Case Report

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Co-infection of COVID-19 and pulmonary tuberculosis: a case report on lung and liver damage

Gudisa Bereda, BSc Pharm, MPH Fellow*

Background: Co-infection with COVID-19 and tuberculosis (TB) often leads to symptoms such as fever, coughing, low blood pressure, altered blood cell counts, elevated liver enzyme levels, and reduced hemoglobin. Additionally, there is significant concern regarding lung damage caused by severe COVID-19, which increases susceptibility to TB.

Case presentation: On May 19, 2022, a 50-year-old retired Black African woman visited the emergency department with a sore throat, a 12 kg weight loss over the previous month, and shortness of breath that began a day earlier. She also experienced muscle weakness, fever, a productive cough, loss of appetite, headache, and 3 days of night sweats. Chest auscultation revealed reduced breath sounds in the right upper and middle lung regions. A throat swab polymerase chain reaction (PCR) test confirmed a COVID-19 diagnosis 36 hours later. During her hospital stay, the patient required continuous oxygen therapy via a nasal cannula at 4 L per minute for the first 5 days. She remained on anti-tubercular medication and received intravenous ceftriaxone at a dosage of 1 g daily for 5 days to prevent hospital-acquired infections.

Clinical discussion: Patients with active pulmonary tuberculosis (TB) have a poorer prognosis and are at risk of developing COVID-19. Both TB and COVID-19 are infectious diseases that primarily target the lungs. Mortality rates associated with COVID-19 are notably higher in individuals with active pulmonary TB.

Conclusion: COVID-19 and Mycobacterium tuberculosis are among the most critical global health challenges. TB infection is linked to a heightened risk of severe COVID-19 outcomes.

Keywords: case report, COVID-19, Mycobacterium tuberculosis, pulmonary tuberculosis, SARS-CoV-2

Introduction

The terms "tuberculosis (TB)" and "coronavirus disease-19 (COVID-19) infection" refer to diseases caused by Mycobacterium tuberculosis and the SARS-CoV-2 virus, respectively[1]. Currently, the COVID-19 pandemic and the global burden of disease due to Mycobacterium tuberculosis remain two of the most significant health challenges worldwide[2]. COVID-19, as an acute viral infection, has a profound impact on the lungs, often leading to hospitalizations and fatalities[3]. The virus can directly cause viral pneumonia through its effects on the lungs [4], and it also significantly affects and challenges the immune system[5]. When COVID-19 and tuberculosis are co-infected, patients with tuberculosis face a higher risk of mortality [6]. There are shared clinical and radiological features between COVID-19 and tuberculosis[7], as both are highly contagious respiratory diseases[8]. Common symptoms of COVID-19 include fever, cough, malaise, myalgia, and sore throat. Some patients may also experience gastrointestinal symptoms such as nausea, vomiting, and diarrhea. Most COVID-19 cases are mild to moderate, with those exhibiting mild symptoms typically requiring only supportive care[9][10]. This case study highlights a retired woman diagnosed with pulmonary tuberculosis and confirmed COVID-19 infection. It further discusses the development of liver damage in patients co-infected with pulmonary tuberculosis and COVID-19. The case report was prepared in compliance with the SCARE criteria[11].

HIGHLIGHTS

- Tuberculosis (TB) and COVID-19 are infectious diseases that primarily affect the lungs.
- Both TB and COVID-19 are highly transmissible respiratory diseases.
- SARS-CoV-2 spreads through respiratory droplets and potentially through airborne particles.
- Co-infections of COVID-19 and tuberculosis exhibit overlapping clinical features, including fever and other symptoms.

Case presentation

On May 19, 2022, a 50-year-old retired Black African woman was hospitalized with complaints of a sore throat, a 12 kg weight loss

Pharmacy Department, All Africa Leprosy, Tuberculosis and Rehabilitation Training Centre, Addis Ababa, Ethiopia

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**Corresponding author. Address: Pharmacy Department, All Africa Leprosy, Tuberculosis and Rehabilitation Training Centre, Addis Ababa 182609, Ethiopia. Tel: + 251 913118492/910790650. E-mail: gudisabareda95@gmail.com (G.Bereda).*

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over the past month, and one day of shortness of breath. She had no family medical or pharmaceutical history and no recent travel history. Her son, a healthcare worker, had been diagnosed with COVID-19 five days prior. The patient had a history of smoking and alcohol use for 20 years and was treated for pulmonary tuberculosis (TB) a year earlier. Her TB treatment involved a 2-month intensive phase with rifampicin 225 mg, isoniazid 150 mg, pyrazinamide 450 mg, and ethambutol 400 mg, followed by a 4-month continuation phase with rifampicin 225 mg and isoniazid 150 mg, to which she adhered strictly. She arrived at the emergency room in stable hemodynamic condition.

