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Progressive Jaundice As An Unusual Presentation Of Hepatosplenic Tuberculosis: A Case Report

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Abstract:
Hepatosplenic tuberculosis is a rare form of extrapulmonary tuberculosis that can present with nonspecific clinical features, making diagnosis challenging. We present a case of a middle-aged patient with progressive jaundice and constitutional symptoms, initially suspected to have a hepatobiliary malignancy. Further investigation including imaging and histopathology confirmed the diagnosis of hepatosplenic tuberculosis. The patient responded well to standard anti-tubercular therapy. This case highlights the importance of considering tuberculosis in the differential diagnosis of unexplained hepatosplenic lesions, especially in endemic regions.

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I. Introduction:

Tuberculosis (TB) continues to represent a major global health challenge, with extrapulmonary forms accounting for approximately 20% of all reported cases [1]. According to the 2021 World Health Organization (WHO) Global Tuberculosis Report, TB remains the leading cause of death from a single infectious agent worldwide [2,3].

Among extrapulmonary sites, hepatosplenic tuberculosis is exceptionally rare and often underrecognized due to its nonspecific clinical manifestations and radiological overlap with other granulomatous, inflammatory, or neoplastic diseases. Presentation with jaundice as a predominant symptom is particularly uncommon, further complicating timely diagnosis.

Herein, we report a case of hepatosplenic tuberculosis presenting primarily with progressive jaundice and constitutional symptoms, confirmed through radiological assessment and histopathological evaluation.

II. Case Report:

A 29-year-old patient with no known medical or surgical history presented with progressive jaundice over a two-month period, accompanied by intermittent abdominal pain and weight loss of approximately 9 kilograms over the preceding 2–3 months.

On physical examination, the patient was icteric, with palpable hepatosplenomegaly. There was no evidence of peripheral lymphadenopathy or stigmata of chronic liver disease.

Laboratory investigations revealed elevated liver enzymes consistent with cytolysis (AST 78 U/L, ALT 92 U/L) and a cholestatic pattern of injury, with total bilirubin of 6.5 mg/dL (direct fraction 4.2 mg/dL), alkaline phosphatase (ALP) 420 U/L, and gamma-glutamyl transferase (GGT) 130 U/L. An inflammatory response was evident, with C-reactive protein (CRP) at 42 mg/L, and leukopenia was noted (2,900/mm³). The prothrombin ratio, serum albumin, protein electrophoresis, and alpha-fetoprotein levels were within normal limits.
Serologic testing for viral hepatitis (HBV, HCV, HAV, HEV) and HIV were all negative.

Abdominal Ultrasonography and Contrast-enhanced CT showed a liver span of 156mm with heterogeneous parenchyma containing multiple hypoechoic areas. Examination of the spleen showed an increased craniocaudal length of 132mm with similar findings to that of the liver (figure 1). Suspicion of malignancy or infection was reported and exclusion of possible tuberculosis (TB) or sarcoidosis was mandated.
Chest radiography and CT scan and were normal.
An ultrasound-guided core needle biopsy was performed and submitted for pathological examination.

Histopathology revealed granulomatous inflammation with caseous necrosis. Ziehl-Neelsen staining revealed acid-fast bacilli and GeneXpert Detected Mycobacterium tuberculosis without rifampicin resistance. A histopathological diagnosis of Hepatosplenic TB was made.

The patient was started on standard first-line anti-tubercular therapy (HRZE – Isoniazid, Rifampicin, Pyrazinamide, Ethambutol) with liver function monitoring.

Within 4 weeks of therapy, the patient showed clinical improvement with reduction in jaundice and systemic symptoms. Follow-up LFTs showed decreasing bilirubin levels. Imaging at 3 months showed regression of hepatic and splenic lesions.

III. Discussion:

Hepatic and splenic involvement in tuberculosis is uncommon and most frequently encountered in disseminated or miliary forms of the disease, particularly among immunocompromised individuals, such as those with concomitant HIV infection. [4-5] Isolated hepatic tuberculosis is hypothesized to originate from a minute intestinal tuberculous focus that gains access to the liver through the portal venous system. [5]

Reed et al. [6] classified hepatic tuberculosis into three distinct morphological patterns: (1) hepatic tuberculosis associated with generalized miliary dissemination—the most prevalent form, observed in approximately 50–80% of patients succumbing to pulmonary tuberculosis; (2) primary miliary tuberculosis of the liver; and (3) localized tuberculoma or tuberculous abscess of the liver. Splenic tuberculosis, analogous to hepatic involvement, may present either as a manifestation of disseminated disease or as an isolated entity. Pathomorphologically, splenic tuberculosis has been categorized into five types: miliary, nodular, tuberculous abscess, calcific, and mixed forms. [7]

The diagnosis of hepatic and splenic tuberculosis is often challenging, as the clinical presentation is typically nonspecific and many patients lack a documented history of prior tuberculosis exposure. Early recognition is essential, as the mortality rate associated with untreated abdominal tuberculosis can reach up to 50%. [8]

Imaging characteristics of hepatosplenic tuberculosis are non-specific and frequently overlap with those of metastatic disease, lymphoproliferative disorders, and other granulomatous conditions such as sarcoidosis or fungal infections. Definitive diagnosis generally requires histopathological or microbiological confirmation, which may necessitate tissue acquisition via image-guided biopsy or laparotomy, particularly for small or indeterminate lesions. Histopathological examination typically demonstrates granulomatous inflammation with central caseous necrosis, surrounded by epithelioid histiocytes, Langhans-type giant cells, and lymphocytic infiltration. [4]

Antitubercular therapy remains the mainstay of treatment, with surgical intervention reserved for select, refractory, or diagnostically challenging cases. [7-8].

Our present case underscores the importance of considering tuberculosis as a differential diagnosis in endemic regions, even in atypical clinical scenarios such as jaundice, particularly when imaging reveals hepatosplenic lesions accompanied by lymphadenopathy.

IV. Conclusion:

Hepatosplenic tuberculosis is a rare and often misdiagnosed condition. Progressive jaundice due to periportal TB lymphadenitis is an uncommon but important presentation. Early diagnosis and prompt anti-tubercular therapy can result in excellent outcomes.

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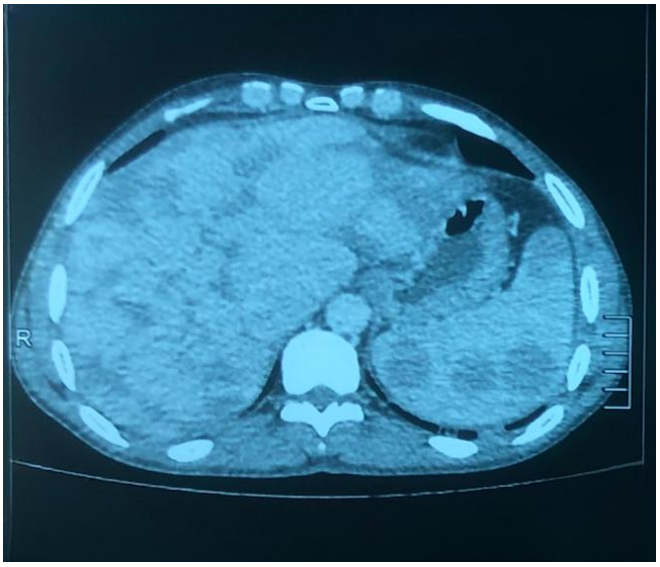

Figure 1: Abdominal Contrast-enhanced CT showing multiple hypodense lesions in the liver and spleen.