MINISTRY OF HEALTH OF THE REPUBLIC OF UZBEKISTAN

TASHKENT MEDICAL ACADEMY

Dadabaeva N.A., Mirahmedova H.T., Abdullaev U.S.,

ACUTE GLOMERULONEPHRITIS

Training and methodological manual

(for teachers, students, clinical residents and masters of medical universities)

Tashkent 2025.

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«Approved»

Vice-Rector for Academic Affairs TMA

Boymuradov Sh.A.

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Compilers:

Mirahmedova H.T. Doctor of Medical Sciences, Associate Professor - Head of the Department of Propaedeutics of Internal diseases №1Tashkent Medical Academy
Dadabaeva N.A., Candidate of Medical Sciences - Associate Professor of the Department of Internal Medicine Propaedeutics №1 of the Tashkent Medical Academy
U.S. Abdullaev - Assistant of the Department of Internal Medicine Propaedeutics №1 of the Tashkent Medical Academy

Reviewers:

Abduazizova N.H. - Candidate of Medical Sciences, Associate Professor, Department of Faculty and Hospital Therapy №1 with Occupational Pathology, Tashkent Medical Academy

Abdullaev A.H. - Doctor of medical sciences, senior researcher, Republican Specialized Scientific and Practical Medical Center of Therapy and Medical Rehabilitation.

This textbook is intended for teachers, students, clinical residents and masters of medical universities.

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Secretary of the HEI Council: G.A. Ismailova

TOPIC: ACUTE GLOMERULONEPHRITIS

Notion

Glomerulonephritis (GN) is an immune-mediated inflammation with predominant initial damage to the tubules and involvement of all renal structures, clinically manifested by renal and/or extrarenal symptoms. Glomerulonephritis (GN) includes a group of morphologically heterogeneous immunoinflammatory diseases with predominant damage to the tubules, as well as with the involvement of tubules and interstitial (interstitial) tissue. GN are independent nosologic forms, but can also occur in many systemic diseases, such as systemic lupus erythematosus, hemorrhagic vasculitis, infectious endocarditis, and others.

Epidemiology

Glomerulonephritis (GN) represents an important section of modern medicine, according to B.I. Shulutko, for 36.76% of all renal pathology. In recent years, there have been many works devoted to the role of endothelial disorders in the genesis of vascular pathology. The prevalence of GN from 2000 to 2005 amounted to 8.3 per 10 thousand population, and the share of GN in the structure of kidney diseases amounted to 4.9%. Glomerulonephritis ranked 4th in the prevalence structure after tubulo interstitial nephritis (1st place), urolithiasis (2nd place) and other diseases (3rd place). In the period 2006-2010, the prevalence of GN increased again from 8.3 to 12.7 per 10,000 population, i.e. 1.5 times.

Motivation

At present, the study of glomerulonephritis is of great importance, as the incidence of this disease among the population has increased and complications have become more frequent. Taking into account the peculiarities of the course, classification, clinic, treatment of the disease, this educational methodological

The manual will be useful for medical students. This manual is based on up-to-date medical data.

In medical practice, glomerulonephritis (nephritis) is not as common as, for example, ischemic heart disease, rheumatic diseases or chronic nonspecific lung diseases. However, their medical and social importance is great, which is determined by the following provisions.

Firstly, acute nephritis, ends in no more than half of cases of recovery; chronic nephritis, especially sub acute, steadily progresses towards chronic renal failure (CKD), often accompanied by edema (nephrotic syndrome) and severe hypertension, leading to disability even before the development of CKD.

Second, young, able-bodied men are more often (and more severely) affected.

Thirdly, although there are methods to replace non-functioning kidneys - dialysis and transplantation, but they are not available to everyone (the need is satisfied by about 1/20), are very expensive and have their own difficulties - attachment to devices, the need for constant immunosuppression, etc. All this makes urgent the problem of early diagnosis and conservative therapy aimed at suppressing the activity of nephritis and inhibition of its progression. A doctor of any specialty should know the symptoms characteristic of glomerulonephritis and be able to choose the right tactics in relation to patients with this disease. It should be emphasized the importance of careful outpatient observation of patients, dietary recommendations, timely detection of exacerbations, and timely referral of patients to nephrologists.

Model of learning technology

Form of training session	Practical training
Class structure	1.Introduction 2.Theoretical part 3.Practical part 4.Analytical part -Situational tasks -Tests
Learning Objective:	Deepening and expanding knowledge of the diagnosis of acute glomerulonephritis.Development of the ability to assess and analyze symptoms in the treatment of patients. Skills and skills of writing a medical history.
The student should know	- etiology and pathogenesis of acute glomerulonephritis - Clinic, diagnosis, differential diagnosis of acute glomerulonephritis - acute glomerulonephritis therapy - a group of drugs for the treatment of acute glomerulonephritis - prevention of acute glomerulonephritis
The student is required to perform	- be able to manage a patient with acute glomerulonephritis - interpret the results of laboratory and instrumental examination methods - Diagnosis and differential diagnosis of acute glomerulonephritis - prescribe therapy
Objectives faculty member:	- To consolidate and deepen the knowledge on assessment and analysis of the general condition of patients with acute glomerulonephritis, to develop skills to rational therapy
Learning outcomes activities:	- assess and analyze the situation and general condition of patients with acute glomerulonephritis - choose an algorithm of actions to make a diagnosis. - develop an algorithm for the treatment of patients with acute glomerulonephritis
Teaching methods	- Interactive methods, management, work with literature
Means and form of instruction	- textbooks, manuals, banners, slides
Assessment	- points

Theoretical part

Etiology

The main etiologic factor is the transferred streptococcal infection (sore throat, pharyngitis, exacerbation of chronic tonsillitis, scarlatina, skin rash). Most often acute glomerulonephritis is caused by 12 and 49 strains of β-hemolytic

group A streptococcus (post-streptococcal glomerulonephritis). Other possible etiologic factors may be hepatitis B virus (causes mainly membranous nephritis), rubella, infectious mononucleosis, herpes viruses, adenoviruses, in some cases acute glomerulonephritis may develop after staphylococcal or pneumococcal infection (non-streptococcal glomerulonephritis)

Acute glomerulonephritis caused by different types of infection are called *infectious-immune* glomerulonephritis.

Along with this may develop acute glomerulonephritis after the introduction (especially repeated) of vaccines, serums, as well as due to individual intolerance hypersensitivity to certain drugs and chemicals, plant pollen, insect venom. It is also possible to develop glomerulonephritis due to alcohol intoxication. These variants constitute a group of *non-infectious-immune glomerulonephritis.*

Genetic predisposition to acute diffuse glomerulonephritis also plays a major role.

Pathogenesis

In the induction of HD, the leading role is given to immune damage factors, reactions of humeral and/or cellular immunity; in further progression, inflammatory mediators and non-immune mechanisms - hemodynamic and metabolic - play an important role. At all stages of development, the leading role is played by persistence of the etiologic factor, known, unfortunately, only in 1/10 patients.

Immunocomplex HS: CECs are deposited on the vascular endothelium in tissues, phagocyte by hepatic Kupffer cells or eliminated through the tubular filter. Once in the tubule, CICs are deposited in the capillary wall, causing micro-circulatory disorders. It is assumed that IRs.

activate blood coagulation factor XII (Hageman's factor) and platelet aggregation, and their release of factor III is carried out by a complement-dependent mechanism. Direct and mediated (through collagen) activation of factor III is possible. The result of hypercoagulation is microthrombosis, which leads to micronecroses. The latter cause reactive inflammation as the third stage of the disease.

