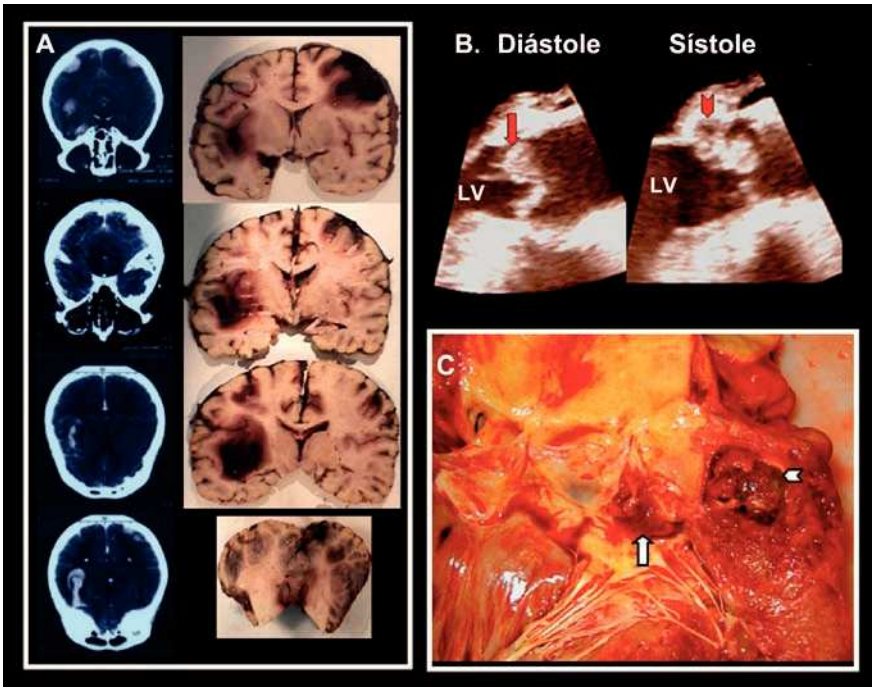

Figure 1 A: Computed tomography and microscopic encephalic slices revealing multiple haemorrhagic areas (supra- and infratentorial regions). B: Large growth on the left coronary aortic sigmoid (arrow), complicated by the presence of perivalvular abscess (arrowhead) and valvular perforation. Trans-oesophageal echocardiogram: the longitudinal image of the aortic root at the level of the valvular plane reveals the aortic growth protruding into the outflow tract of the left ventricle in diastole. C: Necroscopic image: longitudinal opening of the aortic root at the level of the valvular plane showing these same findings.

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Aphasia secondary to left cerebellar infarction

Afasia secundaria a infarto cerebeloso izquierdo

Sir,

The cerebellum is currently considered to modulate several different cognitive processes, including language.[1] Several cases of severe dysarthria grave, agrammatism or mutism secondary to acute cerebellar injuries. Aphasia secondary to cerebellar injuries has been known as crossed cerebral-cerebellar diaschisis, because it has been attributed to the influence exerted by the right cerebellum on the contralateral prefrontal cortical areas by means of the cerebello-pontine thalamo-cortical pathways.[2-4] However, there are scant references to language disorders owing to injuries of the left cerebellar hemisphere.[5]

In the present article, we report the case of a patient who presented acute aphasia after suffering a left cerebellar infarction, and who developed cognitive

impairment months later. This case suggests that both cerebellar hemispheres, and not only the right cerebellar hemisphere, can affect language regulation, as well as other higher functions.

