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Guidelines for conducting a practical lesson with 5th year students of the Faculty of Medicine on the topic:

DIFFERENTIATED DIAGNOSIS AND DIFFERENTIATED TREATMENT OF PYELONEPHRITIS AND GLOMERULONEFRITIS

(the duration of the lesson is 8 hours, the first lesson is 4 hours)

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PURPOSE OF THE LESSON: in the process of clinical analysis of the patient to increase the level (quality) of knowledge and skills of students in the diagnosis (differential diagnosis), formulation of the diagnosis, prevention and treatment of pyelonephritis and glomerulonephritis.

Students should be able to:

1. independently, on the basis of complaints, anamnesis and objective status, suggest a diagnosis of pyelonephritis or glomerulonephritis in a patient

2. appoint a plan for an individual examination of a patient with pyelonephritis and glomerulonephritis.

3. differentiate pyelonephritis and glomerulonephritis.

Motivation of the relevance of the topic.

The most common kidney diseases that lead to the development of chronic renal failure and require further expensive replacement therapy are pyelonephritis and glomserulonephritis. The term "pyelonephritis" refers to a non-specific infectious-inflammatory process that occurs mainly in the renal pelvic-cup system and its tubulointerstitial zone.

Three groups of the population are most at risk of developing this disease: young girls, pregnant women and women in childbirth, and the elderly. In men, the development of pyelonephritis is usually associated with obstructive processes; in boys and young men, pyelonephritis is quite rare.

Under the term glomerulonephritis (GN) is an immunoinflammatory disease of the kidneys with a primary lesion of the glomeruli, but involving both the tubules and the interstitial (interstitial) tissue.

Completely different factors can lead to the development of GN: streptococcal infection (a typical post-infectious lesion), brucellosis, malaria, recketsiosis, treponemal infections, syphilitic GN, GN against the background of persistent HBs antigen, against the background of helminthic invasions, "cold GN", trauma, insolation , intolerance to food and chemicals, drug and vaccine damage to the kidneys, GN on the background of systemic diseases (nodular periarthritis, systemic scleroderma, rheumatoid arthritis, systemic lupus erythematosus, hemorrhagic vasculitis, Gedpasture's disease, etc.). A wide range of etiological factors determines the significant prevalence of GN.

Determining the level of students' preparation. The second level of knowledge: methods of control - a written survey (20 min). Students should know the main issues of etiology, pathogenesis, clinic and diagnosis of pyelonephritis and glomerulonephritis, definition and classification of pyelonephritis and glomerulonephritis and the main stages of differential diagnosis; students should be able to possess propaedeutic skills.

Report of student curators in the Chamber. When reporting a patient, students should pay special attention to the following manifestations of the disease.

Causes of chronic pyelonephritis in women:

- features of the anatomical structure of the urethra (it is wider and shorter, which facilitates the penetration of infection into the urinary tract);
- vulvovaginitis (in this case, infection of the urinary tract is possible);
- pregnancy;
- early postpartum period (predisposes to the penetration of infection into the urinary tract);
- the use of hormonal contraceptives (may contribute to the development of urinary tract dyskinesia and thereby predispose to the development of chronic pyelonephritis).

Ways of penetration of infection into the kidney.

- descending or hematogenous;
- ascending or urinogenic;
- ascending along the wall of the urinary tract.

Factors predisposing to the formation of an infectious process in the urinary tract and kidneys:

- obstruction of the urinary tract,
- instrumental studies of the urinary tract, contributing to their direct infection;
- pregnancy
- diabetes
- non-infectious pathology of the kidneys (GN, etc.),
- immunosuppressive therapy and related changes in immunity and non-specific factors protecting the urinary tract and the body as a whole.

Algorithm for the diagnosis of acute pyelonephritis

Classification of chronic pyelonephritis

By occurrence: primary, secondary

According to the localization of the inflammatory process: unilateral, bilateral, total, segmental

Phase of the disease: exacerbations; remissions.

