VOLGOGRAD STATE
MEDICAL UNIVERSITY
Department of Pathological Anatomy

Kidney disease. Glomerulonephritis. Nephrotic syndrome. Acute and chronic renal failure. Diseases of the urinary system. Diseases of the male reproductive system. Sexually transmitted or predominantly sexually transmitted infections. Diseases of the prostate gland: prostatitis, benign hyperplasia, cancer. Diseases of the testis. Tumors

Guidelines for students
III year of medical faculty
for practical exercises in pathological anatomy

Volgograd, 2020

Diseases of urinary system, male reproductive system.

1. The purpose of the lesson. To study the etiology, pathogenesis, morphological characteristics, complications and outcomes of diseases of the organs of urinary system, male reproductive system.

2. Requirements for the level of the student in the development of the discipline - pathological anatomy. The student must know:

1. Definition, etiology, classification, pathological anatomy of glomerulonephritis.
2. Definition, etiology, classification, pathological anatomy of nephrotic syndrome.
3. Definition, classification, pathological anatomy of diseases of acute and chronic renal failure.
4. Definition, classification, pathological anatomy of diseases of the urinary system.
5. Definition, etiology, classification, pathological anatomy of diseases of the male reproductive system.
6. Definition, classification, pathological anatomy of diseases of the prostate gland: prostatitis, benign hyperplasia, cancer.

3. Theoretical aspects.

Disorders of the urinary system.

Diseases of the male reproductive system.

There are two main groups of kidney diseases, or nephropathy, - glomerulopathy and tubulopathy, which can be either acquired or hereditary.

Glomerulopathy is a kidney disease with a primary and primary lesion of the glomerular apparatus, based on a violation of glomerular filtration. Acquired glomerulopathies include glomerulonephritis, idiopathic nephrotic syndrome, renal amyloidosis, diabetic and hepatic glomerulosclerosis, and hereditary nephritis with deafness (Alport syndrome), hereditary nephrotic syndrome, and forms of familial nephropathic amyloidosis.

Tubulopathies are diseases of the kidneys with the primary leading lesion of the tubules, primarily disturbances in the concentration, reabsorption and secretory functions of the tubules. Acquired tubulopathies are represented by necrotic nephrosis, which underlies acute renal failure, “myeloma kidney” and “gouty kidney”, and hereditary ones - by various forms of tubular fermentopathy.

A large group of kidney diseases are interstitial (interstitial) nephritis, pyelonephritis, kidney stone disease and nephrosclerosis, which often completes the course of many kidney diseases and underlies chronic renal failure. A special group consists of malformations of the kidneys, primarily polycystic, as well as kidney tumors.

GLOMERULOPATHY

GLOMERULONEPHRITIS

Glomerulonephritis is a disease of an infectious-allergic or unknown nature, which is based on a bilateral diffuse or focal non-purulent inflammation of the glomerular apparatus of the kidneys (glomerulitis) with characteristic renal and extrarenal symptoms. Renal symptoms: oliguria, proteinuria, hematuria, cylindruria. Extrarenal symptoms: arterial hypertension, left heart hypertrophy, dysproteinemia, edema, hyperazotemia and uremia.

Depending on the combination of symptoms, the clinic distinguishes between hematuric, nephrotic, (nephrotic syndrome), hypertensive and mixed forms of glomerulonephritis.

Classification of glomerulonephritis takes into account: 1) its nosological affiliation (primary - as an independent disease and secondary - as a manifestation of another disease; 2) etiology (established etiology - usually bacteria, viruses, protozoa and unknown etiologies); 3) pathogenesis (immunologically determined and not immunologically determined); 4) course (acute,

subacute, chronic); 5) morphology (topography, nature and prevalence of the inflammatory process).

Etiology of primary glomerulonephritis. 1) bacterial glomerulonufrit; 2) abacterial glomerulonephritis.

The main pathogens of bacterial glomerulonephritis: □-hemolytic steptococcus (its nephritogenic types), staphylococcus, pneumococcus, viruses, plasmodium malaria. Usually, bacterial glomerulonephritis develops after an infectious disease, as an expression of an allergic reaction of the body to an infectious agent. More often it is angina, scarlet fever, acute respiratory disease, less commonly - pneumonia, erysipelas, diphtheria, meningococcal infection, prolonged septic endocarditis, malaria, sepsis.

Abacterial glomerulonephritis can cause, for example, ethanol - alcoholic glomerulonephritis.

Pathogenesis - the sensitization of the body with a bacterial or non-bacterial antigen with localization of manifestations of hypersensitivity in the vascular glomeruli of the kidneys is of primary importance.

Pathological anatomy. According to the topography, intra- and extracapillary forms are distinguished, according to the nature of the inflammation - exudative, proliferative (productive) and mixed.

