CASE REPORT

Imaging Features of Ketamine-Induced Cystitis and Cholangiopathy: A Report of Two Cases

Komal Chughtai[1*]

[1]Weill Cornell Medicine/New York Presbyterian Hospital, New York, NY 10065, USA

ABSTRACT

The use of ketamine as a recreational drug of abuse is on the rise among young people around the world. Due to this, more and more chronic and avid users are presenting with ketamine-induced intra-abdominal complications. In this report, we present two cases of ketamine bladder (KB) and cholangiopathy (KC) and discuss their radiologic findings. KC should be considered in patients with unexplained abnormal liver function tests, and evidence of biliary ductal strictures and dilation on imaging. KB patients often present with symptoms of cystitis and hematuria, with bladder wall thickening and intra-vesicular bleeding on imaging. Clinicians and radiologists should maintain a high index of suspicion for these complications in patients presenting with biliary and urinary abnormalities and a history of ketamine use, as they can have significant morbidity.

Keywords: *Ketamine, ketamine bladder, ketamine cystitis, ketamine cholangiopathy.*

INTRODUCTION

Ketamine, a dissociative anesthetic first synthesized in the 1960s, has traditionally been used for human and veterinary medical use. It has gained widespread popularity as a recreational drug in the past decade due to its hallucinogenic and euphoric effects. It is a type of arylcycloalkylamine, chemically similar to phencyclidine (PCP) [1, 2]. Ketamine is classified as a Schedule III controlled substance in the United States of America [2]. However, it remains readily available in many parts of the world, contributing to its increasing prevalence as a substance of abuse throughout Asia. The United Nations Office on Drugs and Crime (UNODC) further identifies ketamine as a growing drug of abuse throughout East and South East Asia [3]. Due to this worrisome trend, more and more patients are presenting with previously unknown ketamine-induced pathologies.

Beyond its psychiatric effects, chronic ketamine use has been implicated in a spectrum of abdominal symptoms, collectively posing diagnostic and therapeutic challenges. Among the most frequently reported intra-abdominal conditions are ketamine-associated cystitis (ketamine bladder, KB) and ketamine-associated cholangiopathy (KC), both of which can result in severe morbidity.

In this series, we present 2 cases of ketamine cystitis and cholangiopathy to highlight their radiologic manifestations and shed light on a growing diagnostic challenge.

*Corresponding Author: Komal Chughtai, Weill Cornell Medicine/New York Presbyterian Hospital, New York, NY 10065, USA; E-mail: komal.chughtai@gmail.com
Received: December 31, 2024; Revised: March 13, 2025; Accepted: March 17, 2025
DOI: https://doi.org/10.37184/nrjp.3007-5181.1.23

CASE PRESENTATION

Case 1

A 28-year-old female presented to the emergency department with pelvic pain, hematuria, and passing blood clots with urine. She also experienced burning suprapubic pain, nausea, and vomiting. The patient did not have any prior medical or surgical history. Social history was notable for frequent ketamine use, with occasional marijuana use.

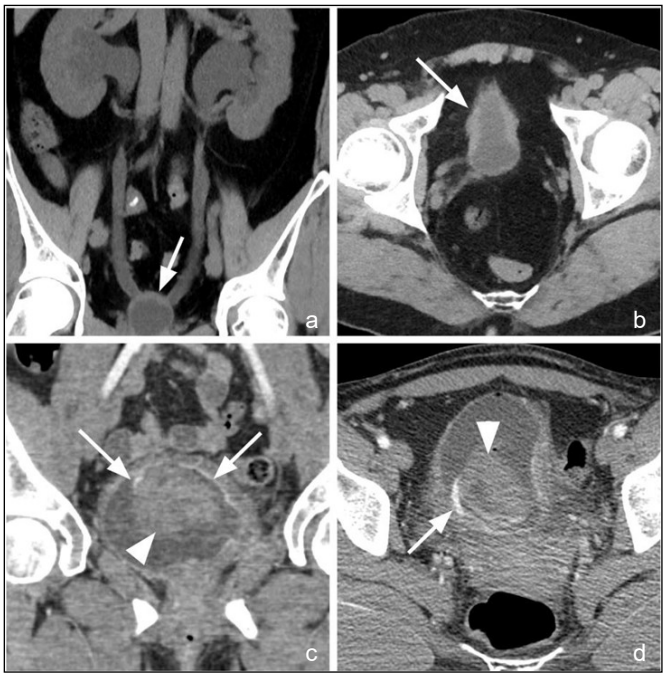

Fig. (1): 28-year-old female presenting with KB. Non-contrast coronal CT **(a)** demonstrated bilateral hydroureteronephrosis, and irregular bladder wall thickening (arrow). Non-contrast axial CT **(b)** demonstrates irregular bladder wall thickening (arrow). No ureteral or bladder calculi were present. Contrast-enhanced urothelial phase CT in coronal **(c)** and axial **(d)** demonstrate contrast extravasation into the bladder (arrows), and a large thrombus in the bladder lumen (arrow heads).

