Annals of Tropical Medicine & Parasitology, Vol. 102, No. 5, 383–390 (2008)

Clinical manifestations of cutaneous leishmaniasis in Sri Lanka — possible evidence for genetic susceptibility among the Sinhalese

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Received 15 October 2007, Revised 11 December 2007, Accepted 14 December 2007

Human cutaneous leishmaniasis (CL) caused by *Leishmania donovani*, a pathogen more usually associated with visceral leishmaniasis, is now endemic in Sri Lanka. This report details the characteristics of 200 patients with locally acquired CL, who were recruited prospectively for an ongoing study into the genetic susceptibility to CL in Sri Lanka. In each case, the CL was confirmed by the demonstration of amastigotes in a direct smear and/or promastigotes in a culture. Although only 82% of the Sri Lankan population is Sinhalese, all 200 patients belonged to this ethnic group. The patients had a median age of 32 years (range=4–80 years). Most of them each had a single, non-tender, non-itching and dry lesion which had started as a papule and then gradually enlarged and ulcerated, with changes in the surrounding skin. None of the patients had any signs of systemic disease. Eleven (5.5%) each had at least one other affected family member. Patients with multiple lesions were most likely to be found in families with more than one affected member ($P=0.002$) but multiple lesions were not associated with diabetes mellitus ($P>0.05$). Although the results of passive detection under-estimate the true occurrence of a disease, the present data point towards enhanced susceptibility to CL among the Sinhalese and/or certain individuals, possibly determined by genetic factors.

The first, known, autochthonous case of human cutaneous leishmaniasis (CL) in Sri Lanka occurred in the 1990s (Athukorale *et al.*, 1992). Prior to that, all known cases of leishmaniasis reported in the country were travellers who had apparently acquired their leishmanial infections in the Middle East (Seneviratne *et al.*, 1995). Since 1992, however, ever more patients with locally acquired CL have been reported, mainly from the North Central, Eastern and Southern provinces of Sri Lanka (Siriwardena *et al.*, 2003). It is unclear, however, whether this trend reflects growing incidence of the disease or improvements in the diagnosis and awareness of the disease, or both (Ihalamulla *et al.*, 2002).

In humans the leishmaniases form a diverse group of disease phenotypes, ranging from self-healing cutaneous lesions to debilitating mucocutaneous infections, subclinical viscerotropic dissemination, and a fatal illness with visceral involvement. Even CL has a range of clinical, histopathological and immunopathological manifestations, which vary with the *Leishmania* species involved, the immune mechanisms of the host, and environmental factors (Akilov *et al.*, 2007). The two pathogenic extremes in CL are represented by mucocutaneous leishmaniasis (associated with hypersensitivity) and anergic diffuse cutaneous leishmaniasis (associated with hyposensitivity). In

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DOI: 10.1179/136485908X300779