Case Report

Ketamine-induced bladder syndrome: Recognition as a key differential diagnosis and the need to curb recreational use

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ABSTRACT

The recreational use of ketamine is becoming increasingly widespread, largely due to its dissociative and anesthetic properties, as well as its accessibility and affordability. A significant and often irreversible impact on the urinary system has recently emerged as a concerning consequence of its recreational use. We present a case report of a young man who has been using ketamine recreationally for less than a year and has developed ketamine bladder syndrome. Within a few months, the patient developed acute kidney injury and some liver damage, as indicated by his blood tests, while continuing to take ketamine.

Key words: Differential diagnosis of cystitis, Ketamine, Ketamine bladder syndrome, Recreational drug

Ketamine, a dissociative anesthetic, is used in the field of medicine to induce and sustain anesthesia, pain management, and depression [1]. Furthermore, owing to its hallucinogenic and dissociative effects, it is used as a recreational drug [2]. Patients with ketamine abuse can present with a variety of debilitating symptoms, including urinary frequency, nocturia, urgency, incontinence, cystitis, hematuria, and bladder or loin pain, causing a profoundly negative impact on quality of life [3]. A nationwide prevalence study in the UK has revealed that 26.6% of regular ketamine users experience at least one urinary symptom, with 51% experiencing an improvement of symptoms on cessation [4]. Approximately 50% of ketamine users experiencing urinary symptoms do not seek medical attention promptly, with symptoms becoming more pronounced after 2 years [5]. Current medical treatment modalities for such patients primarily focus on pain relief and cessation of ketamine, which is the most effective way to prevent severe and irreversible damage that may ultimately necessitate extensive reconstructive surgery.

CASE PRESENTATION

A 20-year-old male presented to the Emergency Department with continuous right flank pain radiating to the loin, sharp in nature, with a pain score of 6/10. The patient was experiencing pain for the past 2–3 months, had worsened by the time of this presentation, and showed no improvement with oral acetaminophen. He also complained of blood in his urine, ongoing for 2–3 months, and increased frequency of urination. There was no history of fever, vomiting, chest pain, or shortness of breath. In addition, he had no history of constipation, diarrhea, dysuria, or urinary retention. He reported using 3 g of ketamine (in powder form) daily, through nasal insufflation, for the past year. The patient has a past medical history of asthma. There were no known drug allergies.

On examination, the patient was alert and oriented to time, place, and person. Vitals were as follows: Blood pressure – 144/84, Pulse – 92, Temp – 37.2, Respiratory rate – 17, Saturation – 95% on air. The chest was clear with equal air entry bilaterally. The abdomen was soft, non-distended, and non-tender. Central nervous system examination showed a Glasgow Coma Scale of 15/15 (E4, V5, M6), and there were no focal neurological deficits. There were no rashes, bruises, or edema. Blood investigations and urinalysis are shown in Tables 1 and 2.

He was treated with oral paracetamol, oramorph, and IV 0.9% sodium chloride. Computed tomography of the kidney, ureter, and bladder showed a shrunken urinary bladder with diffuse mural thickening. Bilateral moderate hydronephrosis with mildly dilated ureters to the vesico-ureteric junction indicates strictures. No renal tract calcification was found. Appearances would support the diagnosis of ketamine-associated lower urinary tract destruction (Fig. 1).

Following a discussion with the urology team, the patient was discharged home on oral antibiotics for a urinary tract infection.

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Table 1: Blood investigations of the patient

Blood tests	Results
PH	7.42
Lactate	0.5
PCO_2	5.29
Calcium	1.19
Glucose	5.4
HCO_3	26.2
Hemoglobin	143
WCC	11.9
NEUT	7.89
PLT	358
LYMP	1.86
EOSIN	0.58
CRP	79
Paracetamol	12
Salicylate	<3
Bilirubin	5
ALT	47
ALP	86
GGT	94
INR	1.2
Na	139
K	3.9
Creatinine	72
eGFR	>90

WCC: White cell count, NEUT: Neutrophils, PLT: Platelet count, CRP: C-reactive protein, ALP: Alkaline phosphatase, ALT: Alanine aminotransferase, GGT: Gamma-glutamyl transferase

Table 2: Urinalysis of the patient

Urine tests	Results
PH	6.5
Specific gravity	1.020
Glucose	Negative
Ketones	Negative
Blood	+++
Proteins	+++
leukocytes	Trace
Nitrites	Negative

When seen in the urology clinic, as an outpatient, it was explained to him that his urinary bladder problem would not resolve and instead worsen if he continued taking ketamine.