The activity of the inflammatory process:

The phase of the active inflammatory process: leukocyturia - L-25,000 / ml or more; bacteriuria - 100,000/ml or more, active L (30% or more); Sternheimer-Malbin cells in 25-50% of patients have an increased titer of antibacterial antibodies in the PHA reaction; ESR over 12 mm/h

The phase of the latent inflammatory process: leukocyturia - up to 2500 / ml; bacteriuria is absent; active L (15-30%); Steinheimer-Malbin cells are absent; the titer of antibacterial antibodies in the PHA reaction is normal; ESR not higher than 12 mm/h

Remission phase: leukocyte- and bacteriuria - absent; active L are absent; Sternheimer-Malbin cells are absent; the titer of antibacterial antibodies in the PHA reaction is normal; ESR - less than 1 mm / h;

Clinical forms:

1. Hypertensive.

2. Nephrotic (rare).

3. Septic.

4. Hematuria.

5. Anemic.

6. Latent (little symptomatic).

7. Recurrent.

Symptoms of chronic pyelonephritis

- pain in the lumbar region (often unilateral) of a aching nature, sometimes quite intense (painful form), can radiate to the lower abdomen, genitals, thigh;

- dysuric phenomena (painful frequent urination due to concomitant cystitis);

- excretion of cloudy urine, sometimes with an unpleasant odor, giving

when standing cloudy sediment (often purulent);

- chilling with severe exacerbation, sometimes transient rises in body temperature to 38.5-39 ° C with normalization by morning.

On examination, you may notice:

- pale skin and visible mucous membranes;

- weight loss (not always);

- pastosity of the face; not typical for chronic pyelonephritis

pronounced edema;

- Pain when feeling or tapping the lumbar

areas (often unilateral);

- a symptom of A.P. Tofilo (1988) - in the supine position, the patient bends the leg at the hip joint and presses the thigh to the stomach,

in the presence of pyelonephritis, pain in the lumbar region increases, especially if you take a deep breath.

The program of examination of a patient with suspected pyelonephritis.

Laboratory research

Complete blood count: signs of anemia, leukocytosis, shift of the blood formula to the left and toxic granularity of neutrophils (with severe exacerbation), increased ESR.

Urinalysis: cloudy urine, alkaline reaction, decrease in urine density; moderate proteinuria, microhematuria, severe leukocyturia, possible cylindruria, bacteriuria (more than 100,000 microbial bodies in 1 ml of urine).

The Nechiporenko test (determination of the content of leukocytes and erythrocytes in 1 ml of urine) - the predominance of leukocyturia over erythrocyturia (normally, the number of leukocytes in 1 ml does not exceed 4,000, erythrocytes - 2,000).

Examination of urine for sterility and determination of the sensitivity of the urine flora to antibiotics.

Zimnitsky test: decrease in urine density during the day (normal urine density during the day ranges from 1.01 to 1.025 kg / l).

Biochemical analysis of blood: an increase in the content of sialic acids, fibrin, seromucoid, α2- and γ-globulins, creatinine and urea (with the development of chronic renal failure), the appearance of PSA.

Immunological blood tests: high titers of antibodies to the O-A2 antigen of Escherichia coli (the leading causative agent of pyelonephritis); decrease in the number and functional activity of T-lymphocytes.

Prednisolone test (used to detect latent pyuria)

Urinalysis according to Sternheimer-Malbin.

Express methods for detecting bacteriuria: Nitrite test, TTX test

Instrumental Research

Plain radiography of the kidney area: a decrease in the size of the kidneys on one or both sides.

X-ray urological examination:

excretory urography - a decrease in the tone of the upper urinary tract, narrowing and elongation of the cups, deformation and convergence of the cups, pyelorenal refluxes, pyelectasis, asymmetry in the size of the kidneys

retrograde pyelography - the deformation of the pyelocaliceal system is determined, it is possible to identify congenital anomalies of the kidneys;

renal angiography - revealed a decrease in the lumen of the renal artery, a decrease in the peripheral blood supply to the kidneys, obliteration of small vessels of the cortical substance.

Ultrasound examination of the kidneys: asymmetry in the size of the kidneys, expansion and deformation of the pyelocaliceal system, diffuse acoustic heterogeneity of the renal parenchyma, compaction of the papillae of the kidneys, shadows in the pelvis (sand, small stones, sclerosis of the papillae), irregularities in the contour of the kidneys, sometimes thinning of the parenchyma.

