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Beyond the Bladder: Ketamine Cholangiopathy With Renal and Electrolyte Complications

Nishat Farjana [1]

1. General Internal Medicine, Barking, Havering And Redbridge University Hospitals NHS Trust, Romford, GBR

Corresponding author: Nishat Farjana, nishat.farjana@nhs.net

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Abstract

Ketamine-induced uropathy is well recognised, but chronic ketamine use can also injure the bile ducts. This ketamine cholangiopathy is less familiar and may mimic primary sclerosing cholangitis (PSC), bile duct stones, or malignancy. We describe a 26-year-old man with many years of heavy ketamine use (about 4 g daily) who was found collapsed and needed three days of intensive care. He had cholestatic liver blood tests without jaundice: alkaline phosphatase (ALP) 1014 U/L, gamma-glutamyl transferase (GGT) 3873 U/L, and bilirubin 7 µmol/L, with normal alanine aminotransferase (ALT), albumin, and international normalised ratio (INR). Magnetic resonance cholangiopancreatography (MRCP) showed a 25 mm narrowed segment of the intrapancreatic common bile duct with upstream dilatation to 12 mm, unchanged from three months earlier, with no stones, a normal pancreas and no convincing features of PSC. Viral, autoimmune, immunoglobulin G4 (IgG4) and carbohydrate antigen 19-9 (CA 19-9) tests were negative. Ketamine cholangiopathy was considered the most likely diagnosis, and the gastroenterology team deferred endoscopic investigation in favour of a repeat MRCP at six months. He also had ketamine uropathy, with bilateral hydroureteronephrosis and a small, contracted bladder with urothelial calcification. His admission was complicated by acute kidney injury (creatinine 85 to 350 µmol/L), which improved after urinary catheterisation, recurrent hyperkalaemia and chronic hyponatraemia attributed mainly to high fluid intake. Repeated intravenous calcium gluconate for hyperkalaemia was followed by a transient, treatment-related rise in calcium. Kidney function and potassium normalised, sodium mildly improved but remained low, and he was discharged with drug and alcohol team follow-up. Clinicians should consider ketamine use in young patients with unexplained cholestasis. Ketamine cholangiopathy can mimic PSC, stones or malignancy, and coexisting uropathy and electrolyte disturbances are important clues and complications. Recognising this pattern can support an individualised approach in which invasive tests such as endoscopic retrograde cholangiopancreatography (ERCP) may be deferred, with close imaging follow-up.

Categories: Internal Medicine, Nephrology, Gastroenterology
Keywords: acute kidney injury, cholangiopathy, hyperkalaemia, hyponatraemia, ketamine, ketamine uropathy, secondary sclerosing cholangitis, substance misuse

Introduction

Ketamine is a dissociative anaesthetic that is increasingly used recreationally in the United Kingdom, particularly by young adults [1]. In England and Wales, 2.0% of people aged 16 to 24 years reported ketamine use in the last year in 2024/2025 [1], and the number of people starting treatment for problematic ketamine use in England increased about 12-fold over the preceding decade [1]. The level of use differs between countries [1]. Its best-known complication is ketamine-induced uropathy, first described as ulcerative cystitis in daily users [2]. Chronic use can lead to a small, contracted bladder, hydroureteronephrosis and acute kidney injury [3,4].

Chronic ketamine use can also damage the biliary tree. In a large cohort of chronic users with urinary symptoms, about one in 10 had liver injury, which was cholestatic in every case [5]. Imaging often shows fusiform dilatation of the common bile duct (CBD), sometimes with distal narrowing or intrahepatic duct changes [6-8]. These appearances can be mistaken for a choledochal cyst [9], and they can mimic primary sclerosing cholangitis (PSC), choledocholithiasis or pancreaticobiliary malignancy, which may lead to invasive investigation [10]. Much of the published evidence on ketamine-related bile duct injury comes from Hong Kong [5,7-9]. The European Association for the Study of the Liver (EASL) now lists ketamine as a toxic cause of secondary sclerosing cholangitis [11], and progression to this condition has been reported in ketamine users [12]. Ketamine cholangiopathy has been reported to improve when ketamine is stopped [13], although progression despite abstinence has also been described [14].