 ISSN: 3007-519X (Online)

The patient was afebrile, with vital signs within normal limits. Physical exam was notable for suprapubic tenderness to palpation. Complete blood count was notable for anemia (hemoglobin 7.5 g/dL). Urine analysis was remarkable for hematuria (>50 red blood cells).

Renal ultrasound demonstrated sonographically normal appearing kidneys. CT abdomen and pelvis without and with contrast demonstrated bladder wall thickening, and a large hyperattenuating blood clot in the bladder lumen **(Fig. 1a-d)**. Additionally, there was extravasation of contrast into the bladder lumen, consistent with active intravesicular hemorrhage **(Fig. 1a-d)**. She was diagnosed with KB.

The patient was treated with intravesicular carboprost and copious bladder irrigation. She was counseled on avoiding future ketamine usage.

Case 2

A 37-year-old male with a history of ketamine abuse disorder, presented with intermittent abdominal pain for the past 4-5 days. He was previously admitted to the hospital for KB and neurogenic bladder. A physical exam revealed tenderness to palpation in the right upper quadrant. He did not exhibit jaundice. The patient was afebrile, with vital signs within normal limits. His liver function tests were notable for mildly elevated alkaline phosphatase.

Ultrasound of the right upper quadrant demonstrated a normal gallbladder, without cholelithiasis. Magnetic resonance cholangiopancreatography (MRCP) demonstrated areas of mild segmental intrahepatic biliary ductal dilation and strictures, most notable in segment 4a, and in the mid-common bile duct **(Fig. 2a-c)**. There was peribiliary enhancement around the dilated ducts on gadobutrol-enhanced T1-weight MRI **(Fig. 2a-c)**. Clinical workup for primary sclerosing cholangitis and autoimmune etiologies was negative. These findings were determined to be caused by KC.

The patient was managed conservatively with supportive care while admitted. His abdominal pain improved over the following week and he was discharged, with counseling on ketamine cessation.

DISCUSSION

Recreational ketamine use has significantly increased around the world, particularly among young people. Recent published studies estimate that approximately

3 million people have used recreational ketamine at least once in their lives in the United States, with most users below the age of 25 years [4]. Due to this trend, more and more patients are presenting with ketamine-associated urinary and biliary complications.

KB, characterized by lower urinary tract symptoms such as dysuria, hematuria, and pelvic pain, is histologically associated with chronic urothelial inflammation and ulceration [2]. This is typically seen in patients reporting chronic and frequent ketamine use. Imaging findings on CT and MRI include circumferential bladder wall thickening, trabeculation, and hemorrhagic products in the bladder lumen [2]. In acute cases, there may be the presence of peri-vesicular fat stranding, and hyper- enhancement of the bladder wall.

On cystoscopy, these patients are found to have ulceration of the bladder urothelium, with granulation tissue and denuded mucosa [2]. In severe cases, urothelial inflammation can progress to involve the ureters and renal collecting systems. Chu *et al.* published a report of 59 KB patients, in which 51% of their study population developed either unilateral or bilateral hydronephrosis, with 4 patients also developing papillary necrosis [5].

