CASE REPORT

Successful Treatment of Cor Pulmonale in Drug-Resistant Tuberculosis-Related ARDS: A Case Report

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ABSTRACT

Introduction: One of the biggest challenges in global tuberculosis (TB) control is the spread of drug-resistant TB. Chronic cor pulmonale is confirmed in a pulmonary TB patient through the mechanism of pulmonary hypertension (PH). Tuberculosis is one of the causes of respiratory failure requiring mechanical ventilation. However, cases of TB requiring mechanical ventilation as the primary cause of respiratory failure are rare. Tuberculous acute respiratory distress syndrome (ARDS) is rare, but it has a very poor prognosis when it does occur.

Case: A 48-year-old female arrived with chronic cor pulmonale decompensated symptoms with drug-resistant pulmonary TB diagnosed by echocardiography. The patient developed ARDS during treatment. Initially, the patient was treated with loop diuretics, oxygen treatment, vasodilators, digitalis, anticoagulant medication, bronchodilators, and empirical antibiotics. As her condition deteriorated, the patient was put on mechanical ventilation and anti-TB medication. The patient's health improved, and she was allowed to return home.

Conclusion: Acute respiratory distress syndrome patients associated with drug-resistant TB may experience decompensated chronic cor pulmonale. In this instance, the main diagnostic method for cor pulmonale is echocardiography. Seldom is drug-resistant pulmonary TB associated with ARDS that results in cor pulmonale being documented. Positive results are linked to early diagnosis and treatment. Following therapy, the patient's health improved, and the patient was permitted to return home with the prescribed drugs.

INTRODUCTION

The whole world was concerned in 1993 about tuberculosis (TB), which was considered to be a global emergency. Now, people are becoming increasingly negligent about TB after they learned that the disease can be cured. The *Mycobacterium tuberculosis* (MTB) organism continues to evolve to combat antagonistic drugs. Both multidrug-resistant (MDR) TB and extensively drug-resistant TB, which have lower cure rates, are triggered by the advent of resistant strains. With the proliferation of resistance mechanisms, total drug-resistant (TDR) TB may emerge.[1]

Drug-resistant TB is a significant challenge for TB therapy and control programs. There were 558,000 new cases of TB worldwide with rifampin resistance in 2017, and of these, 82% developed MDR-TB.[2] Acute respiratory distress syndrome (ARDS) with mechanical ventilation is rarely reported in TB patients. The high mortality rate can be seen in TB patients who develop acute ARDS compared to other causes of ARDS. The range of the mortality rate is 47 to 80%.[3] Furthermore, TB can become a cause of ARDS through a process of sepsis and septic shock.[3]

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Although the World Health Organization (WHO) reports in 2020 indicated that up to 85% of new cases of TB have been successfully treated worldwide, the prognosis for patients who present to an intensive care unit (ICU) with multiorgan failure might still be very bad.[4] One of the leading causes of this mortality in low-income nations is delayed diagnosis and treatment because of a lack of recognition.[4]

Chronic cor pulmonale occurs when the right ventricular (RV) experiences increased afterload, resulting in significant dilation and remodeling.[5] Pulmonary arterial hypertension (PAH) occurs in about 5-10% of all heart conditions and contributes to 20-30% of hospital admissions for heart failure. Both PAH and chronic cor pulmonale can elevate the likelihood of hospitalization and reduce the survival rate. An echocardiographic assessment is a noninvasive method used to evaluate the degree of right ventricular hypertrophy (RVH) and PAH.[6]

Chronic cor pulmonale was pathomorphologically identified in 44.8% of patients who died from pulmonary TB.[7] Throughout their lives, 67.8% of these patients were diagnosed with chronic cor pulmonale.[7] Meanwhile, 32.2% received their diagnosis after death.[7] Among the deceased, chronic cor pulmonale that decompensated and resulted in death was found in 42.5%.[7]

Severe cardiopulmonary dysfunction may result from pulmonary hypertension (PH), a cardiovascular condition that worsens over time. Pulmonary arterial hypertension is a primary category of PH diseases that is distinguished by RV dysfunction and pulmonary vascular remodeling. Right heart failure is the primary cause of the prevalence and death rate in this disease, and is consequently driven by elevated pulmonary artery resistance. Pulmonary arterial hypertension has been linked to inflammation, which plays a vital part in the remodeling of blood vessels. The lesions in the pulmonary arteries in PAH patients are influenced by various immune cells and cytokines, which activate the downstream signaling pathways associated with inflammation.[8] We reported a case of TB linked to ARDS developing into cor pulmonale, even though pulmonary TB is seldom the leading cause of this complication.

