MINISTRY OF HIGHER EDUCATION, SCIENCE AND INNOVATIONS OF THE REPUBLIC OF UZBEKISTAN
MINISTRY OF HEALTHCARE OF THE REPUBLIC OF UZBEKISTAN
TASHKENT MEDICAL ACADEMY

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Pediatrics

Training manual

intended for students of foreign faculties
of higher medical educational institutions

TASHKENT 2024

UDK 616.248-053.2 **BBK** 54.12

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Reviewed and approved at a meeting of the Council of the Tashkent Medical Academy

Protocol No. " " 2024

The textbook was prepared for 3-4 year students of foreign faculty and contains information about common diseases in pediatric practice. The textbook was created on the basis of the requirements of the State educational standard and the educational (modular) program of the science “Pediatrics”. Appropriate for the pediatrics specialty educational programm.

Protocol of the committee was approved by the Academic Council of the Tashkent Medical Academy _____ _____ 2023.

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INTRODUCTION

n an effort to enable our country to join the ranks of developed countries, we are gradually transferring to an improved system of specialist training based on modern medicine, economics, science, culture and technology. We are perfectly aware that our esteemed President Sh. M. Mirziyoyev is concerned about improving the system of education and enlightenment, guiding young people to obtain modern knowledge, and bringing up a comprehensively developed personality.

The Presidential Decree of the Republic of Uzbekistan dated April 20, 2017, No. PQ-2909 "On Measures for Further Development of the Higher Education System" establishes the structure and content of the continuous education system based on modern scientific achievements and social experience, envisaging reforms. To achieve this, it is essential to ensure that the educational process in all forms of the educational system is advanced, scientifically and methodologically grounded with modern methodology. Enhancing the knowledge, skills, and qualifications of the faculty responsible for training medical personnel is one of the pressing tasks of the present.

The aim of education is determined by the development of the social community, the direction of its development, and the content of social relations. Today, the main goal of education organized in the Republic of Uzbekistan is the upbringing of a perfect individual.

This manual is prepared based on a modern approach to diagnosing common diseases among children. The authors of the manual aimed to illuminate the modern approach to diagnosing and treating common diseases.

The manual is intended for students of the 3rd and 4th years of the international faculty "Pediatrics". The manual is created based on the educational (module) program "Pediatrics".

Chapter I

Atypical Pneumonias in Children

Diagnosis and therapy of community-acquired pneumonia in children remain topical issues in pediatrics. The morbidity and mortality rates of this disease remain relatively high. Timely diagnosis and adequate therapy of pneumonia in outpatient settings, especially in young children, pose a significant challenge. In recent years, new data on the etiology of pneumonia have emerged, various disease courses have been identified, and optimal treatment programs have been developed.

Atypical pneumonia refers to a group of acute inflammatory lung diseases caused by rare pathogens and lacking typical symptoms. The European Respiratory Society distinguishes the following four types of pneumonia:

- Community-acquired;
- Secondary (nosocomial);
- Pneumonia in immunocompromised patients;
- Atypical pneumonia (caused by Mycoplasma, Chlamydia, and Legionella).

Epidemiology of community-acquired pneumonia: Worldwide, the incidence of community-acquired pneumonia is 10-12%, affecting approximately 1.5 million people annually. Viral pneumonia accounts for 90% of pneumonia cases in children and up to 39% in adults. Community-acquired pneumonia ranks 6th in mortality in the USA and affects 5% of the working-age population in Russia. According to WHO data, over 2 million children under 5 years old die from community-acquired pneumonia annually.

The clinical picture of atypical pneumonia is characterized by predominance of general toxic syndrome manifestations, while symptoms of bronchopulmonary-pleural syndrome take a back seat. Atypical pneumonia is characterized by epidemiological outbreaks (referring to outbreaks in children's, school, student, and military collectives), as well as familial clusters of respiratory diseases.

This methodological development not only covers classical atypical pneumonia (chlamydial, mycoplasmal, legionella), but also addresses issues of the etiology, clinical presentation, diagnosis, and treatment of cytomegalovirus and Pneumocystis pneumonia. The latter two are more commonly encountered in immunocompromised patients, but their rarity, poor prognosis, and predominance of general toxic manifestations unite Pneumocystis, cytomegalovirus, and classical "atypical" pneumonia.

1.1 Mycoplasmal Pneumonia

Mycoplasma pneumoniae is an independent genus of microorganisms characterized by small size (150-200 nm) and containing both RNA and DNA. The pathogen can replicate in cell-free media and produce toxin (β-hemolysin). Mycoplasmas occupy an intermediate position between viruses, bacteria, and protozoa. They can persist for years in a lipophilic desiccated state at temperatures of -70°C. Streptococcus B, anaerobes (peptostreptococci, bacteroids, pneumococci, staphylococcus aureus, Haemophilus influenzae) Symptoms of pneumonia vary from smoothed clinical presentation to aggressive rapid progression. Without specific treatment, negative consequences including fatal outcomes are possible.

The term "atypical pneumonia" was introduced into clinical medicine in the late 1930s, suggesting that this type of pneumonia differs in its etiology from pneumococcal pneumonia. In the late 1940s, the first pathogen causing atypical disease course, Mycoplasma pneumoniae, was identified. Subsequently, this list was supplemented by Legionella pneumoniae, Chlamydia pneumoniae, and ornithosis pneumonias.

In the overwhelming majority of cases, proper microbiological examination of children with pneumonia is not performed. Mycoplasmas and chlamydia account for 10-45% of cases of tonsillitis/pharyngitis in preschool children, and community-acquired pneumonia accounts for 11-40%. Recurrent bronchitis accounts for 15-20%, and exacerbations of chronic bronchopulmonary diseases occur in 30-40% of cases in children.

Mycoplasmal infection in newborns has a generalized nature, affecting the lung parenchyma. The bronchial and upper respiratory tract tissues remain intact because the pathogen penetrates hematogenously and has a tropism for lung tissue. Mothers of such newborns have a history of obstetric complications (urogenital mycoplasmosis). Children are born with low body weight, pale, with jaundiced skin. Pneumonia develops from the first hours of life. By the end of the first week, meningoencephalitis occurs. According to statistics, these children account for 10-30% of deceased newborns.

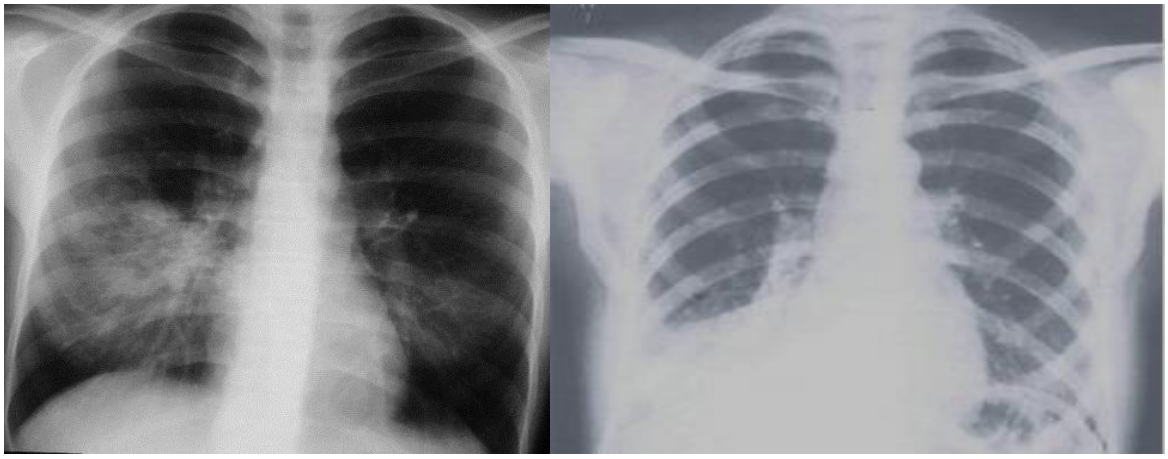
Diagnosis is based on the detection of the pathogen in nasal mucosa smears using fluorescent sera, serological reactions (RSC - increase in complement binding antibody titer). Mycoplasma culture isolation from sputum by seeding on tissue cultures or special media (complicated method) is performed.

Etiology. In the age range of 1-6 months, Chlamydia trachomatis (a consequence of perinatal infection) accounts for up to 20% of cases, and Pneumocystis carinii is relatively rare (in premature infants). Pneumococci and Haemophilus influenzae type b are found in 10% of children (contact with a patient with ARVI). The source of infection is a patient with respiratory mycoplasmosis and a carrier. Transmission routes: airborne-droplet, transplacental. Pneumococcus is the main (more than 50%) pathogen in children aged 6 months to 6 years, and Haemophilus influenzae type b accounts for up to 10% of complicated forms.

Pneumonias caused by M. pneumoniae are observed in no more than 10-15% of patients, Chl. pneumoniae - even less frequently. In the age range of 7-15 years, pneumococcus is the main bacterial pathogen (35-40%), the proportion of atypical pneumonia exceeds 50%, including M. pneumoniae (20-60%) and Chl. pneumoniae (6-24%). The main signs of atypical pneumonia are: small amount of sputum, minor changes on X-ray, mild leukocytosis in blood, severe headaches, high fever, muscle pain, severe weakness, inconsistency of intoxication severity with physical and radiographic data, lack of positive effect from sulfonamides and beta-lactam antibiotics.

Distinctive features of mycoplasmal pneumonia in children: The disease begins with the development of pharyngitis, tracheobronchitis. Subfebrile temperature, persistent cough.
Physical changes - lung percussion does not reveal changes, various rales may be heard, but not necessarily, regional lymph nodes enlargement. Hepatosplenomegaly.

Hematological data - normocytosis, leukocytosis, slight neutrophilia without a left shift in the formula, tendency to eosinophilia, anemia, thrombocytosis, accelerated ESR. The course of the disease is monotonous. Residual effects persist in the form of a dry persistent cough, subfebrility from 2.5 weeks to 2-2.5 months.



1.2 Chlamydial Pneumonia

Etiology. Chlamydia trachomatis. The source of infection is maternal birth canal, preceding conjunctivitis, often between 3 - 19 weeks of life, persistent cough, fever without intoxication, eosinophilia, significant infiltrative changes on X-ray. Distinctive features of chlamydial pneumonia in children. The causative agent is Chlamydia trachomatis. The main transmission routes in newborns are airborne-droplet, aspiration. Usually, the middle and lower parts of the lungs are affected. The pathogen, breaking through the protective barriers, reaches the alveoli, where it causes serous edema, spreading to adjacent lung areas.

Epidemiology: There is no clear seasonality during the course of the disease. For ornithosis pneumonia caused by Chlamydia psittaci, birds (pigeons, parrots, ducks, chickens) are the source of infection. Chlamydiosis is predominantly a pathology of newborns who become infected intranatally during passage through the mother's infected birth canal. In adults, this pathology is considered sexually transmitted infections.

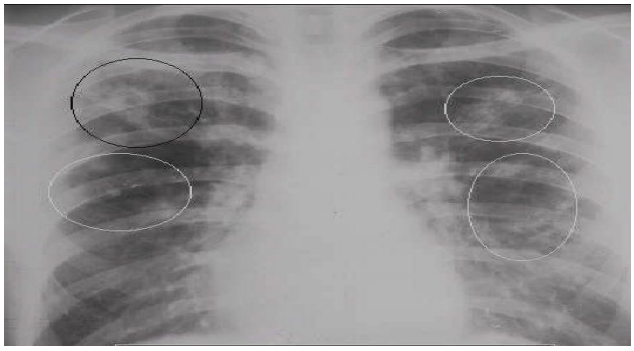
Clinic: The incubation period is 10 days. The onset is accompanied by a general infectious syndrome: weakness, fever (above 39°C), severe headache, bradycardia, muted heart sounds, muscle pains; and angina. Signs of respiratory organ involvement appear in 1-3 days: dry cough, pain in the side, chest. According to physical examination findings, there is a local shortening of the percussion sound, fine bubbling rales, not accompanied by exacerbation of intoxication and tendency to abscess formation. The clinic resembles influenza. The course is prolonged. Fever persists for up to 2 weeks, there may be recurrent waves, asthenization persists for 2-3 months. The prognosis is favorable.

Distinctive features of chlamydial pneumonia in children:

The onset of the disease - acute with fever or asymptomatic onset, without pronounced intoxication and fever, appearance of persistent cough.

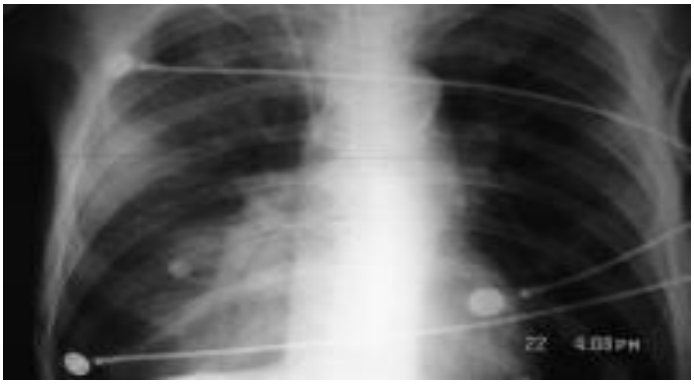
Physical changes - scant changes in the lungs.

Hematological data - hyperleukocytosis, relative neutrophilia with a left shift in the formula, eosinophilia, tendency to anemia and thrombocytopenia, accelerated ESR.



X-ray - Characteristic bilateral patchy shadows.

In newborns infected with chlamydia, unilateral conjunctivitis appears at the end of the 1st to 2nd week of life. The course is slow, and only after 1-2 weeks, mucopurulent discharge from the eyes occurs. During this period, pneumonia may develop (at 4-12 weeks of age), presenting with dyspnea and pertussis-like cough, in the absence of fever and intoxication. Characteristics of the course of chlamydial pneumonia include:



Chlamydial pneumonia

1.3. Legionellosis

Caused by various species of Legionella, is an acute infectious disease. *Etiology.* The genus Legionella includes 9 species: L. pneumophila, L. bozemanii, L. miedadei, L. dumoffii, L. longbeache, L. gonmanii, and others. These are gram-negative rods with pointed ends, 0.3-0.4 μm wide and 2-4 μm long, possessing flagella. They can persist in the external environment for up to a year and grow well on artificial nutrient media. The microbe contains a set of antigenic and toxic components.

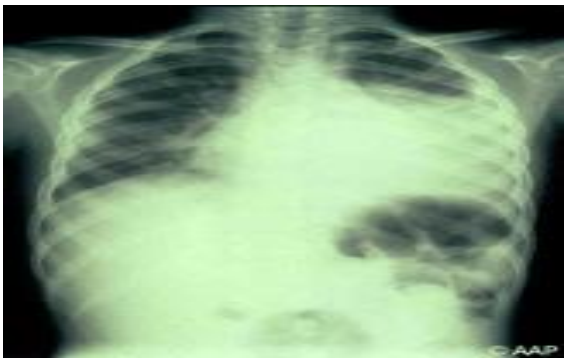
Epidemiology. The natural reservoir of the pathogen is soil. Legionellosis is widespread throughout the year, with epidemic outbreaks occurring in the autumn and summer and sporadic cases reported regardless of the season. Predisposing factors include living near construction sites, and the use of immunosuppressive drugs. Infection occurs via the airborne route. Outbreaks have been described in cases of inhaling tiny droplets of contaminated water formed in air conditioners and ventilators. Transmission from person to person has not been observed, but such a possibility cannot be ruled out.

Pathogenesis. The pathogen enters the upper respiratory tract and affects the bronchioles and alveoli, infiltrating the lung parenchyma. Macrophages and neutrophil leukocytes rush to meet it, destroying bacteria and promoting the release of endotoxin. As a result, parenchymal necrosis, alveolar fibrosis,

atelectasis, and pulmonary emphysema develop. The dissemination of bacteria, their endotoxin, and vascular-adrenal dysfunction cause changes in the cardiovascular, gastrointestinal, central nervous system, kidneys, and provoke the development of DIC syndrome.

Clinical Manifestations. Three variants of legionellosis are distinguished: acute pneumonia, acute alveolitis, acute bronchitis. Acute pneumonia is characterized by an acute onset, fever up to 39-40°C, vomiting, liquid stool, chills, myalgia, severe headaches - a systemic infectious syndrome. Lung involvement manifests as severe coughing and chest pain. The cough later becomes productive, with the expectoration of mucous "rice" sputum. Symptoms of CNS involvement may develop (delirium, hallucinations, dizziness). Acute alveolitis begins like acute pneumonia but progresses to dyspnea and the auscultation of abundant crackling rales over the lungs. In prolonged cases, fibrosing alveolitis of the Hamman-Rich type may develop. Acute bronchitis is rare.

Radiography. Massive infiltrative shadows of much greater intensity than those detected upon examination.



Laboratory and instrumental methods of patient examination.

In CBC (complete blood count), there is neutrophilic leukocytosis (10-15*10^9/L), lymphopenia, and an acceleration of ESR (erythrocyte sedimentation rate) to 60 mm/h or more. In urinalysis, there is proteinuria, hematuria, and cylindruria. Biochemical analysis of blood (BAK) reveals hyponatremia (less than 130 mmol/L). Liver dysfunction is noted in the absence of overt hepatitis (bilirubin levels and transaminase activity are elevated by 2 times, hypoalbuminemia).

Diagnosis is based on detecting specific antibodies in the patient's serum, finding bacteria in bronchial washings, sputum, pleural fluid, as well as material from deceased organs. A fourfold increase in antibody titer is considered diagnostic.

Treatment. Erythromycin in age-appropriate doses is most effective, as well as the tetracycline group and levomycetin. Detoxification, symptomatic therapy, and the use of immunocorrection are indicated.

1.4 Pneumocystis pneumonia.

The causative agent is Pneumocystis carinii, although its taxonomic classification is not yet definitively determined: traditionally, it has been referred to as a protozoan, although there is evidence suggesting that this organism belongs to the fungi. P. carinii is predominantly a pulmonary parasite, thriving in the alveoli of both humans and various animals.

The life cycle of the parasite includes the following stages: cysts - round or oval structures with a diameter of 5-8 μm and a three-layered membrane containing 8 sporozoites; rupture of the cysts and release of sporozoites; maturation of sporozoites into trophozoites and precysts, which then transform into mature cysts. The life cycle occurs within the alveoli, where trophozoites possess numerous extensions that adhere to the alveolar cell surface membrane.

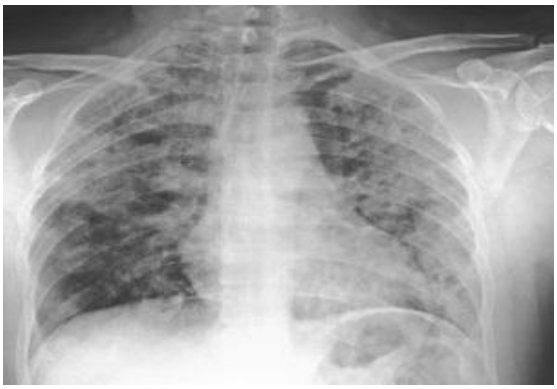
Epidemiology. The vast majority of people become infected with P. carinii in early childhood, as evidenced by the presence of antibodies against P. carinii antigens in virtually every individual. For immunocompetent individuals, this encounter does not lead to pathological consequences. However, in the context of immunosuppression (due to medication, chemotherapy, organ transplantation, stress), P. carinii infection often manifests as severe interstitial pneumonia.

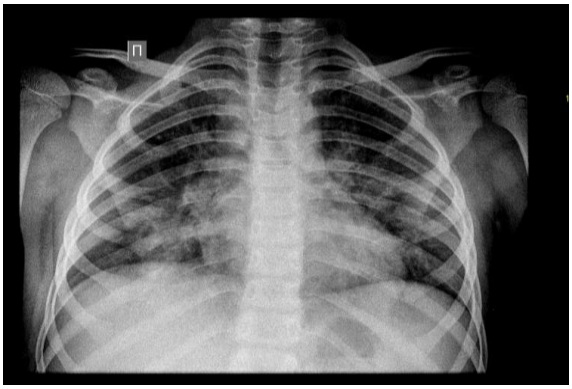
Clinical manifestation. The most common symptoms of Pneumocystis pneumonia include:

- Dry cough (80%)
- Shortness of breath (70%)
- Fever (above 38°C)

While crackles may be rare upon auscultation, they are not typical, and chest pain and sputum production can occur. Notably, there may be a discrepancy between the severity of clinical observations and physical examination findings.

On chest X-ray, the initial sign is an increased bronchial pattern. Subsequently, confluent opacities appear in both lung fields. In CBC, neutrophilic leukocytosis, eosinophilia, anemia, and elevated ESR are noted. The detection of P. carinii in frothy sputum or biopsy material holds diagnostic significance.





- ***Пневмоцистная пневмония*** ***Реб. 10 лет. Диагноз ВИЧ/СПИД, пневмоцистная пневмония***

Treatment:

Currently, there are two widely used and approximately equally effective treatment regimens for Pneumocystis pneumonia (PCP): trimethoprim-sulfamethoxazole (TMP-SMX) and pentamidine, along with several alternative regimens (dapsone, atovaquone, trimetrexate/leucovorin). Combined therapy with pentamidine and TMP-SMX does not offer any advantages. Both main drugs used for PCP treatment have numerous and not completely overlapping side effects.

Considering the high antibacterial activity of TMP-SMX, its use is more justified in the presence of concomitant bacterial infection. Pentamidine is the drug of choice if the patient has a history of allergy to sulfonamide drugs.

The mortality rate with adequately treated PCP is around 20% on average. The earlier the treatment is initiated, the greater the chances of success - if therapy is started when the chest X-ray is still normal or the arterial-alveolar oxygen gradient is below 30 mm Hg, mortality decreases from 45-55% to 10-15%.

1.5 Cytomegalovirus infection (CMVI)

It is most common in childhood. It is more often caused by influenza viruses, parainfluenza, respiratory syncytial virus, adenovirus. It is often complicated by a secondary bacterial infection. The most common viral pneumonia in immunodeficiency conditions is cytomegalovirus pneumonia (an opportunistic infection).

Cytomegalovirus infection (CMVI)

Etiology. The causative agent is Cytomegalovirus hominis from the Herpesviridae family, the virion diameter is 180 nm, and contains DNA. The virus is characterized by low virulence, the ability to lifelong persistence, pronounced immunosuppressive effect, transforming effect on the cell with slow replication. The virus can develop on human fibroblast cultures, as a result of which normal cells turn into cytomegalic (25-40

microns). The appearance of a large intracellular inclusion in transformed cells, separated from the karyolemma by a light rim, gives them the appearance of an “owl's eye".

Epidemiology.

The reservoir and source of infection is a person. The virus is found in blood, cervical and vaginal secretions, semen, breast milk, saliva, urine, feces, and lacrimal fluid. Infection occurs by the transplacental route (with acute or exacerbation of chronic CMVI in a pregnant woman; impaired barrier function of the placenta); contact, fecal oral, aerogenic, through breast milk; iatrogenic (transfusion of blood and its components, organ transplantation).

Pathogenesis.

Having penetrated primarily into the blood, CMV is reproduced in leukocytes (lymphocytes, monocytes) or persists in lymphoid organs. With the development of immunological insufficiency, the virus spreads with the blood flow to various organs and is filtered into liquid media and excretions. Specific changes develop in the affected organs, which determines the clinic.

Clinic.

Acquired CMVI more often occurs in the form of sluggish pneumonia. Congenital CMVI is always generalized in nature. From extrapulmonary lesions, encephalitis, hepatitis, sialoadenitis are noted; eye damage (chorioretinitis, cataracts, optic nerve atrophy), kidneys. According to clinical and radiological data, pneumonia in CMVI differs little from chlamydial, pneumocystic pneumonia. The main symptoms are tachypnea, dyspnea, paroxysmal cough, signs of hypoxia. Radiologically, hyperaeration and diffuse bilateral changes are detected. In the initial stage, the changes create background turbidity. Further, the infiltrate becomes denser, and enlightenments are visible against its background (an air bronchogram). In the UAC, progressive anemia with reticulocytosis, hemorrhagic syndrome, thrombocytopenia, jaundice.

Pathological anatomy.

The morphological picture of CMVI consists of two components: cytomegalic metamorphosis of cells and lymphohistiocytic infiltration of organ stroma. The more pronounced the immunodeficiency, the more cytomegalic cells and less pronounced lymphohistiocytic infiltration. In the lungs, cytomegalic transformation is mainly performed by cells of the alveolar macrophage system and the epithelium of the alveoli, especially those lining bronchovascular cases, interlobular septa, pleural leaflets.

The favorite localization of CMC (cytomegalic cells) are areas of adenomatous rearrangement of lung tissue in atelectases, around granulating ulcers, cysts, foci of pneumosclerosis. In the bronchial tree, CMCs are more often localized in the epithelium of respiratory bronchioles, less often in the epithelium of large bronchi, in the endothelium of the capillaries of the interalveolar septa and the own plate of the mucous membranes of the trachea and large bronchi. Infiltration of the pulmonary interstitium is pathognomonic for CMVI. Diffuse lymphohistiocytic infiltrates in the lungs with signs of interstitial pneumosclerosis and cytomegalic metamorphosis of the epithelium of the alveoli and bronchioles were observed in cases of chronic active CMVI. Similar changes (CMC, lymphohistiocytic infiltration with stroma sclerosis) are observed in the salivary glands, kidneys, and liver.

Diagnostics.

The simplest method is to identify CMC from saliva and urine sediments. Other methods: virus cultivation on fibroblast cultures, enzyme immunoassay and radioimmune, immune blotting are less reliable and expensive. It should be remembered that antibodies to CMV can be transmitted transplacentarly.

Treatment. Specific treatment consists in the use of the following drugs: 1. Nucleoside analogues (embedded in the genome of the virus and block the assembly of viral DNA). This is cytorabine (the daily dose is 100 mg / sq.m in 2 doses). The course is 4-10 days. 2. Zovirax (acyclovir). This drug blocks viral DNA, but does not block its own DNA. The dose for children under 2 years of age is 2.5 mg per kg, in case of severe infection, it is administered intravenously or 0.2 g 5 times a day.

3. Highly titrated gamma globulin from convalescent donors, placental gamma globulin in high doses, as well as immunoglobulins - pentoglobin, sandoglobulin can be used as immunosuppression therapy.

Prevention.

In order to prevent community-acquired pneumonia in children aged 2, 3 and 12 months, pneumococcal (polysaccharide or conjugate), 2,3,4 months hemophilus type b and influenza vaccines are used in children at risk. The expediency of vaccination is due to the fact that these pathogens are the main etiological cause of pneumonia. The duration of immunity is 3-5 years, and the incidence of complicated pneumonia decreases by 25%.

Social and hygienic measures and medical education of the population for the upbringing of a healthy lifestyle of the child: rational nutrition, massage, gymnastics, hardening, improvement of housing conditions, microclimatic conditions of the home (in particular, smoking cessation of parents; ventilation and cleaning of premises).

Control questions

1. Define atypical pneumonia in children;
2. Classification of atypical pneumonia in children;
3. Etiology and pathogenesis of the disease in young children;
4. What is the difference between atypical pathogens and typical ones?
5. Features of mycoplasma pneumonia in children
6. Features of pneumonia caused by chlamydia
7. Which viruses most often cause pneumonia in children?
8. How are the clinical and radiological features of pneumonia manifested?
9. Diagnostic criteria for pneumonia
10. Algorithm of antibacterial therapy of atypical pneumonia in children;
11. Tactics of GP in pneumonia in children.

Chapter II

Bronchial asthma

Bronchial asthma (BA) is the most common chronic respiratory tract disease in childhood. Its frequency continues to grow. The disease, which began in childhood, continues into old age. Long-term bronchial asthma can lead not only to disability of the child, but also to death.Bronchial asthma is an allergic disease characterized by repeated attacks of suffocation (bronchospasm). The disease is based on persistent allergic inflammation of the respiratory tract from the nasal mucosa to the smallest bronchi and bronchioles. The development of bronchial asthma is closely related to the effects of various factors.

Predisposing factors

Atopy: The body produces an increased amount of immunoglobulins E.

Bronchial hyperreactivity is an increased reaction to stimuli in the form of mild and rapid development of obstruction.

Heredity - the risk of developing asthma in a child whose parents have signs of allergy is 2-3 times higher than in a child whose parents are healthy.

