Cutaneous Leishmaniasis in AIDS

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Recent data show that 50% of adult HIV-positive patients are also affected by visceral leishmaniasis (VL) in endemic areas and that 1-3% of AIDS patients become infected with VL in the southwest of Europe [1].

In relation to the immunodeficient state, VL is associated with a poor prognosis [2]. It is therefore suggested that leishmaniasis is an opportunistic infection in HIV-positive patients [3]. Furthermore, since reports of AIDS patients with VL have increased in number, it has been suggested that this infection should be included as a diagnostic criterion for AIDS [3-5].

We report the case of a cutaneous leishmaniasis (CL) whose torpid course, resistance to treatment and histology made us suspect an immunodeficient syndrome. A woman, 39 years old with no relevant antecedents, showed in the left cheek a 2-cm diameter brown nodule that had developed during 1 month (fig. 1). Biopsy showed a huge number of leishmanias within macrophages and confirmed the clinical suspicion (fig. 2). Keto-

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Fig. 1. Chronic facial ulcerated nodule.

Fig. 2. Numerous endocellular Leishman bodies in the dermal infiltrate of macrophages.

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conazole treatment was initiated with a dose of 400 mg/day. The lesion improved but we could not see a total healing because the patient canceled her appointments.

Six months later she came again because the lesion had relapsed dramatically. With the suspicion of an underlying immunological alteration we carried out further explorations.

She was found to have a history of paren-teral drug addiction. Serology for HIV was positive.

Lymphocyte populations were: CD4, 0.04; CD8, 0.7; CD4/CD8 = 0.05. We wanted to discard a VL. Bone marrow aspiration and hepatic biopsy were made with negative results. The cutaneous lesion did not heal under 10 mg of .V-mefhylglucamine given intramuscularly during 5 weeks.

In contrast it enlarged and became ulcerous. This was the reason for the surgical removal. By now and after 6 months, the nodule or any other clinical manifestation of leishmaniasis did not relapse.

CL in HIV-positive patients has been poorly studied. There are only a few reports about VL [6] or CL and herpes simplex virus mixed infections [7,8]. Also the development of the scattering American CL among pa-

tients with AIDS has been considered [9]. In some of these studies the cutaneous infection has a particular virulence and is resistant to the conventional therapies.

We consider interesting to report this case because CL, which is less related to AIDS than VL, has behaved in this case like an opportunistic infection.

We think that more experience is needed, mainly in HIV-positive patients of endemic zones, to be able to clarify whether CL – like VL – can help in the prognosis of these patients or if the case presented, like some others, is a pure coincidence.

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