

# Spontaneous bacterial peritonitis in extrahepatic portal venous obstruction

George Sarin Zacharia, Kavitha Rangan, Sunilkumar Kandiyil, Varghese Thomas

Calicut Medical College, India

## Abstract

Spontaneous bacterial peritonitis is defined by a positive ascitic fluid bacterial culture and an elevated ascitic fluid absolute polymorphonuclear count ( $\geq 250$  cells/mm<sup>3</sup>) without an evident intra-abdominal, surgically treatable source of infection. Transient ascites is well documented in patients with extrahepatic portal venous obstruction but spontaneous bacterial peritonitis complicating extrahepatic portal venous obstruction is extremely uncommon. The postulated reasons for the low incidence of spontaneous bacterial peritonitis in extrahepatic portal venous obstruction includes: lower incidence of ascites; intact hepatic reticuloendothelial system; and a relatively high ascitic fluid protein content. Here we report two cases of spontaneous bacterial peritonitis complicating extrahepatic portal venous obstruction.

**Keywords** Extrahepatic portal venous obstruction, spontaneous bacterial peritonitis, ascites

*Ann Gastroenterol* 2013; 26 (4): 363-364

## Introduction

Spontaneous bacterial peritonitis (SBP) is defined by a positive ascitic fluid bacterial culture and an elevated ascitic fluid absolute polymorphonuclear count ( $\geq 250$  cells/mm<sup>3</sup>) without an evident intra-abdominal, surgically treatable source of infection [1]. SBP usually occurs in a background of cirrhosis and it rarely affects patients without cirrhosis. Cases of SBP have been reported in patients with nephrotic syndrome, heart failure, systemic lupus erythematosus and Budd-Chiari syndrome without liver cirrhosis [2-5]. SBP in extrahepatic portal venous obstruction (EHPVO) is extremely rare with only a single case reported to date [6].

## Case presentations

### Case 1

A 48-year-old male presented with melena of 2 days duration. He was diagnosed to have idiopathic EHPVO at the age of 14 years and had undergone multiple endotherapies and later splenectomy at the age of 15 years. At admission the vitals were stable and physical examination was normal except for

Department of Medical Gastroenterology, Calicut Medical College, India

Conflict of Interest: None

Correspondence to: Dr Thomas Varghese, Professor and Head of the Department, Calicut Medical College, 673008 India, Tel.: +098 460 86770, e-mail: drvarghesethomas@gmail.com

Received 21 February 2013; accepted 9 April 2013

pallor and the presence of abdominal scar. On the fourth day of admission the patient developed fever with abdominal pain and mild abdominal distension due to ascites. Hemogram revealed; hemoglobin 6.1 g/dL, total leukocyte count 6500 cells/mm<sup>3</sup>, platelet count 3.56 lakhs/mm<sup>3</sup>, mean corpuscular volume 65.2 fL and International Normalized Ratio (INR) 1.04. Doppler ultrasound abdomen revealed normal liver, multiple tortuous collaterals replacing portal vein and moderate ascites. Upper gastrointestinal endoscopy revealed mucosal tags in the esophagus (post endotherapy), single column of grade 1 esophageal varix and severe portal hypertensive gastropathy. Ascitic fluid analysis revealed a total leukocyte count of 2400 cells/mm<sup>3</sup>, polymorphonuclear cell count of 1680 cells/mm<sup>3</sup>, with ascitic fluid protein 1.9 g/dL, albumin 0.8 g/dL and serum ascites albumin gradient (SAAG) 2.3. Culture of ascitic fluid yielded heavy growth of *Escherichia coli* (*E. coli*) species.

### Case 2

A 58-year-old male presented with melena, abdominal pain and fever. He was diagnosed to have EHPVO and had undergone splenectomy at the age of 19 years when he presented with massive variceal bleeding in 1973. Physical examination revealed pallor and moderate ascites. Laboratory investigations were as follows; hemoglobin 9.1 gm/dL, total leukocyte count 11700 cells/mm<sup>3</sup>, platelet count 4.04 lakhs/mm<sup>3</sup> and INR of 1.1. Ascitic fluid analysis showed a total leukocyte count of 1600 cells/mm<sup>3</sup>, polymorphonuclear cell count of 1472 cells/mm<sup>3</sup>, total protein 2.5 g/dL, albumin 1.4 g/dL and SAAG 1.9. Ascitic fluid culture yielded growth of *E. coli*. Ultrasound abdomen with Doppler study showed normal liver, cavernous transformation of the portal vein

and moderate ascites. Upper gastrointestinal endoscopy revealed severe portal hypertensive gastropathy and gastric fundal varix. Computerized tomography of the abdomen was consistent with the diagnosis of EHPVO; there was no evidence of cirrhosis of the liver or any features of perforation or inflammation of other organs.

The liver and renal function tests, serum amylase and lipase levels, serum electrolytes and blood glucose levels were normal in both patients. Ascitic fluid glucose, lactate dehydrogenase, triglycerides and adenosine deaminase were also within normal limits. Both patients did not show any features of perforation or obstruction on plain x-ray of the abdomen.

Both patients were managed with infusion of octreotide, proton pump inhibitors, transfusions, hematinics and beta blockers. SBP was treated with Cefotaxime 2 g IV t.i.d. Both patients had relief of fever, abdominal pain and abdominal distension. Repeat ascitic fluid analysis after 48 h of antibiotic therapy showed a decrease in polymorphonuclear cell count to 68 cells/mm<sup>3</sup> and 165 cells/mm<sup>3</sup> in the first and second case respectively. Antibiotics were continued for 5 days and both patients were discharged in a stable state.

