Association of Tuberculosis Status with Neurologic Disease and Immune Response in HTLV-1 Infection

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Abstract

The human T cell lymphotropic virus type 1 (HTLV-1) is the etiologic agent of HTLV-1-associated myelopathy/tropical spastic paraparesis (HAM/TSP). HTLV-1 infected individuals have increased susceptibility to *Mycobacterium tuberculosis* infection but the influence of tuberculosis (TB) on the course of HTLV-1 infection is unknown. The aim of this study was to evaluate the influence of TB on immunological, virologic, and neurologic features of HTLV-1 infection. This is a retrospective analysis of individuals enrolled in a cohort study from an HTLV-1 clinic who were evaluated for past or latent tuberculosis (LTB) and classified clinically as HTLV-1 carriers, probable HAM/TSP and definite HAM/TSP. Spontaneous cytokine production (interferon-gamma [IFN-γ], tumor necrosis factor [TNF], and interleukin[IL]-10), serum chemokines (CXCL9 and CXCL10) and HTLV-1 proviral load were evaluated. Of 172 participants, 64 did not have histories of TB (TB- group), 81 had LTB and 27 had TB in the past (TB+ group). In the TB+ group, there was a higher frequency of HAM/TSP patients (35%) than in HTLV-1 carriers (10%) (OR = 3.8, $p = .0001$). HAM/TSP patients with histories of TB had higher IFN-γ/IL-10 and TNF/IL-10 ratios when compared with HAM/TSP patients without histories of TB. There were no differences in serum chemokine production and proviral load across TB groups stratified on HTLV-1 clinical status. In conclusion, TB may influence the development of HAM/TSP, and patients with these two diseases have an impairment in the modulation of immune response.