

МИНИСТЕРСТВО НАУКИ И ВЫСШЕГО ОБРАЗОВАНИЯ
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GLOMERULONEPHRITIS IN CHILDREN

Учебное пособие

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Издание посвящено одной из актуальных проблем современной
медицины – гломерулонефритам у детей. Заболеваемость гломерулоне-
фритом остается высокой, несмотря на наличие современных методов ди-
агностики и лечения. Рассматриваются вопросы этиопатогенеза, клини-
ческих проявлений, диагностики, педиатрической тактики заболеваний.

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по специальностям 31.05.01 «Лечебное дело», 31.05.02 «Педиатрия».

The publication is dedicated to one of the urgent problems of modern
medicine – glomerulonephritis in children. The disease of glomerulonephritis
remains high, despite the availability of modern diagnostic and treatment meth-
ods. The issues of etiopathogenesis, clinical manifestations, diagnosis, and pe-
diatric tactics of diseases are considered.

It was prepared at the Department of "Pediatrics" of the Medical Insti-
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1. CONCEPT OF GLOMERULONEPHRITIS

Glomerulonephritis is a disease based on immune inflammation with predominant damage to the glomeruli of the kidneys (fig. 1).

The process is accompanied by retention of fluid and salts in the body as a result of the development of secondary circulatory disorders in the kidneys, which ultimately often leads to severe arterial hypertension and fluid overload.

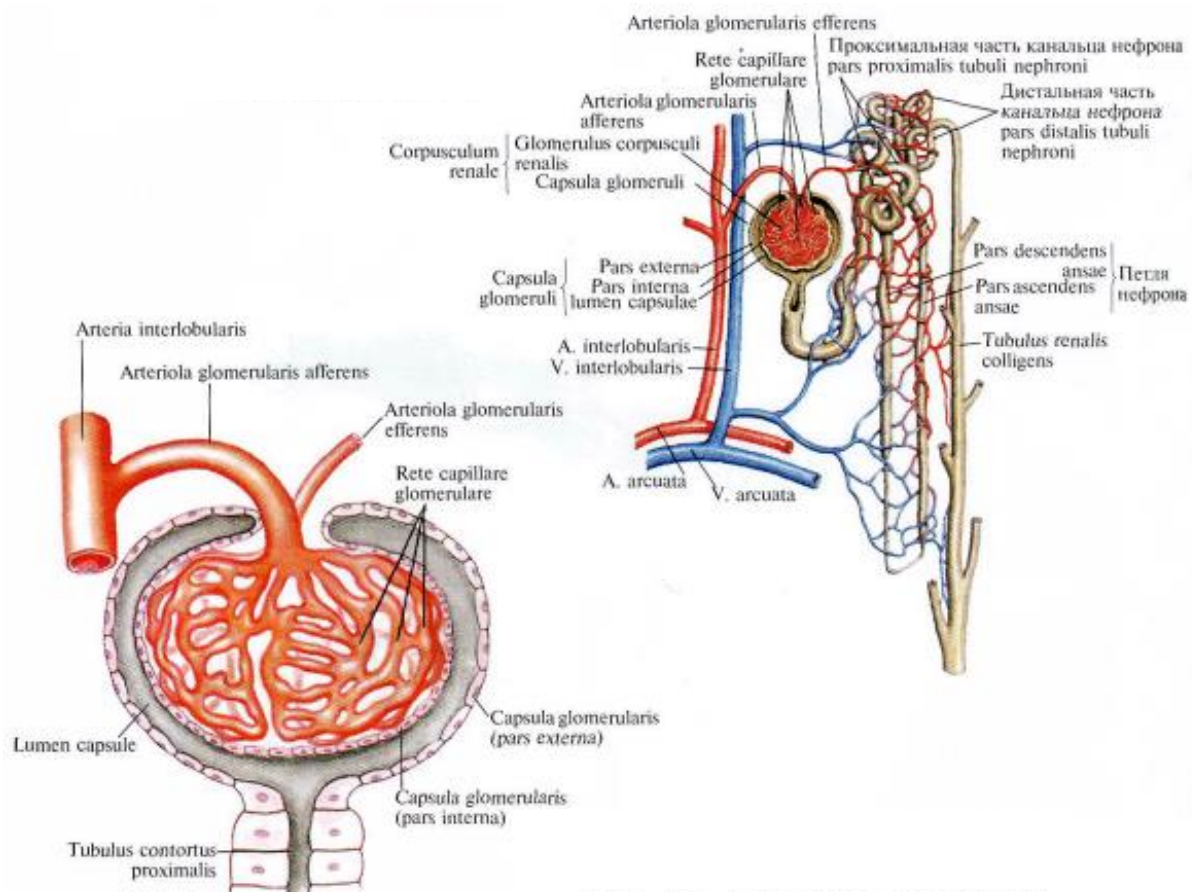


Fig. 1. Structure of the glomerulus (image from the website studfile.net)

There are primary glomerulonephritis, which develops due to the direct impact of an etiological agent on the renal tissue, and secondary, which occurs against the background of systemic connective tissue diseases.

The process may present as acute renal failure, nephrotic syndrome or nephritic syndrome, or as isolated proteinuria and / or hematuria.

These conditions are divided into proliferative or non-proliferative groups, which in turn have various subgroups.

Glomerulonephritis is not a separate independent disease.

Glomerulonephritis is a group of **immune-mediated** glomerular diseases, heterogeneous in etiology, pathogenesis and prognosis, the common feature of which is the inflammatory proliferation of certain resident cells of the glomerulus.

Clinically, glomerulonephritis manifests itself in the form of any glomerular syndrome:

- acute nephritic syndrome (ANS);
- rapidly progressive nephritic syndrome (RPNS);
- chronic nephritic syndrome (CNS);
- nephrotic syndrome (NS);
- isolated urinary syndrome.

1.1. Etiology of glomerulonephritis

Glomerulonephritis in children often occurs after diseases of a streptococcal nature. The leading role in its occurrence is played by β -hemolytic streptococci of group A of the so-called nephritogenic types (4, 6, 12, 18, 25, 49).

In addition to streptococcal antigens, other bacterial antigens, such as staphylococcal and viral ones, may also play a role.

The development of glomerulonephritis is favored by exposure to cold, trauma, insolation, repeated administration of vaccines, chemicals and medications (penicillin group).

Predisposing factors are:

- family history of infectious and allergic diseases;
- increased family susceptibility to streptococcal infection;
- the child has chronic foci of infection, hypovitaminosis, and helminthiasis.

Since the formation of a particular glomerular syndrome depends more on the severity of the pathological process and the involvement of certain glomerular structures in it than on the specific nosological variant of glomerulonephritis, in modern nephrology an important step in establishing a diagnosis and assessing the prognosis is a morphological study of renal tissue.

Diagnosis formulations such as “chronic glomerulonephritis”, “acute glomerulonephritis”, “rapidly progressive glomerulonephritis”, “nephrotic variant of chronic glomerulonephritis” describe not a disease, but a syndrome.

Therefore, these terms are outdated and should only be used to describe the diagnosis when nephrobiopsy is not possible.

1.2. Epidemiology of glomerulonephritis

Glomerulonephritis affects children of any age, but much more often – children aged 3–12 years.

With glomerulonephritis in children over 10 years old, a chronic process is more often observed, and the patient's body usually turns out to be resistant to glucocorticoid therapy.

1.3. Pathogenesis of glomerulonephritis

There are two main mechanisms in the development of glomerulonephritis – immune complex and autoimmune (fig. 2).

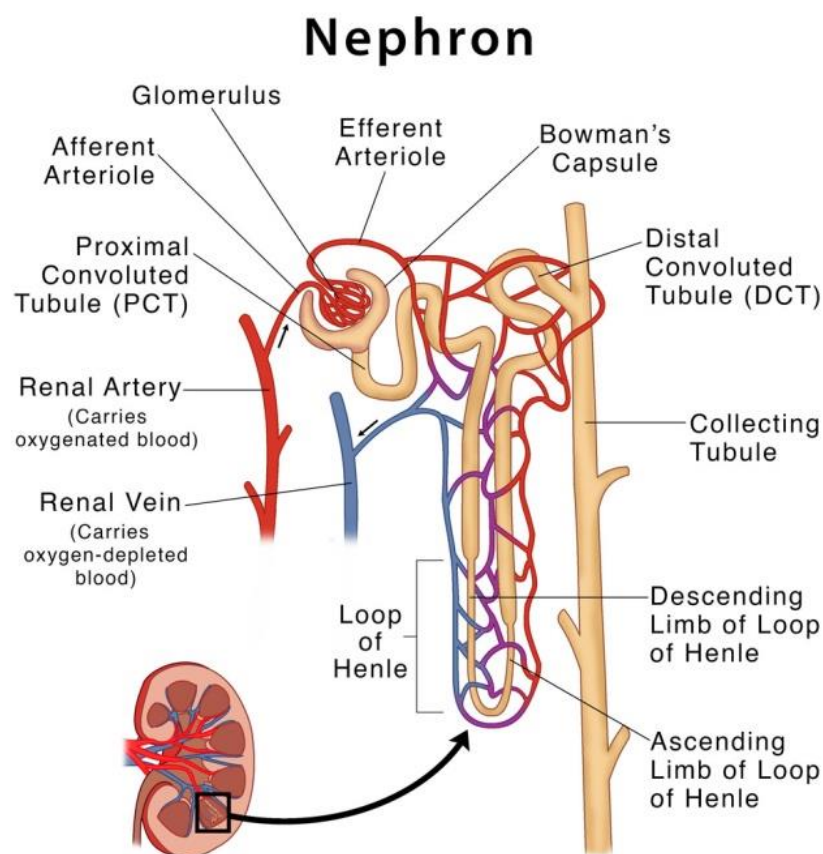


Fig. 2. Pathogenesis (image from the website stylishbag.ru)

In acute post-streptococcal glomerulonephritis, the immunocomplex reaction is of leading importance with the formation of soluble immune complexes in the blood, which activate the complement system, which leads to

damage to the endothelium of the glomerular capillaries with the development of exudative and proliferative processes.

Damage to the endothelium and platelets provokes activation of the hemostatic system and the development of local intravascular coagulation with microthrombosis of the glomerular capillaries.

According to etiopathogenesis, the following variants of glomerulonephritis are distinguished:

- hereditary / genetic;
- idiopathic;
- within systemic diseases;
- secondary (medicinal, associated with infections, paraneoplastic).

In addition, renal amyloidosis and other paraproteinemic lesions belong to the category of glomerular diseases.

1.4. Pathomorphology

Pathomorphology consists of morphological damage to the renal parenchyma.

These may be minimal changes that are observed in all forms of glomerulonephritis, but are most characteristic of nephrotic syndrome.

Focal and/or segmental lesions that occur only in individual glomeruli, occurring in Berger's disease (IgA nephropathy), hemorrhagic vasculitis, etc.

