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Case Report

Deep Vein Thrombosis, Pulmonary Thromboembolism, and Pulmonary Infarction: All in one in a Young Male Patient with Pulmonary Tuberculosis

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Abstract

Pulmonary tuberculosis (PTB) is a known pro-inflammatory state that can predispose individuals to thromboembolic events. We present an uncommon case of a young male with active PTB who developed deep vein thrombosis (DVT), pulmonary thromboembolism (PTE), and pulmonary infarction as complications during the disease course. This case underscores the critical need for prompt identification of thromboembolic events in patients with tuberculosis, the diagnostic value of Doppler and CT pulmonary angiography, and the successful use of anticoagulation alongside modified ATT in achieving favourable outcomes.

Keywords: Pulmonary tuberculosis, deep vein thrombosis, pulmonary thromboembolism, pulmonary infarction, hypercoagulability, anticoagulation, CT pulmonary angiography, Doppler ultrasound, ATT modifications, venous thromboembolism.

Introduction

Pulmonary tuberculosis (PTB) continues to pose a significant public health challenge worldwide, with a disproportionately high impact in low- and middle-income nations. Beyond its classical pulmonary manifestations, PTB has systemic effects, including a propensity to induce a hypercoagulable state[1]. Venous thromboembolism (VTE), though uncommon in tuberculosis, can manifest as deep vein thrombosis (DVT)[2] and pulmonary thromboembolism (PTE)[3], leading to serious complications such as pulmonary infarction. The association between PTB and VTE is often underrecognized due to overlapping clinical features and low index of suspicion. In this case, we describe a young male with active PTB who developed DVT, PTE, and pulmonary infarction, underscoring the importance of early diagnosis and comprehensive management in such presentations[4].

Case Summary

A male of age 25 years with no prior comorbidities and no smoking history or substance abuse presented with acute onset swelling of the left lower limb for three days, along with one-month history of fever, cough, and progressive breathlessness. He had been empirically initiated on anti-tuberculosis therapy (ATT) one month prior based on clinical symptoms, and a family history of pulmonary tuberculosis in his sister which was diagnosed microbiologically.

Clinical examination revealed an edematous, tender, shiny left leg with a positive Homan's sign (Figure 1) and a calf circumference of 30 cm on the left compared to 24 cm on the right, raising suspicion of deep vein thrombosis (DVT). Respiratory examination demonstrated reduced right-sided chest movement, hyper-resonant percussion, and decreased intensity of breath sound in the right interscapular and infrascapular areas. Chest radiography confirmed right-sided pneumothorax along with bilateral inhomogeneous infiltrates (Figure 2).

An intercostal drainage tube (ICDT) was placed, and he was initiated on low molecular weight heparin (LMWH) at a dose of 0.6 mL subcutaneously once daily. Initial laboratory investigations revealed anemia, leukocytosis, and deranged liver function (SGOT/SGPT 110/175 IU/L), prompting initiation of a modified anti-tuberculosis therapy (ATT)

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Figure 1: Patient's left lower limb before treatment

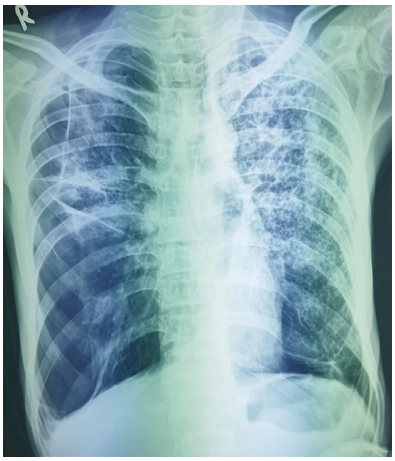

Figure 2: Posteroanterior (PA) chest radiograph demonstrating bilateral inhomogeneous pulmonary infiltrates along with a right-sided pneumothorax

regimen comprising ethambutol 800 mg, and levofloxacin 750 mg once daily, along with broad-spectrum antibiotics and injectable iron sucrose.

