Wide-spread cutaneous leishmaniasis in a patient with reactional leprosy

G C Wickremaratna[1], G M P Sirimanna[2], C Udagedara[3]

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Abstract

A 70 year old farmer from North Central Province presented in August 2001 with wide spread multiple ulcerative plaques of 2 years duration. He has been on treatment for clinico-pathologically proven reactional Leprosy at Anuradhapura without a significant improvement.

Smears from several lesions including lesion over the nose were positive for Leishmania amastigotes. Histology of the nasal lesion demonstrated a dense lymphocyte infiltrate with granulomatous reaction .

Coexisting cutaneous leishmaniasis and leprosy has been reported before in the world literature. However this is the first case of coexisting cutaneous leishmaniasis and leprosy reported in Sri Lanka, and the fist patient to be treated successfully with systemic Sodium Stibogluconate therapy.

Introduction

Over the past several years increasing number of patients with cutaneous leishmaniasis have been encountered in dermatology clinics of Sri Lankan hospitals. Most of these patients have one or two lesions.

We describe a patient who developed widespread lesions of cutaneous leishmaniasis while on treatment for a type II lepra reaction.

Most of the cutaneous leishmaniasis lesions are self limiting or respond well to cryotherapy, but diffuse cutaneous leishmaniasis, mucosal leishmaniasis and recidivans leishmaniasis require systemic therapy.

Case report

A 70 year old farmer from North Central Province, Sri Lanka presented to the National Hospital Colombo in September 2001 with widespread ulcerative plaques of 2 year duration.

Since November 1999 he was on treatment for multi bacillary leprosy and since February 2001 treated for clinico-pathologically proven reactional leprosy, without a significant improvement. He did not have any other significant illnesses in the past.

On examination there were widespread multiple ulcerative plaques on the face, right forehead, bridge of the nose, angle of the mouth, nape of the neck, trunk, upper limbs, right leg and foot. He neither had mucosal lesions nor constitutional symptoms.

Skin lesions were non pruritic, non tender, had areas of skin hyperpigmentation and hypopigmentation with acquired leuconychia. There was a large 7 cm x 5 cm non healing ulcer with a raised edge on the right leg.

There was no lymphadenopathy or hepatosplenomegaly. Examinaton of the cardiovascular, respiratory, gastrointestinal systems were clinically normal.

Nervous system examination revealed thickened bilateral common peroneal nerves. L>R. There were no trophic ulcers, foot drop or objective sensory loss.

Investigations revealed the following; Hb 9.6g/dl WBC 5000 mm^3 (N60 L32 E8). Platelet count 226000/mm^3 ESR 52 mm/1st hour. Liver function tests and renal profile normal. FBS was 100 mg/dl. PPBS was 143 mg/dl and chest x-ray was normal. Ultra sound scan of the abdomen was normal. Mantoux test was negative. HIV test was negative. Lymphocyte subsets analyses showed normal CD4, CD8, Tcells, Bcells and Nkcells counts. Serum Complement C3 was within normal limits. Lymphocyte function tests revealed diminished lymphocyte function following stimulation with concanavalin (a mitogen).

Skin biopsy done in February 2001 showed collections of foamy histiocytes with intermingled lymphocytes in the dermis, several blood vessels were noted with prominent endothelial cells, surrounded by collections of fragmented neutrophils. This biopsy was consistent with lepromatous leprosy with an ENL reaction.

[1]*Senior Registrar in Dermatology, Dermatology Unit, National Hospital Colombo,* [2]*Consultant Dermatologist, Dermatology unit, National Hospital Colombo,* [3]*Consultant Dermatologist, General Hospital Anuradhapura.*

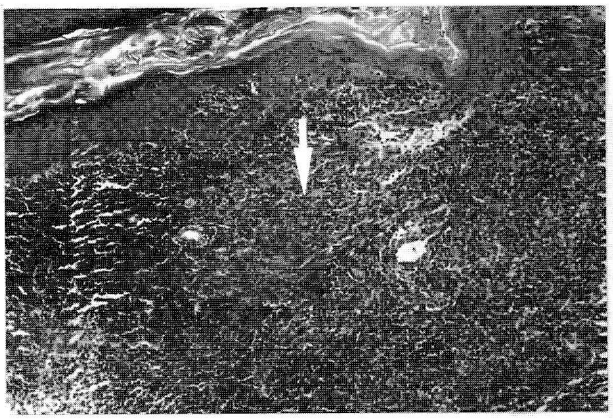

Figure 1. *Histology from the lesion over the nose done in October 2001 showed a dense lymphocytic infiltrate with granulomatous reaction. H & E × 100.*

