Pulmonary Tuberculosis with Deep Venous Thrombosis

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Abstract

Tuberculosis is commonly encountered in developing countries like India. It can present with uncommon hematological manifestations which if not appropriately heeded to can make real diagnosis elusive. The present case highlights rare cooccurrence of pulmonary tuberculosis with deep venous thrombosis, which may at times pose a diagnostic challenge.

Introduction

Tuberculosis is a disorder of protean manifestations. There is paucity of data regarding occurrence of deep venous thrombosis in tuberculosis. Acute phase reactants, haemostatic changes and transient increase in anticardiolipin antibodies have been attributed to link inflammation with deep vein thrombosis in pulmonary tuberculosis1. As venous thromboembolism can be fatal, it is crucial to be proactive in arriving at an early diagnosis and institute prompt treatment2.

Case

A 45 years old male, smoker, non diabetic and normotensive, diagnosed case of sputum positive pulmonary tuberculosis on antitubercular treatment, having completed the intensive phase of the treatment and presently on continuation phase of the treatment regime with Isoniazid 300mg, Rifampicin 450mg and Pyrazinamide 1500mg, presented to medical outpatient department with complaints of swelling of the right leg since one month. The swelling had been initially progressive and associated with calf pain. General physical examination revealed a febrile(101°F oral temperature) male with a body mass index of 24.5. Besides occasional rales at right infraaxillary area, his rest of the systemic examination was unremarkable. The local examination of the right limb showed a swollen, erythematous and tender calf. The mid calf circumference was 11 inches on the right and 8 inches on the left side. The movements in the affected limb would induce calf pain. Peripheral pulses in the limbs were normally palpable on either side. Complete blood count analysis revealed Hb = 7.5g/dl, TLC = 6300/µl (neutrophils of = 49.3%, lymphocytes = 43.2%) and a platelet count of 193000/µl. Moreover his ESR, MCV and MCH were 86.6 fl, 25.5 and 30mm/hr respectively. LFT too was normal. Baseline INR was 1. Antiphospholipid antibody and collagen profile were negative. Kidney function tests, serum electrolytes and arterial blood gas analysis also were unremarkable. Colour doppler of peripheral veins of lower limbs revealed thrombosis of deep veins of right lower limb with thrombus extending to common iliac vein. However inferior vena cava was free of any filling defect and showed normal colour filling of the lumen(Fig:1). 24 hour urinary protein estimation revealed no proteinuria. Bone marrow aspiration revealed erythroid hypoplasia suggestive of a chronic disorder. A CT scan of the abdomen did not reveal any growth or lymphadenopathy causing compression of the intraabdominal vessels. Proten C and Protien S levels were normal.

In view of the doppler findings confirming deep venous thrombosis, the patient was put on an overlap of low molecular weight heparin and warfarin for initial five days followed by escalating dose of warfarin till an INR of 2.5 was achieved. The swelling in the limb subsided and patient was painfree by 10th day of admission. Subsequently he was discharged after 16 days of hospital stay and was put on warfarin 5mg od. He was on our regular follow up for initial four months after which he was lost to follow up.

Discussion

Although deep venous thrombosis in association with tuberculosis is considered a rare occurrence, yet it should be considered particularly in the setting of severe pulmonary or disseminated tuberculosis, as some authors argue that the risk of developing deep venous thrombosis is proportional to the severity of tubercular disease2. The cooccurrence of tuberculosis and deep venous thrombosis is reported to be high during initial phase of the disease. 3, 4 Hypercoagulability in tuberculosis can be attributed to several factors like decreased antithrombin III and protein C, elevated plasma fibrinogen levels, and increased platelet aggregation5, 6. In addition,
systemic inflammatory state prevalent in tuberculosis causes endothelial cell damage which in turn predisposes to local thrombosis. Subtle changes in blood rheologic properties and in the haemostatic system in patients with pulmonary tuberculosis have been reported. Serum fibrinogen level is seen to rise within the first 2 weeks of therapy and then normalise within 12 weeks, which, coupled with impaired fibrinolysis may result in deep vein thrombosis. Another hypothesis favouring a hypercoaguable state in tuberculosis is the increase in concentration of C4 b-binding protein (C4b BP), an acute phase reactant which binds protein S in plasma. Protein S is a cofactor for activated protein C mediated cleavage of Factor VIIIa and Factor Va. Also, experimentally peripheral blood mononuclear cells in tuberculosis can produce IL-1 and TNF-α, the latter causing down regulation of protein C/protein S during sepsis. High frequency of anti-phospholipid antibodies detected in patients with tuberculosis is also mentioned in the literature. Studies have also demonstrated that these haematological parameters worsen during the first 2 weeks of therapy in many cases, but they normalise after a month of anti-tuberculous therapy. The return of these haematological parameters to a normal level is a good indicator of disease control and correlate with sputum conversion in sputum positive tuberculosis patients.

Studies have also demonstrated a possible association between deep venous thrombosis and use of rifampicin with a relative risk of 4.74 in patients treated with rifampicin containing regimens. This does not contraindicate the use of this drug in patients at risk, but such patients need close monitoring. However, thrombosis can also result from venous compression by lymph nodes in ganglionar forms of tuberculosis, as retroperitoneal adenopathies may cause inferior vena cava thrombosis in the absence of any haemostatic abnormalities.

The hypercoagulable state seen in tuberculosis has therapeutic implications as well. In patients with tuberculosis there is a strong reason for prophylactic anticoagulation with heparin and avoiding central venous catheters. Anticoagulant therapy in tuberculosis is also problematic as the antitubercular drugs (INH, rifampicin) are strong enzyme inducers and can interfere with warfarin levels.

Our case highlights the risk of deep venous thrombosis in a patient with pulmonary tuberculosis even in the absence of any specific risk factors for venous thromboembolism. Emphasis is laid on high index of suspicion, early diagnosis, and institution of prompt treatment for deep venous thrombosis while continuing the antitubercular treatment.

References

Illustrations

Illustration 1

Fig 1. Doppler of lower limb veins showing thrombus in right calf veins extending to common iliac vein.
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