Causal factors. Allergens - food, household, epidermal, pollen, fungal, medicinal, viruses, chemicals, vaccines. Under the influence of allergens, sensitization and allergies are formed in children. The formation of various types of sensitization occurs in a certain time sequence. In infants, sensitization to food and drug allergens is initially formed. At the age of 1 year and up to 5 years, sensitization to household, epidermal, fungal and pollen allergens is formed. By the age of 5, the child already has bronchial asthma with polyvalent sensitization. The leading role in the formation of asthma among household allergens is assigned to house dust mites. Favorable conditions for their reproduction are humid air, air temperature from 15 to 24 ° C. Their main habitats are upholstered furniture and beds, but they spread throughout the apartment: soft toys, rugs, wall carpets, household shoes, etc. Mites feed on scales of

the upper layer of human or pet skin, mold, hair, feather, plant food. The cover and secretions of ticks have allergenic properties. Ticks live for about 1 month. The allergenicity of dead ticks persists for months and even years.

The main route of penetration of household allergens into the body is inhalation. Therefore, asthma with household sensitization is characterized by year-round exacerbation, the predominance of attacks of suffocation at night or when cleaning the apartment.

Epidermal allergens include wool, down, feather, dandruff, secretions, saliva of pets and insects (cockroaches), dry fish food. It must be remembered that there are no non-allergenic animals.Pollen sensitization is caused by pollen from plants (oak, birch, poplar, maple, alder, cereals, weeds, etc.).

The effect of causal factors aggravate:

- Respiratory viral infections - viruses damage the mucous membrane, increase its permeability to allergens, and enhance bronchial hyperreactivity.

- Pathological course of pregnancy - contacts with allergens, occupational hazards, irrational nutrition, infectious diseases.

- Prematurity, irrational nutrition of the child

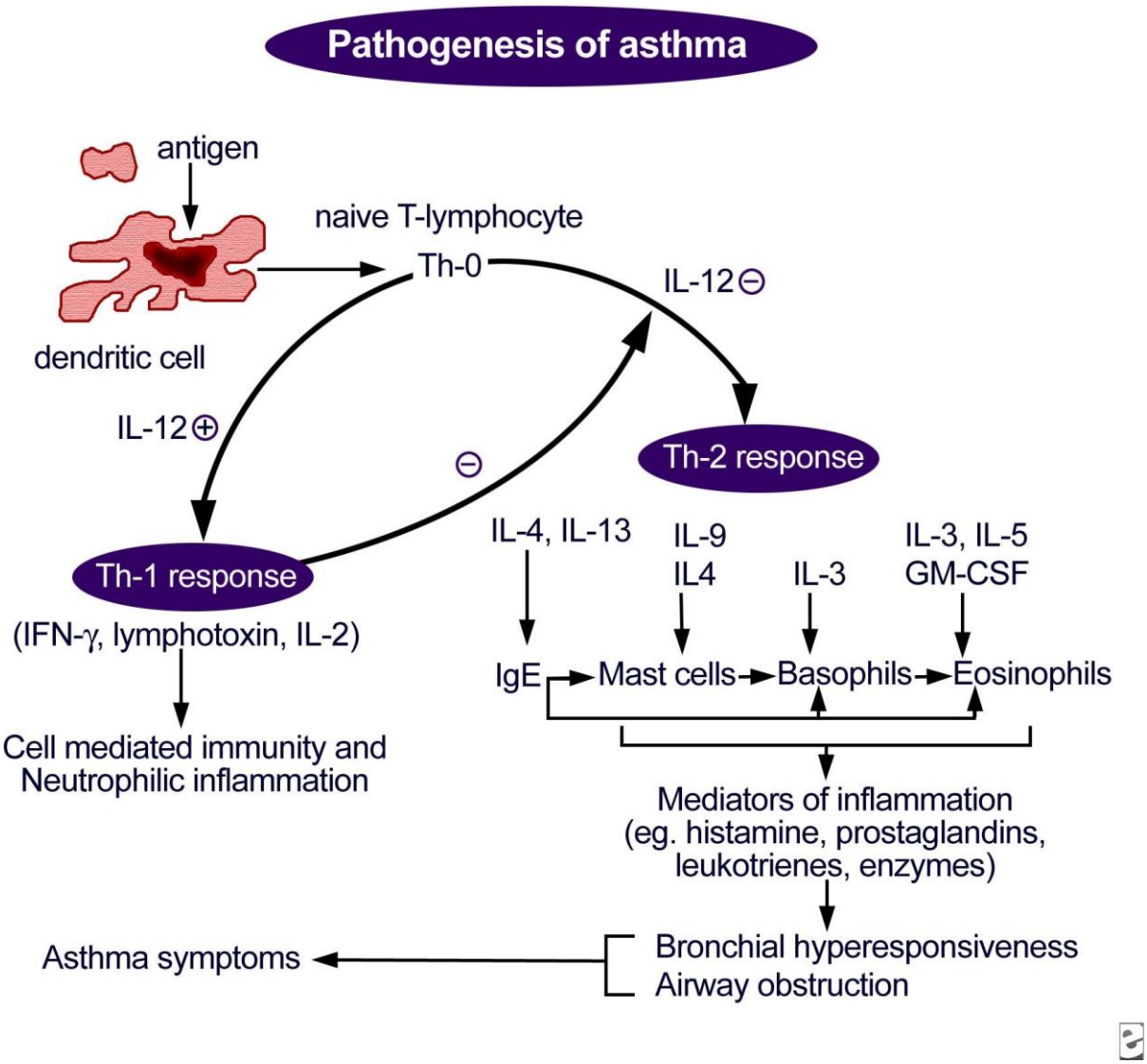
- early artificial feeding, earlier introduction of food products with sensitizing activity into the child's diet.

- The presence of allergic dermatitis and other allergic diseases.

- Passive or active smoking. 20% of children from families where one parent smokes develop asthma at an early age.

- Factors (triggers) that cause an exacerbation of asthma: Allergens, viral respiratory infections, physical exertion, emotional stress, changes in weather conditions.

Pathogenesis. According to the pathogenesis, bronchial asthma can be divided into two variants: immunopathological and nonimmune. Among the immunopathological forms in children, the main role is played by atopic asthma, which is realized according to the type I classification of Gell and Coombs. The reason for this variant is most often non-infectious allergens. The immunocomplex variant of the disease (type III according to Gell and Coombs) is much less common, in the etiology of which, along with non-infectious allergens, infectious ones can also play a role. In individual pathogenesis, these immunopathological forms of BA can be combined.



The non-immunological mechanisms of BA are based on nonspecific bronchial hyperreactivity, which may be genetically determined or acquired against the

background of the course of immunopathological forms of BA. One of the significant factors that increase the reactivity of the bronchi is acute and chronic bronchopulmonary infection. The mechanisms underlying the provoking effect of infection are multifaceted, but ultimately they amount to an increase in the permeability of epithelial barriers of target organs, a decrease in their resistance to the pathological action of specific allergens, and direct excitation of receptors of target organ cell membranes by an infectious agent. The provoking role of infection in children of the first years of life is great due to the presence of increased permeability of epithelial membranes, insufficiency of secretory immunoglobulins, and reduced ability to phagocytosis. The association of exacerbation of allergosis with intercurrent infections is often the basis for an erroneous statement of the infectious and allergic nature of the disease.

One of the modern directions of research on non-immunological mechanisms has been the study of the receptor and metabolic characteristics of patients with bronchial asthma. It has been shown that one of the characteristic features of the disease is the predominance of the cholinergic regulation system over the adrenergic one. This imbalance is determined at different levels: at the level of cell membrane receptors, the ratio of intracellular mediators — cyclic nucleotides (adrenergic system — cyclic adenosine monophosphate, CAMP; The cholinergic system is cyclic guanosine monophosphate (CGMP) and metabolic systems activated by CAMP– and CGMP - dependent mechanisms. It is these mechanisms that determine the ease of occurrence of bronchospastic reactions in asthma patients under the action of various exogenous and endogenous factors. In this regard, one of the common concepts of the etiopathogenesis of bronchial asthma is the so-called "beta-adrenergic blockade theory". It should be assumed, however, that in most cases the detectable changes in the ratio of the components of the cyclic nucleotide system are secondary to immunological ones and only potentiate the manifestation of the pathochemical and pathophysiological phases of immunological reactions. However, in some cases they are the leading link in pathogenesis,

this makes it possible to isolate non-immune BA ("pathoreceptor", with "primary altered bronchial reactivity"). A special variant of non-immune mechanisms is the pathogenesis of the so-called "aspirin bronchial asthma". Aspirin and substances close to it, by inhibiting the cyclooxygenase pathway of arachidonic acid metabolism (prostaglandin synthesis), thereby activate the second pathway of its metabolism — lipoxygenase (synthesis of leukotrienes, a slow-reacting substance of anaphylaxis — MRSA). MRSA, which has a powerful and long-lasting bronchoconstrictor effect, is the main endogenous factor that causes asthma exacerbation.

Classification. Classification of the severity of BA by clinical signs (the number of daytime symptoms per day/week, the number of night symptoms per week, the frequency of use of short-acting beta2-adrenomimetics, the values of PEF or FEV1 and daily fluctuations in PEF (variability) should be taken into account.

- Variants of the course of asthma: atopic, infection-dependent, neuropsychiatric, pathoreceptor and dishormonal.

Step 1: intermittent BA.

Symptoms occur less than once a week. Short exacerbations.

Nocturnal symptoms occur no more than 2 times a month.

- FEV1 or PEF ≥80% of the required values.
- Variability of PSV or OFV1 indicators < 20%.

Stage 2: mild persistent asthma.

Symptoms occur more often than once a week, but less often than once a day.

Exacerbations can affect physical activity and sleep.

Nocturnal symptoms occur more often than 2 times a month.

- OFV1 or PSV ≥80% of the required values.
- The variability of PSV or OFV1 indicators is 20-30%.

Stage 3: persistent moderate asthma.

Symptoms occur daily. Exacerbations can affect physical activity and sleep. Nocturnal symptoms occur more often than once a week.

Daily intake of inhaled B 2 short-acting agonists

actions.

- OFV1 or PSV from 60 to 80% of the required values.
- Variability of PSV or OFV1 indicators > 30%.

Stage 4: severe persistent asthma.

Symptoms occur daily.

Frequent exacerbations.

Frequent nocturnal symptoms

Restriction of physical activity

- OFV1 or PSV ≤60% of the required values.
- Variability of PSV or OFV1 indicators > 30%.

Asthma Classification

	Symptoms		≤5 years of age	>5 years of age	
	Daytime	Nighttime	Exercise tolerance	PEF or FEV1	PEF variability
Mild intermittent	≤2 per week	≤2 per month	Excellent tolerance	≥80%	<20%
Mild persistent	>2 per week, but <1 per day	>2 per month	Exercise symptoms	≥80%	20%-30%
Moderate persistent	Daily symptoms	>1 per week	Frequent exercise symptoms	60%-80%	>30%
Severe persistent	Continual day symptoms	Frequent night symptoms	Exercise severely limited	≤60%	>30%

The presence of at least one sign of the severity of the condition allows you to identify a child in this category. Children with intermittent asthma, but with severe exacerbations, should receive therapy, as with persistent moderate asthma. Children with any degree of severity, even with intermittent asthma, may have severe exacerbations. This type of classification, based on severity, is important in a situation where it is necessary to decide on starting therapy when assessing the patient's condition.

Bronchial asthma in children of this age is characterized by high clinical variability. However, at the same time, it retains all the features of an allergic hereditary disease. The onset of bronchial asthma in 70-80% of patients occurs in early childhood.

The early development of bronchial asthma in children may be facilitated by intrauterine sensitization of the fetus due to increased permeability of the fetoplacental barrier due to various influences. Adverse occupational hazards, active and passive smoking have a possible impact. Among the factors of postnatal sensitization, excessive antigenic exposure in the first 2 years of a child's life is important.

In children of the first year of life, the main sensitizing factors are food allergens. The most significant sensitization is to chicken protein, cow's milk, wheat and other cereals, fish, nuts, cocoa, citrus fruits and other yellow-red colored fruits, berries, and vegetables. From the end of the first year of life, the role of household allergens increases. Sensitization to allergens of house dust and mites is determined at this age in the vast majority of patients. From the age of 3-4, the role of pollen allergens increases as causally significant allergens, the spectrum of which varies in different climatogeographic zones.

Although this phasing in the change of the spectrum of sensitization in young children is characteristic, it is not always observed. Recently, children with bronchial asthma have increasingly experienced early sensitization to a wide range of allergens.

Due to the fact that the possibilities of allergological diagnosis in young children are limited (provocative inhalation tests are not carried out at this age, and skin tests are less sensitive in them — there is often no blister at the site of scarification and the only sign of a positive reaction may be erythema), the role of a carefully collected anamnesis is great.

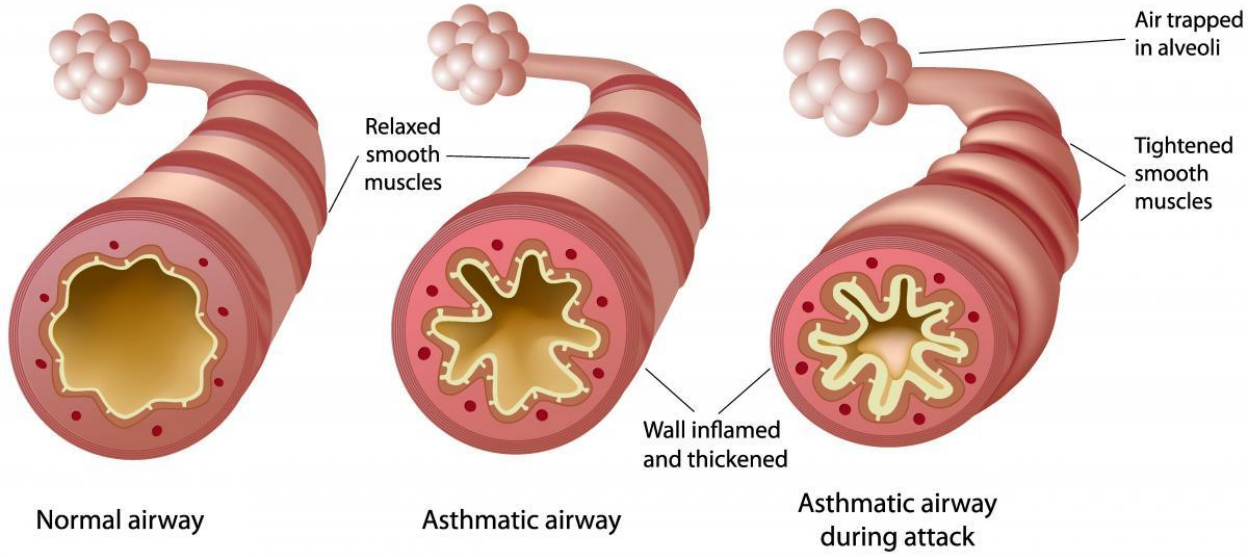
A favorable background for the early development of bronchial asthma in children is perinatal damage to the central nervous system due to the pathological course of pregnancy and childbirth, ante- and intrapartum fetal hypoxia, birth injuries. More than half of patients with bronchial asthma in the first year of life have signs of perinatal encephalopathy of posthypoxic and/or traumatic origin. At least 25% of sick children

have neurological disorders that persist at an older age, leaving an imprint on the course of bronchial asthma.

A detailed analysis shows that in most children, the appearance of the first typical attacks of expiratory dyspnea is usually preceded by manifestations of atopic dermatitis, acute allergic reactions to various foods, medicines, preventive vaccinations (in the form of exanthemums, urticaria, Quincke's edema), repeated respiratory diseases occurring without intoxication and hyperthermia, but with pronounced catarrhal phenomena. Such initial manifestations of respiratory allergies are often regarded by doctors as acute respiratory viral infections, bronchitis, and pneumonia. At the same time, patients are inadequately treated with antibiotics, which only contributes to drug sensitization, manifested by various clinical variants of drug allergies

Typical asthma attacks in early childhood usually develop after contact with a causally significant allergen. At the same time, the appearance of precursors of an attack in 1-2 days in the form of behavior changes (tearfulness, irritability or lethargy), decreased appetite, increased itching of the skin and other manifestations of skin allergies is characteristic. Coughing gradually turns into an obsessive dry cough. Attacks of suffocation develop at any time of the day and are clinically manifested by expiratory or mixed shortness of breath with a predominance of the expiratory component with retraction of the compliant places of the chest, its emphysematous bloating, obsessive (sometimes to vomiting) dry or unproductive wet cough, diffuse dry wheezing in the lungs, as well as widespread wet wheezing of different caliber. Noisy wheezing can be heard from a distance, paleness of the skin is pronounced, cyanosis of the nasolabial triangle, acrocyanosis are noted. In the dynamics of the attack, a dry cough naturally gives way to a wet one. Incomplete or frequent repeated attacks of bronchial asthma in young children can lead to the development of asthmatic conditions. The above-described variant of the seizure period is characterized by a significant peculiarity of clinical and functional manifestations. Children may not complain, their well—being may not suffer significantly - they are, as it were, adapted to constant respiratory failure. Expiratory shortness of breath is not pronounced, but it increases with the slightest

physical effort, the child is worried about a constant unproductive cough, the chest is swollen, scattered wheezes are heard in the lungs against the background of weakened breathing, pallor of the skin, acrocyanosis are pronounced.



Against this background, infection, the development of an acute attack, and other stressful situations can lead to a sharp deterioration of the condition. Depletion of the function of the adrenal cortex and a predisposition to the development of asthmatic conditions in young children require the vigilance of the attending physician and timely adequate therapy.

Due to the anatomical and physiological characteristics of young children (narrow lumen of the bronchial tree, poorly developed muscle layer, significant development of blood and lymphatic vessels), bronchospasm is usually not the leading mechanism in the pathogenesis of bronchial asthma attacks. In the first place is inflammation of the bronchial mucosa, its edema and hypersecretion of mucus, which, according to S.G. Zvyagintseva (1958), causes a peculiar clinical picture of "wet asthma" at this age, less severity and longer duration of attacks. At the same time, along with dry lungs, a large number of different-sized wet wheezes are heard, which persist for a long time, for 5-10 days or more. This course of the disease — in the form of asthmatic bronchitis — often causes overdiagnosis of infectious and inflammatory lung diseases and underdiagnosis of bronchial asthma at this age. Currently, it has been proven that this is a variant of the course of bronchial asthma, typical for young children.

With the age of the child and the duration of the disease, the factors provoking asthma attacks may be physical or psychoemotional stress, tobacco smoke, inhalation of cold, humid or polluted atmospheric air, and other non-specific irritants.

The most frequent provocateur of bronchial asthma attacks in early childhood are acute respiratory viral infections, which have a powerful sensitizing effect on the body, manifested both in increasing the permeability of the damaged mucous membrane of the respiratory tract to various aeroallergens, and in connection with the antigenic properties of the viruses themselves, immunological restructuring of the macroorganism during the infectious process. Provocation of attacks by acute respiratory viral infection, a similar clinical picture, functional and laboratory changes similar to those in obstructive bronchitis (bronchial obstruction in which is mainly associated with infectious causes) make differential diagnosis very difficult. Hiding for a long time under the mask of "acute respiratory viral infections with bronchoobstructive syndrome", "recurrent obstructive bronchitis", bronchial asthma at an early age is often not recognized and patients receive irrational treatment. It was only years later that a number of children showed the transition of the so-called obstructive bronchitis into typical bronchial asthma. According to the results of long-term observations (8-10 years or more after hospitalization due to severe bronchoobstructive syndrome in ARVI), more than half of these children suffer from typical bronchial asthma, not recognized at an early age. At the same time, early diagnosis and timely initiation of appropriate therapy largely determine the outcome of the disease.

Unlike patients with obstructive bronchitis, young children with bronchial asthma are characterized by pronounced hereditary burden of allergic diseases (especially on the maternal side); high frequency of allergic reactions to food, medicines, preventive vaccinations; more pronounced skin allergic manifestations; rapid course of bronchoobstructive syndrome with early onset during acute respiratory viral infections and shorter duration; distinct effect of bronchodilator therapy; pronounced immunological changes. However, the extremely high variability of both clinical signs and laboratory parameters makes it difficult to use them for differential diagnostic

purposes. Practically none of these indicators individually (including the concentration of IgE) can serve as a sufficiently reliable differential diagnostic criterion for obstructive bronchitis and bronchial asthma.

2.1 Modern diagnostic methods:

Skin tests to identify significant allergens: radioimmune and enzyme immunoassay methods for determining total immunoglobulin E and specific immunoglobulins in blood serum; spirometry to assess respiratory function in children over 5 years old; exercise test to detect bronchial hyperreactivity; peak flowmetry - monitoring of PSV (peak exhalation rate) to assess the severity BA and control of the effectiveness of prescribed therapy; sputum examination; lung radiography; bronchoscopy.

Medical history and physical examination

When collecting anamnesis, it is necessary to clarify the following details.

The presence of atopic dermatitis, allergic rhinoconjunctivitis, or a burdened family history of asthma or other atopic diseases.

The presence of at least one of the following symptoms:

- cough, which increases mainly at night;
- recurrent wheezing;
- repeated episodes of difficulty breathing;
- recurrent feeling of tightness in the chest.

The appearance or intensification of symptoms: at night;

in contact with:

- animals;
- chemical aerosols;
- house dust mites;
- pollen;
- tobacco smoke;
- at ambient temperature fluctuations;
- when taking drugs (acetylsalicylic acid, beta-blockers);
- during physical activity;

- with ARVI;
- under severe emotional stress;

During a physical examination, it is necessary to pay attention to the following signs characteristic of asthma:

- Hyperexpansion of the chest.
- Elongation of exhalation or wheezing during auscultation.
- Dry cough.
- Rhinitis.
- Periorbital cyanosis is the so–called allergic shadows (dark circles under the eyes due to venous congestion that occurs against the background of nasal obstruction).
- A transverse fold on the back of the nose.
- Atopic dermatitis.

In children under the age of 5, the diagnosis of AD (asthma disease) is based mainly on the results of a clinical examination. In infants who have had 3 or more episodes of wheezing associated with exposure to triggers, asthma should be suspected, examinations and differential diagnosis should be carried out.

Laboratory and instrumental studies

➢ *Spirometry*. In children over 5 years of age, it is necessary to determine FEV1, FVC and the ratio of FEV1 /FVC. Spirometry makes it possible to assess the degree of obstruction, its reversibility and variability, as well as the severity of the disease. When assessing the indicators of FEV1 and FVC, it is important to take into account ethnic characteristics and age gradations. With normal lung function, the ratio of FEV1 to FVC is more than 80%, and in children, perhaps more than 90%. Any values below these may suggest bronchial obstruction. The diagnosis of AD is also supported by an increase in FEV1 by at least 12% after inhalation of a bronchodilator or in response to trial therapy of HA.

Peak flowmetry (determination of peak expiratory velocity) is an important method of diagnosis and subsequent monitoring of the treatment of asthma. The latest models of peak flow meters are relatively inexpensive, portable, made of plastic and are ideal for use by patients over 5 years old at home for the purpose of daily objective monitoring of the course of asthma. When assessing the indicators of PEF in children, the growth of the child is necessarily taken into account (there are special normograms), but daily monitoring of PEF for 2-3 weeks is more informative to determine an individual indicator. PEF is measured in the morning, when the indicator is at the lowest level, and in the evening before bedtime, when PEF is usually the highest, and in the case of bronchodilators, before and after taking them. Keeping diaries to record symptoms, peak flowmetry results, and treatment plays an important role in AD treatment strategy. Monitoring of PEF can be informative for determining the early symptoms of an exacerbation of the disease. The daily spread of PEF indicators by more than 20% is considered as a diagnostic sign of AD, and the magnitude of deviations is directly proportional to the severity of the disease. The results of peak flowmetry indicate in favor of the diagnosis of AD if the PEF increases by at least 15% after inhalation of a bronchodilator or with a trial appointment of GC.

Detection of respiratory tract hyperreactivity

In patients with symptoms characteristic of asthma, but with normal lung function, studies of the respiratory tract response to exposure to methacholine, histamine or physical activity can help in diagnosing asthma. In the diagnosis of AD, these tests have high sensitivity, but low specificity.

In some children, symptoms of asthma are provoked only by physical activity. In this group, it is useful to conduct a load test (a 6-minute running load protocol). Using this test in conjunction with the determination of FEV1 or PSV may be useful for making an accurate diagnosis of AD.

Laboratory tests

- Skin tests or the determination of specific IgE in blood serum are not informative enough for the diagnosis of AD, but these studies help to identify risk factors and

triggers, on the basis of which appropriate control of environmental factors can be recommended.

Differential diagnosis

- Differential diagnosis of AD most often has to be carried out with the following diseases.
- Dysfunction of the vocal cords (pseudoasthma).
- Bronchiolitis.
- Aspiration of a foreign body or milk in infants.
- Cystic fibrosis.
- Primary immunodeficiency.
- Syndrome of primary ciliary dyskinesia.
- Tracheo- or bronchomalacia.
- Vascular malformations that cause external compression of the respiratory tract.
- Stenosis or narrowing of the respiratory tract associated with the presence of hemangiomas or other tumors, granulomas or cysts.
- Obliterating bronchiolitis.
- Interstitial lung diseases.
- Congestive heart defects.
- Tuberculosis.
- Bronchopulmonary dysplasia.
- Lobular emphysema.
- In the presence of the following symptoms, a disease other than BA should be suspected.

Anamnesis data:

- the appearance of symptoms of the disease at the age of 2 years;
- respiratory distress syndrome and/or ventilator use;
- neurological dysfunction in the neonatal period;

- lack of effect from the use of bronchodilators;
- wheezing associated with feeding or vomiting;
- difficulty swallowing and/or recurrent vomiting;
- diarrhea;
- poor weight gain;
- maintaining the need for oxygen therapy for more than 1 week after an exacerbation of the disease.
- Physical data:
- deformity of the fingers in the form of "drumsticks";
- heart murmurs;
- stridor;
- focal changes in the lungs;
- crepitation during auscultation;
- cyanosis.
- The results of laboratory and instrumental studies:
- focal or infiltrative changes on the chest X-ray;
- anemia;
- irreversible airway obstruction;
- hypoxemia

2.2 Treatment of bronchial asthma

The goals of AD therapy are to achieve and maintain control over the disease.

Treatment of bronchial asthma in children is aimed at:

Preventing the development of life-threatening conditions and death;

Elimination or minimization of clinical manifestations;

Normalization or improvement of respiratory function indicators;

Restoration or maintenance of vital activity, including exercise tolerance;

Reducing the need for bronchodilators;

Prevention of side effects of therapy;

Prevention of disability.

The comprehensive asthma treatment program includes the following components:

Education of sick children and their parents: they should know the goals of treatment, possible ways to achieve them, methods of self-control, inhalation techniques, self-help in case of an incipient attack

Identification and removal (elimination) of factors that provoke an exacerbation of the disease

Rational use of drugs that prevent exacerbation (anti-inflammatory) and alleviate the symptoms of the disease during exacerbation (bronchodilators)

Specific immunotherapy (specific allergovaccination)

Restorative treatment using non-medicinal methods, including sanatorium-resort

Regular medical supervision with correction of therapy.

