periodontitis can become the source of an unpleasant taste in the oral cavity. A feeling of bitter, sour can appear with hepatocholecystitis, gastritis. Perversion or loss of taste is possible with hysteria.

Treatment. Due to the fact that taste disturbances often occur with a lesion localized in the canal of the facial nerve, where taste fibers pass in the β-composition of the facial nerve, with pathology of the lingual nerve (which also includes taste fibers), in the medulla oblongata (the nucleus of the glossopharyngeal nerve), as well as in hysteria, treatment is most often carried out by a neuropathologist according to the

principles of treatment of the corresponding diseases and taking into account their etiology.

11.7.6.3. Salivation disorder

Pathology of the innervation of the salivary glands and damage to the salivary glands themselves and the mucous membrane of the oral cavity often cause impaired salivation.

There are two types of impaired salivation: hypersalivation and hyposalivation (with its extreme degree - xerostomia).

HYPERSALIVATION. The etiological factors causing hypersalivation are acute inflammatory processes of the oral mucosa, accompanied by an unconditioned reflex increase in saliva secretion due to sharp irritation of the mucous membrane, which is a positive protective reaction of the body. In some cases, hypersalivation occurs as a result of direct irritation of the secretory cells of the salivary glands with mercury, iodine when these substances enter the body.

Prolonged hypersalivation is observed in some organic diseases of the central nervous system - parkinsonism, bulbar and pseudobulbar paralysis, in some cases after a stroke, etc. Swallowing disorders in these and other diseases enhances hypersalivation. The cause of hypersalivation may be helminthic invasion.

With neurosis, obsessive states, false hypersalivation is possible, when patients complain of profuse salivation, and this is not confirmed by an objective examination.

Patients with hypersalivation, in addition to a dentist, should be examined by a neurologist and a therapist if indicated.

Treatment. With hypersalivation, treatment in all cases should be aimed at eliminating the underlying disease that caused this condition. A temporary decrease in salivary secretion can be achieved with the use of atropine preparations.

HYPOSALIVATION. The reasons for hyposalivation are diverse: avitaminosis A, B, E, hyposiderosis; salivation decreases in diabetes mellitus, systemic connective tissue diseases, radiation injury, with bilateral chronic parotitis, after surgical removal of the large salivary glands. Dryness of the oral cavity is most pronounced in patients with Sjogren's syndrome. An increase in the tone of the sympathetic nervous system in thyrotoxicosis, in the climacteric period, in neurotic conditions leads to hyposalivation. As a temporary phenomenon, xerostomia can occur in acute infectious diseases, with botulism, after taking certain medications (for example, the atropine group). In people with impaired nasal breathing (polyps, curvature of the nasal septum), constantly forced

breathe through the mouth, dryness of the oral cavity is caused by increased evaporation of fluid from the oral cavity (false hyposalivation). Dry mouth only at night can be the result of sleeping with an open mouth, which is more common in old age. It should be noted that in old and senile age, salivation decreases, therefore, factors that reduce it are stronger in these age groups.

Hyposalivation and especially xerostomia cause in patients a feeling of dry mouth, difficulty in eating and talking, pain from spicy and solid food, burning sensation, roughness of the mucous membrane. Hyposalivation is often associated with dryness of other mucous membranes.

On physical examination, the mucous membrane is poorly moistened or dry, dull. There is little saliva, it is frothy or completely absent. The active growth of microflora

(due to the shutdown of the protective effect of saliva) leads to inflammation of the mucous membrane, the occurrence of erosions, ulcers, injuries when eating. In patients with prolonged hyposalivation, the amount of dental plaque increases, and multiple dental caries occurs.

Treatment. It consists in eliminating the cause of hyposalivation. Reorganization of the oral cavity is mandatory, including prosthetics. Locally, it is recommended to use anti-inflammatory drugs and agents that protect the mucous membrane from irritation (borax on glycerin, lysozyme, peach, sunflower oil, etc.).

As a rule, vitamin therapy is prescribed (vitamins A, Bp B6, B12, C, E), according to indications - sex hormones; iodine preparations inside. A good effect is provided by galvanizing the area of the large salivary glands. Anticholinesterase drugs are successfully used as symptomatic agents, in particular, a 0.5% galantamine solution, 1.0 ml subcutaneously, by electrophoresis, or by mouth daily for 1 month. Prescribe 1% solution of pilocarpine hydrochloride, 4 drops 1-2 times a day

Evaluation Criteria for Monitoring

№	Progress in% and points	Grade	Student knowledge level
1.	96-100	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Shows high activity, creative approach when conducting interactive games. Correctly solves situational problems and test questions. Analyzes independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
2.	91-95	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Analyzes research results independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
3.	86-90	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI.

			Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
4.	81-85	Good "4"	The correct answer to questions about the clinic, diagnosis and treatment of TBI, but there are 2-3 inaccuracies, errors. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
5.	76-80	Good "4"	Correct, but incomplete coverage of the issue. The student knows questions about the clinic and diagnosis of TBI, but is not fully versed in determining the tactics of treatment. Correctly solves situational problems, but the rationale for the answer is not complete. Understands the essence of the issue. Knows, speaks confidently
6.	71-75	Good "4"	Correct, but incomplete coverage of the issue. Correctly solves situational problems, but the rationale for the answer is incomplete. Has 2-3 errors. Knows, tells confidently. Has accurate ideas.
7.	66-70	Satisfactorily "3"	Understands the essence of the issue. Correctly solves situational problems, but cannot substantiate the answer. Has accurate ideas on specific issues.
8.	61-65	Satisfactorily "3"	Has an incomplete understanding of the topic. Makes mistakes when solving situational tasks. Tells not confidently.
9.	55-60	Satisfactorily "3"	Knows, tells not confidently. Has a partial view.
10.	54 and below	Unsatisfactory "2"	Has no exact idea. Does not know.

22- Practical lesson
Topic: Glossalgia. Clinic, diagnosis and treatment

Technological map of the practical lesson

Stages of work	Teacher	Student
1. Stages of preparation (10 minutes)	1. Controls the purity of the audience; 2. Checks students' readiness for classes; 3. Controls attendance;	Listen
2.The introductory stage to classes (15 minutes)	1.Preparation on the topic of content; 2. Preparation of slides for the introductory report; 3. To develop the literature used for the study of the subject;	Write down the topic and listen
3. The main stage (195 minutes)	1. Dividing students into 2 small subgroups, asking questions on the topic; 2. Use of slides and multimedia; 3. Carries out medical work; 4. Combines all information on a given topic, actively the participating students are encouraged and appreciated;	Divided into small groups watching, participating, listening. The student expresses his opinion, complements and asks questions

Тесты

Pain in the tongue, passing during meals, is characteristic
1) for glossalgia
2) for trigeminal neuralgia
3) for catarrhal glossitis
4) for desquamative glossitis
5) for rhomboid glossitis

A disease characterized by burning, tingling,
a feeling of scaldedness in the tongue, disappearing when eating, is called
1) glossalgia
2) HIV infection
3) stomalgia
4) syphilis

Glossalgia is a disease
1) infectious
2) allergic

3) psychogenic
4) hereditary

Complaints of a patient with glossalgia
1) burning sensation in the tongue
2) perversion of taste
3) pain when eating irritating food
4) dry mouth
5) pain in the tongue when moving
6) burning pain in one half of the tongue, aggravated by eating
7) sudden shooting pains in the tongue
8) the feeling of "scalded" tongue, disappearing when eating
An objective examination of the patient with glossalgia reveals
1) regional lymphadenitis
2) no change
3) dryness of the oral mucosa
4) violation of sensitivity in one half of the tongue
5) solitary aphthae in the tongue
6) areas of increased desquamation
7) slight atrophy of the papillae of the tongue

The correct name of the disease with a simultaneous burning sensation in the tongue, lower lip and hard palate
1) glossalgia
2) trigeminal neuralgia
3) neuritis
4) stomalgia

