

Spontaneous bacterial peritonitis with a very high leukocyte count in ascitic fluid caused by *Haemophilus influenzae*

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Abstract: We report on a case of spontaneous bacterial peritonitis (SBP) due to *Haemophilus influenzae* (*H. influenzae*) in an elderly patient with alcoholic cirrhosis. The patient presented with a 5 day history of fever, cough, and fatigue. Abdominal paracentesis revealed a very high neutrophil count (134,800 cells/ μ L). Secondary peritonitis and abdominal abscess were ruled out. Peritoneal fluid culture displayed the growth of *H. influenzae*. The patient was treated with ceftriaxone and showed signs of improvement. Eventually, the patient died due to septic shock caused by other organisms. *H. influenzae* is a very rare cause of SBP. This case report demonstrates that (1) *H. influenzae* should be considered a potential cause of SBP, and (2) a very high leukocyte count in ascitic fluid can be found in patients with SBP.

Keywords: cirrhosis, spontaneous bacterial peritonitis, *Haemophilus influenzae*

Introduction

Spontaneous bacterial peritonitis (SBP) is the most common life-threatening, infectious complication in patients with ascites caused by liver cirrhosis. The organisms most commonly involved in this infection are *Escherichia coli*, *Klebsiella pneumoniae*, and other Gram-negative enteric organisms.¹

Haemophilus influenzae (*H. influenzae*) is a very rare cause of SBP. Large series of cases of SBP do not mention it as a cause, but individual case reports have occasionally implicated this organism in SBP.²

In this case of SBP, the leukocyte count in ascitic fluid was very high. It has been suggested that a very high leukocyte count in ascitic fluid is characteristic of secondary bacterial peritonitis. Our case represents an exception.

Case presentation

A 64-year-old Jewish male of Indian origin was hospitalized in our department, with a 5-day history of fever (39°C), cough, and fatigue. He denied abdominal pain, vomiting, diarrhea, or other symptoms.

His medical history was significant for alcoholic cirrhosis with portal hypertension, splenomegaly, hypersplenism, and ascites. Furthermore, he had diabetes mellitus type 2 (hemoglobin A_{1c} of 7%) and hypertension for years.

His medications included propranolol, lactulose, Aldospirone, omeprazole, and metformin.

On physical examination the patient was alert without encephalopathy or jaundice. The following characteristics of chronic liver disease were present: spider angiomas on his chest, hepatomegaly, splenomegaly, ascites, caput medusa, and pedal edema.

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His abdomen was swollen with tenderness in the right upper quadrant.

Laboratory tests showed a total bilirubin level of 1.76 mg/dL (normal range, 0.21–1 mg/dL); alanine aminotransferase level of 20 U/L (normal values, <40 U/L); aspartate aminotransferase level of 24 U/L (normal values, <40 U/L); alkaline phosphatase level of 147 U/L (normal range, 30–150 U/L); γ -glutamyl transpeptidase level of 73 U/L (normal range, 5–60 U/L); albumin level of 1.3 g/dL; prothrombin time of 15 seconds (international normalized ratio, 1.4); creatinine 2.7 mg/dL. His Model for End-Stage Liver Disease score on admission was 22. Complete blood count showed leukocytosis, 14,000 cells/ μ L, with 85% neutrophils, hemoglobin 7.1 g/dL, and platelets 100,000 cells/ μ L.

Chest X-ray showed mild right pleural effusion with no pulmonary infiltrates, and his abdominal ultrasonograph showed moderate ascites and hepatosplenomegaly. Blood, urine, and pleural fluid cultures were negative.

Abdominal paracentesis was done and revealed 134,800 leukocytes/mm³ (100% neutrophils), a serum-ascites albumin gradient of 1.4; levels of lactate dehydrogenase, amylase, and triglycerides were not elevated, and the level of glucose was within normal limits. The unusually high peritoneal fluid leukocyte count raised the suspicion of secondary peritonitis.

