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Case Report Open Access

Facing Dual Dilemma: The Intersection of Pulmonary Tuberculosis and Pulmonary Embolism

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Abstract

Tuberculosis (TB) incidence has increased in the United States of America (USA) recently due to an increase in migration post COVID pandemic. Tuberculosis is considered a hyper-coagulable state increasing the incidence of both venous and arterial thrombo-embolism. However, pulmonary embolism has not been studied well in patients with pulmonary tuberculosis. Herein we report the case of a 25-year old immigrant male who presented with a one-month history of fever, myalgias, productive cough and pleuritic chest pain. The patient was diagnosed with pulmonary tuberculosis and started on anti-tubercular therapy. Due to the persistence of hypoxia and worsening of chest pain, computerized tomography pulmonary angiogram was done which showed bilateral acute pulmonary embolism. Patient was started on apixaban with a favorable response and improvement in the clinical symptoms. Due to a high mortality associated with pulmonary embolism, this case shows the importance of early recognition of pulmonary embolism in a patient with pulmonary tuberculosis with worsening symptoms despite treatment.

Keywords: Pulmonary; Tuberculosis; Embolism; *Mycobacterium tuberculosis*

Introduction

Tuberculosis (TB) is a global health problem caused by the bacillus *Mycobacterium tuberculosis*. The number of United States of America tuberculosis cases in 2023 were the highest in a decade, according to a new government report. More than 9,600 cases were reported, a 16% increase from 2022 and the highest since 2013 due to an increase in immigrant population. In 2022, TB was the world's second leading cause of death from a single infectious agent, after coronavirus disease and caused almost twice as many deaths as Human Immuno Virus (HIV) or Acquired Immuno Deficiency Syndrome (AIDS) [1]. The disease typically affects the lungs (pulmonary TB) but, can affect other sites as well. Without treatment, the death rate from TB approaches 50% [2]. Pulmonary Embolism (PE) occurs when there is a disruption to the flow of blood in the pulmonary artery or its branches by a thrombus that originated somewhere else. PE remains a significant cause of in-hospital mortality [3]. TB has been suggested as an independent risk factor for venous and arterial thromboembolism, however, there are a few reports of TB complicated by pulmonary embolism [4-7]. We report a newly diagnosed case of pulmonary tuberculosis who was found to have acute pulmonary embolism due to persistent hypoxia and chest pain.

Case Report

A 25-year old immigrant male with no significant past medical history presented to emergency department with a one-month history of fever, myalgias, productive cough and pleuritic chest pain. On examination, patient was ill-appearing, lethargic, febrile, tachycardic, tachypneic, hypotensive with temperature 103°F, heart rate-132/min, respiratory rate-24/min, blood pressure-90/60 mmHg and saturation 88% on room air. The chest examination revealed rhonchi at right upper chest fields. Laboratory results were as follows; haemoglobin-13.6 g/dl, white blood cell count-8.53 × 10(3)/mcL with 83% neutrophils, platelet count 200 × 10(3)/mcL, albumin-2.9 mg/dl, alanine aminotransferase-79 U/L, aspartate aminotransferase-71 U/L, blood sugar-97 mg/dl, procalcitonin-3.79 ng/ml, C-reactive protein-77 mg/L and normal renal functions HIV and hepatitis panel were negative Computerized Tomography (CT) of chest without contrast showed right upper lobe consolidation with associated air bronchograms 6 x 4.5 cm cavitary thick-walled lesion within the right upper lobe, right perihilar consolidation and bilateral lower lobe patchy nodular consolidation. The patient was admitted as bilateral pneumonia with acute hypoxemic respiratory failure, put on high flow oxygen and started on antibiotics. Quantiferon plus tuberculosis was positive. Acid fast bacilli sputum culture or *Mycobacterium tuberculosis* Complex (MTB) Polymerase Chain Reaction (PCR) were positive for *Mycobacterium tuberculosis*. Infectious disease and pulmonology were consulted. The patient was started on Rifampin, Isoniazid, Pyrazinamide and Ethambuto (RIPE) therapy with Isoniazid 300 mg orally q24h+Rifampin 600 mg orally q24h + Ethambutol 800 mg orally q24h + Pyrazinamide 1000 mg orally q24h + Pyridoxine 50 mg orally q24h and high dose steroids. Patient showed mild improvement in his symptoms, was switched from high flow nasal cannula 40 L/min with 65% FiO2 to 4 L nasal canula, maintaining saturation of 96%.