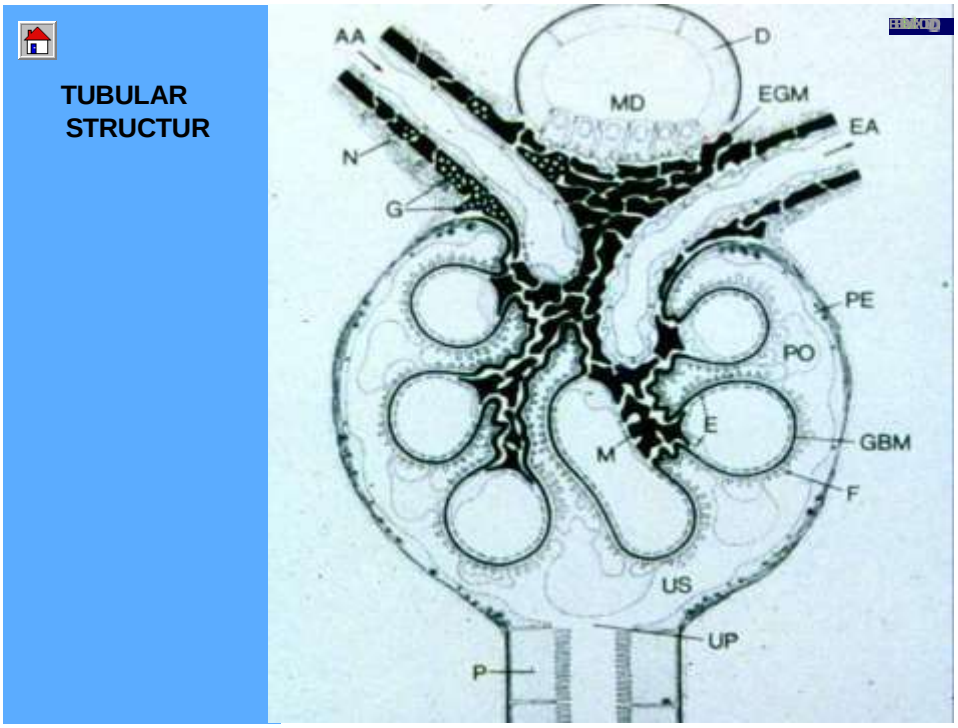
GN with antigenic **mechanism: the** source of antigenemia is the basal tubular membrane itself, which has been previously damaged by chemical or toxic factors. AT are produced directly against the basal membrane. Antigens in immune complexes can be exogenous (antigens of streptococci, staphylococci and other bacteria; antigens of hepatitis B virus and other viruses; foreign protein in serum sickness, etc.) or endogenous.

Among exogenous antigens, streptococcal antigens are of the greatest importance, since post-streptococcal glomerulonephritis is the most frequent variant of acute glomerulonephritis. Streptococcus antigens are its toxins (streptolysin, streptokinase, hyaluronidase), M-protein, lipoproteins, teichoic acid, neuraminidase are also considered as nephritogenic antigens of streptococcus. Sometimes a streptococcal antigen, cytoplasmic protein endostreptolysin with a molecular mass of 46 kilodaltons, is detected in the kidneys as part of immune complexes.

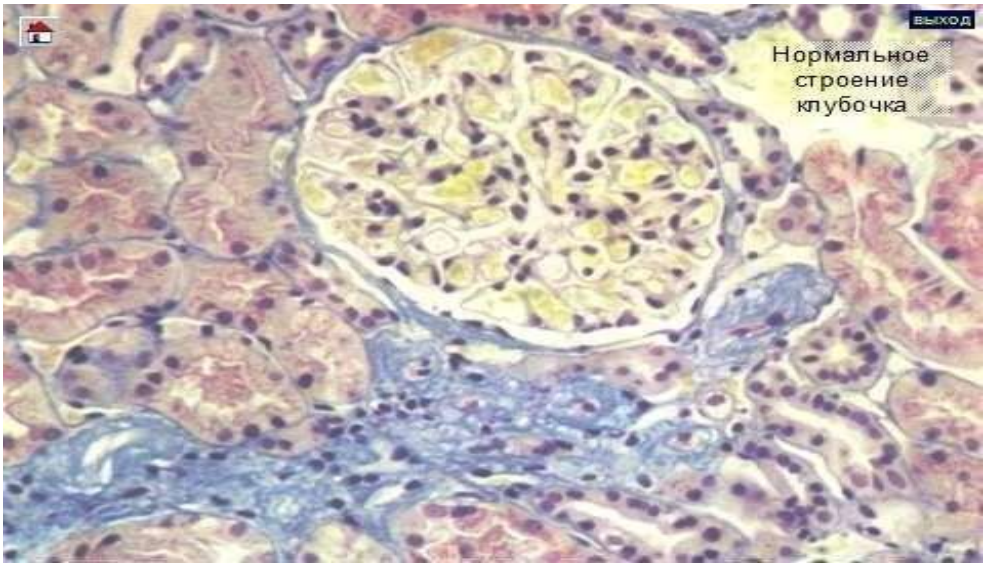
Endogenous antigens are subdivided into extraglobular and glomerular antigens. Extraclubular antigens include antigens (DNA) of cell nuclei in SLE, cryoglobulins, tumor components, etc. Clubular antigens include antigens of renal tubular epithelial cells - glycoprotein GP330, mesangial antigens, as well as antigens of the basal capillary membrane of the tubules (see below), endothelial antigens. The role of these antigens in the pathogenesis of acute glomerulonephritis has been proven experimentally.

Antigens introduced into the kidney or circulating in the blood and subsequently deposited in the kidney induce the formation of appropriate antibodies. The antigen introduced into the kidney interacts with a specific antibody circulating in the blood, resulting in the formation of a local immune complex. In addition, immune complexes formed in the bloodstream are deposited in the kidney.

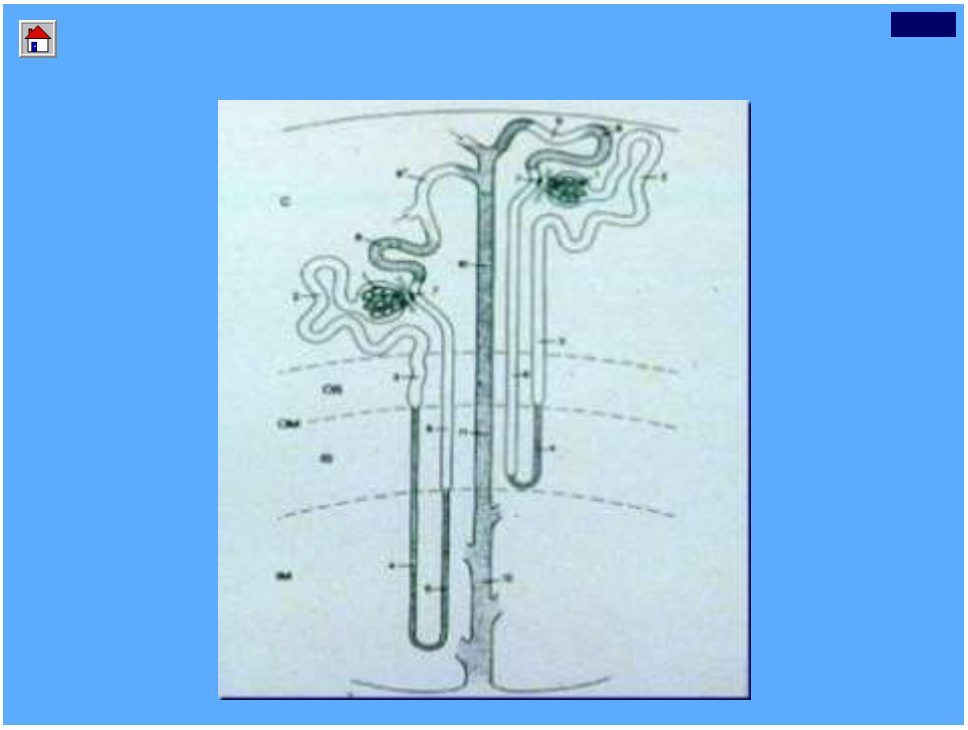
Structure of the tubule and structure of the nephron