This may affect more than 80 % of the glomeruli in diffuse glomerulonephritis.

1.5. Classification of primary glomerulonephritis in children

International Classification of Diseases 10th Revision:

- Block N00–N08 – Glomerular diseases.
- N00 – Acute nephritic syndrome.
- N01 – Rapidly progressive nephritic syndrome.
- N02 – Recurrent persistent hematuria.
- N03 – Chronic nephritic syndrome.
- N04 – Nephrotic syndrome.
- N05 – Nephritic syndrome, unspecified.

- Additional code .9 – Unspecified change.
- Additional codes .2; .3; .4; .5; .6; .7; .8 are established on the basis of kidney biopsy data and cannot be used on an outpatient basis during primary diagnosis.

Examples of diagnosis formulation:

- (N00.9) Acute nephritic syndrome.
- (N01.9) Rapidly progressive nephritic syndrome.
- (N03.9) Chronic nephritic syndrome, stage 2 CKD (eGFR 79 ml/min/73 m²).
- (N04.9) Nephrotic syndrome, stage 2 CKD (eGFR 79 ml/min/1,73 m²).

Stage of CKD (chronic kidney disease).

According to the flow of the process, they are distinguished:

I. Acute diffuse glomerulonephritis.

According to clinical characteristics, it is divided into two forms:

1. Cyclic form, which has a rapid, acute onset of the disease and, as a rule, a relatively rapid recovery. However, even after a course of treatment, patients experience cyclical bursts of mild proteinuria and hematuria for a very long time.

2. Latent. The latter is often classified as a subacute type of glomerulonephritis. It is characterized by a mild clinical picture.

II. Chronic glomerulonephritis.

Divided according to clinical forms into:

1. Nephritic form – characterized by the fact that the leading syndrome is nephritic with signs of kidney inflammation.

2. Hypertensive form – characterized by a predominance among all syndromes – hypertensive.

3. Mixed form or nephritic-hypertensive. As can be understood from the very name of the clinical form, in this situation there are manifestations of both nephritic and hypertensive forms.

4. Latent form. It has virtually no obvious clinical picture except for mild urinary syndrome. This form of acute nephritis very often develops into a chronic form.

5. Hematuric form, which is manifested only by the presence of hematuria.

III. Rapidly progressive glomerulonephritis.

According to etiology and pathogenesis there are:

1. Primary glomerulonephritis – developing as a result of direct morphological destruction of the kidneys (table 1).

2. Secondary glomerulonephritis, which is a consequence of the underlying disease due to infectious pathology, drug use, malignant neoplasms or systemic diseases (systemic lupus erythematosus, vasculitis).

Table 1

Classification of primary glomerulonephritis in children

Form	Process activity	Kidney function status
Acute glomerulonephritis with:	Period of initial manifestations	Without renal dysfunction
nephritic syndrome	High period	With impaired renal function
with nephrotic syndrome	Period of reverse development	Acute renal failure
with isolated urinary syndrome	Transition to chronic glomerulonephritis	Without renal dysfunction
nephrotic syndrome, hematuria and hypertension	Exacerbation period	With impaired renal function
Chronic glomerulonephritis:	Period of partial remission	Chronic renal failure
nephrotic form	Period of complete clinical and laboratory remission	
hematuric form		
mixed form		
Subacute (malignant) glomerulonephritis		

According to clinical and morphological characteristics they are divided into:

1. *Focal segmental glomerular nephritis*. It is characterized by the identification of sclerotic formations in individual capillary loops. Most often, this type of glomerulonephritis develops as a result of prolonged and / or intensive intravenous drug use or HIV infection. Focal segmental glomerular nephritis manifests itself in the form of nephrotic syndrome or persistent proteinuria, which are usually combined with arterial hypertension and erythrocyturia. The course of the process is quite progressive, and the prognosis is extremely unfavorable. This is the most unfavorable of all morphological variants of glomerulonephritis. Rarely responds to active immunosuppressive treatment.

2. *Membranous glomerulonephritis or the second name is membranous nephropathy*. This type of glomerular nephritis is characterized by the presence

of diffuse thickening in the walls of the glomerular capillaries with their splitting and subsequent doubling. Massive deposits also form on the glomerular basement membrane on the epithelial side of the immune complexes. In a third of patients, it is possible to establish a connection between membranous nephropathy and the hepatitis B virus, certain medications, and malignant tumors. It is manifested by the development of nephrotic syndrome and only 15–30 % of patients have arterial hypertension and hematuria. Membranous glomerulonephritis is most predisposed to men and less so to women; it is interesting that the prognosis for treatment is most favorable in women.

3. *Mesangioproliferative glomerular nephritis* is the most common type of glomerulonephritis that occurs in practical urology. Unlike the above, this type meets absolutely all the criteria for glomerulonephritis of an immunoinflammatory nature. It is characterized by expansion of the mesangium, proliferation of its cells and deposition of immune complexes under the endothelium and in it. The main clinical manifestations of mesangioproliferative glomerular nephritis are hematuria and/or proteinuria. Hypertension and/or nephrotic syndrome develop much less frequently.

4. *Mesangioproliferative glomerular nephritis with the presence of immunoglobulin A in the glomeruli*. More often, this type of glomerulonephritis can be found under the name Berger's disease or IgA nephritis. Young men are most susceptible to the disease. The main symptom is hematuria. And half of the patients experience recurrent gross hematuria. If complications such as nephrotic syndrome or hypertension do not join the process, then the prognosis for treatment is quite favorable.

5. *Mesangiocapillary glomerulonephritis*. This is one of the most unfavorable prognosis for treatment of glomerular nephritis, characterized by strong proliferation of mesangial cells with their spread and penetration into the glomeruli. As a result, the characteristic lobulation of the glomeruli and doubling of the basement membranes are formed, characteristic of this type. Quite often, a connection is found between mesangiocapillary glomerulonephritis and cryoglobulinemia or, more often, with hepatitis C. This type of glomerular nephritis is usually manifested by hematuria and proteinuria. Nephrotic syndrome, hypertension and chronic renal failure also often develop. Mesangiocapillary glomerulonephritis is extremely rarely treatable.

According to pathomorphological characteristics:

1. Proliferative.
2. Non-proliferative (table 2).

Pathomorphological variants of GN

Proliferative	Non-proliferative
Endocapillary diffuse proliferative GN (acute, fixation of IR to the endothelium, manifested by acute nephritic syndrome)	GN with minimal changes in the glomeruli (small podocyte foot disease, manifested by proteinuria)
Mesangioproliferative GN (fixation of IR in the mesangial space, proliferation of mesangial cells)	Membranous GN (immune damage – thickening of capillary walls, loss of protein – leads to slow sclerosis, relapsing course)
Mesangiocapillary GN (fixation of IR between podocytes and membrane)	Focal segmental glomerulosclerosis (hyalinosis)
Extracapillary (crescentic) GN – rapidly progressive – fixation of IR and proliferation of the outer layer of the capsule	
Secondary diffuse proliferative fibroplastic GN (CRF)	

Classification of diseases with nephritic syndrome:

1. Acute post-infectious (post-streptococcal) glomerulonephritis:
 - with cyclic reverse development;
 - prolonged and chronic course.
2. Acute nephritic syndrome in systemic diseases (lupus nephritis, Henoch-Schönlein nephritis, other vasculitis).
3. IgA nephropathy.
4. Rapidly progressive glomerulonephritis.
5. Primary and secondary Membrane-proliferative glomerulonephritis.
6. Shunt nephritis.
7. Hemolytic-uremic syndrome.

Formulation of diagnosis:

1. It is necessary to indicate the process: acute, subacute or chronic.
2. Next – the cause, for example, post-streptococcal.
3. Then the form of GN, syndrome and complication are indicated.

Example: Acute post-streptococcal diffuse proliferative glomerulonephritis. Nephrotic syndrome. Transient renal failure.

1.6. Clinical picture of glomerulonephritis in children

Clinical manifestations

Based on clinical manifestations, there are **four variants** of the disease:

- with nephritic;
- nephrotic;
- isolated urinary syndrome;
- with nephrotic syndrome, hematuria and hypertension.

The clinical picture is characterized by Bright's triad:

1. Urinary syndrome.
2. Edema.
3. Arterial hypertension.

Patient complaints:

- swelling;
- shortness of breath / difficulty breathing;
- increased blood pressure for the first time;
- worsening of pre-existing arterial hypertension;
- lowering blood pressure with previously existing arterial hypertension or previously existing normal blood pressure;
- changes in urine color (color of meat slop / tea leaves);
- decrease in the amount of urine;
- weakness;
- loss of appetite / weight loss;
- increased body temperature;
- joint pain;
- muscle pain;
- bone pain;
- skin rashes;
- dry skin, brittleness and thinning of nails; hair loss;
- pain in the mouth / stomatitis / ulcers on the oral mucosa;
- increase in the size of the tongue;
- hearing loss;
- hoarseness of voice;
- crusts in the nose / recurrent nosebleeds / impaired nasal breathing;
- prolonged cough;
- hemoptysis;

- pain and or dry eyes / redness of the eyes;
- pain in the limbs, numbness of the limbs, impaired skin sensitivity;
- diarrhea, bloating.

During the period of initial manifestations, the general condition is disturbed, weakness, increased fatigue appear, appetite decreases, and febrile fever occurs.

Children complain of headaches, sometimes nausea, vomiting, and a small amount of urine excreted per day.

Urine resembles “meat slop”.

Soon, pale skin, pastiness or swelling of the face and limbs, and dull, aching pain in the lower back appear.

Acute nephritic syndrome is a symptom complex that includes the classic triad: edema, hematuria, hypertension.

Nephritic edema is located in areas of loose connective tissue: periorbital edema is pronounced, noticeable in the morning. The face becomes puffy (fig. 3). Then the swelling spreads to the anterior abdominal wall, to the area of the Michaelis rhombus and sometimes to the external genitalia. In the pathogenesis of nephritic edema, the leading role is played by an increase in vascular and tissue permeability.



Fig. 3. Edema in nephritic syndrome (open data: website mavink.com)

With nephritic syndrome, red blood cells (macrohematuria), a small or moderate amount of protein, short-term hypertension, and often oliguria and azotemia appear in the urine.

Nephrotic syndrome is a clinical and laboratory symptom complex characterized by edema and proteinuria (3,5 g/day or more), hypoalbuminemia (less than 30 g/l), and hyperlipidemia.

With significant proteinuria, edema can become massive, spreading to the trunk and limbs.

Fluid often appears in the pleural, abdominal and pericardial cavities.

In the pathogenesis of nephrotic edema, crucial importance is attached to proteinuria, hypoalbuminemia and a decrease in the oncotic pressure of the blood, the movement of fluid from the intravascular to the interstitial space, and hypovolemia.

