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Case Report

Case report on serohepatic tuberculosis (frosted liver): Clinical presentation, imaging, diagnosis, and management ☆,☆☆,★

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ABSTRACT

Hepatic tuberculosis can manifest in various forms, including parenchymal, serohepatic, tuberculous cholangitis and mixed form. Isolated hepatic tuberculosis, specifically in the form of serohepatic tuberculosis, is very rare. Patients with hepatic tuberculosis often present with nonspecific symptoms such as abdominal pain, weight loss, night fever, night sweats, hepatomegaly, and abnormal liver function tests. This case involves a young male with isolated serohepatic tuberculosis who presented to the outpatient department of a tertiary care center with complaints of abdominal discomfort, weight loss, and evening rise in temperature. His liver function tests showed elevated levels of alanine transaminase and aspartate aminotransferase. Ultrasonography of the abdomen revealed multiple subcapsular necrotic lesions in the right lobe of the liver. A contrast-enhanced computed tomography scan of the abdomen showed a few hypodense subcapsular lesions in the right lobe and a minimal subcapsular collection. There was mild thickening and enhancement of the liver capsule and sub capsule, creating a frosted liver or sugar-coated appearance. A small subcentimetric size parenchymal lesion was present in segment VIII, which was in continuity with the subcapsular collection. Fine needle aspiration cytology from the largest subcapsular liver lesion revealed acid-fast bacillus, confirming the tuberculosis diagnosis. A high-resolution CT scan of the chest was performed for further evaluation and showed no abnormalities. The patient is currently being treated with antitubercular therapy.

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Key Clinical Message

Isolated serohepatic tuberculosis is very rare. A frosted liver appearance is seen in computed tomography/ magnetic resonance imaging scans of serohepatic tuberculosis. It is essential for radiologists to maintain a high degree of suspicion and be familiar with the imaging findings of serohepatic tuberculosis for accurate diagnosis.

Introduction

Tuberculosis (TB), caused by an acid-fast bacillus (AFB), mycobacterium tuberculosis is a common infection in developing countries and a re-emerging infection in developed world due to global migration and human immunodeficiency virus (HIV) infection [1]. Hepatic tuberculosis is a form of abdominal tuberculosis, seen in less than 1% of TB infection [2]. Isolated hepatic TB, let alone serohepatic tuberculosis is rare and sparsely documented [3]. Hepatic TB can be seen in one of four different forms, that is parenchymal type, serohepatic type, tuberculous cholangitis and mixed type [4]. Parenchymal tuberculosis can further be classified as micronodular type (nodule size: 0.5-2cm), macronodular type (nodule size > 2cm) and mixed type. Clinical presentation of hepatic TB is very nonspecific like vague abdominal pain, night fever, weight loss, night sweat, hepatosplenomegaly and deranged liver function test [5]. The imaging characteristics of hepatic tuberculosis are subtle and nonspecific. Because these features can overlap with those of various benign and malignant liver lesions, hepatic tuberculosis is frequently misdiagnosed or classified as an indeterminate lesion. Confirmation often requires histopathological and bacteriological diagnosis.

Clinical history and investigations

A 17 years old, otherwise healthy male presented to the outpatient department of a tertiary center with complaints of vague abdominal discomfort and pain for 6 weeks. The pain was dull aching type with no specific localization. There were no aggravating and relieving factors. He also complains of evening rise in temperature. The fever was documented with maximum temperature 101.4-degree Fahrenheit. The fever was not associated with chills and rigor, however associated with significant diaphoresis. The patient complaints of significant weight loss (~ 8 kilogram) in 6 months. He noticed the weight loss because his clothes, shoes and finger rings were loosening. Blood investigation was done which revealed elevated C reactive protein (6.2 mg/L) and erythrocyte sedimentation rate (53 mm/hr). Liver enzymes levels in blood were elevated. Aspartate transaminase and alanine transaminase were 52 U/L and 62 U/L respectively. An ultrasonography (US) abdomen was done which revealed few subcapsular hypoechoic lesions in right lobe, largest measuring ~ 24.2×9 mm (Fig. 1). The lesions showed central liquefied contents within. A contrast computed tomography (CT) chest was performed which showed a subcentimetric size hypodense lesion in segment VIII (Fig. 2A). The lesion is in continuity with subcapsular collection (Fig. 2B). few hypodense lesions with no significant enhancement in right lobe (Fig. 2C). Minimal subcapsular collection was present and there was mild hyperenhancement and thickening of capsule and sub capsule of liver (Fig. 2D).

