

## Commentary

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These two case reports highlight some uncommon but important respiratory conditions associated with two recreational stimulant drugs, Ecstasy and crack cocaine, which are both increasingly used in the UK.

The pattern of cocaine use over the last 20 years has shifted toward inhalation of its purer freebase form, in part because inhaling crack is easier than other methods of administration and the instantaneous euphoric effect is said to be subjectively equivalent to intravenous use. Unlike the well known complications of cocaine use which relate to stimulation of the sympathetic system, the problems with inhaled freebase cocaine are less well recognised and understood. As reported by Kon *et al* in their case report, both acute and chronic lung syndromes associated with crack inhalation are now described in the literature. These various disparate entities have been collectively called "crack lung".

Kon *et al* describe three cases of "crack lung" thought to have been caused by an impure preparation. Shortly after smoking crack from a common source, these patients presented in respiratory distress with interstitial infiltrates which rapidly resolved with supportive treatment only. Similar cases of self limiting crack associated acute pulmonary syndromes consistent with a picture of pulmonary oedema have been reported.<sup>1</sup> The pathogenesis of crack induced pulmonary oedema is not entirely clear. Possible theories include transient left ventricular dysfunction following increased systemic vascular resistance or changes in the alveolar capillary membrane permeability from toxicity of cocaine or adulterants.

The question of an adulterant as a causative agent has been addressed directly in few reports. One report<sup>2</sup> found, as did Kon *et al*, no chemical evidence of an adulterant to account for the pulmonary oedema. It is possible that the separate reports of crack induced pulmonary oedema were caused by similar adulterants rather than the crack alone. Crack cocaine is, however, relatively pure. Few of the usual adulterants in cocaine hydrochloride are able to survive the extraction process. The combination of ammonia and sodium bicarbonate described in this case is unusual, although they are often used individually in extraction. This unusual combination may have produced a causative agent.

Previously published reports of crack induced pulmonary oedema often report a high dose of cocaine<sup>3</sup> in excess of 1 g. It would be interesting to know the quantities of crack consumed in this case. Parallels can be drawn with heroin induced pulmonary oedema, a well recognised entity related to overdose. This,

together with acute infections, especially HIV related, aspiration pneumonitis, alveolar haemorrhage, eosinophilic pneumonias and cardiac causes, should be included in the differential diagnosis in this setting.

Distinguishing between direct effects of the drug itself, dose related phenomena, the role of adulterants, and the complication of multi illicit drug usage in the aetiology of this acute pulmonary syndrome is difficult.

Management of these cases is usually supportive with resolution within days. A separate group presenting with infiltrates may follow a different course.<sup>4</sup> In these cases an interstitial pneumonitis associated with eosinophilic infiltrates has been shown in lung biopsy specimens. These cases can develop respiratory failure and corticosteroids have been an effective treatment.

In summary, the report highlights the need to consider illicit drugs and, in particular, inhaled crack when an otherwise healthy young person presents with acute respiratory distress and diffuse infiltrates on their chest radiograph.

Ecstasy (3,4-methylenedioxymethamphetamine) is a designer drug which has received much publicity in recent months following fatalities associated with its use. With its increasing use in the UK, primarily during raves, various serious adverse reactions have emerged including a hyperthermic syndrome, hepatotoxicity, neurological and psychiatric disorders.

The cases reported by Rezvani *et al* highlight a further rare adverse effect of Ecstasy – namely, the association between its ingestion and the development of pneumomediastinum. It is already known that an association exists between inhaled illicit drugs such as crack cocaine and pneumomediastinum.<sup>1</sup> The postulated mechanism for this association has been of an increase in intrathoracic pressure due to the Valsalva manoeuvre, mouth to mouth breathing used to heighten the drug effects, or violent coughing triggered by the crack. This causes alveolar rupture and dissection of air via the interstitium to the mediastinum.

The mechanism of Ecstasy induced pneumomediastinum, as pointed out by the authors, may be multifactorial. The one previous case report in the literature attributed this to vomiting<sup>5</sup> which can cause pneumomediastinum not only by increasing intrathoracic pressure but by oesophageal perforation which should always be excluded. A direct pharmacological effect of Ecstasy or that of adulterants is also possible.

