1. Nephrolytiasis

5. Stone of kidney

On macropreparations №1 and №5 violation of structure of parenchyma of kidney is presented. It is caused by the presence of stone in kidney, by violation of outflow urine which predetermines the origin of atrophy, sclerotic changes in tissue of kidney.

Urolithiasis (nephrolytiasis) is illness with chronic clinical course, seen in calices, pelvises of kidneys, ureters one or both kidneys the stone of different size, structure and chemical composition appears (phosphates, urates, oxalates and other). Among general factors, which further the development of urolithiasis, important factor is inherited and acquired violations of mineral exchange (calcium, phosphoric, oxaloacetic) and PH colloid condition (development of acidosis), character of feed (advantage is in the meal of carbohydrates and animal albumens), mineral composition of drinking-water (endemic nephrolytiasis), and also deficit of vitamins (avitaminosis A). Inflammatory processes in urinary tracts and urinary stasis are important. These factors are cause the increase of concentration of salts in urine, changes of pH of colloid ballance of urine, formation of colloid (protein) base of the stone. Main reasons of development of nephrolytiasis are: trophic and agile violations of function of calicles, pelvises and ureters (atony of pelvises and ureters, violation of circulation of blood). Morphological changes at nephrolytiasis are various and depend on localization of stone, their sizes, presence of infection, and remoteness of process.

2. Hydronephrosis

On macropreparation is seen the stone of kidney pelvis, which violates the outflow of urine, resulting in pyeloectasia, and in future develops to hydronephrosis with atrophy of kidney parenchyma are presented. On the preparation the kidney is changed into the thin-membrane sack, which is filled by urine. A stone which obturates the ureter causes expansion of not only pelvis but also cavity of ureter higher obturation - hydroureteronephrosis. Thus, there is inflammation of membrane of ureter - uretritis, which ends with stricture; bedsore appears with the perforation of ureter.

32. Hydrocalycosis
In kidney we see that a stone which is situated in calicle, violation of outflow of urine from it cause dilation not only of this cup – hydrocalycosis, but also parenchyma of kidney is atrophied.

Additional infection sharply influences on morphological changes at nephrolytiasis – calculous hydronephrosis (hydroureteronephrosis) becomes pyonephrosis (pyoureteronephrosis). An infection causes pyelitis, pyelonephritis, apostematous nephrite, and also suppuring melting of parenchyma of kidney. Inflammation often spreads on the paranephron and later develops to chronic paranephritis. In such cases kidney becomes bricked up in the thick capsule, built from granulation, fatty and connecting tissue ("testaceous" paranephritis), and sometimes fully changed by scleroused fatty cellulose (fatty changing of kidney).

Complication of urolithiasis is pyelonephritis. Most dangerous are pyonephrosis and suppurous melting of kidney, that in a number of cases is completed by sepsis; rarely there is acute kidney insufficiency. Chronic kidney insufficiency develops at the protracted clinical course of disease, atrophy, fibrous or fatty changing of kidneys.

Death of patients with nephrolytiasis comes from uremia or festering complications.

22. Large greasy kidney

At the proteinuritic stage an amyloid appears not only in pyramids but also in glomerules as an insignificant deposits in mesangium and separate capillary loops, and also in the membrane of arteriole. The sclerosis and amyloidosis of pyramids and boundary layers are considerable, that further an exception and atrophy in deeply located nephrons, reductions of ways of juxteglomelular blood stream and lymph flow in the medullar substance of kidneys. Epithelium of tubules of main parts are in a condition of hyalinedropped and to hydropic dystrophy; cylinders appear in tubules. Kidneys are megascopic, dense, with a pale grey or yellow grey surface. On the section the cortex layer is wide, mat, the medullar substance is grey-red, has a «greasy» kind (large greasy kidney).

