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Detection of *Mycobacterium tuberculosis* in a patient with suspected cystic echinococcosis: a case report

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Abstract

Background Tuberculosis is a global health issue affecting millions of people worldwide. While pulmonary tuberculosis is common, hepatic tuberculosis is rare and accounts for less than 1% of cases. Diagnosis is challenging owing to nonspecific symptoms and its ability to mimic other hepatic diseases.

Case presentation This case report describes a 62-year-old male healthcare worker, originally from the Philippines and of Filipino ethnicity, who was initially suspected to have cystic echinococcosis, an epidemiologically uncommon zoonotic disease in both Austria and the Philippines. Echinococcosis was excluded by ultrasound and serology. Several other differential diagnoses, including pyogenic and amoebic liver abscesses, brucellosis, hepatocellular carcinoma, and hepatic metastases of other malignancies, were also ruled out. Ultimately, a biopsy of the liver lesion was performed, and the tissue tested positive for *Mycobacterium tuberculosis* complex by polymerase chain reaction. The patient was successfully treated with a standard 6-month antitubercular combination therapy.

Conclusion This case highlights the potential for tuberculosis to affect any organ system, including the liver, particularly in patients from high-burden regions.

Keywords *Mycobacterium tuberculosis*, Hepatic diseases, Liver cysts, Case report

Background

Tuberculosis (TB) remains a global health concern, affecting millions of people worldwide. The World Health Organization (WHO) estimates that approximately 25% of the global population is infected with *Mycobacterium tuberculosis* [1]. While pulmonary tuberculosis is the most common form (about 85%), extrapulmonary tuberculosis accounts for the remaining 15% [2]. The anatomical sites most frequently affected by extrapulmonary TB include lymph nodes, pleura, bones, and the genitourinary system [3]. However, hepatic involvement is uncommon and represents less than 1% of all TB cases [4]. Over the past 30 years, reports of abdominal tuberculosis have steadily increased, particularly in regions with a high prevalence of human immunodeficiency virus (HIV) [5]. Clinical and radiological findings are often nonspecific

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and may mimic other hepatic diseases such as malignancies, amoebiasis, or echinococcosis, potentially leading to diagnostic delays and adverse outcomes due to inappropriate treatment [4]. Hepatic tuberculosis may also be associated with elevated tumor markers, further complicating the diagnostic process [6, 7]. Isolated hepatic tuberculosis, as presented in this case report, is exceedingly rare.

Case presentation

A previously healthy 62-year-old male healthcare worker of Filipino ethnicity, born in the Philippines and living in Austria, with more than 40 pack-years of smoking, presented to the interdisciplinary outpatient clinic for echinococcosis at the University Hospital of the Medical University of Vienna (Austria). He reported intermittent, nonspecific abdominal discomfort in the right upper quadrant, which was consistently relieved by bowel movements. The symptoms had progressed slowly over the course of 3 months. The patient denied experiencing fever, cough, nausea, vomiting, diarrhea, notable night sweats, weight loss, or any other constitutional symptoms. On physical examination, moderate tenderness was noted in the right upper quadrant, without signs of hepatomegaly, splenomegaly, jaundice, or ascites. The patient had been living in Austria for over 10 years and was employed as a community nurse. Relevant risk factors included long-term cigarette smoking. However, he denied alcohol consumption or drug use. His last travel abroad was 6 months prior, during a 4-week visit to friends and family in the Philippines. He reported no known contact with individuals diagnosed with tuberculosis and no close contact with animals or pets. Laboratory testing revealed elevated tumor markers with alpha-fetoprotein (AFP) at 6.8 IU/mL (reference range: 0–5.8 IU/mL), carbohydrate antigen (CA) 19–9 at 62.8 kU/L (0–27.0 kU/L), and carcinoembryonic antigen (CEA) at 6.2 µg/L (0–3.8 µg/L). Gamma-glutamyl transferase (GGT) was moderately elevated at 92 U/L (0–60 U/L). Other liver function tests, complete blood count, renal function, and inflammatory markers were within normal limits. Abdominal ultrasound revealed borderline hepatomegaly measuring up to 24 cm and an indistinct, hypoechoic lesion in the left hepatic lobe. According to the 2010 WHO Informal Working Group on Echinococcosis (WHO-IWGE) expert consensus, the lesion was not characteristic of cystic echinococcosis [8]. Although ultrasound remains the cornerstone of cystic echinococcosis (CE) diagnosis, further imaging with computed tomography (CT) or magnetic resonance imaging (MRI) may be indicated in cases with inconclusive findings. Abdominal CT revealed a total of five hypodense, calcified hepatic lesions, the largest measuring up to 4.4 cm, without contrast enhancement in either the arterial or late venous phases (Fig. 1A). Liver MRI showed the lesions to be hypointense on T1-weighted images and hyperintense on T2-weighted images. The lesions exhibited minimal peripheral contrast enhancement without contrast washout but showed marked diffusion restriction, raising suspicion for metastatic liver lesions or primary hepatocellular carcinoma. In addition, intrahepatic cholestasis was noted (Fig. 1B). No intra-abdominal lymphadenopathy or evidence of other abdominal involvement was detected.

