Burns (1993) 19, (3), 239–240 Printed in Great Britain

Ecstasy and Whizz at a rave resulting in a major burn plus complications

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A patient who sustained a major burn following abuse with Ecstasy and Whizz is described. His progress was complicated by hyperpyrexia, acute renal failure and convulsions, all of which have been recently recognized as potential side-effects of Ecstasy abuse. This report further highlights the dangers of substance abuse.

Introduction

Ecstasy (3,4-methylenedioxymethamphetamine) is one of the many new designer drugs. It was originally introduced in the USA in the late 1970s as an 'underground' adjunct to psychotherapy, only becoming illegal in 1985 (Davis et al., 1987). It consists of a psychoactive phenylisopropylamine and is structurally similar to both amphetamine-related sympathomimetics as well as the hallucinogen mescaline (Hubner et al., 1988). It thus not only produces the characteristic clinical picture of amphetamines with hyperactivity, agitation and tachycardia, but is also a euphoriant and has hallucinogenic potential. Its use is increasing and it is often taken in tablet form before and during 'raves'. Whilst often perceived by those who take it as harmless, there is increasing evidence of a wealth of serious, and some fatal, side-effects.

We present a case in which a regular Ecstasy and amphetamine abuser sustained a major burn resulting from the use of these drugs and whose resuscitation and recovery was complicated by several of the recently described side-effects.

Case report

A 19-year-old male was admitted to the Burns Unit having sustained 31 per cent burns to both arms and back, of which 4 per cent was estimated as full thickness skin loss. He had been in a car in which a petrol can had ignited. The exact circumstances remain unclear. The patient had been at a rave and had taken several tablets of Ecstasy, and in the preceding 48 h 2 g of Whizz (a standard amphetamine). He had, over the last 18 months, been a regular abuser of Ecstasy, taking up to 15 tablets a day.

On admission he described and exhibited features of paranoia. His cardiovascular responses were stable. Our usual resuscitation regimen, based on the Roehampton formula with Dextran 110, was instituted. He was electively intubated although adequately ventilating since he had clinical evidence of an inhalation injury with soot in the airways and marked pharyngeal oedema. Abnormally high doses of propofol (480 mg/h; normal dose 70–280 mg/h) were required to maintain adequate sedation. In the 48-h shock phase his urine volumes were satisfactory though it was noted that they were becoming dark with a greenish tinge and contained high levels of haemoglobin and protein. At 2 days postburn early tangential excision and grafting of some of his burns was performed.

On day 5 whilst being weaned off the ventilator his temperature rose to 40°C, he became tachypnoeic, and despite an increased Fio_2 he went into respiratory failure and required reventilation in order to maintain an adequate Pao_2. His urine had become more obviously green and darkened on standing. The green colour was attributed to the propofol infusion and the darkening to the oxidation of free haemoglobin. Radiology of the chest revealed changes consistent with widespread infection or early Adult Respiratory Distress Syndrome. His blood haemoglobin level fell to 9.7 g/dl (from 11.6 g/dl) and his urine output declined. His platelet count however remained elevated, and clotting studies and fibrinogen degradation product levels were normal. Aggressive fluid challenge plus diuretics restored a normal urine output and he was sufficiently well on day 9 for further grafting to his burns.

On day 14 his urine again became dark, his pyrexia increased and over the next few days he went into a high output renal failure (*Figure 1*). His creatinine rose to 444 mmol/l, and his urea to 55.8 mmol/l. Haemofiltration and haemodialysis were started, and

