INTERNATIONAL MULTIDISCIPLINARY JOURNAL FOR RESEARCH & DEVELOPMENT
SJIF 2019: 5.222 2020: 5.552 2021: 5.637 2022:5.479 2023:6.563 2024: 7,805
eISSN :2394-6334 https://www.ijmrd.in/index.php/imjrd Volume 12, issue 03 (2025)

PATHOLOGICAL ANATOMY OF KIDNEY DISEASES, INCLUDING ACUTE AND CHRONIC RENAL FAILURE

Rozimova Etibor Bakhtiyarovna

Assistant of the Department of Anatomy,
Urgench Branch of Tashkent Medical Academy.
Email: etibor8484@gmail.com

Annamuratova Mukhayyo Saporboevna
Assistant of the Department of Military Therapy,
Hematology, and Diagnostics,
Urgench Branch of Tashkent Medical Academy.
Email: muhayyoannamuratova27@gmail.com

Masharifov Xurshidbek Shomurod ugli
2nd-year student of the Faculty of General Medicine
of the Tashkent Medical Academy Urgench Branch
E-mail: khurshidbekmasharifov5@gmail.com

Introduction. Kidney diseases remain a significant concern in modern medicine due to their impact on homeostasis and overall organ function. Recent pathological studies have demonstrated distinct morphological changes in renal tissues associated with both acute and chronic renal failure. By analyzing histopathological samples and clinical data, researchers have identified specific structural alterations that correlate with disease progression and severity. Understanding these changes is essential for improving diagnostic accuracy and developing effective therapeutic approaches.

Acute Renal Failure. Acute renal failure (ARF) is characterized by a sudden decline in kidney function, often associated with ischemia, nephrotoxic damage, or urinary obstruction. Histological examinations of kidney biopsy samples from patients with ARF reveal significant alterations in tubular structures. Tubular epithelial cells often exhibit vacuolization, loss of brush borders, and necrotic changes. In cases of ischemic ARF, studies indicate widespread coagulative necrosis of renal tubules, particularly in the proximal convoluted tubules, along with interstitial edema and inflammatory cell infiltration. In nephrotoxic ARF, toxic substances induce segmental necrosis and cellular detachment, leading to the formation of casts in the tubules. Furthermore, vascular changes such as endothelial swelling and fibrin deposition in glomerular capillaries suggest an impaired microcirculation, which exacerbates tubular damage and delays recovery. Experimental models of ARF confirm that early intervention in cases of ischemic injury can reduce the extent of necrosis, supporting the hypothesis that restoration of blood flow is critical for renal tissue survival.

Chronic Renal Failure. Unlike ARF, chronic renal failure (CRF) develops over an extended period, leading to irreversible structural remodeling of kidney tissue. Histopathological analyses of patients with end-stage renal disease consistently show extensive glomerulosclerosis, interstitial fibrosis, and tubular atrophy. Glomeruli undergo obliteration due to excessive accumulation of extracellular matrix proteins, resulting in thickened and hyalinized capillary loops. In addition, the progressive destruction of nephron structures is accompanied by inflammatory infiltration and fibroblast activation, leading to the replacement of functional parenchyma with fibrotic tissue. A significant

observation in CRF pathology is the association between vascular damage and nephron loss. Studies have identified arteriolosclerosis, characterized by narrowed lumina and thickened vessel walls, as a key factor contributing to chronic hypoxia and progressive nephron dysfunction. Moreover, tubulointerstitial fibrosis appears to play a central role in CRF progression, as demonstrated by increased collagen deposition and myofibroblast proliferation in affected tissues. Longitudinal studies suggest that the extent of fibrosis correlates with declining glomerular filtration rates, reinforcing the importance of early therapeutic interventions aimed at reducing fibrotic changes.

Conclusion. The pathological anatomy of kidney diseases provides crucial insights into the mechanisms underlying renal failure. Findings from histopathological studies highlight the distinct morphological features associated with acute and chronic renal failure, emphasizing the importance of early diagnosis and intervention. While ARF presents with acute tubular necrosis and vascular dysfunction, CRF is marked by progressive fibrosis and glomerulosclerosis, leading to irreversible renal impairment. Future research focusing on targeted antifibrotic therapies and regenerative medicine may offer new strategies for mitigating kidney damage and improving patient outcomes.

References:

1. Brenner B. M., Rector F. C. The Kidney
10th ed. –Philadelphia: Elsevier, 2015. –2736 p.
2. Feehally J., Floege J., Tonelli M., Johnson R. J. Comprehensive Clinical Nephrology. 6th ed. — Philadelphia: Elsevier, 2018. — 1320 p.
3. Levey A. S., Coresh J., Tighiouart H. et al. GFR estimation: from physiology to public health // American Journal of Kidney Diseases. –2020. –Vol. 75, No. 1. –P. 17-26.
4. National Kidney Foundation. Chronic Kidney Disease [Electronic resource]. — Access mode: https://www.kidney.org, free. –Accessed: 18.03.2025.
5. Himmelfarb J. Oxidative Stress in Chronic Kidney Disease: A Dissertation Submitted for the Degree of Doctor of Philosophy. –University of Washington, 2015. –192 p.