1) due to eating warm food
2) due to the impact of positive emotions
3) due to suppression of the pain dominant by a stronger food dominant

Contributes to the development of stomalgia
1) smoking
2) immunodeficiency
3) mental trauma
4) taking antibiotics
Local factors contributing to the development of stomalgia
1) crowded teeth
2) poor oral hygiene
3) lowering the height of the lower part of the face
4) traumatic tooth extraction
5) trauma of the tongue with sharp edges of the teeth

A decrease in the height of the lower part of the face leads to
1) tongue biting

2) to infringement of chorda tympani
3) to infringement of the lingopharyngeal nerve

Stomalgia is more common
1) in adolescents
2) in men 50-60 years old
3) in women 50-60 years old
4) in all age groups
Typical clinical signs of stomalgia
1) dry mouth
2) burning sensation in the mouth increases with food intake
3) burning sensation in the mouth disappears with food intake
4) persistent pain in the tongue increases with food intake
5) profuse white plaque on the oral mucosa
6) numbness of one half of the tongue
7) severe radiating pain occurs suddenly
Typical clinical signs of trigeminal neuralgia
1) dry mouth
2) burning sensation in the mouth increases with food intake
3) burning sensation in the mouth disappears with food intake
4) persistent pain in the tongue increases with food intake
5) profuse white plaque on the oral mucosa
6) numbness of one half of the tongue
7) severe radiating pain occurs

Interactive method
USING THE WEB METHOD
Steps:

1. In advance, students are given time to prepare questions for the lesson.
2. Participants sit in a circle.
3. One of the participants is given a skein of thread, and he asks his prepared question (to which he himself must know the full answer), holding the end of the thread and throwing the skein to any student.
4. The student who received the skein answers the question (at the same time, the person who asked him comments on the answer) and passes the question relay on. Participants continue to ask and answer questions until everyone is on the web.
5. As soon as all students finish asking questions, the student holding the skein returns it to the participant from whom he received the question, at the same time asking his own question, etc., until the ball is completely "unwound".

Text

Stomalgia (syn: glossalgia, glossodynia, paresthesia of the tongue) is a chronic disease with persistent orofacial pain, usually not accompanied by damage to the mucous membrane of the mouth and tongue.

In 1838, Weisse gave the first scientific description of stomalgia. Many works have been published on various aspects of this suffering, but to date, the issues of etiology, pathogenesis and treatment of stomalgia have not been finally resolved.

Due to the unclearness of the mechanism of development of the disease, there is no single designation for stomalgia. Many terms have been proposed: burnt tongue syndrome, glossalgia, or dysesthesia of the oral cavity, glossodynia, vegetative tongue, sensory neurosis of the tongue, glossoperosis. R. M. Basker, D. M. Main (1991) propose a new term for this suffering - "burning mouth syndrome", considering that it more accurately reflects the leading complaint of patients.

Etiology and pathogenesis. A number of authors suggest an increased level of monomers in the acrylic bases of prostheses as the cause of stomalgia, but not all confirm an allergic reaction to acrylates of prostheses as an important cause in the development of the disease. More likely causes are mechanical irritation with prostheses and parafunctional habits. It was believed that in

galvanic currents are of great importance for the development of stomalgia, but the results of recent studies have shown that this is not the case. Stomalgia patients have hypersensitivity to mercury, nickel, cobalt, gold, palladium, copper, zinc and food additives. The hypersensitivity reaction to mercury was the most frequent, however, replacing the amalgam fillings relieved the burning sensation in the oral cavity in less than half of the patients.

Stomalgia may develop after the use of enalopril, captopril and isinopril (angiotensin-converting enzyme inhibitors). A large number of chemical agents and drugs have been described that can cause pathological changes in orofascial localization. With this pathology, approximately 20% of the prevalence of xerostomia is observed. Complaints about dry mouth may not always indicate hypofunction of the salivary glands. Attempts have been made to determine the existence of a link between stomalgia and salivary gland dysfunction. There is evidence of a decrease in the function of the salivary glands in stomalgia.

Given that the majority of patients with stomalgia are menopausal women, many authors suggest that dry mouth may be due to a decrease in the functional activity of the salivary glands during menopause. However, most researchers have shown that there is no significant decrease in the function of the salivary glands during menopause. The cause of xerostomia can be concomitant somatic pathology and the use of certain drugs for its treatment.

In the histological examination of the salivary glands, some authors indicate changes in morphology, while others reject these data.

The clinical picture. With stomalgia, pain can often be classified as dysesthesia of hard-to-determine localization. The pain is superficial and deep, mainly at the tip and along the edges of the tongue, can spread to the mouth, pharynx, lips and be combined with a burning sensation in the mouth, dry mouth, thirst, taste disturbances, anxiety, irritability, and a reduced desire for social interaction. The level of pain in stomalgia, although different in quality, is similar in intensity to toothache.

In the case of dysesthesia and paresthesia, the burning sensation appears in the morning, intensifies during the day and reaches a maximum in the evening, weakening or completely disappearing with food intake. The daily rhythm of occurrence is clearly traced

pain. It is repeated for months with periodic exacerbations. This condition sometimes appears after an affective trauma, death or cancer of a loved one, with personal and professional difficulties.

Most often, women suffer from stomalgia, mainly during menopause. They feel irritation in the area of the mucous membrane of the tongue and mouth, which is usually dry and similar to that of the elderly. The excitement of the tongue joins. Patients examine their language for a long time, trying to detect changes in it. All these symptoms are combined with short-term general excitement, aggravated by hot flashes. There is a fear of the possible development of cancer.

Many patients with stomalgia complain of a taste disorder called dysgeusia. Dysgeusia was detected in 68% of patients with stomalgia, especially for bitter food.

The response to local anesthesia differs in patients in the groups with dysgeusia, burning sensation in the oral cavity and in the group with a combination of dysgeusia and burning.

It was found that dysgeusia is of peripheral origin, since its decrease after the application of local anesthesia is noted. Apparently, local anesthesia can relieve peripheral depression of the central sensory structures in these patients. The lack of influence on the burning sensation of local anesthetics justifies the use of medications acting on the central structures.

Stomalgia symptoms subside with exposure to hot and spicy foods. This effect is associated with the capsaicin found in spicy food. A good effect was revealed when using capsaicin (a preparation of capsicum for external use) in the treatment of orofacial pain. Intraoral triggered trigeminal neuralgia was less responsive to topical capsaicin therapy than stomalgia.

In patients with stomalgia, a correlation was found between the level of beta-endorphin and the severity of stomalgia.

The activation of the sympathoadrenal system was revealed (in the urine of patients, the level of vanillin mandelic acid, which is a metabolite of adrenaline, was determined, the content of which was significantly higher in patients than in healthy people). At the same time, a correlation was found between the level of vanillin mandelic acid and the severity of paresthesia of the tongue. Based on this, the authors concluded that the pathogenetic role of sympathetic activation

adrenal system in the development of stomalgia - an increase in the content of adrenergic substances affects peripheral receptors and central mechanisms of perception of sensations.

Electroencephalographic studies in stomalgia gave conflicting results: in some studies, a shift with a predominance of rhythm activation phenomena was revealed, which is typical for any pain phenomenon, in others, an increased functional activity of synchronizing structures was described.

Stomalgia should be considered along with other pathological manifestations of chronic emotional stress.

Many authors point to the frequent combination of stomalgia with diseases of the gastrointestinal tract, cardiovascular pathology, osteochondrosis, endocrine and autoimmune pathology, and diseases of the nasopharynx. Others do not find a connection between stomalgia and these pathological conditions.

It is assumed that hormonal changes during menopause may be a factor provoking the development of discomfort in the oral mucosa. A number of researchers reported a decrease in the intensity of oral symptoms and psychopathological manifestations (depression, anxiety) in most postmenopausal women who were treated with estrogens. Researchers associate the success of estrogen therapy with the presence of estrogen receptors in the oral mucosa in some women.

