T. T. KOPATS, I. M. ZMACHINSKAJA

KIDNEY DISEASES:

GLOMERULONEPHRITIS,

PYELONEPHRITIS,

CHRONIC KIDNEY DISEASE

Minsk BSMU 2024

МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ РЕСПУБЛИКИ БЕЛАРУСЬ
БЕЛОРУССКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ
КАФЕДРА ПРОПЕДЕВТИКИ ВНУТРЕННИХ БОЛЕЗНЕЙ

Т. Т. Копать, И. М. Змачинская

ЗАБОЛЕВАНИЯ ПОЧЕК: ГЛОМЕРУЛОНЕФРИТЫ, ПИЕЛОНЕФРИТЫ, ХРОНИЧЕСКАЯ БОЛЕЗНЬ ПОЧЕК

KIDNEY DISEASES: GLOMERULONEPHRITIS, PYELONEPHRITIS, CHRONIC KIDNEY DISEASE

Учебно-методическое пособие

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Копать Тереса Тадеушевна
Змачинская Ирина Михайловна

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MOTIVATIONAL CHARACTERISTICS OF THE TOPIC

Total class time: 3 hours.

The purpose of the lesson: to familiarize students with the methodology for assessing the clinical picture, as well as the procedure for using laboratory and instrumental diagnostic methods for glomerulonephritis, pyelonephritis, chronic kidney disease (CKD).

Lesson objectives:

1. To study the clinical manifestations of acute and chronic glomerulonephritis, pyelonephritis.
2. To master the methodology of using these laboratory and instrumental diagnostic methods for glomerulonephritis, pyelonephritis, CKD.
3. Get acquainted with the principles of therapeutic tactics for glomerulonephritis, pyelonephritis, CKD.

Requirements for the initial level of knowledge. To fully assimilate the topic, it is necessary to repeat:

– from normal anatomy: anatomical structure of the kidneys and urinary tract;

– from normal physiology: kidney function.

Control questions from related disciplines:

1. Describe the structure of the nephron.
2. List the main functions of the kidneys.
3. Describe kidney function.

Control questions on the topic of classes:

1. Clinical picture of acute and chronic glomerulonephritis and pyelonephritis.
2. Laboratory diagnostic methods for kidney diseases.
3. Instrumental diagnostic methods for kidney diseases.
4. The concept of CKD.
5. The basic principles of therapeutic tactics for glomerulonephritis, pyelonephritis, CKD.

Tasks for independent work. To master the topic, it is necessary to familiarize yourself with the content of this educational and methodological manual. If you have any questions, it is recommended to write them down for discussion with the teacher at a practical lesson. Next, we should proceed to the final stage — assessment of the degree of assimilation of the topic by answering the questions of the test control.

INTRODUCTION

The urgency of timely diagnosis and treatment of kidney diseases is due to the severity of their complications with the need for high-tech treatment (dialysis and transplantation). The inevitable end of the course of kidney diseases is an irreversible violation of homeostasis due to the increasing death of nephrons.

As a rule, due to the death of part of the nephrons caused by the underlying disease, compensatory functional and structural changes develop in unaffected nephrons: intraclubular hypertension, hyperfiltration, nephron hypertrophy as a result of activation of the intrarenal renin — angiotensin system.

In the early stages of kidney disease, there is a decrease in the functional reserve of the organ itself, in particular, a decrease in the ability to increase the glomerular filtration rate in response to protein loading. At this stage, the course of renal dysfunction is asymptomatic.

The subsequent loss of functioning nephrons (more than 30 % of the norm) leads to more pronounced disorders — an increase in the concentration of nitrogenous metabolites (urea and creatinine), electrolyte imbalance, anemia and other disorders.

The pathomorphological basis is glomerular sclerosis, tubulointerstitial fibrosis, tubular atrophy. From a certain point on, the mechanisms of progression become the same as in primary glomerular, tubulointerstitial or congenital lesions.

ACUTE DIFFUSE GLOMERULONEPHRITIS

Acute diffuse glomerulonephritis — acute immune-inflammatory kidney disease with an initial predominant lesion of the glomeruli and involvement of all renal structures in the pathological process, clinically manifested by renal and (or) extrarenal symptoms.

ETIOLOGY

The main etiological factor is a streptococcal infection (sore throat, pharyngitis, exacerbation of chronic tonsillitis, scarlet fever, erysipelas of the skin). Most often, acute glomerulonephritis is caused by 12 and 49 strains of beta-hemolytic streptococcus group A (post-streptococcal glomerulonephritis). Other possible etiological factors may be hepatitis B virus (causes mainly membranous nephritis), rubella viruses, infectious mononucleosis, herpes, adenoviruses, in some cases, acute glomerulonephritis may develop after a staphylococcal or pneumococcal infection (non-streptococcal glomerulonephritis). Parasitic infestations are pathogens of toxoplasmosis, malaria, schistosomiasis, trichinosis.

Acute glomerulonephritis caused by various types of infection is called infectious — immune.

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

Cooling is an important trigger factor in the development of acute glomerulonephritis, often having independent significance

Genetic predisposition to acute diffuse glomerulonephritis also plays an important role.

PATHOGENESIS

Acute diffuse glomerulonephritis is an immuno-inflammatory disease. There are the following pathogenetic variants of acute glomerulonephritis:

– immunocomplex;

– low-immune;

– due to the appearance of antibodies to the glomerular basement membrane;

– due to antigenic mimicry.

An important link in the pathogenesis of acute glomerulonephritis is the formation and fixation of immune complexes in the kidneys.

Streptococcal antigens — endostreptosin with streptokinase activity, nephritogenic plasma-binding protein — are deposited in glomeruli during the acute phase of streptococcal infection. After 10–14 days, the host's immune response occurs, during which anti-streptococcal antibodies bind to antigens.

