Case Report: A Patient Presenting with Diffuse Cutaneous Leishmaniasis (DCL) as a First Indicator of HIV Infection in India

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Abstract. Opportunistic parasitic infections such as leishmaniasis are common in human immunodeficiency virus (HIV)-infected patients and are usually acquired several days after initial diagnosis of HIV infection. Here, we report on a patient who presented with diffuse cutaneous leishmaniasis (DCL) caused by Leishmania tropica as the first and only clinical manifestation of HIV infection. To the best of our knowledge, this is the first case that illustrates that DCL could be the first clinical indicator of HIV infection. Cutaneous leishmaniasis (CL) and DCL are becoming frequent opportunistic infections in HIV-infected individuals throughout the world. To date, all documented cases of CL and HIV coinfections have been reported in patients who were known cases of HIV and who subsequently developed CL. In this report, we present a case that illustrates that DCL could be the first clinical indicator of HIV infection.

CASE REPORT

A 32-year-old housewife, resident of Bikaner, Rajasthan, India, presented to the dermatology outpatient department of Sardar Patel Medical College with multiple intensely itchy oval to round papulo-nodular lesions of variable size scattered over her limbs (Figure 1), face, and trunk for the last 7 months. History revealed that the lesions first appeared over her limbs and then, gradually spread to her whole body without affecting mucous membranes. New lesions kept on appearing, whereas older lesions gradually increased in size; none of the lesions ulcerated. She never traveled to endemic areas of Kala azar and leprosy. General and systemic examination of the patient did not reveal any abnormality. The laboratory investigations showed anemia (Hb = 8.5%) and lymphopenia (18%). All other investigations, including liver and renal function tests, chest X-ray, and ultrasonography (USG) of the abdomen, were found to be normal. Histopathology of the skin lesions revealed heavy infiltrate of macrophages infected with Leishmania, which was identified as L. tropica by restriction fragment-length polymorphism (RFLP) polymerase chain reaction (PCR)1 (Figure 2). Montenegro test for leishmaniasis was not available, but Mantoux test for tuberculosis (TB) was negative. Human immunodeficiency virus (HIV) infection was suspected, because the patient presented with atypical, generalized, intensely pruritic lesions of cutaneous leishmaniasis (CL); this was confirmed using three different enzyme-linked immunosorbent assay (ELISA) test kits (Tridot, TMB Micro-ELISA, and Combs AIDS; J Mitra & Co., New Delhi, India). Further investigations showed that the CD4 + T cells count was 44 cells/mm³ and that the viral load was 165,400 copies/mL. Highly active antiretroviral therapy (HAART; Zidovudine, Lamivudine, and Efaviranz, Ranbaxy, India) was instituted immediately, and systemic antileishmanial treatment was started simultaneously with intramuscular SSG and hence, was started on Rifampicin (1,200 mg/day) along with HAART. Unfortunately, patient was lost for further follow-up.

DISCUSSION

Infections caused by protozoan Leishmania represent a wide range of diseases, including CL, mucocutaneous leishmaniasis (ML), and visceral leishmaniasis (VL). Incidence of infections caused by Leishmania in HIV-infected patients is increasing throughout the world. Although VL is the most frequent form of Leishmania infection seen in HIV-infected patients, CL and HIV coinfections are also becoming frequent in these individuals.

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Figure 1. Papulo-nodular lesions on the upper (A) and lower extremities (B) in an HIV-infected patient suffering from DCL.
DCL as first manifestation of HIV infection

Almost all treatment, and show frequent realapses. Weinstock and others, which seldom ulcerate, respond poorly to standard antimonial spread non-tender papulo-nodular skin lesions full of parasites, fuse to involve extensive areas of skin, resulting in a condition immune-suppressed patients, lesions of CL may become diff-

Several Leishmania species, including L. major, L. braziliensis braziliensis, and L. infantum, have been identified as causative agents of DCL in HIV-infected patients. Almost all patients with DCL and HIV coinfection are individuals who develop DCL as a secondary opportunistic infection after diagnosis of HIV infection. Our case was unusual for several reasons. First, our patient presented with progressive DCL of 7-months duration without typical clinical signs of HIV infection. Second, to best of our knowledge, this is the first case of DCL caused by L. tropica in an HIV-infected patient. Third, in this patient, there was no visceralization or mucosal spread of L. tropica infection, despite low CD4 + T cell counts. This is surprising given that L. tropica has been shown to visceralize even in immunocompetent individuals. Fourth, this patient did not respond to SSG, which may be because of drug resistance or very low CD4 + counts. Finally, our patient presented with intensely pruritic skin lesions, which have never been reported in DCL caused by other species of Leishmania. Collectively, our case illustrates that L. tropica can cause intensely pruritic, diffuse CL without involvement of mucous membranes and systemic organs in HIV-infected patients and that DCL could be the first clinical indicator of HIV infection. Therefore, we propose that all patients presenting with DCL should be screened for HIV infection.

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REFERENCES


50bp

Figure 2. The restriction analysis pattern of 1 PCR product digested with HaeIII. Lane M = DNA ladder (50 bp). Lane 1 = DNA from skin biopsy of the patient. Lane 2 = DNA from L. tropica WR664 (MHOM/SU/74/K27; positive control). Lane 3 = DNA from a skin biopsy of a healthy person (negative control).