MINISTRY OF HEALTH OF UKRAINE

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Departments of Pediatrics №2

CONFIRMED by

Vice-rector for research and educational work

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METHODOLOGICAL RECOMMENDATIONS ON PRACTICAL CLASSES FOR STUDENTS

International Medical Faculty, course 6

Educational discipline "**PEDIATRICS**"

Approved
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1. **Topic №24**

   Differential diagnosis of glomerulonephritis in children. Conditions associated with hematuria in children. Differential diagnosis of acute poststreptococcal glomerulonephritis in children. Conditions associated with proteinuria in children. Differential diagnosis of nephrotic syndrome in children. Chronic kidney disease in children. Emergency care for acute kidney injury in children. Treatment of glomerulonephritis and chronic kidney disease in children. Conservative treatment of acute kidney injury (AKI) in children and indications for dialysis.

2. Relevance of the topic.

Glomerulonephritis (GN) takes 3-4 place in the structure of diseases of the urinary system in children. Most often, children are sick at the age of 3-12 years. When glomerulonephritis occurs in children over 10 years of age, chronicity of the pathological process is more often observed, and the patient's body is often resistant to GCS therapy. Glomerulonephritis in children is the most common cause of acute or chronic renal failure.

3. Objectives of the lesson:

3.1. General goals:

- get acquainted with the modern definition of GN, etiology, clinical signs, be able to diagnose in children, draw up a plan for therapeutic and preventive measures.

3.2. Educational goals:

- to get acquainted with the contribution of domestic and foreign scientists in identifying the problems of GN that contribute to the development of diseases, to determine the need for prevention of GN in children.

3.3. Specific goals:

- to know:

1. Definition of glomerulonephritis.
2. Prevalence and incidence of glomerulonephritis in children's populations, risk factors for the development of the disease.
3. Pathogenesis of glomerulonephritis.
4. Classification of glomerulonephritis in clinical course, period, degree of activity, presence of renal dysfunction.
5. Characterization of butcher clinical variants of GN in children.
6. Characterization of nephrotic syndrome.
7. Diagnostics of GN.
8. Programs of treatment of the main variants of glomerulonephritis.
9. Differential diagnosis of GN.
10. Complications of glomerulonephritis in children.

3.4. On the basis of theoretical knowledge on the topic:

- to master the techniques / be able to /:

1. Collect anamnesis.
2. Identify the risk factors for the development of glomerulonephritis.
3. Conduct a clinical examination of the nephrologic patient.
4. Select the most informative methods of examination to determine the diagnosis and conduct differential diagnosis.
5. Interpret the results of laboratory tests, ultrasound and X-ray examination.
6. Establish a diagnosis according to the classification.
7. Develop a therapy plan for the child with glomerulonephritis.

4. Materials of pre-classroom independent training (interdisciplinary integration).

№	Discipline	To know	To be able
1.	Previous disciplines		
	1. Anatomy	Anatomical structure of the kidneys and urinary tract	Identify peculiarities in children.
	2. Normal physiology	Functional state of kidneys and urinary tract in children.	Identify peculiarities in children.
	3. Pathological physiology	Diseases of the kidneys and urinary tract: microbial-inflammatory, immune-pathological, metabolic. Congenital malformations of UTS	Correctly assess the nature of the process
	4. Biochemistry	Components of the biochemical blood test, which reflect the function of the kidneys and UTS	Correctly evaluate the results of the study.
	5. Propedeutics of childhood diseases	Anatomical - physiological features of the kidneys and UTS in children of all ages. Semiotics of violations. Survey Methodology.	Conduct a clinical examination of a child with a disease of the UTS.
2.	Next disciplines		
	1. Pediatrics	Methods of diagnostics and differential diagnostics of diseases of kidneys and UTS in children.	Carry out a differential diagnosis with other nosoforms with a similar clinical and laboratory picture.
3.	Interdisciplinary integration		
	1. Glomerulonephritis 2. Chronic kidney disease	Methods of diagnostics and differential diagnostics.	Conduct a clinical examination of the patient. Evaluate the results of paraclinical tests.

5. Contents of the topic.

***Glomerulonephritis* (GN)** is a nonspecific inflammatory disease of the renalglomeruli of imm nologic origin. Most forms appear to be brought about by thepresence of immune complexes in the walls of the glomerular capillaries withactivation of complement and initiation of an inflammatory reaction. It has beengenerally accepted that soluble immune complexes become deposited in theglo meruli, but over the last few years evidence has been produced to suggest thatcirculating antibody r eacts with antigen at fixed sites in the glomerular capillary wall. The other main immunologic mech anism is the activation of complement byantiglomerular basement membrane antibody fixing on to glomerular capillary walls. This mechanism is rare compared with the immune complex mechanism . Immunofluorescence techniques are used to demonstrate immunoglobulins (antibody) and complement give a granular pattern outlining the glomerularcapillary walls in the im mune complex type, and an uninterrupted linear pattern alongthe capillaries in the antiglomerular ba sement membrane antibody type.

Classification:

Acute glomerulonephritis.

I a) with nephritic syndrome - renal edema, arterial hypertension, hematuria, proteinuria;

b) with nephrotic syndrome - marked edema, high proteinuria (3 gr per day and more), hypoproteinemia, blood cholesterol is increased;

c) with nephrotic syndrome, adding arterial hypertension, hematuria;

d) with urinary syndrome

II Renal function : normal, damaged, acute renal insufficiency.

Chronic glomerulonephritis.

a) nephrotic form - marked edema, high proteinuria (3 gr per day and more), hypoproteinemia, blood cholesterol is increased;

b) hematuric form - hematuria with proteinuria;

c) mixed form - edema, hypertension, and urinary syndroms.

II Renal function : normal Damaged Chronic renal insufficiency

Activity of renal process

Period of exacerbation

Period of partial remission

Period of full clinical and laboratory remission

Subacute (malignant) glomerulonephritis

- Without disorders of renal function
- With disorders of renal function
- Chronic renal insufficiency

Pathogenesis of Glomerulonephritis:

The two main prosseses are involved in the pathogenesis of glomerulonephritis.

1. Autoimmune: antibodies (antiglomerular basement membrane) react with an antigen inbthe glomerular basement membrane and produce glomerulonephritis (5% cases).

2. Immune complex theory.

Streptococcal or other antigenes provoke antibody response, and the subsequent antigenantibody complexes in the circulation are deposited in the glomerular cappillary walls. These complexes activate the complement pathway with the liberation ofchemotactic factors causing polymorpho-leucocytic infiltration the release of lysosomal enzymes from neutrophils and the direct effect of the complement system lead to damage of the capillary wall including the glomerular basal lamina.

Clinical features of AGN (nephritic syndrome).

Typical clinical picture is presented now rarely.

• A latent period of from 5 days to 4-6 weeks occurs between
the streptococcal infectious and the abrupt or acute onset of nephritis

• Sings of intoxication (fatigue decreased appetite)

• Edema (periorbital, leg or sacrlal edema or generalized due to salt and water retention)

• Mild or severe hypertension (headaches, visual disturbances secondary to hypertension, rarely hypertensive encephalopathy may be the presenting complains of AGn)

• Sings of left ventricular failure (ortopnoe, breathlessness, 3achycardia)

• Renal impairment manifesting as oliguria or acute renal failure

• Dark urine (cola-colored urine)

• Changes on the retina (spasm of arteries, dilatation of veins, hemorrhages)

• Eclampsia due to cerebral edema and hypertension

• Sometimes the onset of the disease may be insidious with weakness, fatigue and malaise or mild edema as the most prominent symptoms after the history of a sore throat, respiratory disease or other

• In such situation urinalysis should be prescribed

• A history of streptococcal or other infection 1-4 weeks prior to onset
of erythrocyturea, proteinurea with development of edema or hypertension are patogonomic of Acute glomerulonephritis.

