MINISTRY OF HIGHER EDUCATION, SCIENCE AND INNOVATION OF UZBEKISTAN
MINISTRY OF HEALTHCARE OF UZBEKISTAN
TASHKENT MEDICAL ACADEMY
DEPARTMENT OF FACULTY AND HOSPITAL THERAPY № 2, NEPHROLOGY AND HEMODIALYSIS

EDUCATIONAL-METHODICAL COMPLEX

on the module

INTERNAL DISEASES

(For the 5thcourse)

Field of knowledge:	110000 - **Pedagogics**
Branch of education:	510000 - **Healthcare**
Direction of education	5511100- **Professional education** (5510100–
General Medicine)	

TASHKENT – 2023

The working program of the discipline is based on the model program of the discipline of "Therapy", approved by the order No. 107 (Appendix No. 2) of the Ministry of Healthcare of the Republic of Uzbekistan dated Aprel 25, 2019.

The educational-methodical complex was reviewed and approved at the meeting of the Central Methodical Council of TMA on september 19, 2023

"CONFIRMED"
A vice-rector for academic affairs
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2023

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CONTENTS OF THE CURRICULUM COMPLEX FOR THE 5th COURSE OF INTERNAL DISEASES

1. The relevance of the discipline and its place in vocational education

One of the main goals of the national training program is to educate a highly spiritual person, comprehensively developed in medicine, to form his scientific outlook.

Internal medicine is a science that teaches etiology, pathogenesis, classification, clinical course, laboratory diagnostics, clinical recommendations, consequences, complications, prevention, emergency care of common diseases and plays a big role in shaping thinking and enriching the worldview of a general practitioner.

This curriculum is based on the State Educational Standard of the Republic of Uzbekistan and qualification requirements for higher education. On the basis of this program, students will learn to apply modern pedagogical technologies, in the educational process it allows the student to apply the theoretical knowledge gained in practice. With the help of modern medical technologies, the acquired skills are combined with clinical practice.

For the teaching of internal medicine, the subjects of histology, human anatomy serve as the basis for theoretical subjects and internal medicine serves as a theoretical basis for other clinical subjects.

2. The purpose and objectives of the discipline

The purpose of teaching the subject is to form and the ability to substantiate the diagnosis of various variants of typical diseases, as well as in the atypical course of some common diseases, and to teach them the complications and principles of treatment.

Objective of the subject:

In accordance with the specified goals and objectives, after completing the study of the discipline of internal diseases, the student should know:

-etiology, pathogenesis, clinic, complications, prognosis, principles of treatment of the most common diseases of internal organs;

-be able to collect anamnesis and examine the patient by systems to identify the main diagnostic criteria for the disease;

- master the methods of examination and the formation of a preliminary and clinical diagnosis;

- to know and be able to draw up a plan of examination, medical tactics and the appointment of complex treatment;

-be able to interpret the results of additional research;

-Know and be able to use the principles of differential diagnosis and final diagnosis;

-Know basic medical records.

-be able to recommend treatment for various types of internal diseases and teach emergency care.

Guidelines for teaching the subject

Improving the quality of medical education by updating curricula is an ongoing process. HEIs must respond to evolving medical needs, improved practice and scientific advances. Educators need to understand and analyze the relevance of changing circumstances to medical practice and medical education, and they need to update their curriculum accordingly.

№	Lectures topics	The lectures volume
9-semester		
1	Chronic xolecyctitis	**2**
2	Nephrotic syndrome.	**2**
	Total:	**4 hours**

Used foreign literature:

A copy of the following topics was received from Harrison for the 9 semester practical training:

Chronic cholecystitis

The gallbladder is an anatomical part of the liver. This sac-shaped organ is necessary for the deposit of bile synthesized by liver cells, and the excretion of fluid into the small intestine during digestion. The release of bile into the duodenal cavity contributes to the digestion of fatty foods and improved absorption of useful substances. In the organ there is a constant accumulation of secretion, leading to an increase in the viscosity of the fluid. After food enters the intestine, hormones stimulate the relaxation of special valves and the release of bile into the GI tract through special ducts.

Other functions of the organ:

- Maintaining metabolism.
- Improvement of small intestinal motility.
- Excretion of excess cholesterol and bilirubin from the body.
- Activation of enzymes needed to digest protein foods.

The gallbladder is often subjected to pathological effects. Violation of the muscular shell of the organ can impede the secretion of bile - such a process not only harms digestion, but also contributes to the formation of stones that can further completely block the bile ducts and damage the inner membranes of the gallbladder. Normally, the organ does not contain microflora, but with diseases and anatomical defects, pathogenic and opportunistic microorganisms from the intestine may penetrate into the gallbladder.

Causes of occurrence

Various factors can provoke an inflammatory process in the gallbladder. First of all, it is stagnation of bile, which disturbs the functions of the organ and contributes to the emergence of infection. Escherichia coli, streptococci, giardia and other pathogens can penetrate into the organ from the intestine. In this case, the occurrence of infection can be a direct cause of inflammation or a consequence of such a pathological process.

Possible causes:

- Biliary tract obstruction due to an anatomical defect, stones, or valve malfunction.
- Gallstone disease is a common pathology of the gallbladder. Concretions can form in the organ due to a violation of the chemical composition of bile and stagnation of secretion.

- Malignant or benign tumor. A growing neoplasm may impede evacuation of bile from the organ.
- Primary infections. A variety of viruses can infect the gallbladder in patients with HIV infection.
- Violation of motility of the gallbladder and its ducts. With insufficient or chaotic contractility of smooth muscles of the organ, the outflow of bile into the intestine is impeded.

Chronic form of inflammation can occur if the patient did not treat acute cholecystitis. Such pathology persists for many years and significantly worsens the quality of life of a person.

Risk factors

There are various forms of predisposition to the disease, associated with individual characteristics of a person, primary pathologies, nutrition and heredity. Doctors necessarily take into account the presence of risk factors for cholecystitis during examinations.

Key risk factors:

- Female gender and age between 25 and 45 years old.
- Obesity and significant weight loss over several months.
- Taking certain medications. In particular, the risk of inflammation in the gallbladder increases with the use of hormonal drugs.
- Pregnancy.
- Chronic diseases of the intestines, liver and pancreas.
- Surgical treatment of abdominal organs, trauma.
- Chronic foci of inflammation in different parts of the body.
- Prolonged administration of parenteral nutrition.
- Improper diet or prolonged fasting.
- Alcoholic beverage abuse.
- Myocardial infarction and other heart diseases.
- Vascular abnormalities in diabetes mellitus.
- Abnormal throwing of pancreatic secretion into the gallbladder (pancreatobiliary reflux).
- Lack of physical activity.

Effective measures for the prevention of pathology are based on the elimination of risk factors, related to lifestyle and individual patient history.

Forms of the disease

Doctors classify chronic cholecystitis based on the cause of inflammation and the nature of the course of the disease. There is also a classification based on the severity of symptoms.

Basic Forms:

- Chronic calculous cholecystitis is the most common variant of the disease, arising due to blockage of the excretory ducts of the organ by concretions. It is characterized by pronounced symptomatology during exacerbations. The inflammatory process can spread to neighboring anatomical structures, including the diaphragm and pleura.
- Chronic non-calculous cholecystitis is a rarer form of the disorder, often diagnosed in patients with severe disease. In this case, the inflammatory process occurs due to trauma, surgical intervention, severe infection and other causes not related to the formation of stones. With untimely treatment, non-calculous cholecystitis can cause the death of the patient.

The danger of chronic inflammation is due to the sterile symptomatology. Patients pay attention to unpleasant sensations only during exacerbations, as a result of which the disease gradually progresses and causes complications.

Symptoms and signs

The symptomatic picture of the disease depends on the severity of the inflammatory process, the age of the patient and the frequency of exacerbations. The predominant sign is usually pain in the right subcostal region, spreading to the back and central abdomen. If the inflammation affects the diaphragm, there may be pain in the right arm and scapula.

Additional attributes:

- Tension in the abdominal muscles.
- Severe weakness, fatigue.
- The appearance of cold sweat.
- Nausea and vomiting.
- Lack of appetite.
- Liquid stools.
- Abdominal bloating.
- Heart palpitations.
- Shortness of breath.
- Low blood pressure.
- Yellowing of the skin and mucous membranes.

- Chest pain.

Due to insufficient intake of vitamins and minerals in the body, symptoms such as pale skin and constant fatigue occur. Against the background of the development of complications, more severe pathological signs appear. With prolonged sharp pain in the abdomen and persistent fever should seek medical help as soon as possible.

Diagnosis

Necessary examinations can be prescribed to the patient by a gastroenterologist. During the appointment, the doctor will ask the patient about complaints and study anamnestic information. Physical examination allows you to detect jaundice, abdominal bloating and pain in certain parts of the body. Based on the data obtained, the doctor will prescribe the necessary instrumental and laboratory tests.
Necessary diagnostic manipulations:

- Ultrasound examination of the gallbladder and bile ducts. The advantages of the method are safety and real-time imaging of the organs. The doctor can immediately detect gallstones in the gallbladder, changes in the walls of the organ and other pathological signs indicating cholecystitis.
- Blood tests to detect inflammation and infection. With pronounced inflammation in the blood, the number of leukocytes increases. The concentration of liver enzymes is also a diagnostic criterion.
- Collection of bile from the organ by duodenal probing. The obtained material is sent by a specialist to the laboratory for detection of pathogenic microflora.
- X-ray examination of the gallbladder (cholecystography). The doctor receives information about the size, shape and functional activity of the organ. With the help of this study it is possible to detect anatomical anomalies, cholelithiasis or other diseases.

If the doctor is unable to make a diagnosis after requesting the results of the above tests, an additional appointment of gastroscopy, CT scan or laparoscopy is possible. It is necessary to determine the cause of inflammation as accurately as possible.

Treatment

In chronic cholecystitis, the main method of treatment is a special diet that reduces the negative impact on the organ. Medications may be prescribed to eliminate infection and inflammation. If the disease has caused dangerous complications,

such as gallbladder gangrene or peritonitis, it is necessary to perform surgical intervention.

Main Assignments:

- Therapy with the help of therapeutic diet. The patient needs a frequent fractional diet. It is necessary to exclude the constant use of fatty foods, alcohol, beans and fried meat. Observe the diet should be followed for a long time to prevent a relapse of the disease. When removing the gallbladder, patients are prescribed a lifelong diet.
- Antibiotics and anti-inflammatory drugs. For chronic infection, it is recommended to obtain a microflora sample by probing and select an effective antimicrobial agent by laboratory test.
- Antispasmodics and pain relievers to relieve symptoms.
- Diuretic medicines. Medicines of this group are used to improve the excretion of bile into the duodenum.

The main methods of surgical treatment of cholecystitis doctors include removal of the gallbladder or removal of concretions blocking the ducts of the organ. In severe complications of the disease may require complex open intervention in the abdominal cavity.

Complications

Chronic cholecystitis can provoke the development of severe complications even if the symptoms are mild. The main danger is the focus of infection, which can spread to other organs.

Major complications:

- Gangrene of the gallbladder - destruction of the tissues of the organ, leading to a purulent process in the abdominal cavity.
- Peritonitis - extensive inflammation of the peritoneum, causing severe symptoms and intoxication of the entire body. This pathology can occur against the background of perforation of an inflamed gallbladder.
- Inflammation of the pancreas due to infiltration of gallbladder contents into the organ.
- A severe infectious process in which pathogens spread with the bloodstream (sepsis).

Timely surgical treatment can prevent the development of such complications.

Thus, chronic cholecystitis is a frequent consequence of acute inflammation of the gallbladder. Symptomatology of the disease persists for many years and negatively affects the quality of life of a person. Consultation with a gastroenterologist will help the patient to choose an effective treatment for the disease.

Nephrotic syndrome

Nephrotic syndrome is a condition characterized by generalized edema, massive proteinuria (above 50 mg*kg/day or above 3.5 g/day), hypoproteinemia and hypoalbuminemia (less than 20 g/L), hyperlipidemia (cholesterol above 6.5 mmol/l). The term was proposed by E. M. Tareyev in 1923.

Characterized by lesions of the renal tubular apparatus.

Diagnosis

Diagnosis is based on detected changes in blood and urine tests (proteinuria, hyperlipidemia, hypoproteinemia) and clinical data. The clinic of MINS (NS with minimal changes) unfolds gradually, and extrarenal symptoms predominate, especially edematous: there are increasing edema, initially eyelids, face, lumbar region (later may reach the degree of anasarca - widespread edema of subcutaneous tissue), genital organs, ascites, hydrothorax, less often - hydropericardium.. Characterized by significant hepatomegaly due to liver degeneration. The skin becomes pale ("pearlescent" pallor) in the absence of anemia, dry, there are signs of hypovitaminosis A, C, B1, B2, dystrophic changes. There may be brittleness and dulling of hair, on the skin - cracks, from which the fluid leaks out, striae distensae. The child is lethargic, does not eat well, develops dyspnea, tachycardia, systolic murmur at the apex ("hypoproteinemic cardiopathy").

A severe complication in patients with anasarca, i.e., marked hypoproteinemia, may be hypovolemic shock, which is preceded by anorexia, vomiting, sharp abdominal pain. In the observations of N. D. Savenkova and A. V. Papayan (1997) abdominal pain syndrome develops in 23.5% of children with hypoalbuminemia less than 15 g / l, and migrating rye-like erythema in 33.3%, thrombotic episodes in 12.5%, OPN in 3.3% of children with the same severity of hypoalbuminemia, while nephrotic hypovolemic shock is noted only at a serum protein level of less than 10 g / l (5%). As the edema subsides, the decrease in skeletal muscle mass becomes more noticeable.

Arterial blood pressure is usually normal; only up to 10% of children may have transient arterial hypertension. Serum albumin levels in these children are less than 10 g/L.

The content of total protein in plasma (serum) is reduced sometimes up to 40 g / l.

Especially sharply reduced concentration of albumin and g-globulin, while the level of a_2 -globulin is elevated, that is, there is a sharp dysproteinemia. Blood serum has a milky color, in it are found high levels of lipids, cholesterol,

fibrinogen. The level of blood nitrogenous slag is usually normal, and the content of potassium and sodium is reduced. COE is sharply increased (up to 50-70 mm/hour).

Renal symptoms are. oliguria with high relative density (1.026-1.030) of urine and marked proteinuria. In the study of glomerular filtration by endogenous creatinine normal and even elevated values are obtained, but this is a false impression. If we take into account the degree of proteinuria, the glomerular filtration in MINS is always reduced.

Clinical presentation, course and outcome of NS complicating diffuse glomerulonephritis glomerulonephritisare different from those of MINS.

Urinary syndrome in MINS is composed of the following symptoms:

1. proteinuria,

2. oliguria with high relative urine density,

3. cylindruria.

Proteinuria in MINS is usually selective, i.e. plasma proteins with a molecular weight of less than 85,000 (albumin and its polymers, prealbuminsiderophilin, haptoglobin, transferrin, a_1 - and b-globulins, a_1 - and a_2 - glycoproteins, etc.). In most cases, children with selective proteinuria have a better prognosis and are sensitive to glucocorticoid therapy. In the genesis of proteinuria is important and impaired reabsorption of protein in the renal tubules. Non-selective proteinuria, when there are many large-molecule proteins in the urine, is usually a consequence of fibroplastic process, sclerosis, that is uncharacteristic for MINS. Recall that a healthy child over 4 years of age may have up to 100-150 mg of protein in the daily urine.

Oliguria is associated with hypovolemia., hyperaldosteronism.and tubular damage. Due to proteinuria, the relative density of urine is increased, reaching 1.040. The activity of ADH in the blood of patients is high.

