CASE PEPORT

Inferior Venacaval Thrombosis in Active Tuberculosis

*ABI Momen[1], FQ Twinkle[2], FA Quraishi[3]

ABSTRACT

Approximately one third of the world's population is infected with Mycobacterium tuberculosis. Tuberculosis has several mechanisms that induce a hypercoagulable state and can lead to thromboembolic complications. A variety of factors have been postulated to predispose patients with tuberculosis to venous thrombosis that include local stasis due to venous compression by lymph nodes and relative immobility caused by respiratory, alteration in coagulation factors and reactive thrombocytosis. Only a few case reports showing similar findings.

We present a case, man of 26 years old, who presented deep vein thrombus of inferior vena cava from its origin extending up to the entrance of the liver revealed signal changes on L2 vertebrae and CT guided FNAC was negative for malignancy but suggestive of tuberculosis. There might be a possible association between deep venous thrombosis and use of Rifampicin but this does not contraindicate the use of Rifampicin but these patient may need close monitoring.

Key words: Mycobacterium tuberculosis, deep venous thrombosis, inferior vena cava.

Introduction

Tuberculosis persists as a major cause of morbidity and mortality worldwide. As per WHO Global tuberculosis report 2014, in 2013 an estimated 9 million people developed tuberculosis and 1.5 million died from the disease[1]. Tuberculosis can rarely present with venous thrombosis which is rooted in the development of a transient hypercoagulable state in a subset of patients[2]. In a retrospective South African study reported that the prevalence of DVT in tuberculosis is 3.4% within first two weeks after initiation of antitubercular therapy due to endothelial dysfunction by rifampicin[3]. However a more recent Italian study reported the prevalence to be 0.3% in the first month of starting antitubercular therapy, nearly all occurring in hospitalized patients[4]. A variety of factors have been postulated to predispose patients with tuberculosis to venous thrombosis that include local stasis due to venous compression by lymph nodes and relative immobility caused by respiratory [4,5,6],alteration in coagulation factors and reactive thrombocytosis [6]. The haemostatic changes leading to a prothrombotic state are reported to normalize following treatment of the underlying infection[5]. These have included venous thrombosis in lower limb veins, Inferior vena cava, portal vein, cerebral veins and retinal vein [7,8,9,10,11,12,13].

We herewith report a case of inferior vena caval thrombosis (IVC) in patients with evidence of active pulmonary and spinal tuberculosis.

Case report:

A 26-year-old male presented with severe backache for the last 6 months along with fever and weight loss, productive cough, patient was previously diagnosed Pulmonary Tuberculosis 2 months ago based on positive Montoux test, acid-fast bacilli (AFB) in sputum (>30bacilli/field) and high ESR (60) already on anti-tubercular medication 4FDC.

1Dr. Abdul Basit Ibne Momen, Registrar, Department of Medicine, Bangladesh Medical College

2Furial Quraishi Twinkle, Medical Student, Anwer Khan Modern Medical College

3Prof. Firoz Ahmed Quraishi, Professor, Department of Neurology, Anwer Khan Modern Medical College

**Corresponding author*

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General physical examination revealed thin male body mass index of 26. He was apyrexial, haemodynamically stable, SPO2 (FiO2 21%) was 97%. Systemic examination of chest, cardiovascular and abdominal system was unremarkable except engorged veins over the abdomen, right leg was slightly swollen and tender on palpation with prominent veins, tenderness present over the lower back (Fig-1). Routine investigations revealed Hb 11.6 g/dl, leucocyte 8900/µl and platelet were normal (Bilirubin 0.6, ALT 191). Ultrasound of abdomen demonstrated deep vein thrombus of inferior vena cava from its origin extending upto the entrance of the liver (Fig-2). No other risk factors for VTE identified. Baseline INR was 1.14. MRI of spine revealed signal changes on L2 vertebrae (Fig-3) and CT guided FNAC was negative for malignancy but suggestive of tuberculosis. The patient was started on heparin and then switched to warfarin to maintain the desired INR and discharged t come for follow up in 2 weeks.

