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Case report

Primary hepatic tuberculosis presenting as acute liver failure

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Abdominal tuberculosis is a common clinical entity in Indian subcontinent; however, hepatic tuberculosis in the absence of miliary abdominal tuberculosis is restricted to the case reports and small case series in English literature. It mimics common liver diseases like liver abscess and tumors. We report a case of 25-year-old immunocompetent female who presented with features of acute liver failure. Ultrasonography (USG) abdomen revealed multiple hypoechoic lesions. However, patient expired within 48 h of presentation but her liver autopsy revealed multiple epithelioid cell caseating granulomas with positive staining for acid fast bacilli (AFB). Polymerase chain reaction (PCR) was also positive for *Mycobacterium tuberculosis*.

INTRODUCTION

Although hepatic tuberculosis is one of the rare forms of extrapulmonary tuberculosis, isolated hepatic tuberculosis without any evidence of tuberculosis elsewhere is even rarer [1]. A greater awareness of this rare clinical entity may help in commencing specific evidence-based therapy quickly and preventing undue morbidity and mortality.

CASE REPORT

A 25-year-old unmarried immunocompetent female was brought to the emergency department in altered sensorium. She was having low-grade fever associated with decreased oral intake for past 15 days. She had developed abdominal pain, yellowish discoloration of urine and sclera 3 days back along with non-projectile vomiting, constipation and oliguria for last 2 days. Patient developed altered sensorium along with difficulty in breathing night before coming to the hospital. There was no history of headache, sore throat, dysuria, joint pains and photosensitivity. There was no past history of diabetes mellitus, tuberculosis or any other chronic disease. Family history was non-significant.

On examination, patient was drowsy; her BP was 102/70 mmHg but had tachycardia. She had pallor, icterus and ecchymotic patches on the trunk and extremities. On CNS examination Glasgow coma score was 6/15, neck rigidity and kernig's sign was absent. Planters were flexor and deep tendon reflexes were absent in all four limbs. Sensory system, cranial nerves and power could not be tested. Chest examination revealed vesicular breathing with conducted sounds bilaterally. Rest of the systemic examination was unremarkable.

Investigations revealed Hb 10 g/dl, TLC 16 300/mm^3 (70% polymorphs, 30% lymphocytes), platelet count 78 000/mm^3, ESR 80 mm at first hour. Prothrombin time was raised (35 s) and INR was three. Blood culture, Widal test, PBF and rapid antigen card test for malaria, serology for leptospira, dengue and scrub typhus were negative. Urine microscopy was within normal limits and urine culture was sterile. Her Mantoux test reaction was positive (22 × 20 mm). Viral markers for HIV, hepatitis B, C and IgM for hepatitis A, E were negative. ANA, SMA and LKM antibodies were negative. The AST/ALT level was raised at 160 and 294 U/l, respectively. Serum alkaline phosphatase was raised (300 IU). Serum proteins were 6.8 g/dl with serum albumin 3.7 g/dl and serum globulin of 3.1 g/dl. Serum bilirubin was 7 mg/dl (direct 3 mg/dl and indirect 4 mg/dl). Blood urea was 98 mg/dl and serum creatinine was 2.2 mg/dl. Serum copper was 13 mol/l, serum ferritin was 81 pmol/l and serum LDH was 91 U/l which were with in normal range. Rest of the biochemistry profile was also normal. Chest radiograph revealed normal study. Ultrasonography (USG) abdomen showed multiple hypoechoic lesions in liver; largest being 4.5 × 3 cm. There was no evidence of any free fluid or any other organomegaly. FNAC/Biopsy liver was planned to know the exact cause of lesion and rule out malignancy.

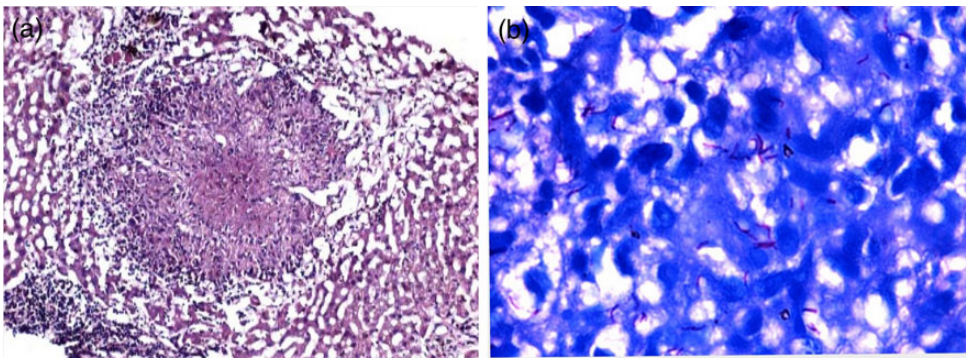

Figure 1: (**a**) H&E stained section of Liver showing necrotizing granuloma. (**b**) ZN staining revealing acid fast bacilli.

