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Spontaneous bacterial peritonitis due to Brucella infection

Brusellanın neden olduğu spontan bakteriyel peritonit

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Brucella infection is a systemic disease, but the microorganism rarely causes infections in the gastrointestinal system such as hepatitis, cholecystitis, colitis and pancreatitis. Spontaneous bacterial peritonitis due to Brucella is extremely rare. Herein, we report a case of cirrhosis complicated with nongranulomatous hepatitis and peritonitis, both due to Brucella. A 63 year-old man with diabetes mellitus was admitted to hospital with complaints of weakness, backache, abdominal pain and abdominal swelling. On the basis of physical examination and laboratory findings, cryptogenic cirrhosis and spontaneous bacterial peritonitis were diagnosed. Due to persistent fever and backache, serum Brucella agglutination test was performed and found to be positive. Brucella melitensis was isolated from ascitic fluid culture. Liver biopsy findings revealed cirrhosis and a nongranulomatous hepatitis which was thought might be due to Brucella infection. Doxycycline and rifampicin, in addition to diuretics were administered for spontaneous ascites infection due to Brucella. A week later, the patient's condition improved and he became afebrile. After two months of therapy, the ascites had almost disappeared.

Key words: Brucella melitensis, nongranulomatous hepatitis, cryptogenic cirrhosis spontaneous bacterial perionitis

Brusella infeksiyonu sistemik bir hastalıktır. Nadiren, hepatit, kolesistit, kolit ve pankreatit gibi gastrointestinal sistem infeksiyonlarına neden olur. Brusellanın sebep olduğu spontan bakteriyel peritonit oldukça nadir görülür. Bu yazıda brusellaya bağlı nongranülomatöz hepatit ve peritonit komplikasyonlu siroz vakası sunuldu. Şeker hastalığı olan 63 yasında erkek hasta halsizlik, sırt ağrısı, karın ağrısı ve karında şişlik nedeniyle hastaneye başvurdu. Fizik muayene ve laboratuvar bulguları değerlendirilerek kriptojenik siroz ve spontan bakteriyel peritonit tanısı verildi. Sırt ağrısı ve ateşinin devam etmesi üzerine Brucella agglütinasyon testi yapıldı ve pozitif bulundu. Asit sıvı kültüründe Brucella melitensis izole edildi. Karaciğer biyopsi bulguları sirozla ve brusella infeksiyonuna bağlı olduğu düşünülen nongranülomatöz hepatitle uyumlu olarak değerlendirildi. Tedaviye diüretiklerin yanısıra doxisiklin ve rifampisin eklendi. Bir hafta sonra hastanın genel durumu düzeldi ve ateşi düştü, iki aylık tedavi sonunda asit sıvısı hemen hemen kayboldu.

Anahtar kelimeler: Brucella melitensis, nongranülomatöz hepatit, kriptojenik siroz, spontan bakteriyel peritonit.

INTRODUCTION

Brucella infections present in two clinical forms: acute and chronic brucellosis, which may resemble a number of diseases. The most prominent symptoms of acute brucellosis are fever, chills, headache, backache and muscular or joint pain. Splenomegaly is usually present and the liver may be palpable. A variety of clinical presentations and complications of brucellosis involving various parts of the body have been reported. It is known that Brucella infection is a systemic disease, but rarely, it may also cause local infections in the gastrointestinal system (i.e. hepatitis, cholecystitis, colitis or pancreatitis) (1-3).

Spontaneous bacterial peritonitis (SBP) is a serious complication of cirrhosis which is seen in 15-20% of advanced cases. The most common pathogenic organisms of SBP are Escherichia coli and Klebsiella pneumonia. Brucella is an extremely rare cause of peritonitis. Herein, we report an interesting case of cirrhosis complicated with nongranulomatous hepatitis and peritonitis, both due to Brucella.

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CASE REPORT

A 63 year-old man was admitted to Erciyes University Hospital with complaints of weakness, backache, abdominal pain and abdominal swelling. He had a history of diabetes mellitus for 13 years. On physical examination, temperature was 38°C. He had ascites and an enlarged spleen.