Emotional changes (especially anxiety and depression) are detected in stomalgia to such an extent that many authors consider this pathology as psychogenic. In recent years, psychological testing has been included in the examination plan for all patients with stomalgia. The most commonly used are the Swedish and Carolina personality scales, the scale of mental functioning, the scale of the quality of life, etc.

It was found that, in comparison with the control group, patients with stomalgia have significantly lower indices in the socialization scale, higher in somatic anxiety in muscle tension and in psychoasthenic scales. According to a number of authors, up to 95% of patients with stomalgia note the presence of anxiety and depression. Patients had significantly higher characteristics of depression (an indicator of the severity of chronic symptoms) than in the sample of patients with somatic pathology, but lower than patients with mental illness with clinical depression. The same correlation can be seen in the level of anxiety.

The most characteristic feature of stomalgia is the presence of depression with sleep disturbance, pronounced neurovegetative reactivity and somatic response to stress. However, latent depression is also revealed. While numerous studies suggest a link between depression and stomalgia, not all support this conclusion unequivocally.

Nerve damage or dysfunction that occurs with oral or facial trauma, as well as after medical interventions, can be a source of pain in stomalgia. Burning pain is characteristic of pain conditions associated with nerve damage. Chronic pain can be associated with changes in the sympathetic nervous system as well as with impaired blood flow.

While peripheral lesions can be provoking factors, the leading factors in the pathogenesis of stomalgia are changes in the central nociceptive structures.

Recent studies have shown that damage or inflammation of peripheral tissues or nerves can create neuroplastic changes in the central nociceptive systems, which are given an important role in stomalgia. Given the possibility of such changes, methods of treating stomalgia associated with damage to facial tissues and nerves (tooth extraction, etc.) should be abandoned: they are usually ineffective and can aggravate neuroplastic changes and the progression of symptoms.

Differential diagnosis. Neuralgia and neuropathy of the lingual nerve have a clinical picture similar to stomalgia, in which sensory disturbances in the form of pain or hypesthesia are more pronounced. However, the main difference between stomalgia is the disappearance of all painful manifestations during a meal.

Treatment. In the treatment of stomalgia, a complex application of therapeutic measures is necessary: drug therapy, physiotherapy, acupuncture, laser therapy, hyperbaric oxygenation and psychotherapy. Drug therapy includes the use of antidepressants, benzodiazepines, antihistamines, sex hormones, vitamins and sialic supplements, antifungal drugs, analgesics, vasoactive drugs, and vegetative tropic remedies.

Antidepressants are widely used for the treatment of painful conditions for more than 30 years. The usual daily doses of antidepressants are 25-100 mg. Doses of antidepressants should be limited if anticholinergic side effects occur (dry mouth, constipation, poor accommodation, urinary retention). Patients, especially those with concomitant cardiovascular pathology, may experience complications (orthostatic hypotension, arrhythmia).

Benzodiazepines are often given to patients with chronic pain, although long-term use can stimulate or worsen depression. The most commonly used are phenazepam at a dose of 0.25-0.5 mg 2-3 times a day, tazepam (daily dose 30-90 mg), mezapam (daily dose 10-30 mg). During the period of exacerbation of discomfort with stomalgia, a course of Relanium is prescribed intramuscularly, and in severe cases of the disease, antipsychotics are used, for example, haloperidol at a dose of 3.6-15 mg per day.

To improve microcirculation, especially in people with vascular diseases and degenerative changes in the cervical spine, use pentoxylline at a dose of 300-600 mg per day, nicotinic acid, nikoshpan, no-shpu, stugeron, cavinton.

It is necessary to use vitamins (parenteral administration of thiamine, riboflavin, pyridoxine, cyanocobalamin, ascorbic acid; oral administration of tocopherol acetate and retinol). It is recommended to take vegetative preparations: anticholinergics (bellaspon, belloid, platifillin); anticholinesterase drugs (galantamine); ganglion blockers (gangleron, benzohexonium). Antihistamines are used - pipolfen, diphenhydramine, suprastin.

In order to inhibit peripheral mechanisms, local anesthetics are used (a suspension of anesthesin on glycerin, solutions of trimecaine and lidocaine) in the form of applications, oral baths, aerosol irrigation.

To reduce the phenomena of xerostomia in order to stimulate the function of the salivary glands, pilocarpine is used at a dose of 5 mg 1 time per day sublingually, vitamin A, potassium iodide. Bromhexin is used to change the consistency of saliva. Sial substitutes of artificial and natural origin are used as replacement therapy, for example salenum, a water-soluble flaxseed extract.

In the complex treatment of stomalgia, acupuncture is being used more and more widely and with high efficiency. The effect of auriculoelectro-

puncture. There is a high therapeutic efficacy from the use of percutaneous electroneurostimulation [Borisova EG, 1993].

Physiotherapeutic procedures are widely used: massage of the collar zone and head, galvanization of the upper cervical sympathetic nodes, endonasal electrophoresis of novocaine, etc. Many authors point to the positive effect of laser therapy, which plays the role of a biostimulator and has an analgesic effect.

A method of treating glossalgias with medical leeches is used, which is especially effective in patients with diseases of the cardiovascular system.

There are indications of the positive effect of the use of hyperbaric oxygenation and the use of oxygen when it is injected with a syringe into the mucous membrane of the mouth, cheeks and on the lateral surfaces of the tongue.

According to a number of authors, an effective course of psychotherapeutic treatment, consisting of hypnotherapy and autogenous training.

Reorganization of the oral cavity, grinding of the sharp edges of the teeth, restoration of the occlusal height are required. Despite the presence of a large number of drugs

and techniques used to treat stomalgia, their effectiveness is low, and therefore requires joint efforts of doctors of different specialties - dentists, neurologists and psychiatrists - to achieve success in the study and treatment of stomalgia

Evaluation Criteria for Monitoring

№	Progress in% and points	Grade	Student knowledge level
1.	96-100	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Shows high activity, creative approach when conducting interactive games. Correctly solves situational problems and test questions. Analyzes independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
2.	91-95	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Analyzes research results independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
3.	86-90	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
4.	81-85	Good "4"	The correct answer to questions about the clinic, diagnosis and treatment of TBI, but there are 2-3 inaccuracies, errors. Thinks creatively. Solves situational

			tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
5.	76-80	Good "4"	Correct, but incomplete coverage of the issue. The student knows questions about the clinic and diagnosis of TBI, but is not fully versed in determining the tactics of treatment. Correctly solves situational problems, but the rationale for the answer is not complete. Understands the essence of the issue. Knows, speaks confidently
6.	71-75	Good "4"	Correct, but incomplete coverage of the issue. Correctly solves situational problems, but the rationale for the answer is incomplete. Has 2-3 errors. Knows, tells confidently. Has accurate ideas.
7.	66-70	Satisfactorily "3"	Understands the essence of the issue. Correctly solves situational problems, but cannot substantiate the answer. Has accurate ideas on specific issues.
8.	61-65	Satisfactorily "3"	Has an incomplete understanding of the topic. Makes mistakes when solving situational tasks. Tells not confidently.
9.	55-60	Satisfactorily "3"	Knows, tells not confidently. Has a partial view.
10.	54 and below	Unsatisfactory "2"	Has no exact idea. Does not know.

