

Ketamine-induced “walnut bladder”: A case report

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ABSTRACT. Ketamine-induced cystopathy is a severe complication of chronic recreational ketamine use, leading to bladder fibrosis and renal impairment. We report a 35-year-old man with long-term intranasal ketamine abuse who presented with severe lower urinary tract symptoms, hydronephrosis, and chronic kidney disease. Cystoscopy and histology revealed a fibrotic, inflamed bladder requiring reconstructive surgery with an ileal neobladder. This case highlights an advanced presentation of ketamine uropathy and emphasizes the need for early recognition to prevent irreversible urinary tract and renal damage.

Keywords: *Ketamine; Dissociative anesthetics; Walnut bladder; Uropathy; Addiction.*

Ketamine is a dissociative anesthetic widely used in both human and veterinary medicine. In recent years, its recreational use has increased steadily, particularly among young adults. Chronic consumption, especially via the intranasal route, has been associated with an emerging condition known as “ketamine-induced cystopathy”, characterized by irritative lower urinary tract symptoms, progressive reduction in bladder capacity, pelvic pain, and chronic renal impairment in advanced stages.

The proposed pathophysiologic mechanism involves direct urothelial injury caused by urinary ketamine metabolites, along with subepithelial inflammation, fibrosis, and microvascular alterations. Although this condition is well documented in Asian and European case series, it remains uncommon in Latin America, leading to delayed clinical suspicion and diagnosis. Early detection is crucial to prevent irreversible renal damage. We present the case of a 35-year-old man with chronic intranasal ketamine use who developed severe ketamine-induced cystopathy and chronic renal impairment.

CLINICAL CASE

A 35-year-old man with no family history of urologic disease had long-standing intranasal ketamine abuse since adolescence, associated with occasional alcohol and cannabis use. Since 2020, he had been under urologic follow-up for

severe bladder dysfunction and recurrent urinary tract infections. In May 2025, he presented with fever, dysuria, marked urinary frequency, and bilateral flank pain. On physical examination, he appeared pale and had bilateral costovertebral angle tenderness. Continuous urinary incontinence was noted.

Laboratory tests revealed elevated serum creatinine, consistent with chronic kidney disease, anemia of chronic disorders, abnormal liver function tests, and blood cultures positive for *Enterococcus faecalis*. Computed tomography (CT) showed bilateral hydronephrosis, inflammatory thickening of the renal pelvis and ureters, and a small-capacity bladder (80 mL) with irregular, thickened walls (Fig. 1). Cystoscopy revealed a diffusely erythematous and friable bladder mucosa. Histopathologic examination demonstrated a fibrotic *lamina propria* with myxoid changes, congested vessels, mononuclear infiltration, and reactive urothelial changes. Alternative infectious or iatrogenic causes (including tuberculosis, radiotherapy, and interstitial cystitis) were excluded.

During hospitalization, targeted antibiotic therapy was administered with resolution of sepsis. Given the severe bladder involvement and persistent pelviccalyceal dilatation, reconstructive surgery was planned. An orthotopic neobladder was constructed using an ileal segment, with a favorable postoperative course, as shown in Fig. 2, which demonstrates adequate filling and no immediate complications.

