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Case Report

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Hepatic tuberculosis mimicking a complex cystic hepatic neoplasm with occult Miliary pulmonary involvement: a case report and narrative review

Mohammed Alkhanafsa[1], Ra'ed Ababneh[2,*], Jamil Wafi[1], Soud Deek[1], Fadel Alqatati[1], Khaled Murshed[3]

[1]Department of Internal Medicine, Hamad Medical Corporation, Zone 37, Al Rayyan Road, Doha, Qatar
[2]Department of Internal Medicine, Hackensack Meridian School of Medicine, 123 Metro Blvd, Nutley, NJ 07110, United States of America
[3]Department of Radiology, Hamad Medical Corporation, Zone 37, Al Rayyan Road, Doha, Qatar, Qatar

*Corresponding author. Department of Internal Medicine, Hackensack Meridian School of Medicine, 123 Metro Boulevard, Nutley, NJ 07110 United States of America. E-mail: raed.ababneh@hmhn.org

Abstract

Introduction: Hepatic tuberculosis is a rare extrapulmonary manifestation that may mimic hepatobiliary malignancy or complex cystic lesions, resulting in diagnostic challenges. Case Presentation: A 36-year-old Filipino woman without recognized immunocompromising conditions presented with fever, weight loss, jaundice, and right upper quadrant pain. Imaging showed a multiloculated cystic lesion in hepatic segments V/VI with focal biliary prominence. Liver biopsy demonstrated necrotizing and non-necrotizing granulomatous inflammation with focally positive Ziehl-Neelsen staining. Tissue MTB-PCR detected *Mycobacterium tuberculosis* without rifampicin resistance, and culture confirmed *M. tuberculosis* complex susceptible to streptomycin, isoniazid, rifampicin, and ethambutol. CT chest showed diffuse bilateral micronodules in a miliary pattern; initial sputum smear and PCR were negative, but final sputum culture became positive at 6 weeks. Standard 2HRZE/4HR therapy was initiated, with clinical and biochemical improvement at 1 month. Conclusion: Hepatic tuberculosis should be considered in atypical cystic hepatic lesions, and tissue diagnosis is essential to avoid unnecessary invasive procedures.

Keywords **hepatic tuberculosis, extrapulmonary tuberculosis, miliary tuberculosis, cystic hepatic lesion, case report**

Introduction

Hepatic tuberculosis is a rare extrapulmonary manifestation and is most often encountered as part of disseminated or miliary disease. Focal hepatic involvement is uncommon and may present with constitutional symptoms, abdominal pain, jaundice, or abnormal liver biochemistry [1]. Imaging is variable and includes micronodular disease, macronodular masses, abscess-like collections, and complex cystic or pseudotumoral lesions that may resemble hepatobiliary malignancy [2]. Pulmonary miliary involvement may be clinically silent and subtle on chest radiography, making CT important when dissemination is suspected [3]. Because imaging alone is often nondiagnostic, tissue sampling with histopathological and microbiological assessment is central to diagnosis and may prevent unnecessary surgery. We report hepatic tuberculosis presenting as a complex multiloculated cystic hepatic lesion with cholestatic liver injury and clinically occult pulmonary involvement in a miliary pattern.

Case presentation

A 36-year-old Filipino accountant, previously healthy, presented with 2 weeks of intermittent high-grade fever, chills, fatigue, right upper quadrant and epigastric pain, jaundice, and 4 kg weight loss. She denied respiratory or gastrointestinal symptoms, previous tuberculosis, known exposure, or recent travel. HIV Ag/Ab was negative, HbA1c was 6.0%, and she had no history of diabetes, corticosteroid or other immunosuppressive use, malignancy, transplantation, or known immunodeficiency.

On admission, she was febrile (38.5°C) and tachycardic (heart rate 125 beats/min). Examination revealed scleral icterus, mild jaundice, and right upper quadrant tenderness without guarding or rebound tenderness. There was no hepatosplenomegaly, lymphadenopathy, ascites, or evidence of chronic liver disease.

Complete blood count and renal function were normal. Liver tests showed a predominantly cholestatic pattern: alkaline phosphatase 349 U/L, gamma-glutamyl transferase 332 U/L, AST 201 U/L, ALT 168 U/L, and bilirubin 23.3 μmol/L. CRP (73 mg/L) and procalcitonin (2.17 ng/mL) were elevated. Eosinophilia was absent.

