most. Neck veins are rarely used, probably due to a combination of difficulty with access and fear of carotid artery puncture. Given the large numbers of intravenous drug abusers, it is not surprising that complications associated with accidental intra-arterial injection sometimes occur. However, a search yielded only two reports of injury to the subclavian artery (both false aneurysms).23

The well demarcated territory outlined in the figure suggests that the site of injection was the thyrocervical trunk. This is the largest branch arising from the first part of the subclavian artery and supplies the shoulder girdle, and parts of the neck and thyroid.4

The pathophysiology of ischaemia from intra-arterial injection is complex. Small vessel embolisation, chemical endarteritis, toxic effect on the endothelium, and vasospasm have all been implicated.5-6 There remains considerable debate about the exact role played by vasospasm. Initial work on an animal model showed no evidence of vasospasm with drugs of abuse.7 However, reduced blood flow was noted when substances frequently “cut” with drugs (for example, corn starch and talcum) were injected into arteries. More recently a paper examining the effect of vasodilator drugs on intra-arterial injection related limb ischaemia concluded that the success of these drugs was related in part to the presence of vasospasm.8

It is recommended that all patients sustaining intra-arterial injection should be admitted for observation.7 The need for further treatment is based on the presence or absence of distal pulses. Where there is evidence of large vessel involvement, angiography is indicated. Various treatments have been used successfully to maintain limb circulation including surgery, heparin, thrombolysis, vasodilator drugs, and prostacyclin infusions.9-11 There have been no randomised trials determined to optimise treatment.

In the patient described here there was no evidence of distal large vessel involvement. The signs visible at the time of presentation represent reactive hyperaemia secondary to initial ischaemia; these changes have previously been described in the upper limb and are known as a “hand trip”.12 This patient’s signs and symptoms rapidly resolved with conservative treatment. Heroin is water soluble, which partly explains why there was little damage to the microcirculation. This is in contrast to temzepam, another drug used by intravenous drug abusers in Glasgow, which solidifies in the microcirculation, often with catastrophic effects.13 The presence of an arterial bruit at the time of first presentation which spontaneously resolved remains unexplained, but may have been due to intimal damage or local haematoma.

This case report outlines a rare presentation of a complication of parenteral drug misuse. Given an aging population of intravenous drug abusers who have increasing difficulty finding venous access, doctors working in A&E departments will undoubtedly see similar complications in the future. They should be aware of the potentially serious nature of intra-arterial injection and should refer these patients to vascular surgeons at an early stage. Finally, the reattendence of the same patient 10 days later, this time with a heroin overdose, suggests that serious complications may not be deterrent to continued substance abuse.

Eccstasy induced retropharyngeal emphysema

Madu Onwudike

Abstract

This is the first reported case of isolated retropharyngeal emphysema caused by ingestion of the amphetamine derivatives “Ecstasy” and “Speed”. The same complication has been reported with marihuana, cocaine, and heroin abuse. The condition resolved spontaneously and this seems to be the experience of others who have reported cases of cervical emphysema and pneumomediastinum associated with substance abuse. Because of the self limiting nature of this condition, extensive investigations may not be neces-


Homerton Hospital,
London E9
M Onwudike

Correspondence to:
Madu Onwudike FRCSEd
1 Thornwood Close, South Woodford, London E18
18H, United Kingdom.

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sary but hospital admission and close observation are still mandatory.

Key terms: amphetamines; ecstasy; cervical emphysema; retropharyngeal emphysema

Case report
A 22 year old male presented to the accident and emergency department with a 24 hour history of epigastric pain and left sided and anterior cervical pain. The pain was severe and associated with marked odynophagia. The symptoms appeared 24 hours after the patient had consumed amphetamine derivatives which included "speed" (methylamphetamine hydrochloride) and "ecstasy" (3,4-methylenedioxyamphetamine). He had taken 1 g of ecstasy by mouth and 1 g of speed, half of which was inhaled while the other half was dissolved in water and ingested. He was a regular drug abuser and had used similar amounts of the same drugs from the same batch two days previously. The only side effect he had suffered in the past was epigastric discomfort, usually relieved by milk.

There was no history of trauma. He had initially dismissed his symptoms as simply a sore throat. However, he reported to hospital when the pain worsened and when he noticed crepitations in his neck. He did not vomit or retch. He had developed a slight cough but there was no fever.

On examination, he was comfortable, afebrile, and had a pulse rate of 78 beats/min and a blood pressure of 110/70 mm Hg. There was tenderness and crepitation on the left side of the neck. Ear, nose and throat examination did not reveal any abnormality. The trachea was central and the chest was clear. There was mild epigastric tenderness but the rest of the abdomen was soft and non-tender. The chest x-ray was normal and in particular there was no pneumomediastinum. The cervical x-ray (figure) showed free air in the retro-pharyngeal space and soft tissues of the neck.

He was admitted for observation. A Gastrografin swallow performed the next day did not show any oesophageal leak. His white cell count was 7.3 × 10⁹/litre and fell to 3.5 × 10⁹/litre after 48 hours.

