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Unusual Presentation of Pulmonary Tuberculosis with Massive Haemothorax Leading to Obstructive Shock: A Case Report

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Abstract
Tuberculosis (TB) remains a major global health burden and continues to pose significant diagnostic challenges due to its nonspecific clinical manifestations. Although pleural effusion is a common extrapulmonary manifestation of TB, massive spontaneous hemothorax leading to obstructive shock is exceedingly rare and potentially life-threatening. We report the case of a 56-year-old male who presented with progressive dyspnea and was initially diagnosed with cardiogenic shock and acute decompensated heart failure at a referring hospital. Physical examination revealed hypotension, tachycardia, tachypnea, elevated jugular venous pressure, and decreased breath sounds over the left lung. Imaging demonstrated massive left-sided pleural effusion with mediastinal shift. Bedside transthoracic echocardiography showed preserved biventricular function with significant extrinsic cardiac compression. Emergency chest tube placement evacuated approximately two liters of blood, resulting in rapid hemodynamic improvement and confirming the diagnosis of obstructive shock secondary to massive spontaneous hemothorax. Despite negative molecular testing for tuberculosis from sputum and pleural fluid, chest computed tomography revealed findings highly suggestive of pulmonary tuberculosis. Anti-tuberculosis therapy was initiated, leading to further clinical improvement. The patient was discharged in stable condition after five days of hospitalization. This case underscores pulmonary tuberculosis as a rare etiology of massive spontaneous hemothorax causing obstructive shock. Early recognition of obstructive shock, prompt mechanical decompression, and thorough etiological evaluation are critical for optimal outcomes. In the absence of microbiological confirmation, radiological findings play a pivotal role in establishing a diagnosis of clinically confirmed tuberculosis, and early initiation of anti-tuberculosis therapy may be lifesaving.

Introduction

Tuberculosis (TB) remains a major global health burden and is currently the second leading cause of mortality worldwide among all infectious diseases. Approximately 45% of global TB cases occur in Southeast Asia, with Indonesia accounting for nearly 10% of the worldwide TB incidence. TB is a highly transmissible disease, particularly in densely

populated and underserved areas. Its nonspecific clinical manifestations often result in delayed diagnosis, suboptimal management, and challenges in disease prevention (Tobin & Tristram, 2024; Kementerian Kesehatan RI, 2024).

Pleural effusion is defined as the abnormal accumulation of excess fluid within the pleural space. The effusion fluid may be classified as either exudative or transudative (Alaei et al., 2016; Cazabon et al., 2017; Floyd et al., 2018; Chakaya et al., 2021). Pleural effusion can represent a manifestation of various pulmonary and systemic disorders; therefore, identification of the underlying etiology is essential to ensure appropriate management (Karkhanis & Joshi, 2012).

Haemothorax is a specific type of exudative pleural effusion characterized by the presence of blood within the thoracic cavity. It is most commonly associated with trauma, coagulopathy, or iatrogenic causes. Haemothorax occurring in the absence of thoracic trauma is referred to as spontaneous haemothorax and represents a rare clinical entity (World Health Organization, 2023; World Health Organization, 2021; Ministry of Health Republic of Indonesia, 2021; Nhung et al., 2020). A large-volume haemothorax may result in significant hemodynamic compromise, including obstructive shock due to mechanical compression of the heart caused by blood accumulation within the thoracic cavity (Manzoor et al., 2021).

Case Description

This is a case of a 56-year-old male diagnosed with cardiogenic shock and acute decompensate heart failure (ADHF) in previous hospital referred to the ER with dyspnea for two months and being progressively aggravating for last one week before admitted. No history of trauma and no past medical history. Physical finding include hypotension, tachycardia, tachypnea with no desaturation and increased jugular vein pressure, distant heart sound, decreased breath sound with dull percussion on left lung.

12-leads ECG showed sinus rhythm with Heart Rate (HR) at 100 times per minute, normal axis, low voltage, poor R wave progression V2-V6 with occasional Premature Atrial Contraction (PAC). Chest X-ray (CXR) revealed massive pleural effusion on left lung with mediastinal shift to the right. Trans-thoracic Echocardiography (TTE) showed normal left ventricle (LV) and right ventricle (RV) function, Left pleural effusion with pressure on Left Atrium (LA) and LV, low cardiac output, low systemic vascular resistance (SVR) and minimal pericardial effusion.

