

## Ruptured Tuberculous Liver Abscess as a Rare Cause of Tuberculous Peritonitis : A Case Report.

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**Abstract:** Tuberculosis is a rare cause of liver abscess even in countries where the tuberculosis is very prevalent. The radiological appearance of the tuberculous abscess is not different from other abscesses. Diagnostic delay may occasionally lead to catastrophic complications like perforation into pleural cavity, retroperitoneum or peritoneum. We present a rare case of multiple tuberculous liver abscesses with one of them rupturing and causing tuberculous peritonitis. The tuberculous etiology should be considered if the patient is not responding to the treatment for pyogenic and amoebic abscess and features of active or healed tuberculosis are present in the abdomen or at any other site.

**Key-words:** tuberculous liver abscess; diagnostic delay; perforation; peritonitis

### INTRODUCTION

Tuberculous liver abscess is very rarely seen with a prevalence of only 0.34% in patients with hepatic tuberculosis<sup>1</sup>. Due to non specific signs, symptoms and imaging features, diagnosis of tuberculous liver abscess is rarely made before aspiration or histopathology<sup>2</sup>. We present a rare case of multiple tuberculous liver abscesses with one of them rupturing and causing tuberculous peritonitis.

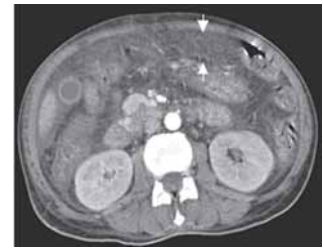
### CASE REPORT

A 55 year old man presented with history of pain in right upper quadrant of abdomen, fever off and on and weight loss for a period of one and half months. Clinical examination revealed a firm hepatomegaly extending to 4 cm below the costal margin and laboratory examinations revealed hemoglobin of 10 mg/dl, white blood cell count of 13,000 /mm<sup>3</sup> and erythrocyte sedimentation rate of 25 mm in the first hour. Chest radiograph showed fibrocalcific changes suggestive of healed Koch's infection in the upper zone of right lung.

Ultrasound of the abdomen revealed hepatomegaly, with multiple anechoic lesions having well defined walls and internal echoes, consistent with appearance of abscess. Calcified lymph nodes were also noted near porta hepatis. One of the abscesses was aspirated under sonographic guidance yielding creamy white pus. The pus was sent routine aerobic and anaerobic cultures which did not result in any growth. The patient was started on broad spectrum antibiotics but did not show any significant improvement. Another pus sample was sent for culture and staining. The condition of the patient deteriorated further. Contrast enhanced CT of the abdomen was done which revealed hepatomegaly with multiple large cystic lesions with well defined walls [fig. 1]. One of the abscesses was communicating with the peritoneal cavity [fig. 1]. CT showed features of tuberculous peritonitis like mild ascites, mesenteric soft tissue stranding, omental caking and also matting of small bowel loops along with calcified lymph nodes in porta hepatis and peripancreatic region [fig. 1 & 2]. Acid fast bacilli were demonstrated in the repeat pus specimen on Ziehl-Nielsen staining and *Mycobacterium tuberculosis* was grown on culture. Patient was started on anti-tuberculous chemotherapy after draining the abscesses under sonographic guidance. Three weeks later patient showed marked clinical improvement. Ultrasound revealed reduction in size of the abscess cavities at follow-up and the patient showed continuous clinical improvement on anti-tuberculous chemotherapy.



**Figure 1.** 55 year old patient with hepatic tuberculosis and tuberculous peritonitis. Contrast enhanced CT axial section showing that one [arrow] of the liver abscesses is communicating with the peritoneal cavity with perihepatic collection of fluid.



**Figure 2.** 55 year old patient with hepatic tuberculosis and tuberculous peritonitis. Contrast enhanced CT axial section showing mesenteric soft tissue stranding, omental thickening [arrows] and fluid collection in perihepatic region.

### DISCUSSION

Hepatic tuberculosis is an uncommonly reported condition and occurs in micro and macronodular forms. Micronodular TB is seen in the form nodules of 0.5 to 2 mm diameter in size. Macronodular form of tuberculosis probably spreads to the liver from the para aortic or portal nodes via the portal vein or hepatic artery<sup>3</sup>. Tuberculous liver abscess is uncommon even in countries where tuberculosis is endemic. The clinical diagnosis of this entity is rarely made as it has non-specific clinical and imaging features and it is also rare. Even in developing countries like India where tuberculosis is so common, diagnosis of tuberculous liver abscess is usually not considered as amoebic and pyogenic abscesses are more common. The diagnosis of tuberculous liver abscess has been made in the majority of cases at laparotomy due to non-detection of mycobacteria in the percutaneous liver aspirate<sup>4</sup>. Alternatively, acid fast bacilli can be demonstrated in the aspirate obtained from the abscess, as seen in our case but often the results are negative.

