

CASE REPORT

VISCERAL LEISHMANIASIS WITH CUTANEOUS ULCER OR CUTANEOUS LEISHMANIASIS IN NEPAL

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Abstract. We document here a case of leishmaniasis in a 12 year old girl with unusual clinical presentations leading to the suggestion that it may be a case of visceral leishmaniasis with an unusual feature of ulcer on the skin of the leg, or a case of cutaneous leishmaniasis, the first such report from Nepal.

Visceral leishmaniasis caused by *Leishmania donovani* is one of the important health problem in different parts of Nepal, including the area in which the laboratory is situated (Koirala, 1995). Non-tender splenomegaly with or without hepatomegaly, wasting and pallor of mucus membrane and also lymphadenopathy are common clinical signs of the disease (Parija, 1996).

We report here a case of leishmaniasis with unusual clinical presentation leading to the suggestion that it may be a case of visceral leishmaniasis with an unusual feature of ulcer on the skin of the leg, or a case of cutaneous leishmaniasis, either one yet to be reported from Nepal.

A 12 year old Nepali girl presented with complaints of irregular intermittent mild to moderate fever; an ulcer on the left foot and discomfort in the left hypochondrium for 8 months. She sought advice from various general practitioners, where she was put on treatment with different antibiotics and antimalarials. She did not respond to any of the treatment.

She was admitted in the hospital. Examination revealed that she was febrile with a temperature of 38.3°C. She was anemic. Spleen was firm, smooth, non-tender and was enlarged upto 8 cm below the coastal ribs, cutaneous examination was marked by a single ulcer on the dorso-medial aspect of left foot just distal to the ankle joint. The ulcer was large and ricular measuring 3 cm in diameter. It had an elevated margin, base of the ulcer being covered with the serous discharge. Other physical examinations were unrewarding.

Laboratory investigations were marked by hemoglobin 7.5 gm/dl, total leukocyte count 3,900/mm³, differential leukocyte count: polymorphy 58, lymphocytes 42, no eosinophils; erythrocyte sedimentation rate (Wintrobe) 67 mm/1st hour. A Mantoux test was non-reactive. Sputum for acid fast bacilli was negative. Peripheral blood smear for malaria parasite were negative. SSS test for leprosy, EKG and X-Ray of chest were normal. The pus culture for aerobic bacteria was sterile after 48 hours of incubation.

Direct microscopy of the bone marrow aspiration fixed with methanol and stained with May-Grunwald Giemsa stain as for blood films showed amastigotes of *L. donovani* (LD bodies). The LD bodies were also demonstrated in the Geimsa-stained buffy coat of the peripheral blood smear. Histopathological examination of thick biopsy from the edge of the skin ulcer showed no LD bodies. Aldehyde test performed with serum of the patient was positive within 20 minutes. Direct agglutination test (DAT) for antibodies to *L. donovani* was 1:2,000 (positive).

The case was diagnosed as visceral leishmaniasis. She was treated with antikala-azar therapy, sodium stibogluconate administered intra-muscularly in a dosage of 20 mg/kg/day for 4 weeks. On the 5th day of treatment, the patient became afebrile. She was discharged from the hospital the next day with the advice to continue the same treatment for a total period of 4 weeks. She was followed up every week for a total period of 3 months after receiving the treatment. The ulcer healed completely within 3 months, leaving a scar. She made an uneventful recovery.

But for the presence of a non-healing ulcer on

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the leg, clinical manifestations such as splenomegaly, anemia and wasting, positive laboratory findings (presence of LD bodies in the bone marrow smear) and a positive aldehyde and DAT test, this case would have been conclusively diagnosed as visceral leishmaniasis, which is frequent in this part of Nepal (Koirala, 1995). The ulcer as such is not a common presenting feature of visceral leishmaniasis. The healing of the ulcer within 3 months of treatment by antimonial therapy strongly suggests the possibility of the case as of one of cutaneous nodule or ulcer which have only been reported from Sudan and rarely from East Africa in cases of visceral leishmaniasis (WHO, 1990). Since presence of such ulcers are not unusual; our patient could be a case of visceral leishmaniasis with a non-healing ulcer possibly caused by a high load of parasites, as LD bodies were seen even in the buffy coat of the peripheral blood smear. Such a case of visceral leishmaniasis presenting with an ulcer would be the first such report from Nepal.

Cutaneous leishmaniasis caused by *Leishmania tropica* has been reported in the neighboring country, India (Parija, 1996). The condition, however, is almost unknown in Nepal, although *Phlebotomus sergenti* and *P. papatasi*, the vectors of the parasite, have been reported from Nepal. The presence

of an ulcer suggestive that of Oriental sore on the dorsomedial aspect of left foot. The healing of the ulcer within 3 months of treatment by antimonial therapy strongly suggests the possibility of the case as one of cutaneous leishmaniasis even though LD bodies were not demonstrated in the full thickness biopsy smear. It is a recognized fact that LD bodies are difficult to demonstrate in cases of chronic cutaneous leishmaniasis. However, the presence of splenomegaly, and serum showing positive aldehyde and DAT tests are findings which go against this diagnosis as these are rarely present or associated with a case of cutaneous leishmaniasis.

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