



MORPHOLOGICAL CHANGES IN KIDNEY INTERSTITIAL TISSUE IN GESTATIONAL PYELONEPHRITIS

Mahkamov Nasirjon Juraevich

Doctor of Medical Sciences, Associate Professor, Andijan State Medical Institute (Uzbekistan)

Tajiboev Temurbek Topvoldi ugli

independent researcher at the Andijan State Medical Institute
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ABSTRACT

At the 24-28th week of pregnancy, developed morphofunctional edema and obstruction in the kidneys may occur, especially compression of the urinary tract leaving the kidney, as well as the addition of infectious factors in various ways; the occurrence of gestational pyelonephritis is determined. With gestational pyelonephritis, the interstitial tissue of the kidney continues with the formation of an acute inflammatory infiltrate as a result of simultaneous attenuation and the addition of an infectious factor.

Relevance of the problem. With pyelonephritis, the infection can enter the kidney through the urinary tract, less often through the hematogenous or lymphogenous route. In 80% of cases, pyelonephritis is caused by E. coli, which normally lives in the intestines and can enter the urinary tract if hygiene is poor. Despite the fact that foreign researchers have studied various regional changes in gestational pyelonephritis, this pathology still occurs. According to Golushko, 52% of pregnant women had gram-negative bacterial infections in vaginal smears. According to J. Delzell in 2017, in pregnant women at a period of 20-26 weeks of pregnancy, a sharp dilation of the urethra was found 1.7 times compared to the norm, dilation of the calyces and calyces of the kidneys.

As a result of this process, urodynamic disturbances and the development of mild forms of gestational pyelonephritis occur in an average of 12% of women. According to Nikiforovsky N.K., Nikiforovskaya E.N., Russia, 2002 study, at a period of 32-36 weeks, dilation of the calyces and calyces of the kidneys by 2.72 times compared to the norm was found in 21 out of 28 cases of the UTT study. It has been established that in the last period of the 3rd and 3rd trimesters of pregnancy, urodynamics are disrupted and the outflow of urine slows down. As a result, the development of gestational pyelonephritis was indicated, but no information was provided on the morphological aspects and immunohistochemical changes in the results obtained, namely the development of purulent pyelonephritis was determined in 12-18% of women who gave birth in the period 3-9 days after birth. This determines the exact period in which treatment tactics will be started.

Material and research methods. The kidney tissue of women who died from pyelonephritis and gestosis developed during pregnancy at the Center for Pathological Anatomy of the Republic. Clinical history. Statistical analysis. Morphological method: hematoxylin-eosin staining and analysis of the results.

Aim of the research. study of pathomorphological and immunohistochemical aspects of gestational pyelonephritis during pregnancy.

Results and discussion. Gestational pyelonephritis continues to affect the peritubular interstitial tissue, in addition to the calyces and calyces of the kidneys. In most cases, foci of acute lymphocytic infiltration are concentrated around the distal part of the collecting ureters in the apical branch of the pyramids. As a result, the outflow of urine is disrupted, the urinary tract becomes inflamed, the functioning of the immune system is disrupted, hormone metabolism is disrupted, if diabetes is present in the body, infection in the urinary tract develops quickly, the MALT structures located in this area and around the mucous membrane decrease sharply and these cells destroy interstitial tissues, and a diffuse arrangement occurs in the form of interstitial nephritis. This leads to an increase in the inflammatory infiltrate, spreading to the cortex and core and progressing to pyelonephritis, and clinically and morphologically acute renal failure or nephrotic syndrome occurs. It is at this point that the clinical signs of preeclampsia and eclampsia are measured, and direct protein filtrates appear in large quantities in the urine. The infectious process in the urinary tract and kidneys spreads, starting from the renal globules, excretory ducts, calyx and calyx due to the dampness of urine. This means that the kidney is enlarged due to damp urine (see Figures 1-2).

