Cephalalgia
An International Journal of Headache
International Headache Society

*Cephalalgia*
32(16) 1227–1229

DOI: 10.1177/0333102412462641
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# Ophthalmoplegic migraine: Could electroencephalogram still be a useful tool to better understand the pathogenetic mechanism?

Dear Sir

We read with great interest the Editorial by Chen and Wang 'Ophthalmoplegic migraine: Migraine variant or cranial neuralgia?' (1), published in a recent issue of *Cephalalgia*, regarding ophthalmologic migraine (OM) pathogenesis. This question emerges periodically, in particular after reports in which different pathologic conditions resemble a primary OM attack such as the one discussed in the Editorial (2); so that the peripheral or central pathogenesis and consequently the classification of OM still remains an unresolved issue.

This is also evident when looking at the literature. Starting from 1980, we find the following titles: 'Ophthalmoplegic migraine: Ischemic or compressive neuropathy?' (3); in 2000 'Ophthalmoplegic migraine: A recurrent demyelinating neuropathy?' (4); in 2002 'Oculomotor ophthalmoplegic migraine: Is it really migraine?' (5); in 2004 'Ophthalmoplegic migraine migrainous or inflammatory?' (6); in 2006 'Ophthalmoplegic migraine: An unresolved problem' (7); in 2007 'Is migraine with cranial nerve palsy an ophthalmoplegic migraine?' (8) and 'Ophthalmoplegic migraine: Inflammatory neuropathy with secondary migraine?' (9); in 2008 'Ophthalmoplegic migraine: Still a diagnostic dilemma?' (10); in 2009 'Ophthalmoplegia with migraine in adults: Is it ophthalmoplegic migraine?' (11); and lastly in 2012 'Ophthalmoplegic migraine: Migraine variant or cranial neuralgia?' (1).

As reported in the Editorial (1), there are aspects that point to a peripheral pathogenesis, but not all the patients present a magnetic resonance imaging (MRI) suggestive of cranial nerve involvement; therefore there are several proposals – different from the current ICHD-II criteria (12) – for a new OM classification that will be discussed in the next International Classification of Headache Disorders. In 2008, Ravinshankar (10) proposed the possibility of identifying a primary OM both with and without MRI ocular motor nerves contrast enhancement; by contrast Friedman, in a 2010 editorial in *Cephalalgia* (13), proposed to consider as primary only the OM attacks in which the affected cranial nerve does not demonstrate contrast enhancement.

Recently, a 23-year-old woman presented to our emergency room, with a history of OM attacks. She had the first attack at the age of 8 years, then she presented during her life one or two migraine attacks each year accompanied by reversible diplopia lasting from 2 to 20 days. She also suffers from migraine without aura with a frequency of about one or two attacks per month; thorough investigations, comprehensive of cerebral digital angiography, found no abnormalities. She came to our attention for an excruciating migraine attack lasting 48 hours fulfilling the ICHD-II 2004 criteria (12), accompanied by diplopia that occurred about 24 hours after the beginning of the attack. Neurologic examination showed a left trochlear cranial nerve palsy.

Cerebral MRI with gadolinium enhancement, performed 3 days after the migraine onset, was normal, whereas an electroencephalogram (EEG) evidenced bursts of diffuse slowing rhythm (Figure 1(a) and (b)). Another EEG performed after a week, when the painful phase had resolved but diplopia was persisting, was still abnormal (Figure 1(c)), whereas a month follow-up EEG, after the complete resolution of diplopia, was normal (Figure 1(d)).

In the literature, there are reports on EEG abnormalities in particular in migraine with aura or in 'complicated migraine attacks' in particular basilar migraine (14,15), supporting cortical spreading depression and the involvement of central nervous system. Further, recent evidence confirms EEG slowing during a migraine attack, related to severity of symptoms, indicating thalamo-cortical hypoexcitability associated with migraine attack (16). With regard to OM, early reports have been studied with EEG but not with neuroimaging (17), whereas subsequently with the widespread use of the neuroimaging in the diagnostic process, reports have been described of neuroimaging without EEG (11). Consequently, patients studied with EEG are very few and a comparison of the two diagnostic procedures in the same patient is rare.