Results

After four days, the patient developed worsening chest pain and had persistent hypoxia. An urgent Computerized Tomography Pulmonary Angiogram (CTPA) showed acute and subacute pulmonary emboli in the ascending and descending branches of the left pulmonary artery and in the descending branches of the right pulmonary artery. Patient

was started on apixaban twice a day. Rifampicin was changed to rifabutin 300 mg orally q24h due to the interaction of rifampicin with apixaban. Lower extremity bilateral venous duplex showed no evidence of Deep Venous Thrombosis (DVT). Thrombophilia workup was negative except for low Protein S levels 52% (Normal: 67%-141%). The echocardiogram showed normal right and left ventricular function. The patient improved with resolution of his chest pain and hypoxia, with saturation of 97% on room air. The patient was discharged in stable condition on modified RIPE therapy and apixaban with no side effects (Figure 1).

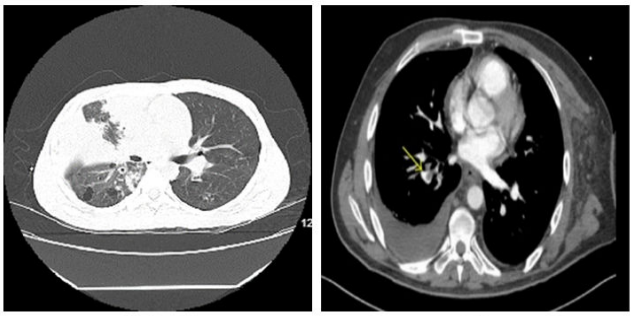

Figure 1: Combination of infectious and thromboembolic pathology in the lungs. **Note:** (A) Computed Tomography (CT) chest without contrast showing right upper lobe consolidation with associated air bronchograms, 6 cm × 4.5 cm cavitary thick-walled lesion within the right upper lobe, right perihilar consolidation; (B) Computerized Tomography Pulmonary Angiogram (CTPA) showed acute pulmonary embolus in the right pulmonary artery.

Discussion

Pulmonary Embolism (PE) leads to pulmonary vascular occlusion and impaired gas exchange and circulation manifesting as dyspnea, chest pain, hypoxemia, shock or sudden death. Prompt recognition of PE is essential because of the high associated mortality and morbidity, which may be prevented with early treatment. A study by Ha et al., showed that 0.6% of tuberculosis patients exhibited PE, deep vein thrombosis or both at or after the time of tuberculosis diagnosis [7]. The factors responsible for thromboembolism in tuberculosis are hypercoagulability, endothelial damage and venous compression due to lymph node enlargement. Hypercoagulability may be due to elevated blood fibrinogen, reduced thrombin III, protein C binding to thrombin and platelet aggregation [4,7,8]. Rifampicin also promotes hypercoagulability and patients taking rifampicin have a 4.5 times higher risk of deep venous thrombosis than those without rifampicin [9]. This is the reason that rifampicin was changed to rifabutin in our case.

Apart from PE, tuberculosis patients are at higher risk of thromboembolism in hepatic veins, cerebral veins and arterial system [10-12]. The risk of PE in tuberculosis is higher in young and middle-aged patients and the occurrence of thrombosis depends on the severity of tuberculosis. Most of the previous cases of PE in tuberculosis were seen in patients with severe pulmonary tuberculosis [13]. In a study by Dentan et al., patients with both active Tuberculosis (TB) and Venous Thrombo-Embolism (VTE) showed higher mortality than patients with only active TB ($P<0.001$) [14]. The diagnosis of PE in patients with pulmonary tuberculosis might get delayed because of overlapping signs and symptoms. In such cases, a high index of suspicion is required especially in young patients with no other risk factors for PE as in our case. The treatment of PE in tuberculosis remains the same as in the general population except for the consideration of drug interactions between rifampicin and anticoagulants. However, the treatment duration is not well defined. In a study by Kouismi et al., treatment with low molecular weight heparin and warfarin was given for 3 months in 25 cases and in three cases, treatment was extended further for 3 months [15].

Conclusion

Patients might need a prolonged duration of treatment with careful monitoring of side-effects and drug interactions. This is one area which needs further investigation. Acute Pulmonary embolism is a life-threatening disease. Due to the high risk of thromboembolism, patients with pulmonary tuberculosis having persistent hypoxia, worsening chest pain and tachycardia should be evaluated for PE. Because of the recent surge in the immigrant population in the United States of America and an increase in the diagnosis of tuberculosis, the clinicians must be aware of this potentially life-threatening and treatable complication.

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