



## Case report

## Bilateral spontaneous pneumothoraces with spontaneous pneumomediastinum: An intravenous methamphetamine complication

Daniel Guck\*, Ryan Munyon

Department of Internal Medicine, Penn State Hershey Medical Center, USA

## ARTICLE INFO

## Keywords:

Pneumothorax  
Pneumomediastinum  
Inflammatory markers  
Methamphetamine use

## ABSTRACT

**Objective:** To present an unusual case and proposed mechanism of bilateral spontaneous pneumothoraces with pneumomediastinum in a patient with intravenous methamphetamine use.

**Case report:** Thin white man presented with confusion and chest pain after intravenous methamphetamine use. Initial workup found bilateral pneumothoraces with pneumomediastinum. Conservative management was initiated and subsequent radiographs and physical examination revealed subsequent improvement in pneumothoraces and pneumomediastinum.

**Conclusion:** Intravenous methamphetamine use increases a wide number of inflammatory markers that can increase the risk of spontaneous pneumothoraces and pneumomediastinum. In patients with known risk factors, methamphetamine use can promote an increased incidence of spontaneous pneumothorax and pneumomediastinum.

## 1. Introduction

Spontaneous pneumothorax with simultaneous pneumomediastinum is an extremely rare complication of methamphetamine use. Spontaneous pneumothorax occurs when there is disruption of the pleural lining allowing air to enter the pleural space. Spontaneous pneumomediastinum occurs in a three-step process in which alveoli rupture, air dissects along the bronchovascular sheaths, and air spreads into the mediastinum [1]. On CT this often presents as linear collections of air along the bronchovascular sheaths (Macklin effect) [2]. Both spontaneous pneumothorax and pneumomediastinum have similar population risk factors: tall and thin body habitus, white, male, and active smoking status [3,4]. Both of these conditions also have an increased incidence in individuals who use the illicit drug methamphetamine [5]. It has been shown that methamphetamine increases inflammatory markers in rats [5] as well as cell signalling leading to cell apoptosis [6]. The proposed mechanism for both of these conditions is that inflammatory markers and barotrauma from methamphetamine use disrupt the alveolar and bronchial membrane leading to these conditions.

## 2. Case presentation

A 28-year-old thin white man with no significant past medical history presented to the emergency department with complaints of

confusion and chest pain after being found naked by police in his front yard. Patient admitted to use of intravenous methamphetamine earlier that day and had trouble remembering the events over the course of the day. Upon arrival to the emergency department, patient was evaluated by the trauma team. Initial vital signs revealed heart rate 100 beats per minute, respiratory rate 22 breaths per minute, and oxygen saturation of 99% on room air. Trauma evaluation, including CT of head and spine was unremarkable for fractures or spinal cord injury. CT chest revealed extensive pneumomediastinum and subcutaneous emphysema along the entire thorax into the neck and tracking along the retroperitoneum down to the level of the iliac crests (Fig. 1), as well as small bilateral pneumothoraces (Fig. 2). The pneumomediastinum caused concern for esophageal tear, however, water soluble swallow study was negative for esophageal pathology. Due to hemodynamic stability, there was no indication for surgical intervention.

After admission to the hospital, the patient was given supplemental oxygen therapy to maintain oxygen saturation of approximately 100% for two days. He had resolving chest pain and was weaned from the supplemental oxygen use. On physical examination, auscultation over the chest wall had subsequent improvement in Hamman's sign and subcutaneous crackles. Chest radiographs three days after admission revealed resolution of bilateral pneumothoraces and interval improvement in pneumomediastinum. He ambulated well without increased dyspnea or chest pain. Psychological counseling was performed for drugs of abuse before the patient was discharged to a drug

\* Corresponding author. 500 University Dr, Hershey, PA 17033, USA,  
E-mail address: [dguck@pennstatehealth.psu.edu](mailto:dguck@pennstatehealth.psu.edu) (D. Guck).

<https://doi.org/10.1016/j.rmcr.2018.05.025>

Received 6 June 2017; Received in revised form 25 May 2018; Accepted 25 May 2018

2213-0071/© 2018 The Authors. Published by Elsevier Ltd. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).



Fig. 1. CT chest revealing subcutaneous emphysema and pneumomediastinum.

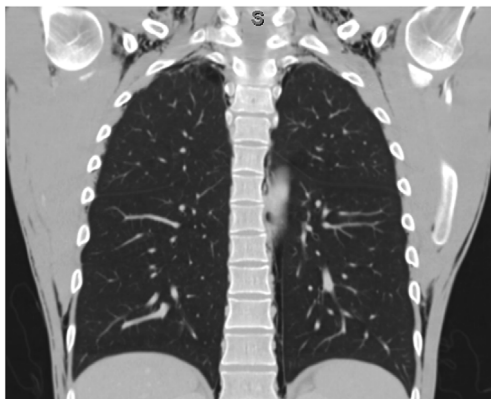


Fig. 2. CT chest revealing small bilateral apical pneumothoraces and neck subcutaneous emphysema.

rehabilitation facility.

