**Note of the editors:** This is a BOWMAN-generated English translation of the officially indexed Spanish-language article, which should be cited as Emergencias 2026;38:398-400. In this translated version, the editors have supervised the process; however, it cannot be ruled out that some errors resulting from the artificial intelligence translation process may have gone unnoticed.

# LETTERS TO THE EDITOR

## Common bile duct dilatation probably due to chronic tusi use

### *Dilatación del colédoco como posible consecuencia del consumo crónico de tusi*

**To the Editor,**

Recreational ketamine use, either alone or as an ingredient of preparations such as tusi or pink cocaine, has increased in recent years.[1,2] Its neuropsychiatric effects as a novel psychoactive substance have already been described, but evidence concerning the systemic consequences of chronic use remains limited, particularly its hepatobiliary effects, which have been associated with cholangiopathy and biliary tract dilatation.[3-6] We present a case of common bile duct dilatation probably associated with prolonged tusi use.

A 25-year-old woman with no known allergies, a past medical history of eclampsia, and several years of chronic tusi use interrupted by short periods of abstinence presented to the emergency department. In recent weeks, she had been taking between 3 and 5 doses daily. While on holiday in Barcelona (Catalonia, Spain), she sought emergency care for a 3-day history of burning abdominal pain located in the epigastrium and right upper quadrant, radiating to the right lumbar region and associated with vomiting (> 15 episodes) and a subjective fever without documented temperature elevation. She reported multiple similar episodes previously in her home city, Madrid, which had prompted emergency visits on two occasions, without a specific diagnosis or subsequent investigations because of poor engagement with the healthcare system. Her general condition was good, and she was hemodynamically stable and afebrile. Physical examination revealed epigastric tenderness without peritoneal signs and a negative Murphy sign. Laboratory tests showed an isolated cholestatic pattern, with GGT of 414 U/L (normal range, 5–40), alkaline phosphatase of 256 U/L (normal range, 46–116), and normal bilirubin, without elevations in transaminases or acute-phase reactants; renal function, coagulation studies, and complete blood count were normal. Abdominal ultrasonography showed intrahepatic bile ducts with a common bile duct measuring up to 6 mm (upper limit of normal) (Figure 1), nonspecific wall thickening, no biliary obstruction, and a gallbladder without stones.

During admission to the emergency department observation area, symptomatic treatment with analgesics, antiemetics, and intravenous fluids was initiated, with progressive clinical improvement. Laboratory tests performed 24 hours later showed no substantial changes. Given the suspicion of ketamine-associated cholangiopathy, she was advised to abstain from tusi and undergo outpatient magnetic resonance cholangiopancreatography. She was discharged after 24 hours with symptomatic treatment and follow-up at her referring center.

Cholangiopathy associated with chronic ketamine use is a rare condition characterized by bile duct dilatation, wall thickening, and cholestatic abnormalities in the absence of mechanical obstruction.[3,5-9] Its pathophysiology remains incompletely understood, although a direct toxic effect on the biliary epithelium and smooth muscle dysfunction, with progressive inflammatory and fibrotic changes, have been proposed.[3,5] This would occur through mechanisms analogous to ketamine-induced ulcerative or sclerosing cystitis, a well-described complication in chronic users, involving urothelial toxicity, chronic inflammation, and fibrosis.[10-12] The coexistence or sequential occurrence of urinary and hepatobiliary involvement suggests a possible shared spectrum of ketamine-induced epithelial injury, dependent on dose and duration of use.[3,5]

In the present case, the absence of an obstructive cause, the biochemical pattern, and the history of intensive tusi use—a probable source of ketamine—support this etiology. A definitive diagnosis has not been established because there has been no subsequent follow-up and no imaging is available beyond the ultrasound performed in the emergency department; the case should therefore be considered probable. Nevertheless, this patient's clinical history highlights the importance of considering emerging organ complications associated with novel psychoactive substances[1,2] and underscores the need for a multidisciplinary approach and close clinical follow-up.[4,5]

Carla Boixeda,
Javier Felipe Del Barrio,
Miguel Ángel Galicia,
Óscar Miró

*Servicio de Urgencias, Hospital Clínic, Barcelona, Spain.*

*cboixedaserra@outlook.es*

**Conflict of Interest Disclosures:** None declared.

**Author Contributions, Funding, and Ethical Responsibilities:** All authors have confirmed their authorship, the non-existence of external funding, and the maintenance of confidentiality and respect for patients' rights in the author's responsibilities document, publication agreement, and assignment of rights to EMERGENCIAS.

