

# Tuberculous Pneumonia as a Primary Cause of Acute Respiratory Distress Syndrome: A Case Report

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## Abstract

**Background:** Tuberculosis is an uncommon and very rare cause of Acute Respiratory Distress Syndrome. Moreover, it has been associated with poor outcomes with a very high mortality rate (40-80%). The clinical symptoms and radiological findings of tuberculous pneumonia that resemble bacterial pneumonia result in difficulties to decide definitive diagnosis. **Case Presentation:** A 35-year-old male patient was admitted to hospital with shortness of breath, fever, cough without expectoration. The patient had history of asthma since childhood. Chest physical examination showed rhonchi and wheezing on both lungs. Initial laboratory findings were leucocytosis, granulocytosis, eosinopenia, increased ALT and ALP levels, blood gas analysis revealed acute respiratory failure. PaO<sub>2</sub>/FiO<sub>2</sub> was 49 mmHg. ECG showed sinus tachycardia and chest X-ray showed consolidation on both lungs. The patient was initially managed for asthma exacerbation and bilateral pneumonia with ARDS. The result of microbiological findings showed acid fast bacilli (AFB) positive. He had been treated with anti-TB first category. Clinical improvement was achieved after 1 week of anti-TB treatment. **Summary:** Tuberculous pneumonia with ARDS is a rare case of pulmonary tuberculosis. The diagnosis is established by a prompt and complete examination that lead to optimal treatment with appropriate anti-TB.

**Keywords:** tuberculous pneumonia, tuberculosis with acute respiratory failure, tuberculosis with ARDS

## Introduction

Tuberculosis (TB) is one of the major health problems in the world, especially in developing countries<sup>(1)</sup>. The disease usually has a slow onset and progression. Tuberculosis patients usually experience coughing, weight loss, anorexia, night sweats, and malaise, which occur for several weeks before getting

sick. Tuberculosis with pneumonia and acute respiratory distress syndrome (ARDS) is very rare<sup>(2)</sup>. Data on TB patients with pneumonia was 10% of community-acquired pneumonia (CAP) cases in Asia<sup>(3)</sup>. Meanwhile, TB with ARDS was reported to account for 1.5-5% of all pulmonary TB cases<sup>(4)</sup>. Based on the description above, we would like to report a case of a 35-year-old man who was diagnosed with TB with symptoms of pneumonia and ARDS.

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## Case Presentation

A 35-year-old man with main complaint of shortness of breath for the past 3 days before being admitted to hospital. Shortness of breath occurred in the early hours of the morning and decreased slightly after using fenoterol and salbutamol sprays. The patient also complained of fever for the past 1 day and cough

with phlegm since 1 week before being admitted to the hospital. There was no weight loss and night sweats. The patient had a history of asthma since the age of 10 and he had never received TB treatment before.

The results of physical examination showed 124 pulse per minute, the respiratory rate was 34 beats per minute, the oxygen saturation was 92% using a non-rebreathing mask of 10 lpm oxygen, and an axillary temperature of 39.1°C. Chest physical examination showed vocal fremitus increased on 2/3 upper of both lung, percussion was dull in 2/3 upper of both lung, bronchovesicular sound in 2/3 upper of both lung, rhonchi in 1/3 lower of right lung and 2/3 lower of left lung, and wheezing in 1/3 upper of both lung. The laboratory examination results showed leukocytosis (14.11/mm<sup>3</sup>), Granulocytosis (94.3%), decreased lymphocytes (3.7%), decreased eosinophils (0.4%), increased SGOT (181 U/L), increased SGPT (155 U/L), hypoalbuminemia. (3.02 g/dL), negative HIV test. The results of blood gas analysis showed respiratory failure with PaO<sub>2</sub>/FiO<sub>2</sub> 49 mmHg. Echocardiography results showed neither volume overload nor acute heart failure. Chest X-ray revealed extensive consolidation in both lung fields (Figure 1A).

The patient was diagnosed with severe pneumonia, severe attacks acute exacerbation of moderate persistent bronchial asthma with ARDS with a differential diagnosis of Allergic Bronchopulmonary Aspergillosis (ABPA) and planned to install a ventilator but the care giver refused. The patient was given nebulized therapy for asthma and empiric antibiotics in the form of Ceftazidime 1000 mg intravenously every 8 hours and Levofloxacin 750 mg intravenously every 24 hours. The day-6 evaluation showed leukocytosis (16.06/mm<sup>3</sup>), granulocytosis (90.7%), lymphocytopenia (5.4%), eosinopenia (0.4%), anemia (9.0 gr/dL), procalcitonin 4.49 ng/mL. The results of blood gas analysis still showed respiratory failure with PaO<sub>2</sub>/FiO<sub>2</sub> 219 mmHg. The results of sputum staining with Ziehl Nielsen, Gram and KOH: no BTA and fungi, formation of positive Gram coccus 3+ with PMN background and 2+ epithelium. The results of aerobic culture found *Streptococcus viridans*,

which is a normal respiratory flora bacteria. The results of blood culture showed no growth of aerobic bacteria and fungi. The results of chest X-ray of post-antibiotics administration still showed a consolidation of air bronchogram in the right supra-parahilar of the left lung and right paracardial that suggested pneumonia (Figure 1b).