The most frequent complications of amyloidosis of kidneys are some infectious diseases (pneumonia, erysipelas, parotitis) which arise up as a result of sharp decline of resistance of organism based on immune insufficiency and violations of methabolism. Complications are possible in connection with arising nephrogenic arterial hypertension (cardiac insufficiency, hemorrhages, and heart attacks). The bilateral thrombosis of the venous system of kidneys is rarely possible; in any stage of acute kidney insufficiency threatens the patient. Reason of such complication is the surplus accumulation in blood of products of albuminous disintegration, reduction of kidney circulation of blood on shock decline of arterial pressure, vascular disorders in connection with the thrombosis of
kidney veins and intercurrent disease. Death of patients with the amyloidosis of kidneys comes as a result of chronic kidney insufficiency and uremia in the last stage of disease.


Nephropathic amyloidosis often enough meets at AA-amyloidosis –secondary, with complicates arthritis, tuberculosis, bronchoectatic illness, and inherited, that arises up at the periodic illness. It goes to show that the amyloidosis of kidneys - it is more frequent in the second illness.

In clinical course of amyloidosis of kidneys distinguish latent, proteinuritic, nephrotic and the nitrogenemetic (uremia) stages. In every stage the morphological changes of kidneys are different and represent the proper dynamics process. In the latent stage external appearance of kidneys is without substantial changes, except of pyramids, in which where found the sclerosis and amyloidosis of straight vessels and collective tubes. In glomerules there are a bulge and two-contour capillary membranes with aneurismatic expansion of lumen of capillaries. There are albuminous granules in the cytoplasm of epithelium of tubules and their lumens. In the intermediate area and pyramids stroma is saturated with the protein of plasma.

26. Grainy dystrophy of kidney

External appearance of organ at this dystrophy: a kidney is megascopic, filling out, of pale kind.

In kidneys at microscopic research there is an accumulation of large corns of protein of bright rose color found in nephrocytes. Thus there is destruction of chondriosome, endoplasmatic net, brush framing. This type of dystrophy of nephrocytes is often met at a nephrotic syndrome and is represented by reabsorbtion insufficiency of convoluted tubules to albumens. This syndrome is one of manifestations of many diseases of kidneys in which the glomerular filter is initially struck (at amyloidosis of kidneys, paraproteinemitic nephropathia and other).

3. Cyanotic induration of kidneys

The origin of it is related to the chronic general venous plethora. Kidney on venous plethora is increased, cyanotic as a result of the increased content of the regenerated hemoglobin. It is dense as a result of violation of lymphcirculation and swollen, and later – leading to excrescence of connecting tissue.
10. Shock kidney.

External appearance of kidney: white grey cortex and stagnant dark- red color of pyramid. Microscopically necrobiotic and necrotic changes are marked in the epithelium of tubules.

Shock is the clinical condition, which is caused by diminishing of the effective cardiac output, violation of autoregulation of the microcirculation system and is characterized by the generalized diminishing of blood supply of tissues, which contributes to the destructive changes of internal organs.

Acute kidney insufficiency is a syndrome which is peculiar to necrosis of epithelium of tubules and deep violations of blood- and lymphocirculation. Acute kidney insufficiency equates with necrotic nephrosis (necronephrosis).

34. Nephrosclerosis

Kidneys are dense with uneven surface due to the presence of considerable scars; there is structural alteration of kidneys

In patho- and morphogenesis two phases are selected. In the first - the sclerosis of kidneys is conditioned by the features of patho- and morphogenesis of the basic disease. In the second - pathomorphogenetic and the clinic-morphological features of nosology are smoothed out, and; conducting become as the syndrome of chronic kidney insufficiency. In the first phase nephrosclerosis engulfs a period of block forming of kidney blood stream on one of structural levels of kidney (arteriolar, glomerular, interstitial), it is the component of morphological manifestations of basic disease.

