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Ketamine uropathy: Clinical experience in a high prevalence center

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Abstract

Objectives: Ketamine uropathy causes inflammatory changes to the urothelium, manifesting as significant lower urinary tract symptoms, small bladder capacity, and pelvic pain. Upper tract involvement and hydronephrosis can occur. Data from UK centers are limited, and no formal treatment guidelines exist.

Patients and Methods: All patients with ketamine uropathy presenting to our unit over an 11-year period were identified through operative and clinic lists, emergency presentations, and a prospectively collected local database. Demographic data, biochemical findings, imaging techniques, and both medical and surgical management were recorded.

Results: A total of 81 patients with ketamine uropathy were identified from 2011 to 2022; however, a large proportion presented from 2018 onwards. The average age at presentation was 26 years (interquartile range [IQR]: 27–34), 72.8% were male, and average follow-up time was 34 months (IQR: 8–46). Therapeutic interventions included anticholinergic medication, cystodistension, and intravesical sodium hyaluronate. Hydronephrosis was present in 20 (24.7%) patients and nephrostomy insertion was required in six. One patient underwent bladder augmentation surgery. Serum gamma-glutamyl transferase and length of follow-up were significantly higher in patients with hydronephrosis. Adherence to follow-up was poor.

Conclusions: We present a large cohort of patients with ketamine uropathy from a small town in the UK which is unusual. The incidence appears to be rising, in-keeping with increasing recreational ketamine use and should be of concern to urologists. Abstinence is a key aspect of management, and a multidisciplinary approach works best particularly as many patients are lost to follow-up. The development of formal guidance would be helpful.

KEYWORDS

drugs and addiction, gamma-glutamyl transferase (GGT), hydronephrosis, ketamine bladder, ketamine uropathy

1 | INTRODUCTION

Ketamine is an *N*-methyl-D-aspartate (NMDA) receptor antagonist and is widely used in healthcare as both a general anesthetic agent and for procedural sedation, producing a characteristic dissociative state with a clinically useful short half-life. It is highly lipophilic and after hepatic metabolism undergoes urinary excretion. Ketamine has gained popularity as a recreational drug for its psychoactive properties, particularly in young adults engaging in the club–drug scene. Ketamine uropathy is an uncommon but well-documented clinical entity causing inflammatory changes to the urothelium, manifesting as significant lower urinary tract symptoms, small bladder capacity, and pelvic pain. These symptoms can be severely debilitating and progress with continued use. Although also referred to as "ketamine bladder" or "ketamine cystitis" these changes can affect the entire urothelium including the upper urinary tract.

The first documented cases of ketamine uropathy were reported in 2007 in small case series both in Canada and Hong Kong.[1,2] Its incidence is directly related to geographical variation in recreational drug habits and as such much of the early clinical experience has come from areas of high usage, notably Eastern Asia and Hong Kong.[3–5] The UK has seen cases in several centers to date. The drug was reclassified in the UK in 2014 from class C to class B controlled drug due to growing concern over the toxic effects on the bladder[6]; however usage remains widespread with annual usage in 16- to 24-year-olds increasing to 3.2% in 2020 from 1.6% in 2015,[7] its highest figure to date.

The exact mechanism through which ketamine and its urinary metabolites damage the urothelium is not completely understood; however, evidence suggests there is a direct, toxic effect on urothelial cells leading to apoptosis. This may be precipitated by ketamine-induced increased intracellular calcium concentrations leading to mitochondrial stress and ATP release.[8] Other evidence suggests that IgE-mediated hypersensitivity and inflammation contributes, as does ketamine-induced loss of E-cadherin disrupting the bladder-epithelial barrier[9]; however, overall, the process is likely to be multifactorial. Typical histological features include denudation of the urothelium, chronic inflammation, and fibrosis. Another notable complication of long-standing ketamine use is the development of biliary dysfunction known as ketamine cholangiopathy, typically presenting with elevated serum liver enzymes and non-obstructive ductal dilatation.[10,11] Interestingly both elevated serum gamma-glutamyl transferase (GGT) and alkaline phosphatase (ALP) have shown to be predictive of upper urinary tract involvement and the development of hydronephrosis[5,12] and may have the potential to be a marker of disease severity.

