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Case Report

A Case Report on Pulmonary Tuberculosis Associated with Deep Vein Thrombosis

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Abstract

Public health problems such as tuberculosis remain a major concern, especially in developing countries. According to WHO (World Health Organization), for every second someone gets infected with tuberculosis. India is the second most populated nation in the world contributes to 23% of TB incident cases annually out of 9.6 million worldwide. The overall morbidity and mortality associated with TB are reduced with early initiation of anti-TB treatment. In India, most antituberculosis regimens are Rifampicin-based under the Revised National Tuberculosis Control Program (RNTCP). Despite its rarity, deep venous thrombosis (DVT) should be considered especially in those with severe disseminated or pulmonary tuberculosis. This case report describes a 31-year-old female with pulmonary tuberculosis initiated on rifampicin-based antituberculosis treatment presented with deep vein thrombosis.

Keywords: Tuberculosis, Deep vein thrombosis, pulmonary tuberculosis

INTRODUCTION

Deep vein thrombosis (DVT) is a common preventable and treatable cause of death worldwide. India reports about 9 million cases per year out of which 1.3-5% are associated with deep vein thrombosis (DVT).Tuberculosis(TB) is one of the most devastating curable communicable diseases in our country. It is estimated that 7 to 8 million newly diagnosed cases of TB are reported worldwide each year [1]. Pulmonary tuberculosis is the most common form of TB. The standard treatment for pulmonary TB comprises 2 months of quadruple therapy with isoniazid (INH), Rifampicin (RMP), ethambutol (EMB), and pyrazinamide (PZA) followed directly by a further 4 months' dual administration of Rifampicin (RMP) and isoniazid (INH) [2]. Rifampicin is an RNA polymerase inhibitor antibiotic, and it's still one of the most powerful antibiotics since its discoverd in the 20th century. It used to be reserved for serious bacterial infections like active and latent tuberculosis (TB) [3]. The presence of a hypercoagulable state among TB patients was postulated as a consequence of the elevated plasma fibrinogen with reactive thrombocytosis, the direct endothelial damage promote by the tubercular bacillus and therefore the use of Rifampicin [4]. Deep vein thrombosis (DVT) is a chronic disease that occurs due to Venus flow obstruction caused by retroperitoneal lymphoma and malignant masses [5]. The exact mechanism of Rifampicin causing DVT is unknown. Rifampicin, make contribute to the hypercoagulable state by decreasing production and increasing clearance of anticoagulant hepatic proteins. Rifampicin can also cause endothelial injury which favors' thrombosis. FDA research report state that the percentage of rifampicin taking patient, where DVT is reported as a side effect is 0.5385% [6]. Here we report a case of a young adult female with pulmonary tuberculosis initiated on rifampicin-based ATT presented with DVT.

CASE PRESENTATION:

A 31-year-old female patient came to the hospital with chief complaints of shortness of breath and fever, weight loss, generalized body pain, and chest pain for one month. Absence of abdominal pain, palpitations.

On examination, the patient was conscious and coherent. The pulse rate was 89 beats per minute. On the chest, examination left-sided basal crept sound is present. Other examinations are unremarkable.Tests revealed elevated urea levels 35 mg/dl (normal range: 6-24 mg/dl), and elevated WBC levels 17300/cumm (normal range: 4000-11000/cumm). Both liver and kidney functions were normal. The montoux test and acid-fast bacillus (AfB) were positive in sputum.

Left lower zone opacity is visible on x-rays of the chest shown in **fig.1**. Hrct demonstrates large nonhomogenous opacities with thin walls, thickened bronchi in the posterior lower lobe, Some enlarged lymph nodes in the pre-tracheal region, and also the presence of tuberculosis shown in **fig.2**.The physician identified pulmonary TB based on laboratory and non-laboratory data. In the hospital, the patient received the following medication

1. Inj pan (pantoprazole 40mg) OD
2. Tab.ATT (Ethambutol-600mg, pyrazinamide- 750mg, Isoniazid- 300mg, Rifampicin-450 mg) - OD
3. Tab. Chymoral forte (Typsin- chymotrypsin1000000 AU) TID
4. Inj. Tramadol (Tramadol Hydrochloride 50 mg) BD
5. Inj. Zofer (Ondansetron 4mg) BD
6. Tab. Benadon (Pyridoxine hydrochloride 20 mg) BD

Ten days later, the patient was discharged from the hospital and advised to continue ATT therapy.