Features of edema:

1. Edema – occurs in 60–80 % of patients;
2. Occurs in the first days of illness;
3. First of all – on the face, then spread to the whole body;
4. In severe cases – up to anasarca;
5. Nephritic edema – hot, dense, does not move;
6. Nephrotic edema – cold, soft, shifting.

Arterial hypertension (AH)

Arterial hypertension (occurs in 60–70 % of patients)

The mechanism of arterial hypertension (fig. 4):

– Activation of the RAAS.

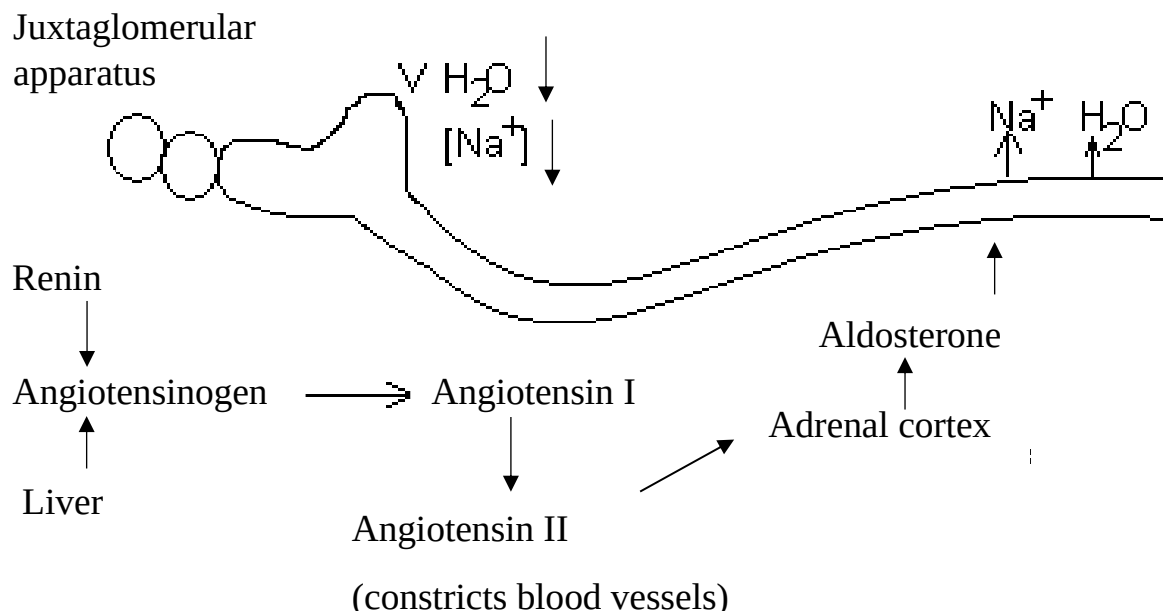


Fig. 4. AH mechanism (image from the website serg-cld.ru)

Arterial hypertension (in 50–80 %)

A uniform increase in systolic and diastolic blood pressure by more than 15–20 mmHg.

Due to this, changes in the fundus of the eye: narrowing of veins, arteries, hyperemia and swelling of the optic nerve nipple, hemorrhages.

Arterial hypertension is often short-term during the period of oliguria.

Features of the clinical picture of acute glomerulonephritis

Acute glomerulonephritis is a syndrome characterized by the sudden appearance of macroscopic hematuria, oliguria, acute renal failure caused by a sharp drop in glomerular filtration, fluid retention and the appearance of edema, hypertension. Proteinuria is less than 3 g/day.

Leading complaints:

- pollakiuria (frequent urination);
- thirst (water-electrolyte imbalance);
- shortness of breath (left ventricular failure begins to form);
- decreased diuresis (severity ranges to oliguria).

Hematuric form (nephritic syndrome)

Etiology: post-streptococcal GN.

Pathogenesis: immune complex GN (type III reactions), with predominant damage to the capillary wall.

Urinary syndrome:

- gross hematuria;
- moderate proteinuria (up to 3 g/l), selective.

Clinical manifestations:

- swelling is small, peripheral, localized mainly on the face;
- marked pallor;
- short-term arterial hypertension during oliguria.

Biochemical blood test:

- slight hypoproteinemia;
- dysproteinemia (increased β -globulins).

Nephrotic form

Etiology: more often – virus-associated GN.

Pathogenesis: most often an immune complex mechanism, with damage to the basement membrane.

Urinary syndrome:

- microhematuria massive proteinuria (more than 3 g/l);
- non-selective;
- Cylindruria.

Clinical manifestations:

- severe peripheral edema;
- cavitary edema (anasarca, hydrocele);
- arterial hypertension during oliguria.

Blood chemistry:

- severe hypoproteinemia;
- hypoalbuminemia < 30 g/l;
- hyperlipidemia.

Mixed form

Etiology: more often – virus-associated GN.

Pathogenesis: combined mechanisms with damage to the basement membrane and capillary wall.

Urinary syndrome:

- gross hematuria;
- massive proteinuria (more than 3 g/l), non-selective;
- Cylindruria.

Clinical manifestations:

- severe peripheral edema (fig. 5).
- cavitary edema (anasarca (fig. 6), hydrocele of cavities)
- persistent arterial hypertension



Fig. 5. Peripheral edema (image from the website mavink.com)



Fig. 6. Anasarca (open data: the website orgma.ru)

Blood chemistry:

- severe hypoproteinemia;
- hypoalbuminemia < 30 g/l;
- hyperlipidemia.

Examples of urine tests:

Patient 5 years old.

- Urine color – s/w;
- Ud. Weight – 1024;
- Protein – 8 g/l;
- Er. – 10–12 in the field of view.
- L – 0–1 in the field of view.

Conclusion: Massive proteinuria, microhematuria. GN, nephrotic form.

Patient 12 years old.

- Urine color – meat slop;
- Ud. Weight – 1020;
- Protein – 2 g/l;
- Er. – 30–32 in the field of view.

Conclusion: Moderate proteinuria, gross hematuria. GN, hematuric form.

Possible outcomes of first exacerbation:

1. Recovery, if after a year the urine test returns to normal. Factors that predetermine it:

- age;
- duration of hospitalization, if short;
- duration and severity of edema and blood pressure, if edema and high blood pressure are quickly eliminated;
- presence of complications, if there were few of them;
- presence of renal failure, if there was none.

2. Clinical recovery after 3–8 months.
3. Morphological recovery up to 3 years.
4. Transition to chronic glomerulonephritis.
5. Fatal outcome is about 1 % (usually rapidly progressive variants of GN).
6. Recovery with a defect (occasional red blood cells and protein in the urine).

To summarize, we can say that AGN consists of **minor syndromes** (urinary, hypertension, edema) and **major syndromes** (nephritic, nephrotic and the formation of renal failure).

Features of the clinical picture of chronic glomerulonephritis

Chronic glomerulonephritis is characterized by a protracted, wave-like, continuously relapsing or latent course with progression, development of sclerotic and fibroplastic changes in the renal tissue.

The diagnosis of chronic glomerulonephritis is established one year after the onset of the disease with continued activity of the process.

According to clinical manifestations, nephrotic, hematuric and mixed forms of chronic glomerulonephritis are distinguished.

Features of the clinical picture of subacute glomerulonephritis

Subacute glomerulonephritis is characterized by a rapid onset, a rapidly progressive, malignant course, difficult to treat and often ending in death 6–18 months from its onset.

In acute glomerulonephritis, there are periods of initial manifestations, peak (2–4 weeks), reverse development of symptoms and recovery (2–3 months), as well as transition to a chronic form.

In chronic glomerulonephritis, the exacerbation period is characterized by high activity and extensive clinical and laboratory symptoms.

During the period of partial remission, clinical manifestations disappear, laboratory parameters and renal function improve.

The period of complete clinical and laboratory remission is characterized by the absence of both clinical and laboratory manifestations of the disease, and its persistence for 5 years or more indicates the child's recovery.

Acute and chronic glomerulonephritis can occur without or with impaired renal function.

Sometimes the course of acute glomerulonephritis may be accompanied by the development of acute renal failure, and chronic glomerulonephritis may be accompanied by the development of chronic renal failure.

Depending on the effect of corticosteroids, hormone-sensitive, hormone-dependent and hormone-resistant forms of the disease are distinguished.

Renal and extrarenal clinical syndromes

1. Renal:

1) isolated microhematuria – red blood cells more than 30 cells/ μ l, more than 10 cells in the visual field (in the absence of urological pathology);

2) recurrent macrohematuria – visible blood in the urine (in the absence of urological pathology);

3) isolated proteinuria – more than 0,3 g/l;

4) isolated urinary syndrome (microhematuria combined with proteinuria less than 3,5 g/l);

5) nephritic syndrome, acute or chronic (edema, arterial hypertension, proteinuria less than 3,5 g/l, gross hematuria);

6) nephrotic syndrome (edema, proteinuria more than 3,5 g/l, hypoproteinemia, hypoalbuminemia +/- hypercholesterolemia) with and without arterial hypertension (see recommendations on nephrotic syndrome);

7) syndrome of rapidly progressive nephritis (proteinuria less than 3,5 g/l, micro- or macrohematuria, rapidly increasing increase in blood creatinine);

8) urinary syndrome in combination with arterial hypertension and a slowly progressive decline in renal function.

2. Pulmonary-renal syndrome:

Rapidly progressive renal failure in combination with severe lung damage (hemoptysis/pulmonary hemorrhage, respiratory failure).

3. Extrarenal clinical syndromes:

1) articular syndrome – arthritis, arthralgia;

2) ossalgia (bone pain);

3) fever of unknown origin;

4) damage to the skin and mucous membranes – urticarial elements (including cold urticaria), erythematous, hemorrhagic, ulcerative-necrotic, photodermatitis, petechiae, ecchymoses;

5) lung damage – repeated “pneumonia”, bronchial asthma;

- 6) eye damage – conjunctivitis, scleritis, episcleritis, iridocyclitis;
- 7) damage to the ENT organs – otitis, sinusitis, nasal / paranasal sinus polyps, tracheal/larynx stenosis;
- 8) polyneuropathy, multiple mononeuritis;
- 9) macroglossia;
- 10) diarrhea.

1.7. Diagnosis of glomerulonephritis in children

Scheme of laboratory and instrumental diagnostics

1. Ultrasound of the kidneys – signs of diffuse changes in the renal tissue.
2. Clinical blood test – leukocytosis, anemia, increased ESR.
3. Assessment of kidney function:
 - urinalysis according to Zimnitsky;
 - used blood (hypoproteinemia, dysproteinemia, hyperlipidemia, increased levels of residual nitrogen, urea, creatinine, CEC, components, complement);
 - Coagulogram (increased fibrinogen);
 - used urine, creatinine clearance;
 - Increased cholinesterase in urine;
 - CBS;
 - Radioisotope renography;
 - Kidney biopsy.

