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Case Report

A case report of cutaneous leishmaniasis

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ABSTRACT

Leishmaniasis is a vector-borne parasitic disease caused by more than 25 species of the hemoflagellate Leishmania. It is transmitted through the bite of an infected sand fly and is widespread in India and around the world. Here, we present a case of a 45-year-old male with cutaneous leishmaniasis in the form of erythematous papules over the abdomen, dorsum of the right hand, and right knee.

1. Introduction

Tropical infections caused by Leishmania spp. can cause diagnostic difficulties for dermatologists. With typical leishmaniasis symptoms in endemic regions, the clinical diagnosis is not challenging. However, in non-endemic countries where cutaneous leishmaniasis (CL) is uncommon, such as India, it can easily be missed. Mycobacterial and deep fungal infections are common differentials when considering a cutaneous lesion with a possible infectious cause. In a country like ours, where tuberculosis is more prevalent, cutaneous leishmaniasis is very likely to be misdiagnosed as cutaneous tuberculosis, especially lupus vulgaris. Cutaneous leishmaniasis predominates in adults and manifests as papules that progress into nodules and ulcers if left untreated.

2. Case Report

A 45-year-old male patient, a farmer by occupation with frequent outdoor activities, presented with multiple lesions that began as erythematous papules over the abdomen, dorsum of the right hand, and right knee for 2 months. All lesions resolved following electrosurgery except one in right knee lesion slowly progressive associated with pruritus and extending borders and currently secondarily infected associated with pain for 1 months. There is no history of trauma prior to the onset of the lesion or at the site of the lesion. On examination, a single hyperpigmented crusted plaque over the right knee measured approximately 9x 7 cm in size. The ulcer floor was moist, smooth, shiny, and filled with serous discharge, and a well-defined raised erythematous margin was present. The systemic examination of the patient revealed no abnormalities. Three years prior, he had cutaneous tuberculosis, for which he received 11 months of antituberculosis treatment. The laboratory investigations (hemogram, renal function tests, liver function tests, and lipid profile) were within the normal limits. ELISA for HIV testing was negative. A skin punch biopsy of the lesion was performed and sent for histopathological examination, with cutaneous leishmaniasis and cutaneous tuberculosis as differential diagnoses. Microscopic examination showed epithelioid cell granulomas containing lymphocytes, histiocytes, and plasma cells. Occasional histiocytes, within these granulomas show intracytoplasmic hematoxylinophilic organisms, 2–4 μm peripherally located oval-shaped

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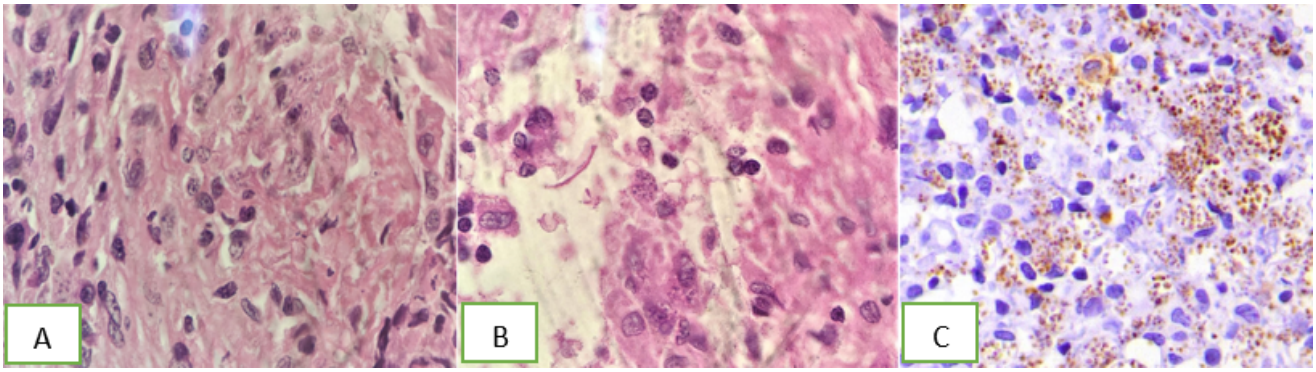

Figure 1: Shows a dermal infiltrate predominantly composed of macrophages; most of them areamastigotes (2–4 μm peripherally located oval-shaped organisms), Leishman Donovan bodies (**A** and **B**, H&E x400), and IHC shows varying numbers of organisms stained with the anti-CD1a antibody (**C**).

organisms, suggestive of Leishman Donovan bodies (Figure 1A&B) and Immunohistochemistry (IHC) showed varying numbers of organisms stained with the anti-CD1a antibody (Figure 1C). Tissue culture and polymerase chain reaction (PCR) confirmed Leishmania donovani as the infecting agent. Patient was treated with intravenous liposomal amphotericin B, which was given intravenously once a day for six days. During the course of treatment, the patient did not experience any fever, or other side effects. The erythema went away three weeks after starting treatment, and the lesion healed.

