Prior works have already suggested a relationship between the cerebellum and cortical functions. The phenomenon of crossed cerebellar diaschisis as a metabolic depression of the cerebellum contralateral to a cortical injury was reported first.[7] In subsequent years, the opposite phenomenon was observed; that is, left cortical dysfunction secondary to contralateral cerebellar injuries.[2-4] Some of these works were based on functional neuroimaging techniques (SPECT or PET) and showed cortical hypoperfusion in the left hemisphere.[3,8] All of this led to these authors' suggesting a lateralization of cerebellar functions to regulate several cognitive processes, wherein the right cerebellar hemisphere would play a major role in language.

Although the concept of crossed aphasia is well documented, there have been very few studies to date that have demonstrated the presence of language problems associated with left cerebellar injuries. Murdoch and Whelan[5] compared scores obtained on different linguistic batteries by a group of patients who had suffered a left cerebellar stroke with a control group and found significant impairment. Based on their findings, these authors advocated the idea that the left cerebellar hemisphere also participated in language regulation as a result of an ipsilateral cortical diaschisis, a datum whose existence had already been referred to previously.[9,10] However, in the article by Murdoch and Whelan, none of the patients were studied in the acute phase nor was any mention made of the language disorder being the main manifestation of the stroke.

We believe that the interest in our case is precisely this, since it is a case of a left cerebellar infarction that only manifested as a language disorder. Furthermore, following the stroke, the patient developed symptoms that were compatible with the cognitive affective cerebellar syndrome described by Schmahmann and Sherman.[6] The more or less sudden appearance of a cognitive impairment following a vascular event does not in any way rule out the presence of previously latent impairment that may not have revealed itself until that time. The evidence of a PET with bilateral temporo-parietal hypoperfusion supports this hypothesis; hence, it is likely that there is an impairment that has gone undiagnosed prior to the stroke, and that the cerebellar infarction played a major precipitating role in its development.

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References

1. Arriada-Mendicoa N, Otero-Sliceo E, Corona-Vázquez T. Conceptos actuales sobre cerebelo y cognición. Rev Neurol. 1999;29:1075-82.
2. Ackermann H, Vogel M, Peterson D, Poremba M. Speech deficits in ischemic cerebellar lesions. J Neurol. 1992;239:223-7.
3. Silveri MC, Leggio MG, Molinari M. The cerebellum contributes to linguistic production: a case of agrammatic speech following a right cerebellar lesion. Neurology. 1994;44:2047-50.
4. Mariën P, Saerens J, Nanhoe R, et al. Cerebellar induced aphasia: case report of cerebellar induced prefrontal aphasic language phenomena supported by SPECT findings. J Neurol Sci. 1996;144:34-43.
5. Murdoch BE, Whelan BM. Language disorders subsequent to left cerebellar lesions: a case for bilateral cerebellar involvement in language? Folia Phoniatr Logop. 2007;59:184-9.
6. Schmahmann JD, Sherman JC. The cerebellar cognitive affective syndrome. Brain. 1998;121:561-79.
7. Baron JC, Bousser MG, Comar D, et al. Crossed cerebellar diaschisis in human supratentorial brain infarction. Trans Am Neurol Assoc. 1980;105:459-61.
8. Botez MI, Léveillé J, Lambert R, Botez T. Single photon emission computed tomography (SPECT) in cerebellar diseases: cerebello-cerebral diaschisis. Eur Neurol. 1991;31:405-12.
9. Rousseaux M, Steinling M. Crossed hemispheric diaschisis in unilateral cerebellar lesions. Stroke. 1992;23:511-4.
10. Beldarrain MG, García-Moncó JC, Quintana JM, Llorens V, Rodeno E. Disachisis and neuropsychological performance after cerebellar stroke. Eur Neurol. 1997;37:82-9.

