Journal of Community Hospital Internal Medicine Perspectives

COACTION PUBLISHING

CASE REPORT

# *Listeria monocytogenes* as a cause of spontaneous bacterial peritonitis: a rare entity

**Bilal Shaikh, DO, Ranjan Pathak, MD*, Naba Raj Mainali, MD and Shobhit Gupta, DO**

Department of Internal Medicine, Reading Health System, West Reading, PA, USA

*Listeria* is an uncommon cause of spontaneous bacterial peritonitis (SBP) in the United States. *Listeria* should be suspected as a cause of SBP when the patient has diphtheria-like organisms on ascitic/blood cultures, iron overload/hemochromatosis, exposure to farm animals, or poor response to empiric therapy within 48–72 h. Diagnosis of SBP is made if the ascitic fluid shows polymorphonuclear cell count $>250$ cells/mm$^3$ without an intra-abdominal source of infection. Ampicillin with or without an aminoglycoside is the treatment of choice. Trimethoprim-sulfamethoxazole is recommended for prophylaxis in patients with a previous episode of *Listeria* SBP.

Keywords: Listeria monocytogenes; *spontaneous bacterial peritonitis*; *cirrhosis*; *ampicillin*

*Correspondence to: Ranjan Pathak, Department of Internal Medicine, Reading Health System, West Reading, PA 19611, USA, Email: ranjanrp@gmail.com

Received: 29 September 2014; Accepted: 20 November 2014; Published: 3 February 2015

Spontaneous bacterial peritonitis (SBP) is found to be the cause of infection in 25% of patients admitted with cirrhosis (1). Enteric gram-negative organisms such as *Escherichia coli* are the most common causes of SBP in adults (1). *Listeria monocytogenes*, however, is an uncommon cause of SBP in cirrhotic patients in the United States with only around 50 cases reported in the literature (2). *Listeria* should be suspected as a cause of SBP if the patient has iron overload/hemochromatosis, history of exposure to farm animals, or poor response to empiric antibiotic therapy within 48–72 h (3). Early effective treatment is essential as the mortality can be as high as 25 to 30% (4, 5).

## Case presentation

A 65-year-old Caucasian woman with a medical history of primary biliary cirrhosis presented with fever, chills, increasing shortness of breath, abdominal pain with distension, nausea, and vomiting for 5 days. She denied any cough, chest pain, palpitations, bipedal edema, paroxysmal nocturnal dyspnea, or obstipation. She denied any alcohol or drug abuse. Family history was negative for any liver disease. Vital signs on admission were as follows: blood pressure 136/61 mmHg, pulse 108/min, respiratory rate 30/min, temperature 38.2°C, and oxygen saturation of 95% on room air. On physical examination, she was anicteric with rales over the left lung base with decreased breath sounds. Her abdomen was moderately distended with diffuse tenderness. However, no rebound tenderness or guarding was elicited. Her white blood cell (WBC) counts were elevated at $14.6 \times 10^9$/L. Her liver function tests revealed an elevated aspartate aminotransferase (AST) of 101 IU/L, total bilirubin of 2.5 mg/dL with a direct bilirubin of 0.8 mg/dL, decreased serum albumin of 2.5 g/dL, and normal alkaline phosphatase, alanine transaminase, and gamma-glutamyl transferase levels. Her lactate was elevated at 8.8 mEq/L with normal serum iron levels. Chest radiography revealed a left pleural effusion with left lower lobe consolidation. Plain and upright abdominal radiography was negative for any air-fluid levels or free gas under the diaphragm. Computed tomography of the abdomen showed cirrhotic liver with portal hypertension, splenomegaly, esophageal varices, and moderate ascites. Diagnostic abdominal paracentesis showed total WBC counts of $10.4 \times 10^9$/L with 8,075/mm$^3$ neutrophils. The serum-ascites albumin gradient was greater than 1.1 g/dL. The ascitic fluid culture and blood cultures came back positive for *L. monocytogenes*.

We considered the differential diagnosis of pneumonia, peritonitis due to viscus perforation, and SBP. SBP due to *L. monocytogenes* was diagnosed on the basis of abdominal tenderness and ascitic fluid neutrophil count of more than 250/mm$^3$ with cultures positive for *Listeria* and absence of secondary causes of peritonitis. Alcoholic hepatitis was unlikely as she had no history of alcohol use.





Citation: Journal of Community Hospital Internal Medicine Perspectives 2015, **5**: 26153 - http://dx.doi.org/10.3402/jchimp.v5.26153

She was started on cefepime and azithromycin for the community acquired pneumonia. She was given ampicillin and gentamycin for the peritonitis based on the antibiotic sensitivity report from the ascitic fluid culture. After the initiation of antibiotics, her repeat paracentesis 48 h later, showed marked decrease in neutrophils from 8,075/mm$^3$ initially to 168/mm$^3$ with sterile blood and ascitic cultures.

