

# *Candida Albicans* in Peritoneal Fluid in a Patient with Hepatic Encephalopathy

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*Candida albicans* is a common human fungal pathogen [1]. *Candida* peritonitis is usually a disease of critically ill surgical patients [2]. Isolation of fungus from ascites fluid in cirrhotic patients is an uncommon event [3] and the clinical significance is unknown. We present the case of a cirrhotic patient who presented with hepatic encephalopathy. She was diagnosed and treated as having spontaneous fungal peritonitis with a temporary good clinical response. We review the literature and discuss the issues regarding the diagnosis and treatment.

## PATIENT DESCRIPTION

A 62 year old woman was admitted to the Internal Medicine department due to hepatic encephalopathy. She had been diagnosed a year previously as having cirrhosis, secondary to hepatitis C virus infection. Complications included ascites, esophageal varices, splenomegaly, hypoalbuminemia and hyperbilirubinemia. She had had recurrent episodes of hepatic encephalopathy, including one a week before her admission secondary to *Escherichia coli* infection, treated with ertapenem.

The patient was admitted in a comatose state which required intubation. She was afebrile and hemodynamically stable. The most impressive physical finding apart from encephalopathy was

ascites. The spleen was non-palpable due to the ascites. There were no obvious triggers for her encephalopathy, such as acute infection, gastrointestinal bleeding, bowel obstruction or use of sedatives. Paracentesis was performed. The ascites fluid cell count was white blood cells 140/ $\mu$ l, with polymorphonuclear cells 25/ $\mu$ l. Gram stain and bacterial cultures were negative. The patient was treated with Laevolac<sup>®</sup> and neomycin.

Three days after admission, *Candida albicans* was identified in fungal culture from peritoneal fluid. Intravenous fluconazole was initiated and the patient was stabilized enough to allow extubation. After 4 days of treatment the encephalopathy resolved, and the ascites cultures were negative. Nevertheless, within 1 week the patient again became encephalopathic despite treatment, and she died from septic shock attributed to extended-spectrum beta-lactamase *E. coli* bacteremia.

## COMMENT

*Candida albicans* is a common human fungal pathogen. It normally serves as a harmless commensal, yet may function as a pathogen among immunocompromised patients or secondary to usage of intravenous catheters [1]. *Candida* peritonitis is usually a disease of critically ill surgical patients. It results from hollow viscus perforation or the treatment of such an event and is associated with significant mortality [2].

Isolation of a fungus from ascitic fluid in cirrhotic patients is an uncommon event. Choi et al. [4] reviewed the cases of 21 cirrhotic patients with ascitic cultures

positive for *Candida* spp. Ten patients had clinical features of peritonitis described as spontaneous *Candida* peritonitis, while 11 patients were classified as having asymptomatic *Candida* isolated from ascites. None of the patients received antifungal therapy. Within 1 year of diagnosis, all patients with spontaneous *Candida* peritonitis died, compared with 54.5% in the asymptomatic group. Ascites fluid polymorphonuclear count > 315 cells/ $\mu$ l had 100% sensitivity in diagnosing spontaneous *Candida* peritonitis [4].

In a recently published study, spontaneous fungal peritonitis was found in 15 of 416 patients (3.6%) diagnosed with spontaneous peritonitis; all of them had a neutrophil count of at least 250 cells/ $\mu$ l. Sixty percent of the patients received antibiotic therapy 90 days prior to the fungal infection. Nosocomial spontaneous peritonitis was significantly more common in the spontaneous fungal peritonitis cohort than in the spontaneous bacterial peritonitis cohort (80% versus 35%,  $P = 0.0009$ ). Only five patients were treated with appropriate antifungal therapy, and only one of them improved. Patients with spontaneous fungal peritonitis had significantly higher 30 day mortality than spontaneous bacterial peritonitis patients (73% vs. 29%) [5].

Previously published data included patients with a higher ascitic neutrophil count than in our patient, and most of the patients did not receive antifungal treatment. Our case is unique because the patient was treated appropriately although she had a very low neutrophil count and her condition improved substantially. Unfortunately she died a few days later due to septic shock and bacteremia.

Candida peritonitis is a rare complication in cirrhotic patients. The definition of an infection versus colonization and its clinical significance and need for appropriate treatment is under debate. After reviewing the literature, we believe that primary fungal peritonitis is underdiagnosed. We suggest that for patients at high risk for fungal peritonitis (prolonged antibiotic therapy, recent hospitalization, and no improvement with empiric antibiotic therapy) [5], fungal cultures from ascitic fluid should be sent for analysis, regardless of neutrophil count. Empiric antifungal therapy should be considered in selected severely ill patients.

This condition is rare and the diagnosis is difficult to ascertain, but our case raises the possibility that positive fungal cultures from ascitic fluid in cirrhotic patients are an indication for infection and not simply harmless colonization, and should be treated appropriately. It seems, however, that regardless of treatment, Candida peritonitis is associated with a poor prognosis in cirrhotic patients. Therefore, larger scale studies are needed to determine both diagnostic and treatment guidelines for this condition.

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