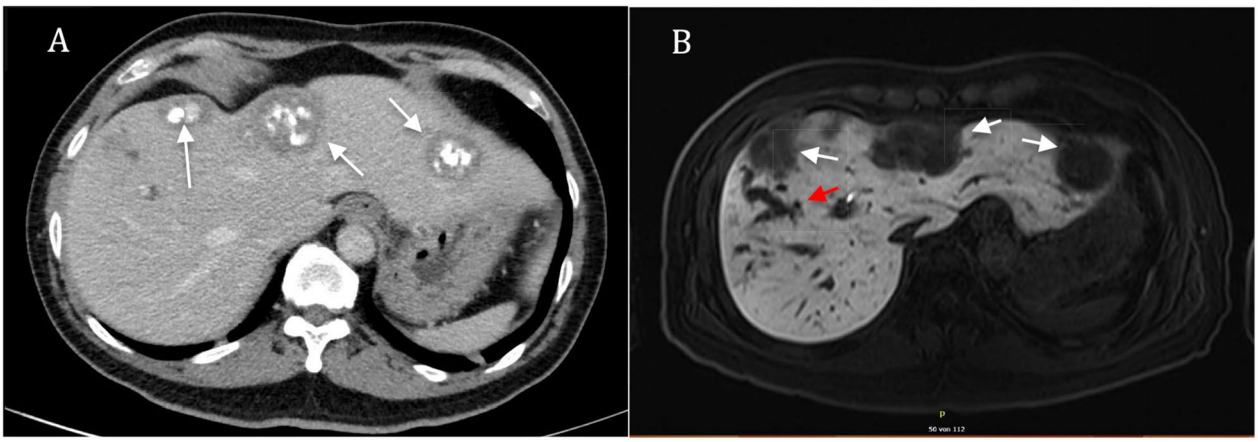

Fig. 1 Abdominal computed tomography (**A**) and abdominal magnetic resonance imaging, axial T1-weighted, fat-suppressed with contrast in hepatobiliary-phase (**B**) of a 62-year-old patient with abdominal discomfort and elevated tumor markers. **A** shows multiple hypodense lesions with central calcification in both lobes (indicated by white arrows). No contrast enhancement raising concern for metastatic liver lesions or primary hepatocellular carcinoma. **B** shows multiple hypointense lesions up to 4.2 cm × 3.8 cm, further raising concern for malignant pathology (indicated by white arrows). Diffuse wedge-shaped hypervascularisation during arterial phase in liver segments VIII and V with dilated intrahepatic bile ducts (indicated by red arrow)

