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Spontaneous Fungal Peritonitis: A Rare Complication of Ascites Secondary to Right Heart Failure: A Case Report

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Spontaneous Fungal Peritonitis: A Rare Complication of Ascites Secondary to Right Heart Failure:A Case Report

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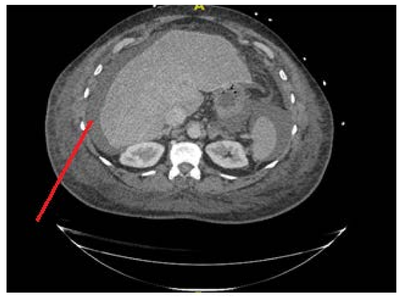
Introduction

- Spontaneous fungal peritonitis (SFP) is an infection defined as a neutrophil count (> 250 cells/mL) in the ascitic fluid with the evidence of positive fungal culture (1) while excluding other intra-abdominal infections. SFP is not as common as spontaneous bacterial peritonitis and has higher mortality rates due to late recognition and difficulty in differentiation between SFP and SBP. (1,2). The common risk factors that have shown to increase the mortality for SFP include hepatorenal syndrome, patient on SBP prophylaxis, elevated APACHE II scores on admission, and elevated lactate levels (2,3). Candida albicans and Candida glabrata are the two most common fungal pathogens responsible for SFP in the cirrhotic population (4). Spontaneous fungal peritonitis is an uncommon phenomenon occurring in a patient with cardiogenic ascites because of high protein content which is generally considered a low risk for infections. Signs and symptoms are indistinguishable from SBP, which may include abdominal pain, distension, guarding, fever and/or tachycardia. We present the second known case of spontaneous fungal peritonitis occurring on the background of cardiac cirrhosis, that was confirmed with fungal cultures growing Candida glabrata and was successfully treated with appropriate antifungal agents.

Case

- A 52-year-old female with a past medical history of chronic obstructive pulmonary disease was admitted to the hospital with a two-week history of abdominal pain and shortness of breath. The abdominal pain was associated with worsening distension. Upon admission, the patient was febrile with Tmax of 101 F, tachycardic with a heart rate of 110 beats/min, and blood pressure of 100/80 mmHg. Cardiovascular examination was pertinent for jugular venous distension. Respiratory examination revealed decreased breath sounds on bilateral lung bases. The abdomen was distended with diffuse abdominal tenderness, flank fullness, an everted umbilicus, and fluid thrill palpable diffusely. Initial laboratory assessment showed normal white blood cell (WBC) count of 7,900/mm^3, absolute neutrophil 6936/mm^3, hemoglobin 15.8 g/dL, hematocrit 60.3, MCV 66.5, platelet count 406 x 103/mm^3, blood urea nitrogen 10 mg/dL, creatinine 0.9mg/dL, total bilirubin 3.40 mg/dL, direct bilirubin 2.30 mg/dL, aspartate aminotransferase 43 U/L, alanine aminotransferase 28U/L, and lipase 43U/L, ammonia of 51, and pro-BNP of 4447. Other chemistries included sodium of 120, potassium of 4.9, chloride of 84, and bicarbonate of 33. Arterial blood gas on admission showed a pH of 7.33 with pCO2 58.7 mm Hg, PO2 75 and bicarbonate of 30 on FiO2 of 100%. An infectious panel of hepatitis A, B, C, and HIV were negative. Autoimmune workup including ANA and anti-smooth muscle antibodies were also negative. Upon initial imaging, a chest x-ray showed right-sided pleural effusion, but CT of the abdomen and pelvis showed bilateral pleural effusions with consolidation on the right, moderate ascites, and liver cirrhosis with no focal lesion. An echocardiogram showed normal left ventricular ejection fraction of 55%, dilated right ventricle, moderate tricuspid regurgitation with right ventricular systolic pressure of 76 mm Hg suggesting severe pulmonary hypertension.
- A diagnostic and therapeutic ultrasound guided paracentesis was done on admission and 400 mL of yellow fluid was removed and sent for analysis and cultures. The patient was empirically started on ceftriaxone for possible spontaneous bacterial peritonitis. On day 3, the patient's clinical status was not improving, so antibiotics were escalated to vancomycin and piperacillin-tazobactam and she was intubated and upgraded to the intensive care unit. The ascitic fluid analysis showed hazy fluid with a specific gravity 1.023 with a WBC count of 23,000, with 92% of polymorphic cells and 8% of mononuclear cells. On day 5, her repeat complete blood count (CBC) showed an elevated WBC count of 14.5/mm3, with the absolute neutrophil count of 12,180/mm3. Preliminary cultures from the ascitic fluid grew yeast and the diagnosis of spontaneous fungal peritonitis was made and started on IV caspofungin. Two sets of blood cultures were negative. The patient responded gradually well to antifungals and got extubated on Day 11. Speciation of fluid was reported on day ten, which grew *Candida glabrata.* Repeat CT scan chest showed significant improvement. Antifungals were continued till the day of discharge on day 16. Infectious disease recommended the patient to be discharged on intravenous Caspofungin and instructed to follow up with the gastroenterologist and infectious disease as an outpatient. She was discharged to a rehabilitation facility. The patient was then followed up for 1-month post discharge and she had shown significant clinical improvement.