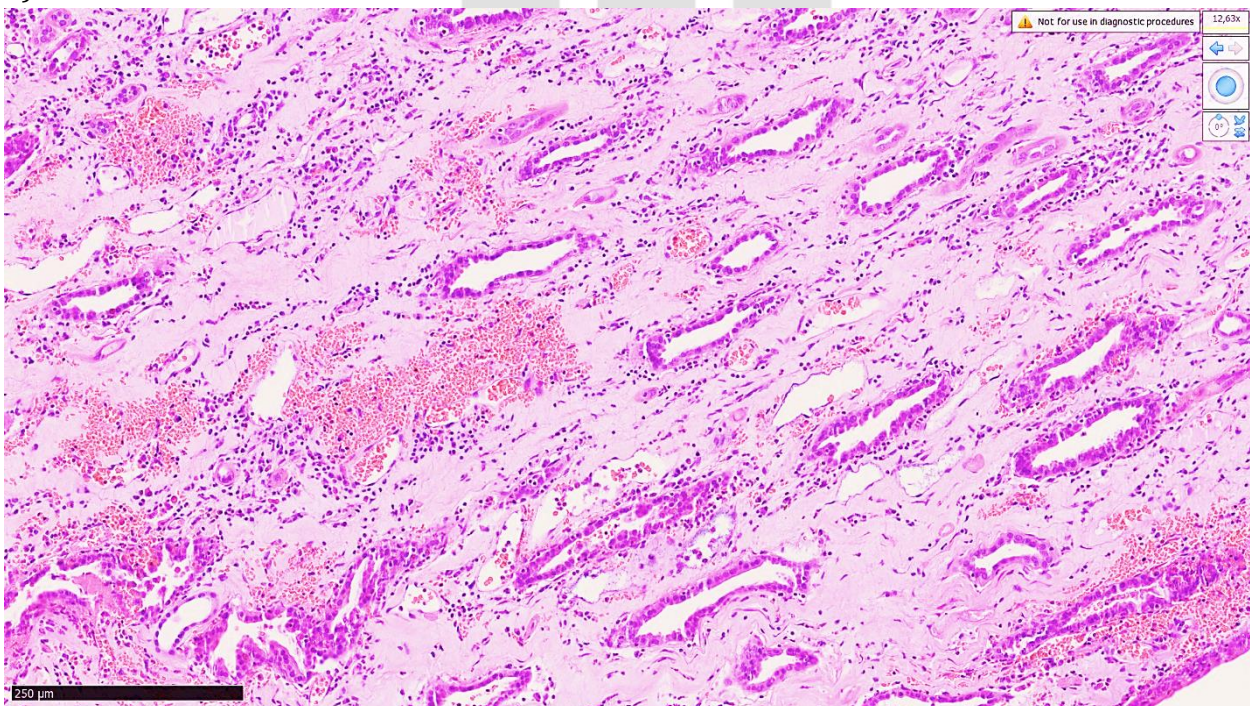


Figure 1. Kidney tissue obtained at autopsy from a pregnant woman at 28 weeks. General view of the terminal tubules of the renal medulla. Most often, peritubular inflammatory infiltrates are detected. Massive expansion of peritubular vessels and foci of diapedetic hemorrhages are revealed. G.E. paint Size 4x10.

