The human T-lymphotropic virus (HTLV) is a potentially immune stimulatory agent through interacting with helper T cells. Autoimmune phenomena are not rare in patients infected by this retrovirus (1).

We read with interest the manuscript by Carvalho et al that was recently published in this Journal. The study evaluated the frequency of connective tissue disorders (CTD) in patients infected by HTLV-I. The authors studied 137 patients (27 with myelopathy). As a whole, Sjögren syndrome (SS) (17.5%) and rheumatoid arthritis (RA) (13.1%) were the most frequent CTD in their survey. Of note, both SS and RA were more significantly found in patients with myelopathy than in asymptomatic carriers (2).

We recently looked at the clinical and laboratory findings of 9 patients with HTLV-I/II-associated myelopathy. Patients were selected on the basis of a preexisting concomitant CTD (3). Eight patients (89%) were female, and the mean age was 49 years. Of interest, 2 patients (22%) were HTLV-II positive. SLE was the most prevalent CTD in our survey (5 patients - 55%). Primary SS was seen in 4 patients (44%), one of them was also diagnosed as presenting associated antiphospholipid antibody syndrome. In general terms, primary or secondary SS were documented in 7 patients (78%). There was no patient diagnosed with RA.

As compared to the above mentioned report, our experience presented different results coupled to a decreased prevalence of myelopathy. Some aspects are worthy of comment, nevertheless. HTVL-II infection was present in 2 of our 9 patients (22%). There is no mention to HTLV-II infection in the Carvalho et al study (3). Also, in contrast to their report (3), we had no patient diagnosed with RA. On the other hand, SLE was frequent in our series, whereas it was extremely rare in the other study (3). Both studies (2,3) agreed as to the highly significant prevalence of SS associated to HTLV infection.

In another strategy, we investigated the presence of antibodies against HTLV-I/II viruses in 69 cases of patients presenting with RA and 33 SLE patients. The frequency of antibodies against HTVL-I was significantly greater in RA patients (7%), as compared to healthy controls (1.28%) (P = 0.004). This finding was not confirmed in our SLE cohort (4).

The apparent discrepancies between both studies may not at least partially be related to differences in patient selection and study design. The relationship of HTLV-I/II infection to autoimmune disorders remains enigmatic, pointing to the need for additional studies on this subject.

REFERENCES