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Case Report

Uncommon association of tuberculosis with deep vein thrombosis in a child: a case report

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ABSTRACT

Tuberculosis (TB) is a major global health issue, particularly in developing countries. While TB commonly affects the lungs, it can spread to nearly any organ system, leading to a range of complications. One rare extrapulmonary manifestation of TB is deep vein thrombosis (DVT), which is even more rarely reported in children. We report a case of a 13-year-old girl with progressive, painful swelling in right lower limb, accompanied by fever, cough and lymphadenopathy. Diagnostic imaging identified DVT in the right external iliac vein. Further investigation confirmed *Mycobacterium tuberculosis* infection. She was treated with anti-tubercular medication and therapeutic anticoagulation. Although uncommon, this case underscores the importance of recognizing this serious complication and need for timely diagnosis and treatment.

Keywords: Disseminated tuberculosis, Deep vein thrombosis, Lymphadenopathy

INTRODUCTION

Deep vein thrombosis (DVT), a type of venous thromboembolism (VTE), is a significant preventable cause of illness and death globally. Risk factors for VTE include age, major surgery, prolonged immobility, oral contraceptive use, cancer, central venous lines, smoking, and genetic predispositions to hypercoagulability.[1] Tuberculosis (TB), with its potential to increase thrombosis risk, is an often-neglected factor in DVT development.[2] Literature suggests a rare but serious connection between TB and DVT, with prevalence rates of approximately 1.5-3.4% as indicated by several studies.[3,4]

CASE REPORT

In 2024, a 13-year-old female presented to the pediatrics department at Lala Lajpat Rai Memorial Medical College, Meerut, with a 15-day history of progressive swelling in her right lower limb. The swelling began in the foot and ascended to involve the entire leg up to the groin.

There was no history of trauma or prolonged immobilization. The patient did not report symptoms of shortness of breath, chest pain, jaundice, reduced urine output, foamy urine, or joint pain. She had experienced intermittent low-grade fever, a non-productive cough, and diffuse, moderate abdominal pain for the past three months. Additionally, she reported a reduced appetite and a documented weight loss of 8 kg over the last three months. There was no known contact with tuberculosis (TB), and she had received the Bacillus Calmette-Guérin (BCG) vaccine at birth.

At presentation, the patient appeared pale and emaciated, with a weight of 24 kg and a height of 124 cm, both below the 3rd percentile for her age. The right lower limb was swollen, tender, and had a limited range of motion, with pitting edema. There was no redness or discharge. Peripheral pulses were intact. Physical examination revealed significant cervical lymphadenopathy, with matted nodes measuring 4 cm on the left side. Chest examination showed signs of consolidation, while the

abdomen was non-tender with a liver span of 10 cm. There was shifting dullness and prominent veins in the lower chest and abdomen.

Blood tests revealed a hemoglobin level of 7 g/dl, a leukocyte count of 5,500/mm³ with a differential showing 70% polymorphonuclear cells and 26% lymphocytes, and a platelet count of 140,000/mm³. The blood smear indicated a normocytic normochromic picture with some microcytic hypochromic red blood cells. The erythrocyte sedimentation rate was 31 mm/hour. Liver and renal function tests were normal, and human immune-deficiency virus (HIV) serology was negative. Serum protein levels were 4.1 g/dl. Prothrombin time was 18.1 seconds with an international normalized ratio (INR) of 1.42.

Chest X-ray displayed multiple bilateral patchy opacities and small nodular opacities. Ultrasound of the chest and abdomen showed consolidation in the right upper lobe, bilateral moderate pleural effusion, and lymphadenopathy in the cervical, left axillary, and left supraclavicular regions. There was also hepatomegaly, moderate ascites, and multiple enlarged lymph nodes in the mesenteric, retroperitoneal, para-aortic, bilateral iliac, and inguinal regions, some with necrosis, conglomeration, and calcification. Notably, a lymph node was compressing the inferior vena cava, common iliac, and right external iliac veins. Doppler ultrasound confirmed partial thrombosis of the right external iliac vein. A 2D echocardiogram revealed pericardial effusion. Contrast-enhanced abdominal computed tomography (CT) scan showed a filling defect in the right external iliac vein, consistent with thrombosis, and confirmed the ultrasound findings. Additionally, the scan revealed thickening of the bowel walls in the ascending and transverse colon, mild mucosal fold thickening, and interbowel fluid, suggestive of tuberculosis.

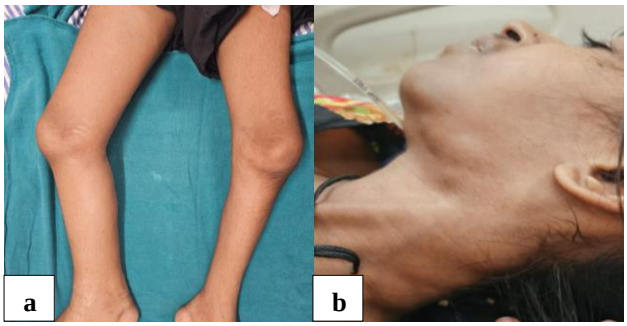

Figure 1: (a) Swollen right lower limb compared to left, and (b) matted cervical lymphadenopathy.

