HTLV-I ASSOCIATED MYELOPATHY IN SALVADOR (NORTHEASTERN BRAZIL)

A. MEIRELES* — E. D. MOREIRA Jr.** — O. A. MORENO-CARVALHO ***
R. BADARO **** — A. MELO *****

SUMMARY — Recent studies of tropical spastic paraparesis have confirmed the existence of human T-cell leukemia virus type-I (HTLV-I) in several tropical areas of the world. In order to determine the role of HTLV-I as an etiologic agent of myelopathies in Salvador, we conducted a clinical and serological study in 43 patients with non-traumatic and non-tumoral myelopathies. We found 9 patients with HTLV-I associated myelopathy (HAM) which points to a new endemic area of HAM.

KEY WORDS: HTLV-I, myelopathy, tropical spastic paraparesis.

Resumo — Recentes estudos têm mostrado a presença de mielopatia associada a infecção por HTLV-I em muitas áreas tropicais do mundo. Com o objetivo de determinar o papel do HTLV-I como agente etiológico de mielopatias em Salvador, realizamos estudo clínico e sorológico em 43 pacientes com mielopatia de etiologia não traumática e não tumoral. Encontramos 9 pacientes com mielopatia associada a HTLV-I (HAM) o que sugere nova área endêmica de HAM.

PALAVRAS-CHAVE: HTLV-I, mielopatia, paraparesia espástica tropical.

Human T-lymphotropic virus type one (HTLV-I) is associated with adult T-cell leukemia/lymphoma (ATL) and tropical spastic paraparesis. HTLV-I associated myelopathy (HAM) has been described in southern Japan, USA, Colombia and Central Africa. In Brazil, several cases have been described in Ceara, Rio de Janeiro, Sao Paulo, and Bahia showing the association of myelopathy and HTLV-I. In a recent study Moreira Jr. & col. described a high prevalence of HTLV-I infection in several risk groups and in a randomized group of 129 patients admitted to a hospital which cares for the low socio-economic class. It was found a prevalence of 19.4% for HTLV-I in the serum of these patients.

The first cases of HAM in Bahia are described in this paper.

METHODS

Sera and CSF of 43 patients admitted in a general hospital in Salvador in a period of 6 months with chronic spastic paraparesis were tested for HTLV-I. Nine patients with progressive chronic spastic paraparesis had serum and CSF positive to HTLV-I.

Antibodies to HTLV-I were detected with a commercially available enzyme immunoassay (EIA). EIA repeatedly reactive samples were further confirmed by a new dot blot confirmatory immunoassay using highly purified HTLV-I viral and recombinant proteins as an
antigen source. Samples were considered positive if antibodies against the both gag (p24) and env (p21E) gene products were present. In CSF samples patterns considered were: cytology (cells/ml number and cytormorphological profile), protein and glucose contents (mg/dL), and gamma globulins, participation (%) and distribution on the protein profile. Other specific antibodies to syphilis, cysticercosis, schistosomiasis, toxoplasmosis and HIV were also tested.

RESULTS
All the patients were adults, with ages that range from 18 to 56 years. Two of the patients were promiscuous and one of them had blood transfusion 8 months before symptoms. They had similar histories with progressive weakness, first in one leg and after in the other leg, that was associated with paresthesias of several degrees of intensity. None of the patients had sensory level. All of them had vegetative disturbances that where characterized by bladder dysfunction (manifested by increase urinary urge or incontinence), constipation and impotence in men.

CSF showed slight increase in the cell number with a range of 4.3 to 25 cells/ml with predominance of lymphocytes and monocytes. There was no relevant number of macrophages, eosinophils and neutrophils. The glucose was normal and the protein ranged from 28 to 68 mg/dL. There was a slight increase in gamma-globulin rate in two of them. One patient had infection by HIV and HTLV1. No other associated parasite or fungus infections were found.

COMMENTS
In recent years, many studies have shown the high prevalence of HTLV1 infection and HAM among different races and high risk groups in several populations in the world. In Brazil, Costa & col. reported 10 patients with clinical evidence of TSP and Cortes & col. concluded that HTLV1 was prevalent in groups at risk for AIDS. Our cases confirm former results of HAM in Brazil and points to an endemic zone of HTLV1 associated myelopathy. Among the patients studied two were promiscuous and only one had received blood transfusion suggesting a vertical source of contamination.

As has been pointed out by Spinha-Franca & col., we also found a slight increase in the cell number as well as in the protein content and gamma globulins rate in the CSF.

Since we have in Salvador a population descendent from black Africans and Iberians this endemia could result from this migration. However, further studies are necessary to assess this question.

REFERENCES