## LETTERS TO THE EDITOR

## Pleural Effusion Due to *Listeria* monocytogenes in a Woman With Cirrhosis of the Liver

To the Editor: Infection due to Listeria monocytogenes tends to affect pregnant women and immunocompromised patients (cancer patients, those undergoing immunosuppressant treatment or infected with the human immunodeficiency virus) though there are cases in the literature that link it to other conditions, including cirrhosis of the liver. The most common clinical presentations are central nervous system manifestations and sepsis, though there are other, very rare forms of infection that affect, for example, the pleural fluid.1 We present the case of a patient with cirrhosis of the liver and pleural effusion complicated by infection with *L monocytogenes*. Response to antibiotic treatment was good.

The patient was a 70-year-old woman with Childs class B cirrhosis of the liver due to the hepatitis C virus, with portal vein thrombosis, episodes of upper digestive tract bleeding, and fluid retention. She had last been admitted with ascites and associated hydrothorax, which responded to treatment with diuretics. She came to the emergency department with ascites, edema, and shortness of breath that had worsened over the previous 2 weeks. Auscultation of the lungs revealed reduced vesicular sounds in the lower two thirds of the left hemithorax. The hemogram showed only thrombocytopenia (76×109 cells/L) and basic biochemistry showed increased levels of creatinine (1.9 mg/dL), bilirubin (3.1 U/L), γ-glutamyl transpeptidase (71 U/L), and alkaline phosphatase (268 U/L), and a decrease in albumin levels (2.3 U/L). A chest x-ray revealed a pleural effusion occupying the lower two thirds of the left hemithorax. The symptoms were diagnosed as a new episode of fluid retention with hydrothorax. The pleural effusion persisted despite raising the diuretic dosage and the patient developed a low-grade fever. Diagnostic paracentesis was performed and a culture of the fluid showed it to be sterile, thus ruling out spontaneous bacterial peritonitis. A thoracentesis performed on the same day showed the following results: white cell count, 4.12×109 cells/L (75% neutrophils); glucose, 262 mg/dL; total proteins, 3.87 g/dL; lactate dehydrogenase, 156 U/L; and pH, 7.33. The fluid extracted was an exudate containing polymorphonuclear cells. This indicated the effusion was probably parapneumonic. L monocytogenes sensitive to ampicillin, gentamicin, and trimethoprimsulfamethoxazole was cultured from the sample of pleural fluid sent for microbiology tests. A thoracentesis performed 2 days later produced

A thoracentesis performed 2 days later produced a culture positive for *Listeria* species and computed tomography of the chest showed a large left pleural effusion (figure). Blood cultures and tests to rule out infection of the central nervous system by *Listeria* species were normal.

Treatment was started with intravenous ampicillin and the clinical and radiological response was good. Due to the development of leukopenia (2.55×10° cells/L) and severe thrombopenia (32×10° cells/L), we replaced the ampicillin with intravenous cotrimoxazole to complete a 4-week course of treatment. Full remission of the effusion was achieved.

To date, 19 cases of pleural fluid infection with *L monocytogenes* have been described, mostly in immunocompromised patients with blood cancer. Only 3 such infections have been described in patients with cirrhosis of the liver. The mechanism by which the bacteria reaches the pleural cavity in cases of cirrhosis of the liver is unclear although spread via the bloodstream from a primary focus of infection (bacterial peritonitis or meningocephalitis) and direct inoculation with the bacteria during thoracentesis have been suggested.

We do not know the exact mechanism by which L monocytogenes reached the pleural cavity in our patient as the bacillus was not isolated in the blood cultures or in the ascitic or cerebrospinal fluids; yet it was isolated after thoracenteses performed on 2 different days. The treatment of choice is usually ampicillin or penicillin, associated with an aminoglycoside—generally gentamicin. Cotrimoxazole is normally used in cases of central nervous system involvement because of its good penetration into the cerebrospinal fluid. A thoracic drain should be inserted in cases of complicated effusion. Treatment may last from a minimum of 2 weeks to a maximum of 6 weeks in immunocompromised patients.6 Based on the results of the antibiogram, we prescribed intravenous ampicillin without aminoglycoside because the patient was suffering from renal insufficiency. The ampicillin was replaced with co-trimoxazole after 2 weeks due to the onset of leukopenia and worsening thrombopenia. Treatment lasted 4 weeks in total.

The prognosis for patients with infection due to *L monocytogenes* and cirrhosis of the liver is unknown because of the small number of described cases. Of the 3 cases published to date, only 1 patient died,

from digestive tract bleeding, specifically related to esophageal varices. The outcome was favorable for our patient, with complete remission of the pleural effusion.

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Computed tomography of the chest, showing right pleural effusion and compressive subsegmental pulmonary atelectasis.