We report a young man with probable ketamine cholangiopathy and coexisting uropathy, whose admission was complicated by acute kidney injury, recurrent hyperkalaemia and chronic hyponatraemia, and by a transient rise in calcium caused by treatment. The case highlights the need to ask directly about ketamine use in patients with unexplained cholestasis, and shows how recognising the pattern can support a cautious, individualised approach to invasive investigation. This article was previously presented as a meeting abstract at the Bangladeshi Doctors' UK National Conference 2026 at Watford, UK on October 24, 2026.

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Case Presentation

A 26-year-old man was brought to the emergency department after being found collapsed in the street. He had used ketamine for many years, recently about 4 g daily, and his last use was the day before he collapsed. He had known ketamine-related bladder disease and chronic hyponatraemia, both documented since 2024. He reported no alcohol use, no other drug use, and no regular medicines. He was underweight.

On arrival, he had cold, clammy skin and low oxygen saturation, requiring supplemental oxygen. His blood pressure was 108/73 mmHg (reference range: 100/60 to 140/100 mmHg), and his lactate was 0.7 mmol/L (reference value: <1.8 mmol/L). He was not jaundiced, and abdominal examination was normal, with no palpable liver. He was treated for suspected sepsis and admitted to the intensive care unit (ICU) for three days. All blood and urine cultures were negative, and antibiotics were stopped.

Investigations

Key blood results are shown in Table *1*.

Test	Result	Reference range
Sodium (chronic, since 2024)	120-125 mmol/L	133-146 mmol/L
Potassium (recurrent peaks)	6.0-6.5 mmol/L	3.5-5.3 mmol/L
Adjusted calcium (after intravenous calcium)	3.30 mmol/L	2.20-2.60 mmol/L
Adjusted calcium (later)	2.49 mmol/L	2.20-2.60 mmol/L
Phosphate	2.27 mmol/L	0.80-1.50 mmol/L
Creatinine (baseline)	85 µmol/L	59-104 µmol/L
Creatinine (peak)	350 µmol/L	59-104 µmol/L
Alkaline phosphatase	1014 U/L	30-130 U/L
Gamma-glutamyl transferase	3873 U/L	<60 U/L
Bilirubin	7 µmol/L	<21 µmol/L
Alanine aminotransferase (admission)	13 U/L	<41 U/L
Alanine aminotransferase (discharge)	16 U/L	<41 U/L
Aspartate aminotransferase (Day 3)	105 U/L	<40 U/L
Albumin	38 g/L	35-50 g/L

TABLE 1: Key blood results during admission

Liver function tests showed a cholestatic pattern without jaundice, with raised alkaline phosphatase (ALP) and gamma-glutamyl transferase (GGT). Alanine aminotransferase (ALT) was normal on admission and at discharge, and aspartate aminotransferase (AST) was 105 U/L on day three. Liver synthetic function was preserved: albumin was 38 g/L, the international normalised ratio (INR) was 1.0 and the prothrombin time was 10.7 seconds, all normal throughout. Creatinine rose from a baseline of 85 µmol/L to a peak of 350 µmol/L. Creatine kinase (CK), parathyroid hormone (PTH) and vitamin D were normal.

A liver screen was negative. This included a viral hepatitis screen, antimitochondrial antibody (AMA), anti-smooth muscle antibody (ASMA) and antineutrophil cytoplasmic antibody (ANCA). Serum immunoglobulin G4 (IgG4) and carbohydrate antigen 19-9 (CA 19-9) were not raised.

Abdominal ultrasound showed a CBD of 10.4 mm with mild intrahepatic duct dilatation. Magnetic resonance cholangiopancreatography (MRCP) showed a 25 mm narrowed segment of the intrapancreatic (distal) CBD, which tapered towards the ampulla, with no filling defects. Above it, the CBD measured 12 mm and the common hepatic duct 8.5 mm, with central intrahepatic duct dilatation (Figure *1*), and the gallbladder was thin-walled and over-distended (Figure *1*).