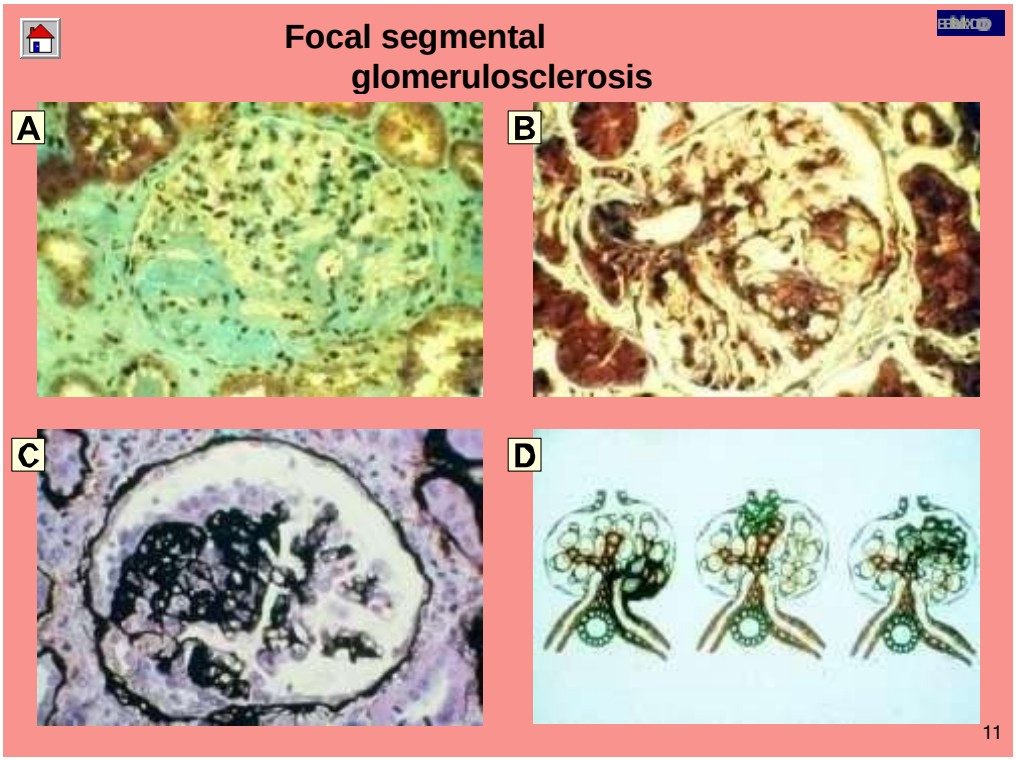
Normal tubular structure



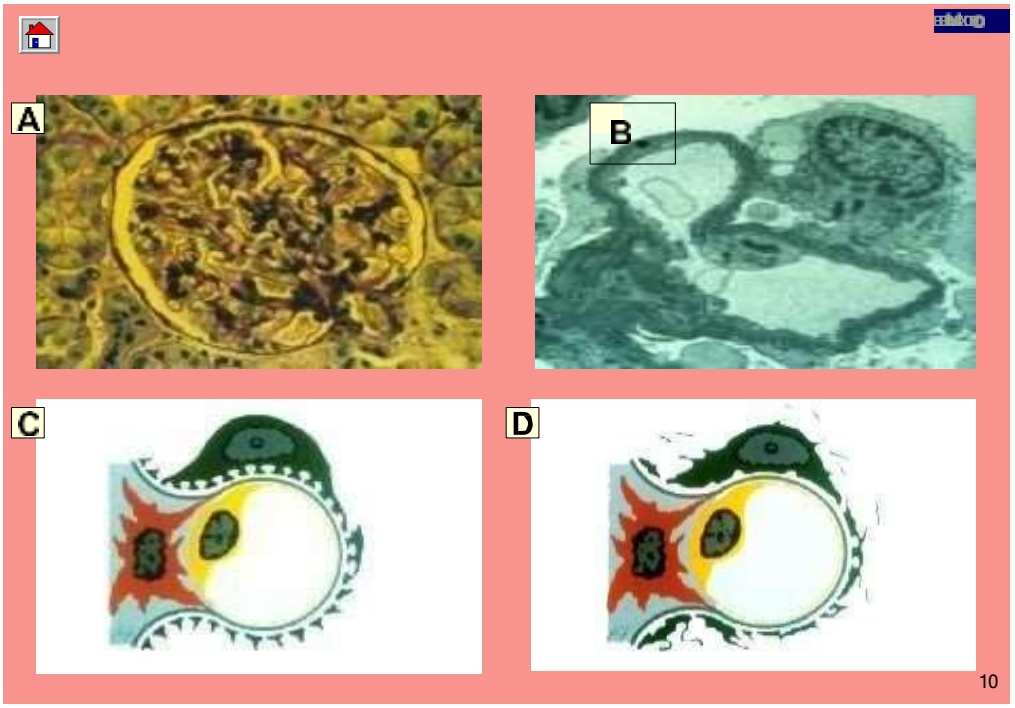
NEPHRON STRUCTURE

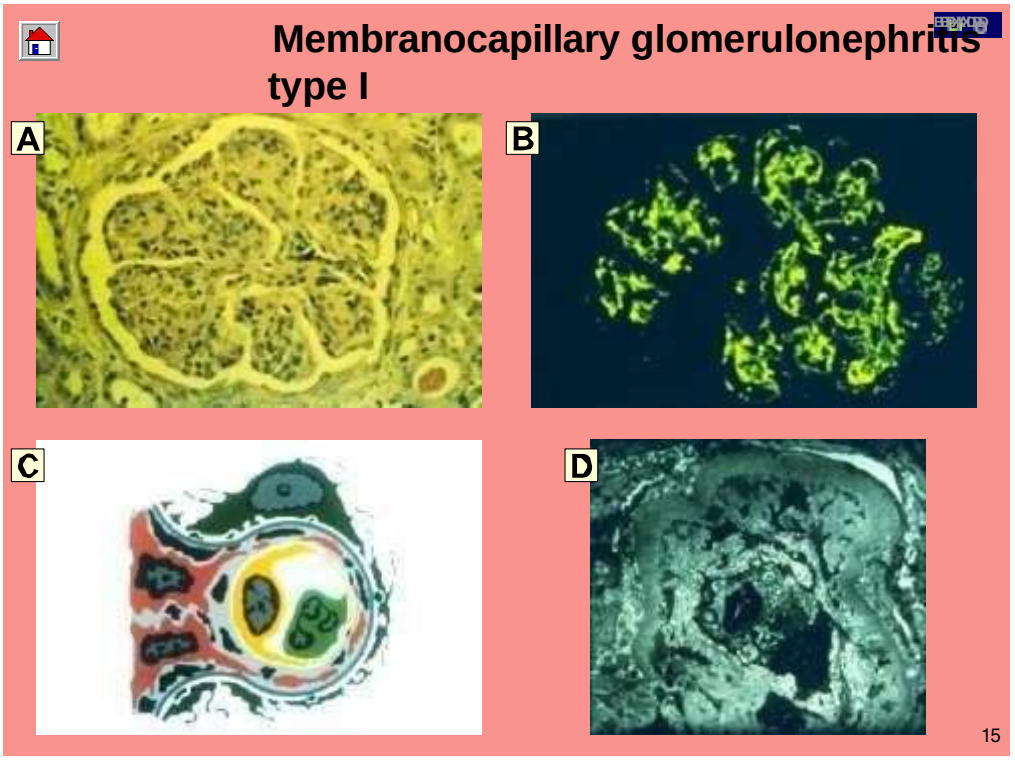


Morphologic variants of chronic glomerulonephritis



Glomerulonephritis with minimal changes





Clinical classification of acute glomerulonephritis:

1. Latent form of AGN;
2. The cyclical form of AGN;

Morphological classification according to Serov V.V., 1987.

