

Isolated Hepatic Tuberculosis in an Adolescent

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ABSTRACT

We report a case of a 15-year-old male presenting with persistent epigastric abdominal pain, significant weight loss, lack of appetite, and nausea over a span of 6-7 months. Clinical examination revealed pallor and splenomegaly, while laboratory investigations indicated microcytic anemia and elevated inflammatory markers. Imaging with ultrasound and contrast-enhanced computed tomography (CT) identified multiple ill-defined hypodense lesions in the liver, enlargement of the spleen, and retroperitoneal lymphadenopathy. Despite the absence of pulmonary symptoms and negative results for Acid Fast Bacilli (AFB), a diagnosis of primary hepatic miliary tuberculosis was established based on elevated serum Adenosine Deaminase levels and a positive Mantoux test. Antitubercular therapy was initiated, resulting in significant clinical improvement and normalization of laboratory values within three months. This case underscores the importance of considering extrapulmonary tuberculosis in adolescents with atypical abdominal symptoms, emphasizing the necessity for thorough evaluation and timely intervention to achieve favorable outcomes.

Keywords: abdominal pain; diagnostic imaging; extrapulmonary tuberculosis; hepatic lesion; isolated hepatic tuberculosis.

INTRODUCTION

Abdominal Tuberculosis (TB) includes involvement of the gastrointestinal tract, peritoneum, lymph nodes, solid organs.^{1,2} Abdominal TB comprises around 5 percent of all cases of TB worldwide whereas liver TB accounts for less than 1% of all tuberculous infections. It is rare due to low tissue oxygen tension in the liver, which is unfavorable for mycobacterial growth.^{3,4} Hepatic tuberculosis has two presentations: military hepatic disease and isolated hepatic disease. Liver involvement in miliary TB occurs in approximately 80 percent of cases of disseminated TB; where TB reaches the liver via the hepatic artery. Isolated hepatic TB occurs in approximately 20 percent of cases; where, TB reaches the liver via the portal vein or gastrointestinal lymphatics or may represent reactivation of latent TB infection.⁴ In endemic regions, hepatic TB can present in the form of hepatic abscess, as an intrahepatic mass, or as granulomatous hepatitis.⁵

CASE PRESENTATION

A 15-year-old male presented to our Tertiary Center with abdominal pains for the past 6-7 months. The pains were localized in the epigastric region, gradually developed, and dull in nature. They were not radiating but were accompanied by 4 to 5 kg weight loss. The patient also reported lack of appetite and nausea. There was no history of fever, cough, vomiting, diarrhea, dysuria, or night sweats. The patient had no previous medical history and no family history of tuberculosis.

On examination, the patient appeared pale, pulse 110 bpm, temperature 96°F, RR 14 pm, and SpO2 98% on room air. The patient had a body weight of 30 kg. On examination of the abdomen, the spleen was palpably enlarged, while the liver was normal in span and non-tender. There was no sign of jaundice or ascites. No lymph nodes were palpable.

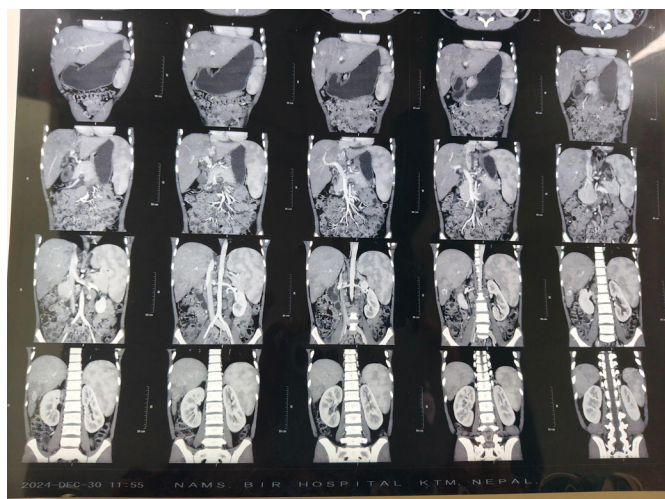


Figure 1. Coronal section of CECT showing section of abdomen.

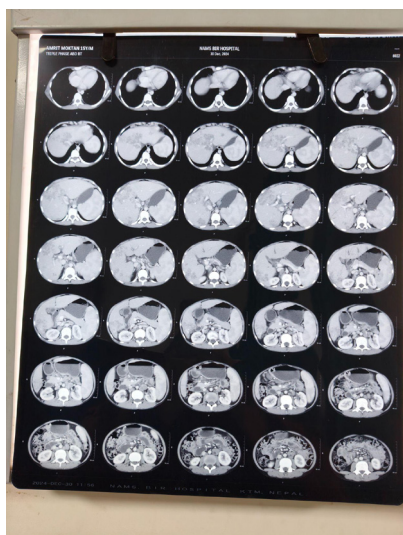


Figure 2. Axial section of abdomen showing hepatic lesions.

A complete blood count revealed microcytic anemia (Hemoglobin = 10.5 g/dL, MCV = 69), while the Erythrocyte Sedimentation Rate (ESR) was 50 mm/first hour. Further studies revealed Aspartate Transaminase at 44, Alanine Transaminase at 16. Alkaline Phosphatase was at a high level of 458 IU/L, with total protein of 8.5. Urea and creatinine were normal (14 and 0.4, respectively); sodium was low (130), while potassium was normal (4.1).