Differential diagnosis of chronic glomerulo- and pyelonephritis

signs	**glomerulonephritis**	**pyelonephritis**
Features of the anamnesis	Exacerbations of streptococcal infections	Indications for ICD, urination disorders
Features of pain	Obtuse bilateral, not always expressed	Pronounced, unilateral, rarely bilateral
Dysuria	not typical	typical
Body temperature	Normal, less often - a slight increase	Exacerbation - sharply increased, without exacerbation - prolonged subfebrile condition
chills	not typical	typical
Leukocyturia, bacteriuria	not typical	typical
Hematuria	Microhematuria is almost constant. Erythrocytes predominate in the Nechiporenko test	not typical Leukocytes predominate in the Nechiporenko test

Proteinuria	Significantly pronounced, cylindruria	less pronounced
Edema syndrome	typical	not typical
Asymmetry of kidney damage	Поражение двустороннее	typical
The state of the pelvicalyceal system according to ultrasound	normal	Signs of damage, often ICD

Main clinical types of GN:
acute, chronic and rapidly progressive.

In the vast majority of cases, GN develops with the participation of immune mechanisms: this is the formation of antibodies, immune complexes (deposited in the glomeruli when the mechanisms for their removal are insufficient), and the renal glomerulus responds to immune damage with two types of pathological reactions: proliferation of cells of the renal glomerulus (mesangial, endothelial, epithelial) and the production of intercellular substance by these cells. Both processes stimulate cytokines (primarily interleukin-1, tumor necrosis factor, platelet-derived growth factor, transforming growth factor beta), which are secreted by cells that infiltrate the renal glomerulus.

Morphological signs of AGN consist of proliferation of glomerular cells, changes in the basement membrane of glomerular capillaries, cell necrosis, sclerosis of glomerular loops, as well as changes in the tubules of the kidneys (dystrophy, atrophy), vessels and interstitium (sclerosis, cell infiltration).

Clinical signs of AGN -

Oliguria - as a result of inflammation, a spasm of the afferent arteriole occurs, the hydrostatic pressure in the glomerulus drops, the pressure in the B-Sh capsule rises. Therefore, renal filtration falls. The reflex release of ADH leads to increased water reabsorption.

Proteinuria

Hematuria.

Rise in blood pressure: due to an increase in BCC. The release of aldosterone increases the processes of sodium reabsorption, which retains water. There is an overflow of all blood vessels, including the vessels of the brain.

Spasm of the afferent arteriole leads to ischemia of the kidney. There is a release of renin - activation of angiotensin I - angiotensin II, a powerful vasopressor and increases in blood pressure.

Edema: decreased glomerular filtration rate, increased reabsorption
water and sodium, increased permeability of the vascular wall,
increased aldosterone and ADH, hypoalbuminemia. (for acute
glomerulonephritis they are less pronounced).

Cylindruria: the tubular apparatus is secondarily disturbed.

Hyperstenuria: because. oliguria, proteinuria.

Complications :

• Convulsive syndrome (eclampsia): pronounced edema, including edema
brain, leading to compression of the substance of the brain.
• Acute heart failure (left ventricular)
• Acute renal failure.

Acute GN

- infectious-immune - develops 6-12 days after the infection (more often - group A beta-hemolytic streptococcus).
- non-infectious-immune - after the introduction of vaccines, sera, due to individual intolerance and hypersensitivity to certain substances, plant pollen, insect venom, due to alcohol intoxication.

Depending on the place of formation of immune complexes, GN with circulating immune complexes (immune complexes arise in the vascular bed, and are localized secondarily in the kidneys) and GN with local formation of immune complexes (directly in the glomerulus) are distinguished.

Antigens in immune complexes can be exogenous (antigens of streptococci, staphylococci, and other bacteria; viral antigens; foreign protein in serum sickness) or endogenous (extraglomerular - antigens of cell nuclei in SLE, cryoglobulins, tumor components; glomerular - mesangial, endothelial, antigens of basal glomerular capillary membranes).

Immune damage is accompanied by an inflammatory response, which eventually ends in repair, leading to various outcomes - from complete restoration of the glomerular structure to global glomerulosclerosis - the basis of progressive renal failure.

In addition to immune mechanisms, hemodynamic disorders play a certain role in the development of AGN. In patients with AGN, the primary factor of circulatory disorders is hypervolemia against the background of a sharply increased water-sodium resorption of the tubules, which plays a leading role in the pathogenesis of hypertension in AGN, in contrast to the concept of angiospasm with renal ischemia and activation of the RAAS, characteristic of CGN.