Sometimes in NS there is massive leukocyturiadue to an immunopathologic process in the kidneys. Leukocyturia is more often transient and is not associated with bacterial infection, i.e. pyelonephritis. The frequency of leukocyturia and erythrocyturia in MINS according to different authors does not exceed 10%.

If there is a large amount of protein in the urine, it can be coagulated in the tubules, taking their shape; on this cast is layered fatty peregenerated renal epithelium - so formed hyaline, granular and waxy cylinders.

Edema. Massive and prolonged albuminuria in a patient with NS eventually inevitably causes hypoproteinemia, as protein loss exceeds the intensity of its

synthesis. Hypoproteinemia leads to a disruption of the Starling equilibrium between hydrodynamic, filtration and colloid-osmotic pressures. This leads to a predominance of fluid outflow from the arterial bed over inflow. Edema begins to appear when albumin levels fall below 27 g/L plasma and develops always if hypoalbuminemia reaches 18 g/L.

Secondary hyperaldosteronism also plays an important role **in the pathogenesis of edema.** hyperaldosteronismtypical of NS. As a result of it in the body retains sodium and, consequently, water, although there is in the blood hyponatremia.

Hypoproteinemia. The main cause of hypoproteinemia in patients with NS is a large loss of albumin with urine and their transfer to tissues. In addition, increased catabolism of albumin, impaired protein synthesizing function of the liver are also important.

Hyperlipidemia. Some authors attribute the increase in the level of low and very low density lipoproteins, cholesterol and lipids (free fatty acids, triglycerides, phospholipids, etc.) in NS to liver dysfunction, others explain this phenomenon by decreased thyroid function. Due to the fact that intravenous administration of albumin solution prevents the increase of hypercholesterolemiaIt is assumed that the increase in blood cholesterol levels is compensatory due to a decrease in albumin content. Since lipidemia in experiment can be obtained after ureteral ligation, it is suggested that hypercholesterolemia and lipidemia in MINS are of renal origin and depend on damage to intermediary metabolism in the tubule enzyme system. Low blood levels of lecithin-cholesterol acetyltransferase, which is excreted in large amounts in the urine, and low activity of lipoproteidlipase are also important in the genesis of hyperlipidemia. lipoprotein lipase. Type IIa and IIb hyperlipidemias are commonly diagnosed in IDDM.

Disorders of phosphorus-calcium metabolism (hypocalcemiaosteoporosis, osteomalacia) are caused by impaired renal function and vitamin D metabolism.

Disorders of iron and trace element metabolism with low blood levels of both iron and zinc, copper, cobalt determine to a large extent the propensity of such patients to anemia, trophic skin disorders, growth retardation, and possibly immunodeficiencies.

Blood viscosity in MINS is increased due to hyperlipidemia, increased platelet adhesiveness. At the same time, the levels of clotting factors (procoagulants) and anti-clotting factors (antithrombin III, proteins C and S) are reduced, which explains the relatively low incidence of decompensated DIC in MINS.

Infections are previously one of the very frequent complications of MINS. Peritonitis was especially frequent, which was caused in most cases by pneumococci, but in 25-50% of cases by E. coli.

Diagnosis

The characteristic clinical and laboratory picture of MINS in the vast majority of cases (90-95%) in children 2-7 years of age allows diagnosis without renal biopsy. A good and rapid response to therapy with glucocorticoids confirms the diagnosis. At the same time, it is advisable to determine the level of IgE in any child with NS, find out the presence of chronic persistent viral infections (hepatitis B , cytomegalyherpes-virus infections, etc.), as positive results significantly complement and modify therapy. The recurrent course of NS is indicated by 2 relapses per year, and frequently recurrent - 3 or more relapses per year. Remission is stated in the absence of proteinuria or its value of less than 4 mg/m^2 per hour and achieving a serum albumin level of 35 g / L. Biopsy is indicated in children with NS under one year of age and over 12 years of age, because they have a very low incidence of MINS.

Differential diagnosis

First of all, Nephrotic syndrome must be differentiated from glomerulonephritis.Nephrotic syndrome should be differentiated from glomerulonephritis, SLE, renal amyloidosis, interstitial nephritis, hepatic vein thrombosis.

Treatment

- Diet - in case of kidney dysfunction, restriction of fluid intake, salt-free, age-optimal amount of protein
- Infusion therapy (albumin, reopolyglucin etc.)
- Diuretics

Diuretics play a major role in the treatment of renal disease, but uncontrolled and prolonged use may result in acute sodium loss and decreased circulating blood volume, hypokalemia and metabolic acidosis. Fasted diuresis with large doses of diuretics, as well as ultrafiltration, in conditions of severe hypoalbuminemia or severe renal failure may be complicated by difficult to control hypovolemic shock or further decrease in glomerular filtration. Therefore, treatment with diuretics is recommended for the shortest possible duration and should be resumed only in cases of markedly decreased diuresis and increasing edema.

For the treatment of nephrotic edema usually used furosemide - 20 - 400 mg orally, 20 - 1200 mg intravenously), which has a fairly powerful and rapid, albeit short-term, action. Similar to furosemide also acts ethacrynic acid (50 - 200 mg / day). Weaker effect hypothiazide, diuretic effect of which is observed in 1 - 2 h after taking 25 - 100 mg of the drug. An important role in the fight against edema play potassium-saving diuretics - triamterene, amiloride, especially spironolactones. spironolactones (aldactoneVerospiron). Verospiron is used in a dose of 25 to 200-300 mg per day, it is most effective in combination with thiazide diuretics, furosemide. Edema - in nephrotic syndrome due to amyloidosis, are characterized by great resistance to diuretics.

- Heparin
- Antibacterial therapy
- Corticosteroids

Glucocorticoids (GCs) - prednisolone (PZ) (medopredprednisol, prednisolone, prednisolone) and methylprednisolone (MP) (metipred, solu-medrol) - are the first drugs of choice in immunosuppressive treatment of HS. GCs affect the redistribution of immunocompetent and inflammatory cells, preventing their entry into the focus of inflammation, inhibit their sensitivity to inflammatory mediators, inhibit the secretion of proinflammatory cytokines such as TNF-α, IL-1, IL-2, IL-6. GCs trigger the processes gluconeogenesispromoting the inclusion of antibodies in carbohydrate metabolism and thereby reducing their amount, tonify capillary wall and reduce hyperemia due to activation and swelling of pericytes. Administration of large doses of GC in the form of "pulses" of MF inhibits the formation of DNA-antibodies, suspends the formation of immune complexes, reduces their mass and promotes their exit from the subendothelial layers of the glomerular basal membrane, increases glomerular filtration and renal blood flow. GC are prescribed to children in all cases of first-onset NS, in relapses of hormone-sensitive NS (as a rule, NSMI), in the progressive course of GN, in combination with other immunosuppressants and so on.

In practice, three regimens of GC therapy are used.

Continuous oral administration of PZ at a dose of 1-2 mg/kg in 2-4 doses taking into account the daily activity of the adrenal cortex (maximum doses of the drug in the morning hours with subsequent reduction, the last dose no later than 16.00) is prescribed at the beginning of treatment to achieve remission.

Alternating (alternative) mode of PZ intake is used when switching to maintenance therapy. It consists in taking the daily dose of PZ every other day, which allows, while maintaining the clinical effect, to significantly reduce side effects: acute - insomnia, euphoria, psychosis, increased appetite; chronic - edema,

obesity, myopathy, striae.The chronic side effects include: edema, obesity, myopathy, myopathy, skin atrophy, hirsutism, acne, osteoporosis, cataracts, increased BP, steroid diabetes; adrenal crisis - acute adrenal insufficiency when the drug is abruptly withdrawn. There is also a variant of the alternating regimen with taking PZ daily for 3 days, followed by a 3-4 day break. In terms of efficacy, both regimens of alternating PZ administration are approximately the same.

MF pulse therapy is used to achieve very high plasma concentrations of HA. It consists in intravenous drip injection during 20-40 min of about 30 mg/kg MF (not more than 1 g per pulse) once every 48 h. The number of injections, as well as single and total doses are determined by the chosen scheme of therapy of this pathology.

Side effects of glucocorticoids may include: insomnia, euphoria, psychosis, increased appetite, edema, obesity, myopathy, striae, skin atrophy, hirsutism, acne, osteoporosis, cataracts, increased BP, steroidal diabetes, adrenal crisis (acute adrenal insufficiency when the drug is abruptly withdrawn)

- Cytostatics

Cytostatic (cytotoxic) drugs (CS). Alkylating agents: cyclophosphamide (cyclophosphamidecytoxan) and chlorambucil (chlorobutin, leukeran) - disrupt cell division by binding to nuclear DNA nucleic acids. Enter the body in an inactive state, activated in the liver. They act indiscriminately on all dividing cells (non-selective immunosuppressants).

Cyclophosphamide is administered orally or in the form of "pulses". The drug is administered orally at the rate of 2.0-2.5 mg/kg/day for 8-12 weeks in the treatment of hormone-dependent or frequently recurrent NS against the background of gradual reduction of the dose of an alternative regimen of PZ, as well as in case of hormone resistance.

Pulse therapy with cyclophosphamide is performed against the background of an alternating course of PZ in hormone-dependent and hormone-resistant NS at a rate of 12-17 mg/kg intravenous drip. The number of "pulses" and the time interval between them depend on the chosen scheme of therapy. Another option - "pulse" once a month for 6-12 months, at a cumulative dose not exceeding 250 mg/kg.

Chlorambucil is administered per os at a dose of 0.15-0.2 mg/kg/day for 8-10 weeks for treatment of hormone-dependent and frequently recurrent NS, less often in hormone-resistant NS, against the background of an alternating course of PP with gradual reduction.

The antimetabolites are azathioprine и methotrexate - are currently rarely used in the treatment of GN. Among the side effects of cytostatics are possible: when using cyclophosphan - nausea, vomiting, leukopenia, hemorrhagic cystitis,

gonadal insufficiency; chlorbutine - pulmonary fibrosis, dermatitis, seizures, hepatopathy, leukopenia.

Practical lessons on the subject

№	Practical topics	The practical training volume
9-semester		
1	Chronic xolecyctitis	**4**
2	Nephrotic syndrome	**4**
3	Amyloidosis of the kidneys	**6**
	Total:	**14 hours**

Chronic cholecystitis

The gallbladder is an anatomical part of the liver. This sac-shaped organ is necessary for the deposit of bile synthesized by liver cells, and the excretion of fluid into the small intestine during digestion. The release of bile into the duodenal cavity contributes to the digestion of fatty foods and improved absorption of useful substances. In the organ there is a constant accumulation of secretion, leading to an increase in the viscosity of the fluid. After food enters the intestine, hormones stimulate the relaxation of special valves and the release of bile into the GI tract through special ducts.

Other functions of the organ:

- Maintaining metabolism.
- Improvement of small intestinal motility.
- Excretion of excess cholesterol and bilirubin from the body.
- Activation of enzymes needed to digest protein foods.

The gallbladder is often subjected to pathological effects. Violation of the muscular shell of the organ can impede the secretion of bile - such a process not only harms digestion, but also contributes to the formation of stones that can further completely block the bile ducts and damage the inner membranes of the gallbladder. Normally, the organ does not contain microflora, but with diseases and

anatomical defects, pathogenic and opportunistic microorganisms from the intestine may penetrate into the gallbladder.

Causes of occurrence

Various factors can provoke an inflammatory process in the gallbladder. First of all, it is stagnation of bile, which disturbs the functions of the organ and contributes to the emergence of infection. Escherichia coli, streptococci, giardia and other pathogens can penetrate into the organ from the intestine. In this case, the occurrence of infection can be a direct cause of inflammation or a consequence of such a pathological process.

Possible causes:

- Biliary tract obstruction due to an anatomical defect, stones, or valve malfunction.
- Gallstone disease is a common pathology of the gallbladder. Concretions can form in the organ due to a violation of the chemical composition of bile and stagnation of secretion.
- Malignant or benign tumor. A growing neoplasm may impede evacuation of bile from the organ.
- Primary infections. A variety of viruses can infect the gallbladder in patients with HIV infection.
- Violation of motility of the gallbladder and its ducts. With insufficient or chaotic contractility of smooth muscles of the organ, the outflow of bile into the intestine is impeded.

Chronic form of inflammation can occur if the patient did not treat acute cholecystitis. Such pathology persists for many years and significantly worsens the quality of life of a person.

F risk actors

There are various forms of predisposition to the disease, associated with individual characteristics of a person, primary pathologies, nutrition and heredity. Doctors necessarily take into account the presence of risk factors for cholecystitis during examinations.

Key risk factors:

- Female gender and age between 25 and 45 years old.
- Obesity and significant weight loss over several months.

- Taking certain medications. In particular, the risk of inflammation in the gallbladder increases with the use of hormonal drugs.
- Pregnancy.
- Chronic diseases of the intestines, liver and pancreas.
- Surgical treatment of abdominal organs, trauma.
- Chronic foci of inflammation in different parts of the body.
- Prolonged administration of parenteral nutrition.
- Improper diet or prolonged fasting.
- Alcoholic beverage abuse.
- Myocardial infarction and other heart diseases.
- Vascular abnormalities in diabetes mellitus.
- Abnormal throwing of pancreatic secretion into the gallbladder (pancreatobiliary reflux).
- Lack of physical activity.

Effective measures for the prevention of pathology are based on the elimination of risk factors, related to lifestyle and individual patient history.

Forms of the disease

Doctors classify chronic cholecystitis based on the cause of inflammation and the nature of the course of the disease. There is also a classification based on the severity of symptoms.

Basic Forms:

- Chronic calculous cholecystitis is the most common variant of the disease, arising due to blockage of the excretory ducts of the organ by concretions. It is characterized by pronounced symptomatology during exacerbations. The inflammatory process can spread to neighboring anatomical structures, including the diaphragm and pleura.
- Chronic non-calculous cholecystitis is a rarer form of the disorder, often diagnosed in patients with severe disease. In this case, the inflammatory process occurs due to trauma, surgical intervention, severe infection and other causes not related to the formation of stones. With untimely treatment, non-calculous cholecystitis can cause the death of the patient.

The danger of chronic inflammation is due to the sterile symptomatology. Patients pay attention to unpleasant sensations only during exacerbations, as a result of which the disease gradually progresses and causes complications.

Symptoms and signs

The symptomatic picture of the disease depends on the severity of the inflammatory process, the age of the patient and the frequency of exacerbations. The predominant sign is usually pain in the right subcostal region, spreading to the back and central abdomen. If the inflammation affects the diaphragm, there may be pain in the right arm and scapula.

Additional attributes:

- Tension in the abdominal muscles.
- Severe weakness, fatigue.
- The appearance of cold sweat.
- Nausea and vomiting.
- Lack of appetite.
- Liquid stools.
- Abdominal bloating.
- Heart palpitations.
- Shortness of breath.
- Low blood pressure.
- Yellowing of the skin and mucous membranes.
- Chest pain.

Due to insufficient intake of vitamins and minerals in the body, symptoms such as pale skin and constant fatigue occur. Against the background of the development of complications, more severe pathological signs appear. With prolonged sharp pain in the abdomen and persistent fever should seek medical help as soon as possible.

Diagnosis

Necessary examinations can be prescribed to the patient by a gastroenterologist. During the appointment, the doctor will ask the patient about complaints and study anamnestic information. Physical examination allows you to detect jaundice, abdominal bloating and pain in certain parts of the body. Based on the data obtained, the doctor will prescribe the necessary instrumental and laboratory tests.