Syndromes in GN:

– ***Urinary syndrome*** (proteinuria (less than 3 gm day), RBCs and casts in the urinary sediment)

– ***Nephritic syndrome*** (abrupt onset of hematuria, proteinnuria (usually associated with non-nephrogenic range),castiuria, oliguria, hypertension)

– ***Nephrotic syndrome*** (proteinurea more than 3,5 gm/day, hypoproteinemia and hypoalbuminemia, severe adema,hyperlipidemia)
– ***Edema*** (mostly is locaried on the face (periorbital), is pale, warm, appears in the morning than decreased, in the second part of the day develops on the legs)
– ***Hypertensive syndrome*** (hypertension is hyperkinetyc and not severe):
• Hypertensive stage: complains on headache, disturbances of vision, insomnia; objective examination reveals high blood pressure, hypertrophy of left ventricular, signs of heart failure, cerebral and cardiac complications
• Stage of chronic renal failure signs and symptoms according to the stage (I - IV).
Nephrotic syndrome (NS) - clinical and laboratory syndrome wich includes:
• *Proteinuriua more than* 1 g/m2 24 hours (3,5-4 g/ 24 hours),
• Hypoproteinemia with hypoalbuminemia less than 25 g/l, hyper-alfa-2-globulinemia,
• hyperlipiduri, lipiduria,
• edema.
Complications of NS
• ***nephrotic crises***
• severe pain in abdomen, associated with peritoneal symptoms,
• fever,
• oliguria and look like thrombosis of mesenterial arteries and need urgent consultation of surgeon),
• skin symptoms migrate erythema
Laboratory findings
• Urine analysis
• the urine may be scanty, brown, smoky of franky bloody.
• From 0.5 to 30 gr/day of protein excreted
• The urinary sediment contains RBCs, RBC cast (are
the pathognomic of glomerulitis from any etiology)
• WBC, renal tubular cells, WBC cast and granular (protein droplets) casts are also may be common
• Urinanalysis Nechyporenko (more than 50 000 RBCs in 1 ml of urine is named as hematuric component)
• Blood analysis (mild anemia (due to hypervolemia), mild leucocytosis, lympocytosis, increased ESR)
Laboratory findings biochemical blood analysis:
• (hypoproteinemia and hypoalbuminemia, hyperlipidemia (hypoalbuminemia trigg ers increased synthesis of all forms of plasma proteins including lipoproteins resulting in hyperlipidemia),
• elevated level of antistrepolysin-titre (more than 1:3000);
• serum complements levels (C3, C4 and the total hemolytic activity) are usually diminished during the active phase of the disease (returns to normal at 6-12 weeks);
• serum urea, creatinine may be elevated due to digurea and creatinine clearance reduced;
• hypercoagubility may result from
– increase urinary loss of antitrombin III
– altered levels and/or activity of protein C
– hyperfibrunogenemia due to increased hepatic synthesis
– impared fibrinolysis
– increased platelet aggregability
Instrumental investigation
• Renal biopsy usually required for diagnosis in adults
• Ultrasound examination may show enlarged kidneys
Parculiaritis of clinical and laboratory signs of CGn according to morphologic changes in kidneys
• Mesangioproliferative Gn (Ig A nephropathy) – isolated urinary syndrome, nephritic syndrome, hematuria in adults
• Mesangiocapillary Gn – nephrotic syndrome, urinary syndrome with hematuric component, hypertension

• Membranosus Gn – nephrotic syndrome (80%) in age 40-50
• Focal and segmental Gn - nephrotic syndrome, hypertension in Afro-Americans
• Minimal change disease - nephrotic syndrome in children
• Fibroplastic Gn - nephrotic syndrome (50%), hypertension, chronic renal failure

Differential diagnosis

Urinary syndrome
• Acute pyelonepritis
• Activation of primary chronic glomerulonephritis
• Toxic nephritis
• Goodpasture's Syndrome
• Hereditary nephritis (Alport's Syndrome)

Nephrotic syndrome
• Amyloidosis
• Diabetic nephropathy
• Colagenic nephtopathy (SLE scleroderma)

Hematuric component
• Malignancy associated nephritis
• Urotuberculosis
• Renal stones

An example of diagnosis

Acute glomerulonephritis, urinary syndrome, hematuric component.
Acute glomerulonephritis, nephrotic syndrome.
Chronical glomerulonephritis, urinary syndrome, hypertensive stage, phase of activation.

Duration of acute glomerulonephritis

• Recovering during first 2-4 weeks or 2-3month
• Prolonged duration (duration more than 4 month, full recovering is 2-3 times rare)
• Negative prognostic feature is nephrotic syndrome, associated with severe hypertension
• Development of chronic glomerulonphritis (urinary syndrome, edema or hypertension are present more than 12 month)

Complications of acute glomerulnepherts

• Eclampsia (angiospastic encephalopathy)
• Acute heart (left ventricular) failure
• Acute renal failure

Treatment of acute glomerulonephritis

Acute glomerulonephritis have to be treated only in speciallised nephrologic department

– Regimen: bed-rest during 2-4-6 weeks until desappearing of edema and normalizing of blood pressure
– Diet № 7a
– Daily record of fluide intake and output
– Restriction of dietary protein if azotemia and metabolic acidosis are present
– Salt free diet (Sodium intake is restricted only when circulation overload, edema, or severe hypertension is present)

The aim of drug therapy is recovering of the patient
• Anti microbal drug
• Symptomatic therapy
• Membranenostabilizative therapy
• Pathogenetic therapy

Antimicrobal therapy

• If a bacterial infection is still present when nephritis is discovered, it should be treated with an appropriated antimicrobial drug
• Semisynthetic penicillins in middle therapeutic doses have to be prescripted

Symptomatic therapy

Edema

• Loop diuretics such as furosemide or lasix (40-400 mg/day or 1-2 gr/day) help in the management of the expanded extracellular fuid volume (side effects: hypocholremic alkalosis, decreasing K, Na level in blood)

• In patients with decreased of furosemide should be prescribed uregit (50-200-500 mg/day orally) or the combination with the thiazides (hypothiazide 25-100 mgm/day)

• 2.4 % solution euphylline 10 ml i/v

• Albumin may help in the management of hypoproteinemia

• Daily weighting to check change in the body fluid status and record of fluid intake and output have to be made in patients which receive diuretics

Hypertensive syndrome

Antihypertensive drug therapy is usually started with single drug, but if there is incomplete response a second drug is added.

One of the following drugs as a single drug treatment can be used:

• ACE inhibitors(or angiotensine II reseptors blockers)

• (loop) diuretics

• calcium channel blockers (non dihydropiridine agents)

If single drug treatment is unsuccessful then the combination therapy may be given as two-drugs or three – drugs therapy

Two – drug therapy:

• calcium channel blocker + diuretic

• ACE inhibitor + diuretic

Triple – drug therapy is used very rare

• calcium channel blocker + diuretic + ACE inhibitor

Such drugs as adelfan or trirezide (which contained fixed doses of several hypotensive drugs) are not good in therapy of hypertensive syndrome

Hematuric component

• Dicinon (etamsilate) 2 ml 12.5% solution twice a day (7-10 days) i/m, then 0.25-0.5 three times a day orally

• Kvarcetin 1.0 in a half of glass of water three times a day.

• Ascorbinic acide 500 mg a day.

• Ascorutine 1 tabl. three times a day

• Rutine and other.

Membranostabilizative therapy

• Have to be prescribed in patients with AGn, urinary syndrome, hematuric component, after prescription of symptomatic therapy.

• Unitiol (5 ml 5% solution i/m during 1 month)

• Dimephosphon (100 mkg/kg/day 1 month)

• Aminochinolytic drugs (delagil – 0.25 two times a day orally 1 month, then 0.25 a day during 5-12 month)

• (side effects: leucopenia, degeneration of retina, allergy, dyspepsia)

• ά-tocoferol (50 mgm/day – 5-12 month)

Pathogenetic therapy have to be used in patients with:

• Gn, nephrotic syndrome after 3-4 weeks from the beginning of the disease, when symptomatic and membranostabilisativetherapy is unsuccessful

Pathogenetic therapy includes:

• Glucocorticoids

• Cytostatics

• Anticoagulants and antiagregative drugs

Glucocorticosteroids

– Prednisolone 1 mg/kg/day for 4-6 weeks followed by decreasing of dosage on 2.5 mg each 5-7 days

– In patients with high activity of patogenetic process pulse-therapy with metylprednisolone (metipred, soly-pred, solu-medrol) (1000 mg/d three days) can be used and then therapy in previous doses
– (Side effects: obesity, hirsutism, disturbances of menstrual function, achne, Cushing syndrome, ulcers of alimentary tract, hyperglycemia, hemorrhagic, pancreatitis, psychiatric disturbances.
– After abrupt discontinuoing of the drug usage can be worsening of the duration of the main disease).