23- Practical lesson
Topic: Pemphigus. Clinic, diagnosis and treatment

Technological map of the practical lesson

Stages of work	Teacher	Student
1. Stages of preparation (10 minutes)	1. Controls the purity of the audience; 2. Checks students' readiness for classes; 3. Controls attendance;	Listen

2.The introductory stage to classes (15 minutes)	1.Preparation on the topic of content; 2. Preparation of slides for the introductory report; 3. To develop the literature used for the study of the subject;	Write down the topic and listen
3. The main stage (195 minutes)	1. Dividing students into 2 small subgroups, asking questions on the topic; 2. Use of slides and multimedia; 3. Carries out medical work; 4. Combines all information on a given topic, actively the participating students are encouraged and appreciated;	Divided into small groups watching, participating, listening. The student expresses his opinion, complements and asks questions

Тесты

Primary lesion element in pemphigus vulgaris
1) bubble
2) bubble
3) blister

A bladder with pemphigus vulgaris is formed
1) intraepithelial
2) subepithelial

Erosion with pemphigus vulgaris to epithelialization
1) prone
2) not inclined

Nikolsky's symptom with pemphigus vulgaris
1) positive
2) negative
Additional survey method required for staging
diagnosis of pemphigus vulgaris
1) bactrioscopic
2) cytological
3) histological
4) research in the rays of Wood
Diagnostic cells for pemphigus vulgaris
1) acantholytic
2) atypical
3) Langhans cells
4) giant multicore
The pathological process in the epithelium leads to the formation Tzanka cells
1) acanthosis
2) hyperkeratosis
3) parakeratosis
4) spogiosis
5) acantholysis
With pemphigus vulgaris, it is affected
1) only the mucous membrane
2) only leather
3) mucous membrane and skin
Pemphigus vulgaris differentiate
1) with herpes simplex
2) with exudative erythema multiforme
3) with leukoplakia
4) lichen planus, erosive form
5) with bullous pemphigoid
6) with atrophic candidiasis

Treatment leads to remission of pemphigus vulgaris
1) antibiotics
2) corticosteroids
3) anabolic steroids
4) tranquilizers
Pemphigus vulgaris is treated by specialists
1) oncologists
2) hematologists
3) dermatologists
4) endocrinologists
The tactics of a dentist in relation to a patient with a vulgar vesiculate (acute period)
1) sanitation of the oral cavity
2) the appointment of corticosteroid hormones
3) referral to a dermatologist
The tactics of a dentist in relation to a patient with a vulgar pemphigus (in remission)
1) sanitation of the oral cavity
2) correction of oral hygiene
3) adjustment of the maintenance dose of corticosteroids

Interactive method
USING THE WEB METHOD

Steps:
In advance, students are given time to prepare questions for the lesson.
Participants sit in a circle.
One of the participants is given a skein of thread, and he asks his prepared question (to which he himself must know the full answer), holding the end of the thread and throwing the skein to any student.
The student who received the skein answers the question (at the same time, the person who asked him comments on the answer) and passes the question relay on. Participants continue to ask and answer questions until everyone is on the web.
As soon as all students finish asking questions, the student holding the skein returns it to the participant from whom he received the question, at the same time asking his own question, etc., until the ball is completely "unwound".

Text

There are 4 clinical forms of pemphigus (pemphigus): vulgar; vegetative; leafy; seborrheic (erythematous).

Most often, the oral mucosa is affected with pemphigus vulgaris (in 75% of patients), so it is of greatest interest to dentists. Pemphigus vulgaris almost always begins with a lesion.

Rice. 11.44. Pemphigus vulgaris. Erosion on the mucous membrane of the tongue, lips, facial skin. Crusts on the red border of the lips.

the mucous membrane of the mouth or larynx, and then already spreads to the skin. Even if the disease begins with skin lesions, lesions of the oral mucosa are almost always subsequently detected.

VULGAR BLADDER (pemphigus vulgaris). Vulgar, or ordinary, pemphigus occurs much more often than other forms. Men and women over 35 years old are ill with this

disease, children are ill very rarely. Etiology. Unknown. There are viral and autoimmune theories of this disease.

The clinical picture. The defeat of the mucous membrane of the mouth and lips with pemphigus is characterized by the formation of blisters with serous or hemorrhagic contents, which have a very thin lining. Due to constant maceration in the oral cavity, the bubbles open very quickly, so it is extremely rare to see them in the oral cavity. The remnants of the former bladder are more often found. In place of the bubbles, round or oval erosions form, which do not heal for a long time. Erosions of a bright red color are located against the background of an unchanged or slightly inflamed mucous membrane (Fig. 11.44). The sizes of erosions in pemphigus are different - from a small abrasion to extensive surfaces of stagnant red color. As a rule, there is no plaque on the surface of erosion, or there may be a thin layer of easily removable fibrinous plaque. Most often, erosion is localized on the mucous membrane of the cheeks (especially in the retromolar region), the lower surface of the tongue, palate and the floor of the mouth. In the absence of treatment, new erosions appear, which, merging with each other, form extensive erosive surfaces without a tendency to healing. The pain is quite severe, most intense when eating and talking. Erosion quickly infects

rush, especially in the unsanded oral cavity. The addition of coccal, fungal flora and fusospirochetosis aggravates the patient's condition, a specific fetid odor from the mouth appears. Salivation intensifies. Saliva macerates the corners of the mouth, painful cracks appear. On the red border of the lips, in the corners of the mouth, blisters and erosion, covered with hemorrhagic crusts, may also appear. Sometimes there is hoarseness, indicating a lesion in the larynx.

On the skin, blisters with pemphigus occur mainly in places of friction with clothing, pressure, maceration (abdomen, back, inguinal folds, etc.). The erosion on the skin formed after opening the blisters is also very painful. Any touch of clothes, linen or bandages to them causes severe pain and forces the patient to be immobile for hours.

Nikolsky's symptom with pemphigus is usually positive. There are three types of it:

if you grab the lining of the bladder or the upper layer of the epithelium at the edge of erosion with tweezers and pull, then the epithelial film detaches on the apparently unchanged healthy mucous membrane and skin. The thin film of the epithelium is very fragile and can be easily dissected with tweezers;

rubbing an unaltered mucous membrane or skin between lesions leads to rapid blistering or erosion;

if you rub the areas located far from the affected area, then the upper layers of the epithelium also peel off from the underlying ones. The second and especially the third varieties of Nikolsky's symptom indicate an increase in the intensity of acantholysis.

With pemphigus, in addition to the skin and mucous membrane of the mouth, other mucous membranes (intestines, stomach, esophagus, pharynx), as well as internal organs and the central nervous system can be affected.

Pemphigus is characterized by a wavy course, periods of exacerbation are replaced by periods of remission, which rarely occur spontaneously, more often under the influence of treatment.

If the process began on the mucous membrane of the mouth, then later, in the absence of appropriate treatment, after 1-6 months, sometimes later it spreads to the skin of the trunk, face and extremities. In this case, the general condition of the patient

deteriorates sharply, intoxication increases, cachexia develops, and 1-2 years after the onset of the disease without treatment with corticosteroids, pain

nye die. However, due to the widespread use of corticosteroids, such cases are now rare. Corticosteroid therapy interrupts the progressive course of pemphigus, and the stage of remission begins.

Diagnosis. It is placed on the basis of clinical manifestations, positive Nikolsky's symptom, results of cytological examination and direct immunofluorescence reaction.

Cytological examination. The method of cytological examination of smears-prints or scrapings from the bottom of erosion is mandatory for the diagnosis of pemphigus. The presence of acantholytic cells in them confirms the diagnosis of acantholytic pemphigus. Acantolytic cells, or Tzank cells, are altered cells of the spiny layer. They have a round shape and are smaller in size than the normal cells of the spiny layer (Fig. 11.45). The nucleus is large relative to the entire cell, its diameter is 1/3 to 72 or more than the diameter of the cell, it is colored dark blue, often having from 1 to 6 or lighter nucleoles. The cytoplasm of cells of heterogeneous color: light blue around the nucleus and dark blue around the periphery. Acantholytic cells are characterized by polymorphism in color, size of cells and nuclei. There are giant multinucleated cells - "monsters". At the height of the disease, the number of acantholytic and multinucleated cells increases dramatically. They merge into a continuous conglomerate of polymorphic cells. During remission and during treatment with corticosteroids, the number of acantholytic cells decreases.

The cytological picture at. vegetative pemphigus does not differ from that of vulgar pemphigus. In seborrheic pemphigus, multinucleated cells, as a rule, are not found, acantholytic cells are found in smaller numbers, they are monomorphic.

