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LETTERS TO THE EDITOR
EDİTÖRE MEKTUP
Neutrocytic ascites in noncirrhotic portal hypertension

Nonsirotik portal hipertansiyonda nötrositik asit

To the Editor

Spontaneous bacterial peritonitis (SBP) is a life-threatening complication of decompensated liver cirrhosis. Bacterial translocation to mesenteric lymph nodes is an important initial step in the pathogenesis of SBP. This process is facilitated by impaired intestinal motility, small bowel bacterial overgrowth and impaired function of the intestinal immune defense (1, 2). The importance of portal hypertension per se as related to small intestinal motility and bacterial overgrowth of the upper gut is poorly understood. Gastrointestinal bleeding facilitates intestinal bacterial overgrowth and is thereby associated with bacteriemia and SBP (3, 4). SBP was not previously reported in noncirrhotic portal hypertensive patients. Here, we report the first case of a patient with noncirrhotic portal hypertension and neutrocytic ascites which developed after gastrointestinal bleeding.

A 65-year-old man, was admitted to our emergency service with bright red vomiting and fatigue. On physical examination, splenomegaly and ascites were found. Gastroscopic examination revealed grade 3 esophageal varices. He was treated with somatostatin infusion and variceal band ligation. Laboratory analyses were as follows: erythrocyte sedimentation rate 46 mm/h, leukocyte 4700/mm^3, hematocrit 30%, hemoglobin 9 g/dl, platelets 90000/mm^3, prothrombin time 13 (control 12) sec, aspartate aminotransferase 45 IU/L, alanine aminotransferase 34 IU/L, alkaline phosphatase 183 IU/L, gamma glutamyl transpeptidase 46 IU/L, total bilirubin 0.84 mg/dl, direct bilirubin 0.41 mg/dl, albumin 3.9 g/dl, and gamma globulin 1.4 g/dl. Hepatitis B surface antigen and antibody to hepatitis C virus were also negative. Ascitic fluid analysis was concordant with portal hypertensive type [glucose 73 mg/dl, LDH 115 IU/L, total protein 1.2 g/dl, albumin 0.7 mg/dl, serum ascites albumin gradient 2.3 g/dl, leukocyte 1200/mm^3, granulocyte 790/mm^3]. Intravenous ceftriaxone 2 g/day was started with presumed diagnosis of SBP. In spite of a negative ascitic fluid culture, normalization of leukocyte count in the third day of therapy with clinical improvement confirmed SBP diagnosis. Probable culture negativity may have been due to inappropriate ascites sampling under emergency conditions. Liver biopsy showed portal fibrosis. In laparoscopic examination the liver was assessed as noncirrhotic. Computerized tomography with portal angiography revealed thrombosis in portal vein and splenic vein, partial thrombosis in superior mesenteric vein and left splenorenal shunt. He was diagnosed with noncirrhotic portal hypertension and neutrocytic ascites with these findings.

This is the first case presented with noncirrhotic portal hypertension and probable SBP. Portal hypertension, by producing congestion and edema of the bowel wall, could increase the passage of bacteria from the intestinal lumen to regional lymph nodes, to the systemic circulation, or to both (5). Portal hypertension is also associated with altered intestinal motor and mucosal function. In acute thrombotic events, a minimal ascites may occur which improves within several days. The radiologic images of the thrombi seemed to be chronic, and the presence of a spontaneous shunt suggested that this clinical condition was a chronic event rather than an acute process. This case showed that neutrocytic ascites associated with

Address for correspondence: Filiz AKYÜZ
Department of Gastroenterohepatology, İstanbul University, İstanbul Medical Faculty, 34590, İstanbul, Turkey
Phone: +90 212 414 20 00/32671 • Fax: +90 212 631 97 43
E-mail: filizakyuz@hotmail.com

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noncirrhotic portal hypertension is probably infectious in origin. Gastrointestinal bleeding may also facilitate SBP even in the absence of hypoalbuminemia, which has an important role in opsonization. Ascites should be analyzed in all patients with cirrhotic and noncirrhotic portal hypertension.

REFERENCES

1. Llovet JM, Bartoli R, Planas R, et al. Bacterial translocation in cirrhotic rats. Its role in the development of spontaneous bacterial peritonitis. Gut 1994; 35: 1648-52.
2. Runyon BA, Squier S, Borzio M. Translocation of gut bacteria in rats with cirrhosis to mesenteric lymph nodes partially explains the pathogenesis of spontaneous bacterial peritonitis. J Hepatol 1994; 21: 792-6.
3. Soriano G, Guarner C, Tomas A, et al. Norfloxacin prevents bacterial infection in cirrhotics with gastrointestinal hemorrhage. Gastroenterology 1992; 103: 1267-72.
4. Zeuzem S. Gut liver axis. Int J Colorectal Dis 2000; 15: 59-82.
5. Hashimoto N, Ohyanagi H. Effect of acute portal hypertension on gut mucosa. Hepatogastroenterology 2002; 49: 1567-70.

Filiz AKYÜZ, Duygu İBRİŞİM, Fatih TUFAN, Fatih BEŞIŞIK, Atilla ÖKTEN

Department of Gastroenterohepatology, İstanbul University, İstanbul Medical Faculty, İstanbul