



Case report

Afebrile tension pyopneumothorax due to anaerobic bacteria: Fistula or gas production?

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ABSTRACT

Pyopneumothorax is characterized by a pleural collection of pus and air requiring emergent thoracic drainage. A 65-year-old diabetic woman presented with a two-week history of fatigue and dyspnea but without fever. Chest computed tomography showed extensive pleural effusion and air in the left pleural cavity, which caused a mediastinal shift with peripheral circulatory failure. There was no evidence of a pulmonary fistula. Anaerobic bacteria were found in the pus smear after microscopy. After 17 days of chest drainage and 18 days of antibiotic treatment, the patient recovered without any complications. The etiology of pyopneumothorax in this case was slowly progressive pyothorax due to gas-producing anaerobic bacteria. In conclusion, we should pay careful attention to serious infectious diseases, including pyothorax, in diabetic patients due to the high prevalence and subclinical symptoms.

1. Introduction

Pyopneumothorax is characterized by a pleural collection of pus and air. Pyothorax is a pleural infection with a mortality rate of 15% and requires early and appropriate treatment [1]. Tension pneumothorax is a life-threatening emergency and requires rapid thoracic drainage. Tension pyopneumothorax usually presents with cough, chest pain, dyspnea, and fever. These symptoms usually progress rapidly within several days because the major cause of pyopneumothorax is a pulmonary fistula due to pneumonia or lung abscess.

We report a case of afebrile tension pyopneumothorax with a gradual progression of symptoms over the course of 4 weeks.

2. Case presentation

A 65-year-old woman had a history of diabetes mellitus (DM) for more than 15 years without any complications. Her most recent glycosylated hemoglobin was controlled to 6.6% with glimepiride, vildagliptin, metformin, and voglibose. The patient had no history of poor oral hygiene or aspiration pneumonia. She presented with a two-week history of fatigue, appetite loss, cough, and dyspnea. She denied having fever in the past 4 weeks despite an elevated white blood cell count (WBC, 13,500 count/mm³) and C-reactive protein (CRP, 24.5 mg/L) in a

blood test performed 4 weeks previously. Since she had no fever or pain, she did not take any antipyretic analgesics or oral antibiotics for the past 4 weeks. Vital signs of the patient were as follows: blood temperature, 35.5 °C; pulse rate, 120/min; blood pressure, 149/86 mmHg; and oxygen saturation, 94% under room air respiration. The patient had pitting edema on her face and both lower legs. A blood test indicated elevated WBC (27,200 count/mm³) and CRP (358.1 mg/L) level.

Whole-body computed tomography (CT) showed extensive pleural effusion with slight bubbles and air in the left pleural cavity, which caused the collapse of the left lung and mediastinal shift to the right (Fig. 1A). We could not detect the presence of a pulmonary fistula that may have caused the left lung to collapse. There was no evidence of gastrointestinal to thoracic fistula or herniation of the stomach and colon into the thoracic cavity. She was admitted to the intensive care unit immediately due to tension pyopneumothorax, suggested by a loss of respiratory variability with a 15 mm diameter of the inferior vena cava (IVC) measured by an echocardiography and a lactate level of 10.6 mmol/L indicated by an arterial blood gas analysis.

During the drainage procedure, a large amount of gas with an intense foul odor was expelled. We placed a 28-French gauge thoracostomy tube on her left chest to avoid tube obstruction. From the tube, 1500 ml of yellow viscous pus drained without air-leakage. The lactate levels decreased to 4.2 and 1.2 mmol/L 1 and 6 hours after drainage,

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respectively. The pitting edema improved with 1400 ml of urination during the first 8 hours after chest drainage. Although *Actinomyces* sp. and some species of anaerobic bacteria were found in the pus smear microscopy, we could not detect the pathogenic bacteria from the pus culture.

We administered intravenous sulbactam and ampicillin and washed the thoracic cavity on day 3–5. Although intrapleural use of tissue plasminogen activator and DNase are effective to pyothorax, we used urokinase instead because they are not permitted by the Japanese insurance system [2]. The patient took analgesics as needed only for the first few days after the chest tube was placed; however, she had no fever since the admission. Chest CT performed on day 9 showed no pulmonary

or bronchopleural fistula (Fig. 1B). The thoracostomy tube was removed on day 17, and antibiotics were discontinued on day 18 because of a drug-induced rash. The patient was discharged on day 21 and recovered from pyopneumothorax without recurrence. Chest CT taken 8 months after the discharge showed full expansion of the left lung, with only slight fibrosis in the lung (Fig. 1C).