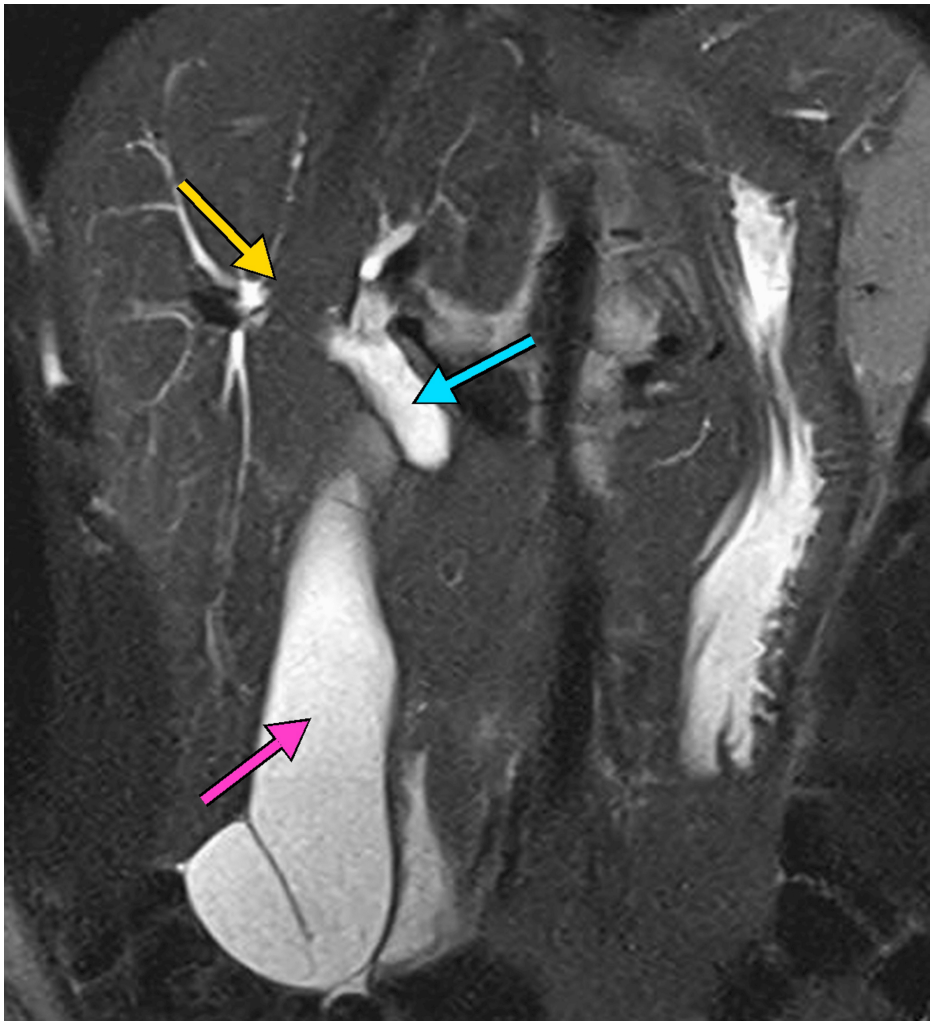


FIGURE 1: Coronal T2-weighted magnetic resonance image of the biliary tree

The yellow arrow shows dilated central intrahepatic bile ducts. The light blue arrow shows the dilated common bile duct (12 mm) above the narrowed intrapancreatic segment. The pink arrow shows the thin-walled, over-distended gallbladder.

There were no stones, the pancreas and pancreatic duct were normal, and there were no convincing features of PSC. The liver was enlarged, and there were periportal and portacaval lymph nodes up to 8.2 mm. The appearances were unchanged from an MRCP three months earlier. The images of that earlier scan were not available to us, so this comparison is based on the radiology report. The gastroenterology team reviewed him and considered ketamine cholangiopathy highly likely. Because he had no jaundice or cholangitis, they deferred endoscopic retrograde cholangiopancreatography (ERCP) and endoscopic ultrasound (EUS), and planned a repeat MRCP in six months with outpatient review.

Ultrasound and MRCP showed bilateral hydroureteronephrosis (Figure *2*), and MRCP also showed ureteric urothelial thickening.

