

Pneumopericardium: Rare Complication of Cocaine Abuse

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ABSTRACT

A 29-year old male presented at the emergency department (ED) with chest pain and localised tenderness of the neck after snorting cocaine. Physical exam showed moderate subcutaneous emphysema on the right side of the neck. No ST-elevation compatible with cocaine-induced cardiac ischemia was seen on ECG and blood analysis was normal (negative troponins). Chest X-ray revealed subtle evidence of pneumomediastinum. Further workup with chest CT-scan confirmed subcutaneous emphysema with a pneumopericardium, large pneumomediastinum and a small pneumothorax. A conservative approach was pursued and the patient was kept overnight for observation. He was discharged from the ED with ambulatory follow up. A control chest CT, performed two weeks later, showed complete resolution of the pneumopericardium, -mediastinum and -thorax.

Pneumopericardium, -mediastinum and -thorax are rare conditions reported after cocaine abuse. A conservative approach is mandated and the outcome is usually uncomplicated.

Introduction

Widespread cocaine abuse leads to an increase in cocaine-related Emergency Department (ED) visits. Increasing amount of reports on respiratory and cardiovascular effects after cocaine abuse is published. Chest pain is the most common complaint of patients with cocaine-associated ED visits [1,2]. Cocaine activates the sympathetic nervous system leading to vasoconstriction, acute rise in arterial blood pressure, tachycardia and arrhythmias. Most frequent cardiovascular complications are acute myocardial infarction and cardiac arrhythmias. Pneumopericardium, -mediastinum and -thorax after cocaine abuse are rarely reported so far and in particular pneumopericardium is extremely rare [1,3].

Case Report

A 29-year old male (weight: 58kg, length: 178cm, BMI: 18.3 kg/m²; BMI normal range: 18.5 kg/m² – 25 kg/m²), without past medical history, presented at the ED with chest pain and localised tenderness of the neck. After attending a social gathering he presented sudden onset of mild oppressing continuous retrosternal chest pain, not irradiating, without any other symptoms (no dyspnea or cough; no nausea or vomiting). He had not encountered any

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trauma and reported healthy the days before. He takes no medications but admits cannabis use on a weekly basis as a teenager, but stopped doing so since he was 25 years old. Hereafter he only smoked marijuana on occasions (on average once a month) and hadn't smoked marijuana in the month prior to admission. He used cocaine once before, approximately one year before admission, and admitted and having snorted cocaine for the second time at the party the night before. He doesn't use any other drugs. As he couldn't sleep due to the cocaine-related sympathetic activation and the continuous chest pain, he smoked some one joint (mix of tobacco and marijuana, approximately 0.32gr marijuana [4]) to ease the pain. He declared smoking marijuana as a normal cigarette without performing prolonged inhalation or Valsalva's manoeuvre. Apart from smoking (5 pack years) he has no other cardiovascular risk factors. Waking up around 12 am he still experienced retrosternal chest pain with slight increased intensity. Additionally he noticed local tenderness at the right side of the neck without muscle tenderness, throat pain or dyspnea. Since pain increased despite taking 1gram paracetamol he presented at the ED.

His vital signs were within normal limits (temperature: 36,1 °C, heart rate: 65 bpm, blood pressure: 125/75 mmHg and equal both sides, respiratory rate: 13/min, saturation 100% on ambient air, GCS 15/15). Physical examination revealed normal respiratory auscultation without adventitious sounds, normal cardiac sounds without murmurs or clear *Hamman's sign* (a crunching sound synchronous with the heartbeat best heard over the precordium; suggestive for pneumopericardium or -mediastinum) [5-7] nor pulsus paradoxus. Thoracic examination revealed moderate subcutaneous emphysema on the right side of the neck. Further physical exam was unremarkable.

Blood analysis was normal (normal haematology and biochemical analysis, no inflammation, troponins < 0,012 ng/mL). Arterial blood gas on ambient air showed a respiratory alkalosis without metabolic compensation (pH 7,50, pCO₂ 29,3 mmHg, pO₂ 119,7 mmHg, base