Necessary diagnostic manipulations:

- Ultrasound examination of the gallbladder and bile ducts. The advantages of the method are safety and real-time imaging of the organs. The doctor can immediately detect gallstones in the gallbladder, changes in the walls of the organ and other pathological signs indicating cholecystitis.

- Blood tests to detect inflammation and infection. With pronounced inflammation in the blood, the number of leukocytes increases. The concentration of liver enzymes is also a diagnostic criterion.
- Collection of bile from the organ by duodenal probing. The obtained material is sent by a specialist to the laboratory for detection of pathogenic microflora.
- X-ray examination of the gallbladder (cholecystography). The doctor receives information about the size, shape and functional activity of the organ. With the help of this study it is possible to detect anatomical anomalies, cholelithiasis or other diseases.

If the doctor is unable to make a diagnosis after requesting the results of the above tests, an additional appointment of gastroscopy, CT scan or laparoscopy is possible. It is necessary to determine the cause of inflammation as accurately as possible.

Treatment

In chronic cholecystitis, the main method of treatment is a special diet that reduces the negative impact on the organ. Medications may be prescribed to eliminate infection and inflammation. If the disease has caused dangerous complications, such as gallbladder gangrene or peritonitis, it is necessary to perform surgical intervention.

Main Assignments:

- Therapy with the help of therapeutic diet. The patient needs a frequent fractional diet. It is necessary to exclude the constant use of fatty foods, alcohol, beans and fried meat. Observe the diet should be followed for a long time to prevent a relapse of the disease. When removing the gallbladder, patients are prescribed a lifelong diet.
- Antibiotics and anti-inflammatory drugs. For chronic infection, it is recommended to obtain a microflora sample by probing and select an effective antimicrobial agent by laboratory test.
- Antispasmodics and pain relievers to relieve symptoms.
- Diuretic medicines. Medicines of this group are used to improve the excretion of bile into the duodenum.

The main methods of surgical treatment of cholecystitis doctors include removal of the gallbladder or removal of concretions blocking the ducts of the organ. In severe complications of the disease may require complex open intervention in the abdominal cavity.

Complications

Chronic cholecystitis can provoke the development of severe complications even if the symptoms are mild. The main danger is the focus of infection, which can spread to other organs.

Major complications:

- Gangrene of the gallbladder - destruction of the tissues of the organ, leading to a purulent process in the abdominal cavity.
- Peritonitis - extensive inflammation of the peritoneum, causing severe symptoms and intoxication of the entire body. This pathology can occur against the background of perforation of an inflamed gallbladder.
- Inflammation of the pancreas due to infiltration of gallbladder contents into the organ.
- A severe infectious process in which pathogens spread with the bloodstream (sepsis).

Timely surgical treatment can prevent the development of such complications.

Thus, chronic cholecystitis is a frequent consequence of acute inflammation of the gallbladder. Symptomatology of the disease persists for many years and negatively affects the quality of life of a person. Consultation with a gastroenterologist will help the patient to choose an effective treatment for the disease.

Nephrotic syndrome

Nephrotic syndrome is a collection of symptoms due to kidney damage. This includes protein in the urine, low blood albumin levels, high blood lipids, and significant swelling. Other symptoms may include weight gain, feeling tired, and foamy urine. Complications may include blood clots, infections, and high blood pressure.

Causes include a number of kidney diseases such as focal segmental glomerulosclerosis, membranous nephropathy, and minimal change disease. It may also occur as a complication of diabetes or lupus. The underlying mechanism typically involves damage to the glomeruli of the kidney. Diagnosis is typically based on urine testing and sometimes a kidney biopsy. It differs from nephritic syndrome in that there are no red blood cells in the urine.

Treatment is directed at the underlying cause. Other efforts include managing high blood pressure, high blood cholesterol, and infection risk. A low salt diet and limiting fluids is often recommended. About 5 per 100,000 people are affected per year. The usual underlying cause varies between children and adults.

Signs and symptoms

Nephrotic syndrome is usually accompanied by retention of water and sodium. The degree to which this occurs can vary between slight edema in the eyelids that decreases during the day, to

affecting the lower limbs, to generalized swelling, to full blown anasarca.

Nephrotic syndrome is characterized by large amounts of proteinuria (>3.5 g per 1.73 m2 body surface area per day, or > 40 mg per square meter body surface area per hour in children), hypoalbuminemia (< 2.5 g/dl), hyperlipidaemia, and edema that begins in the face. Lipiduria (lipids in urine) can also occur, but is not essential for the diagnosis of nephrotic syndrome. Hyponatremia also occurs with a low fractional sodium excretion.

Hyperlipidaemia is caused by two factors:

- Hypoproteinemia stimulates protein synthesis in the liver, resulting in the overproduction of lipoproteins.
- Lipid catabolism is decreased due to lower levels of lipoprotein lipase, the main enzyme involved in lipoprotein breakdown. Cofactors, such as apolipoprotein C2 may also be lost by increased filtration of proteins.

A few other characteristics seen in nephrotic syndrome are:

- The most common sign is excess fluid in the body due to serum hypoalbuminemia. Lower serum oncotic pressure causes fluid to accumulate in the interstitial tissues. Sodium and water retention aggravates the edema. This may take several forms:
  - Puffiness around the eyes, characteristically in the morning. ○ Pitting edema over the legs.
  - Fluid in the pleural cavity causing pleural effusion. More commonly associated with excess fluid is pulmonary edema.
  - Fluid in the peritoneal cavity causing ascites.
  - Generalized edema throughout the body known as anasarca.
- Most of the people with nephrotic syndrome are normotensive but hypertension (rarely) may also occur.
- Anaemia (iron resistant microcytic hypochromic type) may be present due to transferrin loss.
- Dyspnea may be present due to pleural effusion or due to diaphragmatic compression with ascites.
- Erythrocyte sedimentation rate is increased due to increased fibrinogen & other plasma contents.
- Some people may notice foamy or frothy urine, due to a lowering of the surface tension by the

severe proteinuria. Actual urinary complaints such as haematuria or oliguria are uncommon, though these are seen commonly in nephritic syndrome.

- May have features of the underlying cause, such as the rash associated with systemic lupus erythematosus, or the neuropathy associated with diabetes.

- Examination should also exclude other causes of gross edema—especially the cardiovascular and liver system.
- Muehrcke's nails; white lines (leukonychia) that extend all the way across the nail and lie parallel to the lunula

The main signs of nephrotic syndrome are:

- A proteinuria of greater than 3.5 g /24 h /1.73 m2 (between 3 and 3.5 g/24 h /1.73 m2 is considered to be proteinuria in the nephrotic range) or greater than 40 mg/h/m2 in children. The ratio between urinaryconcentrations ofalbumin and creatinine can be used in the absence ofa 24-hour urine test for total protein. This coefficient will be greater than 200–400 mg/mmol in nephrotic syndrome. This pronounced loss of proteins is due to an increase in glomerular permeability that allows proteins to pass into the urine instead of being retained in the blood. Under normal conditions a 24-hour urine sample should not exceed 80 milligrams or 10 milligrams per decilitre.
- A hypoalbuminemia of less than 2.5 g/dL, that exceeds the *liver clearance* level, that is, protein synthesis in the liver is insufficient to increase the low blood protein levels.
- Edema is thought to be caused by two mechanisms. The first being hypoalbuminemia which lowers the oncotic pressure within vessels resulting in hypovolemia and subsequent activation of the renin–angiotensin system and thus retention of sodium and water (underfill hypothesis). Additionally, it is thought that urinary proteases (excreted as a result of significant proteinuria) cause a direct effect by activating the epithelial sodium channel (ENaC) on the principal cell that leads to the reabsorption of sodium and water (overfill hypothesis). Nephrotic syndrome edema initially appears in parts of the lower body (such as the legs) and in the eyelids. In the advanced stages it also extends to the pleural cavity and peritoneum (ascites) and can even develop into a generalized anasarca.
- Hyperlipidaemia iscaused byan increase inthe synthesisoflow and very-low-densitylipoproteins in the liver that are responsible for the transport of cholesterol and triglycerides. There is also an increase in the liver synthesis of cholesterol.
- Thrombophilia, or hypercoagulability, is a greater predisposition for the formation of blood clots that are caused by a decrease in the levels of antithrombin III in the blood due to its loss in urine.
- Lipiduria or loss of lipids in the urine is indicative of glomerular pathology due to an increase in the filtration of lipoproteins.

Complications

Nephrotic syndrome can be associated with a series of complications that can affect an individual's health and quality of life:

- Thromboembolic disorders: particularly those caused by a decrease in blood antithrombin III levels due to leakage. Antithrombin III counteracts the action of thrombin. Thrombosis usually occurs in the kidney veins although it can also occur in arteries. Treatment is with oral anticoagulants (not heparin as heparin acts via anti-thrombin 3 which is lost in the proteinuria so it will be ineffective.) Hypercoagulopathy due to extravasation of fluid from the blood vessels (edema) is also a risk for venous thrombosis.
- Infections: The increased susceptibility of people with nephrotic syndrome to infections can be a result of the leakage of immunoglobulins from the blood, the loss of proteins in general, and the presence of oedematous fluid (which acts as a breeding ground for infections). The most common infection is peritonitis, followed by lung, skin, and urinary infections, meningoencephalitis and in the most serious cases septicaemia. The most notable ofthe causative organisms are *Streptococcus pneumoniae* and *Haemophilus influenzae*.
- Spontaneous bacterial peritonitis can develop where there is ascites present. This is a frequent development in children but very rarely found in adults.
- Acute kidney failure due to hypovolemia: the loss of vascular fluid into the tissues (edema) produces a decreased blood supply to the kidneys that cause a loss of kidney function. Thus it is a tricky task to get rid of excess fluid in the body while maintaining circulatory euvolemia.
- Pulmonary edema: the loss of proteins from blood plasma and the consequent fall in oncotic pressure causes an abnormal accumulation of liquid in the lungs causing hypoxia and dyspnoea.
- Hypothyroidism: deficiency of the thyroglobulin transport protein thyroxin (a glycoprotein that is rich in iodine and is found in the thyroid gland) due to decreased thyroid-binding globulin.
- Vitamin D deficiency can occur. Vitamin D binding protein is lost.
- Hypocalcaemia: lack of 25-hydroxycholecalciferol (the way that vitamin D is stored in the body). As vitamin D regulates the amount of calcium present in the blood, a decrease in its concentration will lead to a decrease in blood calcium levels. It may be significant enough to cause tetany. Hypocalcaemia may be relative; calcium levels should be adjusted based on the albumin level and ionized calcium levels should be checked.
- Microcytic hypochromic anaemia: iron deficiency caused by the loss of ferritin (compound used to store iron in the body). It is iron-therapy resistant.
- Protein malnutrition: this occurs when the amount of protein that is lost in the urine is greater than that ingested, this leads to a negative nitrogen balance.

• Growth retardation: This can occur in cases of relapse or resistance to therapy. Causes of growth retardation are protein deficiency from the loss of protein in urine, anorexia (reduced protein intake), and steroid therapy (catabolism).

• Cushing's syndrome Causes

Histological image of a normal kidney glomerulus. It is possible to see a glomerulus in the centre

of the image surrounded by kidney tubules.

Nephrotic syndrome has many causes and may either be the result of a glomerular disease that can be either limited to the kidney, called *primary* nephrotic syndrome (primary glomerulonephrosis), or a condition that affects the kidney and other parts of the body, called *secondary* nephrotic syndrome.

Primary glomerulonephrosis

Primary causes of nephrotic syndrome are usually described by their histology:

• Minimal change disease (MCD): is the most common cause of nephrotic syndrome in children. It owes its name to the fact that the nephrons appear normal when viewed with an optical microscope as the lesions are only visible using an electron microscope. Another symptom is pronounced proteinuria.

• Focal segmental glomerulosclerosis (FSGS): is the most common cause of nephrotic syndrome in adults. It is characterized by the appearance of tissue scarring in the glomeruli. The term *focal* is used as some of the glomeruli have scars, while others appear intact; the term *segmental* refers to the fact that only part of the glomerulus suffers the damage.

• Membranous glomerulonephritis (MGN): The inflammation of the glomerular membrane causes increased leaking inthe kidney. It is not clear whythisconditiondevelops in most people, although an auto-immune mechanism is suspected.

• Membranoproliferative glomerulonephritis (MPGN): is the inflammation of the glomeruli along with the deposit of antibodies in their membranes, which makes filtration difficult.

• Rapidly progressive glomerulonephritis (RPGN): (Usually presents as a nephrotic syndrome) A person's glomeruli are present in a *crescent moon* shape. It is characterized clinically by a rapid decrease in the glomerular filtration rate (GFR) by at least 50% over a short period, usually from a few days to 3 months.

They are considered to be "diagnoses of exclusion", i.e. they are diagnosed only after secondary causes have been excluded.

Secondary glomerulonephrosis

Diabetic glomerulonephritis in a person with nephrotic syndrome.

Secondary causes of nephrotic syndrome have the same histologic patterns as the primary causes, though they may exhibit some differences suggesting a secondary

cause, such as inclusion bodies. They are usually described by the underlying cause, such as:

- Diabetic nephropathy: is a complication that occurs in some diabetics. Excess blood sugar accumulates in the kidney causing them to become inflamed and unable to carry out their normal function. This leads to the leakage of proteins into the urine.
- Systemic lupus erythematosus: this autoimmune disease can affect a number of organs, among them the kidney, due to the deposit of immunocomplexes that are typical to this disease. The disease can also cause *lupus nephritis*.
- Sarcoidosis: This disease does not usually affect the kidney but, on occasions, the accumulation of inflammatory granulomas (collection of immune cells) in the glomeruli can lead to nephrotic syndrome.
- Syphilis: kidney damage can occur during the secondary stage of this disease (between 2 and 8 weeks from onset).
- Hepatitis B: certain antigens present during hepatitis can accumulate in the kidneys and damage them.
- Sjögren's syndrome: this autoimmune disease causes the deposit of immunocomplexes in the glomeruli, causing them to become inflamed, this is the same mechanism as occurs in systemic lupus erythematosus.
- HIV: the virus's antigens provoke an obstruction in the glomerular capillary's lumen that alters normal kidney function.
- Amyloidosis: the deposit of *amyloid substances* (proteins with anomalous structures) in the glomeruli modifying their shape and function.
- Multiple myeloma: kidney impairment is caused by the accumulation and precipitation of light chains, which form casts in the distal tubules, resulting in kidney obstruction. In addition, myeloma light chains are also directly toxic on proximal kidney tubules, further adding to kidney dysfunction.
- Vasculitis: inflammation of the blood vessels at a glomerular level impedes the normal blood flow and damages the kidney.
- Cancer: as happens in myeloma, the invasion of the glomeruli by cancerous cells disturbs their normal functioning.
- Genetic disorders: congenital nephrotic syndrome is a rare genetic disorder in which the protein nephrin, a component of the glomerular filtration barrier, is altered.
- Drugs (e.g. gold salts, penicillin, captopril):[25] gold salts can cause a more or less important loss of proteins in urine as a consequence of metal accumulation. Penicillin is nephrotoxic in people with kidney failure and captopril can aggravate proteinuria.

By histologic pattern Membranous nephropathy (MN)

- Sjögren's syndrome
- Systemic lupus erythematosus (SLE)
- Diabetes mellitus
- Sarcoidosis
- Drugs (such as corticosteroids, gold, intravenous heroin)
- Malignancy (cancer)
- Bacterial infections, e.g. leprosy & syphilis
- Protozoal infections, e.g. malaria

Focal segmental glomerulosclerosis (FSGS)

- Hypertensive nephrosclerosis
- HIV
- Obesity[
- Kidney loss

Minimal change disease (MCD)

- Drugs, especially NSAIDs in the elderly
- Malignancy, especially Hodgkin's lymphoma
- Allergy
- Bee sting

Membranoproliferative Glomerulonephritis

- Hepatitis C

Genetics

Over 50 mutations are known to be associated with this condition. Pathophysiology

Drawing of the kidney glomerulus.

