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Ketamine Bladder: A Cause of Obstructive Uropathy in a Young Patient

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Abstract Ketamine-associated cystitis is growing in prevalence as the availability and use of illicit drugs increases. Delayed diagnosis and management can lead to poor patient outcomes including irreversible renal impairment. Early diagnosis, ketamine cessation and management of complications is therefore crucial. There are only a few case reports in the literature describing this condition. We report an additional case of a young patient with ketamine-associated ulcerative cystitis causing upstream obstructive uropathy.

Keywords: *ketamine, cystitis, uropathy*

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1. Introduction

Ketamine is an anaesthetic drug which can be misused due to its dissociative effects. Over 25% of patients who use ketamine recreationally have reported urinary tract symptoms with positive correlation seen with drug dose and frequency [1,2]. Ketamine misuse can cause problems with urine storage and voiding and patients often present with lower urinary tract symptoms such as painful haematuria, urinary urgency and frequency. There are only a few case reports in the literature describing this condition which remains poorly understood. We report an additional case.

2. Case Presentation

We present a case of a 21 year old man who presented to his General Practitioner with a history of severe lower urinary tract symptoms. Past medical history included gastro-oesophageal reflux, recurrent urinary tract infections and illicit drug use (ketamine). Blood tests for renal function were normal. Ultrasound imaging demonstrated mild dilatation of the left renal pelvis but otherwise normal appearances of both kidneys. The patient was unable to fill his urinary bladder despite multiple attempts to drink fluids in the ultrasound department. The bladder volume was 45 mls after filling and the patient complained of severe urgency to void. The bladder appeared abnormally shaped with a 22 x 17mm area of thickening in the anterior wall (Figure 1).

The patient was referred to the Urology team for further assessment. Rigid cystoscopy demonstrated very small bladder capacity (<100mls) with diffuse bleeding which prevented distension and adequate examination. CT urogram was therefore requested for further investigation. This demonstrated severe bilateral hydroureteronephrosis with enhancing and thickened ureters and periureteric fat stranding (Figure 2). There was no evidence of urinary tract calculi. The bladder appeared small and contracted with enhancing mucosa and surrounding fat stranding (Figure 3). These findings were in keeping with ketamine-associated ulcerative cystitis with associated bilateral hydroureteronephrosis. The patient was advised to stop ketamine usage and follow-up ultrasound demonstrated resolution of the hydronephrosis.

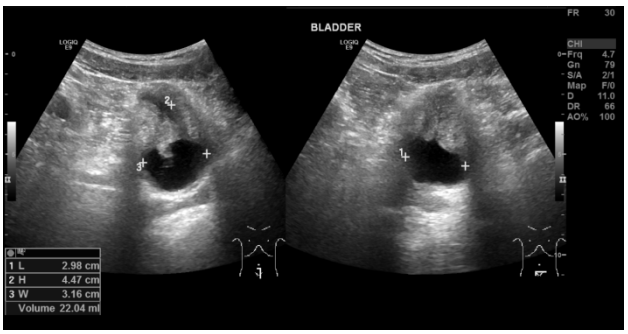

Figure 1. Ultrasound demonstrates thickened urinary bladder wall and reduced bladder capacity

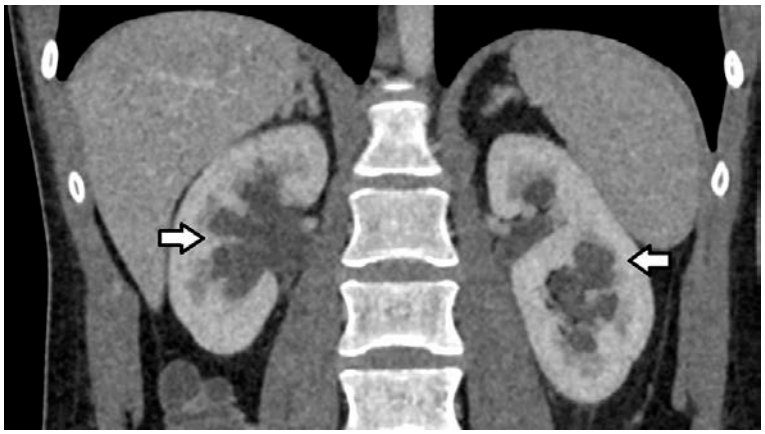

Figure 2. Coronal CT image of the abdomen with contrast demonstrates bilateral hydronephrosis

