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Spontaneous Pneumothorax After Rupture of the Cavity as the Initial Presentation of Tuberculosis in the Emergency Department

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Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Conflict of interest:

None declared

Patient: Male, 65-year-old
Final Diagnosis: Tuberculosis
Symptoms: Cough accompanied by greenish expectoration • chest pain • asthenia • weight loss
Medication: —
Clinical Procedure: Thoracic drainage tube and bronchoscopy
Specialty: Critical Care Medicine

Objective: Rare co-existence of disease or pathology

Background: Spontaneous pneumothorax can be secondary to a wide variety of lung diseases. Spontaneous pneumothorax secondary to pulmonary tuberculosis occurs in rare cases of residual fibrosis with retractions and bullae.

Case Report: We present the case of a 65-year-old male patient from a rural area in the province of Los Ríos in Babahoyo, Ecuador, with no history of contact with tuberculosis. The patient arrived at the Emergency Department of the Regional Hospital of the Instituto Ecuatoriano de Seguridad Social (IESS), Babahoyo, due to acute respiratory failure, preceded by 10 days of evolution due to cough accompanied by greenish expectoration, chest pain, asthenia, and weight loss. On chest radiography, a left pneumothorax and interstitial pulmonary infiltrate were reported. A chest tube was placed, and the patient was intubated and was placed on invasive mechanical ventilation due to severe respiratory failure.

Use of the GeneXpert MTB/RIF System detected *Mycobacterium tuberculosis* without resistance to rifampicin. Ziehl-Neelsen (ZN) staining for the identification of bacillus acid-resistant alcohol was positive in alveolar bronchial lavage.

MALDI-TOF mass spectrometry and phenotypic analysis showed the presence of *Pseudomonas aeruginosa* and *Klebsiella pneumoniae* with carbapenemases resistance mechanism, and the KPC type enzyme was identified. The culture for *Mycobacterium tuberculosis* was positive from the fourth week.

Conclusions: Secondary pneumothorax due to rupture of the polymicrobial cavity and especially of tuberculous origin is a very special form of acute respiratory failure in patients with previous structural pulmonary lesions in the Emergency Department.

MeSH Keywords: Caves • Intensive Care Units • Pneumothorax • Tuberculoma

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Background

Pneumothorax is defined as the presence of gas in the pleural space. A spontaneous pneumothorax typically occurs without history of chest trauma and can be classified as secondary or primary when it occurs in an individual with or without underlying lung disease, respectively [1]. Spontaneous pneumothorax can be secondary to a wide variety of lung diseases. Spontaneous pneumothorax secondary to pulmonary tuberculosis can occur in cases of residual fibrosis with retractions and bullae [2].

The estimated incidence of spontaneous pneumothorax associated with active pulmonary tuberculosis is only approximately 1–2%. Other pulmonary infections, such as necrotizing bacterial pneumonias and particularly *Pneumocystis jirovecii pneumonia* in patients with acquired immunodeficiency syndrome (AIDS), are associated with spontaneous pneumothorax [3].

Latent tuberculosis infection is characterized by an adaptive and specific immune response to *Mycobacterium tuberculosis* (MTB) complex antigens, with no evidence of clinically active TB. The microorganisms that cause latent infection can persist in an inactive phase for several decades, even during the entire life of the host; however, in approximately 5% of all infected individuals, the latent infection progresses to active replication and causes TB disease [4].

Patients with severe tuberculosis who require admission to the ICU typically present with acute respiratory failure, severe/sepsis, and shock/septic, and with multiple organ dysfunction. Patients with acute respiratory failure due to pulmonary involvement most often present with severe radiographic alterations involving the lung lobes [5].

Here, we report the case of a patient with spontaneous pneumothorax due to a possible rupture of a tuberculous cavity with acute respiratory failure in the Emergency Department.

Case Report

Our patient was a 65-year-old man of mixed race, who was a farmer from a rural area in the province of Los Ríos, Ecuador who came to the Regional Hospital of the Ecuadorian Institute of Social Security (IESS), Babahoyo, due to symptoms of cough accompanied by greenish expectoration, chest pain, asthenia, and weight loss. On physical examination, there was a decrease in vesicular murmur to left predominance and bilateral crackles.