The kidney glomerulus filters the blood that arrives at the kidney. It is formed of capillaries with small pores that allow small molecules to pass through that have a molecular weight of less than 40,000 Daltons, but not larger macromolecules such as proteins.

In nephrotic syndrome, the glomeruli are affected by an inflammation or a *hyalinization* (the formation of a homogenous crystalline material within cells) that allows proteins such as albumin, antithrombin or the immunoglobulins to pass through the cell membrane and appear in urine.

Albumin is the main protein in the blood that is able to maintain an oncotic pressure, which prevents the leakage of fluid into the extracellular medium and the subsequent formation of edemas.

As a response to hypoproteinemia the liver commences a compensatory mechanism involving the synthesis of proteins, such as alpha-2 macroglobulin and

lipoproteins. An increase in the latter can cause the hyperlipidemia associated with this syndrome.

Diagnosis

Urinalysis will be able to detect high levels of proteins and occasionally microscopic haematuria.

Ultrasound of a kidney with nephrotic syndrome. There is a hyperechoic kidney without
demarcation of the cortex and medulla.

Along with obtaining a complete medical history, a series of biochemical tests are required in order to arrive at an accurate diagnosis that verifies the presence of the illness. In addition, imaging ofthe kidneys (for structure and presence oftwo kidneys) is sometimes carried out, and/or a biopsy of the kidneys. The first test will be a urinalysis to test for high levels of proteins,[31] as a healthy subject excretes an insignificant amount of protein in their urine. The test will involve a 24-hour bedside urinary total protein estimation. The urine sample is tested for proteinuria (>3.5 g per 1.73 m2 per 24 hours). It is also examined for urinary casts, which are more a feature of active nephritis. Next a blood screen, comprehensive metabolic panel (CMP) will look for hypoalbuminemia: albumin levels of ≤2.5 g/dL (normal=3.5-5 g/dL). Then a Creatinine Clearance CCr test will evaluate kidney function particularly the glomerular filtration capacity.[32] Creatinine formation is a result of the breakdown of muscular tissue, it is transported in the blood and eliminated in urine. Measuring the concentration of organic compounds in both liquidsevaluatesthe capacityofthe glomerulito filter blood. Electrolytes and urea levels mayalso be analysed at the same time as creatinine (EUC test) in order to evaluate kidney function. A lipid profile will also be carried out as high levels of cholesterol (hypercholesterolemia), specifically elevated LDL, usually with concomitantly elevated VLDL, is indicative of nephrotic syndrome.

A kidney biopsy may also be used as a more specific and invasive test method. A study of a sample's anatomical pathology may then allow the identification of the type of glomerulonephritis involved. However,thisprocedure isusuallyreserved for adultsasthe majority of children suffer from minimal change disease that has a remission rate of 95% with corticosteroids. A biopsy is usually only indicated for children that are *corticosteroid resistant* as the majority suffer from focal and segmental glomeruloesclerosis.

Further investigations are indicated if the cause is not clear including analysis of auto-immune markers (ANA, ASOT, C3, cryoglobulins, serum electrophoresis), or ultrasound of the whole abdomen.

Classification

A broad classification of nephrotic syndrome based on underlying cause:

Nephrotic syndrome
Primary Secondary
Nephrotic syndrome is often classified histologically:
Nephrotic syndrome
MCD FSGS MGN MPGN

Differential diagnosis

Some symptoms that are present in nephrotic syndrome, such as edema and proteinuria, also appear in other illnesses. Therefore, other pathologies need to be excluded in order to arrive at a definitive diagnosis.

- Edema: in addition to nephrotic syndrome there are two other disorders that often present with edema; these are heart failure and liver failure. Congestive heart failure can cause liquid retention in tissues as a consequence of the decrease in the strength of ventricular contractions. The liquid is initially concentrated in the ankles but it subsequently becomes generalized and is called anasarca. People with congestive heart failure also experience an abnormal swelling of the heart cardiomegaly, which aids in making a correct diagnosis. Jugular venous pressure can also be elevated and it might be possible to hear heart murmurs. An echocardiogram is the preferred investigation method for these symptoms. Liver failure caused by cirrhosis, hepatitis and other conditions such as alcoholism, IV drug use or some hereditary diseases can lead to swelling in the lower extremities and the abdominal cavity. Other accompanying symptoms include jaundice, dilated veins over umbilicus (caput medusae), scratch marks (due to widespread itching, known as pruritus), enlarged spleen, spider angiomata, encephalopathy, bruising, nodular liver and anomalies in the liver function tests. Less frequently symptoms associated with the administration ofcertain pharmaceuticaldrugs have to be discounted. These drugs promote the retention of liquid in the extremities such as occurs with NSAIDs, some antihypertensive drugs, the adrenal corticosteroids and sex hormones.

Acute fluid overload cancause edema in someone withkidneyfailure. These people are known to have kidney failure, and have either drunk too much or missed their dialysis. In addition, when Metastatic cancer spreadsto the lungsor abdomen it causeseffusionsand fluid accumulation due to obstruction of lymphatic vessels and veins, as well as serous exudation.

- Proteinuria: the loss ofproteins fromthe urine is caused by many pathologicalagents and infection by these agents has to be ruled out before it can be certain that a person has nephrotic syndrome. Multiple myeloma can cause a proteinuria that is not accompanied by hypoalbuminemia, which is an important aid in making a differential diagnosis; other potential

causes of proteinuria include asthenia, weight loss or bone pain. In diabetes mellitus there is an association between increases in glycated hemoglobin levels and the appearance of proteinuria. Other causes are amyloidosis and certain other allergic and infectious diseases.

Treatment

The treatment of nephrotic syndrome can be symptomatic or can directly address the injuries caused to the kidney.

Symptomatic

The objective of this treatment is to treat the imbalances brought about by the illness: edema, hypoalbuminemia, hyperlipemia, hypercoagulability and infectious complications.

- **Edema**: a return to an unswollen state is the prime objective of this treatment of nephrotic syndrome. It is carried out through the combination of a number of recommendations:
  - Rest: depending on the seriousness of the edema and taking into account the risk of thrombosis caused by prolonged bed rest.
  - Medical nutrition therapy: based on a diet with the correct energy intake and balance of proteins that will be used in synthesis processes and not as a source of calories. A total of 35 kcal/kg body weight/day is normally recommended. This diet should also comply with two more requirements: the first is to not consume more than 1 g ofprotein/kg bodyweight/ day, as a greater amount could increase the degree of proteinuria and cause a negative nitrogen balance. People are usually recommended lean cuts of meat, fish, and poultry. The second guideline requires that the amount ofwater ingested is not greaterthan the levelof diuresis. Inorder to facilitate this, the consumption of salt must also be controlled, as this contributes to water retention. It is advisable to restrict the ingestion of sodium to 1 or 2 g/day, which means that salt cannot be used in cooking and salty foods should also be avoided. Foods high in sodium include seasoning blends (garlic salt, Adobo, season salt, etc.) canned soups, canned vegetables containing salt, luncheon meats including turkey, ham, bologna, and salami, prepared foods, fast foods, soy sauce, ketchup, and salad dressings. On food labels, compare milligrams of sodium to calories per serving. Sodium should be less than or equal to calories per serving.
  - Medication: The pharmacological treatment of edema is based on diuretic medications (especially loop diuretics, such as furosemide). In severe cases of edema (or in cases with physiological repercussions, such as scrotal, preputial or urethral edema) or in people with one of a number of severe infections (such as sepsis or pleural effusion), the diuretics can be administered intravenously. This occurs where the risk from plasmatic expansion is considered greater than the risk

of severe hypovolemia, which can be caused by the strong diuretic action of intravenous treatment. The procedure is the following:

1. Analyse haemoglobin and haematocrit levels.
2. A solution of 25% albumin is used that is administered for only 4 hours in order to avoid pulmonary edema.
3. Haemoglobin and haematocrit levels are analysed again: if the haematocrit value is less than the initial value (a sign of correct expansion) the diuretics are administered for at least 30 minutes. If the haematocrit level is greater than the initial one this is a contraindication for the use of diuretics as they would increase said value.

It may be necessary to give a person potassium or require a change in dietary habits if the diuretic drug causes hypokalaemia as a side effect.

- **Hypoalbuminemia**: is treated using the medical nutrition therapy described as a treatment for edema. It includes a moderate intake of foods rich in animal proteins.
- **Hyperlipidaemia**: depending of the seriousness of the condition it can be treated with medical nutrition therapyas the only treatment or combined with drug therapy. The ingestion ofcholesterol should be less than 300 mg/day, which will require a switch to foods that are low in saturated fats. Avoid saturated fats such as butter, cheese, fried foods, fatty cuts of red meat, egg yolks, and poultry skin. Increase unsaturated fat intake, including olive oil, canola oil, peanut butter, avocadoes, fish and nuts. In cases of severe hyperlipidaemia that are unresponsive to nutrition therapy the use of hypolipidemic drugs, may be necessary (these include statins, fibrates and resinous sequesters of bile acids).
- **Thrombophilia**: low molecular weight heparin (LMWH) may be appropriate for use as a prophylactic in some circumstances, such as in asymptomatic people that have no history of suffering from thromboembolism. When the thrombophilia is such that it leads to the formation of blood clots, heparin is given for at least 5 days along with oral anticoagulants (OAC). During this time and if the prothrombin time is within its therapeutic range (between 2 and 3), it may be possible to suspend the LMWH while maintaining the OACs for at least 6 months.
- **Infectious complications**: an appropriate course of antibacterial drugs can be taken according to the infectious agent.

In addition to these key imbalances, vitamin D and calcium are also taken orally in case the alteration of vitamin D causes severe hypocalcaemia, this treatment has the goal of restoring physiological levels of calcium in the person.

- Achieving better blood glucose level control if the person is diabetic.
- Blood pressure control. ACE inhibitors are the drug of choice. Independent of their blood pressure-lowering effect, they have been shown to decrease protein loss.

Kidney damage

The treatment of kidney damage may reverse or delay the progression of the disease. Kidney damage is treated by prescribing drugs:

- **Corticosteroids**: the result is a decrease in proteinuria and the risk of infection as well as a resolution of the edema. Prednisone is usually prescribed at a dose of 60 mg/m2 of body surface area/day in a first treatment for 4–8 weeks. After this period the dose is reduced to 40 mg/m2 for a further 4 weeks. People suffering a relapse or children are treated with prednisolone 2 mg/kg/day tillurine becomes negative for protein. Then, 1.5 mg/kg/day for 4 weeks. Frequent relapses treated by: cyclophosphamide or nitrogen mustard or ciclosporin or levamisole. People can respond to prednisone in a number of different ways:
  - People with Corticosteroid sensitive or early steroid-responder: the subject responds to the corticosteroids in the first 8 weeks of treatment. This is demonstrated by a strong diuresis and the disappearance of edemas, and also by a negative test for proteinuria in three urine samples taken during the night.
  - People with Corticosteroid resistance or late steroid-responder: the proteinuria persists after the 8-week treatment. The lack of response is indicative of the seriousness of the glomerular damage, which could develop into chronic kidney failure.
  - People with Corticosteroid intolerant: complications such as hypertension appear, and they gain a lot of weight and can develop aseptic or avascular necrosis of the hip or knee, cataracts and thrombotic phenomena and/or embolisms.
  - People with Corticosteroid dependent: proteinuria appears when the dose of corticosteroid is decreased or there is a relapse in the first two weeks after treatment is completed.

The susceptibility testing in vitro to glucocorticoids on the person's peripheral blood mononuclear cells is associated with the number of new cases of not optimal clinical responses: the most sensitive people in vitro have shown a higher number of cases of corticodependence, while the most resistant people in vitro showed a higher number of cases of ineffective therapy.

- Immunosupressors (cyclophosphamide): only indicated in recurring nephrotic syndrome in corticosteroid dependent or intolerant people. In the first two cases, the proteinuria has to be negated before treatment with the immunosuppressor can begin, which involves a prolonged treatment with prednisone. The negation of the proteinuria indicates the exact moment when treatment with cyclophosphamide can begin. The treatment is continued for 8 weeks at a dose of 3 mg/kg/day, the immunosuppression is halted after this period. In order to be able to start this treatment the person should not be suffering from neutropenia nor anaemia, which

would cause further complications. Apossible side effect ofthe cyclophosphamide is alopecia. Complete blood
count tests are carried out during the treatment in order to give advance warning of a possible infection.

Prognosis

The prognosis for nephrotic syndrome under treatment is generally good although this depends on the underlying cause, the age ofthe person and their response to treatment. It is usually good in children, because minimal change disease responds very well to steroids and does not cause chronic kidney failure. Any relapses that occur become less frequent over time; the opposite occurs with mesangiocapillary glomerulonephritis, in which the kidney fails within three years of the disease developing, making dialysis necessary and subsequent kidney transplant. In addition children under the age of 5 generally have a poorer prognosis than prepubescents, as do adults older than 30 years of age as they have a greater risk of kidney failure.

Other causes such as focal segmental glomerulosclerosis frequently lead to end stage kidney disease. Factors associated with a poorer prognosis in these cases include level of proteinuria, blood pressure control and kidney function (GFR).

Without treatment nephrotic syndrome has a very bad prognosis especially *rapidly progressing glomerulonephritis*, which leads to acute kidney failure after a few months.

Epidemiology

Nephrotic syndrome can affect any age, although it is mainly found in adults with a ratio of adults to children of 26 to 1.

The syndrome presents in different ways in the two groups: the most frequent glomerulopathyinchildren is minimalchange disease (66%ofcases), followed byfocalsegmental glomerulosclerosis (8%) and mesangiocapillary glomerulonephritis (6%). In adults the most common disease is mesangiocapillary glomerulonephritis (30-40%), followed by focal and segmental glomeruloesclerosis (15-25%) and minimal change disease (20%). The latter usually presents as secondary and not primary as occurs in children. Its main cause is diabetic nephropathy. It usuallypresents ina person fromtheir 40sor 50s. Ofthe glomerulonephritiscases, approximately 60% to 80% are primary, while the remainder are secondary.

There are also differences in epidemiology between the sexes, the disease is more common in men than in women by a ratio of 2 to 1.

The epidemiological data also reveals information regarding the most common way that symptoms develop in people with nephrotic syndrome: spontaneous remission occurs in up to 20% or 30% of cases during the first year of the illness.

However, this improvement is not definitive as some 50% to 60% of people with Nephrotic syndrome die and/or develop chronic kidney failure 6 to 14 years after this remission. On the other hand, between 10% and 20% of people have continuous episodes of remissions and relapses without dying or jeopardizing their kidney. The main causes of death are cardiovascular, as a result of the chronicity of the syndrome, and thromboembolic accidents.

AMYLOIDOSIS

Amyloidosis is a rare disease that occurs when amyloid proteins are deposited in tissues and organs. Amyloid proteins are abnormal proteins that the body cannot break down and recycle, as it does with normal proteins. When amyloid proteins clump together, they form amyloid deposits. The buildup of these deposits damages a person's organs and tissues. Amyloidosis can affect different organs and tissues in different people and can affect more than one organ at the same time. Amyloidosis most frequently affects the kidneys, heart, nervous system, liver, and digestive tract. The symptoms and severity of amyloidosis depend on the organs and tissues affected.

What are the kidneys and what do they do?