Fig-1: Right leg showing engorged veiws

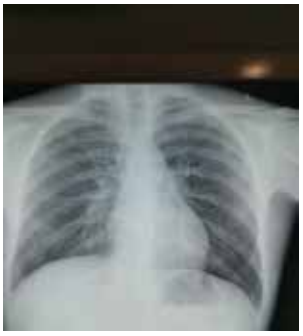

Fig-2: Chest X-ray normal

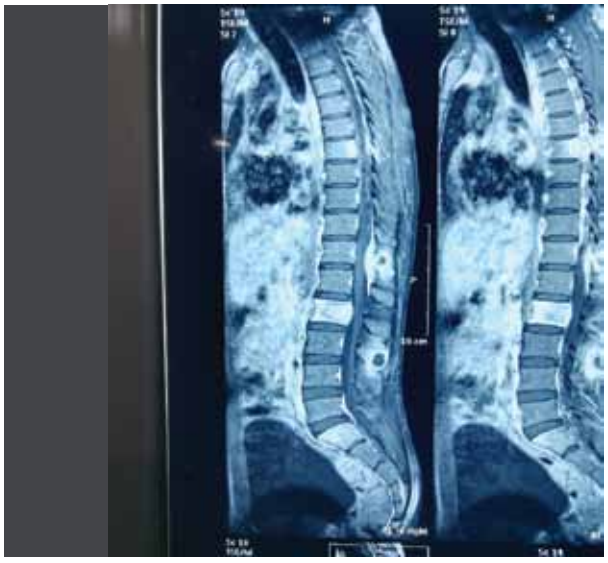

Fig-3: MRI of Spine showing signal changes on L_2 vertebrae

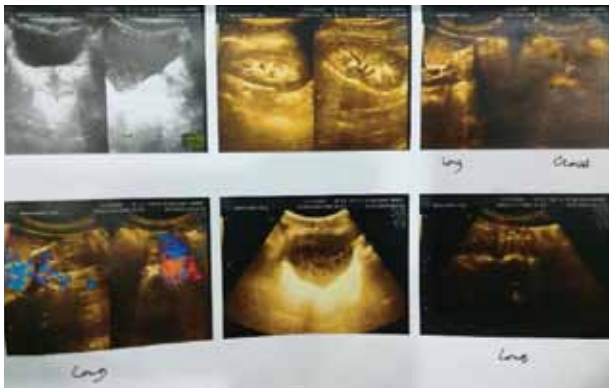

Fig-4: USG of whole abdomen showing deep veiws thrombus in inferior Vena Cava

Discussion

Our patient had extensive inferior vena caval thrombosis which 2 months of initiation of antitubercular therapy in the other. Inferior vena caval thrombosis has been reported only rarely in active pulmonary tuberculosis and only 3 reports exist in English literature. The hypercoagulablity in tuberculosis has been postulated to arise from a number of factors like reduced anti-thrombin III and protein C levels, elevated plasma fibrinogen levels, and an increased aggregation of platelets[5,6] . Serum fibrinogen rises within the first 2 weeks of therapy and subsequently normalizes within 12 weeks. Coupled with impaired fibrinolysis, elevated fibrinogen levels may result in a pro-thrombotic state[6]. Additionally endothelial dysfunction may result in the systemic inflammatory response to tuberculosis and combined with subtle changes in blood rheologic properties[14] may result in intravascular coagulation. A higher frequency of antiphospholipid antibodies and Protein S deficiency has been reported in tuberculosis[2]. Protein S deficiency may result from an increase in concentration of C4 binding protein an acute phase reactant which binds protein S in plasma, and a production of IL1 and TNF-αby peripheral blood mononuclear cells that can result in down regulation of protein C/protein S during any sepsis[15]. 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. Also, retroperitoneal adenopathy may cause inferior vena cava thrombosis in

absence of any haemostatic abnormality [16]. Higher dose of warfarin is necessary to achieve therapeutic INR levels due to rifampicin effect on cytochrome P450 [17].

Conclusion:

An association between inflammation induced by tuberculosis and a hypercoagulable state has been described. Therefore, the occurrence of deep venous thrombosis or pulmonary embolic episodes, should be considered in patients with tuberculosis particularly during the first weeks of treatment. The physician's awareness of these phenomena is important to an early diagnostic suspicion and prompt treatment in order to prevent fatal outcomes.

Conflict of interest: None.

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