Unusual presentation of more common disease/injury

Pneumomediastinum after retching

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Summary

A 25-year-old woman with hirsutism presented to the Emergency Department of the medicine faculty in Gaziantep University with a 4-h History of dyspnoea, dysphagia, coughing, continuous and progressive chest pain radiating to the back and palpation of her neck. The physical examination was significant for diffuse neck and chest subcutaneous emphysema. The x-ray study and CT scan of the chest revealed pneumomediastinum. The patient reported that 6 h before presentation she took spironolactone due to hirsutism and she retched. Retching caused the symptoms. The patient was followed conservatively and fully recovered over the next 7 days. Spontaneous pneumomediastinum developed in this patient with no underlying lung disease, presumably from air leakage secondary to the excessive elevation of intrathoracic pressure due to retching. Pneumomediastinum is an uncommon disease arising most frequently and remains largely underdiagnosed clinically, especially in young, healthy patients.

BACKGROUND

Pneumomediastinum and subcutaneous emphysema are most often found in the setting of oesophageal and chest trauma. These conditions also occur spontaneously in the setting of elevated intrathoracic pressure as a result of mechanical ventilation, excessive coughing, vomiting, Valsalva manoeuvre, childbirth and forceful straining during exercise.^{1–3} It is a rare and self-limiting disease that usually occurs in young adult males without apparent concomitant factors or disease. It has a good clinical course with very rare recurrence and complications.^{4–5} A few cases of pneumomediastinum have been documented in association with cocaine inhalation and marijuana smoking.^{6–7} This is a case report of pneumomediastinum and subcutaneous emphysema occurred after retching due to taking spironolactone.

CASE PRESENTATION

A 25-year-old woman (weight, 56.5 kg; height 164 cm) was ordered spironolactone 200 mg/day due to hirsutism. She used this treatment for 3 days, but no complaints developed. She was admitted to our hospital because of dyspnoea, dysphagia, coughing, continuous and progressive chest pain radiating to the back and palpation of her neck. The pain was acute, severe, unrelenting, and diffuse and was exacerbated by deep inspiration. Six hours ago, she took two spironolactone tablets (without capsule) together, but did not exactly swallow the drugs. She had a sensation of retching and she retched once; then her complaints began. The patient denied any history of trauma, vomiting, coughing or similar symptoms before this episode. She denied any history of recent digestive system invasive procedures, current medications, or known allergies. Her family history was unremarkable.

On physical examination, the patient was calm and cooperative. Cardiac monitoring revealed normal sinus rhythm with a rate of 110/min, and a 12-Lead ECG revealing no abnormalities. On admission, she disclosed a

respiratory rate of 24/min, fever 36.5 °C and systemic blood pressure of 110/67 mm Hg. Snow grip sounds associated with heartbeat (Hamman's sign) were audible on the left anterior chest. No rales were noted on the lungs.

Laboratory data included the following values: haemoglobin, 14.3 g/dl; haematocrit, 41.5%; red blood cell count, $499 \times 10^4 \text{ mm}^3$; white blood cell count, $11\,000/\text{mm}^3$; C reactive protein, 0.37 mg/dl, aspartate aminotransferase, 39 IU; alanine aminotransferase, 31 IU; lactate dehydrogenase, 174 IU and creatine kinase, 31 IU. She had a medical history of operation 1 year ago due to endometrial polip. Chest x-ray showed mediastinum emphysema, with air extending to the tissue of the neck (figure 1). Chest CT revealed marked air accumulation around the aorta, trachea, and pericardium (figure 2). Deep cervical emphysema was also observed (figure 3).

Figure 1 Chest x-ray on admission showed mediastinum emphysema, with air extending to the tissue of the neck.

Figure 2 Chest CT revealed marked air accumulation around the aorta, trachea, and pericardium. Deep cervical emphysema was also observed.

OUTCOME AND FOLLOW-UP

Contrast oesophagogram and bronchoscope showed no abnormal findings. About 15 cm under first construction of lateral oesophagus a defect in 1.5 cm diameter was found by oesophagography.

Perforation was not detected. For the proceeding 4 days, the patient was given nothing orally, supported with nasogastric sonda and intravenous fluid hydration. Her complaints, subcutaneous emphysema, Hamman's sign, and abnormal shadows on chest x-ray disappeared without any surgical treatment. She remained clinically stable and was discharged home after 7 days tolerating a

regular diet. At a clinic visit 2 weeks after discharge, the patient was doing well, with no recurrence of symptoms.

DISCUSSION

Pneumomediastinum, or mediastinal emphysema, is the presence of air in the mediastinum.