Intracapillary glomerulonephritis is a characteristic development of the pathological process in the vascular glomerulus. It may be exudative when the mesangium and capillary loops of the glomerulus are infiltrated by neutrophils. It can be proliferative (productive) - when proliferation of endothelial and especially mesangial cells is noted, the glomeruli increase in size and become "tabular".

Extracapillary glomerulonephritis - inflammation develops in the cavity of the glomerular capsule. It can be exudative - serous, fibrinous or hemorrhagic, and proliferative - characterized by the proliferation of glomerular capsule cells (nephrothelium and podocytes) with the formation of characteristic half moon.

According to the prevalence of the inflammatory process in the glomeruli, diffuse and focal glomerulonephritis is isolated.

Glomerulonephritis with a tubular, tubulo-interstitial or tubulo-interstitial-vascular component is isolated.

Acute glomerulonephritis - usually caused by streptococcus, pathogenesis associated with circulating immune complexes. At the onset of the disease: glomerular hyperemia, infiltration of mesangium and capillary loops with neutrophils. Later, proliferation of endothelial and especially mesangial cells appears, and the exudative reaction decreases. The exudative phase, the exudative-proliferative and proliferative phases are distinguished.

Sometimes: fibrinoid necrosis of the capillaries of the glomerulus and the arterioles with capillary thrombosis and neutrophil infiltration - necrotic glomerulonephritis.

Macro kidneys are enlarged, swollen, pyramids are dark red, bark is grayish-brown with small red specks (mottled kidney). Occasionally, the kidneys are not changed.

The process is reversible, but sometimes the changes persist for more than a year (acute protracted glomerulonephritis), can turn into a chronic one.

Subacute glomerulonephritis - develops in connection with damage to the glomeruli of the kidneys by circulating immune complexes, as well as antibodies. The course is fast (rapidly progressive gl-t): early renal failure occurs - malignant gl. This is usually extrakap.product.gl-t. "half moon" appears due to the proliferation of nephrothelium of glomeruli, podocytes and macrophages, "half moon" squeezes the glomerulus. Capillary loop - fibrinoid necrosis, fibrin thrombi in the lumen. Sclerosis, glomerular hyalinosis, tubular atrophy, renal stromal fibrosis.

Macro kidneys are enlarged, flabby, the cortical layer is wide, swollen, yellow-gray, dull, with red specks, well bordered by dark red medulla (large mottled kidney), or the cortical layer is

red and merges with the full-blooded pyramids (large carcid kidney)

Chronic glomerulonephritis - self-disease. It flows for a long time, latently or with relapses, ends with renal failure. The etiology is unclear; pathogenesis is more often associated with circulating immune complexes. It is represented by two types - mesangial and fibroplastic.

Mesangial hl-t - proliferation of mesangiocytes in response to deposits under the endothelium and in the mesangial immune complexes. Marked expansion of the mesangium of the vascular bundle of the glomeruli and the accumulation of the matrix in it. The processes of mesangiocytes are evicted on the periphery of the capillary loops, in a light microscope - thickening, bypass or splitting of the basement membrane of capillaries. Distinguish between mesangioproliferative and mesangiocapillary variants.

Mesangioproliferative hl-t - the proliferation of mesangiocytes and the expansion of mesangium without significant changes in the walls of the capillaries.

Mesangiocapillary hl - pronounced proliferation of mesangiocytes, diffuse thickening and cleavage of capillary membranes.

In the tubules - dystrophy and atrophy, in the system - cell infiltration and sclerosis.

Macro - the kidneys are dense, pale, with yellow spots with a cortical layer.

Fibroplastic gl-t - the collective form, sclerosis and hyalinosis of capillary loops and the formation of adhesions in the capsule cavity complete the changes characteristic of other morph.types of gl-to. There are focal and diffuse fibrpl.gl.-t. Tubules - dystrophy and atrophy, stroma - sclerosis, vessels - sclerosis.

Macro kidneys are somewhat reduced, with outlined small depressions on the surface, dense, gray-red.

Chr. Hl-t goes into a second wrinkling of the kidneys. The kidneys are reduced, dense, the surface is fine-grained. The fate of sclerosis and atrophy - depressions alternate with areas of hypertrophy. On the cuts, the fabric is dry, anemic, gray, the cortical layer is thin

Micro - atrophy of glomeruli and tubules, replacement of them with connective tissue - clomerulosclerosis and glomerulogialinosis, in some glomeruli - hypertrophy: capsules are thickened, droplets are sclerotic. Tubules - the lumen is expanded, the epithelium is flattened. Arterioles - sclerosis and hyalinosis.

Complications - acute renal failure, with chronic glaucoma - chronic renal failure with uremia, cardiovascular disease, weekly hemorrhage in the brain.

The outcome - acute hl-t - favorable, podo. and xp is unfavorable.

