Dear Editor,

The ability of multiphasic computed tomography (CT) and magnetic resonance (MR) studies in the diagnosis of liver tumours such as hepatocellular carcinoma and cholangiocarcinoma is well established. Nevertheless, potential mimics of malignant tumours abound. Hepatic tuberculosis presenting as hepatic mass is rare and preoperative diagnosis is usually difficult without histological evaluation, in part due to its myriad clinical manifestation and often non-specific imaging appearance. While there is abundant literature detailing the abscess-like imaging appearance of hepatic tuberculosis, the literature detailing the appearance of the macronodular form remains sparse. To the best of our knowledge, the multiphasic contrast enhancement pattern of macronodular tuberculosis has not been previously described. We hereby describe such a case of hepatic tuberculosis and show how the imaging appearance can mimic intrahepatic cholangiocarcinoma. We also describe the multiphasic contrast enhanced MR appearance.

Case Report

A 71-year-old hypertensive male presented with epigastric pain for a duration of 2 weeks. Clinical examination, chest radiography, routine blood tests and hepatobiliary tumour markers were within normal limits. The viral markers for hepatitis B and hepatitis C were negative. Ultrasound and CT abdomen performed revealed a mass lesion in segment 4A of liver. MR scan eventually was performed with images acquired in pre-contrast enhanced, arterial, venous and delayed phases. An irregular mass was detected in segment 4A of liver measuring 4.5 cm x 3.9 cm x 3.0 cm. The lesion was hypointense to the liver pre-contrast, showed slight peripheral enhancement in the arterial phase which was more pronounced in the portal venous phase and showed delayed central contrast enhancement, becoming almost isointense to the liver (Figs. 1a, 1b, 1c). The findings were similar in the CT scan but more conspicuous in MR. In addition, the mass was slightly hyperintense on T2W fat suppressed images. Furthermore, area of capsular retraction is also noted (Fig. 1d). Evaluation with positron emission tomography (PET) showed the mass to be hypermetabolic with maximum standard uptake value (SUVmax) of 6 (Fig. 2). In view of the imaging findings, the working diagnosis was that of an intrahepatic cholangiocarcinoma and the patient subsequently underwent a left hemihepatectomy. Histopathology showed multiple epitheloid granulomas surrounded by fibrosis. Some of these granulomas showed central caseation necrosis. A clinical diagnosis of hepatic tuberculosis was made based on the histology findings. The CT scan performed did not show any other focus of tuberculosis in the abdomen. While special stains for acid-fast bacilli (AFB) organisms were negative, it was felt that the chances of false negative AFB stains were high in view of the histology findings. The patient was started on an anti-tuberculous regime and made an uneventful recovery.

Discussion

Hepatic tuberculosis is one of many granulomatous liver diseases. Tuberculous involvement of the liver is classified as miliary form or localised form. The latter is further subdivided into nodular tuberculosis (tuberculous hepatic abscess and tuberculosis) and tuberculous cholangitis. Focal masses of more than 2 cm are referred to as macronodular tuberculosis.1

Miliary tuberculosis of the liver is the most common type with imaging findings varying from hepatomegaly to multiple scattered focal liver lesions resembling metastases. Tuberculous cholangitis is an extremely rare cause of biliary strictures.

Macronodular tuberculosis is uncommon and often presents as non-specific hepatic masses which may resemble metastases or abscesses.2 The lesions are hypodense on CT and may show peripheral enhancement. At MR, these lesions are hypointense on T1 weighted sequence (T1W) and hyperintense on T2 weighted sequence (T2W). Eventually these lesions tend to calcify.3 This case illustrates a rare and potential pitfall, where an infective process can mimic a liver tumour. In the illustrated, the enhancement pattern of initial peripheral enhancement with delayed central enhancement and the associated capsular retraction led us to the initial working diagnosis of an intrahepatic cholangiocarcinoma. This enhancement pattern and capsular retraction is well-described in intrahepatic cholangiocarcinoma and is believed to reflect the abundant fibrous stroma in cholangiocarcinoma.4,5 We therefore postulate that the similarity in the imaging features between intrahepatic cholangiocarcinoma and macro-nodular tuberculosis in our illustrated case is attributable to the large amount of fibrous tissue seen on histology, surrounding the granulomas.

The overlap between macronodular tuberculosis and malignancy is also seen with PET imaging. This is due to the varied peak standardised uptake values of macronodular
tuberculosis that can range from 2.2 to 21.0,6 which is well within the range to mimic a hypermetabolic malignancy. On microbiology studies, the positivity of AFB stains has been shown to be low in macronodular tuberculosis7 and the presence of caseous necrosis on histology is specific for tuberculosis. This forms the basis of our clinical diagnosis and treatment.

To conclude, we describe the multiphasic imaging appearance of macronodular tuberculosis on MR. This case illustrates the challenge in the diagnosis of macronodular tuberculosis on imaging and demonstrates the overlap in appearance with intrahepatic cholangiocarcinoma. There should be an increased awareness of this potential tumour mimic, particularly in endemic areas, and preoperative biopsy and perhaps patient care report (PCR) may play an important role, to avert unnecessary surgery.

REFERENCES


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