excess 0,3 mmol/L, HCO₃⁻ 22,2 mmol/L, saturation 98,2%, lactate 1,3 mmol/L). ECG revealed sinus rhythm without ST-elevation and normal repolarisation. Chest X-ray showed subtle evidence of pneumomediastinum and right-sided mild subcutaneous emphysema in the neck (fig. 1.A. thick arrow) with air tracking in the centre of the mediastinum (Figure 1.A. thin arrow). There is a small denser line along the right cardiomedial margin (Figure 2) that is often seen in normal X-rays and attributed to an optical illusion called the *Mach Band effect* (visual pattern due to an edge enhancement which manifests as a region of lucency adjacent to convex surfaces). Although in association with subcutaneous emphysema and air tracking in the centre of the mediastinum it's a sign compatible with pneumomediastinum [8]. Since subcutaneous emphysema may indicate presence of pneumomediastinum further workup with chest CT-scan was performed, which confirmed subcutaneous emphysema with a pneumopericardium, large pneumomediastinum and a small pneumothorax (Figure 1B and Figure 3). A conservative approach was pursued and the patient was kept overnight for observation (continuous cardiac and pulse oximetry monitoring and two-hourly blood pressure measurement as well as pain evaluation by the Visual Analogue Scale) and oxygen therapy. Complete pain relief was achieved by analgesics (paracetamol and tramadol). As parameters remained within normal range and clinical re-evaluation was reassuring with resolution of chest pain, he was discharged the next day with expectative approach, oral analgesics and ambulatory follow-up. Control CT after two weeks revealed complete resolution of the free intrathoracic air.

Discussion

Spontaneous pneumopericardium, -mediastinum and -thorax are rare conditions that have been reported after cocaine abuse but diagnostic and therapeutic guidelines remain debatable. Moreover no guidelines for pneumopericardium are described in the current ESC (European Society of Cardiology) guidelines for pericardial diseases [9,10]. Pneumopericardium is an

extremely rare complication of cocaine abuse with so far only 9 published cases to the best of our knowledge [11-18]. So far, the incidence of any relationship between the use of cocaine and spontaneous pneumopericardium is not known. A study with a systematic toxicology screening in patients presenting with spontaneous pneumopericardium/pneumothorax, might solve this question.

Pneumopericardium, -mediastinum and -thorax are defined as the presence of 'free' air in respectively the pericardium, the mediastinum or the pleural cavity [7,19]. It can either be discovered without clear aetiology, referred to as spontaneous or primary, or either secondary to a specific pathologic event (trauma, infection, iatrogenic) [6]. Cocaine-related pneumopericardium, -mediastinum and -thorax are considered as spontaneous or primary as determining the precise source of 'free' air is very difficult and its mechanism remains unclear.

Presumable pathophysiology of pneumopericardium is a pulmonary barotrauma versus microscopic tracheal or oesophageal tear due to the solid contaminants in the crystalline mass inhaled or snorted by the patient [11,15]. Pulmonary barotrauma, also known as the *Macklin effect*, can be explained by a three-step process: alveolar rupture due to abrupt increase in intra-alveolar pressure which leads to air dissection along bronchovascular sheaths, with eventual spreading of the pulmonary interstitial emphysema into the mediastinal and pericardial cavity [7,20-22]. This is the result of either sudden increase in intra-thoracic pressure due to quick nasal insufflation, coughing, sneezing or vomiting, or either deep, forced and prolonged inhalation with Valsalva's manoeuvre [1,3]. Our hypothesis is that the patient's pneumopericardium, -mediastinum and -thorax is the result of a sudden rise in intra alveolar pressure due to cocaine snorting.