The formation of immune deposits in the renal tissue leads to the activation of the complement system, changes in the physico-chemical properties of the basement membrane, mesangium, endothelium, glomerular epithelium. Besides, platelets are activated, producing vasoconstrictive factors involved in the formation of microthrombs, as well as stimulating the proliferation of glomerular cells under the influence of platelet growth factor. Changes in the endothelial surface and collagen matrix activate blood clotting (normally, the physico-chemical properties of the glomerular basement membrane prevent platelet aggregation and local activation of clotting factors).

In the development of acute glomerulonephritis, in addition to immune factors, non-immune factors are also important.

Pathomorphologically, the disease is characterized by a morphological picture of diffuse proliferative glomerulonephritis: infiltration of glomeruli by neutrophils and mononuclear cells, proliferation of endothelial and mesangial cells (intracapillary proliferation). Granular deposits of IgG and complement component C3 are found in the glomeruli. The severity of glomerular damage depends on the nature of the damaging factor and its severity, the localization of pathological changes at the glomerular level (mesangium, basement membrane, endothelium, renal epithelium), the rate of occurrence and the area of the lesion.

Diffuse proliferative glomerulonephritis is not the only morphological manifestation of acute glomerulonephritis. Sometimes there are membranous, mesangioproliferative, membrane-proliferative variants.

CLASSIFICATION

There are several sections in the classification of acute glomerulonephritis:

1. *Etiopathogenesis:*

– infectious — immune;

– non-infectious — immune.

2. *Morphological forms (types):*

– proliferative endocapillary;

– proliferative extracapillary;

– mesangio-proliferative;

– mesangio-capillary (membranous-proliferative);

– sclerosing (fibroplastic).

3. *Clinical forms:*

– classical triadic expanded form (urinary syndrome, edema, arterial hypertension);

– bisyndromic form (urinary syndrome in combination with edema or arterial hypertension);

– monosyndromic form (isolated urinary syndrome);

– nephrotic form.

4. *Complications:*

– acute renal failure;

– acute renal hypertensive encephalopathy (preeclampsia, eclampsia);

– acute heart failure (left ventricular with attacks of cardiac asthma (pulmonary edema) or total).

CLINIC OF ACUTE GLOMERULONEPHRITIS

The triadic classical (expanded) form of acute glomerulonephritis is accompanied by edematous, hypertensive and urinary syndromes.

The onset of the disease is acute. Usually there is weakness, thirst, oliguria, urine the color of “meat slops,” lower back pain, sometimes intense, headache, nausea, vomiting.

Blood pressure within 140–160/90–110, sometimes above 180/120 mm Hg. High and stable arterial hypertension may indicate the possibility of transition to a chronic form.

Swelling quickly appears on the face, eyelids, trunk, which are accompanied by pallor and dry skin.

Urinary syndrome is manifested by a drop in diuresis, proteinuria and hematuria.

The nephrotic form of acute glomerulonephritis is manifested by the predominance of clinical and laboratory signs of nephrotic syndrome:

– massive proteinuria (more than 3 g of protein per day);

– hypoproteinemia (total serum protein — 40–20 g/l (at a rate of 60–85 g/l), due to hypoalbuminemia);

– hyperlipidemia (cholesterol, triglycerides increased by 2 or more times);
– persistent edema, refractory to diuretics.

This form of acute glomerulonephritis is characterized by low severity of arterial hypertension and hematuria.

In clinical practice, the monosymptomatic form, *called isolated urinary syndrome, is relevant.*

The peculiarity of this form of acute glomerulonephritis is the absence of *extrarenal manifestations*: no edema, no hypertension, no changes in the fundus.

The severity of the urinary syndrome is determined by the nature of changes in the urinary sediment with an almost normal specific gravity of urine:

• ***moderate urinary syndrome:***
– proteinuria up to 1g of protein per day;
– hematuria up to 30–50 red blood cells in the field of vision;
– cylindruria (hyaline cylinders);
• ***severe urinary syndrome:***
– proteinuria from 1g to 3 g of protein per day;
– hematuria of 50–100 red blood cells in the field of vision;
– cylindrical (granular, hyaline cylinders);
– maybe leukocyturia;
• ***significantly pronounced urinary syndrome:***
– proteinuria about 3g of protein per day;
– hematuria of more than 100 red blood cells in the field of vision;
– cylindrical (waxy, granular, etc. cylinders);
– leukocyturia (mainly lymphocyturia).

DIAGNOSTICS

Diagnosis of acute glomerulonephritis is based on the data of anamnesis, examination of the patient and a number of laboratory and instrumental studies.

The main research methods include:

1. *General blood test*. Characterized by a slight decrease in hemoglobin concentration, leukocytosis, ESR is moderately elevated.

2. *Biochemical blood analysis* (determination of the content of urea, creatinine, total protein, protein fractions, cholesterol and the entire lipid spectrum, sialic acids, fibrin, seromucoid). Biochemical manifestations of nephrotic syndrome are hypoproteinemia with hypoalbuminemia, dysproteinemia with a predominance of α2- and less often γ-fractions of globulins, hyperlipidemia.

3. *Urine tests*:

– general urine analysis: proteinuria, hematuria, leukocyturia (lymphocyturia). The relative density of urine is slightly reduced;

– urine analysis according to Nechiporenko: micro- and macrohematuria, leukocyturia (lymphocyturia), erythrocyte cylinders;

– Zimnitsky urine analysis: the state of the concentration capacity of the kidneys (daily diuresis, the ratio of day and night diuresis, daily fluctuations in the relative density of urine);

– determination of daily proteinuria — a quantitative method that takes into account daily diuresis and allows you to more accurately assess the dynamics of proteinuria, including under the influence of treatment;

– daily measurement of daily diuresis and the amount of liquid consumed.

4. *Determination of the titer of anti-streptococcal antibodies* (detection of anti-streptolysin O (ASL-O), anti-streptococcal hyaluronidase).

5. *Rehberg–Tareev test* (determination of the presence and degree of decrease in the rate of glomerular filtration and tubular reabsorption by endogenous creatinine).

Additional research methods are:

1. *A smear from the throat to detect streptococci*.