Histopathological studies. It is shown that the main morphological changes in acantholytic pemphigus are acantholysis and edema, resulting in the formation of intraepithelial bubbles. Connections between the cells of the prickly layer are broken - the phenomenon of acantholysis, as a result of which the intercellular bridges melt, gaps form between the cells, and then bubbles. The bottom of such bubbles, as well as subsequently the surface of erosion, is lined mainly with acantholytic cells.

Differential diagnosis. Acantolytic (is

muddy) pemphigus must be differentiated from other bullous lesions of the oral mucosa:

^ exudative erythema multiforme;

^ pemphigoid;

^ drug allergy;

^ bullous form of lichen planus; And Duhring's dermatitis herpetiformis.

Differential diagnosis of acantholytic pemphigus with other diseases accompanied by the formation of blisters is based mainly on the localization of blisters in relation to the epithelium.

So, with exudative erythema multiforme, the bubbles are located subepithelially, Nikolsky's symptom is negative. In addition, exudative erythema multiforme is characterized by an acute onset, seasonality of relapses, pronounced inflammation of the oral mucosa, and short duration of the course.

With bullous pemphigoid, the bubbles are located under the epithelium, their cover is thick, therefore, their lifetime is longer. People over 60 years of age are more likely to

get sick with bullous pemphigoid. Nikolsky's symptom is negative, acantholytic cells are not detected.

Anamnesis (information on medication intake) and the results of allergological tests help to reveal the differences between acantholytic pemphigus and allergic medicinal stomatitis. After discontinuation of the drug - the allergen, stomatitis is quickly eliminated. Bubbles with medicinal stomatitis are located under the epithelium, Nikolsky's symptom is negative, there are no acantholytic cells.

With the bullous form of lichen planus, the bubbles are subepithelial, there is no acantholysis. There are multiple papules around the blisters or in other areas of the oral mucosa, typical of lichen planus.

Dühring's dermatitis herpetiformis is characterized by a subepithelial arrangement of blisters that first appear on the skin. Nikolsky's symptom is negative, acantholytic cells are absent. The blisters are characterized by a significant content of eosinophils in them, eosinophilia is also noted in the blood. Patients with Duhring's herpetic dermatitis have an increased sensitivity to iodine.

In some cases, with pemphigoid, lichen planus and other cystic diseases in the presence of a subepithelial location of the bubbles around them or erosions, the entire layer of the epithelium can quite easily exfoliate; located in the area of inflammation. In this case, the lining of the bladder is thick and ruptures with difficulty. This symptom is called a false Nikolsky symptom, or a symptom of perifocal subepithelial detachment.

Treatment. For the treatment of pemphigus, corticosteroid and cytostatic drugs are currently used. Prescribe prednisolone, methylprednisolone (metipred, urbazone), dexamethasone (dexazone), triamcinolone (polcortolone, kenacort). Treatment with corticosteroids in most patients leads to the disappearance of clinical signs of the disease. However, in the case of discontinuation of corticosteroids, as a rule, relapse occurs, therefore, treatment of patients with pemphigus should be continuous, even in the complete absence of clinical manifestations of the disease. Prednisolone is prescribed in shock doses, depending on the patient's condition, 50-80 mg per day, triamcinolone 40-80 mg, dexamethasone 8-10 mg. Such high, so-called shock doses, patients take until the formation of new bubbles and almost complete epithelialization of erosion stops, after which they slowly reduce the daily dose of the drug. The dose of prednisolone is first reduced by 5 mg every 5 days, later these periods are extended to 7-10 days. Reducing the daily dose is carried out until the minimum, the so-called individual maintenance daily dose, which is administered permanently, is determined. For prednisolone it is usually 10-15 mg, for dexamethasone 0.5-1 mg.

Long-term use of corticosteroid drugs causes various side effects in patients (increased blood pressure, increased glucose in the urine, osteoporosis of bone tissue, provokes thrombosis, etc.). In this regard, in order to reduce complications from corticosteroid therapy, it is recommended to limit food salt and water. Meals should be predominantly protein-based with limited fats and carbohydrates. Inside are prescribed potassium preparations (potassium chloride, asparkam), ascorbic acid, B vitamins, calcium preparations, thyrocalcitonin.

For the treatment of pemphigus, cytostatics are also used, mainly methotrexate. It is prescribed simultaneously with corticosteroids once a week, 35-40 mg.

Local treatment is mainly aimed at preventing secondary infection of erosion and ulcers. It

includes the use of pain relievers in the form of oral baths; antiseptic drugs in non-irritating concentrations; applications to the oral mucosa or lubrication with corticosteroid ointments. After each meal and before the application of corticosteroid-containing ointments, rinsing with warm weak solutions of potassium permanganate, 0.25% chloramine, 0.02% chlorhexidine solution, etc. is necessary. Careful sanitation of the oral cavity is important for the rapid epithelialization of erosions on the mucous membrane. With the defeat of the red border of the lips, applications and lubrication are performed with ointments containing corticosteroids and antibiotics, as well as an oil solution of vitamin A. In case of complications of pemphigus with candidiasis, antifungal drugs are used.

However, even with proper and timely treatment, the prognosis for true (acantholytic) pemphigus remains serious.

Evaluation Criteria for Monitoring

№	Progress in% and points	Grade	Student knowledge level
1.	96-100	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Shows high activity, creative approach when conducting interactive games. Correctly solves situational problems and test questions. Analyzes independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
2.	91-95	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Analyzes research results independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
3.	86-90	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI.

			Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
4.	81-85	Good "4"	The correct answer to questions about the clinic, diagnosis and treatment of TBI, but there are 2-3 inaccuracies, errors. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
5.	76-80	Good "4"	Correct, but incomplete coverage of the issue. The student knows questions about the clinic and diagnosis of TBI, but is not fully versed in determining the tactics of treatment. Correctly solves situational problems, but the rationale for the answer is not complete. Understands the essence of the issue. Knows, speaks confidently
6.	71-75	Good "4"	Correct, but incomplete coverage of the issue. Correctly solves situational problems, but the rationale for the answer is incomplete. Has 2-3 errors. Knows, tells confidently. Has accurate ideas.
7.	66-70	Satisfactorily "3"	Understands the essence of the issue. Correctly solves situational problems, but cannot substantiate the answer. Has accurate ideas on specific issues.
8.	61-65	Satisfactorily "3"	Has an incomplete understanding of the topic. Makes mistakes when solving situational tasks. Tells not confidently.
9.	55-60	Satisfactorily "3"	Knows, tells not confidently. Has a partial view.
10.	54 and below	Unsatisfactory "2"	Has no exact idea. Does not know.

24- Practical lesson
Topic: Pemphigoid clinic and differential diagnostics

Technological map of the practical lesson

Stages of work	Teacher	Student
1. Stages of preparation (10 minutes)	1. Controls the purity of the audience; 2. Checks students' readiness for classes; 3. Controls attendance;	Listen
2.The introductory stage to classes (15 minutes)	1.Preparation on the topic of content; 2. Preparation of slides for the introductory report; 3. To develop the literature used for the study of the subject;	Write down the topic and listen
3. The main stage (195 minutes)	1. Dividing students into 2 small subgroups, asking questions on the topic; 2. Use of slides and multimedia; 3. Carries out medical work; 4. Combines all information on a given topic, actively the participating students are encouraged and appreciated;	Divided into small groups watching, participating, listening. The student expresses his opinion, complements and asks questions

Тесты

Bullous pemphigoid usually occurs in older people.
A) 30 years
B) 40 years old
C) 50 years old
D) 60 years

When pemphigoid is affected.
A) skin and SOPR
B) the mucous membrane of the nose and genitals
B) upper respiratory tract
D) all of the above

The percentage and location of the lesion with pemphigoid.
A) in 10% OCPR

B) 23% skin and nose
B) in 18% of the genitals
D) there is no right answer

60. Pemphigoid is characterized by formation in the mouth ...
A) subepithelial blisters
B) erythematous pemphigus
C) candidiasis
D) there is no right answer

61. Pemphigoid erosions are epithelized through ...
A) 10-15 days
B) 12-16 days
C) 18-25 days
D) 20-30 days

62. Benign non-acantholytic occurs only ...
A) SOPR
B) on the genitals
B) skin
D) on the walls of the nasal mucosa

63. Dif. Diagnosis of bullous pemphigoid should be done ...
A) acantholytic pemphigus
B) Duhring's dermatitis herpetiformis
B) syphilis
D) A and B

64. The favorite localization of blisters in non-acantholytic pemphigus is ...
A) the border of the hard and soft palate
B) cheek and red border of lips
C) gums
D) there is no right answer

65. Mercurialism is ...
A) mercury poisoning
B) lead poisoning
C) bismuth poisoning
D) gold poisoning

.