Laboratory analyses were as follows: haemoglobin 11.4 g / dl, white blood cell count 4100 / mm3 and platelet count 150000 / mm^3. Abnormal biochemical findings were gamma-glutamyl transpeptidase: (GGT) 552 U / L, alkaline phosphatase: 504 U / L, total proteins: 5.2 g / dl, albumin: 2.4 g / dl, LDH: 892 U / L. The prothrombin time was 12 seconds. Ascitic fluid findings were as follows: leukocytes 800 / mm^3 (with 70% lymphocytes), total proteins 2.8 g / dl, albumin 1.2 g / dl. Serum ascide albumin gradient (SAAG) was 1.2 g / dl. The HBs Ag and antibodies for HBs Ag (anti-HBs), HCV (anti-HCV), smooth muscle antigen (SMA), nuclear antigen (ANA), liver-kidney microsomal antigen (LKM-1) and mitochondrial antigen (AMA) were all found to be negative. Congestive gastropathy was observed during upper GI endoscopic examination and a abdominal ultrasonography revealed splenomegaly and ascites, with the portal vein diameter being 14 mm. Due to persistent fever and backache, serum Brucella agglutination test was performed. Brucella serology showed a positive slide test, micro-agglutination titer I/ 1280. The slide test was also positive in peritoneal fluid as was the micro-agglutination test. On the fourth day of admission, gram-negative coccobacilli were noted in blood culture bottles and on day six, they were identified as Brucella melitensis, which was also isolated from ascitic fluid culture. Liver biopsy findings revealed cirrhosis (Figure 1) and a nongranulomatous hepatitis which was thought might be due to Brucella infection (Figures 2-3). Endoscopic retrograde cholangiopancreatography (ERCP) was performed due to increased GGT and alkaline phosphatase levels and a radioopaque shadow compatible with a common bile duct stone was seen.

A diagnosis of cryptogenic cirrhosis was established according to histologic, viral and serologic findings. Doxycycline (100 mg. bid) and rifampin (300m. gdaily), in addition to diuretics were administered for spontaneous ascites infection due to Brucella. A week later, the patient's condition had improved and the became afebrile. After two months of therapy, the ascites has almost disappeared.

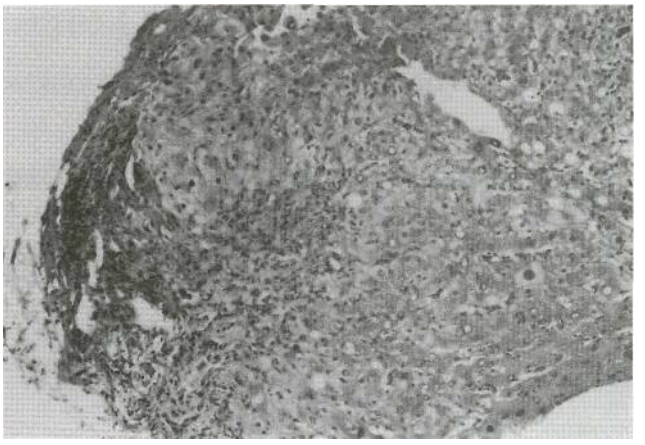

Figure 1. Irregular fibrosis with rare mononuclear inflammatory cells surrounding a macronodule (H&E, xlOO).

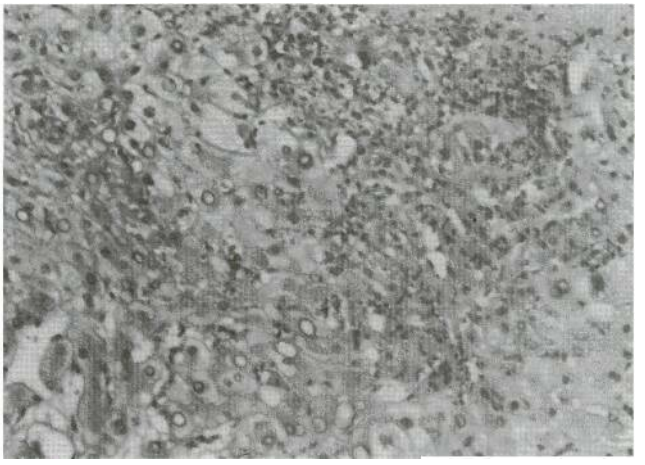

Figure 2. Enlarged portal tract and piecemeal necrosis (H&E, xlOO).

