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Spontaneous bacterial peritonitis due to Brucella Melitensis in a cirrhotic patient

Sirotik bir hastada Brucella Melitensise bağlı spontan bakteriyel peritonit

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Spontaneous bacterial peritonitis is a well-known entity, with a reported incidence of 15-20% in advanced cirrhotic patients. Escherichia coli and Klebsiella pneumoniae are the most common causes of spontaneous bacterial peritonitis; Brucella is extremely rare. We aimed to present one case of such a rare condition in a cirrhotic patient who also had hepatocellular carcinoma. Routine laboratory tests, abdominal ultrasonography and peritoneal fluid examinations were studied in a cirrhotic patient with ascites. Peritoneal fluid white blood cell count was 1300/mm³, with lymphocyte predominance (80%). Peritoneal fluid and blood culture both yielded Brucella melitensis. The patient also had a mass in the right lobe of the liver confirmed as hepatocellular carcinoma by biopsy. Brucella should be suspected as a cause of spontaneous bacterial peritonitis in cirrhotic patients with no response to standard spontaneous bacterial peritonitis treatments and with immunodeficiency such as hepatocellular carcinoma.

Key words: Peritonitis, Brucella, cirrhosis

Spontan bakteriyel peritonit ileri sirotik hastalarda %15-20 oranında rapor edilmiş iyi bilinen bir durumdur. Escherichia coli ve Klebsiella pneumoniae spontan bakteriyel peritonitin en sık nedenleridir, Brucella oldukça nadirdir. Hepatoselüler kanseri de olan sirotik bir hastada böyle nadir bir durumu sunmayı amaçladık. Asitli bir sirotik hastanın rutin laboratuar testleri, abdominal ultrasonografisi ve periton sıvı incelemeleri yapıldı. Periton sıvısında beyaz küre 1300/mm³ idi ve lenfosit hakimiyeti vardı (%80). Periton sıvı ve kan kültüründe Brucella Melitensis üredi. Ayrıca, hastanın karaciğer sağ lobunda biyopsi ile hepatoselüler kanser olduğu doğrulanan bir kitle vardı. Standart tedavilere yanıt vermeyen ve hepatoselüler kanser gibi immün yetmezlik durumu olan spontan bakteriyel peritonitli siroz hastalarında Brucella spontan bakteriyel peritonit nedeni olarak akla gelmelidir.

Anahtar kelimeler: Peritonit, Brucella, siroz

INTRODUCTION

Spontaneous bacterial peritonitis (SBP) is particularly frequent if the cirrhosis is severely decompensated. İnfection is blood-borne, and in 90% monomicrobial; 60-80% are due to aerobic Gram-negative bacteria (1). Escherichia coli and Klebsiella pneumoniae are the most common causes of SBP. It can also be caused by other rare organisms such as Yersinia enterocolitica, Listeria monocytogenes, Pasteurella and Brucella (2-6). Brucella is a disease of domestic and wild animals (zoonosis) that is transmittable to humans. The disease exists worldwide, especially in the Mediterranean basin, the Arabian peninsula, the Indian subcontinent and in parts of Mexico and Central and South America (7). Brucellosis can affect many body sites. In one study, osteoarticular involvement was found in 34 patients (28.3%). Fifteen (12.5%) patients had ocular involvement. Hepatitis, orchiepididymitis, pulmonary involvement and meningitis were found in one (0.8%), four (6.8%), three (2.5%) and one (0.8%) patients, respectively, in brucellosis (8). Seeding of Brucella to the peritoneum is a very rare occurrence (1-4). Herein we present a cirrhotic patient who also had hepatocellular carcinoma with spontaneous bacterial peritonitis due to Brucella.

