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Confirmed COVID-19 in a Patient with Pre-Existing Pulmonary Tuberculosis: A Case Report

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Case Report

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Abstract

Co-infection with COVID-19 and TB frequently results in fever, coughing, hypotension, a changed blood cell count, raised liver enzymes, and decreased hemoglobin. On May 19, 2022, a retired black African woman in her 59s arrived at the emergency room with a tuberculosis diagnosis. She had no recent travel history and was COVID-19-infected. Five days prior to her admission, her sons, who worked in the medical field, were found to be infected with the COVID-19 infection. The patient arrived at the emergency room with weak muscles, a fever, a productive cough, a sore throat, a lack of appetite, a headache, night sweats, and shortness of breath that had lasted for a day. On chest auscultation, there were reduced breath sounds in the right middle and upper lungs. A Mycobacterium tuberculosis infection without rifampicin resistance was detected in a sputum sample using the GeneXpert Mycobacterium tuberculosis and rifampicin tests. After 36 hours, a throat swab polymerase chain reaction test produced positive findings, confirming the presence of the COVID-19 infection. The patient required continuous oxygen at a rate of four liters per minute via a nasal cannula for the first five days while in the hospital, and she is still taking her anti-tubercular medications. In order to combat hospital-acquired infections, she was given therapy with broad-spectrum antibiotics consisting of 500 mg of azithromycin given once a day for five days and 1 g of intravenous ceftriaxone administered daily for five days.

Introduction

The terms "tuberculosis" and "COVID-19 infection" refer, respectively, to the diseases brought on by Mycobacterium tuberculosis and SARS-CoV-2 infection [1]. The two biggest continuing health problems on the globe now are the COVID-19 pandemic and the epidemic of sickness brought on by Mycobacterium TB [2]. Acute viral infections like COVID-19 often and severely impact the lungs, which can result in hospitalizations and fatalities [3]. Viral pneumonia can result from the virus's direct impact on the lungs [4]. SARS-CoV-2 also has a significant impact on and challenges the immune system [5]. When COVID-19 and tuberculosis are co-infected, patients with tuberculosis are more likely to die [6]. There are several clinical and radiological characteristics that COVID-19 and tuberculosis patients have in common [7]. Both are respiratory viruses that are very contagious [8]. Fever, cough, malaise, myalgia, and a sore throat are some of the symptoms of a COVID-19 infection. Some people experience gastrointestinal problems, including nausea, vomiting, and diarrhea. The majority of COVID-19 patients have mild to moderate illness, and those with mild disease frequently only require supportive care [9–11]. This case study shows that the retired woman had pulmonary tuberculosis and a validated COVID-19 infection.

Case Report

On May 19, 2022, a retired black African woman in her 59s was admitted to the hospital with tuberculosis.Muscle soreness, a loss of weight of roughly 12 kg during the previous month, headaches, and a cough were the main symptoms of the admitted female. She had recently lost weight, had nocturnal sweats, and had a history of shortness of breath. Although she had no family medical history,

they did have prior medical and pharmaceutical histories. She had no recent travel history and was COVID-19-infected. Five days prior to her admission, her sons, who worked in the medical field, were found to be infected with the COVID-19 infection. She has a history of drinking or using cigarettes going back 20 years. One year prior, she had pulmonary tuberculosis in her past. She had a history of receiving first-line anti-tuberculosis medication, which included four months of continuous phase treatment with rifampicin 150 mg and isoniazid 75 mg after two months of intensive phase treatment with rifampicin 150 mg, isoniazid 75 mg, pyrazinamide 400 mg, and ethambutol 275 mg. She had adhered strictly to her antitubercular therapy regimen. She arrived at the emergency room with stable hemodynamics.

The patient arrived at the emergency room with weak muscles, a fever, a productive cough, a sore throat, a lack of appetite, a headache, night sweats, and shortness of breath that had lasted for a day. Seven days before being admitted, she was in good health. Her vital signs upon entering the emergency room were as follows: 38.5°C body temperature, 52.8 kg body weight, 1.72 m height, 17.9 kg/m2 body mass index, 113/76 mmHg blood pressure, 19 cycles per minute respiratory rate, 112 beats per minute peripheral pulse, and 88% oxygen saturation in ambient air.

