CASE REPORT
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First Ketamine-Induced Ulcerative Cystitis Cases from Türkiye: Clinical Course, Histopathological and Radiological Findings in Young Female Patients

Serdar Madendere[1], Mert Kılıç[1,2], Ersin Köseoğlu[2], Ayşenur İğdem[3], Tufan Tarcan[2,4]

[1]Vehbi Koç Foundation American Hospital, Clinic of Urology, İstanbul, Türkiye
[2]Koç University School of Medicine, Department of Urology, İstanbul, Türkiye
[3]Vehbi Koç Foundation American Hospital, Clinic of Pathology, İstanbul, Türkiye
[4]Marmara University School of Medicine, Department of Urology, İstanbul, Türkiye

Abstract

Ketamine-induced ulcerative cystitis (KIC) is an emerging urological disorder associated with recreational ketamine use, characterized by inflammation, bladder wall hypertrophy, and involvement of the upper urinary tract. We report two female patients with ketamine-related ulcerative cystitis. The first case represented advanced-stage KIC, with findings including hydronephrosis, bladder fibrosis, and impaired renal function. In contrast, the second case presented at an early stage with clinical and radiological findings that were reversible after complete cessation of ketamine use. Histopathological examination in both cases revealed ulceration, chronic inflammation, and fibrosis. Radiological and cystoscopic findings closely correlated with disease severity. Early recognition of KIC and strict cessation of ketamine use are crucial to prevent irreversible bladder damage and progressive renal deterioration.

Keywords: Ketamine-induced cystitis, ulcerative cystitis, recreational drug abuse

Introduction

Ketamine, introduced in the 1960s as an N-methyl-D-aspartate receptor antagonist, remains widely used as an anesthetic agent (1). However, because of its dissociative and euphoric effects, ketamine has also been used illicitly as a recreational drug (2).

Ketamine-induced ulcerative cystitis (KIC) was first described by Shahani et al. (3) in 2007 in patients presenting with hematuria, urgency, increased daytime frequency, dysuria, and reduced bladder capacity. In some cases, involvement of the upper urinary tract and progression to chronic kidney disease were also reported.

More than 25% of recreational ketamine users develop urinary symptoms, which correlate with both cumulative dose and duration of use (4). Although symptoms may improve following ketamine cessation, irreversible bladder and renal damage may occur in some patients (5,6). Wu et al. (7) proposed a staging system for KIC, classifying the disease into inflammatory, early fibrotic, and severe fibrotic stages.

After obtaining informed consent, we present two young female patients diagnosed with ulcerative cystitis secondary to recreational ketamine use, representing both early and advanced stages of the disease.

Case 1

A 32-year-old woman with a 4-year history of recreational ketamine abuse had discontinued ketamine use six months prior to presentation. Her medical history included cholecystectomy, dyspareunia, endometrioma, and pelvic inflammatory disease. The patient reported the onset of suprapubic pain in April 2022, followed by dysuria in May 2022.

Correspondence: Tufan Tarcan MD, PhD, Koç University School of Medicine, Department of Urology; Marmara University School of Medicine, Department of Urology, İstanbul, Türkiye
E-mail: bilgi@tufantarcan.com **ORCID-ID:** orcid.org/0000-0002-3387-3524
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Her first urological evaluation was conducted in November 2022. At that time, she reported severe lower urinary tract symptoms (LUTS), including increased daytime frequency (approximately hourly), nocturia (approximately hourly at night), dysuria, and bladder pain. Laboratory investigations revealed elevated alkaline phosphatase (ALP) (262 IU/L), gamma-glutamyl transferase (319 IU/L), C-reactive protein (CRP) (36 mg/dL), and eosinophilia (7.3%). Urinalysis demonstrated leukocyturia and hematuria, while urine culture was sterile. Ultrasonography showed bladder wall thickening measuring 9 mm, with no other significant findings.

A cystoscopy performed in November 2022, following hydrodistension, revealed an ulcerative lesion with mucosal bleeding. The maximum bladder capacity under general anesthesia was 200 mL. Urine cytology and biopsies of the lesion were obtained, and fulguration was performed. Histopathological examination demonstrated inflammation, vascular congestion, and fibrosis (Figure 1). The pathological findings were not entirely consistent with interstitial cystitis (IC). Urine cytology revealed reactive urothelial cells, neutrophils, and erythrocytes.