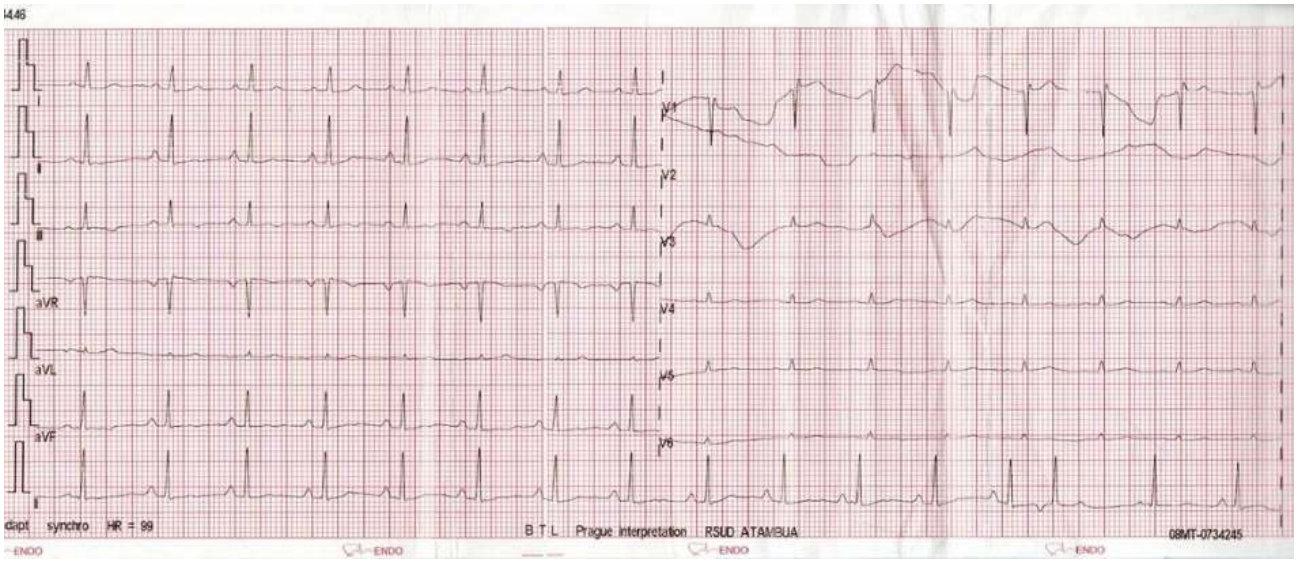

Figure 1. 12-Leads ECG showed SR with low voltage, poor R wave progression and Occasional PAC

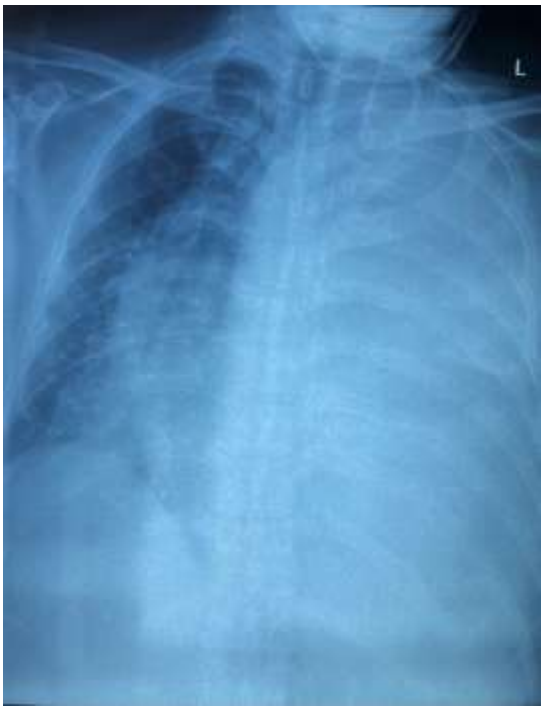

Figure 2. Chest X-Ray showed massive pleural effusion on left lung with mediastinal shift to the right

The patient was managed with fluid restriction, oxygen supplementation, vasopressor support, diuretic therapy, and immediate chest tube insertion, which resulted in the evacuation of approximately two liters of blood and significant clinical improvement. Empirical antibiotic therapy was also initiated. Based on the clinical presentation and response to intervention, the patient was diagnosed with obstructive shock secondary to massive spontaneous haemothorax.

