



Pneumomediastinum and subcutaneous emphysema post cocaine and amphetamine insufflation

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Abstract

A 20-year-old gentleman presented with blood-streaked vomitus after insufflation of an unknown amount of powder cocaine and amphetamine. This was taken with an unspecified amount of alcohol. Other notable symptoms were dysphagia, chest pain, palpitations and the patient reported a 'crunchy' sensation in his chest. A chest x-ray revealed pneumomediastinum and a computerised tomography (CT) trauma confirmed these findings with associated subcutaneous emphysema without an identifiable cause. Follow up investigations included a barium swallow and gastroscopy which showed no obvious perforation but mild gastritis and duodenitis. He was managed conservatively with proton pump inhibitor cover and his symptoms resolved.

Keywords

Cardiothoracic surgery, clinical radiology and imaging, clinical diagnostics, emergency medicine, Boerhaaves syndrome, oesophageal rupture

Introduction

Cocaine use is prominent in the UK with it being the second most used illicit drug after marijuana.¹ It is therefore important to highlight possible complications from its insufflation, both to educate healthcare professionals and prevent unnecessary investigations. Cocaine can be used in various forms; its powder preparation is mainly used for intranasal use or injection, its freebase form also known as crack cocaine is mainly smoked.

The usual symptoms of cocaine intoxication are those related to a hyperadrenergic state. This may include general psychiatric manifestations such as agitation, paranoia and euphoria but may present with more worrying features such as palpitations, chest pain and in some cases loss of consciousness. Where such symptoms are present, it would be prudent to investigate for potentially treatable complications of cocaine use. Such complications include acute coronary syndrome, psychosis, rhabdomyolysis, stroke, and ventricular arrhythmias to name a few.²

Case presentation

A 20-year-old gentleman presented to the emergency department with severe nausea, retching and vomiting following cocaine and amphetamine use over a two-day period. This was taken with an unknown amount of alcohol. He described the vomitus as being streaked with blood though not frank haematemesis. He also had symptoms of dysphagia, chest pain, palpitations and a 'crunchy' feeling in his chest. His past medical history includes smoking, well controlled asthma on inhalers and attention deficit hyperactivity disorder.

With an Early Warning Score of 1 on admission, only his respiratory rate was at the higher end of normal measuring 20 breaths per minute, with other observations being within range. On occasions, his heart rate was elevated at around 110 beats per minute with a subsequent electrocardiogram (ECG) simply revealing a sinus tachycardia. The clinical examination was unremarkable except for the characteristic sign of subcutaneous crepitation in his neck indicating subcutaneous emphysema in this area.

Investigations

A multitude of blood tests were performed to aid diagnosis including a full blood count demonstrating a leucocytosis of $24.7 \times 10^9/L$. It has been reported that amphetamine use can induce a leucocytosis but there is no strong evidence to suggest cocaine is a factor.³ An acute kidney injury (AKI) stage 1 was identified with a creatinine of 154 µmol/L which was thought to be due to gastric losses. The creatine kinase level measured 553U/L which was slightly elevated but not in keeping with rhabdomyolysis.

Paracetamol and salicylate levels were within normal parameters. In terms of his liver function test, there was mild derangement with alanine aminotransferase 121 U/L, alkaline phosphatase 137 U/L and total bilirubin 43 µmol/L with a normal coagulation profile. Electrolyte levels were grossly normal except phosphate levels measured 0.46 mmol/L and calcium levels were 2.64 mmol/L. An arterial blood gas was performed

which demonstrated a mixed alkalotic picture with lactate levels measuring at 1.6 mmol/L.

The treatment was primarily rehydration and replacement therapy for management of cocaine and amphetamine overdose, AKI, and electrolyte disturbances. Initial treatment included intravenous fluid, omeprazole, and phosphate infusions alongside antibiotics to cautiously cover for infection given the possibility of oesophageal rupture.

Imaging

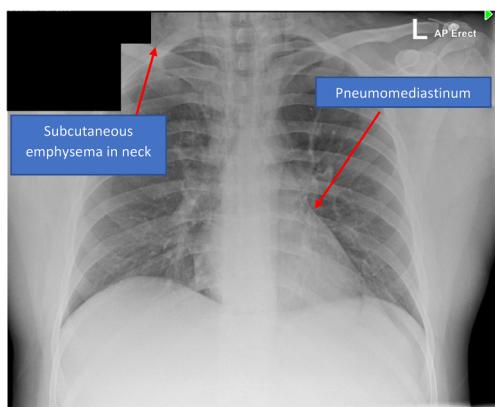
In terms of imaging, a chest X-ray was performed which demonstrated pneumomediastinum with evidence of subcutaneous emphysema (Figure 1). A CT scan was performed which showed no obvious perforation and no other source of emphysema noted (Figure 2). The cardiothoracic team were notified, and they deemed that these findings may be secondary to oesophageal rupture, also known as Boerhaaves syndrome. A barium swallow and gastroscopy were performed which demonstrated no perforation or structural abnormality but there was evidence of gastritis and duodenitis.

As no identifiable cause was found, this was treated as cocaine induced pneumomediastinum and subcutaneous emphysema with air leak likely occurring from the lungs. The mainstay of treatment for this condition is supportive unless perforation has occurred. This gentleman continued to improve with watchful waiting and within a few days his symptoms settled. He was discharged home after 7 days of hospital stay.

Discussion

Barotrauma is a rare but recognised complication of cocaine insufflation with some of the notifiable findings

Figure 1. Chest X-ray demonstrating pneumomediastinum and subcutaneous emphysema in the neck.



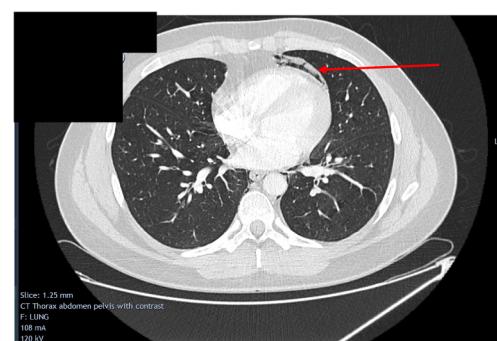
being pneumomediastinum, pneumothorax, pneumorrhachis and subcutaneous emphysema.⁴ There have been few documented cases of the above complications because of cocaine use.^{5–7} The mechanism by which cocaine induces air within the mediastinum is poorly understood but a common theory is that related to pressure changes within the thoracic cavity. Valsalva manoeuvres are commonly performed following snorting of cocaine to maximise the drug's euphoric effect.⁸ This, as well as the predisposition to coughing following its intake, increases intrathoracic pressure causing barotrauma within the chest cavity, thus allowing air to escape into surrounding regions.⁵

It has been reported that forceful emesis can lead to Boerhaaves syndrome, a potentially fatal complication that carries mortality rates of up to 40%.⁹ This can arise from excessive vomiting and was thus an important pathology to exclude in this young, previously healthy individual. Thankfully, this was ruled out with the investigations carried out, but remains an important differential diagnosis in patients presenting with evidence of barotrauma.

Conclusion

- Pneumomediastinum or subcutaneous emphysema secondary to barotrauma can occur because of cocaine insufflation. This is a rare but important clinical finding following cocaine abuse that should be identified early.
- It is an important complication to identify to ensure appropriate investigations are performed and management can be directed appropriately.
- Once more serious underlying pathologies causing pneumomediastinum have been excluded, this can be managed conservatively and is generally considered a benign condition.¹⁰

Figure 2. Ct trauma of chest showing pneumomediastinum.



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