CASE REPORT

ATYPICAL LIVER LESIONS: PRIMARY HEPATIC TUBERCULOSIS

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PJR April - June 2018; 28(2): 159-162

ABSTRACT

Hepatic tuberculosis is seen in 50-80% of patients with disseminated tuberculosis. However, isolated hepatic tuberculosis is rarely encountered in an immunocompetent patient, even in regions with high prevalence of tuberculosis. We report a case of a 43-year-old male who underwent triphasic computed tomography (CT) scan for an uncharacterized hypoechoic liver lesion found on ultrasound. CT scan revealed multiple ill-defined areas in segments VI and IV of the liver, which were reported as atypical liver lesions. Differentials of atypical hepatoma versus hepatic metastasis from an unknown primary were given. Subsequently, biopsy of the lesion was performed and histopathology report revealed chronic granulomatous inflammation with tuberculosis as a likely possibility. The patient was started on antituberculous therapy and responded to treatment. This case emphasizes the diagnostic difficulties of hepatic tuberculosis and the significance of considering this differential in patients with atypical hepatic lesions. It is vital for radiologists to identify unusual presentations of tuberculosis, which is an ever-growing problem in our part of the world.

Keywords: Primary hepatic tuberculosis, hepatic tuberculoma, triphasic CT, atypical hepatic lesions, hepatoma

Introduction

Tuberculosis (TB) is a growing problem worldwide; the calculated annual incidence in Pakistan is 878,000 cases, corresponding to a rate of 497 per 100,000 annually in the population.[1] Consequently, it is vital to recognize the more unusual presentations of tuberculosis.

Intra-abdominal TB has a high mortality and it is a difficult diagnosis to make, often requiring laparotomy. Hepatic involvement in TB is particularly rare; the exact incidence is unknown, likely due to under reporting and unawareness of the disease. Fewer than 100 cases have been reported in the literature, most of which are secondary and associated with military TB.[2] However, isolated hepatic tuberculosis is rarely encountered in an immunocompetent patient, even in regions with high prevalence of tuberculosis.[3]

Case Report

A 43-year-old male patient presented to our hospital with right upper quadrant dull pain since 3 months. There was no history of fever or jaundice. There were no other constitutional symptoms. The chest x-ray did not show any opacities or pleural effusion and ultrasonography of the abdomen revealed a hypoechoic lesion measuring approximately 9 x 5.5 cm in segment VI of the liver. The other abdominal viscera were normal and there was no free fluid seen. Biochemical profile was done which showed mildly raised alkaline phosphatase. Hematological profile was normal. Tumor markers like alpha fetoprotein and carcinoembryonic antigen were normal.

A triphasic CT scan of abdomen revealed mild hepatomegaly with multiple liver lesions within the liver and enlarged lymph nodes at porta hepatis (Fig. 1). The large, ill-defined irregular lesion in segment VI of liver was predominantly hypodense on

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Submitted 25 January 2018, Accepted 7 February 2018

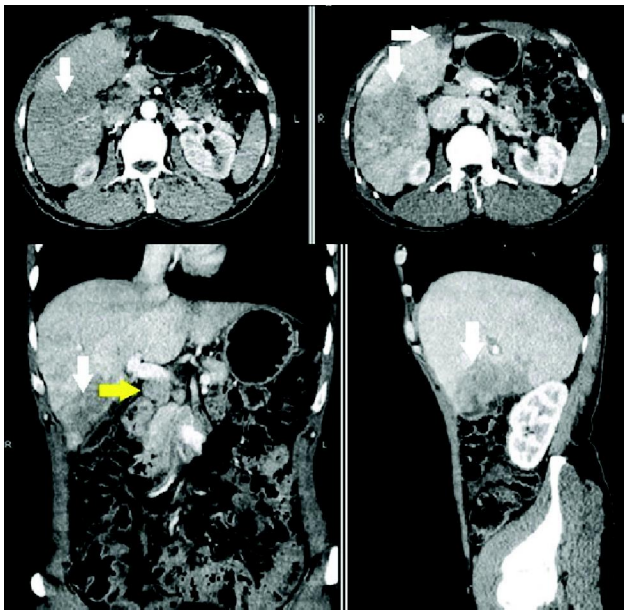

Figure 1: Irregular lesion (white arrows) in segment VI of the liver which was not hypervascular on the arterial phase, and appeared hypodense on the venous/delayed phases. Multiple enlarged lymph nodes at the porta hepatis (yellow arrow).