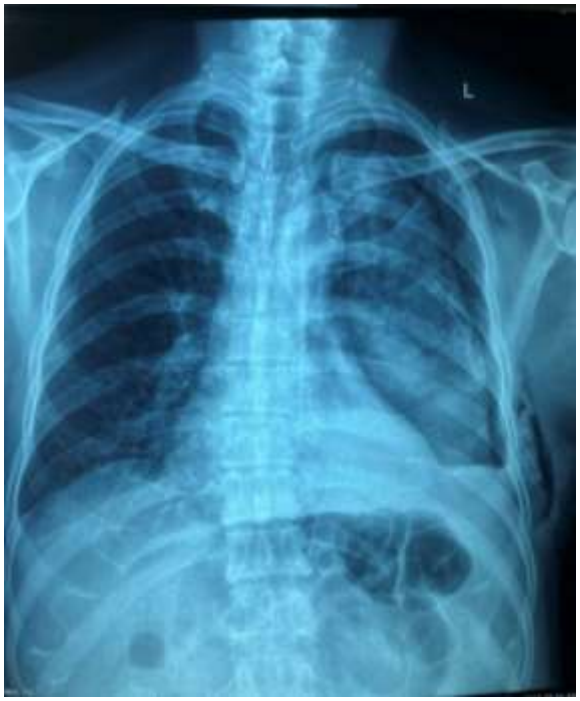

Figure 3. CXR evaluation after 48 hours chest tube placement showed hydropneumothorax on left lung with collapsed left lung and subcutaneous emphysema on lateral left hemithorax

Follow-up chest radiography revealed a left-sided hydropneumothorax with complete collapse of the left lung and subcutaneous emphysema along the lateral aspect of the left hemithorax. Sputum and effusion examination using a rapid molecular test for tuberculosis was performed to identify the underlying etiology; however, the result was negative. Laboratory evaluation demonstrated leucocytosis and mild anemia. Subsequently, the patient was referred for chest computed tomography (CT) to further investigate the underlying cause.

Chest computed tomography revealed left lung atelectasis with multifocal consolidation and diffuse ground-glass opacities, raising suspicion of pulmonary tuberculosis. Consequently, anti-tuberculosis therapy was initiated. The patient demonstrated further clinical improvement and was discharged after five days of hospitalization in stable condition.

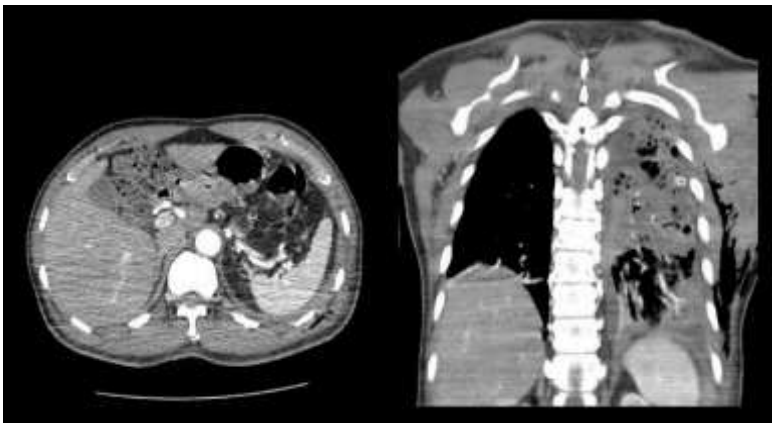

Figure 4. CT Scan showed left lung atelectasis with multifocal consolidation mixed diffused ground glass opacity on the left lung suspected tuberculosis

Discussion

Etiologically, obstructive shock results from impaired diastolic cardiac filling, leading to a reduction in venous return and consequently decreased preload of the right and/or left ventricle (Pai & Behr, 2016; Pai et al., 2016; MacLean et al., 2019; Kumar et al., 2020). Clinical manifestations of shock may include shivering, pallor, hypotension, tachycardia, tachypnea, hypoxemia, oliguria, and altered mental status. However, the most characteristic clinical features of obstructive shock are dyspnea and jugular venous distension. Notably, hypotension may initially be mild, potentially leading to underrecognition of the severity of the condition. Reduced or absent breath sounds over one hemithorax are typical findings in pneumothorax or hemothorax (Kim, 2018; Zotzmann et al., 2022).

In this case, the patient presented with hypotension, tachycardia, and tachypnea without oxygen desaturation or altered consciousness. Physical examination revealed elevated jugular venous pressure and decreased breath sounds with dullness to percussion over the left lung, suggestive of pleural effusion (Porcel, 2018; Zhai et al., 2016; Shaw et al., 2019; Porcel & Light, 2018). Chest radiography demonstrated a massive left-sided pleural effusion with mediastinal shift to the right, corroborating the clinical suspicion. Twelve-lead electrocardiography showed sinus rhythm with a heart rate of 100 beats per minute, normal electrical axis, low-voltage QRS complexes, poor R-wave progression in leads V2–V6, and occasional premature atrial contractions (Light, 2015; Lee et al., 2018; Ali et al., 2019; Martinez & Rodriguez, 2020). Low-voltage QRS complexes, which may represent an electrocardiographic correlate of tamponade physiology, can also be observed in conditions such as pleural effusion, emphysema, obesity, and anasarca (Zotzmann et al., 2022).

