CASE REPORT

Cutaneous Leishmaniasis presenting as generalized multinodular eruption

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ABSTRACT

Human Leishmaniasis has a wide spectrum of clinical presentations. It is caused by infection with intracellular flagellate protozoan parasites belonging to the genus Leishmania. Leishmaniasis is a vector-borne disease transmitted by bite of infected female sandfly. At least 20 species of Leishmania are known to cause human infection and produce distinct clinical forms. Cutaneous leishmaniasis is the most common form of leishmaniasis caused by flagellate protozoa of the genus Leishmania transmitted by sand fly bites. Old World leishmaniasis is endemic in the Mediterranean Sea and the neighbouring countries. *L. tropica, L. major, L. aethiopia* species are responsible for this form of leishmaniasis. I report a case of a 33-year-old-male with a cutaneous leishmaniasis presenting with multiple scaly erythematous plaques on right upper limb, back and both lower limbs with superimposed erosions and crust. Histopathological examinations showed diffuse dermal infiltration consisting of lymphocytes, histiocytes, eosinophil leukocytes and plasma cells. In most, macrophages amastigotes were seen. Because of higher rate of travel and employment abroad increased number of sporadic cases of cutaneous leishmaniasis in non-endemic areas are being reported.

CASE REPORT

A 33-year-old migrant worker presented with a multiple, painless, large, reddish lesions scattered over the arm, the trunk and both lower limbs for more than 10 months [Fig. 1]. The disease had a gradual onset and slowly progressive course, the lesions started as a small red pea-sized asymptomatic papule probably caused by an insect bite. It reached its present size in approximately 7 months, and subsequently, showed very slow spread with no sign of regression. Recurrent superficial erosions, crusting, and pus discharge were reported. The ulcers failed to heal, despite several course of systemic antibiotics. There was

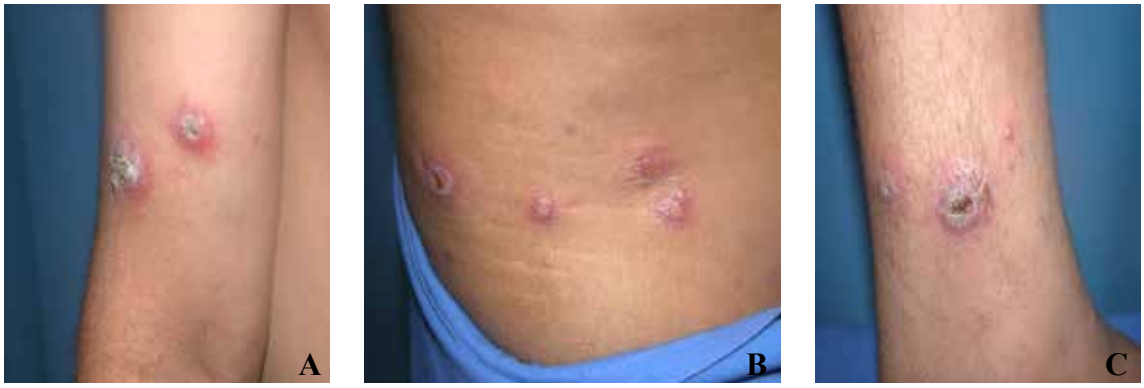

Fig. 1 Multiple scaly erythematous nodules and plaques in right arm (A), trunk (B) and left leg (c).

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no previous history of similar lesions or other skin problems. The patient did not complain of any systemic illness and there was no past medical or drug history of note. No family history of the same condition.

Clinical examination revealed multiple cutaneous, erythematous, infiltrated nodules and plaques of different sizes; the largest was 2 cm in diameter, distributed mainly on the right upper extremities, trunk and both lower extremities. Considering the clinical differential diagnosis of chronic pyoderma gangrenosum, cutaneous leishmaniasis, atypical mycobacterial infection, and deep mycosis, skin biopsy was taken. Patients' routine laboratory and radiological investigations including CBC, CPR, blood sugar, hepatic and renal profile revealed no abnormal findings. Serology for hepatitis B and C and human immunodeficiency virus were nonreactive. X-ray on both limbs showed no attachment of the lesion to underlying bones. Systemic examination was unremarkable.

A punch (5mm) skin biopsy was done from lesion in left leg and histopathological examinations showed hyperkeratosis and an epidermal atrophy. The dermis showed multiple granulomatous infiltrate scattered in upper and mid dermis in diffuse and nodular pattern. The infiltrate was formed mainly of histiocytes admixed with lymphocytes and plasma cells. Hematoxylin and eosin (H & E) section revealed numerous intracellular organisms which were confirmed by Giemsa stain to be Leishman bodies. Periodic Acid Schiff (PAS) stain for fungi and AFB stains for lepra bacilli and tuberculous bacilli were negative [Fig. 2 A & B]. A Clinicopathological correlation was consistent with a diagnosis of cutaneous leishmaniasis.

