

## CASE REPORT

### POST-KALA-AZAR DERMAL LEISHMANIASIS (PKDL): THE FIRST CASE REPORT FROM NEPAL

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**Abstract.** Post-kala-azar dermal leishmaniasis (PKDL) is condition characterized by non-ulcerative lesions of the skin caused by *Leishmania donovani* that is usually seen after the completion of treatment of kala-azar (visceral leishmaniasis). We document the first case report of PKDL in Nepal.

Post-kala-azar dermal leishmaniasis (PKDL, caused by *Leishmania donovani*), has been reported in neighboring India (Parija, 1996). The condition, however, is almost unknown in Nepal, which is endemic for visceral leishmaniasis in the areas adjoining the border with Bihar Province of India although the parasite vector has been reported from Nepal (Koirala, 1995). The majority of patients have history of previous treatment for visceral leishmaniasis. In India, PKDL was reported up to 20% of these post visceral leishmaniasis patients. We report the first case of post-kala-azar dermal leishmaniasis (PKDL) from Nepal.

A 24-year-old Nepali girl presented with a 3-year history of gradually increasing multiple hypopigmented skin lesions on the trunk, upper extremities, face, chin, and right ear. She sought the consultations of various general practitioners and skin specialists; she was prescribed on different topical antibiotics, antifungals and steroids. She did not respond to any of the treatments.

She was seen in the medical out-patient department (OPD) of our Institute. Her history and examination findings were reviewed. While reviewing the history, her document revealed that she had bone marrow *Leishmania donovani*

(LD bodies) positive visceral leishmaniasis; she had received twenty-two intramuscular injections of sodium stibogluconate nearly five years ago. Inspection of her skin lesions revealed multiple hypopigmented macules (10-12 mm in diameter) over the extensor surfaces of the arms and especially the elbows, the back and chin (Fig 1a). A large infiltrated erythematous patch was found on her face and right ear and a few nodules were present on her chin. Peripheral sensation was normal. The general physical examinations was unrewarding; there was no organomegaly.

Laboratory investigations revealed hemoglobin 11.5 g/dl, total leukocyte count 6,500/mm<sup>3</sup>, polymorphs 64%, lymphocytes 36%, no eosinophils, ESR (Wintrobe) 15 mm/1<sup>st</sup> hour; Mantoux test was non-reactive. Slit-skin smears (SSS) for acid-fast bacilli were negative; a skin smear was positive for LD bodies. Bone marrow aspirate for LD bodies and serum for the direct agglutination (DAT) and aldehyde tests were negative. The patient was diagnosed as having post-kala-azar dermal leishmaniasis. She was treated with IM sodium stibogluconate (20 mg/kg/day) for 4 months and was followed-up every week for two months after treatment. The patient's recovery was uneventful: her skin lesions completely disappeared within 4 months and left no scars. A repeat slit-skin smear was negative for LD bodies.

The onset of PKDL is insidious in India: lesions commonly start to appear after an in-

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Fig 1a,b,c—Patient showing lesions of post- kala-azar dermal leishmaniasis.

terval of 1-2 years, sometimes longer (Takahashi and Sato, 1981) and diagnosis may be delayed for as much as 20 years (Munro *et al*, 1972). Hypopigmented macules appear over extensor surfaces of arms, inner sides of thigh, chin and eventually all over the body sparing the scalp, palms, soles, axillae and perineum. Consequently or independently nodules develop in a similar distribution, occasionally external genitalia and mucosa of the nose may be affected (Munro *et al*, 1972). Verrucous, papillomatous and xanthomatous forms are described (Mojan *et al*, 1962). All forms - macular, popular and nodular - may be present in the same patient. PKDL progresses over many years and seldom heals spontaneously. PKDL may be clinically confused with lepromatous leprosy but peripheral nerves are not involved and slit-skin smears are negative for acid-fast bacilli (Bryces, 1996).

In East Africa, PKDL commonly occurs at the end of a course of treatment of VL, presenting as a transient papular rash over face and forearms or as a crop of well-defined rounded papules, less than 5 mm in diameter, that heal spontaneously within a few months (Bryces, 1996). Rarely, late onset and clinical features resemble Indian PKDL (Rashid *et al*, 1986). Indian PKDL is characterized by dermal infiltration with histiocytes, plasma cells and lymphocytes that is variable in distribution extent and intensity. It is more intense and parasites are more numerous in the nodular variety. Epidermal changes are not marked and there is no ulceration.

As a rule, viscera are spared. Immunoglobulin and antibody response are less marked than in VL and tend towards normal in the

more chronic cases. The basis of the change from viscerotropism to dermatropism and of the delicate prolonged balance between infection and immunity that characterize Indian PKDL have not been explained. There are no clear published guidelines for treatment. In principle, treatment with an antimonial should be given in a dose of 20 mg/kg/day intramuscularly at least for four months (Bryces, 1996). This case shows that PKDL should be kept in mind by the clinician while examining non-ulcerative lesions of the skin in Nepal.

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