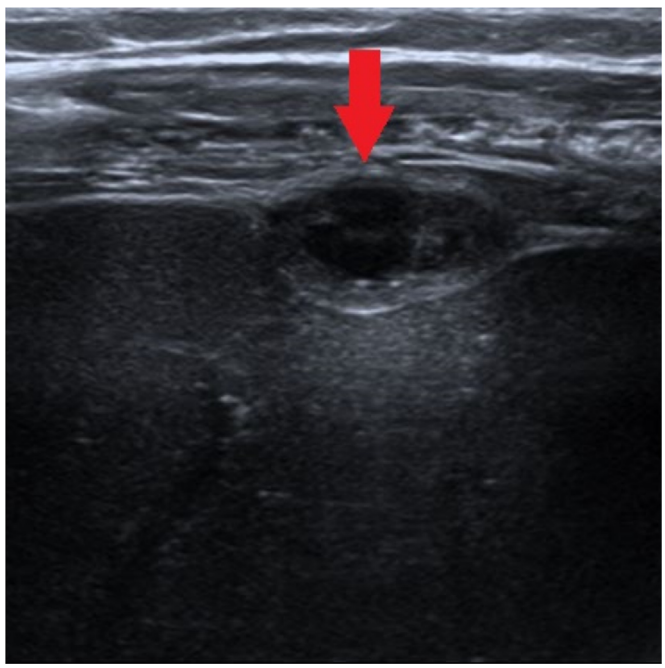

Fig. 1 – High-resolution ultrasound picture of the liver showing a subcapsular hypoechoic lesion (red arrow). The lesion exhibits necrosis and posterior acoustic enhancement.