NEPHROTIC SYNDROME

It is characterized by high proteinuria, dysproteinemia, hypoproteinemia, hyperlipidemia (hypercholesterolemia) and edema.

Classification - primary (idiopathic) and secondary n.s.

Primary N.S. - Lipoid nephrosis, membranous nephropathy, focal segmental sclerosis.

Lipoid nephrosis - children and adults.

Minimal changes in the glomerular filter (electron microscopy) are characteristic - the loss of small processes by podocytes. The membrane is slightly thickened, a slight expansion of the mesangium. The tubules of the main sections of the nephron are enlarged, the epithelium is swollen, contains hyaline drops, vacuoles, neutral fats, cholesterol, fatty degeneration predominates. The tubule epithelium - dystrophy, necrobiosis, atrophy and desquamation, epithelial regeneration. In the lumen of the tubules - hyaline, granular and waxy cylinders. Stroma - edema, lipids, especially cholesterol, lapophages, lymphocytes.

Macro - the kidneys are sharply enlarged, flabby, the capsule takes off easily, the surface is yellow and smooth; the bark is wide, yellow-pale and pale gray, the pyramids are gray-red (large white kidneys).

In the treatment with steroid hormones, the course is favorable, but secondary wrinkling of the kidneys is possible.

Membranous nephropathy - chronic flow, nephr.

Diffuse thickening of the walls of the capillaries due to the neoplasm of the substance of the basement membrane by podocytes in response to deposits in the capillary wall of subepithelial immune complexes, in the absence or extremely weak proliferation of mesangiocytes. in a light microscope - membrane outgrowths in the direction of podocytes between the deposits of immune complexes - the so-called spines on the basement membrane. Channel - severe dystrophy of the epithelium.

Macro - kidneys are enlarged, pale pink or yellow, the surface is smooth.

The outcome is wrinkling of the kidneys and CRF.

Focal segmental glomerular sclerosis - primary and secondary (associated with lipoid nephrosis). In juxtamedullary glomeruli - sclerosis and hyalinosis of individual glomeruli. Detection of lipids both in hyaline masses and in mesangiocytes (turn into foam cells).

The outcome is CRF.

KIDNEY AMYLIDOSIS

One of the manifestations of general amyloidosis

There are: latent, proteinuric, nephrotic and uremic stages.

The latent stage is the amyloid in the pyramids, along the direct vessels and collecting tubes.

The proteinuric stage - amyloid, not only in the pyramids, but also in the glomeruli - small deposits in the mesangium and individual capillaries, in arterioles. Sclerosis and amyloidosis of the pyramids and the boundary layer are pronounced, nephron atrophy, tubules - epithelium - hyaline droplet or hydropic dystrophy, in the lumen of the tubules - cylinders. The kidneys are enlarged, dense, the surface is pale gray or yellow-gray, on the sections the cortical layer is wide, matte, the medulla is gray-pink, “greasy” in appearance (large greasy kidney)

The nephrotic stage - amyloid - in many capillaries of most glomeruli, in arterioles and arteries, along the own membrane of the tubules, but there is no pronounced sclerosis of the cortical substance, and sclerosis and amyloidosis are diffuse in the pyramids and the intermediate zone. The tubules are dilated, clogged with cylinders. In the tubule epithelium - a lot of lipids (cholesterol). The kidneys are large, dense, waxy - a large white amyloid kidney.

The uremic stage is an increase in amyloidosis and sclerosis, the death of most nephrons, their atrophy and sclerosis. The kidneys are of normal size or slightly reduced, very dense, with many cicatricial depressions on the surface (amyloid-wrinkled kidneys). Often - left ventricular hypertrophy (arterial hypertension).

Complications - infection, heart attacks, hemorrhages, heart failure. Arrester.

Death - from chronic renal failure and uremia, acute renal failure, infection.

TUBULOPATHIES

Acute renal failure (ARF)

ARF is a syndrome morphologically characterized by necrosis of the tubular epithelium and deep impairment of renal blood and lymph circulation.

Etiology - intoxication and infection.

Pathogenesis - associated with shock mechanisms.

Distinguish - the initial (shock), oligoanuric stage and the stage of restoration of diuresis.

Macro - kidneys - enlarged, swollen, edematous, capsule tense, easily removed. The cortical layer is wide, beld gray, sharply delimited from the dark red pyramids.

The initial stage is a sharp venous plethora of the intermediate zone and pyramids with focal ischemia of the cortical layer, interstitial edema, tubule epithelium - hyaline droplet, hydropic and fatty degeneration, in the gaps of the tubules - cylinders, sometimes myoglobin crystals.

Oligoanuric stage - necrotic changes in the tubules of the main departments, the changes are focal in nature and are accompanied by destruction of the basement membranes of the predominantly distal tubules - tubulorexis. Interstitium - edema, leukocytes, hemorrhages, in the lumen of the tubules - cylinders, pronounced venous stasis, against which - venous thrombosis.

