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# Headache Following Occipital Brain Lesion: A Case of Migraine Triggered by Occipital Spikes?

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## Abstract

This study describes the case of an 8-year-old boy who developed a genuine migraine after the surgical excision, from the right occipital lobe, of brain abscesses due to selective infestation of the cerebrum by *Entamoeba histolytica*. After the surgical treatment, the boy presented daily headaches with typical migraine features, including right-side parieto-temporal pain, nausea, vomiting, and photophobia. Electroencephalography (EEG) showed epileptiform discharges in the right occipital lobe, although he never presented seizures. Clinical and neurophysiological observations were performed, including video-EEG and polygraphic recordings. EEG showed “interictal” epileptiform discharges in the right occipital lobe. A prolonged video-EEG recording performed before, during, and after an acute attack ruled out ictal or postictal migraine. In this boy, an occipital lesion caused occipital epileptiform EEG discharges without seizures, probably prevented by the treatment. We speculate that occipital spikes, in turn, could have caused a chronic headache with features of migraine without aura. Occipital epileptiform discharges, even in absence of seizures, may trigger a genuine migraine, probably by means of either the trigeminovascular or brainstem system.

## Keywords

electroencephalogram, epilepsy, migraine, wavelet analysis, brain abscess, occipital lobe, *Entamoeba histolytica*

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## Introduction

Migraine and epilepsy are linked by very close symptom profiles, and have common treatment strategies; moreover, it has been demonstrated that the presence of one disorder increases the likelihood of the other.<sup>1</sup> Migraine-like headache is frequently associated with childhood epilepsies,<sup>2</sup> and in particular with occipital lobe epilepsy,<sup>3</sup> both of idiopathic<sup>4,5</sup> or symptomatic origin.<sup>6,7</sup> Among the idiopathic occipital epilepsies, the late-onset variant, or “Gastaut type”,<sup>4</sup> is more frequently associated with migraine<sup>7,8</sup> than the early onset or “Panayiotopoulos type.”<sup>5</sup>

Headache associated with epilepsy can occur as an ictal<sup>8</sup> or postictal phenomenon.<sup>3,9</sup> Postictal headache, following late-onset idiopathic occipital lobe epilepsy, can occur even after a simple visual seizure and may present as a diffuse or unilateral severe, pulsating pain; for these reasons, the occipital seizure may be associated with migraine and clinical features which make them virtually indistinguishable from genuine migraine.<sup>10</sup>

We describe the case of an 8-year-old boy who developed a migraine-like headache following surgical excision of right occipital brain abscesses due to *Entamoeba histolytica*. The boy had EEG epileptiform discharges in the right occipital lobe, but never seizures. We therefore hypothesize that

ongoing occipital lobe epileptiform EEG discharges, even in absence of clinically overt seizures, may have triggered migraine in this patient.

## Case Report

We describe an 8-year-old child, with no relevant previous medical history, who was referred to our center for headache attacks. His father had migraine with aura, and his mother had migraine without aura. At age 5 years, 3 years before our observation, the patient presented severe headache and vomiting, and needed admission. In the emergency room, a computed