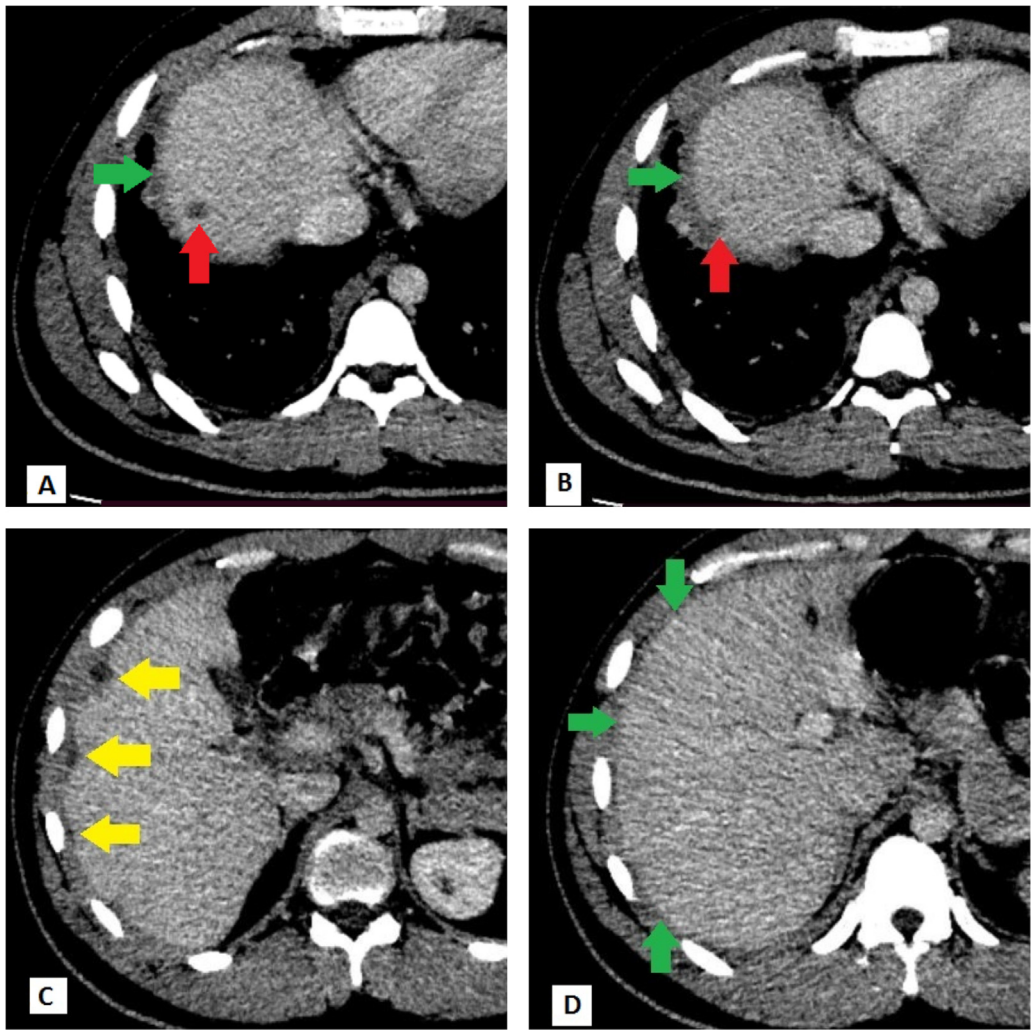

Fig. 2 – Contrast CT images of the liver. (A) shows a subcentimetric hypodense lesion (red arrow) in segment VIII of the liver and a minimal subhepatic collection (green arrow). (B) shows the hypodense lesion (red arrow) in continuity with the subcapsular collection, suggesting rupture of the lesion. (C) shows a few subcapsular hypodense lesions (yellow arrows) in the right lobe of the liver. (D) shows minimal subcapsular collection, mild thickening, and enhancement of the liver capsule and sub capsule.

Differential diagnosis

On the basis of history and clinical examination, following differential diagnosis was considered.

1) Serohepatic tuberculosis
2) Bacterial/ Fungal micro abscess
3) Metastasis
4) Sarcoidosis
5) Lymphoma

Diagnosis and treatment

A fine needle aspiration cytology of the largest subcapsular lesion was done. Pus was aspirated from the lesion. In cytology, AFB were detected (Fig. 3) confirming the lesion to be tubercular. High resolution CT chest was performed to look for any lung involvement. No tuberculous lesions were seen in lung and mediastinum (Fig. 4). Treatment of hepatic tuberculosis is done in the line of extrapulmonary tuberculosis. According to national tuberculosis guideline; Isoniazid (H), Rifampicin (R), Pyrazinamide (Z), Ethambutol (E) are given for 2 months and Isoniazid and Rifampicin for 4 months. Our patient is on antitubercular therapy and has completed 1 month of HRZE with no signs of drug toxicity.

Discussion

Liver is an unfavorable site for TB infection due to low tissue oxygen tension [6]. It can be seen in any age group, but mostly detected in young adults. It may occur primarily or as part of disseminated disease, in which miliary tuberculosis is seen. The route of transmission of disseminated disease is hematogenous, through hepatic artery [2]. The route of

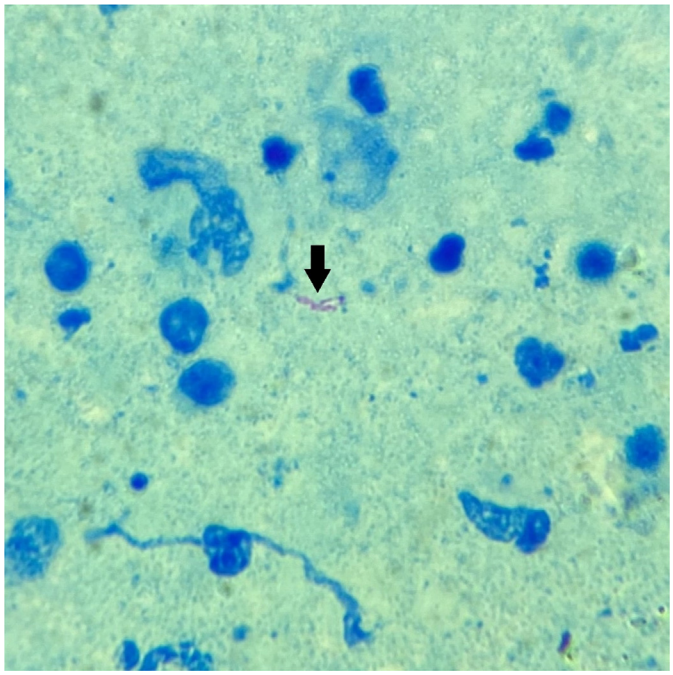

Fig. 3 – Ziehl-Neelsen stain of aspirate from the subcapsular lesion of the liver shows acid-fast bacilli (black arrow).