CASE

A 48-year-old female patient arrived at the emergency department at our hospital with a complaint of shortness of breath that had persisted for one week. The patient complained of a productive cough with thick white phlegm lasting over two months. There were no signs of chest pain or coughing up blood. She had seen several general practitioners, but there had been no change, and it had worsened. When the patient was active, she experienced more shortness of breath and edema of the lower extremities. There was no history of diabetes mellitus, hypertension, chest trauma, congenital heart disease, or interaction with patients who have chronic or long-term coughs. The patient does not smoke. No one else in the family has had comparable issues. The person was diagnosed with TB two years ago, got treatment, and was then said to be healed.

During the vital sign assessment, it was discovered that the patient was conscious (compos mentis) with a blood pressure of 149/122 mmHg, 94 beats per minute for the pulse, 36.5°C for the temperature, 36 breaths per minute for the respiratory rate, and 56% oxygen saturation. The physical examination indicated fine crackles in the lower thirds of the right and left lungs, and edema in both lower limbs. The other physical exams were within normal ranges. Electrocardiography (ECG) revealed a 94 beats per minute sinus rhythm, right axis deviation, and hypertrophy of the right atrium and right ventricle. The blood test showed hemoglobin (Hb) 11.5 g/dl, leukocytes 11,600/uL, and neutrophils 81.9%. Her platelet count was 185,000, urea 30.95, and creatinine 3.39 mg/dL. Arterial blood gas (ABG) showed a pH of 7.2, partial pressure of oxygen (PO_2)=44 mmHg, partial pressure of carbon dioxide (PCO_2)=130 mmHg, and hydrogencarbonate (HCO_3)=24 meq/L. The arterial partial pressure of oxygen (PaO_2)/inspiratory fraction of oxygen (FiO_2) was 48.9. A chest X-ray revealed fibroinfiltrate, numerous cavities in the parahilar and infrahilar regions of the right and left lungs, and cardiomegaly with a raised cardiac apex caused by right ventricular dilatation. Echocardiography revealed dilatation of the right atrium and ventricle. The left ventricle had diastolic dysfunction but a systolic ejection fraction of 77.93%.

She was started on empirical antibiotics. Loop diuretics, nitrate vasodilators, antiplatelets, and angiotensin-converting enzyme (ACE) inhibitors were administered to treat the heart problem. The patient felt short of breath despite being given a non-rebreather mask (NRM) oxygen at 15 lpm, with the saturation increasing to 94%. On the third day of treatment, the patient experienced a decreased consciousness. The patient was put on mechanical ventilation and immediately treated in the intensive care unit (ICU). Despite receiving broad-spectrum antibiotics, the patient showed no signs of recovery. After that, an Xpert MTB/rifampicin (RIF) diagnostic test revealed that the patient resisted rifampicin. Line probe assay (LPA) revealed no resistance to second-line TB medications. The patient's clinical diagnosis was consistent with cor pulmonale brought on by pulmonary

TB and ARDS. In addition to resistant anti-TB medication therapy, which included moxifloxacin, bedaquiline, clofazimine, linezolid, and cycloserine, the patient received heart failure therapy because of the cor pulmonale.

The patient was placed on mechanical ventilation employing a lung protective strategy with a tidal volume of 360 ml (6 ml/kg), positive end-expiratory pressure (PEEP) of 6, and an FiO_2 of 80%. The patient showed increased respiratory effort, rapid breathing, and an elevated heart rate. Her requirement for oxygen remained high until the fourth day in the ICU. We increased the PEEP to 10 and maintained the FiO_2 at 80%. On the fifth day in the ICU, the patient started to show improvement, and PEEP was reduced. On the tenth day, the patient was extubated. On the 15th day, the patient was permitted to return home. The patient's condition improved while they were being monitored. An acid-fast bacilli (AFB) sputum test was then performed on the patient. The results were negative.

In this case, there were respiratory symptoms that worsened from day 3 to day 13 after hospitalization, requiring intubation. The chest X-ray showed bilateral ground-glass opacities. The PaO_2/FiO_2 ratio was 48.9, and left heart failure did not explain the presentation. This case shows that TB must be recognized and considered a cause of ARDS. There was an apparent enlargement of the right heart due to TB, which could be diagnosed as cor pulmonale.