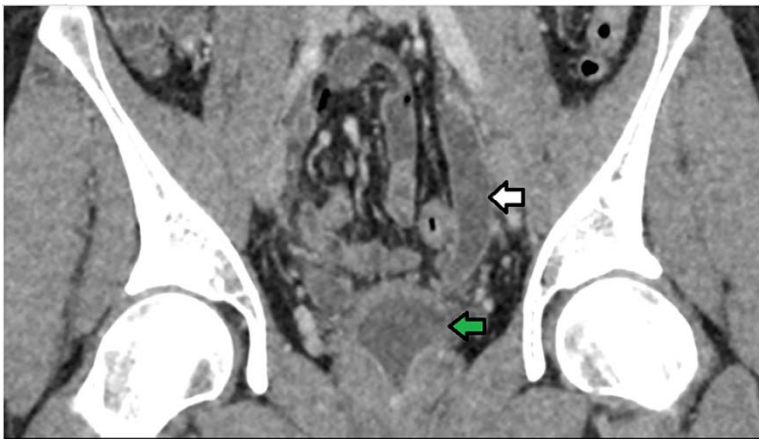

Figure 3. Coronal CT image of the pelvis with contrast demonstrates a thickened urinary bladder (green arrow) and dilated left ureter (white arrow)

3. Discussion

Ketamine-associated cystitis is a relatively new disease which is probably under-diagnosed due to lack of clinical awareness and reduced presentation of patients for medical advice [2]. Our patient's symptoms and investigation findings were typical for long-term ketamine use. The exact pathophysiological of ketamine-associated urinary tract disease is unknown however there are several possible mechanisms [3]. These include: direct toxic effects of ketamine on the renal and bladder interstitial cells leading to structural damage, microvascular endothelial cell injury resulting in ischemia and abnormal neovascularization and autoimmune-induced submucosal oedema [3,4].

Urodynamic studies show detrusor overactivity and reduced bladder adaptability [5]. Cystoscopy often demonstrates reduced bladder capacity with inflammatory changes including neovascularization and ulceration [1]. The reduced bladder compliance can cause vesicoureteral reflux with subsequent hydroureteronephrosis as seen in our patient. Other possible causes of hydroureteronephrosis include papillary necrosis resulting in ureteric obstruction from sloughed papillae and immune-mediated ureteric fibrosis [4].

Treatment of ketamine-associated urinary tract disease depends on the types of complications and disease severity. The mainstay of treatment is cessation of ketamine use and is usually associated with improvement in symptoms [4,6]. Other treatment options are similar to those of interstitial cystitis. Pharmacological management includes non-steroid anti-inflammatory drugs, anticholinergics and pentosan polysulfate [1]. Intravesical instillation of hyaluronic acid solution or botulinum toxin-A bladder injection with hydrodistention may also help improve symptoms [1,5]. Surgical management including bladder augmentation may be necessary in severe cases. Patients with hydronephrosis may require nephrostomy or ureteric stent insertion to preserve renal function.

4. Conclusion

Ketamine-associated cystitis is growing in prevalence as the availability and use of illicit drugs increases. It can progress to involve the upper urinary tract and may cause irreversible renal impairment if it remains untreated. Early diagnosis, ketamine cessation and management of complications is therefore crucial. Clinicians should be aware of the disease and consider it as a possible cause of unexplained lower urinary tract symptoms particularly in young patients. Once the diagnosis is confirmed, a multidisciplinary approach is needed to manage both the urological and psychosocial aspects of the disease to improve patient outcomes.

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