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Isolated Pseudotumoral Hepatic Tuberculosis in an Immunocompetent Adolescent: A Case Report

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ABSTRACT

Isolated hepatic tuberculosis is exceptionally rare, particularly in immunocompetent children and adolescents, and its nonspecific clinical and radiological presentation may mimic infectious or neoplastic liver lesions. We report the case of a 14-year-old boy with no known tuberculosis exposure or underlying immunosuppressive condition who presented with fever, night sweats, anorexia, weight loss, and right upper quadrant pain. Contrast-enhanced computed tomography demonstrated a 6-cm multiloculated pseudotumoral lesion composed of clustered, coalescent hypodense microcollections involving hepatic segments V, VI, and VII, without pulmonary or other extrahepatic involvement. Liver biopsy revealed epithelioid and multinucleated giant-cell granulomas with central caseous necrosis. Molecular testing, acid-fast staining, and mycobacterial culture were not performed. The patient received antituberculous therapy for nine months. Follow-up computed tomography one year later showed complete resolution, leaving only a small retractile parenchymal scar. This case highlights the importance of considering isolated hepatic tuberculosis in an adolescent with an atypical pseudotumoral or abscess-like liver lesion and the central role of image-guided biopsy in avoiding unnecessary surgery.

KEYWORDS

Hepatic Tuberculosis; Tuberculous Liver Abscess; Pseudotumoral Lesion; Adolescent; Liver Biopsy

Abbreviations: ALP: Alkaline phosphatase; ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; CRP: C-reactive protein; CT: Computed tomography; GGT: Gamma-glutamyl transferase; HIV: Human immunodeficiency virus; MRI: Magnetic resonance imaging; PCR: Polymerase chain reaction; TB: Tuberculosis; WHO: World Health Organization; Xpert MTB/RIF: Xpert *Mycobacterium tuberculosis*/rifampicin assay

MAIN ARTICLE

INTRODUCTION

Tuberculosis remains a major global health problem, with an estimated 10.7 million new cases and more than 1.2 million deaths worldwide in 2024 [1]. Although pulmonary tuberculosis is the most common presentation, extrapulmonary involvement accounts for a substantial proportion of cases and may affect almost any organ.

Hepatic tuberculosis is uncommon and usually occurs as part of disseminated or miliary disease. In contrast, isolated hepatic tuberculosis represents less than 1% of all tuberculosis cases and is exceptionally rare in immunocompetent children and adolescents [2,3]. Its clinical and biological manifestations are nonspecific, while its imaging appearance may mimic pyogenic or amebic abscesses, hydatid disease, or primary and secondary hepatic tumors. Consequently, histopathological and microbiological examination of tissue samples is often required to establish the diagnosis and avoid unnecessary surgical intervention [4].

We report the case of a 14-year-old boy with no known tuberculosis exposure or underlying immunosuppressive condition who presented with constitutional symptoms, inflammatory syndrome, and right upper quadrant pain. CT revealed a 6-cm multiloculated pseudotumoral lesion composed of clustered, coalescent hypodense microcollections confined to the right hepatic lobe, with no pulmonary or other extrahepatic involvement. Liver biopsy demonstrated epithelioid and multinucleated giant-cell granulomas with caseous necrosis. The patient showed complete clinical and radiological resolution following antituberculous therapy.

CASE REPORT

A 14-year-old boy with no previous history of tuberculosis and no known contact with an infected individual presented with a one-month history of intermittent, unquantified fever, night sweats, anorexia, and a 3-kg weight loss. He also reported right upper quadrant abdominal pain.

Liver function tests showed moderate cytolysis and a predominantly cholestatic pattern, with AST and ALT levels of 72 and 88 U/L, respectively, an alkaline phosphatase level of 620 U/L, and a gamma-glutamyl transferase level of 146 U/L. Total bilirubin was mildly elevated at 26 $\mu\text{mol/L}$, including a conjugated bilirubin level of 18 $\mu\text{mol/L}$. The white blood cell count was 13,000/ mm^3 , including 8,660 neutrophils/ mm^3 and 2,440 lymphocytes/ mm^3 . Inflammatory

markers were markedly elevated, with an erythrocyte sedimentation rate of 85 mm/h, a C-reactive protein level of 120 mg/L, and an increased fibrinogen level.

Contrast-enhanced abdominal CT revealed a 6-cm multiloculated pseudotumoral lesion composed of clustered, coalescent hypodense microcollections involving hepatic segments V, VI, and VII. The lesion showed peripheral and septal enhancement, without a predominant solid component, giving an abscess-like appearance. No abdominal lymphadenopathy, ascites, peritoneal abnormalities, or splenic involvement was identified. Thoracic imaging was unremarkable, with no evidence of pulmonary or mediastinal tuberculosis. Representative CT images at successive craniocaudal levels are shown in Figures 1-3.

A liver biopsy was subsequently performed. Histopathological examination revealed epithelioid and multinucleated giant-cell granulomas with central caseous necrosis, strongly supporting the diagnosis of hepatic tuberculosis. Mycobacterial PCR, Ziehl–Neelsen staining, and mycobacterial culture were not performed.

