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Isolated hepatic tuberculosis: A rare cause of hepatic mass lesions

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ABSTRACT

Hepatic tuberculosis usually accompanies pulmonary and extrapulmonary tuberculosis. Although isolated hepatic tuberculosis is a very rare condition, it should be considered in the differential diagnosis of a hepatic mass. Here, we report a 42-year-old woman presenting with weight loss, fever, night sweats, and a hepatic mass on the abdominal ultrasonography and magnetic resonance imaging (MRI). Ultrasonography-guided percutaneous needle biopsy demonstrated a caseating granuloma with epithelioid histiocytes and giant cells compatible with the diagnosis of tuberculosis. The patient was treated with four anti-tuberculous drugs for 1 year. She recovered clinically, and her post-treatment abdominal MRI was normal.

Keywords: Isolated hepatic tuberculosis, hepatic mass, differential diagnosis

Case Report

INTRODUCTION

Tuberculosis (TBC) is still an important health issue with considerable morbidity and mortality in both developing and underdeveloped countries. TBC can affect the liver, and hepatic involvement is observed in about 50%-80% of cases of disseminated TBC, although isolated hepatobiliary TBC is less common (1). Hepatobiliary TBC can mimic liver tumors, including hilar tumors causing biliary obstruction, periampullary tumors, cholangiocarcinomas, and liver abscess, or may present with various forms of systemic diseases, like fever of unknown origin (1-3). We here present an isolated hepatic TBC case recovered with anti-TBC treatment.

CASE PRESENTATION

A 42-year-old woman (who approved publication of this paper regarding her disease) from Georgia was admitted to our hospital with a history of anorexia, fever, night sweats, fatigue, and weight loss of 25 kg within the past 6 months. Before her admission to the hospital, she had been treated with ceftriaxone for 13 days without further evaluation. Except for right quadrant tenderness, the physical examination was normal. Liver and spleen were not palpable, and there was no palpable lymphadenopathy. Upon laboratory examinations, hemoglobin level was 8.6 g/dL, hematocrit was 28.8%, white blood cell count was 10,600/mm^3, platelet count was 384,000/mm^3, and erythrocyte sedimentation rate was 96 mm/h. Blood alanine aminotransferase (24 U/L), aspartate aminotransferase (31 U/L), and gamma-glutamyl transpeptidase levels (66 U/L) were within normal limits, but alkaline phosphatase (155 U/L, normal limits=35-104 U/L) was slightly elevated. Brucella agglutination (Wright) test, Brucella IgM and IgG antibodies, anti-human immunodeficiency virus antibody, and hepatitis markers were all negative. Level of the cancer biomarkers carcinoembryonic antigen (CEA), carbohydrate antigen 19-9 (CA 19-9), and alpha-fetoprotein were also considered as normal.

Purified protein derivative (PPD) and QuantiFERON (QuantiFERON TB Gold, Quigen, Hilden, Germany) tests were not performed, since chest x-ray and computerized tomography of the thorax did not reveal any ab-

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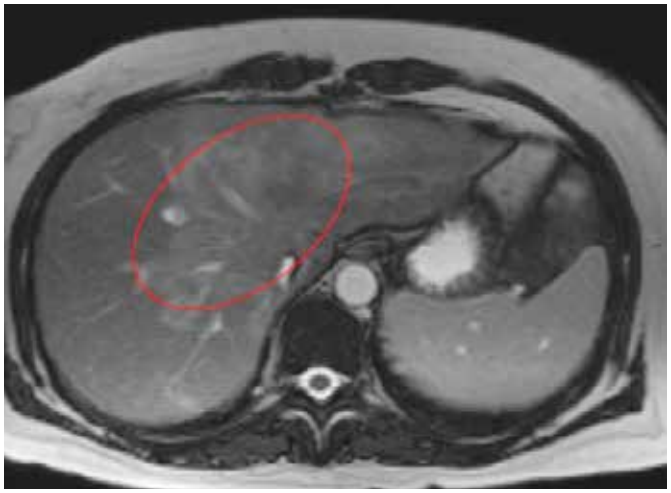

Figure 1. Before-treatment magnetic resonance image: tumor-like hyperintense lesion at T2 infiltrating both lobes of the liver.

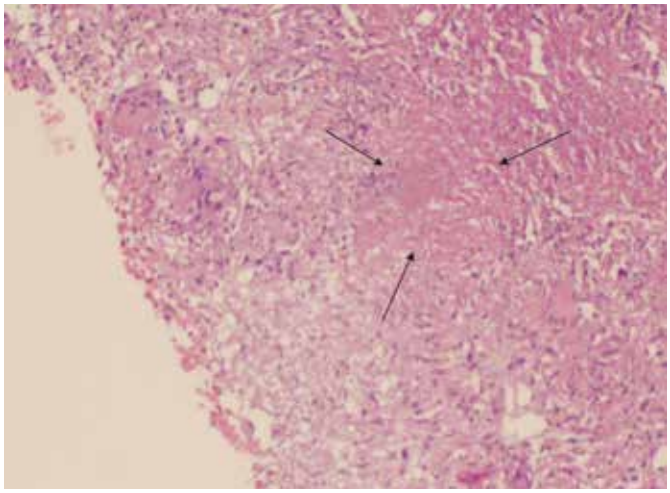

Figure 2. Granuloma consisting of central necrosis and Langhans giant cell among the epithelioid cells (H&E X100).

normal finding. However, a tumor-like mass lesion infiltrating both lobes of the liver was detected by magnetic resonance imaging (MRI) (Figure 1).

