Conservative management was continued and over the course of the next few days his symptoms and signs regressed. A repeat contrast study again demonstrated no leak and with resolution of his clinical picture his CXR improved. The patient was discharged 7 days after admission painfree and eating normally. Since this episode the patient has been asymptomatic and has taken no more MDMA.

Discussion

Pneumomediastinum is a relatively rare condition and when unassociated with obvious perforation of an intrathoracic viscus is termed 'spontaneous'. Spontaneous pneumomediastinum has been described in relation to many different circumstances including asthma, barotrauma secondary to artificial ventilation, laparoscopy[1], oesophagoscopy (without oesophageal perforation), transbronchial biopsy[2], dental extraction, sinus wash out[3], belching[4], violent exercise and drug abuse.

Pneumomediastinum in relation to drug abuse was first noted as a consequence of cocaine inhalation[5] and recently it has been observed in subjects inhaling alkaloid opiates and marijuana. In all these cases it is postulated that the mechanism of pneumomediastinum is an increase in intrathoracic pressure caused by the valsalva manoeuvre (performed by the subject to heighten the effect of the narcotic) leading to rupture of marginal alveoli and resultant 'back-tracking' of the air into the mediastinum via the perivascular spaces around the pulmonary artery. We believe this to be the first case described of an ingested narcotic causing pneumomediastinum and feel that the aetiology here is due to vomiting against a fixed glottis causing a rise in intrathoracic pressure but no oesophageal perforation.

'Ecstasy' use is associated with a number of clinico-pathological states, eg acute psychotic reactions, hyperthermia, hypotension, tachycardia, disseminated intravascular coagulation, adult respiratory distress syndrome (ARDS) and rhabdomyolosis leading to acute renal failure[6]. With its widespread use amongst many young people more sequelae will become manifest with time. We believe that faced with a young patient giving a history of chest (or neck) pain associated with narcotic abuse a physician should immediately get a chest X-ray, and if a pneumothorax, pneumomediastinum or pneumopericardium is noted an immediate contrast swallow should be arranged. If this demonstrates no oesophageal rupture a conservative policy should be adopted (as above) with gradual reintroduction of fluids and food by mouth as the patient clinically improves and the X-ray normalizes.

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'Ecstasy' ingestion: a case report of severe complications

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Keywords: drug abuse; ecstasy; acute renal failure; disseminated intravascular coagulation; rhabdomyolysis

Ecstasy or MDMA (3,4,methylenedioxymethamphetamine) is a Class A controlled drug in the United Kingdom. This case report details the severe complications of disseminated intravascular coagulation (DIC), acute renal failure (ARF), rhabdomyolysis and impaired liver function following the ingestion of a single tablet of ecstasy and a quantity of amphetamine.

Case report

A 23-year-old male was admitted to casualty following a convulsion 3 h after the ingestion of one ecstasy tablet and £5 worth of amphetamine. Significant findings were: agitation, mydriasis, purposeless clonic movements, tachypnoea of 40/min, tachycardia of 130/min, blood pressure 120/80 mmHg, and axillary temperature 40°C. Immediate therapy included oxygen and chlorpromazine 50 mg intramuscularly on two occasions to control agitation. The patient subsequently developed a tachycardia of 160/min and his blood pressure fell to 85/60 mmHg, together with a reduced level of consciousness. Intravenous fluids rapidly restored his blood pressure to 100 mmHg systolic and propranolol 5 mg was administered incrementally to control tachycardia. He was intubated and transferred to intensive care.

On admission, he was sedated, ventilated and monitored invasively. Over 12 h, a progressive metabolic acidosis was noted and the naso-gastric aspirate was bloodstained. He developed haematuria and oliguria despite adequate fluid loading and blood pressure. Frusemide 40 mg was administered intravenously, initially with good effect, but there was biochemical and haematological evidence of rhabdomyolysis, renal and hepatic dysfunction and DIC (Table 1). Toxicology studies revealed an MDMA level of 0.2 mg/l and an amphetamine level of 0.1 mg/l. The patient was treated aggressively with blood and blood products and daily haemofiltration. He was extubated after three days, biochemical and haematological profiles improved, and haemofiltration was continued for 21 days, after which his renal function returned to normal. The patient was discharged from hospital 33 days after his admission.

