Journal of Microbiology, Immunology and Infection (2016) 49, 455—458

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CASE REPORT

Isolated tuberculous liver abscess in an immunocompetent adult patient: A case report and literature review

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Received 26 January 2013; received in revised form 20 August 2013; accepted 11 September 2013
Available online 11 November 2013

KEYWORDS
Immunocompetent;
Liver abscess;
Mycobacterium tuberculosis

Tuberculous liver abscess is a rare disease entity even in endemic areas of *Mycobacterium tuberculosis*. It is usually accompanied by pulmonary tuberculosis or enteric tuberculosis. Further, an isolated tuberculous liver abscess is extremely rare. The disease is diagnosed by laparotomy or postmortem autopsy in most cases, and some authors adopted a 9-month antituberculosis regimen. We herein report a case of an isolated tuberculous liver abscess that initially manifested as persistent fever and general malaise, which was diagnosed by liver biopsy and treated successfully with a 6-month antituberculosis regimen and percutaneous abscess drainage.
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Introduction

Hepatic tuberculosis (TB) is usually accompanied by pulmonary TB or tuberculous enterocolitis. An isolated tuberculous liver abscess is extremely rare. Less than 25 cases

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http://dx.doi.org/10.1016/j.jmii.2013.09.003

were reported in the literature prior to 2003.[1] Hepatic TB is typically associated with the formation of granulomas that may heal with focal fibrosis and calcification or coalesce to form tuberculomas. If the lesion is large enough, necrosis may occur, forming an abscess. Here, we report a case of an isolated tuberculous liver abscess and review the related literature.

Case report

A 76-year-old man with a history of benign prostate hyperplasia and gout visited our infection outpatient department with the chief complaint of intermittent fever for 2 months. Body weight loss (5 kg/mo), abdominal fullness, poor appetite, and general malaise were also observed. No cough, abdominal pain, or dysuria was complained. Physical examination revealed mild right hypochondrial knocking pain. Laboratory data showed leukocytosis, an elevated C-reactive protein (CRP) level, a mildly elevated erythrocyte sedimentation rate (ESR), and hyponatremia. Blood parameters were as follows: white blood cell (WBC) count, 21,300/μL; segment, 88%; hemoglobin, 12.4 g/dL; platelet count, 459,000/μL; CRP, 11.5 mg/dL; blood urea nitrogen, 10 mg/dL; creatinine, 0.87 mg/dL; Na, 133 mEq/L; K, 4.16 mEq/L; glutamate pyruvate transaminase, 19 IU/L; alkaline phosphatase, 70 IU/L; r-glutamyl transferase, 39 U/L; total bilirubin, 0.7 mg/dL; lactate dehydrogenase (LDH), 73 IU/L; uric acid, 5.1 mg/dL; and ESR, 33. Fever of unknown origin was initially suspected. Although sputum specimens were difficult to obtain, TB cultures and acid-fast staining were performed for three sets. Acid-fast staining was negative; further, serum cortisol levels, thyroid function, tumor markers, and routine stool tests were all within normal limits. No red blood cells or WBCs were detected in the stool. Antinuclear antibody testing and urinalysis were also within normal limits. Chest X-ray examination (Fig. 1A) revealed mild blunting of the right costophrenic angle. Because the fever persisted, the patient was admitted for further evaluation. Abdominal computed tomography (CT) revealed a low-density lesion approximately 4 cm in diameter at the S4 segment, which was suspected to be a liver abscess (Fig. 1B). An empirical antibiotic therapy comprising ceftriaxone and metronidazole was administered. Percutaneous abscess drainage was carried out, and pus cultures were performed to detect the presence of bacteria or fungus. The amebiasis antibody test was negative. Abscess pus and blood cultures yielded no subsequent growth. His fever did not subside after 7 days of treatment, and only a small amount of pus was drained from the tube. The percutaneous drainage was repeated, and the tube was not removed until only a small amount of drainage was observed 2 weeks later. The low-grade fever persisted with right-upper-quadrant abdominal pain. Therefore, we scheduled a liver biopsy for a definitive diagnosis. Triphase CT was performed to exclude hepatocellular carcinoma. A moderate amount of pleural effusion was recorded at this time (1 month after admission), and thoracentesis was performed for pleural effusion analysis. Exudative pleural effusion was noted with a low adenosine deaminase (ADA) level, and cytology and TB polymerase chain reaction (PCR) results were both negative [pleural effusion study: WBC count, 2228/μL; red blood cell count, 1277/μL; neutrophil, 9%; lymphocyte, 84%; ADA, 19 U/L; LDH, 108 IU/L (serum LDH: 90 IU/L); protein, 4.5 g/dL (serum protein: 7 g/dL); and Glu: 128 mg/dL]. Abdominal sonography was performed prior to sonography-guided biopsy. The liver biopsy specimen showed Langerhans giant cells and caseous necrosis (Fig. 2), but exhibited negative acid-fast, Gram, periodic acid-Schiff, and Gomori methenamine silver staining. Anti-TB medications including isoniazid, rifampin, ethambutol, and pyrazinamide were administered accordingly. The fever subsided within 1 week, and his appetite restored gradually. Sputum, pleural