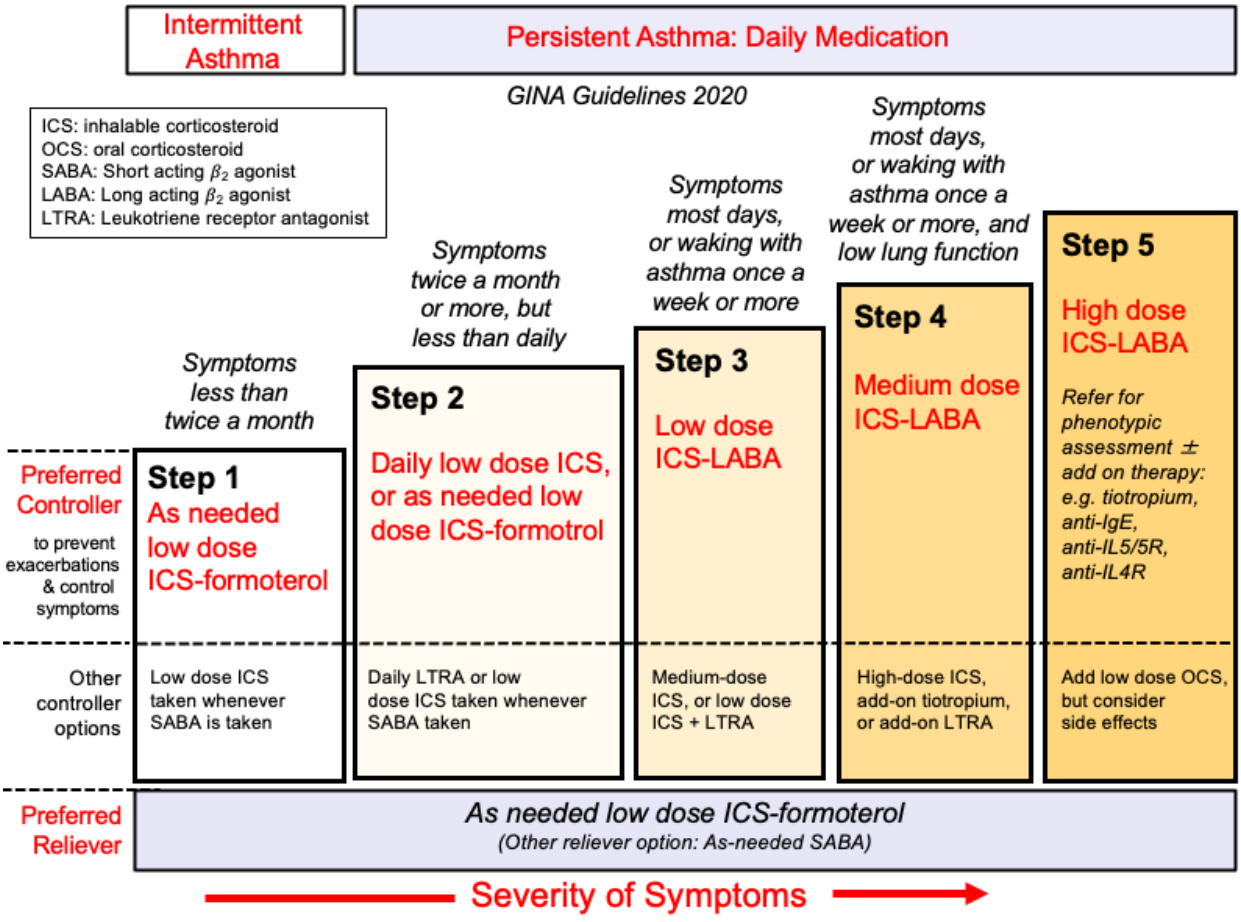
Currently, the principle of stepwise therapy is used in the treatment of AD, i.e. the use of medications in a clear dependence on the severity of the disease. The dose and frequency of medication are increased as the severity of the disease increases or reduced when symptoms are relieved.

Often, anti-inflammatory drugs are prescribed in combination with bronchodilators of prolonged action (β2-adrenomimetics or theophylline preparations).

Primary care at the outpatient stage for a mild attack of asthma includes inhalation of a rapid-acting β2-adrenomimetic from a dosing aerosol inhaler. In moderate to severe attacks, β2-adrenomimetic is used repeatedly at intervals of 20-30 minutes for an hour, then every 4 hours (if necessary). Preference is given to nebulizer therapy. The bronchodilating effect can be enhanced by the addition of apratropium bromide. If the effect is insufficient, prednisone is prescribed orally (1-2 mg / kg / day before 1 year, 10-20 mg at 1-5 years) or parenterally. Budesonide suspension is effective for the treatment of exacerbation of asthma, it can be diluted with saline solution, as well as mixed with solutions of bronchodilators (salbutamol, ipratropium bromide). The dose in children is 0.25-0.5 mg (up to 1 mg) 2 times a day.

In patients with severe exacerbation, not all symptoms may appear in the clinical picture. The appearance of at least one – cyanosis, "mute lung", general weakness, PSV of less than 30% should attract the attention of a doctor to resolve the issue of hospitalization. If the therapy is ineffective, it is necessary to urgently send the patient to the hospital within an hour.

Rational and timely administration of basic therapy for asthma and allergic rhinitis using the principle of a step-by-step approach can ensure long-term remission and prevent the progression of allergic disease.



<u>*Prevention.*</u> *Primary prevention* is aimed at preventing the occurrence of the disease in high-risk individuals. Currently, there are no prenatal measures that could be recommended for the primary prevention of asthma.

Postnatal prevention includes the following measures.

Breastfeeding should be encouraged, its benefits include a protective effect against the occurrence of wheezing at an early age.

Parents of children who smoke should be warned about the side effects of smoking on the child, including an increase in the frequency of wheezing in infancy; they need to be provided with adequate support in the process of quitting smoking. Exposure to tobacco smoke both prenatally and postnatally has an adverse effect on the course of diseases accompanied by bronchial obstruction.

Secondary prevention is aimed at children who have proven the presence of sensitization, but there are no symptoms of AD yet. These are children from risk groups, for the formation of which the use of the following predictors (signs characterizing a high risk of developing AD) is recommended:

Family history of asthma or allergies (the risk of asthma is up to 50%, especially if the heredity is burdened on the mother's side).

The presence of other allergic diseases in the child (atopic dermatitis, allergic rhinitis, the risk of asthma is 10-20%).

An increase in the level of total IgE of more than 30 IU/ml in combination with the detection of specific IgE-AT to cow's milk/chicken egg proteins, to aeroallergens of more than 0.35 IU/ml (in a child with atopic dermatitis or allergic rhinitis, the risk increases to 70%).

For the secondary prevention of AD in risk groups, preventive therapy with cetirizine (ETAC, X) is proposed. The only proven preventive antiallergic effect is shown in the ETAC study, which demonstrated that the administration of cetirizine at a dose of 0.25 mg / kg / day for 18 months to children from the high-risk group (with a burdened allergic history and skin manifestations) with household or pollen sensitization leads to a decrease in the frequency of bronchial obstruction from 40 to 20%. Other earlier studies (X) have established the protective role of specific immunotherapy in risk groups, but the most significant epidemiological study ("Preventive Allergy Treatment Stu-dy") has not yet been completed.

Tertiary prevention is aimed at reducing the effects of provoking factors to improve AD control and reduce the need for drug therapy.

Elimination mode. Compliance with the elimination regimen can help reduce the severity of an existing disease. Frequent contact with allergens in sensitized patients contributes to increased symptoms of asthma, bronchial hyperreactivity, and deterioration of lung function.

Of the allergens with which a person comes into contact in everyday life, allergens of house dust mites, animals (having wool or fur), cockroaches and fungi should be isolated.

Measures to reduce exposure to allergens of house dust mites.

It is necessary to put an impermeable coating on mattresses, pillows and blankets.

Carpets or carpeting should be replaced with linoleum or wooden floors or parquet.

All bedding should be washed weekly in hot (55-60 ° C) water.

Carpets must be treated with acaricides and/or tannic acid.

For cleaning, it is advisable to use a vacuum cleaner with a built-in HEPA filter and a dust collector with thick walls.

Soft toys need to be washed in hot water or periodically frozen. The use of home air ionizers does not lead to a decrease in the severity of asthma symptoms.

It is necessary to eliminate mold foci and prevent high humidity in the apartment throughout the year.

Measures to reduce contact with allergens of pets.

The animal should not be allowed into the bedroom or main living room.

It is necessary to replace carpets or carpeting with linoleum or wooden floors or parquet.

For cleaning, it is advisable to use a vacuum cleaner with a built-in HEPA filter and a dust collector with thick walls.

Even after the complete removal of animals from the house, it may take many months before the concentration of the allergen decreases to acceptable values.

The occupation of an apartment by cockroaches is an important cause of allergic sensitization, especially in urban homes. However, cockroach control measures have only a partial effect.

Food allergies are rarely a factor in exacerbation of asthma, mainly in young children.

Control questions

1. Define BA in children;

1. Classification of bronchial asthma?

2. What risk factors contribute to the development of bronchial asthma. 3.What are the pathogenetic mechanisms of bronchial asthma development?

4. What are the symptoms characteristic of the initial manifestations of the attack period of bronchial asthma?

5. With what diseases are differential diagnostics performed?

6. What are the principles of treatment of bronchial asthma in children?

Chapter III.

Acute rheumatic fever

Acute rheumatic fever (ARF) (synonym – rheumatism, disease Sokolsky – Buyo) is a systemic inflammatory disease of connective tissue with a predominant localization of the pathological process in the cardiovascular system (CVS), developing in people predisposed to it, mainly young people, due to an infection caused by hemolytic streptococcus group A. Currently, a decrease in morbidity is recorded in Uzbekistan. The prevalence is 0.79 per 1000 children. The maximum incidence is at the age of 10-14 years.

Etiology and pathogenesis.

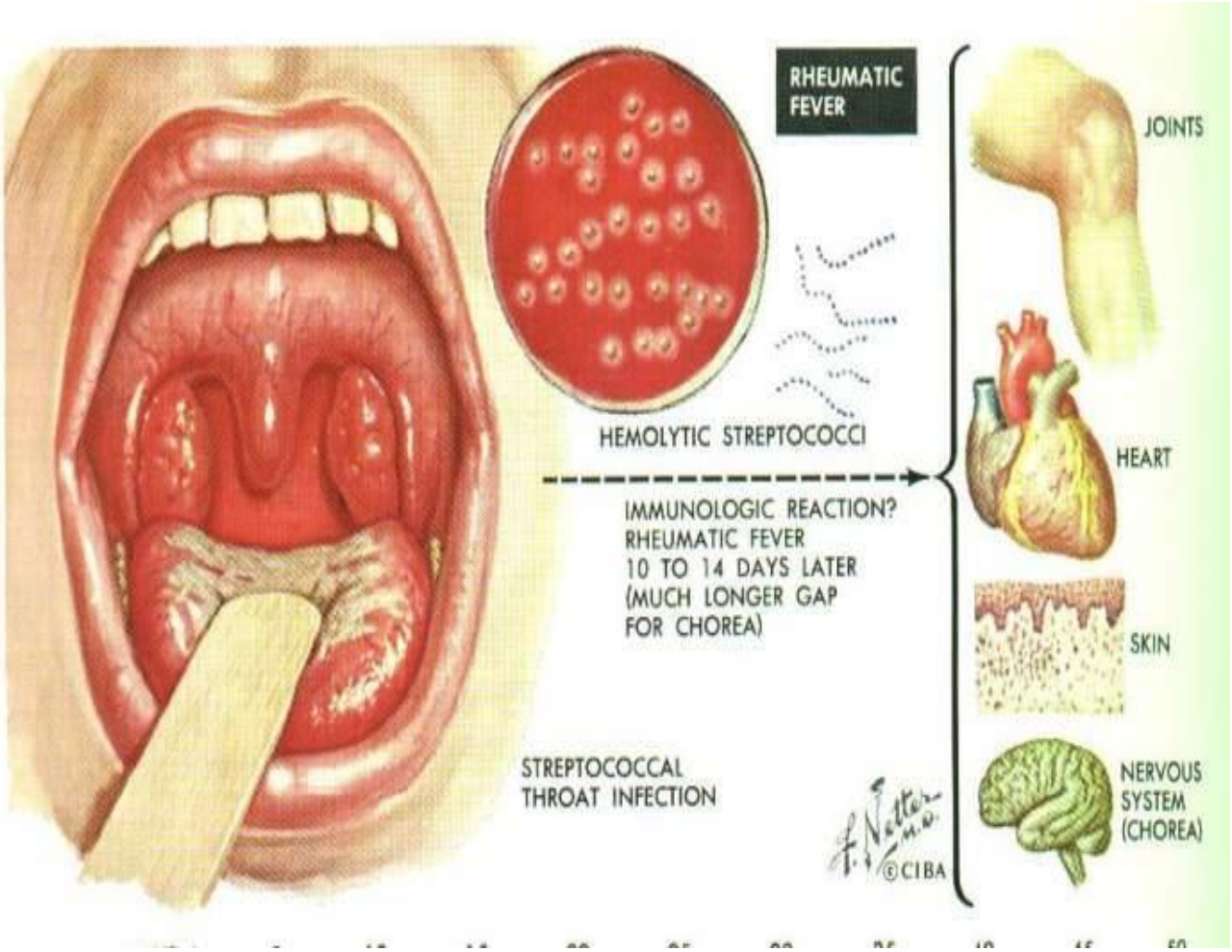
The results of epidemiological data, clinical observations, microbiological, immunological and experimental studies convincingly show the association of streptococcal nasopharyngeal infection (β-hemolytic streptococcus group A) with ARL. In the latter forms of rheumatism, there is no increase in the titers of streptococcal antibodies, and bicillin prophylaxis of rheumatism relapses is not effective. In this regard, many researchers question the role of streptococcal infection in the development of latent, prolonged and recurrent forms of rheumatism. An assumption is made about the allergic (unrelated to streptococcus or other infectious antigens), infectious-toxic or viral nature of these forms of diseases. An important role in the development of the disease is played by the individual sensitivity of the body to streptococcal infection, which is obviously associated with genetically determined changes in humoral and cellular immunity. In rheumatism, antibodies that react with heart tissue are often detected, in particular antibodies that cross-react with both myocardial antigens and streptococcal membranes.

Evidence of the development of autoimmune processes in rheumatism is a violation of humoral and cellular immunity in relation to the antigenic components of connective tissue - structural glycoproteins, proteoglycans, water-soluble components of connective tissue. In rheumatism, an imbalance of immunocompetent peripheral blood cells is revealed; an increase in the total number of lymphocytes due to an increase in the percentage and absolute number of B lymphocytes with a decrease in the percentage and absolute number of T lymphocytes; circulating immune complexes, a shift in the level of immunoglobulins. The body's immune response to one or another

antigenic factor is controlled by genes linked to the HLA tissue compatibility system.

Morphologically, the following phase changes of connective tissue are distinguished in rheumatism: mucoid swelling, fibrinoid changes, necrosis, cellular reactions (infiltration by lymphocytes and plasmocytes, formation of Aschoff-Talalaev granulomas), sclerosis. The rheumatic process ends, as a rule, with sclerosis.

The limitation or reversibility of the process can only be discussed at the stage of mucoid swelling. In the recurrent course of rheumatism, the resulting changes in connective tissue are localized most often at the site of sclerosis or new areas of connective tissue are involved in the process.



3.1 Diagnostic criteria for acute rheumatic fever

Classification of acute rheumatic fever

The modified Jones criteria (2015)

LR populations

Major criteria	Minor criteria
1. Carditis Clinical and/or subclinical 2. Arthritis Polyarthritis only 3. Chorea 4. Subcutaneous nodules 5. Erythema marginatum	1. Polyarthalgia 2. Fever (≥38.5°C) 3. ESR ≥ 60 mm/h and/or CRP ≥3.0 mg/dL 4. Prolonged PR interval after accounting for age variability

Moderate risk to HR populations

Major criteria	Minor criteria
1. Carditis Clinical and/or subclinical 2. Arthritis Monoarthritis or polyarthritis Polyarthralgia 3. Chorea 4. Subcutaneous nodules 5. Erythema marginatum	1. Monoarthralgia 2. Fever (≥38°C) 3. ESR ≥30 mm/h and/or CRP ≥3.0 mg/dL 4. Prolonged PR interval after accounting for age variability

CRP, C-reactive protein; *ESR*, erythrocyte sedimentation rate.
For all patient populations with evidence of preceding Group A streptococcal infection.
Diagnosis of initial ARF: 2 major manifestations or 1 major plus 2 minor manifestations.

Clinical manifestation of rheumatic fever depends primarily on the severity of the process, the severity of exudative, proliferative phenomena, the nature of damage to organs and systems, the duration of the patient's request for medical help from the onset of the disease and previous treatment. In typical cases, the first attack of rheumatism occurs 1-2 weeks after an acute or exacerbation of chronic streptococcal infection (sore throat, pharyngitis, acute respiratory infections). The disease can also develop after severe cooling without prior infection. In some patients, it is not possible to establish a connection between the onset of rheumatism and any effect. Relapses of rheumatism

often occur with intercurrent diseases or after them, surgical interventions, neuropsychiatric and physical stresses. There are 3 periods in the development of the rheumatic process:

The first period (from 1 to 3 weeks) is usually characterized by an asymptomatic course or mild malaise, arthralgia. There may be nosebleeds, pallor of the skin, subfebrile body temperature, increased ESR, titers of streptococcal antibodies (ASL-0, ASG, ASK), ECG changes. This is a pre-disease, or preclinical stage of the disease. During the described period, an immunological restructuring of the body occurs after a streptococcal infection. If this period is recognized, active therapeutic and diagnostic measures could obviously prevent the development of the disease.

The second period is characterized by the appearance of the disease and is manifested by polyarthritis or arthralgia, carditis or damage to other organs and systems. During this period, changes in laboratory, biochemical and immunological parameters, mucoid swelling or fibrinoid disorders are observed. Timely recognition of the disease and appropriate treatment can lead to a complete recovery (if diagnosed in the first 1-7 days from the onset of the disease).

The third period is a period of various clinical manifestations of recurrent rheumatism with latent and continuously recurring forms of diseases.

At the first episode of ARL, its acute course with the involvement of joints in the process, the patient can indicate not only the day, but also the hour of the onset of the disease. In such cases, the disease begins with a rise in temperature to subfebrile or febrile (38-40 ° C), chills and severe joint pain. Due to polyarthritis, the patient may be immobilized. Shortness of breath appears due to heart damage. A similar clinical picture is observed more often in children and young men.

Heart damage in ARL is the leading syndrome. ORL without obvious cardiac changes is rare. The rheumatic process can affect the endocardium and pericardium, but most often develops according to the type of endomyocarditis. The clinical manifestations of the disease depend on the predominance of the inflammatory process in one or another layer of the heart. Due to the fact that in practice it is often difficult to

distinguish the symptoms characteristic of rheumatic myocarditis, endocarditis or pericarditis, the term “rheumocarditis” is used, which means simultaneous damage by the rheumatic process of the myo- and endocardium, which is more often observed at the first attack of rheumatism in the first weeks from its onset. Despite the difficulties, it is still desirable to clarify the localization of the process.

Myocarditis is a mandatory component of rheumocarditis. In about 2/3 of patients, it is certainly combined with endocardial damage. There are primary rheumocarditis, reflecting the initial manifestations of rheumatism, and recurrent, which occurs with relapses of the rheumatic process, more often against the background of damage to the valvular apparatus of the heart.

The diagnosis of ARL is based on the Kissel-Jones diagnostic criteria, revised by the ARA and recommended by WHO for widespread use.

The diagnostic criteria for rheumatic carditis are:

1) pain or discomfort in the heart area;

2) shortness of breath;

3) heartbeat;

4) tachycardia;

5) weakening of the tone at the top of the heart;

6) noise at the top of the heart:

a) systolic (mild, moderate, strong);

b) diastolic;

7) symptoms of pericarditis;

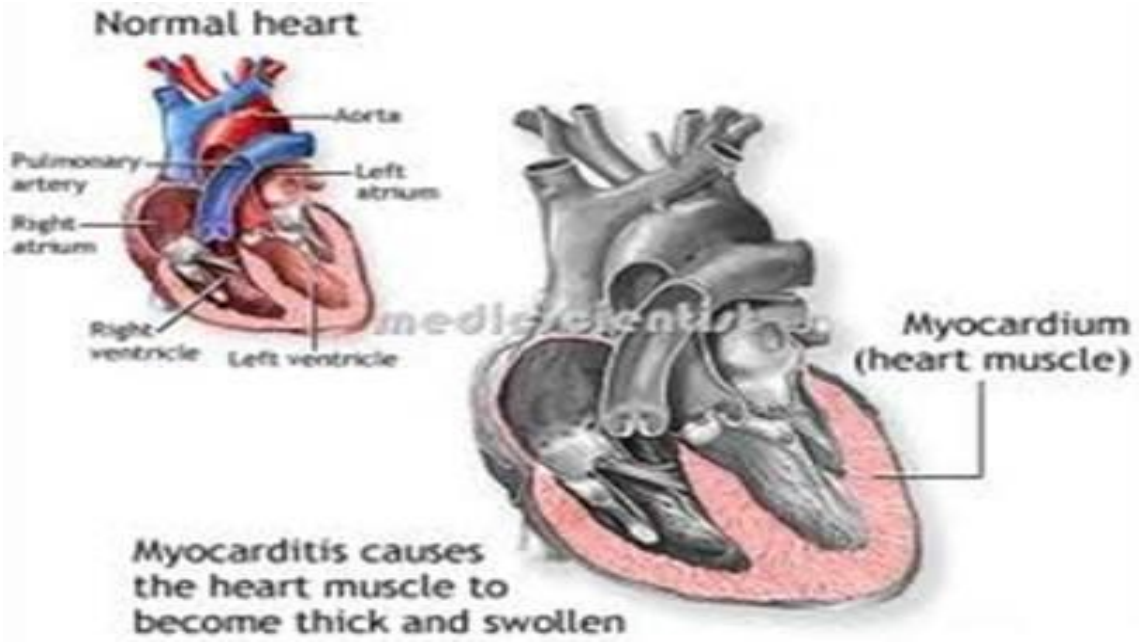
8) increasing the size of the heart;

9) ECG data: a) prolongation of the P-Q interval;

b) extrasystole, nodular rhythm; c) other rhythm disturbances;

10) symptoms of circulatory insufficiency;

11) reduction or loss of working capacity.



The presence of 7 out of 11 criteria in the patient in combination with a previous streptococcal infection makes it possible to make a reliable diagnosis of rheumocarditis. In recent years, the clinical picture of rheumatism has changed significantly: there are many low-symptom forms that occur mainly with damage to the heart (carditis) and joints (arthritis or arthralgia).

Laboratory indicators of the activity of the inflammatory process are poorly expressed. Of the main criteria, the most reliable are carditis and polyarthritis due to a streptococcal infection. Chorea is rare, ring-shaped erythema is observed in 2-5% of patients with active rheumatism, subcutaneous rheumatic nodules are generally not detected in vivo. In this regard, there is a need to develop new diagnostic criteria for primary rheumatism, taking into account the changed features of its course.

The clinical manifestations of the disease are extremely poorly expressed. Pain in the heart area is more often aching or stabbing. They can be anginous, such as angina pectoris, with irradiation to the left shoulder and arm. Such pains are characteristic of damage to the rheumatic process of the coronary vessels and the development of coronaritis. Some patients may be disturbed by a feeling of heart failure. Persistent tachycardia is typical for rheumatic carditis, which persists for a long time after normalization of body temperature and improvement of general condition. The pulse is characterized by great lability, especially after exercise or with negative emotions.

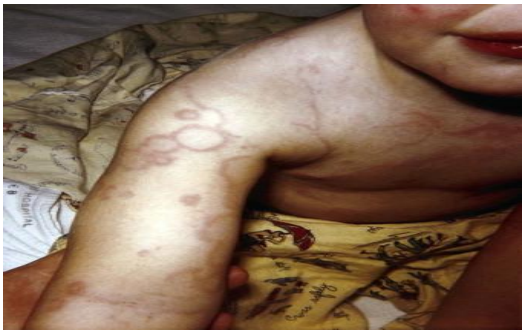
Bradycardia may be detected due to the suppressive effect of the inflammatory process on the sinus node or on the conduction of excitation pulses along the Gis bundle and its branches. Rheumatic endocarditis is difficult to diagnose in the first weeks of the disease, since the clinical picture is dominated by symptoms of myocarditis, largely masking manifestations from the endocardium, and rheumatic endocarditis (valvulitis) is not accompanied by additional subjective symptoms. Rheumatic endocarditis involves the valvular apparatus of the heart in the pathological process and leads to the development of heart disease.

Valvular heart disease, especially mitral, is considered as a "monument to extinct endocarditis." In the latent course of rheumatism, defects develop more often than in acute (22 and 15%, respectively). This is due to the difficulty of timely detection and elimination of latent endocarditis. In rheumatism, aortic valves are affected 2 or more times less often than mitral valves. Tricuspid valves are even less often affected, and very rarely pulmonary artery valves. The clinical diagnosis of endocarditis (primary valvulitis) is based on the evolution of systolic murmur, heard above the region of the apex of the heart, less often in the third intercostal space, to the left of the sternum. Short and soft, then weakening, then increasing at the beginning of the disease, the noise becomes more constant and rough. In the early stage of rheumatic valvulitis, a weak, unstable diastolic murmur may appear, which can be explained by swelling of the valve flaps.

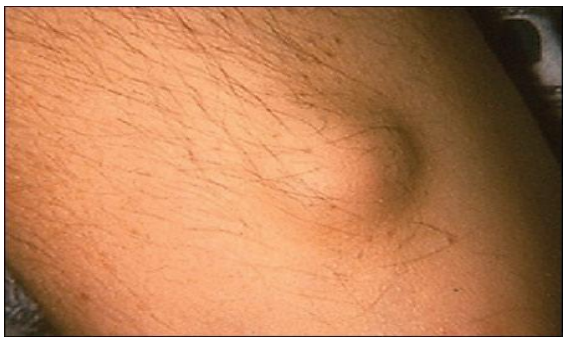
Rheumatic pericarditis is usually combined with rheumatic myo- and endocarditis (pancarditis), develops in the most severe course of the rheumatic process. It is extremely rare. There is a difference between dry (fibrinous) and exudative (serous-fibrinous) pericarditis. With dry pericarditis, patients complain of constant dull pains in the heart area. In an objective study, there is a pericardial friction noise at the base of the heart, to the left of the sternum in the second or third intercostal space. Rough systolic friction can be determined by palpation, it lasts more often for a short time and disappears within a few hours.

Vascular damage is manifested by valvulitis caused by increased vascular permeability and deposition of immune complexes in the walls of capillaries and arterioles. In addition to capillaries and arterioles, veins can also be involved in the pathological process. Rheumatic arteritis of internal organs is the basis of clinical manifestations of rheumatic visceritis: nephritis, meningitis, encephalitis, etc. Rheumatic vasculitis of myocardial vessels is important for the clinic. In such cases, coronary artery disease develops with pain in the heart area, resembling angina pectoris. Rheumatic phlebitis is extremely rare and does not differ in essence from inflammation of the veins of a banal nature. Capillaries with an active rheumatic process are almost always affected, which is manifested by skin hemorrhages, proteinuria, positive symptoms of “tourniquet”, “pinch”.

Skin lesions occur in the form of ring-shaped and nodular erythema, rheumatic nodules, spot hemorrhages, etc. Erythema annulus and rheumatic nodules are considered pathognomonic for rheumatism. Ring-shaped erythema is manifested by pale pinkish-red or bluish-gray spots mainly on the inner surface of the arms and legs, abdomen, neck, trunk. When pressed on the affected skin, the spots disappear, then reappear. The spots do not itch or hurt.



Erythema nodosum is much less common and is not considered characteristic of rheumatism. Erythema nodosum is much more often observed in allergic diseases of a different nature

. 

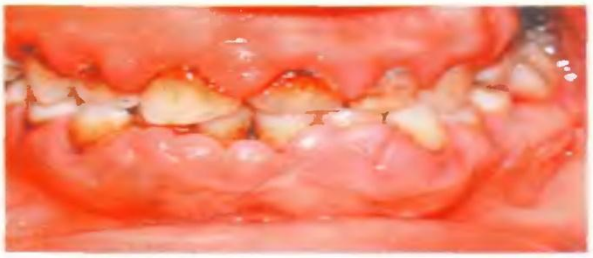


Рис. 64. Валикообразное утолщение десен при ревматизме.

Rheumatic nodules

Lung damage is manifested by pneumonia, pleuropneumonia or pleurisy. Rheumatic pneumonia develops against the background of rheumatism and differs from the banal clinical picture in that it responds well to salicylic therapy and is resistant to antibiotic treatment. Rheumatic pleurisy is in second place after tuberculosis and usually appears against the background of a rheumatic attack or shortly after a sore throat. The clinical features of rheumatic pleurisy include the rapid accumulation and relatively rapid (3-8 days) resorption of a minor serous fibrinous sterile effusion, which never accumulates. Depending on the severity and severity of the process, lymphocytes or neutrophils predominate in the exudate. The dominance of the latter is characteristic of the most severe course of the rheumatic process. Pleural fusion is rare.