Upon arrival, the patient exhibited muscle weakness, fever, productive cough, sore throat, loss of appetite, headache, night sweats, and 1 day of shortness of breath. Seven days prior, she had been in good health. Her vital signs included a body temperature of 38.5°C, weight of 52.8 kg, height of 1.72 m, BMI of 17.9 kg/m^2, blood pressure of 113/76 mmHg, respiratory rate of 19 breaths per minute, heart rate of 112 beats per minute, and oxygen saturation of 88% on room air.

Initial blood tests revealed a blood urea nitrogen of 46 mg/dL, serum creatinine of 2.3 mg/dL, serum sodium of 151 mEq/L, leukocytes at 4620/μL, platelets at 139 200/μL, neutrophils at 65%, hemoglobin at 15.1 g/dL, aspartate aminotransferase (AST) at 68 U/L (normal: 0 -30 U/L), alanine aminotransferase (ALT) at 95 U/L (normal: 0 -35 U/L), erythrocyte sedimentation rate (ESR) at 9 mm/hr (normal: 0 -20 mm/hr), lymphocytes at 26%, and serum potassium at 5.1 mEq/L. Arterial blood gas results showed a pH of 7.37, partial pressure of carbon dioxide ($PaCO_2$) at 29 mmHg, partial pressure of oxygen (PaO_2) at 71 mmHg, and bicarbonate at 18 mEq/L. She received oxygen therapy at 4 L/min via nasal cannula and was later moved to the intensive care unit (ICU).

A chest X-ray revealed pleural empyema, a significant cavity in the right upper lobe, opacity in the right middle lobe, and right hilar fullness. Abdominal examination showed a distended, tight abdomen with fluctuating dullness but no discomfort, rebound tenderness, or palpable lymph nodes. Chest auscultation detected diminished breath sounds in the right middle and upper lung fields. An electrocardiogram (ECG) indicated sinus tachycardia and anterolateral ST depression. A sputum sample tested positive for Mycobacterium tuberculosis via GeneXpert, confirming rifampicin-sensitive TB. The patient had no prior confirmed COVID-19 infection. Nasopharyngeal swabs performed 1 day after admission confirmed COVID-19 infection, prompting her transfer to the ICU.

In the ICU, she received 4 L/min of oxygen through a nasal cannula and was treated with subcutaneous enoxaparin 80 mg every 12 hours for COVID-19-associated complications. Acetaminophen 500 mg was administered as needed for fever. On day 5, enoxaparin was replaced with low-molecular-weight heparin (1 mg/kg daily) for thromboprophylaxis. She also received intravenous ceftriaxone 1 g daily for 5 days to manage hospital-acquired infections, followed by oral azithromycin 500 mg daily for 5 days.

By day 6, her condition stabilized, and she no longer required supplemental oxygen. She resumed her anti-tubercular therapy, which consisted of a 2-month intensive phase (rifampicin 225 mg, isoniazid 150 mg, pyrazinamide 450 mg, and ethambutol 400 mg) and a 4-month continuation phase (rifampicin 225 mg and isoniazid 150 mg).

Follow-up and outcome

The patient remained clinically stable throughout 15-day hospital stay and was discharged on June 4, 2022, following two consecutive negative throat swab results for COVID-19. She was sent home with instructions to follow up at the tuberculosis clinic after achieving two consecutive negative sputum acid-fast bacilli smear results. Additionally, she was advised to continue monthly follow-up visits at the tuberculosis clinic.