Bedside transthoracic echocardiography revealed preserved left and right ventricular systolic function, significant left-sided pleural effusion exerting compressive effects on the left atrium and ventricle, low cardiac output, reduced systemic vascular resistance, and minimal pericardial effusion. These findings support a mechanical obstruction to cardiac filling as the underlying mechanism of shock (Manzoor et al., 2021; Dogrul et al., 2018; Vincent & De Backer, 2013). The fundamental pathophysiology of obstructive shock involves a reduction in left ventricular preload. Although this initially leads to a compensatory increase in heart rate and myocardial contractility, stroke volume and cardiac output ultimately decline.

In patients presenting with shock, laboratory investigations are useful for identifying potential etiologies and associated complications. However, no laboratory parameter is specific for obstructive shock and diagnostic evaluation should not delay prompt therapeutic intervention (Zotzmann et al., 2022). The hallmark of obstructive shock lies in the rapid identification and immediate relief of the underlying mechanical obstruction, which typically results in swift hemodynamic improvement. Although there is concern that vasopressor therapy may increase pulmonary vascular resistance and potentially worsen hemodynamics, maintaining a mean arterial pressure of approximately 65 mmHg remains a reasonable target. Inotropic agents are generally not required and may be harmful due to their potential to induce vasodilation and arrhythmias (Kim, 2018; Zotzmann et al., 2022). In this case, the patient was managed with fluid restriction, oxygen supplementation, vasopressor support, diuretic therapy, and empirical antibiotics.

Immediate thoracic drainage was performed to address the cause of obstructive shock, resulting in the evacuation of approximately two liters of blood and significant symptomatic relief. Except in patients with overt heart failure, thoracentesis is recommended in individuals with more than minimal pleural effusion of unknown etiology. Diagnostic thoracentesis is also indicated in patients with heart failure when atypical features are present, including fever or pleuritic chest pain, unilateral or markedly asymmetric effusions, absence of cardiomegaly, or lack of response to standard heart failure therapy.

Most cases of hemothorax are associated with blunt or penetrating chest trauma or iatrogenic causes such as central venous catheter placement, thoracentesis, pleural biopsy, or cardiac catheterization. Spontaneous hemothorax is rare. Hemorrhagic pleural effusions have been reported in association with malignancy, tuberculosis, uremia, and vascular conditions such as pulmonary infarction. In this patient, further evaluation was undertaken to identify the underlying etiology. Laboratory studies demonstrated leukocytosis, suggestive of an infectious process. Tuberculosis is well known for its nonspecific clinical presentation, which frequently results in delayed diagnosis, inadequate treatment, and challenges in disease control. Patients with active tuberculosis may remain asymptomatic for extended periods and are often diagnosed only after significant disease progression.

Sputum and effusion specimens were examined using a rapid molecular test for tuberculosis, which yielded negative results. However, chest computed tomography demonstrated findings suspicious for pulmonary tuberculosis. Chronic infections such as tuberculosis may induce abnormal neovascularization between the parietal pleura and chest wall, predisposing to aneurysm formation and intrathoracic hemorrhage upon vessel rupture (Kim, 2018; Zotzmann et al., 2022).

Chest radiological examination remains a practical diagnostic modality for detecting tuberculosis-related lesions. Although more costly than sputum examination, imaging offers distinct advantages in specific clinical contexts, such as pediatric and miliary tuberculosis, where bacteriological confirmation is frequently negative (Kementerian Kesehatan RI, 2020).

In this case, despite negative bacteriological testing, radiological findings were consistent with tuberculosis and the patient failed to improve with non–anti-tuberculosis antibiotics, fulfilling the criteria for clinically confirmed tuberculosis. Anti-tuberculosis therapy was therefore initiated. Following several days of treatment, the patient demonstrated a reduction in leukocyte count and minimal reaccumulation of pleural effusion. The patient was subsequently discharged in stable condition with continuation of anti-tuberculosis therapy.

Conclusion

This case highlights pulmonary tuberculosis as a rare yet life-threatening cause of massive spontaneous hemothorax resulting in obstructive shock, where nonspecific symptoms and initially subtle hemodynamic changes can delay diagnosis and underestimate severity. Prompt recognition of obstructive shock, immediate thoracic drainage for mechanical relief, and thorough etiological assessment are essential for favorable outcomes, with radiological evidence proving pivotal for clinically confirmed tuberculosis when bacteriological tests are negative. Early anti-tuberculosis therapy, even without microbiological confirmation, can be lifesaving in patients showing suggestive clinical and imaging features. For future research,

prospective studies should investigate the incidence, optimal diagnostic algorithms (e.g., integrating advanced imaging like PET-CT with rapid molecular tests), and long-term outcomes of empiric anti-TB therapy in similar rare TB presentations to refine management protocols.

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