Upon admission to the emergency department, her blood chemistry showed a blood urea nitrogen of 46 mg/dl, serum creatinine 2.3 mg/dl, serum sodium 151 mEq/L, leukocytes 4,620/L, platelets 139,200/L, neutrophils 65%, hemoglobin 15.1 g/dL, an aspartate aminotransferase level of 68 units/L (normal value: 0–30 units/L), an alanine aminotransferase level of 95 units/L (normal value: 0–35 units/L), an erythrocyte sedimentation rate of 9 mm/hour (normal value: 0–20 mm/hr), lymphocytes 26%, and serum potassium 5.1 mEq/L. An arterial blood gas test showed a potential hydrogen of 7.37, a partial pressure of carbon dioxide of 29, a partial pressure of oxygen of 71, and a bicarbonate of 18. She received 4 liters of intranasal oxygen per minute via a nasal cannula until she was moved to the respiratory isolation room.

On arrival, a chest radiograph revealed a pleural empyema, a significant right upper lobe cavity, right middle lobe opacity, and right hilar fullness. He had no discomfort, rebound tenderness, or visible lymph nodes upon abdominal examination, but did have a bloated, tight abdomen with fluctuating dullness. On chest auscultation, there were reduced breath sounds in the right middle and upper lungs. Her Glasgow coma scale was 10/15 after assessment. With a heart rate of 112 beats per minute, his electrocardiogram revealed anterior-lateral ST depression and sinus tachycardia. A Mycobacterium tuberculosis infection without rifampicin resistance was detected in a sputum sample using the GeneXpert Mycobacterium tuberculosis and rifampicin tests. According to a tuberculosis culture, Mycobacterium tuberculosis was responsive to first-line anti-tuberculosis medications.

She has never before had a proven COVID-19 infection. She was tested for COVID-19 using two nasopharyngeal swabs in the emergency room one day after being hospitalized, and because the results were positive, she was transferred to the intensive care unit. The patient was admitted to an intensive care unit with a diagnosis of previously well-controlled pulmonary TB and a recently confirmed COVID-19 infection after spending two days in the emergency room.

To control the COVID-19 infection, the patient was moved to a respiratory isolation unit. The patient required continuous oxygen at a rate of four liters per minute via a nasal cannula for the first five days while in the hospital, and she is still taking her anti-tubercular medications.

She started receiving 1000 ml of fluid resuscitation (0.9% normal saline) and insulin by drip as soon as she was admitted to a respiratory isolation unit. As part of the protocol for managing SARS-CoV-2, she continued to breathe in oxygen at a rate of 4 liters per minute using a nasal cannula. She received subcutaneous enoxaparin 80 mg 12 hours a day to treat her proven COVID-19 infection. To treat her COVID-19 infection-related fever, she was given 500 mg of acetaminophen as required. After five days, enoxaparin was changed to low molecular weight heparin at a dosage of 1 mg/kg daily as a prophylactic for prothrombotic events. In order to combat hospital-acquired infections, she was given therapy with broad-spectrum antibiotics consisting of 500 mg of azithromycin given once a day for five days and 1 g of intravenous ceftriaxone administered daily for five days. The patient was stable on the sixth day and did not need oxygen treatment. She maintained her treatment for tuberculosis, which consisted of a two-month intensive phase including rifampicin 150 mg, isoniazid 75 mg, pyrazinamide 400 mg, and ethambutol 275 mg, and a four-month continuous phase involving rifampicin 150 mg and isoniazid 75 mg.

Patient perspective

The patient remained clinically stable after 15 days in the hospital and was released on June 4, 2022, after the results of two consecutive negative throat swab tests for COVID-19 infection. The patient was sent home with a follow-up visit at the tuberculosis clinic after two consecutive negative sputum acid-fast bacilli smear results. She was advised to continue monthly follow-up at the tuberculosis clinic.

Discussion

Coronavirus disease is a respiratory illness brought on by the new betacoronavirus coronavirus 2, which causes severe acute respiratory syndrome [12]. The SARS-CoV-2-related disease COVID-19 is a brand-new, lethal illness that is primarily defined by a respiratory condition. The COVID-19 infection has been linked to other respiratory pathogens [13]. Herein, the patient's lungs are seen to be severely impacted by fluid seeping from her small blood vessels in the lungs, which limits her lungs' capacity to absorb oxygen and results in shortness of breath, coughing, and dyspnea.

The lungs and other organs are affected by the bacterial infection known as tuberculosis [14]. In this study, COVID-19 infection caused the patient's airway inflammation to drive airway blockage, making the airways narrower, which increases air velocity and displaces air to unobstructed regions. As a result, the patient's pulmonary tuberculosis got worse. Aerosol droplets containing Mycobacterium tuberculosis are inhaled to spread the disease. The formation of necrotizing granulomas, which primarily affect the lungs but also include other extrapulmonary locations, is what distinguishes it. SARS-CoV-2 spreads through droplets and, most likely, airborne transmission [15]. In this case, the patient is suspected of contracting

COVID-19 from one of her sick sons through airborne droplets caused by his coughing, sneezing, speaking, or breathing in minute liquid particles through his mouth or nose. She may have contracted COVID-19 from him because he shook her mother's hand to greet her and she had close contact with her boys prior to his confirmation of a positive COVID-19 infection. Either airborne droplets or intimate contact with an infected individual have been proven to be the proper route for COVID-19 virus transmission.