Conducting studies with the introduction of contrast agents (IV urography) is prohibited during the acute period!!

Extrarenal laboratory symptoms:

- anemia +/- thrombocytopenia +/- leukopenia;
- anemia +/- thrombocytosis +/- leukocytosis;
- hypereosinophilia (more than 10 % of the total number of leukocytes).

Diagnostic criteria for nephritic syndrome

Acute nephritic syndrome in typical cases is a manifestation of acute glomerulonephritis, which often develops after an infection caused by β -hemolytic streptococcus or other viral-bacterial infections after 1–4 weeks and often has

a cyclic reverse development. The diagnosis is made on the basis of anamnesis (recent infectious disease), clinical manifestations (edema, arterial hypertension) and diagnostic data (fig. 7).

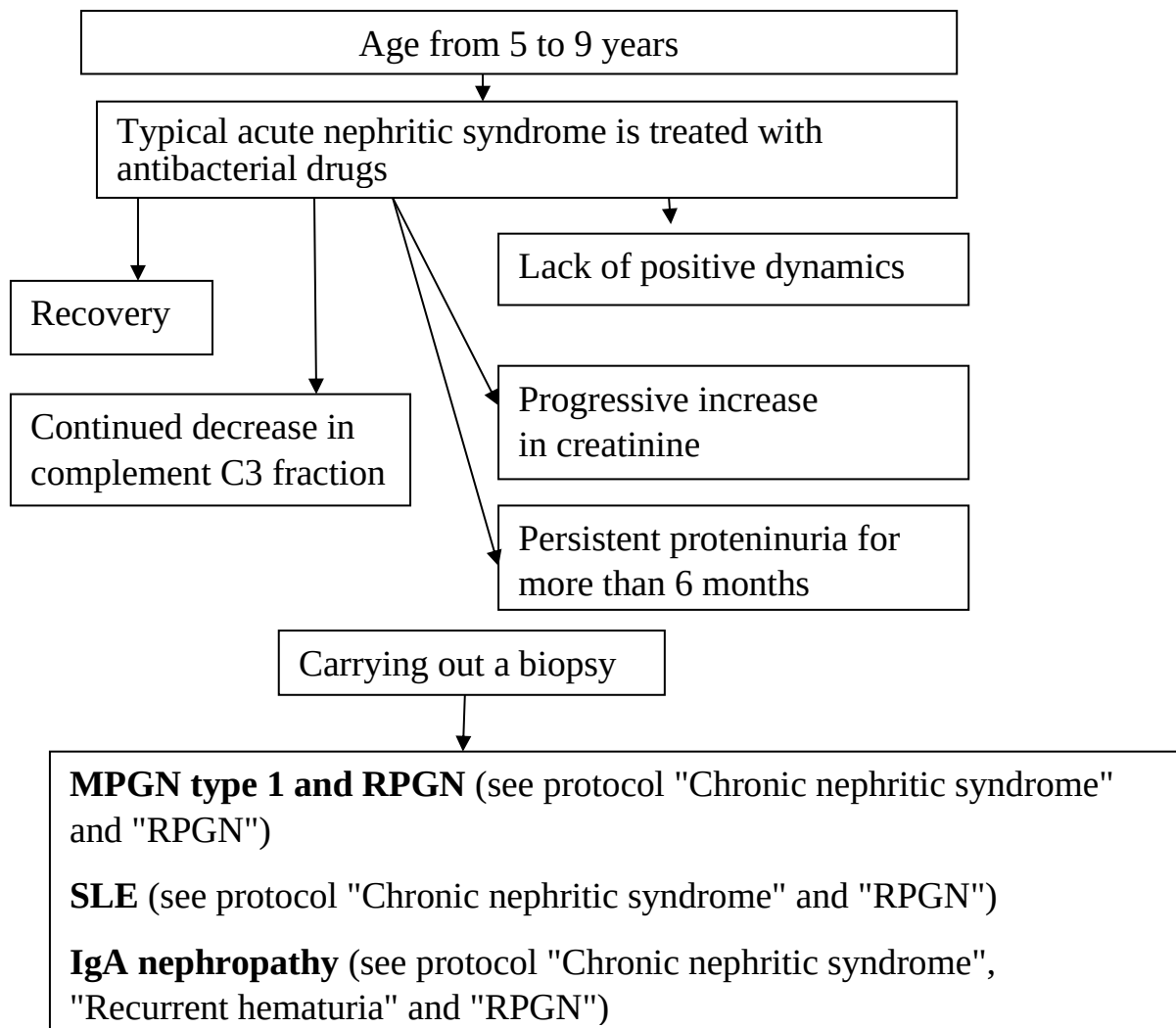


Fig. 7. Diagnosis scheme for acute nephritic syndrome
(image from the website rmg.ru)

Complaints and anamnesis:

- urine is the color of coffee, tea or looks like “meat slop” (macrohematuria).

!Symptoms appear 1 to 4 weeks after a streptococcal or other infection.

- nausea, vomiting, headache;
- moderate short-term arterial hypertension;
- oliguria or anuria.
- rarely left ventricular heart failure (most often hypervolemic) – orthopnea, tachypnea, tachycardia.

On physical examination, dense moderate edema is observed (fig. 8).



Fig. 8. Dense moderate edema
(image from the website serg-cld.ru)

Laboratory diagnosis of glomerulonephritis in children

Clinical blood test: increase in ESR to 20–30 mm/h, mild anemia is possible;

Biochemical blood test: increased blood concentration of creatinine, CRP; the level of total protein and albumin was slightly reduced. Increased ASL-O titer, decreased complement component S3.

!Clinical manifestations of acute nephritic syndrome usually last no more than 2 weeks. Less than 4 % of children with post-streptococcal GN have massive proteinuria (more than 3 g/day). We must remember the possibility of developing crescentic GN with rapidly progressive renal dysfunction. C3 levels usually return to normal 8–10 weeks after infection is detected.

General urine analysis

Urine examination is aimed at identifying glomerular erythrocyturia from macrohematuria (urine the color of “meat slop”) to 10–15 red blood cells in the field of view.

Gross hematuria is caused by thrombosis and rupture of capillary loops, and microhematuria is associated with increased glomerular vascular permeability (fig. 9).

The detection of hyaline and erythrocyte casts in the urinary sediment confirms the glomerular origin of hematuria.

Proteinuria is one of the constant signs of the disease.



Fig. 9. Macrohematuria (image from the website vseproanalizy.ru)

Proteins excreted in the urine are represented mainly by albumin (up to 90 %), seromuroid, α 2-macroglobulins, γ -globulins, etc.

Proteinuria is caused by an increase in the permeability of the basement membrane of the glomerular capillaries for protein molecules, impaired protein reabsorption in the nephron tubules, as well as an increase in catabolic processes in the renal parenchyma.

To verify the morphological variant, it is important to determine the selectivity of proteinuria, which characterizes, first of all, the degree of damage to the glomeruli of the kidneys and the state of glomerular permeability for protein.

Acute glomerulonephritis is characterized by a high degree of selectivity of proteinuria, which is confirmed by studies of the protein spectrum of urine with a predominance of low molecular weight proteins – albumin.

Aseptic leukocyturia may be detected, as indicated by the eosinophilic and lymphocytic nature of leukocytes in the sediment.

Oliguria – a decrease in diuresis by 20–50 % of normal. Occurs due to a decrease in glomerular filtration and an increase in the reabsorption of sodium and water in the initial period of the disease;

Hematuria is observed in most patients and is associated with increased permeability of the basement membranes.

Red blood cells are always changed, leached the color of “meat slop”.

Proteinuria – more than 1 g/day – the leading sign of GN. There are selective and non-selective proteinuria.

The latter is an unfavorable prognostic sign.

Leukocyturia is a variable sign, has an abacterial nature, and is caused by active immune inflammation.

Cylindruria – determined in 60 % of patients. In its structure, it is an inflammatory tubular protein with the inclusion of remnants of formed elements and epithelial cells. Hyaline, erythrocyte, granular cylinders are distinguished.

Instrumental diagnosis of glomerulonephritis in children

– **Ultrasound of the kidneys:** the contours are smooth, the size is within the age norm or slightly increased.

– **ECG** for hypertension reveals left ventricular overload and possible rhythm disturbances.

– **Kidney biopsy:** the following morphological changes may be detected – minor glomerular abnormalities, focal and segmental glomerular lesions, diffuse membranous glomerulonephritis, diffuse mesangial proliferative glomerulonephritis, diffuse endocapillary proliferative glomerulonephritis, diffuse mesangiocapillary glomerulonephritis, dense sediment disease .

!!! Kidney biopsy in acute nephritic syndrome is indicated:

- 1) with a progressive increase in creatinine;
- 2) with persistent proteinuria for more than 6 months;
- 3) with a continued decrease in the C3 fraction of complement.

Indications for consultation with specialists:

- consultation with a rheumatologist, hematologist – if new symptoms or signs of a systemic disease appear;
- consultation with an ophthalmologist – for arterial hypertension;
- consultation with an otolaryngologist – in the presence of an upper respiratory tract infection.

1.8. Differential diagnosis of glomerulonephritis

It is very important to identify a sample of glomerulonephritis, that is, to carry out a differential diagnosis (table 3–5), since the plan and methods of treatment for glomerulonephritis will largely depend on the type of disease.

Table 3

**Differential diagnosis of glomerulonephritis
and pyelonephritis**

Clinical and laboratory signs	Nephritic syndrome	Nephrotic syndrome	Pyelonephritis
Edema	Moderately expressed, unstable	Expressed up to anasarca	There may be pastiness
Hypertension	Up to 150/90 mmHg	Not typical	Not typical
Protein in the blood	Up to 62 g/l	Less than 62 g/l	Within normal limits
Cholesterol	Promoted	Sharply increased	Can be upgraded
Urea, blood creatinine	Promoted	Promoted	Within normal limits
Proteinuria	Up to 3 g/day	More than 3 g/day	Up to 1 g/day
Leukocyturia	Sometimes it may be	Sometimes it may be	Mandatory attribute
Hematuria	Micro-gross hematuria	Absent	Sometimes it may be
Cylindruria	May be	Mandatory	Not permanent
Bacteriuria	Absent	Absent	Mandatory
Intoxication syndrome	May be	May be	Required
Pain syndrome	May be	May be	Required
Dysuric syndrome	Oliguria, anuria	Oliguria, anuria	Mandatory, typical

Table 4

Differential diagnosis of forms of AGN

Option	Hematuria	Proteinuria	Edema	Hypertension
AGN with isolated urinary syndrome	+ (+)	+/-	-	-
AGN with nephritic syndrome	++	1-2 g/day	+	+(+)
AGN with nephrotic syndrome	-	Up to 3 g/day	+++	-
OGN mixed form	+	+	+	+

Table 5

Differential diagnosis of forms of CGN

Form	Hematuria	Proteinuria	Edema	Hypertension
CGN, hematuric form	++	–	–	–
CGN, nephrotic form	–	++	+++	–
CGN, mixed form	+	++	++	+

1.9. Treatment of glomerulonephritis

1. 24-hour hospital for conservative treatment.
2. In the hospital, monitoring of blood pressure, daily diuresis, and laboratory parameters is necessary.
3. In acute nephritic syndrome, patients require antibiotic therapy.
4. Diuretics are also prescribed to relieve edema and reduce blood pressure under the control of daily diuresis.
5. If the course of GN is considered malignant, it is necessary to use immunosuppressive therapy.
6. The patient needs bed rest for a long time.
7. The diet is followed until all extrarenal symptoms disappear and urine analysis sharply improves.
8. The total amount of liquid drunk should not exceed the amount of urine excreted by 200 ml, if there is no edema.
9. The amount of protein is limited at a rate of approximately 1 g per kg of patient weight.
10. The amount of table salt is also limited to 3–5 g per day.