A markedly elevated D-dimer level (6.0µg/ml FEU) warranted intensification of anticoagulation with LMWH 0.6 mL administered subcutaneously twice daily, along with oral dabigatran 110 mg twice a day. Doppler ultrasonography of the lower limb confirmed extensive thrombi in the left common femoral, superficial femoral, and popliteal veins (Figure 3). Sputum CBNAAT confirmed the presence of Mycobacterium tuberculosis, sensitive to rifampicin. Contrast enhanced CT chest followed by CT pulmonary angiography demonstrated a partial thrombotic occlusion of the right lower lobe segmental pulmonary artery, consistent with pulmonary thromboembolism (PTE) (Figure 4A), also revealed a peripheral, wedge-shaped opacity in the right lower lobe, consistent with pulmonary infarction (Figure 4B).

The ICDT was removed after twelve days of insertion. The patient's liver function gradually normalized, full-dose

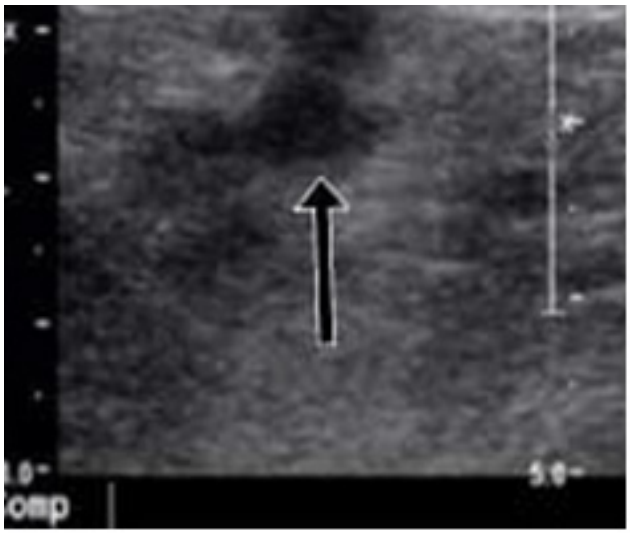

Figure 3: Doppler ultrasound of left lower limb showing deep venous thrombosis (↑)

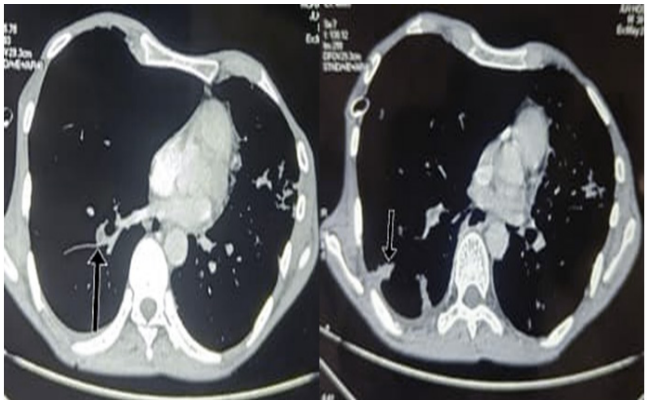

Figure 4: (A and B) Contrast enhanced CT Chest showing Pulmonary thromboembolism involving the segmental branch of the right lower lobe pulmonary artery (↑), and wedge-shaped pulmonary infarction in right lower lobe (↓)

ATT was re-introduced sequentially, and clinical recovery ensued with resolution of leg swelling and normalization of total leukocyte count. He was discharged in a stable condition on oral anticoagulation and continuation of full ATT.

Discussion

Active PTB may be complicated by DVT, reflecting an underlying hypercoagulable state induced by chronic systemic inflammation. Inflammatory cytokines, endothelial dysfunction, and immobilization during illness all contribute to this prothrombotic milieu. The association between PTB and vascular complications, including venous thromboembolism (VTE), has been observed in approximately 1.5%–3.4% of cases, though it is likely underrecognized due to overlapping symptomatology and low clinical suspicion[4,5].