Acute glomerulonephritis:

1. Proliferative endocapillary.
2. Proliferative extracapillary
3. Mesangioproliferative
4. Mesangiocapillary
5. Sclerosing (fibroplastic)AGN:
6. In the exudation phase.
7. Exudative-proliferative phase
8. Proliferative phase
9. Residual phase

Clinic

The disease is characteristic mainly of children and young people. Acute diffuse glomerulonephritis develops in 2-3 (sometimes 1-2) weeks after an infection, most often streptococcal (sore throat, tonsillitis, scarlatina, pyoderma, impetigo).

Hematuria (often macrohematuria), edema, oliguria, hypertension are characteristic. In children, acute GN usually has a cyclic course, with a rapid onset, in most cases ends with recovery. In adults, a sterile variant with urinary changes without general symptoms is more common, gradually taking a chronic course. If the connection with streptococcal infection is proved, titers of antistreptococcal antibodies are elevated and pharyngeal culture is positive, antibiotic treatment for 8-12 days is indicated.

Urinary syndrome: in the urine appear protein (proteinuria), form elements (hematuria, leukocyturia), cylinders (cylindruria).

Proteinuria in AGN is caused by the passage of protein macromolecules through the "pores" of the tubular capillary wall, which under the influence of immune complexes deposited on the basal membrane are enlarged

In addition, immune complexes can cause localized changes

of the capillary wall, increasing its permeability. Through the glomerular "filter" in AGN excrete predominantly albumin - the so-called selective proteinuria (in non-selective proteinuria with urine excrete medium- and high molecular weight proteins, mainly globulins).

The pathogenesis of hematuria is completely unclear. Involvement of the mesangium as well as interstitial tissue involvement are thought to be important. Hematuria may depend on necrotizing inflammation of renal arterioles, renal intravascular hemocoagulation. Erythrocytes pass through tiny ruptures in the basal membrane, changing their shape.

Leukocyturia is due to inflammatory changes in the urinary tract and is uncharacteristic of AGN.

Cylindruria - excretion with urine of protein or cellular formations of tubular origin, having a cylindrical shape and different sizes. Granular cylinders consist of dense granular mass, formed from decayed renal epithelial cells; their presence indicates dystrophic changes in the tubules. Wax-like cylinders have sharp contours and homogeneous structure. Hyaline cylinders are protein formations; it is assumed that they are formed by a glycoprotein that is secreted in the tubules.

Hypertensive syndrome is due to three major mechanisms: 1) sodium and water retention; 2) activation of renin-angiotensin-aldosterone as well as sympathetic-adrenal systems; 3) decreased function of the renal depressor system.

Oedema syndrome is attributed to the following factors: 1) decreased glomerular filtration due to tubular damage; decreased sodium filtration charge and increased sodium reabsorption; 2) water retention due to sodium retention in the body; 3) increased circulating blood volume; 4) secondary hyperaldosteronism; 5)

increased secretion of ADH and increased sensitivity of distal parts of the nephron to it, which leads to even greater fluid retention; 6) increased permeability of capillary walls, which contributes to the escape of liquid blood into the tissues; 7) decreased oncotic pressure of plasma in massive proteinuria.

Acute glomerulonephritis always begins suddenly and first has general manifestations:

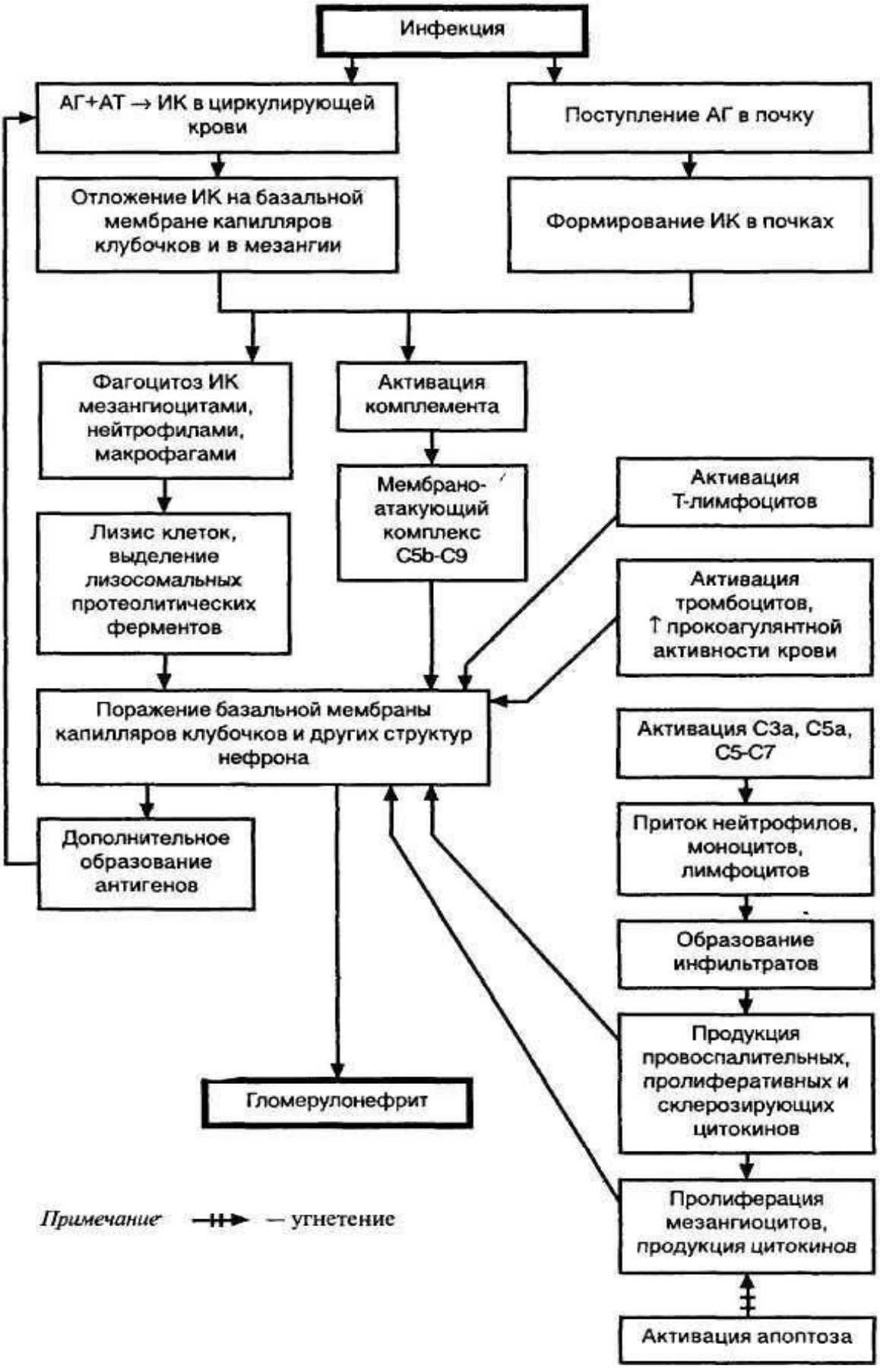
-Elevation of body temperature to critical values;

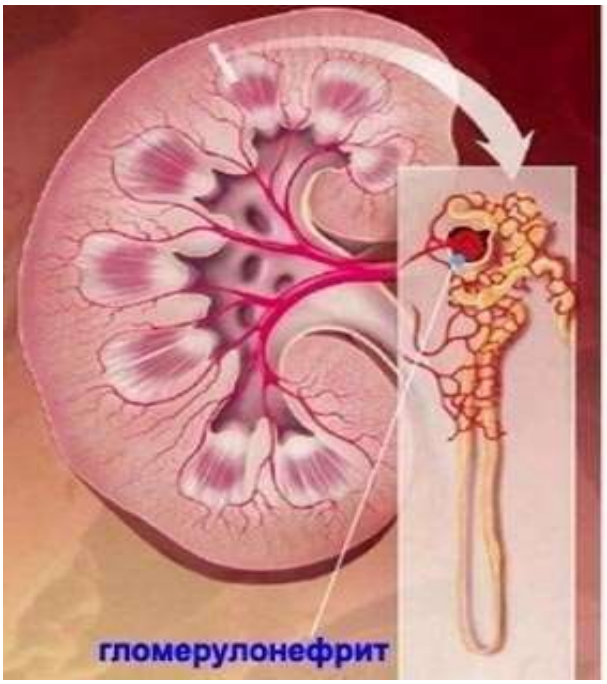
-headache;

-nausea, sporadic vomiting may occur;

-pain syndrome в area lumbar region - this symptom may be absent;

-general weakness, increased drowsiness.


