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BRIEF REPORT

An unusual case of Ecstasy poisoning

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Abstract We describe a case of poisoning with 3,4-methylenedioxymet-amphetamine Ecstasy that presented with all the features suggestive of a fatal outcome, including a creatinine phosphokinase level markedly higher than any previously reported. The patient, a paraplegic, was treated with dantrolene and made a full recovery.

Key words Paraplegia · 3,4-Methylenedioxymetamphetamine (MDMA, Ecstasy) · Hyperthermia · Rhabdomyolysis · Disseminated intravascular coagulation (DIC) · Dantrolene

Introduction

The consequences of poisoning with the "designer" drug 3,4-methylenedioxymetamphetamine (MDMA, Ecstasy) are well known and include hyperthermia, rhabdomyolysis, disseminated intravascular coagulation (DIC) and renal failure. The cases reported in the literature with a full complement of these features mainly have a fatal outcome [1]. We describe the case of a young male paraplegic, who presented in an extremely poor condition but survived to make a full recovery.

Case history

A 26-year-old man (weight approximately 100 kg) who was paraplegic following lumbar spinal injuries after falling from scaffolding 1 year previously, took one tablet of Ecstasy while in a public house with friends. He had taken it previously on a few occasions. While travelling home by bus he began to feel non-specifically unwell, had a progressive decrease in conscious level and developed generalized seizures. He was taken to the casualty department of a small community hospital, where at presentation he was fitting intermittently, had an impaired conscious level and was noted to have a rectal temperature of 41 °C. He was treated with intravenous diazepam in increments of 10 mg and given 60 mg i.v. dantrolene. He was then transferred by ambulance to the intensive care unit in our hospital.

On arrival he was in status epilepticus, despite having received a total of 50 mg diazepam. He was immediately given a further 10 mg diazepam to no effect. The patient was not cyanosed, had vomitus about the mouth and continued to fit. Anaesthesia was induced in rapid sequence with thiopentone, alfentanil and suxamethonium (immediate intubation was required and 1 year had elapsed since the onset of paraplegia: the possible risks of suxamethonium in the paraplegic were thus outweighed by the benefits) followed by intubation and ventilation. Fitting was thus controlled and there was no subsequent grand mal seizure activity. Sedation was maintained with morphine and midazolam by infusion. His temperature at this point was 39.1 °C, he received a further 40 mg dantrolene (i.e. 1 mg/kg in total), then an infusion of 20 mg per h (5 mg/kg per day) and active cooling to 37 °C was started using a Hawksley Ripple Cool blanket. There was no shivering and non-depolarizing neuromuscular junction blocking agents were not used.

Over the next few hours he became progressively hypotensive despite adequate filling confirmed by pulmonary artery catheter. Inotropic support with dobutamine and noradrenaline was started, which rapidly corrected the cardiac index and systemic vascular resistance index to within the desired range. Twelve hours later it became evident that he dad DIC; fresh frozen plasma and platelets were given. His temperature had now normalized and the dantrolene was reduced to 10 mg per h. At 24 h he was oliguric, with evidence of acute renal failure (see Table 1 for details of biochemical and haematological parameters) and gross rhabdomyolysis. Urine contained myoglobin and the serum creatinine phosphokinase was 555000 IU/l. He received renal dose dopamine and urine output was maintained at >30 ml/h. He developed the adult respiratory distress syndrome according to the European–American Consensus Conference diagnostic criteria [2]; pulmonary gas exchange deteriorated to a nadir where a fractional inspired oxygen of 0.8 was required.

By 36 h he was showing multifocal ventricular ectopic beats but remained haemodynamically stable with inotropic support. By 48 h

Table 1 Progress of biological and haematological parameters

	Day					
	1	2	3	4	11	17
Na (mmol/l)	137	133	133	137	137	141
K (mmol/l)	5.8	4.3	4.5	3.5	4.6	3.9
Urea (mmol/l)	9.0	16.3	19.3	24.1	20.4	9.0
Creatinine (μmol/l)	199	206	343	450	170	84
Hb (g/dl)	15.6	11.3	10.9	10.6	9.8	10
WBC ($\times 10^9$/l)	30.7	11.5	9.9	7.1	16.6	15
Platelets ($\times 10^9$/l)	135	46	38	53	258	415
INR	1.8	4.3	3.6	4.8	1.3	
CPK (IU/l)	2800	555000	84000			
Alanine amino-transferase (IU/l)	46	1212	3546	3863	340	154
Bilirubin (μmol/l)	30	50	80	131	86	48

there was evidence of hepatic failure. Acetylcysteine was then given for 72 h (27 g, 13.5 g then 6.75 g, each in 500 ml 5% dextrose over 24 h). At this point dantrolene was reduced to 5 mg/h. He now had gross multisystem failure affecting five organ systems. In addition, there was evidence of colitis, with profuse diarrhoea with mucus (stool samples were negative for culture and *Clostridium difficile* toxin).

Over the next few days with supportive therapy his condition gradually improved; active cooling and dantrolene were stopped at 72 h. A percutaneous tracheostomy was performed at day 10, and at day 17 he was self-ventilating with a tracheostomy mask. He was eventually discharged to the ward and later home after a brief stay in a rehabilitation unit. He has made a full recovery.

Discussion

In Britain there have been many cases of severe or fatal reactions to Ecstasy [1], while very few have been reported in the United States despite its widespread use [3]. This difference has been attributed to the circumstances in which the drug is used [4]. In the United States, Ecstasy is taken alone or at parties, while in Britain it is used in the "rave scene" as a "dance drug". It is known from animal studies that Ecstasy, through central serotonergic mechanisms, causes excessive heat production [5], and it has been proposed that this then triggers rhabdomyolysis and DIC [6, 7]. The "rave" party, with hard and fast dancing, often with little or no opportunity to cool off, is thought greatly to facilitate this hyperthermic process. Our patient, a wheelchair-bound paraplegic, could not have contributed to heat production in the same way.

Many of the cases reported have been fatal [1]. There are no proven criteria at presentation that may be used to predict outcome. The patient described had a temperature of 41 °C, arrived on our intensive care unit from another centre in status epilepticus and had experienced an approximately 8-h delay in the initiation of body cooling. The patient went on to develop failure of at least five organ systems. He also had by far the highest peak creatinine phosphokinase (CPK) level that we have found on a search of the literature [8]. Despite all this, he made a full recovery. Perhaps his high CPK can be explained by intracellular concentration changes in the muscle as a consequence of denervation, a phenomenon well known to anaesthetists in the context of nicotinic receptor upregulation and exposure to suxamethonium.

No other cases of adverse reactions to MDMA in paraplegics have been described in the literature. This case had what are apparently extremely poor prognostic indicators at presentation and yet he survived. It is clear that there are many questions still unanswered about the mechanism by which Ecstasy triggers rhabdomyolysis and DIC.

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