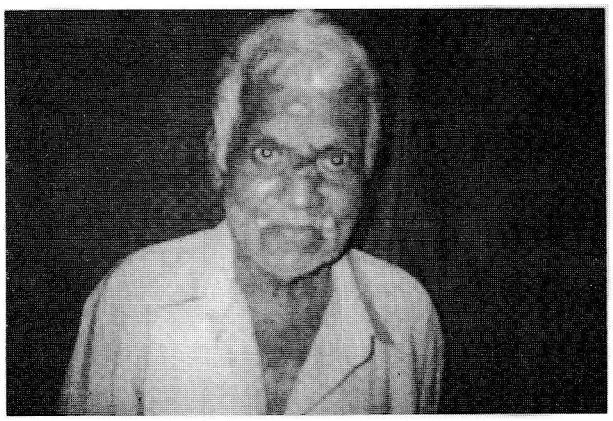

Figure 2. *Multiple ulcerative plaques on the face prior to treatment.*

Figure 3. *Post treatment, at the end of sodium stibogluconate injection course.*

Skin biopsy done in October 2001 showed a dense lymphocyte infiltrate with granulomatous reaction. No specific pathogenic organisms were seen on histology sections.

Skin slit smears were positive for Leishmania amastigotes. He had a negative Formol Gel Test (which is positive in visceral leishmaniasis). Leishmanin Test was not done due to non availability.

Management

Our patient's treatment for reactional leprosy was discontinued in December 2001. His widespread cutaneous leishmaniasis with non healing ulcers was treated with systemic Sodium Stibogluconate. Prior to treatment ECG, CXR, Liver function tests, Serum proteins were repeated. Sodium stibogluconate was given between 5th April 2002 to 14th May 2002 for a period of 20 days, every other day, deep IM (intra muscular) 4.5 ml/day (10 mg/kg b wt=450 mg, 100 mg in 1 ml=4.5 ml). The reported side effects of stibogluconate are nausea, vomiting diarrhoea, abdominal pain, substernal pain, cough, fever, jaundice etc. However our patient had an uneventful complete recovery.

Discussion

Our patient was initially treated at a clinic at Medawachchiya from North Central Province for multi bacillary leprosy (MB). He had continuation of leprosy treatment at Anuradhapura General Hospital. Since February 2001 he was treated for reactional leprosy with multi bacillary Leprosy therapy and corticosteroids.

At Anuradhapura diagnosis of leprosy was based on clinical and histological findings. Histology done in February 2001 was suggestive of leprosy along with thickened peripheral nerves and areas of skin hypopigmentation.

As patient was not responding to reactional leprosy treatment, following were considered. Exacerbation of leprosy and reactional leprosy; both these were unlikely as he was not ill, and did not exacerbate after cessation of reactional leprosy treatment, hence another pathology was considered.

Histology of the lesion over the nose done in October 2001 showed a dense lymphocyte infiltrate with a granulomatous reaction. Fungal studies were negative. Lupus vulgaris was unlikely as lesions were multiple and widespread. There was no caseation on histology, Mantoux test negative, CXR was normal. Lastly leishmaniasis was considered, but pathogenic organisms weres not seen on histology. Skin slit smears stained with Giemsa showed Leishmania amastigotes.

Granulomatous histology and positive skin smears confirmed cutaneous leishmaniasis occurring concommitantly in a reactional leprosy patient. Concomitant cutaneous leishmaniasis and leprosy has been reported before in the world literature[1], but it is rare.

Both leishmaniasis and leprosy have number of similar characteristics. Both are due to obligate intra cellular organisms, both present a spectrum of disease dependent on the host response and the microorganism's virulence factors, both have a histological spectrum with five subgroups[1,2,3]. In leprosy the histological spectrum has been defined by Ridley and Jopling[2] who described a five group classification: the tuberculoid (TT), borderline tuberculoid (BT) borderline (BB), borderline lepromatous (BI), and lepromatous (LL) groups. There is a similar histological spectrum in Leishmaniasis by Bryceson (1969) from tuberculoid TT, through intermediate tuberculoid IT, intermediate TT, macrophage intermediate MI, to macrophage MM.

Patients with lepromatous leprosy have cell mediated immune deficiency which is specific to Mycobacterium leprae[4]. Our patient's lymphocyte subsets show normal numbers, with diminished lymphocyte function and latter may have contributed to his widespread cutaneous leishmaniasis lesions along with the parasitic factors.

This is the first case of coexisting leprosy and cutaneous leishmaniasis reported, and the first case of widespread cutaneous leishmaniasis treated successfully with systemic sodium stibegluconate in Sri Lanka.

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