Инфекция
АГ+АТ → ИК в циркулирующей крови
Поступление АГ в почку
Отложение ИК на базальной мембране капилляров клубочков и в мезангии
Формирование ИК в почках
Фагоцитоз ИК мезангиоцитами, нейтрофилами, макрофагами
Активация комплемента
Мембрано-атакующий комплекс C5b-C9
Активация Т-лимфоцитов
Лизис клеток, выделение лизосомальных протеолитических ферментов
Активация тромбоцитов, ↑ прокоагулянтной активности крови
Поражение базальной мембраны капилляров клубочков и других структур нефрона
Активация C3a, C5a, C5-C7
Дополнительное образование антигенов
Приток нейтрофилов, моноцитов, лимфоцитов
Образование инфильтратов
Продукция провоспалительных, пролиферативных и склерозирующих цитокинов
Гломерулонефрит
Пролиферация мезангиоцитов, продукция цитокинов
Активация апоптоза
Примечание — угнетение



Основные признаки гломерулонефрита:

- гипертония
- отеки
- протеинурия
- гематурия

The clinical and laboratory manifestations of acute glomerulonephritis can be grouped into four major syndromes.

Acute inflammatory bowel syndrome.

The syndrome of acute inflammation of the tubules is characterized by pain in the lumbar region on both sides; increased body temperature; oliguria, reddish or "meat slop" colored urine; proteinuria, microhematuria (rarely macrohematuria), the appearance of cylinders (hyaline, granular, erythrocytic), epithelial cells in the urine; decreased glomerular filtration; leukocytosis, increased sedimentation; increased blood α_2 and γ-globulin. Immunological blood analysis reveals increased IgG, IgM, rarely IgA (normalization of these parameters occurs during the first 2 months from the onset of the disease), circulating immune complexes, transient cryoglobulinemia, fibrinogen complexes with high molecular weight, high titers of antibodies to streptococcal antigens, decreased C3, C5-fractions of complement. Blood coagulation system disorders are also characteristic - hypercoagulability (shortening of thromboplastin time, increased prothrombin index, decreased antithrombin III, suppression of fibrinolytic activity of blood), and as the severity of acute glomerulonephritis grows

gradually increases anticoagulant activity, fibrin and fibrinogen degradation products appear in blood and urine *(markers of DIC).*

Cardiovascular syndrome

The main manifestations of cardiovascular syndrome: dyspnea (reflects heart failure and in some cases - nephrogenic pulmonary edema of varying severity); rarely - hemoptysis (usually in Goodpasture's syndrome); arterial hypertension (sometimes ephemeral); possible development of acute left ventricular failure with the appearance of cardiac asthma and pulmonary edema; tendency to bradycardia; changes in the ocular fundus - narrowing of arterioles, crossing phenomenon, sometimes edema of the optic nipple, spot hemorrhages. On ECG diffuse myocardial changes are determined (decrease in the amplitude of the T-valve in several leads). Arterial hypertension is due to activation of the renin-angiotensin-aldosterone system, an increase in circulating blood volume.

Edema syndrome

The edema syndrome is characterized by "pale" edema mainly in the face, eyelids (appear in the morning), in severe cases may be anasarca, hydrothorax, hydropericardium, ascites. Some patients do not have obvious edema, but there is a daily weight gain, indicating fluid retention in the . Edema is caused by decreased glomerular filtration, increased tubular reabsorption of sodium and water, the development of hypoalbuminemia and decreased oncotic blood pressure, increased secretion of aldosterone and antidiuretic hormone, increased capillary and tissue permeability due to increased activity of hyaluronidase, leading to depolymerization of hyaluronic acid of the main substance of connective tissue.

Cerebral syndrome

Cerebral syndrome is caused by cerebral edema, its characteristic manifestations: , nausea, vomiting, fog before the eyes,

decreased vision, increased muscular and mental excitability, motor restlessness; hearing loss, insomnia.

The extreme manifestation of cerebral syndrome is *angiospastic encephalopathy (eclampsia).* The main symptoms of eclampsia, after a shriek or noisy deep breath appear first tonic, then clonic convulsions of the muscles of the limbs, respiratory muscles and diaphragm; complete loss of consciousness; cyanosis of the face and neck; swelling of neck veins; pupils are wide; blood-colored foam flows from the mouth (tongue biting); noisy, snoring breathing; pulse is rare, tense, blood pressure is high; muscle rigidity; pathological reflexes.

The course of acute glomerulonephritis

Acute diffuse glomerulonephritis is characterized by the following clinical forms:

Acute cyclic: with rapid onset, marked renal and extrarenal symptoms - more common in children and adolescents, usually with recovery.

1. Protracted (acyclic), gradual development of symptoms, slow increase in edema, low severity of arterial hypertension and other symptoms, duration of the disease 6-12 months.

2. Unfolded (with triad of symptoms: edema, hypertension, urinary syndrome) or triadic classical form.

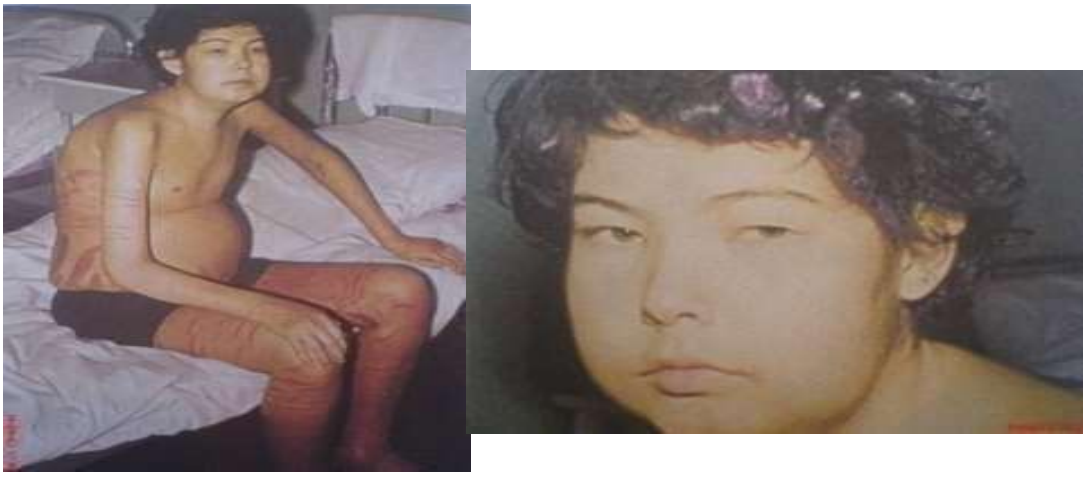
3. Bisyndromic (urinary syndrome combined with arterial hypertension or nephrotic syndrome).

