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Migraine-triggered hemifacial spasm: Another case study[☆]

Espasmo hemifacial desencadenado por migraña: un caso más

Dear Editor:

Hemifacial spasm (HFS) is characterised by involuntary tonic or clonic contractions of muscles innervated by the facial nerve. The pathogenesis of this condition is usually attributed to vascular compression at the emergence of the nerve root from the brainstem. This could be due to the appearance of ectopic discharges and the ephaptic transmission of abnormal impulses. Another potential mechanism could be central hyperexcitability with neural impairment in the facial nerve motor nucleus. To date, 5 cases of HFS caused by migraine have been published.¹⁻³

We present the case of a woman aged 30 with no relevant personal history who had experienced migraine attacks without aura since adolescence. In the previous year, headache episodes became more intense and were sometimes preceded by visual aura. Pain always presented on the right side of the head; upon reaching its maximum intensity, it was accompanied by involuntary contractions of the periocular ipsilateral muscles. Contractions decreased

as pain subsided and only appeared during migraine attacks. These episodes occurred an average of 4 times per month. Initial neurological examination was normal. However, during one of the migraine episodes, we were able to observe the contractions of the right periocular muscles described above. Electromyography (EMG) showed tonic activity of the right orbicularis oculi with high-frequency bursts of increased muscle fibre recruitment (Fig. 1) coinciding with clonic spasms. The study of facial nerve motor conduction and blink reflex showed no relevant abnormalities. Brain MRI and MRA revealed a loop of the right anterior inferior cerebellar artery in contact with the facial nerve (Fig. 2). Treatment with topiramate was prescribed at increasing doses reaching 75 mg per day, which decreased the frequency and intensity of migraine episodes and suppressed the associated muscle spasm.

The temporal relationship between migraine and HFS in the 6 published cases supports the idea of a pathophysiological link existing between those processes. As in prior cases,¹⁻³ our patient presented episodes of HFS that coincided with maximum pain intensity. Therefore, rather than being a migraine aura phenomenon with positive signs, HFS seems to be a consequence of migraine. Husid raised the possibility that HFS could be due to a mechanism of central hyperexcitability related to migraine or to facial nerve compression caused by the dilation of nearby vessels,

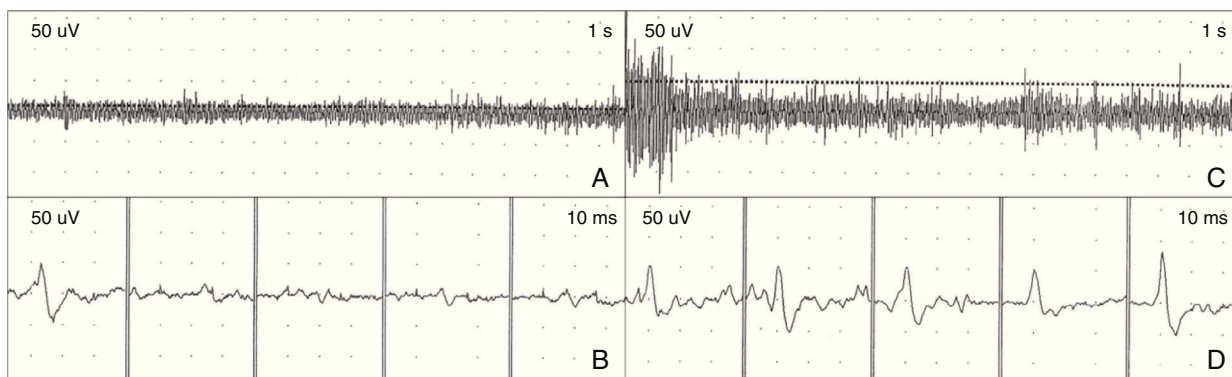


Figure 1 Electromyogram showing spasm of the right orbicularis oculi during a migraine episode. (A, B) Baseline tonic muscle activity coinciding with tonic contraction and reduced palpebral fissure. (C, D) High-frequency bursts of muscle fibre recruitment coinciding with clonic spasms of the upper and lower eyelids.

