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SPONTANEOUS PNEUMOMEDIASTINUM IN A PATIENT WITH MARIJUANA USE

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ABSTRACT

The Macklin effect plays an essential role in the pathogenesis of spontaneous pneumomediastinum. It is the process by which is there is a blunt alveolar rupture that leads to air dissection through the bronchopulmonary sheaths and spreads into the mediastinum. Theoretically, marijuana use can cause spontaneous pneumomediastinum indirectly by inducing rigorous vomiting. We report a case of a healthy 22-year-old male with a history of recent marijuana use who presented with pneumomediastinum and rhabdomyolysis concurrently. After a thorough investigation, we concluded that this patient had spontaneous pneumomediastinum due to the Macklin effect from severe vomiting.

KEYWORDS

Spontaneous pneumomediastinum, Macklin effect

LEARNING POINTS

- Spontaneous pneumomediastinum is the presence of air in the mediastinum that occurs from an unclear aetiology.
- Marijuana use can trigger severe vomiting in patients, leading to spontaneous pneumomediastinum through the Macklin effect.

INTRODUCTION

Spontaneous pneumomediastinum is described as the presence of air in the mediastinum that occurs without a clear, causative aetiology such as trauma or oesophagotracheal injury. One rarely reported association of spontaneous pneumomediastinum includes marijuana use, in which patients often present with nausea and vomiting[1]. Cyclic vomiting from marijuana use and the different smoking techniques can lead to spontaneous pneumomediastinum through the Macklin effect[2]. The Macklin effect is a frequent aetiology for pneumomediastinum in severe blunt chest trauma and describes a three-step pathophysiologic process: there is blunt traumatic alveolar rupture, air dissection along broncho-vascular sheaths, and spread of this blunt pulmonary interstitial emphysema into the mediastinum[3].

CASE DESCRIPTION

A 22-year-old male with no past medical history presented to the emergency department with chest pain, body aches, muscle spasms, nausea and vomiting. His symptoms began

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the day before admission while working as a roofer under hot conditions with temperatures over 38°C. The patient had been unable to eat, drink or urinate, and had persistent nausea and vomiting. He denied shortness of breath, fever and haematuria. The patient used marijuana occasionally but did not smoke. The most recent marijuana use was the day before his symptoms began. On physical examination, his blood pressure was 138/88 mmHg, pulse 93 beats per minute and temperature 37°C; respirations were 20 breaths per minute and the oxygen saturation was 96% while the patient was breathing ambient air. The body habitus was sthenic with the impression of an athlete. The patient was in moderate distress, appeared dehydrated and had tenderness upon palpation of the neck and chest. The remainder of the examination was normal. The chest radiograph showed pneumomediastinum with subcutaneous emphysema of the neck and left chest wall without visible pneumothorax *(Fig. 1)*. A computerised tomography scan of the chest without contrast revealed extensive pneumomediastinum with a mass effect on the heart, extensive subcutaneous emphysema of the neck and left chest wall, and a possible subtle right basilar pneumothorax *(Fig. 2)*. The laboratory findings were significant for leukocytosis with neutrophilia (WBC 13,840/mm^3 and absolute neutrophil count 11,680/mm^3), hyperkalaemia (potassium level 5.4 mmol/l), acute kidney injury (BUN 44 mg/dl and creatinine level 5.4 mg/dl), hypercalcaemia (calcium level 11.3 mg/dl), and high creatinine kinase level (3,867 IU/l). A urine protein dipstick was positive for blood without red blood cells; the urine toxicology report was positive for cannabinoid substance. The diagnoses of rhabdomyolysis with acute kidney injury and pneumomediastinum were made. The patient was admitted to the Department of Internal Medicine. A fluoroscopic oesophagography was ordered to rule out oesophageal injury as a possible cause of the pneumomediastinum, and it later showed no evidence of extravasation. An aggressive intravenous fluid hydration was initiated. Given the patient's history of excessive vomiting, the pneumomediastinum was not found to be a cause of concern as it was consistent with his episodes of retching and caused no significant effects on cardiac function. No intervention was deemed necessary, and the patient was given ondansetron and morphine for nausea and pain, respectively. Following the admission, the rhabdomyolysis with acute kidney injury improved significantly, as well as symptoms of vomiting, and chest and neck pain. His heart rate decreased from 93 to 53 beats per minute and blood pressure decreased from 138/88 mmHg to 127/61 mmHg. The creatinine level decreased to 0.8 mg/dl and the creatinine kinase level went down to 4,700 IU/l. He was able to eat and produce a regular amount of urine. The patient was discharged on the third day of hospitalisation without a follow-up appointment.