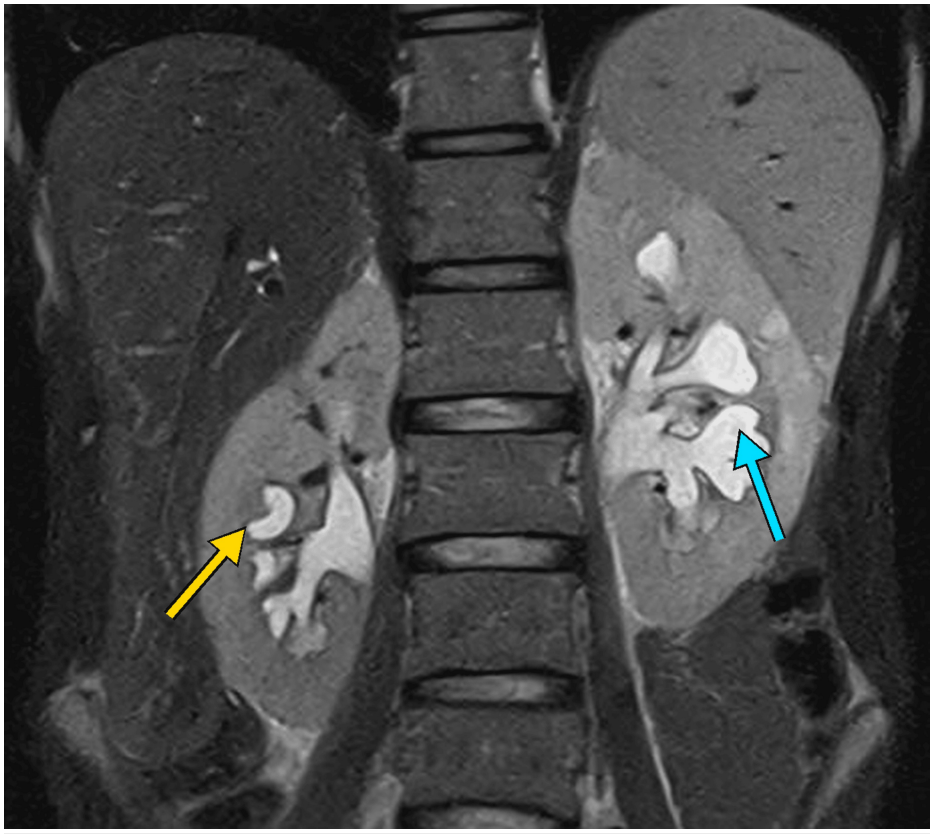


FIGURE 2: Coronal T2-weighted magnetic resonance image of the kidneys

The yellow arrow shows the dilated renal pelvis and calyces of the right kidney. The light blue arrow shows the dilated renal pelvis and calyces of the left kidney.

An earlier computed tomography (CT) scan of the kidneys, ureters and bladder (CT KUB), performed eight months before this admission, showed a small, contracted bladder with calcification along its anterior urothelium (Figure *3*).