2. *Examination of the fundus*. With an increase in blood pressure, the following changes occur: narrowing of arterioles, sometimes the phenomenon of pathological arteriovenous intersection, swelling of the nipple of the optic nerve, spot hemorrhages are possible.

3. *Ultrasound of the kidneys*. The size of the kidneys has not been changed or slightly increased (normally length 75–120 mm, width 45–65 mm, thickness 35–50 mm). The swelling of the kidney tissue is detected. The cup-pelvis system has not been changed.

4. *Blood pressure monitoring.* It is advisable to detect arterial hypertension, especially not noticed by the patient, as well as to verify its severity (according to the study, antihypertensive drugs are prescribed and the adequacy of therapy is monitored).

THERAPEUTIC TACTICS

Therapeutic tactics for acute glomerulonephritis are as follows:

1. *Bed rest* (until the elimination of edema and normalization of blood pressure).

2. *Therapeutic nutrition*:

– restriction of table salt, simple carbohydrates, protein;

– exclusion of extractive substances from the diet and full provision of the body with vitamins and trace elements.

3. *Etiological treatment* (anti-streptococcal or other, depending on the identified pathogen).

4. *Pathogenetic treatment*:

– glucocorticoids;

– immunosuppressants;

– nonsteroidal anti-inflammatory drugs;
– heparin and antiplatelet agents;
– aminoquinoline compounds.

5. *Symptomatic treatment* (treatment of arterial hypertension, edematous syndrome, etc.).

6. *Treatment of complications* (acute left ventricular failure, angiospastic encephalopathy, renal failure).

7. Spa treatment.

CHRONIC GLOMERULONEPHRITIS

Chronic glomerulonephritis (chronic nephritic syndrome) is a heterogeneous group of diseases by origin and pathomorphology, characterized by an immuno-inflammatory lesion of the glomeruli, tubules and interstitium of both kidneys and a progressive course, in as a result, nephrosclerosis and chronic renal failure develop.

ETIOLOGY

Chronic glomerulonephritis may be *a consequence of acute glomerulonephritis*. Along with this, *primary chronic glomerulonephritis* develops without a previous acute period.

The main etiological factors of chronic glomerulonephritis are similar to those of acute glomerulonephritis. Very often, the cause of the disease cannot be found out.

The role *of genetic predisposition* to the development of chronic glomerulonephritis is also widely discussed.

PATHOGENESIS

The mechanism of development of chronic glomerulonephritis is generally similar to the pathogenesis of acute glomerulonephritis, i.e. it is also based on the *immune inflammatory process*, in the development of which the deposition of antibodies and fragments of complement is carried out, the formation of a complementmembrane damaging complex, blood coagulation factors, leukotrienes, cytokines, neutrophils, platelets, macrophages, T-lymphocytes are involved.

The main pathogenetic variants of chronic glomerulonephritis are similar to those of acute glomerulonephritis.

However, there are certain features of the pathogenesis of some morphological variants. A number of authors also emphasize the great role of *the genetic inferiority of the T-cell link of immunity* in the development of chronic glomerulonephritis.

Studies of a number of nephrological centers have allowed us to formulate a hypothesis of the origin of chronic glomerulonephritis. According to her, due to genetic predisposition, there is insufficient intake of early lymphoid (trophic) elements into the kidney. This disrupts the normal physiological repair of individual parts of the nephron and contributes to the formation of inflammatory infiltrate in the kidneys with the participation of T-lymphocytes, mononuclears, the release of a large number of cytokines that enhance the proliferation of glomerular cells, cause damage to all structures of the nephron, primarily the basement membrane, followed by the formation of immune complexes.

It is generally recognized that the development of chronic glomerulonephritis is based on immunopathological processes.

Both ongoing immune inflammatory reactions and non-immune mechanisms of progression are also involved in the progression of the disease:

– the development of progressive renal fibrosis;
– hemodynamic factors;
– metabolic mechanisms;
– coagulation mechanisms;
– tubulointerstitial sclerosis.

The immune-inflammatory process in the kidneys is accompanied by reparative changes, the outcomes of which are different: complete restoration of the glomerular structure (usually under the influence of treatment or less often spontaneous) or with an unfavorable course — the development of progressive fibrosis, which is the basis of chronic renal failure.

Progressive renal fibrosis is caused by hyperfunctionation of glomerular cells and blood cells infiltrating the glomeruli of the kidneys, which is accompanied by excessive accumulation of the connective matrix and at the same time its insufficient utilization.

Mesangial cells play a leading role in the progression of glomerulosclerosis. They have contractile, phagocytic and metabolic activity. As is known, the mesangium is the connective tissue backbone of the glomeruli.

Angiotensin II plays an important role in the development of progressive renal fibrosis. It not only causes intraclubular hypertension, but also stimulates the proliferation of mesangial cells of the renal glomeruli, induces synthesis by smooth muscle and tubular cells of transforming (platelet) growth factor — the main fibroblast growth factor.

Hemodynamic disorders (systemic and arterial hypertension) are the most important factors in the progression of chronic glomerulonephritis.

Chronic progressive glomerulonephritis is characterized by the loss of functioning renal mass, which leads to compensatory hypertrophy and hyperfunction of the preserved renal glomeruli. An increase in their function is always accompanied by a violation of intrarenal hemodynamics —

intraclubular hypertension and hyperfiltration, which provides increased perfusion of the surviving nephrons. Activation of the renin-angiotensin II system also plays an important role, which leads to spasm of efferent arterioles and increased pressure in the glomeruli. Increased pressure in the glomeruli promotes the proliferation of mesangial cells and hyperproduction of the mesangial matrix.

In violation of renal hemodynamics and the progression of glomerulonephritis, an important role is played by a disturbed relationship between vasoconstricting endothelial hormone — endothelin-1 — and vasodilating endothelial factor — nitric oxide. These substances are produced by the endothelium of the renal vessels. In glomerulonephritis, the synthesis of endothelin-1 is activated, which is accompanied by constriction of the renal vessels, a decrease in renal blood flow, ischemic kidney damage and, as described above, stimulation of fibrogenesis. The production of vasodilating factor (nitric oxide) in chronic glomerulonephritis decreases.