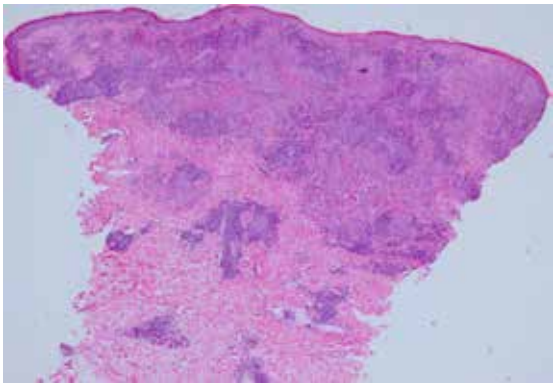

Fig. 2 A Upper and mid dermal granulomatous infiltrate arranged in nodular and diffuse pattern. The overlying epidermis shows hyperkeratosis and atrophy.

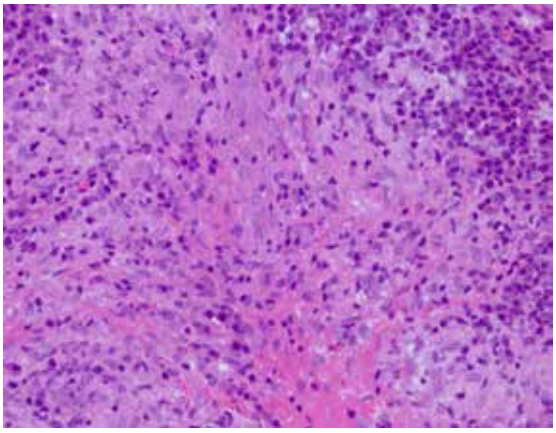

Fig. 2 B The granulomatous infiltrate formed of lymphohistiocytic admixed with plasma cells. Leishman bodies are observed within histiocytes.

FINAL DIAGNOSIS

- Cutaneous leishmaniasis

DISCUSSION

Leishmaniasis is prevalent in approximately 88 countries in the world, with old world disease more prevalent than new world disease.[1] Incubation period for CL is 2–8 weeks. The disease begins as a small erythematous papule at the site of the bite of sandfly. It slowly increases in size up to 2 cm or more and may become a nodule. Central crusting and shallow ulcers develop with

raised border.[2] Lesion persists for few months to a year and gradually heals leaving a slightly depressed scar. *L. tropica* generally produces chronic lesions or treatment resistant lesions.[3] Diagnosis of leishmaniasis can be made by tissue smear which may show organisms as amastigotes on Giemsa stain. Culture on Novy-McNeal-Nicole medium or PCR may help in species identification.[2,3,8] Biopsy from lesion shows diffuse dermal infiltrate composed of varying proportion of histiocytes, lymphocytes, plasma cells, and neutrophils. Amastigotes may be found within dermal macrophages on H & E stain which can be confirmed with Giemsa stain.[6] Immunological diagnosis with enzyme-linked immunosorbent assay (ELISA), Leishman skin tests are not very useful or easily available.[7]

This case showed an unusual presentation of multiple chronic CL on both extremities and trunk reaching a huge size in some lesions. In contrast to self-resolution seen in many cases of CL, this case did not show any sign of resolution over 10 months. As CL can present in a lot of unusual forms, any non-healing chronic lesion even on unexposed body parts should be investigated for leishmaniasis, particularly in endemic areas.

Treatment of CL is often difficult. Multiple treatment options are used throughout the world for cutaneous disease. Antimonials are still the first-line drug in the treatment of CL. Sodium stibogluconate (Pentostam) and meglumine antimoniate glucantime are essentially similar drugs which contain pentavalent antimony (Sb). Sodium stibogluconate can be administered intravenously or intramuscularly while meglumine antimonite should only be given via the intramuscular route. The recommended dose is 20 mg/kg/day for 20-28 days. Dapsone and allopurinol have also been used for the treatment of CL with some success. The mechanism is unclear, although basic biomedical studies have shown that Leishmania cannot make all of their own nucleic acids and, thus, it uses the host's purine through the purine salvage pathway.[8] Besides oral and parenteral medications, local cryotherapy, intralesional infiltration of sodium stibogluconate, local heat therapy, and various topical paromomycin preparations are in practice for many years.

Vaccines for prophylaxis and immunotherapy have been developed and are currently undergoing trials in many countries. The development of molecular biology techniques is also improving knowledge on the structure, evolution, and expression of the Leishmania genome, and the study and definition of the mechanisms that regulate the parasite's biochemical and molecular features will certainly contribute to the development of new and more effective strategies for leishmaniasis treatment.[9]

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