DVT is typically considered in the context of surgery, malignancy, or prolonged immobilization. However, it may

also present at the time of PTB diagnosis, during treatment, or later in the disease course, as seen in our patient[2]. This underlines the importance of including tuberculosis in the differential diagnosis of unprovoked DVT, particularly in endemic regions. Despite this, the literature is sparse, comprising primarily of isolated case reports and small observational studies[5]. Increased awareness, coupled with the widespread availability of non-invasive diagnostic tools like colour Doppler ultrasonography, can enhance early detection and reduce morbidity and mortality from otherwise preventable thrombotic events.

Peripheral oedema in TB patients is often dismissed as a consequence of hypoalbuminemia, but signs such as pain, localized warmth, and asymmetrical limb swelling should prompt evaluation for DVT. In such patients, clinical vigilance is key, and delays in diagnosis may prove fatal. The pathophysiology of DVT in TB appears multifactorial. All elements of Virchow's triad i.e. hypercoagulability, venous stasis, and endothelial injury may concurrently contribute to the thrombotic risk in tuberculosis.

Several prothrombotic alterations are documented in PTB: elevated plasma fibrinogen levels, reduced fibrinolytic activity, and decreased concentrations of natural anticoagulants such as antithrombin III and protein C, and reactive thrombocytosis by, all support a hypercoagulable state[1,2,4]. Furthermore, certain patients exhibit elevated levels of antiphospholipid antibodies, potentially interacting with protein S deficiency to exacerbate thrombotic risk. Notably, hypoprothrombinemia rather than hypercoagulability has been observed in up to one-third of TB cases, contributing to coagulation abnormalities and highlighting the complex hemostatic alterations associated with the disease[4,6,7].

Cytokine-mediated endothelial activation renders vascular intima thrombogenic, while hepatic stimulation may alter synthesis of clotting factors[8]. Prolonged bed rest and disease-related immobility further potentiate thrombosis risk. Additionally, venous compression by tubercular lymphadenopathy can mechanically promote venous stasis[9].

Drug-induced thrombophilia also warrants consideration. Rifampicin, a key component of anti-tuberculosis therapy, has been implicated in increasing the risk of deep vein thrombosis, with some studies reporting a relative risk as high as 4.74.[7] Rifampicin induces hepatic cytochrome P450 enzymes, leading to enhanced metabolism of warfarin and thereby complicating anticoagulation management with vitamin K antagonists. Consequently, while early initiation of anticoagulant therapy is essential in tuberculosis patients with thrombotic events, it must be approached with careful monitoring and individualized dosing strategies. Direct oral anticoagulants (DOACs) such as factor Xa inhibitors offer practical advantages, including fewer drug interactions, rapid onset, and a more favourable bleeding profile compared to traditional warfarin-based regimens.

In summary, this case reinforces the need for heightened clinical vigilance for DVT and VTE in PTB patients, especially those with acute limb swelling or respiratory deterioration. Prompt diagnosis using Doppler ultrasonography and CT pulmonary angiography, coupled with initiation of anticoagulation and tailored ATT, ensures optimal outcomes. As evidence accumulates, integration of thrombotic risk assessment into TB management algorithms may soon become a standard of care.

Conclusion

This case illustrates a rare but clinically significant association of DVT, PTE, and pulmonary infarction in the setting of active pulmonary tuberculosis, underscoring the importance of early recognition and comprehensive identification and treatment of thromboembolic events in tuberculosis patients. The occurrence of deep vein thrombosis, pulmonary thromboembolism, and pulmonary infarction, though rare, can be life-threatening and often go unrecognized due to overlapping symptoms. Prompt diagnosis using Doppler ultrasonography and CT pulmonary angiography, along with timely initiation of anticoagulation and modified ATT, was instrumental in achieving a favorable outcome in this patient. Physicians should remain vigilant for the possibility of venous thromboembolism in individuals with tuberculosis who exhibit new-onset limb edema or progressive respiratory deterioration.

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