Lipid shifts are of the most important importance among metabolic disorders in the progression of chronic glomerulonephritis. They are most often observed in people with nephrotic syndrome, but they also develop in glomerulonephritis without this syndrome.

Changes in lipid metabolism most often consist in an increase in the content of cholesterol, triglycerides, low-density lipoproteins, non-esterified fatty acids in the blood, an increase in the coefficient of atherogenicity. Dyslipidemia leads to the deposition of lipids in the kidneys. Lipid metabolism disorders are accompanied by nephrotoxic effects, and when lipids are deposited in renal structures, an increase in the mesangial matrix is observed simultaneously, which indicates a fibrosogenic effect of lipid metabolism disorders.

Disorders of lipid metabolism in chronic nephritis are accompanied *by activation of lipid peroxidation with the formation of free radicals* and peroxide compounds that have a damaging effect on the kidneys and contribute to the development of fibrosis.

The damaging effect of excessive calcium deposition in the kidneys has also been established, which is especially pronounced in chronic renal failure. The accumulation of calcium in the renal tissue contributes to the development of intraclubular arterial hypertension, mesangium proliferation, and fibrosis progression.

Currently, it has been established that the most important mechanism for the progression of chronic glomerulonephritis is local *intravascular coagulation* of blood with the formation of microthrombs in the glomerular capillaries and the deposition of fibrin in them. Subendothelial fibrin deposits in glomerular capillaries are the most important criterion for the unfavorable prognosis and progression of chronic glomerulonephritis. This is explained by the fact that fibrin deposits formed as a result of local hypercoagulation stimulate

the proliferation of endotheliocytes and mesangiocytes, the formation of connective tissue in the kidneys, reduce microcirculation in the glomeruli, promote the development of ischemia in them. The leading role in the development of intravascular hemocoagulation in the kidneys is played by damage to the endothelium by immune complexes, cytokines, inflammatory mediators, various endotoxins, activated complement.

At the same time, platelet activation, increased adhesive-aggregation function and increased production of transforming growth factor are observed. As a result of these processes, platelet microaggregates are formed, the coagulation link of hemostasis is activated, fibrin deposits are formed, and the synthesis of connective tissue is stimulated.

Tubulointerstitial sclerosis is currently recognized as an important factor in the progression of chronic glomerulonephritis. The main role in the development of tubulointerstitial damage and sclerosis is played by epithelial cells of the renal tubules. They are activated and produce substances that contribute to damage to the renal interstitium and the development of fibrosis. Activation of epithelial cells of the renal tubules is due to the production of cytokines by cells involved in inflammation, as well as protein reabsorption in the renal tubules. Persistent proteinuria has a toxic, damaging effect on the interstitium of the kidney.

Thus, these mechanisms of progression contribute to the development of a long-term inflammatory process that flows in waves (with periods of exacerbations and remissions), which eventually leads to sclerosis, hyalinosis, desolation of the glomeruli, the development of chronic renal failure.

CLASSIFICATION

A group of pathomorphologists — specialists in kidney diseases — under the auspices of WHO has developed a classification of chronic glomerulonephritis based on the results of examination of kidney biopsies using light, electron and immunofluorescence microscopy.

The morphological classification of chronic glomerulonephritis (Thomson, Charleworth, 1994) is as follows:

I. Minimal changes in the glomeruli or their absence according to light microscopy data:

1. Glomerulonephritis with minimal changes.

2. Kidney disease with thinning of the basement membrane.

II. Diffuse glomerular lesion:

1. Membranous glomerulonephritis.

2. Membranous-proliferative (mesangiocapillary) glomerulonephritis:

a) type I — subendothelial deposits;

b) type II — dense intramembrane deposits (disease of dense deposits).

3. Diffuse mesangioproliferative glomerulonephritis:

a) with mesangial deposits of JqA;

b) without mesangial deposits of JqA.

III. Focal lesions of the glomeruli:

1. Focal and segmental glomerulosclerosis and hyalinosis.

2. Focal and segmental proliferative glomerulonephritis:

a) with mesangial JqA;

b) without mesangial JqA.

CLINIC

It is very important for a practical doctor to know the clinical and laboratory symptoms of chronic glomerulonephritis. At the same time, it is necessary to take into account its following clinical forms:

– latent;

– nephrotic;

– hypertensive;

– hematuric;

– mixed.

The latent form (with isolated urinary syndrome) is the most common form of chronic glomerulonephritis. It is characterized by satisfactory well-being, absence of extrarenal symptoms (edema, arterial hypertension, fundus changes). Changes in urine are small: proteinuria (no more than 1–2 g per day), microhematuria, small cylindrical, sufficient relative density of urine.

This form has a long asymptomatic course (10–20 years), is often detected accidentally (during medical examinations to obtain various certificates, sometimes during medical examination or hospitalization for any other diseases — pneumonia, stomach ulcers, etc.), often already at the stage of chronic renal failure.

The nephrotic form (one of the most severe forms of chronic glomerulonephritis) accounts for 10–20 % of all cases.

It is characterized by all manifestations *of nephrotic syndrome*:

– weakness, lack of appetite;

– significantly pronounced persistent edema (hydrothorax, ascites, hydropericardium, anasarca are possible), resistant to diuretics;

– massive proteinuria (over 3–5 g per day);

– hypoproteinemia, dysproteinemia (decrease in the amount of albumins, increase in a2- and γ-globulins);

– hyperlipidemia.

In the nephrotic form, blood pressure is usually normal, arterial hypertension is less common. Laboratory examination also reveals cylindruria, microhematuria (of little character), anemia, and an increase in ESR.

The nephrotic form can be complicated by a nephrotic crisis, with intense abdominal pain, peritonitis-like syndrome, increased body temperature, hypovolemic collapse, intravascular hemocoagulation (development of DIC syndrome), vein thrombosis, including renal, erysipelas-like skin changes in the abdomen, chest, anterior thighs, decreased diuresis, leukocytosis.

