Locally acquired visceral leishmaniasis in Sri Lanka

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(Index words: Liver and bone marrow biopsy, clinical features, LD bodies)

Introduction

Cutaneous leishmaniasis is an established disease in Sri Lanka. The first locally acquired case was detected in 1992 [1]. This was followed a few years later by a number of cases from different parts of the island. The first report of mucosal tissue localisation was in 2005 [2]. Although Sri Lanka has been identified as the country where disease has been acquired in two cases of travel related visceral leishmaniasis in foreigners [3], to date autocthonus visceral leishmaniasis has not been documented in the country. Here we report the first locally acquired case in a resident from the North Central Province of the island.

Case report

A 36-year old woman from the North Central Province presented with a history of abdominal distension for 3 months. There was no history of fever or overseas travel. The patient was pale, and had mild bilateral axillary lymphadenopathy. Massive hepatosplenomegaly was present. There were no skin lesions or pigmentation. The ESR was 160 mm/h. The haemoglobin was 7.9 g/dl, the white cell count 3.6×10^9/l, and the platelet count 186×10^9/l. The liver enzymes and serum bilirubin were within the normal range. The serum proteins were elevated with reversal of albumin: globulin ratio (total proteins 90g/l, albumin 32 g/l, globulins 58g/l). Based on these findings a clinical diagnosis of lymphoma was made. A bone marrow and a trephine biopsy were done, followed by a liver biopsy.

The liver biopsy showed preservation of normal architecture with a prominent periportal inflammatory infiltrate comprising lymphocytes and plasma cells. There was Kupffer cell hyperplasia with numerous amastigotes within them giving the typical dot and dash appearance of Leishman-Donovan (LD) bodies (Figure 1a). The bone marrow showed reactive proliferation of all cell lines and the histiocytes contained numerous amastigotes (Figure 1b).The trephine biopsy also revealed amastigotes within marrow histiocytes (Figure 1c). Giemsa stain performed on the trephine biopsy showed the LD bodies (Figure 1d). Based on the clinical history, histopathological and haematological findings it was concluded that she had visceral leishmaniasis.

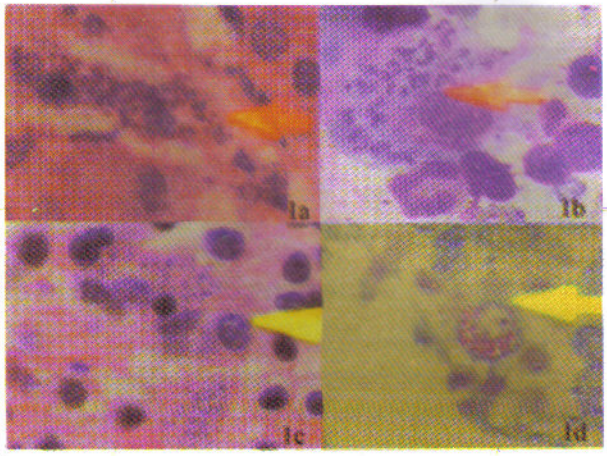

Figure 1a: **The Liver biopsy showing the amastigotes within the Kupffer cells (H & E, 100X).** Figure 1b: **Bone marrow demonstrating the amastigotes within histiocytes (Leishman 100X),** Figure 1c: **Trephine biopsy with amastigotes (H & E, 100X),** Figure 1d: **Trephine biopsy with Giemsa stain showing the amastigotes (Giemsa 100X).**

Discussion

The parasite *Leishmania* is exclusively transmitted by the bite of a female sandfly of the genus *Phlebotomus* or *Lutzomia*. Depending on the species of parasite and immune response of the host, the disease ranges from self-healing cutaneous lesions to a fatal systemic disease. Visceral leishmaniasis, a deadly disease, is particularly prevalent in India, Bangladesh, Brazil, north eastern Africa and in European countries bordering the Mediterranean. Visceral leishmaniasis is a current global health concern due to its co-existnence with HIV infection [4].

Leishmania donovani, the parasite species commonly causing visceralising disease, has been identified as the causative organism for cutaneous leishmaniasis in Sri Lanka [5]. A few studies indicate that the common sandfly *Phlebotomus argentipes* is widely distributed in the island [6].

It is possible that this is the first identification of a prevalent disease [7]. Further testing must be done to specifically identify the parasite by isoenzyme and DNA

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analysis, and epidemiological studies are necessary. Unfortunately, the patient is not willing to undertake any further medical treatment or testing.

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