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Omental Tuberculosis in a Patient with Alcoholic Liver Cirrhosis-the Diagnostic and Treatment Dilemma

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Omental Tuberculosis in a Patient with Alcoholic Liver Cirrhosis-the Diagnostic and Treatment Dilemma

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Abstract

Alcoholic cirrhosis is frequently complicated by spontaneous bacterial peritonitis (SBP) due to the translocation of gut bacteria. However, in immigrants to the USA from parts of the world with high tuberculosis burden, a high degree of clinical suspicion of tuberculous peritonitis should be maintained when a patient presents with symptoms similar to SBP. We describe a case of a 45-year-old Nepali man with a history of alcohol abuse who immigrated to the USA six years prior and presented to the hospital with abdominal pain, night sweats, fevers, and a 10 kg weight loss. A CT scan revealed abdominopelvic ascites, liver nodularity suggestive of cirrhosis, and heterogeneous attenuation of the omentum. A CT-guided biopsy of the omentum was done, and the histopathology revealed non-necrotizing granulomas. However, given the demographics of the patient along with his constitutional symptoms, there was high suspicion for abdominal tuberculosis, and a second CT-guided omental biopsy was done and sent for tissue cultures. Due to the delay in getting microbiological confirmation of tuberculosis and persistent fevers, an empiric trial of isoniazid, rifampicin, pyrazinamide, and ethambutol (RIPE) was started with close monitoring of liver function tests. Since the patient's symptoms improved, the RIPE regimen was continued. Twenty-five days later, tuberculosis was confirmed on omental biopsy tissue culture. This case highlights the diagnostic and treatment challenges associated with omental tuberculosis with liver cirrhosis and suggests a potential role for an empiric trial of RIPE regimen in the appropriate clinical context.

Keywords

omental tuberculosis, abdominal tuberculosis, alcoholic liver cirrhosis, tuberculous peritonitis

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Introduction

Liver cirrhosis is a state of immunosuppression and is an independent risk factor for the development of tuberculous peritonitis by reactivating *Mycobacterium tuberculosis* from dormant foci in the abdomen.¹ It requires a strong clinical suspicion to diagnose tuberculous peritonitis as the presenting symptoms may be non-specific and may mimic spontaneous bacterial peritonitis with other common gut bacteria in patients with liver cirrhosis. Tuberculous peritonitis requires many months of treatment which is sometimes riddled with complications of liver toxicity from anti-tuberculous drugs. Unfortunately, there are no concrete guidelines to help direct therapy in this discrete group of patients. This case aims to highlight the diagnostic and treatment challenges associated with omental tuberculosis in a patient with alcoholic liver cirrhosis.

Case presentation

A 45-year-old Nepali man with a long-standing history of alcohol abuse presented to the hospital with abdominal pain, bloating, night sweats, fevers, and a 10 kg unintentional weight loss within the last few months. The abdominal pain was 7 out of 10 in intensity, localized to the right upper quadrant, and aggravated by positional changes. The patient denied cough, sputum production, difficulty breathing, or hemoptysis. On exam patient was febrile-101.3°F, tachycardic- 112 beats per minute, along with abdominal distention, spider nevi, and scleral icterus. Initial lab workup revealed a normal white cell count of $5.8 \times 10^9/L$, hemoglobin of 14.4 g/dL, and mild thrombocytopenia with platelet count of $148 \times 10^9/L$. Basic metabolic panel were within normal limits. Liver function tests showed elevated aspartate transaminase level of 86 U/L, normal alanine transaminase of 29 U/L, elevated alkaline phosphatase of 167 U/L, elevated total bilirubin of 2.8 mg/dL with a direct bilirubin of 1.5 mg/dL, and low albumin level of 2.7 g/L. INR was moderately elevated at 1.6. Hepatitis panel, HIV, and syphilis screening were negative. An abdominal CT scan revealed liver nodularity suggesting cirrhosis and heterogeneous attenuation of the omentum and mesentery with moderate ascites. However, no evidence of bowel wall thickening, lymphadenopathy, or masses were seen. Ultrasound-guided paracentesis yielded 3 liters of hazy fluid with 3011 nucleated cells with predominantly lymphocytes on analysis and no malignant cells. Patient was admitted for management of suspected spontaneous bacterial peritonitis (SBP) and was started on empiric therapy with Ampicillin/Sulbactam. Ascitic fluid culture, Gram stain, and Acid-fast bacilli (AFB) stains were all negative, and the patient continued to spike fevers, prompting broadening of antimicrobial therapy to Piperacillin/Tazobactam and Fluconazole. He then underwent a CT-guided biopsy of the omentum which was sent for pathology, revealing non-necrotizing granulomatous inflammation and giant cells, with negative Gram, AFB, and fungal stains (Figure 1). However, the morphology of the granulomas was somewhat suggestive of an infective etiology like tuberculosis, per pathology report. Keeping in mind that the patient was an immigrant from Nepal with persistent fevers, night sweats, and abdominal tenderness, tuberculous peritonitis was suspected, and antibiotics were discontinued. Unfortunately, the first time, omental tissue biopsy cultures were not obtained. Given the potential hepatotoxicity of many anti-tuberculous drugs like rifampicin, isoniazid, and pyrazinamide in the context of the patient's liver cirrhosis, confirmatory omental tissue cultures were recommended prior to initiate treatment for tuberculous peritonitis. A second CT-guided biopsy was performed and sent for tissue cultures and polymerase chain reaction (PCR) testing. The patient continued to remain febrile and weak, and the team of physicians decided to try an empiric trial of rifampicin, isoniazid, pyrazinamide, and ethambutol (RIPE) with close monitoring of liver function tests. Five days after starting treatment,