Cytotoxic drugs

• Cytotoxic drugs should be given in refractory cases or if glucocorticosteroids are contraindicated.
• Cyclophosphamide (1.5-2 mg/kg/day), imuran (2-3 mg/kg/day), leukeran, chlorbutin (0.2 mg/kg/day), cyclosporn A (sandimun) or others are given for 4-6 weeks at the nephrologic department and then 4-6 months at home under the control of blood analysis each 7 days.
• Pulse-therapy of cytotoxic drugs (1000-1200 mg of cyclophosphane i/v once a month 5-6 times) at specialized nephologicdepartment can be used
• (Side effects: cytopenia, dyspepsia, hemorrhagic cystitis, toxic hepatitis, sexual dysfunction, infertility)

Anticoagulants and antiagregants

Direct anticoagulants (fraxiparine 0.3-0.6 ml/day subcutaneous 10-14 days, heparin 5000- 10000 subcutaneous 4 times a day 1-1.5 month (under the control of time of blood coagulation or time of bluding) then gradual decreasing of the dose during 1 week)
Side effects: hemorrhages, allergy.
Non – direct anticoagulants (pheniline 0,045 – 0,06/d 1 – 2 month)
Antiagregative therapy (curantyl 200 – 400 mg/d, trental 600 mg/d 2 – 6 month)
In patients with high activity of pathologic process 4-component therapy have to be used (glucocorticosteroids, cytotoxicdrugs, anticoagulants and antiagregants simultaneously)

Treatment of eclampsia

• i/m: 25% solution of magnesium sulfates 10 ml 2-4 times a day;
• 1 ml of 25% solution of aminazine;
• 10 ml of 2,4% solution of euphyllin;
• 80-120 mg of furosemide;
• 30 ml of 40% glucose solution.

Instrumental methods of treatment.

Indications: side effects or nonefficasy of pathogenetic therapy
Contraindications: level of the Hb less than 80 gm/l, hypotension, leucocytopenia, thrombocytopenia, allergy on protein preparations, hemorrhagic complications, ulcer disease.
Types:

• Plasmapheresis (may be safer and more effective when high titers of anti – GBL antibodies are present in the case offulminant immune complex disease)
• Hemosorbtion
• Lymphosorbtion

CHRONIC GLOMERULONEPHRITIS

Chronic glomerulonephritisis the final stage of many different forms of g-n, but often the kidney is so badly damaged that it is impossible to determine the type of g-n that was the forerunner. In particular it is not possible to determine how many cases of chronic g-n were originally acute post-streptococcal g-n.

The kidneys of chronic g-n are reduced in size and have finely granular subcapsular surfaces. They are firm in texture and often have prominent small arteries indicating thickening as a result of hypertension. Hypertension is a feature of chronic g-n. Other clinical features were described in the lecture on renal failure.

Microscopically the glomeruli are solidified either partially or wholly. Tubules show much loss and atrophy, and arteries show intimal thickening. The interstitiumshows fine fibrosis and

contains variable numbers of inflammatory cells. Immunofluorescence and electron microscopy are not of much help in sorting out the antecedent forms of g-n because the glomeruli are usually too severely damaged to enable specific patterns to be recognized.

NEPHROTIC SYNDROME

Nephrotic Syndrome is a disorder in the human body, wherein large amount of protein leaks from the blood into the urine, due to damaged kidneys. This spill eventually leads to depletion of protein levels in the body, an increase in the levels of lipid and causes edema (swelling of body parts due to excessive accumulation of watery fluid). Although, it can occur at any age, children between the age group of 18 months to 4 years are at a higher risk.

Approximately two in every 10,000 individuals suffer from Nephroticsyndrome. Let's evaluate the causes, symptoms and treatment for this kidney disorder.

Causes of Nephrotic Syndrome

Nephrotic syndrome is caused due to the damage to the tiny blood vessels present in the kidney, that are designed to filter waste and excess water from the blood. This condition may arise due to various factors like diseases affecting other parts of the body, such as diabetes and mellitus. A person suffering from glomerulonephritis can also experience Nephrotic syndrome. Non-steroidal anti-inflammatory drugs (NSAIDs), which are harmful for the kidneys, can also lead to this disorder. It is also caused due to allergic reactions stimulated by some insect bites. Nephrotic syndrome may also be a hereditary disorder, though, the chances are very small.

Main causes of NS:

1. Glomerulonephritis

Various forms but membranous and membranoproliferative are commonest

2. Lipoid nephrosis (minimal change disease)
3. Focal and segmental glomerular sclerosis and hyalinosis
4. Generalized systemic diseases:
    a) Diabetes
    b) Amyloid
    c) Systemic lupus SLE
    d) Schonlein-Henoch syndrome (HSP GN)
5. Miscellaneous.

Symptoms of Nephrotic Syndrome

The symptoms of this disorder vary from person to person, but the most common symptoms include:

Edema: Bloating or swelling of the body due to accumulation of water in excessive amounts. It is experienced by 95% of the patients suffering from this disorder. The swelling may be noticed in the face, feet, hands, abdomen etc.

Hematuria: A condition wherein the patient may loose blood while passing urine.

Oliguria: The quantity of urine a person passes, decreases substantially when he is suffering from this syndrome.

Pleural effusion: The person experiences difficulty in breathing, due to the accumulation of water in the space surrounding the lungs.

High-blood pressure: An individual suffering from this disorder experiences high blood pressure regardless of his age.

Other than these symptoms the patient experiences anorexia or loss of appetite, fatigue and the patient appears pale.

Patients may lose as much as 25g of protein in the urine each day and this has the effect of depleting the amount of albumin (and in some circumstances other proteins) in the blood. The decreased levels of serum albumin interfere with the colloid osmotic pressure and cause a loss of fluid

from the capillaries into the subcutaneous tissues. This occurs particularly in the ankles and legs which become swollen due to edema. Effusions of fluid may occur in the peritoneal cavity (ascites) or in the pleural cavities in patients with severe forms of the NS. Serum cholesterol increases and may reach high levels, but not all cases of NS show high serum cholesterol. Various lipids show elevated serum levels, but it is not known why this is so. Doubly refractile fat bodies are found in the urine and probably reflect the fatty changes that take place in the tubular epithelium.

It is important to realize that the NS is not a disease; it is a syndrome caused by many different renal diseases. When a clinician encounters a patient with the NS it is important for him to determine the underlying condition, because the course and prognosis will depend on the underlying disease.

Diagnostic

1. Urine protein exretion is more than 1 g/m^2/day; exretion rates of more than 5 g/day are common in young children. Albumin is the principle urinary protein in NS.
2. Hypoalbuminemia (less than 2,5g/dl) is characteristic. Serum levels of alfa-1 globulin are decreased; levels of alfa-2 globulins, beta globulins, and fibrinogen show a relative or absolute increase.
3. Serum levels of cholesterol, tryglycerides, and total lipids are increased, reflecting both increased production and decreased clearance of lipid. Cholesterol levels vary inversely with serum albumin levels.
4. The urine contains hyaline and granular casts, free lipid, cholesterol-containing bodies, and fatty casts. Microscopic hematuria is present in 25 % of children with NS; gross hematuria or red blood cell casts suggests other glomerularadiseases.
5. Serum BUN and cretinin levels are midly increased in 25 % of children with NS, reflecting reduced intravascular volume; the levels normalize with onset of diuresis. Persistent of worsening azotemia suggests other glomerular diseases.
6. Serum complement levels are normal; a decrease suggests membranoproliferative glomerulonephritis or systemic lupus erythematosus. decreased serum levels of Factor B and Ig G may contribute to increased susceptibility to infection, decreased serum antithrombin III and plasminogen to increased risk of thrombosis.

An individual, who shows the symptoms of Nephrotic disorder, is subjected to a blood test and urine test to measure the amount of protein, cholesterol and sugar in the blood. More sophisticated tests like ultrasound, CT scan, and MRI can be performed for accurate detection of the disorder. A biopsy of the kidney can also be helpful in determining the extent of damage suffered by the organ.

COMPLICATION

Nephrotic children are at increased risk of serious bacterial infection (S.pneumoniae, E. coli, H. influenzae), including septicemia, spontaneous peritonotis, urinary tract infections, and cellulitis. Promptly evaluate children with fever, abdominal pain, dysuria, etc. and initiate appropriate antibiotic therapy as indicated. The risk of deep venous and renal vein thrombosis is increased in nephrotic patients, due to their hypercoagulation state.