Following cystoscopy and hydrodistension, the patient experienced a slight improvement in her symptoms. The patient was recommended an IC-friendly diet and treated with amitriptyline and pentosan polysulfate; however, her symptoms progressively worsened by September 2023. She subsequently developed gross hematuria, anemia, and renal impairment with a serum creatinine level of 1.42 mg/dL. Urodynamic evaluation revealed urinary incontinence with terminal detrusor overactivity, reaching a detrusor pressure of 59 cm H_2O at a bladder volume of 37 mL. Abdominal magnetic resonance imaging demonstrated diffuse bladder wall thickening, bilateral grade 2-3 hydroureteronephrosis, and perivesical inflammation (Figure 2). Intravesical botulinum toxin injection was recommended, but she refused. By March 2024, bladder capacity had further declined to 60 mL.

During psychiatric evaluation, the patient admitted to ongoing ketamine abuse. She was subsequently hospitalized and underwent electroconvulsive therapy. She did not receive any further urological treatment during this period. Six months after cessation of ketamine use, urinary urgency and bladder pain improved markedly. Follow-up urinary ultrasonography revealed a bladder capacity of 68 mL, bilateral grade 1 hydroureteronephrosis, and regression of diffuse bladder wall thickening. The serum creatinine level at follow-up was 1.56 mg/dL.

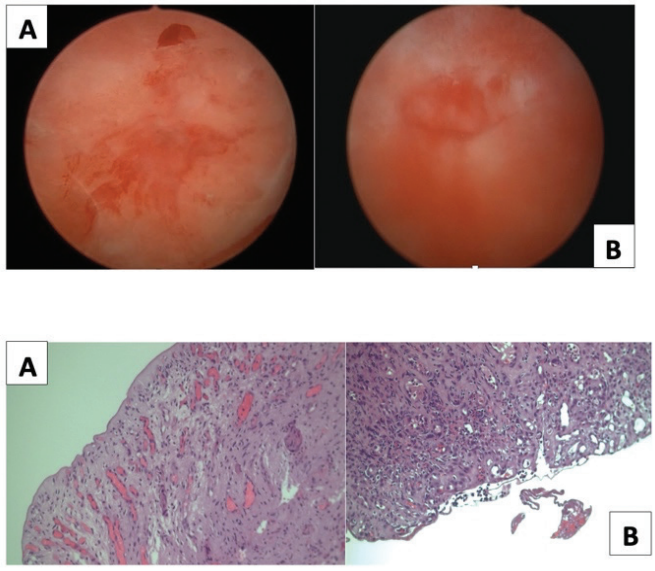

Figure 1. Cystoscopic and histopathological views of ketamine-induced ulcerative bladder lesions

A: Case 1, B: Case 2

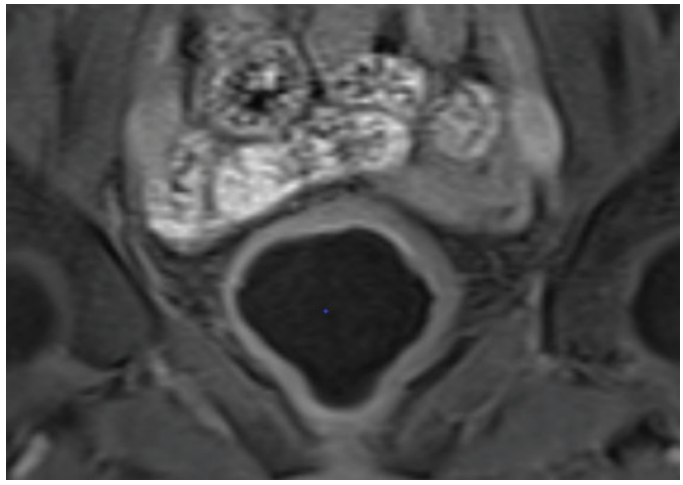

Figure 2. Magnetic resonance imaging of Case 1

Case 2

A 25-year-old woman reported a two-year history of recreational ketamine use via nasal insufflation. Her medical history included premenstrual syndrome and a prior uterine curettage. She presented with a one-week history of dysuria, hematuria, and increased daytime urinary frequency, occurring every 30-60 minutes. Urinalysis demonstrated pyuria, and a urine culture grew *Klebsiella pneumoniae*; oral cefuroxime 500 mg was administered. A cystoscopy performed in March 2024 revealed ulcerative lesions, glomerulations, and petechial

bleeding. The maximum bladder capacity under general anesthesia was 250 mL. Histopathological examination of bladder biopsies showed ulceration and chronic non-specific cystitis with eosinophilic infiltration; IC could not be excluded. Complete cessation of ketamine use and adoption of an IC-friendly diet were recommended. At the 3-month follow-up, the patient's symptoms had resolved and urinalysis findings were within normal limits. Subsequently, she experienced an uncomplicated pregnancy and delivery.

