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Case Report

Acute Respiratory Distress Syndrome as Pulmonary Tuberculosis in a Previously Healthy Adult: A Case Report

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Abstract

Acute Respiratory Distress Syndrome (ARDS) is a critical condition leading to acute hypoxemic respiratory failure, primarily resulting from bacterial sepsis and viral pneumonia. Tuberculosis (TB), generally a chronic lung condition, infrequently manifests as an acute cause of ARDS; but, when it does, it correlates with elevated mortality, especially in TB-endemic areas. Early recognition poses difficulties because of nonspecific clinical and radiological findings, frequently leading to delayed diagnosis and suboptimal outcomes. We describe a 31-year-old previously healthy male who exhibited a 10-day history of high-grade fever, dry cough and progressive dyspnoea, culminating in acute deterioration over a 48-hour period. Upon admission, the patient presented with fever, tachypnoea and significant hypoxemia, exhibiting an oxygen saturation of 76% in room air. Arterial blood gas analysis indicated a PaO_2/FiO_2 ratio of less than 200, meeting the Berlin criteria for acute respiratory distress syndrome. Chest radiography revealed bilateral diffuse alveolar opacities, whereas computed tomography of the thorax displayed extensive ground-glass opacities accompanied by areas of consolidation and centrilobular nodules presenting a tree-in-bud pattern. Despite the administration of non-invasive positive pressure ventilation and empirical broad-spectrum antibiotics, clinical improvement was minimal. The microbiological assessment of induced sputum via cartridge-based nucleic acid amplification testing identified Mycobacterium tuberculosis without evidence of rifampicin resistance. Antitubercular therapy and systemic corticosteroids were commenced, leading to progressive clinical and radiological enhancement. Tuberculosis must be regarded as a significant differential diagnosis in patients exhibiting unexplained ARDS in endemic areas. Timely microbiological confirmation and the swift commencement of antitubercular therapy, coupled with suitable respiratory support, are critical for enhancing patient outcomes.

Keywords: Acute Respiratory Distress Syndrome; Pulmonary Tuberculosis; Hypoxemic Respiratory Failure; *Mycobacterium* Tuberculosis

Introduction

Acute Respiratory Distress Syndrome (ARDS) is a critical clinical condition marked by the rapid onset of hypoxemic respiratory failure, bilateral pulmonary infiltrates, decreased lung compliance and non-cardiogenic pulmonary oedema due to diffuse alveolar damage [1]. This represents a final common pathway of lung injury resulting from various pulmonary and extrapulmonary insults, such as bacterial sepsis, severe pneumonia, aspiration, trauma, pancreatitis and viral infections [2]. Despite progress in lung-protective ventilation strategies and intensive care support, ARDS remains linked to significant morbidity and mortality, especially in patients with moderate to severe disease necessitating ventilatory assistance [3].

Tuberculosis (TB) continues to be a significant global health issue, particularly affecting low and middle-income countries disproportionately. Pulmonary tuberculosis typically exhibits a subacute or chronic clinical course, marked by symptoms such as cough, fever, weight loss and haemoptysis; however, it can occasionally manifest as acute respiratory failure. Tuberculosis is a rare yet acknowledged cause of ARDS, especially in regions where TB is endemic. Initial clinical studies indicated that pulmonary tuberculosis complicated by acute respiratory failure necessitating ventilatory support is linked to unfavourable outcomes and elevated mortality rates [4]. Systematic reviews indicate that ARDS occurs in approximately 1-3% of patients with active tuberculosis, yet it is linked to significantly elevated mortality rates of 40% to 80%, particularly when there is a delay in diagnosis and the commencement of antitubercular therapy [5,6].

The Berlin definition of ARDS established standardized diagnostic criteria that include the timing of onset, specific radiological abnormalities, the exclusion of cardiogenic pulmonary oedema and the severity of hypoxemia. This definition has enhanced diagnostic accuracy and promoted standardized reporting in clinical and epidemiological studies, thereby facilitating the identification of atypical and rare causes of ARDS, such as tuberculosis [7].

The pathogenesis of tuberculosis-associated ARDS is intricate and distinct from that of conventional bacterial pneumonia. Proposed mechanisms encompass hematogenous dissemination of *Mycobacterium tuberculosis*, excessive mycobacterial burden and a dysregulated host immune response. Overactivation of macrophages and T-lymphocytes leads to the secretion of pro-inflammatory cytokines, including tumour necrosis factor-α, interleukin-1 and interferon-γ. This process results in endothelial and epithelial damage, heightened permeability of the alveolar-capillary membrane and diffuse alveolar injury [8,9].