The patient was subjected to a chest CT scan with contrast on the 7th day of treatment. The results showed a tree in bud pattern accompanied by consolidation with air bronchogram in the right and left lung. These findings suggested a picture of pulmonary tuberculosis that was still possibly accompanied by secondary infection with multiple bullae in the superior lobe of the left lung and the superior lobe of the right lung. Multiple nodules in the inferior lobe of the right and left lung, with the largest size of +/- 0.9 cm in the posterobasal of the inferior lobe of the right lung, could represent a tuberculoma (Figure 2). On the 8th day, the patient's clinical condition had not improved. The results of the blood gas analysis still showed respiratory failure with PaO<sub>2</sub>/FiO<sub>2</sub> 139 mmHg. Antibiotic therapy was changed to Meropenem 1000 mg intravenously every 8 hours and Levofloxacin 750 mg intravenously every 24 hours. The patient was also given adjuvant therapy of methylprednisolone 62.5 mg through intravenous injection every 8 hours. Sputum induction was conducted on the 9th day, with Gram stain examination, BTA examination and Xpert MTB/RIF sputum. On the 13th day, the results of morning scanty sputum BTA, with the result of Xpert MTB/RIF MTB detected very low Rifampin resistant not detected. The patient was then treated with a special liver anti-TB regimen Streptomycin 750 mg through intramuscular injection, Levofloxacin 750 mg through intravenous injection, and Etambutol 1000 mg orally on day 13 of treatment because there was still an increase in ALP (115 U/L) and ALT (230 U/L). On the 20th day of treatment, the patient began to show clinical improvement with a significantly reduced breathlessness. The patient was given 3 lpm nasal cannula oxygen therapy, showing moderate hypoxemic blood gas analysis results. The results of chest X-ray on the day 24 (day 11 using OAT

therapy) (Figure 3) showed improvement in the patient's condition.

The patient was then subjected to a bronchoscopy to take sample of Broncho Alveolar Lavage (BAL) on the

29th day of treatment, with the result of *M. tuberculosis*. The patient was discharged from the hospital on the 31st day with 100 mg of INH-sensitized therapy, 750 mg of Levofloxacin and 1000 mg of Etambutol orally.

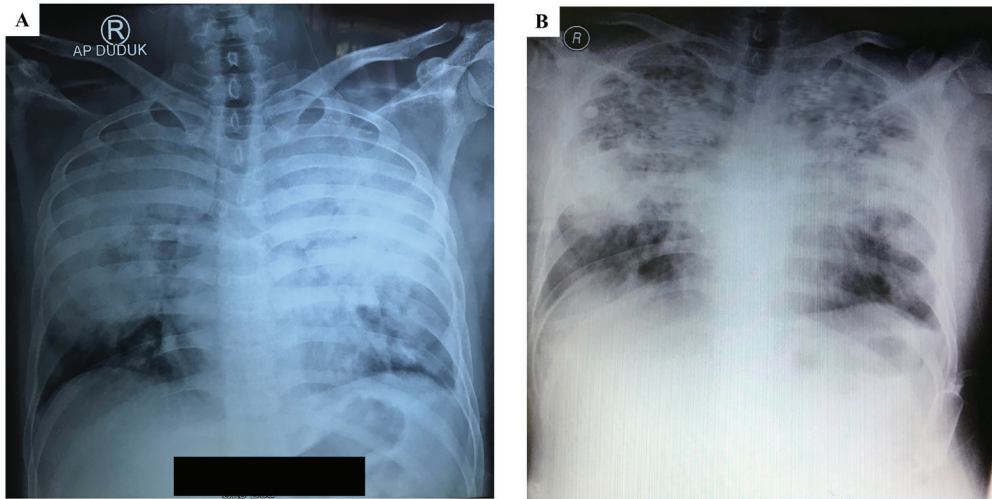
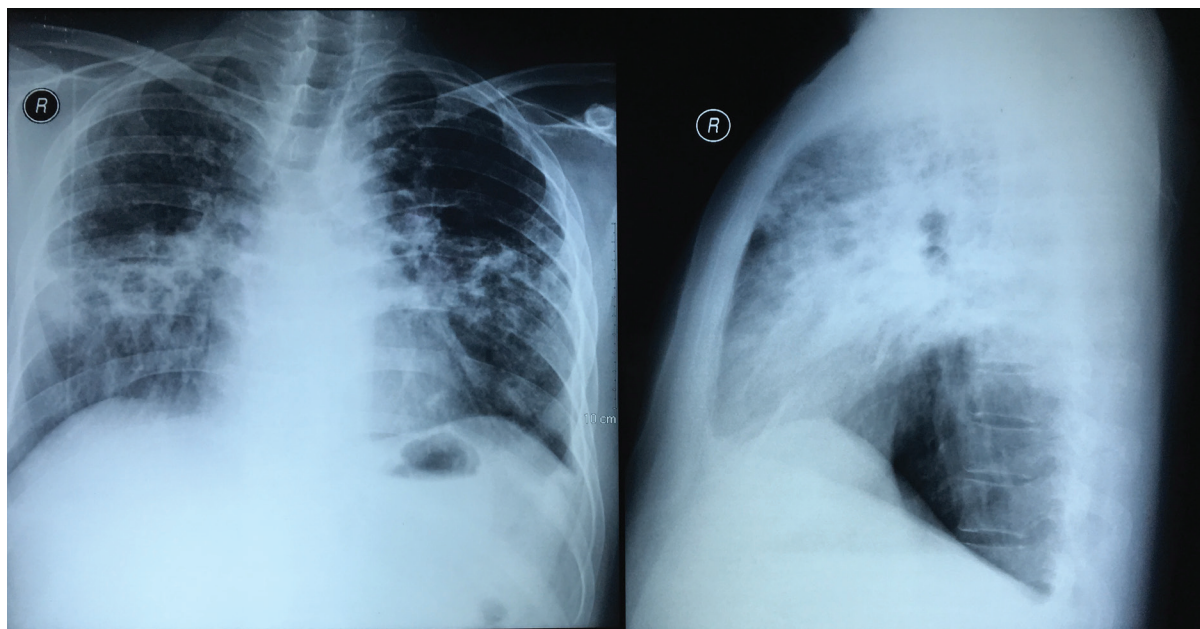