Pathogenetic treatment consists of prescribing immunosuppressive treatment with glucocorticosteroids (GCS).

Indications for this treatment are:

- nephrotic form without increased pressure and hematuria;
- prolonged course of AGN, when proteinuria lasts for more than a month without the development of arterial hypertension and heart failure and if acute renal failure develops;
- the dosage of prednisolone is 1 mg/kg of the patient's weight for 1,5–2 months;

- if high process activity is observed, pulse therapy with methylprednisolone is considered.

Drug therapy

Glucocorticoid therapy regimens:

- Continuous administration at a dose of 1–2 mg/kg per day (single or divided doses) for at least 2 months. Followed by a gradual reduction to a maintenance dose (10–20 mg) for 2 or more months.

- Alternatively, double the dose (usually maintenance) after 1 day.

- Pulse therapy: 0,5–1,0 g of methylprednisolone for 20–40 min after 1 day (total 3–4 g). Contraindications: severe hypertension, cardiomyopathy.

Cytostatics

- Cytostatics are prescribed when nephrotic syndrome develops, resistant to treatment with corticosteroids, when corticosteroids are contraindicated (hypertension, peptic ulcer disease, diabetes mellitus) and if the patient has developed glucocorticosteroid dependence or side effects from their use.

- Treatment with azathioprine (imuran) and mercaptopurine at a dose of 2–3 mg/kg body weight (150–200 mg) per day is considered, cyclophosphamide is prescribed at a dose of 1,5–2 mg/kg body weight (100–150 mg) per day.

- In the acute process, doses should be higher, after which the patient is transferred to maintenance therapy, which is equal to half or a third of the optimal dose. This dosage is maintained for about 4–6 months, and if necessary, longer.

I. Alkylating agents: disrupt cell division and protein synthesis.

- CYCLOPHOSPHAMIDE

per os 2,0–2,5 mg/kg/day;

pulse therapy IV 15 mg/kg;

for CF < 30 ml/min – 10 mg/kg (course of treatment 6 g).

- CHLOROBUTINE per os 0,1–0,2 mg/kg

II. Antimetabolites: inhibit enzymes involved in DNA synthesis.

- AZATHIOPRINE per os 1–3 mg/kg

Anticoagulants

Heparin is prescribed if the patient has developed a nephrotic form of GN, acute renal failure has begun, and DIC has developed in the hypercoagulable stage.

Heparin is injected under the skin of the abdomen at 5,000–10,000 units every 4–6 hours. The daily dose of heparin is 10,000–40,000 units.

The dose is considered adequate, if 5 hours after subcutaneous administration of heparin, the blood clotting time increases by 2–4 times compared to the initial one, and the thrombin time increases by 2 times.

The course of treatment with heparin is usually 6–8 weeks, if necessary – up to 3–4 months.

At the end of the course of treatment, to continue anticoagulant therapy, direct-acting anticoagulants can be prescribed for 12 months in a dose sufficient to maintain the prothrombin index at 45–60 %. Due to the slow development of the anticoagulant effect, direct anticoagulants should be prescribed 1–2 days before the end of heparin treatment. Heparin is discontinued by slowly reducing doses in the last week of treatment.

Antiplatelet agents

Curantil is prescribed for any type of GN, except for hematuria, at a dose of up to 400 mg per day for a period of 2 to 8 weeks.

Then the dose is reduced, and the patient is transferred to a maintenance dose of 50–75 mg per day for up to a year or more.

NSAIDs

While taking NSAIDs, a decrease in glomerular filtration is possible, so their indications for acute hypertension are **sharply limited!!!**

This is important to remember if the patient is forced to use this group of drugs for another disease (if the patient has prolonged proteinuria in the absence of increased blood pressure, edema, oliguria and pronounced activity of the inflammatory process).

Symptomatic treatment:

Symptomatic treatment of arterial hypertension involves the use of almost all groups of drugs that are not contraindicated.

Edema syndrome is treated with diet and fluid restriction; fasting days should be carried out.

If a pronounced edematous syndrome is observed, the use of drugs such as furosemide together with veroshpiron is considered, and hypothiazide is prescribed before the development of renal failure.

Important!

Detect the development of renal failure in a timely manner!

Markers:

- increase in urea above 8,5 mmol/l, creatinine above 110 µmol/l.

Additionally:

- radioisotope renography;
- kidney biopsy.

Treatment of complications of AGN

When treating acute renal failure, it is necessary to limit fluid intake. The diet includes restriction of potassium and protein.

It is important to exclude salt from the diet (in case of acute renal failure, salt is limited to 5 g per day; in case of acute renal failure, salt is excluded altogether).

Large doses of diuretics, heparin, and prednisolone are administered.

If these measures are ineffective, transfer of the patient to hemodialysis is considered.

New agents that correct the immune response:

- mycophenolate mofetil;
- rituximab;
- cyclosporine;
- tacrolimus.

Selective immunosuppressants:

- **Cyclosporine.** Suppresses the activity of T helper cells (CD4+), the production of IL-2, and cytotoxic T cells.

- **Tacrolimus.** Inhibits calcineurin, which leads to suppression of the activity of cytotoxic T cells.

- **Mycophenolate mofetil.** Inosine monophosphate dehydrogenase inhibitor, inhibits the proliferation of T and B lymphocytes.

1.10. Clinical observation

Clinical observation after acute nephritis

Children – 5 years old. For 2 years with a doctor's examination every month for the first six months, and once every 3 months for the next year and a half.

Includes: systematic monitoring of blood pressure, quarterly monitoring of urine and blood tests, creatinine, total protein and blood cholesterol.

Changes in urine tests can last up to 1,5–2 years.

Medical examination of children with CGN

Clinical examination is carried out by a nephrologist and / or pediatrician at the clinic.

Medical examination in the first year after the first attack or last exacerbation:

- First 3 months: preventive examinations – once every 2 weeks.
- Until the end of the year – monthly.
- Each examination – measuring blood pressure and checking for edema.
- Assessment of complications of therapy.
- Resolving issues related to the sanitation of foci of infection.

Laboratory research in the first year:

- Complete blood count – monthly (with cytostatic therapy – 2 times a month) with a study of platelets and reticulocytes.
- Biochemical blood test (total protein, protein fractions, creatinine, urea, cholesterol, β -lipoproteins, glucose, transaminases, bilirubin) – once every 3 months.
- Children receiving cytostatic therapy for a long time have their blood tested for opportunistic infections.
- General urine analysis – monthly and during intercurrent conditions (daily).
- Urine culture for flora – according to indications.
- Zimnitsky test – monthly.

Clinical examination during the period of clinical and laboratory remission:

- Examinations 2 times a year with assessment of physical development, determination of body mass index, assessment of sexual development.
- Annual examination in a specialized hospital.
- Spa treatment.

Preventive vaccinations:

- In the acute period, vaccination according to epidemic indications.
- During the period of remission, vaccination according to an individual schedule: polio vaccine – 2 years after complete remission; other vaccines – after 5 years.

In case of intercurrent infections:

- Bed rest.
- Antibiotic taking into account the nature of the infection for 7–10 days, desensitizing drugs.
- COP 3–5 days.
- Monitoring urine tests – **daily!**

2. ACUTE RENAL FAILURE

Potentially reversible, rapid (hours / days) **cessation of renal excretory function** due to damage to the parenchyma with retention in the blood of products normally removed from the body in the urine.

Frequency 40:1 000 000.

Acute renal failure:

Prerenal:

- BCC disorders (shock, blood loss, exicosis).

Renal:

- Damage to parenchyma.

Postrenal:

- Obstruction of urine flow.

OPN Clinic

- Initial phase;

- Oligoanuric phase (9–11 days).

- A sharp decrease in diuresis – up to 300 ml/day – oliguria, up to 50 ml/day – anuria.

- Increased levels of urea, creatinine.

- Metabolic acidosis.

- Early phase of the recovery period (5–10 days).

- Initial diuresis (up to 400 ml/day).

- Polyuric phase (more than 2000 ml/day).

- Complete recovery of AF (6–12 months).

Treatment of acute renal failure:

- correction of bcc;

- diuretics in combination with cardiotonic drugs (furosemide 30–50 mg/kg/h + dopamine 3 mcg/kg/min);

- antibacterial therapy;

- diet;

- Hemodialysis (**indications:** hyperkalemia above 7 mmol/l, urea above 24 mmol/l).

3. CHRONIC RENAL FAILURE

A symptom complex that develops as a result of the gradual death of nephrons with the progression of chronic renal pathology

Reasons:

- CGN, chronic renal failure, polycystic disease, collagenosis, etc.;
- SKV, JRA, SD;
- Drug-induced nephropathy, hyperparathyroidism, intoxication;
- Urological diseases (USD, tumors).

Classification of chronic renal failure

Stage	Characteristic
Latent	GFR < 50–60 ml/min Creatinine, urea – normal Polyuria (decreased tubular reabsorption)
Compensated	GFR = 30 ml/min Creatinine, urea – normal Polyuria
Intermittent	GFR < 25 ml/min Creatinine and urea are increased Metabolic acidosis
Terminal	GFR < 15 ml/min Uremia (urea > 25 µmol/l) Metabolic acidosis Hyperkalemia, hypernatremia

Chronic renal failure clinic

Complaints:

- decreased appetite;
- fatigue;
- nausea, vomiting;
- headache, hypertension;
- edema.

Upon inspection:

- decrease in body m, atrophy of the pancreas and muscles;
- icteric skin and sclera;
- dry skin, scratching, hemorrhages;
- smell of ammonia from the mouth.

CGN Clinic

Cardiovascular system:

- AH, Myocardial hypertrophy;
- tachicardia, accent of the second tone over the aorta, Pericardial friction rub (uremic pericarditis).

Respiratory system:

- shortness of breath, cough;
- effusion in the pleural cavity;
- pulmonary edema.

Gastrointestinal tract:

- Dyspepsia, Bloating, constipation, Ulcerative defects of the mucous membranes.