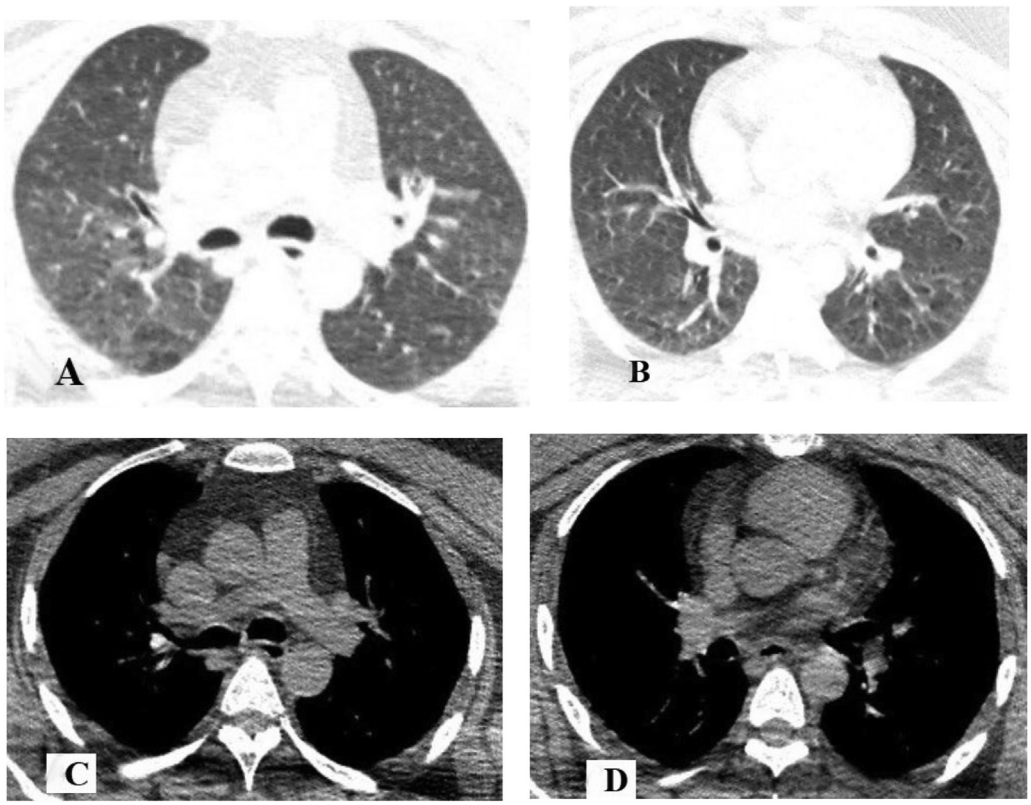

Fig. 4 – A high-resolution CT of the chest was performed to rule out tuberculous lesions in the chest and mediastinum. No obvious tuberculous lesions were seen in the lungs and mediastinum.

transmission of localized disease is portal vein [2]. It is usually associated with gastrointestinal tract TB. Sometimes, lymphatic spread is seen through infected portal lymph nodes or rupture of lymph nodes along portal tract. Hepatic tissue responds by formation of granuloma. Both caseating and noncaseating granulomas are present. The clinical features of hepatic TB are nonspecific. In most of the cases, they are seen as part of disseminated disease. So, pulmonary symptoms are first to be noticed. Isolated hepatic TB may present with vague abdominal discomfort, right upper quadrant pain, evening rise in temperature, night sweat and weight loss. Sometimes, there is biliary obstruction resulting jaundice. Mild hyperbilirubinemia is seen in diffuse hepatic involvement, which is seen in 35% of cases [7]. Elevated liver enzymes, C reactive protein and erythrocyte sedimentation rate are seen in most of the cases with diffuse liver involvement.