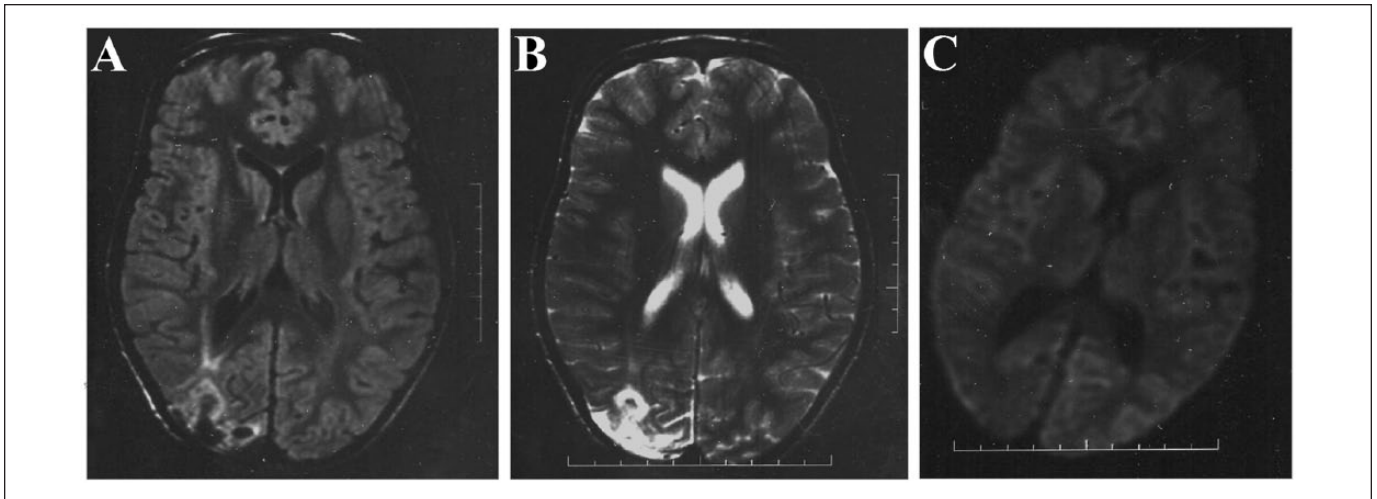
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**Figure 1.** Magnetic resonance (MR) scans performed 2 years after surgery. (A) T1-weighted images; (B) T2-weighted images; (C) diffusion-weighted images (DWI). The MR scans show areas of altered signal in the right occipital lobe, consistent with surgery outcome. No acute pathological process is evident; in particular, no area of altered signal, suggesting brain edema, is present in the DWI.

tomography (CT) scan showed an intracranial heterogeneous lesion in the right posterior parietal and occipital lobes, with perilesional edema and mass effect. Further CT and magnetic resonance imaging (MRI) showed 3 abscesses in the right occipital lobe. Since medical treatment with wide spectrum antibiotics and anti-edema agents failed to reduce the volume of the masses, the abscesses were drained surgically under ultrasound guidance. Three weeks later, a further CT scan showed the recurrence of the lesions (or the development of previously undetected abscesses). Therefore, a direct surgical excision of the abscesses was performed. Histological examination of the capsule, and of the fluid, revealed *E histolytica* trophozoites. The child was not immunodepressed, and the amoebic abscesses were attributed to one of many previous travels in tropical areas. Therapy with metronidazole was followed by the complete resolution of the infective process. The patient never showed any clinical or instrumental evidence of extra cerebral amoebic lesions. After surgery, antiepileptic prophylaxis with phenobarbital 2 mg/kg/d was started, and never withdrawn, although the child never presented seizures. Neuromotor development was normal before and after the amoebic infestation. Despite the removal of brain abscesses, the patient continued to complain of recurrent headaches, with a frequency of 20 to 25 per month. Pain was prevalently localized in right frontotemporal region, occasionally bilateral. During hospitalization, the pain was of moderate to severe intensity, and associated with nausea and photophobia; attacks lasted from minutes to 1 hour, and were usually responsive to acetaminophen, administered only for the treatment of particularly severe attacks. Occasionally, headache was accompanied by diffuse abdominal pain, of severe intensity, also with nausea. Visual symptoms were never reported. Typically, attacks appeared about 1 hour before sleep, and sometimes in the morning while awakening. Blood tests, blood count, and erythrocyte sedimentation rate were normal. Psychological

