Extra-pulmonary tuberculosis presenting as obstructive jaundice

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Abstract

Obstructive jaundice as the sole presentation of extra-pulmonary tuberculosis is a rare entity with only a few cases reported in the literature. Patients with this condition usually present with a protracted illness, jaundice and weight loss, which may be confused with hepatobiliary malignancies. A 45-year-old male alcoholic presented with a history of jaundice associated with low-grade intermittent fever and weight loss of one year duration. Diagnostic imaging techniques revealed multiple ill defined nonenhancing lesions in the liver with a 1.0 x 1.0 cm rounded lesion at the porta hepatis and intrahepatic biliary radical dilatation (IHBRD). A provisional diagnosis of Klatskin tumour (cholangio carcinoma involving the confluence of hepatic ducts) with liver secondaries was made. Subsequent histopathology showed caseating granulomatous lymphadenitis with epitheloid granulomas present periportally. He was treated successfully with a four-drug antituberculous regimen with complete resolution of symptoms.

Key words: tuberculosis, obstructive jaundice, cholestasis, hepatobiliary malignancy, Klatskin tumour.

Introduction

Tuberculosis is a disorder of protean manifestations. Although obstructive jaundice as the presentation of tuberculosis has been described in a few case reports, the diagnosis is often missed or delayed prior to surgery.

Case report

A 45-year-old male presented with a history of jaundice associated with low-grade intermittent fever and weight loss of one year duration. He had developed swelling of the feet, abdominal distension and abdominal pain during the last month. There was no history of haematemesis, malena, pruritus or clay-coloured stools, no haemoptysis or breathlessness. There was no past history of jaundice, blood transfusion, surgical interventions or treatment for pulmonary tuberculosis. He used to consume alcohol regularly but had stopped for one year. He denied any high-risk sexual behaviour.

Physical examination showed a middle aged adult male of normal build and nourishment with a body weight of 54 kg. He was pale, icteric with

bilateral pitting pedal oedema. A 1 x 1 cm firm lymph node was palpable in the left axilla. Vitals were stable. Cardiovascular, respiratory and central nervous system examinations were within normal limits. Abdominal examination showed a tender hepatomegaly 3 cm below the costal margin with a smooth surface. There was no shifting dullness.

Investigations revealed conjugated hyperbilirubinaemia with total bilirubin 10 mg% and direct 4.2 mg%. Alkaline phosphatase (1923 IU/L) and gammaglutamyl transferase (85 IU/L) levels were raised. AST and ALT levels were normal. Serum total protein was low (5.0gm %) with albumin of 2.4 gm%. Prothrombin time was prolonged by 6 seconds which was corrected by vitamin K administration. Blood urea and electrolytes were normal. Haemogram showed Hb 10 gm/dl with normal peripheral smear. Platelet count was reduced (1.69 lakhs/cumm) and ESR was elevated, 50 mm at the end of 1 hour (Wintrobe's method). Urine examination was positive for bile salts and bile pigments and urobilinogen levels were not raised. USG abdomen showed a liver of normal size and echo pattern with intrahepatic biliary radicle dilatation (IHBRD) in the left lobe, splenomegaly (18 cm), normal common bile duct and gallbladder with minimal free fluid. There were no focal lesions. Viral serologies for HIV, HBV and HCV were negative. Blood culture was sterile. Screening for pulmonary tuberculosis, sputum acid fast bacilli (AFB) for three consecutive days, chest X-ray and Mantoux (8 x 8 mm) were negative. Weil-felix, Paul-Bunnel and Brucella serologies were negative. Ascitic fluid analysis revealed a transudate with no cells and negative adenosine deaminase (ADA). Upper GI endoscopy showed no varices. A provisional diagnosis of alcoholic liver disease with biliary malignancy was made.

A contrast CT scan of the abdomen was subsequently done (Figure 1A, 1B) which showed multiple ill-defined nonenhancing lesions in the liver, the largest measuring 1.4 cm. There was a 1.0 x 1.0 cm rounded lesion (? necrotic lymph node) at the porta hepatis with IHBRD seen above this level preferentially in the left lobe. Multiple small para-aortic, periportal and mesenteric lymph nodes were present. Other findings were consistent with USG findings. Left axillary lymph node biopsy and liver biopsy were performed. Histopathology of the lymph node showed caseating granulomatous lymphadenitis. Liver biopsy was consistent with tuberculosis, with epitheloid granulomas present periportally. (Figure 2A, 2B). The patient was started on four-drug anti-tuberculous therapy (ATT). He became afebrile on ATT with regression of jaundice and constitutional symptoms. When last seen he had completed six months of ATT with complete resolution of jaundice. Repeat ultrasound showed normal liver with no IHBRD or focal lesions.

