

## Multidrug-resistant Tubercular Liver Abscess in $\beta$ -thalassemia

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**Background:** Liver abscesses in thalasseemics are predominantly pyogenic. **Case characteristics:** 12-year-old thalasseemic boy with abdominal pain and high grade fever for 15 days. **Observation:** CT abdomen revealed multiple liver abscesses. Pus culture and sensitivity showed multidrug-resistant mycobacteria. Patient was started on second line anti-tubercular drugs. **Outcome:** He responded well with symptomatic improvement and is doing well after 6 months of follow-up. **Message:** Liver abscess in patients with thalasseemia rarely can be tubercular.

**Keywords:** Liver abscess, *Mycobacterium tuberculosis*, *Thalasseemia major*.

Primary tubercular liver abscess (TLA) is rare, even in countries with high prevalence of tuberculosis (TB) [1-4]. Thalasseemics have co-existent immunodeficiency that makes them more susceptible to infection-related morbidity and mortality [5-6]. Liver abscesses in patients with thalasseemia are predominantly pyogenic; tubercular abscesses are very rare. We report a case of tubercular liver abscess in a child with thalasseemia.

### CASE REPORT

A 12-year-old boy presented with dull aching pain in right upper abdomen and lower chest associated with intermittent high-grade fever, weakness and vomiting for 15 days. He was suffering from  $\beta$ -thalasseemia major receiving frequent but inadequate blood transfusions for 11 years, and iron chelation therapy (deferasirox) for 2 years. There was no past history of tuberculosis or contact with a patient of tuberculosis. On physical examination, patient was sick, febrile (102.6<sup>0</sup>F) and pale; pulse rate was 88/min, blood pressure was 104/66 mmHg and respiratory rate was 22/min. Tenderness was present in right hypochondrium; liver and spleen were markedly enlarged. There was no ascites or other palpable mass. Respiratory and cardiovascular examination were normal.

On investigation, hemoglobin was 7.7 g/dL, total leucocyte count was 16000/mm<sup>3</sup> and peripheral smear showed microcytic hypochromic anaemia. Chest X-ray was normal, Mantoux test was negative and three early morning sputum samples were negative for acid-fast bacilli (AFB). Serum ferritin was markedly elevated (2450 mg/dL). Rest of the investigations (including widal test, malarial antigen, urine routine and culture, blood culture HbsAg, Anti HCV, and HIV-ELISA) were within normal limits. Abdominal ultrasound showed heterogeneous, ill-defined, hypo-echoic lesions reaching

upto liver surface in right lobe of the liver suggestive of multiple abscesses. No perihepatic or pleural effusion was seen. A clinical diagnosis of pyogenic liver abscess was made and patient was put on intravenous antibiotics but his fever and abdominal pain persisted even after two weeks of therapy. Repeat ultrasound abdomen showed similar findings. Computed tomography (CT) of abdomen revealed multiple hypodense lesions with perilesional enhancement in right lobe of liver suggestive of multiple abscesses (**Fig. 1**). Pus was aspirated under CT-guidance and sent for examination and culture. Zeil-Neilsen staining and culture demonstrated Acid fast



**FIG. 1** CT abdomen showing multiple hypodense lesions (black arrows) with peri-lesional enhancement in right lobe of liver suggestive of multiple abscesses.

bacilli (AFB) in aspirated pus; there were no amebic trophozoites or fungal elements. Anti-tubercular therapy (ATT) with isoniazid, rifampicin, pyrazinamide, and ethambutol was started along with supportive therapy. Fever and abdominal pain gradually subsided with improvement in his appetite and general condition. However, after 4 month of regular ATT, patient presented again with abdominal pain and intermittent fever. He had a pus discharging sinus in the right hypochondrium. Repeat ultrasound and CT abdomen revealed multiple liver abscesses. Pus culture and sensitivity revealed multidrug-resistant tuberculosis (MDR-TB) as organisms were resistant to both isoniazid and rifampicin. Patient was put on kanamycin, ofloxacin, para-aminosalicylate, etambutol and pyrazinamide, to which he responded well with symptomatic improvement and cessation of discharge. At 6 months of follow-up, he was doing well.

## DISCUSSION

Ruptured tubercular liver abscess presenting as parietal wall swelling is extremely rare with only few reported cases [1-4], that too in adults. Primary liver involvement as tubercular liver abscess is also very rare. Route of infection is usually hematogenous; respiratory and gastrointestinal tracts are primary sources of infection [7,8]. Clinical manifestations of tubercular liver abscess are nonspecific and include anorexia, fever, and vague/localized right upper quadrant pain. Hepatomegaly is common on presentation but jaundice due to extra or intrahepatic obstruction is rare [7,8].

Co-existent immunodeficiency in thalassemic children – both humoral and cell mediated – make them prone to infections. Iron excess deranges the immune system to favor the growth of infectious agents. Other factors include splenectomy resulting in increased susceptibility to encapsulated bacterial infections, transfusion-related allo-antigenic stimulation, transmission of immunosuppressive viruses, iron chelation therapy predisposing to yersinia infections, and zinc deficiency [5-6]. Wang, *et al.* [9] reported 1.6 episodes of severe bacterial infections/100 patient-years in transfusion dependent thalassemic patients with liver being the most common focus, and likely organisms were gram negative bacteria. Tuberculosis in thalassemics can be explained by deficient cell mediated immunity and altered immunological profile. Our case was not

splenectomized but was inadequately transfused and iron overloaded with irregular chelation therapy making him prone to infections.

Clinicians should be aware of unusual co-morbidities in thalassemics, and surveillance for infections is crucial to improve the survival and quality of life. Tubercular liver abscess, although very rare, should be considered in differential diagnosis of hepatic mass lesions, especially multi-loculated abscess in immuno-compromised patients.

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