



Double Mycobacterial Infection in Septic Patient: A Rare Case of Pulmonary Tuberculosis and Leprosy Coinfection

Kirana Paramitha Santosa ¹. Anggar Jito²

¹ General Practitioner, Regional Public Hospital Bantargebang,
Bekasi, West Java, Indonesia

² Departement of Pulmonology and Respiratory Medicine, Regional
Public Hospital Chasbullah Abdulmadjid, Bekasi, West Java,
Indonesia

Corresponding Author : paramithakirana17@gmail.com

Article History :

Received date : 2025/09/27
Revised date : 2025/10/12
Accepted date : 2025/12/23
Published date : 2026/01/20



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ABSTRACT

Introduction: Co-infection of Tuberculosis (TB) and Leprosy is a rare occurrence, even in endemic areas.¹ This co-infection has higher risks of drug resistance, complications, and mortality.^{2,3}

Case Report: A 38-year-old male patient, who had been taking long-term steroids, presented in severe condition, showing signs of sepsis and ARDS. Multilobar infiltrates were found in both lungs, MTB/RIF was positive and sensitive to Rifampicin, BTA was found in slit skin smear test, liver enzyme levels were elevated, D-dimer level was increased (6 mg/L), low PaO₂/FiO₂ ratio (44.25 mmHg), along with an increased AaDO₂ (508.4 mmHg). Despite receiving antimicrobial and anti-tuberculosis therapy, the patient's condition continued to deteriorate, resulting in septic shock and death.

Discussion: The rarity of these cases in the modern literature is likely due to Bacillus Calmette-Guérin (BCG) vaccination in endemic areas, which provides at least partial protection against both diseases. Dual infection is associated with higher morbidity (5.5%) and mortality (37.2%).² Appropriate diagnostic, screening, and therapeutic regimens are needed in clinical practice to prevent the risk of drug resistance and complications. This study aimed to present a complicated case of pulmonary TB and multibacillary leprosy co-infection in a septic patient.

Keywords: pulmonary tuberculosis, leprosy, coinfection, sepsis

INTRODUCTION

Tuberculosis (TB) and Leprosy are chronic granulomatous diseases that have been infecting humans for approximately 9,000 and 4,000 years, respectively. Both diseases share similar geographic endemic locations, primarily in developing countries with poor living conditions. Epidemiological factors contributing to their prevalence include high population density, malnutrition, poor sanitation, and low socioeconomic status. India and Indonesia rank highest in contributing to new TB and Leprosy cases. According to the SDG targets, both diseases should have been eliminated by 2030.^{1,2}

However, co-infection in a single individual is rarely found, even in endemic areas, with an estimated prevalence of 0.02 per 100,000 people.³ The exact incidence of this co-infection in Indonesia is still unknown, but in the last decade, only seven cases have been reported in the national literature. Severe cases of coinfection accompanied by sepsis have never been reported. The rarity of these cases in the modern literature is likely due to Bacillus Calmette-Guérin (BCG) vaccination in endemic areas, which provides at least partial protection against both diseases.⁴

The pathophysiology of co-infection remains uncertain, as it is unclear whether the presence of one infection protects against the other or, conversely, if the host immune response pattern favors the development of another mycobacterial infection. This co-infection poses risks of complications and adverse effects from the medications used for treatments, with clinical, economic, and social impacts. Nevertheless, there is still no specific strategy for detecting populations with active concurrent co-infection.⁵

In this case, the patient presented with complications, having already developed sepsis. Multi-organ dysfunction, including lung and liver dysfunction, was identified through comprehensive physical examination and laboratory tests. Given the rarity of severe co-infection cases and the importance of early detection and appropriate management, this study reports a case of pulmonary TB and multibacillary Leprosy co-infection accompanied by sepsis in a 38-year-old male with long-term steroid use.

CASE REPORT

A 38-year-old male experienced worsening shortness of breath for one week before hospital admission. His symptoms were accompanied by a productive cough that had persisted for six months, intermittent fever, loss of appetite, weight loss, and frequent sweating. The patient also complained of numbness and weakness in both hands, as well as swelling in both legs.

The patient had a history of leprosy in 2022, approximately two years before admission. He had undergone MDT (Multidrug Therapy) treatment from a primary healthcare facility, with monthly check-ups for one year, and was declared cured. However, during treatment in 2022, he developed red plaques and swelling all over his body, suspected to be a leprosy reaction, for which he received methylprednisolone therapy. After being declared cured, he experienced recurrent skin issues and continued taking methylprednisolone independently without professional supervision. He denied any history of lung infection, HIV, heart disease, kidney disease, hypertension, or diabetes.