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HTLV-I- associated myelopathy: A new case in Spain

Mielopatía asociada a virus HTLV-I: un nuevo caso en España

Sir,

The HTLV-I virus was the first retrovirus described in human beings in the nineteen-eighties. It is considered to be the aetiological agent of leukaemia T in adults, tropical spastic paraparesis (TSP), or HTLV-I-associated myelopathy (HAM), uveitis, Sjögren's syndrome, lymphocytic alveolitis, and arthritis.

From an epidemiological perspective, there are between 10 and 20 million people in the world who are infected with the HTLV-I virus; of these, 3,000 have HAM/TSP. The risk of suffering HAM varies between 0.25% and 2.4% in HTLV-I seropositive individuals.[1,2] The virus is endemic in some areas of the Caribbean (Martinique, Jamaica, Trinidad), southern Japan, Central and South

America (mostly in Brazil and Colombia), Sub-Saharan Africa, the Middle East, Melanesia, and the Seychelles Islands. The main routes by which the HTLV-I virus is transmitted in endemic areas are: *a)* sexual; *b)* mother to child (breastfeeding), and to a lesser extent, *c)* blood transfusion, organ transplantation, and parenteral drug addiction.[3]

HAM is found largely in women and is generally diagnosed between the third and sixth decade of life[1]. The usual clinical picture is that of slowly progressing myelopathy characterized by predominantly proximal, asymmetrical, spastic paraparesis, together with disturbances of the sphincters and sexual dysfunction. Between 28 and 49% of the patients report treatment-resistant lumbalgia, with and without radiation to the lower limbs, as an early symptom of the disease.

In 1988, the World Health Organization drafted guidelines on recommendation criteria for the diagnosis of TPS/HAM, later revised in 1989, that made it possible to divide the diagnosis into definite and probable, depending on the clinical presentation and the presence of anti-HTLV-I antibodies in blood and cerebrospinal fluid (CSF).[4-6]

Later on, Brazilian neurologists met with international observers in Brazil in 2002 and 2004 and proposed an amended model for the diagnosis of TSP/HAM[5] that included degrees of diagnostic ascertainment.

Insofar as treatment is concerned, there is no aetiological treatment with clearly proven efficacy. Steroids and plasmaphoresis have been used during the acute phase; the effects of interferon α and β have been studied in patients who suffer from TSP/HAM, but without a clear effect being seen on the progression of the paraparesis or on the disability.

We report the case of a 46-year old Spanish male who consulted due to progressive weakness in his lower limbs. He reported lumbalgia for the last 2 years and in the previous 8 months; he had aggregated progressive weakness of the lower limbs, associated with urinary incontinence, constipation, and sexual dysfunction. As a disease history, two episodes of bilateral uveitis in the preceding two years are noteworthy. With no history of IV drug addiction, transfusions, or sexually-transmitted diseases; involved in a stable relationship for the last 2 years with a woman from South America.

Upon physical examination, moderate, asymmetric, spastic paraparesis was seen, predominantly on the right hand side, with hyperreflexia of the lower limbs, patellar clonus, and bilateral Achilles' clonus; indifferent right plantar skin reflex. He presented sensitivity at the level of D11, with hypoaesthesia and hypopalaesthesia in lower limbs. His gait was right-dominant pyramidal. It was also possible to see symmetrical tremor in the upper limbs at rest, posturing and in action.

The electromyographic examination showed findings compatible with bilateral carpian tunnel syndrome, without any other pathological signs. The magnetic resonance (MR) images of the skull and the cervical spine (fig. 1) were normal, whereas those of the dorsolumbar spine (fig. 2) showed a minimal disk protrusion at D7 without medullary or radicular involvement and L5-S1