Imaging wise, hepatic tuberculosis can be sub classified into parenchymal type, serohepatic type, tuberculous cholangitis type and mixed type. Parenchymal tuberculosis can further be subclassified into micro and macro nodular type. Micronodular TB occurs as a part of miliary tuberculosis. Multiple 0.5-2 cm size hypodense nodules are seen in liver parenchyma. More often than not spleen is also involved. Sometimes, nodule may not be apparent in CT and due to diffuse involvement of liver and spleen, hepatosplenomegaly or diffusely heterogeneous attenuation of liver/ spleen may be present [7]. High resolution US is more sensitive than contrast CT or magnetic resonance imaging (MRI) for detecting small nodules. Differentials of this pattern of hepatic involvement are granulomatous disease, lymphoma, metastasis and fungal infection. In macro nodular TB, which is a localized form, a hypodense nodule/ tuberculoma/ tuberculous abscess of size $>$ 2.0 cm is seen in liver [7]. It is less commonly associated with pulmonary TB. It is often misdiagnosed as pyogenic/ amoebic abscess or metastasis. Findings of serohepatic tuberculosis are classically described as a frosted liver or sugar-coated appearance (classically in T2 weighted image in MRI) [7]. There is a mild subcapsular collection in the liver with a few subcapsular hypodense lesions, and thickening and enhancement of the liver capsule and sub capsule [7,8]. Scalloping of liver is seen because of subcapsular lesions. We postulate that the imaging appearance of serohepatic tuberculosis is due to the subcapsular rupture of a tubercular abscess. The hypodense subcapsular lesions are presumably localized tuberculous pus collections. In our case, a small parenchymal lesion, approximately 4×3 mm in size, was seen in segment VIII. The lesion was in continuity with the subcapsular collection. The subcapsular collection and subcapsular lesions are presumably pus collections following the subcapsular rupture of the parenchymal lesion in segment VIII. The rupture likely occurred quite some time ago. The capsular and subcapsular thickening and enhancement are due to chronic inflammation. Tuberculous cholangitis is seen as areas of multifocal strictures in intrahepatic biliary radicals, miliary calcification along biliary radicals and associated parenchymal atrophy [7,9]. The extrahepatic biliary tracts are uncommonly involved, if involved, there is thickening and irregularity of biliary ducts. Alternet areas of biliary stricture and dilatation mimicking sclerosing cholangitis and IgG4 related cholangitis maybe seen [9]. Sometimes, extrahepatic biliary obstruction may be present due to extrinsic compression by enlarged periportal lymph nodes. Mixed form of all these hepatic tuberculosis may also be seen with overlapping features of all forms.

The above-mentioned findings of hepatic TB may be associated with findings of tuberculous involvement of other organs in abdomen and thorax. Calcification and necrosis of lymph nodes are very specific of tuberculosis [2]. Periportal/ upper retroperitoneal lymphadenopathy, necrotic/calcified lymph nodes, calcification of hepatic/splenic capsule, peritoneal thickening/ nodularity/enhancement, omental caking/nodularity and ascites. Co-existing pulmonary tuberculosis or gastrointestinal tuberculosis is seen in majority of the patients.

Histopathology is the gold standard for diagnosis of hepatic TB [10]. Other old school investigations like tuberculin test, sputum test for AFB, body fluid adenosis deaminase test, sputum culture in Löwenstein–Jensen medium may support the diagnosis.

Origin of Figures 1-4: Origin: Grande International Hospital, Kathmandu, Nepal

Conclusion

Isolated serohepatic TB is extremely rare with very few case reports. Imaging findings are subtle and require high degree of suspicion and expertise. Frosted liver of sugar-coated liver is the term classically used to describe this form of hepatic tuberculosis. Serohepatic TB should be considered as a differential diagnosis when subcapsular lesions or collection are seen in liver and in cases where there is thickening and enhancement of liver capsule and sub capsule.

Data availability statement

The data used in the manuscript will be available for review by the editor-in-chief of this journal if requested.

Author contributions

Prajwal Dahal: Conceptualization, manuscript writing, supervision. **Alina Awale:** Histopathological evaluation. **Sabina Parajuli:** Software, manuscript writing.

Patient consent

A written consent was obtained from the patient for publication of the case report and accompanying images. A copy of written consent will be available for review by the editor-in-chief of this journal if requested.

Ethics Statement

The need for ethical approval was waived and consent from patient was deemed sufficient.

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