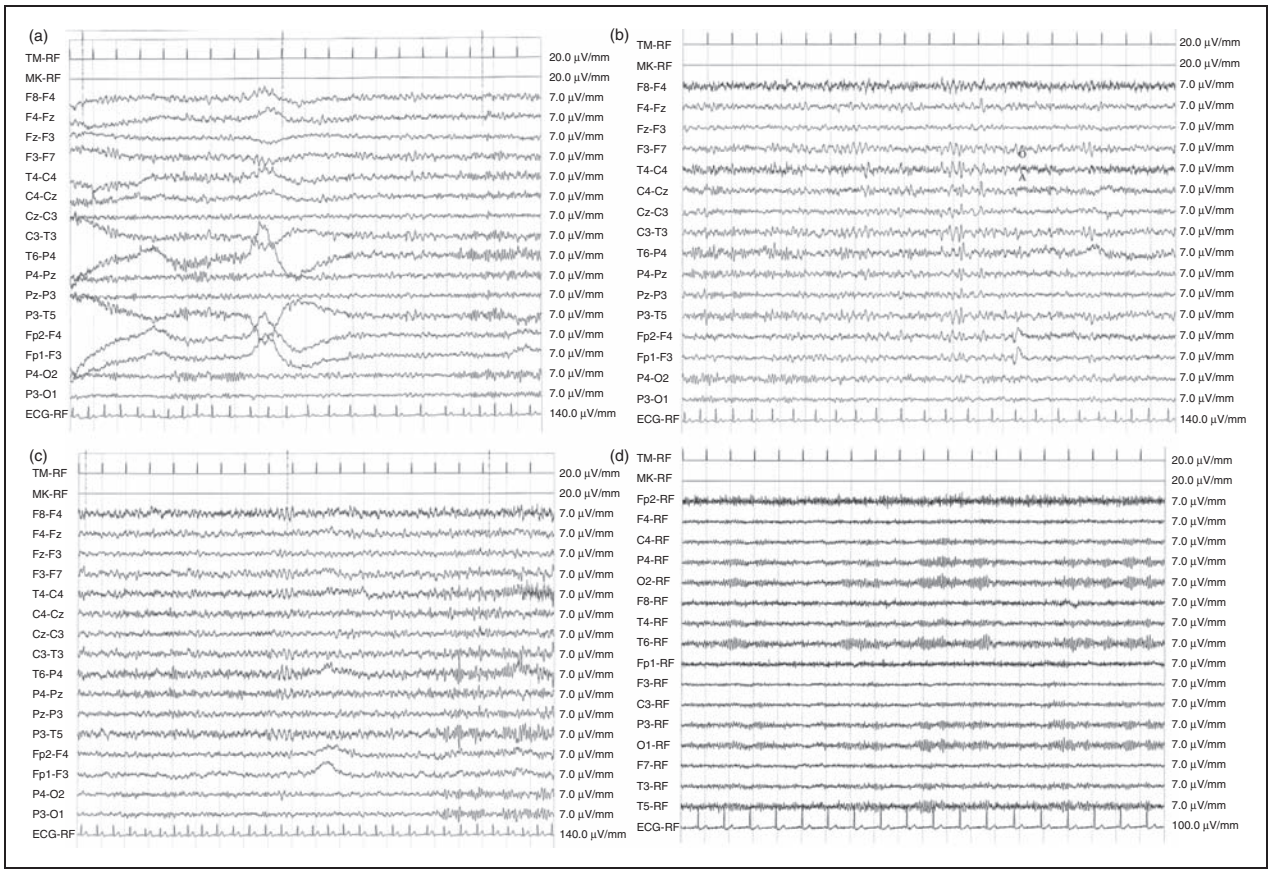


**Figure 1.** Electroencephalogram performed during ophthalmoplegic migraine attack (a, b), showing bursts of diffuse theta activity; after a week during diplopic phase without pain (c), with the persistence of theta activity bursts but with a better organization of the rhythm. Follow-up a month later, without clinical and subjective symptomatology, showing a normalized EEG (d).

The EEG findings in our patient with OM seem to indicate a pattern resembling migraine with aura or complicated migraine attacks.

We believe that EEG could still be useful in OM patients allowing identification of critical abnormalities regardless of MRI abnormalities, that could be helpful both to clarify a central OM pathogenetic hypothesis, and to identify a possible subgroup of primary OM, as proposed by Ravinshankar (10).

Finally, we agree with Chen and Wang that 'more OM case collections and reports are needed to resolve the classification issue of migraine variant or cranial neuralgia' (1).

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