Cutaneous leishmaniasis mimicking Kaposi's sarcoma in an HIV patient

Leishmaniasis cutánea simulando un sarcoma de Kaposi en un paciente VIH

Dear Editor,

Introduction

Leishmaniasis is a disease caused by protozoan parasites, which is widely distributed throughout the world and can affect internal organs and the skin. In immunosuppressed patients, such as those with HIV, the clinical presentation may be atypical and extensive.

Case report

We present the case of a 45-year-old Moroccan male with HIV infection on antiretroviral treatment, undetectable viral load and $CD4^+$ count of 49 cells/μl. He had a history of visceral leishmaniasis (VL) which had been incompletely treated two years earlier, and untreated syphilis of indeterminate duration.

The patient was referred to dermatology for nodular lesions on both forearms which had started to develop a year previously, along with asthenia and joint pain in his hands. Physical examination showed erythematous, violet-coloured nodular lesions on the extensor areas of the forearms (Fig. 1A–C), bilateral inguinal lymphadenopathy and scrotal oedema, with no mucosal involvement.

Analysis revealed leucopenia, anaemia, hypoalbuminaemia and increased acute phase reactants. A full-body CT scan showed axillary, supraclavicular, mediastinal, retroperitoneal and inguinal lymphadenopathy, along with hepatosplenomegaly.

A biopsy of a skin nodule was taken due to suspicion of Kaposi's sarcoma (KS) or cutaneous lymphoma. The histological study showed an inflammatory infiltrate with lymphocytes and histiocytic cells parasitised by numerous *Leishmania* amastigotes (Fig. 2A–D). A biopsy of the jugular lymphadenopathy confirmed the presence of the parasite, identified as *Leishmania infantum*.

The patient was diagnosed with VL and cutaneous leishmaniasis (CL), receiving complete treatment with liposomal amphotericin B, followed by maintenance prophylaxis (2800 mg IV once a week for 3 weeks). The skin lesions resolved with residual hyperpigmentation, the joint pains improved, and the analytical parameters returned to normal.

Discussion

Manifestations of leishmaniasis depend on the species responsible and the patient's immune status.

Old World leishmaniasis is endemic in Asia, Africa, the Mediterranean basin and the Middle East, including *L. donovani, L. infantum, L. chagasi, L. tropica, L. major* and *L. aethiopica.*[1,2] *L. infantum*, whose reservoir is the dog, can cause CL, mucocutaneous leishmaniasis (ML) or VL.[2] CL manifests as papules at the inoculation site, which evolve into ulcerous-crusted plaques or nodules. ML develops with nasal congestion, epistaxis and pharyngeal symptoms. VL causes fever, weight loss, hepatosplenomegaly, lymphadenopathy and anaemia.[1,2]

Among the Old World species, *L. aethiopica* is the only one that causes diffuse cutaneous leishmaniasis (DCL),[2] characterised by subcutaneous nodules on the elbows, knees, face and earlobes. *L. tropica* typically produces fewer than three ulcers on the head, which resolve within two years, whereas *L. major* causes multiple self-limiting ulcers that heal within one year.[2]

New World leishmaniasis is found in Central America, South America and southern Texas, with species such as *L. mexicana, L. amazonensis, L. venezuelensis, L. braziliensis, L. guyanensis, L. panamensis* and *L. peruviana.*[1,2] All of them can cause CL, with *L. mexicana* being the main cause of DCL.[2] *L. braziliensis* causes serious infections, *L. mexicana* can induce Chiclero's ulcer on the outer ear, and