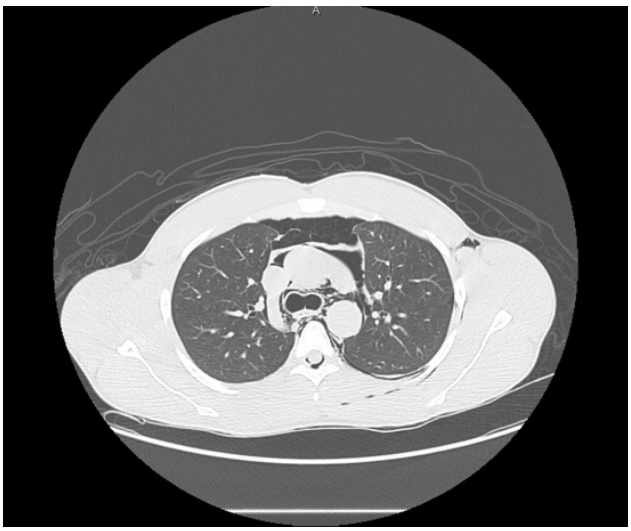


Figure 1. A chest X-ray shows subcutaneous emphysema on the left side of the neck and chest wall.

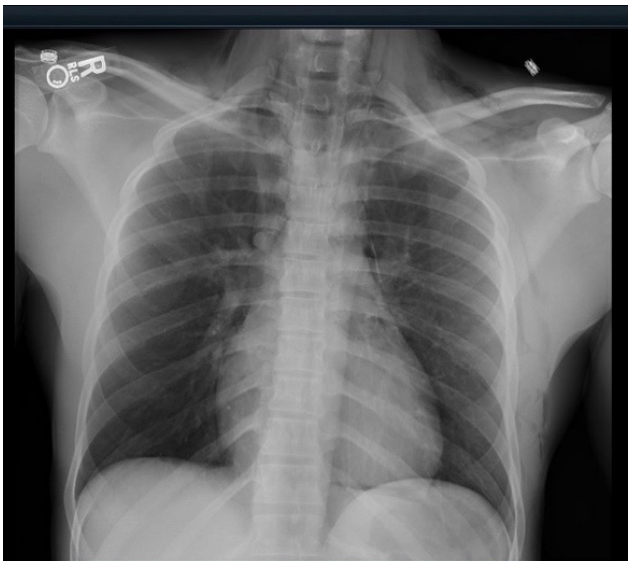


Figure 2. A CT scan of the chest shows pneumomediastinum and extensive subcutaneous emphysema on the left side.

DISCUSSION

This patient presented with classic clinical findings suggestive of pneumomediastinum, including chest pain, dyspnoea, subcutaneous emphysema and neck pain[4-8]. Two common aetiologies of pneumomediastinum are airway (tracheobronchial tree and alveoli) and the gastrointestinal tract. One of the concerning diagnoses of the latter is oesophageal perforation (i.e. Boerhaave syndrome), in which a patient rapidly develops signs and symptoms of mediastinitis and potentially septic shock[7]. Although illicit drug use is a well-known cause of spontaneous pneumomediastinum, only a few studies have associated marijuana with spontaneous pneumomediastinum. One study reported marijuana as a cause of spontaneous pneumomediastinum in 66.6% of patients with spontaneous pneumomediastinum[2]. Marijuana can cause spontaneous pneumomediastinum through the Macklin effect by two proposed mechanisms[2]. One is an attempt at inspiration against a closed airway, known as Müller's manoeuvre[2,9]. Many marijuana smokers use a pipe-like device that requires repeated Müller's manoeuvres[2]. Another is an attempt at forceful expiration against a closed airway, known as the

Valsalva manoeuvre (breathing technique during inhalation and vomiting)[2,10]. Marijuana can cause vomiting through acute and chronic toxicity[11,12]. Small doses of cannabis have anti-emetic properties; on the contrary, large doses of cannabis have pro-emetic properties[11]. An observational study regarding acute cannabis toxicity reported vomiting as one of the clinical manifestations in approximately 19% of all age groups[12]. Cannabinoid hyperemesis syndrome is a paradoxical syndrome characterised by severe vomiting, abdominal pain and nausea in a patient with long-term use of cannabis[11]. A distinctive feature of cannabinoid hyperemesis syndrome is the relief of symptoms in a hot bath or shower[11]. According to our literature review, spontaneous pneumomediastinum is a usually a benign condition that resolves itself[5,8]. Recommended ambulatory treatment includes rest and analgesia[6,7], and there is no need for hospitalisation apart from treating underlying causes[7]. Concerning complications including tension pneumothorax, air embolism and pneumopericardium are rare[5,7]. There are limitations to our study, however; we could not describe the inhalation technique used by this patient. Considering the timeline of symptoms in this patient, we believe that severe vomiting was an acute toxicity from marijuana use.

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