

Spontaneous Fungal Peritonitis as a Rare Complication of Ascites Secondary to Cardiac Cirrhosis: A Case Report

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
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Conflict of interest: None declared

Patient: Female, 52
Final Diagnosis: Spontaneous fungal peritonitis
Symptoms: Abdominal pain • shortness of breath
Medication: —
Clinical Procedure: —
Specialty: General and Internal Medicine

Objective: Rare disease





Background: Spontaneous fungal peritonitis (SFP) is a life-threatening infection which occurs more commonly in patients with liver failure. SFP is not as common as spontaneous bacterial peritonitis (SBP) and has higher mortality rates due to late recognition and difficulty in differentiation between SFP and SBP. Spontaneous fungal peritonitis is extremely uncommon in patients with cardiac ascites due to a high protein content, which predisposes to a low risk of infections.

Case Report: This report presents a rare case of spontaneous fungal peritonitis in a patient with cardiogenic ascites. To the best of our knowledge, this is the second known case of SFP occurring in a patient with cardiac cirrhosis. The patient did not respond to initiation of SBP treatment and after ascitic fluid grew *Candida glabrata*, the diagnosis of SFP was made. The patient's clinical status improved after initiation of intravenous caspofungin.

Conclusions: SFP should be a differential diagnosis in patients who have cardiac or liver cirrhosis, who are not improving with empirical antibiotic therapy for spontaneous bacterial peritonitis.

MeSH Keywords: Ascites • *Candida glabrata* • Peritonitis

Full-text PDF: <https://www.amjcaserep.com/abstract/index/idArt/917757>

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Background

Spontaneous fungal peritonitis (SFP) is an infection defined as a neutrophil count (>250 cells/mL) in ascitic fluid with the evidence of a positive fungal culture [1], while excluding other intra-abdominal infections. SFP is not as common as spontaneous bacterial peritonitis and has a mortality rate due to late recognition and difficulty in differentiation between SFP and systolic blood pressure (SBP) [1,2]. The common risk factors that have been shown to increase the mortality of SFP include hepatorenal syndrome, patient on SBP prophylaxis, high APACHE II scores on admission, increased lactate levels, history of alcoholic cirrhosis, and an elevated Model for End-Stage Liver Disease (MELD) score [2,3]. In the *Candida* species, *Candida glabrata* and *Candida albicans* are the 2 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 [5].

Case Report

This case is of a 52-year-old female with a past medical history of chronic obstructive pulmonary disease who was admitted to the hospital for a 2-week history of abdominal pain and shortness of breath. The abdominal pain was associated with worsening distension. On admission, she was febrile with Tmax of 38.3°C (101°F), tachycardic with a heart rate of 110 beats per minute, and blood pressure of 100/80 mmHg. Cardiovascular examination was positive 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 7900/mm³, absolute neutrophil count (ANC) of 6936/mm³, hemoglobin 15.8 g/dL, hematocrit 60.3, mean corpuscular volume (MCV) 66.5, platelet count 406×10³/mm³, blood urea nitrogen 10 mg/dL, creatinine 0.9 mg/dL, total bilirubin 3.40 mg/dL with direct bilirubin of 2.30 mg/dL, aspartate aminotransferase (AST) 43 U/L, alanine aminotransferase 28 U/L, lipase of 43 U/L, ammonia of 51, pro-BNP (B-type natriuretic peptide) of 4447, and initial albumin of 4.5 g/dL. Other chemistries included sodium of 120 mmol/L, potassium of 4.9 mmol/L, chloride of 84 mmol/L, and bicarbonate of 33 mmol/L. Arterial blood gas

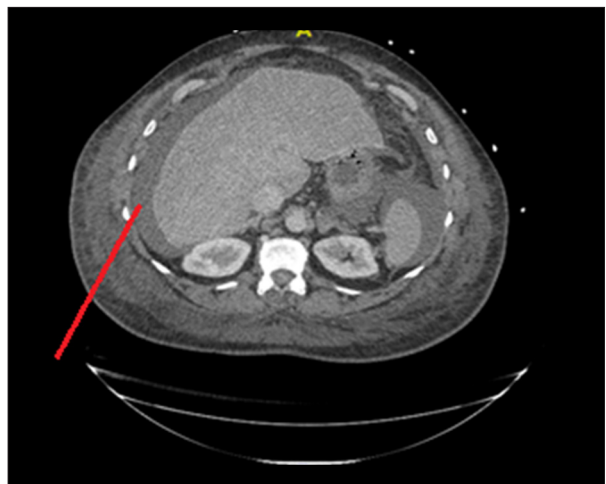


Figure 1. Computed tomography scan demonstrating a moderate amount of ascites, with peritoneal thickening and enhancement consistent with peritonitis.

