A RARE PRESENTATION OF LIVER TUBERCULOSIS AS AN LIVER ABSCESS: A CASE REPORT

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ABSTRACT
Hepatic tuberculosis is uncommon but hepatic abscess is extremely uncommon entity. We report a case of 42 yrs male that developed isolated hepatic abscess not associated with lungs or other organ involvement which was supposed to be a pyogenic liver abscess until report of histological findings arrived. Although tubercular liver abscess is very rare, it should be included in differential diagnosis of abscess of liver and unknown hepatic mass lesions.

Keywords: Liver, Isolated Abscess, Tuberculosis, Pyogenic, Amoebic, Histological Examination

INTRODUCTION
Tuberculosis is common infection in developing countries like India. Though pulmonary TB is most frequent presentation, extra pulmonary disease involving lymph nodes, terminal ileum and peritoneum is not infrequent. Hepatic involvement has been described in 90% of milliary tuberculosis, 75% of extra hepatic TB and 25% of pulmonary TB (Klatkin, 1977).
Essop et al., (1984) have shown that hepatic TB may account for all 1.2% of all TB developed at general hospitals. However isolated hepatic TB without disseminated disease is rare, tubercular abscess is extremely rare entity (Puri et al., 1994). High index of suspicion is required for correct diagnosis and it is only be diagnosed on histopathological examination of liver biopsy specimen.
In this case report we describes a case of isolated tubercular abscess which is a rare entity often presents as pyogenic or amebic abscess.

CASES
A 42 yr old male from middle socioeconomic status was evaluated for pain right hypochondrium, epigastrium, and anorexia, weight loss since 2 months. There was no previous history of TB or contact with any patient of TB. On admission, physical examination revealed a conscious man with pulse rate 80/min, BP 110/70 mm of Hg, and RR was 20/min. there were no jaundice and no lymphadenopathy. Abdominal examination revealed painful hepatomegal, increased liver span 18cm, no splenomegaly and no other abnormality in CVS & CNS were found.
Lab data were-
Hb 10.6g/dl, WBC 13000/mm3, platelets 1.80lachs/mm3, urea 25mg/dl, creatinine 1.0 mg/dl.
Liver enzymes showed total bilirubin 3.5mg/l direct 2.0 mg/l and SGOT SGPT were 89 U/L and 96 U/L respectively.
Patient was non reactive for HbsAg and HIV. Chest X-Ray shows no finding suggestive of TB but revealed little elevation of right side dome of diaphragm.
USG abdomen showed ill-defined, heterogeneous, hypo echoic lesions reaching up to liver surface with cystic area in right lobe of liver which was approximately 7.5*8.4 cm in size suggestive of abscess? Pyogenic.
CT scan showed 9*8*6 cm size abscess in right lobe of liver with no perihepatic collection.
Case Report

USG guided aspiration was done twice but not succeeded only blood aspiration was there, so laparoscopic drainage were planed.

Laparoscopically liver abscess was drained, on which 250 cc frank yellow color pus was come out suggestive of pyogenic abscess. This pus was sent for culture and drain was placed on liver surface.

Patient was started on 3rd generation cephalosporin’s and amino- glycosides with provisional diagnosis of pyogenic liver abscess.

After 3 days pus culture report showed negative report for any bacteriological and fungal infection. Patient symptoms worsened despite injectable antibiotics treatment.

After 5 days repeat CT scan of abdomen showed persistence of multiseptate liver abscess.

In view of patient condition and refilling of multiseptate abscess cavity, a decision to perform repeat laparoscopic drainage was taken.

On repeat same intrapanchymal liver abscess was seen in Rt lobe of liver in which 400cc pus was drained.

At this time biopsy of liver abscess wall was also taken.

Histopathological examination of biopsy showed tubercular follicles with central caseous necrosis surrounded by lymphocytes, multinucleate giant cells and epithelial macrophages which confirms the diagnosis of tubercular abscess.

On confirmation of diagnosis four drug therapy rifampicin, isoniazide, ethambutol and pyrizinamide was started according to schedule. Patient showed drastic improvement postoperatively.

After 4 week follow up pt was asymptomatic and liver size was normal with no abscess cavity on USG.

DISCUSSION

Involvement of liver in Tb can occur in three forms-

1) Commonest as a part of disseminated Tb
2) Diffuse involvement of liver with granuloma formation called granulomatous hepatitis.
3) Third rarest is that granulomatous lesions coalesce to form tuberculoma which undergoes to caseations and abscess formation (Spiegel and Tuczon, 1984).

In all of these three forms the pt are usually symptomatic and associated with a of laboratory abnormalities.

Imaging technique are not helpful in diagnosing isolated hepatic abscess as these are less common and not associated with any peculiar feature which can differentiate them from pyogenic or amoebic liver abscess (Puri et al., 1994).

Liver biopsy is usually required. In histopathological finding of tubercular bacilli with hepatic granuloma with central caseations necrosis on biopsy is characteristics and should be considered diagnosis of tuberculosis until unless proved otherwise (Albrecht, 2003).

Recently PCR assay was demonstrated to be useful in a diagnosis of hepatic Tb (Kawashima et al., 2000; Diaz et al., 1996).

The prevalence of tubercular liver abscess was just 0.34% in pt of hepatic Tb as shown in a study where the pt age ranged from 6 months to 72 yrs with an average age of 39.2 yrs (Essop et al., 1983).

Tubercular liver abscess is frequently confused with hepatoma, amoebic liver abscess and pyogenic liver abscess as in case of out pt. Because of nonspecific clinical presentation the diagnosis of tubercular liver abscess is usually made at biopsy (Balsarkar and Joshi, 2000).

Quadruple therapy with ATT is recommended for 1 yrs which was effectively advocated in our pt (Baveja et al., 2009).

In conclusion isolated hepatic tubercular abscess is very rare, should always be considered in differential diagnosis of pyogenic or amoebic liver abscess. Symptoms and signs are not specific; ultimately diagnosis depends on the demonstration of AFB in pus, aspirate or biopsy specimen or the necrotic tissue. The prognosis with ant tubercular treatment is good.
Case Report

Figure 1: Photomicrograph showing the histological findings. The conglomerate tubercle comprises Langerhans giant cells, epithelioid cells, and lymphocytes.

REFERENCES


