

Case Report

An unusual case of tubercular liver abscess presenting as pericardial effusion

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ABSTRACT

Tropical nations like India frequently have liver abscess. They are typically amoebic or pyogenic abscesses. It can sporadically be a sign of hepatic tuberculosis (TB). Hepatic tuberculosis should be suspected in cases where the patient doesn't present with typical complaints of pyogenic/amoebic abscess without response to commonly used antibiotics. Pyogenic/amoebic liver abscesses show the rare complication of rupture into the pericardium, especially if the abscess is left sided. However, a tubercular pericardial effusion presenting with rupture into the pericardium has rarely been reported. We present to you a case of tubercular pericardial effusion in a known diabetic patient secondary to ruptured liver abscess presenting to the emergency department with chief complaints of breathlessness, chest pain and bilateral pedal edema.

Keywords: Tubercular liver abscess, Pericardial effusion, Breathlessness

INTRODUCTION

The majority of the time, tubercular liver abscesses are linked to immunosuppression, with the lungs or digestive tract serving as the primary sites of infection. It is uncommon for liver tuberculosis to manifest as pseudo tumoral masses without active pulmonary or military tuberculosis.¹ Concomitant involvement of the pericardium with hepatic tuberculous abscesses in the absence of pleuropulmonary and peritoneal involvement has been reported very few times in medical literature before.²

CASE REPORT

70-year-old male patient presented to the emergency department with complaints of breathlessness since 1 day, bilateral pedal edema since 1 day, fever and abdominal pain since 5 days. He gives history of chronic smoking since 40 years – pack years of 40 and chronic alcohol intake since 40 years, having stopped both his habits 2 years ago. He is a known diabetic since 10 years, on oral

hypoglycaemic agents. On presentation, the patient maintained a saturation of 98% at 10 litres of O₂, pulse rate of 110 bpm, blood pressure of 100/60 mm of Hg. On auscultation he has normal vesicular breath sounds bilaterally except basal lung fields where breath sounds were diminished. However, he has muffled heart sounds. On abdominal examination – he has tenderness in the right hypochondrium without any palpable mass. His chest X-ray showed left sided mild pleural effusion.

Patient immediately underwent an electrocardiography (ECG) examination, which showed ST elevation in V2-V6 leads and his serial ECG was no dynamic ST-T changes. There were universal low voltage complexes. He underwent 2D ECHO studies which showed moderate pericardial effusion with ejection fraction 60% without wall hypokinesia. He underwent pericardial sheath needle insertion and there was an immediate gush of yellow turbid fluid of 300 cc volume with dramatic improvement of the patient's vital signs. The pericardial sheath needle was left in situ to allow continuous drainage.

Meanwhile, patient's laboratory findings were as follows: Hb :10.2 g%, white blood cell (WBC) count: 16.3k (N>L), platelets: 4.3 L, SGOT/SGPT: 640/430 IU/l, ALP: 227 IU/l, total bilirubin: 1.5 mg % (direct/indirect = 0.7/0.8), and erythrocyte sedimentation rate (ESR): 45.

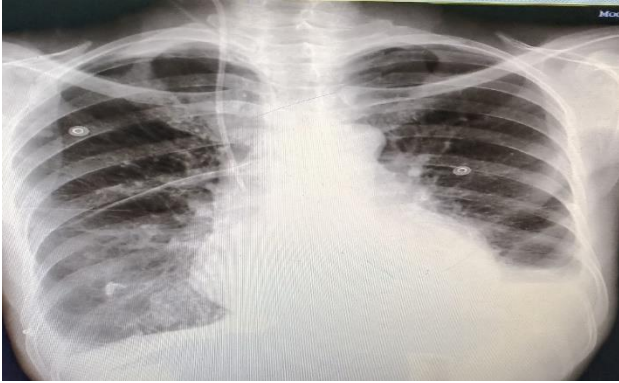


Figure 1: Chest X ray PA view on admission showing left pleural effusion. Note that central venous line is in situ in the internal jugular vein.

An ultrasonography (USG) abdomen pelvis was done which showed solitary liver abscess with subdiaphragmatic collection. Contrast enhanced computed tomography (CECT) abdomen pelvis with intravenous contrast showed an unliquified solitary collection in segment 8 of the liver with capsular rent of 3.3 mm and communication of the collection with the pericardial cavity.

