Disseminated tuberculosis causing isolated splenic vein thrombosis and multiple splenic abscesses

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Tuberculosis is a common infectious cause of splenic enlargement in developing countries, but tubercular splenic abscesses are a rare presentation, found predominantly in immunocompromised populations. We report a case of tubercular splenic abscesses with isolated splenic vein thrombosis in an immunocompetent person.

INTRODUCTION

Extra-pulmonary tuberculosis (TB) is an emerging problem even in developing countries. Abdominal TB is a frequent presentation of the disease. It commonly involves the ileocecal junction, anorectal region, lymph nodes and/or peritoneum. There have been reports of splenic abscesses and venous thrombosis in immunocompromised patients with TB; however, isolated splenic vein thrombus with multiple splenic abscesses has been rarely reported in immunocompetent persons [1, 2].

CASE REPORT

We report a case of 16-year-old female who presented to our outpatient clinic with complaints of low-grade fever, dry cough, along with yellow discolouration of urine, skin and repeated episodes of vomiting of 7 days duration. She also reported chronic diarrhea, amenorrhea and a 10 kg weight loss over a 5-month period. There was no history of abdominal pain, hemoptysis, skin rash, dyspnea, joint pains or photosensitivity. The patient had been on anti-TB therapy (ATT) initiated by a private practitioner 20 days prior to presentation. On presentation the patient was hemodynamically stable, pulse was 104/min regular rate and of good volume, BP was 124/82 mmHg and she was well oriented to time, place and person. The respiratory rate was 18 per min and oxygen saturation was 96%. The patient was febrile (temp-102 F) with mild pallor, scleral icterus and hepatosplenomegaly. The liver was enlarged, firm, 2 cm below the right costal margin at the mid-clavicular line, non-tender with smooth surface while the tip of the spleen was palpable below the left costal margin which was tender on deep palpation. She did not have any peripheral lymphadenopathy, cyanosis, digital clubbing, pedal edema or rash. Rest of the systemic examination was unremarkable.

Investigations revealed a hemoglobin of 10.5 g%, WBC 4800/mm³ with 52% neutrophils and 48% lymphocytes, platelet count 50 000/mm³ and ESR 88 mm in first hour. The peripheral blood film showed normocytic normochromic red blood cells. Blood culture, Widal test, peripheral blood film, rapid antigen card test for malaria and serologies for Leptospira, dengue and scrub typhus were negative. Urine microscopy was within normal limits and urine culture was sterile and was negative for pyogenic bacteria and AFB. Her Mantoux test reaction was positive (22/20 mm). Total bilirubin was 5 mg/dl (normal level 0.2–0.8 mg/dl) with direct, 1.6 mg/dl and indirect, 3.4 mg/dl; AST/ALT level were raised at 141 and 94 U/l, respectively (normal level <40 U/l). Serum LDH, amylase, lipase and alkaline phosphatase were within the normal range. Laboratory tests for viral infections (ELISA for HIV, HBsAg and anti-HCV antibody), connective tissue markers including ANA, APLA and ANCA antibodies were negative. Prothrombin time was elevated at 21 s (control 13 s); INR(1.7) were raised as well. Urine for pregnancy test was negative. Repeated blood cultures for aerobic and anaerobic infections were sterile and renal function tests were also normal. Chest radiograph revealed blunted left cardiopulmonary angle with normal lung parenchyma. Abdomen ultrasonography revealed multiple enlarged peri-pancreatic and
para-aortic lymph nodes. The spleen measured 13 cm with multiple hypoechoic lesions. The liver was also enlarged measuring 14.5 cm with normal echotexture and there was minimal free fluid in pelvis.

Contrast enhanced computed tomography (CECT) chest revealed multiple enlarged necrotic lymph nodes in perivascular, paratracheal, peri-bronchial and subcarinal regions with pericardial effusion, bilateral pleural effusion (left more than right) and consolidation in the left upper lobe (Fig. 1a). Computed tomography (CT) abdomen revealed multiple hypodense coalescing lesions in the spleen and well-defined partial filling defect in splenic vein (suggestive of thrombus) along with thickening of terminal ileum and cecum with retracted ileocaecal junction. The liver, pancreas and both kidneys appeared normal (Fig. 1b). Protein C and protein S levels were normal, factor V leiden mutation, lupus anticoagulant and anti-phospholipid antibody were also negative. Echocardiography revealed minimal pericardial effusion with normal left ventricular ejection fraction. Bone marrow examination revealed hypercellularity with normoblastic erythropoiesis and culture was subsequently positive for *Mycobacterium tuberculosis*.