The diagnosis of isolated hepatic tuberculosis was retained. The patient received antituberculous therapy for nine months, with a favorable clinical and biological outcome. Follow-up CT performed one year later demonstrated complete resolution of the hepatic lesions, leaving only a small retractile parenchymal scar at the site of the previous abnormality. The follow-up appearance is shown in Figure 4.

DISCUSSION

Tuberculosis remains a major global health concern. According to the World Health Organization, an estimated 10.7 million people developed tuberculosis in 2024, and the disease caused more than 1.2 million deaths worldwide [1]. Although pulmonary involvement is the most common presentation, extrapulmonary tuberculosis accounts for approximately 15–20% of active tuberculosis cases in immunocompetent individuals, with a substantially higher proportion among immunocompromised patients [2]. Hepatic tuberculosis is particularly uncommon, representing approximately 0.4% of all tuberculosis cases and 1–3.5% of extrapulmonary forms in non-immunosuppressed populations [3].

Hepatic involvement should be distinguished according to its clinical setting. Microscopic liver involvement is relatively frequent in disseminated or miliary tuberculosis and has been reported in up to 50–80% of advanced disseminated cases. By contrast, clinically apparent isolated hepatic tuberculosis, defined by the absence of pulmonary, gastrointestinal, lymph-node, splenic, or other extrahepatic involvement, accounts for less than 1% of all tuberculosis cases

[2,4]. Tuberculous liver abscess is even rarer, particularly in immunocompetent children and adolescents. The available pediatric literature is consequently limited mainly to isolated case reports and small case series [5,6].

Mycobacterium tuberculosis may reach the liver through the hepatic artery during hematogenous dissemination, through the portal vein from a gastrointestinal focus, through lymphatic channels, or, less frequently, by direct extension from an adjacent tuberculous lesion. The relative resistance of the liver to tuberculous infection has been attributed to its low oxygen tension, the phagocytic activity of Kupffer cells, and the bacteriostatic properties of bile. Hepatic tuberculosis may be classified into miliary, diffuse granulomatous, macronodular or pseudotumoral, abscess-forming, and biliary forms [2,4]. In the present case, the 6-cm pseudotumoral lesion was composed of clustered, coalescent hypodense microcollections with peripheral and septal enhancement, most consistent with the macronodular, abscess-forming form of hepatic tuberculosis.

Isolated tuberculous liver abscess is classically associated with immunosuppression, particularly human immunodeficiency virus infection, malignancy, malnutrition, diabetes, prolonged corticosteroid therapy, or other immunosuppressive treatments.

The clinical and laboratory manifestations of hepatic tuberculosis are nonspecific. The most frequently reported symptoms include prolonged fever, night sweats, anorexia, weight loss, fatigue, and right upper quadrant abdominal pain. Hepatomegaly may be present, whereas jaundice is uncommon and generally suggests biliary involvement. Laboratory abnormalities include leukocytosis, anemia, elevated erythrocyte sedimentation rate and C-reactive protein levels, and abnormal liver function tests. Alkaline phosphatase and gamma-glutamyl transferase are often disproportionately elevated, while aminotransferase and bilirubin elevations are variable. The combination of fever, night sweats, anorexia, a 3-kg weight loss, marked inflammatory syndrome, and a predominantly cholestatic liver biochemical profile in our patient was therefore compatible with, but not specific for, hepatic tuberculosis.

Imaging findings are similarly variable and depend on the pathological stage of the disease. On CT, macronodular hepatic tuberculosis may appear as a solitary or multifocal hypodense lesion with peripheral enhancement, central caseous necrosis, calcification, or a pseudotumoral appearance. Tuberculous abscesses may demonstrate a honeycomb or cluster-like pattern, corresponding to multiple coalescent necrotic foci separated by enhancing septa [4,7]. These findings closely corresponded to the pseudotumoral, multiloculated pattern formed by

clustered, coalescent hypodense microcollections in our patient. Nevertheless, neither CT nor MRI can reliably distinguish hepatic tuberculosis from other infectious or neoplastic lesions, making tissue sampling essential in atypical cases.

In a 14-year-old patient presenting with a solitary or dominant cystic hepatic lesion, the main differential diagnosis is a pyogenic liver abscess. Fever, neutrophilic leukocytosis, elevated inflammatory markers, a cluster sign, and peripheral or septal enhancement may occur in both pyogenic and tuberculous abscesses. An amebic abscess should also be considered, particularly in endemic areas, and typically presents as a solitary right-lobe lesion. Hydatid disease is another important differential diagnosis in endemic regions; daughter cysts, detached membranes, mural calcifications, and positive *Echinococcus* serology may suggest this diagnosis. Fungal abscesses should be considered mainly in immunocompromised patients.