The hypertensive form is observed in 20 % of patients. Her clinical picture is dominated by symptoms caused by hypertension: intense headaches, dizziness, pain in the heart, shortness of breath, palpitations, decreased vision, fog in front of the eyes. A feature of arterial hypertension is a significant increase in diastolic pressure (blood pressure rises to 160–180 / 110–120 mm Hg and above), while it decreases poorly at night.

Pathognomonic for this form of the disease is a fairly early developing and significantly pronounced visual impairment. Examination of the fundus reveals narrowing and tortuosity of the arteries, the phenomenon of crossing, silver or copper wire, single or multiple hemorrhages, swelling of the nipple of the optic nerve.

Characteristic is an early decrease in glomerular filtration, small proteinuria, microhematuria, a decrease in urine density.

This form can be complicated by left ventricular failure (cardiac asthma, pulmonary edema, gallop rhythm).

The hematuric form is observed in 6–8 % of patients. The clinical picture is dominated by macrohematuria or significant and persistent microhematuria. Proteinuria is low, blood pressure is normal, there are no edema or they are insignificant. Hematuria is especially characteristic of glomerulonephritis with deposition of IdA in the glomeruli (Bourget's disease), which is more common in young men.

The mixed form combines the signs of nephrotic and hypertensive forms. It occurs in less than 10 % of cases and is characterized by a steadily progressive course.

During any clinical form of chronic glomerulonephritis, there is a remission phase and an exacerbation phase.

The remission phase is characterized by either a stable satisfactory condition and the absence of clinical and laboratory symptoms of the disease, or a slight hematuria, moderate dysproteinemia and stabilization of blood pressure.

The exacerbation phase is characterized by the appearance or aggravation of the existing clinical and laboratory manifestations of the disease.

Signs of exacerbation of chronic glomerulonephritis are:

– clinical signs: an increase in proteinuria, an increase in hematuria, a sudden progressive nephrotic syndrome, a sharp increase in arterial hypertension, a rapid decrease in renal functions, often accompanied by oliguria and large edema, manifestations of DIC syndrome;

– biochemical signs: increased ESR, increased blood levels of α2-globulins, sometimes γ-globulins, azotemia with normal kidney size, the presence of organ-specific kidney enzymes in the urine (transamidinase, lactate dehydrogenase isoenzymes);

– changes in the indicators of humoral immunity: an increase in the level of circulating immune complexes and the content of immunoglobulins in the blood, a decrease in the level of complement.

The severity of exacerbation is characterized by three degrees of activity (I, II and III).

DIAGNOSTICS

The examination program for patients with chronic glomerulonephritis is the same as for acute glomerulonephritis.

In addition, ultrasound and radioisotope scanning of the kidneys are advisable.

TREATMENT

Therapeutic tactics for chronic glomerulonephritis are as follows:

1. *Bed rest* (until the elimination of edema and normalization of blood pressure). It is necessary to avoid hypothermia, physical overload, etc.

2. *Therapeutic nutrition*:

– restriction of table salt, simple carbohydrates, protein;

– exclusion of extractive substances from the diet and full provision of the body with vitamins and trace elements.

3. *Etiological treatment* (more often it is impossible or does not play a significant role).

4. *Pathogenetic treatment*:

– glucocorticoids (including pulse therapy with methylprednisolone);

– cytostatics (including sandimmun-neoral for steroid-resistant forms of chronic glomerulonephritis);

– nonsteroidal anti-inflammatory drugs;

– anticoagulants and antiplatelet agents.

5. *Symptomatic treatment* (treatment of arterial hypertension, edematous syndrome, etc.).

6. *Phytotherapy*.

7. *Spa treatment*.

ACUTE PYELONEPHRITIS

Acute pyelonephritis is an acute infectious inflammatory kidney disease that first involves the calyx and pelvis, and can also pass to interstitial tissue. Acute pyelonephritis is always associated with the development of infection, the causative agents of which are tropic to the renal tissue.

The symptomatology of the disease is characterized by an increase in body temperature up to a high (40° C) fever with chills and sweating; pain syndrome of varying intensity, often with irradiation to the inguinal region, lower abdomen; dysuria phenomena (discomfort or pain when urinating, frequent urge to urinate), a change in the transparency of urine — the urine is cloudy.

Pyelonephritis is characterized by pyuria — the release of a large number of leukocytes, bacteria, necrotic cells of the urinary tract epithelium.

CHRONIC PYELONEPHRITIS

Chronic pyelonephritis is a chronic nonspecific infectious and inflammatory process with a predominant and initial lesion of the calyx-pelvic system and renal tubules, interstitial tissue with subsequent involvement of glomeruli and renal vessels in the process.

The main etiological factor is the penetration of infection into the urinary tract, the cup-pelvic system, interstitial kidney tissue. Chronic pyelonephritis and its exacerbations are caused by various types of infection.

Factors predisposing to the development of chronic pyelonephritis:

– transferred acute pyelonephritis;

– urological manipulations;

– overcooling;

– urodynamic disorders, i.e. outflow disorders urine of various genesis (stones, tumors, prostate adenoma, urinary tract strictures);

– pregnancy;

– diabetes mellitus;

– chronic infections in the ENT organs and oral cavity;

– genetic predisposition to chronic pyelonephritis.

Ways of infection penetration into the kidney:

– hematogenic;

– ascending or urinogenic pathway;

– ascending along the wall of the urinary tract.

Currently, the role of immune mechanisms in the pathogenesis of the disease has significantly increased, as well as the importance of genetic predisposition.

Under conditions of genetic predisposition, in the presence of urodynamic disorders, reflux and urinary infection, urine stagnation and penetration into the renal tissue of both an infectious agent and immune complexes containing antibodies to it develop, which leads to immune inflammation of the renal tissue, the formation of inflammatory infiltrate and the development of chronic inflammation.

