ISSN- O: 2458 - 868X, ISSN–P: 2458 – 8687
Index Copernicus Value: 49. 23
PubMed - National Library of Medicine - ID: 101731606

*International Journal of Medical Science and Innovative Research (IJMSIR)*
*IJMSIR : A Medical Publication Hub*
*Available Online at: www.ijmsir.com*
*Volume – 3, Issue – 5, October - 2018, Page No. : 205 - 208*

# Disseminated cutaneous Leishmaniasis in a patient who is HIV positive with a flare up as iris -a case report

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**Type of Publication:** Case Report

**Conflicts of Interest:** Nil

**Abstract**

There is a broad spectrum of clinical manifestations of cutaneous leishmaniasis. The usual lesion is a small, red papule which over several weeks becomes darker, crusts in the centre and eventually ulcerates & heals, leaving a raised border. A rare form is the disseminated (diffuse) cutaneous leishmaniasis which can occur among immunosuppressed persons where lesions begin with an initial primary lesion and then disseminates to involve other areas of the skin. The lesions are non ulcerative nodules full of parasites, and are often scattered over the limbs, buttocks, and face. The disease does not involve internal organs.

32 yrs old male presented with multiple swellings in different parts of body with itching. O/E multiple plaques, sub cutaneous nodules and molluscum like lesions were present over extremities and trunk. Biopsy from multiple sites was similar and suggestive of Leishmaniasis. Treated with sodium stibogluconate and lesions responded. 5 months after ART he presented with generalised plaques and oedematous red lesions resembling photo dermatitis. Repeat skin biopsy showed same picture. Treated with sodium stibogluconate again and for past 4yrs he is asymptomatic.

Cutaneous leishmaniasis is caused by different species of genus Leishmania, a protozoon that is transmitted by sand-flies. The result of infection varies from a cutaneous ulcer, to erosive mucosal disease with severe facial disfigurement, to a life threatening systemic infection, depending upon the interaction between Leishmania and the genetic and immunological status of the host. Leishmaniasis and HIV co-infection may intensify the immune defect and is the chief reason for atypical presentation and widespread progression of cutaneous leishmaniasis and its defiance to conventional therapy. IRIS whether paradoxical worsening or unmasking form is very common event after initiation of HAART, especially if CD4 is very low. Paradoxical worsening of diffuse cutaneous Leishmaniasis is still very uncommon.

**Keywords :** Diffuse cutaneous leishmaniasis, HIV infection , Co-infection, IRIS

## Introduction

Leishmaniasis is a protozoal disease caused by Leishmania species. Disease is widely distributed, but majority of cases are from India, Brazil and Sudan[1]. There are many species

of Leishmania that affects human; in India Leishmania Donovani is the prevalent species[2]. The vector transmitting the disease is Phlebotomus species and the

types of species vary from region to region. Motile flagellar forms of Leishmania called as promastigotes are inoculated into skin during blood meal of sand fly. Polymorphs,Macrophages & Dendritic cells phagocytise the organism. Inside phagocytes organism becomes aflagellate, called amastigotes, and it divides by binary fission. They are released from cell by cell rupture and taken by similar cells and further spread takes place via lymphatics[2]. A papulo nodular lesion develops at site of bite & there is regional adenopathy.Satellite lesions develop due to local spread via lymphatics. In diffuse cutaneous leishmaniasis lesions are multiple, disseminated, non ulcerative, nodular, plaques or keloid forms[1]. Internal organs are not involved. Parasite load will be very high. When there is leishmaniasis and HIV co-infection there may be atypical presentation, poor response to therapy, higher incidence of drug toxicity and frequent relapses after treatment[1, 2]. The treatment is same as for immune competent person.

IRIS is a common cause of death and even more common cause of morbidity in patients started on HAART[3]. There are two scenarios; paradoxical symptomatic relapse of a prior infection despite successful microbiologic treatment and unmasking of occult opportunistic infections. Potential mechanisms for the syndrome include a partial recovery of the immune system or exuberant host immunological responses to antigenic stimuli. There is increase in $CD4^+$ T-lymphocyte count and decrease in plasma HIV viral load. Infections, inflammations, auto immune disorders, malignancy etc can develop as IRIS.

**Case report**

32 yrs old male presented with multiple papular and nodular swellings in different parts of body with itching of 2 yrs duration. There was no history of pain. He had persistent fever also and while evaluating, he was found HIV positive, but didn't want a treatment at that time. He was referred to our tertiary care centre for management of HIV infection and skin lesions 2 yrs later when symptoms increased.