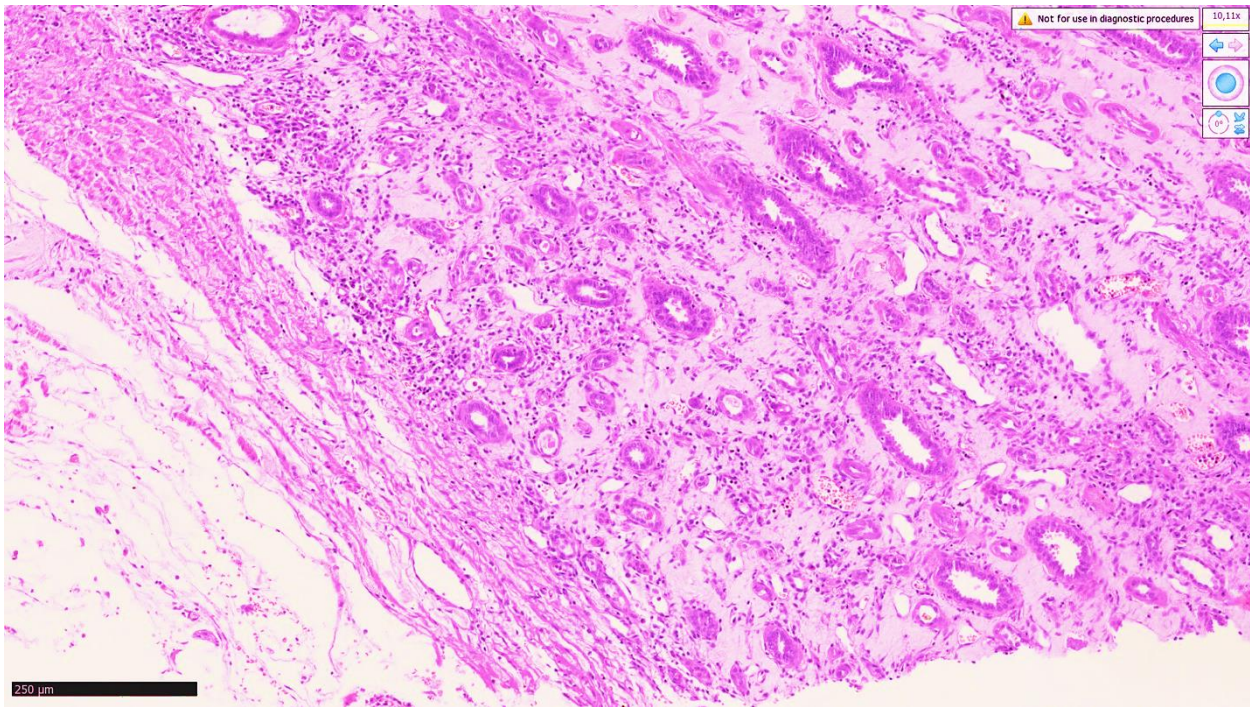


Figure 2. Kidney tissue obtained at autopsy from a pregnant woman at 24 weeks. General view of the terminal tubules of the renal medulla. Diffuse peritubular inflammatory infiltrates are detected. The tubules vary in size and appear as obstructed lesions. G.E. paint Size 10x10.

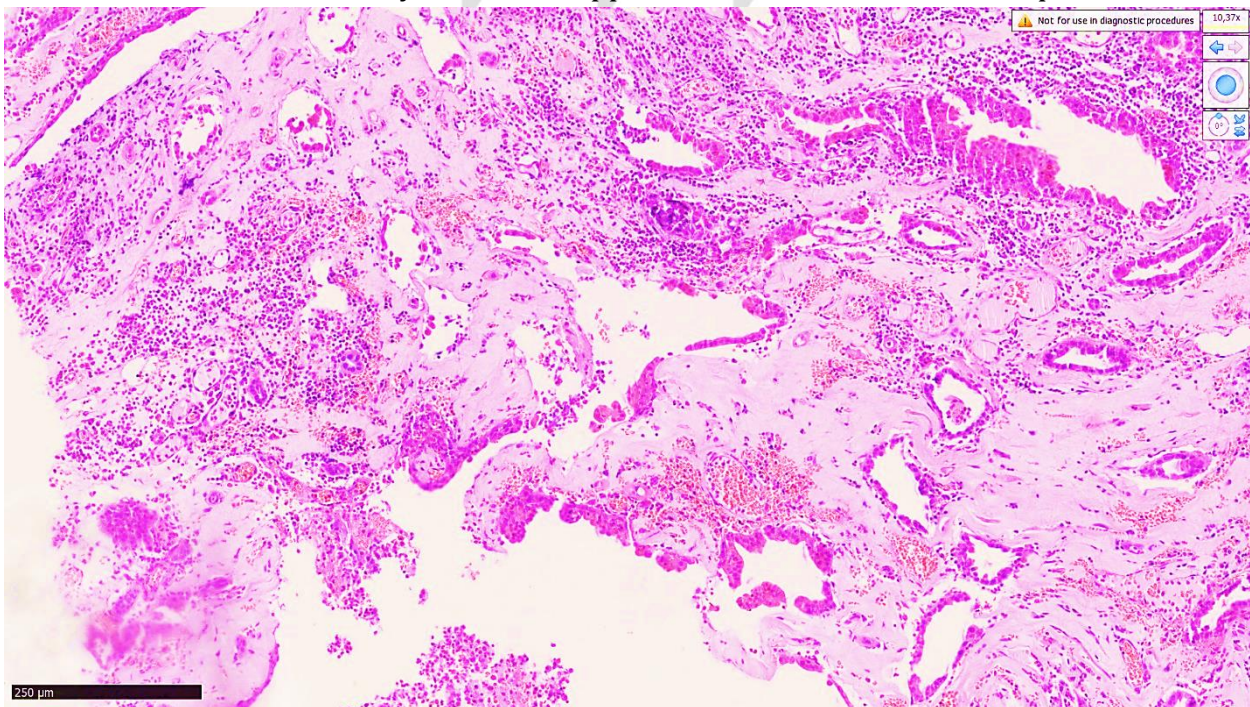


Figure 3. Kidney tissue obtained at autopsy from a pregnant woman at 26 weeks. General view of the terminal tubules of the renal medulla. Diffuse peritubular inflammatory infiltrates are detected. The tubules vary in size and appear as obstructed lesions. A large number of protein deposits were found on the surface of the kidney calyces and the mucous membranes of the kidneys. G.E. paint Size 20x10.