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CASE REPORT

A 65-year-old man with cirrhosis due to hepatitis B virus infection for three years admitted to the hospital with fatigue, arthralgia, abdominal pain and distention of four weeks' duration. He had had two SBP attacks and an attack of hepatic encephalopathy due to esophagus variceal bleeding. He had no history of eating unpasteurized, unsalted cheese made from fresh goat's milk. He had been taking proton pump inhibitors (PPIs), lactulose and propranolol for three years. On admission, he was conscious, pale and icteric. His pulse rate was 65 beats/min, blood pressure 85/53 mm/Hg and body temperature 36.2 °C. Flapping tremor, prominent abdominal tenderness, and distention with ascites were also noted. Other physical findings were unremarkable. Laboratory findings: hemoglobin 11.2 g/dl, hematocrit 30.8%, white blood cell count (WBC) 12800/mm^3, platelet count 115 x 10^9/L, prothrombin time 16.3 s, blood urea nitrogen (BUN) 37 mg/dl, serum creatinine 1.1 mg/dl, sodium 125 mmol/L, potassium 5.1 mmol/L, chloride 102 mmol/L, total protein 5.2 g/dl, albumin 1.9 g/dl, alanine aminotransferase (ALT) 115 U/L, aspartate aminotransferase (AST) 157 U/L, alkaline phosphatase 206 U/L, lactate dehydrogenase (LDH) 275 U/L, total bilirubin 5.7 mg/dl, direct bilirubin 4.3 mg/dl, indirect bilirubin 1.4 mg/dl, and alpha fetoprotein (AFP) >300 IU/ml (0.5-5.5 IU/mL). HBsAg was positive, anti-HBs negative, anti-HBcTotal positive, HBeAg positive, anti-HBe negative and anti- HCV negative. Abdominal ultrasonography showed massive ascites and also a mass in the right lobe of the liver. Peritoneal fluid WBC was 1300/mm^3, with lymphocyte predominance (80%). Biochemical parameters of peritoneal fluid were: protein 1.5 g/dl, albumin 0.7 g/dl and LDH 110 U/L. No organisms were seen on Gram stain of the peritoneal fluid. Ascitic fluid and blood were cultured and ceftriaxone was started 1 g twice a day empirically. However, his clinical condition worsened despite antimicrobial treatment. Three days later serum Brucella agglutination test was positive at a dilution of 1/1280 and at the same time peritoneal fluid and blood culture both yielded Brucella melitensis. Brucella tube agglutination of ascites showed IgM and IgG positivity with ELISA. Therapy was changed to combined therapy with 100 mg doxycycline perorally twice a day plus 1 g ceftriaxone intravenous twice a day and 400 mg ofloxacine perorally twice a day. Peritoneal fluid WBC decreased to 300/mm^3 on the 5th day of the Brucella treatment. Computerized tomography scanning showed a mass in the right lobe of the liver and a biopsy was performed, which confirmed hepatocellular carcinoma. Alcohol injection therapy was planned but his clinical condition worsened and he died on the tenth day of hospitalization due to hepatic failure.

DISCUSSION

Spontaneous bacterial peritonitis is a well-known entity, with a reported incidence of 15-20% in advanced cirrhotic patients (9). The mortality rate for SBP is 33-46%, which increases to 70-88% if the ascitic fluid neutrophil count is >1 x 10^9/L (10). Brucellosis is a systemic infection in which an organ or system of the body can be involved. Gastrointestinal tract and hepatobiliary, nervous, cardiovascular, respiratory, and genitourinary system involvement; and hematologic, cutaneous and ocular complications have also been reported (11). On the contrary, the presence of ascites due to Brucella is an extremely rare finding. Defects in the host defense mechanism play a major role in the pathogenesis of SBP. Patients with decompensated liver cirrhosis have a high risk of bacterial infection and a poorer prognosis. They have defects of the humoral defense mechanism, impaired antibody production and depressed serum complement levels. The cellular defense mechanism is also defective. The function of neutrophils and reticuloendothelial system is depressed. The clearance of enteric organisms from the portal circulation is impaired by portosystemic shunt and impaired Kupffer cell function. Patients with massive ascites are prone to spontaneous bacterial peritonitis due to Gram-negative enteric organisms (12). Ozakyol et al. reported that Brucella had grown in blood and ascitic fluids of a 51-year-old patient with alcoholic cirrhosis and that the Brucella agglutination of the patient was 1/640. In this report it was thought that Brucella peritonitis may have developed due to dysfunction of Kuppfer cells in alcoholism and cirrhosis and diminished cellular immunity (5). The nutritional and immune status of the host, as well as the size of the infectious inoculum and the possible route of transmission can be determinants of brucellosis. For example, the low pH of gastric juices appears to be more effective in preventing Brucella infection. However, PPIs, antiacids and other drugs that decrease gastric acidity have been implicated not only in food- borne brucellosis but also in some other infectious patho-

gens (7). Presence of cirrhosis and hepatocellular carcinoma, which lead to diminished immunity, and use of PPIs may be predisposing factors in our patient. Even if there were not typical Brucella symptoms and signs in our patient, the lymphocyte predominance in the ascitic fluid and arthralgia history of the patient led us to consider that the etiology of the SBP might have been Brucella; diagnosis could have been made with advanced tests. Also, approximately 4% of the patients with ascites have mixed ascites, in which the serum-ascites albumin gradient (SAAG) remains high (>1.1 g/dl), as seen in our patient (1.2 g/dl), reflecting the underlying portal hypertension. Usually an elevated ascitic fluid lymphocyte count provides the clue that something in addition to portal hypertension is the cause of ascites formation. In conclusion, SBP due to Brucella is an extremely rare condition. Brucella as a cause of SBP should be considered in cirrhotic patients who live in areas where brucellosis is enzootic. Also, brucellosis should be suspected in cirrhotic patients who had arthralgia and do not improve with standard SBP therapies.

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