Distinguish

- Low-immune glomerulonephritis (deposition of immune complexes in the kidneys play an insignificant role - the main factor in the pathogenesis of kidney damage in these diseases is the appearance of antineutrophilic cytoplasmic antibodies that cause the release of proteolytic enzymes that damage the kidneys in polyarteritis nodosa, Wegener's necrotizing granulomatosis, idiopathic glomerulonephritis with crescents).

Antibody glomerulonephritis (characterized by the appearance of antibodies to the basement membrane of the glomerular capillaries and can develop as an isolated lesion or as a systemic disease involving the kidneys and lungs (Goodpasture's syndrome)

Glomerulonephritis due to antigenic mimicry (with molecular mimicry, there is a certain similarity between the antigen and renal structures, resulting in a cross-reaction of antibodies with components of the renal tissue).

Clinical and laboratory manifestations of AGN can be grouped into four main syndromes.

1. Acute glomerular inflammation syndrome

pain in the lumbar region on both sides; increase in body temperature; oliguria, reddish urine or the color of "meat slops"; proteinuria, microhematuria (less often macrohematuria), the appearance in the urine of cylinders (hyaline, granular, erythrocyte), epithelial cells; decrease in glomerular filtration; in the blood leukocytosis, increased ESR; increased levels of α2 and γ-globulins in the blood, increased levels of IgG, IgM, circulating immune complexes, fibrinogen complexes with high molecular weight, high titers of antibodies to streptococcal antigens, hypercoagulation is observed, and as the severity of acute glomerulonephritis increases, anticoagulant activity gradually increases, appear degradation products of fibrin and fibrinogen in the blood.

2. Cardiovascular syndrome

Shortness of breath, rarely - hemoptysis; arterial hypertension, acute hypervolemia leads to an expansion of the heart cavities (its dimensions normalize after diuresis is restored and edema subsides), weakening of the first tone, accent of the second tone over the aorta, systolic murmur over the apex (with relative mitral valve insufficiency), in severe cases - gallop rhythm , ECG changes (low voltage, prolongation of the P-Q interval, two-phase and flattening of the T wave, sometimes a shift in the ST interval), occasionally acute circulatory disorders in the peripheral arteries are possible, leading to focal necrosis on the extremities.

3. Edema syndrome.

"pale" edema, mainly in the face, eyelids (appear in the morning), in severe cases, anasarca, hydrothorax, hydropericardium, ascites are possible, some patients do not have obvious edema, but there is a daily weight gain, which indicates fluid retention in the body (edema is due to a decrease in glomerular filtration, increased tubular reabsorption of sodium and water, the development of hypoalbuminemia, increased secretion of aldosterone and antidiuretic hormone, increased tissue and capillary permeability); proteinuria, which can reach high numbers (up to 90 ‰)

Pain in the lumbar region has varying degrees of severity, often symmetrical, depending on the swelling of the kidneys and stretching of their capsule, violations of urodynamics. Dysuric phenomena occur rarely. Oliguria and even anuria are possible more often in the first days of the disease and usually last 2-3 days. Oliguria in acute GN is characterized by a high relative density of urine.

3. Cerebral syndrome

due to cerebral edema, its characteristic manifestations: headache, nausea, vomiting, decreased vision, increased muscle and mental excitability, motor restlessness; hearing loss, insomnia.

An extreme manifestation of cerebral syndrome is angiospastic encephalopathy (eclampsia). The main symptoms of eclampsia: tonic and 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 the neck veins; pupils are wide; blood-colored foam flows from the mouth; breathing is noisy, snoring; the pulse is rare, tense, blood pressure is high; muscle rigidity, pathological reflexes.

Clinical forms of AGN.

1. Acute cyclic: with a rapid onset, severe renal and extrarenal manifestations - more common in children and adolescents, usually ends in recovery.