30. Primary-wrinkled kidney

33. A kidney during hypertensive illness

At hypertensive illness and symptomatic high blood pressure arteriolosclerotic or primary wrinkling kidneys are developed as a result of vascular changes (initially wrinkled kidneys)

Kidneys are dense with a small uneven surface due to the presence of the developed scars; there is structural alteration of kidneys.


13. Big uneven kidney

Kidneys are dense with big uneven surface due to the presence of the developed scars; there is structural alteration of kidneys. Reasons of nephrosclerosis are various. At atherosclerosis atherosclerotic nephrosclerosis (nephrocirrhosis) and wrinkling of kidneys arise up not only initially at hypertensive illness, but also secondary as a result of inflammatory processes in glomerules, in tubules and stroma is the secondary wrinkling of kidneys or the secondary - kidneys are wrinkled. The secondary wrinkling of kidneys is more frequent is complication of chronic glomerulonephritis (secondary nephrotic wrinkling of kidneys), rarer - pyelonephritis (pyelonephrotic wrinkling of kidneys), amyloid nephrosis (kidneys are wrinkled at amyloid nephrosis), urolithiasis, tuberculosis of kidneys, diabetic glomerulosclerosis, heart attacks of kidneys and other. The consequence of nephrosclerosis is development of chronic kidney insufficiency of any etiology.

14. Secondary wrinkled kidney

A kidney is dense with a hilly surface due to the presence of the developed scars; there is structural alteration of kidneys. Chronic kidney insufficiency is a syndrome, morphological basis of which is nephrosclerosis (kidneys are wrinkled), and the most clinical manifestation is uremia. The origin of uremia is explained by deposition in the organism of nitrous slags (urea, urinary acid, kreatinin, indikan), acidosis and deep violation of electrolyte balance. These changes are in protein and electrolyte methabolism, and also in the pH condition cause autointoxication and deep violation of cellular metabolism. A section of dead with uremia is smelled like urine. A reaction with xanthine allows to find out the urea in all organs, especially in lungs, stomach, spleen. Volatile ammoniac substances with strong hydrochloric acid form the pair of chloride ammonium like a cloud. This reaction is tested on section of dead for diagnostics of uremia.

A skin becomes grey as a result of accumulation of urochrome; sometimes, especially on face, it becomes like powdered (chlorides, crystals of urea and urinary acid), that it can be conditioned by hypersecretion of sweat-glands. Often enough hemorrhages and rashes are manifestations of hemorrhagic diathesis appear on skin. There is uraemic laryngitis, tracheitis, gastritis, enteritis and pneumonia, which after the type of exudate belong to fibrinous-necrotic or fibrinous-hemorhragic; is typical edema of uremia in lungs.
There is fatty dystrophy of liver. Quite often on uremia found serous, serous-fibrinous or fibrinous pericarditis, uremic myocarditis; rarer- warty endocarditis. Development of uremic pleuritis and peritonitis is possible. A cerebrum with uremia is pale, filled out with the cells of softening influence and hemorrhages. A spleen is megascopic, reminds septic. Sclerosis and atrophy of kidneys reach extreme degree (mass of kidneys is in a sum 15-20g), appears only at careful research.

18. 19. Apostematous pyelonephritis

On acute pyelonephritis is leukocytic infiltration of stroma and kidneys’ calicles, focuses of necrosis of mucus membrane, picture of fibrinous pyelitis. Miliary abscesses and hemorrhages develop quite often. Tubules are in the dystrophic condition; there are cylinders of cast-off epithelium and leucocytes in their formation. The process has focus or diffuse character.

Kidneys are megascopic, fillings out, overblooded. The cavities of pelvises are extended, filled with turbid urine or by a pus; a mucus membrane is turbid, with hemorrhages. On the cut of kidney - grey-yellow areas are surrounded by overblooded area and hemorrhages, there are shallow abscesses.

20. Infarcts of kidneys

In kidney a white infract appears with hemorrhagic crown- in the area of pale grey color of three-cornered form is expressly marked off. Reason of this is related to violation of blood supply in the renal artery.