Discussion

MDMA is a semi-synthetic hallucinogenic compound related to amphetamine and mescaline. In the UK, MDMA became established within drug using circles at so called 'Acid House' parties in the late 1980s. Little is known of the pharmacology and toxicology of MDMA, but the initial side effects are sympathomimetic[1]. A review of the literature revealed two fatalities in the UK associated with hyperthermia, rhabdomyolysis and coagulopathy[2,3] and four fatalities in the USA due to cardiac complications or accidents[4,5].

Our patients toxicology studies revealed non toxic levels of both MDMA and amphetamine, but the combined level

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Table 1. Selected biochemical and haematological values

	Hours after admission		*Day*			
Parameter	*1*	*12*	*2*	*4*	*5*	*10*
MDMA (mg/l)	–	0.2				
Amphetamine (mg/l)	–	0.1				
Urea (mmol/l)	9.1	17.2	20	10.8	20.5	24
Creatinine (μmol/l)	225	239	393	323	460	570
Total bilirubin (μmol/l)	7	–	36	139	78	31
LDH (U/l)	845	–	5312	1548	1461	727
ALT (U/l)	81	–	289	522	626	284
Alk Phos (U/l)	80	–	69	83	103	102
CK (U/l)			5849			
Base excess	−6.6	−10.8				
Bicarbonate (mmol/l)	19.2	16.2				
Myoglobinuria		Absent				
Platelets (10^9/l)	19	11	17	57	64	266
INR	1.9	1.9	3.6	1.2	1.1	1.1
APTT (s)	46	<115	<115	<115	36	41
Fibrinogen (g/l)	–	1.9	1.8	5.0	5.6	5.2

of 0.3 mg/l was above the toxic threshold of 0.2 mg/l. Brown *et al.*[6] described a non-fatal case of DIC, poor urine output responding to frusemide, toxic hepatitis, and rhabdomyolysis in a patient presenting with MDMA levels of 7 mg/l. This represents true toxicity. Our patient developed similar but more severe complications to those described above in the presence of much lower MDMA levels. This may be an idiosyncratic reaction, but may be related to a contaminant introduced during the manufacture of the drug, or to the circumstances in which it is taken such as energetic dancing, heat exhaustion, dehydration and alcohol consumption. There are clinical similarities with malignant hyperpyrexia and the neuroleptic malignant syndrome with which there may be an association. In addition, he developed reversible acute renal failure which has previously only been reported following amphetamine toxicity[7]. The cause of his renal failure is uncertain but may be related to idiosyncrasy, DIC or rhabdomyolysis, although myoglobinuria was absent.

The treatment of MDMA related morbidity should be early and aggressive and includes: gastric lavage, chlorpromazine, α and β adrenergic blockade, intravenous fluids and passive cooling. A metabolic acidosis, whilst encouraging renal excretion of the drug, may cause precipitation of myoglobinuria in the renal tubules, and sodium bicarbonate should be administered. Blood, urine and gastric lavage samples should be sent for toxicology studies. Although severe complications are rare, they are fulminating and may be rapidly fatal. We advise admission to an intensive care unit with close monitoring in anticipation of the problems reported.

The case report involves an illicit drug that has a street reputation as being harmless. This report proves otherwise. Personal communication has recently revealed four deaths associated with fulminating DIC following MDMA toxicity. More deaths can be expected due to its continuing illegal distribution.

Since acceptance of this paper for publication, The National Poisons Information Service now advocates the early use of dantrolene in the management of severe complications following 'ecstasy'. Chlorpromazine may lower the convulsive threshold and is no longer advised.

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Orbital Kimura's disease

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Keywords: Kimura's disease; orbital muscles

Kimura's disease of the orbit is rare. Only 10 cases proven by histology have been published in the English language literature. In this communication the author presents a case of Kimura's disease with lacrimal gland and orbital muscle involvement in a 21-year-old man who responded to systemic corticosteroid. This case is unique in that orbital muscles are involved.

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Case report

A 21-year-old man presented with progressive proptosis of the right eye and upper eyelid swelling since December 1985. However, he defaulted follow up. In January 1990, he presented again with increased right eye proptosis. On clinical examination, his right eye was protruding forwards and downwards. Adduction of his right eye was slightly impaired, whereas tension of the eyeball, visual acuity and fundal examination were normal. A large right epitrochlear lymph node 2×1.5 cm was palpable.

Upon investigation, eosinophilia was present in the blood counts: WBC 18.6×10^9/l (eosinophil 42%, neutrophil 29%, lymphocyte 20%, monocyte 8%, basophil 1%). The following investigations were either normal or negative: thyroid function tests, autoantibodies, complements,