4. Monosymptomatic.

- Monosymptomatic edema (edema without marked changes in the urine);
- Monosymptomatic hypertensive (predominantly arterial hypertension without edema and marked changes in urine);
- Hematuric (predominates in the clinic);
- c isolated urinary syndrome (without extra renal manifestations, i.e., edema-free and arterial hypertension).

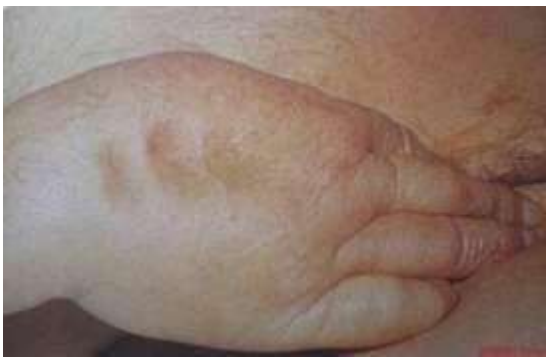
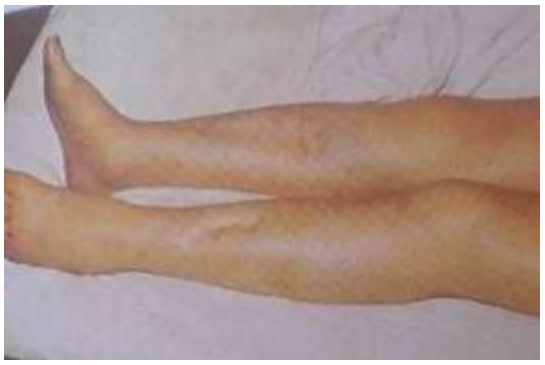
5. Nephrotic (with clinical and laboratory signs of nephrotic syndrome).

Triadic classical (unfolded***)*** *form of* acute glomerulonephritis is accompanied by edema, hypertensive and urinary syndromes. The onset of the disease is acute, sometimes with phenomena of heart failure or seizure syndrome ("cerebral debut"). Usually there is weakness, thirst, oliguria, urine the color of "meat slop", lower back pain, sometimes intense, headache, nausea, vomiting, eclampsia. Blood pressure within the range of 140-160/90-110, sometimes above 180/120 mm Hg, but decreases relatively quickly. High and stable arterial hypertension may indicate the possibility of extracapillary sub acute glomerulonephritis or the transition to a chronic form. Swelling rapidly appears on the face, eyelids, trunk, accompanied by pallor and dryness of the skin. Rarely there are ascites, hydrothorax, hydropericardium. There may be "hidden" edema, determined by weighing the patient. Urinary syndrome is manifested by a drop in diuresis, proteinuria and hematuria.



The monosymptomatic form is characterized by the presence of a single sign in the clinical picture (urinary syndrome, hematuria, edema or arterial hypertension), a calmer course and a relatively favorable prognosis. However, it should be emphasized that the prolonged existence of arterial hypertension has unfavorable

prognostic value, indicating the possible chronicity of the inflammatory process in the kidneys. ***Nephrotic form of*** acute glomerulonephritis is manifested by significant edema, up to anasarca, low severity of arterial hypertension and hematuria, predominance of clinical and laboratory signs of nephrotic syndrome (massive proteinuria, hypoproteinemia, dysproteinemia, hyperlipidemia). The course of the disease is usually long (6-12 months), often with the transition to chronic glomerulonephritis.

The following procedures implied as part of the diagnosis of glomerulonephritis:

1. Interview with the patient (if the patient is a child, information is obtained from the parents):

- how long ago the first symptoms of the disease appeared;
- the intensity with which the symptoms develop;
- whether you have taken any measures to manage your symptoms, such as taking medications to lower your blood pressure or relieve swelling;
- what the body's response has been to the therapy.
- if you've been diagnosed with kidney disease before;
- whether there is a history of glomerulonephritis in relatives;
- whether there has been a previous infectious disease and how it has been since the disease has fully recovered.

2. An examination of the patient is performed:

- the doctor notes the presence and intensity of swelling;
- blood pressure is measured and elevated blood pressure is recorded;
- visually identifies discolored urine.

1. **Laboratory studies**:

- urine - determines its protein and blood content;
- blood - increased leukocyte count and high erythrocyte sedimentation rate will indicate an inflammatory focus in the body;
- blood chemistry - cholesterol, low protein content;
- immune analysis of blood serum - is carried out in case of suspicion of autoimmune cause of glomerulonephritis.

2. **Instrumental examination**. An ultrasound examination is carried out, on which the specialist can note an increase in the size of the kidneys.

At the discretion of the doctor examining the patient and conducting diagnostic measures, a rheumatologist, ENT doctor, ophthalmologist, cardiologist, infectious disease specialist may be invited for consultation.

Differential diagnosis

Differential diagnosis of acute and exacerbation of chronic glomerulonephritis

Signs	Acute glomerulonephritis	Exacerbation of chronic glomerulonephritis
Chronologic relationship co streptococcal. infection	Develops 12 to 20 days after strep throat. infections	Develops 1-3 days after a streptococcal infection
Timing of the build-up of antistreptococcal antibody titers	Before the development of clinical manifestations	After clinical manifestations develop.
Urine density	Normal	Usually decreased Usually
Ratio and severity of hematuria and proteinuria	Hematuria is more pronounced Significant increase in α_2- and γ-globulins	predominant proteinuria, hematuria less pronounced
Changes in protein fractions	Little characteristicNot characteristic	Often hypoalbuminemia, especially in nephrotic syndrome
Anemia Left ventricular hypertrophy	Little characteristic Increased Not characteristic	CharacteristicHaracteristic
Severe angioretinopathy		Very characteristic
Kidney size on ultrasound		ReducedHaracteristic
Azotemia		

Differential diagnosis of acute glomerulonephritis and acute pyelonephritis

Signs	Acute glomerulonephritis	Acute pyelonephritis
Kidney pain Dysuria Chills. Edema Leukocyturia Bacteriuria Hematuria Nephrotic syndrome Characteristic combinations of urinary streaks renal ultrasound	Double-sided Don't are characterized by Not characteristic Characteristic Characteristic Typical Characteristic Frequent Hematuria+ proteinuria+ cylindruria (erythrocytic, hyaline cylinders) Bilateral renal enlargement	Predominantly unilateral Very characteristic Characteristic Characteristic Not characteristic Characteristic Less characteristic characteristicNot characteristic Leukocyturia+ bacteriuria Asymmetric lesion of the PSC

Diagnosis

The diagnosis of acute glomerulonephritis is based on the appearance of edema, headache, arterial hypertension, protein, erythrocytes and cylinders in the urine, increased blood titers of ASO, ACG in persons more often young after angina or acute respiratory disease. In doubtful cases, the diagnosis is verified by morphologic examination of kidney biopsy specimens.

Complications

The following complications may occur in acute diffuse glomerulonephritis:

-acute heart failure (left ventricular or total), develops in no more than 3% of patients and is caused by acute overload of the left ventricle due to arterial hypertension, hyperhydration and myocardial dystrophy; acute renal failure in 1% of patients;

-eclampsia;

hemorrhage;

-acute visual impairment (transient blindness due to retinal spasm and edema).

Forecast

The prognosis in acute glomerulonephritis is determined by a number of factors.

- age (more favorable prognosis in children and young , less favorable in the elderly);
- presence of extrarenal pathologies (in this case, the prognosis is worse);
- the presence of complications (they worsen the prognosis, especially anuria and eclampsia; in eclampsia mortality increases 2-fold);
- morphological variant (worst prognosis by immunofluorescence
- "garland" type of glomerulonephritis);
- concomitant urologic pathology (prognosis worsens).