On physical examination, the patient appeared severely short of breath but was fully conscious. He showed signs of malnutrition, pallor, and clammy skin. His vital signs revealed a blood pressure of 110/57 mmHg, tachycardia (168 beats per minute), hyperventilation (39 breaths per minute), hypoxemia (oxygen saturation of 78% with 15 LPM O₂), and fever (38.4°C). Multiple lesions, including erythematous plaques, hypopigmented macules, xerosis cutis (dry skin), and hypoesthesia were observed on his face and both hands (Figure 1). Motor strength in both hands was slightly reduced. Ulnar nerve thickening and clawing of the 4th and 5th fingers in both hands were noted, along with pitting edema in both legs.



Figure 1. Multiple skin lesions on the dorsum of the hands and ulnar clawing

On thoracic inspection, the chest wall appeared symmetrical in shape and movement, with intercostal retraction observed. Palpation revealed slightly increased tactile fremitus in both lung fields. Percussion was resonant in both lung fields. Auscultation revealed prolonged expiration and coarse wet crackles throughout both lungs, which were later confirmed by a chest X-ray showing infiltrates in the upper, middle, and lower lung fields of both lungs, suggesting active bilateral TB (Figure 2).



Figure 2. Chest X-ray on Day 1 of Hospitalization



Figure 3. Chest X-ray on Day 7 of Hospitalization

Ziehl-Neelsen staining for acid-fast bacilli (AFB) was performed on skin scrapings from the left and right earlobes, face, and the dorsum of both hands, revealing fragmented AFB bacteria (Figure 4). Gram and KOH staining of sputum showed gram-positive cocci (+1), yeast cells (+1), and hyphae.



Figure 4. Fragmented AFB on Skin Scraping Examination

Laboratory tests showed leukocytosis (13,000/uL), hypercoagulopathy (D-dimer 6 mg/L), hyponatremia (122 mmol/L), and impaired liver function (Albumin 2.30 g/dL; SGOT 71 U/L; SGPT 68 U/L; Total Bilirubin 2.46 mg/dL; Direct Bilirubin 1.29 mg/dL; Indirect Bilirubin 1.17 mg/dL). A Molecular Rapid Test (MTB/RIF) was performed on sputum sample to detect *M. tuberculosis* DNA and its resistance to rifampicin. The test result detected *M. tuberculosis* at a medium level and

confirmed sensitivity to rifampicin. Arterial blood gas analysis revealed: pH: 7.47, pCO₂: 22.4 mmHg, pO₂: 35.4 mmHg, P/F ratio: 44.25 mmHg, SaO₂: 69.6%, HCO₃: 16.4 mmol/L, Base Excess (BE): -4.7 mmol/L, AaDO₂: 508.4 mmHg with a non-rebreathing oxygen mask at 15 liters per minute. HIV testing using three different methods was non-reactive.

During the diagnostic and treatment process, consultations were made to the anesthesiology department for intensive care in the ICU, and to the dermatovenereology department for the patient's leprosy. The treatment was then focused on improving the patient's overall condition. A non-invasive ventilator was applied using CPAP with a PEEP of 5 cmH₂O and FiO₂ of 50%, improving the respiratory rate to 28 breaths/min and SpO₂ to 98%. Lactated Ringer's solution was administered as resuscitation fluid, followed by 3% NaCl (500 cc every 12 hours) to correct hyponatremia. The antibiotics given included intravenous Fosfomycin (1 g every 12 hours) and Moxifloxacin (400 mg every 24 hours). Fondaparinux (2.5 mg) was administered to address hypercoagulopathy. Hepatoprotective supplements (both oral and intravenous) and a 20% albumin transfusion were given to improve liver function and correct hypoalbuminemia. Based on clinical considerations, the anti-tuberculosis regimen 2RHZE/4RH (Rifampicin 150 mg, Isoniazid 75 mg, Pyrazinamide 400 mg, Ethambutol 275 mg) was given as FDC (fixed dose combination) tablets per 24 hours. Symptomatic treatments included antipyretics, mucolytics, PPI (Omeprazole), and intravenous steroids (Methylprednisolone). Salbutamol and Budesonide inhalation were administered every 8 hours to relieve breathing difficulty. MDT treatment for leprosy was planned once the patient's condition stabilized.