The patient received 28 days' therapy with ampicillin for *Listeria* SBP. She was discharged from the hospital after a prolonged stay of more than 1 month for multi-organ dysfunction. She is currently undergoing rehabilitation for critical illness myopathy secondary to prolonged ICU stay. She is also being followed by a tertiary care center for liver transplantation.

## Discussion

Cirrhosis leads to general immune dysfunction with a specific cirrhosis-associated immune dysfunction syndrome which is a systemic state of immune dysregulation. Liver houses 90% of the reticuloendothelial cells (such as Kupffer and sinusoidal endothelial cells) that are crucial to the eradication of bacteria. Monocyte migration and bacterial phagocytosis is weakened in cirrhotic patients. Other factors in cirrhotics that reduce immune system function are hyponatremia and hyperammonemia.

*L. monocytogenes* is a gram-positive, motile, aerobic, or facultative anaerobic bacillus. *Listeria* has been known to have predilection for newborns and immunocompromised adults such as pregnant patients, elderly, those on immunosuppressive therapies or continuous peritoneal dialysis cancer, or AIDS patients (6, 7). The use of proton pump inhibitors has also been implicated as a risk factor for SBP (8). Human cases of *Listeria* are usually linked to transmission from food products such as dairy or meat products. Clinically, *Listeria* manifests as septicemia, meningitis, endocarditis, gastroenteritis, or peritonitis (6, 9, 10).

Rheingold et al. described the first case of SBP due to *Listeria* in 1977 (6, 11). *Listeria* should be suspected as a cause of SBP when the patient has diphtheria-like organisms on ascitic/blood cultures, iron overload/hemochromatosis, exposure to farm animals, or poor reaction to empiric therapy within 48–72 h (3). At least 50 cases of *L. monocytogenes* SBP have been described in the medical literature, with many of them found in Spain. This relatively high prevalence of *L. monocytogenes* in Spain is theorized to be secondary to the ingestion of dairy products, raw fruits, and vegetables in that region. It has also been found in Brazil, Mexico, Taiwan, Australia, Israel, and France (12).

Diagnosis of SBP is made with a polymorphonuclear cell count $>250$ cells/mm$^3$ without an intra-abdominal source of infection. SBP can present with hyperthermia or hypothermia, chills, tachypnea, tachycardia, abdominal pain and tenderness, vomiting, diarrhea, or ileus with a leukocytosis on complete blood count. Other possible presentations include worsening of liver function, altered mental status from hepatic encephalopathy, shock, renal failure, or gastrointestinal bleed.

Ascitic fluid culture is positive in only about 40% of cases. Typical organisms are gram-negative agents such as *E. coli* and *Klebsiella pneumoniae*, and gram-positive microbe such as *Streptococcus pneumoniae* which account for about 95% of cases (6). Patients with negative ascitic fluid culture and ascitic fluid polymorphonuclear cell count $>250$ cells/mm$^3$ have culture negative SBP and should still be treated the same as a culture-positive patient. Those patients with bacterascites (positive cultures with a normal ascitic polymorphonuclear cell count) are either colonized secondary to infections outside the peritoneum or early progression of SBP. These patients should be treated as if they have systemic infection if they exhibit signs/symptoms of SBP. If not, it is recommended that a second paracentesis be performed once the culture comes back positive. If ascitic fluid neutrophil count is $>250$ cells/mm$^3$, then the patient should be treated for SBP. If the WBC count is less than 500 cells/mm$^3$, the patient should be followed up. All patients with suspected SBP should have blood cultures drawn before the initiation of antibiotic therapy (13).

Empiric treatment for SBP is usually recommended with a third generation cephalosporin such as cefotaxime, but it does not provide adequate coverage for *Listeria* (6). Ampicillin with or without an aminoglycoside is the treatment of choice. Aminoglycosides should be used carefully because of associated nephrotoxicity and ototoxicity (3). Trimethoprim-sulfamexothazole and erythromycin have been used successfully in the past in patients with penicillin allergy (2). Repeat paracentesis is recommended 2 to 3 days after the initiation of therapy in order to determine therapeutic response on top of clinical progression. A decrease of 25% in ascitic fluid WBC count on repeat paracentesis in 48 h is desirable. If the patient is not responding by this time period, resistant organisms or secondary peritonitis should be suspected. In patients on norfloxacin prophylaxis, gram-positive organisms are usually suspected and cefotaxime is the initial drug of choice. The optimal length of therapy for *Listeria* SBP has not yet been determined but is suggested to be longer than 10–14 days (6, 10). In our case, we treated the patient with ampicillin for 28 days.