Morphologically - damage to interstitial tissue and mucous membranes, damage to the

epithelium covering the surface of the mucous membrane, in the form of cracks of varying degrees, and in some places - the formation of hypercellular focal landscapes. In the submucosal layer, changes are detected in the form of foci of lymphocytic infiltration, interstitial edema and hyperemia of small-caliber vessels. This caused tissue swelling in the form of compressive crush injury and the accumulation of a massive inflammatory infiltrate in the interstitial tissue around the small ureters (see Figure 3).

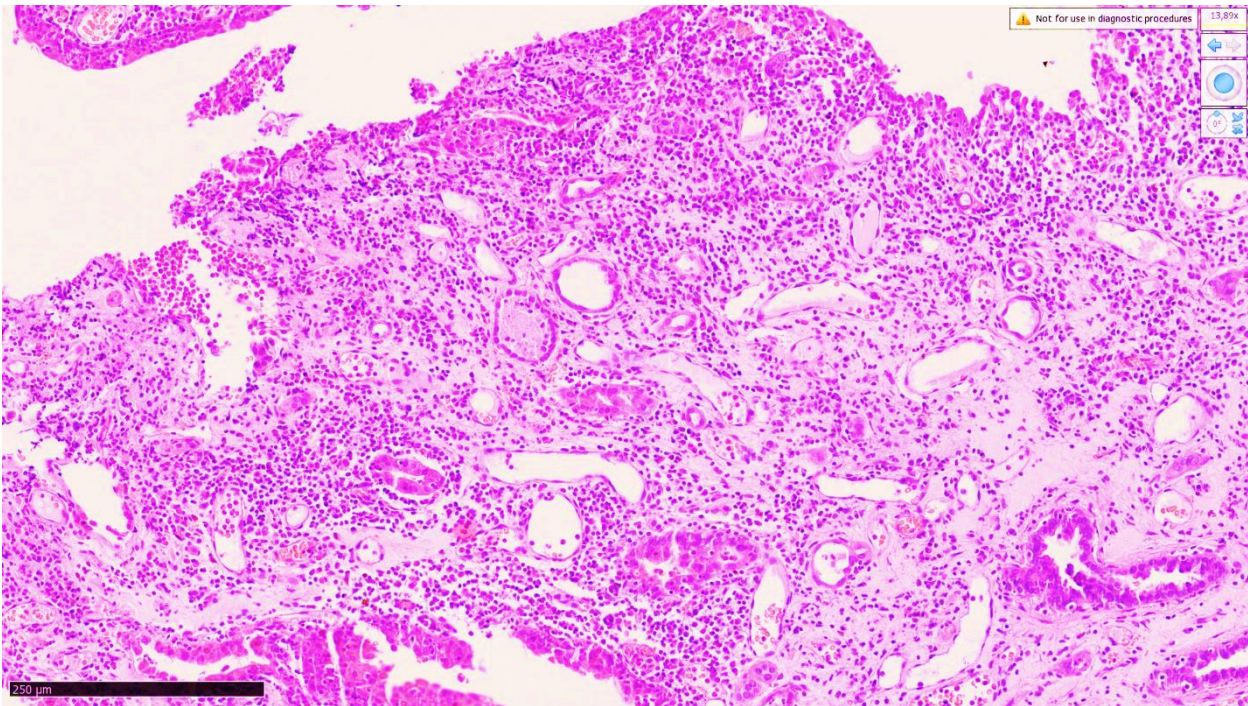


Figure 4. Renal cyst taken from autopsy of a pregnant woman at 24 weeks. The mucous layer consists of multirow single-layer epithelium. Foci of lymphocytic infiltration in the submucosa. The blood vessels are dilated, perivascular edema is detected. G.E. paint Size 20x10.

During pregnancy, under the influence of urine humidification, suppression of the immune system and other factors, the MALT structures under the mucous membrane show a small number of lymphocytes, the presence of fibroblasts and histiocytes from the majority of mesenchymal cells, as well as the presence of homogeneous pink protein deposits. This is one of the changes characteristic of gestational pyelonephritis during pregnancy, and in many cases it continues along with the kidney diseases characteristic of preeclampsia and eclampsia. Neutrophilic infiltration and interstitial edema developing along the perimeter of the submucosal vessels indicate the addition of a bacterial infection to the process (see Fig. 4-5).

