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LYMPHOCYTE ALVEOLITIS IN HAM/TSP PATIENTS

PRELIMINARY REPORT

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SUMMARY — HTLV-I associated myelopathy has been described as a systemic disease characterized by manifestations in several organs outside the nervous system. We report inflammatory pulmonary involvement in patients with diagnosis of HAM.

KEY WORDS: HTLV-I, HAM/TSP, alveolitis, pulmonary involvement.

Alveolite a linfócitos em pacientes com HAM/TSP: registro preliminar

RESUMO — Mielopatia por HTLV-I (HAM) tem sido descrita como doença sistêmica caracterizada pelo acometimento de vários órgãos além do sistema nervoso. Neste registro, estamos relatando o envolvimento pulmonar em pacientes com HAM.

PALAVRAS-CHAVE: HTLV-I, HAM/TSP, alveolite, acometimento pulmonar.

HTLV-I associated myelopathy (HAM) has been described as a neurologic disease characterized by spastic paraparesis, sensitive syndromes, peripheral neuropathy, constipation, bladder disturbance and impotence or decreased libido [5,8]. There have been some reports of systemic non-neurologic manifestations as vasculitis, uveitis, Sjogren's syndrome, arthropathy, cryoglobulinemia, ichthyosis, polymyositis and adult T-cell leukemia/lymphoma associated with HAM [7]. Pulmonary infiltrates in HAM patients were first described in natives of Martinique [2]. In Japan, bronchoalveolar lavage studies showed five patients with increased total cell counts and increased proportion of lymphocytes in lavage fluid, as compared with data found in healthy non-smokin volunteers [9]. One of these patients, with an abnormal chest X-ray, had transbronchial lung biopsy that revealed lymphocyte infiltrations in the alveolar structures which did not resemble leukemia cells.

In order to clarify the clinical picture and pathogenesis of HTLV-I associated myelopathy we have studied the pulmonary involvement in eight patients with HAM.

PATIENTS AND METHOD — Seven women and one man, with ages that ranged from 39 to 67 years, were admitted in this study. The patients presented the basic clinical and CSF features of HAM as has been described elsewhere [5]. CSF and serum samples were collected from the patients and tested for HTLV-I with a commercially available enzyme immunoassay; the specimens were repeatedly reactive in this screening test and further confirmed by Western blot.

All patients were submitted to pulmonary evaluation by a certified pneumologist (K.M.). In each patient was performed hemograma, electrocardiogram, chest X-ray, arterial gasometric studies and spirometry at rest. To accomplish the bronchoalveolar lavage the bronchoscope was wedged into a distal bronchus and 300 ml of sterile saline was used to recover the cells and epithelial lining fluid. The bronchoalveolar lavage was carried out in a single laboratory by the same researcher using identical techniques.

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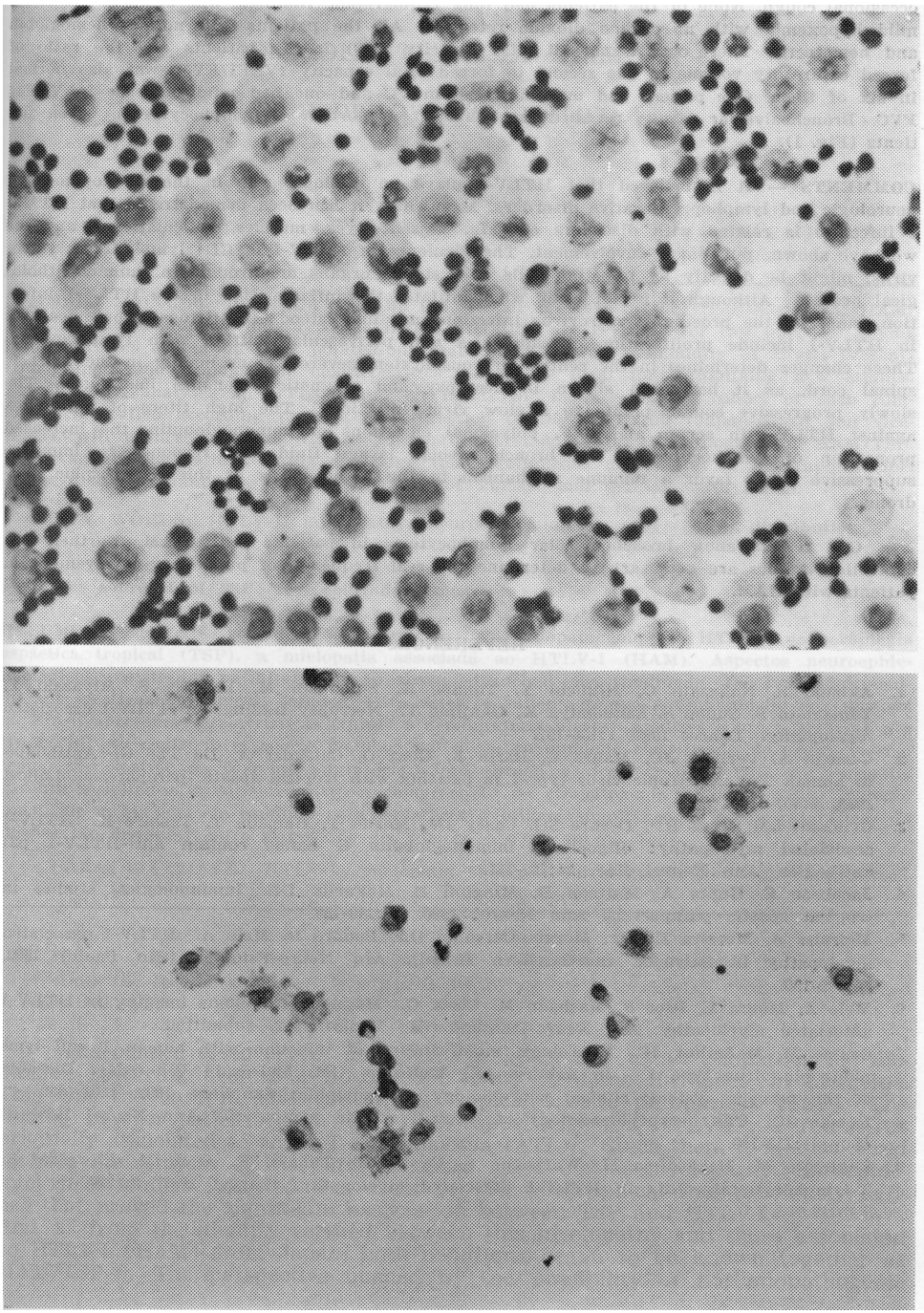