The stage of restoration of diuresis - the glomeruli become full-blooded, edema and kidney

infiltration are reduced, The sites of tubular epithelium necrosis alternate with islet regenerates from light epithelial cells. If the channel membrane is preserved, the epithelium regenerates completely.

Complications: segmental or or total necrosis of the cortical substance of the kidneys.

The outcome is recovery from hemodialysis treatment, death from uremia.

Chronic renal failure (uremia)

Chronic renal failure is characterized by an increase in the concentration of nitrogenous wastes in the blood (azotemic uremia) and signs such as anemia, nausea, vomiting, gastrointestinal bleeding, and skin itching; polyuria and nocturia are often observed.

Clinical manifestations of chronic renal failure develop as a result of impaired normal renal function. The accumulation of nitrogenous toxins develop as a result of a decrease in blood filtration in the kidneys; anemia - as a result of a decrease in the production of erythropoietins by the kidneys, as well as due to hematuria and hemolysis; osteodystrophy often resembles bone damage with rickets (develops as a result of a violation of the metabolism of vitamin D in the affected kidneys, which leads to a decrease in calcium absorption in the intestine and is a cause of stimulation of the parathyroid glands). The process is also complicated by the retention of phosphates in the body. With kidney fibrosis, renin production increases, leading to the development of hypertension.

Pathological anatomy. At the autopsy of the deceased from uremia, the smell of urine is felt. Skin - gray-earthy color as a result of the accumulation of urochrome. Sometimes, especially on the face, it can be as if powdered with a whitish coating (chlorides, crystals of urea and uric acid). Sometimes hemorrhages and a rash as manifestations of hemorrhagic diathesis. Often - uremic laryngitis, tracheitis, pneumonia (usually fibrinous-necrotic or fibrinous-hemorrhagic). Also - pharyngitis, gastritis, enterocolitis of a catarrhal, fibrinous or fibrinous-hemorrhagic nature. In the liver - fatty degeneration. The spleen is enlarged, similar to septic. Very often - serous, serous-fibrinous or fibrinous pericarditis, uremic myocarditis, less often - warty endocarditis. Perhaps uremic pleurisy and peritonitis.

The brain with uremia is pale and edematous, sometimes with foci of softening and hemorrhage.

Pyelonephritis is an infectious disease in which the infection can get into the kidneys by hematogenous (descending) or urinogenous (ascending) route. The most common pathogens are bacteria. Urinary tract infections take second place after respiratory tract infections. For the development of pyelonephritis, vesicoureteral reflux is necessary.

In childhood, boys are more likely to get sick, because they often have various abnormalities in the structure of the urinary tract. From the puberty to middle years, women are more often sick (associated with more frequent injuries of the urethra and pregnancy). After 40 years, men are more often sick due to the development of various diseases of the prostate gland. In addition, predisposing factors are: instrumental examinations and manipulations (for example, catheterization and cystoscopy) and diabetes mellitus.

Acute pyelonephritis develops when infected with pyogenic microorganisms. Clinical manifestations: high fever, chills, and lower back pain. Dysuric manifestations are observed with infections of the lower parts of the urinary system. In the urine, mild proteinuria (up to 1 g / l), leukocytes, leukocyte (white) cylinders and bacteria. The diagnosis is confirmed by bacteriological examination (more than 100,000 microorganisms in 1 ml of urine are a diagnostic sign). Acute pyelonephritis is found at all ages, but most often after the onset of sexual activity and during pregnancy.

Etiology and pathogenesis. Ways of infection of the kidneys: 1) hematogenous (descending) path and 2) urinogenous (ascending) path. Hematogenous introduction of the infection is quite rare (with infectious endocarditis or bacteremia from other sources; pathogens - bacteria, fungi and viruses).

An ascending pathway for the spread of infection is the most common, especially in the

presence of reflux from the lower urinary system. The most common pathogens: gram-negative bacteria (Escherichia coli, Proteus and Enterobacter), which are normal inhabitants of the human intestine. If the disease occurs after catheterization, there may be Klebsiella, Proteus, Enterococcus faecalis, Pseudomonas aeruginosa, etc. In adult women, asymptomatic bacteriuria (5%) (usually Escherichia coli) is quite common, and during pregnancy this percentage increases to 20%.

Factors contributing to the development of acute pyelonephritis:

1. Short urethra in women;
2. Stasis of urine of any etiology.
3. Structural disorders of the urinary tract, predisposing to stasis of urine or leading to communication with infected places, for example, fistulas between the urinary tract and intestines, skin and vagina.
4. Bladder-ureter reflux.
5. Catheterization of the bladder.
6. Diabetes mellitus.

Pathological anatomy. Acute pyelonephritis can be either two-sided or one-sided.