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

3. Expanded (with a triad of symptoms: edema, hypertension, urinary syndrome) or triad classical form.

4. Bisyndromic (urinary syndrome in combination with arterial hypertension or nephrotic syndrome).

5. Monosymptomatic

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

Differential Diagnosis

It is necessary to differentiate with urological diseases accompanied by hematuria (nephrolithiasis, nephroptosis, tumors and tuberculosis of the kidneys, thrombosis of the renal veins), acute pyelonephritis, acute tubulointerstitial nephritis, nephritis as part of a systemic connective tissue disease. According to the "syndromic" principle: the main syndrome of the patient is edematous, a similar sign of the following diseases: heart failure, chronic nephritis, nephrotic syndrome, liver cirrhosis, polyserositis.

Data of additional research methods:

In OAM - Proteinuria, body weight is normal or increased

Nechiporenko test - the predominance of erythrocytes (more than 2 thousand in 1 ml.).

Reberg's test - a decrease in the level of glomerular filtration.

In the KLA - slightly reduced HB and hematocrit.

Biochemical analysis of blood - the level of urea, creatinine is usually not disturbed, an increase in the level of lipids and blood lipoproteins is possible.

Ultrasound data of the study - at first without pathological changes, then a decrease in the size of the kidney, thinning of the parenchyma.

According to kidney biopsy - morphological form of GN:

GN with minimal changes, membranous glomerulonephritis, proliferative intracapillary, proliferative extracapillary, membranous-proliferative glomerulonephritis, fibroplastic (sclerosing) glomerulonephritis, mesangial glomerulonephritis, focal segmental hyalinosis

Forecast.

Mortality in AGN is usually low and is associated mainly with complications - cerebral hemorrhage, acute heart failure, infectious complications and thrombosis.

In 10-15% of cases, acute GN becomes chronic. This can be said if hypertension or edema persists during the year, or proteinuria is above 1‰.

Chronic GN often develops slowly, with an imperceptible onset, less often there is a clear connection with acute GN. In pathogenesis, the main role is played by immune mechanisms, however, non-immune progression factors, which include:

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

Classification :

Downstream Acute, subacute (malignant), chronic.

Chronic glomerulonephritis

primary chronic and secondary chronic.

- clinical forms - latent, nephrotic, hypertonic, hematuric, mixed
- phases - exacerbations, activity I, II, III degree, remission.
- stages - chronic renal failure: compensation, subcompensation, decompensation.

Morphological forms of chronic glomerulonephritis:

Minimal changes (destruction of small processes of podocytes on

against the background of focal swelling, loosening and thickening of the basement membranes and proliferation of the endothelium only in separate loops of glomerular capillaries. Clinically - manifestations of nephrotic syndrome. Diagnosis is possible with electronoscopy.

Membranous glomerulonephritis (sharp diffuse thickening, swelling and splitting of the basement membranes of glomerular capillaries, which can be observed in several - focal, and or in all capillary loops of the glomerulus - diffuse. It is believed that the fixation of immunocomplexes on the basement membrane is the beginning of its damage and increased permeability of the glomerular filtrate for plasma proteins).

Proliferative intracalyllary (proliferation of endothelial and mesangial cells with relatively minor changes in the basement membrane of the glomeruli - occurs in various clinical forms of CGN.

Proliferative extracapillary (the presence of crescents due to the proliferation of epithelial cells of the capsule (nephrothelium) of the glomerulus, which, filling the lumen of the capsule, form crescents, compress the capillary loops of the glomerulus and disrupt blood circulation in them. At the same time, exudative phenomena and loss of fibrin into

the cavity of the glomerular capsule are noted. fibroplastic changes with the development of connective tissue and the death of the glomerulus - the basis of malignant subacute glomerulonephritis.).

Membrano-proliferative glomerulonephritis (combines signs of membranous and proliferative changes in the glomeruli, which are diffuse in nature - clinically it is most often manifested by a latent form of glomerulonephritis.

Fibroplastic (sclerosing) glomerulonephritis (completion of the course of all of the above processes. There are diffuse and focal fibroplastic glomerulonephritis - diffuse sclerosis of the mesangium, vascular loops of the glomeruli with the development of ganglinosis and their sclerosis, with the formation of multiple adhesions between the loops of the glomerular capillaries and the leaves of the capsule. This form of glomerulonephritis often corresponds to nephrotic and hypertensive forms of chronic glomerulonephritis, and, as a rule, it occurs in terminal nephritis).