3. Discussion

Leishmaniasis was named after W.B. Leishman, who discovered organisms in smears taken from the spleen of a patient who had died of dumdum fever in 1901.[1] Leishmania are dimorphic parasites. In the gut of the sandfly or in culture, they exist in promastigote form (10 to 20 μm, spindle-shaped and motile, with a single flagellum). Leishmania exist as amastigote (2-6 μm, round or oval, nonmotile, with a rod-shaped kinetoplast) in the cells of the host reticuloendothelial system.[2] While the infection is a zoonosis in nature, humans are only incidental hosts. The disease is spread by the bites of female sandflies. The organism has a flagellate phase (promastigote) while living inside the vector and a phase when the flagellum is retracted (amastigote), which is the stage found in human infection. The parasite species and the immunological response of the host have a significant impact on clinical symptoms. There are three main types of manly leishmaniasis. In the cutaneous form, erythematous papules first appear at the sandfly bite site. Over the course of a few weeks, these papules grow into nodules and plaques that frequently ulcerate and become crusted. The mucocutaneous form develops when parasites spread from the skin to cause oropharyngeal lesions. Visceral form (kala-azar) is a systemic reticuloendothelial disease characterized by fever, wasting, hepatosplenomegaly, and leukopenia.[3] A conclusive diagnosis is made using smears, cultures, PCR, and histological evaluation of suspected specimens.[4–9]

Histopathological findings show that the epidermis is either ulcerated with secondary changes or it is unremarkable. The superficial dermis shows a dense inflammatory infiltrate predominantly composed of histiocytes, lymphocytes, and plasma cells. In the early stages of leishmaniasis, numerous amastigotes typically distend histiocytes and impart quite a characteristic diagnostic morphology.[4] Weigert iron hematoxylin may stain parasites better than H&E or Giemsa. Immunohistochemistry using the CD1a antibody is more sensitive than H&E. Anti-CD1a, a monoclonal antibody used to stain individual amastigotes.[10]

Histoplasmosis is a pathological differential diagnosis in which the organisms are identical in size to leishmania but are capsulated, exhibit narrow-based budding and have a peri-organism halo devoid of kinetoplasts. Sarcoidosis and other granulomatous diseases may be similar to the late granulomatous stages of leishmaniasis. PCR-based methods may aid in the diagnosis when haematoxylin and eosin (H&E) and Giemsa stains fail to reveal the organisms. It is essential to detect the infection as soon as possible to begin the appropriate treatment and to avoid inadvertent drug use. In the treatment of cutaneous leishmaniasis, antimonial are the first-line medication.[4] Lesions can also be treated with local excision, curettage, or electrocautery, but these procedures probably increase the likelihood of recurrence.[11]

4. Conclusion

For a prompt diagnosis, disease awareness and recognition of various clinical presentations are essential. Cutaneous leishmania may present in unusual forms, any nonhealing chronic lesion should be evaluated for leishmaniasis, especially in endemic region. When evaluating lesions that might have been caused by an infectious agent, cutaneous

leishmaniasis should always be taken into consideration. On the basis of histomorphology and immunohistochemistry, a definitive diagnosis can be made and the right treatment can be started.

5. Source of Funding

None.

6. Conflict of Interest

None.

References

1. Cox FE. History of human parasitology. *Clin Microbiol Rev*. 2002;15(4):595–612.
2. Ghosen S, Kurban AK. Leishmaniasis and other protozoan infections. In: Fizpatrick's Dermatology in General Medicine. 7th edn. New York: McGraw-Hill; 2008. p. 2001–6.
3. Valia RG. IAVL Textbook and atlas of Dermatology 2nd edn. Mumbai: Bhalani Publishing House; 2001. p. 395.
4. Rahman H, Razzak MA, Chanda BC, Bhaskar KR, Mondal D. Cutaneous leishmaniasis in an immigrant Saudi worker: a case report. *J Health Popul Nutr*. 2014;32(2):272–6.
5. De Vries H, Reedijik SH, Schallig HD. Cutaneous leishmaniasis: recent developments in diagnosis and management. *Am J Clin Dermatol*. 2015;16(2):99–109.
6. Rajpar GM, Khan MA, Hafiz A. Laboratory investigation of cutaneous leishmaniasis in Karachi. *J Pak Med Assoc*. 1983;33:248–50.
7. Khan SJ, Muneeb S. Cutaneous leishmaniasis in Pakistan. *Dermatol Online J*. 2005;11(1):4.
8. Kubba R, Al-Gindan Y, El-Hassan AM, Omer AH. Clinical diagnosis of cutaneous leishmaniasis (oriental sore). *J Am Acad Derm*. 1987;16(6):1183–9.
9. Okumura Y, Yamauchi A, Nagano I, Itoh M, Hagiwara K, Takahashi K, et al. A case of mucocutaneous leishmaniasis diagnosed by serology. *J Dermatol*. 2014;41(8):739–42.
10. Ferrufino-Schmidt MC, Bravo F, Valencia BM, Llanos-Cuentas A, Boggild AK, Leboit PE, et al. Is CD1a useful for leishmaniasis diagnosis in the New World? *J Cutan Pathol*. 2019;46(1):90–2.
11. Hepburn NC. Cutaneous leishmaniasis: an overview. *J Postgrad Med*. 2003;49(1):50–4.

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