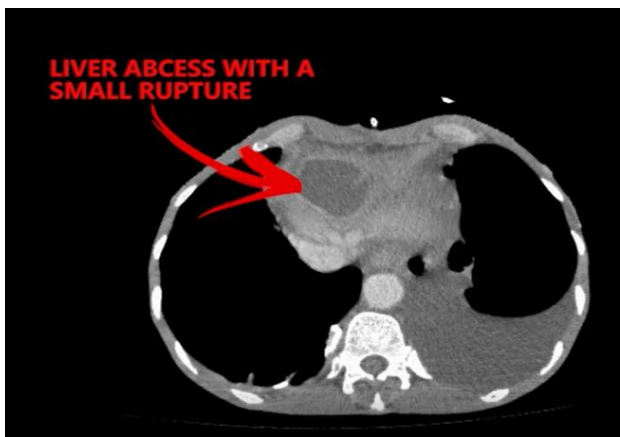


Figure 2: Showing liver abscess with capsular breach.

The pus drained from the pericardium was sent for a battery of investigations: WBC count: 4.3k, protein: 1245 mg%, sugar: 124 mg/dl, and bacterial and viral culture sensitivity: negative screen.

The pericardial sheath drain was kept in situ to allow for continuous drainage. Liver abscess was deemed unliquified to tap. Right sided mild pleural effusion was also aspirated and sent for studies which proved the effusion to be exudative in nature without any organism being cultured.

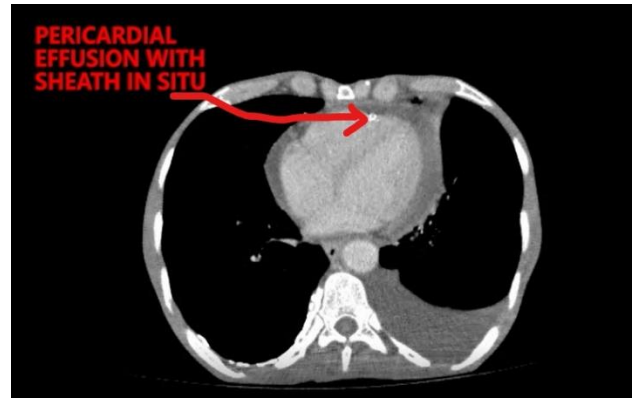


Figure 3: CT thorax showing left pleural effusion with moderate pericardial effusion. Note that pericardial sheath drain is in situ.

Patient was started on empirical antibiotics –treatment started with metronidazole 800 mg thrice a day, injection ciprofloxacin 200 mg twice a day. Fluid resuscitation done as per central venous pressure monitoring. However, the patient showed no improvement with the above mentioned antibiotics. The patient continued to have fever with no cough or expectoration. He continued to have a pericardial output of 60 ml/day due to clinical suspicion, the pericardial sheath pus sample was again sent for Ziehl Neelson (ZN) staining and polymerase chain reaction (PCR). ZN staining came negative for acid fast bacilli.³ However, on polymerase chain reaction, Mycobacterium tuberculosis⁴ was detected. Final diagnosis of tubercular liver abscess was made.

Patient started on hepatoprotective antitubercular drugs-streptomycin 0.75 gms intramuscular OD, levoflox 500 mg OD and ethambutol 800 mg OD. Daily pericardial sheath output monitoring done.



Figure 4: Chest X ray PA view on the day of discharge.

Review ultrasound s/o liquified liver abscess 22 cc collection and USG guided aspiration done.⁶ On continued treatment the pericardial sheath output came to be zero. Review 2 D ECHO studies showed resolution of pericardial effusion. Patient has responded well to

treatment. Liver function test was normal and gradually shifted him to oral antitubercular drugs category I. Patient was discharged in stable condition after removal of the pericardial sheath drain.

DISCUSSION

Hepatic tuberculosis has been classified as a rare form of extrapulmonary tuberculosis. TLA was initially described by Bristowe in 1858.⁷ The majority of the cases reported often involved miliary TB and spread mostly through hematogenous means. The hepatic artery or the portal vein carried bacteria to the respiratory and gastrointestinal tracts, which were the main sources of illness.⁸

Levine divided hepatic TB into a number of presenting types, including tuberculosis of the miliary, primary pulmonary tuberculosis with liver involvement, primary liver tuberculosis, tuberculoma, and tuberculous cholangitis.^{9,10}

