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ERECTILE INSUFFICIENCY AS FIRST SYMPTOM OF HTLV I/II ASSOCIATED MYELOPATHY

CASE REPORT

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ABSTRACT - A case of HTLV-I/II myelopathy in which the initial complaint was erectile insufficiency (EI) is reported. The only abnormalities found on the neurological exam were discrete weakness of the psoas and increased knee jerk reflexes. Diagnosis was made by demonstrating antibodies anti-HTLV I/II in the serum and cerebrospinal fluid (with the techniques of ELISA and Western blot), with confirmation by the polymerase chain reaction (PCR). EI can thus be the first symptom of HTLV-I/II infection and patients with EI of unknown etiology should be tested for HTLV-I/II in endemic areas.

KEY WORDS: erectile insufficiency, HTLV-I/II infection, myelopathy, HAM/TSP, Brazil.

Insuficiência erétil como primeiro sintoma da mielopatia associada ao HTLV I/II: relato de caso

RESUMO - É relatado um caso de mielopatia associada ao HTLV I/II cuja primeira manifestação foi insuficiência erétil (IE). O exame neurológico do paciente apresentava somente discreta fraqueza dos psoas e aumento dos reflexos patelares. O diagnóstico foi feito pelo achado de anticorpos anti-HTLV I/II no soro e no líquor (com as técnicas de ELISA e Western blot) e confirmado pela reação em cadeia da polimerase (PCR). Insuficiência erétil pode ser a primeira manifestação clínica de infecção pelo HTLV I/II e pacientes com IE de etiologia desconhecida devem ser testados para HTLV-I/II em áreas endêmicas.

PALAVRAS-CHAVE: insuficiência erétil, infecção pelo HTLV I/II; mielopatia; HAM/TSP, Brasil.

Erectile insufficiency (EI) is defined as the persistent failure to develop erections of sufficient rigidity for penetrative sexual intercourse[1,2] and is commonly attributed to three main causes: vascular disease, neurological disturbance involving either the central or peripheral nervous system, and psychological factors[3]. It is unusual for EI to be the sole manifestation of diseases of the nervous system[1,2,4].

HTLV-I/II infection can cause a chronic progressive demyelinating disease that predominantly affects the spinal cord, called HTLV I/II associated myelopathy or tropical spastic paraparesis (HAM/TSP). Initial symptoms of HAM/TSP are weakness and spasticity of the lower extremities, often associated with paresthesia, low back pain, and sphincter disturbances[5,6]. EI frequently accompany these symptoms but has never been described as the harbinger of the disease or as first manifestation of HAM/TSP.

We report here a patient presenting with EI in whom HAM/TSP was detected.

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CASE REPORT

RPR, a 36 year old man, was referred by the Urology Clinic for neurophysiological evaluation of EI of acute onset one year before. The patient reported nocturnal erection albeit insufficient for vaginal penetration. There were no micturition symptoms. Upon questioning, he reported constipation of one week duration. Libido was normal. He had no risk factor for EI. Neurological examination revealed slight weakness of the psoas and increased ankle jerks; the plantar responses were flexor. Vibration sense was decreased at the medial maleoli. Injection of papaverine into the penis provoked a normal erection. Hemogram, glucose, creatinine, urea, prolactin, and testosterone levels were normal. Motor nerve conduction studies of the peroneal and posterior tibial nerves as well as sensory conduction of the sural nerve were all normal. The bulbocavernous reflex was unobtainable. Magnetic resonance imaging of the spinal cord and brain were normal. Cerebrospinal fluid cells, protein, and glucose were normal. Anti-HTLV I/II antibodies were detected in serum and spinal fluid by the techniques of ELISA (Ortho, USA), Western blot (Cambridge, USA) and confirmed by a molecular test, the polymerase chain reaction (PCR). Figure 1 shows the electrophoresis of the nested PCR product [primers SK 110 (pol)/SK 44 (tax) and 248/249 (env region)] in 1.8% agarose gel, stained by ethidium bromide. Lanes 1 and 2 are the positive and negative controls, respectively. Lane 3 shows the DNA amplified from the patient's peripheral blood mononuclear cells (PBMCs), with a product of the expected size for the primers used (466 base pairs). The patient has been followed up for four years, his impotence and neurological exam having not changed.

DISCUSSION

Sections through the spinal cord of patients with HTLV-I/II myelopathy show loss of myelin and axons, vacuolar degeneration [7], with a lymphocytic infiltrate, particularly in recent lesions, and proliferative changes in the form of fibroblastic thickening of the meninges and perivascular tissues.

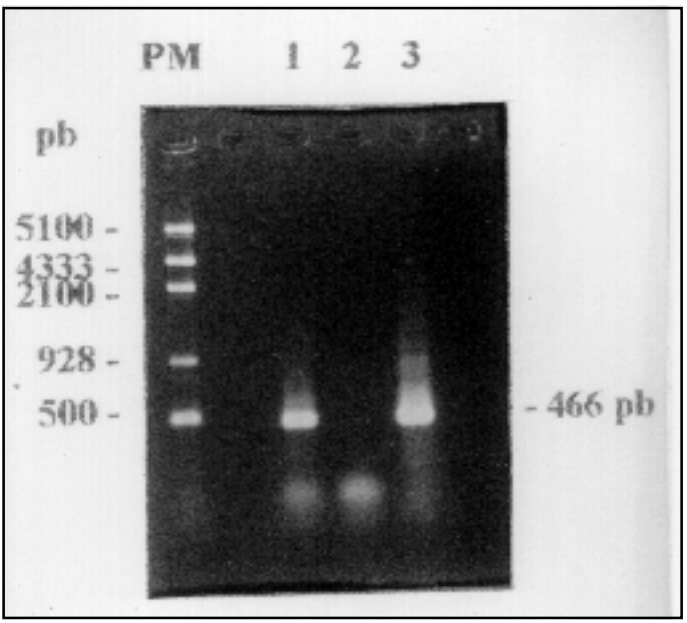

Fig 1. HTLV-I proviral amplification in peripheral blood mononuclear cells (PBMCs) of patient RPR, using nested PCR product with primers SK 110 (pol) / SK 44 (tax) in the external reaction and 248/249 (env region) in the internal. Electrophoresis in agarose gel (1.8%) stained with ethidium bromide: lanes 1 and 2 are the positive and negative controls, respectively. Lane 3 shows the DNA amplified from the patient's PBMCs, with a product of the expected size for the primers used (466 base pairs).

Nerve roots, especially the posterior, are also involved in the inflammatory process[8]. Involvement of the peripheral nervous system may be seen in up to 25 per cent of patients[5]. Our patient had a prolonged RBC latency, possibly indicating a lesion of the peripheral nervous system.

Impotence or decreased libido are common in HTLV-I/II myelopathy[5,6] because of impairment of nervous mechanism of erection caused by the spinal cord lesion or the peripheral nervous system.

The reported seroprevalence of HTLV-I/II in blood donors in Minas Gerais, Brazil is of 0.32%[9] and the finding of antibodies anti-HTLV-I/II in the patient is suggestive that the EI is due to the myelopathy associated to the HTLV-I/II infection. Although the neurological exam of the patient showed soft signs of myelopathy, the EI was the symptom which prompted him to seek medical attention. This is the first report showing that EI can be the first clinical manifestation of this infection. In areas endemic for HTLV-I/II, patients without known causes of EI, in whom soft signs of myelopathy are found, must be investigated for the presence of HTLV-I/II.

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