Patient was a lorry driver by profession who used to travel to different states of India and had high risk behaviour. O/E multiple erythematous to violaceous infiltrative plaques of size1x1cm & 2x2 cm on forehead, cheek, nose, right ear lobe, shoulder and back.(fig 2,3) Multiple umbilicated dome shaped papules with central excoriation were present over upper and lower extremities.(fig 1). Multiple lymph nodes over cervical, axillary and inguinal region were noted. There was no hepato splenomegaly. Possibility of cutaneous fungal infection like sporotrichosis was thought and a biopsy was performed from multiple sites.

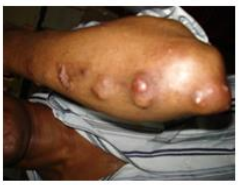

Fig 1

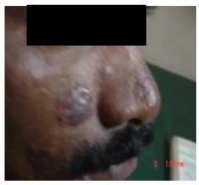

Fig 2

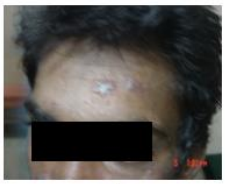

Fig 3

Other lab evaluations were as follows

CBC – Normal. ESR -86mm/hr

LFT – AG reversal with normal liver enzymes

RFT – Normal, RBS and Serum Electrolytes –Normal, VDRL- non reactive.

HBs Ag Negative

Chest X-ray –normal, USG Abdomen – No hepato splenomegaly or intra abdominal node enlargement.

CD4 – 33.

HIV Viral load was not done. Meanwhile biopsy results from multiple sites came as skin with dermal infiltrate showing sheets of macrophages containing amastigotes of Leishmania.(fig 4,5) FNAC of lymph node showed reactive changes.

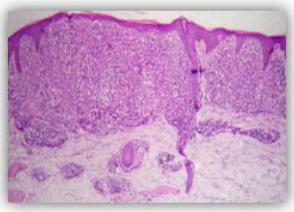

Fig 4

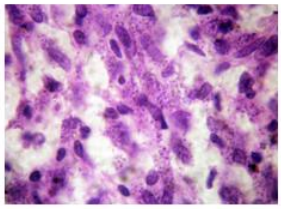

Fig 5

Patient was started on Fluconazole 200 mg daily, but stopped later as there was no response. Amphoterecin B deoxycholate was started at a dose of 0.75 mg/kg/day, On day 5 the renal parameters worsened and hence Amphoterecin was stopped. When creatinin improved Sodium stibogluconate was started, continued for a month, closely monitoring cardiac and renal function. Local infiltration was also given to some huge plaques present over knee. Patient responded and all lesions diminished in size. After 1 month ART- ZLN was started.

5 months after initiating ART patient presented with pruritic papules and plaques over whole body.(fig 6,7,8). Photo dermatitis was considered and managed as. But patient became more symptomatic and a repeat biopsy was done. Biopsy again showed amastigotes inside macrophages. Sodium stibogluconate was started again and continued for another month. Patient responded very well and kept under follow up. There was no relapse even after 4 years. His HIV infection is well controlled.

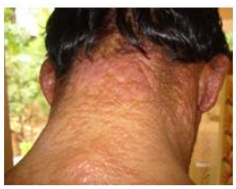

Fig 6

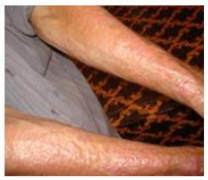

Fig 7

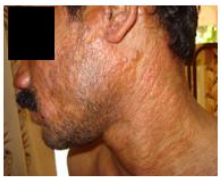

Fig 8

**Discussion**

*Leishmania* species can cause wide spectrum of cutaneous lesions in HIV positive patients. Diffuse cutaneous leishmaniasis is the result of failure of cell mediated immune response on the part of the host and toxigenicity of the parasite. Coexistence of HIV and Leishmania exert cumulative deficiency of cellular immune response[4,5]. I/V drug users are at increased risk. According to WHO 95% of co-infected cases are visceral Leishmaniasis and 5% are cutaneous diseases. CD4 count is less than 200.In immune competent patients lesion may heal spontaneously, but, compromised patients should be treated always because tissue destruction may happen. Several options are available like pentavalent antimonals, Miltifosine, Azoles, heat therapy or cryotherapy[5]. Species identification is helpful for selecting correct drug. IRIS is a dysregulated immune response associated with immune reconstitution after initiating ART. This can be infective or non infective. Tuberculosis, Toxoplasmosis, candidiasis, cryptococcal meningitis, Herpes Zoster etc are common IRIS encountered. Flare up of cutaneous Leishmaniasis is very rarely reported. The treating physician has to be alert after initiating ART regarding occurrence of IRIS especially when CD4 is low. Also the patients should be made aware regarding such consequences; otherwise this may be considered as an adverse reaction to ART and may lead to unnecessary cessation of drug intake.

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