

SEGMENTAL PATHOLOGICAL ALTERATIONS AND REMODELING DYNAMICS OF THE URETERAL WALL IN EXPERIMENTAL URETEROHYDRONEPHROSIS

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Background: Chronic upper urinary tract obstruction triggers structural remodeling of the ureter. Understanding the localized, segment-specific morphofunctional responses along the ureteral tract under chronic obstructive conditions is critical for predicting tissue damage severity, assessing surgical reversibility, and optimizing reconstructive interventions.

Materials and Methods: Experimental ureterohydronephrosis was established in 48 adult Wistar rats (24 male, 24 female) via partial ligation of the upper third of the right ureter using M.V. Novikova's procedure under general anesthesia. Tissue specimens from the abdominal, pelvic, and intramural segments were collected at 3 and 6 months post-surgery. Histological (H&E, Masson's trichrome), immunohistochemical (α -SMA, Collagen I/III), and morphometric assessments were performed to quantify wall thickness, smooth muscle hypertrophy/atrophy, and collagen volume fraction (fibrosis index).

Results: Obstructive injury induced marked progressive structural remodeling that varied significantly across ureteral segments ($p < 0.05$). Total wall thickness increased from a baseline of $415.8 \pm 16.9 \mu\text{m}$ to $602.7 \pm 24.3 \mu\text{m}$ by month 6, accompanied by a sharp elevation in the fibrosis index from 12.4% to 28.9% ($p < 0.05$). Early stages (3 months) were characterized by adaptive urothelial hyperplasia and smooth muscle bundle hypertrophy. Prolonged obstruction (6 months) resulted in decompensatory changes, including urothelial desquamation, vacuolar degeneration, and severe stromal collagen deposition. A distinct proximodistal severity gradient was identified: Intramural > Pelvic > Abdominal. The intramural segment exhibited the most profound destruction of mucosal architecture, extensive myocyte apoptosis/atrophy, and dense perivascular sclerotic replacement.

Conclusion: Chronic urinary obstruction causes heterogeneous, segment-dependent tissue destruction along the ureter, shifting from compensatory hypertrophy to irreversible fibrotic sclerosis. The intramural segment demonstrates the highest vulnerability to ischemic and mechanical damage, making it the primary locus for severe fibrotic remodeling.

