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CORRESPONDENCE

VISCERAL LEISHMANIASIS IN AIDS PATIENT

As far as we know there are 20 cases of visceral leishmaniasis reported in immunocompromised patients with serum antibodies to HIV[1-4, 6-11], eleven of these had AIDS, i.e., were HIV positive with some kind of opportunistic infection or Kaposi Sarcoma and nine were only HIV positive with no clinical manifestation. The clinical data most commonly referred are hepatomegaly, splenomegaly, fever, hypergammaglobulinaemia and parasites were detected in most bone marrow smears. Search for antileishmania antibodies was positive in about 60% of the cases. All cases were treated with antimonial therapy (Glucantime^R) and about 40% of the cases had more than one series or other drug as Amphotericin B or Pentostam.

We report here a case with visceral leishmaniasis in an AIDS patient, the first we had at the University of São Paulo Medical School.

A 37 years old man, promiscuous homossexual, from an endemic area of visceral leishmaniasis (Northeast of Brazil) living in São Paulo (Southeast, non endemic area) for the last 11 months, was admitted at the hospital with a progressive enlarging nodule in the neck for the last 20 days accompained by fever and a weigh loss of 7 kg. On admission he was in good general condition, with hepatomegaly, cervical painful lymph node with 6 cm in diameter and fever. Anaemia was detected (haemoglobin: 10.7 g/dl and 35% haematocrit) as well as leukopenia (2.800/mm^3) with lymphopenia (560/mm^3) and normal platelet count. He had also hypoalbuminaemia (2.1 g/dl) and hypergammaglobulinaemia (2.26 g/dl). Anti HIV atibodies were positive (ELISA and western blot). Many leishmania amastigotes were found in bone marrow smears. Search for acid fast bacilli was positive in cervical lymph node. Treatment with triple scheme for tuberculosis and Glucantime^R (80 ml) was administered. Oral and esophageal **Candida albicans** infection developed leading to worse clinical condition. The treatment with Glucantime^R was changed to amphotericin B (45 ml) but the patient died.

The post-mortem examination showed depletion of T lymphocytes zone in lymphoid organs, disseminated tuberculosis and candidiasis. Leishmaniasis was not seen in the histopathological sections of the organs; however, leishmania antigens were detected by peroxidase antiperoxidase test using anti-leishmania policlonal antibodies mainly within Kupffer-cells.

This case shows that visceral leishmaniasis can occur as an opportunistic infection in HIV positive patients from endemic areas even when not living in this area at the time of the HIV infection. The HIV infection promote changes in the clinical manifestation of the visceral leishmaniasis. In this case the splenomegaly was absent. However, antibodies anti-leishmania were produced. The therapeutic response was affected reinforcing the idea that Glucantime^R action would depend on the integrity of T Lymphocyte immune system. However, the treatment even demanding more than one series or other drug was useful as there was no viable leishmania found. There was only antigenic material present within phagocytic cells. In this case we might consider that leishmaniasis developed either after long incubation period or originated from intracellular quiescent parasites. We have already found leishmania within liver parenchymal cell with no apparent lesion of the cell[5]

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