### 3. Discussion

Risk factors for primary spontaneous pneumothorax include smoking, family history, Marfan syndrome, homocystinuria, and thoracic endometriosis [7]. Secondary spontaneous pneumothorax typically occur in those with established severe lung diseases, such as cystic fibrosis, necrotizing pneumonia, or lung malignancy [8].

Some of the mechanisms for spontaneous pneumothorax and pneumomediastinum from the inhaled formulation of methamphetamine are typically thought to be due to the barotrauma with violent drug inhalation as well as thermal injury [5]. It is also known that the inhaled formulation of methamphetamine can have a dose-dependent increase of inflammatory markers such as tumor necrosis factor (TNF)-alpha and interleukin-6 [9]. Cocaine is another illicit stimulant that, in either the inhaled or intravenous formulation, can increase interleukin-8 which is associated with neutrophil-mediated alveolar toxicity [10]. It is then proposed that similar inflammatory markers that cause alveolar damage in the inhaled formulation can also cause alveolar damage in the intravenous formulation of methamphetamine. Furthermore, if methamphetamine is used in a patient with baseline risk factors for

primary spontaneous pneumothorax or spontaneous pneumomediastinum, the risk of either complication is theoretically increased.

Patients who are clinically stable and have small pneumothoraces can be managed conservatively with supplemental oxygen, but larger pneumothoraces should be managed with a chest tube [11]. Treatment of spontaneous pneumomediastinum is mostly supportive [12]. In the absence of trauma, in patients with dyspnea and findings concerning for spontaneous pneumothorax or pneumomediastinum, the clinician should also inquire as to illicit substance abuse.

### 4. Conclusion

This case reflects that the risk of both spontaneous pneumothorax or pneumomediastinum is increased with the use of intravenous methamphetamine. The proposed mechanism is from an increase in inflammatory markers. In the absence of trauma, patients that present with signs and symptoms of spontaneous pneumothorax or pneumomediastinum should be screened for methamphetamine use. Treatment of pneumothorax is variable depending on the severity of disease, whereas treatment of pneumomediastinum is typically conservative.

### Conflicts of interest

None.

### Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

### References

- [1] S. Sahni, S. Verma, J. Grullon, A. Esquire, P. Patel, A. Talwar, Spontaneous pneumomediastinum: time for consensus, *N. Am. J. Med. Sci.* 5 (8) (2013) 460–464.
- [2] S. Murayama, S. Gibo, Spontaneous pneumomediastinum and Macklin effect: overview and appearance on computed tomography, *World J. Radiol.* 6 (11) (2014) 850–854.
- [3] M. Alnas, A. Altayeh, M. Zaman, Clinical course and outcome of cocaine-induced pneumomediastinum, *Am. J. Med. Sci.* 339 (1) (2010) 65–67.
- [4] J.B. Jougon, M. Ballester, F. Delcambre, T. Mac bride, C.E. Dromer, J.F. Velly, Assessment of spontaneous pneumomediastinum: experience with 12 patients, *Ann. Thorac. Surg.* 75 (6) (2003) 1711–1714.
- [5] W. Tseng, M.E. Sutter, T.E. Albertson, Stimulants and the lung : review of literature, *Clin. Rev. Allergy Immunol.* 46 (1) (2014) 82–100.
- [6] Y.H. Gu, Y. Wang, Y. Bai, M. Liu, H.L. Wang, Endoplasmic reticulum stress and apoptosis via PERK-eIF2 $\alpha$ -CHOP signaling in the methamphetamine-induced chronic pulmonary injury, *Environ. Toxicol. Pharmacol.* 49 (2017) 194–201.
- [7] Y. Guo, C. Xie, R.M. Rodriguez, R.W. Light, Factors related to recurrence of spontaneous pneumothorax, *Respirology* 10 (2005) 378.
- [8] M. Noppen, T. De Keukeleire, *Pneumothorax Respiration* 76 (2008) 121.
- [9] S.M. Wells, M.C. Buford, S.N. Braseth, J.D. Hutchison, A. Holian, Acute inhalation exposure to vaporized methamphetamine causes lung injury in mice, *Inhal. Toxicol.* 20 (9) (2008) 829–838.
- [10] G.C. Baldwin, D.M. Buckley, M.D. Roth, E.C. Kleerup, D.P. Tashkin, Acute activation of circulating polymorphonuclear neutrophils following in vivo administration of cocaine. A potential etiology for pulmonary injury, *Chest* 111 (3) (1997) 698–705.
- [11] A.M. Kelly, D. Kerr, M. Clooney, Outcomes of emergency department patients treated for primary spontaneous pneumothorax, *Chest* 134 (2008) 1033.
- [12] W.L. Dajer-fadel, R. Argüero-sánchez, C. Ibarra-pérez, F.P. Navarro-reynoso, Systematic review of spontaneous pneumomediastinum: a survey of 22 years' data, *Asian Cardiovasc. Thorac. Ann.* 22 (8) (2014) 997–1002.