arterial, portal venous and delayed phase imaging. It measured 10 x 7 cm in anteroposterior and transverse dimensions respectively. Two smaller lesions with similar appearances were also identified in segment IV of the liver. The lesion along the falciform ligament measured 12 x 23 mm and another lesion lying superiorly just below the pericardium measured 8 x 9 mm. There were no signs of chronic liver disease.

Based on CT findings a differential diagnosis of atypical hepatoma versus hepatic metastasis from an unknown primary were given and ultrasound guided biopsy of the lesion in segment VI of the liver was subsequently performed. The histopathology specimen showed moderate periportal inflammation with histocytes and occasional multinucleated giant cells forming granulomas. These granulomas showed surrounding moderate chronic inflammation. Special stains were negative for acid-fast bacilli and fungal organisms. Tuberculosis was reported as a likely possibility based on the histopathological findings.

In keeping with clinical, histopathological and radiological findings patient was started on anti-tuberculous therapy (ATT). On follow up scans performed 2 and 6 months respectively after the start of ATT, the lesions in the liver and lymph nodes at the porta hepatis showed a significant reduction in size (Fig. 2 and Fig. 3).

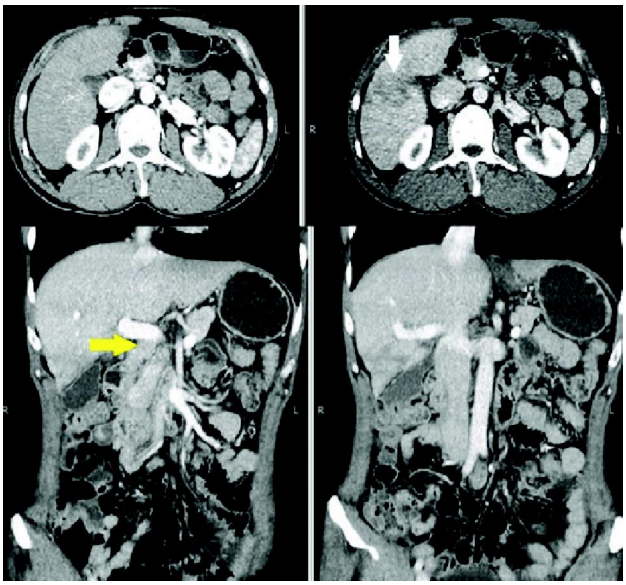

Figure 2: 2 months after initiation of anti-tuberculous therapy. The lesion in the liver (white arrow) and the lymph nodes at the porta hepatis (yellow arrow) have reduced in size.

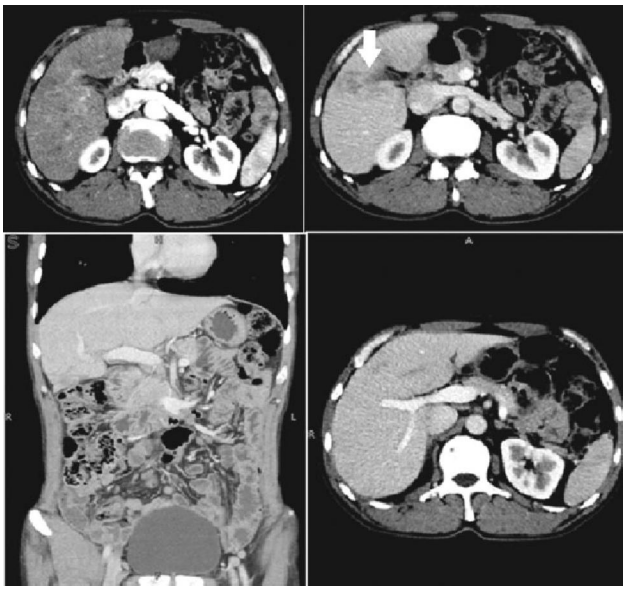

Figure 3: Follow-up scan after 6 months after anti-tuberculous therapy. The lesion in the liver and lymph nodes at the porta hepatis showed significant resolution.