Macro: The kidneys are enlarged, in the cortex - foci of suppuration (abscesses) with radial yellow stripes that suppress the medulla. With hematogenous pyelonephritis, small abscesses are located randomly, mainly in the upper, and with urinogenous infection, in the lower pole of the kidney. Inflammation of the cups and pelvis with accumulation of pus in the lumen of the pelvis.

Micro: polymorphonuclear leukocytes - in the lumen of the tubules, edema and inflammation of the interstitium. During healing, interstitial fibrosis and in inflammatory infiltrate, lymphocytes and plasmocytes predominate.

Complications of acute pyelonephritis.

1. Necrosis of the papillae of the kidney.
2. Pyonephrosis.
3. Paranephritis.
4. Perinephritis.

Chronic pyelonephritis is the cause of chronic renal failure in 15% of patients.

Views:

1) chronic obstructive pyelonephritis;
2) pyelonephritis associated with vesicoureteral reflux

Chronic obstructive pyelonephritis is quite common and is observed in all age groups. Obstruction: 1) mechanical (for example, with stones, with prostatic hyperplasia, tumors, congenital malformations, retroperitoneal fibrosis) or 2) due to paresis of the bladder wall (neuropathic bladder).

In 50% of patients, there was a history of acute pyelonephritis.

In almost all cases of chronic pyelonephritis - vesicoureteral reflux (either as a congenital anomaly, or due to obstruction of the underlying urinary tract in adulthood).

Pathological anatomy.

Macro: Kidney damage is usually asymmetric, characterized by varying degrees of wrinkling of the kidneys. Often - deformation of the pyelocaliceal system. Unlike chronic glomerulonephritis, asymmetric lesions and the presence of deep scars in the cortex are characteristic. In the area of wrinkling - deformation and expansion of the cups.

Micro: interstitial fibrosis with atrophy and expansion of the tubules, in which there are eosinophilic cylinders, which leads to the appearance of a peculiar histological picture resembling the structure of the thyroid gland, therefore such changes are called “thyroidization” of the kidneys (“thyroid kidney”). Foci of inflammation and fibrosis in the interstitium. Inflammatory infiltrate consists of lymphocytes and plasmocytes with a small admixture of neutrophils. Periglomerular sclerosis progresses with the development of global nephron sclerosis. There may be hypertrophy and enlargement of the surviving tubules. With immunofluorescence and electron microscopy, immune complexes in the glomeruli are not detected.

4. Lesson plan

1) To study the following macropreparations, to describe them according to the scheme for the description of macropreparations.

Macropreparations.

1. To study Subacute glomerulonephritis according to a macroscopic picture. Describe the appearance of the kidney, size, texture, color on the surface and in the section; pay attention to the color and presence of red specks in the cortical layer.

2. To study the Kidney cancer on a macroscopic picture. Describe the appearance of the kidney, size, texture, color on the surface and in section; pay attention to the color of the cortical layer, the intermediate zone and the pyramids. Pay attention to the structural features of the tumor, its color in section, size and shape.

3. To study Nephrosclerosis according to a macroscopic picture. Describe the appearance of the kidney, size, consistency, color on the surface and in the section, the state of the layers.

4. To study the Amyloid-lipoid nephrosis on a macroscopic picture. Describe the the appearance of the kidney, size, texture, color on the surface and in the section; pay attention to the color of the cortical and brain layers.

2) Examine the following micropreparations, sketch them, indicate and mark the pathological changes with arrows, using the atlas of micropreparations.

Micropreparations.

1. Subacute (extracapillary) glomerulonephritis. To study the characteristic changes in the glomerular capsule nephrothelium (to find a half moon), the state of the vascular glomerulus, the presence and nature of changes in the tubules.

2. To study Serous extracapillary glomerulonephritis. Indicate the presence and nature of exudate in the glomerular capsule cavity, the state of the vascular glomerulus, the presence and nature of changes in the tubules..

3. To study Kidney amyloidosis. Find areas of amyloid deposition, pay attention to the localization of deposits, the condition of the kidney tubules..

4. Secondarily whrinkled kidney (nephrosclerosis). Indicate sclerosis and hyalinosis of most glomeruli, the condition of the remaining glomeruli (with signs of hypertrophy), the presence and nature of changes in the tubules, arterioles, small and medium arteries, and stroma of the kidney.

3) Examine the following electron diffraction patterns using an atlas of micropreparations.

Electron micrographs.

1. Mesangioproliferative glomerulonephritis (glomerular filter). Slight thickening of the basement membrane with areas of fixation of electron-dense material (immune complexes). Hyperplasia and proliferation of the endothelium. The processes of podocytes are preserved and shortened.2. Mesangiocapillary glomerulonephritis. Mesangial cell proliferation. Mesangial interposition, deposits of immune complexes in the mesangium, a large number of lysosomes.