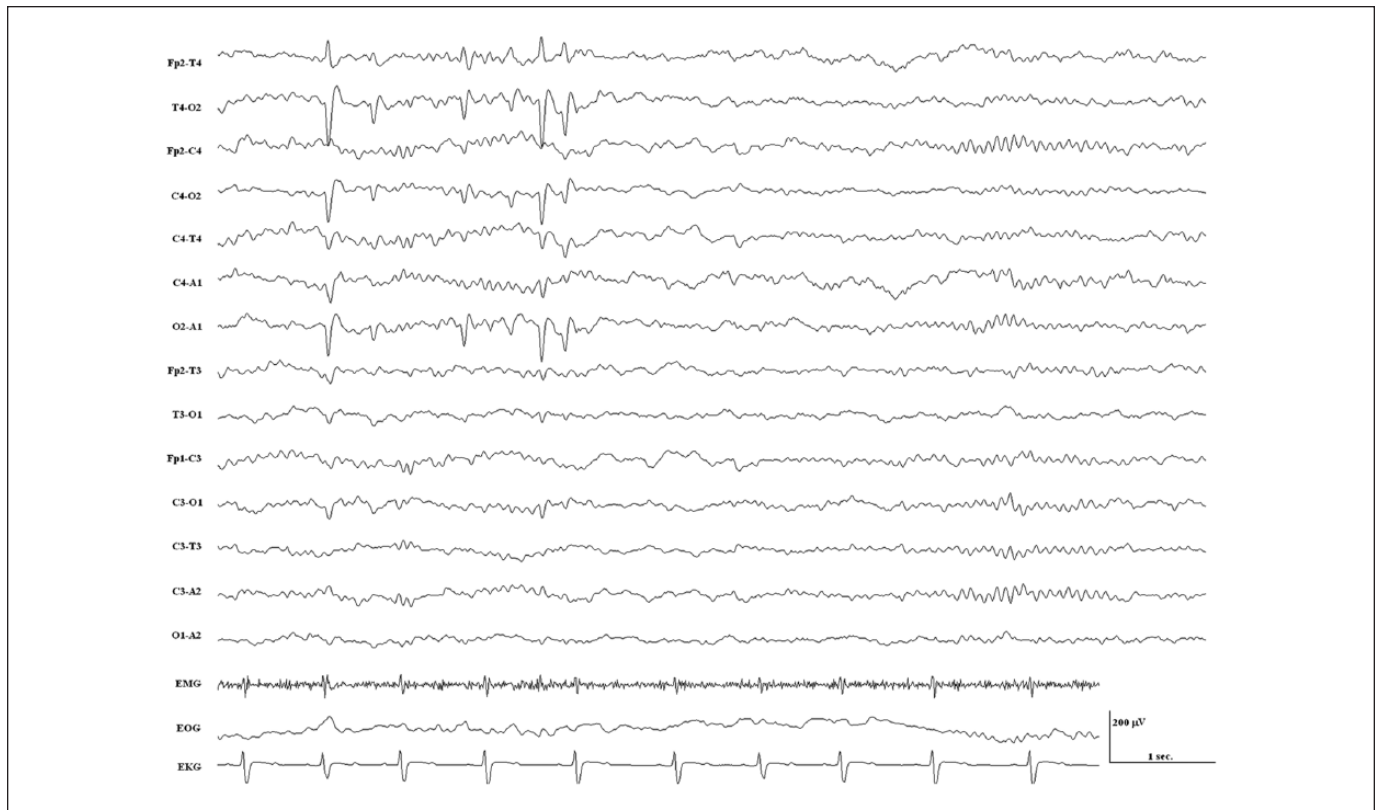
and neuropsychological evaluation did not reveal abnormalities. Neurological examination was normal; in particular no visual field defect was present. MRI performed 2 years after surgery revealed signal abnormalities in the right occipital lobe, consequent to the surgical treatment of the abscesses (Figure 1).

## Methods

A prolonged (10 hours) video-EEG and polygraphic laboratory based recording, including awake and nocturnal sleep, were performed to evaluate the presence of EEG abnormalities. Since the child reported headaches in the evening, prior to nocturnal sleep, the recording was started at 9.00 PM and prolonged up to 7.00 AM the next morning. During this assessment, the boy reported a headache similar to those he had almost daily. The onset of the attack occurred at about 10.00 PM, lasted 15 minutes and resolved spontaneously. Montage included 19 EEG leads, in bipolar reference or referred to the mastoids, bilateral electro-oculogram, submental muscle activity (electromyogram), and electrocardiogram. Spline frequency maps of the wake EEG activity in the dominant alpha frequency (9-11 Hz) were obtained, both during and after the headache attack.