A diagnostic as well as therapeutic pleural tap was done. It was found to be exudative (protein, 4.2 g/dl) and exhibited cow-web coagulum. The test performed on the fluid revealed elevated ADA at 91 U/l (normal, 30 IU/l) and leukocyte count of 2450/mm³ with 70% lymphocytes. Genexpert polymerase chain reaction was also positive for *M. tuberculosis*. Rpo B gene, which confers resistance to rifampicin, was not detected. As the patient did not have any superficial lymph nodes appropriate for biopsy, CT-guided biopsy of paratracheal lymph node and splenic abscess were planned but had to be deferred because of deranged prothrombin time.

Because of concern for drug-related hepatotoxicity, the patient was started on modified ATT consisting of levofloxacin, streptomycin and ethambutol and low-molecular weight heparin for splenic vein thrombus. The patient responded clinically with normalization of AST/ALT levels in 15 days. Regular ATT (rifampicin, isoniazid, ethambutol and pyrazinamide) was reintroduced in a phased manner. Subsequently, the patient did not show any significant elevation of liver enzymes and was put on four drug ATT for 2 months followed by consolidated phase of 4 months on rifampicin and isoniazid. CT abdomen revealed resolution of lesions and decrease in the size of lymph nodes after 3 months. CT angiography showed resolution of splenic vein thrombus completely after 3 months along with regression of spleen size.

DISCUSSION

The common presentations of extra-pulmonary TB are lymphadenopathy, pleural effusion, bone and joint disease, abdominal TB, pericardial disease, meningitis and miliary disease [3]. Splenic TB, especially with abscess formation, is an unusual manifestation and occurs primarily in patients with concurrent HIV infection [4, 5]. However, it may be found as part of severe, disseminated disease in non-HIV-infected patients, as in our reported case. Splenic TB usually occurs following the haematogenous seeding, or by contiguous spread of infection. The incidence of splenic abscess is very low (0.14–0.7%) in various post-mortem studies, and is usually associated with septicemic conditions [6]. In the event of multiple splenic abscesses and splenic rupture the treatment is generally splenectomy. Even in those scenarios, ATT must be used as complementary treatment especially when associated with mesenteric involvement. Our patient responded to ATT alone without the need for surgery.

Deep venous thrombosis (DVT), although a rare event, should be considered particularly in those with severe pulmonary or disseminated TB [7]. Other reports also point to thrombotic phenomena in patients with TB occurring at other sites including hepatic veins and cerebral venous sinuses [8, 9]. Like any other infectious disease, TB can cause thrombosis.
by various mechanisms including local invasion, venous compression or by producing transient hypercoagulable state [10]. Elevated plasma fibrinogen, impaired fibrinolysis coupled with decreased levels of antithrombin III and reactive thrombocytosis appear to favor the development of DVT in pulmonary TB [11].

As splenic vein thrombosis is rare and there are no controlled therapeutic trials assessing the therapeutic role of anti-coagulants, the impact of these interventions remains unknown. Anticoagulation has not been recommended in asymptomatic patients, unless the thrombus extends into the mesenteric vein, posing a risk of mesenteric ischemia [12]. A potential benefit of anti-coagulant therapy is that it may prevent extension of thrombus in the porto-venous system, and postportal or portosystemic collaterals. Although the role of anti-coagulants in this case was not clearly defined and there was a risk of bleeding in the background of deranged liver functions with coagulopathy, we initiated anti-coagulant therapy anticipating recanalization of splenic vein and prevent further complications.

CONCLUSION

The occurrence of isolated splenic vein thrombus with TB has been rarely reported earlier [1, 2]. In our case, though we could not establish histologic or microbiologic evidence of TB in splenic tissue, the resolution of abscesses and splenic vein thrombus with ATT made us confident that TB was the culprit behind this atypical presentation. In developing countries like India, where TB is endemic, TB should be suspected as differential diagnosis of isolated visceral venous thrombosis. This case highlights the idiom that TB can present in uncommon ways.

CONFLICT OF INTEREST STATEMENT

None declared.

REFERENCES