Spontaneous pneumomediastinum and Ecstasy abuse

J A L Pittman, J C Pounsford

Abstract
Ecstasy is an illegal recreationally used drug. A case of a young woman who had taken this drug and was found to have a spontaneous pneumomediastinum is reported. The association of spontaneous pneumomediastinum with drug abuse is discussed. The possible mechanism for this complication of Ecstasy, which has not been previously reported, is discussed. (J Accid Emerg Med 1997;14:335–336)

Keywords: Ecstasy; pneumomediastinum

Ecstasy, or MDMA (3,4-methylenedioxymethamphetamine), is a class A controlled drug. Its side effects and complications are becoming more apparent as its use increases. It has achieved high media profile due to its association with dance parties and “raves” and is most extensively taken by young adults. This case report details a complication of spontaneous pneumomediastinum following the ingestion of Ecstasy.

Case report
A 17 year old female was referred to the medical admission unit by her general practitioner. She was complaining of chest pain and a swollen neck. She gave a history of attending a party fifty six hours previously and ingesting one and a half tablets of Ecstasy. She had then danced, blowing a whistle, almost continuously for eight hours before going home the following morning. She gave no history of vomiting, trauma, or coughing. Twelve hours later she noticed that her neck was swollen and cracked on palpation. She felt short of breath and had some retrosternal pleuritic chest pain that radiated to her back. She had no relevant past medical history. She did not smoke or drink alcohol.

On examination she was not distressed and was apyrexial. She was noted to have surgical emphysema of the neck and a pericardial “crunch” (Hamman's sign). Her pulse was 55 beats/min and blood pressure 120/50 mm Hg. Examination of her chest and abdomen were normal. The white blood cell count was not raised. A chest X ray was taken. This showed lucency between the cardiac border and the pleura consistent with a pneumomediastinum. There was also a small pneumothorax at the right apex.

She was admitted for observation and treated with paracetamol for analgesia. The following day her surgical emphysema had improved and her chest pain eased. Her four hourly observations remained stable. She was discharged and seen in clinic three days later. Repeat chest film showed no change, but clinically she had improved. She failed to keep a subsequent outpatient appointment.

Discussion
Pneumomediastinum is an uncommon but potentially life threatening condition. It is termed spontaneous when not associated with trauma or identifiable pathological processes. In this situation it is usually benign and self limiting, mainly occurring in young males and women in labour.1 Spontaneous pneumomediastinum was first described by Hamman.2 The pathophysiology was proposed soon after this.3 Raised intra-alveolar pressure or reduced interstitial pressure results in the rupture of the alveolar sac and the extravasation of air into the perivascular fascia. The air then tracks back along the vessels into the mediastinum to create the pneumomediastinum. Gas can decompress from there into the neck between the layers of the deep cervical fascia, creating surgical emphysema. Patients generally present with sharp chest pain which is worse on inspiration. Other common presenting symptoms are dyspnoea, neck pain, and dysphagia. Physical examination may be normal, with the most common finding being surgical emphysema of the neck. A pericardial crunch and pneumothorax are also frequently present.4

Spontaneous pneumomediastinum has been reported in conjunction with the taking of recreational drugs.7 Cases have been documented following the inhalation of marijuana,6 cocaine,8 and alkaloid cocaine “crack”.9 Spontaneous pneumomediastinum after inhaling drugs is thought to be due to barotrauma.6 Forceful breath holding following the inhalation of a drug will create a Valsalva type manoeuvre and increase the intra-alveolar pressure, which may cause the alveoli to rupture.

Ecstasy is a commonly used recreational drug. Although patented in 1914, it was only since the late 1980s that the drug has become popular. Ecstasy produces a feeling of euphoria and increased sensuality. As its use increases, so the spectrum of side effects becomes more apparent. These vary from mild jaw stiffness, bruxism, sweating and palpitations to life threatening cardiac, thermoregulatory, and neurological side effects.9 Reports on the long term consequences of Ecstasy abuse are awaited.

We believe this to be a unique case of spontaneous pneumomediastinum following ingestion of Ecstasy. We propose that the spontane-
MDMA induced hyperthermia: a survivor with an initial body temperature of 42.9°C

A Mallick, A R Bodenham

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. (J Accid Emerg Med 1997;14:336–338)

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.4

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