Minor chorea.Damage to the nervous system can manifest itself as minor chorea in children, especially in girls. Against the background of emotional lability, pretentious movements of the trunk, limbs, and facial muscles arise, which increase with excitement and pass during sleep. By the age of 17-18, these phenomena disappear. Encephalitis, diencephalitis, and rheumatic encephalopathy may occur. With heart defects, a common complication is disorders of cerebral circulation - embolisms, vasculitis, syncopal conditions.

Sydenham’s Chorea .Appears in 12-17% of all cases.

Laboratory data.

Changes in the morphological composition of blood in rheumatism are nonspecific. With pronounced activity of the rheumatic process, leukocytosis up to 10-1210 / l with neutrophilosis and a shift to the left, an increase in ESR up to 50-60 mm / h can be observed. Similar changes on the part of the blood are detected with the predominance of articular manifestations of the rheumatic process.

Instrumental data.

For the purpose of early diagnosis of rheumocarditis, a set of methods characterizing the bioelectric, hemodynamic and contractile functions of the heart is used.

Electrocardiography in 1/3 of patients with active rheumatism reveals changes in the P wave in the form of serration, double-hump (decrease or increase in voltage). These changes are not permanent and in primary rheumocarditis disappear as the activity of the rheumatic process is eliminated.

Phonocardiographically, in primary rheumocarditis, a decrease in the amplitude of the I and II tones is detected, in some cases, a splitting of the tone. In about 2/3 of patients, systolic noise is recorded above the apex of the heart or at the Botkin point, which is characterized by the variability of sound oscillations in each cardiac cycle, varying intensity and duration.

X-ray examinations are valuable for establishing a heart defect and the predominance of its type, usually carried out with contrast of the esophagus. Deviations of the esophagus along a large or small radius indicate mitral stenosis or the predominance of stenosis with a combined defect.

Echocardiography in combination with Doppler ultrasonography has high sensitivity and specificity for the diagnosis of rheumocarditis and rheumatic heart defects. The method allows to identify changes in the heart valves and functional disorders. Doppler ultrasonography makes it possible to reliably determine the severity of mitral regurgitation and the blood pressure gradient in the area of the aortic valve. Echocardiographic signs of rheumatic mitral valve endocarditis: the presence of vegetation on the valves, hypokinesia of the posterior mitral flap, mitral regurgitation,

transient domed diastolic bend of the anterior mitral flap. Echocardiography and Doppler ultrasonography reduce the need for cardiac catheterization to diagnose lesions of its valvular apparatus.

Differential diagnosis.

ORL most often has to be differentiated with tonsillogenic heart disease, non-rheumatic carditis, infectious endocarditis, JURA, reactive arthritis, thyrotoxicosis, chronic tuberculosis intoxication. Tonsillogenic myocardial lesions may manifest as myocarditis or cardiomyopathy.

Tonsillogenic myocarditis usually develops during or shortly after angina. Patients often complain of an asthenoneurotic nature. There may be pain in the heart area, a slight displacement of the left border of the heart, a gentle systolic murmur detected auscultatively and on PCG, with relatively calm laboratory parameters (ESR, proteinogram, level of neuramic acids, etc.). If they increase, these indicators quickly return to normal as a result of treatment. There is almost always a pronounced pathology in the tonsils.

Tonsillogenic (functional) cardiomyopathy is observed in chronic tonsillitis. Patients may complain for a long time of dull, aching, stabbing pains in the heart area, which worsen during an exacerbation of tonsillitis, do not go away after taking coronaroactive drugs and weaken or disappear after taking sedatives (valocordin, corvalol, drops of Zelenin, etc.). Functional data are the same as with tonsillogenic myocarditis, but they are determined over several months or years without significant dynamics. Polyarthralgic syndrome is often observed. Laboratory parameters outside the exacerbation of chronic tonsillitis are within the normal range. Prolonged subfebrility may occur for several months. Tonsillogenic cardiomyopathy increases in the presence of chronic (more often decompensated) tonsillitis.

Non-rheumatic carditis usually develops during the period of infection (influenza, SARS, typhus, etc.) or a few days after it. It occurs more often with the flu. Patients complain of aching pains in the heart area. Displacement of the left border of the heart, muffling of the I tone, systolic non-conductive noise at the V point are often determined.

Due to the violation of repolarization processes, the T-wave flattens, the S-T interval decreases. Extrasystoles (supraventricular, ventricular), disorders of atrioventricular conduction, blockage of the Gis bundle, etc. are possible. Laboratory parameters reflecting the destructive inflammatory process have been slightly changed. Non-rheumatic carditis is characterized by a tendency to a prolonged course and recurrence in the absence of endocardial damage and the formation of a heart defect.

Infectious endocarditis occurs, as a rule, with high fever, chills, sweating, tachycardia, hemorrhagic syndrome, enlarged spleen with prolonged increased ESR, anemia, and a positive formolic test. It usually develops against the background of rheumatic or congenital heart disease, less often affects previously intact valves - almost always aortic. In the diagnosis, great importance is attached to the echocardiographic examination of the heart.

Rheumatoid arthritis has to be differentiated from rheumatic polyarthritis. RA is characterized by damage to large joints, the migratory nature of pain, and the good effect of salicylic therapy. In RA, joint pain is constant, it increases in the second half of the night, in the morning, there is stiffness in the joints in the morning. Mainly small joints of the hands are affected. In a relatively short period of time, joint deformity and atrophy of the interosseous muscles develop. There is a violation of the function of the joints. In the blood of 2/3 of patients, RF is determined, a long-term increase in ESR. Radiologically, osteoporosis, narrowing of the articular gap, erosion of articular surfaces, usura, ankylosis are detected.

ARF depends on the nature of the course of the pathological process and the involvement of certain organs and systems in the process. Cutaneous and articular forms of rheumatism usually proceed favorably. Minor chorea ends by the age of 18-20. In pediatric practice, deaths can occur with diffuse myocarditis and meningoencephalitis. The prognosis for rheumatism is determined mainly by the condition of the heart (the presence and severity of heart disease, the degree of myocardiosclerosis and circulatory disorders). The recurrent course is often especially unfavorable in this regard. The timing of the start of treatment of active rheumatism and the degree of reversibility of

the rheumatic process are important. In childhood and adolescence, rheumatism is more severe and more often (in 20-25%) leads to irreversible valvular changes.

3.2 Treatment of acute rheumatic fever.

Currently, the most justified is a three-stage treatment system for acute respiratory infections: the first stage is long-term (4-6 weeks) inpatient treatment in the active phase; the second stage is sanatorium or spa treatment in the post-hospital period, the third stage is dispensary observation in a polyclinic with bicillinomedicamentous treatment. Treatment of ARL should be as early as possible (in the first hours or days - up to 3 days from the onset of the disease), since at this stage there are changes in the connective tissue of the heart and other organs (the phase of mucoid swelling) still reversible; comprehensive, adequate and strictly individual. In case of ARF, the patient must be hospitalized. If this cannot be done, he must observe bed rest at home. The complex of treatment includes therapeutic, protective and motor regimen, rational nutrition, medications and physiotherapy.

The motor mode expands as the activity of the rheumatic process subsides. The food should be varied, rich in proteins, vitamins, phospholipids. Carbohydrate foods are limited. In case of circulatory disorders, food should be predominantly dairy-vegetable with a restriction of salt and liquid.

Treatment.

1.Etiopathogenetic, antiallergic therapy: a) antibiotics, b) nonsteroidal anti-inflammatory drugs (brufen (ibuprofen), voltaren, indomethacin, methindole), c) glucocorticoids, d) immunosuppressive drugs (quinoline, cytostatics, anti-lymphocytic globulin);

2. Antidystrophic agents;

3. Symptomatic remedies for circulatory insufficiency, impaired water-salt metabolism, etc. Of antibiotics, penicillin is indicated 500,000 units intramuscularly 4 times a day for 5 days, then bicillin-5,1.5 million units are administered intramuscularly once every 4 weeks, followed by the transfer of the patient to year-round bicillin prophylaxis. Penicillin has a bactericidal effect on group A streptococcus. If penicillin

is intolerant, erythromycin 250 mg 4 times a day. It is possible to prescribe drugs of the cephalosporin series (kefzol, cefazolin, etc.).

4. Indomethacin (metindol) or voltaren (diclofenac sodium, orthophen, diclonate, etc.) is prescribed at 1-3 mg / kg of weight per day for the entire period of inpatient treatment. Then, after discharge from the hospital, the patient should take 75-100 mg / day of indomethacin (voltaren) for at least 1-2 months. Subsequently, indomethacin (voltaren) during the next 2-3 months. Indomethacin and to a lesser extent voltaren can cause side effects from the central nervous system (headache, dizziness) and the gastrointestinal tract (stomach pain, diarrhea, gastrointestinal bleeding, gastric and duodenal ulcers). In case of intolerance to indomethacin, voltaren and other drugs from this group, as well as if there are contraindications to their appointment, brufen (ibuprofen) is used in a daily dose of 600-1200 mg / day. In some cases, naproxone (naprosin) can be prescribed, which has an anti-inflammatory effect to a greater extent than brufen. However, it can affect the kidneys, lead to aplasia of hematopoiesis, ulceration, followed by gastrointestinal bleeding. The daily dose is 750 mg (250 mg 3 times a day).

5. Glucocorticoid hormones (methylprednisolone, prednisolone) are used with a high degree of activity (III-II) of the rheumatic process and diffuse myocarditis. Glucocorticoids are combined with NSAIDs. The dose of methylprednisolone (medrol, etc.) is 12-16 mg / day, prednisolone is 20-30 mg / day. The main course is carried out for 10-14 days, then gradually the dose of hormones decreases by 2.5-5.0 mg / week. The course of treatment is 4-5 weeks, in severe cases - 8-10 weeks. With severe heart failure and a tendency to hypertension, it is more advisable to use triamcinolone or dexamethasone. Triamcinolone (polcortolone) is used 4 mg 3-4 times a day, dexamethasone - 0.5 mg from 1 to 6 times a day. Since glucocorticoids affect water-salt metabolism, potassium preparations (panangin, asparkam, potassium orotate) are included in complex treatment, with fluid retention - an aldosterone antagonist (aldactone, veroshiron 6-8 tablets / day), diuretics (furosemide 40-80 mg / day, etc.).

6. Aminoquinoline preparations (delagil, plaquenil) are indicated with a decrease in the dose of glucocorticosteroids: delagil 0.25 g or plaquenil 0.2 g once a day in the evening after dinner for 1-3 months; with high immune activity of the disease, prolonged and often recurrent course - up to 6-8 months or a year. These drugs have an immunosuppressive effect. They are combined with salicylates and other NSAIDs.

7. Immunosuppressants - 6-mercaptopurine, imuran (azathioprine), chlorbutin - are indicated only in patients with frequently recurrent and prolonged rheumatism, who are resistant to treatment with both classical antirheumatic drugs, including corticosteroids, and quinoline drugs with their long-term (months-long) use. The dose of 3-mercaptopurine and imuran (azathioprine) is 0.1 -1.5 mg / kg of weight, chlorbutin is 5-10 mg / day.

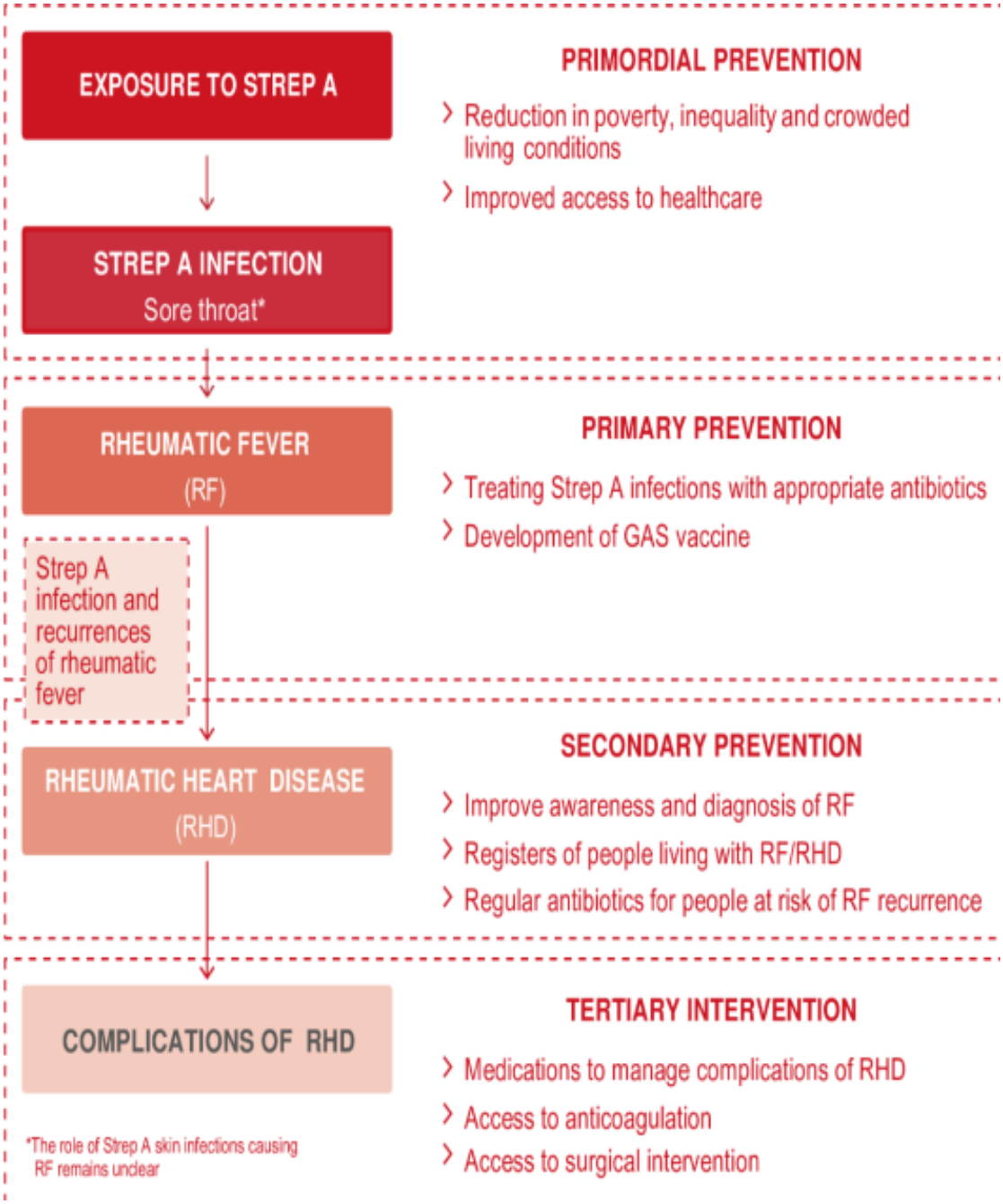
8. Antidystrophic therapy (anabolic steroids, protein hydrolysates, pyramidine derivatives, gamma globulin preparations, etc.) is prescribed, as a rule, in medium therapeutic doses.

Prevention. WHO recommendations for the prevention of acute respiratory viral infections and their recurrence include the following measures.

Primary prevention is reduced to the organization and implementation of a set of national, public and individual measures aimed at preventing the primary incidence of rheumatism. This includes systematic restorative measures: hardening of the body, physical education and sports, reducing, if possible, contacts with patients with streptococcal infection, timely and proper treatment of acute and chronic streptococcal infections. With angina, pharyngitis, acute respiratory infections of streptococcal nature, phenoxymethylpenicillin 250 mg is taken orally 4 times a day. In its absence, bicillin can be administered intramuscularly-5 1 500 000 One meal at a time. In case of severe course of the disease, erythromycin 250 mg is prescribed 4 times a day. Cephalosporins can also be used. Tetracycline-type drugs and sulfonamides are not recommended, since a large number of group A streptococcal strains are resistant to these drugs. In the inactive phase of rheumatism and the named streptococcal infection, in addition to penicillin therapy (600 000 - 800 000 ED / day) antirheumatic drugs are used: voltaren,

indomethacin in a dose of 1-3 mg per kg of weight.

Secondary prevention includes a set of measures aimed at preventing exacerbations, relapses and progression of the disease in people with rheumatism. Patients with rheumatism should be registered at the dispensary. They carry out year-round or seasonal (in spring and autumn) bicillin prophylaxis. Year-round bicillin prophylaxis is preferable. The patient receives 1,500,000 units of bicillin-5 per month for 5 years from the onset of the primary disease or relapse of the disease. Quinoline preparations (delagil, plaquenil) are indicated for frequently recurrent forms of rheumatism. Seasonal bicillin prophylaxis is carried out with bicillin-1 (1,200,000 units 1 time in 4 weeks) or bicillin-5 (1,500,000 units with the same frequency), includes 2-3 such courses. Along with bicillin therapy, one of the antirheumatic drugs is used during the same period: voltaren, indomethacin, etc.



Control questions.

1. Definition of the EAGLE
2.The etiology of ORL
3. The pathogenesis of ARL
4. FRA Clinic
5. Classification of ARL in children

6. Diagnosis and differential diagnosis of ARL in children.

7. Principles of treatment of ARL in children

8. Prevention of ARF in children.

Chapter IV. Juvenile rheumatoid arthritis (JRA)

Juvenile rheumatoid arthritis is a destructive inflammatory joint disease with unknown etiology, complex immunoaggressive pathogenesis, characterized by symmetrical chronic arthritis, systemic damage to internal organs, leading to disability of patients, developing in children under the age of 16 years.

Among rheumatic diseases of childhood, JRA occupies the first place in terms of prevalence. The disease is observed in various regions of the globe with a frequency of 0.05 to 0.6% in the population.

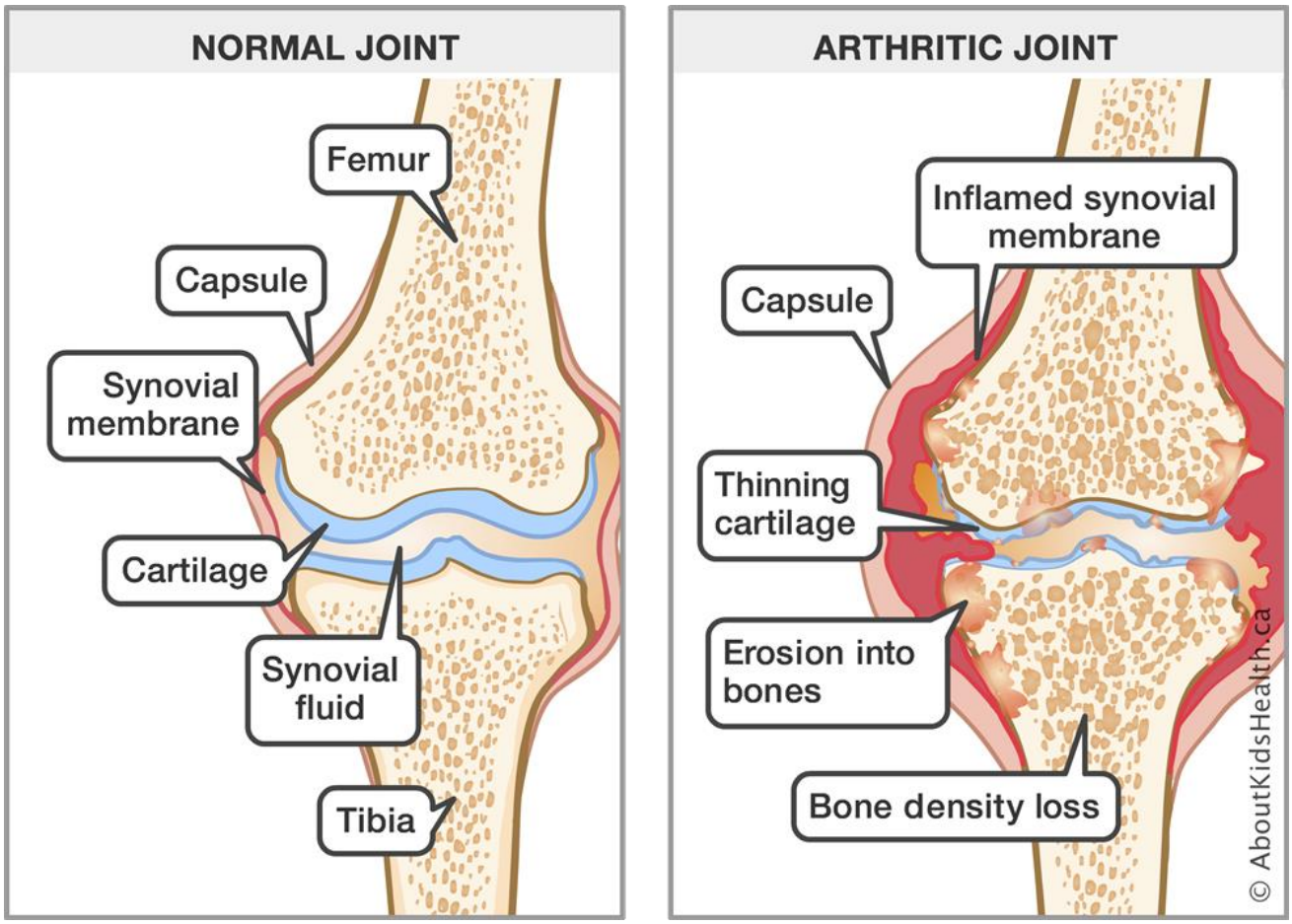
Etiology. JRA is still unknown. Among its causes, a combination of various environmental factors is considered. The inadequate response in patients with JRA is based on their "hypersensitivity to various environmental factors", as a result of which a complex immune response is formed, leading to the development of a progressive disease. A family-hereditary predisposition to rheumatic diseases also plays a certain role.

Pathogenesis. The pathological process in JURA begins in the synovial membrane of the joint with a violation of microcirculation and damage to the cells lining the synovial membrane. In response to the above changes, altered IgG are formed in the patient's body, which are perceived by the immune system as autoantigens. Immunocompetent cells, including plasma cells of the synovial membrane of the joint, produce AT-anti-IgG in response. These AT, called rheumatoid factor, interact with autoantigen in the presence of complement, and immune complexes are formed. CIC has a damaging effect on both the vascular endothelium and the surrounding tissues.

First of all, the synovial membrane of the joint suffers, as a result of which arthritis develops. In the synovial fluid and tissues of the joint, an excessive amount of cytokines of macrophage origin – IL1 and IL6, tumor necrosis factor – alpha is formed. IL1 induces inflammation and destroys cartilage. TNF has the same property. IL6 promotes hyperproduction of proteins of the acute phase of inflammation – C-reactive protein and fibrinogen. There is a further activation of enzyme systems that destroy cartilage. Increased vascular neoplasm, or angiogenesis, due to the action of cytokines on the tissue, also increases the destruction of cartilage. In the process of inflammation, a large number of cells are formed in the tissues of the joint, forming the so-called pannus, or

cloak, covering the surface of articular cartilage, thereby interfering with normal metabolic processes and enhancing the destruction of bone and cartilage formations.

Pathomorphology. Synovial membrane biopsy in the initial period of the disease reveals villous hypertrophy and hyperplasia of the surface layer. Inflammation in the joint in the JURA. As in adult patients, it leads to the formation of erosions and destruction of cartilage. However, these processes develop more slowly in children and in a smaller percentage of cases. The most pronounced changes are in the muscles adjacent to the affected joints.



4.1 Diagnostic criteria of the JRA

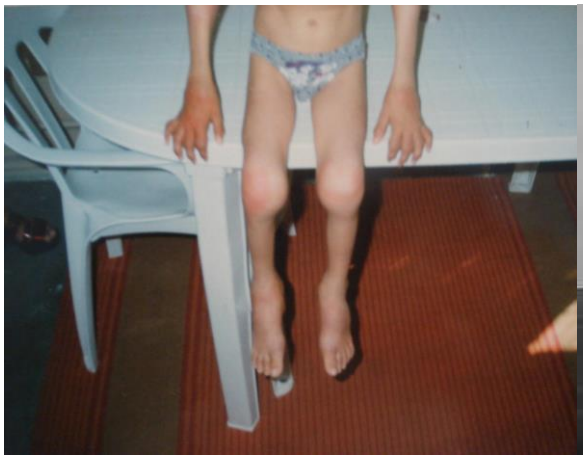
The clinical picture. JRA is diverse. The onset of the disease can be acute or subacute. With an acute onset, body temperature usually rises, soreness appears, and then swelling in one or more joints, more often symmetrical. However, the symmetry of the lesions sometimes becomes apparent not immediately, but within a few days or weeks of the onset of the disease. As a rule, large joints are affected – knee, ankle, wrist, but sometimes small joints of the arms and legs suffer from the very beginning of the disease. The lesion of the joints of the cervical spine is typical for Jurassic. All joints are sharply painful, swollen, and in rare cases the skin around them is hyperemic. Body temperature gradually rises and can reach 38-39 C. At the same time, polymorphic allergic rash often appears on the skin of the trunk and limbs, peripheral lymph nodes, liver and spleen increase. In a general blood test, anemia is detected, often neutrophilic leukocytosis with a shift of the leukocyte formula to the left, an increase in ESR to 40-60 mm / h, an increase in IgG concentration.

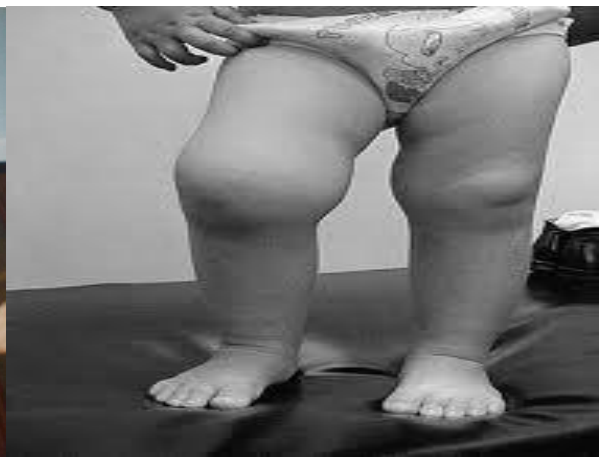
The acute onset of the disease is usually characteristic of severe forms of the JURA – generalized articular or articular-visceral form of the disease with a frequently recurrent course and an unfavorable prognosis. This form is more often observed in children of preschool and primary school age, but it can also occur in adolescents.

The subacute onset of the disease is characterized by less vivid symptoms. Arthritis usually begins with one joint – the knee or ankle. The joint swells, its function is impaired, sometimes even without pronounced pain. The child's gait changes, and children under 2 years old stop walking. The so-called morning stiffness in the joints is observed, which is expressed in the fact that the patient after a night's sleep feels difficulty for some time with joint movements and self-care. He stands up with difficulty, his gait is slow. Morning stiffness can last from a few minutes to 1 hour or more. The process may be limited to one joint for a long time (rheumatoid monoarthritis). This form of the disease, especially in preschool girls, is often accompanied by rheumatoid eye disease – rheumatoid uveitis. The subacute onset of the disease can also occur with the involvement of several joints in the process – more often

2-4. This form of the disease is called oligoarticular. Joint pain may be moderate, as well as exudative changes. The process may involve, for example, two ankle joints and one knee joint and vice versa. Body temperature does not rise, polyadenitis is moderate. This form of JRA is more benign, with less frequent exacerbations.

The articular-visceral form includes five signs: persistent high fever, polymorphic allergic rash, lymphadenopathy, hepatolienal syndrome, arthralgia/arthritis. This form of JRA has two main variants – Still's syndrome, which develops more often in preschool children, and Wissler-Fanconi syndrome, which is usually observed in schoolchildren.





The long-term recurrent course of the JRA can be complicated by secondary amyloidosis, which is facilitated by the constant circulation of immune complexes in the bloodstream. Amyloid is deposited in the walls of blood vessels, in the kidneys, liver, myocardium, intestines, which leads to a violation of their functions. Most often, amyloidosis affects the kidneys, as evidenced by persistent proteinuria with the subsequent development of CRF.