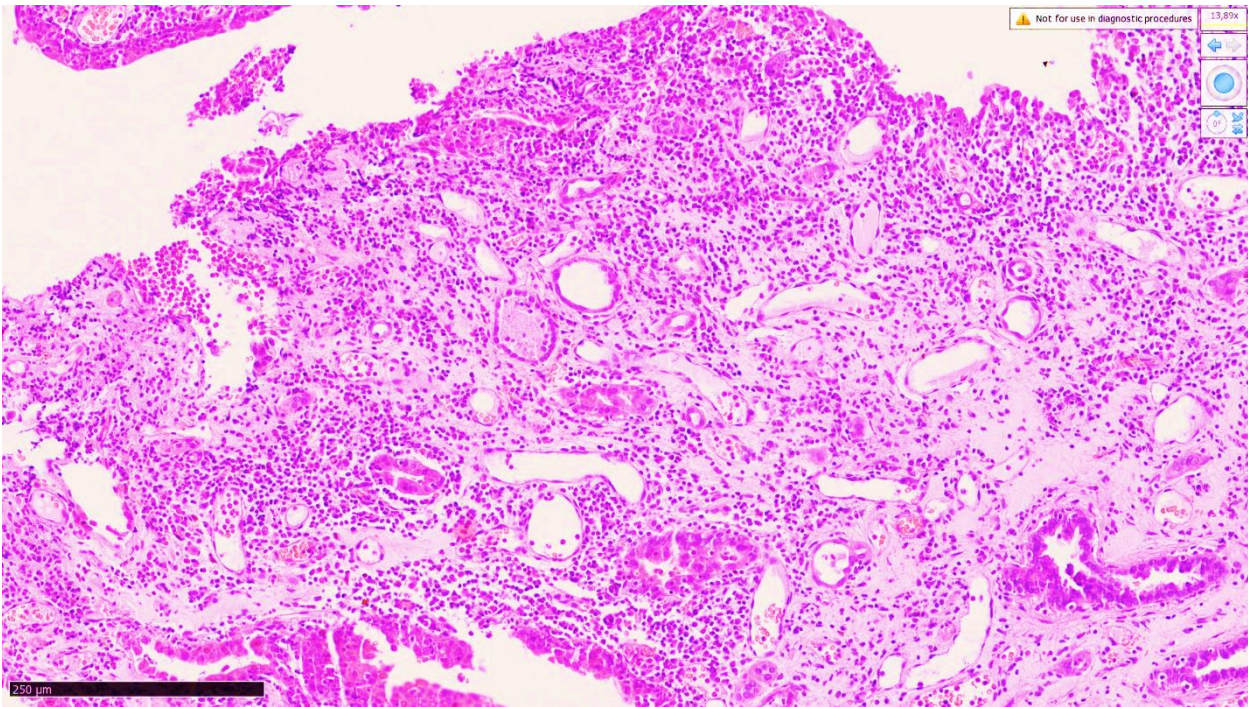


Figure 5. Kidney tissue obtained at autopsy from a pregnant woman at 26 weeks. General view of the terminal tubules of the renal medulla. Most often, peritubular inflammatory infiltrates are detected. Massive expansion of peritubular vessels and foci of diapedetic hemorrhages are revealed. G.E. paint Size 4x10.

As a result, massive dystrophic and necrobiotic changes are observed on the surface of the calyces and calyces up to the collecting duct area, especially in the epithelium of the distal tubules, homogeneous protein structures in the peritubular areas, the presence of mesh protein structures and dark-colored conglomerates in the collecting ducts, clinical-morphological proteinuria, discharge protein from the kidneys in the urine indicates an increase in urine density and an increase in salt deposits. This, in turn, indicates that with gestational pyelonephritis, the composition of diluted urine is rich in organic and inorganic substances that have changed in quantity and quality. As a result, starting from the kidney tissue, it causes dampness of the venous and lymphatic vessels in the urinary tract tissue. When this process begins to develop, due to the development of compensatory processes, pyelo-renal reflux occurs, the calyces and pelvis expand. In parallel with this, dystrophic changes occur in the surrounding tissues and cells due to disruption of the compensatory process in the lymphatic capillaries of the kidneys and urinary tract (see Fig. 5).