However, no typical radiomorphological correlate of an *Echinococcus granulosus* cyst was observed [9]. In addition, enzyme-linked immunosorbent assay (ELISA) testing for antibodies against *E. granulosus* and *Echinococcus multilocularis* was negative, providing no serological evidence of echinococcosis. Initial polymerase chain reaction (PCR) testing for parasites in stool was positive for *Entamoeba histolytica*, but ELISA testing for antibodies against *Entamoeba histolytica*, *Fasciola hepatica*, and *Schistosoma* spp. was negative and therefore deemed unlikely. To exclude co-existing conditions, serologic testing for human immunodeficiency virus (HIV) and viral hepatitis types A, B, and C was performed, all of which returned negative results. Owing to the unclear etiology of the hepatic lesions, and after echinococcosis was ruled out through serological and radiological findings, a CT-guided core needle biopsy of one hepatic lesion was performed. Histopathological examination of formalin-fixed, paraffin-embedded tissue revealed areas of amorphous necrosis surrounded by granulomatous inflammation with lymphocytic infiltration (Fig. 2). No histological evidence of malignancy was found. Stains for fungi, acid-fast bacilli, and amoebic trophozoites were negative. However, molecular analysis using PCR (GeneProof TB-PCR Kit) confirmed the presence of *Mycobacterium tuberculosis* complex.

Genotypic testing for rifampicin resistance using PCR was negative. Ziehl–Neelsen (ZN) staining and PCR testing of sputum samples also remained negative for *Mycobacterium tuberculosis* complex. To exclude other potential sites of infection, CT scans of the brain and lungs were performed, showing no extrahepatic manifestations of tuberculosis. There was no evidence for tuberculous granulomas or active tuberculosis of the lung. The patient received standard antitubercular treatment with rifampicin (600 mg once daily), isoniazid (300 mg once daily), pyrazinamide (1500 mg once daily), and ethambutol (1000 mg once daily) during the 2-month intensive phase, followed by rifampicin and isoniazid during the additional 4-month continuation phase. During treatment, the patient experienced a single episode of moderate, asymptomatic drug-induced liver injury (DILI), characterized by elevated transaminases. Pyrazinamide was suspected as the causative agent. The liver enzyme elevations resolved promptly after temporary discontinuation of therapy and a gradual rechallenge protocol. The

