ABSTRACT... One case of spontaneous bacterial peritonitis (SBP) caused by *Listeria monocytogenes* in cirrhotic patients is reported. In our case, the listeris was isolated from ascites from the ascitic fluid. SBP is a serious and common complication of patients with ascites caused by hepatic cirrhosis and the culture of the ascitic fluid is an important tool for the diagnosis and for the more appropriate treatment. Although a third generation cephalosporin has usually been employed for empiric treatment of SBP, it does not provide adequate coverage against *Listeria* spp. In such cases the use of ampicillin (with or without sulbactam) or sulfamethoxazole-trimethoprim is recommended. The sulfamethoxazole-trimethoprim is used for secondary prophylaxis, instead of norfloxacin. To summarize, *Listeria monocytogenes* infection is a rare cause of SBP, whose treatment should be specific for the bacteria.

INTRODUCTION

Spontaneous bacterial peritonitis (SBP) is the most common life-threatening infectious complication in patients with ascites caused by liver cirrhosis. The diagnosis is established by an elevated ascitic fluid polymorphonuclear count ≥250 cells/mm³ and a positive ascitic fluid bacterial culture. The organisms most commonly involved in this infection are *Escherichia coli*, *Klebsiella pneumoniae*, and other Gram-negative enteric organisms, which account for the majority of cases.

*Listeria monocytogenes* is an uncommon cause of peritonitis, with less than 50 cases reported in the medical literature. Early recognition and treatment is critical because of the high mortality rate. Cefotaxime, or other third generation cephalosporins, have been the mainstay of empiric treatment for SBP, but this class of antibiotics is an inadequate treatment for *Listeria*. Ampicillin alone or in combination with gentamicin is the preferred treatment for *L. monocytogenes*.

In this case report we will describe the clinical presentation and treatment of a patient with long-standing ascites who developed SBP with *Listeria*.

CASE REPORT

A 64 year old Hispanic male with past medical history of hypertension and Cirrhosis of Liver with Ascites presented with complaints of abdominal pain and fever for the past 3 days. Abdominal pain started 3 days back insidiously, in the umbilical and left upper quadrant regions; 8/10 in intensity, dull in nature, non radiating with no relieving factors and aggravated on lying flat and on the left side. Fever is high grade, intermittent, associated with chills and rigors. Patient’s history was not significant for the review of other systems. Patient was diagnosed with Alcoholic Liver disease 3 years back and had on and off ascites. Patient drinks 5-6 beers per day since 40 years, but denies smoking or any drug abuse or high risk sexual behavior.

On presentation, he was hemodynamically stable and with a fever of 101.3 degrees Fahrenheit. The initial laboratory evaluation revealed low WBC count of 2.9 with 80% polymorphonuclear white cells, low platelets of 44,000 and normal metabolic panel. The Liver function tests reveal elevated AST, decreased total protein and albumin, and increased total bilirubin of 3.6 mg/dl with a direct bilirubin of 1.5 mg/dl. Serum ammonia, lipase and amylase were within normal limits.

Chest X ray was normal and the CT scan of the abdomen revealed massive ascites, splenomegaly, shrunken liver, retroperitoneal and splenic varices, and recanalisation of...
umbilical vein.

A working diagnosis of Spontaneous Bacterial Peritonitis was made and a diagnostic abdominal paracentesis was performed.

Patient was started on Cefepime for empiric coverage of the usual spontaneous peritonitis pathogens. Vancomycin was added, for empiric staphylococcal coverage.

The ascitic fluid analysis showed 15 white blood cells and the albumin gradient was 1.1, compatible with subacute bacteria peritonitis. There was no bacterial growth after 48 hours. On the third hospital day, he kept spiking fevers of 100 to 102 degrees Fahrenheit with no rise in white blood cell count.

On the fourth day, the peritoneal fluid cultures grew Listeria species. Based on this culture report, the patient was given Ampicillin and Gentamycin after Infectious Disease specialist consultation. Patient reported gradual improvement in his abdominal pain and the fever spikes subsided. There was no change in the white blood cell count. The patient continued to have Ampicillin and Gentamicin for a total of 14 days.

DISCUSSION

Spontaneous bacterial peritonitis (SBP) is a bacterial infection of ascitic fluid, which arises in the absence of any other source of sepsis.