Discussion

Coronavirus disease (COVID-19) is a respiratory disease caused by the novel beta coronavirus SARS-CoV-2, which leads to severe acute respiratory syndrome[12]. This new, fatal disease is primarily characterized by respiratory symptoms, though it has been linked to other respiratory pathogens[13]. In the case presented, the patient's lungs were severely affected by fluid leakage from small blood vessels, which reduces lung capacity to absorb oxygen, leading to symptoms such as shortness of breath, coughing, and dyspnea.

Tuberculosis (TB) is a bacterial infection that impacts both the lungs and other organs[14]. In this case, the COVID-19 infection exacerbated the patient's airway inflammation, narrowing the airways, increasing air velocity, and redirecting air to unaffected regions. This led to a worsening of her pre-existing pulmonary tuberculosis.

Mycobacterium tuberculosis spreads through aerosolized droplets when infected individuals cough, sneeze, speak, or breathe [15]. The patient likely contracted COVID-19 from one of her sick sons through airborne droplets, or from close contact, such as handshaking, prior to his confirmed positive test. Both airborne transmission and intimate contact are established routes for the spread of SARS-CoV-2. Clinical signs such as fever, fatigue, chest discomfort, cough, weight loss, night sweats, and lack of appetite are common for tuberculosis and often overlap with symptoms of COVID-19 [16]. In this patient, the symptoms included muscle weakness, fever, productive cough, sore throat, loss of appetite, headache, night sweats, and shortness of breath, all of which are characteristic of both infections.

Active tuberculosis stimulates the immune system, and COVID-19 infection further amplifies the immune response, potentially leading to an uncontrolled immune reaction and cytokine storm, which can cause pulmonary and multi-organ dysfunction. COVID-19 has been shown to deplete T-cells, rendering them non-functional, which may contribute to the reactivation of tuberculosis[17]. Mycobacterium tuberculosis also manipulates macrophages to reduce their defensive capacity, further weakening the immune system.

Additionally, COVID-19 can lead to pneumonia, intubation, ICU admission, and even death, with severe complications such as acute respiratory distress syndrome, sepsis, kidney failure, and multi-organ failure. Older individuals and those with comorbid conditions are at higher risk of mortality [18]. In this case, the patient displayed elevated liver enzymes, likely due to either the tuberculosis medications or COVID-19-induced hepatitis. Her blood tests showed increased levels of aspartate aminotransferase (68 U/L) and alanine aminotransferase (95 U/L), indicative of liver injury.

The anti-tubercular medications pyrazinamide, isoniazid, rifampicin, and ethambutol, commonly included in her

treatment plan, are known to increase hepatic enzyme levels. Of these, pyrazinamide is the most potent in raising hepatic enzyme levels, followed by isoniazid, rifampicin, and ethambutol. These drugs are primarily associated with hepatotoxicity as a side effect. The degree of liver damage can vary, ranging from mild to severe, with each medication contributing to the overall risk of liver toxicity. This combination of drugs heightens the likelihood of hepatic injury.

In addition to immune system damage, the increased hospitalizations and ICU stays associated with COVID-19 further exacerbate liver function impairment. Inflammatory mediators released during COVID-19 infection contribute to this process. Currently, reverse transcription polymerase chain reaction (RT-PCR) is the most sensitive diagnostic test for COVID-19, as it detects viral RNA[19]. Regular testing with nasopharyngeal swabs remains essential for accurate diagnosis.

Conclusion

The COVID-19 pandemic presents a significant risk to individuals with tuberculosis. Those with tuberculosis who contract COVID-19 may face worse outcomes, as pre-existing pulmonary disease is associated with higher mortality rates in COVID-19 patients. Pyrazinamide, followed by isoniazid, rifampicin, and ethambutol, is associated with an increase in hepatic enzyme levels. All the medications in her anti-tubercular regimen have hepatotoxic effects, ranging from severe to less common forms of liver toxicity. COVID-19 impairs liver function by triggering the release of various inflammatory mediators into the bloodstream.

Ethical approval

This case report did not require review by ethics committee.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A Copy of the written informed consent is available for review by the editor-in-chief of this journal on request.

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Author's contribution

G.B. contributed to the preparation of the proposal and participated in preparing the first draft of the manuscript and edits of the manuscript. The author checked and confirmed the final version of the manuscript.

Conflicts of interest disclosure

The authors declared that they have no competing interest.

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