Methods



CT scan demonstrating a moderate amount of ascites, with peritoneal thickening and enhancement consistent with peritonitis.

Results

- Cardiac cirrhosis is an uncommon but well-documented condition characterized by the presence of signs and symptoms of chronic liver disease along with the history of cardiac failure. (5) (6). Usually, it presents with sign and symptoms consistent with congestive heart failure along with clinical features of portal hypertension. (7). The basic pathophysiology includes hypoxic hepatitis due to impaired arterial perfusion (forward failure) and increased hepatic congestion (backward failure) (8). This backward flow, which is most commonly seen in right heart failure, causes an increase in liver lymphatic production resulting in ascites (9).
- Ascites due to heart failure is generally considered at low risk for infections because of the high protein content in the ascitic fluid. According to the gut hypothesis, patients with heart failure will have increased congestion in the gut which can lead to chronic intestinal damage allowing bacteria to translocate to the ascitic fluid(10) However, an acute episode of exacerbation may also cause further damage, leading to increase translocation of the fungal organism (11). Ascitic fluid should be evaluated by laboratory testing and imaging. Ascitic fluid retrieved by diagnostic paracentesis should be analyzed for lactate dehydrogenase, cytology, total cell count and differential, total protein, albumin, bacterial and fungal culture sensitivities(12). Cell count and the differential is significantly important for the diagnosis of spontaneous peritonitis (polymorphonuclear neutrophils >250 cells/ml). Along with the bacterial gram stain and culture sensitivity, fungal stain and culture sensitivities are necessary due to an increasing prevalence of SFP in patients with cirrhosis (13).
- The most common species causing SFP are Candida albicans, with other causative fungal agents being *Candida glabrata*, *Candida krusei*, *Cryptococcus* spp, and *Aspergillus* spp(14). Risk factors for SFP include elevated Child-Pugh and Model for End-Stage Liver Disease score, history of prophylactic antibiotics, low ascitic fluid protein (<1 g/dL), recent hospitalization and hepatorenal syndrome(3,15).
- The diagnosis of SFP require ascites fluid culture and sensitivity however polymerase chain reaction and beta-D glucan can also be done if the clinical suspicion is high. These tests are faster than conventional culture and sensitivities (15,16). Treatment with echinocandins is recommended as soon as the diagnosis is made. However, the prognosis is poor if there is a delay in appropriate antifungal therapy, especially with severe underlying disease. (17). Karvellas et al also showed that the delay in the diagnosis and treatment of SFP can lead to poor prognosis with a mortality of 100% (2). De-escalation to systemic fluconazole is recommended when sensitivity tests are available, which is also beneficial in reducing the rise of resistant organisms (18).

Conclusion

- To conclude, SFP is considered a serious complication of cardiac cirrhosis. A better understanding of the etiology of SFP with a quick diagnosis should be done promptly with an urgent evaluation of ascitic fluid and treatment with broad-spectrum antifungal therapy.

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