the patient's fevers resolved, and his symptoms improved; hence RIPE regimen was continued. He was discharged with outpatient follow-up at the county health clinic to complete tuberculosis treatment. Twenty-five days later, *Mycobacterium tuberculosis* was confirmed on the omental biopsy tissue cultures though the PCR was inconclusive. He continued to improve till the end of the 12-month treatment period and tolerated it well with no derangement in liver function tests to suggest drug hepatotoxicity.



Figure 1. CT scan of the abdomen showing heterogeneous attenuation of the omentum as shown by the red arrow.

Discussion

Liver cirrhosis is a state of immunosuppression due to reticuloendothelial dysfunction. It is well-established that patients with liver cirrhosis are at a higher risk of reactivation of latent tuberculous bacilli in abdominal macrophages, facilitating the development of tuberculous peritonitis, as seen in our patient.¹ Tuberculosis (TB) can affect any organ in the abdomen but particularly affects the peritoneum, constituting 31% to 58% of all cases of abdominal tuberculosis.² Interestingly, in a study done in the USA, more than 50% of the cases of peritoneal tuberculosis had underlying alcoholic liver cirrhosis.¹

The most common presenting features of tuberculous peritonitis are ascites (71%), abdominal pain (64.5%), weight loss (61%), and fever (59%), but the classically described doughy

abdomen associated with tuberculous peritonitis is rarely seen.² Sometimes it can present as worsening of liver function or increasing/resistant ascites occurring despite diuretic treatment.³ These are non-specific symptoms that can mimic spontaneous bacterial peritonitis, thus posing a diagnostic challenge as seen in our patient and a high degree of clinical suspicion is essential to make a diagnosis of tuberculous peritonitis.

Peritoneal tuberculosis is a paucibacillary disease making it tough to diagnose on routine AFB stains of ascitic fluid (sensitivity 0% to 40%). PCR, particularly Xpert MTB/RIF, which has an overall sensitivity of 83.1% to diagnose other types of extrapulmonary tuberculosis, has not been well studied in ascitic fluid samples for the diagnosis of intra-abdominal tuberculosis. Laparoscopy with peritoneal/omental biopsies is the investigation of choice to diagnose tuberculous peritonitis. Tissue from the biopsy should be sent for mycobacterial cultures as well as routine histopathology. Histopathology typically shows caseating granulomas with Langerhans giant cells and lymphocytes.⁴ In our patient, the first sample omental biopsy sample showed non-necrotizing granulomas, which has only been reported in a few cases in the medical literature.^{5,6,7} Unfortunately, the first sample was not sent for mycobacterial tissue cultures prompting a second CT-guided biopsy to get the tissue sample. The sensitivity of cultures has been reported between 38% and 92%, but it usually takes 2 to 8 weeks for final results, which proves challenging to make treatment decisions, as with our patient.^{2,4} Ultimately, since the patient's clinical condition continued to decline, the team chose to give an empiric trial of anti-tuberculous drugs even without final culture data in hand.

There are no concrete guidelines regarding antimycobacterial drugs used in patients with alcoholic liver cirrhosis. The treatment of tuberculous peritonitis is frequently complicated by poor drug tolerance, hepatotoxicity, and an increased risk of multidrug resistance. Patients receiving anti-tuberculous therapy should get a baseline and regular follow-up liver function tests. It is proposed that anti-tuberculous therapy can be guided by the Child-Turcotte-Pugh score,⁸ on which our patient scored 10 i.e., Class C. It is recommended that only one potentially hepatotoxic drug be used for patients in this category, with rifampicin preferred over isoniazid, while pyrazinamide should not be used. However, our patient received all four anti-tuberculous drugs and tolerated them well with no hepatotoxicity on close monitoring. There is some role for the empiric trial of anti-tuberculous therapy when clinical suspicion for TB is high, as shown by some studies.⁹

Conclusion

In patients with underlying liver cirrhosis, the diagnosis of peritoneal tuberculosis is often challenging as it can mimic SBP. Omental tuberculosis should be high on the differential diagnosis when epidemiological and demographic factors point towards the possibility of TB. Confirming the diagnosis of tuberculosis by tissue biopsy culture is generally required before subjecting the patient to the many months of hepatotoxic antitubercular therapy, especially in a patient with liver cirrhosis. However, in patients who clinically deteriorate and clinical suspicion for tuberculous peritonitis is high, anti-tuberculous therapy can be potentially started even before the results are confirmed on tissue cultures, as delayed therapy can worsen clinical outcome.

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