Treatment

The main methods of treatment of acute glomerulonephritis are regime, diet and medication which are determined by pediatricians, depending on the health of a sick child, treatment may be carried out in hospital or at home. In the acute period it is necessary to prescribe bed regime and warm the child. Bed rest should be observeduntil recovery of diuresis, reduction of swelling, lower blood pressure and elimination of massive haematuria (usually no more than 3 - 4 weeks).

The basis used diet is to limit the content of sodium in the diet, a temporary restriction of protein and fluid. "Therapy hunger and thirst" should not be applied, because it causes the collapse of the endogenous protein with hyperasotemia. In the first period of the disease salt-free diet must be

used. The number of drunken fluid diuresis must exceed the previous day at 400 - 500 ml (to compensate for extrarenal losses). The total caloric content of food must meet the age requirements at the expense of fats and carbohydrates. During the first 4-6 weeks of illness some limitation of protein are desirable (up to 1-1,5g per 1kg body weight per day) with subsequent transition to physiological norms.

In acute glomerulonephritis of the child's nutritional products, causing allergy and maintaining and strengthening hypertension and edema, are excluded. Products containing potassium must be added in the ration: potatoes, raisins, dried apricots, bananas. Only at renal failure the protein is limited to (1,0-1,5 - 2g / kg) at the expense of meat, fish, cottage cheese.

Hereditary Nephritis (Alport's Syndrome)

This dangerous form of glomerulonephritis occurs in families and appears to be widespread in the country. Both males and females are affected but males have a much worse prognosis and die during early adult life; females fare much better, although a certain number will die from renal failure.

The clinical picture is varied but hematuria is the commonest symptom, coming on sometimes in childhood and tending to be recurrent. Proteinuria varies in its severity and some patients may have a nephrotic syndrome. Another feature is deafness of a high frequency type, but this is not always present; in some affected families some of the siblings may have deafness without the renal lesion. The course of those with renal involvement is a gradual development of chronic renal failure, particularly in the affected males. The genetic analysis seems consistent with *autosomal dominant inheritance* of a pleiotropic gene, with variable penetrance and expressivity.

The pathologic picture in the early stages is a focal form of glomerular involvement in which *segmental areas of bland sclerosis* occur in the glomeruli. Some cases show a proliferative picture in the glomeruli. Gradually, more and more glomeruli become sclerotic with concomitant tubular loss. *Foam cells* containing numerous different lipids are found in the interstitium. At one time foam cells were considered to be a specific change; this is not so; foam cells are found in patients with the nephrotic syndrome of various different origins. However, the presence of interstitial foam cells in a patient without the nephrotic syndrome should arouse the suspicion of hereditary nephritis. Another changesof interest in hereditary nephritis is the appearance of the glomerular capillary walls under the electron microscope. This change consists of a *splitting of the basement membrane into several layers* accompanied by varying numbers of small round dark particles. This change is common in hereditary nephritis. In addition to the morphologic changes described in GBM, recent studies have shown the absence from the GBM of the so-called Goodpasture antigen.

Differential diagnostics of hematuric and nephrotic forms of chronic glomerulonephritis

Sign	**Hematuric form**	**Nephrotic form**
Edema	Insignificant	expressed
Hematuria	Significant (macro hematuria)	Insignificant, unstable
Daily proteinuria	Up to 1 g / day	> 2 g / day
Arterial hypertension	Often	is possible
Cholesterol level, total lipids	N	Elevated
Total protein	N	Reduced
Albumin of blood	N	Reduced

Differential diagnosis of acute glomerulonephritis and pyelonephritis.

Sign	PN	Acute GN

Communication with the disease	ARVI	2-3 weeks after streptococcal infection
Symptoms of intoxication	+	+
Body temperature	>38	-
Dysuria	Possible nocturia	-
Pain syndrome	+	Rarely in the back
Leukocytosis	+	±
ESR	Up to 30 mm / h	More than 30 mm / h
Bacteriuria	+	-
Osmolarity of urine	Reduced	N
Glomerular filtration rate	N	Reduced or N

Differential diagnosis of tubulointerstitial nephritis and chronic glomerulonephritis.

Sign	Glomerulonephritis, hematuria form	Interstitial nephritis
Causes of development	Acute and chronic streptococcal infection	Metabolic disorders, drugs, viral infection, kidney dysplasia, vascular, physical, allergic factors
Edema	+	Join at later stages
Hypertension	Joins at later stages	Joins early
Lumbar pain	-	+
Rise in temperature	-	+
Hematuria	When exacerbating the process	It is possible
Proteinuria	+	Not high
Glucosuria	-	It is possible

Differential diagnostics of interstitial and hereditary nephritis

Sign	Hereditary nephritis	Interstitial nephritis
Kidney disease in family members	+	Rarely
Loss of hearing in proband in the family	Often	_
Anomalies of sight	In 20%	-
The most common clinical signs	Stigma diesembriogenesis	Abdominal pain, dysuria, intoxication, decrease (increase) of blood pressure
Pyelectasis, defects of kidney development	Often	Rarely
Relative density of urine	Normal for a long time	Reduced
Hematuria	More often microhematuria	Expressed
Leukocyturia	Rarely	Often, abacterial
Increase in kidney area (ultrasound)	-	+
Morphobioptic data	Focal-segmental glomerulitis	Infiltration of interstitial lymphocytes and plasma cells, tubular atrophy, fibrosis, scarring in interstitium

Uremic coma

Uremic coma develops at the chronic kidney diseases with severe functional insufficiency. A coma develops slowly with proof head ache and vomit, common trouble, insomnia.

Gradually a child becomes indifferent, sleepy, soporosis and eventually falls in a deep coma. In the last phase a child lies with expression of indifference, pale, with the easy edema of eyelids and narrowed pupils. A skin is dry, swollen. Breathing is deep, often to the type Chayne-Stokes or Kussmaul. Cardiac activity is broken. There are high cardiac shove, increased II tone above an aorta, promotion of AP. Friction of pericardium. In vomit there are masses admixture of blood . Toxic diarrhea is present. There is the white disk of visual nerve on an eyeing botton. Tendon reflexes are promoted. Convulsions appear in the final stage of disease. In a blood: anaemia, nitrogenemia, increase of urinary acid and creatinini, decline of alkaline reserve. In urine: decline of specific gravity, cylinduria, erythrocyturia, proteinuria.

A coma at eclampsic uremia (pseudouremia, chloremia) can occur very acutely, suddenly to the exposure of signs of acute glomerulonephritis. More frequently begins from generelized tonico-clonic convultions and at once is deep. In most cases develops at presence of signs of acute diffuse glomerulonephfitis, to which precede great head ache, vomit, disorders of sight and ear. Quite often there are the additional focal symptoms related to the spasm of cerebral vessels: pathological reflexes, hemianopsia, aphasia, temporal paresises, fibril convultions of muscles and other

During the attack of convultions the breathing is stridorous, breathing normalization comes after an attack. Tones of heart are loud, high cardiac shove, the accent of II tone on an aorta, increase of AP. Skin is pale, edenematous, pupils are narrow, stagnant, edenematous nipples and spasm of vessels on an eyeing botton. In a blood: hyperelectrolitemia with hyperchloremia and hyperkaliemia, hypocalciemia. In urine: oliguria, haematuria, proteinuria, cylindrurtia.

The hypochloremia coma develops at unrestrained vomit: poisonings, toxicoinfections, acute gastro-intestinal disorders, surgical diseases of digestive channel (impassability, invagination), afteroperative vomit, protracted acetonemic vomit, massive and protracted sweatness, reception of saldiuretics.

It develops slowly. A child becomes adynamic with hyperreflexia. Quite often there is tetania, catalepsy, fibril convulsions. Sharp lines of face, eyeballs are soft, a skin is dry, turgor is reduced. Breathing is superficial and increased. Tachycardia, arterial blood pressure is low. Blood: high hematocriti, hypernitrogenemia, hypochloremia, hypokaliemia. In urine: small proteinuria and erythrocyturia.

