Spontaneous bacterial peritonitis caused by Listeria monocytogenes

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SUMMARY

A case of spontaneous bacterial peritonitis caused by Listeria monocytogenes in a patient with non-alcoholic cirrhosis is reported. The patient was initially treated with cefotaxime, antibiotic often selected as empiric therapy of all episodes. The increasing cephalosporins implication of Listeria spp to this type of infection and the intrinsic resistance of all isolates to 3rd generation are noted.

Key words: Listeria monocytogenes, spontaneous peritonitis, cirrhosis

Spontaneous bacterial peritonitis (SBP) is a relatively rare complication in patients with liver disease. retrospective studies showed that the incidence of SBP in this group of patients is about 10 percent.¹ Enteric bacteria with predominance of Escherichia coli are the most frequently recovered microorganisms followed by Streptococcus pneumoniae and group A streptococci.¹ Recently, Listeria monocytogenes has been increasingly implicated in SBP.²⁻⁷ Most patients has a liver cirrhosis as underlying disease. we present another case of spontaneous bacterial peritonitis due to L. monocytogenes in a patient with non-alcoholic cirrhosis.

CASE REPORT

A 71-year old woman was admitted to the hospital

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N. Evgenidis, Department of Gastroenterology, Hippokration General Hospital, Konstantinoupoleos 46, Thessaloniki, Greece with fever, jaundice and ascites. The patient had a history of cirrhosis with oesophageal varices since 1994 and she was treated with ursodesoxycholic acid. Almost five months before the episode, the patient suffered from anorexia, weight loss and deterioration of liver function and she was treated with corticosteroids, ursodesoxycholic acid and chlortalidone. On admission, the physical examination revealed a fever of 37.5°C, abdominal tenderness and oedema in both lower extremities. Laboratory evaluation showed the following values: Ht 31%, haemoglobin 10,6 g/dl, WBC 11.900 cells/mm³ with left shift, SGOT 162 U/L, SGPT 142 U/L. Paracentesis of the abdomen yielded a cloudy peritoneal aspirate that contained 13.200 cells/mm³ with predominance of neutrophils (88%). The diagnosis of spontaneous bacterial peritonitis was established and the patient was treated empirically with intravenous administration of cefotaxime (8 g/day). Samples of peritoneal aspirate was inoculated into two blood culture bottles that were incubated aerobically and anaerobically at 37°C under continuous monitoring blood-culture system (Organon Teknika, Turnhout, Belgium). Gram-stained smears prepared from the sediment of centrifuged peritoneal fluid revealed no organisms. After 48h of incubation, a grampositive rod was recovered that was oxidase negative, catalase positive and motile. The isolate was identified by Vitek system (bioMerieux Vitek Inc, Hazelwood, Mo) and API Coryne (bioMerieux) as L. monocytogenes. Serotyping of isolate with specific antisera (Difco) showed that the isolate belonged to the serovar 4b. Two blood cultures, drawn concomitantly with peritoneal aspiration, yielded no growth.

Antimicrobial susceptibility testing by the E-test method (AB Biodisk, Solna, Sweden) showed that the isolate was susceptible to penicillin G, amoxicillin, amikacin, gentamicin, clindamycin, erythromycin, piperacillin/tazobactam, meropenem, ciprofloxacin and vancomycin, but it was resistant to cefuroxime, cefotaxime and ceftriaxone. On the basis of the culture results, treatment with cefotaxime was discontinued and replaced with piperacillin/tazobactam administrated intravenously (4.5g every 8h). Clinical improvement was rapidly noted and the fever subsided. However, the therapy was changed to ciprofloxacin five days later because of deterioration of hepatic function. A second specimen of peritoneal fluid, taken before the replacement of antibiotic, yielded the same organism. On day 8, a third specimen of peritoneal fluid was obtained that was sterile. However, the patient presented signs of worse progress of liver disease and died.

DISCUSSION

Listeria monocytogenes is a gram-positive, non-sporeforming short rod, motile, facultatively anaerobic. It is widely distributed in the environment, often isolated from soil, water and sewage, and in many foods, including dairy products, meat, vegetables and seafood. From clinical human specimens, the organisms is rarely isolated. Most infections occur in neonates and in adults with impaired cell-mediated immunity due to immunosusppressive therapy, malignancies or pregnancy.8 Other factors that have been reported to be associated with listeriosis include heart and renal disease, diabetes mellitus, hepatic cirrhosis and organ transplantation.8 L. monocytogenes causes a wide spectrum of clinical syndromes ranging from mild influenza-like illness to meningitis, meningoencephalitis and septicaemia. In pregnant women mainly causes spontaneous abortion, stillbirth or early-onset infection.8 Recently, scattered reports of spontaneous bacterial peritonitis due to L. monocytogenes have appeared in the literature.²⁻⁷ The number of such cases has significantly increased during the last years suggesting that this pathogen can cause infections more frequently in this group of patients than it was initially believed.

Listeria monocytogenes is susceptible to numerous

antimicrobial agents that include penicillins (except oxacillin), first –and second- generation cephalosporins, macrolides, aminoglycosides, tetracyclines, trimethoprim-sulfamethoxazole, carbapenems, rifampicin and chloramphenicol but it shows natural resistance to broadspectrum cephalosporins. As the bacterium may be confused with diphtheroids, the use of a third-generation cephalosporin as empirical therapy of this type of infection might be fatal.

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