# A rare presentation of visceral leishmaniasis with diffuse cutaneous involvement mimicking lepromatous leprosy

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## Abstract

A 61 year old male, presented with generalized skin coloured asymptomatic dermal nodules for 5 weeks. He had a history of hypertension, right sided cerebrovascular accident and diabetes mellitus (DM).

He had hepatomegaly but no lymphadenopathy, hypopigmented patches or thickened peripheral nerves.

Two differential diagnoses considered were lepromatous leprosy (LL) and diffuse cutaneous leishmaniasis (DCL).

Slit skin smear and skin biopsy didn't reveal any evidence of leprosy which excluded LL. Though amastigotes were not seen in light microscopy, culture and PCR of both skin and bone marrow were positive for Leishmania, suggesting visceral leishmaniasis (VL).

He was started on IV sodium stiboglunate 20 mg/kg/day. After one week worsening of thrombocytopenia led to intra cerebral haemorrhage to which he succumbed.

The immuno-suppression by the disease itself and the diabetes mellitus could have led to haematogenous spread to skin resulting in his current presentation. However post kala-azar dermal leishmaniasis is also a possibility.


## Case report

A 61 year old male from Western province presented with widespread skin nodules for 5 weeks duration. These painless, skin colored nodules have initially appeared on face, and subsequently spread rapidly in a cephalo-caudal manner. He denied having a contact with a person with leprosy. There was no history of foreign travel. He did not have long standing low grade fever, night sweats, significant loss of weight or loss of appetite. Patient was also on treatment for hypertension and left sided ischaemic stroke. He was recently diagnosed with diabetes mellitus (DM).

Examination of the skin revealed generalized, non-tender multiple skin coloured and erythematous dermal nodules without any ulceration (Figure 1, 2 and 3). There were no hypopigmented patches or thickened peripheral nerves.

On systemic examination he was found to have hepatomegaly. There was no clinically detectable splenomegaly or lymphadenopathy.

The differential diagnoses considered were lepromatous leprosy (LL), diffuse cutaneous leishmaniasis (DCL), visceral leishmaniasis (VL) and a haematological malignancy.

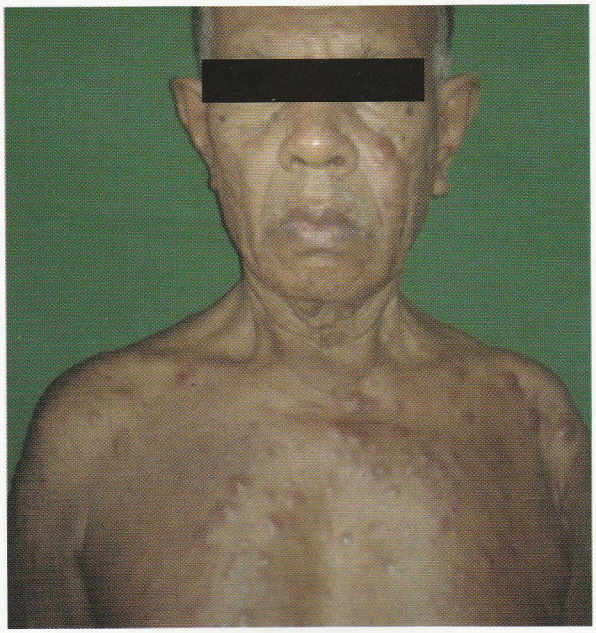

**Figure 1.**

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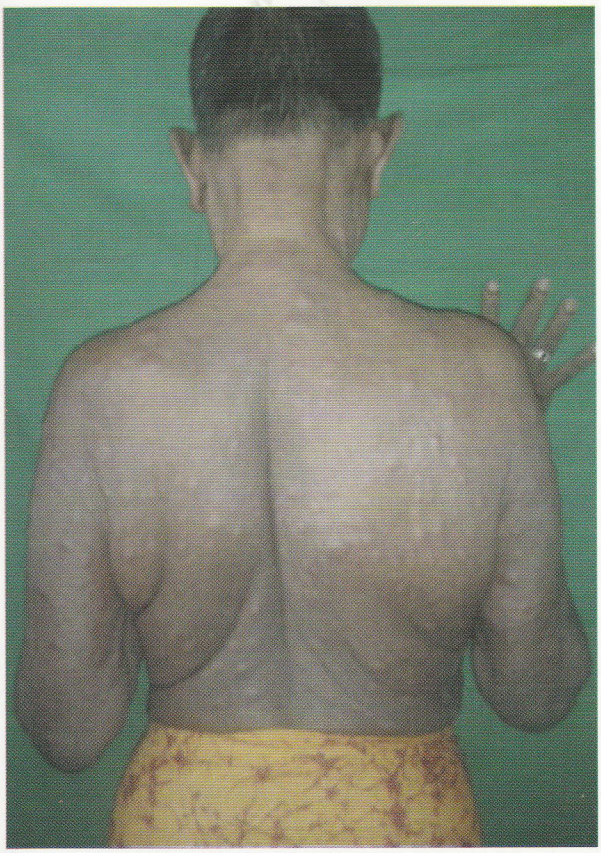