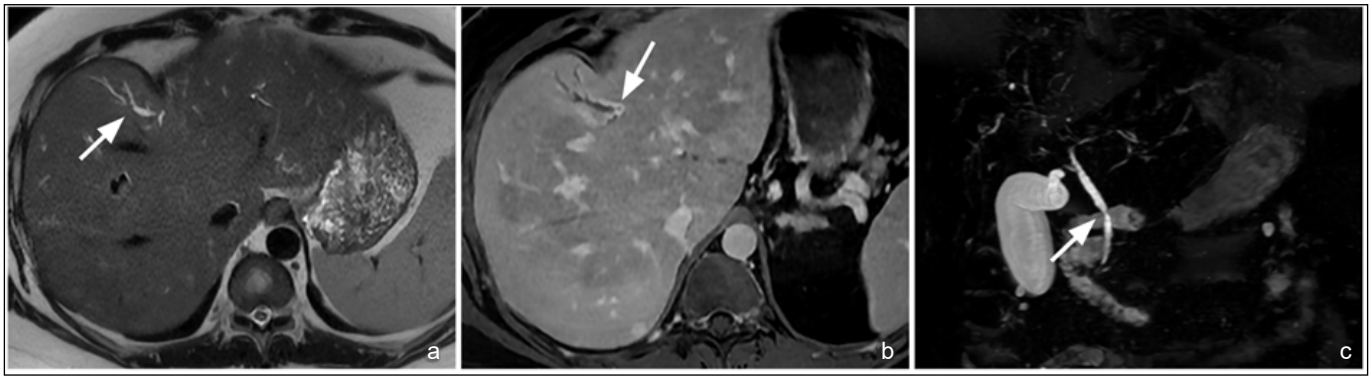

Fig. (2): 37-year-old male presenting with KC. T2-weighted MRI **(a)** demonstrates biliary dilation most pronounced in segment 4a (arrow). Gadobutrol-enhanced T1-weighted MRI in delayed phase **(b)** demonstrates peribiliary enhancement in segment 4a, with biliary stricture (arrow). Coronal MRCP image **(c)** shows a stricture in the common bile duct (arrow).

KC, though less commonly recognized, is a serious biliary complication characterized by chronic inflammation and sclerosis of the biliary ducts. Patients typically present with jaundice, abdominal pain, and elevated liver enzymes. Contrast-enhanced MRI with MRCP are the best imaging modalities to access KC. Imaging findings include intra- and extrahepatic bile duct dilation and strictures. Acute biliary inflammation can be accompanied by peribiliary enhancement of MRI.

While the imaging features of KC are similar to primary sclerosing cholangitis, its mechanism is not clearly understood. One proposed hypothesis is that ketamine leads to the blockage of NMDA receptors in smooth muscle cells within the biliary ducts may lead to their dysfunction [6]. Furthermore, it may also cause decreased relaxivity of the sphincter of Oddi, ultimately causing upstream biliary stasis [6].

CONCLUSION

With recreational ketamine use around the world continuing to rise, it is paramount for clinicians and radiologists to keep ketamine-induced complications on the differential for patients presenting with abdominal symptoms.

CONSENT FOR PUBLICATION

Not applicable.

CONFLICT OF INTEREST

The author declares no conflict of interest.

ACKNOWLEDGEMENTS

Declared none.

REFERENCES

1. Zarantonello P, Bettini E, Paio A, Simoncelli C, Terreni S, Cardullo F. Novel analogues of ketamine and phencyclidine as NMDA receptor antagonists. Bioorg Med Chem Lett 2011; 21(7): 2059-63.
DOI: https://doi.org/10.1016/j.bmcl.2011.02.009

2. Morgan CJ, Curran HV. Independent scientific committee on drugs. Ketamine use: A review. Addiction 2012; 107(1): 27-38.
DOI: https://doi.org/10.1111/j.1360-0443.2011.03576.x

3. United Nations Office on Drugs and Crime, Synthetic Drugs in East and Southeast Asia: Latest developments and challenges. 2024; Available at: https://www.unodc.org/roseap/uploads/ documents/Publications/2024/Synthetic_Drugs_in_East_and_ Southeast_Asia_2024.pdf

4. Ni A, Lee Cantrell F, Clark RF. Ketamine exposure demographics and outcomes over 16 years as reported to US poison centers. Am J Emerg Med 2018; 36(8): 1459-62.
DOI: https://doi.org/10.1016/j.ajem.2018.04.066

5. Chu PS, Ma WK, Wong SC, Chu RW, Cheng CH, Wong S, *et al.* The destruction of the lower urinary tract by ketamine abuse: A new syndrome? BJU Int 2008; 102(11): 1616-22.
DOI: https://doi.org/10.1111/j.1464-410X.2008.07920.x

6. Bartoli A, Cursaro C, Seferi H, Andreone P. Secondary sclerosing cholangitis After SARS-CoV2: ICU ketamine use or virus-specific biliary tropism and injury in the context of biliary ischemia in critically ill patients? Hepat Med 2023; 15: 93-112.
DOI: https://doi.org/10.2147/HMER.S384220