Received: 20 June 2023 | Revised: 30 March 2024 | Accepted: 13 May 2024

DOI: 10.1002/ccr3.9016

CASE REPORT

Clinical Case Reports Open Access WILEY

Sphincter of oddi dysfunction induced by ketamine: A case report

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Key Clinical Message

Chronic ketamine use can lead to sphincter of oddi dysfunction (SOD), causing various hepatobiliary complications. Recognizing substance abuse history is vital for early detection. Timely intervention can prevent irreversible liver and pancreas damage.

Abstract

Ketamine is commonly abused as a recreational drug worldwide due to its ability to induce euphoria-like effects. Ketamine abuse is associated with many hepatobiliary side effects ranging from cholestasis to biliary sepsis and death. Here we present a case of a young 29-year female with upper abdominal pain due to SOD resulting from chronic use of ketamine. SOD can result in obstruction or dysfunction of the bile and pancreatic ducts. Ketamine induces SOD by activation of the muscarinic receptors in the sphincter of oddi. Detail history of substance abuse is crucial for early identification of ketamine-induced SOD. Early identification and treatment of this rare condition can prevent permanent injury to the liver and pancreas.

KEYWORDS

biliary diseases, drug abuse, low-dose ketamine, sphincter of oddi dysfunction

1 | INTRODUCTION

Ketamine is used as a recreational drug (street ketamine) due to its ability to induce euphoria and a trance-like state. Street ketamine can be inhaled, swallowed, or injected. It is abused in many parts of the world.[1] Although the recreational dosage is 15%–20% lower than the amount used for anesthesia, the extended and widespread recreational use of this substance has led to an increase in both side effects and fatalities.[2] In the United States, more than 2.3 million teens and adults used ketamine in their lifetime.[2]

Although the effects of ketamine on the urinary bladder have been widely reported, its effects on the bile duct and its management have not been extensively studied.[3] We report a rare case of the sphincter of oddi dysfunction (SOD) in a young female who presented to the hospital with abdominal pain.

2 | CASE HISTORY/ EXAMINATION

A 29-year-old female with a history of recreational ketamine use for the past 8 years presented to the emergency department with right upper quadrant abdominal pain associated with nausea and non-bilious and non-bloody vomiting.

At the presentation, her vitals were stable. Physical examination was unremarkable except for the tenderness in the right upper quadrant, without guarding or rebound tenderness.

3 | METHODS (DIFFERENTIAL DIAGNOSIS, INVESTIGATION, AND TREATMENT)

Lab studies revealed an Alkaline Phosphatase (ALP) of 116 U/L. Chest X-ray did not show any cardiopulmonary pathology. Electrocardiogram showed a normal sinus rhythm. Ultrasound of the abdomen revealed a prominent common bile duct (CBD) without gallbladder pathology. computed tomography (CT) of the abdomen and pelvis showed dilatation of the CBD measuring up to 9 mm.

She was admitted to the hospital for the workup of CBD dilation and pain management. During hospitalization, her pain was managed adequately with analgesics; however, her liver enzymes continued to increase (Table 1).

An inpatient gastroenterology consultation was done, and a shared decision was made to perform an Endoscopic retrograde cholangiopancreatography (ERCP), given her symptoms of right upper quadrant pain, ketamine abuse, the elevation of liver enzymes, and dilatation of the CBD without obstructive pathology. ERCP revealed the dilatation of CBD and Type 2 SOD (Figure 1). Sphincterotomy was performed (Figure 2) and post-sphincterotomy fluoroscopy was done which suggested reduced CBD dilatation.

4 | OUTCOME AND FOLLOW-UP

Sphincterotomy resulted in subsequent symptom resolution and eventual discharge with outpatient gastroenterology follow-up. The patient was happy with the treatment provided.