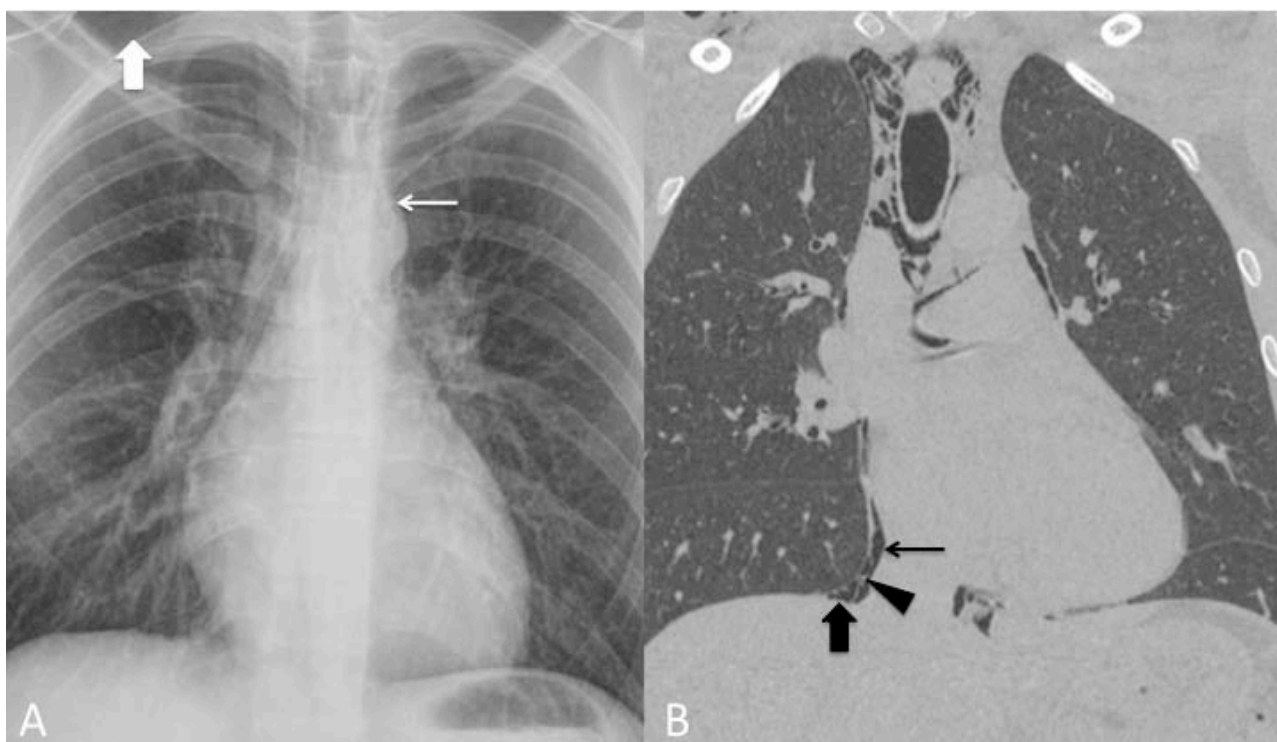


Figure 1: Chest X-ray (A) and Chest CT (B).

On both pictures subcutaneous emphysema is apparent at the right side of the neck (thick white arrow) as well as air tracking in the centre of the mediastinum indicative for mediastinal emphysema or pneumomediastinum (thin white arrow). Chest CT reveals a pneumopericardium (thin black arrow) and a small pneumothorax (thick black arrow). The pericardium can be seen between the pneumopericardium and pneumothorax (black triangle).

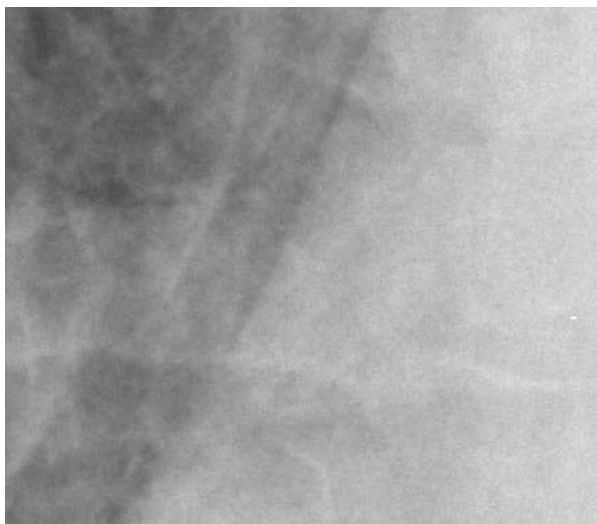


Figure 2: Mach band effect versus pneumomediastinum. Zoom in on the right cardiomeastinal border showing the edge enhancement along the cardiac margin.

