TOXOPLASMA GONDII MYELITIS IN A PATIENT WITH ADULT T-CELL LEUKEMIA-LYMPHOMA

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ABSTRACT - Adult T cell leukemia-lymphoma (ATL) caused by HTLV-I may be associated with severe immunosupression and several opportunistic infections. Toxoplasmic encephalitis is a common central nervous system opportunistic infection in severely immunosupressed patients, however spinal cord involvement by this parasite is rare. In this paper, we report a case of toxoplasmic myelitis in a patient with ATL.

KEY WORDS: HTLV-I, immunosupression, Toxoplasma gondii, myelitis.

Mielite por Toxoplasma gondii em um paciente com leucemia-linfoma de células T do adulto

RESUMO - Leucemia de células T do adulto (ATL), causada pelo HTLV-I, pode estar associada com imunossupressão severa e muitas infecções oportunistas. Encefalite por toxoplasmose é uma infecção oportunista do sistema nervoso central em pacientes imunossuprimidos, no entanto o envolvimento da medula espinal por este parasita é raro. Neste artigo, apresentamos um caso de mielite em um paciente com ATL.

PALAVRAS-CHAVE: HTLV-I, imunossupressão, Toxoplasma gondii, mielite.

HTLV-I is associated with several diseases as arthropathy, uveitis, dermatitis and interstitial pneumonia. This retrovirus is also the etiologic agent of HTLV-I associated myelopathy/tropical spastic paraparesis (HAM/TSP) and adult T-cell leukemia-lymphoma (ATL) ¹. The association of ATL and HAM/TSP has been described and it is considered to be uncommon^{2,3}. Patients with ATL present severe immunosupression and are susceptible to several opportunistic infections as *Cryptococcus neoformans, Pneumocystis carinii, Strongyloides stercoralis, Candida albicans, Cytomegalovirus, Mycobacterium avium and Aspergillus^{4,5}, to mention some. The protozoa <i>Toxoplasma gondii* is the most common infectious organism responsible for meningoencephalitis in patients with AIDS⁶. Nevertheless, myelitis caused by this agent is considered to be uncommon even in immunosuppressed patients⁷.

We describe a case of Toxoplasma gondii myelitis in a patient with ATL.

CASE

A 51 year-old, Afro-Brazilian male patient was admitted to the University Hospital/Federal University of Bahia with a two months complaint of progressive weakness in lower limbs evolving to paraplegia 1 month ago; he had intermittent fever and lost around 12 Kg of body weight. He also noticed some kind of skin lesions in the lower limbs during the last three months and, more recently, leg edema. On physical examination the patient appeared underweight and chronically ill, with an axillary temperature of 37.5° C and a BMU= 17.8 Kg/m^{2} . Erithematous lesions were observed in the thorax and limbs and lymphadenopathy was found in the neck. In the abdominal examination the liver and the spleen were increased and the bladder was palpable. On the neurologic examination the patient was somnolent and the motor examination revealed decreased muscle strength, hypotonia and atrophy of all limbs; the deep reflexes of the lower limbs were increased. The hematocrit was 30%, the hemoglobin was 9.9 g/dl; the white-cell count was 104,800 with 73% of lymphocytes. The platelets count was

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393,000. The sodium was 140 mEq/l, potassium 5.0 mEq/l, urea nitrogen 71 mg/dl, creatinine 2.1 mg/dl, uric acid 11 mg/dl, calcium 5 mg/dl, protein 6.3 mg/dl and albumin 3.8 g/gl. The serum aspartate aminotransferase (ALT) 54 U/l and lactic dehydrogenase (LDH) 386 U/l. The X-ray of the chest showed irregularly enlarged mediastinum. S. stercoralis and E. histolytica were identified in the stool examination. The CSF examination yielded a colorless fluid that contained 1 cell/mm³; glucose was 53 mg/dl and protein 22 mg/dl. Thiabendazol and allupurinol were started. The patient evolved febrile and with negative blood cultures. Bone marrow examination showed low grade non-Hodgkin's pleomorphic lymphoma. At this time intratecal chemotherapy with methrotexate, hydrocortisone and ARAc was initiated. On the ninth day of admission the hematocrit was 22%, the hemoglobin was 7.3 g/dl, the white-cell count 10,200 with 95% lymphocytes, platelets 72600, BUN 18 mg/dl, creatinine 1.1 mg/dl, uric acid 3 mg/dl and LDH 1757 U/l. Five days later the patient died in respiratory failure. Antibodies to HIV were negative and to HTLV-I were positive.

