Spontaneous bacterial peritonitis by *Clostridium* species and antimicrobial therapy

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Spontaneous bacterial peritonitis (SBP) is common in decompensated cirrhosis. Its incidence is 10–30% in patients with ascites and chronic liver disease. Mortality is almost 25% in patients with SBP [1, 2]. We are presenting a very rare case of SBP secondary to clostridium species.

A 65-year-old man known to have chronic hepatitis C (treatment naive), poly-substance abuse, Child-Pugh C cirrhosis, and gastroesophageal reflux disease (on chronic acid suppression) was admitted due to fever and generalised abdominal pain for 3 days. He had a history of paracentesis three times in the past. He was on ciprofloxacin 500 mg twice daily maintenance because of previous SBP (culture negative neurocytic ascites) for 1 month. Vitals were normal except fever of 101 F. On general physical examination, the patient was cachectic, lethargic, had scleral icterus, gynaecomastia, and spider angiomata on the chest; the abdomen was distended and tender, with dilated veins radiating from the umbilicus. Pulmonary, cardiovascular, and neurological examinations were unremarkable.

Labs showed white blood cells (WBCs) of 12.6 × 10^9 cells/l, total bilirubin 4.6 mg/dl, international normalised ratio (INR) was 1.67, and serum albumin was 26 g/l. Paracentesis was done and 5.6 l fluid was removed followed by administration of 37.5 g of albumin in three divided doses, fluid analysis showed polymorphonuclear count (PMN) of 12,996 cells/mm^3, ascitic fluid albumin of 12 g/l, and glucose of 58 mg/dl (3.2 mmol/l). Ultrasound showed large abdominal ascites, the liver was small, and there was some hepatofugal flow in the main portal vein (Figure 1). Computed tomography (CT) scan of the abdomen revealed massive abdominal and pelvic ascites with a shrunken liver with a nodular undulating contour consistent with liver cirrhosis, splenomegaly was appreciated, and no bowel pathology was noticed (Figure 2).

Intravenous cefotaxime 2 g every 8 h was started empirically, and the ascitic fluid culture was positive for clostridium species (unspecified microbiologically) in two bottles. Blood cultures were negative for bacteraemia. Stool assay was negative for *Clostridium difficile*. Intravenous metronidazole 500 mg every 8 h was added. The patient improved clinically on the third day after addition of metronidazole. Repeat paracentesis was done on the fifth day and 1.1 l of ascitic fluid was drained. Fluid analysis revealed a significant decrease in PMN count to 190 cells/mm^3 and repeat ascetic fluid cultures were negative. The patient clinically improved and was discharged on the seventh day on oral metronidazole 500 mg three times a day for 7 more days. Maintenance ciprofloxacin was continued for SBP prophylaxis.

Ascitic fluid with polymorphonuclear cells (PMN cells) more than 250 cells/mm^3 is called SBT [3]. Common microorganisms causing SBP are Gram-negative bacteria including *Escherichia coli*, *Klebsiella* species, and *Enterobacter* species. Gram-positive organisms are involved in 20% of cases and anaerobes in 3% of cases. Risk factors for Gram-positive and multidrug-resistant bacteria are recent hospitalisation, invasive procedures, and quinolone prophylaxis. The mechanism includes immune dysfunction due to portosystemic shunting, decreased phagocytic activity, and genetic polymorphism of toll-like receptor and nucleotide-binding oligomerisation domain 2 (NOD2) leading to bacterial translocation (BT).

Extraintestinal migration of bacteria or bacterial product can lead to SBP [4]. Ours is a very rare case of SBP due to clostridium species. Anaerobes rarely cause

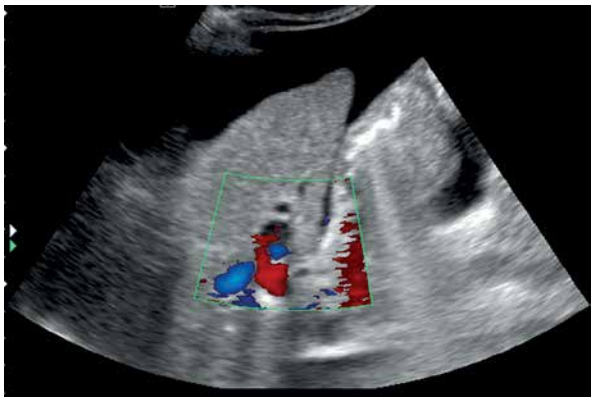

Figure 1. Ultrasound abdomen shows small liver and hepatofugal flow in the portal vein

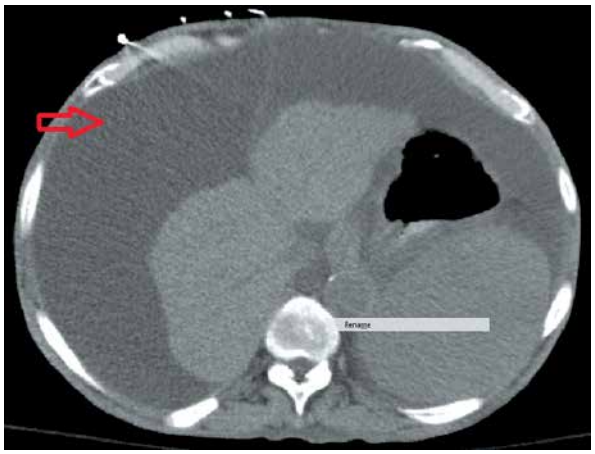

Figure 2. Computed tomography abdomen axial view showing large ascites (red arrow) and shrunken liver

SBP although they are predominant in the small intestine and colon, probably because of high oxygen tension of the ascetic fluid and relative inability to cross intestinal mucosa. Achlorhydria and decreased intestinal motility cause small intestinal bacteria overgrowth, which can cause SBP by bacterial translocation [5].

Spontaneous bacterial peritonitis usually presents with fever and abdominal pain. Diagnostic paracentesis should be performed earlyto avoid delayed diagnosis [6]. If the PMN cell count is greater than 250 cells/mm^3, empiric antibiotics should be started. Commonly used antibiotics are third-generation cephalosporins, amoxicillin-clavulanate, or quinolones.

In healthcare-associated SBP significant failure is noticed with the above antibiotics. A repeat diagnostic paracentesis should be done within 48 h of the start of treatment if clinical improvement is not appreciated [4].

Spontaneous bacterial peritonitis is associated with poor prognosis. Early paracentesis with fluid culture is recommended for a better outcome. In the absence of indication, excessive use of acid suppression in cirrhotic patients should be avoided.

Conflict of interest

The authors declare no conflict of interest.

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