Particular shape

In this form, the progression of the JRA leads to persistent deformation of the joints with partial or complete restriction of mobility in them. Up to 25% of children become disabled

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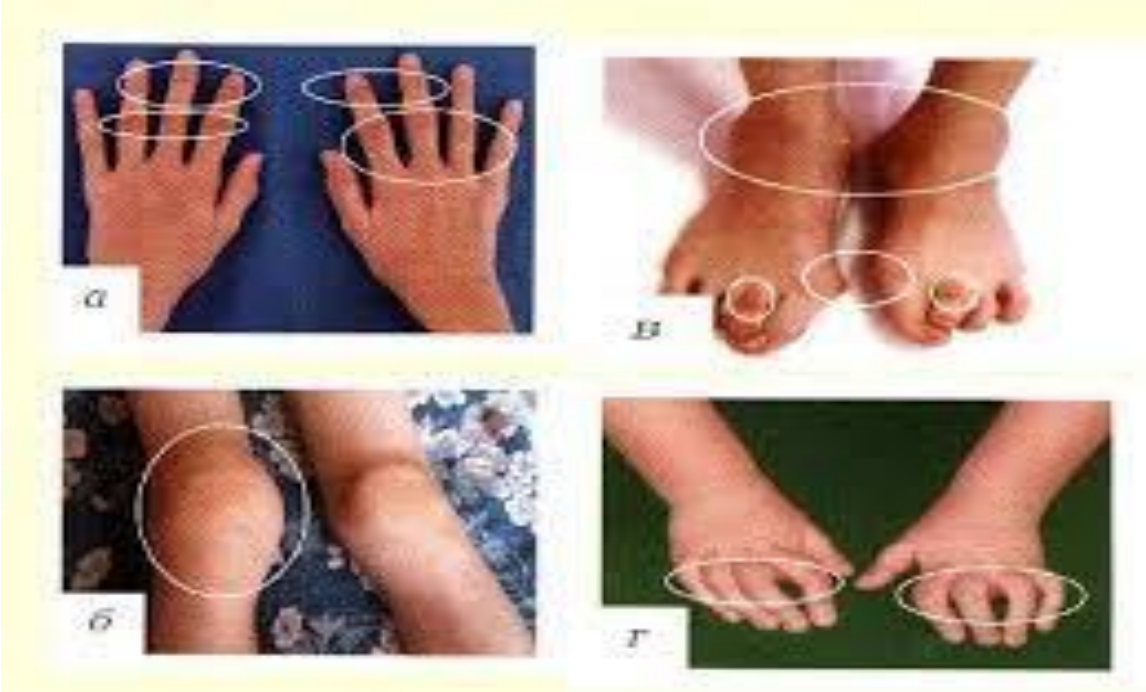


Classification

The following classification is used:

Form:

-mostly articular

-systemic (with damage to the heart, RES, lungs, with vasculitis, with polyserositis)

- Still's syndrome

- Wissler-Fanconi syndrome

-Articular form with eye damage – rheumatoid uveitis

Course:

-rapidly progressing

-moderately progressive

-slowly progressing

Activity, degree I II III

The presence of rheumatoid factor in the blood

-rheumatoid factor "+"

-rheumatoid factor "-"

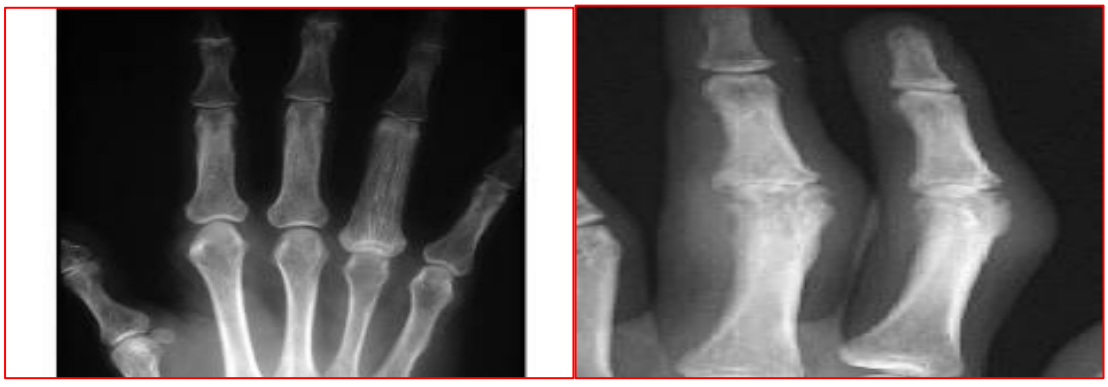
Functional disorders, grade I II III

Diagnostics. JRA often presents difficulties, especially in the early stages of the disease.

Diagnostic criteria:

1. Arthritis lasting 3 months or more.
2. Arthritis of the second joint, which occurred after 3 months and later.
3. Symmetrical lesion of small joints.
4. Joint contractures
5. Tendosinovitis or bursitis.
6. Muscular atrophy.
7. Morning stiffness.
8. Rheumatoid eye disease.
9. Rheumatoid nodules.
10. Effusion into the joint cavity

Radiological signs. Osteoporosis, small-cystic restructuring of the bone structure of the epiphysis, narrowing of the articular cracks, bone erosion, ankylosis of the joints.



Нарушение роста костей. Поражение шейного отдела позвоночника.

Laboratory signs. Positive rheumatoid factor, positive synovial biopsy data, depending on the number of identified positive signs determine the degree of probability of the disease:

3 signs - probable JRA

4 signs - definite JRA

8 signs - classic JRA

X-ray criteria of changes in the joints according to the classification of American radiologist Steinbrocker, subdivided into four stages, are also used:

I look - osteoporosis without destructive changes

Stage II - inconspicuous cuts of skin and hair, slight vasoconstriction of vessels, single hair ulceration

Stage III - significant destruction of cartilage and bone, pronounced narrowing of the articular gap, multiple usuras, subluxations, ulnar deviation

Stage IV - symptoms of stage III combined with ankylosis

As a result of rheumatoid inflammation, changes develop in the joints, which have three degrees, depending on the nature and severity of their dysfunction. The degrees of joint dysfunction are as follows:

I follow - reasonable planning of professional activities (especially in school), but not full co-regulation of self-regulation

Stage II - deprivation of the ability to perform professional activities and moderate limitation of self-care

Stage III - loss of self-sustaining ability and inability to care for oneself

The differential diagnosis of JRA should be made with rheumatic arthritis, reactive arthritis, ankylosing spondylitis, Reiter's disease.

Rheumatic arthritis: arthritis or arthralgias occur 2-3 weeks after a sore throat; joints are affected symmetrically; pain and swelling last from a few days to 2-3 weeks; arthritis resolves without sequelae.

Reactive arthritis: association of arthritis with a specific infection; asymmetric joint involvement; positive serologic tests; effect of antibiotic therapy

Bechterew's disease: early bilateral sacroileitis; slowly progressive mono-, oligoarthritis; hip joint often affected; exostoses of heel bones;

Reiter's disease: association with chlamydial infection; asymmetric arthritis of lower extremities; unilateral sacroileitis; reversibility of arthritis.

4.2 Treatment of JRA

Treatment for JRA should be carried out comprehensively and in stages. In the active period of the disease, patients need inpatient treatment, in the inactive period they need outpatient supervision and sanatorium treatment. During the period of exacerbation, treatment includes NSAIDs, in severe cases in combination with glucocorticoids and immunosuppressants, as well as with normal human immunoglobulin.

The main drugs used in the treatment of JRA

NSAIDs: diclofenac, acetylsalicylic acid, indomethacin, ibuprofen, naproxen

Glucocorticoids

Basic drugs: quinoline, methotrexate, sulfasalazine, cyclosporine.

Immunotherapy: human normal immunoglobulin

Local therapy intra-articular administration of drugs, mainly glucocorticoids, temporary immobilization of the joint with a removable splint, various physiotherapeutic methods of treatment, physical therapy, massage. In the presence of contractures, skeletal traction is applied, mechanotherapy is performed on special equipment.

Intraarticular administration of glucocorticoids is indicated for signs of active inflammation and exudation

Apply no more than 1 time per month

The following treatment is advisable: early initiation of basic therapy, nonsteroidal anti-inflammatory drugs (NSAIDs), intra-articular administration of glucocorticoids (HA) in the presence of synovitis, local therapy with anti-inflammatory ointments and gels, massage, physical therapy, rehabilitation measures (due to the frequent development of disability associated with joint deformity).

Schemes for the use of basic drugs:

Methotrexate — 7.5—10 mg/m2 /week. The course of treatment is at least 2 years. It may have a hepatotoxic effect.

Plaquenil — at a body weight of > 33 kg, 6.5 mg / kg / day. The course of treatment is long. The main side effect is the development of retinopathy.

Sulfasalazine — 30-40 mg / kg / day (the dose should be increased to the calculated one gradually). The course of treatment is long. It can have a hepatotoxic effect and cause cytopenia (mainly leukopenia).

NSAID application schemes:

Voltaren — 2-3 mg / kg / day. The main side effect (as with most NSAIDs) — ulcerative lesions of the mucous membrane of the gastrointestinal tract. It can be taken for many years under the supervision of gastroscopy.

Brufen (ibuprofen) — 30-40 mg / kg / day for a long time (from several months to several years). It may have a hepatotoxic effect.

Naproxen (naprosin) — 10-20 mg / kg / day for a long time (from several months to several years). It can be prescribed only to children after 10 years of age.

Flugalin (flurbiprofen) — 5 mg / kg / day for 2-3 months.

Aspirin is used less often — 60-80 mg / kg / day, but not more than 2.5—3.0 g / day. The course of treatment is 2-3 months.

Treatment of systemic variants of JRA

The purpose of treatment is to stop systemic manifestations, joint syndrome (if present) and the activity of intercurrent infection. To do this, apply:

- 1) pulse therapy with methylprednisolone in low doses (achieving a rapid anti-inflammatory effect). The dose of the drug is 5-10 mg / kg per 1 injection; the number of injections is from 1 to 5 every day or every other day, depending on the severity of myopericarditis, pneumonitis, vasculitis, polyserositis, fever;
- 2) immunoglobulin for intravenous administration (achieving a substitute and "mild" immunosuppressive effect). The dose of the drug is 0.3—1 g / kg per course; it is administered daily or every other day, depending on the severity of the condition;
- 3) antibiotics (in case of systemic manifestations, leukocytosis with a neutrophil shift to the left, the presence of an active intercurrent infection). Preference should be given to broad—spectrum drugs (aminoglycosides - amikacin is

better, cephalosporins of the third and fourth generations, etc.), prescribing them intravenously or intramuscularly for 7-10 days; the dose of the drug depends on the age of the patient;

- 4) intra-articular administration of methylprednisolone or diprospan (in the presence of exudate, pain syndrome and dysfunction, it allows to stop joint syndrome, prolong the anti-inflammatory effect of pulse therapy with methylprednisolone). The dose of drugs when administered to large joints is 1.0 ml; in medium — 0.5—0.7 ml; in small joints of the hands — 0.1—0.2 ml. GC is injected into each joint no more than 1 time in 1-3 months;
- 5) oral HC (with the ineffectiveness of pulse therapy with methylprednisolone, intra-articular administration of HC, immunoglobulin therapy for intravenous administration) at a dose of 0.2-0.3 mg / kg / day, but not more than 0.5 mg / kg / day; duration of administration — no more than a year. Oral HA is not advisable to use as first-line drugs for long-term treatment of JRA, given their severe side effects;
- 6) NSAIDs — voltaren is the safest and most effective at a dose of 2-3 mg / kg / day;

The greatest problems from taking hormone therapy that appear in the patient can be remembered using the abbreviation "CUSHINGOID MAP“

- Cataracts
- U – Ulcers
- S – Strial
- H – Hypertension
- I – Infections complications (infectious complications)
- N – Necrosis of bone (avascular) (avascular osteonecrosis)
- G – Growth retardation (cessation of growth)
- O – Osteoporosis
- I – Inercralased intracranial pressure (increased intracranial pressure)
- D – Diabetes mellitus (diabetes mellitus)
- M – Myopathy
- A – Adipose tissue hypertrophy (hypertrophy of adipose tissue)
- P – Pancreatitis

JRA is a lifelong disease, however, with the right therapy and systematic supervision of a rheumatologist, a long–term remission with a satisfactory quality of life is possible. With a frequently recurrent course, systemic manifestations of the disease, the prognosis is more pessimistic – disability occurs early, active life is limited. Relatively favorable. In 1/3 of patients, as a result of the chronic progressive course, significant restrictions of movement in the joints occur, contractures and ankylosis develop.

The development of uveitis and secondary amyloidosis sharply worsens the prognosis.

Prevention.

There is no primary specific prevention.

Nonspecific: rehabilitation of foci of chronic infection, medical supervision of children with altered reactivity.

To prevent relapse, it is recommended that dispensary supervision of children suffering from JRA.

Control questions

1. Definition of JRA;
2. The etiology of Jurassic;
3. Pathogenesis of Jurassic;
4. JRA Clinic;
5. Classification of the JRA in children;
6. Diagnosis and differential diagnosis of JRA;
7. Principles of treatment of JRA in children;
8. Prevention of JRA;

Chapter V

Chronic gastritis and gastroduodenitis in children. (HGD)

Chronic gastritis (HCG) is a chronic inflammatory recurrent disease of the mucous membrane (SOH) and submucosa of the stomach, which is accompanied by cellular infiltration, impaired physiological regeneration. HCG with inadequate treatment is prone to the gradual development of atrophy of the glandular apparatus and the progression of disorders of the secretory, motor and endocrine functions of the stomach. Unlike therapeutic practice, HCG in children is only an isolated disease in 10-15%. Antral gastritis is more common in combination with duodenitis - gastroduodenitis.

Chronic gastroduodenitis (HCG) is a chronic recurrent inflammatory disease that is accompanied by a nonspecific structural restructuring of the mucous membrane and glandular apparatus of the stomach and duodenum (dystrophic, inflammatory and regenerative changes) with various secretory and motor disorders.

HCG is the most common form of chronic gastroduodenal diseases. The structure of diseases of the stomach and duodenum is 58-74 % . The diagnosis should take into account the presence of risk factors for the development of HCG and hereditary predisposition. The clinic of the disease depends on the phase and severity of the inflammatory process, the state of secretory function of the stomach, motor evacuation disorders of the stomach and duodenum. With exacerbation of HCG, as with peptic ulcer disease, the following clinical syndromes: painful, dyspeptic and chronic nonspecific intoxication. The characteristic color of the clinical picture is given by the concomitant pathology of the hepatobiliary system, intestines, and pancreas. Clinical symptoms are similar to manifestations of peptic ulcer disease, but there is often no seasonal frequency of pain syndrome, night pain is infrequent.

Exogenous risk factors for the occurrence of HCG:

• alimentary - food “in dry water”, abuse of spicy and fried foods, deficiency of protein and vitamins in the diet, consumption of dietary supplements, violation of the rhythm of nutrition, etc.

•psychoemotional factor - stress, depression

• environmental factors: the state of the atmosphere, the presence of nitrates in food, poor quality of drinking water

• medicines – nonsteroidal anti-inflammatory drugs (indomethacin, acetylsalicylic acid, corticosteroids, etc.)

• bad habits - smoking, alcohol

• parasitic infections (especially giardiasis)food allergies and intolerance to certain food

• poor condition of the dental system

• hormonal dysfunctions

Endogenous factors of HCG

• HP infection

• bile reflux into the stomach

• Endocrine disorders

The development of highly effective therapeutic technologies, endoscopy, morphological examination of the ESH, some biochemical and bacteriological techniques made it possible to distinguish the following variants of gastritis (Sydney classification) into the following independent types:

Type A gastritis (endogenous, autoimmune gastritis)

Endogenous gastritis occurs due to the production of autoantibodies to the lining cells of the stomach. In children, 1-3% of all cases of gastritis are rare. This variant of gastritis is characterized by primary atrophic changes localized in the bottom and body of the stomach, decreased gastric secretion, increased gastrin content in the blood.

Type B gastritis (bacterial)

HP - associated gastritis. In children, this form of gastritis accounts for 80-85% of all gastroduodenal pathology. It has been proven that the pathogenesis of chronic type

B gastritis is based on persistent HP infection, which is confirmed by the fact that this microorganism is found in the pyloric department in the vast majority of patients. The route of infection is oral with food or with endoscopic manipulations, probing (for the pathogenesis of HP infection, see the lecture on peptic ulcer disease presented on the website in the pediatrics section).

Type C gastritis (reactive, chemical gastritis, reflux gastritis)

The determining role in the pathogenesis of gastritis C is played by duodenogastric reflux with the discharge of bile acids that disrupt the coolant and damage the epithelium (reflux gastritis). Among other reasons for this variant of gastritis, NSAIDs (acetylsalicylic acid, etc.) occupy a leading place. Due to the antiprostoglandinic effect of NSAIDs, the production of bicarbonates and mucus is blocked by the subsequent formation of erosions, violation of microcirculation.

The modern classification of HCG, which is used by Ukrainian pediatricians and recommended by the Ministry of Health of Ukraine (2000) mainly corresponds to the "Sydney Classification" (1990)

HCG in children usually occurs in the presence of a hereditary tendency and the listed risk factors. The clinic of chronic gastritis is diverse and depends on the nature of disorders of the secretory, evacuation function of the stomach, age and characterological characteristics of the child. The following clinical features of chronic gastritis in the period of exacerbation associated with the state of hydrochloric acid secretion are noted: With increased (or normal) secretion of hydrochloric acid (more often type B gastritis)

Pain syndrome: intense and prolonged, associated with eating. Early pain is typical for fundal gastritis, and late pain for antral gastritis). Pain at night. There is no clear connection with the time of year, diet disorders. In older children, palpation shows moderate pain in the epigastrium and pyloroduodenal zone.

Dyspeptic syndrome: “acidic” belching, belching with air, heartburn, nausea, tendency to constipation.

The syndrome of nonspecific intoxication and asthenia is variable. Attention is drawn to vegetative instability, irritability, rapid exhaustion during mental and physical loading.

With reduced secretion of hydrochloric acid

(more often type A gastritis)

The pain syndrome is weakly expressed, characterized by dull diffuse pain in the epigastrium. After eating, there is a feeling of heaviness and overflow in the upper abdomen; pain occurs and increases depending on the quality and volume of food. On palpation, there is a slight “diffuse” soreness in the epigastrium.

Dyspeptic syndrome prevails over pain. There is regurgitation of food, nausea, a feeling of bitterness in the mouth, decreased appetite, flatulence, unstable stools. With gastritis with reduced secretory activity, there is a decrease in appetite, aversion to certain foods (cereals, dairy dishes, etc.). The syndrome of nonspecific intoxication is significantly pronounced, asthenia prevails. Patients are pale, body weight is reduced due to a violation of the gastric stage of digestion and secondary disorders of the pancreas, in severe cases, manifestations of polyhypovitaminosis, anemia are noted. In domestic pediatric practice, much attention is paid to determining the state of the acid-forming function of the stomach, which affects not only the features of clinical manifestations of gastroenterological pathology, but also makes it possible to prescribe antisecretory therapy and reparants more reasonably.

The modern method for determining the acid-forming function of the stomach is intragastric PH-metry. This method allows you to determine the PH of the body and the antrum of the stomach. The normal PH of the stomach on an empty stomach in children over 5 years of age is 1.7-2.5, and after administration of histamine — 1.5-2.5. The antrum of the stomach, which neutralizes acid, has a PH above 5. (Shabalov M.P., 1999). That is, the difference between the PH of the body and the antrum is normally above 2 indicates a compensated state. A decrease in this difference indicates a decrease in the neutralizing properties of the antrum and acidification of the duodenum (decompensated state). In some medical institutions that are deprived of the opportunity to conduct

intragastric PH measurement, the PH of the stomach is examined by a fractional method using a variety of irritants. The concept of HCG is clinical and morphological. The most complete picture of the gastric lesion is provided by a comprehensive study of biopsies of the antrum, fundus and angle of the stomach. It should be emphasized that from the point of view of leading gastroenterologists, the diagnosis of gastritis is unlawful without morphological examination of the stomach. As a preliminary diagnosis before morphological examination, it is recommended to use the term non-ulcerative dyspepsia. Modern HCG treatment regimens depend on the type of gastritis, secretory function of the stomach, the age of the child, the state of the autonomic nervous system and his psychoemotional state.

Given that a significant amount of abdominal pain in children is psychogenic in nature, the clinical diagnosis of chronic gastritis needs to be verified endoscopically and histologically and only then begin therapy, taking into account the presence of combined gastroenterological, somatic and psychosomatic pathology.

Chronic gastroduodenitis (HCG) is a chronic recurrent inflammatory disease that is accompanied by a nonspecific structural restructuring of the mucous membrane and glandular apparatus of the stomach and duodenum (dystrophic, inflammatory and regenerative changes) with various secretory and motor disorders.

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Pain syndrome: The most characteristic abdominal pain is aching, prolonged, which occurs in the morning on an empty stomach and 1, 5-2 hours after eating. There is often an acute, paroxysmal short-term nature of pain, which is localized in the epigastrium, right hypochondrium, around the navel. The pain increases after eating and physical loading.

With erosive hyperacid HCG, hunger, night and late pain are combined. Palpation is marked by diffuse soreness in the epigastrium, a positive symptom of Mendel, the pyloroduodenal zone, with erosions, local muscle tension is possible.

Dyspeptic syndrome: frequent belching, heartburn, prolonged nausea, heaviness after eating, a feeling of bitterness in the mouth, flatulence, constipation, more rarely unstable stools.

Nonspecific intoxication syndrome: emotional lability, frequent headaches, irritability, general weakness and asthenization.

In the treatment of patients with HCG and HCG in modern conditions, in order to conduct adequate diagnosis and treatment, it is necessary to perform the necessary amount of paraclinical examination methods:

Laboratory tests:

a) mandatory (one-time):

• Clinical blood test;

• clinical urine analysis;

• total protein and protein fractions of blood;

• tests for Helicobacter pylori (rapid urease, bacteriological, respiratory urease test, serological (ELISA), ELISA analysis of the concentration of HP antigen in feces, PCR);

b) if necessary:

• stool analysis for latent blood (Gregersen reaction);

• histological (cytological) examination of biopsies using the histological diagnostic method of Helicobacter pylori — the "gold standard";

• immunogram;

Instrumental studies and diagnostic criteria:

Necessarily:

• fibroesophagogastroduodenoscopy with targeted biopsy and rapid HP diagnostics (for erosive HCG — twice);

• intragastric PH-metry (or fractional examination of gastric contents) — at a time;

• Ultrasound of the abdominal cavity organs — once to identify concomitant pathology.

If necessary:

• X-ray examination of the stomach and duodenum (motor evacuation disorders, developmental abnormalities);

• rheography;

• others in accordance with the nature of background and combined pathology

5.1 Principles of treatment of chronic gastritis and gastroduodenitis in children

Principles of treatment of type A gastritis. Substitution therapy is carried out aimed at restoring the functioning conditions of the stomach close to normal, compensation for atrophic processes in the coolant.The main method of therapy is therapeutic nutrition. In the acute phase, diet No. 1a is prescribed, which provides functional, mechanical, thermal and chemical cooling of coolant and 5-6 meals a day. Dishes that irritate the body are excluded from the diet (pickles, smoked meats, rich soups, marinade, spicy seasonings, fried meat and fish). Given that patients often do not tolerate whole milk, grape juice, sour cream, it is advisable to exclude them from the diet. The use of salt, strong tea and coffee, and their surrogates is limited. As the inflammation is eliminated, gradually increasing functional stimulation of the fundal glands is shown. For this purpose, diet No. 2 or even No. 15 is prescribed. At the same time, it is necessary to limit the consumption of fatty meats and fish, refractory animal fats, fried potatoes, pancakes, canned foods, smoked meats, spices. Milk is replaced with fresh fermented milk products (yogurt, kefir, cottage cheese, non-spicy cottage cheese).

Fresh and black bread, pastry products, cream, sour cream, cabbage, grapes, which causes gas formation in the intestines, are limited.

Anticholinergic and antacid drugs for type A gastritis are not prescribed. In the presence of pain and dyspeptic syndromes, a good effect is achieved with internal administration or intravenous injections of metoclopramide, sulpiride, no-shpa, butylskopolaminbromide (buscopan).

Enveloping and astringent herbal remedies are widely prescribed: infusion of plantain leaves, plantaglucide granules, yarrow, chamomile, mint, St. John's wort, valerian root. Herbal infusions are taken orally 1/3 1/2 cup 4-5 times a day before meals for 2-4 weeks.

In order to stimulate the secretory function of the stomach, herbal combined preparations can be used that stimulate secretion: herbogastrin, gastric drops herbion, plantain and its preparations (plantaglucide).

For substitution purposes, a solution of hydrochloric acid, pepsin, and other drugs are used. To improve the trophism of COOLANT, agents are used that enhance microcirculation, protein synthesis and reparative processes: nicotinic acid preparations, vitamins B and C inside and in injections, methyluracil, solcoseryl. With concomitant megaloblastic anemia, vitamin B12 injections are additionally prescribed. In the stage of subsiding exacerbation, methods of physiotherapy and treatment with mineral waters can be used. Outside of the exacerbation of the disease, sanatorium treatment is recommended for patients.

Principles of treatment of type B gastritis

Given that the predominant number of cases of type B gastritis is caused by HP, the treatment of this form of gastritis is based on the eradication of helicobacter infection. Schemes of anti-helicobacter therapy (see the lecture on peptic ulcer disease). At the same time, dietary nutrition has not lost its role in the initial stages of treatment. The N1 diet is prescribed, which moderately reduces the mechanical and chemical effects on coolant. The number of meals increases to 4-6 times a day. During the period of exacerbation, with significant severity of the pain syndrome, antispasmodics can

additionally be prescribed – drotaverine (drotaverine-KMP, no-shpa, no-x-shpa), halidor, papaverine. In some cases, the cholinolytics atropine and buscopan are effective. With high acidity of gastric juice, antisecretory drugs from the group of selective M— cholinolytics – pirenzepine (gastrocepine) are prescribed for up to 4 weeks. The dose in preschool age children in the form of tablets is 12.5 mg 2 times a day, and in schoolchildren 25 mg 2 times a day.

Histamine H2 receptor blockers (famotidine, ranitidine) for a period of 2 weeks. Children over 10 l. famotidine is prescribed at a dose of 0.02-0.04 g before bedtime. After completing the course of antisecretory therapy, complex antacids such as phosphalugel, or drugs that contain algedrate with magnesium hydroxide (almagel, almol, maalox) are used. It is possible to prescribe diosmectite (smecta) in a dose for older children of 2 liters. — 6-9 g / day in the form of an aqueous solution for several doses. At the end of the course of treatment, before confirming the effectiveness of eradication therapy, especially with residual dyspeptic and painful manifestations, courses of cytoprotectors – sucralfate (ankrusal, venter) may be prescribed. The dose of sucralfate in children is 0.5 g – 1.0 g. 4 times per dose (including 1 time per night) for a month.

In order to improve the trophism of coolant, sea buckthorn oil and multivitamin preparations can be used for a period of 3-4 weeks. In complex therapy, the appointment of tranquilizers for 2-3 weeks is justified – diazepam (seduxen, sibazone), tazepam, etc. Herbal sedatives are effective – valerian extract, persene.

Principles of treatment of type C gastritis

In the treatment of type C gastritis (reflux gastritis) occurring with impaired motility, duodenogastric and gastroesophageal reflux, the appointment of metoclopramide (raglan, cerucal) is indicated which normalizes the closing function of the cardia. Metoclopramide also reduces gastrointestinal reflux, accelerates gastric emptying and increases the resistance of coolant to damage. Occasionally, side effects are possible in the form of hyperkinetic phenomena, drowsiness, tinnitus, dryness of the mucous membranes of the mouth.

Domperidone (motilium) normalizes gastric motility. This drug acts milder than cerucal, less likely to cause side effects. In gastroesophageal reflux, the use of the selective cholinomimetic cisapride is promising (to be used with caution in cases of disorders of the cardiac conduction system). In order to neutralize the aggressive effect of bile on coolant, phosphalgel is prescribed, which, in addition to its antacid effect, adsorbs bile acids and has an enveloping effect. Sucralfate (ankrusal, venter, ulgastran, sucreise) has a good cytoprotective effect. The mechanism of action of the drug in reflux gastritis consists in the formation of complex compounds with tissue proteins in the area of the damaged mucous membrane. Sucralfate adsorbs pepsin and bile acids, increases the resistance of the mucous membrane to the acid-peptic factor. The cytoprotective effect is diosmectite (smecta).