**Figure 2.**

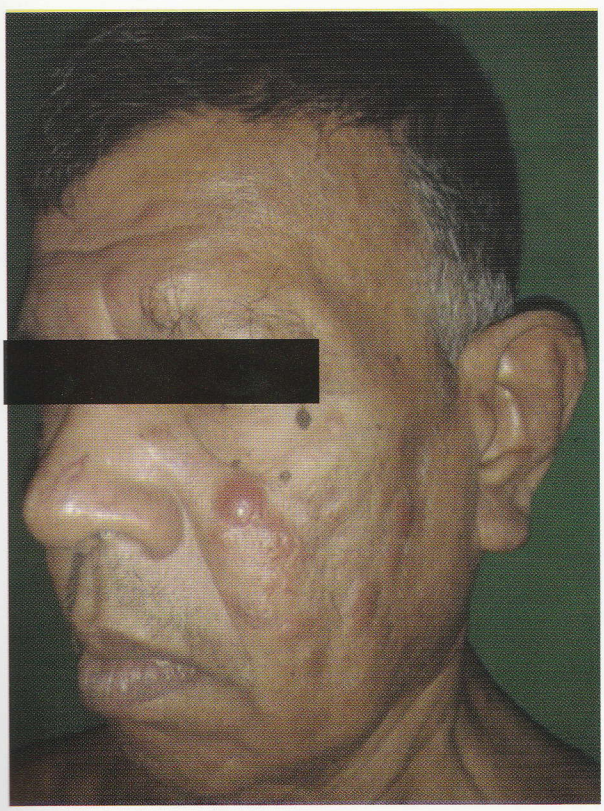

**Figure 3.**

**Laboratory investigations**

FBC showed neutrophil leukocytosis with WBC - $71\times10^9$ /L (N -70%, L- 20%, E -10%), haemoglobin - 8 g/dl, platelet - $100\times10^9$/L and blood picture revealed neutrophil leukocytosis, normochromic normocytic anaemia with mild thrombocytopenia. FBS-25.9 mmol/dl, ESR -25 mm, CRP - 0.5 mg/dl. HIV screening was negative, liver profile and renal function tests were normal. LDH level was 580U/l. There was mild hepatomegaly with a normal echo pattern and a mild splenomegaly in USS of abdomen and pelvis. Slit skin smear (× 4) and skin biopsy were negative for leprosy.

Though clinical features were suggestive of LL, repeatedly negative SSS and skin biopsy made the diagnosis of leprosy unlikely.

Histology of skin biopsy showed lympho-plasmacytic infiltrate without granuloma formation. Neither the skin histology nor slit skin smear did show Leishmania amastigotes. But culture and PCR from the skin biopsy became positive for leishmaniasis.

Histological examination of the bone marrow showed hyperplastic myelopoiesis with mildly suppressed erythropoiesis and megakaryopoiesis with normal cellular morphology. Immune stains also did not reveal any abnormal cells. Bone marrow aspirate for culture and PCR became positive for leishmaniasis.

Presence of bone marrow involvement and absence of parasites in the skin smears made the possibility of DCL unlikely.

Within two weeks of initial evaluation after hospital admission the WBC became normal, but his anaemia and thrombocytopenia persisted.

Patient was started on IV sodium stibogluconate (SSG) 20 mg/kg/day. After one week of SSG platelet count further dropped to $50\times10^9$/L, and his SSG treatment was stopped. Two days later he developed worsening of his right sided weakness, and platelet count had reduced to $12\times10^9$/L. CT scan of brain revealed an intra-cerebral and sub arachnoid haemorrhage.

Inspite of platelet transfusions and a good control of blood pressure, he further deteriorated and succumbed to his illness. Due to unavoidable reasons pathological postmortem was not done.

## Discussion

Leishmaniasis is a spectrum of chronic infections caused by species of *Leishmania* . There are four major clinical patterns: cutaneous, mucocutaneous, diffuse cutaneous and visceral leishmaniasis.

In Sri Lanka human leishmaniasis is caused by *Leishmania donovani*, which is the species more widely known to cause VL.

Clinical categorization of leishmaniasis in this patient was challenging. Possibilities include Diffuse Cutaneous Leishmanaiasis (DCL), Post Kala Azar Dermal Leishmaniasis (PKDL) and Disseminated Leishmaniasis (DL).

DCL is less likely due to the presence of visceral involvement and negative skin microscopy. PKDL following subclinical VL is another possibility. Disseminated leishmaniasis (DC) due to haematogenous spread from bone marrow to skin also needs to be considered Immuno-suppression due to disease itself and diabetes could have played a role in causing this.

The transient very high WBC count is not a previously documented finding with either VL or disseminated leishmaniasis.

Due to the presence of a potentially visceralizing parasite in Sri Lanka as the cause of cutaneous leismaniasis, this case report highlights the importance of proper assessment and follow up.

Another important message is that leishmaniasis should always be considered in the list of differential diagnosis in the presence of diffuse skin nodules resembling LL.

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