## Discussion

Spontaneous bacterial peritonitis was first recognized by Harold Conn in the 1964. SBP is the infection of ascitic fluid that occurs in the absence of visceral perforation or intra-abdominal inflammatory focus. Over 60% of the SBP episodes are caused by Gram-negative enteric bacilli like *E. coli* and *Klebsiella pneumoniae*. The key pathogenic mechanism initiating SBP is bacterial translocation, a process through which enteric bacteria cross the intestinal barrier and infect the mesenteric lymph nodes, thus entering the blood circulation and ascitic fluid. The high rate of bacterial translocation in cirrhosis is due to intestinal bacterial overgrowth, loss of integrity of intestinal mucosal barrier and local immune system [7]. The intestinal bacterial overgrowth in patients with cirrhosis plays a key role and is mainly attributed to delayed intestinal transit time. In healthy people, the Kupffer cells collaborate with neutrophils in the process of bacterial extraction from the circulation. In patients with hepatic cirrhosis, because of intra and extrahepatic shunts, the bacteria bypass the Kupffer cells, with resultant bacteremia and ascitic fluid inoculation [8]. Low serum and ascites complement levels also predispose to bacteremia and eventual bacterial proliferation within ascitic fluid [8].

Transient ascites is reported in about 13% of patients with EHPVO [9]. SBP complicating prehepatic portal hypertension is extremely rare and to date there is only one published case report [6]. The postulated reasons for the low incidence of SBP includes the low occurrence of ascites, intact hepatic reticuloendothelial system and a relatively high ascitic fluid protein content in patients with EHPVO [6].

Clinical manifestations of SBP are often nonspecific. Approximately 10% of the patients with SBP are asymptomatic.

The most frequently encountered symptoms and signs are fever, abdominal pain, signs of hepatic encephalopathy, abdominal tenderness, diarrhea, ileus and shock [1]. SBP is diagnosed when the ascitic polymorphonuclear leucocytes exceed 250 cells/mm<sup>3</sup> and bacteriological cultures isolate only one germ [10]. Empirical antibiotic therapy must be initiated immediately after diagnosis of SBP, without waiting for the results of ascitic fluid culture [10]. Cefotaxime is currently the drug of choice as it covers most causative organisms and because of its high ascitic fluid concentrations during therapy [10]. The recommended dose is 2 g IV t.i.d. for 5 days. Infection resolution is obtained in 77-98% of patients. If ascitic fluid neutrophil count fails to decrease to less than 25% of pre-treatment value after 2 days of antibiotic treatment, there is likelihood of failure to respond to therapy and this should raise the suspicion of infection caused by antibiotic resistant bacteria [10].

Ascitic fluid analyses in our patients were consistent with the diagnosis of SBP. There were no features to suggest visceral perforation or intra-abdominal focus of sepsis. The prompt response to antibiotic therapy and an uneventful recovery without any surgical interventions also favors the diagnosis of SBP in our patients. The reasons for development of SBP in our patients with EHPVO might be multifactorial such as ascites induced by gastrointestinal bleeding, the postsplenectomy status and the low ascitic fluid protein level.

In conclusion, SBP may rarely develop in patients with EHPVO with ascites.

## References

1. Hoefs JC, Canawati HN, Sapico FL, Hopkins RR, Weiner J, Montgomerie JZ. Spontaneous bacterial peritonitis. *Hepatology* 1982;**2**:399-407.
2. Chuang TF, Kao SC, Tsai CJ, Lee CC, Chen KS. Spontaneous bacterial peritonitis as the presenting feature in an adult with nephrotic syndrome. *Nephrol Dial Transplant* 1999;**14**:181-182.
3. Rajatanavin R, Pombegara CN, Pektanan P, et al. Spontaneous bacterial peritonitis in systemic lupus erythematosus: a case report. *J Med Assoc Thai* 1981;**64**:406-412.
4. Gómez del Olmo V, Patier de la Peña JL, Moreno-Cobo MA, Meseguer M. Spontaneous bacterial peritonitis caused by *Listeria monocytogenes* in ascites of cardiac origin. *Rev Clin Esp* 2010;**210**:421-422.
5. Barrio J, Castiella A, Gil I, García-Bengoechea M, Arenas JJ. Spontaneous bacterial peritonitis by *Campylobacter fetus* in Budd-Chiari syndrome without liver cirrhosis. *Liver* 1999;**19**:69-70.
6. Gupta N, Sahni V, Singh P, Das A, Kar P. Spontaneous bacterial peritonitis in isolated splenic vein thrombosis with portal hypertension. *Indian J Gastroenterol* 2006;**25**:263-264.
7. González Alonso R, González García M, Albillos Martínez A. Physiopathology of bacterial translocation and spontaneous bacterial peritonitis in cirrhosis. *Gastroenterol Hepatol* 2007;**30**:78-84.
8. Wiest R, Garcia-Tsao G. Bacterial translocation in cirrhosis. *Hepatology* 2005;**41**:422-433.
9. Sarin SK, Aggarwal SR. Idiopathic portal hypertension. *Digestion* 1998;**59**:420-423.
10. Rimola A, Gracia-Tsao G, Navasa M, et al. Diagnosis, treatment and prophylaxis of spontaneous bacterial peritonitis: a consensus document. International Ascites Club. *J Hepatol* 2000;**32**:142-153.