Fig. 1. Above: bronchoalveolar lavage fluid after cytocentrifugation in a patient with HAM note the increased number of lymphocytes. Below: bronchoalveolar lavage fluid after cyto centrifugation in a normal control. Papanicolaoustain, X400.

RESULTS — One patient refered fatigue and breathlessness with exertion and three had occasional cough. None of the patients had lung radiographic findings. One of them presented mild hypoxemia with no carbon dioxide retention. All the patients had normal hemoglobin and hematocrit. There was a normal vital capacity (VC) in six patients and the ratio of forced expiratory volume in one second to forced vital capacity (VEF1/FVC) was also normal in six of them. One patient had mild decrease in VC and one had mild decrease in VEF1/FVC. Bronchoalveolar lavage revealed an increased proportion of lymphocytes in seven patients (Fig. 1).

COMMENTS — It is believed that HTLV-I plays an etiologic role in the development of neurologic and lymphoproliferative disorders. Meanwhile, it has been demonstrated that HTLV-I infection is related with disorders of skin, vessels, eyes, muscles, articulations 7 and, as we have shown, pulmonary involvement. The virulence of HTLV-I, a CD4 lymphotropic retrovirus, might be directly by infecting cells of systemic organs or through an immunopathological process. Although it is not clear what initiates the inflammation in the HTLV-I infection, many of the process of retrovirus infections are understood *3,4*. Histopathologic features in HTLV-I include proliferation of capillaries and perivascular cuffing with lymphocytes *1*. These changes determines in the central nervous system severe lesions mainly in the thoracic spinal cord, as it has been shown in necropsy and magnetic resonance imaging *1,6*. The slowly progressive course points to a slow virus infection. The high titers of antibodies against HTLV-I in serum and CSF, polyclonal elevations of immunoglobulin, the increased proportion of lymphocytes in the bronchoalveolar lavage fluid and response to immune-suppressive drugs favor a immune mechanism in the pathogenesis of this pleomorphic syndrome.

Our results show bronchoalveolar lymphocytosis in patients with HAM. Further and controlled studies are necessary to determine the real frequency of pulmonary involvement in patients with HAM.

REFERENCES

1. Akizuki S, Nakazato O, Higuchi Y, Tanabe K, Setohushi M, Yoshida S, Miyasaki Y, Yamamoto S, Sudou S, Sannomiya K, Okajima T. Necropsy findings in HTLV-I associated myelopathy. Lancet 1987, 1:156-157.
2. Gessain A, Vernant JC, Maurs L, Barin F, Gout O, Calender A, De Thé G. Antibodies to human T-lymphotropic virus type-1 in patients with tropical spastic paraparesis. Lancet 1985, 2:407-410.
3. Grimaldi LM, Roos RP, Devare SG, Casey IM, Maruo Y, Hamada T, Tashiro K. HTLV-I associated myelopathy: oligoclonal immunoglobulin G bands contain anti-HTLV-I p24 antibodies. Ann Neurol 1988, 24:727-731.
4. Jacobson S, Gupta A, Mattson D, Mingioli E, McFarlin DE. Immunological studies in tropical spastic paraparesis. Ann Neurol 1990, 27:149-156.
5. Meireles A, Moreira ED Jr, Moreno-Carvalho OA, Badaró R, Melo A. HTLV-I associated myelopathy in Salvador (northeastern Brazil). Arq Neuro-Psiquiat (São Paulo) 1992, 50:189-190.
6. Melo A, Moura L, Rios S, Machado M, Costa G. Magnetic radiologic imaging in HTLV-I associated myelopathy. Arq Neuro-Psiquiat (São Paulo) 1992, submitted.
7. Osame M, McArthur JC. Neurologic manifestation of infection with human T cell lymphotropic virus type I. In Asbury AK, McKhann GM, McDonald WI (eds): Diseases of the nervous system: clinical neurobiology. Philadelphia: Saunders, 1992, 1331-1339.
8. Roman GC. The neuroepidemiology of tropical spastic paraparesis. Ann Neurol 1988, 23 (Suppl):113-130.
9. Sugimoto M, Nakashima H, Watanabe S, Uyama E, Tanaka F, Ando M, Kawasaki S. T-lymphocyte alveolitis in HTLV-I associated myelopathy. Lancet 1987, 2:1220.