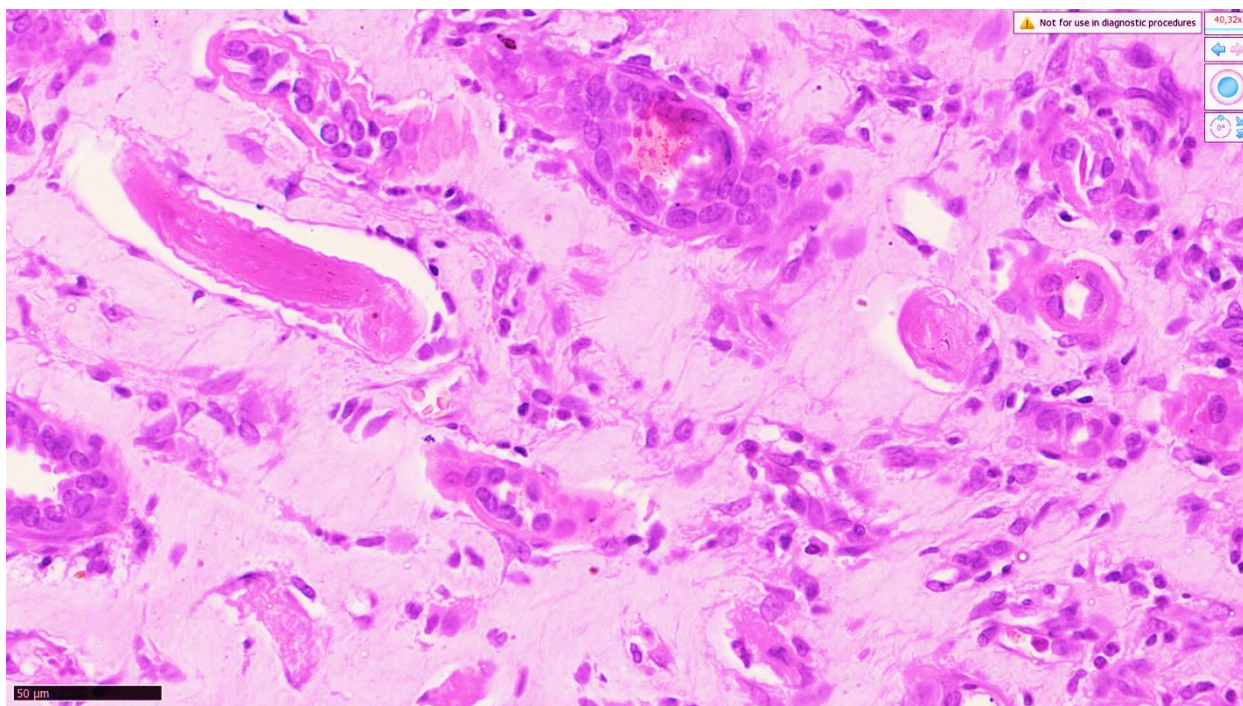


Figure 6. Kidney tissue obtained at autopsy from a pregnant woman at 26 weeks. General view of the terminal tubules of the renal medulla. Fibrinoid thickenings and obstructed tubules are found along the perimeter of the distal collecting ducts. The stroma is homogeneous and this is also considered plasmatic. G.E. paint Size 40x10.

Entry of urine into interstitial tissue leads to edema, tissue hypoxia, and histamine production. Acute hyperemia of the vessels in this area (venous vessels) causes changes in the form of plasmatic swelling. As a result, protein-rich tissue fluid dilates the lymphatic vessels, increasing the flow of lymphatic fluid and increasing the load on the lymph nodes. Disruption of local lymphatic circulation causes dynamic and resorptive insufficiency, morphologically varicose dilation of lymphatic vessels, the relative absence of their valves, sudden expansion and deformation of lymphatic vessels, expansion of peripheral and medullary sinusoids in the lymph nodes. The above changes lead to diffuse narrowing and compression of the tubules due to an increase in the volume of renal tissue and interstitial edema. Clinically and morphologically, these changes manifest themselves in the form of nephrotic syndrome (see Fig. 6).

Conclusion, during pregnancy, there is a morphofunctional blockade of plasmatic interstitial spaces and the flow of urine from the kidney to the bladder, accumulation of urine in the kidney and parallel drying of all perinephric lymph nodes, while there is a loss of the drainage function of most local blood and lymphatic vessels. This confirms that gestational pyelonephritis occurred with a sharp dilation of the vessels of the mucous membrane of the cup and pelvis and swelling of the mucous membrane. As a result, the kidneys become diffusely hypofunctional, and urodynamics are impaired due to closure, narrowing, and plasma staining of the distal collecting ducts. This ultimately manifests itself as acute renal failure.

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