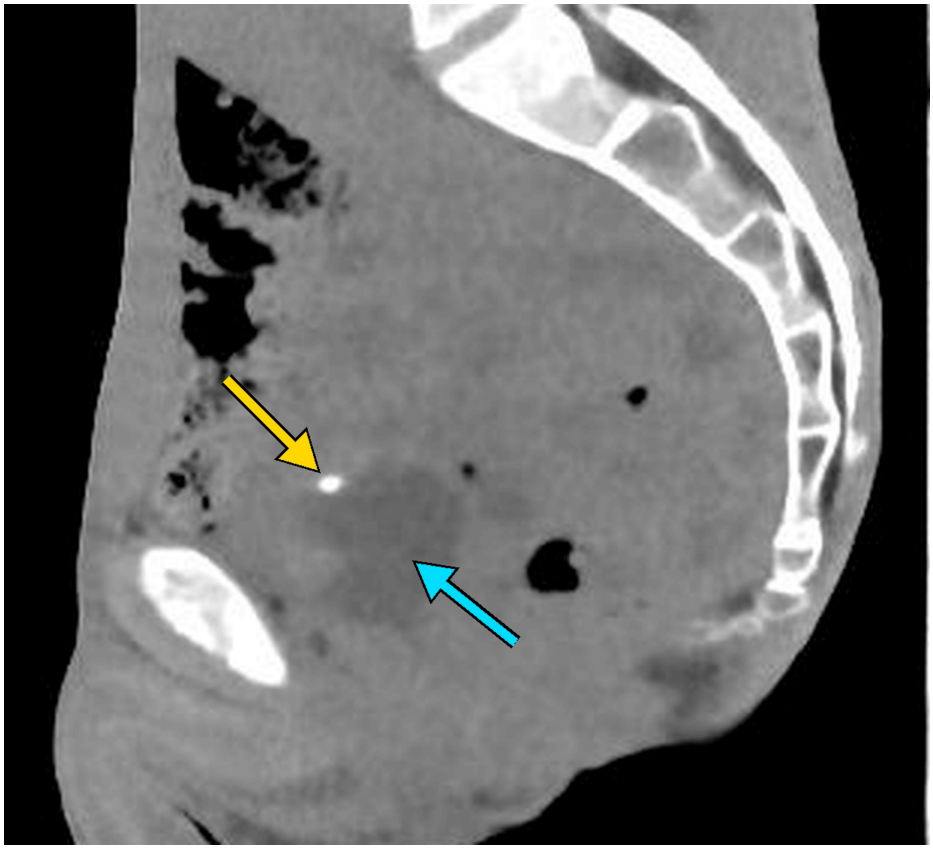


FIGURE 3: Sagittal computed tomography image showing a contracted bladder with urothelial calcification

Non-contrast computed tomography (CT) of the kidneys, ureters and bladder, performed eight months before this admission. The yellow arrow shows calcification along the anterior urothelium of the bladder. The light blue arrow shows the small, contracted bladder.

Management

Hyperkalaemia recurred at 6.0-6.5 mmol/L and was treated on three occasions in line with the trust guideline. Contributing factors were acute kidney injury, chronic urinary tract obstruction and a potassium-containing oral nutritional supplement. After repeated doses of intravenous calcium gluconate given as part of this treatment, adjusted calcium reached 3.30 mmol/L on day six, with a normal serum albumin of 38 g/L. Ionised calcium was normal the next day, and adjusted calcium fell to 2.49 mmol/L on day eight. No other cause of hypercalcaemia was identified, and parathyroid hormone and vitamin D were normal, so the rise was attributed to the intravenous calcium. The renal team advised a low-potassium diet, oral sodium bicarbonate 500 mg three times daily, a potassium binder, and a change to a low-potassium nutritional supplement. They also advised giving intravenous calcium only when there were electrocardiogram (ECG) changes.

Serum osmolality was 287 mOsm/kg with a urea of 20 mmol/L, giving an effective serum osmolality of about 267 mOsm/kg, consistent with hypotonic hyponatraemia. Urine osmolality was 204 mOsm/kg. The urine was not maximally dilute, so reduced kidney function may also have limited water excretion. Thyroid function was normal (thyroid-stimulating hormone 1.99 mU/L, free thyroxine 15.4 pmol/L), and a 9 am cortisol of 789 nmol/L made adrenal insufficiency unlikely. The endocrine team attributed the chronic hyponatraemia mainly to dilution from high fluid intake, and fluid was restricted to 1.5 litres per day. A urinary catheter was inserted on day eight, when creatinine peaked at 350 µmol/L, and kidney function then improved. The urology team advised that no urinary stent or nephrostomy was needed at this point because the acute kidney injury was improving, and a urology outpatient review has been booked.

Outcome

The patient's creatinine and potassium returned to normal before discharge. His sodium mildly improved but remained low. ALP fell to about 800 U/L and GGT also fell, although neither had returned to normal. He agreed to stop using ketamine and was discharged with follow-up from the drug and alcohol team. A repeat MRCP is planned in six months.

The timeline of events is summarised in Table 2.