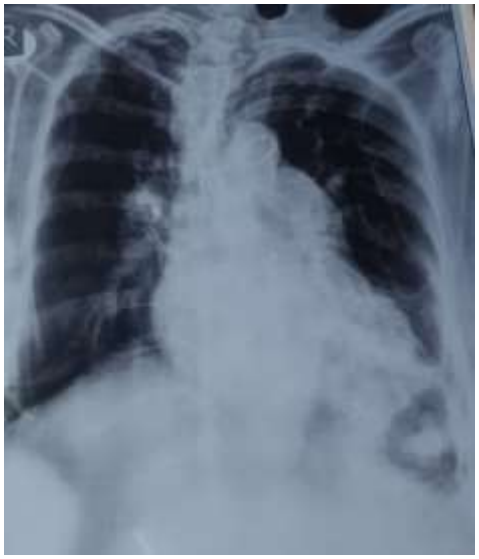

Fig.1

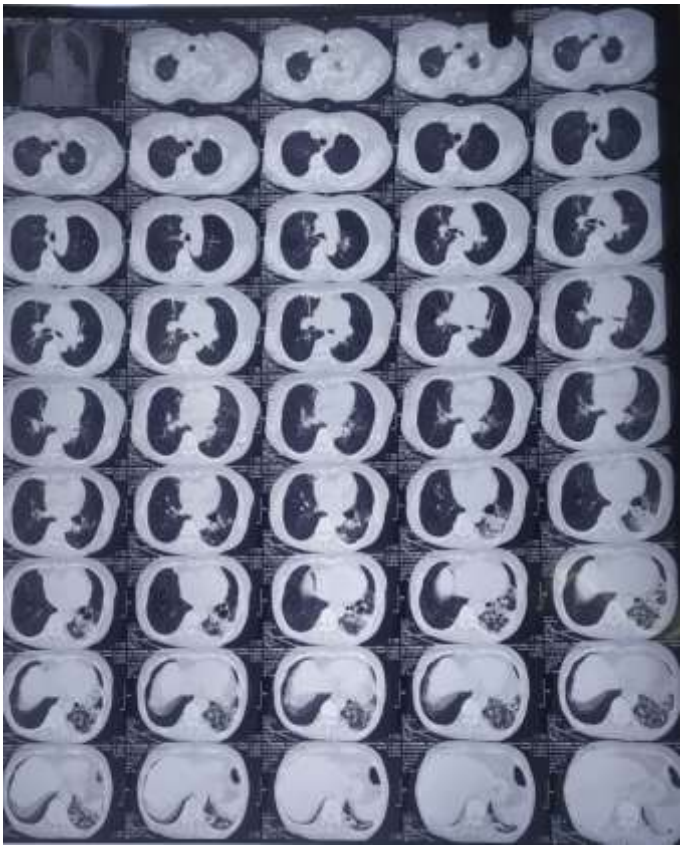

Fig.2

After 10 days of painful swelling of the left lower limb, the patient was brought back to the hospital (**Fig.3**). On physical examination no evidence of cellulitis is present. In a color Doppler USG of the left lower limb, we found that the common femoral, superficial femoral, popliteal, anterior, and posterior tibial veins visualized were noncompressible, distended, and filled with heterogeneous hypoechoic thrombosis without flow. Based on the color Doppler of the left lower limb physician confirmed DVT and it was caused by antitubercular medication. The patient was started on low molecular weight heparin (LMWH) 40mg subcutaneous injection. Five days after treatment, swelling and pain began to decrease. A continuous intravenous (IV) heparin was administered to the patient up until 48 hours before managing the patient, after which the patient was switched to oral apixaban twice daily at 10 mg for seven days and then reduced to 5 mg on the same schedule for the remainder of the six-month anticoagulant therapy. After few days the patient's condition had stabilized and patient was discharged from the hospital and advised to come back for a follow-up visit in the hospital.

Fig.3

DISCUSSION

Most patients who manifest symptomatic VTE have a pulmonary embolism (PE) while two-thirds have DVT alone. A 31-year-old female patient presented with pulmonary tuberculosis. Following guidelines from the Revised National TB Control Programme (RNTCP), treatment was administered. Six months of ATT therapy was started. After one month of ATT therapy, the patient developed deep vein thrombosis (DVT).

Virchow's triad,including hypercoagulability, venous stasis, and endothelial dysfunction, might all contribute to VTE in TB. A high plasma fibrinogen and factor VIII level, as well as reactive thrombocytosis, may cause hypercoagulability. Furthermore, these changes improved within 4 weeks of ATT [7]. After one month of ATT therapy, then patient developed DVT.

Rifampicin has also been associated with DVT, with a relative risk of 4.74 in patients treated with rifampicin-containing regimens [8]. So our patients also develop DVT after using rifampicin based antituberculosis therapy.

ATT should be immediately started supplemented with anticoagulant therapy as hemostatic changes improve during the 1st month of treatment [9]. Our patient also received low molecular weight heparin and apixaban for deep vein thrombosis treatment. Our patient was discharged from the hospital after receiving treatment. The Patient was advised to take anticoagulant therapy for six months during ATT therapy.

CONCLUSION

Pulmonary tuberculosis causes deep vein thrombosis rarely. Special attention is paid to tuberculosis patients receiving antituberculosis therapy. To improve outcomes, early diagnosis and treatment are essential. Prompt diagnosis and treatment can prevent increased mortality. It is important to initiate antituberculosis treatment along with anticoagulant therapy throughout the initial stages of the disease to avoid the potentially fatal complications that may occur. A physician should pay special attention to start of an antitubercular medication containing rifampicin to ensure a successful outcome.

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