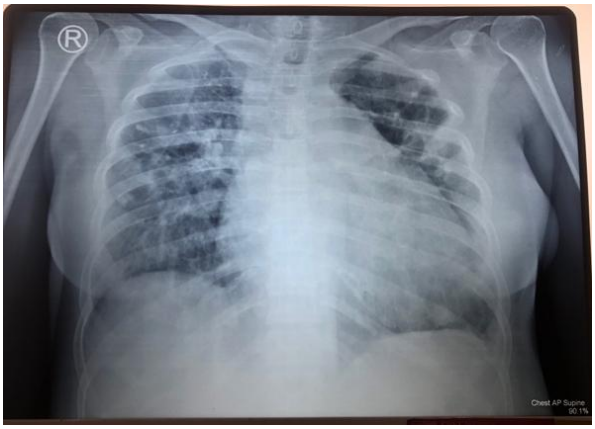

Figure 1. Chest X-ray showing bilateral infiltrates

DISCUSSION

Cor pulmonale is a disorder characterized by alterations in the anatomy and function of the heart's right ventricle, usually due to respiratory disease. In respiratory diseases, resistance to blood flow increases, which occurs in the pulmonary circulation. This condition is suspected to be PH. It can lead to right ventricular failure.[9,10]

Pulmonary artery hypertension is a severe cardiovascular condition marked by pulmonary vascular remodeling. It leads to elevated pulmonary artery pressure and eventual right ventricular failure. Pulmonary hypertension is a mean pulmonary arterial pressure (mPAP) over 20 mmHg, confirmed through hemodynamic evaluation via right cardiac catheterization.[11,12]

Pulmonary vascular dysfunction, characterized by irreversible tissue remodeling and occlusion, is often regarded as the principal pathogenic characteristic in the development of PAH. Alongside vascular remodeling, various perivascular inflammatory cells, namely mast cells, dendritic cells, monocytes, T and B lymphocytes, and macrophages, accumulate in the pulmonary perivascular spaces and surrounding remodeled pulmonary vessels.[13,14]

The increased pulmonary dead space fraction in ARDS contributes to the elevated afterload, significantly increasing the potential for cor pulmonale and right ventricular failure in affected people.[10,15,16] Increased pressure afterload, myocardial hypoxia, and metabolic dysfunction contribute to maladaptive remodeling of the right ventricle, significantly impacting morbidity and mortality in affected patients.[17]

In recent decades, numerous classical therapeutic agents for PAH have been extensively utilized in clinical settings. However, the prognosis for PAH patients remains inadequately improved. Current clinical drugs for treating PAH primarily consist of vasodilators, which do not address the fundamental pathophysiology of pulmonary vascular dysfunction. Targeted treatments focusing on inflammation offer a new pathophysiological approach with considerable therapeutic promise for treating PAH. These potential drugs may function as independent or complementary therapies by enhancing vascular remodeling. Recent studies have shown that various existing and novel immunosuppressants yield promising results in preclinical and clinical trials. Currently, no immunomodulator has been approved for the treatment of PAH. Considering the significant role of inflammation and immunity in this disease, anti-inflammatory and immunotherapeutic approaches may represent novel strategies for the clinical management of PAH.[8]

The right ventricular and pulmonary circulation function become uncoupled due to this rise in RV afterload. Since the right ventricle lacks a contractile reserve, its only adaptive reserve mechanism is dilatation, which causes left ventricular compression, deteriorating oxygen supply, and circulatory failure.[18] Cor pulmonale can occur in ARDS, pulmonary embolism, and patients using mechanical ventilation.[16,19] The routine assessment of RV function is critical in identifying cor pulmonale. They are often

treated in the emergency room before being transferred to the ICU.[16]

The symptoms of ARDS include severe hypoxemia, decreased lung compliance, and protein-rich alveolar edema. With a 28-day death rate of 30% in recent cohorts, ARDS continues to be a serious public health concern despite modest indications of mortality improvement over the past few decades. One of the key components of ARDS pathophysiology is pulmonary vascular dysfunction, which leads to a certain amount of PH, larger levels of which are linked to morbidity and death. Endothelial dysfunction, pulmonary vascular occlusion, elevated vascular tone, extrinsic vessel occlusion, and vascular remodeling all contribute to the development of PH.[18]