Most often, recovery occurs within 1 month to 1 year (about 70% of patients). Transition to the chronic form is possible (about 28% of patients), the reasons for which are individual characteristics of the organism, delayed diagnosis and hospitalization, inadequate therapy, exposure to infections, hypothermia and physical overstrain. The transition to the chronic form is indicated by the persistence of any extra renal signs and/or proteinuria for a year. Recovery beyond this period is sometimes possible. In rare cases, less than 1% (mainly in the elderly and children), death occurs. The causes of death may be circulatory failure, renal eclampsia, cerebral hemorrhage, acute renal failure, malignant course of glomerulonephritis. It should be remembered that acute glomerulonephritis under the influence of unfavorable factors (cooling, physical overexertion, infections, etc.) can transform into a malignant form (rapidly progressive glomerulonephritis).

Examination program

1. CBC, urine, feces.
2. Daily measurement daily diuresis и amount drunk

fluids.

3. Examination of urine according to Zimnitsky and Nechiporenko (determination of the number of erythrocytes, leukocytes, cylinders in 1 ml of urine).
4. BAC: determination of urea, creatinine, total protein, protein fractions, cholesterol, β-lipoprotein, sialic acids, fibrin, seromucoid.
5. Reberg-Tareyev test: determination of glomerular filtration and tubular reabsorption by endogenous creatinine.
6. renal ultrasound

Treatment

1. Bed rest.

2. Diet Table 7: the content of proteins is somewhat limited, fats and carbohydrates - within physiological norms. Food is prepared without salt. Salt is given to the patient in the amount specified by the doctor (3-6 g). The amount of free fluid is reduced to an average of 1 liter. Exclude extractive substances of meat, fish, mushrooms, sources of oxalic acid and essential oils. Cooking without mechanical and with moderate chemical sparing. Meat and fish (100-150 g per day) boiled. The temperature of food is normal. **Chemical composition and caloric content of dietary table number 7**: carbohydrates - 400-450 g (80-90 g sugar); proteins - 80 g (50-60% animal), fats

- 90-100 g (25% vegetable), calories - 2700-2900 kcal; free fluid - 0, 9-1, 1 liter. **Mode of nutrition**: 4-5 times a day.

3. Drug therapy:

4. Pathogenetic therapy

5. Glucocorticosteroids
6. Cytostatics
7. Anticoagulants
8. Antiaggregants
9. Symptomatic therapy
10. Hypotensive drugs

11. Diuretics
12. Drugs that improve blood rheology
13. NSAIDS
14. Vitamin therapy

Immunosuppressive therapy is used to suppress activity: glucocorticoids, non-selective cytostatics and cyclosporine A.

Glucocorticoids (GCS), possessing immunosuppressive and anti-inflammatory action, в within for several remain the main means of pathogenetic therapy of nephritis for several decades. More often used orally - prednisolone in high (1-2 mg / kg per day) or moderately high (0.6-0.8 mg / kg per day) doses, daily in 2-3 doses or once a day in the morning, long-term (1-4 months), с followed by slowly decreasing. Alternating regimen is also possible, when the patient takes every other day. every other day once in the morning double daily dose. Athigh activity of renal inflammation for rapid achievement of very high concentrations of HA in plasma is indicated "pulse therapy" - intravenous administration of ultra-high doses (0.8-1.2 g) of methylprednisolone or prednisolone.

Cytostatics in the treatment of nephritis are used somewhat less frequently than GCs because of the greater severity of side effects. Alkylating compounds such as cyclophosphamide (at a dose of 1.5-2 mg/kg per day) and chlorbutine (at a dose of 0.1-0.2 mg/kg per day) are usually used; the antimetabolite azathioprine is less effective, although less toxic. The decline in the white blood cell count occurs over several days or weeks. During this period, it is important to check the peripheral blood leukocyte count every 2-3 days so that if it drops to the lowest tolerable level, the dose can be reduced or withdrawn.

The most serious side effects of alkylating cytostatics are bone marrow suppression, development of infections and gonadal failure. Other complications include hepatitis, alopecia, hemorrhagic cystitis, gastrointestinal disorders and elevated

risk of developing tumors.

A very interesting selective immunosuppressant cyclosporine A (CsA), used for a long time in transplantology and in recent years in therapeutic nephrology. The initial dose of CsA for adults in the nephrology clinic is 3-5 mg/kg, for children - 6 mg/kg per day. Thereafter, the dose depends on tolerability, side effects and serum concentrations, which should be checked regularly.

CsA is indicated first of all for patients with minimal changes and FSGS with frequent recurrence of NS or steroid-resistant NS, in case of complications of steroid and cytostatic therapy. The most serious complications of CsA treatment are arterial hypertension and nephrotoxicity.

The following guidelines should guide the administration of immunosuppressive therapy.

- Immunosuppressive therapy is always indicated in highly active HN.
- First-time NS, especially without hematuria and hypertension, is always an indication for GC treatment.
- In rapidly progressive forms of nephritis (with a rapid increase in creatinine level), it is obligatory to prescribe immunosuppressants - large doses of GC and cytostatics inside and/or in the form of "pulses".

Not only immunosuppressive therapy, but also the so-called non-immune nephroprotective therapy can stabilize the course of GN, slow down its progression, and sometimes even lead to its reverse development. At the present stage we can talk about four methods of nephroprotective therapy, the effect of which on the progression of GN has been proven or is being studied. These are:

- inhibitors angiotensin-converting enzyme и angiotensin II receptor blockers;
- heparin sodium;
- dipyridamole;
- and polypidemic drugs, primarily statins.

Practical part

The practical part of the lesson is devoted to the treatment of patients in a specialized nephrology department. First, the teacher demonstrates patients with different stages of CKD and conducts a clinical review. Then allocates them to independent supervision, at the end of which, students report patients, demonstrating the practical skills. The teacher evaluates the step-by-step performance of skills according to the criteria developed in department

Step-by-step mastery, execution and evaluation of practical skills

Professional questioning and examination of the patient with

AGN Objective:

Establishing a trusting relationship with the patient;

assessment of the patient's personality and attitude to the disease (internal picture of the disease);

obtaining the information needed for diagnosis;

Identification of other sources of information (relatives, other doctors, etc.);

assess the state of consciousness and mental status of the patient, his/her position, general appearance, condition of external coverings and individual body parts.

Interviews and examinations are mandatory for all patients in well-lit rooms, or in the doctors' office, in a trusting environment.

Patient management

Numbers	Activity Algorithm		Expected attributes
	Sequence of actions	**Types of action**	
1.	Gathering passports data	Interview with the patient o	Age, nationality, profession, place of residence
2.	Collection of characteristic complaints for the disease я	Interview with the patient o	Edema, decreased diuresis, headaches, dizziness, general weakness
3.	Gathering medical history	Interview with the patient o	The disease develops acutely, latently, gradually.
4.	Gathering anamnesis lives	Interview with the patient o	Hereditary diseases, hereditary predisposition
5.	Evaluate general conditions	Examination of the patient	Relatively satisfactory, medium severity. Pale skin, swelling of the face (face "nephritic"), trunk
6.	Objective examination	Visual and physical surveys	Palpation and percussion: skin pale and dry, swelling under the eyes, BP above 130/80 mmHg.
7.	Compose pla examinations	Clinical reasoning	OAC - anemia, erythrocytopenia, acceleration of COE. OAM: proteinuria, hematuria, cylindruria. Zimnitsky's test: hypoisosthenuria, nicturia. Blood chemistry: hypoproteinemia, hypocholesterolemia, increased urea and creatinine
8.	Diagnosis	Clinical reasoning	Establishment clinical diagnosis based on classification .
9.	Spend differential - diagnostic	Clinical reasoning	Acute, chronic glomerulonephritis. Acute and chronic pyelonephritis. Kidney amyloidosis Tuberculosis KidneyChronic kidney disease Arterial arterial disease hypertension.

