TUBERCULAR ABSCESS IN AN IMMUNOCOMPETENT PATIENT WITH CHRONIC LIVER DISEASE

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ABSTRACT

A 67 year old man presented with swelling of abdomen, anorexia and weight loss with a previous history of alcoholism. Subsequently it was diagnosed to be a case of tubercular liver abscess in a background of chronic liver disease due to alcoholism which is very rare in an immunocompetent patient.

Key words: Tubercular liver abscess, Chronic liver disease, immunocompetent

INTRODUCTION

Though hepatic tuberculosis is not a rare disease entity, tubercular liver abscess (TLA) is extremely rare even in a country where tuberculosis is an alarming public health problem. It is usually associated with foci of infection either in the lung and/or gastrointestinal (GI) tract [1] or with an immunocompromised state. An isolated or primary TLA with no evidence of tuberculosis elsewhere is even rarer. The rarity of this clinical entity prompted us to present this case which involves an immunocompetent adult with an isolated hepatic tubercular abscess and with no foci of infection in the lungs or GI tract.

CASE REPORT

67 yrs old married hindu male business by occupation was admitted with swelling of the abdomen, anorexia & weight loss for last 3 months. Swelling of the abdomen was painless and gradually increasing. There was also history of drainage of 1 lit of ascitic fluid in the local hospital 1 month back. There was mild decrease in appetite and significant weight loss from 52 kg to 46kg during last 3 months. There was no h/o facial puffiness, pedal swelling, decrease urine output, fever, respiratory distress, palpitation, chest pain, cough, haematemesis, melena, jaundice. There was also no h/o blood transfusion or promiscuous sexual activity. He was previously alcoholic, started taking alcohol 38 yrs back and took daily for 10-12 yrs and then stopped. He is also smoker but non diabetic, non hypertensive. He was treated with diuretics in local hospital but abdominal swelling persisted. He had a past h/o jaundice 32 yrs back. He was diagnosed

as a case of gout 25 yrs back but is on irregular medication. There was no past h/o or contact with tuberculosis.

On examination- there was mild pallor & he was underweight (BMI-17.968). There was no jaundice, clubbing or lymphadenopathy. There was fullness of abdomen, prominent epigastric & flanks vein, palpable left lobe of liver, splenomegaly (3cm below the left costal margin). Shifting dullness was present. Other systemic examination was within normal limit.

On investigation, there was normocytic, normochromic anaemia with raised ESR (Hb%-9 gm%, ESR-120).Fasting blood sugar, urea, creatinine, Na+, K+ were within normal limit. Liver function test showed reversal of Albumin/Globulin ratio & increased globulin(albumin- 3.2gm/dl, globulin-4.6gm/dl), SGOT-33 IU, SGPT-16 IU, ALP-214 IU. Protrombin time was 15.2 sec with INR 1.44. Uric acid was 6.8 mg /dl. Ultrasonography of abdomen revealed mild increased coarse echotexture of liver, PV -13 mm, spleen-13.8 cm, moderate ascites. Ascitic fluid examination showed cell count-200 cu mm (L95N5),sugar-105mg/dl protein-7.7gm/dl, albumin-2.8gm/dl, SAAG- 0.4, ADA-32.6, gm stain, AFB stain & malignant cell- negative. Chest x ray was normal, Sputum for AFB, HBsAg, Anti HCV were all negative. UGI endoscopy showed gastroduodenitis. CECT of whole abdomen revealed hepatomegaly with low density lesion (abscess) in the right lobe of liver (Figure 1) , moderarate ascites. CECT abdomen didn't reveal any omental thickening, lymph nodes, ileo-caecal mass orother gut pathology except ascites and liver abscess. CT guided FNAC from liver SOL showed degenarating inflammatory cells, granulomatous collection of epitheloid cells, multinucleated giant cells and caseous necrosis and Z-N stain revealed acid fast bacilli (Figure 2).

So it is a case of tubercular liver abscess with ascites in an immunocompetent patient in the background of chronic liver disease due to alcoholism.

We treated the patient with HRZE and after 1 month patient's appetite improved, ascites decreased, liver enzymes were normal but Uric acid level increased to 17mg/dl. We stopped pyrazinamide and started ofloxacin. The patient is now doing well. CT scan of abdomen after 1 year revealed only a small organised lesion without any calcification.