Only few cases of ruptured tuberculous liver abscesses have been reported in the literature. In three cases tuberculous liver abscess ruptured into the right pleural cavity with two of them also having communication with bronchial tree<sup>5,6,7</sup>. Jain et al reported a case in which a tuberculous liver abscess ruptured into the retroperitoneum<sup>2</sup>. No case of tuberculous liver abscess rupturing into the peritoneum has been reported in the literature. In our case, the patient's CT scan

showed features of tuberculous peritonitis like mesenteric thickening giving a stellate appearance, omental thickening, free and loculated ascites and matting of bowel loops. There were also calcified periportal and peripancreatic lymph nodes. Following aspiration of the abscesses the patient responded well to the antituberculous chemotherapy. So even though amoebic and pyogenic liver abscess are more common, the possibility of tuberculous liver abscess should always be considered if the patient is not responding to the antibiotics, and also has findings suggestive of active or healed tuberculosis like calcified periportal and peripancreatic lymph nodes and lung lesions as in our case. Delay in diagnosis may lead to complications, multiple operations and morbidity as rupture of abscess occurred in our case<sup>7</sup>.

It is concluded that in a case of multiple liver abscesses, the possibility of tuberculous liver abscess should be considered especially if the patient is not responding to the conventional treatment for pyogenic

and amoebic abscess and features of active or healed tuberculosis are present in the abdomen or at any other site. Delay in the diagnosis may result in complications like rupture of abscess.

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# Unilateral Ocular Myasthenia Gravis following Acute Sinusitis-A Case Report.

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**Abstract:** Myasthenia is an autoimmune disease in which IgG antibodies directed against the acetylcholine receptors at the neuromuscular junction prevent normal muscle contraction and lead to muscle weakness. Myasthenic muscle weakness of acute onset can occur following identifiable stimuli. We describe a case of a 40 years old lady with sinusitis and who subsequently underwent a sinus endoscopy, and following this within days developed sudden onset isolated, unilateral, painless ptosis. She was then diagnosed as ocular myasthenia and treated with pyridostigmine to which she responded well.

## INTRODUCTION

Myasthenia gravis is caused by antibodies to post synaptic acetylcholine receptor and evidence indicates that AchR abs must be present in circulation prior to development of a 40 years old lady with sinusitis and who subsequently underwent a sinus endoscopy, within days developed sudden onset isolated, unilateral, painless ptosis. Such rapid evolution of myasthenia following an identifiable stimulus has been previously described<sup>1</sup>. It is speculated that the remote effects of autoinflammation secondary to tissue microtrauma leads to sudden increase in muscle permeability and greater exposure of receptors to antibodies, with resulting acute impairment of neuromuscular transmission<sup>1</sup>.

## CASE REPORT

A 40 years lady had complaints of cough, cold and headaches of a few days duration, was diagnosed as a case of right sided frontomaxillary sinusitis and treated by an ENT specialist with antibiotics and a subsequently a sinus endoscopy. She eventually improved and became asymptomatic.

However, within 7-10 days of endoscopic procedure, she complained of sudden onset drooping of the right eyelid, which characteristically worsened in the evenings. She had no headaches, periorbital pains, dimness or double vision, numbness on the face or limb weakness. She was not on any medications and had no history of recent vaccination.

Then she was seen at our clinic and examination revealed a definite right sided ptosis with evidence of fatigability. There were no signs of local inflammation, fever or tenderness. The rest of her gross neurological and general physical examination was normal. A probable

diagnosis of ocular myasthenia was made and she was referred for a neurologist opinion.

Subsequent investigations revealed a normal MRI Brain including both orbits, normal S. TSH and Blood sugars. She underwent a repetitive nerve stimulation test which was noncontributory. This was followed by a tensilon (edrophonium) test. Edrophonium is a short acting anticholinesterase that prolongs the presence of neurotransmitter acetylcholine at the neuromuscular junction. This revealed a striking improvement in her ptosis and reported as strongly positive. She was then started on oral pyridostigmine following which her ptosis settled. Three months after initial diagnosis, patient is symptom free.

## DISCUSSION

Myasthenia is an autoimmune disease in which IgG antibodies directed against the acetylcholine receptors at the neuromuscular junction prevent normal muscle contraction and lead to muscle weakness<sup>2</sup>. A long acting oral anticholinesterase such as pyridostigmine is the first line of treatment. Immunosuppression with prednisone is used when symptom improvement is inadequate<sup>3</sup>. This case demonstrates the importance of considering neurotransmitter diseases in the approach to new onset focal weakness that involves the ocular or bulbar musculature.

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