

## Spontaneous bacterial peritonitis caused by *Listeria monocytogenes*: a case report and literature review

Cláudia Cardoso, Isabelle Cremers, Ana Paula Oliveira

Department of Gastroenterology, Centro Hospitalar de Setúbal, Setúbal, Portugal.

### ABSTRACT

Spontaneous bacterial peritonitis (SBP) is a common and often serious complication of long standing ascites in the presence of advanced liver disease.<sup>1</sup> We report a case of a 51-year-old man with alcoholic cirrhosis admitted to our department with jaundice, ascitis and lower limbs edema. A diagnosis of spontaneous bacterial peritonitis was made and empiric therapy with cefotaxime was prescribed with no response. Three days later *Listeria monocytogenes* was detected in peritoneal fluid culture and amoxicillin was initiated according to in vitro sensibility test. Despite adequate antibiotic therapy, the patient died one week later.

**Key words.** Alcoholic cirrhosis. Gram positive rod.

### INTRODUCTION

The diagnosis of SBP is made when there is an elevated ascitic fluid absolute polymorphonuclear (PMN) count (i.e.,  $\geq 250$  cells/mm<sup>3</sup>) without an intra-abdominal surgically treatable source of infection.<sup>1</sup>

The prevalence of SBP in cirrhotic outpatients is 1.5-3.5%, whereas in inpatients it is nearly 10%, with half of the episodes being present at the time of hospital admission while the rest is acquired during hospitalization.<sup>2</sup>

Patients with SBP may have one of the following:

- Local symptoms and/or signs of peritonitis: abdominal pain, abdominal tenderness, vomiting, diarrhea, ileus.
- Signs of systemic inflammation: hyper or hypothermia, chills, altered white blood cell count, tachycardia, and/or tachypnea.

- Worsening of liver function.
- Hepatic encephalopathy.
- Shock.
- Renal failure; and
- Gastrointestinal bleeding.

However, it is important to point out that SBP may be asymptomatic, particularly in outpatients.<sup>2,3</sup>

When first described, SBP mortality exceeded 90% but it has been reduced to approximately 20% over the years due to an increased awareness of this condition, routine use of diagnostic paracentesis and prompt prescription of adequate antibiotic therapy.<sup>4,5</sup>

Ascitic fluid bacterial culture is positive in about 40% of cases. Gram negative agents of the *Enterobacteriaceae* family, such as *Escherichia coli* and *Klebsiella pneumoniae*, and gram positive cocci, such as *Streptococcus pneumoniae*, are isolated in about 95% of patients, as the result of their translocation from the bowel into the peritoneal cavity.<sup>6-8</sup>

*Listeria monocytogenes* is a gram positive rod that has been increasingly recognized as a pathogen in immunocompromised, with an estimated annual incidence of 0.2 cases per 100,000 population in Europe and United States. It has been isolated as the infectious agent in cases of meningitis, endocarditis, bacteremia with or without sepsis and, more rarely, SBP.<sup>10</sup>

Correspondence and reprint request: Cláudia Cardoso, M.D.  
Rua Camilo Castelo Branco  
2910 Setúbal, Portugal.  
E-mail: claudiamarcal@gmail.com

## CASE REPORT

A 51-year-old man with alcoholic cirrhosis was admitted to our hospital with a three day history of ascitis, peripheral edema and jaundice. The patient denied fever, changes in level of consciousness, vomits, abdominal pain, diarrhea, ileus or gastrointestinal bleeding.

Physical examination revealed a temperature of 36.7 °C, no signs of encephalopathy, blood pressure, heart and respiratory rates within normal ranges, jaundice of the skin and sclerotics, a large volume ascites and peripheral edema.

Blood tests showed:

- Hemoglobin 12.7 g/dL (range 13-17).
- Leucocytes 13,200/ $\mu$ L (range 4,500-11,400) with 84.8% PMN.
- Platelets 130,000/ $\mu$ L (range 150,000-450,000).
- Total bilirubin 5.98 mg/dL (range 0.2-1).
- International normalized ratio-1.8 (range 0.8-1.2).
- Creatinine serum level-1.0 mg/dL (range 0.7-1.3), and
- Albumin-2.3 g/dL (range 3.5-5).