The patient had a pathological history of valvuloplasty performed approximately 3 years ago at a tertiary-level hospital in Guayaquil, Ecuador. His mother had a history of acute

myocardial infarction. No history of allergies or smoking was noted. The patient received continued treatment due to aortic valve replacement with Warfarin 5 mg once a day and Carvedilol 12.5 mg in the morning and 6.25 mg in the afternoon. His EKG revealed the presence of a left anterior hemiblock.

The patient came to the Emergency Department of the Regional Hospital of the Ecuadorian Institute of Social Security (IESS), Babahoyo, for acute respiratory failure (ARF) and was admitted to the ICU due to poor ventilatory mechanics, with inadequate management of secretions and use of accessory muscles, as well as with arterial blood gas analysis with mixed acidosis, for which orotracheal intubation (OIT) was requested for ventilatory mechanical assistance, and he was placed on invasive mechanical ventilation (IMV). The patient presented the following vital signs: systolic blood pressure (PA) 104/54 mmHg, partial oxygen saturation (SpO₂) 80%, respiratory rate (RR) 36 rpm, and heart rate (HR) 120 beats/min. A chest X-ray showed a left pneumothorax; therefore, a thoracic drainage tube was placed (Figure 1).

During his ICU stay, the patient received IMV support under sedation with Propofol at a dose of 1 mg/kg/h and analgesia with fentanyl at a dose of 1 mcg/kg/h, with the following programmed ventilatory parameters: IPPV mode assisted with tidal volume (vt) 460 ml (8 ml/Kg/weight/ideal), inspiratory time (TI) 1 s, flow 45 L/min, positive end-expiratory pressure (PEEP) 5 cm of H₂O, respiratory rate (RR) 12–14 breaths/min, and fraction inspired oxygen (FIO₂) 45%. For the calculation of ideal Vt of ideal body weight (IBW), we used the formula 55.5±2.3 (height in inches – 60) for men and 45.5±2.3 (height in inches – 60) for women [16]. Patient ventilation parameters were: minute volume (MV) 7.3 L/min; exhaled tidal volume 460 ml; maximum airway pressure (P_{imax}) 29 cm of H₂O, plateau pressure (P_{plt})p: 20 cm of H₂O, average pressure (P_m) 10 cmH₂O/L/s, and compliance (CSTA) 31.9 ml/cm of H₂O.

His arterial blood gas analysis (ABG) showed pH 7.17, pCO₂ 35 mm/Hg, PO₂ 62.8 mm/Hg, HCO₃ 12.7 mmol/L, Base excess (EB) 13.4, and SO₂ 88.6%, which indicates metabolic acidosis due to shock caused by dehydration and severe hemodynamic compromise caused by tension pneumothorax.

The patient remained in the ICU with a central venous catheter, nasogastric tube, bladder catheter, and chest tube to drain the pneumothorax, with an initial diagnosis of acute respiratory failure with additional pneumonia due to non-specific microorganisms.

A CT scan of the thorax showed tree-like infiltrate, some consolidations in the upper lobe, lower right, and in the upper left lobe, pleural effusion on the right side, and left pneumothorax with the presence of chest tube insertion (Figure 2).

