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Case Report

Extensive Deep Vein Thrombosis: A Rare Manifestation of Disseminated Tuberculosis

Shekhar Swaroop[1], *Paras Singla*[2], *Upendra Baitha*[3*], *Gaurav Gupta*[3], *Amandeep Singh*[3]
1- Department of Gastroenterology, All India Institute of Medical Sciences, New Delhi, India
2- Department of Pediatrics, All India Institute of Medical Sciences, New Delhi, India
3- Department of Medicine, All India Institute of Medical Sciences, New Delhi, India

*** Corresponding author:** Dr. Upendra Baitha, Associate Professor, Department of Medicine, *All India Institute of Medical Sciences*, New Delhi, 110029, India.
Email: drupendrabaitha@aiims.edu

ABSTRACT

A 20-year-old male with disseminated tuberculosis (pulmonary, pleural, and lymph node TB) presented with unusual manifestation in the form of extensive deep vein thrombosis (DVT) involving bilateral popliteal veins, femoral veins, iliac veins, and infra-hepatic inferior vena cava (IVC). Upon follow-up with the patient after two months of therapy, there was a significant clinical improvement, and edema and tenderness of the lower limb had decreased.

KEYWORDS: tuberculosis, diagnosis, deep vein thrombosis

INTRODUCTION

It is well known that a chronic inflammatory state predisposes to deep vein thrombosis (DVT), but the literature on tuberculosis predisposing to DVT is scarce. Only a few case reports have reported the association between the two conditions. Tuberculosis is such a common infection in developing countries that clinicians should be aware that DVT can be one of the unusual manifestations of tuberculosis.

CASE REPORT

A 20-year-old male with no comorbidities or addictions presented with a history of dyspnoea, undocumented on and off low-grade fever, dry cough, significant weight loss and heaviness over the left side of the chest for the past five months, and painful swelling of bilateral lower limbs extending till thigh and visible veins over the abdomen for the past four months (Figure 1). There was no history of wheezing, bluish discoloration, palpitation, orthopnoea, or paroxysmal nocturnal dyspnoea.

On examination, the patient had tachypnoea and tachycardia. A general physical examination revealed bilateral enlarged posterior cervical lymph nodes, which were matted, tender, and firm in consistency without any redness or the local temperature rise over the lymph node, swelling, or discharging sinus or fistula. Examination of the lower limb revealed bilateral swollen and tender lower extremities extending to the thigh. Upon examination of the respiratory system, the trachea was shifted to the right side, with decreased expansion on the left side. On percussion, there was dullness over the left lung till the 4th intercostal space posteriorly and 5th intercostal space anteriorly. On auscultation, there was decreased air entry on the left side of the chest. Abdominal examination revealed dilated and engorged veins with a direction of blood flow from below upward, suggesting inferior vena cava obstruction.

On presentation, a chest X-ray was done, which revealed bilateral pleural effusion with massive left-sided pleural effusion. An intercostal drainage tube

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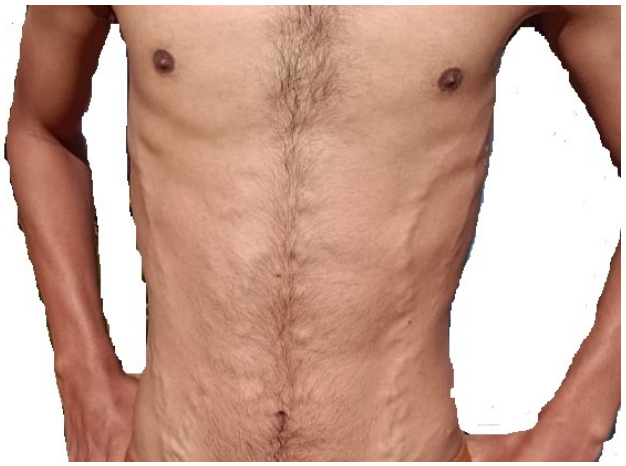
Figure 1: Collateral veins visible over patient's abdomen with extensive Deep Vein Thrombosis with disseminated Tuberculosis