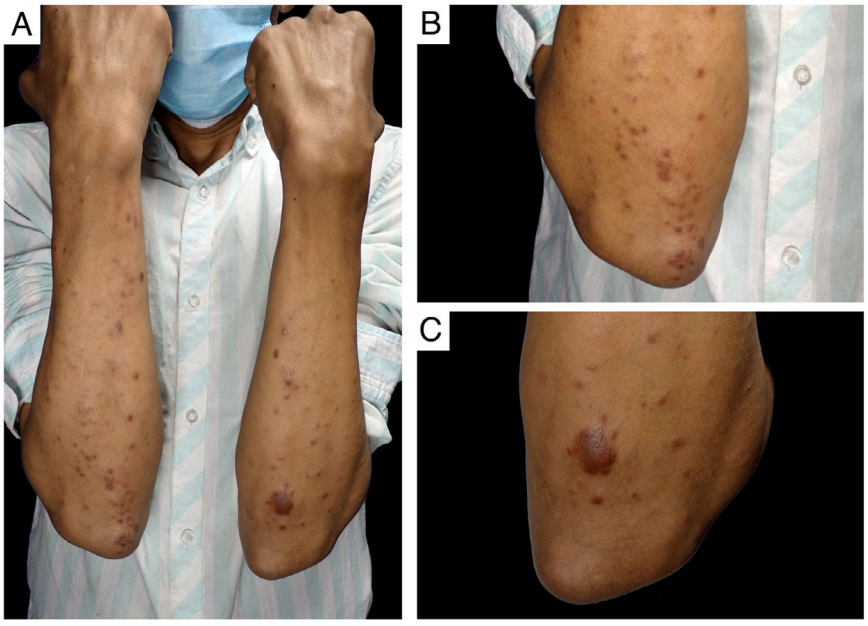

Fig. 1. A–C) Clinical images of the patient. A) Red-purple papules, plaques and nodules on the extensor surface of both forearms. In more detail, note that the lesions acquire a violet tone (B) and in some cases become nodular (C).

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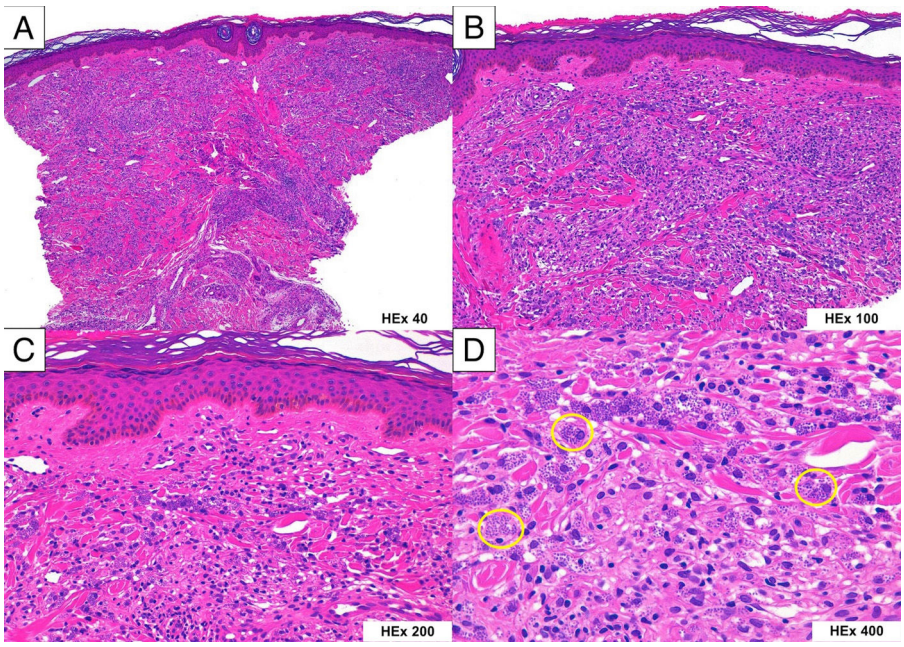

Fig. 2. A–D) Histological sections with haematoxylin-eosin (H&E) staining of skin biopsy. A) An inflammatory infiltrate can be seen extending into the deep reticular dermis (H&E ×40). B) Note how the infiltrate acquires a more diffuse distribution on the surface and more nodular at depth (H&E ×100). C) We can see that the infiltrate is predominantly composed of histiocytes and lymphocytes (H&E ×200). D) We can see numerous *amastigotes of Leishmania*, predominantly in the cytoplasm of the histiocytes (yellow circles) (H&E ×400).

L. panamensis is associated with non-healing ulcers extending into the lymph channels.

Co-infection of *Leishmania* and HIV is common, with VL predominating, although cases of solitary CL have also been described.[2,3] Most leishmaniasis in patients with HIV appears in advanced stages (77%–90% have fewer than 200 $CD4^+$ cells/μl). Both pathogens target macrophages, and VL accelerates HIV progression and increases susceptibility to opportunistic infections.[3]

For this reason, in immunosuppressed patients, and specifically in patients with HIV with low CD4 count, CL manifestations are atypical, more severe and extensive,[2] as occurred in our patient. In the literature, diffuse papulonodular rashes,[5] kaposiform and nodular lesions in patients with HIV and VL,[4] several cases of DCL in the context of co-infection with HIV or within the immune reconstitution syndrome, some of them mimicking post-kala-azar dermal leishmaniasis (papular rash on the face and upper extremities during, or several months after, treatment of visceral disease),[3,5–8] or forms of CL simulating cutaneous lymphoma[9] have all been described.

In conclusion, leishmaniasis should be considered in the differential diagnosis in immunosuppressed patients with atypical skin lesions, and samples should be taken for histology and microbiology for correct diagnosis.

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