MDMA induced hyperthermia: a survivor with an initial body temperature of 42.9°C

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Abstract
A young male survived hyperpyrexia (42.9°C) following MDMA (“Ecstasy”) ingestion. He developed convulsions, rhabdomyolysis, metabolic acidosis, and respiratory failure. This was successfully managed by assisted ventilation, aggressive fluid therapy, and the early administration of dantrolene, in addition to cooling measures. This is the first report of a survivor with such a severe hyperpyrexia.

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Keywords: MDMA; Ecstasy; hyperthermia; dantrolene

In recent years the use of 3,4-methylenedioxyxymethamphetamine (MDMA, “Ecstasy”) has increased in the USA, the United Kingdom, and continental Europe.1 Severe and sometimes fatal adverse reactions are increasingly encountered in various accident and emergency (A&E) departments. These include hyperthermia, metabolic derangements, seizures, hypertensive crises, cardiac dysrhythmias, disseminated intravascular coagulation (DIC), rhabdomyolysis, acute renal failure, hepatic toxicity, cerebrovascular accidents, and psychiatric disturbances.2 3 Survival with a core temperature greater than 42°C is rare.3

Case history
A 19 year old male presented after collapsing in a local night club. The patient was reported to have ingested three tablets of Ecstasy four hours previously. At presentation, he was obtunded, with a Glasgow coma score of 7 (eye opening 2, vocalisation 2, motor response 3). He was sweating profusely, with a rectal temperature of 42.9°C, central cyanosis, and a respiratory rate of 44 breaths/min. A narrow complex tachycardia of 192 beats/min was present but blood pressure was 150/90 mm Hg. His pupils were widely dilated.

The patient arrived in the A&E department with seizures, which were controlled with 10 mg diazepam intravenously. In view of the seizures and the marked hyperpyrexia, he was anaesthetised using thiopentone sodium and suxamethonium, and his airway was secured by tracheal intubation. Anaesthesia was maintained with increments of midazolam, and muscle relaxation was continued with an infusion of atracurium. Hyperthermia was treated with cooling measures (moistened towels, ice packs, fanning, and body exposure). At the same time, dantrolene was given intravenously as 60 mg boluses at 10 minute intervals to a total of 180 mg until the body temperature was brought down to 38°C.

The patient was transferred to the intensive care unit. Sedation was maintained with infusions of alfentanil and propofol. From the time of admission he required a total of 6 litres of crystalloid over a period of six hours for resuscitation, with continuous monitoring of central venous pressure. Over the ensuing few hours he became progressively hypotensive despite adequate central venous pressure. A pulmonary artery catheter was inserted and the wedge pressure was found to be adequate. He
needed an adrenaline infusion at 0.1 μg/kg/min to maintain his circulation. Aliquots of 50 mmol sodium bicarbonate were given twice to correct a metabolic acidosis found on repeated arterial blood gas analyses. Urine output was maintained at more than 2 ml/kg/h. There were no further seizures in the intensive care unit. From the time of presentation to A&E department, normal oxygenation proved difficult to achieve. Blood gases are shown in Table 1. Despite the application of positive end expiratory pressure of up to 10 cm H₂O, PaO₂ was 6.9 kPa on an FiO₂ of 1.0. Secretions were minimal and there was no evidence of aspiration of gastric contents. Chest X ray showed bilaterally increased vascular markings. Hypoxia resolved spontaneously over the next 24 hours, and the patient was extubated on day 2. There were no detected residual deficits on a neurological assessment and he was discharged home on day 6. Full blood count, coagulation profile, and blood biochemistry were all within normal limits. Serum sodium concentration was within normal range. Creatine kinase reached a maximum of 42 120 IU/l, falling to 6000 IU/l after five days. Serum aspartate transaminase (AST) rose to 1400 IU/l, returning to normal after five days. MDMA was detected both in blood and urine. Urine examination revealed myoglobinuria of more than 100 mg/dl. Despite myoglobinuria, this patient did not show any signs of renal impairment, as blood urea and serum creatinine remained within normal range.

On follow up at six weeks, the patient had not taken any more MDMA and there was no abnormality on gross neurological evaluation.

**Discussion**

MDMA is commonly used in the "rave" scene, where strenuous exertion and subsequent dehydration are generally thought to be partly responsible for the hyperthermic syndrome. MDMA-induced hyperthermia, as suggested from animal evidence, is due to an acute increase in central 5-hydroxytryptamine (5-HT) and dopamine levels, and their synergistic action on hypothalamic centres. The severity and duration of hyperthermia appear to be important prognostic indicators in this condition. Complications seem to be related to duration and degree of hyperthermia and they increase in number and severity if not treated promptly. The concentration of circulating endotoxins and cytokines, tumour necrosis factor α (TNF-α), interleukin-1 (IL-1), and interleukin-6 (IL-6) are increased in the presence of very high body temperature and they can lead to multisystem organ failures. The patient in this report had a peak temperature of 42.9°C which was quickly brought down, within an hour of arrival, with both intravenous dantrolene and cooling measures. We attribute the survival of our patient to prompt and rapid control of hyperthermia in the A&E department. It would not have been possible to bring down the body temperature within an hour with cooling measures alone. Moreover, external cooling can lead to peripheral vasoconstriction and a raised core temperature.

As the patient was hypoxaemic and had a full stomach, we used suxamethonium to secure the airway quickly, although its known side effects of hyperkalaemia and precipitation of malignant hyperpyrexia are of potential concern in these group of patients. In vitro muscle testing for malignant hyperpyrexia in one patient did not substantiate this. Non-depolarising relaxants such as atracurium or the more rapid acting rocuronium may be suitable alternatives in such a situation.

Dehydration was corrected by aggressive fluid therapy with isotonic 0.9% saline over a period of six hours, with central venous pressure and pulmonary artery wedge pressure monitoring as a guide. There have been reports of three deaths due to dilutional hyponatraemia and acute water intoxication following MDMA ingestion. This may result from drinking excessively large volumes of water.
Seat belt injuries and sigmoid colon trauma

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Abstract
Colonic seat belt injuries are rare but carry higher mortality rates than small bowel injuries. The case of a 44 year old man is described who had severe sigmoid colon compression injury from his seat belt a few days after a road traffic accident.

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Keywords: seat belt; sigmoid compression

Case report
A 44 year old male driver with no significant past medical history presented to the accident and emergency department two days after a road traffic accident with increasing abdominal pain and constipation.

He was febrile, dehydrated, and abdominal examination showed severe peritonitis. There was a faint resolving bruise over the lateral margin of the left iliac fossa. Digital rectal examination was normal.

He was the driver in a road traffic accident when he skidded and hit a lamp post. The front passenger died of severe head injury and two of the three back seat passengers sustained variable physical injuries. He was treated in another city hospital for minor facial wounds.