Central Nervous System (CNS):

- uremic encephalopathy, polyneuropathy, coma.

Musculoskeletal system:

- Osteodystrophy;
- Osteoporosis.

Principles of therapy

- treatment of the underlying disease;
- bed rest;
- diet;
- monitoring fluid intake;
- correction of electrolyte disturbances;
- correction of azotemia;
- treatment of anemia;
- blood pressure correction;
- treatment of osteodystrophy;
- treatment of concomitant pathology;
- DIALYSIS!

TEST TASKS

1. THE CLASSIC TRIAD OF NEPHRITIC SYNDROME INCLUDES:

- 1) hypertension;
- 2) peripheral edema;
- 3) hyperlipidemia;
- 4) massive swelling, up to anasarca;
- 5) hematuria.

2. FEATURES OF NEPHRITIC EDEMA:

- 1) cold, soft, shifting;
- 2) hot, dense, do not move;
- 3) hot, soft, shifting.

3. HEMATURIA IN NEPHROTIC SYNDROME:

- 1) absent;
- 2) sometimes it can be;
- 3) present.

4. KIDNEY BIOPSY IN ACUTE NEPHRITIC SYNDROME IS INDICATED:

- 1) with a progressive increase in creatinine, with persistent proteinuria for more than 6 months, with a persistent decrease in the C3 complement fraction;
- 2) with persistent proteinuria for more than 2 months, with a persistent decrease in the C3 complement fraction;
- 3) with a progressive increase in creatinine.

5. HOW LONG AFTER THE ONset OF THE DISEASE CAN THE DIAGNOSIS OF CHRONIC GLOMERULONEPHRITIS BE ESTABLISHED:

- 1) 3 months;
- 2) 6 months;
- 3) 1 year;
- 4) 5 years.

6. WHAT SYNDROME IS CHARACTERISTIC FOR ARTERIAL HYPERTENSION?

- 1) nephritic syndrome;
- 2) nephrotic syndrome;
- 3) pyelonephritis.

7. WHAT INDICATORS ARE CHARACTERISTIC FOR A MIXED FORM OF FIRE?

- 1) hematuria;
- 2) proteinuria;
- 3) edema;
- 4) arterial hypertension.

8. WHAT INDICATORS ARE CHARACTERISTIC FOR FIRE WITH NEPHROTIC SYNDROME?

- 1) hematuria;
- 2) proteinuria;
- 3) edema;
- 4) arterial hypertension.

9. GLOMERULONEPHRITIS IS

- 1) a disease with an immune-mediated mechanism of damage to the glomerulus;
- 2) disease caused by hereditary changes in the structures of the glomerulus;
- 3) any nephropathies with initial damage to the glomerulus.

10. URINARY SYNDROME IN GLOMERULONEPHRITIS IS CHARACTERISTIC:

- 1) Leukocyturia;
- 2) Glucosuria;
- 3) Proteinuria;
- 4) Cylindriuria;
- 5) Erythrocyturia.

11. CHRONIC GLOMERULONEPHRITIS IS MORE COMMON IN CHILDREN OF THIS AGE:

- 1) at 3 years old;
- 2) at 7–8 years old;
- 3) over 10 years old.

12. IN DIFFUSE GLOMERULONEPHRITIS, WHAT PERCENTAGE OF GLULULAR FILTRATION IS AFFECTED?

- 1) 50–60 %;
- 2) 60–70 %;
- 3) more than 80 %.

13. WHAT IS MOST CHARACTERISTIC OF THE LATENT FORM OF CHRONIC GLOMERULONEPHRITIS:

- 1) the leading syndrome is nephritic with signs of kidney inflammation;
- 2) the predominance among all syndromes is hypertensive;
- 3) presence of manifestations of nephritic and hypertensive forms;
- 4) has virtually no obvious clinical picture except for mild urinary syndrome. This form of acute nephritis very often develops into a chronic form.

14. WHAT SHOULD BE INCLUDED IN THE THERAPEUTIC DIET FOR ACUTE GLOMERULONEPHRITIS:

- 1) salt-free diet;
- 2) limiting protein in the diet;
- 3) diet with sufficient calorie content;
- 4) high protein diet due to loss of protein in urine;
- 5) age-appropriate nutrition without restrictions.

15. WHAT MECHANISM IS THE BASIS OF THE DEVELOPMENT OF GLOMERULONEPHRITIS IN CHILDREN:

- 1) immune reactions;
- 2) physical inactivity;
- 3) hormonal background;
- 4) stress factor.

16. WHICH KIDNEY STRUCTURE IS MOST IMPACTED BY ANTIGEN-ANTIBODY IMMUNE COMPLEXES IN GLOMERULONEPHRITIS IN CHILDREN:

- 1) Loop of Henle;
- 2) tubules;
- 3) glomeruli;
- 4) calyces.

17. WHICH OF THE LISTED STUDIES ALLOWS THE EVALUATION OF RENAL CONCENTRATION FUNCTION?

- 1) urine analysis according to Nechiporenko;
- 2) determination of blood urea;
- 3) Zimnitsky test;
- 4) definition of CBS.

18. GLOMERULAR FILTRATION CAN BE JUDGED BY:

- 1) Zemnitsky test;
- 2) blood protein formula;
- 3) Rehberg test;
- 4) the amount of residual nitrogen;
- 5) Amburger's test.

19. INDICATIONS FOR CONSULTING AN OPHTHALMOLOGIST FOR GLOMERULONEPHRITIS IS:

- 1) infectious eye diseases;
- 2) deterioration of the patient's general condition;
- 3) arterial hypertension.

20. WHAT IS THE LEADING INDICATOR IN OAM IN GBV?

- 1) proteinuria;
- 2) hematuria;
- 3) oligouria;
- 4) leukocyturia.

21. INDICATIONS FOR PRESCRIPTION OF CYTOSTATICS ARE:

- 1) contraindications to treatment with GCS (hypertension, peptic ulcer, diabetes mellitus);
- 2) development of nephrotic syndrome resistant to treatment with corticosteroids;
- 3) isolated urinary syndrome;
- 4) glucocorticosteroid dependence;
- 5) development of nephritic syndrome.

22. SUBACUTE GLOMERULONEPHRITIS IS:

- 1) a disease that develops as a result of infectious pathology or drug use; for malignant neoplasms or systemic diseases;
- 2) a disease with a rapid onset, a rapidly progressive, malignant course, difficult to treat and often ending in death 6–18 months from its onset;
- 3) protracted, wavy, continuously relapsing disease, with the development of sclerotic and fibroplastic changes in the renal tissue.

23. A 6-YEAR-OLD BOY WAS ARRIVED TO A CHILDREN'S HOSPITAL WITH COMPLAINTS OF PROGRESSIVE SWELLING AROUND THE EYES, WHICH SPREADED TO THE LEGS. URINE IS POSITIVE FOR NEPHROTICA PROTEINURIA BUT NOT HEMATURIA. SERUM ALBUMIN IS LOW. HE HAS BEEN DIAGNOSED WITH NEPHROTIC SYNDROME. WHICH OF THE FOLLOWING WOULD NOT BE PART OF HIS INITIAL TREATMENT PLAN?

- 1) limiting salt;
- 2) diuretics
- 3) bed rest;
- 4) low fat diet;
- 5) corticosteroids.

24. A 5-YEAR-OLD GIRL WAS ADMISSITED WITH PERIORBITAL EDEMA AND PROTEINURIA. HER MOTHER REPORTS THAT SHE HAD A COLD LAST WEEK. ON EXAMINATION YOU NOTE A PULSE RATE OF 70 BPM AND A BLOOD PRESSURE OF 95/53. WHAT PROTEIN(S) WILL BE LIKELY TO BE FOUND IN HIGH QUANTITIES IN THIS PATIENT'S URINE?

- 1) IgG;
- 2) IgA;
- 3) Albumin;
- 4) Albumin and IgG;
- 5) Albumin, IgG and IgA.

25. 11-YEAR-OLD BOY WITH EDEMA AND PROTEINURIA EXCEEDING 3.5 G IN 24 HOURS. YOU SUSPECT THAT THIS PATIENT HAS EXPERIENCED A LOSS OF POLYANIONS THROUGH THE GLULULAR BASAL MEMBRANE. WHICH OF THE FOLLOWING RESULTS WILL CONFIRM YOUR DIAGNOSIS?

- 1) leukocytes in urine;
- 2) red blood cells in urine;
- 3) selective albuminuria;
- 4) crystals with negative birefringence in urine.

26. WHAT ARE THE MAIN MECHANISMS IN THE DEVELOPMENT OF GLOMERULONEPHRITIS?

- 1) immunocomplementary and antigenic;
- 2) immune complex and autoimmune.

27. CONSOLIDATED FACTORS FOR THE DEVELOPMENT OF GLOMERULONEPHRITIS INCLUDE:

- 1) exposure to cold;
- 2) insolation;
- 3) reintroduction of vaccines and medications;
- 4) viruses, bacteria;
- 5) everything is correct except 4;
- 6) all answers are correct.

28. PRESISTING FACTORS OF GLOMERULONEPHRITIS INCLUDE:

- 1) the presence of foci of infection in the child;
- 2) hypovitaminosis;
- 3) helminthiasis;
- 4) all answers are correct;
- 5) all are correct except 3.

29. SECONDARY CAN BE CALLED NEPHRITIS DEVELOPED IN A PATIENT WITH:

- 1) diffuse connective tissue disease;
- 2) subacute bacterial endocarditis;
- 3) systemic vasculitis;
- 4) congenital anomaly of the genitourinary system.

30. MEMBRANOSE NEPHROPATHY IS CHARACTERIZED BY:

- 1) detection of sclerotic formations in individual capillary loops;
- 2) the presence of diffuse thickening in the walls of the glomerular capillaries with their splitting and subsequent doubling.

31. DURING THE PERIOD OF COMPLETE CLINICAL AND LABARATORY REMISSION IS CHARACTERIZED BY:

- 1) absence of clinical and laboratory manifestations of the disease;
- 2) the absence of only laboratory manifestations of the disease;
- 3) the absence of only clinical manifestations.

32. HEPARIN IS USED IN CASES:

- 1) nephrotic form of GN;
- 2) nephritic form of GN;
- 3) acute renal failure;
- 4) DIC syndrome in the hypercoagulation stage.

33. FOR WHAT FORM OF GN IS CURANTIL (ANTIAGREGANT) NOT PRESCRIBED?

- 1) hematuric;
- 2) nephrotic;
- 3) all of the above;
- 4) none of the above.

34. THE DIAGNOSIS IS MADE PRIMARILY ON THE BASIS OF:

- 1) data from immunofluorescent study of kidney biopsy;
- 2) clinical picture (recurrent gross hematuria);
- 3) increased level of Ig-A in blood serum.