Abdominal ultrasound showed focal prominence of the right intrahepatic biliary ducts with adjacent heterogeneous hepatic parenchyma. The common bile duct was not dilated. MRCP demonstrated a 3.6 × 3.1 cm complex multiloculated cystic lesion in hepatic segments V and VI with focal intrahepatic biliary prominence and

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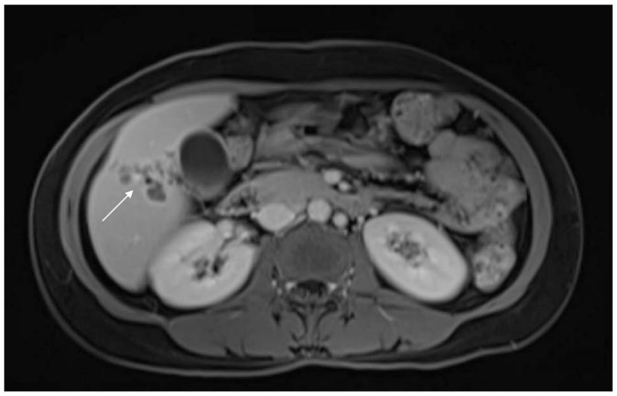


Figure 1 Axial contrast-enhanced MRI abdomen demonstrating a complex multiloculated cystic lesion within hepatic segments V and VI.

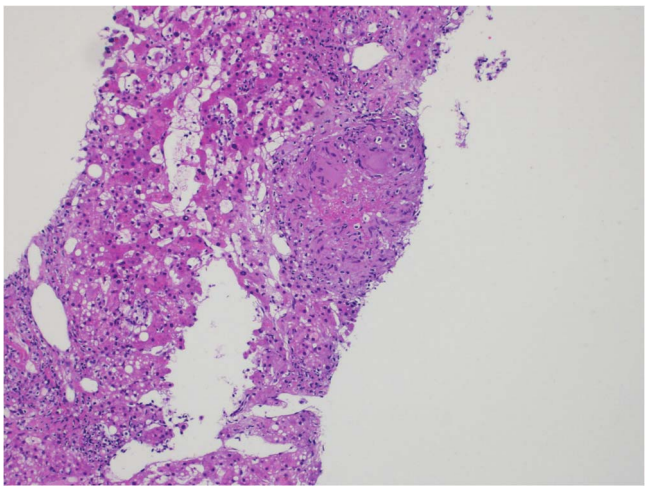


Figure 2 Photomicrograph showing liver parenchyma infiltrated by a necrotizing granuloma (hematoxylin and eosin stain, ×100).

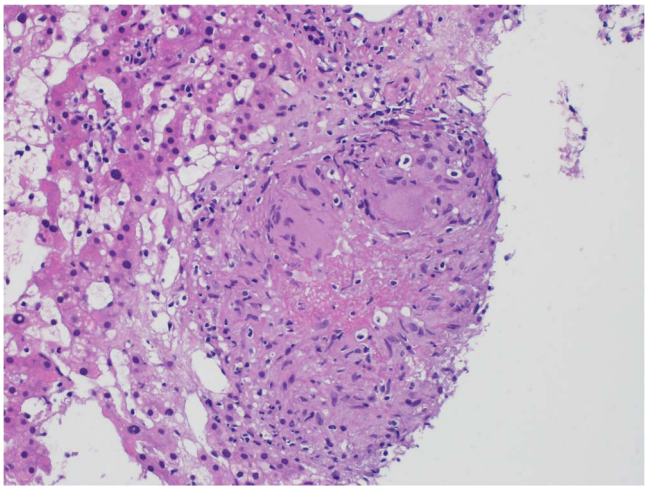


Figure 3 High-power view showing a well-formed granuloma with central necrosis, the adjacent liver parenchyma shows macrovesicular steatosis (hematoxylin and eosin stain, ×200).

no extrahepatic biliary obstruction (Fig. 1). There was no abdominal lymphadenopathy, splenomegaly, ascites, bowel abnormality, or additional hepatic lesion.

The differential diagnosis included cystic hepatic neoplasm, autoimmune/inflammatory hepatobiliary disease, pyogenic abscess, parasitic disease, and granulomatous infection. Tumor markers revealed mildly elevated CA 125 (63.5 U/mL) and CA 19–9 (43.4 U/mL), while CEA was normal, contributing to concern for a neoplastic mimic. Given the cholestatic liver injury and focal biliary abnormalities, autoimmune hepatobiliary disease was also considered; ANA, AMA, ASMA, anti-LKM antibodies, tissue transglutaminase antibodies, and IgG subclasses including IgG4 were negative. Echinococcus serology and stool ova/parasite examination were also negative.