The kidneys are two bean-shaped organs, each about the size of a fist. They are located just below the rib cage, one on each side of the spine. Every day, the two kidneys filter about 120 to 150 quarts of blood to produce about 1 to 2 quarts of urine, composed of wastes and extra fluid. The urine flows from the kidneys to the bladder through tubes called ureters. The bladder stores urine. When the bladder empties, urine flows out of the body through a tube called the urethra, located at the bottom of the bladder. In men, the urethra is long, while in women it is short.

The kidneys are two bean-shaped organs, each about the size of a fist.

What types of amyloidosis affect the kidneys?

Primary amyloidosis and dialysis-related amyloidosis are the types of amyloidosis that can affect the kidneys.

Primary Amyloidosis of the Kidneys

The kidneys are the organs most commonly affected by primary amyloidosis. Amyloid deposits damage the kidneys and make it harder for them to filter wastes and break down proteins. When the kidneys become too damaged, they may no longer be able to function well enough to maintain health, resulting in kidney failure. Kidney failure can lead to problems such as high blood pressure, bone disease, and anemia—a condition in which the body has fewer red blood cells than normal.

Dialysis-related Amyloidosis

People who suffer from kidney failure and have been on long-term dialysis may develop

dialysis-related amyloidosis. This type of amyloidosis occurs when a certain protein, called beta-2 microglobulin, builds up in the blood because dialysis does not remove it completely. The two types of dialysis are

Hemodialysis. Hemodialysis uses a special filter called a dialyzer to remove wastes and extra fluid from the blood.

Peritoneal dialysis. Peritoneal dialysis uses the lining of the abdominal cavity—the space in the body that holds organs such as the stomach, intestines, and liver—to filter the blood.

Dialysis-related amyloidosis is a complication of kidney failure because neither hemodialysis nor peritoneal dialysis effectively filters beta-2 microglobulin from the blood. As a result, elevated amounts of beta-2 microglobulin remain in the blood.

What are the signs and symptoms of primary amyloidosis of the kidneys?

The most common sign of primary amyloidosis of the kidneys is nephrotic syndrome—a collection of signs that indicate kidney damage. The signs of nephrotic syndrome include

albuminuria—an increased amount of albumin, a protein, in the urine. A person with nephrotic syndrome excretes more than half a teaspoon of albumin per day.

-hyperlipidemia—a condition in which a person's blood has more-than-normal amounts of fats and cholesterol.

-edema—swelling, typically in a person's legs, feet, or ankles and less often in the hands or face.

hypoalbuminemia—a condition in which a person's blood has less-than-normal amounts of

albumin.

Other signs and symptoms of primary amyloidosis may include:

fatigue, or feeling tired

shortness of breath

low blood pressure

numbness, tingling, or a burning sensation in the hands or feet weight loss

What are the symptoms of dialysis-related amyloidosis? The symptoms of dialysis-related amyloidosis may include

-pain, stiffness, and fluid in the joints.

-abnormal, fluid-containing sacs, called cysts, in some bones.

-carpal tunnel syndrome, caused by unusual buildup of amyloid proteins in the wrists. The symptoms of carpal tunnel syndrome include numbness or tingling, sometimes associated with muscle weakness, in the fingers and hands.

Dialysis-related amyloidosis most often affects bones, joints, and the tissues that connect muscle to bone, called tendons. The disease may also affect the digestive

tract and organs such as the heart and lungs. Bone cysts caused by dialysis-related amyloidosis can lead to bone fractures. Dialysis-related amyloidosis can also cause tears in tendons and ligaments. Ligaments are tissues that connect bones to other bones.

How is primary amyloidosis of the kidneys diagnosed?

A health care provider diagnoses primary amyloidosis of the kidneys with a medical and family history

-a physical exam

-urinalysis

-blood tests

-a kidney biopsy

Medical and Family History

Taking a medical and family history may help a health care provider diagnose amyloidosis of the kidneys. He or she will ask the patient to provide a medical and family history.

Physical Exam

A physical exam may help diagnose primary amyloidosis of the kidneys. During a physical exam, a health care provider usually

-examines a patient's body to check for swelling

-uses a stethoscope to listen to the lungs

-taps on specific areas of the patient's body

Urinalysis

A health care provider may use urinalysis—the testing of a urine sample—to check for albumin and amyloid proteins in urine. The patient provides a urine sample in a special container at a health care provider's office or a commercial facility. A nurse or technician can test the sample in the same location or send it to a lab for analysis. More-than-normal amounts of albumin in urine may indicate kidney damage due to primary amyloidosis. Amyloid proteins in urine may indicate amyloidosis.

Blood Tests

The health care provider mayuse blood teststo see how well the kidneys are working and to check for amyloid proteins and hyperlipidemia. A blood test involves drawing a patient's blood at a health care provider's office or a commercial facility and sending the sample to a lab for analysis. Blood tests for kidney function measure the waste products in the blood that healthy kidneys normally filter out. Hyperlipidemia may indicate nephrotic syndrome. Amyloid proteins in blood may indicate amyloidosis.

Kidney Biopsy

Only a biopsy can show the amyloid protein deposits in the kidneys. A health care provider may recommend a kidney biopsy if other tests show kidney damage. A kidney biopsy is a procedure that involves taking a piece of kidney tissue for examination with a microscope. A health care provider performs a kidney biopsy in a hospitalwith light sedation and local anesthetic. The health care provider uses imaging techniques such as ultrasound or a computerized tomography (CT) scan to guide the biopsy needle into the kidney and take the tissue sample. A pathologist—a doctor who specializes in diagnosing diseases—examines the tissue in a lab for amyloid proteins and kidney damage.

The biopsy results can help the health care provider determine the best course of treatment. **How is dialysis-related amyloidosis diagnosed?**

A health care provider diagnoses dialysis-related amyloidosis with □ □urinalysis

-blood tests

-imaging tests

A health care provider can use urinalysis and blood tests to detect the amount of amyloid proteins in urine and blood. Imaging tests, such as x-rays and CT scans, can provide pictures of bone cysts and amyloid deposits in bones, joints, tendons, and ligaments. An x-ray technician performs imaging tests in a health care provider's office, an outpatient center, or a hospital. A radiologist— a doctor who specializes in medical imaging—interprets the images. A patient does not require anesthesia. X-ray image showing amyloid deposits in the wrist

How is primary amyloidosis of the kidneys treated?

A health care provider treats primary amyloidosis of the kidneys with the following:-medication therapy, including chemotherapy

-a stem cell transplant

-treating other conditions

Medication therapy. The goal of medication therapy, including chemotherapy, is to reduce amyloid protein levels in the blood. Many health care providers recommend combination medication therapy such as

- melphalan (Alkeran), a type of chemotherapy
- dexamethasone (Decadron), an anti-inflammatory steroid medication

These medications can stop the growth of the cells that make amyloid proteins. These medications may cause hair loss and serious side effects, such as nausea, vomiting, and fatigue.

Stem cell transplant. A stem cell transplant is a procedure that replaces a patient's damaged stem cells with healthy ones. Stem cells are found in the bone marrow and develop into three types of blood cells the body needs. To prepare for a stemcell transplant, the patient receives high doses of chemotherapy. The actual transplant is like a blood transfusion. The transplanted stem cells travel to the bone

marrow to make healthy new blood cells. The chemotherapy a patient receives to prepare for the transplant can have serious side effects, so it is important to talk with the health care provider about the risks of this procedure.

Treating other conditions. Primary amyloidosis has no cure, so treating some of the side effects and other conditions seen with the disease is essential. Other conditions may include

- anemia—treatment may include medications
- depression—treatment may include talking with a mental health counselor and taking medications
- fatigue—treatment may include changes in diet and activity level
- kidney disease—treatment may include medications to help maintain kidney function or slow the progression of kidney disease

A patient and his or her family should talk with the health care provider about resources for support and treatment options.

How is dialysis-related amyloidosis treated?

A health care provider treats dialysis-related amyloidosis with -medication therapy newer, more effective hemodialysis filters surgery a kidney transplant

The goal of medication therapy and the use of newer, more effective hemodialysis filters is to reduce amyloid protein levels in the blood. Medication therapy can help reduce symptoms such as pain and inflammation. A health care provider may treat a person with dialysis-related amyloidosis who has bone, joint, and tendon problems, such as bone cysts and carpal tunnel syndrome, using surgery.

Dialysis-related amyloidosis has no cure; however, a successful kidney transplant may stop the disease from progressing.

Eating, Diet, and Nutrition

Researchers have not found that eating, diet, and nutrition play a role in causing or preventing primary amyloidosis of the kidneys or dialysis-related amyloidosis. People with nephrotic syndrome may make dietary changes such as

- limiting dietary sodium (PDF, 167 KB) , often from salt, to help reduce edema and lower blood pressure

- decreasing liquid intake to help reduce edema and lower blood pressure

- eating a diet low in saturated fat and cholesterol to help control more-than-normal amounts of fats and cholesterol in the blood

Health care providers may recommend that people with kidney disease eat moderate or reduced amounts of protein. Proteins break down into waste products that the kidneys filter from the blood. Eating more protein than the body needs may burden the kidneys and cause kidney function to decline faster. However, protein intake that is too low may lead to malnutrition, a condition that occurs when the body does not get enough nutrients.

People with kidney disease on a restricted protein diet (PDF, 112 KB) should receive blood tests that can show low nutrient levels. People with primary amyloidosis of the kidneys or dialysis-related amyloidosis should talk with a health care provider about dietary restrictions to best manage their individual needs.

Learning as much as you can about your treatment will help make you an important member of your health care team.

Clinical Trials

The National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) and other components of the National Institutes of Health (NIH) conduct and support research into many diseases and conditions

4. Analytical part

4.1. Cluster, conceptual table organizers Cluster formation rule

1. Write down everything that comes to your mind. Don't discuss the quality of your ideas, just write them down.
2. Ignore spelling mistakes and other factors that stop the letter.
3. Do not stop writing untilthe allotted time is up. If you suddenly stop coming up with ideas, then keep drawing on paper until new ideas come to you.

CONCEPTUAL TABLE

It provides a comparison of two or more aspects of the studied phenomenon, concept, and ideas.

It develops the skills of systematic thinking, bringing information into structure, systematization.

They get acquainted with the con table of rules. They ident comparables, distinguish characteristics according t

Individually or in small gro complete the conceptual table.

- Comparable by length (opinion are placed;
- Various descriptions are written, carried out according to the com the bed.

Presentation of work resul

Each group evaluates the other groups. 15 points if all requirements are fulfilled

Grou A clear and p clear answer (5)

PROBLEM BASED LEARNING NEPHROTIC SYNDROME.

The patient is 63 years old and has been suffering from chronic pyelonephritis since 43 years old. He was treated many times. Recently, he started to feel bad. Complaints: severe headache, back pain, ringing in the ears, swelling of the face and legs, loss of appetite, general weakness.

Lens: overall condition is relatively satisfactory. The skin is pale, the eyelids and legs are swollen. There is no vesicular breath or wheezing in his lungs. The heart sounds are muffled, the pulse is rhythmic at 84 beats per minute. AQB 100/70 mm.

etc. Abdomen is soft, painless. Liver along the rib cage. The spleen is painless. Pasternasky's symptom is bilaterally positive. Analysis: UQA Er-3.4*10*12 g/l, HB-96 G/l. r/k. -0.9, L-4.6, Echt – 22mm/h. USA: protein 4.8, L-8-10/1, hyaline and waxy cylinders. Biochemistry: ALT 09, bilirubin 14.6 micron/l, urea 8.7 micron/l, creatinine 0.25 micron/l, protein 49 mmol/l, cholesterol 90 mmol/l.

2. Supporting question/trigger.

What disease should be differentially diagnosed with? Glomerulonephritis, Chronic kidney failure.

3. Purpose

1. Studying the etiology, pathogenesis, clinical symptomatology of the disease to the students 2. Teaching laboratory-instrumental diagnostics and rational therapy, prevention of complications, rehabilitation.

4. Special tasks of teaching:

• Determination of clinical signs of nephrotic syndrome • Determination of X-ray changes of nephrotic syndrome • Reasoning of nephrotic syndrome

• Analyzing, checking and strengthening the student's acquired knowledge • Comparative diagnosis with the clinic of chronic kidney failure

• Basic principles of nephrotic syndrome treatment

5. One of the methods of developing questions used for the search for evidence is the formulation of PICO questions:

Population - patients/population Intervention - intervention Control - control
Outcome is a result

In the case of the above, it could be:

Population - patients/population = patients with patients Intervention = reduction of collagen synthesis

Sontrol - control = Cuprenil, glucocorticosteroids, 4-aminoquinoline derivatives, NYAQV, lidase, antiaggregants, drugs that improve blood rheological properties separately or in combination) + alternative interventions

Outcome = reduction of collagen production, pain in the joints, freezing of the fingers, difficulty in swallowing.

Make as many questions as you can for the above situation. Then use the PICO formula and modify this question wherever possible.

1. What is the etiology of nephrotic syndrome? 2. Pathogenesis of nephrotic syndrome?

3. Classification of nephrotic syndrome? 4. Clinical picture of nephrotic syndrome?

5. General inspection, palpation, percussion and hearing indicators? 6. Laboratory-instrumental conclusions?

7. Methods of diagnosis of nephrotic syndrome? 8. Comparative diagnosis of nephrotic syndrome?
9. The main principles of treatment of nephrotic syndrome? 10. Course of nephrotic syndrome, prognosis?

COLLECTION OF CASES CASE: Renal amyloidosis Study subject: Internal diseases. Topic: Renal amyloidosis

The main purpose of the case:

To make students interested in this problem by widening the material. Introducing the latest news in the diagnosis and treatment of this disease. Arousing interest in independent work with literature. To have deep and modern knowledge, broad outlook, independent thinking potential in the diagnosis and treatment of renal amyloidosis, to justify the relevance of the clinical course of the disease and modern methods of diagnosis and treatment at the current level. Drawing up a treatment plan and developing measures to prevent the disease.

Expected results of educational activities:

• Being able to diagnose kidney amyloidosis early, showing its causes and pathogenesis;

• Explain the treatment plan for renal amyloidosis and disease prevention measures;

• Application of theoretical knowledge in solving problematic tasks; • Identifying a problem and finding a solution to solve it.

In order to successfully implement this case, students must have the following knowledge and skills:

The student should know:

- The etiology, pathogenesis, classification, diagnostic methods, basic principles of treatment, dispensation, rehabilitation and prevention of renal amyloidosis

The student must:

- learnsthe subject independently; clarifiesthe nature ofthe problem, puts forward ideas, examines information from a critical point of view, learns to make independent decisions; has his own point of view and makes a logical conclusion; works independently with educational information; compares, analyzes and summarizes data.

A student should have:

- communicative skills; presentation skills, collaborative work skills; to the skills of analyzing problematic situations.

List of recommended literature for using sources: • Evidence-based medicine.

• Clinical recommendations. Evidence-based medicine • PubMed articles.

Medical history

Diagnostics of diseases of the internal organs. A.N. Okorokov. Moscow 2005 Lechenie bolezney vnutrennix organov. A.N. Okorokov. Moscow 2005 Clinical recommendations for practicing physicians. Moscow 2002

Evidence-based medicine. Moscow 2003

Description of the case based on technological features:

The main source ofthis case is a cabinet, a record, and it is described on the basis of life situations. The main object of the case is person-oriented. This is an organizational and institutional case, based on data, situations and questions. It is moderately structured and aimed at creating knowledge and skills on the educational subject intended for training. According to didactic purposes, cases are aimed at presenting problems, solving them, analyzing and evaluating them. This case can be used in the subject "Internal diseases" and in open class hours.

