Oro-Pharyngeal Mass in a Patient With HIV Infection

Luis Mérida Rodrigo,[a] Julián Olalla Sierra,[a] Alfonso del Arco Jiménez,[a] and Isabel Rodrigo Fernández[b]
[a]Servicio de Medicina Interna, Hospital Costa del Sol, Marbella, Málaga, Spain
[b]Servicio de Anatomía Patológica, Hospital Costa del Sol, Marbella, Málaga, Spain

CASE STUDY

Male, 65-year-old, with human immunodeficiency virus infection (HIV), category C3, and cirrhosis due to hepatitis C virus, treated with abacavir, lamivudine, zidovudine, fosamprenavir, ritonavir, spironolactone, and prophylaxis against *Pneumocystis jirovecii* with TMP/SMX. He came to the clinic due to pharyngeal and retrosternal odynophagia lasting 1 week. Over the previous week he had presented fever in the evenings, increase in abdominal perimeter and oedemas on his legs.

The examination revealed an ulcerovegetative lesion measuring over 4 cm on the soft palate and the uvula (Figure 1), hepatosplenomegaly, abdominal distension, ascitic waves, and oedemas on the legs with fovea. The blood work-up revealed: LDH, 682 U/L; albumin, 1.93 g/dL; haemoglobin, 8.2 g/dL; 1420 leucocytes/µL (44% neutrophils), and 84 000 platelets/µL.

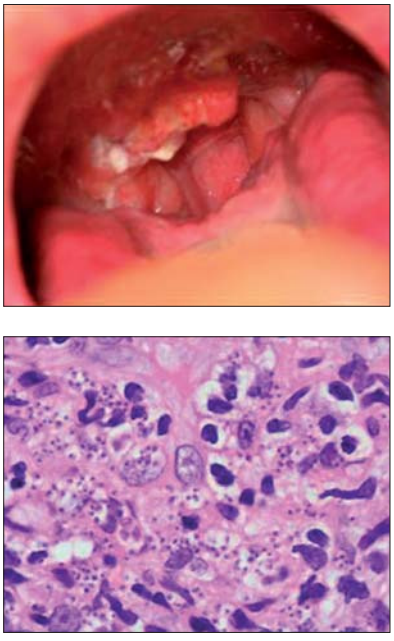

Figure 1. Ulcerovegetative lesion on the soft palate. Biopsy of the pharyngeal mucosa. Numerous leishmanias in the cytoplasm of the histiocytes.

With a suspected neoplasia of the soft palate, a biopsy of the lesion was performed using direct laryngoscopy and marked epithelial hyperplasia and an intense lymphohistiocytic inflammatory infiltrate was observed, with presence in the histiocytes of sandflies typically diagnostic of leishmaniasis (Figure 1). Due to thrombocytopenia, treatment was begun with intravenous antimonials at a dose of 20 mg/kg/day. In the first few day he presented fever, with the formation of abscesses at the infusion sites on the peripheral accesses, therefore the treatment was changed to liposomal amphotericin B at doses of 3 mg/kg/day, on days 0, 1, 2, 3, 4, and 10; his progress was satisfactory, with a marked reduction in Leishmania parasites on the control biopsy (Figure 2).

DISCUSSION

Visceral leishmaniasis is a parasitosis transmitted by *Phlebotomus sandflies*, although the HIV infection may alter the transmission mechanisms.[1] The forms of presentation in patients co-infected by HIV may have atypical locations,[1] one of which, namely the pharynx, often occurs in the course of a visceral leishmaniasis years later or in connection with lesions in the nasal cavity, pharynx, and tongue, in the form of nodules or ulcerous lesions, in which dysphonia is the most frequent symptom.[2] These develop when a visceral leishmaniasis becomes chronic; the parasite

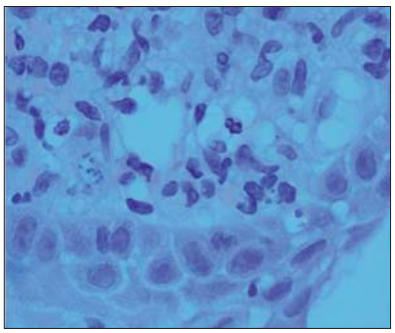

Figure 2. Marked reduction of *Leishmania in* the cytoplasm of the histiocytes in the pharyngeal mucosa after treatment with antimonials and 5 doses of liposomal amphotericin B.

The authors have not indicated any conflict of interest.

Correspondence: Dr. L. Mérida Rodrigo.
Servicio de Medicina Interna. Hospital Costa del Sol.
Avda. de la Aurora, 46, 2.º A. 29006 Málaga. España.
E-mail: LUISMERO@terra.es.

Received June 27, 2007.
Accepted for publication November 29, 2007.

takes up residence in the mononuclear phagocytic system and control of the disease depends on cell immunity.[3] A peculiarity of leishmaniasis associated with HIV infection is its tendency to relapse (25%-61%), normally precociously, generally within 1 year. Some studies have shown that the relapsing course of infection by *Leishmania donovani* in patients with HIV is linked to certain factors: positive serology to *Leishmania* in the initial episode, female gender, incomplete treatment of the initial episode, onset of a first relapse, and absence of secondary prophylaxis of leishmaniasis. It would have been necessary to aspirate bone marrow and produce a culture to discard isolated mucocutaneous leishmaniasis, but our patient refused. Treatment is based on antimonials, amphotericin or surgery for the most resistant forms.[4] Due to the emergence of resistance, adverse effects and co-infection with HIV, the treatment has changed in recent years.[2] The response rate to antimonials is 30%-50%; to liposomal amphotericin B at a dose of 1 mg/kg/day for 15 days, 60%,[3] and miltefosin at a dose of 2.5 mg/kg/day for 28 days, 94%.[5] In our case, because we could not discard isolated oro-pharyngeal leishmaniasis, it was decided to apply medical treatment. The therapy described for our case with liposomal amphotericin B reduces the duration of hospitalization and costs, with a response rate similar to that of classic treatments.[6]

Therefore, in immunodeficient patients we have to consider the possibility of this kind of infection in lesions with a clinical appearance of neoplasia produced in the oral cavity.

REFERENCES

1. Sinha PK, Pandey K, Bhattacahra SK. Diagnosis & management of leishmania/HIV co-infection. Indian J Med Res. 2005;121:407-14.
2. Benítez D, Miranda C, Navarro JM, Morillas F, Martín J, de la Rosa M. Varón de 36 años con disfonía resistente al tratamiento médico convencional. Enferm Infecc Microbiol Clin. 2001;19:233-4.
3. Benítez D, Miranda C, Navarro JM, Morillas F, Martín J, de la Rosa M. Leishmaniasis aislada laríngea y cultivo de médula ósea. Enferm Infecc Microbiol Clin. 2002;20:133-4.
4. Pintado V, Lopez-Velez R. Visceral leishmaniasis associated with human immunodeficiency virus infection. Enferm Infecc Microbiol Clin. 2001;19: 353-7.
5. Rosenthal E, Marty P. Recent understanding in the treatment of visceral leishmaniasis. J Postgrad Med. 2003;49:61-8.
6. Bodet E, Andreu L, Ruiz Giner A, Fortuny JL, Palomar V. Leishmaniasis laríngea: presentación de dos casos clínicos. ORL-DIPS. 2002;29;131-4.