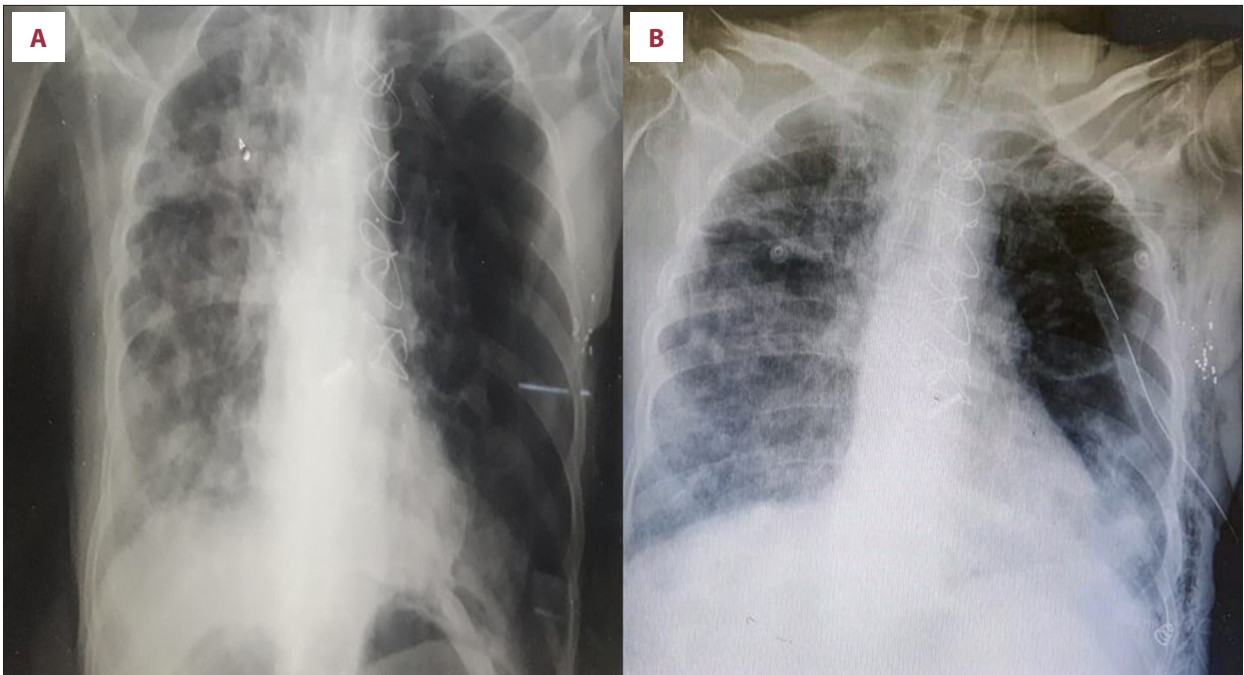


Figure 1. CT scan of the thorax. (A) Axial section of the pulmonary window. (B) Coronal section of the pulmonary window showing tree-like infiltrate, some consolidations in the upper lobe, lower right, and in the upper left lobe, pleural effusion on the right side, and left pneumothorax with the presence of chest tube insertion.