Help on a prehospital stage at uremic coma

1. **At sopor to wash a stomach and intestine by 2-3 % sodium solution hydrocarbonati (**washing of stomach in the comatose state is conducted only after intubation).
2. To limit the receipt in the organism of salt, proteins.
3. With the purpose of the detoxication intravenously in drops introduction of 5 % Glucose solution 10-20 ml/kg of mass with 2-3 ml 5 % solution of ascorbic acid.
4. At severe vomit and diarrhea to reduce hypochloremia intravenously in drops introduction 5 % of Glucose solution in half with isotonic solution of sodium chloride .
5. At vomit: Motillium – 1/2-1 tablets 2 times per day, Bromopridi 1 candle (10 mg) 1-2 times for a day, Cerucali – 0,05 mg/kg the masses (for one occasion dose) intramuscular, Clemastini – 0,5-1 mg (for one occasion dose) intramuscular to children of 6-12 years old .
6. At convulsions: Seduxeni 0,5 % solution intramuscular or intravenously streamly in one occasion dose 0,1 ml/kg the masses, but not more than 2-3 ml; 20 % solution of Oxybutirati sodium 70-100 mg/kg of mass intravenously streamly slowly on 20 ml isotonic solution of chloride sodium or 5 % Glucose solution.

7. Oxygen therapy by clean moistened oxygen.
8. Hospitalization in the intensive unit or chamber of intensive therapy.

Help on a hospital stage

1. Intravenously in drops 5 % Glucose solution , isotonic solution of sodium chloride or Ringer solution in even proportions in the total dose of 15 ml/kg of mass + volume which equals day's diuresis + amount of the liquid lost with vomit and diarrhea, on every degree of the promoted temperature of body ad 5 ml on every kilogram of mass.
2. At oliguria intravenously in drops 15 % Mannitoli solution 0,5-1 g/kg of masses per day, dividing into 2-3 introductions. Parallel is appointed 1 % solution of Lazix intravenously streamly in one occasion dose 1-2 mg/kg of mass, 2,4 % Euphyllini solution in one occasion dose 2-3 mg/kg of mass intravenously streamly.
3. For liquidation of acydosis intravenously in drops very slowly 4 % solution of sodium hydrocarbonati 4 ml/kg of mass, to enter by drops doses under the control ABB.
4. At hyperkaliemia 10 % Glucose solution intravenously in drops from a calculation 0,5 g/kg of mass with insulin (1U of insulin for a 4-5 g of Glucose), 10 % solution of Calcium gluconatis 0,5-1 ml/yr of life intravenously streamly slowly.
5. At acute cardiac insufficiency intravenously in drops Dophamini from the calculation of a 3-5 mkg/kg of mass in 1 minute (4 % to divorce solution of Dophamini in 400 ml 5 % Glucose solution , in 1 ml will be 500 mkg Dophamini).
6. At severe anaemia transfusion of red corpuscles mass 5 ml/kg of mass of body.
7. At convultions: 0,5 % solution of Seduxeni intramuscular or intravenously streamly in one occasion dose 0,1 ml/kg of mass, but not more than 2-3 ml, or 20 % solution of Oxybutirati sodium 70-100 mg/kg of mass intravenously streamly slowly on 20 ml isotonic solution of sodium chloride or 5 % Glucose solution .
8. Oxygen therapy by 40 % moistened warmed-up to 22-24 0C oxygen through a nasal catheter.
9. Lespenephrili 20-30 drops 8-10 times per.
10. Washing of stomach (if in a coma, after intubation) and intestine by 2-3 % of solution of sodium hydrocarbonati; enterosorbents: absorbent carbon 1 g/kg of mass, Enterodesi 2-3 g 3-4 times per a day; Sorbiti, Xyliti – 0,3g/kg of mass.
11. At absence of effect - hemodialisis, which the indications are the increasing the blood urea to 20-33 mmol/l, creatinini – to 0,64-1,2 mmol/l, potassium – to 6-6,5 mmol/l, decline of alkaline reserve below 12 mmol/l, during glumerules filtration below 5 ml/mn.

6. Materials of methodological support of the lesson.

6.1 Tests.

1. A boy, 5 years, enrolled in a clinic with an assumption of acute glomerulonephritis. The examination revealed the presence of edema on the face, lower limbs. Indicate obligatory objective research to clarify the diagnosis?

A. Measurement of blood pressure
B. Determination of body temperature
C. Palpation of the kidneys
D. Record of diuresis
E. Palpation of ureteric points

2. A child of 14 years of age is observed for chronic glomerulonephritis, mixed form for 6 years. Edema syndrome BP 160/110 mm Hg The skin is pale. Blood urea - 20 mmol / L, blood creatinine - 0.12 mmol / L. Clearance on endogenous creatinine - 20 ml / min. Determine the leading method of renoprotektivnoy therapy patient:

A. Dialysis therapy
B. Angiotensin-converting enzyme inhibitors

C. Saluretics
D. Corticosteroids
E. Adrenoblockers

3. A girl, 8 years, has come up with complaints about changes in the color of urine. The condition is satisfactory, there is no visible edema. BP - 105/60 mm Hg Urine color "meat wash". Preliminary diagnosis: Acute glomerulonephritis. To clarify the diagnosis should determine the presence of hidden edema. For this purpose, it is necessary to conduct:
A. Sample by Zimnitsky
B. Blister test
C. Clearance on endogenous creatinine
D. Control of diuresis
E. Test the tourniquet

4. In the 10-years boy there are edema on the face, lower extremities, headache, urine of dark red color. BP - 140/90 mm Hg 7 days before, there was sore throat, fever. Urine test - a protein of 2.5 g / l, white blood cells - 8-10 in n / zor, red blood cells - entirely, cylinders - hyaline 2-3. Daily proteinuria - 1.5 g. The blood protein is 65.8 g / l. An blood - white blood cells 9.2 g / l, ESR 26 mm / hour. The ASL-O titre is 625 IU / ML. Acute glomerulonephritis is suspected. Indicate the most likely cause of the disease:
A. Respiratory viruses
B. Enterovirus
C. Streptococcus
D. Staphylococcus
E. E. colon

5. In a child, 10 years, 3 weeks after the streptodermia, edema developed, and the urine of the color of "meat wash" appeared. BP - 130/80 mm Hg, Diuresis - 550 ml. Total urine analysis - protein - 0,85 g / l, eritr. - entirely, L - 8-10, cylinders in hyaline - 1-2. Established preliminary diagnosis: Acute glomerulonephritis of streptococcal etiology. Assign an optimal antimicrobial drug:
A. Sulphanilamides
B. Aminoglycosides
C. Cephalosporins 2 generations
D. Cephalosporins 3 generations
E. "Protected" penicillins

Answers: 1 -A, 2 -B , 3 -B , 4 -C, 5 - E.

6.2. List of recommended reading:

- *b*asic*:*

1. Kliegman, R.M., St Geme, J.W., Blum, N.J., Shah, S.S., Tasker, R.C., Willson, K.M., & Behrman, R.E. (Eds.). (2019). Nelson Textbook of Pediatrics (21st ed.). 4264 p.
2. Ghai ”Essential pediatrics ” 9th Edition. 2019. 814 p.
3. Mayer U, Schmitz J, Bräsen JH, Pape L. Crescentic glomerulonephritis in children. Pediatr Nephrol 2020; 35:829.

- *additional:*

1. Daliriani R., Mahyar A., Ayazi P., Ahmadi G. et al. Neurological Manifestations of renal Diseases in Children in Qazvin // Iranian journal of child neurology (2016), 10(3):24-7 http://www.ncbi.nlm.nih.gov/pubmed/27375752
2. Vogt B. et. al. Nephrology Update: Glomerular Disease in Children. http://www.ncbi.nlm.nih.gov/pubmed/27163763

3. Rovin BH, Caster DJ, Cattran DC, Gibson KL, Hogan JJ, Moeller MJ, et al. Management and treatment of glomerular diseases (part 2): conclusions from a Kidney Disease: Improving Global Outcomes (KDIGO) controversies conference. *Kidney Int*. (2019) 95:281–95. doi: 10.1016/j.kint.2018.11.008.
4. Balasubramanian R, Marks SD. Post-infectious glomerulonephritis. Paediatr Int Child Health. 2017 Nov;37(4):240-247.