Empirical antibacterial therapy for suspected cholangitis consisted of ceftriaxone plus metronidazole for 7 days, followed by piperacillin-tazobactam for 4 days and meropenem for 2 days because fever and inflammatory abnormalities persisted. After approximately 2 weeks without meaningful response, ultrasound-guided core biopsy of the heterogeneous right hepatic area yielded two cores (1.5 and 1.8 cm). Histopathological examination showed multiple necrotizing and non-necrotizing granulomas (Figs. 2 and 3). Ziehl-Neelsen staining was focally positive for acid-fast bacilli. Tissue MTB-PCR detected *Mycobacterium tuberculosis* DNA with no rifampicin resistance detected, and mycobacterial culture confirmed *M. tuberculosis* complex. Susceptibility was demonstrated to the agents tested: streptomycin, isoniazid, rifampicin, and ethambutol. The exact molecular assay and mycobacterial culture platform were not documented in the available clinical record.

Further evaluation for pulmonary involvement showed negative initial sputum AFB smear and MTB-PCR, with preliminary mycobacterial culture negative at 14 days. QuantiFERON-TB Gold, obtained during subsequent TB evaluation, was positive. Chest radiography was reported as normal on initial interpretation (Fig. 4); however, subsequent CT of the chest demonstrated diffuse bilateral pulmonary micronodules consistent with a miliary pattern (Fig. 5). The final sputum culture became positive for *M. tuberculosis* at 6 weeks, confirming pulmonary tuberculosis.

At treatment initiation, the patient weighed 51 kg. The patient was started on rifampicin 600 mg, isoniazid 300 mg, pyrazinamide 1600 mg, and ethambutol 1100 mg once daily, with pyridoxine 50 mg daily, planned as 2 months of HRZE followed by 4 months of HR.

At the 1-month follow-up, the patient remained adherent to therapy and reported no treatment-related adverse events. Fever had not recurred, and her appetite, abdominal pain, and jaundice had improved. Liver biochemistry also showed improvement, with AST decreasing to 43 U/L, ALT to 18 U/L, ALP to 290 U/L, and total bilirubin to 15.5 μmol/L. Selected laboratory parameters at admission and at the 1-month follow-up are summarized in Table 1. No repeat abdominal or chest imaging had been performed at the time of the follow-up visit.

Discussion

Hepatic tuberculosis is an uncommon and diagnostically challenging manifestation of tuberculosis, most commonly occurring as part of disseminated or miliary disease. Focal hepatic involvement is rare and may present with nonspecific symptoms including fever, abdominal pain, weight loss, abnormal liver enzymes, and jaundice. These findings overlap with cholangitis, hepatic abscess, inflammatory biliary disorders, and malignancy. Liver biochemical abnormalities are variable; however, a cholestatic pattern, particularly alkaline-phosphatase

Table 1 Selected laboratory parameters at admission and 1-month follow-up.

Laboratory parameter	At admission	1-month follow-up	Reference range
Hemoglobin	12.7	12.4	12.0–16.0 g/dL
White blood cell count	6.1	5.2	4.0–10.0 $\times 10^3/\mu$L
Platelet count	240	320	150–400 $\times 10^3/\mu$L
Eosinophils	0.1	0.1	0–6%
AST	201	43	0–35 U/L
ALT	168	18	0–35 U/L
Alkaline phosphatase	349	290	35–104 U/L
Gamma-glutamyl transferase	332	Not repeated	6–42 U/L
Total bilirubin	23.3	15.5	5–21 μmol/L
C-reactive protein	72	Not repeated	$<$5 mg/L
Procalcitonin	2.17	Not repeated	$<$0.05 ng/mL

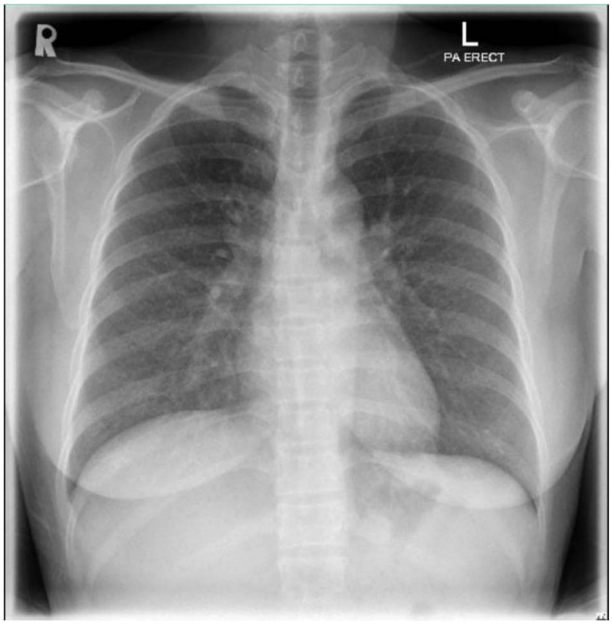


Figure 4 Chest radiograph showing no definite miliary pattern on initial interpretation.