The patient has been under observation for several months since his initial presentation. During this period, he has been hospitalized multiple times due to continued recreational ketamine use, which has resulted in acute kidney injury and abnormal liver function. Repeated blood tests revealed elevated creatinine levels (109 μmoL/L), a reduced glomerular filtration rate of 84 mL/min, and elevated liver enzymes with alanine aminotransferase at 160 U/L, alkaline phosphatase at 359 U/L, and gamma-glutamyl transferase at 1212 U/L. The patient has been advised to stop using ketamine and offered rehabilitation.

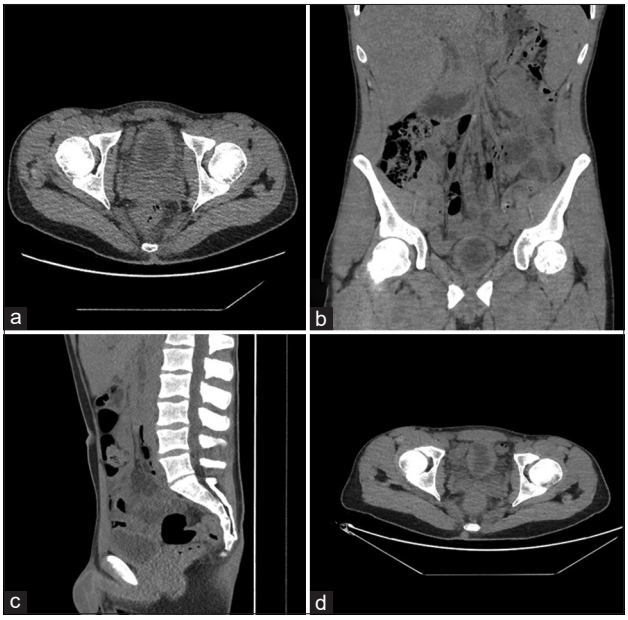

Figure 1: (a-d) Computed tomography of the kidney, ureter, and bladder shows a shrunken urinary bladder with diffuse mural thickening

DISCUSSION

Ketamine was initially created as an alternative to phencyclidine for use as an anesthetic. It functions as a non-competitive antagonist of the N-methyl-d-aspartate receptor complex and is utilized in several medical applications, including anesthesia, treatment for epilepsy, pain relief, and depression management [4]. As a recreational drug, ketamine can be administered in several ways, including injection, inhalation, oral consumption, or smoking in various forms. This multifaceted substance is known for its powerful anesthetic, analgesic, stimulant, and psychedelic effects. One notable effect, often referred to as the "K-Hole," can induce a sensation of dissociation from one's body and surroundings, leading to an experience akin to floating outside of oneself. The effects of the drug are brief, and users often develop tolerance rapidly, which leads them to require higher and more frequent doses to achieve similar results [1,5,6].

The most frequently observed toxic consequence associated with ketamine abuse is abnormalities of the urinary tract, which include ulcers, cystitis, and fibrosis, leading to urinary incontinence, hematuria, bladder overactivity and shrinkage, eventually leading to hydroureter and hydronephrosis, a clinical entity known as "ketamine bladder syndrome" [5-7]. Cholestasis related to chronic ketamine abuse has also been described recently.

Early cessation of ketamine use remains the best opportunity for symptom resolution and preventing further multi-system damage [7-9]. Furthermore, the recurrence of symptoms after resuming ketamine use highlights the importance of ketamine abstinence. Many studies support the fact that abstinence leads to symptomatic improvement. Significantly lower pain urgency frequency (PUF) scores and a larger voided volume have been reported in people who had abstained from ketamine use for a year as compared to active ketamine abusers. This trend toward higher voided volumes and lower PUF scores was observed as

the duration of ketamine cessation increased, although neither variable was statistically significant [10-12]. A similar follow-up study of 101 participants abstaining from ketamine abuse and 218 active ketamine users exhibited statistically significantly lower PUF scores and a higher bladder capacity in the abstinence group [13]. In addition, abstinence was the sole protective factor associated with a reduction in symptoms, increased voided volume, and greater bladder capacity [14].

Raising awareness about these risks is essential, especially among recreational users and health-care providers who may not routinely discuss the adverse effects associated with ketamine. Educational programs focusing on and educating ketamine users about the signs and symptoms of urinary complications associated with its use will help curb this menace and also make them aware of the importance of seeking help if they experience such issues. By improving awareness and understanding of ketamine's potential dangers, we can encourage safer practices among users and promote more effective prevention and intervention strategies within health-care systems. Abstinence from ketamine is fundamental in treating ketamine-induced bladder syndrome, as well as other liver and gallbladder diseases related to ketamine abuse.

CONCLUSION

Ketamine abuse has garnered increasing attention due to its potential to cause significant health issues, particularly ketamine-induced urinary symptoms. Many users may be ignorant about the association between excessive use of ketamine and serious bladder problems, including inflammation, increased urinary frequency, and even ulcerative cystitis. These conditions arise from the drug's irritating effects on the urinary system, often resulting in chronic pain and a lower quality of life for affected individuals.

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