The diagnosis of SBP is made by diagnostic paracentesis and it has been found that the number of neutrophils within ascitic fluid is a remarkably accurate indicator of infection. The neutrophils count of > 250 cells/mm² is considered diagnostic for neutrocytic ascites and empiric antibiotic therapy can be initiated while awaiting results of blood and ascitic fluid cultures. The current gold standard for diagnosis of SBP is the isolation of a single pathogen. The most frequently isolated agents are the gram-negative rods, specially Escherichia coli and Klebsiella pneumoniae, and gram-positive cocci, such as Streptococcus pneumoniae.

In our case report the ascitic fluid culture confirmed SBP caused by Listeria sp. SBP caused by Listeria monocytogenes is a very rare infection. Rheingold et al. were the first to describe spontaneous bacterial peritonitis due to Listeria monocytogenes in cirrhotic patients in 1977. More than 40 cases were reported later and most of them occurred in Spain. The reason for the great incidence in this country is unknown. Cirrhosis has been related in approximately fifty percent of cases and main etiology has been alcoholic cirrhosis. Although many authors have suggested a third-generation cephalosporin such as cefotaxime for the empiric treatment of SBP, it does not provide adequate antibiotic coverage against Listeria spp. In such cases, recent reviews have suggested the use of ampicillin (with or without sulbactam) or sulfamethoxazole-trimethoprim. The use of this last antibiotic is recommended for secondary prophylaxis, instead of norfloxacin. Initially our patient had been treated with a third-generation cephalosporin, but the antibiotic was changed after the isolation of Listeria.

The optimal duration of therapy for SBP caused by Listeria spp. has not been determined. Factors that may determinate the duration of therapy include the resolution of neutrocytic ascites, sterilization of blood and ascitic cultures, and the presence of the organism in sheltered sites, such as the brain, joints, or an abscess. The mortality rate of peritonitis caused by Listeria monocytogenes is high. Sivalingam et al. (1992) observed the mortality rate for the 12 cases of Listeria peritonitis was 25%.

The morphology of Listeria spp. can be confused with that of other gram-positive bacteria and caution should be taken to differentiate from enterococci (catalase-negative and CAMP test negative) or group B streptococci (bile-esculin negative). Because of the characteristic motility of this bacterium in semisolid agar, the motility test can also be used to differentiate it from other gram-positive bacteria. An umbrella-like pattern of growth can be seen several millimeters below the agar surface.

We concluded that the culture of the ascitic fluid is an important tool for the diagnosis. It is more important in
special cases of less common pathogens infection. Listeria spp. is a pathogen that can rarely cause spontaneous bacterial peritonitis in cirrhotic patients.

The overall mortality of cirrhotic patients with listerial infected ascites has been estimated to be 30% and patients die within six days after contracting the infection. The increasing incidence of L. monocytogenes requires early recognition and specific treatment. Infection with L. monocytogenes should be suspected in patients with end-stage liver disease and inadequate response to conventional medical treatment within 48–72 h.

A high index of suspicion should also be maintained in patients with hemochromatosis, impaired cell-mediated immunity, exposure to farm animals and Gram-positive-like organisms in peritoneal fluid or blood.

CONCLUSION

Patients with chronic liver disease have been recently identified as a risk group for invasive listeriosis, including SBP. The risk of listeriosis is markedly increased among newborns, pregnant women, the elderly, and patients with impaired cell-mediated immunity. Iron overload in end-stage liver disease may predispose to SBP caused by Listeria. Listeria is resistant to third-generation cephalosporins, the standard empiric treatment for SBP. Ampicillin/sulbactam and gentamicin should be instituted early, particularly in patients who do not respond promptly to conventional antibiotic regimen.

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REFERENCE


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Correction

The amendment of the Professional Vol:17, No.02 (Prof-1530) on page 211 is as under;

HER 2 NEGATIVE METASTATIC BREAST CANCER;
LOW DOSE SEQUENTIAL DOCETAXEL - CAPECITABINE CHEMOTHERAPY AS FIRST LINE TREATMENT. A CLINICAL TRIAL OF CANCER RESEARCH GROUP PAKISTAN

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