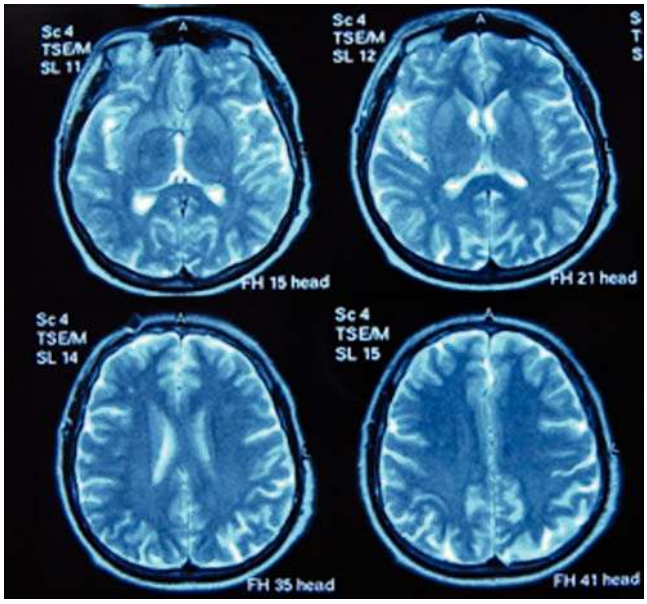

Figure 1 Magnetic resonance image of the skull, axial slice in T2 sequence, with no observable areas of hyperintensity in the white sub-cortical substance.

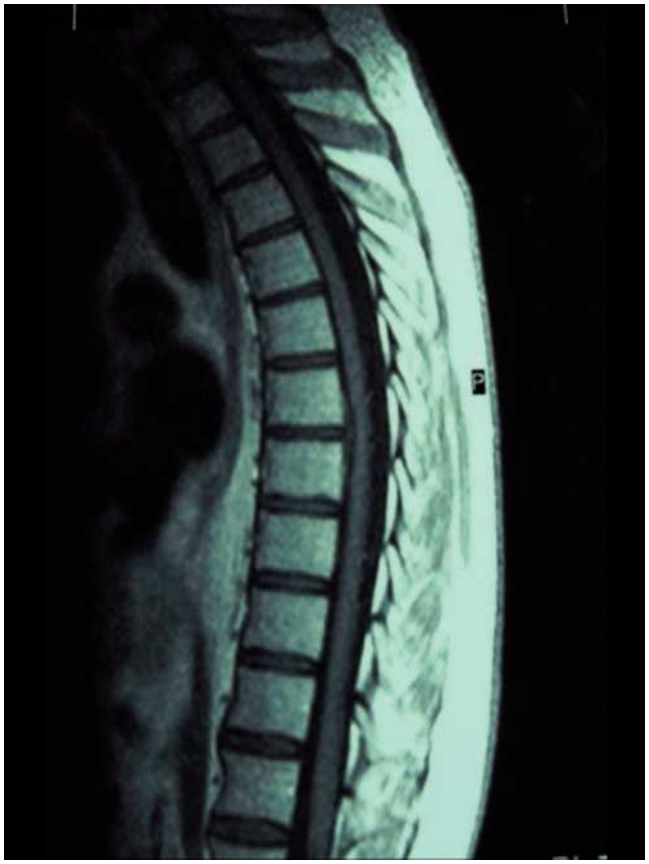

Figure 2 Magnetic resonance image of the dorsolumbar spine, sagittal slice in T2 sequence, with small disk protrusion of D7-D8.

disk hernia without ponytail or medullary cone involvement.

The biochemical study carried out did not reveal any alterations in thyroid hormones, vitamin B_{12}, folic acid, long-chain fatty acids or immunoelectrophoresis. The CSF analysis showed leukocytosis with a clear mononuclear predominance and mild proteinorachia. The serological studies in blood and CSF were negative for syphilis and HIV, but positive for HTLV I-II (his spouse turned out to be seronegative).

In Europe and the United States, the cases reported correspond to immigrants from endemic areas or autochthonous populations that have travelled to these regions or have had sexual relations with people of such origin.[3] In Spain to date, 7 cases of HAM/TSP have been reported; two were in immigrants from endemic areas, four were Spaniards who had lived in these areas and exact information is not available for the other case.[7-10] The method of transmission is also unknown in our patient.

In recent years, the increase in migratory flows to Europe from endemic areas may bring about an increase in the incidence of infections by the HTLV-I virus in the decades to come, as well as of the other illnesses associated with it.

