CASE REPORTS

Spontaneous Bacterial Peritonitis Caused by S. paratyphi A

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Abstract
Spontaneous (primary) bacterial peritonitis (SBP) due to S. paratyphi A is relatively uncommon. Clinical manifestations of SBP vary widely from severe to slight or absent, necessitating laboratory investigation of ascitic fluid. The disease is confirmed by number of neutrophils >250/mm³ associated with or without bacterial growth in ascitic fluid culture from diagnostic abdominal paracentesis. Here, we present a case of S. paratyphi A SBP occurring in a patient with chronic liver disease and portal hypertension. The patient was treated with intravenous cefotaxime with good clinical response.

Introduction
Spontaneous (primary) bacterial peritonitis (SBP) is characterised by spontaneous infection of ascitic fluid without any intraabdominal, surgically treatable source and is a frequent and severe complication of ascites. In adults, it occurs frequently in association with cirrhosis of liver. The disease is also caused by metastatic malignant disease, postnecrotic cirrhosis, chronic active hepatitis, acute viral hepatitis, congestive heart failure, systemic lupus erythematosus, lymphoedema and in patients with no underlying disease. SBP can occur in up to 30% of hospitalised cirrhotic patients with ascites and can have a 25% mortality rate. We report a case of SBP in which S. paratyphi A was isolated from ascitic fluid.

Case Report
A 53-year-old man was admitted in our hospital for fever, yellowish discoulouration of skin and swelling of legs for 10-15 days. He was a known case of chronic liver disease (CLD) with distension of abdomen for last 10 years which was gradually worsening. He was diagnosed to have CLD with portal hypertension. There was no past history of enteric fever.

Initial physical examination revealed a temperature of 101°F, blood pressure 110/80 mm Hg, pulse 80/min, icterus, and bilateral pedal oedema with normal chest and heart examinations. There was symmetrical abdominal distension with mild diffuse tenderness on palpation with normal bowel sounds. Blood tests showed: WBC 11,310/mm³, haemoglobin 8 g/dl, platelets 150,000/mm³, bilirubin-total 5.0 mg/dl (by modified Jendrassik Grof method), elevated liver enzymes, decreased serum albumin (2.0 g/dl) with normal creatinine and blood electrolytes. A diagnostic paracentesis was done which showed a leucocyte count of 750/mm³, majority being neutrophils, with protein and albumin at 2.8 g/dl and 0.2 g/l respectively. His serum-ascitic albumin gradient was 1.8 g/dl, consistent with significant portal hypertension. Ultrasound of the liver showed features consistent with CLD, splenomegaly, moderate ascites with no identifiable focal lesion.

Considering the high leucocyte count and the clinical condition of the patient, SBP was suspected and intravenous (IV) cefotaxime (CTX) was empirically started. However,
2 days later, ascitic fluid culture showed growth of \textit{S. paratyphi A} which was sensitive to ampicillin, chloramphenicol, cotrimoxazole, ciprofloxacin, CTX, azithromycin and resistant to nalidixic acid. Oesophageal variceal ligation was done and he was treated with IV fluids, IV albumin, diuretics, abdominal paracentesis and IV CTX. A favourable clinical outcome was noted and he was discharged on day 5 on oral ciprofloxacin and azithromycin.

**Discussion**

SBP is a common complication in patients with a severe CLD. The pathogenesis of SBP is the bacterial translocation from the gut into mesenteric lymph nodes, leading to bacteraemia and seeding of the ascitic fluid.\(^1\) This is facilitated by a diseased liver and altered portal circulation resulting in a defect in the normal filtration process.\(^3\) As there was no history of enteric fever in our patient, the cause of \textit{S. paratyphi A} as the aetiological agent of SBP remained unexplained. Blood culture was not done for this patient. Previous case series reported SBP due to \textit{Salmonella enteriditis} from three patients with advanced CLD, portal hypertension and no clinical and microbiological evidence of \textit{Salmonella} infection other than in the ascitic fluid.\(^4\)

Common offending organisms in SBP are \textit{Escherichia coli} and other gut flora. However, gram positive bacteria e.g., \textit{Streptococcus viridans}, \textit{Staphylococcus aureus}, and \textit{Enterococcus sp.}, are also found.\(^1\) \textit{Salmonella paratyphi A} is a rare cause of SBP. There has been a case report of \textit{Salmonella sp.} group A from bacterascitis-a variant of SBP.\(^5\) A single organism is typically isolated in SBP as in our case. When more than two organisms are isolated from ascitic fluid, secondary bacterial peritonitis due to perforated viscus should be considered.\(^1\)

This case falls in the “classical SBP” variant having typical symptoms suggestive of peritoneal infection such as fever, abdominal pain, a high PMN cells count with a positive ascitic fluid culture by a single organism. In our case, the diagnostic paracentesis and the prompt initiation of empiric antibiotic based on ascitic PMN count has contributed to the early clinical improvement and survival of the patient.

The first-choice in the treatment of SBP in cirrhotic patients is CTX for 5 days in the dose of 4-8 g/day.\(^6\) Empirical coverage for anaerobes is not required.\(^3\) Our patient was treated with IV CTX and stepped down to oral ciprofloxacin and azithromycin at the time of discharge with good clinical response. On further follow-up at 6 weeks patient has been doing well.

**Conclusion**

Although SBP due to \textit{S. paratyphi A} is rare, the outcome is favourable when the condition is diagnosed early and treated adequately with broad spectrum antibiotics. Physicians should be alert of this uncommon causative organism in SBP. However, over the years with advances in medical science, SBP is less life-threatening, with improvement in diagnosis, prophylaxis and therapy.

**References**