Neoplastic differentials include a necrotic hepatocellular carcinoma, particularly the fibrolamellar subtype occurring in adolescents without underlying cirrhosis, undifferentiated embryonal sarcoma, lymphoma, and necrotic metastasis. Hepatoblastoma and mesenchymal hamartoma are considerably less likely at 14 years of age because they predominantly occur in children younger than five years. A complicated hepatic cyst, inflammatory myofibroblastic tumor, and rare biliary cystic tumors may also be considered. Patient age, clinical presentation, serum alpha-fetoprotein and other tumor markers, epidemiological context, and the presence or absence of extrahepatic abnormalities can help prioritize these diagnoses, but histological analysis is frequently required [7].

The biological diagnosis of hepatic tuberculosis is challenging because the disease is often paucibacillary. Tuberculin skin testing and interferon-gamma release assays may support previous exposure to *Mycobacterium tuberculosis*, but they cannot distinguish active tuberculosis from latent infection and may be negative in active disease. When aspiration or biopsy is performed, samples should ideally be submitted for histopathological examination, acid-fast bacillus staining, mycobacterial culture, nucleic acid amplification testing such as Xpert MTB/RIF or Xpert Ultra, and drug-susceptibility testing [8]. Culture remains important because it provides microbiological confirmation and allows susceptibility assessment, although its sensitivity may be reduced by the low bacillary burden.

Histologically, the most suggestive finding is necrotizing granulomatous inflammation composed of epithelioid histiocytes and Langhans-type multinucleated giant cells surrounding central caseous necrosis. In the present case, the demonstration of epithelioid and giant-cell

granulomas with caseous necrosis strongly supported the diagnosis of hepatic tuberculosis. The favorable clinical response and complete radiological resolution under antituberculous treatment further strengthened the diagnosis.

Medical therapy is the cornerstone of treatment. For most drug-susceptible extrapulmonary forms in children and adolescents, current guidelines generally recommend an initial two-month intensive phase combining isoniazid, rifampicin, pyrazinamide, and ethambutol, followed by four months of isoniazid and rifampicin [9]. However, because of the rarity of hepatic tuberculosis and the absence of controlled studies specific to this localization, treatment durations ranging from 6 to 12 months have been reported in pediatric cases [5,6]. The nine-month treatment administered in our patient therefore falls within the durations described in the literature, although it is longer than the standard six-month regimen currently recommended for most extrapulmonary forms.

Because isoniazid, rifampicin, and pyrazinamide may cause hepatotoxicity, liver function tests should be assessed before treatment and monitored closely, particularly in patients with abnormal baseline liver biochemistry. Percutaneous aspiration or drainage may be indicated for large liquefied abscesses, persistent sepsis, diagnostic sampling, impending rupture, compressive complications, or an inadequate response to medical treatment. Surgery should be reserved for complicated lesions, failure of percutaneous management, biliary obstruction, or cases in which malignancy cannot be excluded [2,10].

The favorable outcome observed in our patient, with complete disappearance of the hepatic lesion on CT one year later and persistence of only a small retractile parenchymal scar, illustrates the effectiveness of antituberculous treatment. Residual fibrosis, parenchymal retraction, and occasionally calcification are recognized healing patterns. This case highlights the importance of considering isolated hepatic tuberculosis in the differential diagnosis of an abscess-like or pseudotumoral liver lesion in an adolescent, even in the absence of known tuberculosis exposure, pulmonary disease, or overt immunosuppression. Early image-guided biopsy may establish the diagnosis, avoid unnecessary hepatic surgery, and permit timely initiation of appropriate therapy.

CONCLUSION

Isolated hepatic tuberculosis should be considered in the differential diagnosis of an atypical pseudotumoral or abscess-like liver lesion in an adolescent, particularly in tuberculosis-endemic regions, even in the absence of known exposure, pulmonary disease, or overt

immunosuppression. Because clinical, laboratory, and imaging findings are nonspecific, image-guided biopsy plays a central role in establishing the diagnosis and excluding infectious and neoplastic mimics. Whenever possible, biopsy specimens should also undergo acid-fast staining, molecular testing, mycobacterial culture, and drug-susceptibility testing. Early recognition enables effective medical treatment, avoids unnecessary hepatic surgery, and is generally associated with a favorable outcome, as illustrated by the complete resolution of the lesion with only a small residual retractile scar in our patient.

FIGURES:



Figure 1. Axial contrast-enhanced CT image at the level of hepatic segment VII showing the cranial component of the multiloculated pseudotumoral lesion, composed of clustered hypodense microcollections in the right hepatic lobe.



Figure 2. Axial contrast-enhanced CT image at the junction of hepatic segments V and VII demonstrating clustered, coalescent hypodense microcollections with peripheral and septal enhancement, collectively forming a multiloculated pseudomass.



Figure 3. Axial contrast-enhanced CT image at the level of hepatic segment V showing the caudal extension of the multiloculated pseudomass formed by coalescent hypodense microcollections.



Figure 4. Follow-up non-contrast abdominal CT obtained one year later showing complete resolution of the previous hepatic lesions, with a small retractile residual parenchymal scar at the previous site of involvement.

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