## Results

Baseline wake EEG showed focal slowing over the right temporo-parieto-occipital leads, which is consistent with the site of the surgical intervention. During wake and, more often during sleep, clear epileptiform discharges, consisting of spike and spike-and-slow wave complexes could be detected in the same sites (Figure 2). Occasionally, epileptiform activities spread over contralateral and anterior regions. During the recording, about 30 minutes before bedtime, an acute episode of headache



**Figure 2.** Ten seconds of EEG and polygraphic recording from sleep stage II NREM. High-voltage spikes are present on the right temporo-occipital leads, with low-amplitude contralateral projection. EMG, submental muscles surface electromyogram; EOG, electro-oculogram; EKG, electrocardiogram; NREM, non-rapid eye movement. Calibration is in the lower right corner.

occurred. Pain was throbbing, intense, localized in the right forehead, associated with nausea, photophobia, and phonophobia. No vomiting, visual disturbances or impairment of consciousness were observed; the patient did not show tearing, conjunctival injection, eyelid edema or nostril congestion. The episode remitted spontaneously after about 1 hour. EEG did not show epileptiform discharges during the headache (Figure 3). Moreover, neither epileptiform discharges nor isolated spikes were detected prior to the onset of the headache, during the acute bout, or in the 30 minutes following.

During the headache, analysis of EEG spline frequency maps showed clear asymmetry of the dominant alpha rhythm frequency, in the 9- to 11-Hz range, which appeared to be markedly reduced in the right temporo-parieto-occipital regions (Figure 4).

## Discussion

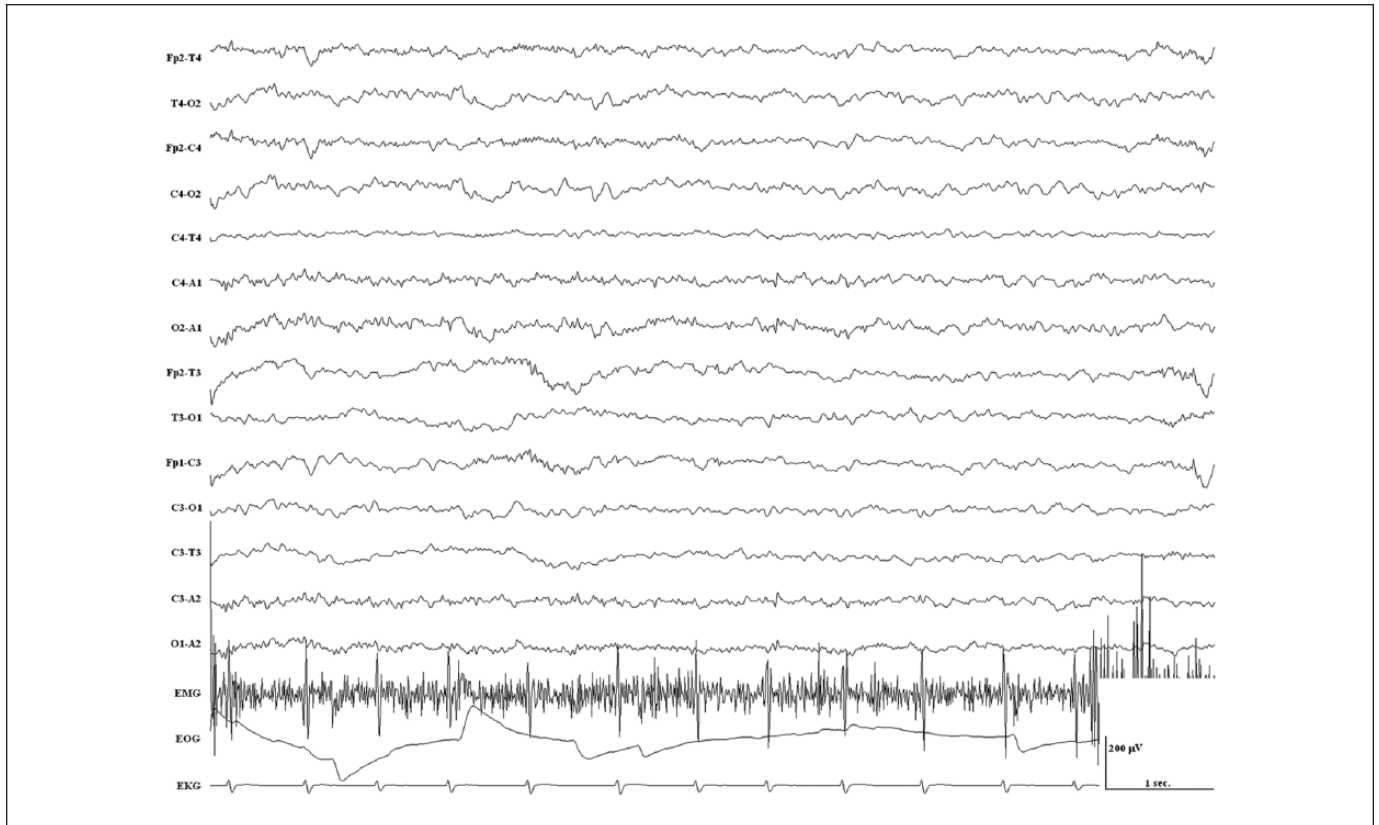
We describe a child who reported, following a surgical occipital brain lesion, headaches with migraine features. EEG showed right occipital spikes and spike-and-slow wave paroxysm, but, despite the presence of interictal epileptiform EEG activity, the boy never presented with clinically overt seizures. It is likely that prophylaxis with phenobarbital, started immediately after the surgery, prevented seizures.