CLASSIFICATION

1. By origin:
– primary pyelonephritis;
– secondary pyelonephritis.
2. On localization of the inflammatory process:
– one-sided (right, left);
– two-sided;
– total (affecting the entire kidney);
– segmental (affecting the segment or kidney site).
3. Phase of the disease:
– the phase of exacerbation;
– remission phase.
4. Activity of the inflammatory process:
– active inflammatory process;
– latent inflammatory process;
– remissions.

Clinical forms:

– septic;
– latent;
– recurrent;
– anemic;
– hematuric.

CLINICAL PICTURE

Complaints:

1. specific:

– pain in the lumbar region of a nagging nature or quite intense, even with irradiation to the lower abdomen;

– dysuric phenomena;

– the release of cloudy urine, sometimes with an unpleasant odor, giving a cloudy sediment when standing (often purulent);

– transient rises in body temperature to 39.5–41° C (with a decrease in the morning to 38) and chills;

2. general: weakness, decreased performance, headaches, poor sleep, decreased appetite

DIAGNOSIS OF ACUTE AND CHRONIC PYELONEPHRITIS

1. General blood test (leukocytosis, leucocyte formula shift to the left, increased ESR).

2. Biochemical blood analysis: determination of the level of urea, creatinine, total protein, protein fractions, sialic acids, CRP.

3. Urine tests:

– general urinalysis (leukocyturia, bacteriuria, proteinuria);

– urine analysis according to Nechiporenko (increase in the number of leukocytes);

– urine analysis according to Zimnitsky (change in daily diuresis — polyuria, oliguria; nocturia; decrease in relative urine density — hypostenuria, isohypostenuria).

4. Rehberg–Tareev test (determination of the presence and degree of decrease in the rate of glomerular filtration and tubular reabsorption by endogenous creatinine).

Additional research methods:

1. Blood pressure monitoring.

2. Fundus examination.

3. Ultrasound examination of the kidneys (asymmetry of kidney size, expansion and deformation of the calyx-pelvic system, diffuse acoustic heterogeneity of the renal parenchyma, compaction of the papillae of the kidneys, shadows in the pelvis (sand, stones, papillary sclerosis), irregularities in the contour of the kidneys, sometimes a decrease in the thickness of the parenchyma).

4. Radioisotope scanning of the kidneys (asymmetry of kidney size, diffuse nature of changes).

5. Radioisotope renography(decreased secretory-excretory function of the kidneys on one or both sides).

6. X-ray examination (an increase in the renal-cortical index and the appearance of Hudson's sign — a decrease in the thickness of the parenchyma at the poles, the detection of a decrease in the tone of the upper urinary tract, pyelorenal reflux, pyelectasia and asymmetry of the kidneys).

7. Retrograde angiography (deformation of the pelvic-pelvic system is revealed).

8. Renal angiography (there is a decrease in the lumen of the renal artery, a decrease in peripheral blood supply to the kidneys, obliteration of small vessels of the cortical substance (performed in case of difficult diagnosis by previous methods or for differential diagnosis).

9. Chromocystoscopy (there is a violation of the excretory function of the kidneys on both sides or on one side).

10. Kidney biopsy.

PRINCIPLES OF THERAPEUTIC TACTICS OF ACUTE AND CHRONIC PYELONEPHRITIS

1. Regime (bed rest until the symptoms of the disease are eliminated).

2. Therapeutic nutrition.

3. The appointment of specific therapy aimed at correcting the disease (antibacterial therapy after determining the sensitivity of flora; anti-inflammatory drugs; drugs that promote detoxification; drugs that improve renal blood flow, symptomatic therapy).

4. Herbal medicine.

5. Spa treatment.

CHRONIC RENAL FAILURE

Chronic renal failure (CRF) is a pathological symptom complex caused by a sharp decrease in the number and function of nephrons, which leads to disruption of excretory and endocrine kidney function, homeostasis, disorder of all types of metabolism, acid-base balance, activity of all organs and systems.

Renal insufficiency is an intoxication of the body caused by impaired renal function (as a result of the progression of nephrosclerosis as a result of various diseases).

The main causes of CRF are:

1. Damage to glomeruli, tubules, interstitial kidneys: chronic glomerulonephritis, subacute glomerulonephritis, chronic interstitial nephritis, chronic pyelonephritis, tuberculosis and amyloidosis of the kidneys.

2. Congenital kidney diseases: polycystic disease, hypoplasia, Alport syndrome (hereditary glomerulonephritis with progressive renal insufficiency in combination with decreased hearing acuity and, less often, vision).

3. Systemic diseases: systemic lupus erythematosus, systemic scleroderma, rheumatoid arthritis, dermatomyositis, nodular periarteritis, hemorrhagic vasculitis and other systemic vasculitis, myeloma.

4. Heart and vascular diseases: arterial hypertension, renal artery stenosis.

5. Endocrine diseases and metabolic disorders (diabetes mellitus, hyperparathyroidism, gout).

6. Obstructive diseases of the upper (stones, tumors, retroperitoneal fibrosis) and lower (anomalies of the neck of the bladder and urethra, urethral strictures, adenoma and prostate cancer, bladder cancer) urinary tract with the addition of chronic pyelonephritis.

Under the influence of etiological factors, the number of functioning nephrons and glomerular filtration decrease, fibroplastic processes develop with the replacement of nephrons with connective tissue; compensatory hyperfiltration takes place in the preserved nephrons, which contributes to their progressive lesion and aggravation of structural changes.

Increased load on functioning nephrons is the main non-immune mechanism of progression of CRF.

A sharp decrease in the mass of active nephrons in CRF is due to the development of the following main pathogenetic factors:

1) violation of the excretion of the end products of protein metabolism, which leads to the development of azotemia: the level of urea and creatinine increases (normally in the blood serum urea — 2.5–8.3 mmol/l, creatinine — 0.08–0.1 mmol/l);

2) impaired ability of the kidneys to concentrate and excret urine:

• the rhythm of urine excretion is disrupted (nocturia)

• daily diuresis changes:

– polyuria > 1500 ml;

– oliguria < 500–600 ml;

– anuria < 200 ml;

• the relative density of urine and its daily fluctuations change: in the morning not < 1018, the difference is < 8 units, hypostenuria, isostenuria;

3) electrolyte imbalance develops (depending on the stage of CRF, the level of electrolytes in the blood serum increases or decreases).