21. Chronic pyelonephritis

Surface of kidney of large humped, on cut cicatrix tissues which is alternated from relatively unchanged parenchima of kidney; pelvises are extended, membranes of them are thickened, white. The changes of renal tissue on chronic pyelonephritis often have focus character: the cells of interstitial inflammation, atrophy and sclerosis are surrounded by unchanged renal tissue in which it is possible to find the signs of regeneration hypertrophy. Such ability of process stipulates the characteristic type of kidneys at a chronic pyelonephritis. The consequence of chronic pyelonephritis is pyelonephritic wrinkled kidney. Thus found even cicatrix wrinkling, formation of dense
joints between kidney tissue and capsule, sclerosis of pelvises and cellulose of pelvis, asymmetry process in both kidneys. Although these signs are relative, but allow to distinguish pyelonephrotic nephrosclerosis from nephrosclerosis and nephrocirrhosis of other etiology.

28. Pyonephrosis

On Acute pyelonephritis progress of festering process causes connecting of abscesses and formation of carbuncle of kidney, connection of festering cavity with the pelvis (pyonephrosis), passing of process to the fibrous capsule and paranephron. Sometimes necrosis of papillae of pyramids (papillonecrosis) develops as a result of the toxic "influencing of bacteria in the conditions of urinary stasis, most patients with diabetes melitus are suffering. Sometimes pyelonephritis becomes the source of sepsis. At limitation of festering process, when scarring is, the chronic abscesses of kidneys appear. At a chronic pyelonephritis, especially one-sided, the origin of nephrogenic arterial hypertension is possible and arteriolosclerosis in the second (to unchanged) kidney. The bilateral pielonefrotichne wrinkling of kidneys ends with chronic kidney insufficiency.

8. A metastasis of cancer into kidney

15. 24. Adenoma of kidney

9.36. Cancer of urinary bladder

In urinary bladder is a tumour with exophytic growth, without clear borders, infiltrates surrounding tissues. After a histological structure more frequent in the urinary bladder there is transition- cellular cancer.

42. Hypernephroid cancer of kidney

On preparation tumour of yellow- grey color without clear borders, infiltrates surrounding tissues are presented.

4. Cancer of kidney
On preparation tumor of grey color without clear borders, infiltrates surrounding tissues are presented.

17. Adenocarcinoma of kidneys

On preparation tumor of grey color without clear borders, infiltrates surrounding tissues are presented.

43. Cancer of pelvis of kidney

On preparation tumor of yellow- of grey color without clear borders, infiltrates surrounding tissues are presented. After histostructure planocellular cancer is more frequent.

35. Fibroma of kidney

Fibroma is a tumor which develops from connecting (fibrous) tissue. It has the appearance of knot of different sizes. At microscopic research a tumor is built from the differentiated connecting tissue; the wisps of fibres and vessels are located in different directions.

7. Cystic disease of infant type

29. Small-cystic kidney

Cystic disease of kidneys of children is inherited as autosomal-recession type; cystic disease of kidneys of adults -as autosomal-dominant. Development of cystic disease of kidneys is caused by violation of embriogenesis in the first weeks, that is accompanied with formation of glomerular, tubular and excretory cysts. Glomerular cyst does not have connection with kidney tubules, that causes early development of kidney insufficiency. Tubular cysts which appear from widening tubules, and excretory, that appear from collapsible tubes, slowly increased in connection from impediment of their emptying and here arrive at considerable sizes. Cysts squeeze kidney parenchyma, and there are atrophy, sclerotic and inflammatory processes in it. The membrane of cysts is teared sometimes, that is favourable in support of inflammatory process in parenchyma of
kidneys. It should be noted that manifestations of cystic disease appear more, more malignantly course has the disease; lingering illness passes without symptoms

