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Hepatic Tuberculosis: A Diagnostic Challenge – Case Report and Literature Review

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Abstract

Hepatic tuberculosis is a rare but clinically important form of extrapulmonary tuberculosis that can mimic hepatic neoplastic or inflammatory lesions. Diagnosis often relies on a combination of clinical, imaging, and, most importantly, histological and microbiological findings. Management is based on appropriate antitubercular therapy, with particular attention to the risk of hepatotoxicity and coinfections (HBV, HCV).

We report the case of a 51-year-old female patient, followed for renal failure and ampullary adenoma, admitted for acute pancreatitis with incidental discovery of multiple hepatic lesions on imaging. The initial workup suggested metastases, but liver biopsy confirmed hepatic tuberculosis. This case illustrates the diagnostic challenge of this rare condition.

Keywords: Hepatic tuberculosis, Extrapulmonary tuberculosis, Mycobacterium tuberculosis, Antitubercular therapy, Liver biopsy.

Introduction

Tuberculosis, caused by Mycobacterium tuberculosis, primarily affects the lungs but can also involve various organs, resulting in extrapulmonary forms. Among these, hepatic tuberculosis corresponds to a particular localization of active infection. The first case was described in 1858 by Dr. John Syer Bristowe, a British physician [1]. In 1905, more than two decades after Koch's discovery of the bacillus, Rolleston and McNee proposed a classification distinguishing miliary (disseminated) hepatic tuberculosis from localized (isolated) forms [1].

Although the global prevalence of tuberculosis has markedly declined since the introduction of antitubercular therapy in the 1940s, the disease remains a major public health issue, especially in developing countries. Extrapulmonary forms, including hepatic tuberculosis, persist and still present significant diagnostic challenges due to their atypi-

cal clinical and radiological presentation [2].

Case Presentation

We report the case of a 51-year-old woman with a history of cholecystectomy (2012), moderate renal insufficiency under treatment since 2018, and ampullary adenoma diagnosed three years earlier. She had no known chronic liver disease, no alcohol consumption, no personal or family history of tuberculosis, and no evidence of immunodeficiency.

She was admitted for management of stage A acute pancreatitis. Clinically, she presented with isolated epigastric pain, with tenderness in the epigastrium and right hypochondrium. Lipase was more than three times the normal value.

CT scan of the abdomen revealed multiple well-defined oval hepatic lesions, hypodense with peripheral ring enhancement ("target" appearance), initially suggestive of secondary deposits. No intrahepatic bile duct dilation was noted, associated with signs of stage A pancreatitis. Abdominal ultrasound confirmed these findings. (Figure 1) (Figure 2).

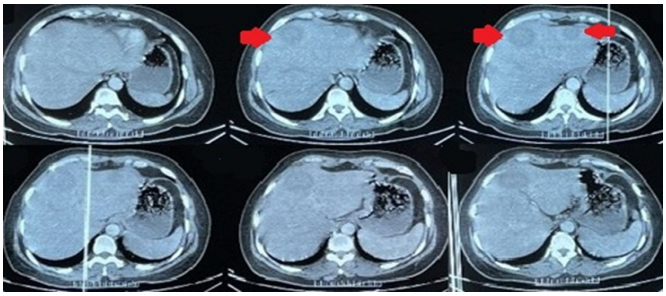

Figure 1: Hypodense hepatic lesions with a peripheral halo, showing a "target sign" (red arrow).

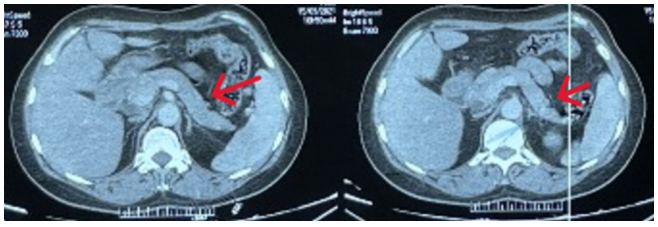

Figure 2: CT scan image showing the normal appearance of the pancreas (red arrow).

Biliary MRI showed multiple well-demarcated hepatic lesions, with dilatation of the common bile duct, suggestive of an odditis, but without intrahepatic bile duct dilatation (Figure 3).

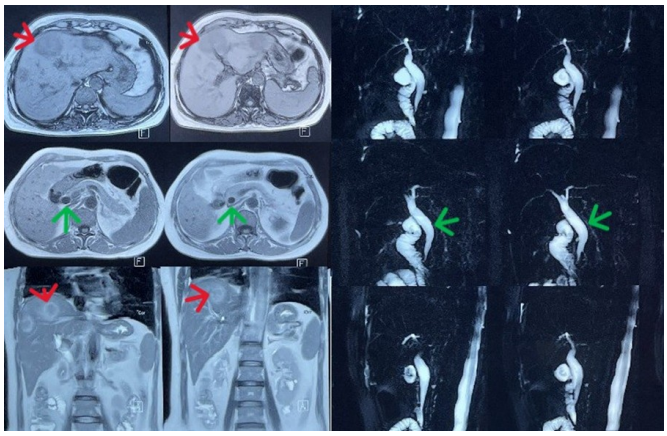

Figure 3: MRCP image showing well-defined hepatic lesions (red arrow) with dilatation of the common bile duct (green arrow).

Following clinical improvement, upper GI endoscopy with lateral vision was performed, showing an adenomatous appearance of the papilla without malignant transformation. Biopsies revealed nonspecific chronic fibro-inflammatory changes without malignancy. Colonoscopy, performed to search for a digestive primary, was normal.