Clinical event:

A paramedic came to the home of a 42-year-old patient. Complaint: nausea, vomiting, diarrhea, weight loss, loss of appetite, constant headache, severe general weakness. He got sick 8 years ago, received inpatient treatment, was treated during attacks. During the attack, according to the patient's words, swelling on the face and legs, elevation of AQB was observed. Urine has changed to "meat wash" color. His condition has worsened for 10 days.

Objective: body temperature is 37.3°C. his general condition is serious, his face is swollen. He is thin, his skin is pale, dry, and his mouth smells of ammonia. Shortness of breath, few moist rales in the lower extremities. NOS 24 in 1 minute. The left relative border of the heart is 1 cm beyond the midline. Heart tones are muffled, accent of the 2nd tone in the aorta. The pulse is 76 in 1 minute, rhythmic, tense. AQB 170/100 mm.s.ust. The tongue has a moist discharge. Abdomen is soft, with a little pain in the iliac region..

Questions:

1. What additional examination methods are used to determine the diagnosis?

2. In your opinion, which diseases should be compared with comparative diagnosis? 3. List the diagnostic criteria.

4. What is your diagnosis and justify it? 5. Create a treatment plan.

Task: based on the condition of a patient with chronic kidney disease, make an initial diagnosis, apply the necessary examination methods, and draw up a reasonable treatment plan.

• Chronic kidney disease is a complex of clinical symptoms associated with the disruption of the homeostatic activity of the kidneys as a result of irreversible changes (progressive destruction of nephrons) caused by chronic diseases (glomerulonephritis, pyelonephritis, amyloidosis, etc.). Statistics. The incidence rate of BE varies widely, ranging from 8 to 95 per 100,000.

CAUSES: hereditary nephropathies, kidney dysembryogenesis, chronic glomerulonephritis, chronic pyelonephritis

diabetes, obstructive diseases, hypertension, gout, drug-related nephropathy

The goal of treatment: to reduce the rate of progression of SBYE while maintaining the patient's quality of life satisfactorily.

Tasks: exclusion of factors that cause damage to intact nephrons: taking nephro-toxic drugs is prohibited, the level of infection and intoxication is minimized; - reduction of azotemia due to optimization of paresis mode; - control of kidney-related hypertension; - correction of anemia, electrolyte and metabolic disorders.

Diet

One of the methods of treatment of patients with SBYE. Low protein diet

1. Treatment of AG has a pathogenetic nature, as it slows down fibroplastic processes in the kidney. ß-blockers, vasodilators, Ca antagonists, and thiazide diuretics are used for snoozing.

2. Treatment of acidosis: sodium bicarbonate is administered intravenously, but the amount is given based on a special formula and the single dose should not exceed 200 ml.

3. Correction of phosphorus-calcium metabolism disorders: Sa carbonate, amphogel, alukol are used and fasting.

4.Erythrocyte mass, testosterone,retabolil, erythropoietyare used against anemia. Lidocaine 100 mg intravenously and cholestyramine 5 grams twice a day are used against skin itching.

5. Intestinal dialysis: 1 liter of a solution containing Na chlorine, Sa chlorine, bicarbonates, sorbitol or mannitol is drunk in 1 hour, then diarrhea starts and nitrogen substances are released. This is done 2-3 times a week during the intermittent period of SBE.

In the terminal stage, replacement therapy and chronic hemodialysis are used.
In order to successfully implement the case, it is necessary to have knowledge and skills about the anatomical structure and physiology of the urinary system.
Solving the case allows you to achieve the following results: • Consolidation of knowledge on the mastered topic;
• Transfer of knowledge and skills in individual and group analysis of the problem and the adopted solution;
• Development of logical thinking;
• Acquisition of independent decision-making skills;
• Check the level of assimilation of educational information.
METHODICAL GUIDELINES FOR STUDENTS ON STEP-BY-STEP ANALYSIS AND SOLUTION OF A PRACTICAL SITUATION
1. Instructions to students
2. 2.1 Problem: Inpatient diagnosis and treatment of patients with chronic kidney failure
2.2. Small problem Analysis of the patient's appearance
Analysis of disease history and life history Analysis of examination results
Selection of necessary inspection methods
Analyzing the results of the examination and conducting a comparative diagnosis Determination of treatment tactics.
2.3. Solving algorithm
Analysis of the patient's appearance includes: - constitution, stature, gait
- face, body
2. Anamnesis analysis: - medical history
- transmitted diseases
- family and social anamnesis 3. Inspection analysis
- palpation of the kidneys - percussion of the kidneys
- auscultation of lungs and heart
4. Selection of necessary diagnostic methods: - general analysis of blood and urine
- blood biochemical analysis
- determination of protein in daily urine - acute phase tests
- Zimnitsky, Nechiporenko, Addis-Kokovsky tests - EKG
- UTT kidneys - chest X-ray
5. Analysis, comparison and comparative diagnosis of the obtained results: - glomerulonephritis
- pyelonephritis
- congenital kidney defects - with nephropathies
6. Selection of treatment tactics - use of traditional medicine
- use of kidney transplantation - use of hemodialysis

Option for the teacher to solve and analyze the case The main issue in the case Timely and correct diagnosis ofchronic kidney failure, development ofa treatment plan, extension of the remission period, development of prophylactic measures.

Ways to solve the problem situation

• Let's get acquainted with the case and its information supply We determine the level of problem solving

• Chronic kidney failure is a complex of clinical symptoms associated with the disruption of the homeostatic activity of the kidneys as a result of irreversible changes (progressive destruction of nephrons) caused by chronic diseases (glomerulonephritis, pyelonephritis, amyloidosis, etc.). Statistics. The incidence rate of BE varies widely, ranging from 8 to 95 per 100,000.

CAUSES: hereditary nephropathies, kidney dysembryogenesis, chronic glomerulonephritis, chronic pyelonephritis diabetes, obstructive diseases, hypertension, gout, drug-related nephropathy

The goal of treatment: to reduce the rate of progression of SBYE while maintaining the patient's quality of life satisfactorily.

Tasks: exclusion of factors that cause damage to intact nephrons: taking nephro-toxic drugs is prohibited, the level of infection and intoxication is minimized; - reduction of azotemia due to optimization of paresis mode; - control of kidney-related hypertension; - correction of anemia, electrolyte and metabolic disorders.

Low protein diet

Correction of phosphorus-calcium metabolism disorders: Sa carbonate, amphogel, alukol are used and fasting.

Erythrocyte mass, testosterone, retabolil, erythropoiet are used against anemia. Lidocaine 100 mg

intravenously and cholestyramine 5 grams twice a day are used against skin itching.

Intestinal dialysis: 1 liter of a solution containing sodium chloride, sodium chloride, bicarbonates, sorbitol or mannitol is drunk in 1 hour, then diarrhea begins and nitrogen substances are excreted. This is done 2-3 times a week during the intermittent period of SBE.

In the terminal stage, replacement therapy and chronic hemodialysis are used. Treatment of patients with SBE in the terminal stage

1. Peritoneal dialysis (used in 10% of patients) - in which various substances from blood and body fluids are dialyzed through a solution injected into the abdominal cavity. Peritoneum acts as a dialysis barrier.

2. Hemodialysis - the products of nitrogen exchange are removed from the blood by diffusion through a semi-permeable membrane. This session lasts 5-6 hours, is held 2-3 times a week, and prolongs the life of patients. Contraindications: - heart failure with dampness in large and small blood circulation; - infectious diseases; - oncological diseases; -tuberculosis; - period of attack of gastrointestinal ulcers; - severe liver diseases - mental patients; - hemorrhagic syndrome

3. Consequencesofkidneytransplantation: SBE canrarely be reversible. In most cases, the process ends with a terminal stage, which requires the correction of kidney function. Exacerbation of SBE occurs due to acute dehydration and severe sodium restriction, excessive intake of diuretics, urinary tract infection and obstruction, hypercalcemia, and hyperuricemia. According to Snuning, arterial hypertension, expressed proteinuria, smoking, and hyperlipidemia are the factors that accelerate SBE.

Let's get acquainted with the given situation

After carefully reading the data once again, we mark the lines that are important for us. Before moving fromone paragraphto another, weread it two orthreetimesand get into itscontent. We underline the important points in the case with a pencil. We draw your attention to the main concepts and expressions given in the description of the situation.

3. Analysis of the problem situation.

We will prepare the presentation. In this case, we find all possible solutions to the problem: ***Problem Type:***

Problem 1. List the factors that cause the development of chronic kidney failure. Causes of the problematic situation

• hereditary nephropathies, kidney dysembryogenesis, chronic glomerulonephritis, chronic pyelonephritis

diabetes, obstructive diseases, hypertension, gout, drug-related nephropathy Vaziyatdan chiqib ketish harakatlari

Diet should be observed. Problem Type:

Problem 2. Principles of drug treatment of chronic kidney failure. Causes of the problematic situation

exclusion of factors that cause damage to intact nephrons Actions to get out of the situation

- it is forbidden to take nephro-toxic drugs, the level of infection and intoxication is minimized; -reduction of azotemia due to optimization of paresis mode; - control of kidney-related hypertension; - correction of anemia, electrolyte and metabolic disorders.

Problem Type: Problem 3.

Treatment of chronic renal failure in the terminal stage Actions to get out of the situation

It is necessary to identify the disease in the early stages and apply rational treatment measures

Description of the case based on technological features:

The main source ofthis case is a cabinet, a record, and it is described on the basis of life situations. The main object of the case is person-oriented. This is an organizational institutional case, based on data, situations and questions. The volume is moderately structured and aimed at creating knowledge and skills on the training subject. According to didactic purposes, cases are aimed at presenting problems, solving them, analyzing and evaluating them.

This case can be used in the subject "Internal diseases" and in open class hours. Clinical event:

Patient Z., 35 years old, COMPLAINTS: pain in the joints of the legs, difficulty walking, severe pain in the first toe in the evening, redness, swelling, sleep disorders, headache, nausea, general weakness .

Lens: overall condition is average. Skin mucous membranes are pale, clean. Vesicular breath in the lungs. Heart tones are muffled, tachycardia. AKB 160/90 mm. cm. Pulse 92 in 1. The tongue is wet and clean. The abdomen is soft and

painless. Diarrhea and urination are normal. Redness of the right big toe, pain in this finger, limited movement, limping. Tophus is observed in the left elbow joint.
Laboratory tests: General blood analysis: NV-100 g/l, L-5.4, ROE-27mm/s.
General urine analysis: protein - 0.033‰, L - 8-10/1, epithelium - 4-6/1, salts - urates ++ UZI: the presence of salt in the left kidney was detected.

Questions:

1. What additional examination methods are used to determine the diagnosis?
2. In your opinion, which diseases should be compared with comparative diagnosis? 3. List the diagnostic criteria.
4. What is your diagnosis and justify it? 5. Create a treatment plan.

Task: based on the condition of a patient with nephrotic syndrome, make a preliminary diagnosis, apply the necessary examination methods, and draw up a reasonable treatment plan Nephrotic syndrome is a complex of clinical laboratory symptoms, including massive proteinuria, protein, lipid, water-salt metabolism disorders, as well as swelling up to the development of anasarca, which develops in primary or other, often systemic diseases of the kidney.

Etiology

I Primary: Glomerulonephritis, Pyelonephritis, Nephropathy of fetuses, Renal tumors II Secondary

BTDK, RA, Systemic vasculitis, septic endocarditis, bronchi, lungs, chronic purulent diseases of the bone and joint system, tuberculosis, diabetes, allergic diseases, when taking D-pencillamine, gold preparations and other drugs

Pathogenesis: immunological disorders, increased glomerular filtration for proteins, development of hypoalbuminemia, decrease in oncotic pressure of plasma, and loss of fluid to tissues,

hypovolemia leads to increase in production of renin, aldosterone and antidiuretic hormone. sodium and water are retained, sodium concentration decreases in oligouria and urine, sodium and water enter the tissues and swelling increases, hypoalbuminemia increases the formation of lipoproteins. the amount of lipids, cholesterol and phospholipids always increases

Nephrotic syndrome clinic

general weakness, pain in the lower back, loss of appetite, daytime nausea, nausea, swelling, oliguria, thirst, mouth sores, swelling in the face, legs, body, chest, later development of anasarca, ascites, hydropericardium, hydrothorax, dry skin , nails and hair are brittle, dull

• enlargement of the liver, enlargement of the left border of the heart, weak systolic murmur at heart rate, arterial pressure is expected or normal, nephrotic crisis, straining syndrome, hypokalemia

Passing

• Episodic (constant)

• Persistent – regardless of active treatment in 5-8 years SBE • Progressive – in 1-3 years SBE

Laboratory indicators

Signs of anemia, increased ECHT, hypoproteinemia, hypoalbuminemia, increased 2 and globulins, , hypercholesterolemia., creatinine, increased urea, massive proteinuria (more than 3.5-5 g/day), cylinduria, microhematuria, high urine density, SBE does not develop, foamy urine

is an early sign. Verification program

Total blood analysis, Zimnisky, Nechiporenko's urinalysis, daily proteinuria analysis, fractions of total protein, cholesterol, triglycerides, lipoproteins, urea, creatinine, Reberg-Tare test, Kidney ultrasound, Radioisotoprenography and kidney scan, ECG, Eye examination

Treatment of nephrotic syndrome No. 7 B

Treatment of kidney pathology

Protein solutions, parenteral administration of plasma Diuretics (furosemide). Potassium stores - veroshpiron

Anticoagulants (heparin 10 thousand units 2 times a day under the control of blood clotting) Antibiotics - in secondary infection

NYAQV has a temporary antiproteinuric effect APF inhibitors - captopril - 50-100mg per day Hypolipidemic means - statins

In nephrotic crisis - filling the circulating blood volume with plasma substitutes, antikinin agents (parmidine). antihistamines, antibiotics

Plasma ultrafiltration - in torpid transition to diuretics

Nephrectomy with subsequent chronic hemodialysis and kidney transplantation.

In order to successfully implement the case, it is necessary to have knowledge and skills about the anatomical structure and physiology of the spine, joints, ligament apparatus.

Solving the case allows you to achieve the following results: • Consolidation of knowledge on the mastered topic;

• Transfer of knowledge and skills in individual and group analysis of the problem and the adopted solution;

• Development of logical thinking;

• Acquisition of independent decision-making skills;

• Check the level of assimilation of educational information.

METHODICAL GUIDELINES FOR STUDENTS ON STEP-BY-STEP ANALYSIS AND SOLUTION OF A PRACTICAL SITUATION

Instructions to students

2.1 Problem: Diagnosis and treatment of patients with nephrotic syndrome in inpatient conditions

2.2. Small problem Analysis of the patient's appearance

Analysis of disease history and life history analysis Analysis of examination results

Selection of necessary verification methods

Analyzing the results of the examination and making a comparative diagnosis Determination of treatment tactics.

