

## Case Report

# TB ascitis in cirrhosis of liver - A case report

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## Abstract

To diagnose the cause of ascitis in cirrhosis of liver is difficult clinically as fluid is also present in cirrhosis with portal hypertension. The other diseases causing ascitis can also occur in cirrhosis of liver with portal hypertension. In tropical countries like India tuberculous ascitis is very common, hence tuberculous ascitis can occur even in presence of cirrhosis of liver with portal hypertension. In this case report, 45 years old male presented with tuberculous ascitis in cirrhosis of liver. This points out that tuberculous ascitis should be thought of even in presence of cirrhosis of liver or cirrhosis of liver with portal hypertension. The patient recovered completely with anti-tubercular treatment without any complications or toxicity. The role of anti-tubercular treatment and its toxicity in the presence of liver cirrhosis is also discussed.

## Key words

Cirrhosis of liver, Tuberculous ascitis, Drug toxicity of ATT.

## Introduction

Cirrhosis of liver is a relatively common condition. Although the exact prevalence in India is unknown, the autopsy studies in USA showed a prevalence of 5-10%. In addition to Hepatitis B and C there has been a massive increase in alcoholism [1] in India contributing to an increase in chronic liver disease including

cirrhosis. This leads to an immunodeficiency status [1] and along with malnutrition which is commonly present in such patients, may increase the risk of tuberculosis.

In general population, pulmonary tuberculosis accounts for 80-85% of all cases [2]. However, in patients with cirrhosis, extra pulmonary tuberculosis is more common. Peritoneal

tuberculosis occurs in 3 forms: (i) Wet type with ascitis; (ii) Encysted (loculated) type with a localized abdominal swelling; and (iii) Fibrotic type with abdominal masses composed of mesenteric and omental thickening, with matted bowel loops felt as lump(s) in the abdomen. A combination of these types are also common [3]. Ascitis due to peritoneal tuberculosis may be difficult to diagnose in the setting of liver cirrhosis where portal hypertensive ascitis is common [4]. Here we have presented a case report demonstrating association between tuberculous ascitis and cirrhosis of liver.

### **Case report**

45 years old male presented with complaints of abdominal distension of gradual onset, shortness of breath, diffuse pain abdomen and swelling of both lower limbs, history of weight loss of 5 kg over a period of 6 months. There was no history of fever, haemoptysis, vomiting, and chest pain. He was not a known case of diabetes, hypertension, tuberculosis, bronchial asthma, epilepsy. He was a non smoker and non alcoholic. He was on diuretic therapy for the distension of abdomen. With diuretic therapy, he was not relieved of his symptoms.

On examination, patient was dehydrated. His temperature was normal, pulse rate (PR) - 74/min, blood pressure (BP) - 110/70 mm of Hg, respiratory rate (RR) - 18/min. Examination of Cardio vascular system was normal, Respiratory system was normal, Central nervous system was normal. Gastro intestinal tract examination - per abdomen was soft, diffuse tenderness was present all over, distended, dull note on percussion, ascitis present, hepatosplenomegaly was present.

On Investigations, Complete Urine Exam revealed albumin - nil, sugars - nil, Complete Blood Picture: Hb - 11.2 gm/dl, RBC - 4.9 million/mm<sup>3</sup>, WBC - 6000/mm<sup>3</sup>, DC - Neutrophils - 62%, Eosinophils - 2%, lymphocytes - 34%, Monocytes - 2% and Basophils - 0%, Platelets - 1 lakhs/mm<sup>3</sup>, ESR -

35mm 1<sup>st</sup> hour, RBS - 107 mg/dl, Blood Urea - 28 mg/dl, Serum creatinine - 0.9 mg/dl, Serum calcium - 9.9 mg/dl, LFT - Serum bilirubin - 0.2 mg/dl, Direct - 0.5 mg/dl, SGOT - 57 U/L, SGPT - 46 U/L, ALP - 170 U/L, Total protein - 6.1 gm/dl, Albumin - 2.8 gm/dl, Globulin - 3.3 gm/dl, HbsAg - Reactive, HIV 1 and 2 - Nonreactive, HCV - Nonreactive, Ascitic Fluid Analysis showed straw coloured fluid, Glucose - 101 mg/dl, Protein - 0.7 gm/dl, Albumin - 0.2 gm/dl, TLC - 1000 cells/cumm, Lymphocytes - 98%, Neutrophils - 02%. ADA - 54 u/l, Ultrasonogram of Abdomen - Small nodular liver with increased echogenicity in irregular areas seen. Spleen is enlarged, 10 cm, portal vein - dilated 14 mm, multiple gall stones, Kidneys size - right kidney 9.3x4.2 cm with parenchymal calculi, left kidney 9.2x4.3 cm, with moderate ascitis, no lymph nodes, was suggestive of Cirrhosis of Liver with portal hypertension, Upper GI Endoscopy showed oesophageal varices, Chest X-ray - Normal. Sputum for AFB was negative. Montoux test was positive.