The origin of the headache in our patient is questionable since it does not match the International Classification of Headache Disorders (ICHD-II)<sup>11</sup> criteria either for chronic postcraniotomy headache or chronic post-nonbacterial infection headache.

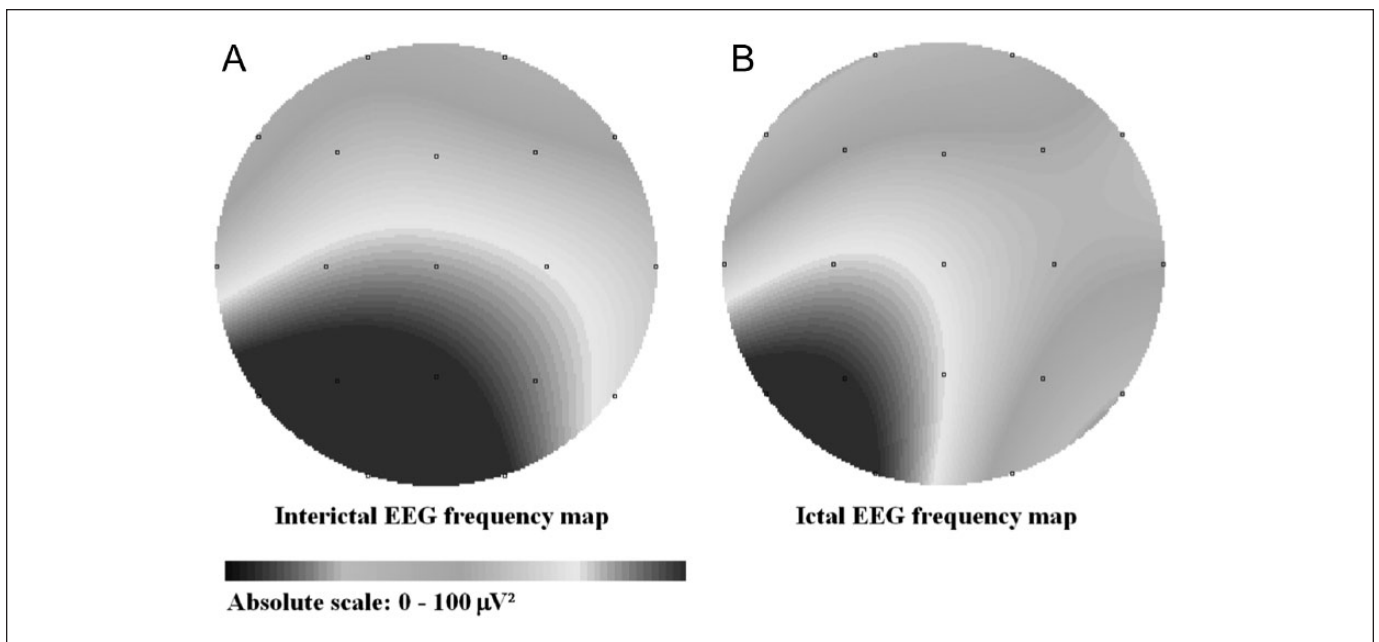
Chronic postcraniotomy headache (point 5.7.2 in the ICHD-II, 2004)<sup>11</sup> can be ruled out because of the clinical features of the pain. Chronic postcraniotomy headache induces pain involving the area of craniotomy, whereas in our patient the pain was prevalently frontotemporal, unilateral or bilateral. Similarly, a chronic post-nonbacterial infection headache (A9.4.2, ICHD-II)<sup>11</sup> could be hypothesized, but the ICHD-II itself clearly states that “there is little evidence for the existence of chronic headache attributed to non-bacterial infections.”