Ultrasonography (USG) findings showed an ill-defined heterogeneous hypoechoic lesion in segment V/VI of the right lobe of the liver, which suggests a very early hepatic abscess. The radiologist recommended a Triphasic Contrast-Enhanced CT Study of the abdomen with a hepatic protocol for further exploration. The abdominal USG also showed thickening of the gallbladder wall, splenomegaly of 14.40 cm, and increased periportal echogenicity/cuffing. Subsequently, the pediatrician ordered the serum

Adenosine Deaminase which was 85 U/L, suggestive of tuberculosis, although a chest X-ray was normal.

Subsequently, a Contrast-Enhanced Computed Tomography (CECT) of the abdomen and pelvis with hepatic protocol was ordered which confirmed mild ascites, splenomegaly (splenic index of 1080), and mesenteric and retroperitoneal lymphadenopathy (some calcified). In both lobes of the liver, Multiple ill-defined hypodense lesions (micronodular) were noted, of which the largest coalescing lesion appeared to measured 67×49×48 mm (Tr×AP×CC) in segment V, with no foci of calcifications. In post-contrast study, there is mild enhancement in the portal venous phase noted. A few calcified lesions, probably calcified granulomas, were identified in the liver, the largest of which is measuring 21×20×15 mm (AP×CC×Tr) in segment VIII. No filling defects are seen in the celiac artery, hepatic artery, superior mesenteric artery, splenic artery, splenic vein, or portal veins.

The radiologist concluded his CT report by mentioning likely features of hepatic tuberculosis. Following the CT findings, the Mantoux Test was done and gave a positive result of 18 mm after 72 hours. However, both Acid-Fast Bacilli (AFB) microbiology reports were negative, confirming the absence of pulmonary TB. Diagnosis of Primary Hepatic Miliary TB has been made.

Anti-Tuberculosis Treatment (ATT) was initiated with a quadruple regimen of isoniazid, rifampicin, pyrazinamide, and ethambutol. After roughly one month of treatment, the symptoms had improved, the patient reported good appetite and had gained almost 5 Kg of weight from the baseline of toilet elegance. The laboratory tests were back to normal after three months of treatment.

DISCUSSION

HTB is classified into three types: miliary TB from generalized infection, primary hepatic miliary TB, and the rarest nodular lesion tuberculoma.^{6,7}

The liver and biliary system are involved mainly due to miliary lung tuberculosis. Isolated primary TB of the liver is rare. Liver and spleen involvement is said to have a frequency of involvement between 80 and 100%.⁸ The main route of spread of the bacillus to the liver in the case of tuberculoma is through the portal vein, whereas it is through the hepatic artery in miliary TB.¹⁴

The most common clinical manifestations of hepatic TB are fever, malaise, abdominal pain, distention, and weight loss. Splenomegaly, hepatomegaly, ascites, and rarely jaundice are other signs. Furthermore, acute respiratory symptoms (cough and sputum production) can occur in the case of miliary hepatic TB.⁹

Hypoechoic or complex lesions and calcifications characterize the ultrasound findings of hepatic TB, while

hypodense or calcified lesions on CT with or without peripheral rim or central enhancement characterize the CT findings in hepatic TB.⁹ The characteristics of hepatic TB detected using MRI included hypointense on T1WI and hyperintense on T2WI, with or without the said central enhancement. Mass-like lesions usually show heterogeneous enhancement with necrosis on CT and MRI.¹⁰

With more accurate assessment and improved sampling, liver biopsy comes up as a validated diagnostic parameter. However, this is not always deemed necessary, since microbiological and histological findings may provide a more accurate diagnosis. Intermittent elevation of ALT (normal range: 0–200 U/L) and aspartate transaminase (typical range: 0–200 U/L) is common. Usually, higher levels of ALT and AST are seen in jaundiced patients. A systematic review by Hickey AJ et al. showed that the most frequent abnormalities found in hepatic TB include ALP (normal range: 200–750 U/L) and GGT (normal range: 100–400 U/L). In our case report, jaundice did not develop in the patient.¹¹

The duration of treatment has been established by the National Institute for Health and Clinical Excellence (NICE) guidelines from the United Kingdom, American Thoracic Society (ATS) CDC, and IDSA all of which recommend

six (6)-month treatment. This includes a 2-month intensive phase of treatment with isoniazid, rifampicin, pyrazinamide, and ethambutol or streptomycin followed by a 4-month period of continuation with isoniazid and rifampicin.^{12,13}

This case emphasizes the need for heightened clinical awareness of extrapulmonary tuberculosis, especially in adolescents, where atypical symptoms and non-specific laboratory findings may delay diagnosis. It serves as a reminder of the critical integration of clinical acumen, appropriate diagnostic imaging, and targeted therapy in improving patient outcomes in cases of suspected infectious etiology affecting the liver. Regular follow-up will be essential to monitor any potential complications and to ensure the complete resolution of the disease.

CONCLUSIONS

Patient presenting with atypical abdominal symptoms should also raise high suspicion of isolated hepatic tuberculosis emphasizing the need for timely evaluation for favourable outcome.

Conflict of Interest: None

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