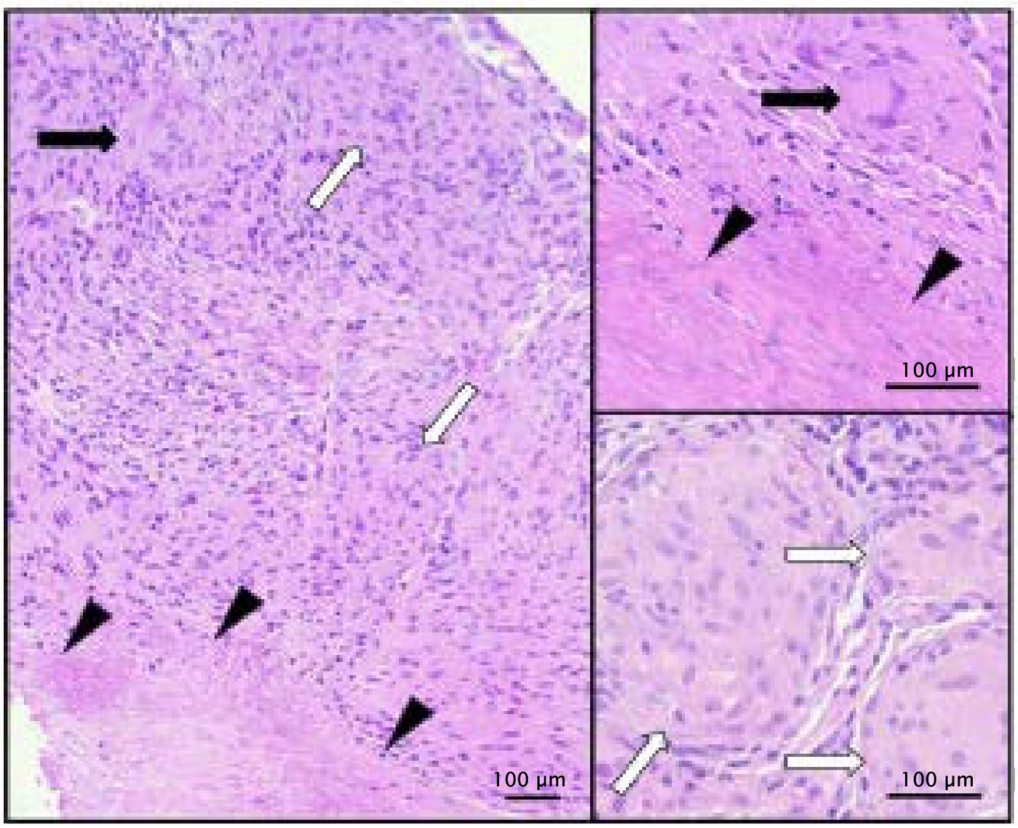

Fig. 2 Microscopic histopathology image of liver biopsy. It shows multiple epitheloid granulomas (indicated by white arrows), multinucleated giant cells (indicated by black arrows), and amorphous, necrotic material (indicated by black arrowheads)

patient responded well to the full 6-month course, with complete resolution of symptoms and subsequent clinical cure.

Discussion and conclusion

This case emphasizes the importance of considering isolated hepatic tuberculosis as a differential diagnosis in patients presenting with elevated tumor markers and hepatic lesions. Isolated hepatic tuberculosis is a rare manifestation. Mycobacteria can reach the liver via the portal vein, spreading from the gastrointestinal tract, and form nodular lesions greater than 2 mm in size. However, in most cases, hepatic tuberculosis is part of miliary disease, with hematogenous dissemination from the lungs via the hepatic artery, resulting in multiple micronodular lesions smaller than 2 mm in diameter [10]. The low oxygen tension in hepatic tissue is believed to create an unfavorable environment for mycobacterial growth [10]. If left untreated, hepatic tuberculosis can lead to serious complications by undergoing caseous central necrosis, which can form tuberculous abscesses [11, 12]. These abscesses may rupture into the peritoneal cavity and lead to tuberculous peritonitis, a life-threatening complication with high mortality [13]. The rarity and nonspecific clinical and radiological features of hepatic tuberculosis make it diagnostically challenging. It often mimics other hepatic pathologies such as malignancies or echinococcosis, potentially leading to delays in diagnosis and inappropriate treatment [4]. There are no pathognomonic radiographic features of hepatic tuberculosis. On CT scan, isolated hepatic TB typically appears as hypodense nodules without contrast enhancement, similar to necrotic liver tumors. Internal nodular or irregular calcifications are suggestive of tuberculomas and may serve as a diagnostic clue [13]. However, focal calcifications are not specific and may also be found in other granulomatous liver diseases such as histoplasmosis, echinococcosis, or sarcoidosis [14].

Furthermore, hepatic tuberculosis can be associated with elevated tumor markers, adding to the complexity and diagnostic challenge [6, 7]. Several case reports have described extrapulmonary TB being initially misdiagnosed as malignancy owing to misleading radiologic features and elevated tumor markers [6, 7, 11]. While tumor markers such as AFP, CEA, and CA 19-9 are valuable in the management of various malignancies, their specificity is limited in certain benign conditions, including tuberculosis [15]. AFP expression increases in hepatocyte injury and regeneration, leading to mild elevation in benign liver diseases, including hepatic tuberculosis [16]. CA19-9, although mainly associated with pancreatic cancer, may rise due to intrahepatic cholestasis, as seen in our patient [17]. Elevated CEA levels, typically associated with gastrointestinal malignancies such as hepatocellular carcinoma, can also be elevated due to infections, including tuberculosis [18]. Hepatic inflammation and necrosis caused by *Mycobacterium tuberculosis* might stimulate hepatocytes and obstruct biliary tracts, leading to increased serum levels of AFP, CEA, and CA19-9. Therefore, sole reliance on tumor markers without clinical, radiological, and histopathological correlation may lead to misdiagnosis. In our case, a CT-guided core biopsy, combined with histopathological examination and PCR, was essential for timely diagnosis. Histologically, hepatic tuberculosis is characterized by granulomatous inflammation, either caseating or non-caseating. Caseating granulomas typically consist of central necrosis surrounded by epithelioid histiocytes, Langhans giant cells, and peripheral lymphocytic infiltration [19]. While ZN staining remains a valuable diagnostic modality for tuberculosis, its sensitivity can be limited, especially in extrapulmonary tuberculosis where bacterial load is typically lower than in pulmonary tuberculosis. Therefore, a negative result does not exclude mycobacterial infection. PCR has a significantly better sensitivity and specificity, rapid turn-around time, and the ability to detect even minimal amounts of mycobacterial DNA. Furthermore, PCR, unlike ZN staining, allows for differentiation of *Mycobacterium tuberculosis* complex from nontuberculous mycobacteria (NTM), which can also cause hepatic disease but requires different management [20]. In our patient, ZN staining did not reveal acid-fast bacilli, and PCR testing confirmed the diagnosis of hepatic tuberculosis. Mycobacterial culture, while the gold standard for diagnosing tuberculosis, has certain limitations. It has a high specificity and allows for drug susceptibility testing. However, mycobacterial culture has a long turnaround time of many weeks and might not capture the disease in extrapulmonary sites owing to a low bacterial burden.