Figure 1. (a) Chest x-ray on the first day and (b) the third day



Figure 2. Thoracic CT-scan on day 7



**Figure 3. Chest x-rays on day 24 and day 11 using anti-TB drugs show improvement.**

### Discussion

Several cases of tuberculous pneumonia reported that cough with phlegm occurred prior to respiration and other systemic complaints. The resulting acute respiratory failure also confuses the likelihood of TB because the incidence is very low among hospitalized pulmonary TB patients<sup>(5)</sup>. The diagnosis of tuberculous pneumonia can be made should the patient has a high fever with signs of severe toxicity, thoracic physical examination showing signs of consolidation, the presence of consolidation of a minimum area in one lobe, and microbiological examination of sputum revealing tuberculous puncture<sup>(6)</sup>. ARDS in TB patients is an extremely rare occurrence<sup>(7)</sup>. ARDS criteria include onset within 1 week of known clinical insult, or deteriorating or recent respiratory system,  $SpO_2/FiO_2 \leq 315$  with  $SpO_2 \leq 97\%$ , features of bilateral opacity evaluated by ultrasonography or chest X-ray should available, respiratory failure unrelated to heart failure or fluid overload, the need of objective assessment such as echocardiography to rule out hydrostatic edema in the absence of risk factors<sup>(8, 9)</sup>.

Anti-TB drugs are therapy given to TB patients with a combination of at least 4 types of drugs at a dose in

accordance with the patient's body weight. Treatment requires a minimum of 6 months in category 1, given in 2 stages, namely the intensive stage (2 months HRZE) and the advanced stage (4 months HR) to prevent recurrence and resistance<sup>(10)</sup>. Patients with a threefold increase in liver enzymes before starting anti-TB therapy can be given a special liver regimen according to WHO guidelines (2HRES/6HR or 2SHE/10HE). Tuberculosis is rarely considered a diagnosis should the patient has features of acute pneumonia and respiratory failure. This can lead to the use of antibiotics that have anti-TB therapeutic effects such as fluoroquinolones which temporarily improve clinical conditions, increase the resistance of fluoroquinolones used as monotherapy, thereby accelerating the occurrence of XDR and pre-XDR cases<sup>(2)</sup>.

Tuberculous pneumonia therapy is similar to TB therapy in general. Corticosteroids are used as adjuvant therapy in TB treatment to overcome this inflammatory reaction (11). Corticosteroids work by inhibiting the release of cytokines that play a role in constitutional symptoms and tissue damage. In addition, corticosteroids make it easier for anti-TB drugs to enter the granuloma and destroy micobacterium tuberculosis. The use of corticosteroids can make patients more susceptible to

other infections<sup>(3, 11, 12)</sup>. The benefits of corticosteroids in tuberculous pneumonia with acute respiratory failure have been reported in several studies. Tuberculous pneumonia patients with acute respiratory failure who received corticosteroid therapy showed lower mortality than those who did not receive corticosteroid therapy (56.7% vs 77.8%)<sup>(8)</sup>. The prognosis of tuberculous pneumonia patients has improved with the use of corticosteroids, in which corticosteroids are a favorable prognostic factor for tuberculous pneumonia patients<sup>(10, 13)</sup>. Corticosteroids play a role in improving the clinical situation in TB patients when used as adjunctive therapy<sup>(14, 15)</sup>.

In conclusion, a 35-year-old man was reported having symptoms of acute pneumonia with ARDS. The 2-week pneumonia therapy showed no improvement. Moreover, the results of chest CT scan with contrast and Xpert MTB/RIF examination of sputum induction detected MTB. The patient was given anti-TB therapy and experienced improvement both clinically and radiologically.

**Conflict of Interest:** The authors declare that they have no conflict of interest.

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Statement of Ethics

The present case report adhered to the Declaration of Helsinki. Informed consent for publication was obtained from the patient.

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