2.3. Solving algorithm

1. Analysis of the patient's appearance includes: - constitution, stature, gait
- face, body
2. Anamnesis analysis: - medical history
- transmitted diseases
- family and social anamnesis 3. Inspection analysis
- palpation of the kidneys - percussion of the kidneys
- auscultation of lungs and heart
4. Selection of necessary diagnostic methods: - general analysis of blood and urine
- blood biochemical analysis
- determination of protein in daily urine - acute phase tests
- Signs of Zimnitsky, Nechiporenko, Addis-Kokovsky - EKG
- UTT kidneys - chest X-ray
- Radioisotope renography and kidney scan
5. Analysis, comparison and comparative diagnosis of the obtained results: - renal amyloidosis
- glomerulonephritis - pyelonephritis
- chronic kidney failure - with nephropathies
6. Selection of treatment tactics - use of traditional medicine
- use of surgical procedures - use of plasmapheresis

6. Practical skills

№	List of practical skills	number	Necessary equipment (equipment) for practical training
9- semester			
1	Peakflowmetry for nosologies	1	textbooks, literature, photographs, phantoms, dummies, simulators,
2	Deciphering pathological changes	1	

	on the ECG with nosologies		equipment, tables, training and control tests, computer programs, evening duty for clinical subjects, volunteering, work in similation centers.
3	Blood pressure measurement	1	
4	The use of inhalers for bronchial obstruction	1	
5	First aid for choking	1	

Algorithm for step-by-step implementation of practical skills:

PEAK FLOWMETRY

№	Sequence of execution
1	The patient must sit comfortably
2	Set the scale to "0"
3	Treating the peak flow meter with alcohol
4	The patient should take a deep breath
5	The patient should exhale with all his might at the peak flow meter.
6	Bring the scale to "0" and repeat the procedure twice
7	Record the highest result in the protocol
8	Monitor during the day (morning, afternoon, evening)
9	Observe the dynamics of the peak flow meter
10	Rinse and dry the peak flow meter

ECG INTERPRETATION IN NOSOLOGIES

№	Sequence of execution
1	Know how to place the electrodes correctly
2	Record at least 4 PQRST cardiac cycles per lead
3	Identify sources of excitement
4	Calculate number of heart contractions
5	Determination of the electrical axis of the heart
6	Determination of the position of the heart
7	Give the correct conclusion

BLOOD PRESSURE MEASUREMENT

№	Sequence of execution
1	Blood pressure measurements are taken with a break of 3-5 minutes while the patient is sitting. When measuring blood pressure, the patient should be calm and not talk
2	Measurement of blood pressure begins with the right arm, the patient's shoulder should be at the same level with the heart
3	The bottom of the cuff should be 2.5-3 cm above the elbow joint
4	The brachial artery is found in the ulnar cavity, and a phonendoscope is placed there.
5	Air is supplied to the cuff until the pulse disappears and air is released
6	The appearance of the first tone corresponds to systolic blood pressure, the disappearance of tones - to diastolic pressure
7	Pulse is measured by the interval between systolic and diastolic pressure
8	Air is removed from the pressure gauge cuff.
9	Air is removed from the pressure gauge cuff.
10	And these actions are performed by both hands

TECHNIQUE USE OF INHALERS IN CASE OF ASTHMA ATTACK

№	Sequence of execution
1.	Shake the inhaler several times
2.	Hold the inhaler with your hand and open the lid vertically so that your fingers do not cover the inhaler openings
3.	Exhale deeply. Pinch the nozzle with your lips, press down on the inhaler and inhale as much as possible through the nozzle
4.	Hold your breath for a few seconds. Exhale slowly
5.	Close the inhaler lid

FIRST AID FOR AN ATTACK OF SUFFOCATION

№	Sequence of execution

1.	Eliminate the factor that caused the asthma attack
2.	Give patient inhalation of short-acting beta2-agonists (salbutamol, berotec) - 1 dose every 20 minutes
3.	Intravenous administration of aminophylline 2.4% - maximum dose 5.6 mg / kg. maintenance dose - 6 mg / kg.
4.	Intravenous administration of methylprednisolone at the rate of 1-5 mg / kg of body weight (in 10 ml of 0.9% sodium chloride solution).
5.	Monitoring the effectiveness of bronchodilator therapy (disappearance of dryness, wheezing in the lungs during auscultation, improvement of the general condition of the patient).

Information supply of student independent work

Independent work assignments in the field of internal medicine are compiled based on the model program and approved by the head of the department. In the assignment given to the student, initial instructions and recommendations for independent work are recorded.

Textbooks and training manuals, methodological manuals and instructional data sets, scientific and public periodical publications, relevant information on the Internet, previous work on the given topic are used as a source of information for the student to perform independent work. works and others serve.

The management of the academy created conditions for students to use computer equipment and the Internet effectively to complete independent work on time.

Based on the above, the department recommends the following forms for the student's independent work:

• Preparation of abstract on the subject of TMI (10-15 pages).

• Document preparation on the subject of TMI (15-20 min.) provided that the text is submitted.

• Organization of a presentation on the topic of TMI, with the condition of submitting a report or abstract.

• To provide information about modern clinical-diagnostic examination methods, scientific technologies.

• Development and formal submission of handouts (tables, schemes, pictures, albums, graphs, organizers).

• Development of tests on the topic of TMI (at least 20 tests for one topic). • Creating and conducting a script of interactive games on the topic of TMI.

• Development of crosswords, scanwords, chainwords, rebuses on the subject of the training.
• Working with scientific literature (writing magazine articles, monographs, abstracts and reports on Medline), printing 10 pages.

Handouts: Situational issues

1. The patient is 63 years old and has been suffering from chronic pyelonephritis since 43 years old. He was treated many times. Recently, he started to feel bad. Complaint: severe headache, back pain, ringing in the ears, swelling of the face and legs, loss of appetite, general weakness. Lens: overall condition is relatively satisfactory. The skin is pale, the eyelids and legs are swollen. There is no vesicular breath or wheezing in his lungs. The heart sounds are muffled, the pulse is rhythmic at 84 beats per minute. AQB 100/70 mm. etc. Abdomen is soft, painless. Liver along the rib cage. The spleen is painless. Pasternasky's symptom is bilaterally positive. Analysis: UQA Er-3.4*10*12 g/l, HB-96 G/l. r/k. -0.9, L-4.6, Echt – 22mm/h. USA: protein 4.8, L-8-10/1, hyaline and waxy cylinders. Biochemistry: ALT 09, bilirubin 14.6 micron/l, urea 8.7 micron/l, creatinine 0.25 micron/l, protein 49 mmol/l, cholesterol 90 mmol/l.
I. Your diagnosis:
A. Chronic pyelonephritis in remission
B. Secondary amyloidosis, nephrotic stage* V. Primary amyloidosis
G. Chronic glomerulonephritis
II. Method of additional examination to substantiate the diagnosis of renal amyloidosis: A. Detection of Bence-Johnson protein in urine
B. UTT of the kidneys and X-ray of the chest V. Kidney, gum and rectal mucosal biopsy*
G. Determination of protein fractions in blood and urine

2. The patient is 45 years old, his complaints are: severe weakness, swelling of the whole body, headache, ringing in the ears, pain in the joints, morning sickness, as well as shortness of breath and palpitations. In the anamnesis: the swelling suddenly appeared 3 months ago, he drank medicinal herbs. He has been suffering from rheumatoid arthritis for 8 years and receives inpatient and sanatorium treatment every year. In recent days, his condition has worsened, the swellings have increased, the pains in the joints have increased, and panting has appeared. General condition is serious, skin: pale, anasarka. Vesicular breath in the lungs, wet wheezes with small bubbles in the lower parts. Heart sounds are muffled, soft systolic murmur is heard at the peak, pulse is 88 beats per minute. AQB 120/80 mm. Liver +1-1.5 cm. Pasternasky's symptom is 2-sided positive. UQA: Er -

3.4*10*12 g/l, Hb 80 g/l, ECHT 53 ml/h, leu-10*10*9. USA: protein 3.8‰, waxy cylinders. Er-8-10/1, ley 8-10/1, epit 0-1/1. Biochemistry, analysis: total protein 50 g/l, cholesterol 10 micro/l, creatinine 2.5 μmol/l. Amyloid protein in kidney and other organ biopsies.

I. Your diagnosis:

A. primary amyloidosis nephrotic stage

B. RA, polyarthritis, secondary amyloidosis, terminal stage* C. secondary amyloidosis, proteinuric stage

D.RA, polyarthritis renal secondary amyloidosis

II. Assign additional treatment to the main treatment of the disease: A. Detoxification therapy: 5% glucose solution, diuretics

B. diet, protein and salt removal from the diet, fluid restriction, plaquinil, diuretics

C. restriction of protein in food, fluid intake at the rate of urine output + 30 ml of fluid, hemodialysis*

D. Surgical treatment

3. Patient S, 78 years old, complains: loss of appetite, headache, ringing in the ears, drowsiness, weakness, frequent diarrhea. Anamnesis: only suffered from ORVI in his youth. Lens: the general condition is satisfactory, skin covers are pale, wet. Vesicular breath in the lungs. Heart sounds muffled, rhythmic, bradycardia, accent II in the aorta. Abdomen is soft, painless. Pasternasky's symptom is 2-sided weakly positive. Diuresis has not changed. Watered inside. Analysis: UQA: Nv – 90 g/l, L – 6.4 ECHT – 18 mm/s. USA - protein - 6 ‰ solution. 4-6/1 L – 4-6/1 hyal. And waxy cylinders. Total protein – 49 g/l, urea – 9.8 mmol/l, creatinine 0.5 mmol/l, cholesterol – 89 mmol/l

I. Your diagnosis:

A. Chronic glomerulonephritis, initial stage. B. Primary renal amyloidosis

C. Senile amyloidosis, initial stage *

D. Secondary amyloidosis, terminal stage.

II. Which additional examination method confirms the diagnosis: A. Kidneys are LONG

B. Kidney R-graphy

C. Renal biopsy * D. EKG

4. Patient SH. 63 years old, complaints: sweating, rapid fatigue, loss of appetite, drowsiness, swelling of legs. Anamnesis: suffered from pulmonary tuberculosis in his youth. Lens: the general condition is satisfactory, skin covers are pale, moist. Weak vesicular breath in the lungs. Heart tones are muffled, rhythmic. Pulse 96 1 min. Pasternasky's symptom is 2-sided weakly positive. Analyzes: UQA hem.90

g/l ECHT –26 mm/s. Total protein - 56 g/l, hypoalbuminemia. General urine analysis: protein 0.132 ‰.

I. Which additional examination method helps to determine the diagnosis: A. Blood biox. analysis: urea and creatinine; EKG.

B. Kidneys and liver UTT.

C. Biopsy of kidneys and gingival submucosa. D. All of the above *

II. Your initial diagnosis:

A. Intensification of the tuberculosis process. B. Secondary renal amyloidosis, latent stage* C. Primary renal amyloidosis.

D. Pulmonary sarcoidosis

5. Patient K. 77 years old, primary amyloidosis was diagnosed, in the analysis: proteinuria 4.5 ‰, hypoproteinemia 45 g/l, creatinine 0.225 mmol/l CF 40g/l.

I. Recommendations for the patient about fluid, protein, salt intake:

A. Salt restriction, protein 20-40 g/s (taking into account CF, proteinuria and proteinemia), liquid - separate + 300 ml *

B. Exclusion of salt, equal to the amount of protein, liquid-separated

C. Exclusion of salt, protein is not less than 80-120 g./s, limitation of liquid amount= separated -200 ml (in case of taking into account the massiveness of tumors and KF)

D. Salt restriction, protein exclusion, fluid restriction, management of edema with diuretics.

II. A group of drugs effective in the treatment of amyloidosis: A. GKS and nonsteroidal anti-inflammatory drugs

B. Immunodepressants and vitamins C. Antibiotics and biostimulants

D. 4-aminoquinoline drugs and unitiol*

6. Patient K. 77 years old, Primary amyloidosis was diagnosed, in the analysis: proteinuria 4.5 ‰, hypoproteinemia 45 g/l, creatinine 0.225 mmol/l CF 40g/l.

I. Recommendations for the patient about fluid, protein and salt consumption:

A. Salt restriction, protein 20-40 g/s (taking into account CF, proteinuria and proteinemia), liquid - separate + 300 ml *

B. Exclusion of salt, equal to the amount of protein, liquid-separated

C. Exclusion of salt, protein is not less than 80-120 g./s, restriction of liquid amount= separated -200 ml (taking into account the massiveness of tumors and KF)

D. Salt restriction, protein exclusion, fluid restriction, management of edema with diuretics. II. A group of drugs effective in the treatment of amyloidosis:

A. GKS and nonsteroidal anti-inflammatory drugs B. Immunodepressants and vitamins
C. Antibiotics and biostimulants
D. 4-aminoquinoline drugs and unitiol*

7. Patient S, 78 years old, retired. Complaints: loss of appetite, headache, ringing in the ears, drowsiness, weakness, frequent diarrhea. In anamnesis: no diseases.
Analyzes: UQA: er-3,4. Nv – 90 g/l, R/K 0.9 L – 6.4 ECHT – 18 mm/s. USA - protein - 6 ‰ solution. 4-6/1 L – 4-6/1 hyal. and waxy cylinders. Total protein – 49 g/l, urea – 9.8 mmol/l, creatinine 0.5 mmol/l, cholesterol – 8.9 mmol/l
I. Your diagnosis:
A. Chronic glomerulonephritis, initial stage. B. Primary renal amyloidosis
C. Senile amyloidosis, initial stage *
D. Secondary amyloidosis, terminal stage.
II. Which additional examination method confirms the diagnosis: A. Kidneys
B. Kidney R-graphy C. Renal biopsy * D. EKG

9. Bemor Karimov S. 20 years old. He came to the clinic with the following complaint: urine to look like an interesting meat yuvundus, headache, swelling under the eyes. On examination, the face is slightly swollen, pale, Pasteurpasiky's sign is positive on palpation.
1. Do you have a diagnosis? A. Acute glomerulonephritis. B. Acute pyelonephritis.
C. renal amyloidosis.
D. chronic glomerulonephritis.
2. What additional inspection should be done? A. roengenoscopy.
B. UTT
C. Rebert test.
D. general analysis of urine.
10. Patient Akhmedov V. came to the clinic for pain in the lower back, fever rising to 37.5, weakness, dark circles under the eyes and slight swelling when he got up in the morning. The condition is satisfactory. came with a complaint. The color is a little pale in the vision, lekasites 15-20, protein 0.66 in the general analysis of urine. Pasterpasky's symptom is positive.
1. Your diagnosis?
A. collected pyelonephritis. B. acute gromerulonephrine. C. renal tuberculosis.
D. acute pyolonephrine. E. renal amyloidosis.
2. What additional inspection should be done? A. X-ray
B. UTT C. Kidney
D. Ziminovsky's symptom. E. Robert's symptom.