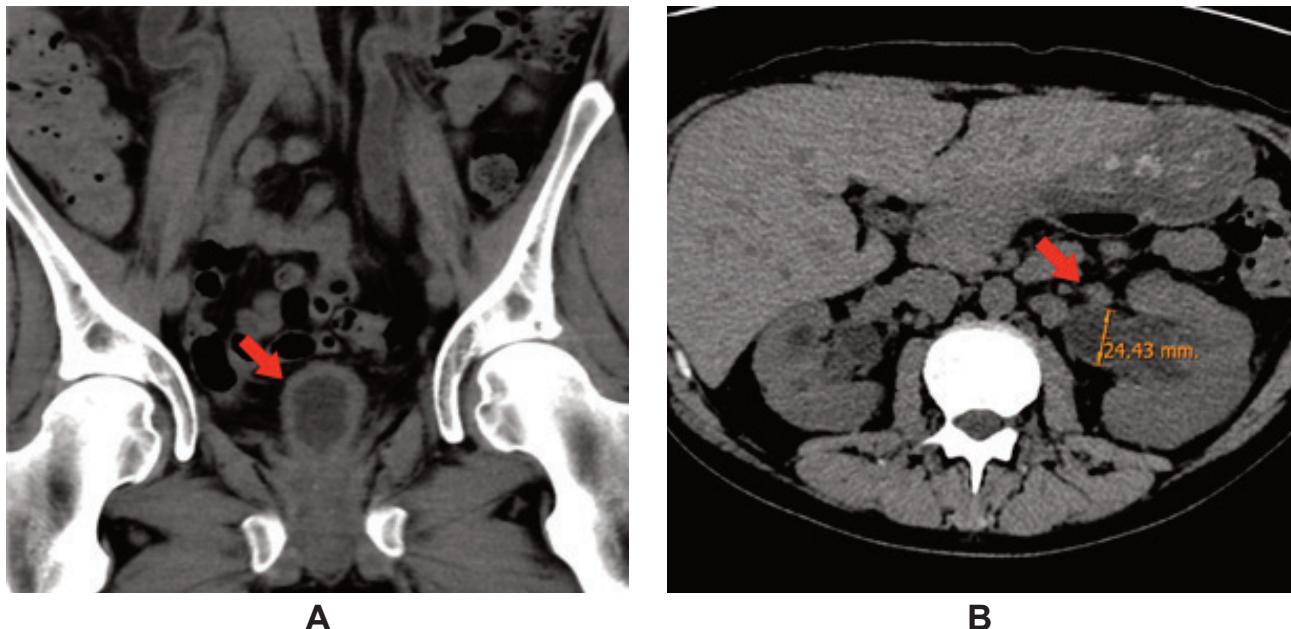


Figure 1. Abdominopelvic CT images. A. Coronal view showing a low-capacity bladder with diffuse mural thickening (red arrow), consistent with chronic cystopathy. B. Axial view showing a dilated ureter measuring 24.4 mm (red arrow), consistent with uronephrosis secondary to obstructive bladder dysfunction (Credits: courtesy of the authors).

On follow-up, the patient showed symptomatic improvement, a reduction in infectious episodes, and partial recovery of renal function. He remains under urologic and nephrologic follow-up, with psychological support to maintain abstinence.

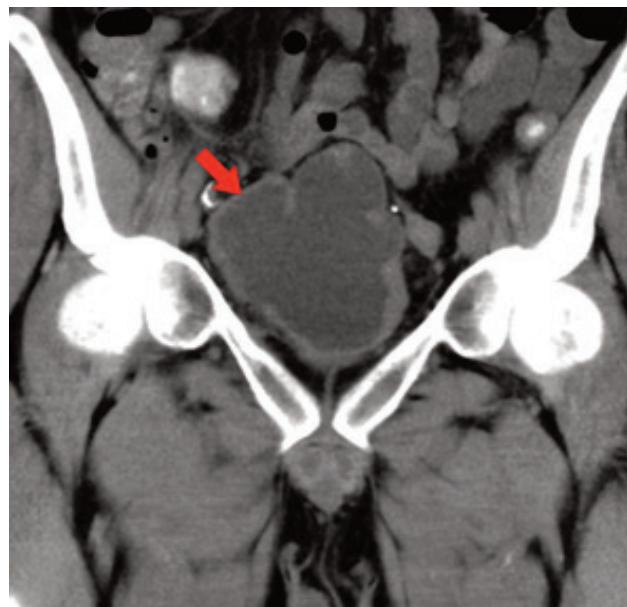


Figure 2. Postoperative coronal CT demonstrating an orthotopic neobladder created from a small bowel segment (red arrow), showing satisfactory filling (Credits: courtesy of the authors).

DISCUSSION

Ketamine cystopathy is an emerging complication of chronic recreational use and represents a diagnostic challenge in regions where the prevalence of abuse is low or underreported. Its pathophysiology is related to the direct toxicity of urinary metabolites on the bladder epithelium, oxidative damage, and inflammatory processes leading to fibrosis and loss of bladder compliance.¹⁻³

The clinical presentation typically includes urinary urgency, frequency, nocturia, suprapubic pain, and hematuria.^{4,5} Over time, bladder contracture, secondary hydronephrosis, and renal function deterioration may develop.⁶⁻⁸ Diagnosis is primarily clinical, supported by imaging studies, cystoscopy, and histopathologic findings. In the present case, the finding of a markedly reduced bladder capacity with diffuse wall thickening and biopsy evidence of inflammatory fibrosis confirmed the toxic etiology.

Management depends on the stage of disease: in early phases, cessation of ketamine use and medical therapy may stabilize symptoms.⁹⁻¹² In advanced stages, when bladder capacity is below 100 mL or upper urinary tract involvement is present, reconstructive surgery with enterocystoplasty is the most effective option.¹³⁻¹⁶

The relevance of this case lies in its late presentation, with severe bladder and renal damage requiring reconstruc-

tive surgery. Reports from Latin America remain scarce, underscoring the importance of considering ketamine cystopathy in the differential diagnosis of chronic bladder dysfunction in young adults.¹⁷

CONCLUSION

Ketamine cystopathy should be suspected in patients presenting with chronic lower urinary tract symptoms and a history of recreational use, even in the absence of recent

exposure. Early recognition is essential to prevent progression to chronic kidney disease and to preserve bladder function. The present case illustrates an advanced form of this condition and reinforces the need to raise awareness among the medical community regarding its clinical manifestations and timely management.

Conflicts of interest

The authors declare no conflicts of interest.

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