6.3. Indicative map for independent work with literature.

№	Main Task	Directions	Answers
1	**2**	**3**	**4**
1.	To get acquainted with the purpose and tasks of the class	To formulate the definition of the GN in children	Formulate the concept of multifactorial disease and its functional character.
2.	Epidemiology	Know the prevalence of GH among children. List the risk factors for the development of GN in children.	The incidence of post-streptococcal glomerulonephritis is an average of 32.4 cases per 100,000 children. In recent decades, in developed countries, there has been a reduction in the incidence of glomerulonephritis to 10-15% of all glomerulonephritis, which is associated with improved socioeconomic conditions. In developing countries, post-streptococcal glomerulonephritis is the cause of 40-70% of all glomerulonephritis. The peak incidence falls on preschool and primary school age (5-9 years), less than 5% of children suffer from glomerulonephritis before the age of 2. Post-streptococcal glomerulonephritis in 2 times more common in boys.
3.	Etiology	List the main etiological pathogens of GN	I infectious Bacteria: beta-hemolytic streptococcus group A, enterococci, pneumococci, staphylococci, corynebacterium, klebsiella, salmonella, mycoplasma, ischemia, meningococci. Viruses: hepatitis B, measles, Epstein-Barr, rubella, chicken pox, cytomegalovirus, and rarely a herpes simplex virus. Parasites: plasmodia malaria, toxoplasma, schistosomi. Mushrooms: candy Non-infectious. Alien proteins

			Serums
4.	Pathogenesis	Determine the paths of pathogens penetration under GN. To characterize the main pathogenetic mechanisms in children with GN.	In the pathogenesis of acute glomerulonephritis in children, two mechanisms can be identified: immunocomplex and nonimmunocomplex.
5.	Classification	Know the classification of GN in children.	Be able to diagnose.
6.	Clinic	Indicate the main symptoms and their characteristics in the state of the GN.	General signs are clinical signs, functional disorders.
7.	Diagnosis	Determine the basic methods of clinical and paraclinical examination at the state of the GN	Be able to evaluate the results of laboratory and functional research methods.
8.	Differential diagnosis	List diseases with similar clinical and laboratory manifestations	Pyelonephritis, interstitial nephritis, hereditary Alport nephritis, Berger's disease
9.	Treatment, prophylaxis	Determine the main directions of treatment, prevention and prophylactic examination for children with GN.	Pay attention to the complexity of diagnosis of the urinary system.

7. Materials for self-control on the quality of training.

A. Questions.

1. Definition of glomerulonephritis.
2. The prevalence and morbidity of glomerulonephritis in children's populations, risk factors for the disease.
3. The pathogenesis of glomerulonephritis.
4. Classification of glomerulonephritis.
5. Clinical picture of glomerulonephritis depending on the form.
6. Treatment programs for the main variants of glomerulonephritis.
7. Complications of glomerulonephritis in children.
8. Differential diagnosis of GN.
9. Prevention and prognosis of GN in children.

B. Tests.

1. A child 6 years of age during the examination revealed lethargy, subfebrel hyperthermia, edema, hypertension, urinary syndrome with oliguria, hematuria with leached red blood cells, selective proteinuria, low leukocyturia; hypoalbuminemia, low leukocytosis, eosinophilia, ESR 30-40 mm / h. This is a clinic:

A. Acute pyelonephritis;
B. Acute glomerulonephritis;
C. Acute cystitis;
D. Amyloidosis of the kidneys;
E. Chronic renal failure.

2. In Sergiyka 10 years after 2 weeks after the suffering quinine there was a headache, lower back pain, body temperature increased to 37.8 ° C. The objective examination revealed pallor of the skin,

the presence of pastosity of the lower extremities, slight puffiness under the eyes in the morning, hyperhidrosis syndrome, obstruction of the tongue, urine of color "meat wash". What disease can you think of?
A. Acute pyelonephritis.
B. Acute cystitis.
C. Acute myocarditis.
D. Rheumatism.
E. Acute glomerulonephritis.

3. In Nicholas 12 years after 2 weeks after the suffocation of quinsy, headache, low back pain, body temperature increased to 37.8 ° C. The objective examination revealed pallor of the skin, the presence of pastosity of the lower extremities, slight puffiness under the eyes in the morning, hyperhidrosis syndrome, obstruction of the tongue, urine of color "meat wash". What research should be conducted first of all to clarify the diagnosis?
A. Test for the activity of the inflammatory process.
B. General blood test.
C. General Urine Test.
D. Zimnitsky test.
E. ECG.

4. A boy, 8 years old, enrolled in inpatient treatment. Two months so he suffered from angina, after which there was a headache, an ailment. Mother noticed the girl's swelling under her eyes. Before admission to the hospital increased swelling on the face; appeared on the limbs and trunk. When looking at the skin pale, edema on the face, limbs and trunk. Heart rate 80 for 1 min, blood pressure 110 and 70 mm Hg In the biochemical analysis of blood protein 50 g / l, cholesterol 10 mmol / l, urea 6 mmol / l, creatinine 0.088 mmol / l. In the analysis of urine a specific gravity 1,010, a protein of 5,8 g / l, leukocytes 4-5, erythrocytes (leached) 20-22, hyaline cylinders 3-4. What syndrome is leading in this patient?
A. Edema.
B. Nephritic.
C. Nephrotic.
D. Isolated uric.
E. Intoxication.

5. A girl, 8 years old, enrolled in inpatient treatment. Two months so he suffered from angina, after which there was a headache, an ailment. Mother noticed the girl's swelling under her eyes. Before admission to the hospital increased swelling on the face; appeared on the limbs and trunk. When looking at the skin pale, edema on the face, limbs and trunk. Pasternatsky sign is positive on both sides. Heart rate 80 for 1 min, blood pressure 110 and 70 mm Hg In the biochemical analysis of blood protein 50 g / l, cholesterol 10 mmol / l, urea 6 mmol / l, creatinine 0.088 mmol / l. In the analysis of urine a specific gravity 1,010, a protein of 5,8 g / l, leukocytes 4-5, erythrocytes (leached) 20-22, hyaline cylinders 3-4. What proteinuria is present in the patient?
A. Massive.
B. Moderate.
C. Average.
D. Selective
E. Postnal.

6. A girl, 12 years of age, after a severe acute respiratory viral infection, drew attention to the urine of red color. When reviewing the deviations in the objective status it was not found. BP - 115/60 mm Hg, Urination painless, 6 times a day. General urine test - protein 0,55 g / l, white blood cells - 6-8,

red blood cells - entirely, salts - uraty, mucus a little q-ty. Acute glomerulonephritis is suspected. Highlight the leading disease syndrome:
A. Increase of hydrophilicity of tissues
B. Hypertension
C. Leukocyturia
D. Hematuria
E. Dizurichny

7. In a boy, 12 years old, 3 weeks after postponed streptodermia appeared macrohematuria, low-grade temperature, abdominal pain. When reviewing the deviations in the objective status it was not found. BP - 140/80 mm Hg . Pasternatsky sign is negative. Urination painless, 6 times a day. General urine test - protein 0,25 g / l, white blood cells - 8-10, Red blood cells - entirely, salts - entirely, mucus is a small amount. Set up a preliminary diagnosis:
A. Acute glomerulonephritis
B. Dismetabolic nephropathy
C. Acute pyelonephritis
D. Nervous stomach disease
E. Acute cystitis

8. In a boy of 3 years, swelling on the face, lower limbs, anterior abdominal wall, ascites are observed. BP - 90/50 mm Hg, urine test - a protein of 4.2 g / l, white blood cells - 5-6, erythrocytes - 8-10, cylinders - hyaline 2-3. The blood protein is 48,6 g / l. Cholesterol of blood - 8.2 mmol / l. Established preliminary diagnosis: Acute glomerulonephritis. Specify the form of the disease:
A. Nephritis syndrome
B. Nephrotic syndrome
C. Nephritis syndrome with hematuria
D. Nephrotic syndrome with hematuria and hypertension
E. Isolated urinary syndrome

9. In a boy of 3 years, after an ARVI there was an edema syndrome like anasarka. BP - 90/50 mm Hg. Urine test - a protein of 5.2 g / l, leukocytes - 5-6, erythrocytes - 6-8, Cylinders - hyaline - 2-3. Total blood protein - 41.8 g / l, albumin - 40.1%. Blood cholesterol -9.3 mmol / l. A preliminary diagnosis was established: Acute glomerulonephritis with nephrotic syndrome. Specify the leading mechanism of development of proteinuria in a child:
A. Damage to the interstitial tissue of the kidneys
B. Reduction of oncotic pressure
C. Damage to the basement membrane of the glomerular capillaries
D. Strengthening catabolic processes in the body
E. Renal intravascular coagulation

10. A 2-year boy suffering from food allergy has a pronounced anasarca-type edema syndrome. BP - 90/50 mm Hg. Common an. urine - protein 5.2 g / l, leukocytes - 5-6 per field, erythrocytes - 6-8 per field, cylinders - hyaline 2-3 per field. Total blood protein - 41.8 g / l, albumin - 40.1%. Blood cholesterol -9.3 mmol / l. A preliminary diagnosis was established: Acute glomerulonephritis with nephrotic syndrome. Specify the main direction of pathogenetic therapy for a child:
A. Saluretics
B. GCS
C. Anticoagulants
D. Quinoline preparations
E. Antihistamines

Answers: 1 -B, 2 -E, 3 -C, 4 -C, 5 -A, 6 -D, 7 -A, 8 -B, 9 -C, 10 -B.

