

Tension pyopneumothorax caused by *Burkholderia cenocepacia*: a rare case report in an immunocompetent individual

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Abstract

Tension pyopneumothorax is a medical emergency that occurs when air and pus build up in the pleural space. This case report

describes a 47-year-old diabetic female who presented with tension pyopneumothorax due to *Burkholderia cenocepacia*. The patient was successfully treated with a 24-French tube thoracostomy and antibiotics. To the best of our knowledge, this is the first case of tension pyopneumothorax caused by *Burkholderia cenocepacia* to be reported.

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Introduction

Tension pyopneumothorax is a medical emergency that occurs when air and pus build up in the pleural space, causing the affected lung to collapse and putting pressure on the heart and the other structures in the chest. It can quickly lead to respiratory failure and cardiac arrest if not treated promptly. Herein, we report a successful treatment of tension pyopneumothorax caused by *Burkholderia cenocepacia* in an immunocompetent individual.

Case Report

A 47-year-old female farmer presented with a cough with greenish expectoration and fever for 20 days. She also complained of dyspnea - Modified Medical Research Council (mMRC) functional class II for 20 days, which progressed to mMRC functional class IV for the preceding 2 days. She gave a history of left-side pricking chest pain for the previous 2 days. She denied any history of alcohol consumption or smoking. She reported loss of appetite and weight for the preceding 20 days. Her history was significant for type II diabetes mellitus. She did not suffer from any chronic lung diseases, such as cystic fibrosis. On physical examination, the patient was tachypneic; blood pressure was 84/50 mm of Hg, and pulse rate was 120 beats/minute. Room air oxygen saturation at presentation was 88%, and arterial blood gas analysis reported pH of 7.45, partial pressure of oxygen in the arterial blood of 48 mm of Hg, partial pressure of carbon dioxide of 33 mm of Hg, bicarbonate of 22, and sulfur dioxide of 88%. On chest auscultation, breath sounds were absent on the left side. A complete hemogram revealed anemia with leucocytosis viz. hemoglobin – 8.7 g/dL, total leucocyte counts – 16,700 per microliter, and platelet count – 453,000 per microliter. Hepatic, renal, and thyroid function tests were reported normal.

Chest roentgenogram showed left-side homogenous opacity with air-fluid level and mediastinal shift to the right (Figure 1a). Immediate left tube thoracostomy was done using a 24-French intercostal tube; 600 mL of pus was drained (Figure 1b). Following tube thoracostomy, the patient's room air saturation improved to 94%, and blood pressure increased to 118/74 mm of Hg. The patient was admitted and received antibiotics empirically, and continuous

drainage of pleural fluid was done. Blood cultures drawn before initiating antibiotics were sterile.

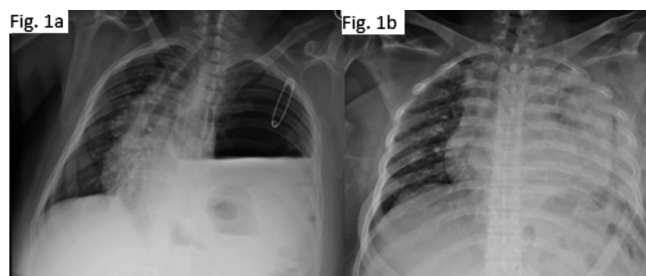


Figure 1. a) Chest roentgenogram showing left side hydropneumothorax with mediastinal shift to the right; b) post-tube thoracostomy chest roentgenogram.

Chest high-resolution computed tomography taken 3 days later showed left-sided moderate pleural effusion with multiple air pockets (suggestive of septated effusion) (Figure 2). As the chest tube drain was insignificant, we administered 250,000 units of streptokinase in 20 mL saline solution through the chest tube in an attempt at enzymatic debridement to avoid more invasive treatment. The pleural effusion showed resolution after three days of intrapleural streptokinase; then the chest tube was removed (Figure 3).

Initially, the patient was suspected to have tubercular empyema. However, empyema fluid and sputum PCR for mycobacterium tuberculosis were negative. The pleural fluid culture showed growth of *B. cenocepacia* sensitive to ceftazidime, co-trimoxazole, levofloxacin, meropenem, and minocycline (Figures 4 and 5). However, the sputum pyogenic culture was sterile. Based on the empyema fluid culture report, antibiotics were changed to inj. ceftazidime and inj. trimethoprim/sulfamethoxazole [1]. She received intravenous antibiotics for two weeks. She made a complete clinical



Figure 2. High-resolution computed tomography axial section showing left side pleural effusion with air pockets.

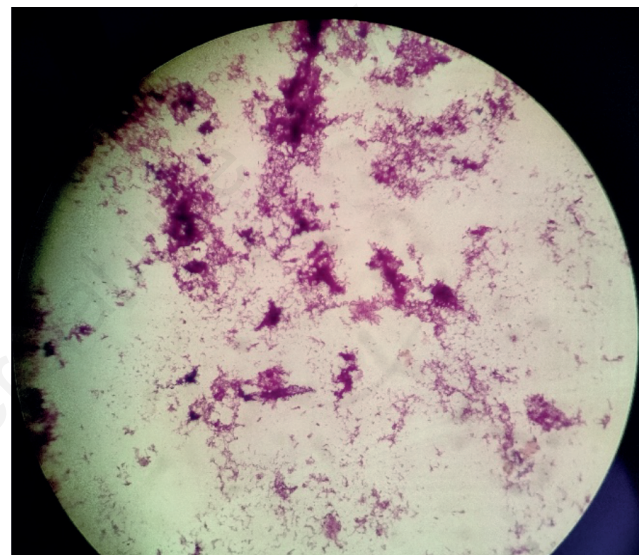


Figure 4. Gram stain showing gram negative bacilli.