Time	Event
Eight months before admission	CT KUB: small, contracted bladder with anterior urothelial calcification
Three months before admission	MRCP: narrowed intrapancreatic CBD with upstream dilatation
D1	Found collapsed; treated for suspected sepsis in the emergency department; admitted to the ICU; ALT 13 U/L, albumin and INR normal
D2	Ultrasound: CBD 10.4 mm, mild intrahepatic duct dilatation and bilateral hydroureteronephrosis
D3	AST 105 U/L
D4	MRCP: 25 mm intrapancreatic CBD narrowing, unchanged from three months earlier; moved from the ICU to the ward
D6	Creatinine 186 µmol/L; adjusted calcium 3.30 mmol/L after repeated intravenous calcium gluconate for hyperkalaemia
D7	Creatinine 170 µmol/L; ionised calcium normal
D8	Creatinine peak 350 µmol/L; urinary catheter inserted; adjusted calcium 2.49 mmol/L
D23	Discharged: creatinine and potassium normal, ALP about 800 U/L and GGT falling but not yet normal, ALT 16 U/L, sodium mildly improved but still low; drug and alcohol team follow-up
Six months after discharge	Repeat MRCP planned

TABLE 2: Timeline of events (D1 is day 1, the day of admission)

CT KUB: computed tomography of the kidneys, ureters and bladder; MRCP: magnetic resonance cholangiopancreatography; CBD: common bile duct; ICU: intensive care unit; ALT: alanine aminotransferase; INR: international normalised ratio; AST: aspartate aminotransferase; ALP: alkaline phosphatase; GGT: gamma-glutamyl transferase

Discussion

This case shows probable ketamine cholangiopathy occurring together with established ketamine uropathy, complicated by acute kidney injury, two electrolyte disturbances and a transient, treatment-related rise in calcium. Ketamine cholangiopathy was considered the most likely diagnosis, based on the pattern of findings and exclusion of other causes, without tissue sampling.

Recognising the pattern

Our patient's findings match published descriptions. Wong et al. found cholestatic liver injury in 9.8% of chronic ketamine users with urinary tract dysfunction, with bile duct injury on every biopsy taken [5]. Yu et al. reported fusiform CBD dilatation without obstruction in 69% of ketamine users who had imaging, and the degree of dilatation correlated with the duration of use [7]. Seto et al. described several MRCP patterns of ketamine-related cholangiopathy in drug users [8]. A systematic review of case reports found a mean patient age of about 26 years [10], the same as our patient. Our patient had all of these features: heavy long-term use, a young age, cholestasis without jaundice, a distal CBD narrowing and coexisting uropathy.

The mechanism is not fully understood. Proposed explanations include direct toxicity from ketamine and its metabolites excreted in bile, and chronic inflammation leading to fibrosis and stricturing [10,14]. In some patients, this process may progress to secondary sclerosing cholangitis [12].

Excluding other causes

The differential diagnosis of a distal CBD narrowing with upstream dilatation in a young adult is wide. Choledocholithiasis was unlikely because MRCP showed no stones or filling defects. Chronic pancreatitis and pancreatic head tumours were unlikely because the pancreas and pancreatic duct were normal. Cholangiocarcinoma and other malignant strictures were considered less likely because the narrowing tapered distally, was unchanged over three months, and CA 19-9 was not raised. However, CA 19-9 has limited accuracy, so a normal value does not exclude malignancy [11].

PSC is diagnosed on MRCP by multifocal strictures and dilatations, and only after secondary causes of sclerosing cholangitis have been excluded [11]. Our patient had a single distal narrowing without the multifocal pattern of PSC, and ANCA was negative. IgG4-related sclerosing cholangitis can also cause distal bile duct strictures. Serum IgG4 was not raised, although serum IgG4 alone has limited accuracy in separating this condition from PSC and cholangiocarcinoma [11]. Secondary sclerosing cholangitis has many causes, including critical illness, ischaemia, infection and toxins, and ketamine is now listed among the toxic causes [11]. Secondary sclerosing cholangitis due to critical illness was unlikely, because the bile duct changes were already present three months before this ICU admission. A choledochal cyst was also considered, because ketamine-related CBD dilatation has been mistaken for a choledochal cyst on imaging [9]. The heavy ketamine use, the coexisting uropathy and the stable distal narrowing favoured ketamine cholangiopathy.

