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LETTERS TO THE EDITOR

EDİTÖRE MEKTUP

Hepatic abscess in brucellosis: A case report

Brusellaya bağlı karaciğer absesi

Dear editor

A 49 year-old female patient, was admitted to hospital with complaints of intermittent fever, wide spread joint pain, perspiration and upper right quadrant abdominal pain.

She resided at her village for a few monts each summer, having contact with animals (some of which had abortions) and eating animal produce, particulary unpasteurized cheese.

On physical examination, the liver was palpated four cm below the the right costal arch at the mid clavicular line. Results of laboratory investigations were as follows: Hb: 9.7 gr/dl, WBC 16700 mm3 sedimentation rate 73 mm/hour. Blood culture was negative. In serum biochemical investigations, AST was 56 U/L, alkaline phosphatase: 385 U/L and GGT: 159 U/L.

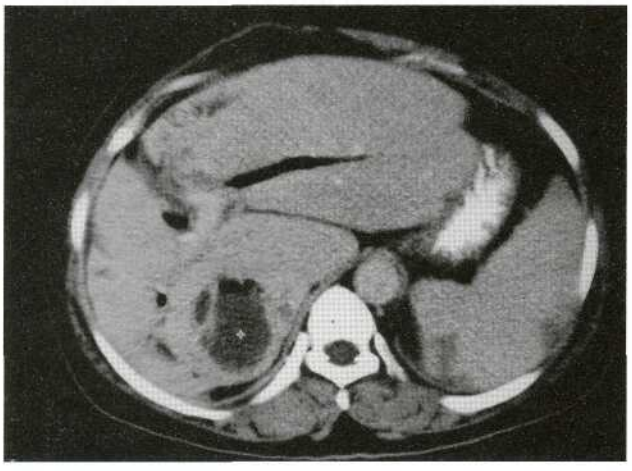

Figure 1. Collection at the right lobe of liver, with septation 3x5 cm in size.

Ultrasonography and computerized axial tomography (CAT) investigation revealed a collection that was 3x5 cm in size with septation at the right lobe of the liver below the diaphragm (Figure 1). Brucella agglutination test was found to be positive at 1/360 titrations. Oral treatment of rifampicin 600 mg/day (every morning), tetracycline 250 mg qid and ciprofloxacin 500 mg bid was commenced. Clinical findings improved after the first week and after two weeks, ultrasonography showed the abscess in the right lobe to be substantially improved. Medical treatment was continued for two months and at the end of this period, laboratory results had returned to normal ranges and brucella agglutination test was negative. Follow up ultrasonograpy and CAT established that the liver abscess had with calcification remaining in the region.

Although liver involvement is frequent in patients with brucellosis, hepatic abscess develops after a long latent period in patients with chronic brucellosis.

Brucellosis is seldom isolated from the liver abscess or blood cultures (2, 6). In our study, it could not be isolated in blood culture. Although the definitive diagnosis is made with isolation of the causative agent from abscess or blood cultures, in cases where this is not possible, diagnosis is made on the basis of patients history and confirmed with serological test (2, 3). In our case, the clinical findings, history, positive serological tests and complete response to medical treatment confirmed the diagnosis.

Absence of any disorder that could cause septic fever and pyogenic abscess, such as cholangitis,

Address for correspondence: Dr. Ertuğrul KAYAÇETİN
Türkiye Yüksek İhtisas Hastanesi Gastroenteroloji Kliniği, Ankara

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dramatic presentation of disease, multiple visceral involvement and unusual localization of infarctions have been reported (1,2). Involvement of hepatic and gastrointestinal tract arteries have also been reported although hepatic involvement and hepatic aneurysm rupture are rarely seen (8-10). Necrotizing vasculitis of appendix vermiformis and acute appendicitis as a clinical manifestation of PAN are also uncommon; the largest series in the literature consists of 12 cases reported by Moyana who demonsrated the close relationship between necrotizing arteritis of appendix vermiformis and polyarteritis nodosa (11). In the acute phase, fibrinoid necrosis and mixed type inflammatory cell infiltration involving the whole wall are observed.

Since our patient had no previous history of polyarteritis nodosa, his abdominal pain was initially thought to be due to a gastroenteritis. In spite of the medical treatment, however, his general condition and liver function tests deteriorated and further radiological investigations, especially CT scan and selective celiac angiography, led to the diagnosis of subcapsular hematoma and suspicion of underlying polyarteritis nodosa, preoperatively. Multiple heterogenous lesions in segment three, six and seven of the liver in the CT scan indicated subcapsular hematoma, and multiple aneurysmal dilatations of the hepatic, splenic and renal arteries in selective celiac angiography led us to consider the possibility of underlying polyarteritis nodosa, although we did not expect to find necrotizing appendicitis preoperatively. Histopathologic examination of the liver biopsies supported the diagnosis of polyarteritis nodosa as did histopathologic examinations of appendix vermiformis, which was not expected.

Since PAN is an uncommon vasculitis and the symptoms are non-specific, the diagnosis is always difficult. The lesions may occur in visceral organs and diagnosis often depends upon microscopic tissue examination (12). In our patient, a provisional diagnosis was initially made by angiography and subsequently confirmed by tissue biopsy. The American College of Rheumatology have established ten criteria for diagnosis of polyarteritis nodosa and stated that a patient with vasculitis may only be diagnosed with polyarteritis nodosa if at least three of these 10 criteria are present (13). In our patient, the presence of five of these criteria (diastolic pressure >90mm Hg, myalgias, muscular weakness, weight loss >4 kg, angiographic abnormalities and PMN in a medium sized artery supported the diagnosis of polyarteritis nodosa.

Necrotizing vasculitis of appendix vermiformis and acute appendicitis as a clinical manifestation of PAN is uncommon. Hepatic aneurysm rupture is also rarely seen (14). Indeed, more than one complication in a patient is fairly rare. Intrahepatic bleeding and hemobilia with complicated peritonitis have been reported in only one case with perirenal and hepatic hematoma in only one other (10,14). In our case hepatic aneurysmal rupture and necrotizing appendicitis were seen simultaneously. Although PAN may occur with visceral infarction, the involvement of liver and appendix vermiformis in the same case is very unusual and thus its rarity is worthy of report.

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