B. Tasks for self-control with answers.

1. A child of 8 years entered the department with complaints of weakness, increased fatigue, loss of appetite, headache, pink color of urine. A week before the disease was affected by ARVI. When reviewing BP - 130/90, skin is pale, facial pastosus, in the general analysis of blood - hemoglobin 105 g / l, red blood cells 3,2 t / l, leukocytes -10,5; ESR-30 mm / h, in the general analysis of urine - specific gravity - 1025; protein-0,99; white blood cells - 10; erythrocytes-50; cylinders for hialin 7-8;

A. Determine the preliminary diagnosis.

B. Speed of glomerular filtration, calculation for Schwartz.

C. The plan for laboratory examination of a child.

D. Instrumental survey.

E. Lines of dispensary observation.

Etalon response:

A. Acute glomerulonephritis with nephritic syndrome

B. GFR = 0,0484 x height (cm) / creatinine of blood (mmol / l)

C. Biochemical blood analysis with proteinogram, cholesterol, creatinine, urea; glomerular filtration rate (GFR); coagulogram; immunological examination with determination of ASL-O, IgG, IgM, IgA, complement (C3-fraction); Research of daily protein excretion, urine analysis according to Nechiporenko, Zimnitsky test

D. Control of blood pressure, body weight; ECG; Ultrasonography of the kidneys and organs of the abdominal cavity

E. Dispensary observation for 5 years after normalization of clinical and laboratory parameters

2. In a child of 3 years, which was considered previously healthy, without noticeable provocative factors, swelling appeared on the face, trunk, legs. The general condition is hardly broken. BP - 90/60 mm Hg During examination, proteinuria was detected up to 4 g / day, hypoproteinemia, hypercholesterolemia.

A. Determine the preliminary diagnosis

B. Make a patient survey plan

C. List the groups of drugs for pathogenetic program treatment

D. Calculation and scheme of the appointment of glucocorticoids, taking into account the circadian rhythm of the adrenal glands

E. Variants of nephrotic syndrome depending on reaction to program therapy of glucocorticoids

Etalon response:

A. Nephrotic syndrome

B. Biochemical blood test with the definition of creatinine, urea; calculation of glomerular filtration rate (GFR); liver tests (ALT, AST, bilirubin and its fractions); hematocrit; coagulogram; study of electrolyte composition of blood (potassium, sodium, chlorine); immunological examination with determination of ASL-O, IgG, IgM, IgA, complement (C3-fraction); Urine analysis by Nechiporenko, Zimnitsky test; bacteriological examination of urine; ECG; Ultrasound of the kidneys and organs of the abdominal cavity; blood pressure control, body weight;

C. Glucocorticoids; cytostatics; antiagregants and anticoagulants; oxyquinoline derivatives.

D. 1.5 - 2 mg / kg / day (for prednisolone). Dosage: 9.00 - 50%; 12.00 - 30%; 15.00 - 20%.

E. Hormonal; partially hormone-susceptible; hormone-resistant; hormone-negative hormone-dependent

3. Sasha M.parents, 3 years, turned to the neurologist with complaints of lethargy, swelling in the face and legs, dreams of appetite, pain in the abdomen.

From the anamnesis it was revealed that the boy often suffers from colds, and two weeks ago he suffered a sore throat. Treatment at home: biseptol, pharyngosept, multivitamins. Genealogical and social anamnesis without features.

Objectively: the condition is of medium severity, the skin is pale, the blue under the eyes, the eyelids are swollen, the swelling on the legs. In the throat - mucous physiological stains, lymph nodes up to 0.5 cm in diameter, slightly painful, not soldered to the surrounding tissue. Subcutaneous fat layer is developed satisfactorily. From the side of the heart and respiratory organs, pathology has not been revealed. The abdomen is soft at palpation, there is a slight soreness, the liver and spleen are not enlarged.

In the general analysis of urine: protein 14 g / l, relative density 1030, alkaline reaction, erythrocytes up to 20 in the field of view, leukocytes 8-10 in the field of view hyaline cylinders.

In a general blood test: E-$4.0x10^{12}$ / L, HB-100 g / L, Le-$4.7x10^{9}$ / L, ESR-69 mm / h. Biochemistry of blood: residual nitrogen 35.7 mmol / l, urea 13.48 mmol / l, total protein in blood 46.8 g / l.

A. Formulate and justify a preliminary diagnosis.

B. Name additional symptoms to clarify the diagnosis, tell us about the method of their detection.

C. Tell us about the amount of pre-hospital care and the rules of transportation to destination.

D. Make a plan for diagnostic studies in the hospital, tell about the patient's preparation for them and the principles of treatment.

E. Tell about the method of sampling in Zimnitskiy test.

Standards of answers:

A. Acute glomerulonephritis. The conclusion is based on the history and complaints: swelling in the face and legs, lethargy, decreased appetite, abdominal pain. The provoking factor of the onset of this disease was postponed 2 weeks ago angina. Data objective examination: pallor of the skin, "blue" under the eyes, puffiness of the eyelids, legs, moderate soreness with palpation of the abdomen. Laboratory test data: in the analysis of urine, the high protein content up to 14 g / l, the appearance of erythrocytes up to 20 in the field of vision, hyaline cylinders, in the analysis of the blood ESR-69 mm / h (sharply accelerated), HB-100 g / L (reduced) , the biochemical analysis of blood, the residual nitrogen was increased to 35.7 mmol / l, urea increased to 13.48 mmol / l, the total protein decreased to 46.8 g / l.

B. Additional symptoms that can be detected: increased blood pressure, headache, oliguria, dysuric manifestations, fever.

C. The child must be hospitalized. First aid is the symptomatic treatment and trans-porting of the patient to a hospital in a prone position.

D. Diagnostic and treatment program in the hospital:

Diagnostic program

- general blood analysis
- general urine analysis;
- urine analysis in Zimnitskiy and Addis-Kakovskiy;
- Daily protein excretion;
- Biochemical blood test (determination of total protein and protein fractions of urea, creatinine, residual nitrogen, CRP)
- clearance of creatinine
- coagulogram;
- Kidney ultrasound

excretory urography;

- kidney biopsy by indicators;
- consultation of ENT, genetics, urologist;
- treatment program
- bed rest for 3-4 weeks;

- With a diet to exclude table salt, irritating, extractive, allergenic products, the intake of protein to reduce the amount of liquid to limit;

- basic therapy: diuretics, antihypertensives, antibiotics, ascorutin;

- In severe cases, pathogenetic therapy: corticosteroids.

After discharge from the hospital, the child should be observed at the nephrologist, from dispensary registration to remove after 5 years, if within the last year there is remission.

E. The trial according to Zimnitsky is carried out according to the algorithm of manipulation.

4. A 13-year-old girl consulted a nephrologist with complaints of overweight, the appearance of a large amount of protein in urine tests when trying to stop taking glucocorticoids. Patient from 3 years, when after the transferred angina there were edemas, oliguria, proteinuria up to 14 g / l. Since then, constantly receives 15 mg of prednisolone per day.

Objectively: well-being is satisfactory. The skin of the face is red, with cyanotic striae on the forehead, hips and lateral surface of the abdomen. The subcutaneous fat layer is unevenly developed: excessive deposition in the face, neck, chest, abdomen. Lymph nodes are not enlarged. Nasal breathing is not disturbed. Palpation, percussion and auscultation of changes on the part of the respiratory organs were not detected. The area of the heart is not changed and the borders are not expanded. The tones are clear, rhythmic. BP - 115/60 mm Hg on both hands. The abdomen is soft, painless. The liver and spleen are not enlarged. Stool and urination are not violated.