Without tissue sampling, malignancy, IgG4-related disease and PSC cannot be completely excluded, and PSC cannot be ruled out on one stable scan. The gastroenterology team considered ketamine cholangiopathy highly likely. He had no jaundice or cholangitis, the narrowing was unchanged over three months and CA 19-9 was not raised, so ERCP and EUS were deferred, with a repeat MRCP at six months and outpatient gastroenterology review. ERCP carries recognised risks, including pancreatitis, bleeding, cholangitis and perforation [15].

Stopping ketamine

Stopping ketamine is the main treatment. Tkachuk et al. reported biliary changes that improved after abstinence [13]. However, Nyirenda et al. described a patient whose cholangiopathy progressed despite abstinence and who needed biliary stenting [14]. Early recognition and support to stop ketamine are therefore important, and follow-up imaging is needed.

Renal and electrolyte complications

The acute kidney injury was probably partly obstructive. He had bilateral hydroureteronephrosis and a small, contracted bladder, both recognised complications of ketamine uropathy [3,4], and kidney function improved after a urinary catheter was inserted on day eight, when creatinine peaked. The hyperkalaemia had several contributing causes: acute kidney injury, chronic urinary tract obstruction and potassium in an oral nutritional supplement. Repeated intravenous calcium gluconate was followed by a transient rise in adjusted calcium to 3.30 mmol/L, with a normal serum albumin and normal ionised calcium the next day. Current United Kingdom guidance suggests intravenous calcium for hyperkalaemia when ECG changes are present, and it also describes the use of potassium binders [16]. In line with this guidance, the renal team advised giving intravenous calcium only when ECG changes were present, and a potassium binder was started. The chronic hyponatraemia was hypotonic and was attributed mainly to dilution from high fluid intake, although the urine was not maximally dilute, so reduced kidney function may also have contributed. Thyroid dysfunction and adrenal insufficiency were unlikely, and the hyponatraemia was managed with fluid restriction. Sodium improved only mildly and remained low at discharge, so it needs ongoing follow-up.

Limitations

This report has several limitations. It describes a single patient, so the findings cannot be generalised, and a causal link between ketamine and the bile duct changes cannot be proven. The history of ketamine use was self-reported, and the exact duration and dose over time were unknown. No liver biopsy was performed, so the diagnosis rests on the clinical pattern, the imaging and the exclusion of other causes. EUS and ERCP with brush cytology were not performed, so a small malignant or IgG4-related stricture cannot be completely excluded, and PSC cannot be ruled out on one stable MRCP. The images of the earlier MRCP were not available, so the comparison relies on the radiology report. The long-term course after stopping ketamine is uncertain. Published cases describe both improvement [13] and progression despite abstinence [14]. Whether this patient stays abstinent, and whether his bile ducts improve, will only be known at follow-up, including the planned repeat MRCP. Fluid intake was not recorded, so the dilutional cause of the hyponatraemia could not be confirmed directly.

Conclusions

Ketamine use should be considered in young patients with unexplained cholestasis and a dilated or narrowed bile duct, especially in settings where recreational ketamine use is common, and clinicians should ask about it directly. Ketamine cholangiopathy can mimic PSC, bile duct stones or malignancy, and ketamine exposure alone does not exclude these other causes. Coexisting ketamine uropathy is an important clue, and it brings its own complications, including urinary tract obstruction, acute kidney injury and electrolyte disturbances. Recognising this pattern can support an individualised decision about invasive tests such as ERCP and EUS, with close imaging follow-up.

Stopping ketamine is the key treatment, and patients need support from drug and alcohol services. Because the long-term course after abstinence is uncertain, follow-up imaging is needed. Kidney injury and electrolyte disturbances need multidisciplinary care, and treatments such as intravenous calcium should be given only when they are clearly indicated.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Nishat Farjana

Acquisition, analysis, or interpretation of data: Nishat Farjana

Drafting of the manuscript: Nishat Farjana

Critical review of the manuscript for important intellectual content: Nishat Farjana

Disclosures

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