The most effective cytoprotectors are synthetic analogues of prostoglandins misoprostol (sitotek, arboprostyl, etc.). According to the mechanism of action, they may be promising for use in children, since they reduce basal and stimulated gastric secretion, stimulate repair processes. However, these drugs often cause dyspeptic phenomena, a number of undesirable effects from the reproductive system, allergic reactions and therefore can only be used in adolescents with erosive gastritis. Isolated chronic gastritis or duodenitis in children are rare, more often we meet with gastroduodenitis. From the point of view of leading pediatricians of HCG, children with a history of gastroenterological pathology have a pre-ulcerative condition.

The basic principles of treatment mainly correspond to the principles of HCG therapy and depend on the period of the disease, the nature of clinical and endoscopic changes, the state of secretory function of the stomach and disorders of the motor evacuation function of the stomach and duodenum. In case of exacerbation, it is necessary to determine the conditions of treatment (inpatient or outpatient), the regime of physical activity. Prescribe dietary measures taking into account the patient's condition (table No. 1 or No. 5).

The complex therapy includes:

• In the presence of HP: eradication anti-HP therapy (usually for 7 days);

• Antisecretory drugs: histamine H2 receptor blockers for 2-3 weeks, selective M1 cholinolytics (pirenzepine for 4 weeks).

If necessary, antacids are added with cytoprotective and sorption effect for 10-14 days.

Take 1 pack 3-4 times a day;• Prokinetics (domperidone) are prescribed in the presence of reflux and duodenostasis for 10 days.• Antispasmodics (drotaverine, papaverine, metacin) — for 7-10 days;• Sedatives and tranquilizers, sedatives of plant origin.

After the cancellation of antisecretory drugs, reparants are prescribed — smekta, sucralfate, liquiriton, sea buckthorn oil for a period of 4-6 weeks. At the same time, concomitant pancreatic pathology is being treated with the appointment of enzyme preparations.

Adolescents may be prescribed intestinal antispasmodics (dicetel, pinaverium bromide), laxative drugs (macrogol) and others for constipation.

In the stage of exacerbation of the disease, physical methods of treatment are used — electrotherapy, thermal therapy. Laser and magneto-laser therapy are used to normalize the motor evacuation function of the stomach and increase the trophism of the coolant. Reflexotherapy is used among non-medicinal methods of treatment.

In clinical remission: phytotherapy, balneotherapy, physiotherapy, physical therapy, non-medicinal alternative therapy. The length of stay in the hospital is on average 21 days (with erosive HCG — up to 28 days).

It is advisable to continue inpatient treatment at a local pediatric gastroenterological sanatorium. Referral to domestic balneological resorts in Mirgorod, Truskavets, Morshin and others is carried out in the presence of a sufficiently stable clinical remission.

Dispensary monitoring for HCG and HCG has been carried out for 5 years since the last exacerbation, the frequency of examinations is at least twice a year.

Patients are examined by a pediatrician once every 6 months and a pediatrician-gastroenterologist once a year. Fibrogastroduodenoscopy is performed at least 1 time

per year. With erosive HCG, the frequency of examinations increases to 3 times a year, endoscopic examination — 2 times a year).

A child with HCG or HCG is subject to removal from the dispensary register, subject to 5-year clinical and endoscopic remission.

Criteria for the effectiveness of dispensary supervision:

Complete clinical remission – absence of pain syndrome and dyspeptic disorders, reduction of endoscopic and histological signs of process activity

Complete clinical, histological and morphological remission - absence of clinical symptoms, reduction of endoscopic and histological signs of inflammation and HP infection activity

After Hp eradication, the inflammatory reaction of the COOLANT disappears within 2-4 months and, in the absence of clinical manifestations, does not require additional treatment.

With an exacerbation of HCG And HCG, physical education is contraindicated for schoolchildren. 6 months after the onset of remission, physical education classes are held in a special group, and if you feel well and transfer loads after another 3 months – preparatory. Classes in the main group are not allowed.

Prevention

In case of gastroduodenal pathology, it is very important to observe the principles of age-related nutrition, to protect the child from physical and emotional overloads.

Secondary prevention includes adequate and timely therapy, follow-up of a GP and regular consultations with a gastroenterologist, sanatorium treatment.

Control questions

1. Define chronic gastritis
2. What risk factors for HCG and HCG do you know?
3. What is the pathogenesis of chronic gastritis
4. What is the classification of gastritis
5. Give a clinical description of gastritis of type A, B,C

6. Define gastroduodenitis

7. What laboratory diagnostic methods do you know?

8. How is the treatment, medical examination, prevention of HCG and HCG carried out?

Chapter VI.

Features of the course of peptic ulcer of the stomach and duodenum in children.

Peptic ulcer disease is a chronic and recurrent disease prone to progression, involving in the pathological process, along with the stomach and duodenum, organs of the digestive system, the development of complications that threaten the patient's life.

The proportion of peptic ulcer disease (PUD) is 13.5% of all gastroenterological diseases in children, with duodenal ulcers (DU) prevailing (90-95%). However, in 5-8% of cases, peptic ulcer disease is complicated by perforation, penetration, perivisceritis, stenosis, gastrointestinal bleeding. The latter pose a real threat to life, since even minor blood loss is much more difficult for children than for adults.

Etiology and pathogenesis

According to modern concepts, UD is considered as a polyethological disease, the causes of which include:

- hereditary predisposition
- perinatal pathology (threat of miscarriage, gestosis of pregnancy, pathology in childbirth, perinatal damage to the central nervous system), leading to fetal hypoxia, including gastric hypoxia
- allergic inflammation of the gastric mucosa (SOH), in which tissue eosinophilia is noted and the number of cells producing Ig E is increased.

A change in the nature of nutrition, including:

1. Early transfer to artificial feeding

2. lack of fresh fruits and vegetables; macro- and microelements; dietary fibers that act as enterosorbents

3. an excess of refined proteins and carbohydrates in the diet, as well as the consumption of large amounts of fats and canned foods, which may include pesticides, organometallic compounds, mycotoxins, antibiotics, hormones.

Schoolchildren have an irregular, unbalanced diet environmental degradation, including the ingestion of xenobiotics, heavy metal salts, and various metabolites into the body with water intestinal dysbiosis, the use of frequent and long-term courses of antimicrobial and ulcerogenic drugs (corticosteroids, NSAIDs)

tuberculosis, syphilitic, fungal lesions of the gastrointestinal tract (GI tract) are not uncommon in children; helminthiasis, parasitic diseases ; common infectious diseases: viral hepatitis, rotavirus infections, dysentery, salmonellosis .

Other representatives of the microbial flora: bacteroids, lactobacilli, fusobacteria, staphylococci, streptococci, neisseria, etc., which, being conditionally pathogenic flora, enhance gastric alkalinization while increasing the proteolytic activity of gastric juice other somatic diseases: diseases of the liver, pancreas, intestines, cardiovascular system, kidneys, blood diseases occurring with anemia; diseases of the endocrine system (diabetes mellitus), inactivity accompanied by motor evacuation disorders, transformation of the regime towards greater employment, which contributes to the formation of stress factors , urbanization, in particular, early industrial activity of children, poor financial security, alcoholism of parents

neurovegetative and psychoemotional disorders in children, especially in adolescence violation of the activity of the sphincters of the digestive system, manifested by reflux, insufficiency of the sphincter apparatus

imbalance and intensity of metabolic processes, the formation of neurohumoral and endocrine regulation, imperfection of immunogenesis, most often manifested during puberty.

Secondary immunodeficiency conditions, often found in children, especially those living in ecologically unfavorable regions.

The pathogenetic role of Hp in ulceration is due to their ability to colonize the pyloroantral part of the gastric mucosa and form foci of gastric metaplasia in the duodenal bulb.

Hp activates the complement system, causing complement-dependent inflammation, and immunocompetent cells, whose lysosomal enzymes damage epithelial cells of the gastric and duodenal mucosa, inhibit the synthesis and secretion of gastric and duodenal mucus glycoproteins, reducing the resistance of the gastric and duodenal mucosa, thereby contributing to the proteolytic "breakthrough" of the mucosa with enhanced retrodiffusion + ions.

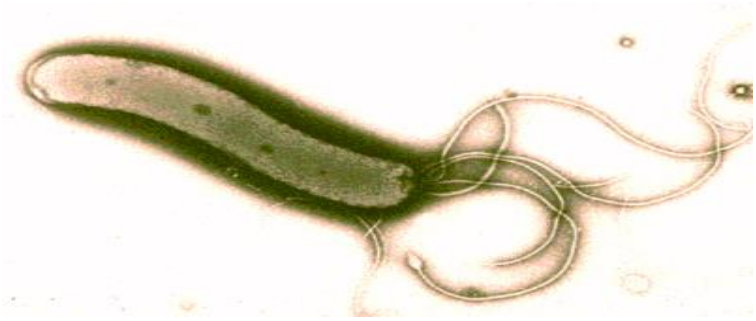
The role of helicobacter in pathogenetic processes is that it secretes a special protein – an inhibitor of hydrochloric acid secretion, and also activates proteases and phospholipases that violate the integrity of the epithelial layer, activates catalase and alcohol dehydrogenase, which can damage the epithelial layer.

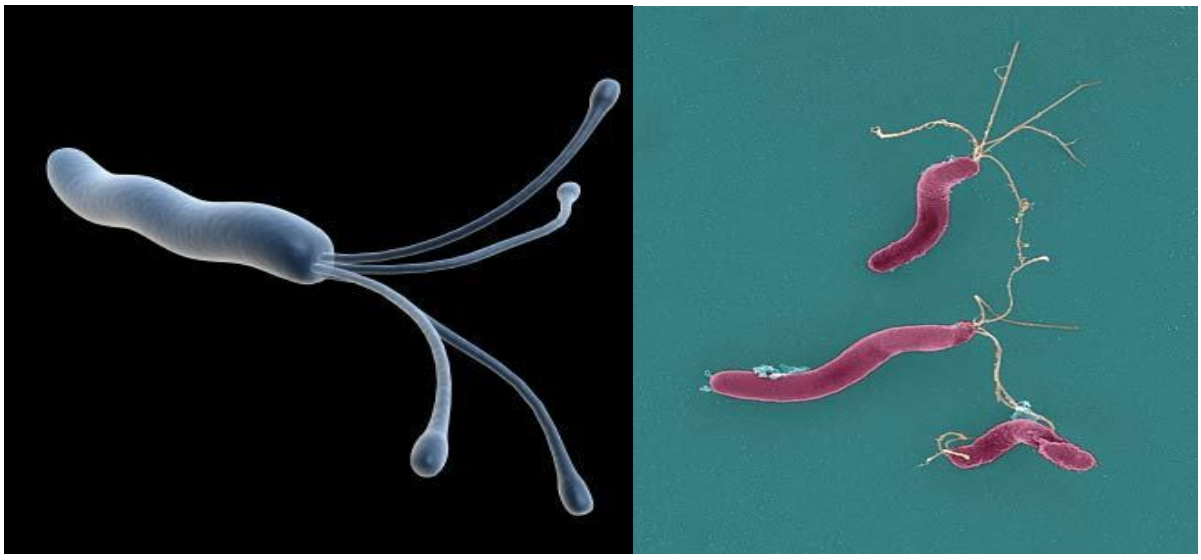
Among the endogenous factors of the development of UD, the most important importance is attached to the ratio between aggressive factors affecting the mucous membrane of the stomach and duodenum (the level of acid production, high proteolytic activity, especially due to the increased content of pepsinogen) and the state of its protective elements (the nature and degree of mucosation, including the exchange of glycoproteins and glycosaminoglycans, the production of prostaglandin E2, bicarbonates, secretory immunoglobulin A, blood flow in the coolant). It has been shown that in case of , the balance is disturbed towards a decrease in protective factors, which creates conditions for damage to the coolant and duodenum.

An important role is assigned to gastroduodenal motility, due to the neuropsychiatric stress of the patient, especially pronounced in children with a parasympathetic orientation of the vegetative status. Increased motor activity leads to accelerated evacuation of acidic contents from it into the duodenum, morphological changes in the coolant.

The presence of duodenogastric reflux promotes the entry of fatty acids into the stomach, damages the protective mucous barrier. This situation is aggravated by the

presence of Hp, which penetrates into the protective mucus layer and then adheres to the integumentary epithelium of the antrum of the stomach. Binding to the epithelium is accompanied by the development of local inflammatory and systemic immune reactions, leading to degeneration of the protective mucus layer with tissue destruction. The inflamed mucous membrane is very sensitive to acid and pepsin and, eventually, ulcerative depression may develop. As a result of emptying the stomach, its acidic contents come into contact with the epithelium of the duodenal bulb, as a result of which gastric metaplasia develops in it. The high and specific Hp agent for gastric epithelium causes infection of areas of metaplastic epithelium in the duodenum. Thus, at present there is every reason to assert that Hp plays a leading role in the development of nuclear weapons.





6.1 Diagnostic criteria for peptic ulcer disease in children.

Classification of peptic ulcer disease

By localization:

1. In the stomach

2. In the duodenum.

3. In the stomach and duodenum

Clinical and endoscopic stage:

fresh ulcer;

onset of epithelialization of the ulcerative defect;

healing of the ulcerative defect of the mucous membrane with preserved duodenitis;

clinical and endoscopic remission.

By severity: mild ; moderate; severe

Complications: bleeding; perforation; penetration; stenosis

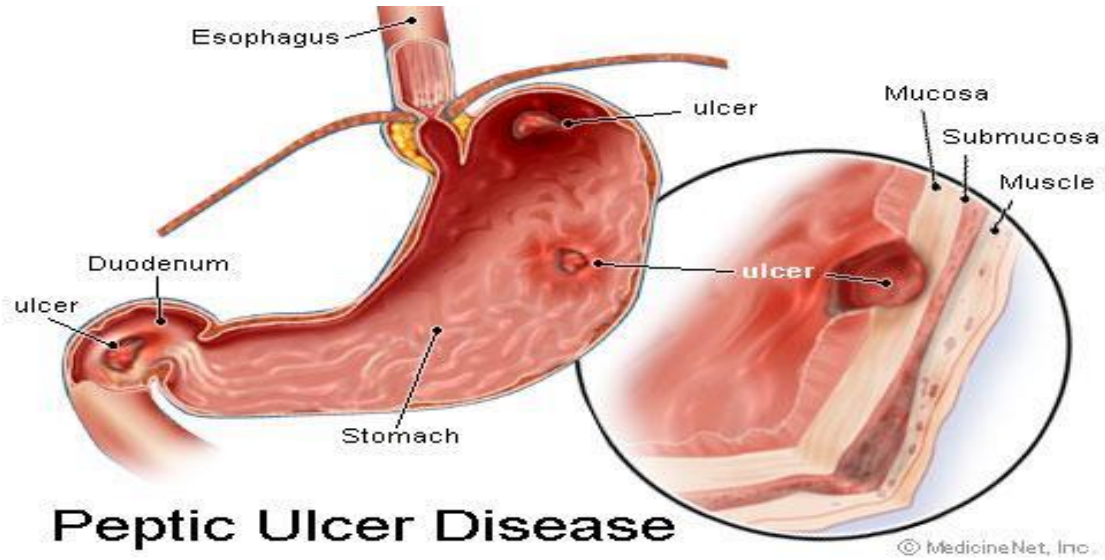
Clinic. The clinical manifestations of peptic ulcer disease in children depend on the stage of the disease and the location of the ulcer. The most constant and important symptom is pain. A characteristic feature of this pain is the connection with eating. Even in cases where the pain is more or less constant, it can be detected to increase it some time after eating. Pain can occur immediately after eating – after 30-60 minutes (so-called early pain), or after 2-3 hours (late pain). Of great diagnostic importance are night pains, which are often very intense and often disappear after eating (a glass of milk, kefir, a few sips of water). The pain is paroxysmal, cutting, stabbing, radiating to the back, right shoulder, shoulder blade.

The localization of pain is determined by the location of the ulcer: pain occurs more often in the epigastric region and to the right of the midline. During the period of exacerbation of pain, patients look for the most comfortable position in which the pain decreases. More often, this is a pose with a bent torso and legs drawn to the stomach, sitting in bed or lying on your side. If the ulcer is located on the front wall of the stomach, then pain relief may be in a position on the back or arching backwards. Despite the greatest diagnostic value of the pain syndrome, in some patients it may be absent: This is observed in cases of so-called “mute” or latent ulcers.

It is known that such severe complications of peptic ulcer disease as bleeding and perforation develop precisely in those patients who did not have pronounced pain. One of the most common and earliest symptoms of peptic ulcer disease is heartburn. It often precedes the appearance of pain and can also be periodic in nature: hungry, nocturnal heartburn.

Belching, nausea and vomiting in patients are somewhat less common than pain and heartburn. Vomiting is associated with pain.

The following sequence of symptoms can be established: heartburn – pain – nausea – vomiting - relief of dyspeptic syndrome. Appetite in children is most often preserved, sometimes even enhanced. The tongue is covered with a white coating, moist. Constipation is typical for patients with an exacerbation of the disease. The seasonality of pain and dipepsy syndromes (spring, autumn) is noted. Examination of children reveals signs of moderate chronic intoxication and hypovitaminosis. Palpation of the abdomen determines soreness, and local muscular protection of the anterior abdominal wall in the upper abdomen and right hypochondrium.



Complications of peptic ulcer disease:

1. Bleeding accompanied by vomiting with an admixture of blood, melena (black stools), weakness, dizziness, tachycardia (palpitations);

2. Penetration (penetration of ulcers into other organs), characterized by persistent pain syndrome, sharp pains radiating into the back, vomiting that does not bring relief, and persistent heartburn;

3. Perforation (breakthrough of an ulcer into the abdominal cavity), which occurs acutely and is accompanied by sharp pain in the epigastric region, tension of the anterior abdominal wall and symptoms of irritation of the peritoneum. Diagnostics. Observations in the gastroenterological clinic indicate that a thorough study of the anamnesis, analysis of risk factors, assessment of clinical manifestations of the disease, examination of the child and palpatory examination of the abdominal organs make it possible in 70-80% of cases to recognize the disease of the stomach and duodenum in a timely manner. Difficulties in diagnosis at the early stages of the disease are often associated with the involvement of other digestive organs (pancreas, biliary system, intestines) in the pathological process, which causes the "blurring" of clinical manifestations of peptic ulcer disease in children.

Additional methods of examination of the stomach and duodenum can be divided into three groups:

1. Methods based on the study of morphological features of the stomach and duodenum (X-ray examinations, gastroduodenofibroscopy, histological, histochemical examination of biopsies of the gastroduodenal mucosa).

2. Methods for studying the functional state of the gastroduodenal system (fractional gastric probing, pH-metry, radiotelometry, manometry, etc.).

3. Methods for the detection of Helicobacter pylori.

The main thing in confirming the diagnosis of peptic ulcer disease today is gastrofibroduodenoscopy. It is carried out in the dynamics of the disease to assess the condition of the ulcer under the influence of therapy. Depending on the characteristics of the clinical course and the results of additional research methods, a gastroenterologist makes a diagnosis and prescribes a complex of therapy.

6.2 Treatment

Treatment of peptic ulcer disease in children involves such main directions as:

- elimination of Hp infection;

- increasing the protective properties of the mucous membrane of the stomach and duodenum;

- effects on the autonomic nervous system in order to correct the balance between its departments.

Diet therapy and regimen moments in peptic ulcer disease are similar to those in chronic gastroduodenitis. It is advisable to hospitalize a child in a specialized pediatric gastroenterology department if the disease worsens. However, in some situations, when a child is not in contact and expresses a categorical protest against hospitalization, treatment at home is acceptable.

Psychotherapy is very important, especially in older children and adolescents. It is advisable to spend it at the same time with your parents. Of the general activities, walks in the fresh air after meals are recommended – at least 30-40 minutes. Do not take a horizontal position for 2-3 hours after eating. A night's sleep should be 8-10 hours. Children are contraindicated from sudden physical exertion, which causes a sudden increase in intra-abdominal pressure: jumping, intense running, weight lifting. The diet is based on the form of the disease and the acidity of the gastric juice. Meals should be fractional: 4-5 times a day, in small portions. The maximum break between meals should not exceed 4 hours. The last meal is at 19-20 o'clock. Foods that enhance bile secretion are excluded from the diet: vegetable and animal fats in their pure form, fried foods, yolks, caviar, cream, fatty sour cream, cakes and pastries. It is advisable to use sour-milk products, not whole milk.

Highly carbonated drinks are strictly contraindicated for all children with chronic inflammatory diseases of the stomach and duodenum “Coca-Cola” Pepsi-Cola, Fanta and others. Prolonged use of chewing gum (more than 10-15 minutes) is also a harmful factor. It is necessary to achieve a regular stool. If you are prone to constipation, you should increase your intake of vegetables, especially beets. The diet includes prunes, dried apricots, dried fruits in steamed form. With a tendency to diarrhea, vegetables are

excluded from the diet. Preference is given to semolina and rice porridge, fresh cottage cheese.

Drug therapy. Depending on the presence of a Helicobacter infection, various antibacterial agents are prescribed. The process of eliminating this microorganism is called “eradication".

According to the recommendations of gastroenterologists, Hp eradication is mandatory for gastroduodenal ulcers in case of infection, both during exacerbation and during remission and in patients with atrophic gastritis. Antihelicobacteric therapy is recommended for non-ulcerative dyspepsia; for therapy with non-specific anti-inflammatory drugs; for reflux esophagitis with prolonged use of antisecretory drugs; after surgical treatment for complicated peptic ulcer disease. Antihelicobacteria therapy is desirable for asymptomatic course, which is more common in children; the presence of concomitant non-gastroenterological diseases (lung pathology, allergic diseases, pathology of the endocrine system).

Currently, standard regimens (protocols) for the treatment of H.pylori in children and adults have been defined:

One-week triple therapy using proton pump inhibitor drugs (omeprazole) together with: - metronidazole and clarithromycin; or with amoxicillin and clarithromycin; or amoxicillin and metronidazole

One-week triple therapy with bismuth preparations together with: tetracycline and metronidazole or tinidazole.

One-week “quadro” therapy: omeprazole + bismuth preparation together with: - tetracycline and metronidazole or tinidazole.

The appearance of a bacterium in the body a year after treatment is regarded as a re-infection and therapy for the eradication of Helicobacter pylori is again prescribed. When prescribing anti-helicobacter therapy, it is necessary to study the intestinal microflora and prescribe biologics, both during and after antibacterial treatment. Given the familial nature of Hp infection, it is recommended that therapy courses be prescribed

to all relatives permanently residing with a sick child. The second important component of anti-ulcer therapy is the selection of antacids and antisecretory drugs.

With increased and preserved acid-forming function of the stomach, maalox, almagel, phosphalgel are used. The use of ranitidine and famotidine is effective. These drugs block basal acid formation for 12-24 hours. Drugs from the group of proton pump inhibitors that affect the transport of hydrogen ions due to enzyme blockade (omeprazole, pantoprazole, lansoprazole) are becoming increasingly popular among pediatric gastroenterologists. The complex of therapy also includes drugs from the group of M-cholinolytics – gastrocepin. These products increase the protective properties of mucus and reduce the damaging effect of gastrin. If there is a discharge of duodenal contents into the stomach, enterosorbents are used: enterosgel, smecta, cholestyramine, activated charcoal. In order to restore proper peristalsis of the gastrointestinal tract, motilium is used. One of the basic drugs for the treatment of peptic ulcer is sucralfate (Venter), which restores the properties of the mucous membrane of the stomach and duodenum. Colloidal bismuth preparations (De-nol, ventrisol, bismofalk, etc.) are widely used in pediatrics in the presence of an ulcerative defect.

As symptomatic, protective drugs (protecting) the mucous membrane are used: solcoseryl, actovegin. However, since these drugs are obtained from the blood of calves, the attitude towards their use is currently being reviewed due to the epidemic of spongiform encephalopathy (mad cow disease). Depending on the presence of concomitant pathology of other organs, additional medications are prescribed.

At the stage of rehabilitation, physiotherapy, acupuncture, phytotherapy, homeopathic remedies are used.

Stages of polyclinic rehabilitation:

1. Improve your daily routine, eliminate bad habits.

2. Courses of preventive treatment (antacids, cholinolytics, pepsin). The course of treatment is at least 1 month, 1 time per quarter for 2-3 years. In the future, 2 times a year.

3. In case of recurrent peptic ulcer disease, treatment should be continued for 2 years. In the future, preventive treatment.

4. Spa treatment.

Prevention.

Primary prevention of peptic ulcer disease provides for the protection of the child from physical and emotional overloads, compliance with the principles of age-related physiological nutrition, timely detection and rehabilitation of chronic foci of infection, timely treatment of other lesions of the digestive system (helminthic infestations, giardiasis, intestinal infections, etc.)

Secondary prevention consists in regular examination by a GP and a gastroenterologist, diet, seasonal preventive therapy, and limiting school workload. It is of great importance to ensure a well-groomed psycho-emotional environment at home and at school.

Control questions

1. Etiopathogenesis of peptic ulcer disease in children;

2.Classification of peptic ulcer in children peptic ulcer in children;

3. clinical features of peptic ulcer disease in children;

4.Diagnosis and differential diagnosis of peptic ulcer disease in children;

5. Modern approaches to the treatment and prevention of peptic ulcer disease in children;

6. Prevention and rehabilitation of peptic ulcer disease in children;

Chapter VII

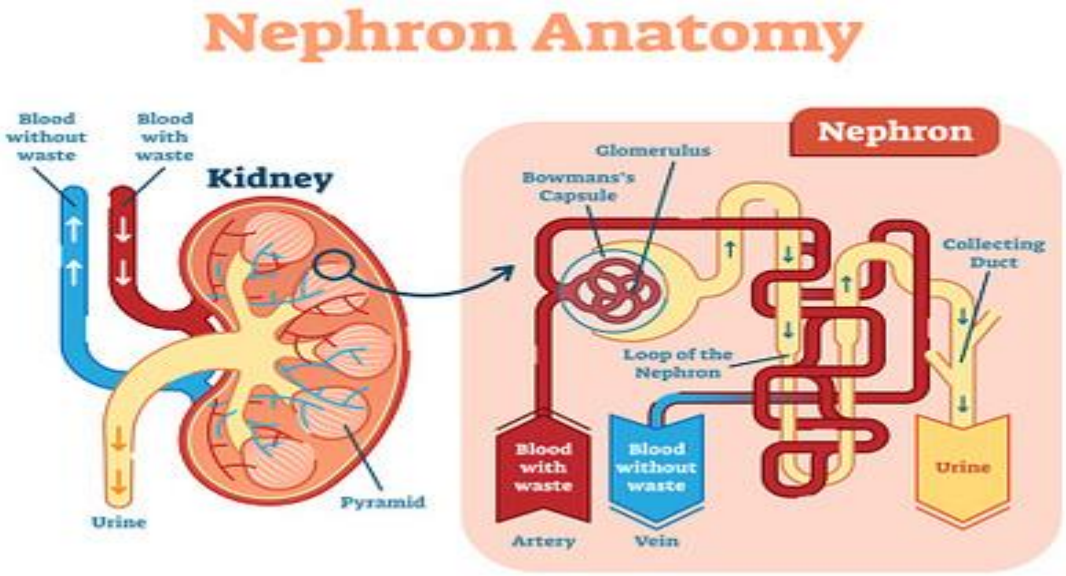
Acute and chronic infectious glomerulonephritis in children.

Glomerulonephritis (GL) is a heterogeneous group of acquired kidney diseases, different in etiology, clinical and morphological manifestations, course and outcome, mainly characterized by immune inflammation with primary glomerular lesion and

secondary involvement of renal tubules and interstitium in the pathological process. Acute, rapidly progressive and chronic glomerulonephritis are isolated.

Terminology.

Glomerulonephritis, or immune glomerulopathy, is a heterogeneous group of diseases characterized by the presence of immunological and clinico-morphological signs of inflammation associated primarily with glomerular lesions.



The term "jade" is often used as the equivalent of GN. In fact, nephritis should be talked about when there are inflammatory changes in various parts of the nephron.

Diffuse glomerulonephritis is a clinical term emphasizing the presence of bilateral bacterial inflammation in the renal tissue with predominant primary involvement of glomeruli. It also includes morphological variants with segmental and focal glomerular lesions. The term "primary GN" emphasizes that GN develops independently of systemic connective tissue diseases, systemic vasculitis, or chronic infections. Diffuse GN and primary GN are synonymous. Acute GN implies the development of kidney damage after streptococcal disease and often has a cyclical course.