5 | DISCUSSION

SOD is a condition characterized by abnormal muscular valve function regulating the flow of bile and pancreatic juice into the duodenum. SOD can result in obstruction or dysfunction of the bile and pancreatic ducts, leading to symptoms including abdominal pain, nausea, vomiting, and diarrhea. The prevalence of SOD is estimated to be 1.5% in the general population and up to 29% in patients with chronic abdominal pain.[4]

Ketamine is a dissociative anesthetic drug used for pain management and sedation. Ketamine has a short half-life, undergoes extensive first-pass metabolism in the liver, and is primarily excreted through urine and bile.[5] Although some studies have shown that ketamine can induce the contraction of the sphincter, leading to obstruction of the bile and pancreatic ducts, more studies are still required to confirm this.[6,7] The mechanism by which ketamine induces SOD is not fully understood, but it is thought to involve the activation of the muscarinic receptors in the sphincter of oddi. It has been shown to enhance the release of acetylcholine, a neurotransmitter that activates the muscarinic receptors in the sphincter. This increases the tone and contraction of the sphincter, which can obstruct the bile and pancreatic ducts.[6,8] This can result in elevated liver enzymes, jaundice, and pancreatitis.

The diagnosis of SOD due to ketamine abuse is based on the patient's history of ketamine use, symptoms of biliary or pancreatic dysfunction, and imaging studies such as magnetic resonance cholangiopancreatography (MRCP) or endoscopic retrograde cholangiopancreatography (ERCP). MRCP can detect any structural abnormalities in the bile and pancreatic ducts, while ERCP can measure

TABLE 1 Progression of liver enzymes.

Liver function test	On the day of hospitalization	Day 1	Day 2	Day 3	Day 4	One Week	Normal range
Total Bilirubin (mg/dL)	0.2	0.2	0.2	0.3	0.2	0.4	0–1.2
Direct bilirubin (mg/dL)	0	0	0	0	0	0	0–0.3
AST (Aspartate aminotransferase, U/L)	16	148	168	91	489	71	10–40
ALP (Alkaline phosphatase, U/L)	116	139	165	173	241	99	40–129
ALT (Alanine aminotransferase, U/L)	18	113	151	520	520	86	7–56

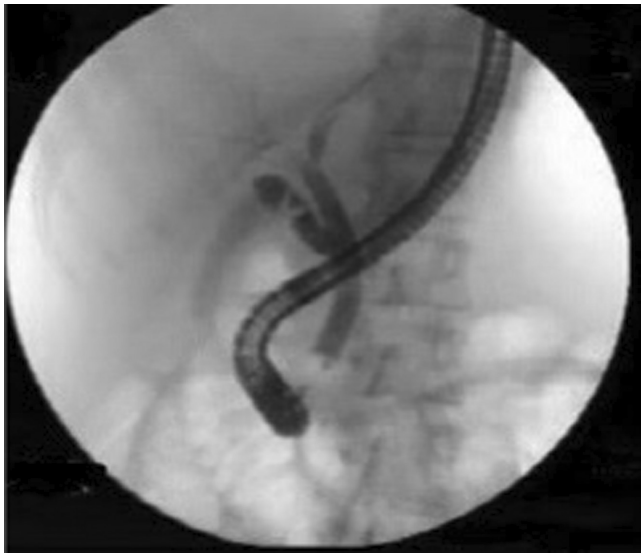

FIGURE 1 ERCP showing obstructive cholangiogram.

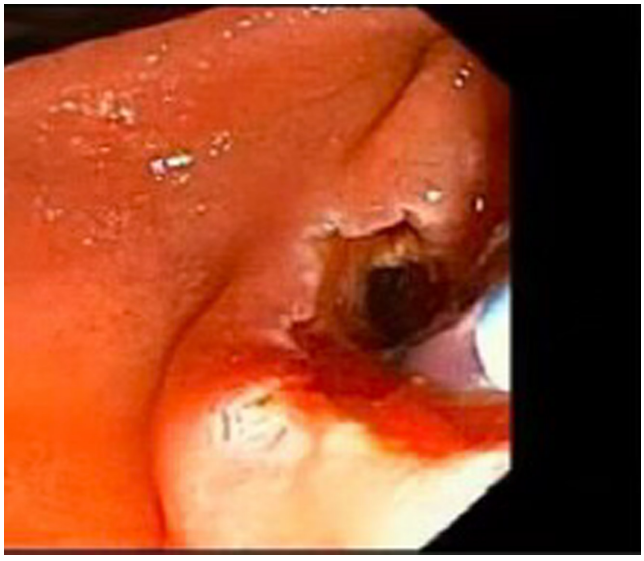

FIGURE 2 During sphincterotomy.