Based on history, clinical examination and investigations patient was diagnosed as a case of TB ascitis with Cirrhosis of Liver. The patient was kept on anti-tubercular therapy for 6 months. Patient was followed up every month. He did not suffer any side effects of ATT. His ascitis subsided completely. This shows that TB ascitis can occur in cirrhosis of liver. With anti tubercular treatment, the ascitis is completely subsided.

### **Discussion**

This index case was presented to highlight the importance of suspecting unusual causes for ascitis in a patient with liver cirrhosis. Patients with liver cirrhosis generally have impaired cellular immunity. These patients demonstrate impaired delayed type of hypersensitivity; hence there is high likelihood of false negative tuberculin test. In addition abdominal tuberculosis is a paucibacillary disease and AFB smears are generally negative in such patients.

The diagnostics of tuberculosis in cirrhotics, is similar to disease in general population.

A specific diagnostic problem occurs when peritoneal tuberculosis [5] is suspected in a patient with liver cirrhosis, with both conditions can present with ascitis. Tubercular ascitis should be suspected in the clinical setting of a new onset of ascitis in a patient with stable compensated liver cirrhosis or when there is increasing or resistant ascitis occurs despite diuretic treatment in cirrhosis, especially when there is background of constitutional symptoms such as anorexia, fever, weight loss, etc. A high index of suspicion is required to exclude tuberculous ascitis and ascitic fluid examination should be done in all such cases. Tubercular ascitis in the setting of cirrhosis reveals a high SAAG, high protein ascitis with a lymphocyte predominant high cell count fluid. PCR for *Mycobacterium tuberculosis* may be positive.

The need for critical review of treatment of tuberculosis in cirrhotics arises because 3 of the 5 first line anti-tubercular drugs are potentially hepatotoxic. The administration of these drugs can lead to worsening Liver Function Tests with decompensation of stable cirrhotics and sometimes cause fulminant hepatic failure with a high mortality. Broadly, the more advanced the liver disease the less the number of hepatotoxic drug should be used. It must be remembered that pyrazinamide has the highest hepatotoxicity followed by rifampin and isoniazid. Safer anti tubercular drugs are Ethambutol, quinolones, aminoglycosides and cycloserine [6].

There are broadly two categories of treatment

- Cirrhotic patient with essentially normal baseline liver function tests. Such patients may be treated with standard 4 drug regime for 2 months followed by 2 drugs for remaining 4 months (total 6 month treatment). Since Pyrazinamide is potentially the most hepatotoxic drug, it may be completely avoided and a 9 month 3 drug regime

may be used. Regular monitoring of Liver Function Tests is recommended.

- Cirrhotics patients altered baseline liver function tests.

According to 2010 WHO guidelines [7], depending on the severity of the disease and degree of decompensation, the following regimen can be used, by altering the number of hepatotoxic drugs. One or two hepatotoxic drugs may be used in moderately severe disease whereas hepatotoxic drugs are completely avoided in decompensated cirrhosis.

- Two hepatotoxic drugs
  - 9 months of Isoniazid, Rifampin and Ethambutol (until or unless isoniazid susceptibility is documented)
  - 2 months of Isoniazid, Rifampin, Ethambutol and Streptomycin followed by 6 months of Isoniazid and Rifampin
- One hepatotoxic drug
  - 2 months of Isoniazid, Ethambutol and Streptomycin followed by 10 months of Isoniazid and Ethambutol
- No hepatotoxic drugs
  - 18-24 months of Streptomycin, Ethambutol and Quinolones

Regular LFT monitoring should be done in all cirrhotic patients receiving anti-tubercular treatment and drug therapy may be stopped /altered as per the LFT reports.

Hepatotoxicity due to antitubercular treatment is more commonly observed in patients with liver cirrhosis. In the general population, the criteria for stopping anti tubercular treatment is

- $AST / ALT > 3$ times upper limit of normal and symptomatic
- $AST / ALT > 5$ times upper limit of normal even if asymptomatic.
- Raised bilirubin

No clear guidelines are available for patients with cirrhosis. However, as a general principle a rising trend of liver abnormalities on 2 consecutive testing may be an indication for stopping the treatment. The absolute level of transaminases cannot be used as the sole criteria in cirrhotics. Any rise in S. Bilirubin should be treated with great caution and hepatotoxic drug treatment stopped immediately.

Treatment should be stopped and re-started after serum bilirubin and transaminase return to near normal. Drugs are re-started in a sequential fashion starting with rifampin first followed by isoniazid and lastly pyrazinamide which may be avoided altogether. The prognosis of patients with liver cirrhosis depends on the stage of disease and associated complications. Overall, the 1 year mortality is 34%, whereas patients with complications admitted in ICU have 1yr mortality rate of 69% .The mortality rate in patients with tuberculosis who have not received treatment (or delayed treatment) is >50%.The prognosis in patients of liver cirrhosis who develop tuberculosis is poorer compared to either disease alone .

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