Diffuse Cutaneous Leishmaniasis in an HIV Sero-positive patient – A Diagnostic Dilemma

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INTRODUCTION
Protozoan parasite belonging to the genus leishmania and is transmitted by the bite of a female sandfly [1]. Human leishmaniasis is usually classified as visceral, localized or diffuse cutaneous, and muco-cutaneous [2]. Cutaneous leishmaniasis can become disseminated (diffuse) in HIV-positive patients and is clinically characterized by multiple (>200), atypical, widespread infiltrations of skin lesions; and it weakens response to classic treatment [3]. Clinical diagnosis is confirmed by demonstrating amastigotes in Giemsa stained cytological smear and skin biopsy. We report an HIV positive patient with the unusual presentation of diffuse cutaneous leishmaniasis diagnosed on fine needle aspiration cytology in a non-endemic area.

CASE PRESENTATION:
A 45-year-old male diagnosed to be HIV positive for the past 5 years, presented with an asymptomatic skin-colored eruption on his face, neck and extensor aspect of left forearm. There was no history of prior trauma, ulceration, systemic symptoms, or travel outside Karnataka.

Cutaneous examination revealed multiple discrete, skin-colored, non-tender nodules on the extensor aspect of left forearm (Figure 1). The palms, soles and mucosal surfaces were unaffected. He had no systemic complaints and physical examination revealed neither pulmonary abnormalities nor organomegaly.

His complete blood count, urinalysis, hepatic and renal function tests, chest X-ray, ultrasound of abdomen, and lipid profile were within normal limits. CD4 T cell count was 250cells/mm³.

Biopsy from the skin nodule was done which was inconclusive, showing sheet of macrophages, with chronic inflammatory cell infiltrate (Figure 2). So fine needle aspiration was advised. Fine needle aspiration of
skin nodules over the extensor surface of forearm showed non-caseating granulomas (Figure 3) with presence of numerous macrophages harboring leishmania amastigotes (Lieshman-Donovan bodies) (Figure 4). Repeat biopsy of the skin nodule was advised and histopathological examination revealed many macrophages harboring leishmania amastigotes (Figure 5). A polymerase-chain-reaction (PCR) assay performed on a second biopsy specimen was positive for leishmania.

So based on clinical, cytological, histopathological features and polymerase-chain-reaction a diagnosis of cutaneous leishmaniasis was confirmed.

Figure 1. Clinical photograph showing diffuse skin lesions over forearm.

Figure 2. Histopathology showing macrophages with chronic inflammatory cells.

Figure 3. Fine needle aspiration showing non caseating granulomas with macrophages harboring leishmania amastigotes.

Figure 4. Fine needle aspiration showing a macrophage harboring leishmania amastigotes.

Figure 5. Histopathology showing macrophages harboring leishmania amastigotes.
DISCUSSION

Fine needle aspiration though a blind procedure; it has been a well established tool for the diagnosis of skin lesions. The skin lesion aspirates contain most of the information necessary not only for the diagnosis of cutaneous leishmaniasis but also for other inflammatory and neoplastic processes [5].

Fine needle aspiration is easier, less painful and more cost effective than the previous conventional scraping method/biopsy followed by histopathology. The diagnostic accuracy of skin aspiration, however, is dependent on the skill and experience of the personnel who perform the aspiration, prepare the slides and interpret the results. Since no local anesthesia is required, the process can be done in an outpatient setting as well [6].

The newer serologic techniques, such as ELISA (enzyme-linked immunosorbent assay), IFAT (indirect immunofluorescent antibody test), and others, are largely research tools with greatest use in seroepidemiological surveys [7].

Leishmaniasis is one of the top five diseases targeted by the WHO Special Program for Research and Training in Tropical Diseases. About 1.5 million new cases are documented each year, and over 350 million people live in areas of active parasite transmission[1].

Clinical variants of leishmaniasis (cutaneous, mucosal, and visceral) and clinical status (subclinical, self healing, disseminated, death) are largely determined by parasite species and host cell mediated immunity (CMI) response. Several arguments suggest that dissemination of lesions is more determined by host immunosuppression [IL-10 more than INF-Y] than by the virulence of the species involved. The species involved in diffuse CL are Leishmania braziliensis, Leishmania amazonensis, and Leishmania aethiopica. In India, Leishmania donovani has been reported in post-kala azar dermal leishmaniasis (PKDL) associated with HIV. Leishmania pifanoi and Leishmania infantum have been found in CL with HIV co-infection[2].

Amastigotes represent the host form of leishmaniasis. To ensure that the visualized structures are amastigotes rather than other organisms (Histoplasma spp), one should look for characteristic size (2-4µm in diameter), shape (round or oval), and internal organelles (the nucleus and the kinetoplast). Other disorders mimicking cutaneous leishmaniasis include traumatic ulcerative lesions, foreign body reactions, infected insect bites, impetigo, fungal and mycobacterial infections, sarcoidosis and neoplasms [6].

Other lesions such as sporotrichosis and bacterial ulcer, both of which are frequent with in regions where leishmaniasis is endemic, may mimic the presentation of cutaneous leishmaniasis. Artifacts from the staining method that mimic L.D. bodies include air bubbles, nuclear debris and stain particles [8].

Confirmation of infection is also important because treatment is expensive, toxic, and difficult to administer. Lesions may re-activate and produce the progressive and disfiguring mucosal form if not adequately treated [6].

CONCLUSION

Diffuse Cutaneous Leishmaniasis should be suspected in cases of HIV and fine needle aspiration should be advised for early diagnosis and treatment.

REFERENCES


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