According to a study where patients with hepatic TB ranged in age from 6 months to 72 years with an average age of 39.2 years, the prevalence of TLA was just 0.34%. The illness's symptoms are frequently unclear and include fever, nagging stomach pain, anorexia, and weight loss. Hepatomegaly is a typical physical observation. An extremely uncommon TLA symptom, jaundice may be brought on by extra- or intrahepatic blockage. Jaundice and the extent of liver damage are not directly correlated. Hepatoma, pyogenic liver abscess, and amoebic liver abscess are frequently confused with TLA.¹¹ The diagnosis of TLA is typically made at autopsy or sporadically after a laparotomy due to the non-specific clinical presentation.¹²

Our patient was treated on the lines of amoebic liver abscess because of the endemicity of the condition in the Indian subcontinent and a negative screen for bacteria and virus. Amebiasis is endemic in India, and antibodies can develop even in *Entamoeba histolytica* non-invasive infections and last for years after a clinical cure. Because of this, amoebic serology data may be inaccurate, and if a high index of clinical suspicion is not maintained, valuable therapeutic time may be lost.

TLA radiological findings are not very specific. Findings from computed tomography (CT) and ultrasound (USG) scans typically show distinct stages of illness, from granulomatous tubercles to fibrosis and calcification in the healing stage. Although rare studies have shown hyperechoic lesions as well, USG findings of hepatic tuberculosis typically show hypo-echoic lesions, as was reported in our case. The final determination of TLA's diagnosis therefore hinges on the presence of AFB in pus, aspirate or biopsy specimens, necrotic tissue, or both.

AFB is more frequently seen in caseous necrotic material, but other studies have shown that even the absence of AFB should not prevent a diagnosis, particularly in a nation with a high TB incidence rate like ours. Recent studies have

shown that PCR may quickly identify *Mycobacterium TB* and speed up the choice to start therapy, making it an effective diagnostic tool for hepatic tuberculosis. PCR results for tuberculous hepatic granulomas were at least 57% more positive than those from other types of TB diagnostic procedures. Another benefit is that *M. tuberculosis* may be distinguished from other mycobacteria using PCR analysis, which saves a lot of valuable time.¹³

Histopathological examination of the specimens from lesions is essential for the exact diagnosis. Low sensitivity of both acid-fast staining (from 0% to 45%) and culture (from 10% to 60%) mean diagnosis can still be difficult. In this case, the aspirate from pericardial sheath was negative for AFB on staining adding to the diagnostic dilemma. Diaz et al found that at least 57% of the hepatic granulomas caused by tuberculosis gave positive PCR test results.

A high index of clinical suspicion is necessary for the diagnosis of a tuberculoma or a tubercular liver abscess. Tubercular liver abscess should always be kept in mind as a differential diagnosis especially in elderly individuals in endemic countries like ours who aren't responding to the established line of management. This helps to decrease the chances of development of antibiotic resistance – an issue which already has resulted in disease causing organisms being resistant to most of the antibiotics being used in the world of medicine currently. The situation is likely to worsen in this era of prescription of the overuse and abuse of these drugs, as well as the pharmaceutical industry's lack of new drug research as a result of diminished economic incentives and onerous regulatory constraints, have all been linked to the challenge of antibiotic resistance.

Many of the germs that the Centres for Disease Control and Prevention (CDC) has identified as posing urgent, serious, and worrying concerns have already caused a significant clinical and financial burden on the American health care system, patients, and their families. It is imperative to work together to enact new regulations, redouble research efforts, and explore crisis management strategies.

The best medical course of action for a tuberculous liver abscess is still up for dispute. According to Gracey, the huge size and dense fibrous tissue around the abscesses may make it difficult for antibiotics to reach their intended target. Antitubercular medication quadruple therapy is advised for one year. In the right circumstances, systemic ATT in addition to percutaneous draining of the abscess has been employed. TLAs have also been successfully treated in other instances by transcatheter antitubercular medication infusion and percutaneous drainage.

In our instance, the patient reacted effectively to systemic ATT and was getting better when examined for the last time six weeks after beginning ATT.

CONCLUSION

This is an uncommon instance of an isolated hepatic tubercular abscess in an immunocompetent adult without any lung or GI tract foci. A high index of suspicion should be maintained when dealing with a space-occupying lesion in the liver because the clinical presentation of an isolated TLA is so unusual that it puts the treating physician's clinical judgement to the test. This will allow for the use of evidence-based specific management rather than empirical therapy. If a hepatic tubercular abscess is detected early and treated quickly, the majority of patients have great prognoses.

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