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PATHOMORPHOLOGICAL ALTERATIONS OF THE RENAL PELVIS IN DIABETES-ASSOCIATED NEPHROLITHIASIS

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Abstract

Diabetes mellitus is a chronic systemic metabolic disorder that profoundly affects renal physiology and significantly increases susceptibility to kidney stone formation. Diabetes-associated nephrolithiasis is characterized not only by metabolic derangements but also by distinct structural and pathological changes within the renal pelvis. Persistent hyperglycemia, insulin resistance, altered urinary composition, oxidative stress, and recurrent urinary tract infections create a pathological microenvironment that promotes crystal nucleation, aggregation, and retention within the pelvicalyceal system. Continuous mechanical irritation by calculi, combined with chronic inflammation and diabetic microangiopathy, leads to progressive urothelial damage, subepithelial fibrosis, vascular remodeling, and impairment of pelvic peristalsis. These alterations contribute to urinary stasis, obstruction, hydronephrosis, and secondary renal parenchymal injury. This article provides a comprehensive analysis of the pathogenetic mechanisms, histopathological characteristics, and clinical implications of renal pelvic pathology in diabetes-associated kidney stone disease based on contemporary experimental and clinical data.

Keywords: diabetes mellitus, nephrolithiasis, renal pelvis, urothelial injury, fibrosis, diabetic microangiopathy

Introduction

Diabetes mellitus represents one of the most prevalent metabolic disorders worldwide and is a leading contributor to chronic kidney pathology. In addition to classical diabetic nephropathy, growing evidence indicates that diabetes significantly increases the incidence and recurrence of kidney stone disease. Epidemiological studies demonstrate that diabetic patients have a 1.5–2-fold higher risk of nephrolithiasis compared with non-diabetic individuals [1].

The renal pelvis serves as a critical anatomical and functional component of the urinary tract, ensuring effective urine transport from the kidney to the ureter. In the setting of diabetes-associated nephrolithiasis, the renal pelvis becomes a primary target of pathological alterations due to prolonged exposure to supersaturated urine, recurrent infections, and repeated mechanical trauma caused by calculi. Despite its clinical importance, renal pelvic pathology in diabetic stone disease remains insufficiently characterized. Elucidation of these changes is essential for understanding disease progression and improving therapeutic outcomes. The pathogenesis of renal pelvic damage in diabetes-associated nephrolithiasis is multifactorial and involves complex metabolic, inflammatory, and vascular mechanisms. Chronic hyperglycemia leads to increased urinary glucose excretion, creating favorable conditions for bacterial colonization and recurrent urinary tract infections. Infectious agents further enhance inflammation and promote the formation of infection-related stones [2].

Insulin resistance plays a pivotal role in altering renal acid–base regulation. Reduced ammonium production results in persistently acidic urine, which promotes uric acid crystallization. These stones frequently localize within the renal pelvis, where they exert sustained mechanical pressure on the urothelium [3]. In parallel, advanced glycation end products and reactive oxygen species induce oxidative stress, damaging epithelial cell membranes and disrupting protective glycosaminoglycan layers.

Diabetic microangiopathy contributes significantly to pelvic pathology by impairing microcirculation, leading to chronic ischemia, delayed tissue regeneration, and increased susceptibility to fibrotic remodeling.

Histological examination of the renal pelvis in diabetes-associated nephrolithiasis reveals a characteristic pattern of pathological alterations. The urothelial layer often exhibits focal desquamation, epithelial thinning, and areas of erosion due to continuous mechanical irritation by calculi. In some regions, compensatory hyperplasia or squamous metaplasia develops as an adaptive response to chronic injury [4].

The subepithelial connective tissue demonstrates pronounced inflammatory infiltration composed primarily of lymphocytes, macrophages, and plasma cells. Persistent inflammation activates fibroblasts, resulting in excessive collagen deposition and progressive fibrosis of the pelvic wall. This fibrotic transformation reduces tissue elasticity and compromises normal pelvic peristalsis.

Vascular pathology is a prominent feature, including capillary basement membrane thickening, endothelial cell degeneration, and perivascular fibrosis, reflecting diabetic microangiopathy. In advanced stages, dystrophic calcification may be observed within fibrotic areas, further impairing pelvic compliance and urinary flow.

The described structural changes have significant functional consequences. Loss of urothelial integrity increases susceptibility to infection and hematuria, while fibrotic remodeling disrupts coordinated pelvic contractions, promoting urinary stasis. These factors create a self-perpetuating cycle that facilitates stone retention and recurrent stone formation.

Prolonged obstruction at the level of the renal pelvis may result in hydronephrosis, increased intrarenal pressure, and progressive atrophy of renal parenchyma. In diabetic patients, impaired immune defense and delayed tissue repair exacerbate disease severity and increase the risk of severe complications, including chronic pyelonephritis and urosepsis [5].

Conclusion

Diabetes-associated nephrolithiasis induces complex and progressive pathomorphological changes in the renal pelvis driven by metabolic dysregulation, chronic inflammation, oxidative stress, and microvascular injury. Urothelial damage,

inflammatory infiltration, fibrotic remodeling, and vascular alterations constitute the structural basis of pelvic dysfunction in this condition. These changes play a critical role in stone persistence, urinary obstruction, and secondary renal impairment.

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