Text

... This group of diseases includes diseases that are outwardly similar to pemphigus:
♦ Lever's bullous pemphigoid, or actually non-cantolytic pemphigus (according to ND Sheklakov);

♦ cicatricial pemphigoid, or muco-synechial bullous dermatitis, pemphigus of the eyes;

♦ benign non-acantholytic pemphigus of the oral mucosa only (according to BM Pashkov).

The etiology of pemphigoid is not fully understood. All types of pemphigoid are characterized by the absence of acantholysis in the epithelial cells, the subepithelial arrangement of the blisters, pronounced inflammation, the absence of acantholytic cells, and the negative Nikolsky symptom. There may be a positive symptom of subepithelial perifocal detachment.

Pemphigoid proceeds benignly, the general condition of patients suffers little, the prognosis for life is favorable.

BULLY PEMFIGOID (pemphigoid bullosa). The disease usually occurs in people over 50 years of age. The skin and mucous membranes of the mouth, nose, genitals are affected. In about 10% of cases, the disease begins with lesions of the oral mucosa.

The clinical picture. Characterized by education

subepithelial blisters in the mouth. Against the background of edematous and hyperemic mucous membrane of the mouth, tense blisters with serous or hemorrhagic contents, 5–20 mm in diameter, appear. They persist for several hours, sometimes days, then erosions, covered with fibrinous plaque, form in their place. Unlike acantholytic pemphigus, erosions are epithelized after 10-15 days without scarring and atrophy, but may reappear after a certain time. The favorite localization of blisters with non-acantholytic pemphigus is the border of the hard and soft palate, cheeks. The disease lasts for months and years and may disappear spontaneously.

Sometimes the pathological process can be localized on the gum. In this case, the mucous membrane of the gingival margin from the vestibular surface is hyperemic, edematous, bleeding (Fig. 11.46). Nikolsky's symptom in this case is often positive, but acantholytic cells are not detected. The course of the disease is persistent and prolonged.

Pathological anatomy. Subepithelial blisters are histologically determined. In the lamina propria of the mucous membrane, edema and inflammatory infiltration are noted (Fig. 11.47).

Differential diagnosis. Differentiation of bullous pemphigoid follows primarily from: A acantholytic pemphigus; ▲ Dühring's dermatitis herpetiformis.

Treatment. The most effective treatment for non-acantholytic pemphigus are corticosteroid drugs (prednisone 20-30 mg per day). In some cases, a good effect is achieved by combined treatment - prednisolone, broad-spectrum antibiotics, antimalarial drugs, multivitamins.

CUTTING PEMFIGOID (pemphigus cicatrical), or muco-synechial atrophy bullous dermatitis, pemphigus of the eyes. For a cicatricial pemphigoid, it is characteristic that at the site of the former blisters, scars, adhesions and atrophy are subsequently formed. The mucous membranes, mainly the conjunctiva of the eyelids, and sometimes the skin are affected. As a result, narrowing of the palpebral fissure can occur, immobility of the eyeball and blindness appear.

Treatment. Should be done in conjunction with an optometrist.

The same drugs are prescribed as for the treatment of bullous pemphigoid.

Forecast. For life, the prognosis is favorable, however, sometimes developing blindness and cicatricial deformities of the mucous membranes can significantly reduce the working capacity of patients.

BENEFICIAL NONACANTOLYTIC BLADDER ONLY OF THE ORAL MUCOSA. Described by BM Pashkov and ND Sheklakov in 1959. With this disease, for many years, bubbles appear only on the mucous membrane of the mouth, without appearing in other places.

Clinical features of the course do not differ much from non-acantholytic pemphigus. The blisters in this disease are smaller (3-10 mm in diameter), located subepithelially, without the phenomena of acantholysis; arise in

in some patients almost continuously, in others with remissions from several days to several weeks. Subjective complaints of patients are reduced to a feeling of a slight burning sensation and constriction of the mucous membrane. Erosions after blistering are rapidly epithelized without leaving scars and atrophy.

Treatment. Carry out antihistamines, small doses of corticosteroid drugs (15-20 mg prednisolone) in combination with antimalarial drugs. Prescribe vitamins C and P. Local treatment is the same as for pemphigus

Evaluation Criteria for Monitoring

№	Progress in% and points	Grade	Student knowledge level
1.	96-100	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Shows high activity, creative approach when conducting interactive games. Correctly solves situational problems and test questions. Analyzes independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
2.	91-95	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Analyzes research results independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.

3.	86-90	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
4.	81-85	Good "4"	The correct answer to questions about the clinic, diagnosis and treatment of TBI, but there are 2-3 inaccuracies, errors. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
5.	76-80	Good "4"	Correct, but incomplete coverage of the issue. The student knows questions about the clinic and diagnosis of TBI, but is not fully versed in determining the tactics of treatment. Correctly solves situational problems, but the rationale for the answer is not complete. Understands the essence of the issue. Knows, speaks confidently
6.	71-75	Good "4"	Correct, but incomplete coverage of the issue. Correctly solves situational problems, but the rationale for the answer is incomplete. Has 2-3 errors. Knows, tells confidently. Has accurate ideas.
7.	66-70	Satisfactorily "3"	Understands the essence of the issue. Correctly solves situational problems, but cannot substantiate the answer. Has accurate ideas on specific issues.
8.	61-65	Satisfactorily "3"	Has an incomplete understanding of the topic. Makes mistakes when solving situational tasks. Tells not confidently.
9.	55-60	Satisfactorily	Knows, tells not confidently. Has

		"3"	a partial view.
10.	54 and below	Unsatisfactory "2"	Has no exact idea. Does not know.

25- Practical lesson
Topic: Lichen planus etiology and pathogenesis. Clinic, diagnostics, treatment.

Technological map of the practical lesson

Stages of work	Teacher	Student
1. Stages of preparation (10 minutes)	1. Controls the purity of the audience; 2. Checks students' readiness for classes; 3. Controls attendance;	Listen
2.The introductory stage to classes (15 minutes)	1.Preparation on the topic of content; 2. Preparation of slides for the introductory report; 3. To develop the literature used for the study of the subject;	Write down the topic and listen
3. The main stage (195 minutes)	1. Dividing students into 2 small subgroups, asking questions on the topic; 2. Use of slides and multimedia; 3. Carries out medical work; 4. Combines all information on a given topic, actively the participating students are encouraged and appreciated;	Divided into small groups watching, participating, listening. The student expresses his opinion, complements and asks questions

Text

Lichen planus (lichen ruber planus) is a chronic inflammatory disease of the skin and mucous membranes, manifested by the formation of keratinized papules.

This disease is more common in women aged 40-60 years. With lichen planus, along with skin lesions, changes in the mucous membrane of the mouth and the red border of the lips are often noted. An isolated lesion of the oral mucosa is quite common. According to EI Abramova, simultaneous lesions of the skin and oral mucosa were noted in 25% of patients, and isolated localization on the oral mucosa without skin lesions was established in 75% of patients. Lichen planus is localized on other mucous membranes: genitals, anus, conjunctiva, esophagus, stomach, urethra.