3. Discussion

We reported a case of slowly progressive and afebrile tension pyopneumothorax that may have been caused by gas-producing anaerobic bacteria. Although tension pyopneumothorax is a condition so rare

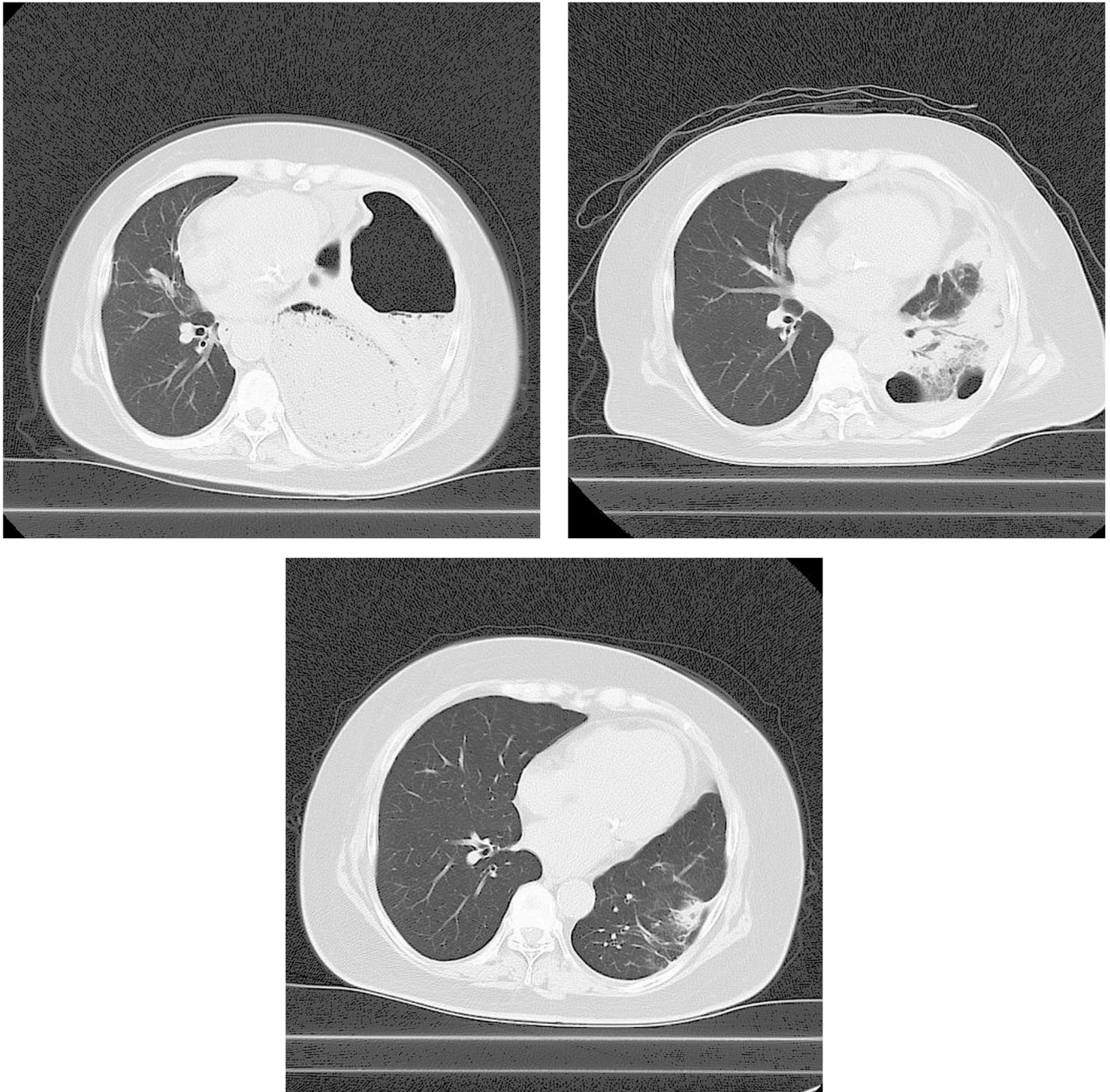


Fig. 1. Chest computed tomography (CT) images at the level of the lower lobe of the lung in chronological order. A was taken on the day of admission. The image shows a massive pleural effusion with slight bubbles, air in the left pleural cavity, collapse of the left lung, and mediastinal shift to the right. However, a pulmonary fistula that may have caused the left lung to collapse was not observed. B was taken 9 days after drainage. This image shows the modest expansion of the left lung, with remaining pleural gas, pulmonary consolidation, and fibrosis. C was taken 8 months after discharge. This image shows the improvement of the consolidation with the full expansion of the left lung.

that only 19 literature can be found on PubMed [3], this is the first report that tension pyopneumothorax can slowly develop during several weeks.

We failed to detect the pathogenic bacteria despite plenty of pus; however, the findings of the pus smear microscopy and the odor of the intrapleural gas suggested that the main pathogen was anaerobic bacteria which account for 30% of pyothorax [4]. Moreover, pyothorax associated with aspiration pneumonia is reported to be caused by mixed bacterial flora consisting of aerobic and anaerobic bacteria [5]. In contrast, we suspect that *Actinomyces* sp. was not the pathogenic bacteria in the present case despite the finding of the pus smear microscopy. Treatment for pulmonary actinomycosis requires prolonged high dose antimicrobial therapy, that is, intravenous beta-lactam antibiotics for 2–6 weeks and following oral antibiotics for 6–12 weeks [6]. Thus, the treatment course of the patient was inconsistent with pulmonary actinomycosis; the patient recovered from pyopneumothorax without recurrence by only 18 days of intravenous antimicrobial therapy.

Given the elevated inflammatory reaction in a blood test measured 4 weeks before admission, the patient may have already developed pneumonia or pyothorax at that point. Pyopneumothorax caused by a pulmonary fistula results in immediate collapse of the lung, which is inconsistent with this patient's course. Gas-production by some anaerobic bacteria can cause pyopneumothorax [7,8]. This etiology can appropriately explain the slow course of the patient. The loss of respiratory variability of IVC and the elevated lactate level suggested peripheral circulatory failure due to the mediastinal shift. Metformin-induced lactic acidosis, severe sepsis, and/or cardiac damage were assumed to be the differential diagnoses for the elevated lactate level. However, these causes were excluded by the rapid improvement of the lactate level and pitting edema within several hours after chest drainage.

The patient had no fever despite the development of pyothorax sufficient to accumulate 1500 ml of pus in the pleural cavity. Fever is one of the most common symptoms of pyothorax, being present in 80% of cases [4]. Afebrile pyothorax may be caused by the patient's immunocompromised condition due to DM or by the low virulence of the pathogenic bacteria. Indeed, the immunocompromised condition due to DM affects the prevalence of pyothorax. Comorbidity with DM is the second most frequent complication of pyothorax [4]. Mechanisms of immune dysfunction derived from DM consist of suppression of cytokine production, leucocyte recruitment, defect in pathogen recognition, dysfunction of leucocyte, and inhibition of antibody and complement effector [9]. These mechanisms, especially suppression of cytokine production including interleukin-1 and 6, may be associated with afebrile pyothorax.

In conclusion, pyopneumothorax, usually an emergency due to its rapid exacerbation, may develop slowly and without fever over several weeks. A gas-producing pyothorax by anaerobic bacteria may cause the situation due to the low virulence and/or host's immunocompromise state.

Patient consent

We obtained written informed consent for publication of this case

report and accompanying images.

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Disclosure statement

KF has received honoraria from Boehringer Ingelheim. TM has received honoraria from Bristol Myers Squibb, Chugai Pharmaceutical, AstraZeneca, Novartis, and Boehringer Ingelheim. The other authors have no conflicts of interest to declare.

Declaration of competing interest

KF has received honoraria from Boehringer Ingelheim. TM has received honoraria from Bristol Myers Squibb, Chugai Pharmaceutical, AstraZeneca, Novartis, and Boehringer Ingelheim. The other authors have no conflicts of interest to declare.

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