23. Large cystic kidney

39. Cysts in kidneys

16. Cyst of kidney

Cystic disease of kidneys is the inherited disease of kidneys with bilateral cystic disease of part relatively developed parenchyma- tubules and collapsible tubes. External appearance of kidneys on cystic disease looks like bunch of grapes. Tissue of kidneys consists of great number of cysts of different size and form, filled by serousy fluid, colloid semi-fluid masses of chocolate color. The membrane of cysts is covered by cube epithelium; sometimes in it found wrinkled vascular glomerule. Renal tissue between cysts is atrophied. Often cystic disease connects with cystic disease of liver, ovaries, lungs and pancreas.

Complications of cystic disease can be pyelonephritis, suppuration of cysts, rarely in the membrane of cysts there are malignant tumours (cancer). Consequences are unfavorable. Patients die from progressive kidney insufficiency and axotemic uremia.

40. Large mottled kidney

37. Hemorrhagic glomerulonephritis

Macroscopically kidney on acute glomerulonephritis is megascopic, languid, the layer of cortex is extended, overblooded, in it and under a capsule it can be visible red (mottle kidney).

Caused by β-hemolitic streptococcus of group A types 12, 4 and 1 (poststreptococcal, bacterial glomerulonephritis). Pathogeneticaly this immunologically conditioned disease which has an imunocomplex mechanism of defeat. Duration of disease is 1,5-12 months

At morphological research in the glomerules of kidneys acute plethora of capillaries is determined, infiltration as reaction neutrophilic leucocytes on immune complexes, located on BMC.
27. Subacute glomerulonephritis

Macroscopically kidneys at subacute glomerulonephritis are megascopic, languid, the layer of cortex is wide, fawn color with red, pyramids are sharply overblooded, red color (large mottle kidney), sometimes a cortex layer is acutely overblooded and in color merges with red pyramids; such kidney is named a large red kidney.

Subacute glomerulonephritis usually ends with development of acute or chronic kidney insufficiency, the correction of which is provided with either chronic hemodialysis, or transplantation of kidneys.

25. Chronic glomerulonephritis

This disease is characterized by long course- more than 12 months, develops latently or recidives, has different clinical forms. Etiology of it is unknown, the number of authors considers that 60 % researches caused by passing of acute glomerulonephritis to chronic. The basic mechanism of origin of chronic glomerulonephritis is immunocomplex and rarer antibody.

Histologically chronic glomerulonephritis is presented by two types: mesangial and fibroplastic glomerulonephritis. Chronic glomerulonephritis ends, as a rule, with secondary wrinkling of kidneys (the secondary wrinkled kidneys)

On macropreparation the kidney is diminished in a size, dense, a surface is fine-grained, cortex and medullar layers are refined, greyish color. On histological research in hollow areas found the sclerosis of glomerul and tubules, atrophy of nephrocytes with expansion or collapse of space of tubules. In the areas of thrusting out glomerules are hypertrophied, part of vascular loops is sclerosed. Epithelium of tubules in a condition of albuminous dystrophy, a space is extended. Membranes of large arteries are sclerosed, is possible elastofibrosis. In stroma there are limphohistiocytic infiltrates.

Chronic glomerulonephritis is accompanied with periphery changes. The hypertrophy of heart develops as a result of the increased arterial pressure, in arteries are the phenomena of elastofibrosis, atherosclerosis. Medullar, cardiac and kidney arteries are most often injured. It sometimes creates large difficulties in differential diagnostics of chronic glomerulonephritis and hypertensive illness.

Chronic glomerulonephritis is often complicated by acute or chronic kidney insufficiency, sometimes there is a hemorrhage in a cerebrum. Acute cardio-vascular
insufficiency is possible. All of these complications can be reasons of death of patient. More frequent all the patients in the final stage of kidney insufficiency die from axotemic uremia. The unique methods of treatment in this case of chronic glomerulonephritis is hemodialysis or transplantation of kidney.