In addition to the above options, I also distinguish mesangial glomerulonephritis (mesangiomembranous, mesangioproliferative, mesangiocapillary), as well as focal segmental hyalinosis - the deposition of immune complexes in the subendothelial space of the basement membrane of the glomerular capillaries and in the mesangium, which leads to proliferation of endothelial and mesangial cells.

Etiology and pathogenesis:

• Infections: streptococci (beta-hemolytic streptococcus group

A) and other bacteria and viruses (hepatitis B, Epstein-Bar, measles, rubella, etc.)

• Systemic connective tissue diseases: SLE, hemorrhagic vasculitis, periarteritis nodosa, Goodpasture's, Wegener's syndromes.

• Serum, drug disease.

In the occurrence of the disease, an autoimmune mechanism takes place. Streptococcus toxins, damaging the glomerular apparatus, cause damage to the basement membrane proteins. Proteins become foreign and AT begins to be produced against them. They interact with basement membrane proteins and cause further damage.

Clinic of chronic glomerulonephritis:

Latent form: isolated urinary syndrome, in the absence of

extrarenal signs of the disease. Daily proteinuria does not exceed 1 g; in a routine study, it most often fluctuates between 0.033 - 1.0 g / l. This stage is characterized by a slight erythrocyturia (5 - 10, less often 30 - 50 erythrocytes in the field of view) and cylindruria. There are no edema, or pastosity under the eyes. BP is long and persistently maintained at a normal level. There are no signs of left ventricular hypertrophy. The appearance of these symptoms is possible with an exacerbation of the process. Kidney function remains normal for a long time. This form is the most favorable in terms of prognosis, but often the diagnosis is established only at the stage of CRF.

The nephrotic form is less common than the latent form and manifests itself as a nephrotic syndrome: a lesion of the tubule with degenerative changes. The epithelium undergoes vacuolization and dystrophy. Podocytes and their processes degenerate. The basement membrane is exposed. In some places, it thickens, in others it becomes thinner, the pore diameter increases sharply and permeability increases (“moth eaten away”). As a result: Massive proteinuria (10 - 60%) hypoproteinemia and massive edema (fluid is not retained in the vascular bed because oncotic pressure drops and moves into the tissues). As a result of the fall of the bcc, the vascular wall value receptors are excited, act on the adrenal cortex, and aldosterone is produced, which increases sodium reabsorption in the tubules. The latter, entering the bloodstream, increases the osmotic pressure of the blood and excitation of the posterior lobe of the pituitary gland occurs through osmo receptors. As a result, water reabsorption increases and oliguria occurs. Due to hypoonkia, the fluid in the vascular bed is not retained and again goes into the tissues - a vicious circle is closed - renal edema is formed (protein-free, soft, after pressure a hole remains). The concentration ability of the kidneys at the beginning does not suffer (sp. weight is normal). In the final stage, hypoisostenuria and true azotemic uremia develop. The development of iron deficiency anemia is associated with a large loss of protein. the transport protein, ferritin, is lost.

Hypertensive form (the leading symptom is arterial hypertension with a slight severity of the urinary syndrome and the absence of edema. In some patients, pastosity may be noted. With an exacerbation of the process, in addition to an increase in blood pressure, patients experience LV hypertrophy, accent II tone over the aorta, systolic murmur over the base of the heart and over the apex Changes in the ischemic nature and rhythm disturbances, conduction (on the ECG), narrowing of the retinal vessels, hemorrhages, hypertensive crises, acute myocardial ischemia, circulatory disorders, acute heart failure are possible.

Hematuric form (significant and persistent hematuria in the absence of edema, hypertension and low proteinuria). It occurs relatively rarely. The diagnosis is made with the exclusion of another pathology, which is manifested by proteinuria (TUMORS of the kidneys,

bladder, polyps, ICD, Berger's disease - Ig nephropathy)

Mixed form. (Combination of syndromes) In prognostic terms, the most unfavorable. The life expectancy of patients is 3-5 years, maximum - 8 years.

Features of the differential diagnosis are associated with the clinical type of chronic glomerulonephritis (CGN). CGN should be differentiated from acute glomerulonephritis, amyloidosis, gouty kidney, etc.

- Idiopathic (bright GN)

CGN

- As part of a systemic disease (SLE, rheumatoid arthritis, etc.)

a) The latent type of GN should be differentiated:

1. with chronic pyelonephritis (high leukocyturia, bacteriuria,
pain syndrome and fever, asymmetric kidney damage).