Epidemiological studies indicate that, despite advancements in lung-protective ventilation strategies and adjunctive therapies like prone positioning, ARDS remains associated with considerable mortality, especially in severe instances [10]. In nations with a significant tuberculosis burden, including India, tuberculosis must be regarded as a crucial differential diagnosis for patients exhibiting unexplained ARDS or inadequate response to broad-spectrum antibiotics, as highlighted by global tuberculosis surveillance data [11,12]. The timely microbiological confirmation and immediate commencement of antitubercular therapy are essential factors influencing survival outcomes.

Case Summaries

A 31-year-old male with no known comorbidities reported to the emergency room with a 10-day history of high-grade intermittent fever, dry cough and progressively worsening shortness of breath, culminating in an acute exacerbation of dyspnoea over the prior 48 hours. No history of haemoptysis, chest discomfort, wheezing, orthopnoea, recent travel or exposure to harmful inhalants was noted. He possessed no antecedent history of tuberculosis, diabetes mellitus, chronic pulmonary disease or immunosuppressive treatment. No known contact with a TB patient occurred. He abstained from smoking and refrained from alcohol consumption.

Upon admission, the patient presented with fever 102°F, tachypnoea (28 breaths/min), tachycardia (128 beats/min) and hypoxemia, with an oxygen saturation of 76% on room air. Blood pressure measured 110/70 mmHg. He had severe respiratory difficulty, utilizing accessory muscles for breathing. Chest auscultation revealed bilateral diffuse inspiratory crackles, although cardiovascular, per abdomen and neurological assessments were unremarkable.

Arterial blood gas analysis while on supplemental oxygen revealed significant hypoxemia, indicated by a PaO_2/FiO_2 ratio of less than 200, thereby meeting the Berlin criteria for mild Acute Respiratory Distress Syndrome (ARDS). Chest radiography demonstrated bilateral diffuse alveolar opacities seen in Fig. 1.

While Computed Tomography (CT) scan of the thorax exhibited multiple centrilobular nodular opacities showing tree in bud appearance and extensive ground glass opacity in bilateral lungs with areas of consolidation showing air bronchogram sign within in the bilateral lower lobes in Fig. 2. Laboratory tests revealed increased inflammatory markers while renal and hepatic functions remained normal. Procalcitonin levels were diminished, blood cultures were negative and serological testing for human immunodeficiency virus was negative. RT-PCR for COVID, Influenza A and B, Parainfluenza virus, Respiratory syncytial virus and Rhinovirus were negative.

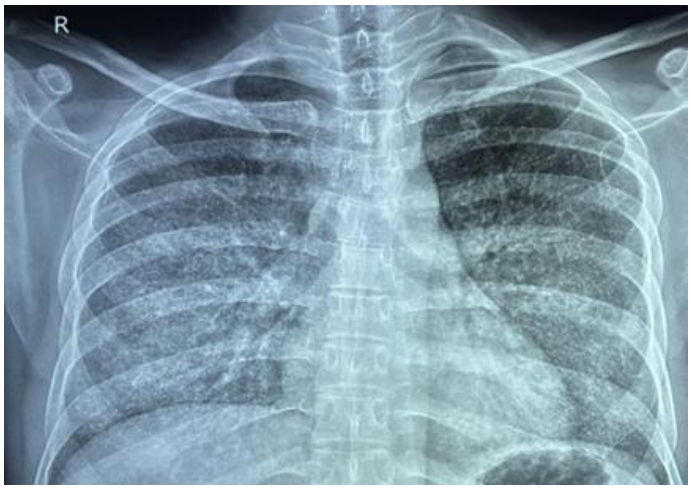

Figure 1: Chest X-ray shows bilateral diffuse alveolar opacities.