Acute respiratory distress syndrome is severe and frequently lethal. Respiratory viruses, smoke particles, and saltwater inhalation are some causes of ARDS. Systemic problems, including sepsis, pancreatitis, or blood transfusions, can potentially result in ARDS. Unfortunately, there are not many choices for treating ARDS outside of last-resort measures like mechanical ventilation and extracorporeal support techniques, even though the death rate is high (40%).[19]

People affected by post-TB lung disease (PTLD) have a shortened life expectancy and increased risk of recurrent TB, but the predictors of long-term outcomes are not known. Multiple episodes of TB, drug-resistant TB, delays in diagnosis, and possible smoking are all risk factors for PTLD. Pulmonary vascular disease in the post-TB population due to lung damage is often found in patients with advanced cor pulmonale.[20]

Active TB and PTLD, as well as host-related and environmental-related elements, probably contribute to the multifactorial predisposing risk factors for PH development.[21] It is also predicted that almost 10% of the TB population suffers from active TB PH.[22] Individuals who have a history of drug-resistant TB are more prone to developing post-TB PH. Limited data exist on the link between drug-resistant TB and post-TB PH.[23]

The effective management of cor pulmonale involves optimizing the ventilator settings to reduce driving pressure and improve gas exchange, utilizing prone positioning to enhance pulmonary function and reduce RV afterload.[18] Pharmacological interventions and extracorporeal membrane oxygenation (ECMO) should be considered for severe cases.[24] Echocardiography is necessary to diagnose RV dysfunction, and specific markers such as tricuspid annular plane systolic excursion (TAPSE) are crucial.[10] Pharmacotherapy for PAH and, in extreme situations, cutting-edge mechanical support systems, such as oxygenated RV assist devices, may be part of management.[25]

Finding and treating the triggering factors, enhancing contractility using inotropes, decreasing afterload, and adjusting volume to enhance diastolic ventricular interactions with medications that target the pulmonary circulation are all examples of effective therapeutic management techniques.[26] Therapy for right heart failure due to cor pulmonale focuses on optimizing preload, reducing afterload, and enhancing contractility. This includes pharmacologic treatments, invasive monitoring, and potentially mechanical circulatory support, tailored to the underlying cause of PH.[27] Agents like phosphodiesterase-5 inhibitors and soluble guanylate cyclase stimulators can improve RV function by targeting pulmonary vascular resistance.[26] Good therapy for right heart failure requires the rapid recovery of adequate arterial blood pressure. Volume optimization with vasopressors and/or inotropes is necessary.[28]

Pulmonary TB can manifest as either parenchymal or extraparenchymal disease. Mortality rates for TB patients as the primary cause of respiratory failure necessitating mechanical ventilation range from 47% to 80%.[29] Acute respiratory distress syndrome as a manifestation of TB is infrequently documented.[29,30] Approximately 3.4% of hospitalized TB patients must be admitted to the ICU. The prognosis for patients who required ICU hospitalization is dismal and has a significantly elevated mortality rate relative to other etiologies of severe pneumonia. The primary reason for ICU admission related to TB is acute respiratory failure, which may arise from pneumonia or ARDS. This is followed by septic shock accompanied by multiple organ dysfunction, adrenal insufficiency, and neurological complications, particularly tubercular meningitis. Tuberculosis patients necessitating ICU admission are predominantly immunocompromised, often due to human immunodeficiency virus (HIV) coinfection, and typically present with underlying miliary or disseminated TB. Pulmonary TB manifesting as ARDS is uncommon. However, it is a prevalent reason for the admission of TB patients to the ICU.[31]

Drug-resistant TB is estimated to account for 13% of all deaths attributable to antimicrobial resistance worldwide. This is due to continuous resistance and transmission from person to person. Inadequate access to efficient treatment and delayed diagnosis make this worse.[32] Drug-resistant TB has become a global burden related to antimicrobial resistance. There is a need for effective antibiotic management and focused efforts to create new regimens targeting drug-resistant strains. To reduce the spread of drug-resistant strains, contact tracing, active mapping of social contacts, and long-term follow-up for symptoms in TB patients can be conducted. To limit and prevent further transmission and the progression of the disease, prophylaxis is

needed for patients who have come into contact with drug-resistant patients. To prevent the evolution of drug-resistant strains, it is crucial to develop shorter treatment regimens that quickly eradicate all populations of mycobacteria while simultaneously inhibiting the metabolic processes that contribute to drug tolerance, the emergence of resistance, and mutagenesis.[33]