Spontaneous pneumomediastinum (SPM) was first reported by Hamman⁸ in 1939, occurs without an apparent precipitating factor or diseases in younger and thin people. SPM is relatively rare and has a benign, self-limited course. Several authors reported that some patients with SPM have a current and previous history of bronchial asthma⁹; the others have anorexia nervosa. SPM has been well known, and may be caused by manual straining, cough bouts, shouting, sneezing, inhalation use, childbirth and lung function testing.¹⁰ This group most commonly presents with subcutaneous emphysema, chest pain, and neck or cervical discomfort.^{4 11 12} Other common symptoms may include dysphonia, dysphagia and coughing. The diagnosis of SPM is often suggested by a thorough patient history and physical examination and confirmed on diagnostic imaging. The clinical diagnosis of subcutaneous emphysema can be made on physical examination by palpating subcutaneous air. Pneumomediastinum often can be diagnosed on a plain chest x-ray study.^{4 5 13-15} As shown in our case, signs of pneumomediastinum on chest x-ray study include radio-lucent streaks from free gas that may be vertical as it tracks along the left side of the heart, retrosternal, pre-cardiac or peri-tracheal. In some documented cases of pneumomediastinum, the plain chest x-ray study does not always demonstrate the pneumomediastinum, and a chest CT scan is necessary to make the diagnosis.¹⁶⁻²⁰ This above scenario is a classic case of pneumomediastinum, a relatively rare clinical entity with incidence reported from 1 : 800 to 1 : 42 000 emergency department visits.²¹

SPM is distinguished from pneumomediastinum in that pneumomediastinum is secondary to an identifiable aetiology such as invasive procedure, mechanical ventilation, pneumothorax or other obvious disease process. Clinical presentation of SPM includes retrosternal chest pain with radiation to the back in 90% of cases. Persistent cough is described in 42%; dysphonia, throat or jaw pain in up to 25%²¹ and dyspnoea in approximately 8% of cases.²¹ Physical examination reveals subcutaneous air in 73% of

Figure 3 Cervical emphysema was also observed in lateral cervical x-ray.

reported cases.²² Hamman's sign, although less common, is pathognomic of pneumomediastinum.²¹ A low-grade fever may be present and is thought to be a result of cytokine release.²³ Vital signs may reveal only slight tachycardia in a normotensive patient. The common mechanism is considered to be sudden increased intrathoracic pressure induced by an effected Valsalva manoeuvre, but some patients have no evident triggers. A pathogenetic sequence of SPM, delineated by Macklin,²⁴ was that when the air pressure in the alveoli increases rapidly, free air can leak from ruptured alveoli and produce interstitial emphysema, resulting in an accumulation of air along the bronchovascular tissue sheath and moving into the mediastinum. Although most people usually have no medical problems during air travel, air travel may present risks of pulmonary disorders, including pneumothorax and pulmonary thromboembolism, in some patients.²⁵

It is possible that, by trapped gases, a high intrathoracic pressure might be developed and induces consequent rupture of marginal pulmonary alveoli, allowing bubbles of air to dissect along the vascular sheaths and connective tissue planes to the mediastinum. The exact mechanism of pneumomediastinum in our patient is unknown. However, we suppose that her thin and tall build (body mass index, 16.9 kg/m²) and medical history of hirsutism have some relationship to the development of pneumomediastinum. Furthermore, we speculated that there were fragile areas such as bullae adjacent to the mediastinum, although the chest CT of our patient could detect no bullae and ruptured alveoli. Therefore, pressure change by retching influenced these areas and induced rupture of oesophageal mucosa.

Although largely considered a benign, self-limited entity, it is important to consider the rare but potential serious complications that can be associated with SPM, as well as the underlying disease processes that predispose the condition. Complications that should be considered and ruled out in these patients include current or impending airway compression, cerebral and coronary gas embolism, tension pneumomediastinum, pneumopericardium, oesophageal rupture and perforation of a viscus (eg, the oesophagus) or the tracheobronchial system. These pathologies should be searched for with any new-onset pneumomediastinum having concomitant dyspnoea, stridor, altered level of consciousness or haemodynamic instability.

Underlying medical conditions to consider in these patients include obstructive lung disease, gastro-oesophageal reflux disease, respiratory tract infection, epilepsy and bulimia; these medical conditions should be identified and medically managed. Finally, an attempt to elicit precipitating factors such as Valsalva manoeuvre, illicit drug inhalation, vigorous or cyclical vomiting, intractable hiccups, childbirth, weight lifting, scuba diving, and helium gas inhalation will likely help identify the specific aetiology.

Competing interests None.

Patient consent Obtained.

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