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LETTERS TO THE EDITOR

Reversible posterior leucoencephalopathy in a patient with spinal cord injury

Leucoencefalopatía posterior reversible en un paciente con lesión medular

Dear Editor:

Patients with spinal cord injury at T6 or a higher level often suffer autonomic dysreflexia. This problem can affect up to 30%90% of high paraplegic and quadriplegic patients, according to different series.

Autonomic dysfunction in these patients responds to the loss of normal supraspinal control, but the reorganization of synapses and neuronal plasticity in the injured spinal cord also has an influence. Clinically, it is manifested as hypertension, sweating, headache and bradycardia. Dysreflexia crises are often triggered by stimuli such as pressure ulcers, urinary retention or rectal distension. The most troubling component of these cases are hypertensive crises with potential risk to life, which require intensive, emergency management.[1]

We report the case of a 65-year-old male with traumatic cervical myelopathy at C4 level, ASIA B. The patient presented a sudden loss of vision in both eyes accompanied by blood pressure of around 200/100mmHg. His level of consciousness deteriorated within a few hours, having no response to stimuli, as well as a general-type crisis. The patient was afebrile with negative meningeal signs. We noted squinting to the right side and absence of visual threat reflex.

The EEG showed continuous delta activity in posterior regions without epileptiform activity. T2 and FLAIR sequences in the cranial MRI scan showed multiple hyperintense images in the white matter of the pons, cerebellar hemispheres and both occipital lobes (fig. 1).

The hypertension was difficult to control and required the use of captopril, furosemide, amlodipine and labetalol at full doses. The patient improved, with a normalization of the level of consciousness, vision and ocular motility in the first 24h of evolution. Subsequently, on two separate occasions, he presented two-hour episodes of blurred vision and bradypsychia, which coincided with uncontrolled blood pressure.

A control cranial magnetic resonance imaging scan one month later showed complete remission of the hyperintense images found in the initial study (Fig. 2).

The diagnosis was reversible posterior leukoencephalopathy related to autonomic dysreflexia.

Reversible posterior leukoencephalopathy is a recently described clinical and radiological entity (Hinchey et al, 1996). The diagnosis is very descriptive, as it is a process that settles in the white matter (although it often affects the grey matter as well), has a clear tropism for the posterior brain regions (although there are cases of atypical distribution) and in most cases ends in resolution of the clinical symptoms and image alterations.[2]

It has been related with many causes, among which are arterial hypertension, particularly in the context of renal failure and pregnancy. Other causes include immunosuppressive drugs such as cyclosporine, cytostatic drugs such as cisplatin, blood diseases such as thrombotic thrombocytopenic purpura, connective tissue disorders and vasculitis.[3,4]

The association of this entity with autonomic dysreflexia in the context of spinal cord injury is very unusual. In the largest series published to date, which included 36 patients, 2 cases occurred in quadriplegic patients with C5 spinal cord injuries. There is another case reported in a patient with a C5 lesion.[5,6]

The clinical spectrum includes headache, seizures, alterations of consciousness, focal deficits and visual impairment ranging from blurred vision to cortical blindness. The pathophysiological basis is vasogenic oedema. In cases linked to hypertension, it is due to cerebral autoregulation failure, leading to arteriolar vasodilatation and blood-brain barrier breakdown. The propensity for the posterior territories is justified by the lesser sympathetic innervation of the vertebrobasilar territory with a more precarious autoregulation.[7]

The characteristic findings in magnetic resonance imaging are hyperintense images in the T2 and FLAIR sequences, predominantly in the subcortical white matter of the posterior regions. Diffusion studies (DWI) and apparent diffusion coefficient (ADC) maps distinguish the vasogenic oedema characteristic of this entity from the cytotoxic oedema typically found in cerebral stroke.[8]

Cases of reversible posterior leukoencephalopathy are very rare in patients with cervical or high dorsal spinal cord injury. However, the physician should maintain a high index of suspicion because its early recognition and proper treatment make complete patient recovery possible.