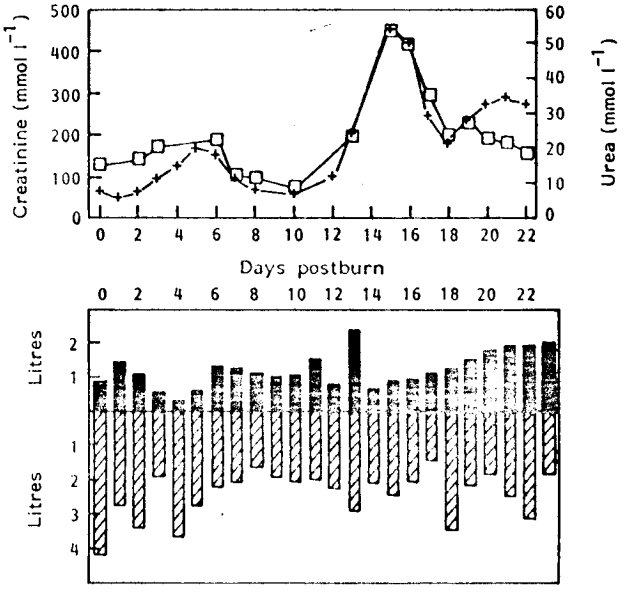

Figure 1. Plasma urea and creatinine, and fluid balance. —□—, Creatinine; —×—, urea; ■, urine output; ▨, fluid input.

after 10 days he stabilized and his renal function returned to normal.

At 4 weeks he was about to be discharged, when he had a grand mal fit and became unconscious. He was transferred to a neurosurgical unit where CT scanning revealed multiple echogenic areas within the brain suggestive of either cold abscesses or multiple infarcts. He was treated conservatively with antibiotics and made an unremarkable recovery, the lesions becoming smaller on repeat CT scanning. He was discharged at 10 weeks postburn on anticonvulsant therapy.

Discussion

Whilst the clinical and pathological effects of amphetamine overdose are well recognized, those arising from Ecstasy overdose are only just beginning to emerge. Ecstasy has gained widespread acceptance amongst those who attend 'raves' and is taken prior to and during such occasions. It is relatively cheap, costing between £15 and £25 (US$22–37) per tablet. Because intravenous administration is not required it is more readily available and there is a false sense of safety regarding its use.

It produces a euphoric sensation, together with a feeling of closeness. In one survey 20 per cent of users experienced visual hallucinations (Peroutka et al., 1988), Sympathomimetic effects include hyperactivity, sweating, mydriasis, tachycardia and a dry mouth. In overdose the user initially gets paranoia and psychoses, and then eventually delirium. One of the commoner side-effects is hyperpyrexia (Chadwick et al., 1991). This effect is also recognized by the organizers of 'raves', and often a cooler area is set aside where the overly heated Ecstasy taker can 'chill-out', hence the expression.

In our patient the classical paranoia and hyperactivity were seen on presentation. The history was unclear but there can be little doubt that the cause of the burn was related to the altered state of consciousness induced by the drug.

Our patient twice lapsed into renal failure despite adequate fluid replacement and without any evidence of renal hypoperfusion. The cause of renal failure was probably multifactorial. Three mechanisms for amphetamine-related renal failure have been reported, all of which may be valid for Ecstasy: a direct toxicity (Foley et al., 1984), tubular damage secondary to hyperpyrexia-induced rhabdomyolysis (Screaton et al., 1992) and finally, secondary to disseminated intravascular coagulopathy (DIC) (Ginsberg et al., 1970). Although free haemoglobin was present in the urine implying intravascular haemolysis, DIC was excluded by the aforementioned tests. Hyperthermia was noted prior to the episodes of renal failure and an associated rhabdomyolysis remains a strong possibility. Although myoglobinuria was never demonstrated it is very transient and as such readily missed. Other markers of rhabdomyolysis are of little value in burned patients. Lastly a direct toxicity of Ecstasy causing acute interstitial nephritis cannot be excluded. The additional factor of sepsis including renal failure is possible, though we felt that in this patient this was less likely.

At 4 weeks postburn our patient had recurrent fits and on CT had multiple echogenic areas representing either infarcts or abscesses. There was no previous history of epilepsy. There is one previous report implicating Ecstasy with fits and cerebral infarction (Harris and de Silva, 1992). We feel that this case may represent another such association.

Ecstasy abuse has also been linked to a number of other side-effects. Common ones include impaired concentration, depression, recurrent flashbacks and prolonged neuropsychiatric syndromes (Creighton et al., 1991; MCann and Ricaurte, 1991). On a more sinister note there are also reports of jaundice, acute hepatitis, cardiac arrhythmias, acute chest pain and sudden death (Gorard et al., 1992; Henry, 1992).

Clearly Ecstasy is not a safe drug as is so often perceived by those who take it. There are a number of very serious and potentially fatal side-effects, some of which are highlighted in this report.

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Paper accepted 18 November 1992.

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