We present the case of an eighty-three year old, right-handed male with no prior medical history of note or prior cognitive impairment. The patient was admitted due to a week-long clinical syndrome consisting of difficulty to express himself, disorientation, and nervousness. The onset of symptoms had been sudden and was associated with a left hemicranial headache. The systemic examination was unremarkable; he was afebrile and had normal blood pressure. The patient maintained a good degree of consciousness. Of note was the fact that his speech was slow and fairly inarticulate, with frequent mistakes in naming. He understood simple orders, although he was unable to do so when faced with several sequences. He also found it very difficult to read and write. The patient presented no other neurological focality, except for minimal lateralization toward the left on walking. The clinical diagnosis upon admission was ischaemic stroke in the territory of the left middle cerebral artery (LMCA). Nevertheless, the CAT scan of the brain performed in the Emergency Room revealed left cerebellar hypodensity without mass effect compatible with recent ischaemia; hence, it was hypothesized that there had been several simultaneous ischaemias in different territories, possibly due to an embolus. The blood test, ECG, and chest X-ray were all normal. The cerebral MRI (fig. 1) confirmed the existence of acute ischaemia in the left cerebellar hemisphere, but failed to reveal other hyperintense areas in the diffusion study or old injuries in either hemisphere. The patient was given AAS for antiaggregant treatment and sent home.

After being released from hospital, the language disorder persisted, with the addition of progressive cognitive impairment in the form of greater disorientation, apathy, stiff and obsessive character, aggressiveness, and daytime hypersomnia. The patient required help for tasks such as getting dressed or personal hygiene. A new cerebral MRI ruled out the presence of new injuries or hydrocephaly secondary to compression of the IV ventricle. In the assessment carried out two months later, relatively inarticulate speech continued with anomia and agrammatism. Both reading and writing had improved, and verbal and written comprehension was virtually recovered. The patient continued to be temporally and spatially disoriented, although his short-term memory remained intact. He was collaborative with the physician during the visit, although he was irritable when comments were made by relatives. He scored 22/30 on the Mini Mental Test.

The cerebral PET (fig. 2) ordered revealed predominantly temporo-parietal hypometabolism at the left cerebellar and bilateral cortical level. The patient was treated with neuroleptics, with improvement of the behaviour disorders, although his cognitive impairment has persisted.

In 1998, Schmahmann and Sherman published what they called the "cognitive-affective cerebellar syndrome" in a group of patients with different kinds of cerebellar injuries.[6] This disorder includes impairment of executive functions, visual disorganization, personality changes, and language disorder (dysprosodia, agrammatism, and anomia). The hypothesized explanation for these alterations is the de-activation of the prefrontal area of the left hemisphere secondary to the loss of excitatory impulses coming from the cerebello-pontine thalamo-cortical pathways.

Figure 1 Diffusion magnetic resonance image revealing hypersignal in the left cerebellar hemisphere compatible with acute ischaemia.

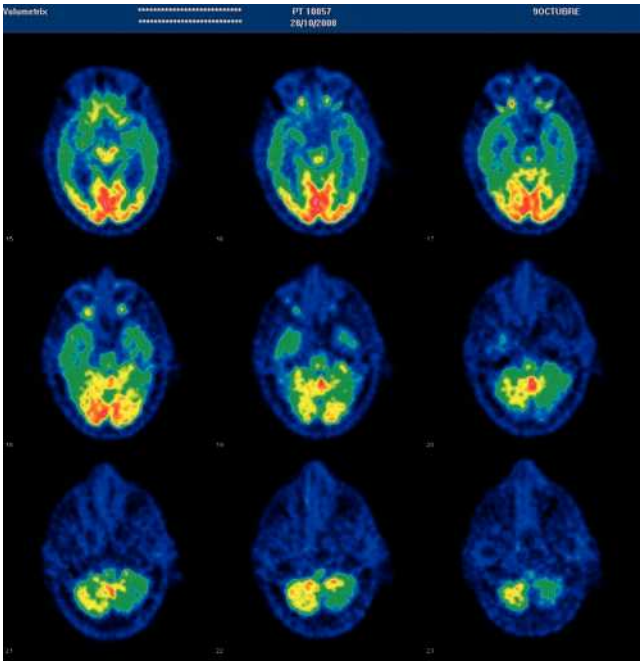

Figure 2 Brain PET performed months after the stroke. Hypometabolism is seen in the left cerebellar and cortical (bilateral and diffuse) areas with a fairly symmetrical, predominantly temporo-parietal distribution.

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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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