10.	Composition pla treatments	Clinical reasoning	Diet #7. Corticosteroids.Cytostatics Antiaggregants Anticoagulants. Diuretics Hypotensive drugs

The steps to be performed and their evaluation:

№	Event	Don't fulfilled	Completely correctly executed
1	Questioning passport data	0	5
2	Gathering of complaints	0	10
3	Collection of medical history	0	15
4	Gathering a life history	0	10
5	Epidemiologic, allergic history	0	5
6	Objective examination of the patient	0	10
7	Drawing up a survey plan	0	5
8	Correct diagnosis	0	10
9	Differential diagnosis	0	10
10	Making a treatment plan	0	10
	Total	0	100

INTERPRETATION OF ELECTROCARDIOGRAM IN HD AND ITS EVALUATION

Purpose: To master the skill of ECG interpretation. Steps to be performed and their evaluation:

№	Event	Don't executed(0 points)	Completely correctly executed
1	Determination of heart rhythm	0	20
2	Determination of the electrical axis of the heart	0	20
3	Calculation of the amplitude of teeth and duration of intervals	0	20
4	Determining the location of the STi segment cogs T	0	20
5	Formalization of ECG report	0	20
6	Total		100

RECOMMENDATIONS.

№	Event	Don't executed(0 points)	Completely correctly executed
1	Mode	0	10
2	Diet	0	10
3	Correct Choosing right dietary table according to the diagnosis	0	20
4	According to the diagnosis, the severity of the disease. и stage prescription basic therapy	0	20
5	According to the diagnosis, severity and stage of the disease, prescribe symptomatic therapy	0	20
6	Preventive measures	0	20

Interactive teaching methods

USE BUSINESS GAME "WHO IS BIGGER? WHO Faster?" (Script)

1. The job requires:
2. Cards with questions on the topic (the number of cards is equal to the number of students in the group; each contains 5 questions)
3. StopwatchWork progress:
4. The game is played orally
5. Students take turns pulling out question cards
6. Within 3 minutes, each student orally answers a series of questions (5) written on a card.
7. The teacher counts number correct answers 5.In All students participate in the game
8. Total playing time 45 minutes
9. The questions that have not been given correct are discussed
10. Students' answers are evaluated according to the following form: Each of the 5 questions is evaluated for 20 points.
11. Correct answers to 5 questions -100 points
12. Correct answers to 4 questions - 80 points
13. Correct answers to 3 answers - 60 points
14. Correct answers to 2 questions - 40 points
15. Correct answers to 1 question - 20 points.
16. Received student score is taken into account in grading
17. In the instructor's learning log make a note about the game.

A SET OF QUESTIONS FOR CONDUCTING A BUSINESS GAME:

For internalize topic can be quizzes

questions, solve situational problems and tests

1.The concept of acute glomerulonephritis

2.Etiology of acute glomerulonephritis

3.Pathogenesis of acute glomerulonephritis.

4. Clinical classification of acute glomerulonephritis 5.Morphologic classification of acute glomerulonephritis 5.Morphologic classification of acute glomerulonephritis

6. Major clinical syndromes

7.Clinic of acute glomerulonephritis

8. Laboratory methods of examination

9. Results of laboratory examination methods

10.Instrumental examination methods

11.Differential diagnosis of acute glomerulonephritis 12.Complications of acute glomerulonephritis

13.Diet in acute glomerulonephritis

14.Treatment of acute glomerulonephritis

15.Prevention of acute glomerulonephritis

Analytical part

Situational tasks

Task #1. Young man, 20 years old after hypothermia began to note increasing swelling in the face, arms, legs, general weakness, decreased diuresis. He was treated as an inpatient at his place of residence without much effect. 2 months later he was hospitalized in the nephrology department. On examination the skin was pale, dry. Swelling on the face, arms, legs, ascites. Percussion dulling of pulmonary sound below the angle of the shoulder blades, there at auscultation breathing is sharply weakened.Heart tones are muffled.BP 90/60 mm. Hg. Daily proteinuria 6 grams.

1. Formulate a clinical diagnosis?
2. What diseases should be differentiated from?
3. Treatment Plan.

Task #2 A 16-year-old patient was admitted with complaints of headaches, swelling on the face, trunk, shortness of breath, decreased amount of urine. 20 days ago after hypothermia there was high t-body up to 38-39º for 5 days, cough, runny nose, sore throat. After treatment with antibiotics the condition improved. 5 days ago suddenly noticed redness of urine, swelling under eyes. In 2 days the swelling spread to the abdomen, lower back, the amount of urine decreased to 300 ml per day, headaches, shortness of breath appeared. Objectively: face puffy, pale. Swelling of the anterior abdominal wall, lumbar. In the lungs in the lower parts of the dulling. Auscultatory: sharp weakening. The borders of the heart are dilated to the left by 1-2 cm. Heart tones muffled, bradycardia, accent of the II tone on the aorta. Pulse 56 per min. Rhythmic. BP 140/110 In the bowman's capsule fluid with a level 2 cm below the umbilicus. Urine analysis: color of meat slop, specific gravity 1030 protein 9,9‰. Er 40-50/1 changed. Leuk 5-8/1 cylinders hyaline 2-3/1, granular 3-4/1. Urine analysis according to Zimnitsky uz weight 1026- 1034. Daily diuresis 500 ml.

I. Make a preliminary diagnosis:

II. What diseases should be differentially diagnosed?

Tests

What is the cause of low back pain in acute glomerulonephritis:

A) bacterial inflammation

B) proteinuria

D) hyperstenuria

E) Swelling of the kidney as a result of immune inflammation *

F) hematuria

Changes in urine not specific for acute glomerulonephritis:

A) leukocyturia

B) hematuria

D) proteinuria

E) cylindruria

F) bacteriuria.

Maximum increase in antistreptolysin O titer in acute glomerulonephritis:

A) in the first 3 weeks of illness *

B) in the first 6 months of illness

D) after 1 year

E) in 2 years

F) in 3 years

Physician's tactics in case of suspected acute glomerulonephritis:

A) treat at home

B) Get an EKG.

D) consultation with a neurologist

E) immediate hospitalization *

F) prescription of sanatorium treatment

When is daily proteinuria a pathology?

A) more than 10 mg / day.

B) more than 30 mg / day.

E) more than 50 mg/day *

F) more than 100 mg/day.

F) more than 150 mg / day

Renal anuria can be caused by:

A) Acute glomerulonephritis*

B) Acute urethritis

D) Chronic epididymitis

E) Cervical cancer

F) Urethral cancer

Macrohematuria is characteristic of all kidney diseases except.

A) acute glomerulonephritis.

B) hypernephroma.

D) renal amyloidosis*

E) Infarction of the kidney

F) kidney stone disease.

How does acute glomerulonephritis manifest itself?

A) proteinuria

B) edema

D) hypertension

E) any of the following *

F)hematuria

What disease is characterized by the given urine analysis: protein 0.165‰, leuk.8-10; altered erythrocytes 20-30 in the field of view. Hyaline cylinders 2- 3 in the field of view.

1. Chronic pyelonephritis
2. Kidney amyloidosis
3. Acute glomerulonephritis*
4. Lupus nephritis
5. Urolithiasis

Pollakiuria is:

1. Frequent urination*
2. Infrequent urination
3. Increased urine output
4. Painful urination
5. Urinary incontinence

OSCE Control Questions:

1. Anatomo-physiological features of the urinary system
2. Clinical classification of AGN
3. Main clinical syndromes of AGN
4. Etiopathogenesis of acute glomerulonephritis
5. Clinic of acute glomerulonephritis
6. Laboratory methods of examination
7. Results of laboratory examination methods

8. Instrumental diagnostic methods
9. Differential diagnosis of acute glomerulonephritis
10. Complications of AGN
11. Diet for AGN
12. Treatment of SUDs
13. Prevention of SUDs

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