The pathological examination of the spinal cord revealed a diffuse meningomyelitis with densely infiltrating mononuclear cells (Figs 1 and 2). The lesions consisted of multifocal nodules predominantly of lymphocytes and hystiocytes (microglial nodules) involving the white and gray matter, sometimes with perivascular distribution. Tachyzoites and cysts of *Toxoplama gondii* were frequently found. Similar lesions were found in the brain stem, cerebellum and cerebrum associated with an intense and diffuse meningeal involvement. The presence of tachyzoites was also demonstrated in lymph nodes, lungs and spleen.

DISCUSSION

Toxoplasma encephalic involvement in patients with AIDS is common, although myelopathy by this agent is rare. So far 14 cases in the medical literature were reported, 13 in patients with HIV infection⁷. The last case was a patient with hemolytic anemia using ciclofosfamide⁸.

There are evidencies that HTLV-I infection causes mild immunosuppression increasing risks to opportunistic infections such as strongyloidiasis, tuberculosis and hanseniasis⁹. Additionally, patients with ATL are deeply immunosuppressed and frequently infected by opportunistic agents more frequently than patients with Hodgkin and non-Hodgkin lymphoma^{4,5}. The analysis of the 14 cases of myelopathy by *T. gondii* described in the literature showed that the diagnosis was done by biopsy in 7 cases, by necropsy in 2 cases and the others by favorable response to specific therapy⁷.

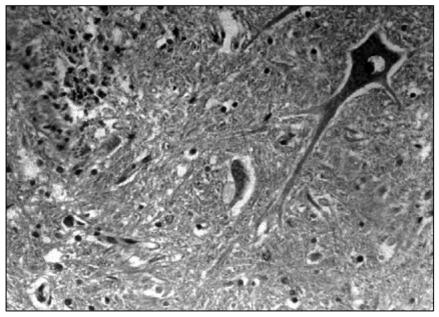


Fig 1. Spinal cord-tissue specimen at level of gray matter showing a microglial nodule (hematoxylin and eosin, x200).

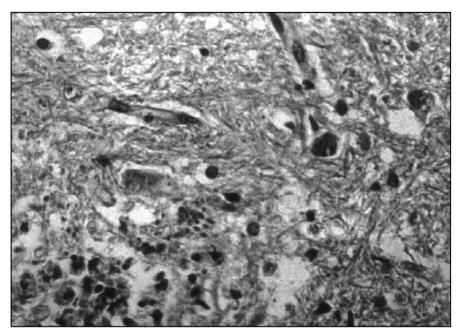


Fig 2. Detail from Figure 1 showing heavy parasitism by T. gondii, with single forms and pseudocysts, near of microglial nodule (hematoxylin and eosin, x400).

In the present report the myelopathy was initially attributed to neoplastic infiltration or an association of ATL and HAM/TSP, although the sub acute evolution and the characteristics of the sensitive syndrome are not frequent in HAM/TSP. The survival of our patient was shorter than described in the literature in patents with ATL 10 . We believe that the lethality was due to disseminated T. gondii as observed in the necropsy.

Even though it is a single case report, we believe it is important to consider the possibility of opportunistic infections as *T. gondii* in the differential diagnosis of acute and sub acute myelopathies in patients with ATL.

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