However, patient developed septic shock and required mechanical ventilation with triple inotropic support but she could not survive beyond 48 h of hospitalization.

Autopsy was conducted and microsections from liver revealed multiple caseating epithelioid cell granulomas with mononuclear inflammatory cell infiltrate. Acid fast bacilli (AFB) staining and polymerase chain reaction (PCR) for *Mycobacterium tuberculosis* was positive (Fig. 1). Microsections of lungs, spleen, kidneys and gut showed mild congestion. The brain showed edema along with congestion whereas the heart was unremarkable. Chemical analysis of viscera was negative which ruled out any drug intoxicity.

With autopsy report, consistent investigations and no evidence of tuberculosis anywhere in the body except liver, diagnosis of primary hepatic tuberculosis with acute liver failure and encephalopathy was made.

DISCUSSION

Hepatic tuberculosis has been classified by Levine as miliary tuberculosis, pulmonary tuberculosis with hepatic compromise, primary hepatic tuberculosis, focal or tuberculous abscess and tuberculous cholangitis; most common form being the miliary tuberculosis [2]. The respiratory and GI tract are the major sources of infection and bacilli travel there via hepatic artery or the portal vein. Tuberculosis involving only liver is considered to be rare, it being an unfavorable site for the growth of mycobacteria due to the low oxygen tension of liver [3].

The most common clinical findings are abdominal pain, fever and weight loss. Hepatomegaly is frequently found. The laboratory investigations frequently show an increase in alkaline phosphatase, with normal to mildly increased transaminases, anemia, hypoalbuminemia, hypergammaglobulinaemia and hyponatremia [4]. Ultrasonography may reveal hypoechoic nodes and, rarely, hyperechoic nodules. Due to the polymorphism of the lesions on imaging, resembling primary hepatic neoplasms or metastasis, diagnosis is difficult without a histopathological examination [5]. For this reason, biopsies are often required and sometimes diagnosis is made on autopsy.

Demonstration of AFB on ZN staining and caseous necrosis on biopsy can be very useful pathological tools, but their absence cannot exclude the diagnosis [6]. Nonspecific findings include Kupffer cell hyperplasia, focal hepatocytic necrosis with lymphocytic infiltration of hepatic parenchyma and portal inflammation [5]. Recently, PCR has been found to be a useful diagnostic tool for hepatic tuberculosis as it enables rapid identification of *M. tuberculosis* and expedites a treatment decision. At least 57% of tuberculosis hepatic granulomas gave positive PCR results compared with other conventional diagnostic techniques for TB [7].

Hepatic TB is a curable disease with relatively good outcomes; however, a delay in diagnosis can have fatal consequences. It warrants increased awareness about its varied presentations, diagnostic pitfalls and recommended treatment protocols especially for physicians practicing in tuberculosis endemic areas. It has been recommended that patients with isolated hepatic TB should be treated with standard regimens despite the potential hepatotoxicity of these drugs [8].

Our patient presented as acute liver failure with hepatic encephalopathy with multiple hypoechoic lesions on USG. However, patient expired within 48 h of presentation but her liver autopsy revealed multiple caseating epithelioid cell granulomas with positive staining for AFB and PCR. Similarly, isolated hepatic tuberculosis presenting as jaundice and abdominal distension has been reported in literature by Shastri *et al*. [9] However, hepatic TB presenting as acute liver failure has not been reported in literature.

Isolated hepatic tuberculosis, although rare, should be included as one of the differential diagnosis of liver mass in areas endemic for tuberculosis, especially when the patient presents with upper abdominal pain, fever and hepatomegaly. The best method of diagnosis is hepatic biopsy. Demonstration of caseating granulomas on histopathology is frequently considered as a diagnostic criterion. However, newer techniques like PCR can be helpful in patients negative for AFB.

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