The authors mention that spontaneous pneumomediastinum can be caused by violent exercise. Indeed, the paper they cite<sup>6</sup> suggests that physical exertion is a contributory factor in 25% of cases of spontaneous pneumomediastinum. It is highly likely therefore that, during the intense prolonged vigorous dancing of a rave, enough physical exertion occurs to cause pneumomediastinum. These are likely to be exercise induced rather than directly induced by Ecstasy. Further information on

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whether the first patient reported took his Ecstasy at a rave would have been useful, and the amount of physical exertion undertaken by both patients would have been relevant to dissecting out the aetiology.

It is interesting that the context in which Ecstasy is taken in the UK appears to define a unique spectrum of adverse reactions. The intense dancing in the hot environment of a rave with inadequate rehydration can exacerbate Ecstasy induced hyperthermia, with associated metabolic acidosis, disseminated intravascular coagulation, rhabdomyolysis, and acute renal failure.<sup>7</sup> This sequence of events is rarely seen if Ecstasy is not mixed with physical exertion. Spontaneous pneumomediastinum may be a further rave specific adverse reaction of Ecstasy to be added to the list.

The report by Rezvani *et al* highlights the need to consider the diagnosis of pneumomediastinum as well as pneumothorax and

pneumopericardium in young illicit drug users with pleuritic chest pain. This is true whether they inhale their drugs or not, and is especially important to consider because the clinical signs are not always obvious.

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- 4 Forrester JM, Steele AW, Waldron JA, Parsons PE. Crack lung: an acute pulmonary syndrome with a spectrum of clinical and histopathologic findings. *Am Rev Respir Dis* 1990;142:462-7.
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- 6 Abolnick I, Lossos IS, Breuer R. Spontaneous pneumomediastinum: a report of 25 cases. *Chest* 1991;100:93-5.
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## Expandable metal stents for non-malignant bronchial obstruction

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### Abstract

An expandable metal stent was inserted to relieve bronchial obstruction following lobectomy for localised squamous carcinoma which had not been relieved by bronchoplasty with a Goretex flap. This resulted in substantial improvement in lung function and exercise tolerance for nine months, following which severe inflammation around the stents required residual pneumonectomy.

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In recent years bronchial stents have been used as palliative measures for bronchial obstruction in malignant conditions, but seldom in benign disease. The following case illustrates the potential use of such stents in a non-malignant setting.

### Case report

A 53 year old woman presented with haemoptysis and a left upper lobe mass on her chest radiograph. After staging this was shown to be a localised squamous carcinoma and she

underwent a left upper lobectomy with stapling of the bronchial stump. Postoperatively she developed shortness of breath with wheeze at 24 hours and rigid bronchoscopy revealed severe narrowing of the left lower lobe bronchus at the level of the resected upper lobe. At immediate reoperation the diagnosis was made of bronchial stenosis from bronchial tenting (excess resection of the upper lobe origin from the main bronchus caused by excess pulling with the stapler). The narrowed segment of the residual main bronchus was widened along the whole length of the affected bronchus using a patch of Goretex (surgical bronchoplasty); a sleeve resection was not suitable because of the length of its stenosis. The patch appeared satisfactory and she made a good early postoperative recovery with a reasonable exercise tolerance.

Six weeks later she had become short of breath on 10 minute exertion and on lying down; she had a wheeze, and persistent cough not responding to antibiotics or bronchodilators. The wheeze was most prominent on the left side and there was poor air entry to the left lung. Fibrooptic bronchoscopy revealed bronchial stenosis from the bronchoplastic flap which collapsed in inspiration. The patient was referred for endoscopic stenting. Spirometric tests showed a forced expiratory volume in one second ( $FEV_1$ ) of 1.36 litres and a forced vital capacity (FVC) of 1.80 litres. Further investigation did not reveal any evidence of tumour recurrence. Rigid bronchoscopy confirmed occlusion of the left lower bronchus due to inward bulging of the Goretex with some additional distal stenosis.

Three Gianturco expandable wire stents, 2.5 cm long by 2 cm maximum external diameter, were sequentially inserted along the narrowed bronchus, resulting in visibly improved pat-

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