2. with amyloidosis (kidney biopsy - amyloid, history of tuberculosis
rheumatoid arthritis, suppurative diseases).

3. with gouty kidney (increased uric acid, tophi,
gouty arthritis).

b) Hematuric type of GN:

1. with a tumor.

2. with urolithiasis.

3. with tuberculosis

(computed tomography, x-ray and ultrasound studies reveal a tumor, stones; a study of the nature and source of hematuria - altered or unchanged erythrocytes, a three-glass test: to exclude tuberculosis - biological tests for mycobacteria, a biopsy of the bladder mucosa, kidneys).

c) Hypertensive type of GN should be differentiated:

1. with hypertension: with nephrogenic hypertension
non-crisis persistent hypertension, relatively good subjective
high blood pressure tolerance, young age.

2. with renovascular hypertension (doppler ultrasound).

3. keep in mind Kohn's syndrome, pheochromocytoma (examine in the blood
aldosterone, catecholamines).

d) Nephrotic type of GN:

1. with amyloidosis of the kidneys in the nephrotic stage (amyloid in biopsy
kidney or rectal mucosa, gums).

2. with diabetic nephropathy (presence of diabetes mellitus,
widespread microangiopathy, infectious complications).

3. with SLE (facial erythema in the form of a "butterfly", arthralgia, trophic disorders, fever, LE - cells, antibodies to DNA,
thrombocytopenia).

Assessment of the functional state of the kidneys

Compensation stage: there are no signs of intoxication, a urine sample according to Zimnitsky and residual blood nitrogen are normal

Stage of subcompensation: usually the disappearance of edema, isohyposthenuria,
decrease in glomerular filtration, nocturia, polyuria. blood creatinine
200-300μmol/l.

Stage of decompensation (renal failure): signs of uremia,
weight loss, anemia, hemorrhages, oliguria. In the blood: further increase
urea, creatinine, acidosis, electrolyte imbalance.

Retinopathy.

Determining the phase of the disease:

Exacerbation, relapse: severe clinical and laboratory symptoms
diseases;

Remission: a satisfactory general condition, the absence or low severity of renal and extrarenal signs of the disease.

Criteria for the diagnosis of acute glomerulonephritis: acute development of the urinary syndrome (proteinuria, hematuria), transient arterial hypertension and / or edema in the absence of these symptoms in the past, the absence of left ventricular hypertrophy and azotemia.

Criteria for the diagnosis of chronic glomerulonephritis: stability of the urinary syndrome and other manifestations (edema, arterial hypertension), duration of the disease, impaired renal function, in the presence of arterial hypertension - signs of left ventricular hypertrophy.

Clinical diagnosis:

It is formed on the basis of the most common classification by V.M. Tareeva. The morphological classification of V.V. Serov.

The clinical type of nephritis, renal function (presence and stage of renal failure), and if there is a biopsy, the morphological type of nephritis are taken into account.

For example:

• Chronic glomerulonephritis of mixed type (morphological - focal segmental glomerulogialinosis) in the stage of chronic renal failure (anemia, azotemia, arterial hypertension.

• Chronic glomerulonephritis of latent type with preserved function kidneys.

• Chronic glomerulonephritis of nephrotic type (membranous) with early signs of renal failure.

Preliminary diagnosis: based on leading complaints and clinical manifestations, as well as indicators of additional studies.

differential diagnosis. It is necessary to differentiate pyelonephritis with glomerulonephritis, with systemic diseases of the connective tissue, with atherosclerotic lesions of the kidneys, with amyloidosis and kidney lesions in hypertension and diabetes mellitus.

Clinical diagnosis: according to the accepted classification with the rationale for the form and phase of the disease, the severity and existing complications in a particular case.

Conducting classes in a thematic classroom. Analysis of the features of the etiology, pathogenesis, clinic and treatment of a particular patient. Indicate the main methods of non-drug exposure (changing lifestyle, nutrition, giving up bad habits, doing physiotherapy exercises). The main groups of drugs and their mechanisms of action, the main indications and contraindications for use and the rationale for choosing a particular drug from pharmacological groups.

The final part of the lesson: control of the acquired knowledge - solving situational problems without possible options for correct answers.

Summary.