The patient underwent computed tomography of the abdomen with oral contrast, and no evidence of perforation or abdominal abscess was found.

A peritoneal fluid culture was positive for *H. influenzae*, and the patient was treated with ceftriaxone. Abdominal paracentesis was repeated 48 hours after beginning antibiotic treatment and showed a significant decrease of leukocyte count to 3,000/mm³. After hospitalization for 2 weeks the patient improved significantly; his fever resolved, and the patient felt better and was discharged. Three days later, the patient readmitted with septic shock due to *Escherichia coli* and methicillin-resistant *Staphylococcus aureus*. Unfortunately, despite antibiotic therapy, the patient died.

Discussion

The epidemiology of SBP pathogens has recently changed; in particular, there has been an increase in infections due to Gram-positive cocci. Studies¹ have suggested that these changes are associated with the long-term hospitalization of patients with end-stage liver disease and the use of prophylactic antibiotics after an initial episode of SBP.

However, Gram-negative bacteria remain the most common pathogens associated with SBP.¹

H. influenzae as a cause of SBP is rare and is mentioned only in a few case reports. Large case series do not mention *H. influenzae* as a cause of SBP.²

Haemophilus species are common inhabitants of mucosal surfaces of the human upper respiratory tract. They can provoke respiratory tract infections, otitis, meningitis, and other life threatening disease. However, little is known about the ability of *Haemophilus* species to colonize other sites.³ *H. influenzae* has been cultured from feces, jejunal fluid, and the genital tract of normal persons as well as from 6% of appendices removed surgically from children with appendicitis.³

The proportion of *H. influenzae* strains versus *Haemophilus parainfluenzae* is reported to be the same in both fecal isolates and oropharynx isolates. This data strengthens the assumption that *Haemophilus* species found in the gastrointestinal tract originate from the oropharynx and saliva.³

Our patient presented with fever and cough; we think that the source of *H. influenzae* was the respiratory tract. Three routes of transmission from the respiratory tract to the ascitic fluid were suspected: (1) *H. influenzae* bacteremia, (2) infection of the pleural effusion (empyema), (3) gut colonization by *H. influenzae* which originated from the upper respiratory tract flora.

Our evaluation revealed negative blood cultures, and the pleural effusion was transudate and cultures were negative.

It is well known that a large proportion of cirrhotic patients have hypochlorhydria,⁴ which is considered a risk factor for colonization of the upper gut with mainly Gram-positive flora; especially viridans *Streptococci*, coagulase-negative *Staphylococci*, and *Haemophilus* species.⁵

We assume that the origin of *H. influenzae* in the ascitic fluid is intestinal due to gut colonization from the upper respiratory flora, specifically in the absence of a systemic infection.

In this case of SBP, the leukocyte count in ascitic fluid was very high. It has been suggested that a very high leukocyte count in ascitic fluid is characteristic of secondary bacterial peritonitis.⁶ Our case represents an exception.

The ascitic fluid neutrophil count is the best determinant for early diagnosis of SBP. An ascitic fluid neutrophil count >250/mm³ is consistent with the diagnosis of SBP. Ascitic fluid leukocyte and neutrophil counts in our patient were higher than those typically seen in SBP. In patients with ascites and cirrhosis, the presence in ascitic fluid of more than 5,000–10,000 leukocytes/mm³ may suggest that

the peritonitis is secondary. As mentioned above, secondary peritonitis was ruled out.⁷ Patients with SBP and a neutrophil count $>1,000/\text{mm}^3$ seem to have high mortality.⁸ This case report demonstrates that (1) *H. influenzae* should be considered a potential cause of SBP and (2) a very high leukocyte count in ascitic fluid can be found in patients with SBP.

Disclosure

The authors report no conflicts of interest in this work. The authors confirm no financial interest in a business or commercial entity that relates to the manuscript.

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