The management of ketamine uropathy is complex and requires a multi-disciplinary approach. A one-stop service model has been shown to be effective[13,14]; however, national guidelines on the management of ketamine uropathy do not exist at present. Our unit comprises a urology area network in the North of England with a population of approximately one million patients. Despite our relatively small size we have noted a high prevalence of ketamine uropathy. The aims of this study are to present our experience with this patient group who often present significant clinical management challenges.

2 | PATIENTS AND METHODS

All patients with ketamine uropathy presenting from 2011 to 2022 were identified. This was achieved via screening of theater listings for patients undergoing cystodistension, clinic lists, emergency presentations, and a prospectively collected local database. Inclusion criteria required documented established recreational ketamine use in the presence of typical lower urinary tract symptoms as diagnosed by a urologist. Data extracted included baseline demographics, operative findings including bladder capacity at cystodistension, histology, radiological evidence of upper tract disease, biochemical results, and management strategies.

Continuous data were compared using an independent *t*-test and Fisher's exact test for categorical variables. *P* values <0.05 were taken to be significant. Statistical analysis was carried out using IBM® SPSS® Statistics version 28 software.

3 | RESULTS

In total, 81 patients with ketamine uropathy were identified from 2011 to 2022; however, there was a significantly higher incidence toward the end of this time period with 61 presenting from 2018 onwards (Figure 1). Baseline data are summarized in Table 1. Of the total cases, 71/81 (87.7%) originated from a single hospital. The average age at the time of data extraction was 30 years (IQR: 27–34) while the average age at presentation was 26 years (IQR: 23–29). More cases were diagnosed in males (59/81, 72.8%). Average follow-up time was 34 months (IQR: 8–46) with 21 (25.9%) patients lost to follow-up due to lack of attendance; however, many patients with longer total follow-up would frequently be lost to services for extended periods of time before being re-referred. Four patients were discharged owing to

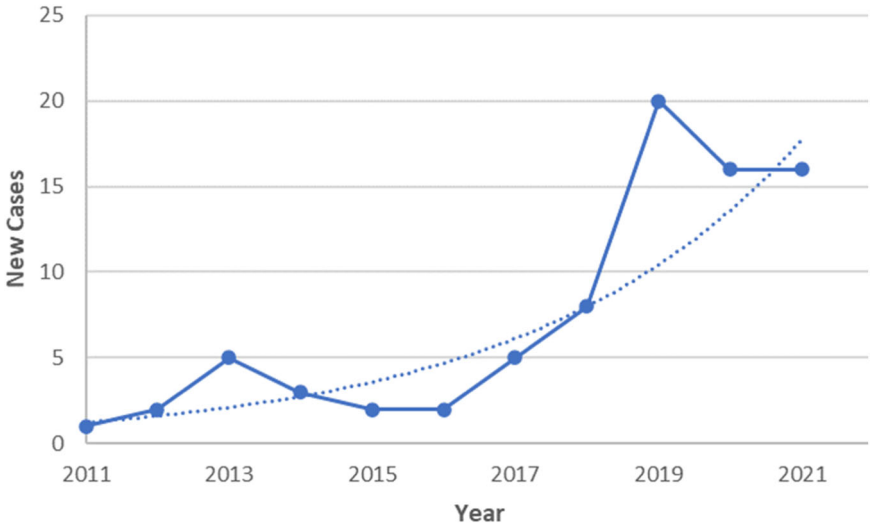

FIGURE 1 Incidence of new diagnoses of ketamine uropathy per year (2011–2021).

TABLE 1 Baseline demographic data for all patients with ketamine uropathy.

Variable	Value ($n = 81$)
Gender (male, percentage)	59 (72.8%)
Age (median, IQR)	30 (27–34)
Age at presentation (median, IQR)	26 (23–29)
Follow-up time, months (mean, IQR)	34 (8–46)
Serum GGT level, U/L (mean, IQR)	575 (83–579)
Serum ALP level, U/L (mean, IQR)	123 (31–170)
eGFR, mL/min/1.73 m^2 (mean, IQR)	82 (90–90)
Hydronephrosis (percentage)	20 (24.7%)
Bladder capacity at cystodistension, mL (mean, IQR), $n = 49$	260 (162–350)

Abbreviations: ALP, alkaline phosphatase; eGFR, estimated glomerular filtration rate; GGT, gamma-glutamyl transferase; IQR, interquartile range.

recurrent active use and only four were discharged due to improvement in symptoms. Daily usage of ketamine was not well documented, however, varied between 1.75 and 7 g. A diagnosis of mental health illness such as anxiety/depression was recorded in 25/81 (30.9%) patients.