Liver biopsy was done few weeks after starting Anti tubercular drugs which revealed fibrosis around portal tracts, bridging fibrosis, regenerative nodules with a HISTOLOGY ACTIVITY INDEX (HAI - Knodell Score) of 3. Strict alcohol cessation and spironolactone has been introduced.

Figure 1. CECT of Abdomen showing low density lesion (abscess) in the right lobe of liver.

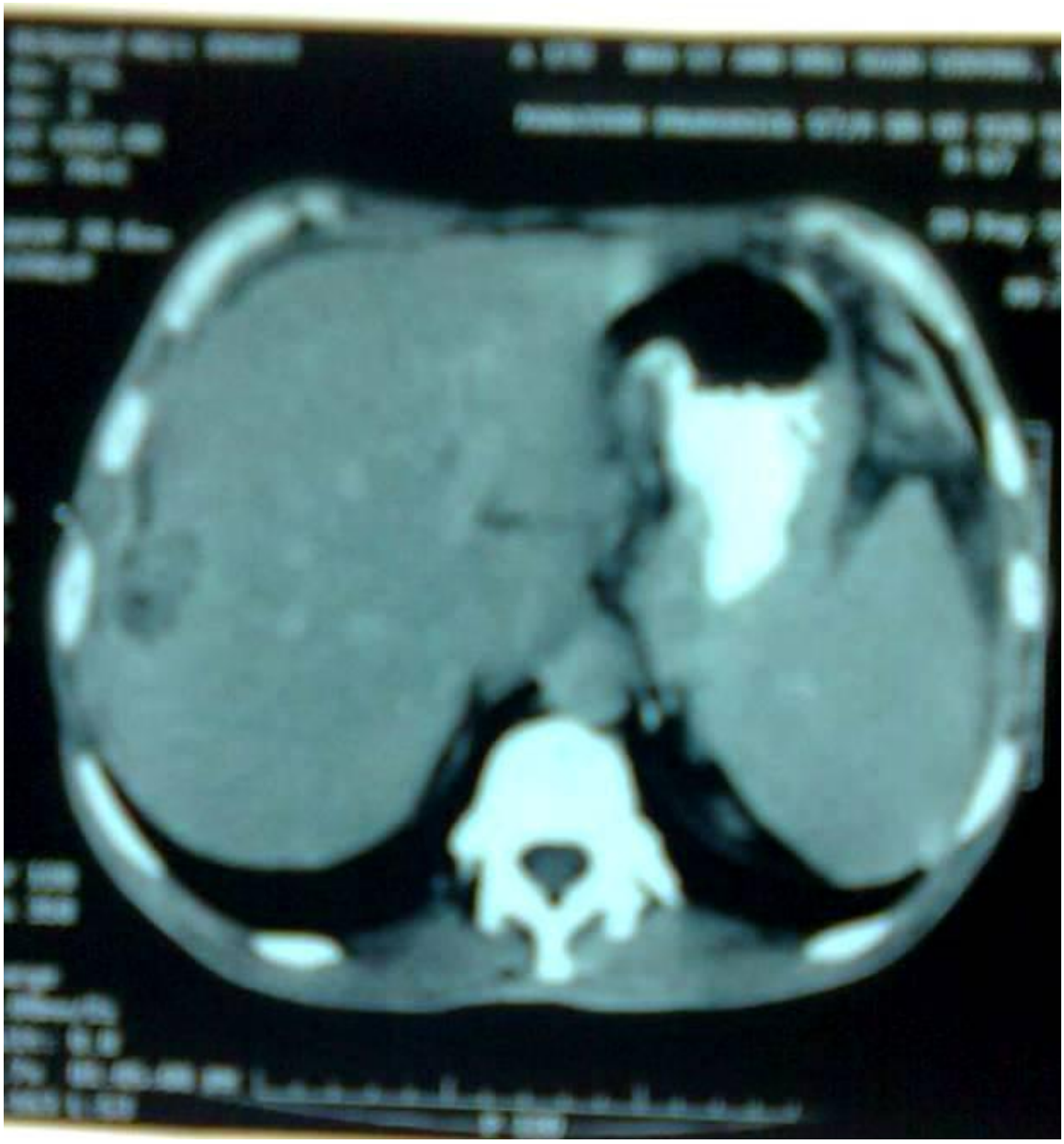
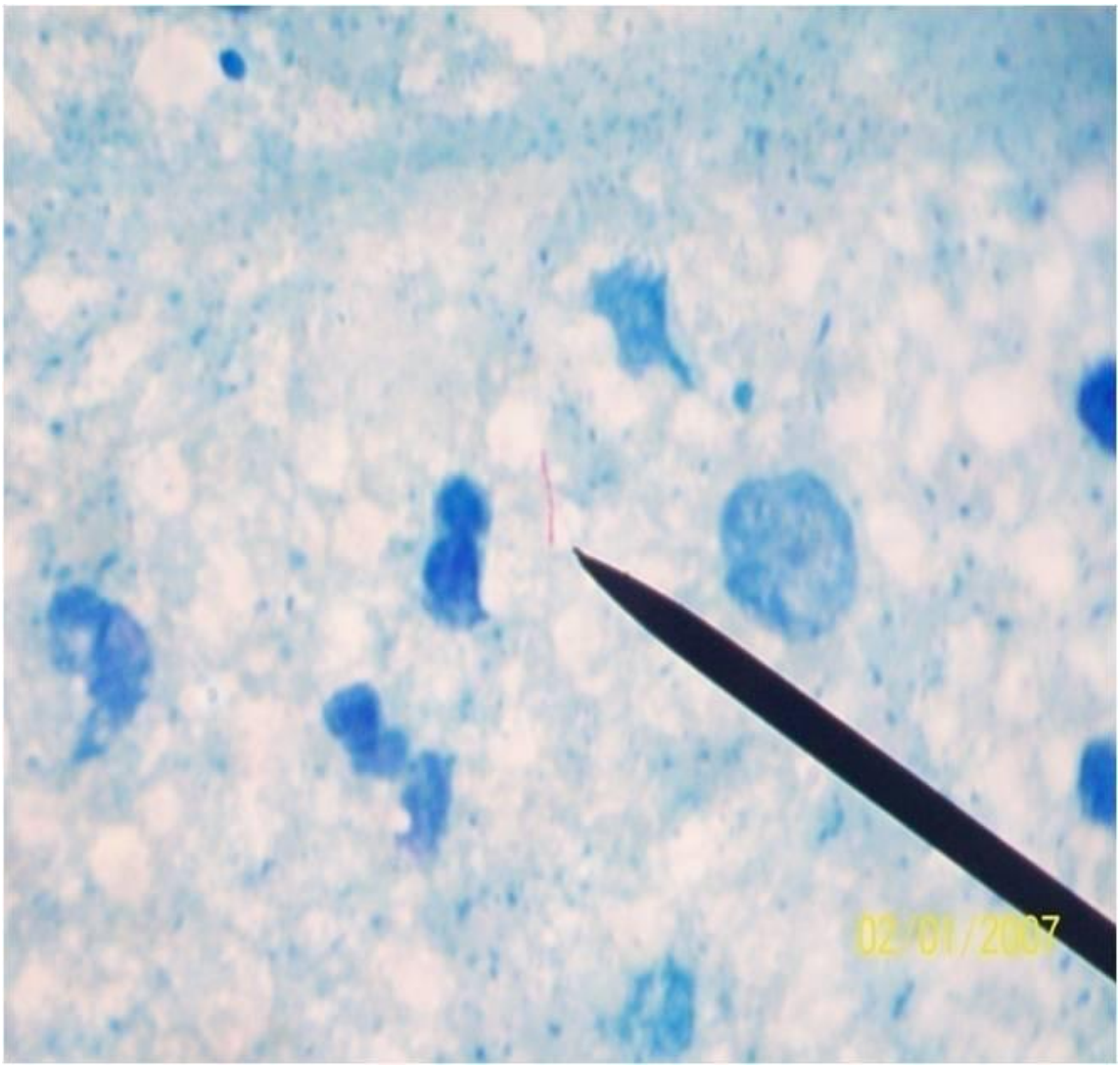


Figure 2. FNAC from liver SOL(Z-N stain) showing Acid Fast Bacilli.