The patient underwent intensive care for 10 days. On day 5, Fosfomycin was discontinued and replaced with Meropenem (1 g every 8 hours). By day 7, clinical and radiological improvements were observed, with oxygen saturation maintained at 93% using a non-rebreathing mask with 15 LPM O₂, and lung infiltrates appeared to be reduced (Figure 3). However, on day 9, the patient's condition deteriorated, showing an increasing AaDO₂ (626.0 mmHg) and D-Dimer level (>10 mg/L). The patient was declared deceased on day 10 due to septic shock. Based on the data collected during hospitalization, including history, physical examination, laboratory findings, and radiological results, the final diagnosis was concluded as a new case of pulmonary TB, multibacillary leprosy, and sepsis.

DISCUSSION

Tuberculosis (TB) and Leprosy are global health burdens. According to WHO data from 2022, Indonesia had the second-highest number of new TB cases after India and the third-highest number of new Leprosy cases after India and Brazil. Both diseases are granulomatous infections caused by aerobic, acid-fast, Gram-positive bacilli from the *Mycobacterium sp* genus. They are primarily transmitted via droplets. TB primarily affects the lungs, whereas Leprosy manifests in the skin and peripheral nerves.⁶

Co-infection in a single individual is rare, even in endemic areas, with an estimated prevalence of 0.02 per 100,000 people. Dual infection is associated with higher morbidity (5.5%) and mortality (37.2%). The time interval between a patient's Leprosy and TB diagnoses varies significantly, with the largest study reporting a delay ranging from six months to 15 years. Typically, Leprosy precedes TB, but cases of TB occurring first have also been documented.⁷ In this case, the patient was diagnosed with TB approximately two years after being diagnosed with Leprosy.

A systematic review on co-infection conducted by Luciana et al. reported that most cases involved young males, with pulmonary TB being the most common type of TB, and multibacillary (MB) Leprosy being the most common form of Leprosy. In this case, the patient had clinically, radiologically, and laboratory-confirmed pulmonary TB and multibacillary Leprosy. The occurrence of TB and MB Leprosy co-infection is suspected to be linked to the patient's immune response pattern.⁵ The suppression of cell-mediated immunity in Leprosy patients—characterized by defects in Toll-like receptor 2, chemokine ligand-2, and tumor necrosis factor-alpha—prevents proper restriction of *M. tuberculosis* (MTB) growth. This increases the risk of latent TB reactivation or new TB infection.⁶ However, conflicting theories exist, including the concept of cross-immunity. This is supported by findings that the BCG vaccine provides protection against both Leprosy and TB, as evidenced by conversions in intradermal lepromin and tuberculin skin tests.⁸

The Mantoux test and QuantiFERON Gold (IGRA) tests, if positive in Leprosy patients, can be misleading when diagnosing TB co-infection. Similarly, the presence of acid-fast bacilli (AFB) in sputum does not confirm TB, as AFB can also be found in the sputum of Leprosy patients (especially

in the lepromatous type), creating a diagnostic dilemma. Therefore, evidence of active TB on chest X-ray and a positive TB culture are considered the gold standard for diagnosing active TB in Leprosy patients.⁸ In this case, a molecular rapid test (MTB/RIF) was performed alongside a chest X-ray to establish a definitive TB diagnosis and initiate appropriate treatment as soon as possible.

According to the 2020 National Medical Service Guidelines for Leprosy Management and the 2024 Clinical Practice Guidelines by PERDOSKI, the management of TB-Leprosy co-infection involves administering anti-tuberculosis therapy (OAT) and multidrug therapy (MDT) for Leprosy simultaneously. During TB treatment, Rifampicin is given at the standard TB dosage. The duration of Leprosy treatment remains consistent with the prescribed period for paucibacillary (PB) or multibacillary (MB) Leprosy. If TB treatment is completed, Leprosy treatment should continue following the WHO MDT blister packs.^{9,10} Rifampicin is a bactericidal drug effective for both TB and Leprosy. If Rifampicin is administered monthly to a Leprosy patient unknowingly co-infected with TB, the risk of drug resistance increases. Therefore, early detection of co-infection is crucial, especially in Leprosy patients showing TB symptoms.⁷ In this case, the patient did not have Rifampicin resistance, as confirmed by the MTB/RIF. Thus, TB management followed the 2021 National Medical Service Guidelines for Tuberculosis Management and the 2022 WHO Consolidated Guidelines, in which the 2RHZE/4RH regimen was initiated for new cases of drug-sensitive pulmonary TB.¹¹

