



Inferior Vena Cava Thrombosis Complicating Tuberculosis

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Abstract

Tuberculosis (TB) remains a major global health problem in developing countries with a myriad of presentations and complications. Deep Venous thrombosis is a rare complication of this disease. We report an unusual case of Inferior vena cava thrombosis complicating isolated lymphadenopathy tuberculosis. A 23 year-old male presented with a three months history of moderate abdominal pain associated with anorexia, asthenia, weight loss, fever and night sweats. The patient's abdominal and chest Computed Tomography (CT) revealed cervical, axillary and abdominal lymph nodes associated with a floating thrombus in the inferior vena cava. *Mycobacterium tuberculosis* (MT) infection was confirmed by the bacteriological and histopathology examination of the axillary lymph node biopsy specimen. He was treated with low-molecular-weight heparin, acenocoumarol and antituberculous drugs. His general state improved and he responded well to anticoagulation therapy.

Keywords: Tuberculosis; Venous thrombosis; Prognosis

Introduction

Tuberculosis (TB) remains a major global health problem in developing countries. Clinical presentations depend on localizations and with a myriad of presentations and complications. Tuberculosis infection can be of pulmonary or extrapulmonary type [1]. Like other infectious diseases, TB can cause thrombosis by various mechanisms such as local invasion, venous compression or by producing a transitory hypercoagulable state. Although deep venous thrombosis is considered a rare event and because it can be fatal, it is crucial to suspect it to perform an early diagnosis and initiate prompt treatment [2].

We report an unusual case of Inferior vena cava thrombosis as a complication of isolated tuberculous abdominal lymphadenopathy in a young man.

Case Presentation

A twenty-three-year-old Tunisian male soldier presented with a three months history of abdominal pain. He was an active smoker of one pack per day for the past of six years, a moderate alcohol consumer and had sexual risk behaviors. There was no history of past illness and no family history of chronic disease. The patient had received BCG vaccination at birth and had never contacted a patient with TB. The patient denied recent trauma, surgery, immobilization, or family history of thrombotic disorder.

The abdominal pain was insidious in onset, slowly progressive, moderate, generalized and was associated with anorexia, asthenia, weight loss and fever. The fever was low-grade and not associated with chills or rigors, but was associated with night sweats.

On examination he was afebrile and had multiple cervical, supra clavicular, axillary and inguinal lymph nodes. Abdominal examination was normal including per rectal examination. Clinical examination of other systems was clinically normal.

Laboratory findings on admission revealed a normal WBC count (6100 G/mm^3) normochromic normocytic aplastic anaemia (haemoglobin 10 g/dl), lymphopenia (lymphocyte count $=700/\text{mm}^3$), normal platelet count ($293000/\text{mm}^3$) with high Erythrocyte sedimentation rate (ESR = 90 mm in the first hour) and elevated C-reactive protein (27 mg/l). His renal and liver functions were normal. Coombs' test was positive. A chest X-ray film was normal.

Ultrasound examination of abdomen showed multiple lymph nodes. The patient's abdominal and chest computed tomography revealed cast of adenomegaly to axillary left supra clavicular necrotic center and lower left jugulo carotidienne, cast of the hepatic hilum adenomegaly coelio mesenteric, aortic lateral inter aortico cellular and retro cave. The scan also showed a floating thrombus in the inferior vena cava in renal stretched to the common and internal right iliac vein (Figure 1).

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Figure 1: MRI: floating thrombus in the inferior vena cava in renal stretched to the common and internal right iliac vein.

A Mantoux test showed pronounced erythema with a duration of 13 mm.

Histopathology examination of the left axillary lymph node biopsy specimen revealed the presence of granulomas in the lymph node tissues with marked epithelioid cell changes as well as giant cells formation associated to caseation and necrosis. Another biopsy specimen was found to contain acid-fast bacilli. *Mycobacterium tuberculosis* (MT) infection was confirmed by culture on Lowenstein-Jensen agar and MT organisms were found to be sensitive to all first-line antituberculosis drugs. Serologic tests for HIV, HBV and HCV were negative. Because of the unusual location of the patient's thrombosis, an evaluation was performed for hematologic disorders. Laboratory assessment of protein C and S activity, cardiolipin antibodies and antithrombin III activity was unrevealing. Malignancy was ruled out with physical exam, imaging and histopathology examination of the lymph node biopsy. The patient was put on low-molecular-weight heparin (Innohep 0.5 ml/day) and 15 days later, acenocoumarol (1 mg/day) was started after biopsy. The target International Normalized Ratio (INR) of 2.0-3.0 was achieved, 35 days later, with a daily acenocoumarol 10 mg dose (5 mg twice/day).