**Editor in Charge:** Javier Jacob Rodríguez.

**Article not commissioned by the Editorial Board and with external peer review.**

## References

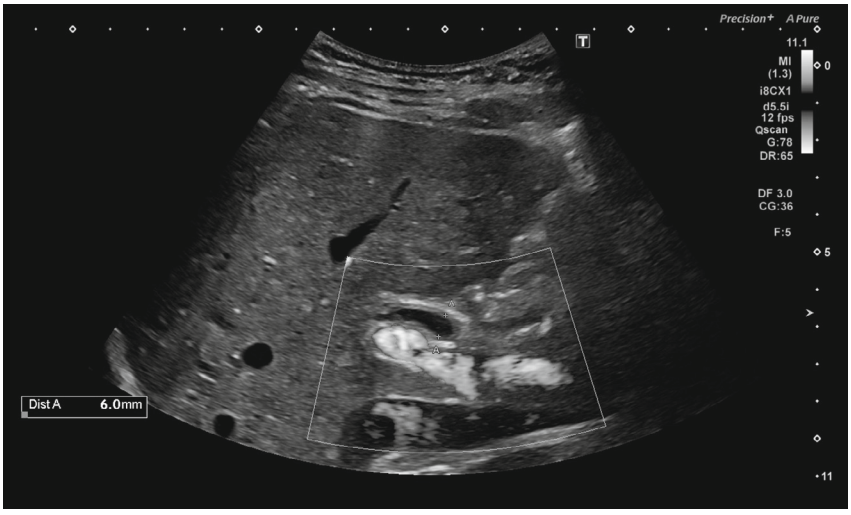



**Figure 1.** Intrahepatic bile ducts with a common bile duct measuring up to 6 mm.

1 European Union Drugs Agency. Ketamine in Europe: EMPACT situation report. Luxembourg: Publications Office of the European Union; 2026. (Accessed 30 March 2026). Available at: https://www.euda.europa.eu/publications/joint-publications/ketamine-europe_en
2 United Nations Office on Drugs and Crime. World drug report 2023. Viena: UNODC; 2023.
3 Teymouri A, Nasoori H, Fakheri M, Nasiri A. Features of biliary tract diseases in ketamine abusers: a systematic review of case reports. J Med Case Rep. 2024;18:84.
4 Ng SH, Tse ML, Ng HW, Lau FL. Emergency department presentation of ketamine abusers in Hong Kong: a review of 233 cases. Hong Kong Med J. 2010;16:6-11.
5 Tkachuk B, Swain L, Burak KW, Swain MG. Chronic ketamine use resulting in a reversible cholangiopathy: a case series from a single Canadian centre and review of the literature. Can Liver J. 2026;9:55-63.
6 Wong GL, Tam YH, Ng CF, Chan AW, Choi PC, Chu WC, et al. Liver injury is common among chronic abusers of ketamine. Clin Gastroenterol Hepatol. 2014;12:1759-62.
7 Lui KL, Lee WK, Li MK. Ketamine-induced cholangiopathy. Hong Kong Med J. 2014;20:78.e1-e2.
8 Lo RS, Krishnamoorthy R, Freeman JG, Austin AS. Cholestasis and biliary dilatation associated with chronic ketamine abuse: a case series. Singapore Med J. 2011;52:e52-5.
9 Yu WL, Cho CC, Lung PF, Hung EH, Hui JW, Chau HH, et al. Ketamine-related cholangiopathy: a retrospective study on clinical and imaging findings. Abdom Imaging. 2014;39:1241-6.
10 Selby NM, Anderson J, Bungay P, Chesterton LJ, Kolhe NV. Obstructive nephropathy and kidney injury associated with ketamine abuse. NDT Plus. 2008;1:310-2.
11 Shahani R, Streutker C, Dickson B, Stewart RJ. Ketamine-associated ulcerative cystitis: a new clinical entity. Urology. 2007;69:810-2.
12 Chu PS, Kwok SC, Lam KM, Chu TY, Chan SW, Man CW, et al. 'Street ketamine'-associated bladder dysfunction: a report of ten cases. Hong Kong Med J. 2007;13:311-3.