An ultrasonography-guided percutaneous liver biopsy was performed, and histopathological examination of the specimen showed caseating granulomas with epithelioid histiocytes and giant cells (Figure 2). Acid-fast bacilli (AFB) could not be detected on smear examination, and polymerase chain reaction (PCR) for *Mycobacterium tuberculosis* was negative in the biopsy specimen.

Despite negative AFB and PCR, anti-TBC treatment was started based on the presence of the caseating granuloma, which is characteristic of TBC. The patient was treated with a combination of rifampicin, isoniazid, ethambutol, and pyrazinamide for 1 year, with no adverse effects were reported, and totally recovered clinically with a normal end-of-treatment abdominal MRI (Figure 3).

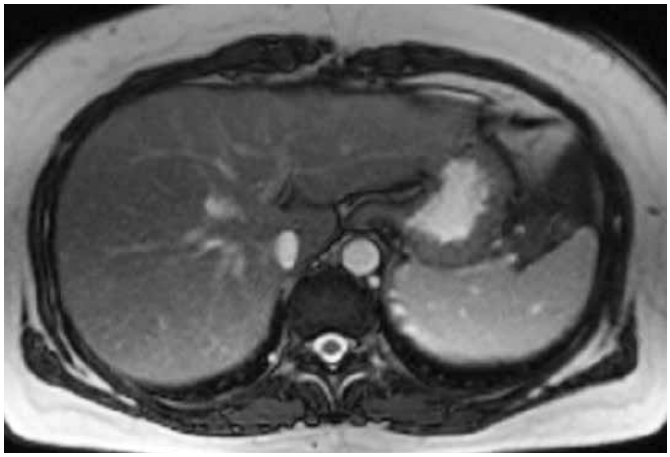

Figure 3. After-treatment magnetic resonance image: normal appearance.

DISCUSSION

Hepatic involvement of TBC in the absence of miliary or pulmonary/extrapulmonary TBC is very rare and constitutes less than 1% of all cases (4). In 1995, Senturk et al. (5) reported two cases of primary macronodular hepatosplenic tuberculosis with ultrasonography, computerized tomography, and magnetic resonance imaging findings, confirmed by biopsy. Hepatic TBC is classified into three categories: miliary, granulomatous, and localized hepatic form (6). The pathophysiology of localized hepatic TBC is similar to other forms of extrapulmonary TBC, which results from hematogenous dissemination of the infection through the hepatic artery, the portal vein, or lymphatics (7-9).

Unfortunately, the clinical features of hepatic TBC are non-specific. The most common presenting symptoms were reported to be weight loss (64%), loss of appetite (64%), abdominal pain (57.1%), fever (50%), and jaundice (42.3%) (3). Jaundice may occur as a result of lymphadenopathy at the porta hepatis, obstruction in the common bile duct, or portal inflammatory stricture. Hepatomegaly and splenomegaly are found in 95% and 18%-55% of the cases, respectively (2). Liver enzymes are elevated in only 25% of patients. In the present case, there was only a mild elevation in ALP and no abnormality in AST and ALT levels, and no hepatomegaly and splenomegaly were detected.

Radiological investigations in cases of hepatic TBC usually mimic other common diseases. Ultrasonography may show hypoechoic or, rarely, hyperechoic nodular lesions or a solid hepatic mass. Computerized tomography findings demonstrate a hypodense mass or a cystic lesion (4,5,9,10). Our patient had a tumor-like mass lesion expanding to both lobes of the liver on the abdominal ultrasonography and MRI.

Histopathological examination is needed for the exact diagnosis of hepatic TBC (11). Hepatic granulomas may also be observed in other infectious diseases, such as brucellosis, in-

fectious mononucleosis, chronic hepatitis, and fungal infections, or non-infectious diseases, like early-stage primary biliary cirrhosis, sarcoidosis, Hodgkin's disease, Crohn's disease, drug hypersensitivity, and extra-hepatic biliary obstruction (2,4). Epithelioid granuloma can be demonstrated in liver TBC in 80%-100% of cases, caseating necrosis in 30%-83%, and AFB on smear examination in 0%-59% of the cases (7). In our case, we could not detect AFB on direct examination. Detection of mycobacterial DNA on tissue specimen by PCR has 88% sensitivity and 100% specificity. In one study, PCR was positive in 57% of hepatic granulomas (4). In our case, PCR test was also negative on the tissue specimen.

Hepatic TBC treatment includes the standard four-drug regimen with rifampicin, isoniazid, ethambutol and pyrazinamide—similar to any other extrapulmonary tuberculosis (4,7). Our patient was successfully treated with these anti-TBC drugs for 1 year, and no adverse effect was observed during the treatment period. Control MRI investigation after treatment was normal.

In conclusion, isolated hepatic TBC can mimic other diseases presenting with a mass in the liver, but it should especially be considered in the differential diagnosis of patients coming from endemic areas for TBC.

Ethics Committee Approval: N/A.

Informed Consent: Written informed consent was obtained from patient who participated in this study.

Peer-review: Externally peer-reviewed.

Author contributions: Supervision - B.Ç., N.T., G.B.; Data Collection&/or Processing - N.T.K.; Literature Search - N.T.K.; Writing - N.T.K.; Critical Reviews - N.T., G.B.

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