The creation of the Xpert MTB/RIF diagnostic test assay marked a substantial advancement in the global diagnosis of TB and the identification of rifampicin resistance. Xpert MTB/RIF should be employed as the primary diagnostic test for TB and rifampicin resistance detection in individuals exhibiting signs and symptoms of pulmonary TB. The application of molecular LPA to identify resistance to second-line anti-TB medications and to isoniazid and rifampicin is necessary.[34]

Smear microscopy, culture, and phenotypic drug susceptibility testing (pDST) should be replaced by Xpert MTB/RIF as the primary diagnostic test for TB and rifampicin resistance (RR) detection in sputum in individuals without a prior TB history (≤5 years) or with a distant TB treatment history (>5 years since treatment completion). Xpert Ultra should be used as the first diagnostic test for people who have had a history of TB and whose treatment was completed within the last five years. It is for TB and RR detection in sputum, replacing pDST, culture, and smear microscopy. Additional testing with Xpert Ultra is not allowed if the first test results in a positive trace.[34]

Truenat MTB or MTB Plus can be the primary diagnostic test for TB, supplanting smear microscopy or culture. A positive result from Truenat MTB or MTB Plus allows for using Truenat MTB-RIF Dx as an initial test for rifampicin resistance, replacing phenotypic drug susceptibility testing. Additionally, moderately complex automated nucleic acid amplification tests can be utilized on pulmonary specimens to identify pulmonary TB, rifampicin, and isoniazid resistance instead of culture and phenotypic drug susceptibility testing.[34]

Treatment for drug-resistant TB is more challenging than that for non-drug-resistant TB. Drug-resistant TB comes in a variety of forms, including extensive drug resistance (XDR), MDR, and RR. Only rifampin causes RR-TB to become resistant. Other first- or second-line medications do not. Resistance to at least two of the most effective anti-TB drugs, isoniazid and rifampin, is known as MDR-TB. It was discovered that 82% of the 558,000 RR-TB cases that were reported globally in 2017 were MDR-TB.[2]

In patients with MDR/RR-TB on long-term treatment, a minimum of four TB drugs should be started. All three drugs should be from group A, and at least one should be from group B. If only one or two group A drugs are used, both group B drugs should be added. If the patient cannot be treated with medications from group A and B, then group C drugs are added to supplement them.[35]

Group A drugs are fluoroquinolones. These include levofloxacin and moxifloxacin. Other drugs are bedaquiline and linezolid. Both of these drugs are highly effective and are highly recommended to be given to patients unless there are contraindications. Group B drugs are clofazimine, cycloserine, or terizidone. These two drugs are recommended to be given to patients unless contraindicated. It is also a second-choice agent. Group C drugs include all other drugs that can be used when group A and B drugs cannot be given to the patient.[35]

In this case, the patient was a female with a history of previous TB, no history of hypertension, diabetes mellitus, or heart failure. She came with right heart failure and ARDS. She was treated in the ICU with chronic cor pulmonale medication and given broad-spectrum antibiotics. The patient then experienced a decrease in consciousness and was immediately given mechanical ventilation. A TCM test was conducted after considering other diagnoses, such as TB. The result was rifampicin resistance. After being given TB medication, she got better. In TB patients, we must always consider the possibility of concurrent diagnoses such as ARDS, especially in patients from developing countries, even if there are no clear comorbidities or immunosuppression. She came in with a worsening shortness of breath and lower extremity edema, indicating right ventricular heart failure due to chronic cor pulmonale.

CONCLUSION

Acute respiratory distress syndrome patients associated with drug-resistant TB may experience decompensated chronic cor pulmonale. In this instance, the primary diagnostic method for cor pulmonale is echocardiography. Seldom is drug-resistant pulmonary TB associated with ARDS that results in cor pulmonale documented. Positive results are linked to early diagnosis and treatment. Following therapy, the patient's health improved, and she was permitted to return home with the prescribed drugs.

Consent

Written informed consent was obtained from the patient.

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Conflict of Interest
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Author's Contribution
AS: Conceptualization, writing the original draft preparation, supervision, and review.

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