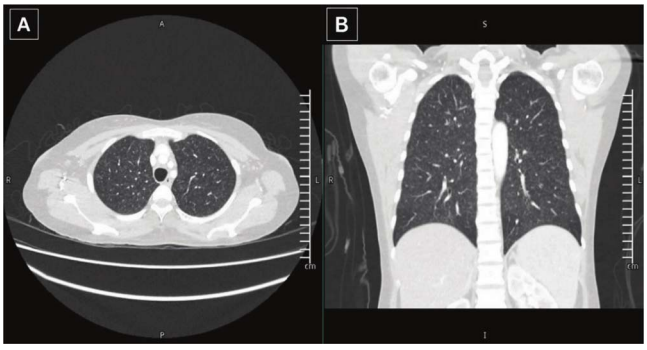


Figure 5 (A/B). Computed tomography (CT) of the chest demonstrating diffuse bilateral pulmonary micronodules distributed throughout both lungs, consistent with miliary tuberculosis, shown in axial (A) and coronal (B) views.

elevation, is well recognized, although hepatocellular and mixed patterns may occur [1]. Tuberculous granulomas may involve hepatic lobules or portal regions, with lobular involvement more typical of miliary disease and portal involvement of localized disease [1].

In this patient, fever, jaundice, weight loss, cholestatic liver injury, focal intrahepatic biliary abnormalities, and a complex multiloculated cystic lesion raised significant concern for biliary cystic neoplasm. Mild elevation of CA 19–9 and CA 125 further complicated the assessment, although these markers may increase in inflammatory and infectious conditions. Infectious, parasitic, and autoimmune/inflammatory hepatobiliary disorders were also considered. Persistent fever and inflammatory abnormalities despite sequential broad-spectrum antibacterial therapy strengthened the indication for tissue diagnosis.

The radiological appearance of hepatic tuberculosis is highly variable and may include micronodular lesions, macronodules, abscess-like collections, calcified lesions, or complex cystic masses. This overlap with neoplastic and infectious liver lesions frequently results in delayed diagnosis and unnecessary invasive procedures [2]. In our patient, histopathology demonstrated necrotizing and non-necrotizing granulomatous inflammation with focally positive Ziehl–Neelsen staining. Tissue MTB-PCR detected *M. tuberculosis* DNA with no rifampicin resistance detected, and culture confirmed *M. tuberculosis* complex, with susceptibility demonstrated to the agents tested (streptomycin, isoniazid, rifampicin, and ethambutol). These concordant findings established hepatic tuberculosis.

A focused narrative comparison of seven recent clinically similar cases is summarized in Table 2 [4–10]. Several mimicked cystic neoplasm, cholangiocarcinoma, metastatic disease, hepatocellular carcinoma, or liver abscess. Khalid et al. described a cystic lesion interpreted as cystadenoma that required resection before tuberculosis was recognized [4], while Acevedo-Rodriguez et al. reported cystic hepatic lesions with biliary dilatation mimicking Caroli disease or cholangiocarcinoma [10]. In contrast, image-guided biopsy established the diagnosis in our patient and avoided unnecessary surgery.

Another important aspect was the discrepancy between pulmonary investigations. Despite the absence of respiratory symptoms, initial sputum AFB smear and MTB-PCR were negative, and chest radiography was reported as normal on initial interpretation; however, CT chest revealed diffuse bilateral micronodules compatible with miliary tuberculosis. The final sputum mycobacterial culture subsequently became positive for *M. tuberculosis* after 6 weeks, confirming pulmonary tuberculosis and supporting disseminated disease. CT is more sensitive than chest

Table 2 Summary of recent case reports (2023–2026) of hepatic tuberculosis presenting as cystic, pseudotumoral, malignant-appearing, or abscess-like hepatic lesions.

