RESEARCH NOTE

Relationship between the Human T-lymphotropic Virus Type 1Infection and Clinical Manifestations of Tegumentary Leishmaniasis in the Colombian Pacific Coast

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The human T-lymphotropic virus type 1 (HTLV-1) was the first retrovirus reported in humans, and it is a cellular transforming agent (BJ Poiesz et al. 1980 *Proc Natl Acad Sci USA 77*: 7415-7419). It has the capacity to produce tumors of T-cells, neurologic diseases and possibly immunosuppression (M Popovic et al. 1984 *Science 226*: 459-462). The HTLV-1 has been associated with the etiology of several diseases, such as the adult T-cell leukemia/lymphoma (K Takatsuki et al. 1977 *Excerpta Medica*: 73-77), HTLV-1-associated myelopathy (GC Roman & M Osame 1988 *The Lancet I*: 651) and tropical spastic paraparesis (A Gessain et al. 1985 *The Lancet II*: 407-410). Moreover, an interaction has been observed between retroviral infection and clinical expressions of some diseases such as leprosy, infective dermatitis and strongyloidiasis (L Legranade et al. 1990 *The Lancet 336*: 1345-1347, A Blank & F Rosso 1996 *Acta Médica Colombiana 21*: 122-126), that occur occasionally as hyperinfection in carriers of HTLV-1, as in the case of strongyloidiasis. Immunosuppression induced by viral infection may contribute to the development of these disease presentations. Among the inhabitants of the Colombian Pacific Coast there is a relatively high seropositivity of 2.8% for HTLV-1 (G Roman 1988 *Ann Neurol 23* (Suppl): s113-s120, C Arango et al. 1990, p. 377-383. In William A Blattner, *Human Retrovirology: HTLV-1*, Raven Press Ltd., New York). Because tegumentary leishmaniasis is frequent in this area, and presents with a broad clinical spectrum, and subclinical infection is more frequent than the disease, we postulated the possible existence of a relationship between chronic/severe clinical expressions of tegumentary leishmaniasis and coinfection with HTLV-1 evidenced by the presence of antibodies in the serum. We analyzed 92 serum samples from individuals residing in Tumaco (Nariño); 23 were obtained from patients with chronic disease (duration of disease > 6 months); 23 from patients with acute disease (duration of disease < 3 months), and 46 from individuals with subclinical infection (positive leishmanin test, without evidence of either active lesions or scars compatible with leishmaniasis). Antibodies to HTLV-1 were detected by latex particle agglutination (Serodia HTLV-1 Fujerebio Inc., Tokyo, Japan). Two samples (2.2%) were positive for antibodies to HTLV-1, one having a titer of 1: 64, and the other 1: 32 (Table). These two sera were confirmed using western blot (Problot HTLV-1 Fujerebio Inc., Tokyo, Japan). Sera are considered positive for antibodies to HTLV-1 when they react with the glicoprotein 46 (gp46) a membrane or envelope protein, and two of the following pro-

TABLE

Frequency of serum antibodies anti-HTLV-1 in individuals with different outcomes of infection by *Leishmania viannia*

Group	% positivity [a]
Subclinical infection	2/46 (4.3%)
Chronic infection > 6 months	0/23 (0%)
Acute infection < 3 months	0/23 (0%)
Total	2/92 (2.2%)

a: antibody titer > 1: 16

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teins: p53, p24 or p19 structural proteins. The samples that were positive by agglutination also were positive by immunoblot. One sample was positive for the gp46, p53 and p24; the other for the gp46, p24 and p19.

Both positive samples corresponded to individuals with subclinical infection. The frequency of seropositivity for antibodies anti HTLV-1 in the study population was similar to the prevalence observed for the general population from the Pacific Coast. Therefore there is no evidence of an association between HTLV-1 and a more severe clinical presentation of dermal leishmaniasis.

Although the HTLV-1 infection may produce immunosuppression, coinfection evidently does not trigger the development of disease in the individuals with subclinical *Leishmania* infection. On the other hand, it is unknown whether the course and time of evolution of HTLV-1 infection could have bearing on the development of disease in individuals with subclinical infection with *Leishmania* parasites or if modulation of the immune response by HTLV-1 infection might favor a subclinical outcome of *Leishmania* infection.