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Bronchoalveolar T-lymphocytosis in HTLV-1-associated Myelopathy

To the Editor:

Chronic progressive spastic paraparesis occurring in tropical areas and southwest Japan is frequently associated with high antibody titers to HTLV-I in serum and cerebrospinal fluid.\(^1\)\(^2\) We recently reported that T-lymphocyte alveolitis occurred in patients with HTLV-I-associated myelopathy (HAM).\(^3\) The presence of such pulmonary lesions were also described by Vernant and associates in patients with HTLV-I-associated tropical spastic paraparesis (HTLV-I-TP).\(^4\)\(^5\)

In the present study, bronchoalveolar lavage (BAL) fluid had an increased proportion of lymphocytes in patients with HAM (ten women and three men, 50 ± 17 percent); compared to HTLV-I-negative normal control subjects (ten nonsmoking male volunteers, 14 ± 3 percent). Increased BAL lymphocytes consisted mainly of CD4+ cells (78 ± 17 percent) and CD4+ /CD8+ ratios (1.5 ± 0.8) were similar to those of normal control subjects (1.3 ± 0.7).

Serum levels of soluble interleukin-2 receptors (IL-2R), a marker of T-cell activation,\(^6\) were significantly elevated in patients with HAM compared to normal control subjects (685 ± 210 vs 286 ± 49 U/ml, p < 0.01). Soluble IL-2R levels were detectable in BAL fluid from HAM patients and BAL levels were approximately 13 times higher than those in serum (166 ± 103 vs 18 ± 13 U/mg albumin, p = 0.001). BAL IL-2R levels in HAM patients correlated well with the number of lymphocytes and T-cells in BAL fluid (r = 0.68, p < 0.05, and p = 0.64, p < 0.05, respectively). These results suggest that T-lymphocytes infiltrating the lungs of HAM patients are activated locally to produce IL-2R.

Our data show that bronchoalveolar T-lymphocytosis occurs in patients with HAM, suggesting that immunologic mechanisms play an important role in the development of pulmonary lesions in HAM.

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Bronchoscopic Aspect of Pulmonary Aspergilloma

To the Editor:

We read with interest the paper by Smith et al\(^7\) reporting a patient in whom pulmonary aspergilloma was visualized and biopsied by flexible fiberoptic bronchoscopy. We would like to report another case similar to the one described by Smith et al also diagnosed this way. In this form of Aspergillus lung disease, clinical diagnosis is usually based on radiologic and immunologic criteria; nevertheless fiberoptic bronchoscopy is a simple, easy procedure for confirmation in some cases.

A 40-year-old man was admitted with high fever and a chest x-ray film showing a cavitated consolidation of the right upper lobe. During endoscopic examination with an Olympus B2 fiberoptic bronchoscope, we were able to introduce the tip through the B2b bronche into the cavity. Putrid, purulent debris was seen and removed. Culture showed growing for E coli, but anaerobes, mycobacteria or fungi were not isolated. The patient improved when treated with penicillin and tobramycin, and chest x-ray film revealed a cavity with a crescent-shaped air space. Because serum precipitins were found to be negative, fiberoptic bronchoscopy was newly performed and the cavity entered. A fungus ball was seen (Fig 1) and a biopsy taken. Histopathologic examination showed septate hyphae, and aspergilloma was confirmed after right superior lobectomy.

Bronchoscopic visualization of a fungus ball has been rarely reported; in fact, we could only find one other reference in the literature.\(^8\) We also performed fiberoptic bronchoscopy in another eight patients suffering from aspergilloma, but we couldn't enter the cavity in any of them.\(^9\) We assume it was possible in this case due to the great cavity's size, as in Smith's patient as well. However, we want to point out that definitive diagnostic findings were obtained only when bacterial superinfection was overcome.