Discussion

Obstructive jaundice as the sole presentation of extra-pulmonary tuberculosis is a rare entity with only a few cases reported in the literature. The cause of jaundice in tuberculosis is multifactorial. Most case reports describe obstructive jaundice secondary to tuberculous involvement of the biliary tree, pancreas or ampulla of Vater and post-tubercular biliary stricture [1-6]. Jaundice can also occur secondary to compression of bile ducts by enlarged lymph nodes in the porta hepatis [7]. Biliary obstruction due to periportal tuberculous adenitis is a rare condition which is difficult to diagnose and treat. Patients with this condition usually present with a protracted illness, obstructive jaundice and weight loss which may be confused with hepatobiliary malignancies. The diagnosis is often missed or delayed and the patient

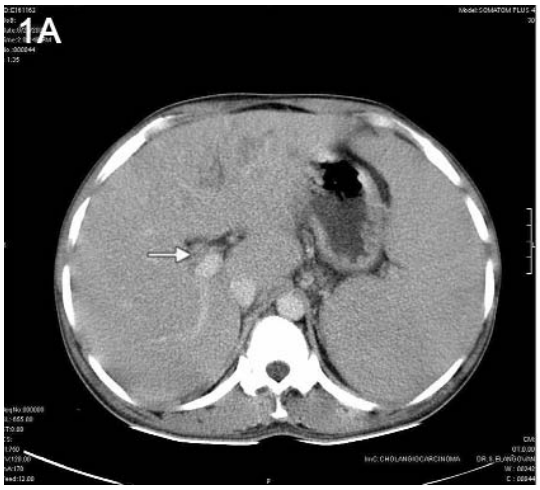

Figure 1A. CT film showing enlarged lymph node at porta hepatis (arrow)

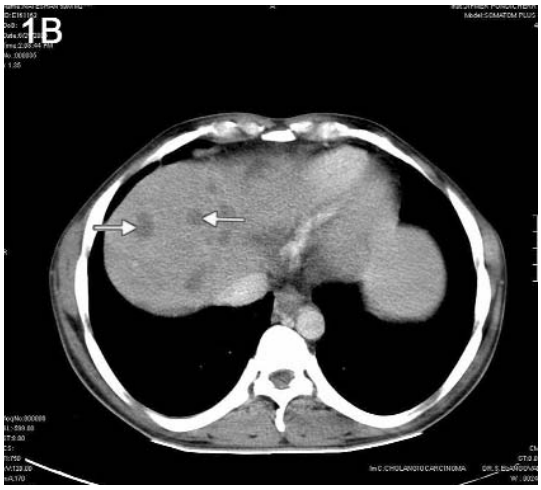

Figure 1B. CT film showing multiple focal lesions in the liver (arrow)

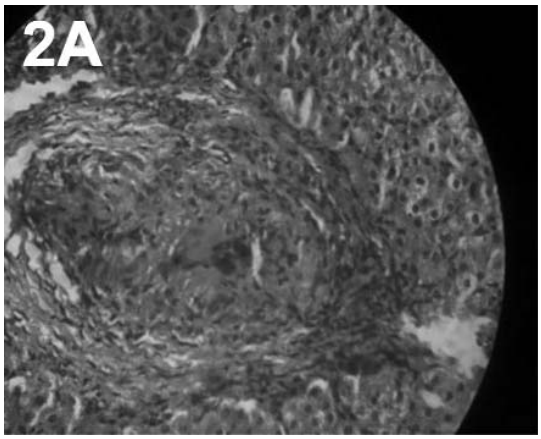

Figure 2A. Hepatic granuloma low power view

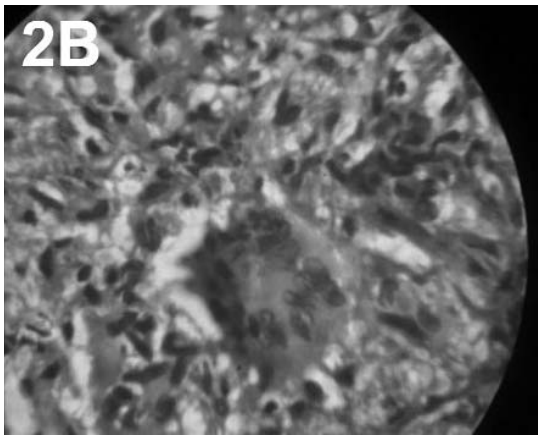

Figure 2B. Hepatic granuloma high power view

is subjected to unnecessary surgical procedures. Our patient presented with weight loss, deep jaundice and abdominal distension. Laboratory data showing cholestasis, IHBRD and multiple focal lesions in the liver with periportal lymphadenopathy suggested the possibility of hepatobiliary malignancy. In view of fever and peripheral lymphadenopathy infectious aetiologies such as viral hepatitis, tuberculosis and brucellosis were also considered in the differential diagnosis but the initial work-up for them was negative. The histopathology report suggesting tuberculosis came as a welcome surprise.

Local hepatic tuberculosis (primary hepatic) refers to involvement of the hepatobiliary tract by TB without any apparent involvement elsewhere or only with local lymph node or splenic involvement [8]. Here the tubercle bacillus is said to reach the liver through the portal vein from the gastrointestinal tract rather than the hepatic artery as in case of miliary tuberculosis [9]. The general belief is that it is a histopathological entity only. Treatment of hepatobiliary tuberculosis does not differ from that of other forms of tuberculosis but poses its own challenge as most of the antitubercular drugs are hepatotoxic.

Conclusions

This case is a reminder to physicians to be on the lookout for uncommon presentation of this common condition. The importance of a tissue diagnosis in all cases of obstructive jaundice is stressed.

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