

Spontaneous bacterial peritonitis from *Salmonella*: an unusual bacterium with unusual presentation

Harshal Rajekar · Chun-Tao Wai · Kang-Hoe Lee ·
Sin-Yew Wong · Kai-Chah Tan

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Abstract Spontaneous bacterial peritonitis (SBP) is a common cause of morbidity and mortality in patients with advanced cirrhosis and portal hypertension. While gram-negative rods and *Enterococcus* species are the common offending organisms, *Salmonella* has also been recognized as a rare and atypical offending organism. Atypical features of *Salmonella* SBP include both its occurrence in cirrhotic patients with immunosuppressive state and its lack of typical neutroascitic response. Diagnosis is often delayed as it requires confirmation from ascitic fluid culture. We report a case of *Salmonella* SBP occurring in a patient with decompensated cryptogenic cirrhosis with concurrent low-grade non-Hodgkin lymphoma and prior treatment with rituximab. Physicians should be aware of the atypical presentation, especially in cirrhotic patients who are immunosuppressed.

Keywords Spontaneous bacterial peritonitis ·
Rituximab · Live donor liver transplant · Cirrhosis

Introduction

Spontaneous bacterial peritonitis (SBP) due to *Salmonella* is uncommon in cirrhotic patients with ascites, and it is often associated with an immunosuppressed state and often presents without typical signs or symptoms of SBP [1, 2]. We report a case of SBP due to group B *Salmonella* in a patient with decompensated cryptogenic cirrhosis and portal hypertension.

Case

A 61-year-old female Arab patient was referred to our center for management of decompensated cryptogenic liver cirrhosis. She has type II diabetes mellitus since adolescence and hypertension of 5 years' duration. She was also diagnosed with B-cell follicular non-Hodgkin lymphoma (NHL), stage IIIA, involving gastric mucosa and abdominal lymph nodes, 8 months ago. She was treated with rituximab (anti-CD20 monoclonal antibodies) and dexamethasone. However, she developed grade IV hepatic encephalopathy after three cycles of rituximab and chemotherapy was stopped.

Since the onset of encephalopathy, she started complaining of worsening ascites and lethargy, and had two more episodes of grade IV hepatic encephalopathy. By February 2008, she was referred to our center for evaluation for live donor liver transplantation (LDLT). She had no history of variceal bleeding or SBP and was not on prophylactic antibiotics. Initial physical examination showed moderate ascites and bilateral ankle edema, with normal chest and heart examinations. Blood tests showed normal creatinine and electrolytes, bilirubin 82 μ M, albumin 26 g/l, alanine transaminase 22 U/l, aspartate transaminase 43 U/l, and alkaline phosphatase 73 U/l. Complete blood cell count showed slightly low hemoglobin at 11.3 g/dl, low platelets at 65,000/mm³, and normal white cells at 5,050/mm³. INR was elevated at 1.31. Computed tomographic (CT) scan revealed cirrhotic liver, splenomegaly, moderate ascites, and small, subcentimeter retroperitoneal lymphadenopathy. No gallstones were seen on CT or ultrasound scan. Positron emission tomography CT scan did not reveal any fluorodeoxyglucose-avid lesions to suggest active lymphoma, and ascitic fluid cytology was negative for lymphoma cells.

H. Rajekar · C.-T. Wai (✉) · K.-H. Lee · S.-Y. Wong ·
K.-C. Tan

Asian Center for Liver Diseases and Transplantation, Gleneagles
Hospital Annexe Block #02-37, 6A Napier Road,
Singapore, Singapore 258500
e-mail: dr_desmond_wai@yahoo.com.sg

While she was undergoing evaluation for LDLT, she complained of acute onset of generalized abdominal pain. There was no fever, diarrhea, vomiting, or urinary symptoms. Clinically, she was afebrile and did not have tachycardia, but there was right-sided abdominal tenderness. A diagnosis of SBP was suspected and the patient was admitted for diagnostic paracentesis and empiric antibiotics therapy. Urinalysis and urine culture were unremarkable. Initial ascitic fluid analysis results showed a leukocyte count of $110/\text{mm}^3$, majority being lymphocytes, with lactate dehydrogenase, protein, and albumin at 54 U/l, 11 g/l, and 5 g/l, respectively. Her serum ascitic albumin gradient was 21 g/l, consistent with significant portal hypertension. However, 2 days later, ascitic fluid culture showed presence of group B *Salmonella*, which was sensitive to both ceftriaxone and ciprofloxacin. She was treated with an intravenous dose of ceftriaxone for a week followed by an oral dose of ciprofloxacin. Intravenous albumin infusion was also given concurrently to prevent renal impairment. Subsequent blood and stool cultures did not show growth of any organisms. She recovered well from the infection, with resolution of abdominal pain and tenderness; she underwent LDLT 2 weeks after diagnosis of *Salmonella* SBP. During laparotomy, no intra-abdominal lymphadenopathy was noted and explant liver did not show any evidence of active lymphoma. She has been recovering well from the transplant and remained well at her last follow-up 6 weeks posttransplant.

Discussion

SBP is a common complication of patients with decompensated cirrhosis, occurring in 10% to 30% of cirrhotic patients with ascites, with a mortality of up to 30%. Standard investigation includes ascitic fluid analysis for cell count analysis, biochemical analysis, and cultures. Third generation cephalosporins with intravenous albumin infusion are the standard treatment. Definitive diagnosis does not require a positive culture because an ascitic fluid neutrophil count of more than $250/\text{mm}^3$ is sufficient for diagnosis, as culture results often take a minimum of 48 h to be available [3, 4].

Common offending organisms in SBP are *Klebsiella pneumoniae*, *Escherichia coli*, and *Enterococcus* species [5, 6]. Non-typhoidal *Salmonella* is a rare cause of SBP. Previous case series reported various immune-compromised states, such as AIDS and non-hepatic cancer, as risk factors for *Salmonella* SBP [1, 2]. In our patient, her immunosuppressive state could be related to her prior administration of rituximab, an anti-CD20 monoclonal antibody. Depletion of peripheral B lymphocytes,

important for humoral response to bacterial antigens, has been shown to last for up to 2 years after its administration [7, 8]. In addition, her underlying low-grade NHL and diabetes mellitus may have also contributed to her immunosuppressive state.

Salmonella SBP is an unusual form of SBP as it has also been reported to be asymptomatic, with no overt systemic signs of infection. There may be little biochemical evidence, or neutroascitic response in ascitic fluid analysis, and the diagnosis often requires confirmation on ascitic fluid culture [3]. As seen in our patient, her only symptom was abdominal pain and initial ascitic fluid analysis did not reveal any neutroascitic reaction or elevation of lactate dehydrogenase and confirmation of SBP was made only 48 h later when ascitic culture results returned.

In summary, we reported a case of a cirrhotic patient who was immunosuppressed from follicular NHL and prior use of rituximab, presenting atypically for SBP that was subsequently diagnosed as *Salmonella* SBP. Physicians should be aware of this atypical presentation and routine ascitic fluid culture should be performed in patients suspected of having SBP, particularly in cirrhotic patients with an immunosuppressive state.

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