was inserted on the left side, and around 3 liters of pleural fluid was drained over five days. Pleural fluid analysis was suggestive of exudative effusion. Adenosine deaminase (ADA) and lactate dehydrogenase (LDH) in the pleural fluid were raised. Cytology of the pleural fluid did not reveal any malignant cells. Routine blood investigations revealed an elevated erythrocyte sedimentation rate (ESR). Contrast-enhanced computed tomography (CECT) of the chest and abdomen revealed multiple centrilobular nodules in tree-in-bud patterns in bilateral lung fields with multiple enlarged conglomerated necrotic supraclavicular, mediastinal, and periportal lymph nodes with bilateral empyema. Ultrasound screening was done for lower limb swelling, which revealed thrombosis of the bilateral femoral and popliteal vein, which was followed by a CT angiogram of the lower limb, which showed thrombotic occlusion of bilateral popliteal veins, femoral veins, iliac veins, and infra-hepatic IVC with collateral formation. Lymph node excisional biopsy revealed large areas of geographic necrosis with palisaded foamy histiocytes and few epithelioid cell granulomas. GeneXpert for MTB was positive and sensitive to rifampicin. We also considered other causes and risk factors for DVT, both acquired and inherited, and ruled them out with appropriate history, physical examination, and investigations.

The patient was started on anti-tubercular treatment (ATT) for disseminated tuberculosis and on low molecular weight heparin (LMWH) overlapped with warfarin for DVT and was monitored with international normalized ratio (INR) and the dose titrated accordingly. After starting ATT, the patient resolved symptoms and was discharged on the 15th day of ATT. LMWH and warfarin were stopped after eight days. On follow-up after two months of therapy, there was a decrease in lower limb swelling and tenderness, the fever had resolved, and the pleural effusion resolved.

DISCUSSION

Tuberculosis is one of the most common infections encountered in developing countries, and so is the case in India. DVT can have so many causes, including both inherited and acquired causes and inflammatory diseases are one of them. Inflammatory diseases and infections have been known for a long to cause DVT.[1] Various mechanisms have been proposed for inflammation-induced thrombosis. Inflammation increases procoagulant factors and inhibits natural anticoagulant pathways and fibrinolytic activity, causing a thrombotic tendency. Chronic inflammation may cause endothelial damage, resulting in the loss of physiologic anticoagulant, antiaggregant, and vasodilatory properties of endothelium. On the other hand, coagulation also augments inflammation, mainly using thrombin-induced secretion of proinflammatory cytokines and growth factors. Platelets may also trigger inflammation by activating the dendritic cells.[2,3]

However, the association of tuberculosis with DVT has begun to be reported in the last few years with only case series and case reports on the same.[4-9] The mechanisms responsible for the development of DVT in patients with tuberculosis are not fully understood, and all three parts of Virchow's triad, including hypercoagulability, endothelial dysfunction, and stasis, may predispose to DVT in TB.[6]

Treatment of this condition requires special mention of the interaction of warfarin with rifampicin. CYP2C9 metabolizes warfarin, and rifampicin is an enzyme inducer. Co-administration of both warfarin and rifampicin can make it very difficult to reach the target INR with warfarin, and higher doses of warfarin may be required. Also, evidence is there to show that rifampicin itself can predispose to thrombosis risk.[10] So, this aspect of treatment and whether we need to modify treatment for the same need to be studied. Our patient did not show any progress in thrombosis post-ATT. Also, INR was measured frequently, and with routine follow-up, warfarin was continued for three months without any complications.

CONCLUSION

Such an extensive thrombosis in a patient with disseminated tuberculosis without any retroperitoneal lymph node involvement and IVC obstruction has never

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been reported. Therefore, clinicians need to be aware that TB can predispose to extensive DVT and can be life-threatening because of the high risk of pulmonary embolism.

CONFLICTS OF INTEREST STATEMENT

The authors declare no conflict of interest.

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None

AUTHORS' CONTRIBUTIONS

SS: writing the draft

PS: review

UB: supervision

GG: editing

AS: conceptualization

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