Treatment for hepatic tuberculosis follows the same standard regimen used for pulmonary and other extrapulmonary forms. It consists of an intensive phase with isoniazid, rifampicin, pyrazinamide, and ethambutol, followed by a continuation phase with isoniazid and rifampicin. Treatment typically lasts 6 months, though prolonged therapy may be required in severe cases. Monitoring of liver function is crucial owing to the risk of drug-induced hepatotoxicity [13]. The development of drug-induced liver injury during our patient's treatment served as a reminder of the potential side effects of antitubercular treatment. Prompt recognition and temporary discontinuation of therapy, followed by gradual reintroduction, led to full recovery and successful treatment completion. There is no evidence in

current literature suggesting that hepatic tuberculosis is a specific risk factor for antituberculosis drug-induced liver injury (AT-DILI).

This case underscores the diagnostic challenges of isolated hepatic tuberculosis, particularly when presenting with elevated tumor markers and imaging findings that mimic malignancy. We highlight the importance of thorough diagnostic evaluation, including histopathological examination and PCR testing. It is important for clinicians to consider extrapulmonary tuberculosis as differential diagnosis, especially in patients from TB-endemic regions, even in the absence of classic symptoms. Furthermore, the potential for drug-induced liver injury underlines the need for careful treatment monitoring. To date, no comprehensive studies have investigated the mechanisms underlying tumor marker elevation in TB. Further research is warranted to better understand the role of tumor markers in the diagnosis and monitoring of hepatic tuberculosis.

Abbreviations

TB	Tuberculosis
PCT	Polymerase chain reaction
WHO	World Health Organization
HIV	*Human immunodeficiency virus*
AFP	Alpha-fetoprotein
CA	Carbohydrate antigen
CEA	Carcinoembryonic antigen
GGT	Gamma-glutamyl transferase
IWGE	Informal Working Group on Echinococcosis
CE	Cystic echinococcosis
CT	Computed tomography
MRI	Magnetic resonance imaging
ELISA	Enzyme-linked immunosorbent assay
ZN	Ziehl–Neelsen
MLST	Multilocus sequence typing

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Author contributions
HL was the physician who provided clinical diagnosis and treatment of the patient; SD, JFH, FW, and MS analyzed and interpreted the patient data; histopathological analysis was performed by PK and SL; SD wrote the original draft preparation; JFH, HL, and MS reviewed the manuscript; all authors have read and agreed to the published version of the manuscript.

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Data availability
The data underlying the findings of this study are available from the corresponding author upon reasonable request, with the approval of the participating investigators.

Declarations

Ethics approval and consent to participate
We declare that our institution does not require ethics committee approval for retrospective case reports analyses of patients' records and histological data.

Consent for publication
Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests
This case report has no competing interests.

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