References

1. Joseph R. Zunt. Co-infection: retrovirus and others CNS infections. American Academy of Neurology, 2007 Annual Meeting.
2. Kaplan JE, Osame M, Kubota H, Igata A, Nishitani H, Maeda Y, et al. The risk of development of HTLV-I- associated myelopathy/ tropical spastic paraparesis among persons infected with HTLV-I. J Acquir Immune Defic Syndr. 1990;3:1096-101.
3. Carod-Artal FJ, Mourao Mesquita H, Da SIveira Ribeiro L. Manifestaciones neurológicas y discapacidad en pacientes que padecen mielopatía asociada al HTLV-I. Neurología. 2007;22: 1-9.
4. Report of World Health Organization Scientific Group on HTLV-I infection and associated diseases. Kagoshima, Japan 10-15 december 1988.
5. Castro Costa C, Araujo A, Marreto M, Takayanagui O, Sohler M, Da SIva E, et al. Proposal for diagnostic criteria of tropical spastic paraparesis/ HTLV-I associated myelopathy (TSP/ HAM). AIDS Res Human Retrovir. 2006;22:931-5.
6. Salamano R, Valiño J, Savio E, Scaramelli A, Pietra M, Russi J, et al. Paraparesia asociada al HTLV-I. Una nueva enfermedad en Uruguay: a propósito de dos casos clínicos. Rev Med Uruguay. 1998;14:69-72.
7. Soriano V, Calerón E, Cilla G, Aguilera A, Merino F, Gutierrez M, Grupo Español para el Estudio de la Infección HTLV-I/II. Análisis multicéntrico de la infección por HTLV-I y HTLV-II en España. Med Clin (Barc). 1993;100:492-6.
8. Soriano V, Gutierrez M, Vallejo A, Aguilera A, Calerón E, Franco E, Grupo Español para el Estudio de la Infección HTLV-I/II. Infección por HTLV-I en España. Análisis de 2 casos identificados hasta noviembre de 1994. Med Clin (Barc). 1995;105: 246-50.
9. Gámez J, Matas L, Caballero E, Solans R, Cervera-Alvarez C, Rauer N, et al. Paraparesia espástica por HTLV-I: identificación temprana de un nuevo caso. Revisión de la casuística española. Neurología. 2001;16:172-5.
10. Carbone J. Activación crónica de células T y B en la mielopatía asociada al HTLV-I. Neurología. 2002;17:121-2.

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Atypical migraine progressing from nummular headache to epicrania fugax

Un caso de cefalea atípica con evolución de cefalea numular a *epicrania fugax*

Sir,

Pareja et al[1] recently reported a new headache syndrome or variant called epicrania fugax. We present here a further case of headache with similar characteristics and a peculiar evolution.

Twenty-three year old female with no prior illnesses of interest with the exception of infrequent tension headache. For the last few months, she has presented shooting pain on a specific point of her scalp of less than 1 cm in diameter and located in the left parietal area. Said pain lasted between 2 and 3 seconds, was moderate-severe in intensity, and was sometimes followed by mild pain for several hours. This occurred 3 to 4 times a day without any trigger. Neurological examination was normal, except for the presence of a hypersensitive point in the area of the pain. Haemogram showed an MCV of 77 and the magnetic resonance of the head was normal. Two months following her first visit, the characteristics of the pain were similar; however, in a new examination 5 months later, she reported changes in the previous few weeks: the pain occurred once or twice a day; it always radiated to the ipsilateral eye, occasionally accompanied by tearing and rarely radiated to the left ear. She had no symptoms between paroxysms. The remaining characteristics were similar: stabbing-type pain, moderate to severe in intensity and lasting for 2 to 3 seconds. She followed several treatments (gabapentin, tramadol, vitamin complexes, flunarizine) with no clear response to any of them (slight temporary improvement with gabapentin and flunarizine).

Our patient is a young woman who debuted with stabbing, epicranial pain lasting very few seconds, at a single point on her scalp innervated by the first trigeminal branch, moderate-severe in intensity, with no other symptoms, which occurred several times per day. Between paroxysms, she began presenting mild pain in