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MORPHOLOGICAL FEATURES OF THE JUXTAGLOMERULAR APPARATUS IN EXPERIMENTAL HYDRONEPHROSIS

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Background. Hydronephrosis develops as a result of urinary tract obstruction and leads to profound morphofunctional alterations in the renal parenchyma. Along with the structural components of the nephron, the juxtaglomerular apparatus (JGA) is also affected. As a key component of the renin–angiotensin system, the JGA plays a crucial role in regulating renal hemodynamics. Therefore, investigating the morphological changes of the juxtaglomerular apparatus in hydronephrosis contributes to a better understanding of the disease pathogenesis and may improve opportunities for its early diagnosis.

Objective. To evaluate the morphological alterations of the renal juxtaglomerular apparatus in an experimental model of hydronephrosis and to determine their pathogenetic significance.

Materials and Methods. The study was conducted 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. Morphological evaluation was performed on days 7, 14, and 30 after surgery. Kidney tissue specimens were stained with hematoxylin and eosin (H&E), periodic acid–Schiff (PAS), and Van Gieson's stain. Morphometric analysis was carried out using ImageJ software, and statistical analysis was performed with standard statistical software packages.

Results. Progressive ischemic, dystrophic, and sclerotic changes were observed in the renal parenchyma during the development of experimental hydronephrosis. The diameter of the glomeruli gradually decreased, while Bowman's space became enlarged. The proximal tubular epithelium exhibited vacuolar degeneration, desquamation, and atrophic changes. Juxtaglomerular cells demonstrated a reduction in the number of secretory granules, nuclear hyperchromasia, and pyknosis, whereas dystrophic changes were identified in the macula densa cells. Morphometric analysis revealed a statistically significant decrease in the number of juxtaglomerular cells and the intensity of renin granules, accompanied by progressive interstitial fibrosis and thickening of the arteriolar walls. These alterations were closely associated with impaired renal microcirculation and the development of nephrosclerosis.

Conclusion. Morphological and morphometric alterations of the juxtaglomerular apparatus in experimental hydronephrosis represent important structural indicators of declining renal function. Destructive changes in the JGA contribute to reduced activity of the renin–angiotensin system, impaired renal microcirculation, and the progression of nephrosclerosis. The findings provide a scientific basis for the early morphological

diagnosis of hydronephrosis, assessment of disease progression, and the development of pathogenetically targeted therapeutic strategies.

Keywords: hydronephrosis, kidney, juxtaglomerular apparatus, morphology, morphometry, nephrosclerosis, ischemia, renin–angiotensin system.