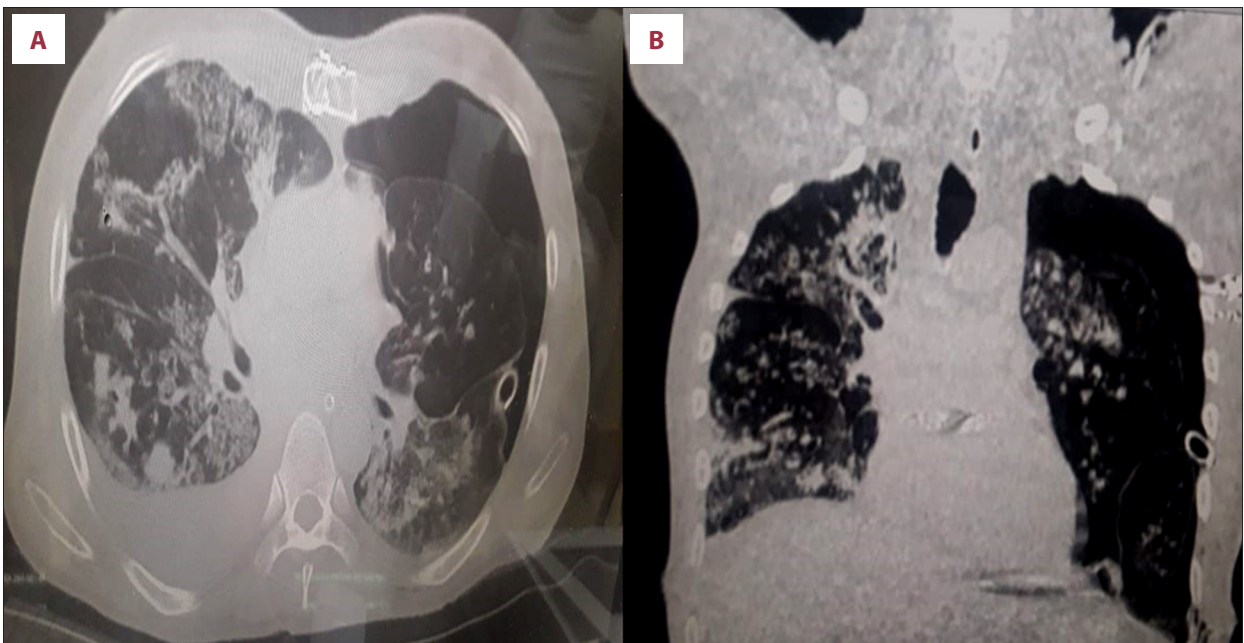


Figure 2. (A) Posterior-anterior view of shows bilateral diffuse opacities, predominantly in the right lung field, with left pneumothorax. (B) The pleural drainage tube is observed on the left side.

Laboratory exams

Complete blood count analysis revealed: leukocytes 14 200 mm³, hemoglobin 8.9 g/dl, hematocrit 28.9%, mean corpuscular volume (MCV) 77 fl/red cell, mean concentration of hemoglobin

(MCH) 23, 7 picograms/cell, mean cell hemoglobin concentration (MCHC) 30.6 grams/deciliter, red blood cell count 374 mm³, platelets 166 000 mm³, Monocytes (%) 3.2, eosinophils (%) 1.4, lymphocytes (%) 3.9, neutrophils (%) 91.3, and basophils (%) 0.2.

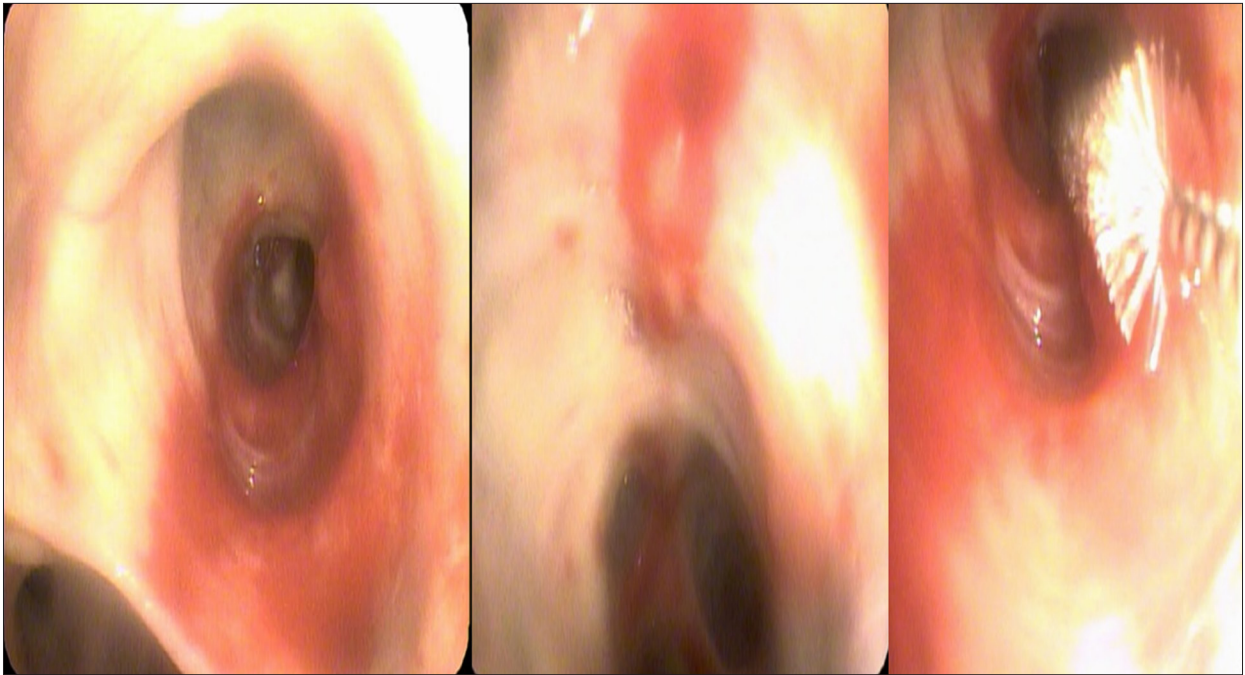


Figure 3. Bronchoscopes images showing mucosal image in left bronchus with deep infiltration without active bleeding and cavitary image and bronchial brushing of cavitary lesion with the presence of a small air leak.