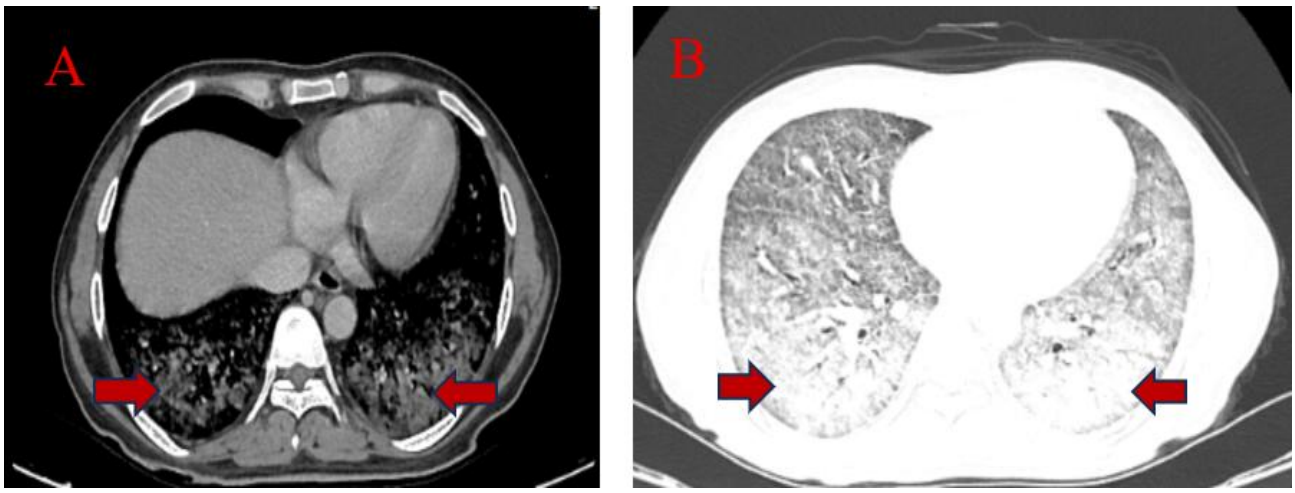

Figure 2: (A& B): Multiple centrilobular nodular opacities showing tree in bud appearance and extensive ground glass opacity in bilateral lungs with areas of consolidation showing air bronchogram sign within in the bilateral lower lobes.

The patient was admitted to a high-dependency unit and treated with Non-Invasive Positive Pressure Ventilation (NIPPV) utilizing bilevel positive airway pressure, in conjunction with conservative fluid management and meticulous monitoring. Empirical broad-spectrum antibiotics were commenced for suspected severe pneumonia; however, no substantial clinical improvement was observed after 72 hours. Given the endemic context, inadequate response to medications and the lack of an alternative diagnosis, tuberculosis was presumed.

Follow Up

The microbiological assessment of induced sputum with Cartridge-Based Nucleic Acid Amplification Testing (CBNAAT) identified *Mycobacterium tuberculosis*, exhibiting no resistance to rifampicin. Standard first-line antitubercular medication was commenced alongside supplemental systemic corticosteroids. The patient exhibited progressive enhancement in oxygenation and respiratory symptoms, facilitating the transition from non-invasive ventilation to low-flow oxygen. He was discharged in stable condition on continuation phase antitubercular therapy. At follow up, remained asymptomatic with significant radiological improvement.

Discussion

Tuberculosis-associated ARDS is a rare yet severe manifestation of pulmonary tuberculosis, presenting considerable diagnostic and therapeutic challenges. ARDS typically results from bacterial sepsis or viral pneumonia; however, tuberculosis is an often-overlooked cause, especially in endemic areas [4,5]. The incidence of ARDS in patients with active tuberculosis is low; however, the mortality rate is significantly high, highlighting the necessity for early recognition and treatment [5,6].

The pathophysiology of tuberculosis-associated Acute Respiratory Distress Syndrome (ARDS) exhibits distinct differences compared to ARDS resulting from conventional bacterial pneumonias. The hematogenous spread of *Mycobacterium tuberculosis* may lead to extensive pulmonary involvement, eliciting a heightened immune response from the host. The overproduction of pro-inflammatory cytokines results in injury to endothelial and epithelial cells, heightened permeability of the alveolar-capillary membrane and diffuse alveolar damage [8,9]. Histopathological studies indicate that hyaline membrane formation and diffuse alveolar damage occur in TB-associated ARDS, akin to other ARDS causes, but are driven by a chronic infectious process instead of an acute insult [1].