Corticosteroids are commonly used to manage Type I and Type II Leprosy reactions and neuropathy. Several studies have reported cases of pulmonary TB emerging in Leprosy patients on long-term corticosteroid treatment.¹ However, a study by Agnes et al. found that Leprosy patients experiencing reversal reactions who were treated with steroids did not necessarily develop TB reactivation—likely because the steroids were either administered at low doses or higher doses for a short duration.⁶ In this case, before admission, the patient had been self-medicating with corticosteroids (Methylprednisolone 12 mg/day) for about a year without professional supervision. Aside from immunocompromised status, other factors that increase the risk of co-infection include overcrowded living conditions, malnutrition, poor sanitation, and low socioeconomic status.¹²

The patient presented to the hospital in critical condition, with extensive lesions caused by co-infection, along with signs of sepsis and acute respiratory distress syndrome (ARDS). According to the 2021 Sepsis Campaign Guidelines, SIRS criteria, NEWS, or MEWS are preferred over qSOFA for sepsis assessment. In this case, the patient met all four SIRS criteria, along with an identified source of infection. With sepsis confirmed, the patient received antimicrobial therapy within 1 hour of diagnosis and crystalloid fluid resuscitation as the first-line treatment. Although the guidelines recommend High-Flow Nasal Oxygen (HFNO) over noninvasive ventilation (NIV) for Type 1 respiratory failure, further research is still needed. In this case, the use of CPAP (PEEP 5 cmH₂O, FiO₂ 50%) initially showed a positive response, improving the respiratory rate to 28 breaths/min and SpO₂ to 98%. Additionally, intravenous venous thromboembolism (VTE) prophylaxis was administered to prevent disseminated intravascular coagulation (DIC), deep vein thrombosis (DVT), and pulmonary embolism, following recommendations for critically ill ICU patients.¹³

Until the production of this article, there were no reported cases of TB-Leprosy coinfection with sepsis in modern literature. *Mycobacterium leprae* is a slow-growing bacterium, which typically does not directly cause sepsis. However, several literatures have reported sepsis cases in leprosy patients. In severe forms of leprosy, such as necrotizing erythema nodosum leprosum (NENL) and lucio leprosy, secondary infections that are not properly managed can lead to sepsis.^{14,15} On the other hand, TB is often a subacute infection, but in some cases, it can present as sepsis or septic shock, with poor outcomes and a hospital mortality rate of up to 80%. The clinical picture of tuberculosis sepsis (TBS) includes: hematological abnormalities such as disseminated intravascular coagulation (DIC), altered mental status, kidney failure, refractory hypotension, and ARDS. However, TBS may be indistinguishable from bacterial sepsis. Most cases present with a diffuse multilobar consolidation pattern on chest radiographs.¹⁶ TB spreads both lymphatically and hematogenously, leading to a systemic response. Proinflammatory mediators trigger leukocytosis, stimulate the release of cytokines, activate coagulation and fibrinolysis, and ultimately lead to widespread multi-organ damage through tissue ischemia, cytopathic injury, increased programmed cell death (apoptosis). Intravascular fluid shifts, vasopressin inhibition, and increased vasoactive mediators (e.g., nitric oxide) contribute to hypotension or shock. In the lungs, endothelial damage disrupts blood flow and

increases microvascular permeability, leading to interstitial and alveolar pulmonary edema. This results in diffuse alveolar damage, which causes significant ventilation-perfusion (V/Q) mismatch and ARDS.¹⁷

CONCLUSION

This case study showcases a rare but fatal co-infection of pulmonary tuberculosis and multibacillary Leprosy in a sepsis patient with long-term steroid use and immunocompromised status. While TB-Leprosy co-infection is uncommon, it should not be overlooked, as it significantly increases patient mortality. Therefore, appropriate diagnostic, screening, and treatment regimens are essential in clinical practice to prevent drug resistance and complications. Screening for TB in Leprosy patients—and vice versa—is crucial. Steroids can be used to manage Leprosy reactions, but dose adjustments may be necessary to achieve the best outcomes. Finally, patient education and supervised treatment are vital to prevent unsupervised self-medication, which can worsen the patient's condition.

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