3. Membranous glomerulonephritis (glomerular filter). A sharp uneven thickening of the basement membrane (BM); podocyte hyperplasia (PD), destruction of their processes; deposits of deposits (D).

4. Lipoid nephrosis. Loss of small processes by podocytes, the basement membrane is not changed, fusion of podocytes with the membrane is noted.

4) Solve the following situational problems (case study) using the tutorial.

Situational tasks (case study).

Case 1

Patient K., 38 years old, over the past 2 months. There is a rapid and progressive decrease in renal function with the development of severe oliguria. The disease developed after severe streptococcal tonsillitis, there was an increase in proteinuria, dysproteinemia, hyperlipidemia, arterial hypertension. Performed a puncture biopsy of the kidneys. The microscopic picture of a biopsy: the formation of characteristic cellular figures, half moon, which obliterate the space between the capsule and capillary glomerulus, fibrin filaments between the layers of cells in the half moon, dystrophic and necrobiotic changes in the tubular epithelium.

Questions: 1) What kidney disease has occurred? 2) What is the morphological type of the described kidney disease (according to the microscopic picture)? 3) The prognosis of the described disease?

Case 2

Patient P., 9 years old, was admitted to the hospital with complaints of weakness, red color of urine, a sharp decrease in the amount of urine excreted per day. Ill 3 weeks after suffering streptococcal tonsillitis. When examined in a hospital: the amount of urine per day is 600 ml; urinalysis: red blood cells - 25-30 in the field of view, cylinders, protein; blood test: a decrease in the amount of total protein, an increase in urea, creatinine, residual nitrogen. Performed a puncture biopsy of the kidneys. Microscopically in a biopsy specimen: glomeruli are sharply enlarged due to proliferation and swelling of endothelial cells, neutrophils in the lumens of capillary loops; in the stroma - edema, dystrophy of the tubular epithelium, in the lumen of the tubules - erythrocyte cylinders.

Questions: 1) What kidney disease has occurred? 2) What is the morphological type of the described kidney disease (according to the microscopic picture)? 3) The prognosis of the described disease?

Case 3

Patient V., 5 years old, was admitted to the hospital for examination. In the urine - a significant amount of protein (more than 5 g / day); in the blood - hypoalbuminemia (plasma albumin level less than 3 g / 100 ml); swelling from the anamnesis of life: some time ago a preventive vaccination. Performed a puncture biopsy of the kidneys. Microscopically in a biopsy sample: with light microscopy, glomeruli are intact (histologically normal glomeruli), lipids in proximal tubule cells; with electron microscopy, there is a simplification of the architecture of epithelial cells with flattening and swelling of the legs of the processes (the so-called disappearance of the legs of the processes); with immunofluorescence microscopy, deposits of immunoglobulins or complement are not detected.

Questions: 1) What syndrome does the patient have? 2) What kidney disease caused this syndrome? 3) What other diseases can cause this syndrome? 4) The prognosis of the described disease?

Case 4

A 40-year-old patient suffering from urolithiasis was admitted to the hospital in an extremely serious condition and died of progressive intoxication. At autopsy: the kidneys are slightly enlarged, flabby, on the cuts - in the pelvis and cups - "coral" stones and pus, the tissue of the kidneys is saturated with pus; microscopically: multiple foci of purulent inflammation with the melting of renal tissue, tubular necrosis; the liver - large-drop fat and protein (granular) hepatocyte

dystrophy, myocardium - granular dystrophy of cardiomyocytes. In paranephric tissue - multiple cavities filled with pus.

Questions: 1) What kidney disease did the patient have? 2) What complication developed?

5) Answer the following questions of the current test control.

QUESTIONS OF THE CURRENT TEST CONTROL:

Select all correct answers.

1.With immunologically caused glomerulonephritis, glomerular damage involves:
a) mast cells
b) macrophages,
c) platelets
g) neutrophils,
e) mesangial cells.

Select all correct answers.

2. Hematuria is a characteristic clinical sign:
a) glomerulonephritis,
b) malakoplakii,
c) nephrolithiasis,
g) renal cell carcinoma,
d) papillomas of the bladder.

SITUATIONAL OBJECTIVE

In a 6-year-old boy, 2 weeks after an acute upper respiratory tract infection, oliguria, proteinuria, hematuria and generalized edema appeared. An examination of the kidney biopsy revealed glomerular hypercellularity as a result of proliferation of endothelial and mesangial cells and glomerular infiltration by neutrophils and macrophages.

Choose one correct answer

3. Conclusion:
a) acute glomerulonephritis,
b) lunar glomerulonephritis,
c) diffuse proliferative glomerulonephritis,
g) lipoid nephrosis,
e) membranous nephropathy.

Choose one correct answer

4. In a patient who had streptococcal tonsillitis, after 3 weeks there were swelling on the face in the morning, urine acquired the color of meat slops, headache was noted. Macroscopic view kidney:
a) primary shriveled,
b) "big whites",
c) "big motley",
d) secondary-wrinkled.