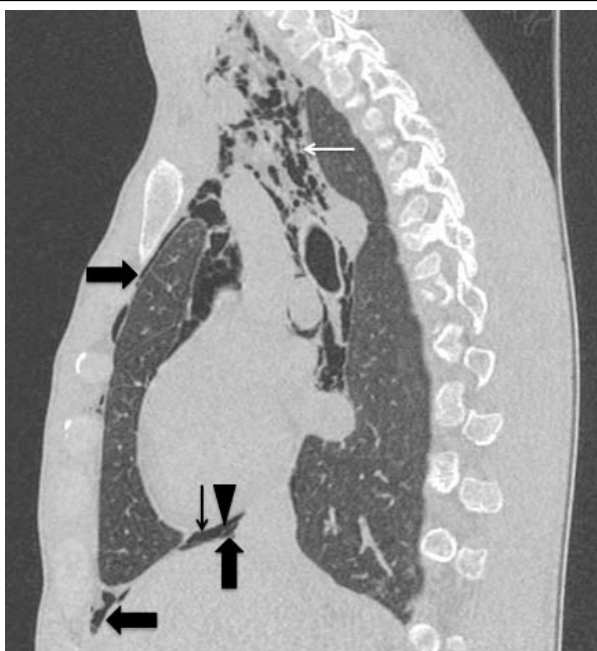


Figure 3: Chest CT showing extensive subcutaneous and mediastinal emphysema compatible with large pneumomediastinum (white arrow), a pneumopericardium (thin black arrows; with pericardium marked by the black triangle) and small pneumothorax (thick black arrows).

The patient denied coughing or vomiting and didn't perform prolonged inhalation or a Valsalva's manoeuvre. As he declared already having chest pain before smoking marijuana and having smoked marijuana as a normal cigarette without Valsalva's manoeuvre; marijuana is less likely to be the cause of the pneumopericardium, -mediastinum and -thorax.

Just as the patient described in our case, most published patients presenting with spontaneous

pneumopericardium and/or pneumomediastinum are young, thin, males without previous medical history. The most frequent complaint is chest pain, followed by neck pain, dyspnea and cough. Less frequent symptoms are odynophagia, hoarseness and feeding problems [7,9,15,17]. Emergency physicians should be alert for patients meeting these criteria as they have a considerable higher risk of developing pneumopericardium and/or pneumomediastinum.

Spontaneous pneumopericardium and -mediastinum can be diagnosed on plain chest X-ray although chest CT remains the gold standard. A CT-scan is also helpful in excluding secondary causes of pneumopericardium. Further invasive diagnostic studies are not routinely recommended in spontaneous pneumomediastinum and should only be performed in highly suspicious cases of oesophageal or tracheal rupture [9]. In all published cases so far, no patient was diagnosed with oesophageal rupture. Since our patient didn't present any digestive complaints and he declined performing prolonged Valsalva's manoeuvre we didn't perform invasive diagnostics such as oesophagoscopy/gastroscopy or bronchoscopy. Echocardiography may be useful in evaluating unstable patients with suspicion of tamponade due to the pneumopericardium. As our patient was stable during the whole observation and didn't present a pulsusparadoxus no echocardiography has been performed.

Current guidelines for spontaneous pneumothorax and pneumomediastinum recommend a conservative approach with outpatient follow-up in selected patients with minimal or no symptoms. These patients should receive clear advice to return in case of worsening dyspnea, chest pain or fever [19,23]. Treatment of spontaneous pneumopericardium is not clearly identified since the rarity of its presentation. It is considered a benign disease, which responds well to conservative treatment, consisting of bed rest, oxygen therapy and analgesic medications. All reported cases were treated conservatively with complete resolution over a period of one to two weeks. Oxygen therapy should be considered as its consumption increases the diffusion

pressure of nitrogen in the interstitium, promoting the rapid absorption of free air in the mediastinum and possibly as well in the pericardium [7,9]. In none of the cases the patients developed pericarditis or mediastinitis. Therefore antibiotic treatment should only be considered in patients at risk for pericarditis (presenting with fever, blood sample showing elevated inflammatory parameters or pneumopericarditis secondary to oesophageal rupture) and should not be initiated empirically [9].

Conclusion

Cocaine-related complaints are seen more often in the ED as cocaine abuse is increasing in current society. Chief complaint of cocaine abuse is chest pain. Although pneumomediastinum and pneumothorax after cocaine abuse have been reported, they are rare complications; yet pneumopericardium is even more rare and not well documented. The emergency physician should be attentive for other pathologies than solely the most common (acute myocardial ischemia or arrhythmia) in patients with chest pain after cocaine abuse. Patients meeting the criteria of a young thin male without previous medical history have an increased risk of developing pneumopericardium- and/or pneumomediastinum. Short observation with oxygen therapy and cardiac monitoring may be considered. When ECG shows no signs of cardiac ischemia or arrhythmia and serial cardiac marker testing is negative in asymptomatic or mildly symptomatic patients with spontaneous pneumopericardium an expectative approach is warranted since most patients have spontaneous resolution within one or two weeks.

