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## Migralepsy; a controversial entity

### Migralepsia, una entidad controvertida

*Dear Editor,*

The term migralepsy was used for the first time in 1960 to describe a case of ophthalmic migraine followed by typical symptoms of epileptic seizure.[1] But it is not until 2004 that migraine-triggered epilepsy or migralepsy was included (under point 1.5.5) in the 2nd edition of the International Classification of Headache Disorders (ICHDII)[2] where it is defined as an epileptic seizure occurring during or within 1 hour after a migraine aura.

We report here the case of a 15-year-old female with a family history of migraine with aura (father and grandmother). From the age of 10 years, the patient had presented paroxysmal seizures with a visual aura in the form of flashes of light and zigzagging lines in the centre of her field of vision, moving slowly and crossing over each other. After 20 or 30 minutes, she presented clonic movements on the right hand side of her body, occasionally extending into generalized seizures in which she lost consciousness, with tonic-clonic jerking in all four limbs. These seizures continued with intense left hemicranial cephalea accompanied by photophonophobia and nausea. She was initially diagnosed as having migraine with aura and treatment was begun with gabapentin at a dose of 300 mg every 12 h. Subsequently, an electroencephalogram (EEG) was performed while she was awake and this revealed an acute left temporal focus; she was then diagnosed as having complex focal epilepsy and treatment was begun with levetiracetam 2,000 mg/day. At age 15, she was once more admitted to our department following a new episode. Magnetic resonance (MR) scan of the brain presented no alterations. The EEG in vigil revealed persistent acute activity in the left temporal region figure 1. The patient and her family reported 1 to 2 episodes per month despite good compliance with medication. In view of the diagnosis of migraine-induced epilepsy, it was decided to replace her treatment with 600 mg per day of valproic acid; after 8 months of follow-up, total remission of her clinical symptoms has been achieved, with a slight asymmetry in the check-up EEG in vigil.

The theory most commonly accepted with respect to the pathophysiological substrate of migraine aura is the phenomenon of propagated cortical depression. This phenomenon consists in a wave of neuronal and glial depolarization extending from the visual area throughout the occipital pole (visual aura) and, less frequently, to the parietal sensory cortex (sensory aura) and the motor area (motor aura); thus, hypoperfusion/hypometabolism occurs in the area.[3] This cortical depolarization is capable of activating the trigeminal-vascular system, inducing the release of vasoactive peptides (CGRP and VIP) in the leptomeningeal area, giving rise to vasodilatation and sterile inflammation causing migraine pain.[4] Migraine aura and epilepsy share a common pathophysiological substrate. On the one hand, depolarization processes are activated during the propagated depression phenomenon, involving stimulant amino acids and their receptors (glutamate and NMDA receptors) which also participate actively in epileptic seizures. On the other hand, the biochemical mechanism of propagated depression gives rise to a state of local hyperexcitability with certain similarities with the increased stimulant tone involved in the pathophysiology of epilepsy.[5] Therefore, in both entities there is a state of cerebral hyperexcitability that would be more marked in epilepsy than in migraine aura and intermediate in migraine-induced epilepsy.

There is evidence that migraine *per se* may represent an epileptic seizure and may even in some cases be the sole manifestation of epilepsy.[6] For this reason, it is recommended to perform an EEG during and between migraine crises in selected patients with prolonged episodes of migraine or poor response to treatment.[7]

Our patient presented recurrent bouts of focal epileptic crises in the course of her visual migraine auras and fulfilled the current diagnostic criteria of the ICHDII for migraine-induced epilepsy (table 1). There are some discrepancies with respect to these criteria, as set out in the papers by Maggioni et al.,[8] who present several cases of epileptic seizures preceded by migraine without aura and propose to include epileptic seizures triggered by a migraine with or