In general analysis of urine without pathology. Nitrogen excretory function is not broken. There are no abnormalities in the protein blood spectrum, lipid and blood sugar levels.

A. Formulate and justify a preliminary diagnosis.

B. Name additional symptoms to clarify the diagnosis, tell us about the method of their detection.

C. Tell us about the amount of pre-hospital care and the rules of transportation to destination.

D. Make a plan for diagnostic studies in the hospital, tell about the patient's preparation for them and the principles of treatment.

Standards of answers:

A. Chronic glomerulonephritis, nephrotic form, hormone-dependent, without disturbance of renal function. The conclusion is based on the history of the disease: in urinalysis, the appearance of a large amount of protein when trying to stop taking glucocorticoids, is recorded in the nephrologist from 3 years after the angina, when edema, oliguria and proteinuria reached 14 g / l. Constantly received prednisolone at 15 mg per day. Objective data: the skin of the face is red, cyanotic striae are noted on the skin surface, the subcutaneous fat layer is excessively developed. Respiratory organs and heart were not found. Stool and siechevipuskanney not broken, the analysis of urine without pathology. Biochemical blood test without changes.

B. Additional symptoms that can be detected in this disease:

- pain in the abdomen and lower back;

- microhematuria, proteinuria

- Increased blood pressure;

- dysuria

myocardial hypertrophy

- appearance of Cushing's syndrome;

C. First aid: during the exacerbation of chronic glomerulonephritis, hospitalization is indicated. Introduction of diuretics, hypotensive drugs. Transportation to the nephrologic department in uncomplicated form in the sitting position.

D. Diagnostic program. General analysis of blood and urine, urine tests for Zimnitskiy and Addis-Kakovskiy, daily protein excretion, biochemical blood analysis (total protein and its fractions, urea, residual nitrogen, creatinine, CRP, cholesterol, sialic acids), creatinine clearance, coagulogram, ultrasound kidney and immunogram, excretory urography, consultation of ENT, genetics, urologist, kidney biopsy according to indications.

Therapeutic program:

- bed rest 3-6 weeks
- diet with protein restriction to 1.0-1.5 g / kg and salt exclusion, after improvement, a diet with a protein restriction of up to 2 g / kg and sodium chloride up to 3 grams per day is prescribed. The diet excludes meat, fish and mushroom decoctions, as well as products, often cause allergic reactions (chocolate, citrus fruits, strawberries, carrots, eggs, etc.);
- severely restrict physical activity;
- pathogenetic therapy: immunosuppressants, anticoagulants, disaggregants. Restore and do not stop taking prednisolone at 15 mg per day
- if necessary, to appoint non-steroidal anti-inflammatory drugs: brufen, orthophene, indometacin, diuretics, antihypertensives and cardiac drugs.

After discharge from the hospital, the child is observed with a nephrologist, constantly takes prednisolone, a blood test is performed on a monthly basis, the urine is analyzed, blood pressure is measured, biochemical blood test, Zimnitsky test once a quarter, and the endogenous creatinine clearance is determined. Investigate the fundus 2 times a year. Ultrasound and X-ray examination by the appointment of a nephrologist.

5. A 10-year-old child with a complaint of headache, fatigue, and puffiness of the face was hospitalized on the fourth day of the illness. In the general analysis of urine hematuria. Arterial pressure 160/100 mm Hg.

A. Preliminary diagnosis
B. Make a plan for examining the patient
C. Determine the hematuria
D. Criteria of nephritic syndrome
E. List the groups of drugs used to correct arterial hypertension

The standard of the answer:

A. Acute glomerulonephritis
B. Biochemical analysis of blood with the study of the parameters of nitrogen metabolism of electrolytes; glomerular filtration rate (GFR) liver tests; coagulogram; Immunological examination with the determination of ASL-O, IgG, IgM, IgA, complement (C3-fraction); Study of daily protein excretion, urine analysis according to Nechiporenko, Zimnitsky's test, bacteriological urine test Control of blood pressure, ECG body weight; Ultrasound of the kidneys and organs of the abdominal cavity; examination of the fundus; radionuclide studies;
C. Insignificant 15-20 erythrocytes and the field of view is moderate 25-100 erythrocytes and the field of view is significant more than 100 erythrocytes and the field of view.
D. Edema, hypertension, hematuria, proteinuria (up to 1.5-2 g / day), a violation of the nitrogen excretory function of the kidneys
E. Blockers of angiotensin II receptors (irbesartan 2-6 mg / kg / day, losartan 1-2 mg / kg / day, candesartan 0.5 mg / kg / day, telmisartan 1 mg / kg / day). Inhibitors of ACE-IAPF (enalapril 0.1-0.3 mg / kg / day, lisinopril 0.1-0.3 mg / kg / day; Quadropril 0.05-0.1 / kg / day, monopril 0.2-0 , 3 mg / kg / day, moexipril 0.05-0.1 / kg / day). Calcium blockers (diltiazem 5-8 mg / kg / day, felodipine 0.1-0.2 / kg / day). Moxonidine (Physiotens) 0.005 mg / kg / day. Loop diuretics (furosemide 1-2 mg / kg / day).

8. Materials for classroom self-study.

8.1. The list of educational practical tasks that must be completed during practical exercises.

1. Collect history, highlight the points that indicate the nature of the inflammatory disease.
2. Identify the most informative features of the disease during objective and laboratory and instrumental examination of the patient.
3. Clinical diagnosis in modern classification.

9. Instructional materials for mastering professional skills.

9.1. Methodology for performing work, stages of implementation

1 .Evaluate the data and medical history of the disease, seen risk factors that play a role in causing the disease.
2. Conduct the clinical examination of the patient.
3. Make a plan to further investigation.
4. Evaluate the results of laboratory and instrumental examination of the patient.
5. Formulate a clinical diagnosis according to the classification

10. Materials for self-control of mastering knowledge, abilities, skills.

10.1 Tests.

1. Which of the given nosological forms is not included in the classification of acute glomerulonephritis:
A. GN with nephritic syndrome
B. GN with nephrotic syndrome
C. GN with isolated urinary syndrome
D. Interstitial nephritis
E. GN with nephrotic syndrome, hematuria, hypertension

2. The boy is 11 years. 2 weeks after the transferred streptococcal angina was hospitalized in the nephrologic department in connection with acute glomerulonephritis. The most probable pathogenetic basis of the disease?
A. Violation of urodynamics
B. Immediate damage to glomerulus streptococcus
C. Circulatory or histotoxic hypoxia of renal tissue.
D. Damage of the basement membrane by glomerul antibodies or immune complexes
E. Violation of renal hemodynamics and trophic disorders

3. A 6-years boy received complaints about facial swelling, headaches, red color of urine. When examined, blood pressure 140/90 mm. In urine analyzes, the protein is 1.2 g / l, red blood cells are all over the field of view. What is the most likely diagnosis?
A. Nephrolithiasis
B. Acute glomerulonephritis
C. Tuberculosis of the kidneys
D. Interstedium nephritis
E. Pyelonephritis

4. A 7-years boy is on treatment for a month. At admission, pronounced edema was observed, proteinuria - 7,1 g / l, protein in daily urine - 4,2 g. Biochemical analysis of blood contains hypoproteinemia (43,2 g / l), hypercholesterolemia (9,2 mmol / l). Which of the listed variants of glomerulonephritis is more likely to occur in a patient?
A. The hematuric
B. Nephritic
C. Isolated urinary tract
D. Nephrotic
E. Mixed

5. A boy 8 years old entered the children's department with complaints of large swelling. Previously, the child was treated three times for relapses of the nephrotic syndrome. 2 times - GCS (GC), the last time GC one cytostatic with a positive effect. The study revealed proteinuria - 6 g / day, total blood protein - 48 g / l, cholesterol - 8.9 mmol / l. Which of the following is the most acceptable method of treatment for a sick child?

A. GCS
B. GCS + cyclosporine
C. Indomethacin
D. Delagil
E. GCS + cytotoxic agents

Answers: 1 -D, 2 -D, 3 -B, 4 -D, 5 -E