The level is normal:

• potassium — 3.5–5.5 mmol/l;

• sodium — 135–155 mmol/l;

• calcium — 2.2–3.0 mmol/l;

4) violation of acid-base balance (blood pH is normal = 7.35–7.45);

5) the hematopoietic function is disrupted, which leads to the development of anemia.

CLINIC

Most often, the disease proceeds latently, then appear:

– minor symptoms (polyuria, thirst, dry skin);

– dyspetic disorders (nausea, hiccups);

– sleep disorders.

Further, due to the increase in azotemia, electrolyte and other disorders, the clinical picture develops:

– gingivitis, stomatitis, gastritis, colitis (manifested by nausea, vomiting, decreased and then loss of appetite, diarrhea, weight loss);

– laryngitis, bronchitis (due to the release of urea);

– itching of the skin (excretion of urea through the skin);

– hemorrhages (impaired liver function, impaired hematopoiesis);

– symptoms of uremic encephalopathy;

– heart — rhythm disturbance (electrolyte imbalance).

Next — coma.

DIAGNOSIS OF CRF SEVERITY

It is based on the following diagnostic criteria:

1. Determination of urea and creatinine.

2. Determination of glomerular filtration and tubular reabsorption.

The Rehberg–Tareev test:

– glomerular filtration rate (normally — 80–120 ml/min);

– tubular reabsorption (normally — 97–98 %);

– creatinine (normally — 0,08–0,1 mmol/L);

– daily diuresis;

– day and night diuresis.

3. Determination of the relative density of urine during the day (Zimnitsky test).

4. Blood pH.

5. Blood electrolyte composition (K, Na, Sa).

6. The severity of anemia.

Depending on the severity, the following are distinguished stages of CPN:

– latent;

– compensated;

– intermittent;

– terminal.

The latent stage. There are no complaints. Diuresis is normal. Zimnitsky's test is the norm. Urea — up to 8.8 mmol/l; creatinine — up to 0,18 mmol/l. GFR — 45–60 ml/min. Hb is more than 100 g/l. K, Na, Sa — normal. Blood pH is normal.

Compensated stage. Complaints: nausea, dry mouth, fatigue. Diuresis — mild polyuria (2–3 liters of urine per day). Zimnitsky's test — the difference in the figures of the relative density of urine is less than 8 units; urea — 8.8–10.0 mmol/l; creatinine — 0.2–0.28 mmol/L. GFR — 40–30 ml/min. Hb —100–80 g/l. Na — slightly reduced; K, Ca — normal. Blood pH is normal.

The intermittent stage. Complaints: severe weakness, constant thirst, frequent vomiting, lack of appetite, sleep disturbance, headache. Diuresis — pronounced polyuria (> 3 liters). Zimnitsky's test — isostenuria, hypostenuria (relative density of urine 1012–1010). Urea — 10.1–19.9 mmol/l; Creatinine — 0.3–0.6 mmol/l. GFR — 30–20 ml/min. Hb — less than 80 g/l. Na, K, Sa — reduced. Moderate metabolic acidosis (blood pH — 7.3).

Terminal stage. Complaints: constant vomiting, diarrhea, headache, cramps, drowsiness, itching, cachexia. Diuresis is an oliguria that turns into anuria. Zimnitsky's sample — the relative density of urine is 1008 and lower. Creatinine — more than 0.6 mmol/l; urea — more than 20 mmol/l; GFR — less than < 20 ml/min. Nv — 40 g/l and below. Na is lowered, then rises. K and Sa are elevated. Severe metabolic acidosis.

CRITERIA FOR CHRONIC KIDNEY DISEASE

CKD is a supranosological concept that unites all patients with any pathological changes on the part of the kidneys that persist for 3 months or more according to laboratory and instrumental studies and/or the presence of impaired renal function in the form of a decrease in the glomerular filtration rate (DOCKS (2002, Congress of the American Society of Nephrology).

The diagnosis of CKD does not cancel the nosological verification of diseases and is not a mechanical combination of chronic kidney injuries of various nature. The transition to a broader concept is caused by the need to focus not on the expensive treatment of the final stage of kidney disease (dialysis, transplantation), but on the preventive direction. It is very important to detect the disease or the threat of developing renal pathology early and to develop methods for the prevention, prediction and treatment of nephropathies at the initial, still reversible stages.

The concept of CKD is more universal and to a greater extent than the term CPN, corresponds to the tasks of prevention and nephroprotection. The development of the concept of CKD means a shift of emphasis from the terminal stage to the early ones, ensures continuity of patient management.

The criteria for determining CKD are:

– the presence of any clinical markers of kidney damage confirmed at intervals of 3 months or more: changes in blood tests (increased urea and creatinine, changes in acid-base state, electrolyte composition, etc.) or urine (albuminuria, proteinuria, persistent hematuria, leukocyturia, decreased specific gravity, etc.);

– the presence of any markers of irreversible structural changes of the organ detected once during visualization or morphological examination (cysts, hydronephrosis, transformation of kidney size, etc.);

– reduction of GFR less than 60 ml/min/1.73 m^2 for 3 months or more with the presence of other signs of kidney damage or without.

GFR reflects the percentage of functioning nephrons: a value of less than 60 ml/min/1.73 m^2 corresponds to the death of more than half of them. The criterion of “durability” is considered to be a period of 3 months. During this time, acute nephropathies, as a rule, end in recovery or signs of chronization of the process are revealed. The main criterion is considered to be GFR, since creatinine levels begin to rise only with serious violations of renal functions, up to 50 % of which may be lost even earlier.

CLASSIFICATION OF CKD STAGES

Stage 1 — all cases of chronic kidney disease, manifested by any of the above signs of pro-preserve kidney function (GFR > 90 ml/min/1.73 m^2).

Stage 2 — signs of nephropathy are accompanied by a slight decrease in kidney function (GFR 60–89 ml/min/1.73 m^2).