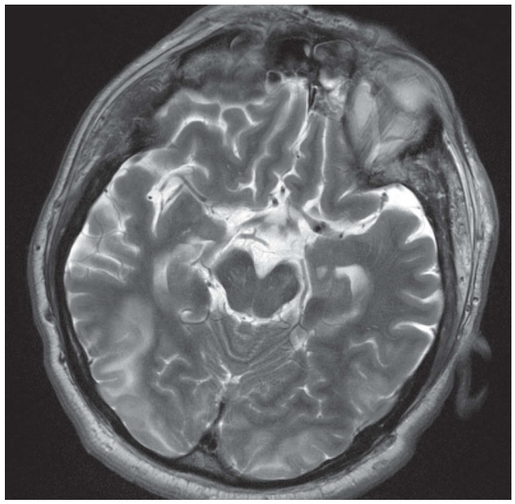

Figure 1 T2 axial MRI scan conducted at the beginning of the condition, showing hyperintense images in both occipital lobes, more marked on the right side.

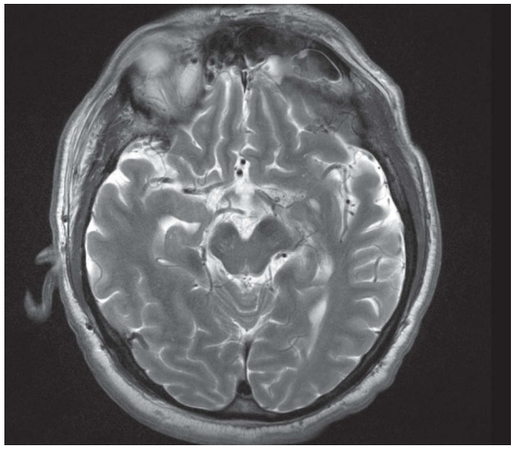

Figure 2 T2 axial MRI scan, showing resolution of the previous image alterations.

References

1. Mallory B. Autonomic Dysfunction in Spinal Cord Disease. In: Lin VW, Cardenas DD, Cutter NC, Frost FS, Hammond MC, Lindblom LB, et al., editors. Spinal cord medicine. Principles and practice. New York: Demos Medical; 2003. p. 477-500.
2. Hinchey J, Chaves C, Appignani B, Breen J, Pao L, Wang A, et al. A reversible posterior leukoencephalopathy syndrome. N Engl J Med. 1996;334:494-500.
3. López-García F, Amorós-Martínez F, Sempere AP. Síndrome de leucoencefalopatía posterior reversible. Rev Neurol. 2004; 38:261-6.
4. Caminero AB. Síndrome de encefalopatía posterior reversible. Neurología. 2005;20:327-31.
5. Lee VH, Wijdicks EF, Manno EM, Rabinstein AA. Clinical spectrum of reversible posterior leukoencephalopathy syndrome. Arch Neurol. 2008;65:205-10.
6. Chaves CJ, Lee G. Reversible posterior leukoencephalopathy in a patiente with autonomic dysreflexia: a case report. Spinal Cord. 2008;46:760-1.
7. Schwartz RB, Mulkern RV, Gudbjartsson H, Jolesz F. Diffusionweighted MR imaging in hypertensive encephalopathy: clues to pathogenesis. AJNR. 1998;19:859-62.
8. Covarrubias DJ, Luetmer PH, Campeau NG. Posterior reversible encephalopathy syndrome: Prognostic utility of quantitative Diffusion-Weighted MR images. AJNR. 2002;23: 1038-48.

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Convulsive status epilepticus associated with a tramadol overdose

Crisis epiléptica convulsiva relacionada con sobredosis de tramadol

Dear Editor:

We describe the case of a 15-year-old male without previous history of epilepsy who presented tonic-clonic seizures during the day, followed by refractory convulsive status epilepticus (CSE). The family and perinatal history contained no relevant data. At age 9 he was diagnosed with osteoid osteoma. As he was not a candidate for surgical intervention, treatment with non-steroidal anti-inflammatory drugs (NSAIDs) was started; normally, naproxen treatment was sufficient to relieve the pain. Two weeks before admission, he presented an exacerbation of pain that showed no response to NSAIDs and led to tramadol treatment being started. After an oral dose of 25mg, he suffered complex verbal auditory hallucinations involving commands and suggestions, paranoid delusions and finally confusion psychosis. The symptoms were resolved within 24h without medical intervention. His mother stopped the administration of tramadol, but on