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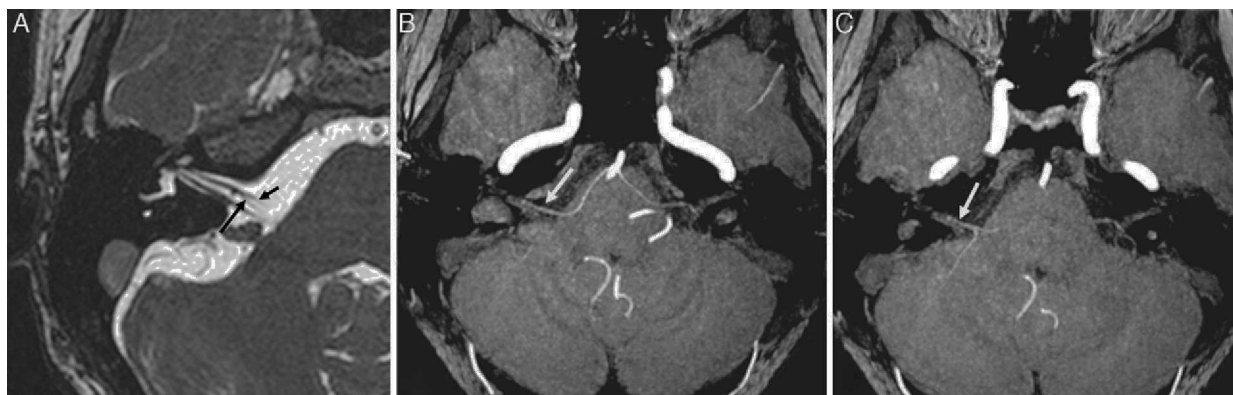


Figure 2 High-resolution MRI at the level of the cerebellopontine angle. (A) 3D-FIESTA MR image (three-dimensional fast imaging employing steady-state acquisition) showing vascular contact (long arrow) with the right facial nerve (short arrow). (B, C) Three-dimensional time-of-flight MRA (3D-TOF MRA) showing a vascular loop of the right anterior inferior cerebellar artery (arrow) in the internal auditory canal.

which in turn is produced by the trigeminovascular system in cases of migraine attacks.² On the other hand Cuadrado et al. suggested that potentiation of certain trigeminofacial reflexes could cause HFS during a migraine episode.³ Barahona et al. combined all these hypotheses by proposing that central hyperexcitability phenomena, along with activation of trigeminofacial reflexes due to nociceptive stimuli arriving in the caudate nucleus of the trigeminal nerve, plus the possible compression of the facial nerve caused by the dilation of vessels that are in contact with the nerve, may provoke episodes of HFS during migraine attacks in predisposed patients.¹

We believe that migraine-triggered HFS, like migraine-triggered seizure, could be included among the complications of migraine headaches.⁴ Both central and peripheral mechanisms may be involved in the pathogenesis of this condition.

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Respect for dementia patients' autonomy and the need for interdisciplinary cooperation[☆]

Respeto a la autonomía del paciente con demencia y la necesidad de colaboración interdisciplinar

Dear Editor:

It was with great interest that we read Dr. Álvaro's compelling review on competency in dementia patients,

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published in *Neurología*.¹ Beginning with an overview of the philosophical and legal background of the concept of informed consent (IC), he alerts us to the risk that IC might substitute key elements of doctor-patient interaction and be thought of as a mere legal tool.

Obtaining a patient's IC as a general rule has been investigated as a possible obstacle to routine clinical activity since the requirement first entered into force and up to the present day. Studies cite, for example, the difficulties it poses for research involving patients with dementia.^{2,3} Nevertheless, Lidz, Appelbaum, and Meisel stated in 1988 that the problem was not the doctrine of IC, but rather, the way that doctrine was typically implemented.² Social change was followed by legislative change, and as of the 1990s, Spanish autonomous communities such as the Canary Islands (11/1994), Andalusia (2/1998) and Catalonia (21/2000) had introduced laws providing for informed consent. This current culminated with the enactment of Law 41/2002 for the regulation of patient autonomy and the rights and obligations