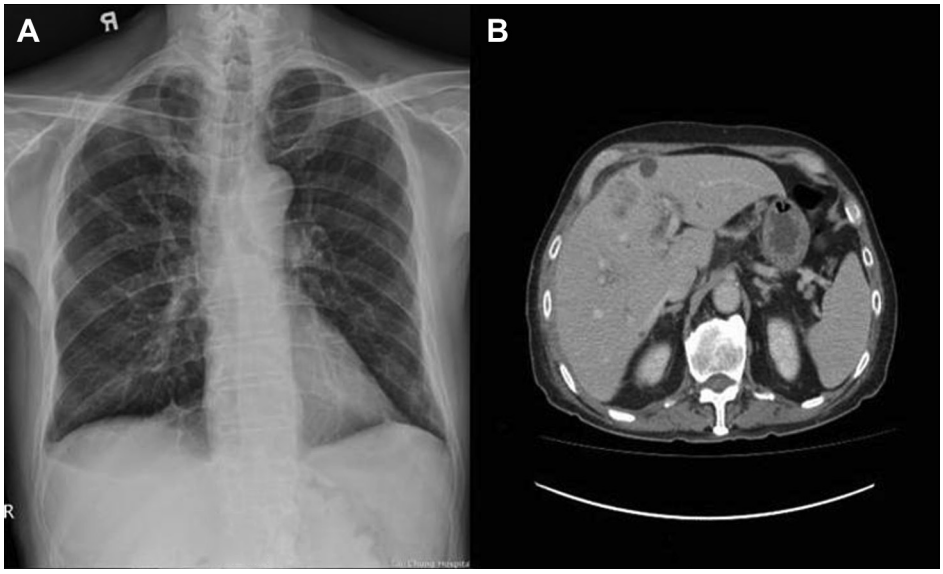

Figure 1. (A) At the first visit to the outpatient department, mild blunting of the right costophrenic angle is noted on the chest X-ray image. (B) A computed tomography scan shows a heterogeneous low-density lesion at the S4 segment and an adjacent cystic lesion.

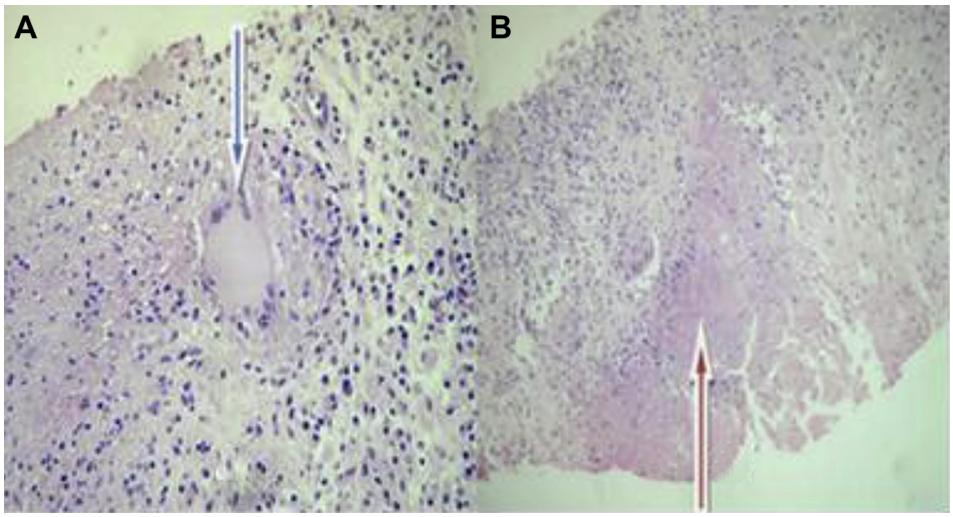

Figure 2. (A) Langerhans giant cells are observed in the liver biopsy specimen (blue arrow) (H&E stain, 200×, photograph by Dr Lai-Ching Wong). (B) An area of caseous necrosis was present in the liver biopsy specimen (red arrow; H&E stain, 200×, photograph by Dr Lai-Ching Wong). H&E = hematoxylin and eosin.

fluid, and tissue *Mycobacterium tuberculosis* biopsy cultures were eventually negative. Because no abdominal symptoms (abdominal pain or diarrhea) and sign (abdominal tenderness) were reported and routine stool examination was normal, TB enterocolitis was excluded. The patient completed the 6-month course of medication (2 months with isoniazid, rifampin, ethambutol, and pyrazinamide, followed by 4 months with isoniazid, rifampin, and ethambutol) without severe adverse events. The only side effect was elevated uric acid levels, which declined after pyrazinamide was discontinued. The follow-up abdominal CT scan revealed that the lesion at the S4 segment disappeared. WBC count, CRP, and ESR all returned to normal levels. The patient's appetite and body weight returned to that exhibited previously. Follow-up was continued for 9 months, and the patient is still healthy.