Liver function tests were normal, and tumor markers "CAE ; CA 19.9" were negative.

At multidisciplinary team meeting, an ultrasound-guided liver biopsy was decided. Histopathology revealed necrotizing tuberculoid granulomatous inflammation, consistent with hepatic tuberculosis.

The patient received standard antitubercular therapy: 2 months of isoniazid, rifampicin, pyrazinamide, and ethambutol, followed by 4 months of isoniazid and rifampicin. She showed favorable clinical, biological, and radiological evolution.

Discussion

Hepatic tuberculosis refers to Mycobacterium tuberculosis infection of the liver, either in isolation (rare) or as part of miliary or diffuse abdominal tuberculosis [1]. Most cases (≈79%) result from systemic dissemination, corresponding to miliary hepatic tuberculosis. Isolated localized forms have been reported in South Africa, the Philippines, and India [1].

Globally, tuberculosis remains a major health issue with rising incidence [3]. This entity mostly affects individuals aged 11–50 years, with a peak in the second decade of life [4].

The bacilli can reach the liver via the hepatic artery (from lungs) or via the portal vein (from the gastrointestinal tract). Ingested bacilli may cause intestinal ulcers, then spread through the portal vein, producing primary hepatic involvement (granulomatous hepatitis). Over time, ulcers may heal, leaving isolated hepatobiliary tuberculosis [5,6].

Abdominal pain is the most frequent symptom (40–83%), followed by fever (30–100%) and hepatomegaly (10–100%). Less common signs include jaundice (0–60%), splenomegaly (0–40%), and ascites (5–25%) [5].

Hepatic TB may be macronodular or miliary. Macronodular form: solitary or multiple liver masses, mimicking tumors. Miliary form: multiple microabscesses, hypo- to isoechoic on US, hypodense on CT, mimicking metastases or lymphomas [9]. Lab tests may show elevated ALP, GGTP, and sometimes mild transaminase elevation. Histopathological or bacteriological confirmation remains essential [4].

Liver tissue sections may show necrotizing or non-necrotizing granulomas. Tuberculous granulomas typically contain a peripheral rim of lymphocytes and plasma cells, epithelioid histiocytes, and multinucleated giant cells, with or without central caseous necrosis. Acid-fast bacilli may be demonstrated with Ziehl–Neelsen stain or auramine–rhodamine fluorescent stain [7].

Macronodular tuberculomas can mimic metastatic carcinoma, hepatocellular carcinoma, or other necrotic liver lesions. Despite imaging clues, biopsy remains necessary [8].

Primary hepatic TB is managed with standard quadruple therapy (isoniazid, rifampicin, pyrazinamide, ethambutol) for 2 months, followed by 4–7 months of rifampicin + isoniazid [9]. Surgery may be needed in localized complicated cases (abscess, obstructive jaundice, portal hypertension, confusion with malignancy). Procedures include enucleation, local excision, segmentectomy, lobectomy, or drainage [2,5]. Monitoring is important due to risk of hepatotoxicity from ATT (particularly isoniazid, rifampicin, pyrazinamide) [10].

Conclusion

Hepatic tuberculosis remains a rare and diagnostically challenging condition due to nonspecific clinical features. Imaging combined with fine-needle biopsy remains the most reliable diagnostic strategy. Antitubercular therapy usually ensures good outcomes, though surgery may be indicated in selected cases.

References

1. Hickey, A.J., Gounder, L., Moosa, M.Y.S. et al. A systematic review of hepatic tuberculosis

with considerations in human immunodeficiency virus co-infection. BMC Infect Dis 15, 209 (2015).
2. Wu Z, Wang WL, Zhu Y, Cheng JW, Dong J, Li MX, Yu L, Lv Y, Wang B. Diagnosis and treatment of hepatic tuberculosis: report of five cases and review of literature. Int J Clin Exp Med. 2013;6(9):845-50.
3. Akkahadsee P, Sawangjit R, Phumart P, Chaiyakunapruk N, Sakloetsakun D. Systematic review and network meta-analysis of drug-induced liver injury prevention in TB therapy. Sci Rep. 2023;13(1):19880.
4. Zheng SM, Lin N, Tang SH, Yang JY, Wang HQ, Luo SL, et al. Isolated hepatic tuberculosis with portal vein thrombosis and HBV coinfection. World J Clin Cases. 2021;9:9310–9.
5. Varshney P, Kumar Kapoor V. Hepatopancreatobiliary tuberculosis: a review. Turk J Surg. 2024;40(2):95-103.
6. Niyogi D, Goel M, Shinde RS, Patkar S. Primary hepatic tuberculosis: a rare event. Ann HPB Surg. 2019;23(1):80.
7. Tobin EH, Tristram D. Overview of tuberculosis. StatPearls Publishing; 2025.
8. Bahurupe S, Dhote S, Phatak S, Mitra K, Onkar P. Liver Tuberculosis Presenting As Fever of Unknown Origin. Cureus. 2023;15(10):e47889.
9. Hu N, Wu Y, Tang M, Luo T, Yuan S, Li C, Lei P. Case report: Hepatic TB mimicking HCC in HBV cirrhosis. Front Med. 2022;9:1005680.
10. Johnkennedy N, Abiodun AE, Ifeoma UH, Hope O, Ikechukwu NE, Nnedimma NC. Alterations in hepatic function biochemistry in TB patients on therapy. Indian J Med Healthc. 2012;1:12–5.