Bladder Calcification Secondary to Ketamine

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INTRODUCTION

Ketamine is a dissociative anesthetic agent, able to induce and maintain anesthesia.[1] Effects on consciousness and propensity to cause hallucinations and out of body experiences have led to the use of ketamine as a recreational drug in recent years. Its use among 16 to 25 year olds is currently estimated at 1.9% in the UK.[2]

Ketamine-associated bladder dysfunction, including lower urinary tract symptoms and pathology secondary to ketamine, is a newly described phenomenon.[3,4] Case reports suggest that young patients who regularly abuse ketamine may be prone to ulcerative cystitis, detrusor overactivity, reduced bladder capacity, thickened bladder wall, hydronephrosis, and hematuria.[3-6] We report a case which has demonstrated the additional finding of the bladder calcification.

CASE REPORT

A 28-year-old white British man presented with a 5-year history of pelvic pain and urinary frequency. He was voiding hourly during the day and a number of times during the night. He admitted regularly using ketamine and ecstasy on approximately a weekly basis for the past 6 years. His past medical history included depression and mild asthma, for which he did not take inhalers. Of note, he had never traveled outside Europe.

His regular medication consisted of trazodone hydrochloride capsules (for depression) and alimemazine 10 mg once a day (for insomnia). He had previously found no benefit from solifenacin succinate.

Urine dipstick showed a trace of blood and protein. Midstream clean catch urine was ster-

ile. Complete blood count was normal, as was renal function with an estimated glomerular filtration rate of >90 mL/min/1.73 m^2. C-reactive protein was slightly elevated at 16 mg/dL, and liver function tests (LFTs) were mildly raised (aspartate aminotransferase 47, alanine transaminase 56, and gamma glutamyl transpeptidase 111 IU/L), and corrected calcium was within the normal range at 2.22 mmol/L. A Chlamydia screen was negative.

Ultrasonography of the bladder revealed a reduced bladder capacity of 150 mL with the suggestion of a bladder stone. A computed tomography urography also demonstrated a 16 × 10 mm bladder calculus with no evidence of other urinary tract pathology (Figure 1).

Cystoscopy revealed a small non-occlusive prostate, open bladder neck, and no evidence of a urethral stricture. Both ureteral orifices were dilated, and the bladder was generally inflamed with two areas of exophytic calcification on the anterior wall, which were removed with biopsy forceps. Maximal bladder capacity under anesthetic was 300 mL (Figure 2).

Biopsies of the bladder wall demonstrated chronically inflamed and congested transitional mucosa, with surface ulceration and focal calcification (Figure 3). There was no evidence of urothelial atypia as seen in previous case series.[7] Chemical analysis of the calcified deposits demonstrated them to be 100% calcium phosphate.

Figure 1. Computed tomography scan showing bladder calcification.

Figure 2. Bladder calcification at cystoscopy.

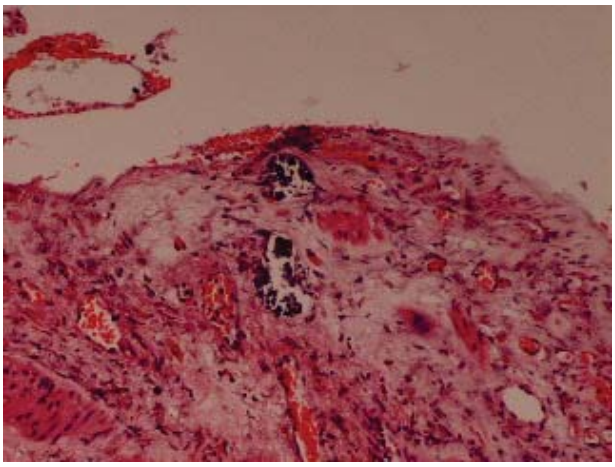

Figure 3. Histology showing focal calcification and total ulceration of urothelium (×20).

DISCUSSION

It seems highly likely that this patient's symptoms were secondary to ketamine-induced pathology in the bladder. Unlike previous cases reported, he displayed macroscopic and microscopic calcification of the anterior bladder wall.

With the urological side effects of ketamine recently being described, it is likely that there are a large number of symptomatic patients who remain undiagnosed due to lack of knowledge about the urological side effects of ketamine. With the increasing use of ketamine as a recreational drug,[2] it seems only likely that subjects with ketamine-associated bladder dysfunction will increase.

Ketamine is also being linked with abnormal LFTs and bile

duct pathology, which may explain our subject's deranged LFTs. However, no radiological evidence was seen on computed tomography and this was not investigated further.[8,9]

Due to the illegal nature of ketamine abuse, it is unlikely that patients will volunteer that they have been using this substance. It is therefore crucial that recreational ketamine use is specifically asked about when taking the history in order to identify these patients early. Early identification and patient education to encourage abstinence from this substance may prevent more serious damage to the renal tract.

Furthermore, it is important that researchers into the wider uses for ketamine in healthcare, such as chronic pain relief[10] and catheter-related discomfort,[11] also appreciate the potential serious urological side effects of chronic ketamine use.

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CONFLICT OF INTEREST

None declared.

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