Case Report

Necrotizing renal vasculopathy resulting in chronic renal failure after ingestion of methamphetamine and 3,4-methylenedioxymethamphetamine (‘ecstasy’)

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Introduction

A case is described of chronic renal failure developing after the use of oral methamphetamine and 3,4-methylenedioxymethamphetamine (‘ecstasy’) with histologically proven necrotizing angiitis confined to the kidney.

Case

A 30-year-old man presented having been unwell for 1 week with symptoms of lethargy, nausea and vomiting, breathlessness and a reduction in urine output. At the time of admission, he was on regular medication with temazepam and the antidepressant fluoxetine. He subsequently admitted that 10 days prior to admission he had taken amphetamine and 3,4-methylenedioxyamphetamine ‘ecstasy’ tablets at a party. He denied ever using intravenous drugs and there was no clinical evidence to doubt this.

When examined on admission he was apyrexial. He was tachycardic, pulse 110, and hypertensive with a blood pressure of 190/100. His heart sounds were normal but he had signs of fluid overload with a raised jugular venous pressure and crepitations over both lung fields.

Baseline investigations revealed that he was in renal failure, with Na 134 mmol/l, K 5.7 mmol/l, urea 61.3 mmol/l, creatinine 1562 μmol/l, bicarbonate 22 mmol/l, glucose 3.7 mmol/l, C-reactive protein 21 mg/l, haemoglobin 8.9 g/dl, white cell count 14.4 × 10⁹/l, platelets 102 × 10⁹/l, INR 1.1. Hepatitis B and C serology were negative. Anti-neutrophil cytoplasmic antibody titres (p and c) were negative. Chest X-ray confirmed pulmonary oedema. He commenced haemodialysis via a temporary double lumen subclavian catheter. A subsequent ultrasound scan showed two normal sized kidneys and he underwent ultrasound-guided percutaneous renal biopsy.

An empirical course of oral steroids (initial dose prednisolone 60 mg daily) was given but there was no improvement in renal function and he remained on regular haemodialysis. His compliance with fluid and dietary restrictions was poor and he had repeated emergency admissions with fluid overload and hyperkalaemia necessitating extra dialysis. His ECG, which initially had been normal, subsequently showed changes of marked left ventricular hypertrophy and strain.

He attended for a regular haemodialysis session and collapsed with a cardiac arrest shortly after arrival. His serum potassium on this occasion was 7.8 mmol/l. Despite prolonged attempts resuscitation was unsuccessful.

Pathology

Renal biopsy

The most marked changes on renal biopsy involved the arterioles and small arteries. A range of features were present, from fibrinoid necrosis with insudation of fibrin and red cells to oedematous intimal thickening with occlusion of the lumen consistent with a necrotizing vasculopathy. There were very few inflammatory cells present in the vessels. The glomeruli showed lobulation due to mesangial thickening with little cellularity. Some contained focal/segmental necrotizing lesions. There were no crescents. The tubules showed secondary damage and contained casts. There was little interstitial inflammation (Fig. 1).

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Necrotizing renal vasculopathy after ingestion of ‘ecstasy’

Post-mortem findings

The lungs were heavily oedematous weighing 1250 g (right) and 1065 g (left); in addition, a large wedge-shaped infarct was present in the right lower lobe associated with an embolus. The heart was enlarged (weight 560 g) with a hypertrophied and enlarged left ventricle. This was associated with a congenitally abnormal aortic valve the cusps of which were swollen and deformed with partial fusion of the commissures. Histology showed myxoid degeneration. The kidneys were macroscopically normal, but showed healed arteritis of small arteries with narrowing of the lumen and focal/segmental sclerosis of glomeruli. There was no evidence of arteritis in any of the major organs.

Discussion

A systemic disorder associated with characteristics of polyarteritis nodosa has previously been reported in a study of 14 drug abusers in 1970 [1]. Their clinical presentation varied from the asymptomatic to those with signs and symptoms of renal failure, hypertension, pulmonary oedema, pancreatitis and neuropathy.

Vascular changes including arterial aneurysms and sacculations were seen in the kidney, liver, pancreas and small bowel at selective angiography which was used in diagnosis. These changes were confirmed at post-mortem in the four patients with a fatal outcome. Histologically they had a necrotizing angiitis (as in polyarteritis nodosa) involving medium-sized and small arteries in most organs, with inflammatory infiltrate, fibrinoid necrosis and luminal narrowing.

The 14 patients involved had taken a wide range of narcotics, stimulants, hallucinogens and depressants, making it impossible to define a single aetiologial agent, although methamphetamine taken intravenously was the most commonly used drug. None of the patients had taken 3,4-methylenedioxymethamphetamine (MDMA or ‘ecstasy’). Less than 30% of the patients had positive serology for hepatitis B. An isolated case of angiographically and histologically confirmed necrotizing angiitis in a methamphetamine user has been reported in association with hepatitis B-antigen positive hepatitis [2].

Methamphetamine and ‘ecstasy’ may cause acute reversible renal failure due to acute tubular necrosis secondary to hypotension, rhabdomyolysis, disseminated intravascular coagulation [3–6] (DIC) and hyperpyrexia. Methamphetamine-induced acute interstitial nephritis proven on renal biopsy has been described [7]. ‘Ecstasy’ may have similar direct nephrotoxic effects but renal biopsy is usually impossible because of the presence of DIC [3]. Recreational use of ‘ecstasy’ has increased greatly over the last 10 years and it is often perceived to be a ‘safe’ drug for occasional use. Methamphetamine has been used since the 1960s. Its effects are well established. There have been a number of recent reports of deaths from arrhythmias, hyperpyrexia and DIC after the use of ‘ecstasy’, as well as suggestions that it may have long-term adverse effects on the brain [8,9] and liver [10].

Our patient is the first reported case of chronic renal failure developing after the use of oral methamphetamine and ‘ecstasy’ with histological evidence of necrotizing angiitis and negative hepatitis serology.

References


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