Biochemical analysis revealed: glucose 114.3 mg/dl; electrolytes sodium 141 meq/L, potassium 3.2 meq/L, chlorine 111 meq/L, calcium 7.9 meq/L, BUN 15 mg/dl, and creatinine 0.9 g/dl. During his initial admission, the patient received specific treatment for severe community-acquired pneumonia with ampicillin/sulbactam plus clarithromycin. The *antifimic* treatment with primary scheme (rifampicin-isoniazid, pyrazinamide, and ethambutol) was started immediately after the positive result for tuberculous mycobacteria was obtained from bronchoalveolar lavage.

Bronchoscopy findings

The patient underwent a bronchoscopy at 24 h, in which a sharp pale palate mucosa and areas of erythematous stippling and cavitary image in the apical segment of the right upper lobe were noted. Bronchioloalveolar lavage (BAL) and bronchial brushing of the site of injury were performed and samples were taken for cultures of mycobacteria and fungi (Figure 3).

Microbiological diagnostics

Results were obtained at 4 h after performing the bronchoscopy using a molecular biology technique with the use of MALDI-TOF mass spectrometry (Brucker Daltonics) and by additional phenotypic tests (Optochin and Vitek 2). These results were determined using a disc diffusion method according to CLSI recommendations [6] with detection of *Pseudomonas aeruginosa* and *Klebsiella pneumonia* and with mechanism of

resistance to *KPC* (producing strains of carbapenemase) and GeneXpert MTB/RIF System for detection of *Mycobacterium tuberculosis* determined without resistance to rifampicin examination in straw. Ziehl-Neelsen (ZN) staining for the identification of bacillus acid-resistant alcohol was positive in alveolar bronchial lavage.

Conventional cultures of bronchioloalveolar lavage (BAL) fluid confirmed the presence of *Pseudomonas aeruginosa* and *Klebsiella pneumonia* with carbapenemases resistance mechanism, and the *KPC* type enzyme previously reported with the use of MALDI-TOF mass spectrometry and by additional phenotypic analysis. The Löwenstein-Jensen culture method was positive at 4 weeks. Colistin treatment was administered at an initial dose of 300 mg followed by 100 mg every 8 h plus 500 mg of nebulized amikacin every 12 h.

On day 10, a new CT scan of the thorax control showed complete resolution of the pneumothorax with the presence of an interstitial pattern, more left pulmonary cavern and pleural effusion, and a small amount of bilateral right predominance. The patient presented with hemodynamic instability on day 13 of hospitalization, requiring an increased dose of vasopressor and inotropic support with norepinephrine and dobutamine. An episode of atrial fibrillation with rapid reverse ventricular response was noted, and the patient died on day 13.

Discussion

Secondary spontaneous pneumothorax usually occurs as a complication of the underlying lung disease. The diseases most commonly associated with this entity are obstructive pulmonary disease, cystic fibrosis, cavitated cancers, necrotizing pneumonia, Pneumocystis pneumonia, and tuberculosis [7]. Under these conditions, the lung tissue is hyper-distended and the pulmonary alveoli in hyper-aeration can come into contact with the pleural cavity and rupture. The existence of lung caverns in contact with the intrapleural space can also cause secondary pneumothorax.

Destruction of the pulmonary parenchyma with loss of the metalloproteinase matrix is the main characteristic of pulmonary tuberculosis [8]. Cavities appear when large granulomas with central liquefaction and erosions with discharge of their contents in the underlying airway are produced. Most studies that report larger cavities after the initial presentation of the disease suggest an acute presentation event in pulmonary tuberculosis [7]. In addition, the amount of adjacent fibrosis is not correlated with the age of the lesion, and the cavities are generally observed adjacent to extensive zones of condensation [9]. Regardless of its location in the pulmonary lobe of the tuberculous cavities, in the vast majority of cases it has a subpleural localization; however, there is little evidence of cavitory rupture or pneumothorax as the initial presentation of severe respiratory insufficiency [10].