Select all correct answers.

5. Jade syndrome is:
a) oliguria,
b) arterial hypertension,

c) severe hypoproteinemia,
g) hematuria,
e) azotemia.

Choose one correct answer
6. Morphology of acute post-streptococcal glomerulonephritis:
a) loss of small processes of podocytes,
b) membranous transformation,
c) extracapillary productive glomerulonephritis,
d) intracapillary productive glomerulonephritis.
SITUATIONAL OBJECTIVE
A 23-year-old patient became acutely ill after hypothermia. There was an increase in blood pressure, hematuria and swelling on the face. Despite treatment, the phenomena of renal failure accrued. After 6 months, the patient died of uremia.

Choose one correct answer
7. The main disease:
a) subacute glomerulonephritis,
b) acute post-streptococcal glomerulonephritis,
c) renal amyloidosis,
g) IgA nephropathy,
e) renal cell carcinoma.

Choose one correct answer
8. The morphological equivalent of the disease:
a) tumor growth,
b) glomerular hypercellularity,
c) membranous transformation
g) deposition of masses of amyloid,
e) the formation of half moon in glomeruli.

Select all correct answers.
9. Morphological characteristics of extracapillary productive glomerulonephritis:
a) proliferation of nephrothelium and podocytes with the formation of a half moon,
b) nodules of Kimmelstyle-Wilson,
c) the formation of the half moon,
g) protein dystrophy of the tubular epithelium,
d) fibrin in the lumen of the glomerulus capsule,
e) proliferation of podocytes.

Establish compliance
10. Nephrotic syndrome: Disease:
1) primary, a) renal amyloidosis,
2) secondary. b) diabetic nephropathy,
c) membranous nephropathy,
g) focal segmental glomerular hyalinosis,
e) lupus nephritis,
e) lipoid nephrosis.

Choose one correct answer

11. A man with hepatitis B has developed nephrotic syndrome. In the study of kidney biopsy, a thickening of the glomerular basement membrane was found, which contained deposits of immunoglobulins. With electron microscopy, the deposits were located subepithelially. Conclusion:
a) acute glomerulonephritis,
b) chronic glomerulonephritis,
c) membrane proliferative glomerulonephritis, type I,
g) membrane proliferative glomerulonephritis, type II,
e) membranous nephropathy.

Choose one correct answer

12. In a 2-year-old boy with severe edema (anasarca), which developed shortly after an acute respiratory viral infection, significant albuminuria, hypoproteinemia, and hyperlipidemia were detected during laboratory tests. Changes disappeared with the use of corticosteroid therapy. Conclusion:
a) disease of minimal changes (lipoid nephrosis),
b) focal segmental glomerular hyalinosis,
c) membranous nephropathy,
g) acute glomerulonephritis,
e) rapidly progressive glomerulonephritis.

Choose one correct answer

13. A patient who uses drugs has developed proteinuria. In the study of kidney biopsy, obliteration of the capillary loops of some glomeruli was found. Conclusion:
a) amyloidosis,
b) lunar glomerulonephritis,
c) focal segmental glomerular hyalinosis,
g) disease of minimal changes (lipoid nephrosis),
e) nodular or diffuse diabetic glomerulosclerosis.

Select all correct answers.

14. Complications of chronic glomerulonephritis:
a) chronic renal failure,
b) hyperglycemic coma,
c) anemia,
g) cardiovascular failure,
e) cerebral hemorrhage.

Choose one correct answer

15. During a postmortem examination in a man of 60 years old, suffering from chronic glomerulonephritis for 12 years, small, dense, fine-grained kidneys, fibrinous inflammation of the serous and mucous membranes, degenerative changes in the myocardium and liver, and pulmonary edema were found. Cause of death:
a) myocardial dystrophy,
b) diphtheria colitis,
c) fibrinous pneumonia,
g) fibrinous pericarditis,
e) uremia.

Choose one correct answer
16. Sign of acute renal failure after cardiogenic shock:
a) oliguria,
b) polyuria,
c) hematuria,
g) proteinuria,
e) glucosuria.

Choose one correct answer
17. The patient underwent gastrectomy for cancer; the operation was accompanied by massive bleeding. In the postoperative period, despite the replenishment of blood loss, persistent anuria persisted, on the 5th day the patient died. Morphological expression of persistent anuria:
a) lunar glomerulonephritis,
b) necrotic nephrosis,
c) intracapillary productive glomerulonephritis,
g) membranous transformation,
e) loss of small processes of podocytes.

Select all correct answers.
18. In a patient who suffered during an earthquake and was removed from under the rubble of a building with a sagging leg, persistent anuria was observed on the 2nd day. Pathogenetic factors of the crash syndrome:
a) hypertensive crisis,
b) stress
c) shock
g) myoglobinuria,
d) albuminuria.