References

1. Cregler LL, Mark H. (1986). Special report Medical complications of cocaine abuse. *N Engl J Med.* 315: 1495-1500.
2. Restrepo CS, Carrillo JA, Martinez S, Ojeda P, Rivera AL, et al. (2007). Pulmonary Complications from Cocaine and Cocaine-based Substances: Imaging Manifestations. *RadioGraphics.* 27: 941-956.
3. Underner M, Perriot J, Peiffer G. (2017). Pneumomediastinum and cocaine use. *Press Med.* 46: 249-262.
4. Ridgeway G, Kilmer B. (2016). Bayesian inference for the distribution of grams of marijuana in a joint. *DrugAlcdep.* 165: 175-180.
5. Hamman L. (1937). Spontaneous interstitial emphysema of the lungs. *TrAssoc Am Physicians.* 52: 311-319.
6. Caceres M, Ali SZ, Braud R, Weiman D, Garrett HE. (2008). Spontaneous Pneumomediastinum: A Comparative Study and Review of the Literature. *Ann Thorac Surg.* 86: 962-966.
7. Sahni S, Verma S, Grullon J, Esquire A, Patel P, (2013). Spontaneous Pneumomediastinum: Time for Consensus. *North Am J Med.* 5: 460-464.
8. Zylak CM, Standen JR, Barnes GR, Zylak CJ. (2000). Pneumomediastinum Rivisted. *RadioGraphics.* 20: 1043-1057.
9. Kim KS, Jeon HW, Moon Y, Kim YD, Ahn MI. (2015). Clinical experience of spontaneous pneumomediastinum: diagnosis and treatment. *J Thorac Dis.* 7: 1817-1824.
10. Adler Y, Charron P, Imazio M, Badano L, Esquivias GB, et al. (2015). 2015 ESC Guidelines for the diagnosis and management of pericardial diseases. *Eur Heart J.* 36: 2921-2964.
11. Albrecht CA, Jafri A, Linville L, Anderson HV. (2000). Cocaine-Induced Pneumopericardium. *Circulation.* 102: 2792-2794.
12. Katzir D, Klinovsky E, Kent V, Shucri A, Gilboa Y. (1989). Spontaneous Pneumopericardium: Case Report and Review of the Literature. *Cardiology.* 76: 305-308.
13. Leitman BS, Greengart A, Wasser HJ. (1988). Pneumomediastinum and pneumopericardium after cocaine abuse. *Am J Roentgenol.* 151: 614.
14. Savader SJ, Omori M, Martinez CR. (1988). Pneumothorax, Pneumomediastinum and pneumopericardium: complications of cocaine smoking. *J Fla Med Assoc.* 75: 151-152.

15. Adrouny A, Magnusson P. (1985). Pneumopericardium from cocaine inhalation. *N Engl J Med.* 313: 48-49.
16. Garcipérez de Vargas FJ, GómezBarrado JJ, Luis MoyanoCalvente S, Amaya García MJ, Marcos G. (2013). Pneumopericardium and pneumomediastinum in a diabetic and cocaine user patient. *EndocrinolNutr.* 60: e3-4.
17. Brander L, Ramsay D, Dreier D, Peter M, Graeni R. (2002). Continuous left hemidiaphragm sign revisited: a case of spontaneous pneumopericardium and literature review. *Heart.* 88: e5.
18. Schweitzer VG. (1986). Osteolytic sinusitis and pneumomediastinum: deceptive otolaryngeologic complications of cocaine abuse. *Laryngoscope.* 96: 206-210.
19. MacDuff A, Arnold A, Harvey J. (2010). Management of spontaneous pneumothorax: British Thoracic Society pleural disease guideline 2010. *Thorax.* 65: 18-31.
20. Macklin CC. (1939). Transport of air along sheaths of pulmonic blood vessels from alveoli to mediastinum. *Arch Intern Med.* 64: 913-926.
21. Wintermark M, Schnyder P. (2001). The Macklin Effect: A Frequent Etiology for Pneumomediastinum in Severe Blunt Chest Trauma. *Chest.* 120: 543-547.
22. Murayama S, Gibo S. (2014). Spontaneous pneumomediastinum and Macklin effect: Overview and appearance on computed tomography. *World J Radiol.* 6: 850-854.
23. Tschopp JM, Bintcliffe O, Astoul P, Canalis E, Driesen P, et al. (2015). ERS task force statement: diagnosis and treatment of primary spontaneous pneumothorax. *EurRespir J.* 46: 321-335.