Dear Editor,

Visceral leishmaniasis (VL) is a systemic disease, caused by Leishmania donovani complex. It is estimated that the incidence of human visceral leishmaniasis exceeds 500,000 worldwide and accounts for 75,000 deaths annually. Moreover, due to increasing rate of HIV infection in areas endemic for VL, HIV-VL co-infection in the adult population is being reported frequently. The clinical signs in human include protracted fever, hepatosplenomegaly, substantial weight loss, progressive anemia and may be occasionally fatal. L. infantum is responsible for VL in children in the Mediterranean basin and Iran. The diagnosis of VL is complex, because commonly occurring diseases such as malaria, typhoid fever and tuberculosis may show similar clinical features. Moreover, some of VL cases have been misdiagnosed as autoimmune hepatitis, acute lymphoblastic leukemia and malignant lymphoma. Most of these misdiagnosed patients are reported from non-endemic regions where physicians do not expect the occurrence of the disease. Moreover, atypical cells and different blast may be observed in bone marrow aspiration of VL patients.

In 2004, a 2.5-year-old boy from the eastern district of Fars Province was referred by a general practitioner to a hematologist in Shiraz with the chief complaint of prolonged fever and pancytopenia in blood cell count and impression of leukemia. The patient was referred from eastern Fars Province where VL was not endemic. Bone marrow aspiration showed acute myeloid leukaemia (AML-M2). The patient was admitted to Nemazee hospital where he underwent on abdominal sonography, showing mild hepatosplanomagaly. The patient received chemotherapy with cytarabine (cytozar), danamycin, vp16, methotrexate and hydrocortisone. Laboratory findings provided on the following day were as follows: Hb 12 mg/dL, white blood cell 3200/µL and platelet 67000/µL; liver function test showed protein 7.3 mg/dL, Alb 3.2 mg/dL, glob 4.1 mg/dL, AST 220 IU/L, ALT 105 IU/L, ALK-Ph 108 IU/L, D-Bill 0.1 mg/dL and T-Bill 0.3 mg/dL; ESR 6 cm after the first hour; biochemical tests showed normal serum Na, K, BUN and creatinine; on cytological examination, no blast cell was found in cerebrospinal fluid. After a week, a markedly hypocellular marrow with dirty background, suggestive of recent toxic effect, was seen in bone marrow aspirate. White blood cell and platelet count, gradually decreased during the ensuing 20 days, were 1000/µL and 30,000/µL, respectively. Leishman bodies were observed on repeated bone marrow aspiration performed two months later, when anti-leishmanial antibody titer of 1/128 was found by indirect fluorescent antibody test (IFAT). However, liver function tests were normal and therapy with glucantime was administered. After 18 days, no response was seen and the fever did not subside and the therapy was replaced by amphotericin B. Body temperature gradually decreased during 7 days. New bone marrow aspiration showed a hypercellular marrow with decreasing...
number of leishman bodies as compared with the previous report. The examination of bone marrow aspirate also revealed the absence of megakaryocytes and normal myeloid and erythroid maturations. Amphotericin B was administrated for 20 days and the patient was discharged in good condition.

Five months later, the patient was readmitted to the hospital with chief complaint of progressive abdominal distention. On physical examination, the patient had marked splenomegaly. Hematologic examinations showed Hb 6mg/dL, white blood cell 2000/µL and platelet 144000/µL. Anti-leishmanial antibody titers demonstrated by IFAT and direct agglutination test (DAT) were 1/526 and 1/3200, respectively. Duodenal and liver needle biopsies showed numerous leishman bodies, which was indicative of parasite dissemination. Unfortunately, the patient died on the same day.

The highly endemic areas of VL in Iran are different parts of Fars and Bushehr provinces in South, the districts of Meshkin-Shahr and Moghan in Northwest and Qom province in Central Iran. However, sporadic cases of VL have been reported from other parts of Iran.5-7 The unfortunate haste and oversight of hematologist in performing primary bone marrow aspiration accounted for the failure in reaching a correct diagnosis. The presence of Leishman bodies in bone marrow aspirate is highly specific for the diagnosis of VL, although demonstration of parasite maybe time-consuming and requires a prolonged search. Moreover, serologic and molecular methods must be performed to arrive at a definitive diagnosis.8 In conclusion, this is the first report of visceral leishmaniasis misdiagnosed as AML-M2. Furthermore, in endemic or even non-endemic areas, patients with clinical signs similar to VL must be referred to hematologist or pathologist who will then perform bone marrow aspiration after obtaining patient’s consent, with due emphasis on parasite demonstration in bone marrow aspirate. In regard to reactivation of parasite and sensitivity to infection of immunocompromised and immunosuppressed patients,9,10 the attention of physicians are drawn to such underlying conditions.

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Q Asgari,1 M Fakhar,1,2 MH Motazedian,1 F Cheraqali,3 E Banimostafavi1

1Department of Medical Parasitology and Mycology, School of Medicine, Shiraz University of Medical Sciences, Shiraz, Iran, 2Department of Medical Parasitology and Mycology, School of Medicine, Sari University of Medical Sciences, Sari, Iran, 3Department of Pediatric, Gorgan University of Medical Sciences, Gorgan, Iran

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