Albumin with antibiotics has been shown to decrease the occurrence of hepatorenal syndrome from 30 to 9% compared to the use of antibiotics alone. The European Association for the Study of the Liver suggests that albumin be administered at admission because it has been shown to decrease the incidence of hepatorenal syndrome and has a survival benefit. It has been particularly recommended in those patients with serum bilirubin $>4$ mg/dL or serum creatinine $>1$ mg/dL (13).


Citation: Journal of Community Hospital Internal Medicine Perspectives 2015, **5**: 26153 - http://dx.doi.org/10.3402/jchimp.v5.26153

In patients who survive an initial episode of SBP, the recurrence rate within the next year is 70% (6). Norfloxacin prophylaxis is indicated in cirrhotic patients with acute gastrointestinal bleed (with or without ascites), as primary prophylaxis in patients with low total protein in ascitic fluid, and as secondary prophylaxis in cirrhotic patients with previous episodes of SBP (3, 13). Trimethoprim–sulfamethoxazole is recommended for prophylaxis in patients with a previous episode of *Listeria* SBP.

## Conflict of interest and funding

The authors have not received any funding or benefits from industry or elsewhere to conduct this study.

## References

1. Bonnel AR, Bunchorntavakul C, Reddy KR. Immune dysfunction and infections in patients with cirrhosis. Clin Gastroenterol Hepatol 2011; 9(9): 727–38. doi: 10.1016/j.cgh.2011.02.031.
2. Ahmad M, Krishnan A, Kelman E, Allen V, Bargman JM. *Listeria monocytogenes* peritonitis in a patient on peritoneal dialysis: A case report and review of the literature. Int Urol Nephrol 2008; 40(3): 815–19. doi: 10.1007/s11255-008-9411-2.
3. Brann OS. Infectious complications of cirrhosis. Curr Gastroenterol Rep 2001; 3(4): 285–92. doi: 10.1007/s11894-001-0051-2.
4. Jammula P, Gupta R. *Listeria monocytogenes*-induced monomicrobial non-neutrocytic bacterascites. South Med J 2002; 95(10): 1204–6.
5. Nolla-Salas J, Almela M, Gasser I, Latorre C, Salvadó M, Coll P. Spontaneous *Listeria monocytogenes* peritonitis: A population-based study of 13 cases collected in Spain. Am J Gastroenterol 2002; 97(6): 1507–11. doi: 10.1111/j.1572-0241.2002.05798.x.
6. Cardoso C, Cremers I, Oliveira AP. Spontaneous bacterial peritonitis caused by *Listeria monocytogenes*: A case report and literature review. Ann Hepatol 2012; 11(6): 955–7.
7. Jayaraj K, Di Bisceglie AM, Gibson S. Spontaneous bacterial peritonitis caused by infection with *Listeria monocytogenes*: A case report and review of the literature. Am J Gastroenterol 1998; 93(9): 1556–8. doi: 10.1111/j.1572-0241.1998.00482.x.
8. Jayasinghe S, Connor M, Donaldson S, Austin H, Foster A. Spontaneous bacterial peritonitis due to *Listeria monocytogenes*: Importance of enrichment culture. J Clin Pathol 2010; 63(9): 835–6. doi: 10.1136/jcp.2009.074641.
9. Tablang MVF. Spontaneous bacterial peritonitis caused by infection with *Listeria monocytogenes*. Case Rep Gastroenterol 2008; 2(3): 321–5. doi: 10.1159/000151579.
10. Frachtman S, Lu L, Lau M, Greenberg S. Spontaneous bacterial peritonitis due to *Listeria monocytogenes*: A case report and a review of *Listeria monocytogenes* peritonitis. Infect Dis Clin Pract 2009; 17(1): 63–5. doi: 10.1097/IPC.0b013e31818127a2.
11. Rheingold OJ. Spontaneous peritonitis of cirrhosis due to *Listeria monocytogenes*. Ann Intern Med 1977; 87(4): 455. doi: 10.7326/0003-4819-87-4-455_2.
12. European Association for the Study of the Liver. EASL clinical practice guidelines on the management of ascites, spontaneous bacterial peritonitis, and hepatorenal syndrome in cirrhosis. J Hepatol 2010; 53(3): 397–417. doi: 10.1016/j.jhep.2010.05.004.
13. Toyoshima MTK, Apanavicius A, de Matos Soeiro A, de Almeida GMD, Arai MH. *Listeria monocytogenes* peritonitis in cirrhotic patients: First description in Brazil. Rev Inst Med Trop São Paulo 2006; 48(5): 291–3.