Case Report

Bilateral Pulmonary Embolism Presenting as Lung Abscess in a Tuberculosis Patient: A Rare Presentation

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Abstract

Pulmonary embolism is a life-threatening medical emergency associated with high mortality and morbidity which is likely to be missed if a high index of suspicion is not maintained. Classical triad of chest pain, dyspnea, and hemoptysis is seen only in a minority of cases. Both inherited and acquired conditions are involved in its etiology. A case of bilateral pulmonary embolism presenting as lung abscess in a 65-year-old female with active tuberculosis is being discussed here.

Keywords: Acquired, inherited, life-threatening, triad

Introduction

Tuberculosis (TB) is a major public health problem worldwide since time immemorial, and India contributes about a quarter of the cases. Pulmonary embolism is common in a patient with prolonged immobility, congestive heart failure, recent surgery, systemic hypertension, atherosclerosis, old age, malignancy, nephrotic syndrome, and use of oral contraceptives by females. Rarely, TB can also predispose to it. Present or past episodes of deep-vein thrombosis (DVT) play a very important role in the development of pulmonary embolism. Lung abscess usually occurs secondary to aspiration and periodontal disease with anaerobic and Gram-negative organisms mainly involved in its etiology. Rarely can it be the presentation of pulmonary embolism.

Case Report

A 65-year-old female presented to the outpatient department with a history of fever, breathlessness, central chest pain, and swelling in bilateral lower limbs of 1-month duration, confining her to bed. Her husband had also taken one episode of treatment for pulmonary TB. No history of diabetes, hypertension, dyslipidemia, malignancy, or coronary heart disease was present. She had undergone one episode of anti-TB treatment for microbiologically confirmed pulmonary TB for 6 months from revised national tuberculosis control program (RNTCP) 2 years back and was declared cured. She had no history of any surgery in the recent past and had attained her menopause 20 years back. On examination, the patient was cyanosed and bilateral pitting pedal edema was present along with engorgement of neck veins. Tachycardia was present (PR – 118 bpm) along with tachypnea (RR – 26/min). Blood pressure was normal (104/72) and oxygen saturation was low (SpO_2 – 58% without oxygen). Amphoric breath sounds were present in the right infraclavicular area with a dull note on percussion along with rib crowding and other features of volume loss. Chest X-ray showed a homogeneous opacity on the right side with a horizontal fluid level and fibrotic changes in the left upper lobe with infiltrates in the left lower lobe [Figure 1], and there was marked progression from her previous chest X-rays which warranted for a contrast-enhanced computed tomography (CT) of the thorax. Routine blood investigations were done. Complete blood count, renal function tests, random blood sugar, serum uric acid, fasting lipid profile, and serum electrolytes were within normal limits. Serum albumin level was decreased (2.4 g/dl) and the total leukocyte

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count was 8800. Screening for HIV and hepatitis B surface antigen was negative. Sputum acid-fast bacillus staining and pyogenic culture were negative. Sputum was sent for cartridge based nucleic acid amplification test (CBNAAT), and *Mycobacterium tuberculosis* was detected which was sensitive to rifampicin. Electrocardiogram revealed sinus tachycardia with right ventricular hypertrophy and P pulmonale [Figure 2]. CT of the thorax revealed acute pulmonary embolism in both the pulmonary arteries (right > left) with pulmonary hypertension along with dilated right and left ventricles and right lung abscess with significant volume loss in the right side and fibrocalcific opacities with traction bronchiectasis and collapse of the left upper lobe and tree in bud opacities in the left lower lobe suggestive of active infection [Figure 3]. Two-dimensional echocardiography, protein C, protein S, antithrombin III, and antiphospholipid antibodies could not be analyzed due to the economic constraints of the patient and limitation of investigations in our laboratory. Serum C-reactive protein (CRP) was negative and Doppler study of bilateral lower limbs was within normal limits.

O_2 therapy was initiated following which her saturation improved. The patient was started on anti-TB chemotherapy and low-molecular-weight heparin followed by warfarin. There was no indication for thrombolysis. Antibiotics with Gram-negative and anaerobic coverage were started. Bronchodilators, antipyretics, and diuretics were given for symptomatic relief.