A biopsy of the cervical lymph node indicated necrotizing lymphadenitis, and a gastric aspirate tested positive for acid-fast bacilli. This confirmed the diagnosis of disseminated tuberculosis. The patient was started on a four-drug antitubercular regimen. For anticoagulation, she was initially given low-molecular-weight heparin for seven days, then transitioned to warfarin at a dose of 0.2 mg/kg/day, with adjustments made to maintain an INR between 2 and 3. INR levels were closely monitored to account for potential interactions with rifampicin.

The patient recovered well. She became afebrile within two weeks, and the swelling and pain in her leg improved within four weeks. A follow-up Doppler ultrasound of the right leg showed a residual thrombus with partial re-canalization and flow, indicating ongoing resolution. She was continued on antitubercular therapy and warfarin, with regular follow-ups. She is now in good health, has regained her appetite, and has gained 3 kg in the past month.

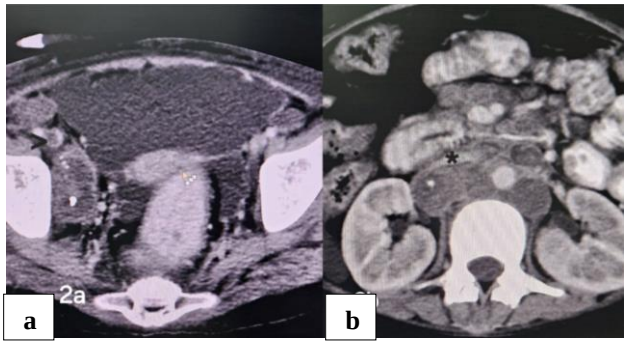

Figure 2: CECT abdomen images (a) filling defect in right external iliac vein suggesting thrombus, marked >, and (b) IVC compressed by the enlarged retroperitoneal lymph nodes, marked *.

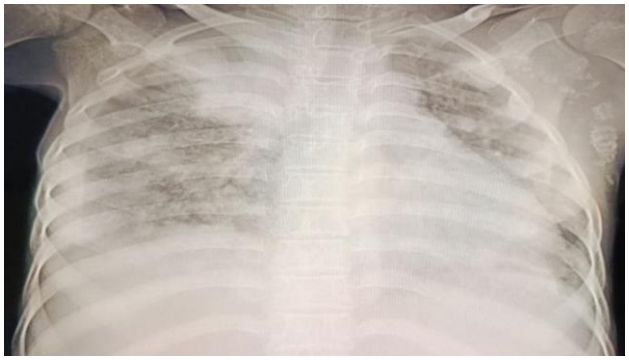

Figure 3: Chest X- ray showing bilateral patchy fluffy and nodular opacities.

DISCUSSION

Disseminated tuberculosis is characterized by the involvement of two or more non-contagious sites or organ systems due to the lympho-hematogenous spread of *Mycobacterium tuberculosis*. It can lead to vascular complications, which typically reflect the severity of the disease, with DVT being one such complication. Literature has highlighted active tuberculosis as an emerging risk factor for the development of DVT.[2,4-7]

Cases of TB associated with DVT are rarely reported in the pediatric population, although such cases are somewhat more documented in adults.[2,4-7] For instance, Jayesh et al reported a case of DVT in an 11-year-old in Kolkata in 2015, while Geeta et al described a case of

abdominal TB complicated by DVT and intracranial sinus thrombosis.[4,5] In both instances, DVT appeared after the initiation of anti-tubercular therapy, and neither case involved venous compression by enlarged lymph nodes as a cause of DVT.

The development of DVT in TB patients is multifactorial, involving hypercoagulability, endothelial injury, and venous stasis. In children, TB-induced DVT can present and impact differently than in adults due to variations in immune responses, which may affect inflammatory and coagulation pathways. TB's proinflammatory nature, characterized by monocyte and macrophage activation and the release of interleukins and cytokines, can contribute to endothelial injury and increase the risk of DVT. Other factors include elevated fibrinogen levels, impaired fibrinolysis, decreased antithrombin III, and reactive thrombocytosis.[8] Venous thrombosis can also result from compression by enlarged lymph nodes, though this can occur without bleeding abnormalities. Additionally, there may be an association between TB and antiphospholipid antibodies or protein S deficiency.[9] Some studies suggest a possible link between rifampicin use and increased DVT risk, though this does not preclude its use, but patients should be monitored carefully.[10]

In our case, intra-abdominal lymphadenopathy led to compression of the inferior vena cava and right external iliac vein, causing thrombosis a mechanism consistent with those discussed.

CONCLUSION

The simultaneous occurrence of DVT and TB presents diagnostic and treatment challenges. Clinicians should be vigilant for DVT in patients with disseminated TB, particularly if they show unexplained limb swelling or pain. Doppler ultrasonography is an essential diagnostic tool for these cases. Managing DVT in TB patients generally involves anticoagulation therapy, but close monitoring is necessary due to potential interactions with TB medications and bleeding risks.

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