3.1 | Patient symptoms

Typical symptoms reported by patients with ketamine uropathy were predominantly storage lower urinary tract symptoms and pelvic pain. Urinary frequency, urgency, and a small capacity bladder were common. Some experienced intermittent visible hematuria. Pelvic pain was a common feature and was severe in cases despite analgesia. Symptom improvement with abstinence was variable with some showing a near-complete resolution while others had persistent, debilitating symptoms despite self-reported abstinence. Incontinence or bladder outlet obstruction was not reported.

3.2 | Medical therapy

Uptake of anticholinergic or β-3 agonist prescribing was high with 62/81 (75.3%) of patients prescribed at least one medication from these classes while 31/81 (38.3%) were on dual therapy, most commonly Solifenacin and Mirabegron. Intravesical sodium hyaluronate was used in 11 patients and 3 received Botulinum therapy. Response to oral therapy was generally good with some symptomatic improvement; however, in the presence of continued ketamine use this was significantly limited.

3.3 | Upper tract involvement and imaging

Overall, 70/81 (86.4%) patients received upper urinary tract imaging. This was using ultrasound alone in 53/81 (65.4%) and 17/81 (21%) were further evaluated with computed tomography (CT) imaging. Radioisotope renography (Tc-99m MAG3 renogram) was used in three patients; however, it was noted to be poorly tolerated in some cases. Hydronephrosis was present in 20/70 (28.6%) of patients who received upper tract imaging and was bilateral in 16/20 (80%). Typical CT findings were of a small, contracted bladder with bilateral vesicoureteric reflux and hydroureteronephrosis down to the vesicoureteric junction (VUJ) (Figure 2); however, four patients had evidence of stricture formation (Figure 3). A univariate analysis of demographic and biochemical

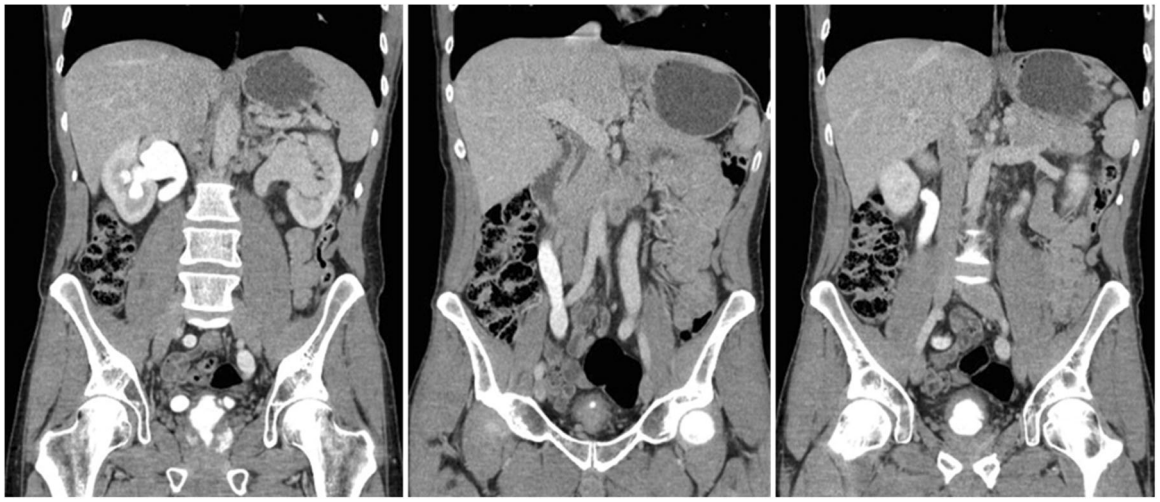

FIGURE 2 Computed tomography with urinary contrast in a 38-year-old male highlighting typical features of ketamine uropathy with bilateral hydroureteronephrosis secondary to reflux from a small-capacity thick-walled bladder.