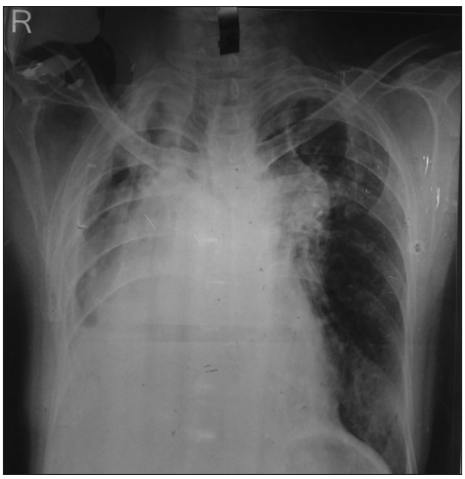

Figure 1: Chest X-ray posteroanterior view showing bilateral lung lesion

Discussion

Association of pulmonary embolism with TB is a rare occurrence, and only a few cases are reported in the literature. DVT confirmed by venography has been observed in 3%–4%of patients with pulmonary TB,[1] and one-third of these manifests as pulmonary embolism. Death occurs in 12% of pulmonary embolism cases within 1 month of diagnosis.[2] Factors responsible for the development of thromboembolism are still unclear. All the three elements of Virchow's triad, namely hypercoagulability, venous stasis, and endothelial dysfunction, seem to be operational in the development of pulmonary embolism. Infections such as TB can rarely lead to thromboembolism by enhancing platelet aggregation; increasing fibrinogen, fibrin degradation products, plasminogen activator inhibitor and through associated deficiencies of protein C, protein S and antithrombin III.[3,4] Rifampicin is also implicated in the increased incidence of DVT in a study.[5] Pro-inflammatory cytokines released during the disease can make the endothelium more thrombogenic and also enhance synthesis of clotting factors by the liver.[6,7] Enlarged lymph nodes compressing the veins can also lead to thrombosis.[8] High frequency of antiphospholipid antibodies detected in patients with TB has also been mentioned in the literature. CRP has been implicated in platelet aggregation associated with TB. Pulmonary infarction is seen in only 10 percent of the cases of pulmonary embolism due to the dual nature of blood supply.[9] Aseptic liquefaction and cavitation occur in infarcts >4 cm in size.[10] Cavitation occurs in about 4%–7% cases of pulmonary infarction,[11] in which superinfection with anaerobic and Gram-negative organisms can occur.

Antituberculous chemotherapy (ATT) and anticoagulant therapy should be started at the earliest, and coagulation abnormalities associated with TB will be corrected within about 12 weeks. Rifampicin may interfere with the action of oral anticoagulants such as warfarin by inducing cytochrome P450, thereby reducing its efficacy close to 50%.

Conclusion

A high index of suspicion should be maintained for diagnosis of pulmonary embolism at the earliest, as it is associated with a very high mortality rate. Anticoagulant therapy should be instituted, and thrombolysis should be considered in patients with hemodynamic instability. Surgical embolectomy is another feasible option in well-equipped centers.

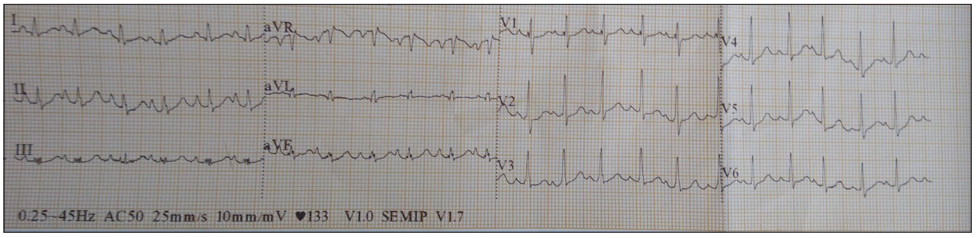

Figure 2: Electrocardiogram showing sinus tachycardia with right ventricular hypertrophy and P pulmonale

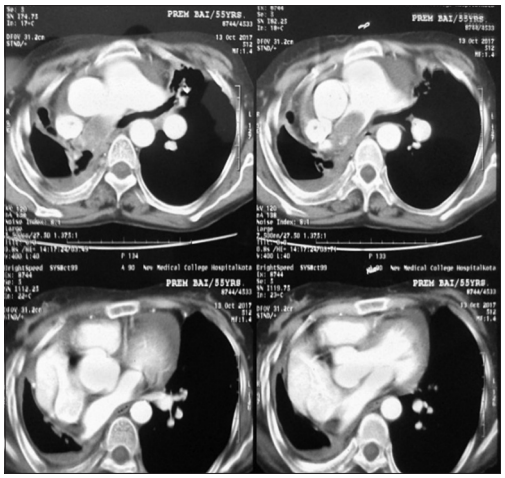

Figure 3: Contrast-enhanced computed tomography of the thorax demonstrating filling defect and cavitation with air-fluid level

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Conflicts of interest

There are no conflicts of interest.

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