Study/year	Patient and presentation	Imaging/initial impression	Diagnostic confirmation	Pulmonary/ extrahepatic involvement	Treatment/ outcome
Khalid et al. [4], 2026	46F; upper abdominal pain and weight loss	Bilobed cystic segment VII lesion with a small biliary channel; interpreted as hepatic cystadenoma	Surgical resection; granulomatous inflammation on histopathology	Reported as isolated hepatic TB	Standard ATT for 9 months; clinically well after treatment
Li et al. [5], 2025	44F; abdominal pain, reduced appetite, fever, and night sweats	Right-lobe tuberculous abscess; later heterogeneous lesion with internal septations	US-guided biopsy with chronic granulomatous inflammation; TB DNA positive	No pulmonary symptoms; tuberculous peritonitis described	Four-drug ATT; symptoms essentially resolved by 6 months
Singla et al. [6], 2025	45 M; RUQ pain, fever, and jaundice	Hepatolithiasis, left hepatic duct stricture, and left-lobe atrophy; malignancy suspected	Left lateral segmentectomy; nonnecrotizing granulomatous inflammation; AFB negative	No miliary or pulmonary disease identified	ATT after surgery; asymptomatic without residual/recurrent disease at 6 months
Labied et al. [7], 2025	51F; hepatic lesions found during evaluation of an ampullary tubulovillous adenoma	Multiple hepatic lesions with heterogeneous enhancement; initially interpreted as metastases	Percutaneous liver biopsy confirmed pseudotumoral hepatic TB	No pulmonary involvement reported; HIV negative	Six-month ATT; complete MRI resolution
Seong et al. [8], 2025	35 M; chronic RUQ pain, anorexia, malaise, and weight loss	Solid segment VI/VII lesions; imaging and FNAC favored HCC	Hepatic segmentectomy; caseating granulomas and Langhans giant cells	Chest CT clear; isolated hepatic disease	ATT initiated postoperatively
Shahzad et al. [9], 2024	55F; RUQ pain, fever, weight loss, and altered bowel habits	Hepatic lesions mimicking metastatic disease on imaging	Targeted liver biopsy showed necrotizing granulomatous inflammation with caseous necrosis	No active pulmonary TB	ATT; favorable follow-up without reported treatment toxicity
Acevedo-Rodriguez et al. [10], 2023	54F; recurrent abdominal pain, jaundice, fever, vomiting, weight loss, and asthenia	Multiple cystic hepatic lesions with intrahepatic/extra-hepatic biliary dilatation; Caroli disease and cholangiocarcinoma considered	Operative lesion biopsy and histopathology supported hepatic TB	Primary/extrapul-monary hepatic TB reported	Six-month standard regimen (2 months HRZE, then 4 months HR)

AFB, acid-fast bacilli; ATT, anti-tuberculous therapy; FNAC, fine-needle aspiration cytology; HCC, hepatocellular carcinoma; HR, isoniazid and rifampicin; HRZE, isoniazid, rifampicin, pyrazinamide, and ethambutol; RUQ, right upper quadrant; TB, tuberculosis; US, ultrasound.

radiography for subtle miliary involvement; therefore, initially negative respiratory investigations do not exclude miliary disease when extrapulmonary tuberculosis has been established [3].

Compared with previously reported cases (Table 2), this case is distinctive because of a complex multiloculated cystic hepatic lesion, cholestatic injury, microbiologically confirmed hepatic tuberculosis, and clinically silent miliary pulmonary involvement. This case highlights the importance of considering tuberculosis in atypical cystic hepatic lesions and the value of tissue diagnosis in preventing unnecessary surgery. The early clinical and biochemical improvement observed at 1 month was encouraging; however, the short follow-up duration and absence of repeat hepatic or chest imaging limit assessment of definitive treatment response and radiological resolution.

Conclusion

Hepatic tuberculosis should be considered in patients with constitutional symptoms, cholestatic liver injury, and atypical cystic hepatic lesions. Image-guided biopsy with histopathological and microbiological confirmation can establish the diagnosis and prevent unnecessary surgery.

Ethics Approval and Consent to Participate

Ethical approval for this case report was waived by the Medical Research Center (MRC), Hamad Medical Corporation, Doha, Qatar, in accordance with institutional guidelines for single-patient case reports. The study was conducted in accordance with institutional ethical standards.

Consent for Publication

Written informed consent was obtained from the patient for publication of this case report, including clinical information and accompanying images. The patient reviewed the manuscript and provided consent prior to submission.

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Authors' contributions

Mohammed Alkhanafsa: Conceptualization, data collection, manuscript drafting, and revision. Ra'ed Ababneh: Data collection, literature review, and manuscript revision. Jamil Wafi: Data collection, literature review, and manuscript revision. Soud Deek: Clinical data interpretation and critical revision of the manuscript. Fadel Alqatati: Clinical evaluation, data interpretation, and critical revision of the manuscript. Khaled Murshed: Radiological interpretation and critical review of the imaging-related content. All authors reviewed and approved the final version of the manuscript.

Conflicts of interest

The authors declare that they have no conflicts of interest relevant to this work.

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Data availability

All data supporting the findings of this case report are included within the manuscript. Additional data may be available from the corresponding author upon reasonable request, subject to patient confidentiality and institutional regulations.

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