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LETTERS TO THE EDITOR
EDİTÖRE MEKTUP
A case of visceral leishmaniasis misdiagnosed as autoimmune hepatitis

Otoimmün hepatit tanısı ile izlenen bir visseral leishmaniasis olgusu

Tho the Editor,

A two-year-old girl was admitted to our clinic with the complaints of fever, abdominal distension, anorexia and irritability for the last five months. Physical examination revealed hepatosplenomegaly. Before hospitalization of the patient in our center, she had been evaluated at the local hospital. Hypochromic microcytic anemia, elevated hepatic transaminase levels, hypoalbuminemia, hyperglobulinemia, and high sedimentation rate and C-reactive protein level were detected. They could not demonstrate any specific pathogen with blood, urine and throat cultures. Viral serology, Salmonella and Brucella antibodies were negative. Direct Coombs', smooth muscle antibody, and liver-kidney microsomal antibody were negative. Antinuclear antibodies (ANA) and antids DNA were found to be positive. Abdominal ultrasound demonstrated hepatosplenomegaly with a uniform increase in the parenchymal echogenicity. On histologic examination, the liver structure seemed to be preserved. Hepatocyte apoptosis, sinusoidal inflammation and Kuppfer cell hypertrophy were noted. Portal mixed inflammation was striking in some areas and histiocytic aggregates forming microgranulomas were seen, but no specific microorganism was detected with special stains. In addition to inflammatory changes, there were wide areas of hemorrhagic necrosis involving zones 3 and 2. Bone morrow aspiration was found normocellular but eosinophilia was detected. The patient was diagnosed at the local center as autoimmune hepatitis based on these findings. Prednisone and azathioprine were started. After four weeks of therapy, the patient's clinical and laboratory findings worsened and she was referred to our clinic for evaluation of liver transplantation. During the hospitalization period we observed high fever. In light of the clinical and laboratory findings, such as fever, hepatosplenomegaly, hyperglobulinemia and eosinophilia, we suspected visceral leishmaniasis (VL); amastigotes were shown by repeated bone marrow examination. After 20 days of glucantime therapy, fever and organomegaly resolved. Serum aminotransferases, total protein, albumin, immunoglobulin levels, ANA and antids DNA were all performed and all were found to be within normal limits six weeks after discharge.

Autoimmune hepatitis (AIH) is characterized by the presence of interface hepatitis and plasma cell infiltration on liver biopsy examination, hypergammaglobulinemia and autoantibodies. Diagnosis requires the exclusion of other conditions that resemble AIH (2).

Hepatosplenomegaly, elevated hepatic transaminase levels and hyperglobulinemia are common findings both in VL and AIH. The presence of autoantibodies in patients with VL has been reported before (3-6). The positivity of autoantibodies to ribonucleoproteins in VL is due to polyclonal activation of lymphocytes and molecular mimicry between leishmanial antigens and ribonucleoproteins (3-7).

In our patient, amastigotes could not be demonstrated at the local hospital, and because of the autoantibody positivity the patient was misdiagnosed as autoimmune hepatitis. Although there are diagnostic problems in VL in nonendemic areas, VL with the classical triad is a common childhood

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protosoal disease in our country. VL is a potentially fatal infection, especially in immunocompromised patients. For this reason, in endemic areas, VL should always be excluded, even in patients already diagnosed as having autoimmune disorders, before starting the immunosuppressive therapy.

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