He received also anti-tuberculosis treatment: HRZE 4 tablets/day (isoniazid (200 mg/day), rifampin (480 mg/day), pyrazinamide (1200 mg/day) and ethambutol (800 mg/day) for 2 months and then isoniazid (200 mg/day) and rifampin (480 mg/day) for 8 months. No adverse effects were seen. His general state improved and he responded well to anticoagulation therapy.

Discussion

Tuberculosis is a major killer worldwide mainly affecting people of developing countries. There were an estimated 8 million new cases of TB, resulting in 1.9 million deaths, with the greatest burden of disease in developing nations. Actually, tuberculosis is a disease with a wide variety of clinical presentations and recently, there is a relative increase in the incidence of extrapulmonary involvement. Lymph node is the most common extrapulmonary site of tuberculosis [1].

Vascular complications associated with mycobacterium tuberculosis infection had been reported in the literature and occurred in 1.5% to 3.4% of tuberculosis infection. Venous thromboembolism is a rare complication of tuberculosis and can be the presenting feature of TB, occur a few days after the diagnosis or late in the course of the disease, even in patients on ATT [2].

The factors commonly associated with the pathogenesis of thrombosis are: alteration in the wall of the vein, alteration in the blood constituents and slowing of the stream [2,3].

Tuberculosis has several mechanisms that induce a hypercoagulable state and can lead thromboembolic complications. The association between inflammation, haemostatic changes and hypercoagulable state has been established in tuberculosis recently. Hypercoagulability in tuberculosis is attributed to decreased antithrombin 3 and protein C, elevated plasma fibrinogen level, increased platelet aggregation and reactive thrombocytosis. Apart from high frequency of antiphospholipid antibody levels in a patient with tuberculosis, deficiency of protein S has been mentioned [3,4]. But, in our patient all these were normal.

Cytokines by their pro-inflammatory character will activate the vascular intima and make thrombogenic endothelium. They will also lead to a stimulation of hepatic synthesis of coagulation proteins [5,6]. These risks of hypercoagulability are increased by immobility and bed rest because of the morbidity caused by the disease. However, thrombosis can also result from venous compression by lymph nodes in ganglionar forms of TB, as retroperitoneal adenopathies may cause inferior vena cava thrombosis in the absence of any haemostatic abnormalities [7,8].

Many reports demonstrate that thrombotic phenomena in patients occurs in several sites including hepatic veins, the vena porta, jugular vein, iliac vein, inferior vena cava. Retroperitoneal para-aortic lymphadenopathy is commonly seen in tuberculosis and have been reported to cause mechanical venous obstruction in few patients in literature [3,9]. Tubercular lymph nodes may cause IVC thrombosis either by a large collective matted mass or due to minor obstruction in the presence of an underlying predisposed heredity thrombophilic state. In our case, protein C, S, antithrombin 3 levels and activated partial thromboplastin time (for lupus anticoagulant) were normal, thus eliminating a predisposed thrombophilic state. Hence, large matted tubercular lymph node masses are the probable explanation for the IVC and iliofemoral thrombosis in our patient.

Regardless of the cause, prompt recognition of IVC thrombosis is important because of the potential acute complications. It carries a higher risk of pulmonary embolism than lower extremity deep venous thrombosis with 33% reported. There is also risk associated with clot propagation including extension to the renal veins and extension to the hepatic veins. Though rarely reported, critical limb ischemia secondary to phlegmasia cerulean dolens is another potential complication [10]. Physical exam findings and symptoms are variable and dependent on the degree and location of occlusion. Once suspected, the diagnosis of IVC thrombosis is established through imaging. Computed tomography with contrast and Magnetic Resonance Imaging (MRI) has been shown to be equally sensitive. The gold standard imaging modality is venography, though this is invasive and time consuming, but it is the preferred method if surgical intervention is planned [6,7,10]. In our case, CT identified the presence of caval thrombosis.

Frequently, a higher dose of acenocoumarol is necessary to achieve therapeutic INR levels, because of rifampicin effects on cytochrome P450. Additionally, this drug may also contribute to the hypercoagulable state by decreasing production and increasing clearance of anticoagulant hepatic proteins [4,10].

Conclusion

Thromboembolic disease is to search systematically at the TB view of the risk of occurrence of this complication particularly in extensive and severe forms. Prophylactic anticoagulation finds its

indications in these forms. Inferior vena cava thrombosis may be one of the atypical presentations of tuberculosis and the possibility of retroperitoneal tubercular lymphadenitis should be considered in cases of unexplained thrombosis.

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