The early diagnosis of tuberculosis in patients with ARDS presents significant challenges. Clinical features and radiological findings frequently exhibit nonspecific characteristics and show significant overlap with severe bacterial or viral pneumonias [5,6]. As a result, tuberculosis is often not considered during the initial assessment, leading to a delay in the commencement of antitubercular therapy and suboptimal outcomes. Clinical indicators, including inadequate response to broad-spectrum antibiotics, low procalcitonin levels, lack of an alternative microbiological diagnosis and relevant epidemiological risk factors, should initiate prompt evaluation for tuberculosis [8,11].

The ventilatory management of TB-associated ARDS adheres to established principles of ARDS management, which encompass adequate oxygenation, conservative fluid strategies and lung-protective ventilation. Prone positioning as an adjunctive strategy has demonstrated an improvement in survival rates for patients with severe ARDS [10]. Although numerous reported cases necessitate invasive mechanical ventilation, certain patients can be effectively managed with Non-Invasive Positive Pressure Ventilation (NIPPV) with careful monitoring, thus preventing complications associated with intubation [4]. In this case, the early application of NIPPV alongside the timely commencement of antitubercular therapy led to positive clinical outcomes.

The use of adjunctive corticosteroids in tuberculosis-associated acute respiratory distress syndrome is a subject of ongoing debate. Several studies indicate that corticosteroids may reduce excessive inflammatory responses and enhance oxygenation in specific patients [8,9]. Early initiation of antitubercular therapy is the most significant factor influencing survival.

Conclusion

Tuberculosis represents a rare yet significant etiology of acute respiratory distress syndrome, especially in regions where the disease is endemic. The presentation of ARDS is frequently unexpected, potentially resulting in delayed diagnosis because of nonspecific clinical and radiological characteristics. This case underscores the necessity for a heightened index of suspicion for tuberculosis in patients presenting with unexplained ARDS or inadequate response to empirical treatment. Timely microbiological confirmation and the swift commencement of antitubercular therapy, coupled with suitable respiratory support, are essential for enhancing patient outcomes.

Conflict of Interest

The authors declared no potential conflicts of interest with respect to the research, authorship and/or publication of this article.

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Data Availability Statement

Not applicable.

Ethical Statement

The project did not meet the definition of human subject research under the purview of the IRB according to federal regulations and therefore, was exempt.

Informed Consent Statement
Informed consent was taken for this study.

Authors' Contributions
All authors contributed equally to this paper.

References

1. Richeldi L, Ferrara G, Fabbri LM. Diffuse alveolar damage in miliary tuberculosis. Eur Respir J. 1993;6(7):1023-7.
2. Lee PL, Jerng JS, Chang YL. Patient mortality of pulmonary tuberculosis requiring mechanical ventilation. Eur Respir J. 2003;22(1):141-7.
3. Agarwal R, Aggarwal AN, Gupta D. Tuberculosis-associated acute respiratory distress syndrome: A systematic review. J Intensive Care Med. 2007;22(3):156-66.
4. Kim JY, Park YB, Kim YS. Prognostic factors of tuberculosis-associated acute respiratory failure. Int J Tuberc Lung Dis. 2008;12(12):1465-70.
5. Phua J, Badia JR, Adhikari NKJ. Has mortality from acute respiratory distress syndrome decreased over time? A systematic review. Am J Respir Crit Care Med. 2009;179(3):220-27.
6. Ranieri VM, Rubenfeld GD, Thompson BT. Acute respiratory distress syndrome: The Berlin definition. JAMA. 2012;307(23):2526-33.
7. Guérin C, Reignier J, Richard JC. Prone positioning in severe acute respiratory distress syndrome. N Engl J Med. 2013;368(23):2159-68.
8. Sharma SK, Mohan A. Acute respiratory distress syndrome in tuberculosis: A review. Indian J Med Res. 2013;137(3):385-97.
9. Bellani G, Laffey JG, Pham T. Epidemiology, patterns of care and mortality for patients with acute respiratory distress syndrome in intensive care units. JAMA. 2016;315(8):788-800.
10. Dheda K, Barry CE III, Maartens G. Tuberculosis. Lancet. 2016;387(10024):1211-26.
11. Mohan A, Sharma SK. Immune mechanisms in tuberculosis-associated acute respiratory distress syndrome. Clin Chest Med. 2019;40(4):733-44.
12. World Health Organization. Global tuberculosis report. Geneva: World Health Organization. 2025.