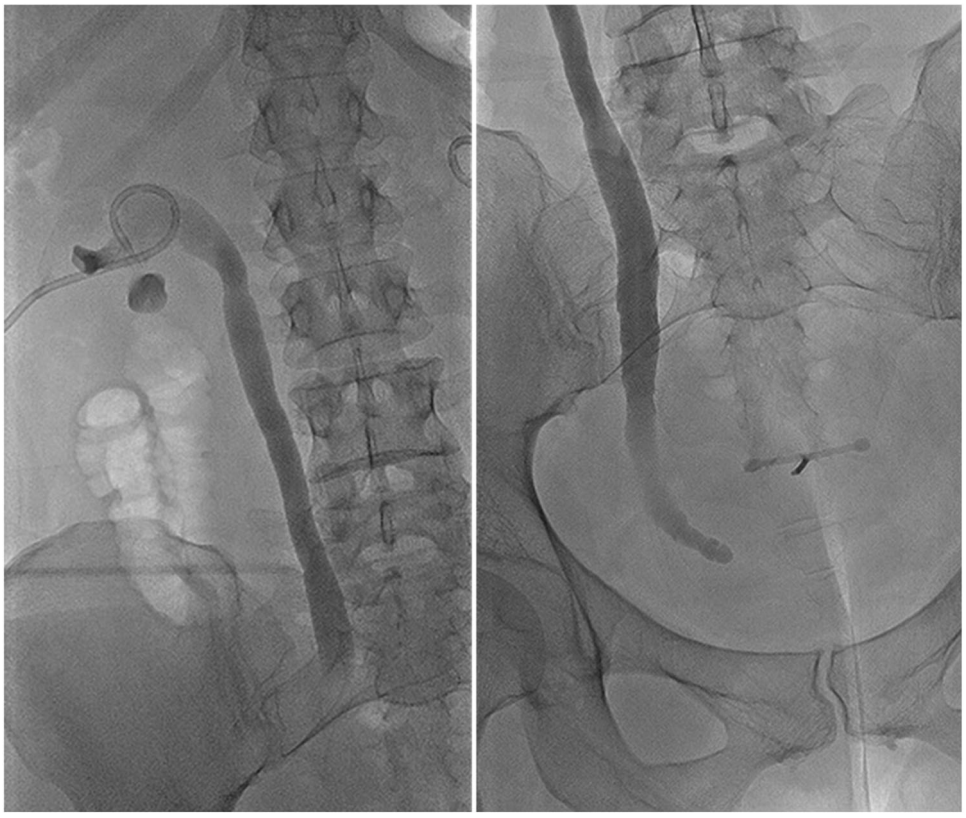

FIGURE 3 Left prone nephrostogram in a 34-year-old female with a 9-year history of ketamine abuse showing hydronephrosis with a significantly dilated ureter down to the level of the vesicoureteric junction where a stricture prevents contrast passage into the bladder.

factors comparing patients with and without hydronephrosis is shown in Table 2. Total follow-up time ($p = 0.003$), average serum GGT ($p = 0.012$), and alanine transaminase ($p = 0.021$) were all significantly higher in patients with hydronephrosis. Average renal function was worse in the hydronephrosis group ($p = 0.001$).

3.4 | Operative interventions

Flexible cystoscopy was almost universally poorly tolerated and as such was not offered routinely. There were 60 patients who underwent cystodistension and maximum bladder capacity was recorded in 49/60 (81.7%) with an

TABLE 2 Univariate analysis comparing patients with and without hydronephrosis.

Variable	No hydronephrosis ($n = 50$)	Hydronephrosis ($n = 20$)	p Value
Gender—male	38 (76%)	11 (55%)	0.076
Age	29.8 ± 4.37	31.2 ± 4.93	0.131
Age at presentation	26.2 ± 4.36	27.4 ± 4.44	0.152
Follow-up time (months)	27.2 ± 22.9	56.2 ± 39.9	0.003
Serum GGT (U/L)	339 ± 320	1188 ± 1526	0.012
Serum ALT (U/L)	105 ± 102	197 ± 180	0.021
eGFR (mL/min/1.73 m^2)	88.8 ± 5.59	65.4 ± 30.4	0.001
Bladder capacity (mL)	270 ± 126 ($n = 29$)	235 ± 123 ($n = 13$)	0.202

Note: Figures represented as mean ± SD or a percentage value for categorical data.

Abbreviations: ALT, alanine transaminase; eGFR, estimated glomerular filtration rate; GGT, gamma-glutamyl transferase.

average of 260 mL (50–550 mL). Typical macroscopic appearances were of a small capacity, friable, and fibrotic bladder. Microvascular hemorrhage was common. Response to cystodistension varied with some reporting major improvement in symptoms while some felt it was of little benefit. Two extraperitoneal bladder perforations occurred at cystoscopy without biopsy which were managed conservatively, although one required a short period of organ support from intensive care, which is an unusually adverse response to an extraperitoneal bladder perforation. A second cystodistension was performed in 21 patients and a small number received this on further occasions.