Discussion

We present two cases of KIC that represent different stages of the disease: one with an advanced-stage presentation and one with an early-stage presentation. Both cases involved young women. Although previous studies have reported that approximately 80% of patients with KIC are male, female patients tend to exhibit higher symptom severity scores (8). The clinical presentation of KIC can mimic carcinoma in situ, IC in women, and prostatitis in men, which may delay accurate diagnosis (9).

In our first case, laboratory evaluation revealed elevated CRP and eosinophil levels in the peripheral blood, along with abundant leukocytes and erythrocytes on urinalysis, despite a sterile urine culture. In contrast, the second case had a documented urinary infection, accompanied by pyuria and microscopic hematuria. Typical laboratory findings in KIC include sterile pyuria, microscopic hematuria, and, in some cases, elevated liver enzymes such as ALP (3,10). In the first case, peripheral eosinophilia and renal dysfunction suggested upper tract involvement, consistent with advanced-stage disease. Previous studies have demonstrated that elevated serum immunoglobulin E, eosinophilia, and eosinophilic infiltration of the bladder wall are associated with increased bladder pain severity (11).

The primary histopathological feature of KIC is inflammation, which underlies the development of LUTS. Persistent inflammation leads to collagen deposition and progressive fibrosis, ultimately resulting in a contracted bladder and, in advanced stages, involvement of the upper urinary tract (12,13). In both of our cases, histopathological examination of bladder biopsies demonstrated prominent inflammatory cell infiltration and fibrosis. It has been suggested that eosinophilic infiltration and detrusor muscle hypertrophy are distinguishing pathological features of KIC compared with other inflammatory bladder conditions such as IC (14). Nevertheless, clinical presentation and radiologic findings remain essential components of the differential diagnosis.

In our first case, the earliest radiological finding was increased bladder wall thickness. Following approximately one year of ketamine abuse, an magnetic resonance imaging scan revealed bilateral grade 2-3 hydronephrosis, perivesical inflammation, marked bladder wall thickening, and contracted bladder. Previous studies have shown that patients with IC typically exhibit bladder wall thinning, whereas patients with KIC exhibit bladder wall thickening (15). Recently, Betancur et al. (16) also reported a case of KIC that demonstrated significant bladder wall thickening on computed tomography. In addition, ureteral involvement and perivesical inflammation are features that more clearly distinguish KIC from other inflammatory bladder disorders (17). These findings support the notion that radiological changes correlate with both the intensity and duration of ketamine abuse.

Hydrodistension and an IC-friendly diet provided temporary symptomatic relief in both of our cases, consistent with previous reports (18,19). Nevertheless, absolute cessation of ketamine use remains the cornerstone of management (4). Although conservative management may alleviate symptoms in early stages, patients with advanced disease often require surgical interventions such as augmentation cystoplasty or urinary diversion (18-20). Recurrence is common following resumption of ketamine use (21).

To our knowledge, these cases represent the first reported instances of KIC from Türkiye. Although the short-term follow-up period limits our ability to draw conclusions regarding long-term renal outcomes, our findings illustrate the clinical and radiological progression of KIC and underscore the potential for partial recovery when the disease is recognized early and ketamine use is discontinued.

Conclusion

KIC should be considered in the differential diagnosis of ulcerative cystitis in young adults presenting with refractory LUTS. Radiological and cystoscopic findings are valuable in distinguishing KIC from other inflammatory bladder diseases. Early diagnosis and strict cessation of ketamine use, supported by a multidisciplinary approach, are essential to prevent irreversible bladder dysfunction and progressive renal damage.

Ethics

Informed Consent: Written informed consent was obtained from the patients.

Footnotes

Authorship Contributions

Surgical and Medical Practices: S.M., M.K., E.K., A.İ., T.T., Concept: S.M., M.K., E.K., T.T., Design: S.M., M.K., E.K., T.T., Data Collection or Processing: S.M., M.K., E.K., T.T., Analysis or Interpretation: S.M.,

M.K., E.K., T.T., Literature Search: S.M., M.K., E.K., T.T., Writing: S.M., M.K., E.K., T.T.

Conflict of Interest: No conflict of interest was declared by the authors.

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