Choose one correct answer
19. The main condition for the complete regeneration of the epithelium with necrotic nephrosis:
a) the conservation of single glomeruli,
b) the integrity of the tubular basement membrane,
c) pronounced lymphoid-plasmacytic infiltration,
d) fibroblasts in the stroma,
e) moderate stromal edema.

Choose one correct answer
20. Allergies to drugs in the kidneys are most often found in the form of:
a) lunar glomerulonephritis,
b) focal necrotizing glomerulonephritis,
c) acute tubulointerstitial nephritis,
g) acute pyelonephritis,
e) acute papillary necrosis.

Choose one correct answer
21. Etiology of acute pyelonephritis: a) immune complexes,
b) viruses
c) gram-negative bacteria
g) gram-positive bacteria
d) mycobacteria.

Select all correct answers.

22. In a 25-year-old pregnant woman, her body temperature increased to 38 ° C, dysuria and pain in the lumbar region on the right appeared. Numerous neutrophils and bacteria in the urine. Acute pyelonephritis was diagnosed. Risk factors:

a) age
b) gender
c) pregnancy
d) errors in the diet,
e) fever and pyuria.

Choose one correct answer

23. Kidneys with asymmetric corticomedullary scars, severe deformity of the pyelocaliceal system, stromal sclerosis with the appearance of sharply expanded atrophic tubules, filled with eosinophilic masses and periglomerular sclerosis form in the outcome:

a) chronic glomerulonephritis,
b) chronic pyelonephritis,
c) amyloidosis,
g) hypertension
e) diabetic nephropathy.

Select all correct answers.

24. Predisposing to the development of urolithiasis conditions:

a) sickle cell nephropathy,
b) hyperparathyroidism,
c) gout
g) amyloid nephropathy,
e) hyperoxaluria.

Choose one correct answer

25. Most renal cell carcinomas grow from the epithelium:

a) glomeruli,
b) tubules,
c) cups
d) pelvis.

6. List of recommended literature:

Basic literature:

1. “Basic pathology” Vinay Kumar, Ramzi S. Cotran, Stanley L. Robbins, 1997.

Additional literature:

1. “Pathology. Quick Review and MCQs” Harsh Mohan, 2004.
2. “Textbook of Pathology ” Harsh Mohan, 2002.
3. “General and Systemic Pathology” Joseph Hunter, 2002.
4. “General and Systematic Pathology ” Ed. J.C.E. Underwood – Edinburgh: Churchill Livingstone, 1996 (2th).
5. “Histology for Pathologist” Ed. S.S.Sternberg – Philadelphia: Lippincott Raven Publ, 1997 (2th).
6. “Histopathology. A Color Atlas and Textbook” Damjanov I., McCue P.A. – Baltimore, Philadelphia, London, Paris etc.: Williams and Wilkins, A Waverly Co., 1996.
7. “Muir’s Textbook of Pathology” Eds. R.N.M. MacSween, K. Whaley London: ELBS, 1994 (14th).
8. “Pathology” Eds. Rubin, J.L. Farber – Philadelphia: Lippincott Raven Publ, 1998 (3th).
9. “Pathology Illustrated” Govan A.D.T., Macfarlane P.S., Callander R. – Edinburgh: Churchill Livingstone, 1995 (4th).
10. “Robbins Pathologic Basic of Disease” Eds. R.S.Cotran, V.Kumar, T.Collins – Philadelphia, London, Toronto, Montreal, Sydney, Tokyo: W.B.Saunders Co., 1998 (6th).
11. “Wheater’s Basic Histopathology. A Color Atlas and Text” Burkitt H.G., Stevens A.J.S.L., Young B. – Edinburgh: Churchill Livingstone, 1996 (3th).
12. “Color Atlas of Anatomical Pathology” Cooke R.A., Steward B. – Edinburgh: Churchill Livingstone, 1995 (10th).
13. “General Pathology ” Walter J.B., Talbot I.C. – Edinburgh: Churchill Livingstone, 1996 (7th).
14. “Concise Pathology” Parakrama Chandrasoma, Glive R. Taylor.
15. “Pathology” Virginia A. LiVolsi, Maria J. Merino, John S. J. Brooks, Scott H. Saul, John E. Tomaszewski, 1994.
16. “Short lectures on pathology” Zagoroulko A., 2002
17. “Robbins pathologic basis of diseases” Cotran R., Kumar V., Collins T.
18. “General pathology” Dr. Fatma Hafez, 1979.
19. “Anderson’s Pathology ” Damjanov I., Linder J. – St. Louis: Mosby Inc., 1995 (10th).

https://www.volgmed.ru/ru/depts/list/69/
https://volgmu-pat-anat.3dn.ru/
https://webpath.med.utah.edu/