35. IN THE TREATMENT OF IG-A NEPHRITIS GLUCOCORTICOSTEROIDS AND CYTOSTATICS:

- 1) used for severe proteinuria;
- 2) never used;
- 3) always used.

36. ACUTE GLOMERULONEPHRITIS IS MORE COMMON AT AGE:

- 1) from 1 year to 3 years;
- 2) 4–5 years of life;
- 3) 6–7 years of life;
- 4) 8–9 years of life;
- 5) over 10–12 years old.

37. WHICH OF THE FOLLOWING RENAL SYMPTOMS IS MANDATORY IN THE CLINIC OF ACUTE GLOMERULONEPHRITIS:

- 1) abdominal pain;
- 2) oliguria;
- 3) hematuria;
- 4) pain in the lumbar region;
- 5) severe proteinuria.

38. THE MAIN GROUP OF DRUGS FOR THE TREATMENT OF THE NEPHROTIC FORM OF CHRONIC GLOMERULONEPHRITIS ARE:

- 1) anticoagulants;
- 2) diuretics;
- 3) glucocorticosteroids;
- 4) antibiotics.

39. WHAT DRUG IS USED TO PREVENT COMPLICATIONS OF NEPHRITIC FORM OF GLOMERULONEPHRITIS?

- 1) Josamycin;
- 2) Ceftriaxone;
- 3) Cefazolin;
- 4) Tetracycline.

40. MAIN SYMPTOM CHARACTERISTIC OF ALPORT SYNDROME:

- 1) pain in the right iliac region;
- 2) hearing loss;
- 3) stomatitis;
- 4) arthralgia.

41. PROTEINURIA SHOULD BE TALKED ABOUT THE AMOUNT OF PROTEIN IN THE URINE:

- 1) traces;
- 2) less than 0,03 g/l;
- 3) more than 0,03 g/l;
- 4) more than 0,06 g/l;
- 5) more than 0,09 g/l.

42. TERMINAL HEMATURIA OCCURS IN ALL OF THE LISTED DISEASES EXCEPT:

- 1) stones or tumors of the bladder neck;
- 2) varicose veins in the bladder neck area;
- 3) chronic glomerulonephritis;
- 4) colliculitis;
- 5) acute cystitis.

43. IN CHILDREN OF EARLY AND PRESCHOOL AGE, ACUTE GLOMERULONEPHRITIS IS MORE OFTEN:

- 1) with hypertension syndrome;
- 2) visually impaired;
- 3) with changes in the fundus;
- 4) with nephrotic syndrome;
- 5) with severe isolated hematuria.

44. WHICH OF THE FOLLOWING SYMPTOMS IN ACUTE GLOMERULONEPHRITIS IS NOT EXTRARENAL:

- 1) symptoms of intoxication;
- 2) edema syndrome;
- 3) changes in urine color – redness;
- 4) Hypertension syndrome;
- 5) changes in the fundus.

45. WHAT BLOOD STUDIES REFLECT THE ACTIVITY OF THE INFLAMMATORY PROCESS IN THE RENAL TISSUE:

- 1) proteinogram;
- 2) seromucoid;
- 3) C-reactive protein;
- 4) blood electrolytes;
- 5) blood fibrinogen.

46. LABORATORY INDICATORS OF ACTIVITY OF ACUTE GLOMERULONEPHRITIS WITH NEPHRITIC SYNDROME ARE:

- 1) acceleration of ESR;
- 2) hyperfibrinogenemia;
- 3) gross hematuria;
- 4) proteinuria up to 1 g/l;
- 5) all of the above.

47. IN NEPHROTIC SYNDROME EDEMA:

- 1) common;
- 2) anasarca;
- 3) facial swelling;
- 4) swelling is loose, soft, asymmetrical, mobile;
- 5) swelling “dense”.

48. WHAT IS NOT CHARACTERISTIC FOR ACUTE GLOMERULO-NEPHRITIS?

- 1) urinary syndrome;
- 2) damage to target organs in the presence of hypertension;
- 3) reversible decrease in glomerular filtration;
- 4) positive Pasternatsky symptom.

49. WHAT COMPLICATIONS CAN OCCUR IN ACUTE GLOMERULONEPHRITIS?

- 1) surge arrester;
- 2) hemorrhage in the brain;
- 3) nephrotic encephalopathy;
- 4) uremia;
- 5) acute heart failure.

50. IN WHAT TIME AFTER AN INFECTION DOES ACUTE GLOMERULONEPHRITIS DEVELOP?

- 1) 10–12 days;
- 2) 3–4 days;
- 3) 7 days;
- 4) 30 days;
- 5) 2 months.

ANSWERS TO TEST TASKS

Question	Answer	Question	Answer
1	1, 2, 5	26	2
2	2	27	5
3	1	28	4
4	1	29	1, 2, 3
5	3	30	2
6	1	31	1
7	1, 2, 3, 4	32	1, 3, 4
8	2, 3, 4	33	1
9	1	34	1
10	1, 3, 4, 5	35	1
11	3	36	5
12	3	37	3
13	4	38	3
14	1, 2, 3	39	1
15	1	40	2
16	3	41	3
17	3	42	3
18	3	43	3
19	3	44	3
20	1	45	1, 2, 3, 5
21	1, 2, 4	46	5
22	2	47	1, 2, 4
23	3	48	2, 4
24	3	49	1, 2, 3, 4, 5
25	3	50	1

SITUATIONAL TASKS

Problem № 1.

An 8-year-old child and his mother contacted their local pediatrician with complaints of general malaise, a feeling of weakness, swelling of the eyelids, facial swelling in the morning, dull, aching, prolonged pain of low intensity in the lumbar region, and a change in the color of urine to dark brown.

The child suffered from a sore throat a month ago. After recovery, during one of the walks the child was caught in the rain and was cold. After a few days, he began to urinate less frequently and little by little. The appetite decreased, the child turned pale. Swelling appeared on the face.

The child grew and developed in accordance with his age. Family history: mother has hypertension, father has chronic glomerulonephritis.

On examination: the condition is of moderate severity. Body temperature 36,8 °C. The face is pale, puffy, the eyelids are swollen, the eyes are narrowed. The skin and mucous membranes are pale, clean, moist. BH – 20 per min.

Heart sounds are muffled, there are no murmurs, heart rate is 70 per min. Blood pressure – 130/80 mm Hg.

The abdomen is soft and painless. The lower edge of the liver is palpated at the edge of the costal arch, painless.

The kidney area is not changed, the kidneys are not palpable, the tapping symptom is weakly positive on both sides.

In the general analysis of urine: color – “meat slop”, relative density – 1018, protein – 3,066 g/l, erythrocytes – 22–24 per field of view, leukocytes – 1–2 per field of view.

Analysis for antistreptolysin – O: 621,1 IU/ml.

Task: formulate a clinical diagnosis.

Answer: Diagnosis: acute post-streptococcal glomerulonephritis. Nephritic syndrome. The form of the disease and the nature of complications (acute renal failure?) need clarification.

Problem 2.

A 12-year-old boy was hospitalized in the nephrology department of a children's hospital with complaints of weakness, nausea, vomiting, and pain in the lower abdomen.

Considers himself sick for 2 weeks after suffering from ARVI. On the last day before hospitalization, the child urinated only once a day. His appetite decreased and he lost 2 kg.

On admission: Body temperature 37,2 °C, puffy face, massive swelling of the face, torso, and lower extremities.

On examination: the skin and mucous membranes are pale, dry, interic sclera. BH – 20 per min. Heart sounds are muffled, the accent of the second tone is over the aorta, heart rate is 82 per minute. Blood pressure – 150/105 mm Hg. Art. The abdomen is soft, painful, and bloated. The lower edge of the liver is palpated at the edge of the costal arch, painless. The kidney area is not changed, the kidneys are not palpable, the tapping symptom is positive on both sides.

Performed: Clinical blood test – no changes; biochemical blood test: urea 14,4 mmol/l, creatinine 112 mmol/l, total cholesterol – 5,3 mmol/l, total protein – 60 mmol/l, albumin – 24 g/l. GFR <25 ml/min; General urine analysis – relative density 1024, protein 4 g/l, erythrocytes 0–1 in the field of view, leukocytes – 0–1 in the field of view.

Task: formulate a clinical diagnosis.

Answer: Chronic glomerulonephritis, nephrotic form, CRF, intermittent stage.

Problem 3.

A 13-year-old girl was admitted to the nephrology department with complaints of weakness, nausea, vomiting, pain in the lower abdomen, and the appearance of brown urine when urinating. Considers himself sick for 2 weeks after suffering a respiratory illness. The local pediatrician began treatment for cystitis. During outpatient treatment, lethargy and nausea increased. Urine tests show protein 0,099 %, red blood cells 15–20 per field of view. The girl was hospitalized in a specialized department.

Upon admission and subsequently, the condition was of moderate severity. There is slight pastiness of the eyelids, blood pressure is 150/105 mm Hg. Art. Decreased diuresis. Clinical blood test: ESR 24 mm/h. Biochemical blood test: urea 14,4 mmol/l, creatinine 73 mmol/l, gamma globulins 22,1 %. In urine tests: relative density 1003–1012, protein 0,66 %, red blood cells 25–30 in visual field.

In the hospital, with a targeted history taking, it was possible to find out that at the age of 8 the girl suffered from scarlet fever, against which traces of protein, gross hematuria, and leukocyturia were found. Subsequent urine tests were not monitored.

In the hospital, as the disease progressed, elements of a maculopapular and hemorrhagic rash began to appear on the girl's face, skin in the torso and limbs, which after a few days began to gradually fade and disappeared without a trace.

Task: make a diagnosis according to the classification.

Answer: Chronic glomerulonephritis, hematuric form, period of exacerbation, with impaired renal function.

Problem 4.

Patient A., 18 years old, a student, consulted a doctor at the emergency department with complaints of general malaise, weakness, pain in the lumbar region on both sides, severe constant headache, and reddish urine. Urination is painless. He considers himself sick for about 3 weeks: after severe hypothermia, the body temperature increased to 38,0 °C, and pain in the throat appeared when swallowing. She went to the clinic at her place of residence, where lacunar tonsillitis was diagnosed and antibacterial therapy was prescribed. On the seventh day, the symptoms of sore throat were relieved, but general weakness remained. Five days ago, against the background of increased fatigue, aching pain in the lumbar region, headache, and an increase in body temperature to 37,5 °C appeared. Two days ago the amount of urine decreased and became reddish in color.

On examination: condition of moderate severity, body temperature 37 °C. Height – 158 cm, weight – 72 kg.

The skin and visible mucous membranes are pale, clean, of normal moisture. The face is puffy, there is dense swelling on the upper and lower extremities, the skin above them is warm and pale. Peripheral lymph nodes are not enlarged. The chest is normosthenic, symmetrical, and evenly participates in breathing. BH – 22 per min.