Nephrostomy insertion was required in six patients for hydronephrosis and renal impairment, four were bilateral and two were unilateral. The average improvement in eGFR after nephrostomy insertion was 25.4 mL/min/1.73 m^2 (0–55). Two of these patients have had bilateral nephrostomies in place for over 2 years while awaiting an appropriate time for reconstructive surgery. One patient underwent subtotal cystectomy and substitution cystoplasty and four patients were referred for consideration of reconstructive surgery. Other surgical procedures performed included an emergency cystoscopy and washout for hematuria, cystolitholapaxy, and ureteroscopy and biopsy for a ureteric lesion. One patient has formed calcium phosphate (brushite) bladder stones recurrently.

3.5 | Histology

Histology was available from 18 patients: 17 from biopsy at cystoscopy and 1 from subtotal cystectomy. All showed changes in the urothelium in-keeping with ketamine uropathy including extensive urothelial denudation, chronic inflammation, and reactive atypia. Extensive replacement of the lamina propria with fibrous tissue and fibroblastic proliferation was also seen. There was one case of squamous metaplasia and two showed florid proliferation of von Brunn nests. There were no cases of carcinoma in situ (CIS) or malignancy.

3.6 | Mortality

Over the study period two (2.5%) patients died, both related to complications of ketamine use. A 31-year-old male with an 8-year history of ketamine use and longstanding bilateral hydronephrosis who was still actively using developed pyelonephritis, acute renal failure, and sepsis leading to renal abscesses and ultimately multiple organ failure. The second, a 35-year-old male who had been referred for reconstructive surgery, also still actively using, developed progressive ketamine cholangiopathy and died due to fulminant hepatic failure.

4 | DISCUSSION

We present what is to our knowledge the largest reported cohort of patients with ketamine uropathy in the United Kingdom to date. This is unusual given that most cases came from a single district general hospital in a town with a population of approximately 244 000 residents. Previous UK studies have been reported in larger urban areas and tertiary referral centers. It is unclear what socioeconomic factors have led to this high prevalence, although local deprivation is higher than the England average.[15] The area is 97% Caucasian. There appears to be a relationship between duration of abuse and

development of hydronephrosis and declining renal function. Ketamine continues to increase in popularity among young people with the lifetime usage rate in 16–24 year-olds rising to 5.6% in 2020.[7]

During the initial assessment of a patient with ketamine uropathy, key features to obtain from the patient history include an estimation of duration and amount of use, as well as assessment of symptom severity such as the pelvic pain and urgency/frequency (PUF) score and a measurement of functional bladder capacity.

Biochemical markers such as serum creatinine, GGT, and ALP are helpful in on-going assessment, predicting hydronephrosis and for highlighting active ketamine use.[5,11] In our cohort we have found that GGT in particular was significantly higher in patients with hydronephrosis and is also our opinion that it is raised during periods of active use although this was difficult to confirm retrospectively. Ketamine cholangiopathy is an emerging pathology and one which needs further investigation; however, abstinence is likely to be important in limiting progression.

Initial diagnostic workup should include upper tract imaging to assess for the development of hydronephrosis. Ultrasound is widely used as a first-line investigation for hydronephrosis; however, when present this requires further imaging to differentiate between vesicoureteric reflux and ureteric stricture formation. This was most commonly performed using CT. Radioisotope imaging such as a MAG3 renogram is an alternative although this can be poorly tolerated by patients with ketamine uropathy affecting its diagnostic accuracy. As with flexible cystoscopy, cystogram and urodynamic studies are also poorly tolerated and of limited use,[4] hence were not routinely offered.

Abstinence is key in limiting the progression of urothelial damage and patient symptoms; however, this is not always easy to achieve as relapse rates are high. Therefore, the involvement of local addiction services is essential. Symptomatic improvement and even resolution of hydronephrosis have been reported with abstinence in some cases.[5,16] In our patients hydronephrosis was significantly more common in those with longer follow-up and total duration of use, highlighting the need for help with long-term and sustained abstinence. Many patients with severe pelvic pain report needing the analgesic effects of ketamine to cope with their symptoms which leads to an unfortunate cycle of disease progression. Ideally, a specialist pain service should be involved to initiate opioids and gabapentioids, if necessary, in a patient group at risk high risk of addiction and further substance misuse.

