Original Research Paper

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Clinical Research

CASE REPORT- DEVELOPMENT OF DEEP VEIN THROMBOSIS IN A TUBERCULOSIS PATIENT AFTER START OF ANTITUBERCULAR TREATMENT

Dr Priyadarshini Behera — MD (Pulmonary Medicine), Assistant Professor, Dept of Pulmonary Medicine, IMS and SUM Hospital, Bhubaneswar

Dr Sonali Parida — MD (Pulmonary Medicine), Senior resident, Dept of Pulmonary Medicine, IMS and SUM Hospital, Bhubaneswar

Dr Sadananda Barik — Consultant Pulmonologist and Intensivist, Kalinga Hospital Limited, Bhubaneswar

ABSTRACT Tuberculosis is a highly prevalent disease worldwide especially in developing countries. It leads to lots of morbidity and mortality. Deep vein thrombosis as direct consequences of TB and therapeutic consequences of hypercoagulable state in patient receiving antitubercular therapy has also been reported. We are presenting a case report of a young patient who developed deep vein thrombosis following start of antitubercular drugs.

KEYWORDS : Deep Vein Thrombosis, Tuberculosis, Antitubercular treatment

Introduction:
Global incidence of Tuberculosis is 104 lakhs with a mortality of 14 lakhs (1). This deadly disease not only has various direct damaging results but also has important indirect consequences.

There is a complex interplay of acute phase reactants, haemostatic variations, temporary rise of anticardiolipin antibodies involved in deep vein thrombosis formation in Pulmonary tuberculosis (2). Thus, it is important to create awareness regarding this association to prevent life threatening complications.

Case report:
A 22-year-old female presented to emergency with complaint of low grade intermittent fever, shortness of breath and cough with mucoid expectoration for 1 month. There was also loss of appetite and weight loss of 5 kg in the last 3 months. She gave history of pulmonary TB twice in the past, first 12 years back and second 4 years back. On examination she had bilateral pedal oedema, hypotension and hypoxemia (BP- 90/60, SpO2- 88 % with room air). There were bilateral coarse crepitations on auscultation of chest. Her ABG revealed Type 1 respiratory failure. Chest X-Ray revealed bilateral bronchiectasis with cavities in the right upper zone (Fig. 1). ECG revealed inverted T waves in the leads V1- V4. 2D-Echocardiography revealed global hypokinesia, moderate LV systolic dysfunction with an ejection fraction of 39 %. She responded well to intravenous fluids and antibiotics, inhalational bronchodilators and oxygen initially. She had a TLC count of 22,000/cu mm with neutrophilia. Her renal function tests and liver function tests were within normal limits. Viral markers like HBsAg, anti HCV and HIV were negative.

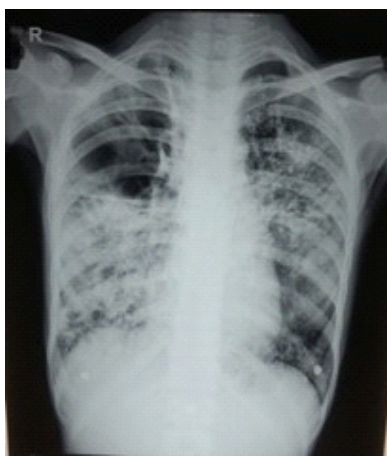

Fig 1 (Chest X-ray: right upper lobe cavitary lesions and bilateral lung opacities

HRCT thorax revealed cavities in right upper and middle lobes with bilateral bronchiectasis and tree in bud nodules (Fig 2). Her sputum was negative for AFB. Her sputum for Cartridge-based nucleic acid amplification test (CBNAAT) revealed presence of Rifampicin sensitive *Mycobacterium tuberculosis.* So she was started on standard ATT for retreatment case.

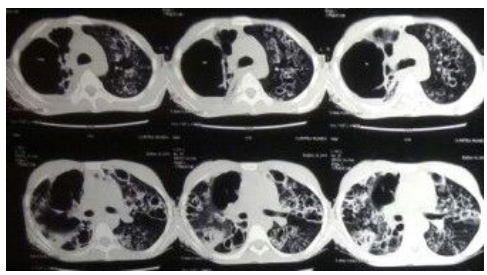

Fig 2. HRCT thorax revealed cavities in right upper and middle lobes with bilateral bronchiectasis and tree in bud nodules

On seven days of start of ATT she complained of severe pain in her right leg and clinically there was swelling of right leg with calf tenderness and a positive Homan's sign. An urgent USG venous Doppler of both lower limbs was done which revealed acute deep vein thrombosis involving right lower limb veins with cranial extension up to common iliac veins (Fig 3).

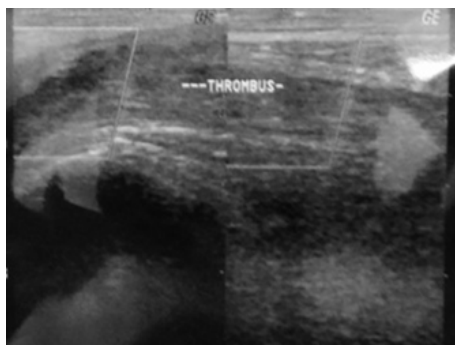

Fig 3. Thrombosis in right femoral vein

She was immediately started on Enoxaparin 60mg subcutaneous twice daily along with tablet Acenocoumarol. When the INR of 2 was achieved, Enoxaparin was discontinued and Acenocoumarol was continued. The patient improved symptomatically with reduction in the limb swelling and pain. Her fever subsided and her vitals significantly improved. She was discharged with ATT and Acenocoumarol and advised for regular follow up.

Discussion:

Vascular and haematological disturbances are rare in pulmonary tuberculosis. TNF α and IL- 6 produced by interaction between mycobacterial products and monocyte macrophage system make the internal surface of blood vessels thrombogenic (3). Increased fibrinogen, defective fibrinolysis, decreased anti thrombin III, reactive thrombocytosis are key factors in development of DVT in TB patients (4). There is a possible association between DVT and use of rifampicin with a relative risk of 4.74 in patients using a rifampicin based therapy (5). Therapeutic consequences of hypercoagulable state in tuberculosis are also important. Prophylactic heparin anticoagulation and avoidance of central venous catheters are recommended in TB patients (6). Besides, DVT management is quite difficult in TB due to concurrent use of enzyme inducers like rifampicin which can alter warfarin levels. A study by Turken et al showed that haemostatic disturbances improved within 4 weeks of commencing anti tubercular therapy (7). Hence early initiation of anti tubercular therapy also prevents serious haemostatic disturbances.

Conclusion:

In our case the patient developed DVT without presence of any risk factors. In all patients with TB, clinical suspicion, prompt diagnosis, treatment as well as monitoring while continuing anti tubercular therapy is very essential.

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