Case reports

A commentary on the following two case reports appears on pp 962–3.

"Crack lung" caused by an impure preparation

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Abstract

Pulmonary complications of crack cocaine have been reported mainly from American centres. Crack usage is now on the increase in the UK. Three cases of "crack lung" are reported in patients who acquired the drug from the same source. The pulmonary syndrome they developed was due to an impure form of crack.

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Crack, a potent smokable form of cocaine, is now increasingly available in the United Kingdom.[1] The acute clinical syndrome of "crack lung" has been reported previously but the role of adulterants in its pathogenesis is not clear.[2] We report three cases of "crack lung" in subjects who presented on the same evening after having obtained crack cocaine from the same source. This was probably due to an impure preparation of the crack cocaine.

Case reports

Three patients (two women aged 36 and 25, and one man aged 25) presented within two hours of each other with identical histories of acute onset of chest tightness, sharp pleuritic retrosternal pain, cough, and marked dyspnoea coming on a few minutes after smoking crack cocaine obtained that evening from the same drug dealer. All were regular crack smokers of at least 12 months duration but none had noted adverse reactions in the past. All smoked approximately 20 tobacco cigarettes daily and two had occasionally smoked cannabis. All three denied use of other drugs. All three were agitated and distressed, febrile (35.7–38.8°), tachycardic (100–130 beats per minute), and tachypnoeic (22–36 breaths per minute). Examination was otherwise normal; there were no added sounds in the chest. Investigations revealed neutrophilia and raised levels of C reactive protein in all cases but no eosinophilia (table 1). All three were hypoxaemic and hypocarbic and all had reduced peak expiratory flow rate (39–65% of predicted normal). The electrocardiograms were normal but chest radiographs in each case showed distinct perihilar and lower zone interstitial infiltrates.

Following admission their symptoms resolved rapidly. They received 60% oxygen for 24 hours by which time arterial blood gas tensions whilst breathing room air had returned to normal. All had plasma benzoylecgonine levels above 500 μg/l indicating recent exposure to cocaine. Antinuclear factor, rheumatoid factor and complement levels were all normal. Pulmonary function tests (FEV_1 = forced expiratory volume in one second; FVC = forced vital capacity; TLC = total lung capacity; RV = residual volume; T_{LCO} = transfer factor of the lung for carbon monoxide; K_{CO} = diffusion coefficient) performed 12 hours after admission were within the normal range in two patients but showed a considerable obstructive defect (FEV_1 63% of predicted, FVC 88% of predicted, FEV_1/FVC ratio 31%) in the third. Repeat radiographs 48 hours after admission showed complete clearing in all three. Only one patient returned for follow up and reported no further symptoms and no further usage of crack cocaine.

Table 1 Investigations on admission and pulmonary function at 12 hours (percentage of expected values)

	Woman aged 35	*Man aged 25*	*Woman aged 25*
Investigations carried out on admission			
Arterial pH	7.529	7.44	7.43
$Paco_2$ (kPa) on air	3.89	4.2	4.32
Pao_2 (kPa) on air	8.0	6.6	8.8
Neutrophil count ($\times 10^9$/l)	16.6	12.0	12.9
C reactive protein (mg/l)	22	30	24
PEFR (l/min)	255 (65%)	230 (39%)	250 (63%)
Investigations carried out at 12 hours			
PEFR (l/min)	245 (63%)	520 (88%)	400 (100%)
FEV_1 (l)	0.95 (35%)	3.75 (94%)	2.9 (100%)
FVC (l)	3.02 (88%)	4.45 (92%)	3.87 (108%)
FEV_1/FVC (%)	31	84	93
K_{CO} (mmol/min/kPa/l)	1.58 (87%)	1.63 (87%)	1.75 (93%)

$Paco_2$, Pao_2 = arterial carbon dioxide and oxygen tensions; FEV_1 = forced expiratory volume in one second; FVC = forced vital capacity; K_{CO} = diffusion coefficient for carbon monoxide; PEFR = peak expiratory flow rate.

Discussion

Crack, which is a potent and highly addictive crystalline form of cocaine, is increasingly available in the United Kingdom, accounting for about 35% of all cocaine seized by police and customs.[1] Reported pulmonary complications of crack include pulmonary haemorrhage, pneumothorax, pneumomediastinum, asthma, obliterative bronchiolitis, hypersensitivity pneumonitis, non-cardiogenic pulmonary oedema, eosinophilic lung disease and persistent gas transfer abnormalities.[2 3]

We report a new pulmonary complication, characterised by acute onset interstitial pneumonitis which resolved rapidly in a few hours without specific therapy, which was almost certainly due to an adulterant rather than the crack itself. Our three cases all developed acute respiratory symptoms and fever immediately after smoking the same batch from one dealer. They reported a reduction in the usual euphoria suggesting that the crack had been adulterated. They had never experienced these

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% pure[6] and may contain various additives including sugars, ephedrine, caffeine, lignocaine, talc, strychnine, and quinine.[4] 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 persisted[2] and where there has been evidence of pulmonary eosinophilia in lung biopsy or bronchoalveolar 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.

1 Institute for the Study of Drug Dependence (ISDD). *Drug notes 5. Cocaine and crack*. Revised 1993.
2 Ettinger NA, Albin RJ. A review of the respiratory effects of smoking cocaine. *Am J Med* 1989;**87**:664–8.
3 Forrester JM, Steele AW, Waldron JA, Parsons PK. Crack lung: an acute pulmonary syndrome with a spectrum of clinical and histopathological findings. *Am Rev Respir Dis* 1990;**142**:462–7.
4 Shannon M. Clinical toxicity of cocaine adulterants. *Ann Emerg Med* 1988;**17**:1243–7.
5 Hannon DJ, Adler AG. Crack abuse: do you know enough about it? *Postgrad Med* 1990;**88**:141–6.
6 Rinfret M. *Cocaine price, purity and trafficking trends*. NIDA Research Monograph 1991;**110**:297–303.
7 O'Donnell AK, Mappin G, Sebo TJ, Tazelaar H. Interstitial pneumonitis associated with "crack" cocaine abuse. *Chest* 1991;**100**:1155–7.
8 Oubeid M, Bickel JT, Ingram EA, Scott GC. Pulmonary talc granulomatosis in a cocaine sniffer. *Chest* 1990;**98**:237–9.
9 Cooper CB, Bai TR, Heyderman E, Corrin B. Cellulose granulomas in the lungs of a cocaine sniffer. *BMJ* 1983;**286**:2021–2.
10 Oh PI, Balter MS. Cocaine induced eosinophilic lung disease. *Thorax* 1992;**47**:478–9.

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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 which has become established as a recreational drug in the UK since the late 1980s. Both cases recovered without incident, but it is important that the possible complications of this drug be publicised so that at risk subjects can be diagnosed early and managed appropriately.
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Keywords: Ecstasy, pneumomediastinum.

Ecstasy, or 3,4-methylenedioxymethamphetamine (MDMA), is a semi-synthetic hallucinogenic compound related to amphetamine and mescaline. In the UK, MDMA became re-established 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,[1] 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, unrelated to posture, worse on 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 apyrexial and tachycardic. There was no evidence of surgical emphysema. Cardiovascular examination revealed a pericardial crunch (Hamman's sign),[2] but otherwise examination was unremarkable. His chest radiograph showed the presence of a pneumomediastinum which was confirmed by echo-