A diagnostic paracentesis was performed showing a serum-ascites albumin gradient  $\geq 1.1$  g/dL with 19,600 leucocytes/ $\mu$ L (absolute neutrophil count of 16,450). A diagnosis of SBP was made and intravenous cefotaxime 2 g every 12 h was promptly prescribed after ascitic fluid inoculation into aerobic blood culture. Despite empiric antibiotic therapy, there was a worsening of peripheral and ascitic fluid white blood cell count at 48 h analytical control.

Three days after the admission, ascitic fluid culture yielded a positive result for *Listeria monocytogenes*. Cefotaxime was discontinued and the patient started amoxicillin 2.2 g iv every 8 h according to in vitro sensibility test, that had also revealed resistance of this rod to cefotaxime.

However, despite appropriate antibiotic, a rapid clinical and analytical deterioration was observed with the onset of encephalopathy and oliguric renal failure that led to patient's death 7 days after the admission.

## DISCUSSION

The first case of SBP due to *Listeria monocytogenes* was described in 1977 by Rheingold, *et al.*<sup>11</sup> Since then, about 40 cases have been reported in the literature.<sup>9,11</sup>

*Listeria monocytogenes* is a gram positive facultative anaerobic rod that has been isolated from water, soil, sewage, dust, decaying vegetable matter, poultry and dairy products. It is also found in the fecal flora of many mammals, including 5% of asymptomatic healthy adults.<sup>12</sup>

This organism is particularly likely to infect newborns and adults with impaired cell mediated immunity such as pregnancy, old age, immunosuppressive therapy, malignancies, continuous ambulatory peritoneal dialysis and acquired immunodeficiency syndrome. It appears to have a tropism for placenta, meningis and endocardium.<sup>13-17</sup>

One of the implicated mechanisms in the development of SBP is a disturbance in gut flora with bacterial overgrowth and intestinal translocation. Cirrhosis predisposes to the development of bacterial overgrowth, possibly because of altered small intestinal motility, and to an increased intestinal permeability that favors intestinal translocation. Therefore, it seems reasonable to presume that the transmission of *Listeria monocytogenes* occurs by fecal-oral route with subsequent colonization of the bowel and translocation into the peritoneal cavity.

It is also known that iron enhances the growth of this bacteria *in vitro*. Individuals with end stage liver disease can have increased body iron stores that may favor *Listeria* induced SBP.

Infection with *Listeria spp* should be considered when there are gram positive bacilli-like organisms presence in the peritoneal fluid or in the blood, in patients with previously mentioned epidemiological risk factors and when there is an inadequate response to conventional medical treatment in the first 48 to 72 h.<sup>18,19</sup>

Although current guidelines suggest a third generation cephalosporin such as cefotaxime for the empiric treatment of SBP,<sup>6</sup> it does not provide an adequate antibiotic coverage against *Listeria spp*. In such cases recent reviews have suggested the use of ampicillin (with or without an aminoglycoside) or trimethoprim-sulphamethoxazole for 10 to 14 days. However, the optimal antibiotic therapy and its duration are not well defined.<sup>9</sup>

In patients who survive an episode of SBP, the cumulative recurrence rate at one year is approximately 70%. The administration of prophylactic antibiotics has proved to reduce the level of recurrence to 20%. Norfloxacin (400 mg/day, orally) is the treatment of choice. Alternative antibiotics include ciprofloxacin (750 mg once weekly, orally) or co-trimoxazole (800 mg sulfamethoxazole and 160 mg trimethoprim daily, orally), but evidence is not as

strong as that with norfloxacin.<sup>6</sup> *Listeria monocytogenes* usually is not sensitive to norfloxacin and the use of trimethoprim-sulfamethoxazole is advised in these patients.<sup>1</sup> The probability of survival at 1 year after an episode of SBP is 30-50% and falls to 25-30% at 2 years. Therefore, patients recovering from an episode of SBP should be considered for liver transplantation.<sup>20</sup>

## CONCLUSION

*Listeria monocytogenes* induced SBP is a rare condition. Clinicians should have a high index of suspicion when a patient does not respond promptly to conventional antibiotic therapy, especially if risk factors for this agent transmission are present. The optimal antibiotic therapy and its duration are not well defined. Ampicillin in monotherapy or in association with an aminoglycoside has been advised as the standard therapy and trimethoprim-sulphamethoxazole seems to be more appropriate for secondary prophylaxis than norfloxacin.