the sphincter's pressure and diagnose SOD.[8,9] The treatment of SOD due to ketamine abuse includes cessation of ketamine use and using medications to relax the sphincter, such as nitrates, calcium channel blockers, and anticholinergics. Endoscopic sphincterotomy, a procedure that involves cutting the sphincter, can also relieve the obstruction of the bile and pancreatic ducts.[9]

In conclusion, SOD due to ketamine abuse is a serious medical condition that can lead to biliary and pancreatic dysfunction. It is essential to identify the condition early to prevent complications such as pancreatitis and hepatic damage. Treatment involves cessation of ketamine use and the use of medications or endoscopic sphincterotomy to relieve the obstruction of the bile and pancreatic ducts.

6 | CONCLUSION

Ketamine is commonly abused among teens and adults worldwide. Ketamine-induced sphincter dysfunction can be life-threatening if an early diagnosis is not made. Physicians should be aware of this rare finding in patients with acute abdominal pain. Early diagnosis and management can prevent further hepatic and pancreatic injury.

AUTHOR CONTRIBUTIONS

Nava Raj Sharma: Conceptualization; methodology; writing – original draft; writing – review and editing. **Arjun Basnet:** Conceptualization; methodology; writing – original draft. **Saral Lamichhane:** Conceptualization; methodology; writing – original draft; writing – review and editing. **Kripa Tiwari:** Conceptualization; methodology; writing – original draft. **Jeffy Varghese:** Conceptualization; methodology; writing – original draft. **Sudarshan Gautam:** Conceptualization; methodology; writing – original draft. **Madalasa Pokhrel:** Conceptualization; methodology; writing – original draft.

FUNDING INFORMATION

None.

CONFLICT OF INTEREST STATEMENT

None.

DATA AVAILABILITY STATEMENT

All data regarding this case has been reported in the manuscript. Please contact the corresponding author if you are interested in any further information.

CONSENT

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of written consent is available for review by the Editor-in-Chief of this journal on request.

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REFERENCES

1. Lankenau SE, Sanders B, Bloom JJ, Hathazi D. Towards an explanation of subjective ketamine experiences among young injection drug users. *Addict Res Theory*. 2008;16(3):273-287. doi:10.1080/16066350801983749
2. Liu Y, Lin D, Wu B, Zhou W. Ketamine abuse potential and use disorder. *Brain Res Bull*. 2016;126:68-73. doi:10.1016/j.brainresbull.2016.05.016
3. Srirangam S, Mercer J. Ketamine bladder syndrome: an important differential diagnosis when assessing a patient

with persistent lower urinary tract symptoms. *Case Reports*. 2012;2012:bcr2012006447. doi:10.1136/bcr-2012-006447
4. Bistritz L. Sphincter of oddi dysfunction: managing the patient with chronic biliary pain. *World J Gastroenterol*. 2006;12(24):3793-3802. doi:10.3748/wjg.v12.i24.3793
5. Dinis-Oliveira RJ. Metabolism and metabolomics of ketamine: a toxicological approach. *Forensic Sci Res*. 2017;2(1):2-10. doi:10.1080/20961790.2017.1285219
6. Al-Nowfal A, Al-Abed Y. Chronic biliary colic associated with ketamine abuse. *Int Med Case Rep J*. 2016;2016:135-137. doi:10.2147/IMCRJ.S100648
7. Varadarajulu S, Wilcox CM. Prospective evaluation of ketamine on sphincter of Oddi motility. *Gastrointest Endosc*. 2006;63(5):AB281. doi:10.1016/j.gie.2006.03.726
8. Nyirenda TJ, Shirazi-Nejad A, Soliman AS. Persistent ketamine-induced cholangiopathy: an approach to management. *Cureus*. 2020;21:e11611. doi:10.7759/cureus.11611
9. Afghani E, Lo SK, Covington PS, Cash BD, Pandol SJ. Sphincter of Oddi function and risk factors for dysfunction. *Front Nutr*. 2017;4:4. doi:10.3389/fnut.2017.00001

How to cite this article: Sharma NR, Basnet A, Lamichhane S, et al. Sphincter of oddi dysfunction induced by ketamine: A case report. *Clin Case Rep*. 2024;12:e9016. doi:10.1002/ccr3.9016