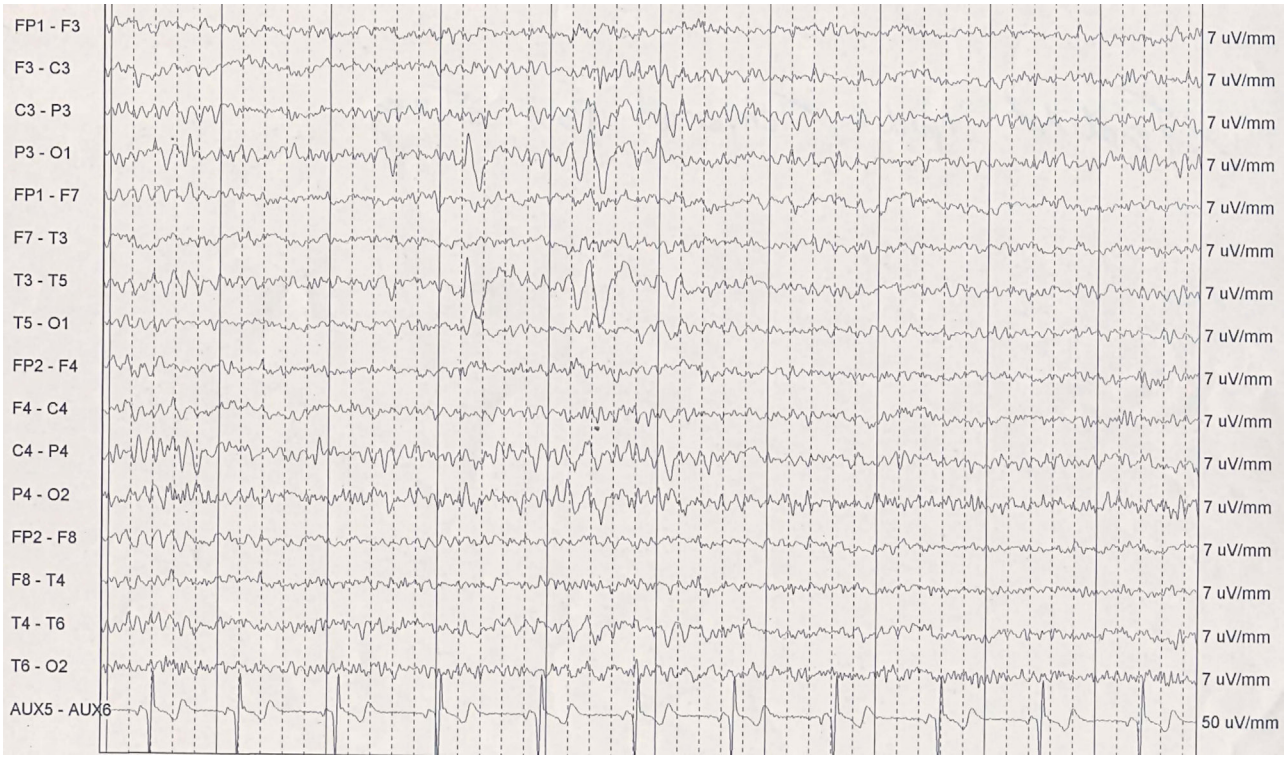


**Figure 1** Electroencephalogram of vigil: acute left temporal focus.

**Table 1** International Classification of Headache Disorders II (ICHD-II)

| |
|---|
| Hemicrania epileptica (7.6.1) |
| **Diagnostic criteria:** |
| Headache lasting seconds to minutes, with features of migraine, fulfilling criteria C and D |
| The patient is having a partial epileptic seizure |
| Headache develops synchronously with the seizure and is ipsilateral to the ictal discharge |
| Headache resolves immediately after the seizure |
| Post-ictal headache (7.6.2) |
| **Diagnostic criteria:** |
| Headache with features of tension-type headache or, in a patient with migraine, of migraine headache and fulfilling criteria C and D |
| The patient has had a partial or generalised epileptic seizure |
| Headache develops within 3 hours following the seizure |
| Headache resolves within 72 hours after the seizure |
| Migraine-triggered seizures (migralepsy) (1.5.5) |
| Description: a seizure triggered by a migraine aura |
| **Diagnostic criteria:** |
| Migraine fulfilling criteria for 1.2 Migraine with aura |
| A seizure fulfilling diagnostic criteria for one type of epileptic attack occurs during or within 1 hour after a migraine aura |

without an aura within the term migraine-induced epilepsy. They conclude that both entities have a shared pathophysiological substrate as functional MR and magnetoencephalography studies have shown the presence of silent cortical depression in cases of migraine without aura.[9] Sances et al.[10] reported a case and identified in the literature a total of 50 potential cases of migraine-induced epilepsy but, following a systematic review, only 2 (6%) fulfilled the diagnostic criteria for migralepsy. The main diagnostic errors are that most of the cases published as migraine-induced epilepsy are actually occipital crises with post-ictal cephalea.