Our patient presented to the Emergency Department with a spontaneous pneumothorax, probably due to the rupture of a cavity, most likely of tuberculous origin, without ruling out the possibility of multidrug-resistant polymicrobial association. However, another potentially associated factor is the presence of other microorganisms that cause lung cavities, such as *Klebsiella pneumoniae* and *Pseudomonas aeruginosa*, which are common causes of community-acquired pneumonia in patients with chronic structural lung lesions. However, the presence of multiple pulmonary cavities that cause coalescence and gangrene in the lung parenchyma [9] was not observed in the bronchoscopy of our patient, although this result does not rule out the possibility of participation of these bacteria in the genesis of pneumothorax due to cavitory rupture.

Studies show that the extension of cavitation is a very dynamic process, with preferential formation in sites of high mechanical stress near the necrotic granuloma, suggesting that mechanical stress can weaken the underlying lung tissue, with subsequent air entrapment and cavity expansion [11]. Our patient had a ruptured cavity that caused a spontaneous pneumothorax and acute respiratory failure with immediate transfer to the ICU for management of respiratory failure and pneumothorax drainage.

The literature describes the association of tuberculosis and pneumothorax in various circumstances associated with miliary tuberculosis [12], as well as in some cases with remnant cavitory lesions that become visible after the drainage of pneumothoraxes, some of them reported retrospectively [14] and with diagnosis based on radiographs of the cavity after expansion of the pneumothorax, while other cases have been reported in children [15].

We present a case in which the pneumothorax presented by initial rupture of a cavity, probably of tuberculous origin, without ruling out the possibility of polymicrobial association of multidrug-resistant polymicrobial association. Rapid and timely diagnosis is made possible by rapid performance of bronchoscopic visualization and the processing of samples in the laboratory with results obtained 4 h after bronchoscopy using a molecular biology technique, as well as by additional phenotypic tests and the GeneXpert MTB/RIF system for the detection of *Mycobacterium tuberculosis* [6].

However, in patients with *Pseudomonas aeruginosa* and *Klebsiella pneumoniae* with carbapenemases resistance mechanism, the KPC type enzyme is the main cause of progression to septic shock and death.

Conclusions

We report the case of a 65-year-old man with a history of valvuloplasty approximately 3 years ago, who came to our Emergency Department due to acute respiratory failure (ARF) and was admitted to the ICU due to poor ventilatory mechanics. The unusual complications such as secondary pneumothorax resulted from primary parenchymal disease with cavity formation. *Mycobacterium tuberculosis* was confirmed by polymerase chain reaction (PCR) (GeneXpert MTB/RIF) and culture. Chest tube drainage allows for rapid expansion and resolution, but in a polymicrobial infection with carbapenemases resistance mechanism, the KPC type enzyme is the main cause of progression to septic shock and death.

Department and Institution where research study was conducted

Intensive Care Unit, Ecuadorian Institute of Social Security (IESS), Babahoyo, Ecuador.

Conflict of interest

None.

Abbreviations

ICU – Intensive Care Unit; **MTB** – *Mycobacterium tuberculosis*; **ARF** – acute respiratory failure; **BAL** – bronchioloalveolar lavage; **EAB** – acid-base balance; **Vt** – tidal volume; **Vmin** – volume of minutes; **RR** – respiratory rate; **HR** – heart rate; **pH** – negative algorithm of concentration of hydrogen ions; **pCO₂** – concentration of carbon dioxide; **PO₂** – arterial oxygen partial pressure; **SaO₂** – arterial oxygen saturation; **FiO₂** – fraction of

inspired oxygen; **EB** – excess base; **ABG** – arterial blood gases; **HCO₃** – establishes the concentration of buffer or blood bicarbonate; **OTI** – orotracheal intubation; **Mcg** – micrograms; **SpO₂** – partial oxygen saturation; **PA** – systolic/diastolic blood pressure; **IMV** – invasive mechanical ventilation; **AIDS** – acquired immunodeficiency syndrome.

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