The surgical emphysema resolved after 72 hours and patient was able to eat and drink normally. He was discharged four days after admission.

Discussion
Causes of cervical emphysema and pneumomediastinum have been extensively reviewed elsewhere. The pathological anatomy and physiology have been comprehensively described by Maunder et al and by Rose and Veach.

Most cases of non-traumatic cervical and mediastinal emphysema are thought to be due to rupture of marginal alveoli and subsequent dissection of air along the pulmonary vascular sheath to the hilum. From the hilum decompression occurs along natural fascial planes to the neck. The alveoli ruptures because of a sudden increase in pressure (barotrauma) often associated with a Valsalva manoeuvre. This is believed to be the mechanism for the cervical emphysema seen in asthma, cough, weight lifting, labour, and intravenous and inhalational drug abuse. It has also been reported that in cases of drug abuse there is a general reduction in lung interstitial pressure which facilitates alveolar rupture by increasing the pressure gradient.

The pathophysiology also explains the frequent coexistence of drug related cervical emphysema and pneumomediastinum. Riccio and Abbott have reported a case of isolated retropharyngeal emphysema associated with free-basing cocaine which resolved spontaneously. It would appear that those presenting very early are more likely to be found with pneumomediastinum. After a variable length of time, the air tracks into the neck to present as cervical and retropharyngeal emphysema.

Review of published reports suggests that cervical and mediastinal emphysema associated with drug abuse or due to barotrauma from other causes is usually benign and self limiting. This has led many authors to conclude that extensive investigations are often unnecessary in the typical young and otherwise fit male patient. Our patient had a Gastrografin swallow and most clinicians would consider this investigation essential for differential diagnosis.

Because of the potential for the development of life threatening complications such as pneumothorax, tension pneumomediastinum, air block (occlusion of the airways due to the splinting action of air within the connective tissue of the lung), and pulmonary vascular obstruction, hospital admission is mandatory. Management should in most cases be limited to bed rest, observation, and reassur-
An unusual cause of hiccups

S Perry, J Stevenson

Abstract
A case of persistent hiccup associated with cavitating pulmonary tuberculosis is reported. Though tuberculosis presenting with hiccup is rare, tuberculosis is again on the increase and clinicians should remain alert to the possibility of this diagnosis.


Key terms: hiccup; pulmonary tuberculosis

Case report
A 52 year old unemployed plasterer presented to an accident and emergency department with a five day history of hiccups. He complained of weight loss, haemoptysis, and night sweats. He was a cigarette smoker of 35 years, intermittently abused alcohol, and had been exposed to tuberculosis as a young adult. There was a past medical history of vagotomy and pyloroplasty at the age of 24 years and transthoracic vagotomy at the age of 30 years for peptic ulcer disease.

On examination he was hiccuping, hoarse, emaciated, and pyrexial (37.5°C). Clinical examination of the chest revealed left upper lobe collapse but was otherwise normal.

A chest x ray showed emphysema, elevation of the left diaphragm, and irregulat cavitating opacities in the left upper lobe with hilar lymphadenopathy (figure). The patient was admitted for further investigation with a provisional diagnosis of carcinoma of the lung.

There was a normochromic normocytic anaemia (Hb 10.8 g/dl), raised erythrocyte sedimentation rate (24 mm per hour), and raised alkaline phosphatase (171 U/litre; normal range 45-140 U/litre). Sputum was positive for acid- and alcohol-fast bacilli, identified as Mycobacterium tuberculosis. Bronchoscopy showed no evidence of a carcinoma.

Two days after admission he was started on rifampicin 120 mg, isoniazid 50 mg, and pyrazamide 300 mg four times daily, with ethambutol 1 g daily. Chlorpromazine 25 mg three times daily was prescribed on admission to control the hiccups, which stopped three days later. He was discharged after four weeks of inpatient care and is currently under review.

Discussion
Persistent hiccup lasting more than 48 hours is uncommon, and tuberculosis presenting as hiccup is rare.1 The incidence of tuberculosis is again increasing. In Scotland there were 2033 cases reported in 1965, followed by a steady decline to 425 cases in 1987. By 1993 reported cases had increased to 460.2

Hiccup is an involuntary forceful inspiration, with poorly understood pathophysiology. A reflex arc has been proposed with the phrenic nerves, vagi, and T6-12 sympathetic fibres as the afferent limb.3 The “hiccup centre” is thought to be located in either the brainstem respiratory centre or the cervical cord between segments C3 and C5.4 The efferent limb is the phrenic nerve.5 Whether hiccup has a purpose is unclear. Hiccups are common in utero and may be a primitive reflex to prevent amniotic fluid aspiration6 or to prepare the respiratory muscles for breathing after delivery.7 Some investigators have suggested that hiccups may have no physiological function.8

Most episodes of hiccup associated with acute gastric distention and alcohol ingestion are short lived and resolve spontaneously. Chronic hiccup is defined as an attack lasting longer than 48 hours.9 Such episodes may be
Ecstasy induced retropharyngeal emphysema.

M Onwudike

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