1. A 20-year-old patient came to the department with the following complaints: shortness of breath after physical activity, stabbing pain in the heart area, body temperature rising to 37.8°C. He associates the onset of the disease with staying in the cold. Objective: the general condition of the patient is moderate, the skin is pale, the lips are cyanotic. Vesicular breathing is heard in the lungs. The boundaries of the percussive heart are widened to the right and left. Heart sounds are muffled, pericardial friction noise is heard. Pulse 100 beats per minute. The liver is not enlarged, I, II on ECG, T is negative on AVL. In the general blood analysis, leukocytosis, ECHT is equal to 30 mm / s. Your diagnosis:
A. NSD, cardiac type
B. Acute rheumatic fever
C. Infectious-allergic myocarditis. Pericarditis* D. Systemic lupus erythematosus, lupus-carditis
What additional methods of examination should be conducted to confirm the diagnosis? A) ExoKS, chest X-ray*
B) Acute phase tests, UTT
V) X-ray chest X-ray, AlT, AsT, LDG

2. The patient, 25 years old, turned to the doctor with the following complaints: palpitations, throbbing pains in the heart area, shortness of breath after physical exertion, an increase in body temperature up to 37-38°C, severe weakness. He connects the onset of the disease with viral hepatitis. Lens: the general condition is medium, the skin is pale, the lips are cyanotic. Heart boundaries are slightly shifted to the right and left. Heart tones are muffled, extrasystole. Pulse 96 beats 1min. AQB 100/70 mm wire. above equal to Blood analysis: leukocytosis, with a shift to the left, ECHT 26 mm/s. Urine analysis is unchanged. Your diagnosis:
A) NSD is cardiac type
B) Acute rheumatic fever
C) Infectious myocarditis*
D) TQB, lupus-carditis
What additional tests should be performed to confirm the diagnosis:
A) Acute phase tests, coagulogram, blood smear
B) Chest X-ray, blood biochemical analysis
C) ECG, ExoKS, Acute phase tests*
TESTS:
1. Specify the type of amyloidosis:
A. local, tumorous amyloidosis*
B. autoimmune
C. pediatric amyloidosis

D. infantile amyloidosis
E. juvenile amyloidosis

2. Show the theory of amyloidosis pathogenesis:
A. theory of dysproteinosis*
B. theory of dysglycogenesis
C. infectious
D. theory of mitochondrial secretion
E. theory of chromosome aberrations

3. Show the stage of secondary amyloidosis:
A. proteinuric (latent)*
B. pre-clinic
C. clinical
D. outbreak
E. terminal

4.Give a characteristic sign for genetic amyloidosis:
A. nephrotic*
B. hemorrhagic
C. angiotrophic
D. cardiac

5. Symptoms of nephrotic syndrome are often:
A. cylindruria *
B. proteinuria 3.5g/milk
C. hypoproteinemia
D. dysproteinemia
E. hypercholesterolemia

6. Indicator of proteinuria in neurotic syndrome:
A. More than 3.5 g/milk*
B. Less than 3.5 g/milk
C. 1 g/milk
D. Up to 3 g/milk

7. A characteristic sign for the nephrotic stage of amyloidosis:
A. anemia*
B. hyperkalemia

C. hypernatremia
D. hyperproteinemia
E. hyperglycemia

8. Specific laboratory indicators for the nephrotic stage of amyloidosis:
A. Slight increase in creatinine*
B. hyperkalemia
C. hypernatremia
D. hyperproteinemia
E. hyperglycemia

9. The main difference between nephrotic syndrome and amyloidosis:
A. pronounced dysproteinemia (hypoalbuminemia + hyper-alpha2- and hypergammaglobulinemia)*
B. expressed dysproteinemia (hyperalbuminemia + hyperV globulinemia)
C. hyperglycemia
D. appearance of Bensa-Jones protein in urine

10. Material is taken for biopsy in amyloidosis:
A. From the mucosa or submucosa of the sigmoid colon*
B. From the mucosa or submucosa of the stomach
C. From the mucosa or submucosa of the duodenum
D. From the hair follicle

11. Show 5 types of renal amyloidosis according to etiological and pathogenetic signs:
A. genetic*
B. primary*
C. secondary*
D. old age*
E. local tumor amyloidosis*
F. autoimmune
G. in infants
H. in children
I. in teenagers

12. Show 4 theories of amyloidosis pathogenesis:
A. theory of dysproteinosis*
B. immunological theory*

C. the theory of local cell secretion*
D. mutational theory*
E. theory of dysglycogenesis
F. infectious theory
G. theory of mitochondrial secretion
H. theory of chromosomal aberrations
13. Show 4 clinical stages of secondary amyloidosis:
A. dysproteinemic (preclinical, preamyloidosis)*
B. proteinuric (latent)*
C. nephrotic*
D. azotemic (uraemic)*
E. to the clinic
F. clinical
G. is on the rise H. terminal

14. Show 3 syndromes characteristic of genetic amyloidosis:
A. nephrotic*
B. neuropathic*
C. Cardiopathic*
D. hemorrhagic
E. angiotrophic
F. cardiac

15. Show 4 additional (in addition to the 3 main) syndromes observed in genetic amyloidosis:
A. gastrointestinal damage*
B. fever*
C. anemia*
D. ECHT increase*
E. nephrotic
F. neuropathic
G. cardiopathic
H. hemorrhagic I.
hypothermic

16. In amyloidosis, indicate 3 zones where material can be obtained for biopsy:
A. mucosa and submucosa of the sigmoid colon*
B. mucous and submucous layer of gums*
C. skin with subcutaneous base*

D. mucosa and submucosa of the stomach
E. Mucous and submucosa of the duodenum
F. hair

17. List 3 specific tests that help distinguish secondary amyloidosis from other types:
A. detection of Bence-Jones protein in urine*
B. thermoprecipitation test*
C. Increased Ig G*
D. daily protein loss
E. Increased Ig A
F. hypoproteinemia
18. Name the 4 main syndromes in primary amyloidosis:
A. enteropathy*
B. Cardiopathic*
C. neuropathic*
D. nephrotic*
E. hemorrhagic
F. splenomegalic
G. cardiac
H. cholestatic
19. Name the 3 main groups of diseases that cause secondary amyloidosis:
A. chronic infectious diseases (mostly tuberculosis)*
B. chronic purulent diseases (bronchiectasis, lung abscess, osteomyelitis, etc.)*
C. connective tissue systemic diseases and rheumatoid arthritis.*
D. acute infectious diseases
E. acute purulent diseases of lungs and bones
F. deforming osteoarthrosis and gout
20. List 6 laboratory changes characteristic of primary amyloidosis:
A. hypochromic anemia*
B. leukocytosis*
C. ECHT increase*
D. hyperfibrinogenemia*
E. cholesterolemia*
F. hyper-beta-lipoproteinemia*
G. erythremia
H. leukopenia
I. hyperproteinemia
21. Amyloid is composed of 3 components?

A. component that forms fibrils (F-component)*
B. plasma component (R-component)*
C. hematogenous inclusions (albumins, globulins, fibrin, SIK, etc.)*
D. carbohydrate that forms fibrils
E. serum component (F-component)
F. hemoglobin supplements

22. Indicate the main 3 ways to influence the treatment of amyloidosis:
Amyloidosis developed against the background of
A. to the main disease (secondary)*
B. to mechanisms of pathogenesis*
C. to the main clinical syndromes*
D. etiological
E. palliative
F. physiotherapeutic

23. Show the 3 groups of diseases that lead to the prevention and exacerbation of secondary amyloidosis and the method of its treatment:
A. long-term special treatment for chronic infections (tuberculosis, brucellosis, wounds)*
B. in chronic purulent diseases (bronchoectatic disease, various abscesses, osteomyelitis) -
complex treatment with antibiotics, sanitization of purulent foci, surgical treatment if necessary *
C. in connective tissue systemic diseases - complex treatment using basic drugs and symptomatic treatment *
D. in acute infectious diseases - their quick treatment
E. in acute purulent diseases - antibacterial treatment
F. in deforming osteoarthrosis - complex treatment

24. Show 6 ways of influencing pathogenetic mechanisms to reduce amyloid synthesis:
A. daily consumption of raw liver (80-120 g)*
B. aminoquinoline preparations (delagil, resorcin, xingamine)*
C. unitiol*
D. colchicine*
E. immunostimulants*
F. dimethylsulfoxide*
G. daily consumption of raw meat (80-120 g)

H. glucocorticoids
I. immunosuppressants

25. Show 3 methods and amounts of protein replacement in amyloidosis:
A. 1.5 g/kg protein intake (90-120 g/milk)*
B. native and dry plasma (100-200 ml IV per day for 3-5 days)*
C. plasma albumin (100 ml IV per day for 3-5 days)*
D. 1 g/kg protein intake (50-90 g/milk)
E. hemotransfusion (NEW blood, erythrocyte mass)
F. casein-type protein hydrolysates

26. Show 5 types of renal amyloidosis according to etiological and pathogenetic signs:
A. genetic
B. primary
C. secondary D. old age
E. local tumor amyloidosis F. autoimmune
G. in babies H. in children I. in teenagers
27. Show 4 theories of amyloidosis pathogenesis:
A. dysproteinosis theory
B. immunological theory
C. the theory of local cell secretion
D. mutational theory
E. theory of dysglycogenesis
F. infectious theory
G. theory of mitochondrial secretion
H. theory of chromosomal aberrations

28. Show 4 clinical stages of secondary amyloidosis:
A. dysproteinemic (preclinical, preamyloidosis)
B. proteinuric (latent)
C. nephrotic
D. azotemic (uremic)
E. to the clinic
F. clinical
G. is on the rise H. terminal

29. Show 3 syndromes characteristic of genetic amyloidosis:
A. nephrotic

B. neuropathic
C. cardiopathic
D. hemorrhagic
E. angiotrophic
F. cardiac

30. Show 4 additional syndromes (in addition to the 3 main ones) observed in genetic amyloidosis:
A. Gastrointestinal damage
B. fever
C. anemia
D. ECHT increase
E. nephrotic
F. neuropathic
G. cardiopathic
H. hemorrhagic
I. hypothermic

31. In amyloidosis, indicate 3 zones where material can be obtained for biopsy:
A. mucosa and submucosa of the sigmoid colon
B. mucous and submucous layer of gums
C. skin with a subcutaneous base
D. mucosa and submucosa of the stomach
E. Mucous and submucosa of the duodenum
F. hair

32. List 3 specific tests that help distinguish secondary amyloidosis from other types:
A. detection of Bence-Jones protein in urine
B. thermoprecipitation test
C. Increased Ig G
D. daily protein loss
E. Increased Ig A
F. hypoproteinemia

33. Name the 4 main syndromes in primary amyloidosis:
A. enteropathy
B. cardiopathic
C. neuropathic

D. nephrotic
E. hemorrhagic
F. splenomegalic
G. cardiac
H. cholestatic

34. Name the 3 main groups of diseases that cause secondary amyloidosis:
A. chronic infectious diseases (often tuberculosis)
B. chronic purulent diseases (bronchiectasis, lung abscess, osteomyelitis, etc.)
C. connective tissue systemic diseases and rheumatoid arthritis.
D. acute infectious diseases
E. acute purulent diseases of lungs and bones
F. deforming osteoarthrosis and gout

35. List 6 laboratory changes characteristic of primary amyloidosis:
A. hypochromic anemia
B. leukocytosis
C. ECHT increase
D. hyperfibrinogenemia
E. cholesterolemia
F. hyper-beta-lipoproteinemia
G. erythremia
H. leukopenia
I. hyperproteinemia

36. Amyloid is composed of 3 components?
A. component that forms fibrils (F-component)
B. plasma component (R-component)
C. hematogenous inclusions (albumins, globulins, fibrin, SIK, etc.)
D. carbohydrate that forms fibrils
E. serum component (F-component)
F. hemoglobin supplements

37. In the treatment of amyloidosis, indicate the main 3 ways to be affected:
A. amyloidosis developed against the background of the main disease (secondary)
B. to mechanisms of pathogenesis
C. to the main clinical syndromes
D. etiological
E. palliative

F. physiotherapeutic

38. Show the 3 groups of diseases that lead to the prevention and exacerbation of secondary amyloidosis and the method of its treatment:
A. long-term special treatment for chronic infections (tuberculosis, brucellosis, wounds).
B. in chronic purulent diseases (bronchoectatic disease, various abscesses, osteomyelitis) -
complex treatment with antibiotics, sanitization of purulent foci, surgical treatment if necessary
C. in connective tissue systemic diseases - complex treatment using basic drugs and symptomatic treatment
D. in acute infectious diseases - their quick treatment
E. in acute purulent diseases - antibacterial treatment
F. in deforming osteoarthrosis - complex treatment

39. Show 6 ways of influencing pathogenetic mechanisms to reduce amyloid synthesis:
A. daily consumption of raw liver (80-120 g)
B. aminoquinoline preparations (delagil, resorcin, xingamine)
C. unitiol
D. colchicine
E. immunostimulants F. dimethylsulfoxide
G. daily consumption of raw meat (80-120 g) H. glucocorticoids
I. immunosuppressants

40. Show 3 methods and amounts of protein replacement in amyloidosis:
A. 1.5 g/kg protein consumption (90-120 g/milk)
B. native and dry plasma (100-200 ml IV per day for 3-5 days)
C. plasma albumin (100 ml IV per day for 3-5 days)
D. 1 g/kg protein intake (50-90 g/milk)
E. hemotransfusion (NEW blood, erythrocyte mass)
F. casein-type protein hydrolysates

41. Show 3 types of protein products that can be consumed in amyloidosis:
A. meat
B. eggs
C. liver
D. shade

E. cottage cheese F. milk
G. sour cream

42. List 4 clinical signs that help to diagnose amyloidosis:
A. presence of disease causing amyloidosis (clinical or anamnestic data)
B. emergence or exacerbation of proteinuria, or development of nephrotic syndrome
C. The presence of disease that does not cause amyloidosis, but the presence of proteinuria or nephrotic syndrome
D. stable severe heart failure, malabsorption syndrome, polyneuropathies (if other causes are not identified)
E. acute diseases of internal organs
F. high proteinuria
G. stagnant tumors
H. dysproteinemia

43. Show 3 signs that help distinguish proteinuria in amyloidosis from acute or chronic glomerulonephritis:
A. Relatively slow progression of kidney damage in amyloidosis
B. amyloidosis is not associated with acute respiratory diseases
C. permanent microhematuria in glomerulonephritis (20% of cases in amyloidosis)
D. relatively rapid progression of kidney damage in amyloidosis
E. Amyloidosis is associated with streptococcal infection Macrohematuria is observed in
F. amyloidosis

44. List the 5 main components that should be considered when making a clinical diagnosis of amyloidosis:
A. type of amyloidosis
B. stage of amyloidosis (proteinuric, nephrotic, terminal)
C. functional status of kidneys (presence, level of kidney failure)
D. primary disease (in secondary amyloidosis)
E. condition ofother organs (heart, liver, nervous system, etc. in idiopathic /primary/ amyloidosis)
F. process activity
G. passage
H. X-ray stage
I. loss of working capacity

45. Show the 3 groups of diseases that lead to the prevention and exacerbation of secondary amyloidosis and the method of its treatment:
A. long-term special treatment for chronic infections (tuberculosis, brucellosis, wounds).
B. in chronic purulent diseases (bronchoectatic disease, various abscesses, osteomyelitis) -complex treatment with antibiotics, sanitization of purulent foci, surgical treatment if necessary
C. in connective tissue systemic diseases - complex treatment using basic drugs and symptomatic treatment
D. in acute infectious diseases - their quick treatment
E. in acute purulent diseases - antibacterial treatment
F. in deforming osteoarthrosis - complex treatment

46. There may be etiological factors of acute pericarditis, indicate the wrong one:
A) viruses
B) bacteria
C) fungi
D) alcohol*
E) drugs

47. Pericardial friction noise in acute dry pericarditis:
A) 3-component*
B) 2-component
C) 1-component
D) Everything

Basic and additional educational literature and information sources List

Main literature:

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3. Gadaev A. G., Karimov M.Sh. Internal medicine propaedeutics. Textbook. Tashkent. Editor's Publishing House. 2012

Additional literature:

1. Harrison's Principles of Internal Medicine-19th Edition – 2015
2. Internal diseases, Gadaev A.G. T: Turon Zamin Ziyo, 2014, 2019
3. Internal diseases, Bobojanov S. T: New age generation, 2008
4. Internal diseases, Sharapov UF T: Ibn Sina, 2003

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6. Окороков А.Н. Диагностика болезней внутренних органов М., 2005г. 465 стр.
7. А.В. Гордиенко, Hospital therapy, Ст. Петерсбург.: СпетсЛит., 2016, 533 С. textbook.
8. Рябов С.И. Внутренние дисеасес Волуме 1. Саинт-Петерсбург.: СпетсЛит., 2015, 783 S. Textbook 9. Ryabov S.I. Vnutrennie disease Volume 2. St. Petersburg.: SpetsLit., 2015, 575 S. Textbook
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Internet sites

1. www.ziyonet.uz;
2. www.lex.uz;
3. www. cardiosite.ru;
4. www. cardiologia.ru;
5. www.bilim.uz;
6. www.gov.uz;