DISCUSSION

In extrapulmonary tuberculosis, hepatic tuberculosis has been regarded as a rare form of Tuberculosis[2]. Most of the cases described usually occurred in association with miliary tuberculosis, mainly through hematogenous dissemination. The respiratory and GI tracts were the major sources of infection and bacilli travelled there via hepatic artery or the portal vein[3]. Rolleston and McNee classified hepatic TB into the miliary and local forms in 1929. Since then, researchers have described three morphologic types of hepatic TB: (a) miliary TB of the liver associated with generalized miliary TB; (b) primary miliary TB of the liver without involvement of other organs; and (c) a primary nodular lesion termed tuberculoma or a frank abscess[4]. Of these, primary tuberculous abscess of the liver is the rarest,but has been reported sporadically, with only few cases previously reported in the literature.Tubercular liver abscess was firstdescribed by Bristowe in 1858 [5].

The prevalence of TLA was just 0.34% in patients with hepatic tuberculosis as shown in a study where the patient age ranged from 6 months to 72 years with an average age of 39.2 years [6]. Symptoms of the disease are commonly non-specific and include fever, vague abdominal pain, anorexia and weight loss . Hepatomegaly is a common physical finding. Jaundice is a very rare manifestation of TLA and may be caused by extra- or intrahepatic obstruction. No clear relationship exists between the degree of liver involvement and jaundice . TLA is frequently confused with hepatoma, pyogenic liver abscess and amoebic liver abscess. Because of the non-specific clinical presentation, the diagnosis of TLA is usually made at autopsy or occasionally after laparotomy has been performed .In our case although there was anorexia and weight loss but fever was absent and clinical features were suggestive of chronic liver disease with potal hypertension. But low SAAG ascites pointed us that ascites was not due to potal hypertension (which led us to perform a C.T.Scan of abdomen and subsequently diagnostic C.T. guided. F.N.A.C.from liver SOL), which was further corroborated by improvement of ascites after treatment with anti tubercular drug.

The radiological findings of TLA have a low specificity [7]. USG and computed tomography (CT) scan findings usually reflect different stages of disease varying from granulomatous tubercles with or without caseous necrosis to fibrosis and calcification in the healing stage. USG findings of hepatic tuberculosis usually show hypo-echoic or hyperchoic lesions,but in our case the lesion was so small and peripherally located that it was not revealed by ultrasonography. The ultimate diagnosis of TLA depends upon the demonstration of AFB in pus, aspirate or biopsy specimen or the necrotic tissue. Recently, PCR has been found to be a useful diagnostic tool for hepatic tuberculosis [7] as it enables rapid identification of *Mycobacterium tuberculosis* and expedites a treatment decision. At least 57% of tuberculous hepatic granulomas gave positive PCR results compared to other conventional diagnostic techniques for TB . Another advantage is that PCR analysis can distinguish *M. tuberculosis* from other mycobacterium saving a lot of precious time. As acid fast bacilli was found in FNAC in our patient there was no need for doing PCR.

Medical treatment of tuberculous liver abscess is still a subject of debate. Gracey postulates that thick fibrous tissue around the abscesses and their large size may prevent antibiotics from reaching the target . Quadruple therapy with antitubercular drugs is recommended for 1 year. Percutaneous drainage of the abscess, combined with systemic ATT has been used in appropriate cases [4]. In some other cases, TLAs have been successfully treated by percutaneous drainage combined with transcatheter infusion of antitubercular drugs [8]. In our case we started with Isoniazid (300mg), Rifampicin(450mg), Ethambutol(800mg), Pyrzinamide(1200mg), Benadon (20mg)per day,after 1 month of starting treatment patient gained 2 kg wt and ascites disappeared but uric acid level level increased to 11 mg/dl inspite of taking Allopurinol 300mg/day,then we stopped Pyrazinamide and started Oflxacin 400mg/day. At the end of 1 yr of treatment patient was responded with decrease in liver SOL size.

CONCLUSION

This is a rare case of an isolated hepatic tubercular abscess without any pulmonary and GI tract foci in an immunocompetent adult. The clinical presentation of an isolated TLA

is so atypical that it challenges the clinical acumen of the treating physician, and hence a high index of suspicion should be maintained when dealing with a space-occupying lesion in the liver so that evidence-based specific management may be undertaken instead of empirical therapy. The prognosis of a hepatic tubercular abscess is excellent for the majority of patients if diagnosed early and prompt treatment is administered.

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