Discussion

By 1858, Bristowe had reported 12 cases of solitary tubercles of the liver with cavity formation among 167 instances of tuberculous ulceration of the intestine.[2] However, hepatic TB was reported sporadically and infrequently thereafter. A study from South Africa showed that liver TB accounts for only 1.2% of all TB cases.[3] Isolated hepatic TB accounted for 0.3% of new TB cases.[4] In another study, tuberculous liver abscess occurred only in 0.34% of patients with hepatic TB.[5] Tuberculous liver abscesses are more likely to occur in children and specific ethnic groups including African Americans and Igorots.[2] A study in 1990 indicated that only 13 cases of isolated liver abscess had been described in the English literature[6] and less than 25 cases of isolated liver abscess had been documented prior to 2003.[1]

The most frequent symptoms of patients with hepatic TB are fever, weight loss, and abdominal pain.[3,7] Right hypochondrial tenderness, hepatomegaly, and splenomegaly are the most common findings.[3] Laboratory data demonstrate leukocytosis and anemia,[1,7] and liver function and bilirubin level are within normal limits; however, alkaline phosphatase level is mostly elevated.[1,3] Acid-fast staining and TB cultures were negative in most cases of tuberculous liver abscess.[8,9] The findings of our patient were comparable to these results. The only difference is the normal alkaline phosphatase level in our patient.

Three forms of hepatic TB have been described. The first is diffuse hepatic involvement, seen along with pulmonary or miliary TB. The second form is diffuse hepatic infiltration without pulmonary involvement, which was previously called primary miliary TB of the liver. The third is focal liver tuberculoma or abscess.[3]

Tubercle bacilli reach the liver via hematogenous dissemination; the point of entry in the case of miliary hepatic TB is the hepatic artery, whereas that for focal liver TB is the portal vein.[2,3] Because no symptoms and signs of enterocolitis were observed during the whole disease course, colonoscopy was not arranged for the patient.

Abdominal sonography reveals a hypoechoic lesion in most cases of TB liver abscess. It also happened in the present case. However, a hyperechoic lesion was reported in one case.[1] CT usually demonstrates a low-attenuation lesion with or without ring enhancement,[6] and septated (i.e., honeycomb-like) abscesses can be visualized by ultrasound or CT.[4,7]

Isolated hepatic TB is difficult to diagnose. Invasive procedure is always needed. Exploratory laparotomy or autopsy led to the final diagnosis in the past literature.[1] Now, biopsy is increasingly being adopted.[5,7] Although biopsy is less invasive, false-negative results are possible if the necrotic tissue is obtained instead of the margin of the lesion. Surgery may then be required to make a diagnosis if the biopsy of the abscess wall is not diagnostic. Meanwhile, differential diagnosis of abscess from the necrotic tumor is very important. TB can be diagnosed by culture, PCR, or pathology. In the present case, the diagnosis was made according to the pathology findings of the liver biopsy. Although caseous necrosis does not represent TB

exclusively, the treatment outcome of anti-TB regimen confirms the diagnosis indirectly. The lack of a positive TB culture and PCR results did not influence the final diagnosis. A treatment course lasting 9 months was adopted in some cases.[3,10] In the present patient, a 6-month standard antituberculous regimen with percutaneous abscess drainage led to a successful result.

No proven TB culture or PCR results were found in the present case, but meaningful pathologic results were obtained. Sputum TB culture, sputum acid-fast stain, pleural acid-fast stain, TB culture, and TB PCR were all negative. Bronchoscopic biopsy was not carried out because of the lack of thoracic symptoms. Further, pleural biopsy was not arranged because TB pleurisy was not suspected due to low ADA levels of pleural effusion. No associated chest or alimentary tract symptoms were found. No diabetes mellitus, malignancy history, or immunosuppressive drug use was traced. Although the patient did not provide informed consent to perform HIV testing, which means that HIV infection cannot be excluded objectively, he presented no risk factors for HIV or signs of opportunistic infections. Nine months after completing the treatment, the WBC count is still within the normal limit and no leukopenia or lymphopenia has been found.

Isolated hepatic TB is extremely rare. High awareness is required to make a successful diagnosis. Biopsy is a better initial choice than laparotomy because it is less invasive. Percutaneous abscess drainage is a good adjunct for treatment.[6,9] Although universal consensus has not been reached on the treatment duration of TB liver abscess, in our experience, 6 months of standard antituberculous regimen is feasible when accompanied by drainage. This approach may avoid the need for laparotomy, open drainage, and hepatic resection.

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