

HTLV-1-ASSOCIATED MYELOPATHY: A CASE REPORT

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BACKGROUND

Human T-Lymphotropic Virus Type 1 causes a chronic progressive myelopathy called tropical spastic paraparesis (TSP) in the Caribbean or HTLV-1-associated myelopathy (HAM) in Japan. This retrovirus is endemic in some areas such as Central and South America and transmission occurs through sexual contact, contaminated blood, breastfeeding. HAM/TSP is a chronic, progressive inflammatory myelopathy characterized by a slowly progressive paraparesis associated with lower-extremity paresthesia, neuritic pains in the back and legs, bladder dysfunction. Onset is generally after age 30 years and it can lead to inability to walk unaided in 10-13 years. The diagnosis is based on clinical and laboratory criteria and requires an appropriate clinical picture with HTLV1 positive serology in blood and cerebrospinal fluid. There is no recognized effective treatment in HAM/TSP but some authors described partial benefit of plasmapheresis or repeated treatments with methylprednisolone.

CASE REPORT

A 50-year-old male, born in Peru, presented with a five month history of walking difficulty characterized by sense of leg stiffness after some distance walked. He felt unsteady on his feet and complaint also cramps and legs pain. Physical examination showed spastic-ataxic gait, bilateral leg spasticity with moderate weakness, lower limbs hyperreflexia and Babinski sign, slightly reduced vibratory sense at both halluces, ankles and knees. The remainder of the general and neurologic examination was unremarkable. Laboratory tests including complete blood count, metabolic panel, electrophoresis, HIV-HBV-HCV serology, vitamin B12, neuronal nuclear antibodies (Yo-Hu-Ri), GAD antibodies test, syphilis serology were all within normal limits except for an increase of neutrophils with relative lymphopenia. The study of lymphocytes population showed a prevalence of CD8 T cells, suggesting a viral infection. CSF examination showed a moderate lymphocytosis, discretely increased proteins, increased IgG with a oligoclonal pattern on electrophoresis and increased IgG ratio and intrathecal synthesis. Cranial and spinal contrast-enhanced MRI was normal. Electrophysiology showed slowing of lower limbs somatosensory evoked potentials. HTLV-1 antibodies were detected in patient's serum and CSF. Polymerase chain reaction was positive for HTLV-1 DNA in both whole blood and CSF. During hospitalization he was treated with high dose methylprednisolone (1000 mg per day for 5 days) associated with baclofen, then he periodically returned to our hospital to undergo treatments with corticosteroid.

CONCLUSIONS

Even if HAM is a rare condition in our country, this case highlights the need to consider HTLV-1 infection in patients with progressive myelopathy, due to the increasing diversity and integration of the European and Italian population.

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