Etiology. Not fully clarified. There is a non-progenous, viral, toxic-allergic theory of the occurrence of this disease. Lichen planus usually occurs against the background of

chronic diseases of the gastrointestinal tract, hypertension, diabetes mellitus, neuroses, imbalance of the immune system, etc. Most patients showed a significant increase in vascular wall permeability and a decrease in immunological reactivity. Trauma (sharp edges of teeth, poor-quality prostheses, galvanic phenomena, allergic reaction to plastic of prostheses, etc.) is of a certain importance in the development of lichen planus of the oral mucosa.

The clinical picture. The main morphological element of the lesion is a keratinized papule of a round or polygonal shape up to 2 mm in diameter. On the skin, papules are usually flat, with a waxy sheen, pinkish or bluish-red in color. On the oral mucosa, due to keratinization of the epithelium and constant maceration, they acquire a whitish-gray color, standing out against the background of a normal or hyperemic mucous membrane. A characteristic feature of lichen planus is the tendency of papules to merge in the form of a pattern resembling a lace mesh, snowflakes, tree-like branches, sometimes rings, stripes. Papules rise slightly above the level of the mucous membrane, giving it roughness. On the dorsum and lateral surface of the tongue, papules, merging, often form hyperkeratic plaques of various sizes, reminiscent of leukoplakia; the papillae in this area are smoothed. In smokers, papules are more pronounced and large, leukoplakia spots are often superimposed on them. On the red border of the lips, papules can merge, forming a whitish strip, sometimes taking a stellate shape. Lichen planus on the red border and mucous membrane of the lips often leads to glandular cheilitis. The most typical localization of lichen planus is on the mucous membrane of the cheeks at the place where the large molars fit with the transitional folds, on the lateral surfaces of the tongue and the back with a transition to the lower surface in the area of the large molars. Less commonly, the lips, gums, palate, floor of the mouth are affected.

Рис. 11.39. Типичная форма красного плоского лишая. Множественные папулы, образующие узор на слизистой оболочке щеки.

Rice. 11.39. Typical form of lichen planus. Multiple papules forming a pattern on the buccal mucosa.

Due to the variety of clinical manifestations of lichen planus in the oral cavity, the following forms are distinguished: typical (simple), exudative-hyperemic, erosive-ulcerative, bullous, hyperkeratotic.

Typical shape. More common than others. Whitish-nacreous papules are located separately or in the form of patterns, lace, fern leaves, rings, stripes on the apparently unchanged oral mucosa (Fig. 11.39). With such a typical picture of lichen planus, subjective sensations are minimally expressed and can be manifested by a burning sensation, tightness, roughness, dryness of the oral mucosa. Quite often, the disease is asymptomatic and can be detected by chance when examined by a dentist.

Rice. 11.40. Exudative hyperemic form of lichen planus. Against the background of severe hyperemia of the mucous membrane of the gums, multiple papules.

Exudative hyperemic form. Less common. Papules are located on a hyperemic, edematous mucous membrane (Fig. 11.40). This form is accompanied by more pronounced pain sensations, burning, pain, aggravated by eating spicy food, talking. Against the background of an inflamed, hyperemic mucous membrane, the pattern of papules can lose the clarity of their outlines and even partially disappear. But in the process of reverse development, when the edema and hyperemia of the mucous membrane decrease, the papule pattern reappears.

Erosive and ulcerative form. Heaviest of all forms. It can arise as a complication of typical and exudative-hyperemic forms as a result of erosion of the area of the affected mucous membrane by various traumatic factors (sharp edges of teeth, dentures, galvanic phenomena, etc.). With this form, on the hyperemic and edematous mucous membrane of the mouth there are erosions, sometimes ulcers, around which, against a background of pronounced inflammation, papules typical for lichen planus are located in the form of a pattern (Fig. 11.41). Erosions or ulcers of irregular shape, covered with fibrinous plaque, after removal of which bleeding easily occurs. They can be single, small, painful, but they can also be multiple with pronounced pain. Such erosion and ulcers last for a long time, sometimes for months, even without epithelialization for years. Often, under the influence of treatment, they partially or

completely epithelize, but soon they recur again in the same or another part of the mucous membrane, sometimes even immediately after stopping treatment. Sometimes in place of long-existing erosions and ulcers, there are areas of atrophy of the mucous membrane. In some cases, long-term erosions and ulcers can be aggravated.

Bullous form. It is rare. It is characterized, along with the typical rash of whitish-pearly papules, by the appearance of blisters with a diameter of 1 to 10 mm. Blisters can be with serous or hemorrhagic contents, they open rather quickly. Their life span is from several hours to 2 days. The erosion bubbles formed at the site of the erosion are quickly epithelized, which distinguishes the bullous form of lichen planus from the erosive-ulcerative one. The duration of the course of the bullous form can be different, sometimes blisters can appear for many months. Blisters can appear on the oral mucosa at the same time as papules or join them later. Sometimes they precede the appearance of papules, which makes it difficult to diagnose lichen planus.

Hyperkeratotic form. It is very rare.

Rice. 11.42. Hyperkeratotic form of lichen planus.
Extensive hyperkeratotic plaque on the dorsum of the tongue, around which there are multiple papules.

It is characterized by the presence of various shapes and outlines of hyperkeratotic plaques, towering above the level of the mucous membrane with sharp boundaries (Fig. 11.42). Around the foci of hyperkeratosis there are papular rashes typical of lichen planus. Most often, this form is localized on the mucous membrane of the cheeks and the back of the tongue.

The described forms of lichen planus can transform one into another. So, as a result of complications, the typical form can turn into exudative-hyperemic and erosive-ulcerative. The transformation process is determined by the influence of general (somatic diseases) and local factors. The presence of sharp edges of teeth and dentures, dissimilar metals, dentoalveolar anomalies and deformities, untreated periodontitis, tartar contribute to the transition of lichen planus from a typical form to a more severe one.

Lichen planus is a chronic disease characterized by a persistent long course. It can last for decades with alternating exacerbations and remissions, the duration of which is influenced by severe general diseases and the presence of local traumatic factors in the oral cavity.

Lichen planus on the oral mucosa can become malignant. More often this occurs in elderly people who have suffered from erosive-ulcerative or hyperkeratotic forms for a long time. Signs of a malignant

The formation of lichen planus is the formation of compaction at the base of the lesion, increased keratinization processes, the appearance of vegetation on the surface of long-term non-healing and non-curable erosions and ulcers.

Pathological anatomy. Pathohistological changes in the epithelium are characterized by the presence of hyper- and parakeratosis, as well as acanthosis (Fig. 11.43). In some cases, the formation of a granular layer is detected. In the stroma, edema, a diffuse inflammatory infiltrate (mainly from lymphocytes and plasma cells) are found, the cells of which penetrate through the basement membrane into the epithelium (exocytosis), as a result of which it is indistinctly distinguishable. The infiltrate almost never penetrates into the deeper layers and its own lamina of the mucous membrane, it comes close to the epithelium, as if supporting it from below. Depending on the type of lesion, some differences are determined. In case of erosive-ulcerative lesions, in addition to the phenomena of hyper- and parakeratosis, ulceration and destruction of the epithelium, hemorrhages in the mucous layer itself are noted. The infiltrate is determined directly under the basal membrane. Bullous lesions develop under the epithelium as a result of extensive edema in the connective tissue. The vesicles are located subepithelially, under them there is a massive round-cell infiltration into the lamina propria of the mucous membrane. Typical for

lichen planus epithelium changes in erosive-ulcerative and bullous forms are determined in areas bordering with a defect.

Diagnostics. In typical cases, especially in the presence of lesions on the skin, the diagnosis of lichen planus is not difficult. It is more difficult to make the correct diagnosis with an isolated lesion of the oral mucosa.

Differential diagnosis. Differential diagnosis of lichen planus must be carried out with:

▲ leukoplakia; And candidiasis;

▲ lupus erythematosus;

And papular syphilis; And allergic stomatitis; A chronic trauma; Bowen's disease.