Transient alterations were observed in the cerebral MR images: hyperintensity in the T2-weighted fluid-attenuated inversion recovery (FLAIR) and proton density sequences of patients with migraine-induced epilepsy.[11] The hypotheses put forward to explain this phenomenon include the interruption of the blood-brain barrier, parenchymatous and meningeal hyperperfusion, oedema and vasospasm. These alterations, which may persist for several days (from 1 to 3 weeks), might be caused by reversible neuronal dysfunction and must not be confused, in any way, with false lesions of the cerebral parenchyma.

Our case shows the importance of a precise diagnosis, given that patients with migraine-induced epilepsy may benefit from low doses of "dual anti-epileptics", thus giving optimum control of the episodes of migraine and epilepsy with a single drug. From our point of view, it would be necessary to review the criteria for migraine-induced epilepsy as it might be an under-diagnosed entity due to the current strict diagnostic criteria.

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# Diffusion tensor tractography in vanishing white matter disease ☆

## Tractografía por tensor de difusión en un síndrome de la sustancia blanca evanescente

*Dear Editor,*

Letters published in earlier issues of this journal by Pato Pato et al.[1] and Piñeiro et al.[2] have aroused great interest and we agree with the possibility that this condition might be under-diagnosed. In this sense, we should like to describe a case with similar characteristics at our unit and we contribute our experience with diffusion tensor tractography, a new technique that might be useful in the diagnosis of this entity.

An 18-year-old male from Seville, without any history of consanguinity between his parents, suffered cranioencephalic trauma following a motorcycle accident without wearing a helmet and, in consequence, weakness in the right limbs, for which reason he was admitted to our department. His personal history of note included cranioencephalic trauma at 8 years of age which caused right hemiparesis and required admission to the intensive care unit for 10 days with total recovery on discharge. The neurological examination revealed proportional right hemiparesis (3/5) with pyramidalism and gait ataxia. The rest of the neurological and general examination was normal. The general analyses performed, including study of thyroids and lipids, were normal; cranial CT scan revealed a dilatation of the ventricular system and the magnetic resonance (MR) of the skull showed, in addition, extensive hyperintense areas in T2 of the white matter at the supratentorial level, affecting all lobes and the posterior arms of both internal capsules, compatible with demyelinization. Also noteworthy was generalized cortical-subcortical atrophy and marked thinning of the corpus callosum with cystic areas inside (fig. 1). Using diffusion tensor tractography, it was possible to observe the absence of crossed fibres in the central portion of the corpus callosum and an anomalous arrangement of the same in the genu, the cingulate gyrus and the splenium

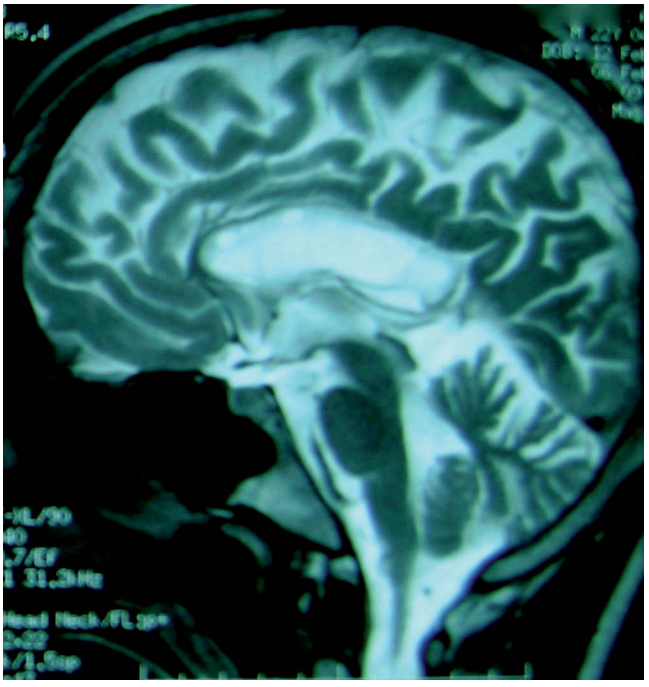

**Figure 1** Magnetic resonance image with marked thinning of the corpus callosum with cystic areas inside (T2 sequence).

☆ This paper was partially presented as a Poster at the 32nd Annual Meeting of the Andalusian Neurology Society.