## REFERENCES

1. Hoefs JC, Canawati HN, Sapico FL, Hopkins RR, Weiner J, Montgomerie JZ. Spontaneous bacterial peritonitis. *Hepatology* 1982; 2: 399-07.
2. Nousbaum JB, Cadranel JF, Nahon P, Nguyen Khac E, Moreau R, Thévenot T. Diagnostic accuracy of the Multistix 8 SG reagent strip in diagnosis of spontaneous bacterial peritonitis. *Hepatology* 2007; 45: 1275-81.
3. Plessier A, Denninger MA, Consigny Y, Pessione F, Francoz C, Durand F. Coagulation disorders in patients with cirrhosis and severe sepsis. *Liver Int* 2003; 23: 440-48.
4. Tandon P, Garcia-Tsao G. Bacterial infections, sepsis, and multiorgan failure in cirrhosis. *Semin Liver Dis* 2008; 28: 26-42.
5. Garcia-Tsao G. Current management of the complications of cirrhosis and portal hypertension: variceal hemorrhage, ascites, and spontaneous bacterial peritonitis. *Gastroenterology* 2001; 120: 726-48.
6. Caly WR, Strauss E. A prospective study of bacterial infections in patients with cirrhosis. *J Hepatol* 1993; 18: 353-58.
7. Fernández J, Navasa M, Gómez J, Colmenero J, Vila J, Arroyo V. Bacterial infections in cirrhosis: epidemiological changes with invasive procedures and norfloxacin prophylaxis. *Hepatology* 2002; 35: 140-48.
8. Wong F, Bernardi M, Balk R, Christman B, Moreau R, Garcia-Tsao G. Sepsis in cirrhosis: report on the 7th meeting of the International Ascites Club. *Gut* 2005; 54: 718-25.
9. 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: 1556-58.
10. Rheingold OJ, Chiprut RO, Dickinson GM, Schiff ER. Spontaneous peritonitis of cirrhosis due to *Listeria monocytogenes*. *Ann Intern Med* 1977; 87: 455-56.
11. Arias Miranda IM, Nuño Mateo FJ, Noval Menéndez J, Fonseca Aizpuru EM, Menéndez Calderón MJ. Listeriosis in the adult. Revision of 10 cases. *An Med Interna* 2004; 21: 75-78.
12. Suárez MM, Bautista RM, Almela M, Soriano A, Marco F, Bosch J, Martínez JA, et al. *Listeria monocytogenes* bacteriemia: analysis of 110 episodes. *Med Clin (Barc)* 2007; 129: 218-21.
13. Riviero GA, Torres HÁ, Rolston KV, Kontoyiannis DP. *Listeria monocytogenes* infection in patients with cancer. *Diagn Microbiol Infect Dis* 2003; 47: 393-8.
14. Nolla-Salas J, Plasencia A, Gasser I, Almeda M, Coll P, Anto JM. Estudio clinic-epidemiológico de la listeriosis humana en Barcelona (1990-1991). *Med Clin* 1994; 103: 41-5.
15. Chang J, Powles R, Metha J, Paton N, Treleaven J, Jameson B. Listeriosis in bone marrow transplant recipients: incidence, clinical features and treatment. *Clin Infect Dis* 1995; 21: 1289-90.
16. Doganay M. Listeriosis: clinical presentation. *FEMS Immunol Med Microbiol* 2003; 35: 173-5.
17. Bierhoff M, Krutwagen E, van Bommel EF, Verburgh CA. *Listeria* peritonitis in patients on peritoneal dialysis: two cases and a review of the literature. *Neth J Med*. 2011; 69: 461-4.
18. Lorber B. Clinical listeriosis-implications for pathogenesis. In: Miller AL, Smith JL (eds.). *Foodborne listeriosis*. Society for Industrial Microbiology. New York: Elsevier Science; 1990, p. 41-49.
19. Nieman RE, Lorber B. Listeriosis in adults: a changing pattern. Report of eight cases and review of literature. *Rev Infect Dis* 1980; 2: 207-27.
20. 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: 397-17.