We hypothesize that the ongoing epileptiform EEG discharges in the right occipital lobe, secondary to the surgical lesion, may have triggered a genuine migraine, in the absence of overt occipital epileptic seizures. Numerous observations may support this hypothesis, as reported in a meticulous recent review.<sup>12</sup>

Our patient presented headache attacks with typical migraine features: unilateral throbbing pain, photophobia, phonophobia, nausea, and, sometimes, vomiting. Migraine or migraine-like headache is very frequently associated with occipital lobe epilepsies, both of idiopathic<sup>4,5</sup> or symptomatic<sup>7</sup> origin. In these



**Figure 3.** Ten seconds of EEG and polygraphic recording in wake, during the acute headache attack. Notably, no activation of right-side epileptiform activities is present. EMG, submental muscles surface electromyogram; EOG, electro-oculogram; EKG, electrocardiogram. Calibration is in the lower right corner.



**Figure 4.** Frozen spline frequency maps of 60-second artifacts-free EEG activity in the interictal recording (A, on the left) and during the headache attack (B, on the right). Maps show the topographic distribution of the dominant background activity (alpha band peak frequency, frequency range 9-11 Hz). Color scale expresses the absolute power (in  $\mu V^2$ ).

cases, headaches can occur either as an ictal<sup>8,13</sup> or a postictal phenomenon.<sup>3,9</sup> Some investigators have even suggested that the headaches may be preictal.<sup>14,15</sup> Postictal headache can occur even after minimal, simple visual seizures; it can be lateralized, throbbing, and completely indistinguishable from a migraine attack.<sup>10,16</sup>

Furthermore, based on the high clinical, pathophysiological and therapeutic overlap, Parisi et al<sup>17</sup> recently proposed to add to the forthcoming ICHD-3 classification<sup>18</sup> a new entity, namely the “ictal epileptic headache” (IEH), in order to define a condition diagnosed when a headache attack is the only clinical feature of epileptiform discharges.