Chronic GN is the equivalent of the undulating, prolonged, latent course of diffuse GN. At the same time, the progressive nature of the process and the development of sclerotic and dystrophic changes in the renal tissue are emphasized. "Rapidly

progressing" GN is the most unfavorable form of the disease with an acute onset and malignant development, often leading to CRF after several months. The hematuric form of GN is a clinical definition that emphasizes the predominance of erythrocytes in urinary syndrome. This group of pathologies includes IgA, nephropathy (Berger's disease). Under the "mask" of the hematuric form of GN, various glomerulopathies of tubulointerstitial tissue and kidney vessels are hidden.

The mixed form of GN is a severe disease with pronounced renal and extrarenal manifestations, which has a progressive character due to the fact that the morphological basis is sclerosing forms of glomerular lesions. The diagnosis of "chronic GH with moderate or isolated urinary syndrome" should be supported by biopsy data. The diagnosis should indicate the type of morphological changes, the course variant, the degree of activity of the process and the nature of renal dysfunction in this case.

Etiology.

In the etiology of acute glomerulonephritis (OGN), the following are distinguished:

1. Streptococcal infection (more often it is beta-hemolytic streptococcus group A, type 1; 2; 4, after suffering from diseases such as sore throat, streptococcal impetigo, etc.);

2. Staphylococcus;

3. Pneumococcus;

4. Viruses: cytomegaloviruses, HBV viruses (cause fire in children in the Japanese population);

5. Plasmodia of malaria, pathogens of syphilis.

Fire can also occur due to other reasons:

1. vaccinations, medicinal substances;

2. Snake and bee bites;

3. hypothermia of the body.

Fire can also have a hereditary nature. These are Alport syndrome and Goodpasture syndrome.

Glomerular basement membrane (GBM) is a three-layer matrix with a thickness of about 300 nm. It consists of glycoproteins, includes fibropeptin, laminin, type IV collagen, negatively charged heparan sulfate proteoglycans.

There are various variants of type IV collagen chains, denoted respectively from a1 to a6 type IV collagens. Each chain encodes its own gene. These genes are arranged in pairs on 3 different chromosomes. Two of the type IV collagen chains are associated with the most important clinical syndromes: Alport, Goodpasture.

The pathogenesis of GN (modern concepts).

Currently, there is no previous opinion in the pathogenesis of OGN on how damage to the BMC (basement membrane of the capsule) occurs. There are many theories explaining the deposition of deposits on BM, but we will focus on the two most basic ones, which fully explain the damage to the glomeruli of the kidneys. The inflammatory reaction of the renal glomeruli (proliferation of epithelial and mesenchymal cells, exudative changes) develops under the action of IR with the participation of such secondary pathogenetic factors as the coagulation system, the complement system.

Nephritogenic streptococci contain a certain (unique) AnH that exhibits pathogenic properties. It binds to BMC, which, as we know, has a negative charge due to negatively charged heparan sulfate proteoglycans. As a result, structures are linked. During this process, connective tissue is disorganized. For a fixed AnH, the corresponding immunoglobulin G is produced, but immunoglobulins M and A. can also be detected. But at the age of 3 years, the structure of BMC includes a neonatal type of AnH, which binds to the non-collagenic structure of BMC. For this type of AnG AnT are not formed or are formed very slowly. This explains the absence of Goodpasture syndrome in children under 3 years of age.

The second mechanism. An infection enters the child's body. Accordingly, Ig G2, Ig G-3, and Ig A. are produced on the ingested AnG, which circulate in the blood. But Ig G has little affinity with macrophage receptors (whose function is the elimination of IR), so IR circulates in the blood for a long time. The IR fixation on the can also be

carried out due to the charge. I.e., a positively charged IR is attracted to the GBM. As a result, immune inflammation develops in BMC, which reflects an immediate type of hypersensitivity reaction. This mechanism is typical for acute and subacute GN. If connective tissue acts as AnH (as well as its non-collagenic structure), then autoimmune inflammation develops, i.e. BMC contains autologous ICS, which is manifested by a delayed hypersensitivity reaction. Its expression is the mesangial forms of GN. In particular, the immunocomplex mechanism is more common than the antibody one. Humoral reactions are responsible for the defeat of BMC - a large role is assigned to the complement system.

Pathological anatomy.

According to the topography, intra- and extracapillary processes are distinguished. By the nature of inflammation: exudative, proliferative, mixed. Along the course of the process: 1) acute; 2) subacute; 3) chronic. According to the prevalence of inflammation of the process, there are: 1) diffuse, 2) focal.

Acute GN — in the first days, glomerular hyperemia is expressed, then leukocyte infiltration of the endothelium and especially mesenchymal cells joins, therefore, the following phases are distinguished

1. Exudative;
2. Exudative-proliferative;
3. Proliferative.

Sometimes there may be a picture of necrotic acute GN. The kidneys are enlarged, swollen. The pyramids are dark red in color, the bark is grayish-brown in color, with small specks on the surface of the incision. A picture of a mottled kidney. These changes may be reversible, but they can persist for up to a year and transform into chronic GN.

Subacute GN is a rapidly progressive, malignant GN. It is characterized by extracapillary productive changes in nephrothelium and podocytes with the formation of characteristic half-moons. A picture of a large red kidney.

Chronic GN occurs in two main forms:

A. Change of mesangial cells in the form of: 1) mesangioproliferative - relatively benign course, 2) mesangiocapillary glomerulonephritis — rapid development leading to renal failure. The kidneys have the form: dense, pale, with yellow spots in the cortical layer.

B. Fibroplastic - the predominance of the processes of sclerosis and hyalinosis. This process can be focal and diffuse. The kidneys are usually reduced in size, they become dense, their surface is fine-grained.

Clinic.

It is not possible to briefly characterize the clinical course, manifestations and laboratory parameters in acute GN.

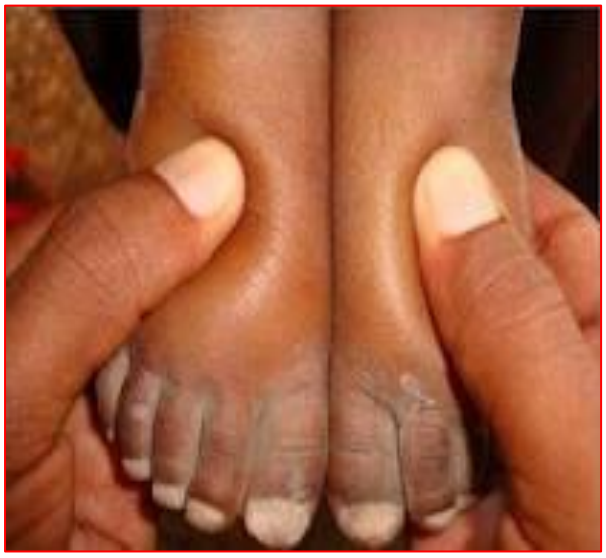
Acute GN (AGN) occurs 7-14 days after pharyngitis or 14-21 days after pyoderma caused by beta-hemolytic streptococcus group A, as well as acute GN after a viral infection, i.e. in the post-influenza period. In all patients (85-95%), the course of AGN (acute glomerulonephrit) develops into three periods that determine the cyclical course of the disease.

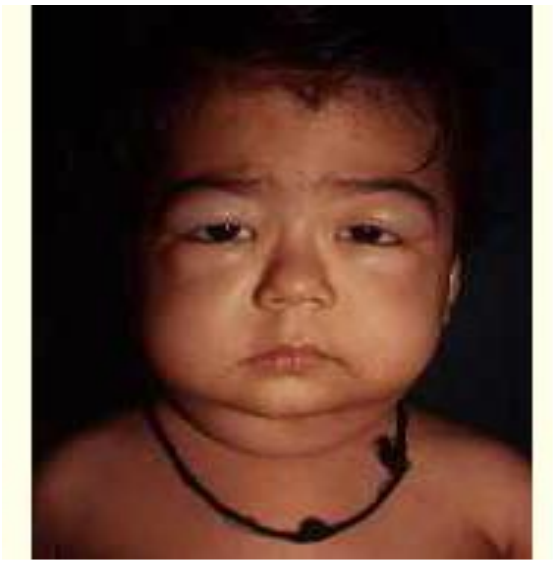
1. the initial period;
2. the period of reverse development of symptoms;
3. the period of complete clinical and laboratory remission.

In 10-15% of cases, a non—cyclical (unfavorable) course of the disease is possible, during which the transformation of AGN into a chronic form (CGN) is possible. A fatal outcome is also possible due to complications such as acute kidney injury.

Within 4-6 weeks, many patients show puffiness of the face, pallor of the skin, and a decrease in diuresis up to 200-300 ml / day. The urine is cloudy, sometimes the color of meat slops. Blood pressure is moderately elevated (130/80 - 150/90 mmHg). Sometimes there are vague pains in the abdomen and lumbar region, headache and vomiting appear. In the next 5-7 days, hypertension may increase in the absence of changes in the fundus. During this period, hematuria can reach macrohematuria. Proteinuria increases to 0.5—3.5 g / day, some patients have small, short-term (3-5 days)

leukocyturia (10-20 ml per day), as well as erythrocyte cylinders. Anemia, leukocytosis, and eosinophilia are often observed. Kidney functions are preserved or slightly impaired. Oliguria is combined with a high relative density of urine (up to 1013), unlike oliguria in acute renal failure, which manifests itself with a significant restriction of renal functions. Glomerular filtration is slightly reduced (up to 50-60 ml/min in endogenous creatine clearance), there may be a limitation of the function of osmotic concentration and excretion of H+ ions, maximum reabsorption of C6H1206. There is an increase in serum urea (hyperazotemia of the "acute period"), which persists for 3-5 days.





Extrarenal manifestations:

Ailments, poor appetite, lethargy, nausea.

Swelling (mainly on the face, in the evening on the shins, ankles).

Hypertension: headache, blood pressure, changes in the vessels of the fundus, changes in the cardiovascular system.

Pallor of the skin (facies nephritica).

With a cyclic course, from the end of 2-3 weeks, the reverse development of symptoms begins with a gradual decrease and elimination of hypertension and edema, hematuria, proteinuria. If individual symptoms persist for more than 6 months, we can

talk about a prolonged course of AGN, for more than 1 year - about the transition to a chronic form. In the initial period, the FIRE manifests itself:

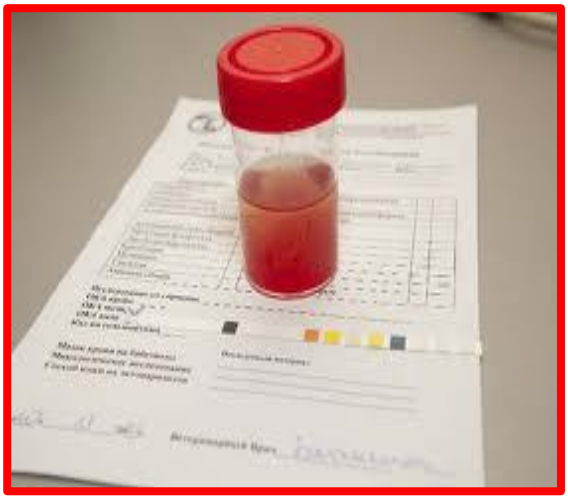
1) acute nephritic syndrome - edema, hypertension, hematuria, proteinuria - 2 g/day or more;

2) isolated urinary syndrome - hematuria, proteinuria without edema and hypertension;

3) nephrotic syndrome - edema, proteinuria of more than 2.5 g / day, hyperlipidemia, hypo- and dysproteinemia. In the initial period, eclampsia and acute heart failure are possible against the background of hypertension, as well as minor complications such as acute renal failure.

Nephrotic syndrome, as defined by Academician of the Russian Academy of Medical Sciences V. Yu. Shanin, is a pathological condition of the body due to the loss of plasma proteins with medium-sized molecules in urine and the reaction of homeostasis maintenance systems to a decrease in their concentration in blood plasma.

In the process of proteinuria, small and medium-sized proteins (up to 5.5 nm) are lost to the body - these are albumin (3.4 nm), transferrin (4.0 nm), immunoglobulin G (5.5 nm), proteins responsible for blood clotting, erythropoietin, as well as transport proteins.



In response, the liver increases its lipoprotein production functions. The mass of the liver increases, since together with albumin and transferrin, large proteins are formed

that have not been exposed to ultraviolet light — these are apolipoproteins B, E, fibrinogen. Their content in blood serum is increasing. The composition of blood plasma has changed dramatically. Onco- and osmotic pressures are falling. This causes plasma to exit from the vascular bed into the interstitium - edema forms. The volume of extracellular fluid increases due to the areactivity of the kidneys in relation to the natriuretic peptide, as well as pathological changes in the renal parenchyma.

Chronic GN is considered in the case when AGN becomes chronic (5-15% of cases). Chronic GN (CGN) develops in the following diseases:

1) membranoproliferative GN;

2) membranous nephropathy;

3) Alport syndrome;

4) systemic lupus erythematosus (SLE);

5) diffuse glomerulosclerosis;

6) hemorrhagic vasculitis;

7) fast-progressing GN;

8) Berger's disease.

There are three forms of CGN:

Nephropathic form - in preschoolers. It has a recurrent course. It is clinically manifested by edema of varying severity, proteinuria over 2.5 g / day, dysproteinemia, hyperlipidemia. Morphologically, there are minimal changes in the glomeruli, less often mesangioproliferative and other changes; kidney functions are preserved. The prognosis is favorable when using corticosteroid and cytostatic drugs. Hematuria and hypertension are not observed.

The mixed form is mainly found in schoolchildren. Clinically

It is characterized by a combination of nephrotic and hematuric syndromes. They can also be combined with hypertension (the prognosis is unfavorable). Early there is a violation of glomerular filtration and concentrating function of the kidney. Diffuse mesangioproliferative (MPGN), mesangiocapillary (MCGN) or fibroplastic glomerulitis with a tubulointerstitial component (TIC) is detected in the kidney biopsy. The

progression of this form of CGN depends on the timing and methods of the chosen treatment.

The hematuric form is manifested only to urinary syndromes in the form of hematuria - from micro to macro. It can be combined with minor proteinuria (up to 1 g / day). Kidney functions are preserved, blood pressure is normal. In the biopsy of the kidney, focal, less often diffuse MPGN, dystrophic changes in the epithelium of the tubules, focal sclerosis of interstitial tissue are detected. During luminescent microscopy, deviations of Ig G, M, complement, fibrinogen are noted. In 40-50%, isolated or total deviation of immunoglobulin A is noted. Serum levels of immunoglobulin increase at normal Ig O levels. This variant of GN is called Berger's disease - after the name of the author who described it (or Ig A - GN). A development link is assumed

diseases with persistent viral infection, not excluding the role of gluten in antigenic stimulation with subsequent hyperproduction of Ig A

7.1 Diagnosis and differential diagnosis.

AGN is diagnosed by sudden onset after exposure to the previously listed infectious (more often streptococcal) and non-infectious factors, as well as the presence of a combination of the main symptoms: edema, hypertension, urinary syndrome. OGN must be differentiated from the recurrence of CGN, which is characterized by a duration of more than 1 year, the absence or low severity of the exudative component in various morphological types of glomerulitis; from secondary GN, which developed against the background of SLE or other pathology; from GN, which developed against the background of congenital malformation of the kidneys such as dysplasia; from pyelonephritis.

HCG, its hematuric form, is diagnosed based on the presence of isolated urinary syndrome (hematuria, minor proteinuria), preservation of kidney function, Verger's disease, based on elevated levels of Ig A, Ig A-containing CIC, glomerular deposits, mainly Ig A. This form of GN must be differentiated from interstitial nephritis of

metabolic genesis or other nature, which can manifest itself as hematuria and proteinuria, restriction of tubular functions.

Laboratory tests conducted in case of suspected GN:

- urine tests;
- determination of serum complement (CZ and C4);
- determination of antibodies to streptococcus in serum;
- sowing from the throat, scraping from the skin;
- determination of serum albumin content;
- detection of the antinuclear factor;
- detection of hepatitis B markers;
- determination of the titer of antinuclear cytoplasmic antibodies.

Acute diffuse GN must be differentiated from toxic kidney lesions, acute pyelonephritis, DIC syndrome or related pathological conditions - hemolytic—uremic syndrome, thrombotic, thrombocytopenic purpura, exacerbation of CGN, hereditary nephritis. Toxic kidney damage in children develops in acute infectious diseases resulting from the toxicosclerotic effects of microorganisms on the vascular system of the kidney. Toxic kidney damage is accompanied by mild urinary syndrome (traces of protein in urine, microhematuria, single cylinders and renal epithelial cells in urine sediment), which are detected only in the midst of an infectious disease, and then disappears. AGN is difficult to distinguish from primary acute pyelonephritis. But the latter is characterized by pronounced general intoxication, anorexia, nausea, vomiting, weight loss, thirst, pronounced dysuric phenomena, significant leukocyturia with a predominance of neutrophiluria, bacteriuria. AGN is characterized by: edematous syndrome, arterial hypertension, hematuria, oliguria, impaired renal concentration function. In the anamnesis of children with hemolytic-uremic syndrome (Gasser-Ovren syndrome), which also has to be differentiated from AGN, acute respiratory diseases, acute gastrointestinal infections, sometimes taking several medications. The disease develops in young children (up to 2-3 years old). The hemolytic-uremic syndrome is

characterized by acutely developing hemolytic anemia, thrombocytopenia with severe hemorrhagic syndrome. An acute kidney injury may develop.

AGN with a prolonged course has to be differentiated from CGN during the period of exacerbation. It is necessary to carefully study the anamnesis, clarify the presence of past kidney disease and foci of chronic infection and sensitization in the body.

It should be noted that the exacerbation of CGN usually occurs 2-4 days after any infection, whereas the duration of the initial period with AGN is significantly longer (14-21 days); the patient has a history of symptoms of general intoxication, moderate pallor of the kidney and its dryness, the presence of arterial hypertension, anemia, hypoproteinemia with dysproteinemia, glomerular disorders and renal tubular functions for a long time and the duration of the renal process for more than 1 year indicate a chronic course of GN.

7.2 Treatment.

1. The mode and maintenance of electrolyte balance;
2. Antibacterial therapy;
3. Vitamin therapy;
4. Antihypertensive therapy;
5. Diuretics;
6. Pathogenetic therapy;

6.1.Glucocorticoids;

6.2. Immunosuppressive agents.

1. The mode and maintenance of electrolyte balance.

In case of FIRE, hospitalization is mandatory, bed rest for at least 1.5 months from the onset of the disease. The transfer to semi-bed rest provides for staying on the verandas. With a free hospital regime, the child should be on walks. Children are shown massage, diet.

Violation of ionic equilibrium - hypokalemia - leads to: muscle weakness and paresis leading to apnea, constipation and intestinal obstruction, as well as an increase in the likelihood of cardiac arrhythmias, fibrillation.

The maximum rate of administration and concentration of potassium solutions?

For correction, K+ must be administered intravenously (IV), and the concentration [K] in solution should not exceed 40 meq / l (if injected into a peripheral vein) and 80 meq / l (if injected into a central vein). The infusion rate should be no more than 0.3 meq/ kg/h. Faster administration can lead to phlebitis and paraesthesia, as well as cardiac arrest. If the child has a life-threatening condition (rhythm disturbances, fibrillation), then the infusion rate is increased to 1 meq / kg / h.

The loss of sodium is called hyponatremia. Correction is performed when clinical symptoms of this condition appear. They can be different: from gastrointestinal disorders, changes in mental status to seizures and coma. Such patients need to be injected with a 3% hypertensive solution. The introduction of 1 ml of the solution increases the concentration of sodium by 1 mEq/ l.

2. Antibacterial therapy.

In post-streptococcal GN, active antibacterial therapy mainly with penicillin or semi-synthetic drugs of the same group is mandatory for at least 1.5 months from the onset of the disease. In the presence of foci of infection, 1.5 - 2 months after the onset of the manifestation of the disease, it is necessary to carry out surgical rehabilitation for chronic tonsillitis.

3. Vitamin therapy.

Moderate doses of vitamin C for angioprotective effect. Vitamin (retinal) prevents the disorganization of connective tissue. Vitamins of group D have an antirachitic effect. Vitamins of group B are also prescribed.

4. Antihypertensive therapy.

Antihypertensive drugs are divided into 4 groups. Group 1 includes sedatives of central action — valerian, motherwort, etc. They are prescribed in cases of signs of exogenous hypercorticism. The 2nd group includes various antiadrenergic agents (rauwolfia

preparations, clofelin, ganglioblockers). The 3rd group includes nitroprusside n apressin. These drugs are practically not used in the treatment of GN. In the treatment of GN, drugs belonging to the 4th group are most often used - diuretics. Recently, attention has been drawn to the antihypertensive agent nifedipine, a calcium channel blocker, as well as to captopril.

5. Diuretics.

With edema of a nephritic nature, it is usually sufficient to use antihypertensive drugs against the background of a salt-free diet and bed rest. With nephrotic or mixed genesis of edematous syndrome, diuretics are used, which, depending on the mechanism of their action, can be conditionally divided into three groups.

Group 1 combines saluretics (dichlotiazide, furosemide, uregit, brinaldex). Dichlotiazide is widely recommended for the treatment of edematous syndrome in GN. Furosemide — with unexpressed or poorly expressed nephrosclerotic changes, the diuretic effect occurs within up to 4 hours at a dose of 1-5 mg / kg of body weight of the child. Unlike hypothyazide, furosemide has a lesser calyuretic effect. In conditions of a sharp decrease in kidney function, primarily due to sclerotic processes, certain transport systems are disrupted, which affects sensitivity to the action of diuretic drugs.

The 2nd group includes drugs that delay potassium in the body (aldactone n veroshpiron).

The 3rd group of drugs includes drugs that change osmotic pressure in the vascular bed. These include mannitol, rheopolyglucine. Osmotic diuretics can also include albumin and salt-free protein injected into the bloodstream in large quantities.

Differences between thiazide group diuretics and furosemide.

Dichlotiazide inhibits the reabsorption of sodium and chlorine in the initial part of the distal tubule with a higher probability than in the Henle loop. The onset of action is 1-2 hours after administration, the peak of action is 4-6 hours after administration. The effect is more prolonged compared to furosemide. Thiazides are less effective in edema, but more effective in hypertension, are indicated for hypercalciuria, are effective in

correcting acidosis caused by pathology of the proximal renal tubules, can cause hypovolemia, hypokalemia.

The effect of furosemide is based on the fact that it acts on the ascending knee of the Henle loop, thereby blocking Na, K, Cl - combined transport. In addition, it reduces the reabsorption of calcium and magnesium. It works 5 minutes after intravenous administration and 1 hour after oral administration. Peak 2 hours after administration.

6. Pathogenetic therapy.

Immunomodulation plays an important role, since excessive antibody formation often develops against the background of a primary hypoimmune condition. Equally significant is the inhibition of the effect of inflammatory mediators, complement, blood protease activity, platelets, kinins, lymphokines, histamine, prostaglandins.

6.1 Glucocorticosteroids - their widespread use in the treatment of GN is associated with their catabolic effect on mesenchymal cells, which leads to inhibition of proliferative sclerotic processes. This is especially pronounced when prednisone is combined with anticoagulants and antiplatelet agents.

6.2 Immunosuppressive agents are divided into several groups according to their mechanism of action:

Group 1 - antimitotics (cyclophosphane and chlorbutine);

Group 2 - antimetabolites (6-mercaptopurine, azathioprine);

Group 3 - enzymes (L-asparaginase);

Group 4 - antibiotics (chloramphenicol, mitocin C, actinomycin C, etc.);

Group 5 - alkaloids (vinblastine, vincristine, colchicine).

The combination of prednisolone with chlorbutine and heparin is indicated in the form of GN, when it is necessary to obtain a rapid diuretic effect or in cases of suspected fibroblastic changes in renal tissue. For the same purpose, the 4th component, curantil, is included in the complex of therapeutic agents. This so-called 4-component therapy of GN is most widespread in the treatment of membranoproliferative GN. In these cases, azathioprine at a dose of 2-3 mg / kg day is usually used as a cytostatic agent, not chlorbutin. Heparin is prescribed at a dose of 150-200 units / kg of body weight / day

parenterally, curantil — 3-5 mg / kg / day. The dose of prednisolone is usually about 1 mg / kg / day, with an intermittent or alternative course of therapy lasting at least 6 months.

It is important to know that glucocorticosteroids are contraindicated in RF, renal tissue dysplasia, sclerosing variants of GN, in the presence of severe hormone resistance, complications of hormone therapy (seizures, cataracts, steroidal diabetes, osteoporosis, gastrointestinal ulcers, persistent arterial and intracranial hypertension, herpes), in the presence of previous infections.

Control questions

1. Define glomerulonephritis;
2. Classification of GN in children;
3. Etiology and pathogenesis of GN in children;
4. How are the clinical and bacteriological features of GN manifested?
5. Diagnostic criteria of GN
6. What diseases should be treated with a differential diagnosis?
7. Principles of treatment of GN in children.
8. The tactics of GP in children with GN.

Test and Cases

1. 12 years old patient complains on an increase in body temperature to 39- 40C, intense pain in the ankle joints, especially at night, morning stiffness in the joints, disappearing in the afternoon. Objectively: the soft tissues in the area of both ankle joints are swollen, local moderate hyperthermia is detected by touch. Active and passive movements in them are limited in volume, difficult and sharply painful. Painless, pea-sized nodules soldered to aponeurosis are palpated on the extensor surfaces of both shins. In the blood test, leukopenia is noted, an increase in ESR to 40 mm/ hour, CRP++, RF is absent, and the content of α-2 globulins is increased to 15%. On the Rögram of both ankle joints, signs of effusion into the joint cavity and compaction of periarticular tissues were found.

task:
1. Formulate a nosological diagnosis;
2. List the process activity indicators;
3. What stage of the process do the listed Rh signs correspond to?

Task 2.
The boy is 15 years old, has been ill since the age of 3, when polyarthritis of symmetrical joints appeared against the background of subfebrile temperature – wrist joints, small joints of the hands, elbows. By the end of the first year of the disease, structural changes in the joints developed with the formation of ankylosis in the small bones of the wrist. To date, there is a noticeable lag in growth (especially in the upper extremities). The affected joints are spindle-shaped deformed, there are flexor contractures. No pathology was detected on the part of the internal organs. The skin is clean and pale. The feeling of morning stiffness persists until noon. Body temperature ranges from 37.4 to 37.6 C. Laboratory data reveal moderate hypochromic anemia and minor neutrophilic leukocytosis up to 10 X 103/ l, accelerated ESR up to 15 mm/hour, CRP+, RF positive, increased content of α-2-globulins up to 12%.
task:
1. Formulate a nosological diagnosis;
2. What is pannus?
3. Describe the articular syndrome in this disease.

Task 3.
A 6-year-old girl has been ill for three years. They are concerned about pain in the knee joints (including at rest), morning stiffness lasting up to 30 minutes, and subfebrility. Objectively: the condition is of moderate severity. The skin is pale; low nutrition. The knee joints are deformed: spherical in shape; the skin above the knee joints is of normal color; moderate local hyperthermia is detected palpationally above them. Movements in the knee joints are painful. The volume of active and passive movements in them is sharply limited. The gait has been changed (the patient moves with the help of crutches). No pathology was detected on the part of the internal organs. Bilateral uveitis is determined. A knee joint examination revealed the presence of the following signs of the disease: epiphyseal osteoporosis, cartilage loosening, narrowing of the articular gap, single erosions. Laboratory data: ESR – 15 mm/hour, CRP+, RF – negative, an increase in the level of α-2-globulins up to 12%.
Task:
1. Formulate the intended diagnosis.
2. Tactics of treatment of a particular patient.
3. Principles of outpatient follow-up for this disease

Task 4.
A 14-year-old boy has been suffering from chronic gastroduodenitis since the age of 9. Heredity is burdened: the paternal grandfather and the father have peptic ulcer disease.