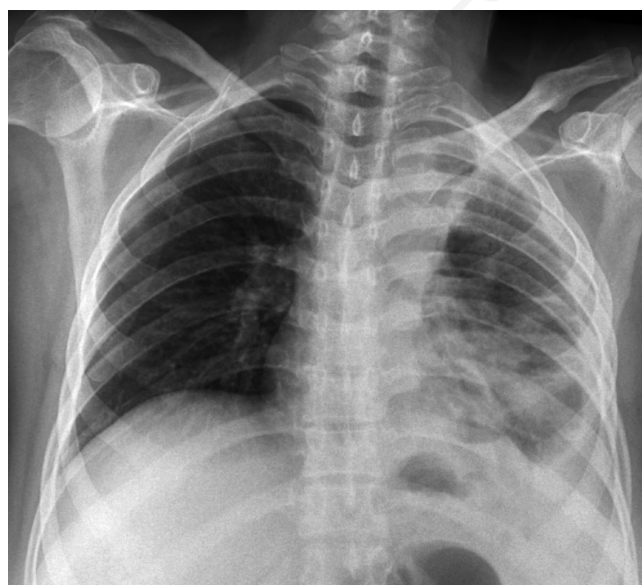


Figure 3. Chest roentgenogram showing improvement in pleural effusion post intrapleural streptokinase.

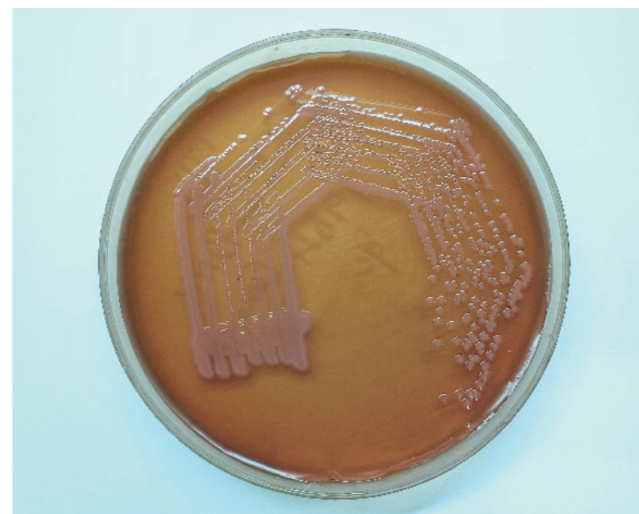


Figure 5. MacConkey agar showing non-lactose fermenting colonies.

recovery at the time of discharge. A follow-up chest roentgenogram showed evidence of radiological recovery.

Discussion and Conclusions

Tension pyopneumothorax is a life-threatening complication of pneumonia or lung abscess eroding into the pleural space. Pyopneumothorax develops by connection to the airways through a pleural defect, and tension pyopneumothorax may supervene if a one-way valve mechanism exists. Tension pyopneumothorax has also been described as a complication of *Streptococcus pyogenes* pharyngitis and ruptured esophagus secondary to Barrett's esophagus and Boerhaave's syndrome in an alcoholic patient with recurrent vomiting [2-4]. This is the first case of tension pyopneumothorax caused by *B. cenocepacia* to be reported. *B. cenocepacia* complex is ubiquitous in nature, and its members are most commonly found on plant roots, soil, and moist environments. William Burkholder, an American microbiologist, described it as the causative agent of bacterial rot in onion bulbs [5]. *B. cenocepacia* is a member of the *B. cepacia* complex. It constitutes a group of phenotypically similar but genetically distinct gram-negative aerobic bacilli. It is a widely known lung pathogen in patients with cystic fibrosis and chronic granulomatous disease. However, only a few cases of *B. cepacia* have been reported in immunocompetent individuals.

B. cepacia is also reported to be associated with renal failure requiring dialysis, indwelling central lines, recent abdominal surgery, and invasive procedures such as bronchoscopy or tracheostomy in ICU patients [6]. Serious comorbidities, including diabetes mellitus, chronic obstructive pulmonary disease, congestive heart failure, and malignancy, also act as risk factors for *B. cepacia* bacteremia [7]. Our patient was diabetic, but no other risk factors were present. In our patient, the source of infection could have been environmental, as she was a farmer by occupation. Moreover, the organism grown was pan-sensitive. No hospital outbreaks were reported before or after the isolation, thus making the possibility of nosocomial infection unlikely. Although *B. cepacia* is rarely known to cause community-acquired infections in immunocompetent patients, few cases have been reported. Karanth *et al.* reported a case of community-acquired *B. cepacia* infection presenting as pyopneumothorax in an immunocompetent individual [8].

Treatment of *B. cenocepacia* infection requires a multidisciplinary approach involving antibiotics, supportive care, and management of underlying comorbidities. There is no consensus on the optimal antibiotic regimen, and the choice of antibiotics should be based on local antibiotic susceptibility patterns and the severity of the

infection. In severe cases, combination therapy with multiple antibiotics may be necessary. Our patient was treated with a combination of ceftazidime and trimethoprim/sulfamethoxazole. Our patient had septated pleural effusion with an insignificant drain for which intrapleural streptokinase was given [9]. The patient was not affordable for tissue plasminogen activator and DNase.

To conclude, *B. cepacia* infection may also present outside the nosocomial setting, even in an immunocompetent individual. Appropriate antibiotic treatment should be initiated as per the sensitivity pattern at the earliest. Clinicians should be aware of the possibility of *B. cenocepacia* infection in patients with pyopneumothorax even in tubercular endemic regions.

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