Clinical characteristics such as fever, exhaustion, chest discomfort, coughing, weight loss, night sweats, and a lack of appetite have been recognized as indicators of tuberculosis screening that detect signs and symptoms. Patients with COVID-19 also frequently display these symptoms. Therefore, individuals who have both COVID-19 and tuberculosis co-infections have the same set of clinical traits [16]. In this study, the patient symptoms were: weak muscles, a fever, a productive cough, a sore throat, a lack of appetite, headaches, night sweats, and shortness of breath. She experienced the clinical symptoms of both tuberculosis and the COVID-19 infection, which include fever, coughing, shortness of breath, and weak muscles.

Active tuberculosis infection stimulates the host's immunological response, and COVID-19 infection enhances the immune load even more. This might result in an out-of-control immune system and cytokine storm that cause pulmonary and multiorgan dysfunction. Tuberculosis reactivation is also explained by T-cell depletion brought on by the COVID-19 infection. Patients with COVID-19 had much lower T-cell numbers, and even the remaining T-cells were no longer functional [17]. The mycobacterium tuberculosis tricked the macrophages into lowering their defense against it, which decreased the immune system of the patient in this study. In this case report, COVID-19 infection boosts signaling pathways, increases interferon I production, and activates immune cells' chemotaxis.

Similar to the previous coronavirus, which attached to the target cell via angiotensin-converting enzyme 2, the SARS-CoV-2 virus is thought to attach to the host target cell in a similar manner. SARS-CoV-2 virus carriers may have complications that result in pneumonia, intubation, admission to an intensive care unit, and possibly death. Serious injuries, such as acute respiratory distress syndrome, sepsis, acute kidney and heart failure, and multi-organ failure, can be brought on by COVID-19. Higher mortality risks include being older and having more comorbid conditions [18]. Herein, the exhibited patient had increased liver enzymes that were either thought to be owing to tuberculosis medications or to COVID-19-related hepatitis. According to her blood tests, she had elevated levels of the liver damage markers aspartate aminotransferase (68 units/L) and alanine aminotransferase (95 units/L). As pyrazinamide > isoniazid > rifampicin > ethambutol raise hepatic enzymes, all of her anti-tubercular regimen's adverse effects produce hepatotoxicity, ranging from severe to uncommon hepatotoxicity. Through immune system damage and an increase in hospitalizations or critical care unit stays, COVID-19 infection causes liver damage.

There are several diagnostic tests for COVID-19 that have different sensitivity levels currently available. The recommended approach to make the diagnosis is still reverse transcription—polymerase chain

reaction—for detecting viral ribonucleic acid [19]. Two nasopharyngeal swabs were used for regular reverse transcription polymerase chain reaction COVID-19 testing in the emergency room, and she tested positive on the same day of her admission. At least three consecutive sputum specimens, each taken at intervals of 8 to 24 hours, with at least one being an early morning specimen, are required for the diagnosis of mycobacterial tuberculosis [20]. GeneXpert is a fast diagnostic test that is more sensitive than a sputum smear for the diagnosis of mycobacterial TB [21]. In this instance, a GeneXpert Mycobacterium tuberculosis and rifampicin test on sputum revealed a positive result for Mycobacterium tuberculosis infection without rifampicin resistance.

She started receiving 1000 ml of fluid resuscitation (0.9% normal saline) and insulin delivery by drip as soon as she was admitted to the respiratory isolation room. She continued to breathe in oxygen at a rate of 4 liters per minute using a nasal cannula as prescribed by the SARS-COV-2 treatment regimen. To treat her confirmed COVID-19, she was given subcutaneous enoxaparin 80 mg 12 hours a day.

Conclusion

The COVID-19 epidemic poses a serious hazard to tuberculosis sufferers. Individuals with tuberculosis who develop COVID-19 infection may experience worse results since underlying pulmonary disease is linked to greater mortality in patients with COVID-19 infection.

Declarations

Consent for publication

Written informed consent was obtained from the patient for publication of this case report.

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None

Competing interests

The author has no financial or proprietary interest in any of the materials discussed in this article.

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