My grandfather died of a perforated stomach ulcer. The child is being monitored and treated at the polyclinic at the place of residence. Over the past year, there has been a deterioration in the condition. He became more irritable, restless sleep, there was a distinct seasonality of exacerbations, a Moyningan rhythm of pain. The last 2 weeks. They are concerned about intense paroxysmal pain in the epigastrium and pyloroduodenal zone, with irradiation in the back, at night, on an empty stomach, late; sometimes vomiting occurs, without blood impurities. During palpation of the abdomen, a muscular defense. A positive symptom of Mendel.
Fibrogastroduodenoscopy: pronounced inflammatory changes in the mucous membrane of the stomach and duodenum. There is an oval-shaped defect on the mucous membrane of the bulb measuring 1.5 cm in diameter, surrounded by an inflammatory shaft with clear edges.
Task:
1. Formulate the intended diagnosis.
2. Tactics of treatment of a particular patient.
3. Principles of outpatient follow-up for this disease

Task 5
A 10-year-old boy was admitted to the nephrological department of the children's city hospital again with a diagnosis of glomerulonephritis. At the age of 9, swelling of the face, shins, and scrotum appeared against the background of chickenpox. Additional examination revealed proteinuria up to 4 g/day, hypo- and dysproteinemia, hypercholesterolemia, microhematuria up to 5 red blood cells per day. At the beginning of the disease, short-term hypertension up to 140/100 mmHg was noted, and blood urea levels increased to 10.6 mmol/l. Prednisone treatment at a dose of 2 mg / kg / day was effective, but when the dose was reduced to less than 15 mg / day, proteinuria and edema reappeared. Over the past year, I have had SARS three times and angina 1 time. Against the background of intercurrent diseases, relapses of kidney disease occurred. The edematous syndrome became more persistent over time. With the last exacerbation of the disease, proteinuria and moderate peripheral edema persist, hypertension persists for a long time in the range of 130-150 / 90-110 mmHg. In urine tests: protein-3.3 g / l, leukocytes - 10-15, erythrocytes – 5-10 in n /a.
Task:
1. Formulate the intended diagnosis.
2. Tactics of treatment of a particular patient.
3. Principles of outpatient follow-up for this disease

Task 6
A 9-year-old boy suffers from food and drug allergies, frequent acute respiratory infections, and sore throats. He suffered rubella, after which there were swelling on his face in the morning, fatigue, decreased appetite, headache. I was in the village, I was not observed by a doctor. After another 2 months, he suffered ARVI, which was accompanied by hoarseness of voice, an increase in body temperature to 39.5 C for 5

days. Against the background of the disease, oliguria and a progressive increase in edema are noted. He was hospitalized. Upon admission, anasarca, blood pressure 150/100 mmHg, macrohematuria were detected. Additional examination revealed anemia, hypoproteinemia, hypocholesterolemia, and an increase in urea levels to 14.5 mmol/L. In the future, despite the ongoing therapy with prednisone, chlorbutine, heparin, hypotensive and diuretics, the condition progressively worsened. After 4 months. from the onset of the disease, seizures occurred against the background of hypertension (BP-220/120 mmHg), proteinuria (20 g / day), creatininemia (0.12 mmol / l).
Task:
1. Formulate the intended diagnosis.
2. Tactics of treatment of a particular patient.
3. Principles of outpatient follow-up for this disease

Task 7
A 15-year-old patient was examined in the gastroenterology department for pain in the epigastric and pyloroduodenal regions. Intraventricular pH-metry revealed an increase in the acid–forming function of the stomach body: pH of basal secretion – 1.4; stimulated - 1.1. Fibrogastroscopy: diffuse hyperemia and edema of the gastric mucosa, hyperplastic lymphoid follicles in the antrum, spotted hyperemia of the mucous membrane of the duodenum 12. A urease test and enzyme immunoassay revealed HP (Helicobacter pylori).
Task:
1. Formulate the intended diagnosis.
2. Tactics of treatment of a particular patient.
3. Principles of outpatient follow-up for this disease

Task 8
A 10-year-old boy has been ill since the age of 5. From the very beginning of the disease, he developed generalized articular syndrome with damage to the cervical spine. Upon admission to the hospital, he complains of arthralgia, pronounced joint deformity, an increase in body temperature to 39C, weakness, morning stiffness in the joints that does not weaken throughout the day. Objectively: the condition is severe, the patient is pale, lags behind in physical development. There is a polymorphic rash on the skin of the face, trunk, and limbs. There is a spherical deformation of the knee joints and a fusiform one of the elbow joints. Their palpation is painful, the local temperature of the soft tissues in the area of these joints is moderately elevated. Similar symptoms were also found in the cervical spine. Peripheral lymph nodes are enlarged, painless, not soldered to surrounding tissues, the size of a pea. The boundaries of the heart are expanded, the tones are muted. A systolic murmur is heard at the top of the heart. Splenomegaly. Laboratory data: increased ESR up to 40 mm/hour, severe hypochromic anemia,

leukopenia, increased content of α-2-globulins up to 15%, CRP++, RF positive. The joint Rö-gram shows signs of osteoporosis with destruction of articular cartilage.
Task:
1. Formulate the intended diagnosis.
2. Tactics of treatment of a particular patient.
3. Principles of outpatient follow-up for this disease

Task 9
A 10-year-old girl complains of decreased appetite, pain in the umbilical zone of a blunt nature that occurs 1-2 hours after eating. The pain decreases with food intake. He has been ill for 2 years. Objectively: the skin is pale, the abdomen is soft, painful with deep palpation in the epigastrium. With FGS, the mucous membrane of the stomach and duodenum 12 is hyperemic, edematous, and covered with mucus. N.pylori ++.
Task:
1. Formulate the intended diagnosis.
2. Tactics of treatment of a particular patient.
3. Principles of outpatient follow-up for this disease

Task 10
The boy is 5 years old, his parents turned to the local doctor with complaints of paroxysmal cough, wheezing, shortness of breath.
I grew up and developed well until I was 2 years old. At the age of 3, during another acute respiratory viral infection, a suffocation attack occurred, which was stopped by inhalation of salbutamol only after 4 hours. In the future, the attacks were repeated 1 time in 3-4 months, were associated either with acute respiratory viral infections, or with eating chocolate, citrus fruits. Family history: father and paternal grandfather have bronchial asthma, mother has eczema. Got sick 3 days ago. Against the background of an increase in body temperature to 38.2 C, runny nose and sneezing were noted. On examination: the condition is of moderate severity. Body temperature 37.7 C, paroxysmal cough, wheezing breathing with prolonged exhalation. BH – 32 in 1 min. The mucous membrane of the pharynx is slightly hyperemic, granular. The chest is swollen. Above the lungs is a percussion sound with a boxy tinge. Whistling dry and wet wheezes are heard on both sides. The heart tones are slightly muted. Heart rate – 88 beats in 1 min.
General blood test: Hb – 120 g/l, Er – 4.6x10/L, Lake – 4.8x10/L, p/I – 3%, c – 51%, e – 8%, L – 28%, m – 10, ESR – 5 mm/hour.
Task:
1. Formulate the intended diagnosis.
2. Tactics of treatment of a particular patient.
3. Principles of outpatient follow-up for this disease

Task 11

The girl is 6 years old. Complaints of paroxysmal cough, wheezing.On artificial feeding from 2 months. She suffered from childhood eczema until the age of 1. It does not tolerate chocolate, strawberries, eggs.
Family history: the child's mother has recurrent urticaria, the father has gastric ulcer.
At the ages of 3 and 4, in May, outside the city, the girl had attacks of suffocation, which were stopped on their own when moving to the city. The real attack occurred after eating chocolate. On examination: the condition is of moderate severity. The skin is pale, blue under the eyes. There is dryness, peeling, and scratching on the cheeks, behind the ears, and in the natural folds of the arms and legs. The language is "geographical", there are jams in the corners of the mouth. The breathing is whistling, audible in the distance. Exhalation is prolonged. BH 28 in 1 min. Above the lungs, a percussive sound with a boxy tinge, auscultative: a mass of dry wheezes over the entire surface of the lungs. The boundaries of the heart: the right one is 1 cm inside from the right edge of the sternum, the left one is 1 cm inside from the left mid-clavicular line. The tones are muted. Heart rate – 72 beats/min. The belly is soft, used. Liver +2 cm. The spleen is not palpable. The chair is daily, decorated.
General blood test: Hb – 118 g/L, Er – 4,3x10/L, Lake – 5,8x10/L, p/I – 1%, c – 48%, E – 14%, L – 29%, M – 8%, ESR – 3 mm/hour.
Chest X-ray: pulmonary fields of increased transparency, increased bronchopulmonary pattern in the basal zones, no focal shadows.
1. What is your diagnosis?
2.Differential diagnosis
3. Therapeutic tactics
Task 12
Inna, 8 years old, has been suffering from bronchial asthma for 5 years. Daytime attacks daily, night attacks 1-2 times a week. The real attack developed after playing with the cat. The position in bed is forced – orthopnea, noisy breathing, expiratory shortness of breath, distant wheezing. The chest has a barrel shape. Periorbital cyanosis is pronounced. Additional muscles take part in the act of breathing.
Percussion over the lungs is a box sound, auscultation is harsh breathing, dry whistling wheezes. PSV is 34% of the norm.
Task:
1. Formulate the intended diagnosis.
2. Tactics of treatment of a particular patient.
3. Principles of outpatient follow-up for this disease
Test questions on pediatrics
1. Based on what symptoms can juvenile rheumatoid arthritis be suspected in a 3-year-old child:
A.pain in the knee joints and their swelling
B.allergic rash
B.abdominal pain
G.expanding the boundaries of the heart
D.enlargement of the liver and spleen

2. Which of the following can contribute to the development of juvenile rheumatoid arthritis in a child
A.psoriasis in the sister
B.Constitutional allergy
B. nosebleeds
G.Biliary dyskinesia
D.a history of acute intestinal infection

3. What clinical manifestations are characteristic of the development of juvenile rheumatoid arthritis in a 3-year-old child?
A.no changes in the blood test
B.stiffness in the morning in the symmetrical middle joints of the legs
B.Normal temperature
D.allergic rash
D.systolic heart murmur

4. Which of the above may not be a risk factor for the development of juvenile rheumatoid arthritis in children?
A.the presence in the anamnesis of exudate-catarrhal diathesis
B.frequent acute respiratory infections
B. Reaction to vaccination
D.temperature rise in acute respiratory viral infections
D.joint injury

5. The minimum mandatory examination for suspected diffuse connective tissue disease includes
A. General blood test
B. General urine analysis
B. biochemical blood test (proteinogram)
G. ECG
D. Echocardiography

9. The main pathomorphological changes in joints in jura include:
A. proliferative and fibroplastic processes
B. sclerosis of the joint capsule and periarticular tissues
B. formation of granulation tissue (pannus)
G. Exudative component
D. hemorrhage in the joint (hemarthrosis)

10. The examination plan for the JURA must include
A. General blood test
B. General urine analysis

V. Zimnitsky test
G. Holter monitoring
D. Echocardiography
E. determination of rheumatoid factor

11. Basic anti-inflammatory drugs in the treatment of JRA include
A. glucocorticoids
B. cytostatics
B. Gold preparations
G. of the quinoline series preparation

12. Treatment of gastritis B includes:
A. antiparasitic
B. Enzyme therapy
B. stimulants of gastric secretion
G. Antacid preparations

13. The attack period of bronchial asthma is characterized by:
A. respiratory dyspnea
B. itchy nose
B. lacrimation
D. dulling of the pulmonary sound
D. painful dry cough

14. Rheumatic lesions of the nervous system in children are usually observed:
A. diencephalitis
B. violation of cerebral circulation
B. small chorea
G. forms with ticks
D. cerebral vasculitis

15. Newborn pneumonia must be differentiated from everything except:
A. edematous hemorrhagic syndrome
B. perinatal encephalopathy
B. primary atelectasis
G. aspiration syndrome

16. For the diagnosis of diseases of the stomach and duodenum 12, use:
A. colonoscopy
B. esophagogastroduodenoscopy
B. irrigoscopy
G. retrograde pancreatic cholanography

17. Treatment of an acute attack of rheumatism with the phenomena of carditis and high activity necessarily includes everything except:
A. antibacterial drugs of the penicillin series
B. acetylsalicylic acid preparations
B. glucocorticosteroids
G. Sedatives

18. The minimum mandatory examination for suspected diffuse connective tissue disease includes:
A. FGDS
B. Duodenal sensing
B. proteinogram
G. UZ DG
D. ECHOCG
E. Bicycle ergometry

19. Prednisone for glomerulonephritis is indicated:
A. in all cases
B. with hematuric form
B. with nephrotic form
G. Contraindicated

20. Acute glomerulonephritis with nephrotic syndrome is characterized by:
A. leukocyturia
B. small proteinuria
B. massive proteinuria
G. microhematuria

21. Antihistamines of the 2nd generation include all drugs except:
A. loratodin
B. suprastin
V. zirtek
G. astemizole

22. Still's syndrome in rheumatoid arthritis is characterized by:
A. copious allergic rash
B. lymphopolyadenia
B. lesion of the cervical vertebrae
G. Kidney lesions

23. For the eradication therapy of peptic ulcer of the duodenum, all drugs are used, except:
A. de nola

B. flemoxin
V. klacida
G. gramurina

24. The features of an attack of bronchial asthma in young children are:
A. an abundance of dry wheezes
B. shortness of breath of a mixed nature
B. Forced situation
G. Inspiratory dyspnea

25. The methods of morphological diagnosis of chronic gastritis include all but:
A. FGDS
B. Mucosal biopsies

B. Chromogastroscopy
G. Intraventricular pH-metry

26. The basic therapy of bronchial asthma includes all drugs except:
A. Cromoglycanate Na
B. intal
B. Histaglobulin
G. tailed

27. Acute pneumonia in children is characterized by the following syndrome:
A. lack of intoxication
B. respiratory failure without signs of obstruction
B. diffuse changes in the lungs
G. Forced situation

28. The following antibiotics are effective in atypical pneumonia:
A. penicillins
B. cephalosporins
B. Macrolides
G. Aminoglycosides

29. The classification of acute pneumonia includes all forms except:
A. ochagovoy
B. focal drain
B. bronchiectatic
G. Segmental

30. The Zimnitsky test allows you to identify:
A. nicturia

B. leukocyturia
V. oxaluria
G. hematuria

31. The duration of antibacterial therapy in children with acute pneumonia with rapid onset of effect is:
A. 5 days
B. 6-7 days
B. 7-10 days
D. 10-14 days

32. For the treatment of pneumonia on an outpatient basis, it is prohibited to use:
A. penicillins
B. cephalosporins
V. lincomycin
G. Macrolides

33. The symptoms of acute pneumonia are all except:
A. shortness of breath
B. cyanosis
B. tachypnea
G. dry whistling wheezes

34. Gastritis with increased secretory function is characterized by:
A. pain after eating of a nagging nature
B. pain on an empty stomach, or 1.5-2 hours after eating, intense
B. belching with rotten food
G. Diarrhea

35. For the express diagnosis of helicobacter pylori infection,:
A. Identification of the microorganism
B. determination of titers of specific antibodies
V. "De-nol test"
G. Histological examination
36. During an objective examination of a patient with peptic ulcer of the duodenum, a positive symptom is noted:
A. Kera
B. Kerniga
V. Mendel
G. Murphy

37. Mendel's symptom is characterized by pain in:
A. palpation in the gallbladder area

B. pounding along the rib arc
B. shaking in the projection of small and large curvature of the stomach
G. pressure between the legs of M.sternoclaidomastoideus

38. The basic therapy of glomerulonephritis includes everything except:
A. Diet therapy
B. Physical therapy
B. Antibiotic therapy
G. antihistamines

39. Chronization of glomerulonephritis can be discussed if the activity of the process persists:
A. More than 3 months
B. more than 6 months
B. more than 1 year
G. More than 3 years

40. Gastritis with reduced secretory function is endoscopically characterized:
A. hypertrophy of the mucous membrane

B. subatrophic and atrophic mucosal changes
B. erosive changes
G. hemorrhages of the mucous membrane

41. In children with chronic gastritis, sanatorium treatment is indicated:
A. in the first 3 months after the exacerbation
B. 3 months after the exacerbation
B. 6 months after the exacerbation
G. 1 year after the exacerbation

42. In the initial period of rheumatic attack, it is shown:
A. penicillin
B. cefazolin
V. levomycetin
G. bicelin
D. gentamicin

43. With frequent recurrence of peptic ulcer of the duodenum, it is recommended
A. monotherapy
B. Double therapy
B. Triple therapy
G. Quadrotherapy

44. Gastritis associated with Helicobacter pylori refers to
A. Autoimmune (type A)
B. bacterial-conditioned (type B)
B. mixed (type A and B)
G. chemically-toxically induced (type C)

45. With chronic gastroduodenitis, abdominal pain
A. late
B. Night
B. Early and late
G. Late and night

46. The main side effect of NSAIDs is
A. gastrotoxic
B. hepatotoxic
B. Nephrotoxic
G. Cardiotoxic

47. Clinical signs of gastric ulcer perforation are:
A. dagger pain in the epigastric region and vomiting, which does not bring relief
B.subfebrile temperature
B. shingles in the abdomen
G.pronounced flatulence

48. Glomerulonephritis is a disease
A. Immunocomplex
B. non-immune
B. Microbial-inflammatory
G. Infectious

49. Nephrotic syndrome is characterized by
A.significant proteinuria, hypoalbuminemia
B.hypolipidemia
B. Hypertension
G. Hypofibrinogenemia

50. Laboratory indicators of activity in the hematuric form of glomerulonephritis are
A. Accelerated ESR
B. hyperfibrinogenemia
B. macrohematuria
G. All of the above

51. In what form of glomerulonephritis can abacterial leukocyturia be observed at the beginning of the disease?
A . with a hematuric form
B. with a mixed form
B. with nephrotic form
G. in all forms of glomerulonephritis

52. Which of the diuretics is advisable to use in a patient with glomerulonephritis with anasarca and the threat of pulmonary edema?
A. veroshpiron
B. veroshpiron in combination with hypothyazid
V. laziks
G. Uregit

53. Which symptom is not typical for cystitis?
A. pain in the lumbar region
B. pollakiuria
B. dysuria
G. pain in the suprapubic region

54. Hereditary nephritis is often combined
A. with hearing loss
B. with congenital heart disease
B. with glucosuria
G. with pyelonephritis

55. Rheumatic attack is most often preceded by
A. streptococcal infection
B. Injury
B. sepsis
G. intestinal infection

56. Rheumatic joint damage is observed
A. morning stiffness
B. recurrent polyarthritis
B. Nocturnal localized pain
G. benign course of polyarthritis

GLOSSARY

Abdominal syndrome - from Latin abdominalis – abdominal

Vitamin deficiency is a type of vitamin deficiency characterized by an almost complete lack of intake of any vitamin into the body. Polyavitaminosis is characterized by the non-absorption of several vitamins into the body at once.

Agranulocytosis (aleikia, granulocytopenia) is the absence of neutrophilic granulocytes (white blood cells) in the blood or a decrease in their number below 750 in 1 µl. Since it is these cells that protect the body from various infections, agranulocytosis is characterized by severe infections of various localizations.

Adynamia is a decrease or complete cessation of motor activity of a person (animal) or a separate organ.

Acrocyanosis is a cyanotic coloration of the skin of the extremities caused by venous congestion, more often with insufficiency of the right heart.

Alimentary - from the Latin alimentarius - food.

Allergization is the process of an organism acquiring hypersensitivity to any allergen substance (one or more), i.e. the occurrence of allergies. Allergies are at the root of allergic diseases (for example, bronchial asthma, hay fever, etc.).

Anamnesis is a set of information about a patient, about his disease, obtained by interviewing the patient himself and (or) people who know him. It is used to establish the diagnosis, prognosis of the disease, and the choice of optimal methods of its treatment and prevention.

Anasarca is a common swelling of the subcutaneous tissue.

Anatoxin is a bacterial exotoxin that has lost its toxicity as a result of prolonged exposure to formalin, but has retained its antigenic properties. It is used for immunotherapy and immunoprophylaxis.

Anaphylaxis is an immediate allergic reaction that occurs with parenteral administration of an allergen; common manifestations are anaphylactic shock, serum sickness; local manifestations are inflammation, edema, sometimes tissue necrosis.

Anemia is a condition characterized by a decrease in the number of red blood cells and (or) hemoglobin in the blood. Anemia leads to hypoxia. Causes: blood loss (posthemorrhagic anemia), increased hemorrhage (hemolytic anemia), hematopoiesis disorder (hypochromic anemia if the blood color index is less than 0.85 and hyperchromic anemia if the color index is higher than 1.05).

Anergy - 1. The absence of an organism's reaction to irritation; 2. A decrease in mental, motor and speech activity.

Anorexia is a lack of appetite in the presence of a physiological nutritional need.

An antigen is a substance (usually of a protein nature) that, when ingested, causes a specific immune reaction with the formation of an antibody.

Antibodies are serum globulins (proteins) formed in response to antigens entering the bloodstream.

Anuria is the absence of urine, occurs when the kidneys are damaged or the urinary tract is blocked (for example, by a stone).

Apnea is a temporary respiratory arrest.

Areflexia is the absence of deep tendon reflexes.

Arthralgia is pain in one or more joints.

Arthritis is an inflammation of a joint or several of its elements.

Aspiration - 1) penetration of foreign substances into the respiratory tract during inhalation; 2) procedure for sucking out the contents of a cavity or pathological focus.

Ascites is an accumulation of fluid in the abdominal cavity (dropsy of the abdomen). Causes: heart failure, cirrhosis of the liver, peritonitis, etc.

Atelectasis is the collapse of the pulmonary alveoli with compression of the lung, blockage of the bronchus (for example, a tumor), pneumonia, etc.

Auscultation is a study of internal organs based on listening and analyzing sound phenomena related to the activity of these organs.

Autoinfection is a disease caused by its own conditionally pathogenic microflora, which acquires pathogenic properties under unfavorable conditions for the body.

Acidosis is a decrease in the acidity index due to insufficient ventilation of the lungs (respiratory acidosis) or an irregular etiology (metabolic acidosis).

Bradycardia is a slowing of the heart rate to less than 60 per minute. It occurs normally (in athletes) and in various pathologies: it can accompany myocardial infarctions, cardiac conduction disorders, etc.

Inflammation is the body's reaction to a pathogenic irritant, manifested by a local increase in vascular permeability, tissue dystrophy and cell proliferation.

Gastralgia is a stomach pain.

Гематурия - наличие крови в моче (эритроцитов), видимой простым глазом (макрогематурия) или только при помощи микроскопа (микрогематурия); является симптомом ряда заболеваний и травматических повреждений почек и мочевыводящих путей. К таким заболеваниям относятся: гломерулонефрит, мочекаменная болезнь (при приступах), воспаление мочевого пузыря и мочеиспускательного канала, а также травмы мочевой системы.

Hepatolienal syndrome is a combined enlargement of the liver and spleen due to the involvement of both organs in the pathological process.

Hepatomegaly is a significant increase in liver size. It occurs in hepatitis, cirrhosis of the liver, and liver dystrophy.

Hepatosplenomegaly is a simultaneous significant enlargement of the liver and spleen. It is often found in cirrhosis of the liver.

Hepatotoxicity is the property of various chemical and pharmacological substances to have a toxic effect on liver cells.

A hepatocyte is a liver cell.

Hydropericardium is an accumulation of non-inflammatory fluid in the pericardial cavity.

Hydrothorax is an accumulation of non-inflammatory fluid in the pleural cavity.

Hyperbilirubinemia is an increase in bilirubin content in the blood, externally manifested by jaundice of the skin and sclera.

Hyperventilation is increased ventilation of the lungs due to increased and increased breathing.

Hypervitaminosis is a pathological condition of the body as a result of excessive intake of vitamins into it

Hyperglycemia is an increase in the concentration of glucose in the circulating blood on an empty stomach.

Hyperemia is an increase in blood filling in any area of the peripheral vascular system; limited redness of the skin area.

Hypercalcemia is an increase in the calcium content in the blood plasma.

Hypertension is a persistently high blood pressure.

Hyperthermia is an increase in body temperature. It is a protective reaction of the body, it is not recommended to "knock down" it if it does not exceed 38 ° C.

Hyperuricemia is an increase in the content of uric acid in the blood, characteristic of gout and some other diseases.

Hyperuricuria is an increased secretion of uric acid by the kidneys, occurs in uric acid diathesis, gout, and other diseases.

Hyperfermentemia is an increased content of enzymes in the blood serum.

Hyperesthesia is an increase in skin sensitivity to irritants.

Hypovitaminosis is a type of vitamin deficiency that develops due to insufficient intake of a certain vitamin into the body. It often occurs in the spring.

Hypoglycemia is a reduced blood sugar content. Normally, fasting blood sugar is 3.3 - 5.5 mmol/l.

Hypocalcemia is a decrease in the level of calcium in the blood plasma until the development of seizures.

Diarrhea is diarrhea.

Dysuric disorders are disorders of urination (pain, pain, burning, as well as increased urination).

Dysbiosis is a change in the composition and quantitative ratio of bacteria that normally inhabit the human intestine (as well as the skin, upper respiratory tract, oral cavity).

Dyspepsia is a digestive disorder in the gastrointestinal tract.

Dysproteinemia is a violation of the ratio between the protein fractions of blood plasma, characteristic of conditions accompanied by a violation of the protein-forming function of the liver (hepatitis, cirrhosis of the liver).

Diuresis is the amount of urine released over a certain period of time (day - daily diuresis, hour - hourly diuresis). In a healthy adult, the daily diuresis is 75% of the amount of liquid consumed.

A diuretic is a drug that causes increased urinary excretion.

Jaundice is a syndrome caused by the accumulation of bilirubin in the blood and tissues of the body (both direct and indirect), which leads to jaundice staining of the skin, mucous membranes and sclera.

Ventilator - artificial ventilation of the lungs.

The immune complex (antigen - antibody complex) is a stable compound of antigen and antibody that circulates in the blood for a long time and has the property of damaging healthy cells.

Clonic-tonic seizures are symmetrical tonic contractions of all muscle groups.

Clonic seizures are rhythmic contractions of individual skeletal muscles or muscle groups.

Glomerular filtration is the process of filtering blood plasma in the renal glomerulus.

Coma is a state of deep depression of the functions of the central nervous system, characterized by complete loss of consciousness, loss of reactions to external stimuli and a disorder in the regulation of vital body functions.

Crepitation is a characteristic crunching, determined by auscultation of the lungs

Leukopenia is a decrease in the number of leukocytes in the peripheral blood.

Lymphangiitis is an inflammation of the lymphatic vessel, usually from the focus of inflammation to the regional lymph node.

Lymphopenia is a decrease in the number of lymphocytes in the blood.

Lipotropic substances are compounds with the ability to prevent fatty

Macrophages are connective tissue cells with active mobility and pronounced ability to phagocytosis - the absorption and destruction of foreign cells (see phagocytes).

A mixed infection is a mixed infection when, in addition to one pathogen of an infectious disease, several are found in the body.

Myocarditis is an inflammation of the heart muscle; it is manifested by signs of impaired contractility, excitability and conduction.

Mucolytics are drugs that dilute sputum.

The mucociliary apparatus is the ciliary cells of the bronchial mucosa, which provide purification of the bronchial tree by promoting bronchial secretions.

Nonspecific humoral immunity factors are a variety of plasma proteins with antimicrobial properties or properties to activate other immune mechanisms. These include proteins of the complement system, lysozyme, transferrin, and C-reactive protein

The nephron is a structural unit of the kidney, represented by a glomerular capsule and tubules. Performs the function of blood filtration and urination.

Oliguria is a decrease in urine excretion. It can be physiological (in newborns, with increased sweating, etc.) and pathological (with renal failure, shock, hemolysis).

Omphalitis is an inflammation of the skin and subcutaneous tissue in the navel area.

Palpation is a method of examining the body or organs using touch.

Paraclinical studies are laboratory research methods.

Percussion is a method of examination consisting in tapping on the human body and listening to specific percussion sounds.

The perinatal period includes the time before childbirth — antenatal, during childbirth — intranatal and after childbirth — neonatal periods.

Pneumosclerosis is an overgrowth of scar connective tissue in the lungs, which leads to a violation of their function.

Pneumothorax is an accumulation of air in the pleural cavity.

Polyarthritis is an inflammatory injury to many joints at the same time.

Polyuria is an increase in the volume of urine excreted per day over 1800 ml.

Reabsorption is the process of reabsorption of certain substances from the renal tubules back into the blood, for example, glucose.

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