Percussion above the lungs there is a clear pulmonary sound. Breathing is vesicular, no wheezing. The pulse is rhythmic, 98 per min, blood pressure – 160/100 mm Hg. Art. The apex beat is not visually or palpably determined. The boundaries of relative cardiac dullness: right – along the right edge of the sternum, upper – the lower edge of the third rib, left – 1 cm inward from midclavicular line. Auscultation: heart sounds are dull, rhythmic, heart rate – 78 per min. The abdomen is symmetrical, soft, painful in the projection of the kidneys. The dimensions of the liver according to Kurlov are 9×8×7 cm. Palpation of the right hypochondrium is painless, the edge of the liver is not palpable. The effleurage symptom is positive on both sides.

In analyses.

Clinical blood test: hemoglobin – 105 g/l, red blood cells – $3,2 \times 10^{12}/l$, color index – 0,9; platelets – $270 \times 10^9/l$, leukocytes – $10,7 \times 10^9/l$, erythrocytes – 4 %, band cells – 5 %, neutrophils – 9 %, segmented neutrophils – 70 %, lymphocytes – 11 %, monocytes – 6 %. ESR – 23 mm/h.

General urine analysis: “meat slop” color, cloudy, pH – acidic, specific gravity – 1008; protein – 3,5 g/l, sugar – none, leukocytes – 1–2 in the field of

view, renal epithelium – 12–20 in the field vision, red blood cells – a large number, cylinders: hyaline – 10–12, granular – 6–8 in the field of view, salts – no.

Daily proteinuria – 7,3 g/l, daily diuresis – 650 ml.

Biochemical blood test: total bilirubin – 12,4 $\mu\text{mol/l}$, direct – 3,2, indirect – 9,2 $\mu\text{mol/l}$, creatinine – 0,96 mmol/l, glucose – 4,3 mmol/l, cholesterol – 8,0 mmol/l, potassium – 3,9 mmol/l, total protein – 56 g/l, albumin – 35 %, α_1 – 3,5 %, α_2 – 10,5 %, β – 13,6 %, γ – 27,4 %, fibrinogen – 4,2 g/l.

ECG: sinus rhythm, heart rate – 64 beats per minute. The electrical axis is tilted to the left.

Diffuse dystrophic changes in the myocardium of the left ventricle.

X-ray of the chest organs: without pathology.

Task:

1. Assume the most likely diagnosis.
2. Justify your diagnosis.
3. Draw up and justify a plan for additional examination of the patient.
4. Create a treatment plan for this patient. Justify your choice.
5. Give recommendations for further treatment, determine the prognosis.

Answer:

1. Acute post-streptococcal glomerulonephritis, symptomatic arterial hypertension, grade 2, risk 4. Acute renal failure.

2. The onset of kidney damage shortly after hypothermia and sore throat (10 days), no indication in the anamnesis of the presence of other causes. A combination of hypertension, gross hematuria and nephrotic syndrome (edema, daily proteinuria – 7,3 g/l, hypercholesterolemia – 8,0 mmol/l, hypoalbuminemia – 35 %).

ARF: signs of oliguria, azotemia: creatinine – 0,96 mmol/l.

3. The patient is recommended to: determine eGFR by creatinine and the ratio of albumin to creatinine; urine analysis according to Nechiporenko to clarify the cellular composition of urine sediment, immunogram to clarify the autoimmune process; LE cells in the blood, antinuclear antibodies AT to DNA and Sm – antigen to exclude kidney damage in SLE; AT to streptococcal antigens to confirm the etiology of the disease and determine treatment tactics; Ultrasound of the abdominal organs and kidneys; kidney biopsy to confirm and determine the form of glomerulonephritis.

4. Strict bed rest for 2–4 weeks until swelling is eliminated and blood pressure decreases. Restriction of liquid and table salt, protein restriction.

Etiotropic treatment: antibacterial therapy – if there is a proven connection between AGN and streptococcal infection and (or) the presence of obvious foci of chronic infection: (cephalosporins, for allergies – fluoroquinolones).

Elimination of edema: diuretics – Furosemide, Diuver.

Antihypertensive drugs (if bed rest, water and salt restrictions are ineffective): Hypothiazide 25 mg per day, Losartan 25 mg 2 times a day, Diltiazem 40 mg 2 times a day.

Anti-inflammatory and immunosuppressive therapy: glucocorticoids (Prednisolone) 40 mg per day; if immunosuppressants are ineffective (Cyclophosphamide 150 mg per day).

Improving microcirculation and preventing thrombosis: Heparin, Curantil.

5. After the symptoms of nephritis are relieved, the patient should be under “D” observation for 1 year, and symptomatic treatment is carried out.

The prognosis for timely treatment is favorable. Recovery is possible (up to 70 % of cases), transformation into chronic glomerulonephritis, death (up to 5 % with rapidly progressive glomerulonephritis with crescents).

Problem 5.

A 6-year-old boy was admitted to the department with complaints of weakness, decreased appetite, and swelling.

A child from the second pregnancy, which was threatened with termination at 4 months. Delivery on time, with stimulation. Birth weight 3200 g, length 51 cm. Breastfed for up to 3 months. Preventive vaccinations by age. I suffered from atopic dermatitis until I was 3 years old. He suffered from chicken pox. I often suffered from ARVI. Family history: mother – dermatitis, chronic tonsillitis; my maternal grandmother has bronchial asthma.

The child fell ill 16 days after suffering from the flu. Edema syndrome appeared. Subsequently, the swelling increased and diuresis decreased.

Upon admission to the hospital, the condition was of moderate severity. BP = 95/45 mm Hg. Heart rate – 82 beats/min. The skin is pale. Severe swelling of the face, legs, feet, anterior abdominal wall, lumbar region. Borders of the heart: right - along the right edge of the sternum, left – along the left midclavicular line. Heart sounds are somewhat muffled. The abdomen is soft and painless on palpation. Liver +2 cm from under the costal margin. The spleen is not palpable. I excreted 300 ml of urine per day.

Clinical blood test: HGB – 161 g/l, RBC – $5,2 \times 10^{12}/l$, MCV – 82, MCH – 28, MCHC – 31 g/l, RDW – 10,5 %, PLT – $480 \times 10^9/l$, MPV – 8,3,

PDW – 12 %, PCT – 0,4 %, WBC – $10,1 \times 10^9$ /l, p/i – 3 %, s/i – 36 %, eosinophils – 7 %, lymphocytes – 52 %, monocytes – 2, ESR – 37 mm/h.

General urine analysis: color – yellow, complete transparency, pH – 6,5, density = 1030, protein – 6,3 g/l, bilirubin – negative, urobilinogen – 1 mg/l, ketone bodies – negative, glucose – negative, leukocytes – 3–5 in p/z, erythrocytes – 0–1 in the field of vision, cylinders: hyaline 5–6 in the field of view, granular 3–4 in the field of view.

Biochemical blood test: total protein – 41 g/l, albumin – 19 g/l, seromucoid – 0,44, CRP ++, cholesterol – 13 mmol/l, total lipids – 13,2 g/l (normal – 1,7–4,5), potassium – 3,81 mmol/l, sodium – 137,5 mmol/l, urea – 5,1 mmol/l, creatinine – 96 μ mol/l (normal – up to 110 μ mol/l). Endogenous creatinine clearance: 80,0 ml/min. Coagulogram: fibrinogen – 4,5 g/l, prothrombin – 130 %.

Biochemical urine analysis: protein – 2,5 g/day (normal – up to 0,2 g/day), oxalates – 28 mg/day (normal – up to 17).

Ultrasound of the kidneys: the kidneys are located in a typical location, the echogenicity of the cortical layer is moderately diffusely increased.

Task:

1. Make a diagnosis.
2. What is the reason for the appearance of proteinuria in this disease?
3. Give reasons for the diagnosis.
4. Make an examination plan. Name functional methods for studying the kidneys.
5. Assess the functional state of the kidneys.
6. Make a treatment plan.
7. What diet should you follow for this disease?
8. What is the duration of clinical observation?

Answer:

1. Acute glomerulonephritis with nephrotic syndrome. Active period, stage II, Impaired Kidney Function-0.

2. Proteinuria – loss of protein from 1 to 24 g/day, mainly albumin. The reason is an increase in the permeability of the glomerular filter, a decrease in protein reabsorption.

3. Substantiation of diagnosis. Anamnesis: illness 16 days after a viral infection, there was an increase in edema. Objectively pallor, severe swelling, oliguria, leukocytosis, lymphocytosis, increased ESR, proteinuria, +CRB, seromucoid, hypoalbuminemia (N 53-65), hypoproteinemia, hyperlipidemia, reduced clearance. endogenous creatinine (N 120), moderate increase in echogenicity of the renal cortex, oxaluria.

4. Research: concentration function according to Zimnitsky (N 1010–1025, hypersthenuria up to 1030>, hyposthenuria <1,010, isosthenuria during the day 1012–1014, nocturia predominates – nighttime diuresis over daytime. N 2:1, polyuria – increased daily diuresis 2 times or more.

f glomeruli: glomerular filtration rate: N 120 ml/min = endogenous creatinine clearance, urinary protein excretion should be at least 150 mg/day.

Tubular f assessment: determination of tubular secretion and reabsorption. The total concentration function of the kidneys (proximal and distal tubules) is reflected by tubular reabsorption, which can be determined by the following formula: $KR = (KF - MD) / KF * 100 \%$, where KR is tubular reabsorption; GF – glomerular filtration; MD – minute diuresis.

Emergency urography – according to indications.

5. Functional state of the kidneys – reduced functional activity of the glomeruli (proteinuria, reduced clearance, hypoproteinemia, hypoalbuminemia, oliguria).

6. Treatment – bed rest, diet with limited salt.

Prednisologist 1,5 mg/kg

Ampicillin IM 500 mg × 3 times a day, for 7 days.

Anticoagulants: heparin – 5000–10000 units under the skin of the abdomen every 4–6 hours.

Cytostatics are prescribed when nephrotic syndrome develops, resistant to treatment with corticosteroids, when corticosteroids are contraindicated (hypertension, peptic ulcer disease, diabetes mellitus) and if the patient has developed glucocorticosteroid dependence or side effects from their use. Cyclophosphamide is prescribed at a dose of 1,5–2 mg/kg body weight (100–150 mg) per day.

Curantil is prescribed for any type of GN, except for hematuria, at a dose of up to 400 mg per day for a period of 2 to 8 weeks.

7. Diet – with restriction of animal protein and Na, dairy-vegetable (protein 1,5/kg body weight per day, salt – 2 g per day). Drinking – 50 % more than the age norm (tea, compote).

8. Clinical observation for 2–3 years. For 3 months, OAM every 10 days, urine according to Nechiporenko once every 2–3 weeks.

During the 1st year, OAM, urine according to Nechiporenko, urine culture, AS monthly, then once every 3 months. Zimnitsky test, blood test – once every 6 months.

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