In erosive leukoplakia, in contrast to lichen planus, the keratinization area looks like a solid grayish-white plaque slightly rising above the level of the mucous membrane, around which there is no inflammation, there are no papules. Unlike lichen planus, leukoplakia is localized mainly in the anterior parts of the oral cavity, more often in the mucous membrane of the corners of the mouth and cheeks.

Differential diagnosis with candidiasis is based on differences in clinical manifestations. With candidiasis there is no clear pattern of papules; when scraping, the white plaque is partially or completely removed, while the papules of lichen planus are not removed. The final diagnosis is made on the basis of bacterioscopic data.

Lesions on the oral mucosa with lupus erythematosus are localized mainly on the lips, palate, and less often on the cheeks. They are hyperemic, infiltrated, hyperkeratosis is present only within the focus of inflammation, in the center of which is atrophy, which does not happen with lichen planus. Syphilitic papules are much larger, round or oval in shape. Their surface is covered with a grayish-white bloom, which can be easily removed by scraping. Pale treponemas are found in smears from the surface of naked erosion.

From allergic stomatitis, lichen planus differentiates on the basis of anamnesis data, disappeared

changes in the lesion after elimination of the action of the allergen, the results of allergological tests.

It is necessary to distinguish lesions of the oral mucosa with lichen planus from similar changes under the influence of local traumatic factors, in which the area of the lesion corresponds to the action of the stimulus, after the elimination of which the focus of inflammation disappears.

The bullous and erosive-ulcerative form of lichen planus must also be differentiated from other diseases in which there are blisters and erosion. These are exudative erythema multiforme, acantholytic and non-acantholytic pemphigus, herpes simplex, erosive and ulcerative form of chronic lupus erythematosus.

The hyperkeratotic form must be distinguished from verrucous leukoplakia and cancer.

Treatment. Patients with lichen planus should undergo a thorough examination to identify somatic diseases. Particular attention is paid to the state of the gastrointestinal tract, the nervous system, to determine - indicators of blood pressure and blood glucose. If a pathology is detected, the patient is subject to treatment by the appropriate specialists.

All patients with lichen planus undergo a thorough sanitation of the oral cavity, which the dentist begins with the elimination of traumatic factors. Sharp edges of teeth and dentures should be sanded down. If there are dissimilar metals in the oral cavity, it is necessary to remove them or at least one of the dissimilar metals. Removable plate dentures should be made of colorless plastic with an inner elastic layer. It is advisable to replace amalgam fillings with cement ones, especially if their placement coincides with the period of lichen planus appearance. It is recommended to quit smoking and eating irritating food, and maintain good oral hygiene.

Drug therapy for lichen planus depends on its shape.

For all forms of the disease, it is advisable to prescribe sedatives (valerian, bromine preparations, tranquilizers). For typical and exudative-hyperemic forms, vitamin A is prescribed orally (3.44% solution of retinol acetate in oil or 5.5% solution of retinol palmitate in oil) 10 drops 3 times a day with meals, in courses of 1 , 5-2 months with 2-month breaks. In addition, long-term intake of B vitamins, especially niacin, is prescribed. Simultaneously oil

a solution of vitamin A is used for applications on the mucous membrane of the mouth in the affected area.

In exudative-hyperemic and erosive-ulcerative forms, effective use of 1% nicotinic acid, which is administered under the lesions, 1 ml together with 1% solution of novocaine or trimecaine every other day, for a course of 15-20 injections. If the injections are poorly tolerated by patients, nicotinic acid is administered orally at 0.05-0.1 g 3 times a day after meals, and under the lesions they do novocaine (trimecaine) blockade 2-3 times a week, for a course of 10- 12 injections.

A good effect is given by the appointment of tigazone (tigason) 10 mg 3 times a day, taken with meals and washed down with milk. After 2-4 weeks of treatment, in case of good tolerance, you can increase the dose: 25 mg 3 times a day. The entire course of

treatment lasts 6-8 weeks. In case of relapses, repeated courses of treatment are carried out.

For the treatment of the erosive and ulcerative form of lichen planus, honsuride is used, which is used in the form of applications 2-3 times a day or injections of 1 ml under the lesion element every other day (0.1 g of honsuride is diluted in 10 ml of a 0.5% solution of novocaine or isotonic sodium chloride solution). Honsurid stimulates the process of epithelialization of erosions and ulcers, has an anti-inflammatory effect.

With pronounced symptoms of inflammation of the oral mucosa, erosions or ulcers, anti-inflammatory rinses (chamomile solution, 0.5% chloramine solution, 0.02% furacilin solution, etc.) are applied topically with subsequent applications to the lesions of oil solutions of vitamins A, E, ointments Solcoseryl, Actovegin, corticosteroid-containing, 1% dibunol ointment. To increase the resistance of the oral mucosa and stimulate its regeneration, applications or oral baths with 0.25% lysozyme solution are used. Treatment of erosive-ulcerative and bullous forms at the Department of Skin Diseases of the Moscow Medical Institute is carried out using a combined method: prednisolone 20-25 mg per day every other day (triamcinolone 16-20 mg, dexametazone 3-3.5 mg), hingamin (delagil) 0.25 g 1-2 times a day for 4-6 weeks and nicotinic acid (0.05 g 3 times a day after meals) or xanthinol nicotinate (complamin, theonikol) 1 tablet 3 once a day or intramuscularly for 1.5 months. The dose of prednisolone is reduced by 5 mg every 7-10 days. Such treatment, in the absence of contraindications, accelerates the epithelization of erosions and ulcers, and reduces the inflammatory process. It is possible to treat with only one of these drugs (prednisolone or delagil), but it will be less effective. In the presence of limited erosive-ulcerative lesions, it is effective to inject them with a suspension of hydrocortisone (or a solution of prednisolone) and a 5-10% solution of hingamine. Injections are carried out 1 time in 3 days, 1 - 1.5 ml for each erosion, for a course of 8-12 injections. Such courses can be repeated in 3-4 months. When treating corticosteroid drugs, one should take into account their possible side effects, in connection with which during the treatment it is necessary to carry out a blood test (general clinical and glucose), measure blood pressure, prescribe potassium, calcium, multivitamins, and recommend a diet with a reduced amount of table salt.

If there are contraindications to the use of corticosteroid preparations, then in case of erosive and ulcerative lichen planus, it is recommended to conduct hyposensitizing therapy with repeated courses of histaglobin (several courses with a two-month break; 2 ml under the skin 2 times a week, for a course of 8-10 injection).

It should be remembered about the possibility of malignancy of long-existing and not amenable to treatment of erosions, ulcers, foci of hyperkeratosis. In these cases, their excision is necessary, followed by histological examination. Cryodestruction gives good results.

Lichen planus is a disease that does not always respond well to treatment. Relapses often occur after stopping treatment. The decisive importance in their prevention belongs to the elimination of local traumatic factors and the successful treatment of somatic diseases. In order to increase the body's defenses, repeated courses of vitamin therapy, auto-hemotherapy are carried out, pyrogenal, prodigiosan, thymogen and other agents are used in a hospital.

Evaluation Criteria for Monitoring

№	Progress in% and points	Grade	Student knowledge level
1.	96-100	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Shows high activity, creative approach when conducting interactive games. Correctly solves situational problems and test questions. Analyzes independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
2.	91-95	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Analyzes research results independently. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
3.	86-90	Fine "5"	Complete correct answer to questions about the clinic, diagnosis and treatment of TBI. Summarizes and makes a decision. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.
4.	81-85	Good "4"	The correct answer to questions about the clinic, diagnosis and treatment of TBI, but there are 2-3 inaccuracies, errors. Thinks creatively. Solves situational tasks correctly. Understands the essence of the issue. Knows, tells confidently. Has an accurate idea.

5.	76-80	Good "4"	Correct, but incomplete coverage of the issue. The student knows questions about the clinic and diagnosis of TBI, but is not fully versed in determining the tactics of treatment. Correctly solves situational problems, but the rationale for the answer is not complete. Understands the essence of the issue. Knows, speaks confidently
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