At first view, these forms of “epileptic migraine,” that is, migraine originating as either an ictal or a postictal phenomenon, seem to be ruled out in our case. The boy never presented any clinically overt epileptic seizure, or even simple visual hallucination. Moreover, the video-EEG recording across the headache episode did not show any epileptiform activity before, during (Figure 2), or immediately after the headache attack. Likewise, the analysis of EEG during the headache may be consistent with a migraine attack.<sup>19</sup> EEG, in fact, showed a slight interictal asymmetry of the alpha rhythm, with predominance in the left occipital lobe (Figure 4A); such alpha asymmetry was greatly enhanced during the headache attack, with virtually total disappearance of alpha frequencies over the right occipital areas (Figure 4B). Various studies demonstrated that alpha asymmetry and unilateral alpha suppression are regarded as a typical, although nonspecific, EEG features of acute migraine.<sup>19-21</sup>

In contrast to the “ictal” findings (during the headache attack), the prolonged video-EEG showed copious epileptiform activity, consisting of focal right occipital-temporal, high-amplitude spikes, and spike-and-slow wave discharges, particularly enhanced during non-rapid eye movement sleep (Figure 3). Similar unequivocal epileptiform EEG activities (spike-and-wave complexes) can be found in epilepsy, but never in migraine (with the exception of peculiar forms, such as basilar migraine, migraine with prolonged aura, or alternating hemiplegia).<sup>19</sup> The pattern of occipital-posteromotor (O-PT) spike-wave paroxysms is an EEG abnormality observed primarily in occipital epilepsy of childhood and basilar migraine; occasionally O-PT spike-and-wave complexes, not associated with epileptic seizures, may be associated with headache.<sup>6</sup>

Consequently, we hypothesize that, even though the EEG recording did not show epileptiform discharges during the acute headache, the genuine migraine attack in our patient might represent the ictal epileptic manifestation. During the past decade, Parisi and colleagues,<sup>22-24</sup> in numerous articles, stressed the concept of the ictal epileptic headache (IEH) events as “sole” ictal epileptic manifestation. In this way, the reported migraine attack could, at least in part, follow the new IEH criteria proposed.<sup>17</sup>

Occipital lobe structures are involved in the pathogenesis of migraine with visual aura,<sup>25</sup> and some observations suggest

occipital involvement in migraine without aura.<sup>26,27</sup> Modification of excitability in the occipital cortex, whether hypoexcitability<sup>28</sup> or hyperexcitability,<sup>29</sup> may be involved in the pathogenesis of migraine. These modifications of neuronal excitability are directly linked with the phenomenon of cortical spreading depression (CSD), which has a key role in migraine.

In 2 exhaustive recent reviews, Parisi et al<sup>12,24</sup> clearly defined the complex interaction between migraine attacks and seizures, explaining that the onset and propagation of CSD and an epileptic focus are triggered when these neurophysiological events reach a certain threshold, which is lower for CSD than for seizure. In particular, migraine attacks can be the result of a trigeminovascular system activation by an epileptic discharge, which rarely can not be recorded at the scalp but only by deep stereo-EEG recording.<sup>13,30</sup> Thus, the fact that EEG recording did not show epileptiform discharges during an acute episode of headache, in the described patient, is not enough to exclude that the migraine attack was the only ictal epileptic manifestation.

The hypothesized pathogenesis of migraine in our patient is, moreover, in accordance to the mechanism proposed for the onset of postictal migraine in children with occipital lobe epilepsy. In these patients, occipital seizure discharge may trigger genuine migraine through trigeminovascular or brainstem mechanisms.<sup>10,31</sup> Occipito-postero-temporal spikes may represent a nonspecific epileptiform abnormality that may occur in children with epilepsy (idiopathic or symptomatic), absences or even in patients with no evidence of seizures. A close association between migraine and occipito-postero-temporal spike-and-wave discharges has been reported by several authors<sup>4,32</sup> and, as stated by Talwar et al,<sup>6</sup> “it is not surprising that most these patients (up to 80%) had seizures, migraine, or both.”

In conclusion, occipital epileptiform discharges, even in absence of seizures, may trigger a genuine migraine, probably by means of trigeminovascular or brainstem systems.

### Declaration of Conflicting Interests

The author(s) declared no conflicts of interest with respect to the research, authorship, and/or publication of this article.

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