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Date: 19th July-2026

"Ensuring the integration of science and education on the basis of innovative technologies.". Collection of scientific papers on materials of the international scientific-practical conference 19.07.2026,Pub. "ICP", Helsinki, Finland, 60 p.

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UDC 616.61-007.63-092:611.61:616-076.4

PATHOMORPHOLOGICAL ASSESSMENT OF MORPHOLOGICAL CHANGES IN THE RENAL MICROCIRCULATORY BED IN EXPERIMENTAL HYDRONEPHROSIS

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Background. Hydronephrosis develops as a consequence of impaired urinary outflow, resulting in renal parenchymal ischemia, microcirculatory disturbances, and progressive dystrophic and sclerotic changes. Identification of morphological alterations in the renal vasculature and interstitial tissue is essential for understanding the pathogenesis of the disease, improving early diagnosis, and optimizing therapeutic strategies.

Objective. To perform a pathomorphological assessment of morphological changes occurring in the renal microcirculatory bed in an experimental model of hydronephrosis.

Materials and Methods. The study was carried out on adult male albino laboratory rats weighing 200–250 g. The animals were divided into control, hydronephrosis, and recovery groups. Experimental hydronephrosis was induced by partial ureteral ligation. Examinations were performed on days 7, 14, and 30 after surgery. Kidney tissue samples were stained with hematoxylin and eosin (H&E), periodic acid–Schiff (PAS), and Van Gieson's stain, followed by microscopic and morphometric analyses. Statistical analysis was performed using Student's *t*-test.

Results. Experimental hydronephrosis was associated with progressive pathological changes in the renal microcirculatory bed. Thickening of the arteriolar walls, vacuolar degeneration of endothelial cells, thickening of the basement membrane, and signs of hyalinosis were observed. A reduction in capillary diameter indicated worsening tissue hypoxia and ischemia. The interstitial tissue demonstrated lymphocytic infiltration, fibroblast proliferation, and increased collagen deposition, leading to the development of interstitial fibrosis. Morphometric analysis revealed a significant increase in arteriolar wall thickness, a decrease in capillary diameter, and a several-fold enlargement of the interstitial fibrotic area. These findings confirmed progressive impairment of nephron blood supply and the development of nephrosclerosis.

Conclusion. Morphological alterations in the renal microcirculatory system represent one of the key pathogenetic mechanisms of experimental hydronephrosis. Ischemic, dystrophic, and sclerotic changes involving the arterioles, capillaries, and interstitial tissue contribute to the progressive decline of nephron function. Morphological and morphometric assessment enables the early detection of these alterations and provides valuable insights into the pathogenetic mechanisms of hydronephrosis, thereby supporting future studies aimed at preventing the progression of nephrosclerosis.

Keywords: hydronephrosis, kidney, microcirculation, ischemia, interstitial fibrosis, nephrosclerosis, morphology, morphometry.