on admission showed a pH of 7.33 with pCO₂ 58.7 mmHg, PO₂ 75, and bicarbonate of 30 mmol/L on FiO₂ of 100%. Hepatitis A, B, and C panel and human immunodeficiency virus (HIV) were nonreactive. Autoimmune workup including ANA and anti-smooth muscle antibodies were also negative. Initial imaging of the chest x-ray showed only a right-sided pleural effusion, but computed tomography (CT) of the abdomen and pelvis showed bilateral pleural effusions with consolidation on the right, moderate ascites, and liver cirrhosis with no focal lesion (Figure 1). An echocardiogram showed normal left ventricular ejection fraction of 55%, dilated right ventricle, moderate tricuspid regurgitation with right ventricular systolic pressure of 76 mmHg 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 was not able to maintain saturation above 70% on room air after high flow oxygen via venti mask and non-rebreather and had increased respirations of more than 35 breaths per minute; the decision was made to intubate. Antibiotics were also changed to vancomycin and piperacillin-tazobactam. The ascitic fluid analysis showed hazy fluid with a specific gravity 1.023 with a WBC count of 23 000 mm³, with 92% of polymorphic cells and 8% of mononuclear cells, ascitic albumin of 3.1 g/dL, with a serum ascites albumin gradient (SAAG) of 1.4 g/dL. 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 intravenous caspofungin. Two sets of blood cultures were negative. The patient responded gradually

Table 1. Spontaneous fungal peritonitis in the background of cardiac cirrhosis.

| Reference | Year | Age | Risk factors | SAAG ratio | Speciation; Day received | Complications | Treatment | Successful outcome |
|------------------|------|-----|---------------------------------------------------|------------|----------------------------------|---------------------------------------------------------------------------------|-------------------------------------------------------|--------------------|
| Wang et al. [20] | 2017 | 50 | Alcohol abuse, CHF with reduced ejection fraction | 2.0 g/dL | <i>Candida glabrata</i> ; Day 17 | No respiratory distress or intubation | Piperacillin-tazobactam IV caspofungin | Yes |
| Present case | 2019 | 52 | COPD | 1.4 g/dL | <i>Candida glabrata</i> , Day 10 | Pleural effusion; acute hypoxic respiratory failure with indication to intubate | Vancomycin, piperacillin-tazobactam IV caspofungin | Yes |

SAAG – serum ascites albumin gradient; CHF – congestive heart failure; IV – intravenous; COPD – chronic obstructive pulmonary disease.

to antifungals and was extubated on Day 11. Speciation of fluid was reported on Day 10, which indicated *Candida glabrata*. Repeat CT scan of the chest showed significant improvement. Antifungal therapy was continued until the day of discharge on Day 16. Infectious Disease consult recommended the patient to be discharged on intravenous caspofungin and instructed to follow up with the gastroenterologist and infectious disease unit as an outpatient. She was discharged to a rehabilitation facility. The patient was then followed up 1-month post discharge, and she showed significant clinical improvement.

Discussion

Cardiac cirrhosis is a rare but well-documented disease characterized by the presence of signs and symptoms of chronic liver disease along with a history of cardiac failure [6,7]. Usually, it presents with sign and symptoms consistent with congestive heart failure along with clinical features of portal hypertension [8]. The basic pathophysiology includes hypoxic hepatitis due to impaired arterial perfusion (forward failure) and increased hepatic congestion (backward failure) [9]. This backward flow, which is most commonly seen in right heart failure, causes an increase in liver lymphatic production resulting in ascites [10].

Ascites due to heart failure is generally considered at low risk for infections because of the high protein content in the ascitic fluid. The high protein level is optimal for creating a bactericidal environment along with an intact reticuloendothelial system, which differs than SBP [11]. According to another theory, the gut hypothesis states that patients with heart failure will have increased congestion in the gut which leads to chronic intestinal damage, allowing bacteria to translocate to

the ascitic fluid [12]. However, an acute episode of exacerbation may also cause further damage, leading to increase translocation of the fungal organism [12]. 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, and bacterial and fungal culture sensitivities [13]. Cell count and the differential is very important to make the diagnosis of spontaneous fungal peritonitis (polymorphonuclear neutrophils >250 cells/mL). Along with the bacterial Gram stain and culture sensitivity, fungal stain and culture sensitivities are necessary due to the rising prevalence of SFP in patients with cirrhosis [14].

The common fungi causing SFP are *Candida albicans*, *Candida glabrata*, *Candida krusei*, *Cryptococcus* spp, and *Aspergillus* [15]. Risk factors include elevated Child-Pugh and Model for End-Stage Liver Disease (MELD) score, history of prophylactic antibiotics, low ascitic fluid protein (<1 g/dL), recent hospitalization, and hepatorenal syndrome [3,16].

Diagnosing SFP requires culture and sensitivity of the ascitic fluid, 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 tests [16,17]. 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 [18]. As this is the second known case of SFP from cardiac cirrhosis, aggressive management was done immediately to salvage the patient and prompt for a quick recovery compared to the first diagnosed case of SFP with cardiogenic ascites. In the first diagnosed case, there was a delay in diagnosis from getting the speciation of cultures, versus

in our case, immediate cultures including fungal cultures were taken to make a diagnosis of SFP with appropriate sensitivities. Karvellas et al. also reported 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 [19].

Conclusions

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. There has been only one other successfully treated documented case of cardiac cirrhosis from SFP, hence this case provides additional literature on the importance of keeping SFP as a differential diagnosis in patients with cirrhosis (Table 1).

Disclaimer

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