features previously with crack usage. Notably, they had few clinical signs apart from decreased peak flow and tachypnoea but were hypoxaemic and had interstitial shadowing on the chest radiographs. These clinical features resemble case reports from North America in whom non-cardiogenic pulmonary oedema developed after smoking crack.4 However, our three cases recovered rapidly over a few hours without specific treatment.

Crack is produced from “street” cocaine (normally cocaine hydrochloride) by the addition of a base (normally sodium bicarbonate). This mixture is boiled, resulting in the formation of a residue of crystalline free base (crack) cocaine.5 The crystals or “rocks” are then smoked. Crack cocaine is about 50-90% pure6 and may contain various additives including sugars, ephedrine, caffeine, lignocaine, talc, strychnine, and quinine.6 A case of acute pneumonitis associated with silica accumulation has been reported after smoking crack cocaine.7 There have also been reports of pulmonary granulomatosis secondary to talc and cellulose contamination but these cases had only sniffed cocaine and had less acute presentations.8,9 In our cases it later transpired that the dealer had prepared the crack in an unusual way, having mixed cocaine with ammonia as well as sodium bicarbonate before boiling. We were unable to identify a specific impurity from a small residual sample of the crack which caused the acute pneumonitis in these three patients.

Corticosteroids have been used in cases where the acute pulmonary syndrome has persisted1 and where there has been evidence of pulmonary eosinophilia in lung biopsy or bronchial/alveolar lavage samples.3,10 A short course of corticosteroids should therefore be considered after initially ruling out any other causes of pulmonary infiltrate, particularly of an infectious nature.

As the use of crack cocaine increases in the UK and other countries there may be a concomitant rise in associated pulmonary syndromes, some of which may be due to adulterants rather than the crack itself, as these three cases show.


Ecstasy induced pneumomediastinum

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Abstract

Two cases are reported of pneumomediastinum induced by the use of Ecstasy, a semi-synthetic hallucinogenic compound related to amphetamine and mescaline. In the UK, MDMA became reestablished as a recreational drug in the late 1980s, particularly amongst those in their late teens and early twenties. Simultaneously there has been a rise in reported cases of associated detrimental effects, and the National Poisons Unit estimates that there have been 30-50 Ecstasy related deaths. Here we report two cases of Ecstasy induced pneumomediastinum.

Case reports

A 20 year old man presented after having taken one tablet of Ecstasy earlier that evening. He complained of a sudden onset of central chest pain unrelieved by either deep inspiration and movement. There was no history of vomiting although he did feel nauseated and had retched at least once. There was no past medical history of note and he was not taking any regular medication. On examination he was apyrexic and tachycardic. There was no evidence of surgical emphysema. Cardiovascular examination revealed a pericardial crunch (Hamman's sign); but otherwise examination was unremarkable. His chest radiograph showed the presence of a pneumomediastinum which was confirmed by echo-
Ecstasy induced pneumomediastinum

Cardiography, he was treated conservatively with analgesia and intravenous fluids. A Gastrografin swallow undertaken to exclude a ruptured oesophagus was negative. A CT scan of his chest again confirmed the presence of a pneumomediastinum, but showed no other pathology. Conservative management was continued and over the course of the next few days his symptoms and signs subsided. Serial chest radiography confirmed a gradual resolution of the pneumopericardium.

The second case was an 18 year old man who presented with pleuritic chest pain of sudden onset associated with shortness of breath eight hours after having taken an Ecstasy tablet at a party the night before. He also noticed the crackling of air under his arms, chest, up his throat, and a change in the quality of his voice. On examination he was apyrexial, tachycardic, and tachypnoeic with evidence of subcutaneous emphysema involving his neck, arms, and chest. Hamman's sign was negative. His chest radiograph revealed mediastinal and surgical emphysema present along the lateral chest walls and in the cervical soft tissues. With conservative management his clinical picture improved and serial chest radiography confirmed resolution of the pneumomediastinum and surgical emphysema.

Discussion

An increasing number of complications have been described in association with Ecstasy including disseminated intravascular coagulation, acute renal failure, rhabdomyolysis, impaired renal function, acute psychotic episodes, convulsions, ARDS, and death.1 Spontaneous pneumomediastinum - that is, pneumomediastinum not associated with obvious perforation of an intrathoracic tissue - is uncommon, being first described in 1939 by Hamman.2 Spontaneous pneumomediastinum results from rupture of marginal pulmonary alveoli which allows bubbles of alveolar gas to dissect along the vascular sheaths and connective tissue planes to the mediastinum. Spontaneous pneumomediastinum has been described associated with many different factors including vomiting, asthma, violent exercise, diabetic ketoacidosis,1 infections including Pneumocystis carinii and Cryptococcus neoformans,1 parquat intoxication,2 following transbronchial biopsies,1 endoscopy, and dental extraction. Pneumomediastinum has also been described in association with other illicit drugs. This is thought to be secondary to inhalation techniques. Smoking crack cocaine usually involves deep inspiration followed by the Valsalva manoeuvre and coughing. There has been one previous case report of Ecstasy induced pneumomediastinum in the literature.3 It is unlikely that the mechanism involved in these cases is a direct pharmacological action of MDMA. Furthermore, there is no evidence that the drug was taken in overdose as both cases reported had taken one tablet only, although the doses of drugs taken illicitly are unknown. In the absence of a clear history of trauma or retching (as in the first case), it is difficult to postulate a mechanism for the induction of pneumomediastinum. However, the absence of such a history may represent lack of recollection. An alternative explanation could be that the pneumomediastinum is secondary to the severe physical exercise which is often associated with its use. Currently, Ecstasy is used more for its "energy enhancing" effects than for the hallucinogenic effects which were more important for those using it in the past. Another possibility is the presence of contaminants in the preparation which may influence its actions or have actions of their own.

A greater awareness of the possible complications of Ecstasy, especially in the at risk population, will enable such cases to be diagnosed earlier and managed appropriately.

Ecstasy induced pneumomediastinum.

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Thorax 1996 51: 960-961
doi: 10.1136/thx.51.9.960

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