We report a more frequent use of cystodistension than other studies which is due mainly to historical departmental practices before the formation of our urology area network. Responses varied significantly between patients, and it is difficult to predict which patients will benefit. All biopsies taken at cystodistension confirmed ketamine cystitis with no cases of neoplasia and therefore given the increased perforation risk we do not recommended routine biopsy in the absence of an obvious lesion. We believe the only indications for diagnostic cystoscopy are to investigate visible hematuria as there are two case reports of malignancy within the bladder in patients presenting with ketamine cystitis[17,18]; however, the histology was atypical and unclear whether related to ketamine use.

Intravesical treatments which have been trialed include glycosaminoglycan (GAG) layer replacement therapy (e.g., sodium hyaluronate) and injection of Botulinum toxin A (Botox). These were used in relatively few patients in our cohort; however, good short-term relief of symptoms, particularly with Botulinum therapy, was reported. It has been suggested that Botulinum use has been associated with short-term symptomatic improvements,[19] although this is not universal and robust evidence regarding long-term effects is lacking at present. Its use is likely to be futile with concurrent ketamine use and it is our departmental policy to confirm 6 months of cessation with urinary testing before offering surgical intervention such as cystodistension or Botulinum therapy.

Reconstructive surgery may ultimately be necessary for refractory cases. Suitable patient selection presents a dilemma as the incidence of surgical complications in this group can be high[20]; however, this varies by study.[21] For cases where bladder reconstruction or replacement is offered, it is our practice to insist on a period of abstinence of at least 6 months (confirmed by urinary testing) as rates of significant perioperative complications are high, as is relapse postoperatively causing progressive upper tract disease.[22] Due to the risks of bladder augmentation in ketamine users such as ureteric strictures and anastomotic leak, suitable patient selection is paramount. Only one patient in our study period underwent subtotal cystectomy and substitution cystoplasty. In severe cases of upper urinary tract disease renal auto-transplantation has been performed.[23] At the time of writing, three patients in our cohort are being considered for reconstructive surgery; however, continued ketamine usage remains a barrier to definitive urinary tract management.

When managing a new patient presenting with ketamine uropathy we recommend a stepwise approach, primarily focusing on ketamine cessation and guiding patients to local drug and addiction services, counseling, and chronic pain services. It is important to stress that

disease progression is likely and minimal improvement in symptoms despite medical therapy is to be expected with continued active use. Initial medical therapy can be commenced with anticholinergic/β-3 agonist titrated to response. We now screen for active ketamine use from presentation with urinary testing and only consider offering invasive treatments such as cystodistension or Botox, if required, after 6 months of abstinence. Reconstructive surgery should be reserved for refractory cases however requires careful patient selection.

A key finding from our experience managing ketamine uropathy is the poor patient compliance with follow-up. We found that over the duration of a patient's total follow-up time, with an average of 34 months, there would be frequent periods of nonattendance at clinic appointments sometimes spanning months to years before re-presenting with symptoms. This is a limitation of this study as in most cases individual follow-up was not continuous and at the end of the study period 25.9% of patients had been lost to follow-up. The lack of validated patient symptoms scores used to categorize lower urinary tract symptoms is a weakness but due to the length of study period we were unable to assess this consistently. A further limitation is that we have been unable to verify abstinence during follow-up as toxicology screening is usually reserved for those being considered for surgical intervention. It is our experience that cycles of abstinence and relapse are common and patient-reported abstinence is not always accurate. Due to the unusually high incidence of ketamine uropathy in our unit we continue to record new cases prospectively.

5 | CONCLUSIONS

We report our experience of managing a large and increasing number of patients with ketamine uropathy. The incidence appears to be increasing and should be of concern to urologists. At present the best treatment strategy has not been clearly defined and the development of formal guidance would be beneficial, particularly for centers less familiar with ketamine uropathy. Abstinence remains a key aspect of management and a multidisciplinary approach works best.

AUTHOR CONTRIBUTIONS

Data collection: George Sturgess, Robin Shepherd, and Alison Downey. *Initial drafting of manuscript*: George Sturgess. *Manuscript review*: Ian Beckley, Robin Shepherd, and Alison Downey.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

Data are available upon request.

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