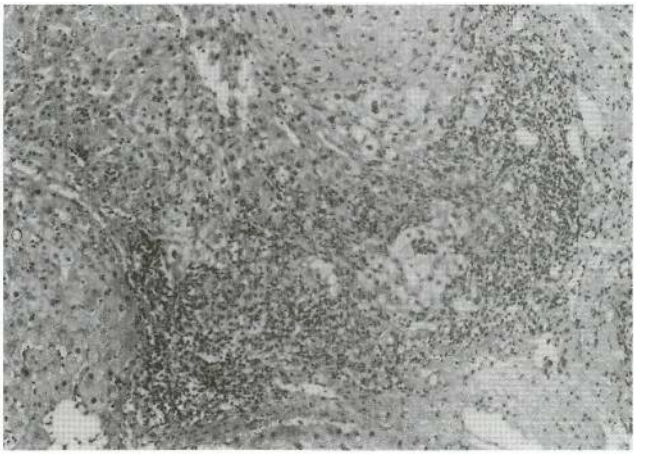

Figure 3. Inflammatory cells marching into parenchyma through the destroyed limiting plate (H&E, x200).

DISCUSSION

The mortality rate for SBP is 33-46%. Ifthe ascitic neutrophil count is more than 1000 / mm3, this rate increases to 70-88% (4). Patients with ascites caused by diseases other than cirrhosis rarely have SBP. Defects in the host defense mechanism play a major role in the pathogenesis of SBP. There are frequent infections in cirrhotic patients, as their defenses against infectious agents are altered, and bactericidal and opsonic activites in the ascites of cirrhotic patients are reduced (4-6). Although Escherichia coli and Klebsiella pneumonia are the most common etiological organisms, a few unusual organisms such as Yersinia enterocolitica, Listeria monocytogenes and Brucella melitensis may cause SBP (5-10). Brucella is usually caused by ingestion of unpasteurised dairy products or infected raw liver (7). Once Brucella coccobacilli are ingested, they enter the lymphatic system via the gastrointestinal system. A hematogenous dissemination ensues and is then followed by colonisation of Brucella in reticuloendothelial-rich organs such as the liver, spleen, lymph nodes, bone marrow and kidney. Brucella rarely causes infections in the gastrointestinal system such as hepatitis, cholecystitis, colitis and pancreatitis. Spontaneous bacterial peritonitis due to Brucella is extremely rare. To our knowledge, this is the sixth reported case of culture-proven spontaneous bacterial peritonitis caused by Brucella melitensis in a cirrhotic patient (4, 7-10). In our patient, there was not only cirrhosis and SBP caused by Brucella melitensis, but also nongranulomatous hepatitis due to brucella. The has been no reported case of both cirrhosis and hepatitis in the literature.

The World Health Organisation recommends a six-week course of doxycycline and rifampicin or doxycycline and streptomycin for the treatment of brucellosis (9). Patients with neurobrucellosis or Brucella endocarditis may require longer courses and treatment should be continued until the patient recovers (11). Although there is no such consensus about the duration of treatment for Brucella peritonitis, it was decided to give antibiotics until the ascites resolved, which took two months in this case.

In conclusion, Brucella may cause SBP in cirrhotic patients with ascites. It could be thought that the histopathological changes in our patient may have been due to a nongranulomatous Brucella hepatitis rather than cirrhosis, but the presence of fibrosis and regeneration nodules in the liver and in particular, findings consistent with portal hypertension such as congestive gastropathy and portal vein dilatation, refute such a suggestion. Furthermore, it is known that in Brucella hepatitis, hepatic lesions resolve with antimicrobial therapy and in the absence of other causes, cirrhosis does not occur, despite the severity of inflammation (1). Althoughsome organisms, such as tuberculosis, may directly initiate peritonitis (12), a hepatic (or cirrhotic) ascites always precedes the development of Brucella peritonitis.

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