CASE REPORT

Acute kidney injury requiring haemodialysis following ingestion of mephedrone

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SUMMARY

A 25-year-old man was found to have acute kidney injury (AKI) following ingestion of mephedrone. He presented to this local emergency department with worsening bilateral loin pain. He became oligoanuric, serum creatine peaked at 1214 $\mu\text{mol/l}$ and he required several sessions of haemodialysis before kidney function began to improve. The mechanism of AKI and legal aspects of the use of mephedrone are discussed.

BACKGROUND

Mephedrone has become an increasingly common recreational drug, with an unknown prevalence of adverse effects. There is little in the literature about mephedrone use associated with acute kidney injury (AKI), the pathophysiology of why it develops and the prognosis for renal recovery is unknown. We describe a case of AKI attributed to mephedrone, to highlight its potential dangers and promote awareness.

CASE PRESENTATION

A 25-year-old gentleman presented to this local emergency department after waking early in the morning with worsening severe bilateral loin pain. He admitted to ingestion of a large amount of mephedrone, consuming speed (methamphetamine), diazepam and snorting cocaine over the previous 3 days. He had used these substances intermittently over the past 5 years, but had never taken as much mephedrone before, or on consecutive days. He denied any current or previous injection of drugs. He had no other relevant medical background, and no family history of renal disease. He said he had passed no urine that day. Upon examination he had bilateral renal angle tenderness. The rest of the systemic examination was unremarkable. He was haemodynamically stable with a blood pressure of 140/70 mm Hg, and heart rate 90 bpm. His temperature was 37.0°C. He passed only 10–20 ml urine over the first 24 h.

INVESTIGATIONS

His admission blood tests showed a serum creatine concentration of 654 $\mu\text{mol/l}$, with a potassium of 4.7 mEq/l. Bicarbonate was low at 16 mmol/l. Haematology and clotting were normal, but his creatine kinase (CK) was elevated at 1183 U/l. Urinalysis showed 2+ of blood and protein. Renal ultrasound scan excluded hydronephrosis and demonstrated normal-sized kidneys. A CT renal angiogram revealed diffuse swelling of both kidneys, without any evidence of renal infarction.

Antineutrophil cytoplasmic antibody and other autoantibodies were not detected.

DIFFERENTIAL DIAGNOSIS

Initial thought owing to the amphetamine-like drug abuse by patient was whether the patient had developed renal infarcts or rhabdomyolysis owing to poor fluid intake coupled with drug ingestion. However, the initial CK level was only modestly elevated, and it started to decrease after initial presentation despite deteriorating renal function.

TREATMENT

He was initially given opioid analgesia, intravenous normal saline and a dose of co-amoxiclav to cover against pyelonephritis (later stopped). Sodium bicarbonate was administered in view of the metabolic acidosis. The oligoanuria persisted and repeat blood tests showed deteriorating renal function, with serum creatine concentration peaking at 1214 $\mu\text{mol/l}$. In view of the poor urine output and worsening renal function he was started on haemodialysis.

OUTCOME AND FOLLOW-UP

After two sessions of dialysis (5 days into admission), his renal function began to recover spontaneously, and he started passing more than 30 ml/h of urine. His serum creatine concentration decreased by approximately $-200 \mu\text{mol/l}$ daily, and was 335 $\mu\text{mol/l}$ on discharge. The patient did not have a renal biopsy as his renal function improved spontaneously. When he was seen in the clinic 2 days post discharge his renal function had improved further, with a serum creatine concentration of 233 $\mu\text{mol/l}$.

DISCUSSION

Mephedrone (4-methylmethcathionone) is a synthetic cathinone, also known as 'meow meow', 'MCat', 'snow' and 'bubbles'.^{1 2} It is one of a group of synthetic cathinones structurally similar to a naturally occurring alkaloid cathinone, in *Catha edulis* (a khat plant).³ It was made an illegal class B drug in the UK from April 2010, owing to a rapid increase in its use and links to deaths.^{3–5} It is often obtained as a white powder, marketed as 'plant food' or 'bath salts', via the internet or 'head shops'.^{3 5}

Information on the type and prevalence of adverse effects of mephedrone is limited to results of drug surveys, poison information services and case reports.¹ Many reports are not confirmed by toxicology results, since many laboratories do not have the facilities to process an assay, and a result would unlikely be back in time to assist

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management.¹ As in this case, mephedrone is often not used in isolation, and so the use of other stimulant drugs may contribute to its effects.¹ Mephedrone may be taken by various routes of administration most commonly by nasal insufflation and oral ingestion, but also by intravenous/intramuscular injections and rectally.^{6–8} Information from sources described hereinbefore suggest its stimulant effects are similar to those of other drugs such as amphetamine, cocaine and ecstasy.⁹ Reported side effects include sweating, nausea and vomiting, headache, palpitations, agitation, insomnia and cool extremities.^{2–6}

There are two cases in the literature of AKI following the use of 'bath salts' (combination of mephedrone and another stimulant cathinone-derived substance called methylenedioxypyrovalerone),^{10–11} although the patient required dialysis treatment in only one of these.¹¹ As in this case, rhabdomyolysis may have contributed, but was unlikely to explain the degree of AKI because the CK concentration was not very high. Adebamiro *et al*¹⁰ speculated that these synthetic cathinones cause renal arteriolar vasospasm, leading to renal ischaemia and acute tubular necrosis (ATN). This mechanism has been proposed by others, mainly based on the close structural and pharmacological relationship between mephedrone and substances such as ecstasy and amphetamine.^{11–12} Mephedrone may also be directly nephrotoxic, leading to ATN.¹⁰ All cases reported have either developed mild AKI or have recovered kidney function with dialysis treatment.¹¹ Regunath *et al*¹¹ suggested that the low molecular weight of mephedrone would permit its removal by dialysis.

It is unclear what changes the legal status have had or will have on the prevalence of use of mephedrone and other synthetic cathinones.² The pattern of recreational drug use has changed over the past 10 years or so, and will likely continue to do so.^{13–14} Owing to most information on mephedrone toxicity being derived from self-reporting and case reports, the longer term effects of its use are from speculation using other structurally similar substances.³ It is important for healthcare professionals to be aware of current drug use patterns and the potential medical consequences.

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Learning points

- Mephedrone use may be a rare cause of acute kidney injury.
- Identifying drug use in patients is vital, especially as no assay is commonly used to identify mephedrone.
- Healthcare professionals need to be vigilant on the changing patterns of drug use and their potential health consequences.

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