3-Stage I — the disease is accompanied by a moderate decrease in kidney function (GFR up to 30–59 ml/min/1.73 m^2).

Stage 4 means severe renal dysfunction (GFR 15–29 ml/min/1.73 m^2).

Stage 5 corresponds to terminal CRF (GFR < 15 ml/min/1.73 m^2 or dialysis treatment) (Table).

The ratio of the stages of CKD and CRF
(by N. A. Lopatin, I. N. Kuchinsky)

Stage of CKD	SKF	Stage of CPN
C1	> 90	–
C2	89–60	–
C3a	59–45	latent
C3b	44–30	compensated
C4	29–15	intermittent
C5	< 15	terminal

Principles of CKD treatment tactics:

1. The appointment of specific therapy aimed at correcting the primary disease that led to the development of CKD.
2. Detection and treatment of concomitant pathology.
3. Prevention and treatment of cardiovascular complications.
4. The use of drugs that reduce the content of nitrogenous slags.
5. Prevention and treatment of complications of decreased renal function (hypertension, anemia, acidosis, etc.).
6. Preparation for therapy of terminal renal insufficiency (decrease in GFR less than 30 ml/min, etc.).
7. Replacement of kidney function by dialysis or transplantation in the presence of symptoms of uremia (GFR less than 15 ml/min, etc.).

It is important to remember that the diagnosis of “chronic kidney disease” is recommended to be indicated after the underlying disease and, in each case, strive to: identify the etiological cause or several of them, reflect the stage of GFR, the albuminuria/proteinuria index and the type of renal replacement therapy.

SELF-CONTROL OF TOPIC ASSIMILATION

1. Glomerulonephritis is characterized by the following complaints of patients:

a) pain in the lumbar region;
b) cough;
c) hemoptysis;
d) nausea attacks;
e) swelling on the face.

2. The main cause of acute glomerulonephritis:

a) staphylococcus;
b) β-hemolytic streptococcus of group A;
c) β-hemolytic streptococcus group B;
d) β-hemolytic streptococcus type 4;
e) viruses.

3. Clinical forms of chronic glomerulonephritis include:

a) nephrotic;
b) compensated;
c) latent;
d) hypertensive;
e) triad.

4. Clinical forms of acute glomerulonephritis include:

a) classical triad;
b) mixed;
c) nephrotic;
d) arterial;
e) urinary.

5. Establish a correspondence between the stages of chronic renal failure and the creatinine content in the blood:

a) compensated;	1) up to 0.18;
b) intermittent;	2) 0.2–0.28;
c) latent;	3) 0.3–0.6;
d) terminal;	4) more than 0.6.

6. Establish the correct sequence of anamnesis collection in patients with acute glomerulonephritis:

a) anamnesis of the disease;
b) complaints;
c) objective methods of examination of the patient;
d) anamnesis of life.

7. Acute glomerulonephritis includes symptoms:

a) pain in the lumbar region;
b) urine the color of meat slops;
c) dry cough;
d) nausea;
e) heartburn.

8. The latent form of chronic glomerulonephritis is characterized by:

a) urinary syndrome;
b) arterial hypertension;
c) fundus changes;
d) edema;
e) all of the above.

9. The hypertensive form of chronic glomerulonephritis is characterized by:

a) severe arterial hypertension;
b) pronounced changes in the fundus;
c) pronounced edema;
d) massive proteinuria;
e) all of the above.

10. Nephrotic syndrome is characterized by:

a) massive proteinuria;
b) hypoproteinemia;
c) severe arterial hypertension;
d) hyperlipidemia;
e) pronounced persistent edema.

11. Glomerulonephritis is characterized by a predominance in urine tests:

a) protein;
b) red blood cells;
c) leukocytes;
d) bacteria;
e) salt crystals.

12. Glomerulonephritis is characterized by a predominance in urine tests according to Nechiporenko:

a) protein;
b) red blood cells;
c) leukocytes;
d) bacteria;
e) salt crystals.

13. The Zimnitsky test allows you to evaluate:

a) daily diuresis;
b) violation of the ability of the kidneys to concentrate and excret urine;
c) glomerular filtration rate;
d) azotemia;
e) metabolic acidosis.

14. The Rehberg–Tareev test includes:

a) daily diuresis;
b) glomerular filtration rate;
3) tubular reabsorption;
c) blood creatinine;
d) the relative density of urine.

15. To determine the stage of chronic renal failure, the following indicators are used:

a) creatinine;
b) glomerular filtration rate;
c) daily diuresis;
d) bilirubin;
e) blood pressure.

16. The main complaints of patients with pyelonephritis include:

a) lumbar pain, fever, dysuric complaints;
b) pain in the lumbar region;
c) increase in blood pressure;
d) increase in body temperature;
e) all of the above.

17. Pyelonephritis is characterized by a predominance in urine analysis:

a) piurias;
b) hematuria;
c) proteinuria;
d) albuminuria;
e) all of the above.

18. The criteria for determining CKD are as follows:

a) the presence of clinical markers and changes in blood and urine tests at intervals of 3 months or more;
b) the presence of clinical markers with an interval of 3 months or more;
c) the presence of changes in blood and urine tests at intervals of 3 months or more;
d) all of the above;
e) none of the above.

19. In the classification of CKD, there are:

a) 3 stages;
b) 2 stages;
c) 5 stages;
d) 4 stages;
e) the stages are not distinguished.

20. The classification of CKD is based on the definition of:

a) SKF;
b) creatinine levels;
c) the level of hypoproteinemia;
d) proteinuria;
e) severity of AG.

Answers: 1 — a, e; **2** — b; **3** — a, c, d; **4** — a, c; **5** — a2, b3, c1, d 4; **6** — b, a, d, c; **7** — a, b; **8** — a; **9** — a, b; **10** — a, b, d, e; **11** — a, b; **12** — b; **13** —a, b; **14** — b, c, d; **15** — a, b, c; **16** — a; **17** — a; **18** — a; **19** — c; **20** — a.

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