Discussion

Tuberculous involvement of the liver is usually part of disseminated disease. The hepatic parenchyma can show a diffuse pattern of involvement in the form

of multiple small-sized military nodules.[4] Hepatic tuberculosis can be seen in around 50-80% of patients with disseminated tuberculosis.[5]

On the other hand, isolated hepatic tuberculosis is rarely encountered in clinical practice with only a few sporadic cases and short series available in the current literature.[4] The clinical presentation is non-specific and a high degree of suspicion is required to diagnose this entity which can be medically managed easily but if left untreated can lead to hepatic failure, increasing morbidity and ultimately mortality.[6] Criteria for diagnosis of primary hepatic tuberculosis include recent evidence of tubercular infection, demonstration of necrotizing granulomatous inflammation in liver and absence of old tubercular disease elsewhere.[7]

Although there is no standard classification system available for hepatic tuberculosis, Levine[8] classified hepatic involvement in tuberculosis into five patterns: miliary tuberculosis, concomitant hepatic and pulmonary disease, primary (isolated) hepatic tuberculosis, tubercular hepatic abscess, and tubercular cholangitis. The imaging manifestation of the tubercular hepatic disease can be wide ranging but can be broadly categorized into miliary pattern, nodular tuberculosis with serohepatic variant and tubercular cholangitis.[9] Hypoechoic nodules are usually seen at ultrasonography, though rarely the appearances may appear hyperechoic. CT findings usually reveal a round hypodense lesion with slight peripheral enhancement and, occasionally, areas of focal calcification.[10] MRI of hepatic TB shows a hypointense nodule with a hypointense rim on T1-weighted imaging. T2-weighted imaging shows a hypointense, isointense or hyperintense nodule with a less intense rim.[11] Multiple lesions of varying density may also be identified, indicating that there are lesions in different pathologic stages coexisting in hepatic TB, including TB granuloma, liquefaction necrosis, fibrosis or calcification.[12] Therefore, it is very difficult to diagnose primary hepatic TB using imaging modalities.

Clinical manifestations, laboratory findings, and imaging studies are varied and non-specific, so pathologic examination of liver lesions is essential to confirm hepatic tuberculosis. Therefore, percutaneous fine needle biopsy is an excellent diagnostic method and lead to the avoidance of a laparotomy.[13] The histologic findings often achieve the diagnosis accurately and in a review of 96 patients with a predominantly hepatic presentation of tuberculosis, the histological findings were granulomas (95.8%), caseation (83.3%), fatty changes (42%), portal fibrosis (20%) and acid-fast bacilli in association with granulomas (9%).[14] Low sensitivity of both acid-fast staining (from 0% to 45%) and culture (from 10% to 60%) mean diagnosis can still be difficult.[8] However, the use of polymerasechain reaction to directly detect the presence of Mycobacterium tuberculosis is increasing and may improve sensitivity rates.[15] This case illustrates the minimally invasive investigation, diagnosis, and treatment of primary hepatic tuberculosis.

The WHO protocol for the treatment of pulmonary TB (two months of rifampin, isoniazid, ethambutol, and pyrazinamide and then 4 months of rifampin and isoniazid) has been adopted for treatment of hepatic TB with good results. The appropriate duration for treatment of hepatic TB is a matter of conflict. Usually 6-12 months duration is appropriate for most of the patients.[16]

Conclusion

Primary focal involvement of liver in tuberculosis is a rare entity, but with the increasing worldwide incidence of TB, it is a diagnosis that must be considered, not only in patients considered at high risk but also in an immunocompetent hosts presenting with upper abdominal pain, fever and jaundice with hepatomegaly. It is difficult to diagnose on clinical basis and on imaging alone; therefore, a biopsy should be done to avoid unnecessary surgical procedures especially in tuberculosis endemic areas like Pakistan. Appropriate treatment initiated early can result in marked recovery whereas failure to recognize this entity can prove to be fatal. Our aim was to highlight the fact that in endemic countries like Pakistan, tuberculosis should be kept in mind as a differential of an atypical hepatic mass in an immunocompetent host.

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