# Should "migralepsy" be considered an obsolete concept? A multicenter retrospective clinical/EEG study and review of the literature

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## ARTICLE INFO

## ABSTRACT

The few reports that have been published on the current International Classification of Headache Disorders, Second Edition (ICHD-II), criteria for migralepsy and hemicrania epileptica have highlighted the considerable confusion regarding this "hot topic" within both headache and epilepsy classifications (ICHD-II and International League Against Epilepsy [ILAE]). Indeed, the ICHD-II describes a *migraine-triggered seizure* as a rare event in which a seizure occurs during migraine aura; on the other hand, *hemicrania epileptica* is described as an "ictal headache" that occurs "synchronously" with a partial seizure. To confuse matters even further, neither the term *migralepsy* nor the term *hemicrania epileptica* is included in the currently used ILAE classification. On the basis of both a review of "migralepsy" cases in the literature and 16 additional retrospective multicenter cases, we suggest that the term *migraine-triggered seizure* or *migralepsy* be deleted from the ICHD-II classification until unequivocal evidence is provided of its existence, and that the term *ictal epileptic headache* be introduced into the ILAE classification.

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## 1. Introduction

Although not fully elucidated, a relationship between migraine and epilepsy has long been postulated, with clinical and epidemiological studies demonstrating that both entities are highly comorbid [1–12]. As both these disorders are characterized by transient paroxysmal episodes of altered brain function, one condition may be mistaken for the other [1–12]. Epilepsy and migraine may either coexist independently in the same individual or be associated by chance; the outcome of this comorbidity is that one of these disorders may lead to, or mimic, the other.

Although the nature of this association is unclear, several plausible explanations do exist, including: the two disorders coexist by chance; headache is part (or even the sole ictal phenomenon) of seizures or the postictal state; both disorders share a common underlying

etiology; epilepsy mimics the symptoms of migraine (as in benign childhood epilepsy); lastly, migraine with aura triggers seizures, a phenomenon referred to as *migralepsy* [1]. Recently, Parisi et al. [13–22] suggested that the term *ictal epileptic headache* be used for patients whose headache rarely represents the sole ictal epileptic manifestation; on the basis of articles and cases previously published by both their group [13–22] and other groups [2,23–34], they suggested that the "migralepsy concept" might not exist at all, and that headache is simply the first ictal epileptic symptom in most "migralepsy" cases [13–22]. In other words, "migralepsy" probably represents an epileptic event that starts with an "ictal epileptic headache" followed by other sensory/motor/autonomic ictal epileptic signs/symptoms.

Nonetheless, the term *migralepsy* ("migraine-triggered seizure") is currently included in the International Classification of Headache Disorders, Second Edition (ICHD-II); its use is based on the fulfillment of two criteria: (1) migraine fulfilling criteria for 1.2 Migraine with aura (MA); (2) a seizure fulfilling the International League Against Epilepsy (ILAE) classification diagnostic criteria for one type of epileptic attack occurring within 1 hour of a migraine aura.

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In this article, we review and discuss all the "migralepsy" cases previously described in the literature (Table 1), adding a multicenter retrospective series of sixteen new "potential" cases (Table 2).

## 2. "Migralepsy" and "ictal epileptic headache" cases in the literature

The term *migralepsy* was first used in 1960 by Lennox and Lennox [1] to describe a condition wherein "ophthalmic migraine with perhaps nausea and vomiting was followed by symptoms characteristic of epilepsy" [1].

There are at least 50 potential cases of migralepsy reported in the literature [1–3,5,8,23–31] (Table 1). However, the diagnosis in the majority of these cases is uncertain because the information available is not clear (38%), the cases do not fulfill the current ICHD-II criteria (30%), or the diagnosis is questionable (28%) (Table 1) [31]. Indeed, most of the previous reports of "migralepsy" are complicated cases that do not provide a meaningful and unequivocal migraine–epilepsy sequence or are occipital seizures that imitate migraine with aura [4,8,10–12]. The term *migralepsy* does not appear either in previous classifications of the ILAE or in the recent report by the ILAE Commission on the Classification and Terminology of seizures and epilepsies [36], whereas the current ICHD-II [35] defines migralepsy as one of the complications of migraine, in the paragraph coded 1.5.5, in which migralepsy is referred to as a "migraine-triggered seizure."

Another interesting aspect is that, according to the definition provided in the ICHD-II, migralepsy is considered to be associated with MA attacks alone; by contrast, some authors have described a migraine without aura (MO) attack as representing a trigger for an epileptic seizure [14,30,31], and (unlike the current ICHD-II criteria) the development of an epileptic seizure more than 1 hour after an MA attack is not excluded [30].

As regards the ictal EEG abnormalities in these types of patients, it should be borne in mind that although unequivocal epileptiform abnormalities usually point to a diagnosis of epilepsy, the lack of clear epileptic spike–wave activity is frequent in other ictal autonomic manifestations, such as Panayiotopoulos syndrome [19,20], as well as in patients with a deep epileptic focus arising, for example, from the orbitomesial frontal zone [2,17–20,37]. In such cases, ictal epileptic EEG activity might be recorded either from the scalp or by stereo-EEG recording as a "theta" or even "delta" shape without any spike activity. Interestingly, there may, on rare occasions [2], be an isolated epileptic headache that has no associated ictal epileptic manifestations or scalp EEG abnormalities but whose ictal epileptic origin can be demonstrated by depth electrode studies (see patient 2 in Laplante et al. [2]).

With respect to the ictal EEG recording in the more recently published "ictal epileptic headache" (IEH) cases, it should be stressed that there is no specific EEG picture; a number of associated EEG patterns have instead been recorded during migraine-like complaints [13,16–22,32–34], including: (1) high-voltage, rhythmic, 11- to 12-Hz activity with intermingled spikes over the right temporo-occipital regions [32,33]; (2) high-voltage theta activity intermingled with sharp waves over the occipital region [13], or over the frontal regions with repetitive subsequent high-frequency rhythmic theta discharges [34]; and (3) bilateral continuous spike-and-slow-wave discharges [28]. Furthermore, a photoparoxysmal response [25], combined with complaints about a light pulsating headache, has been reported during intermittent photic stimulation [13].

Nevertheless, as reported for other autonomic manifestations, EEG discharges in patients with IEH do not, despite being detectable by scalp EEG, display any specific cortical patterns [20,22,38].

It is noteworthy that complete remission of the headache and of the epileptic abnormalities in most of these patients with "ictal epileptic headache" was achieved not by means of specific antimigraine drugs, but following intravenous administration of diazepam [13,28,33,34] or phenytoin [32]. Therefore, the anticipation of the cortical spreading depression (CSD) event suggests that the effectiveness of antiepileptic drugs (AEDs) in the prophylaxis phase and the pathophysiology of the initial part of the "migralepsy sequence" are due to the fact that such cases are merely "ictal epileptic headaches" followed by other autonomic–sensory–motor ictal epileptic signs/symptoms.

Transient brain MRI abnormalities, persisting for days, have been reported in about 6% of patients with migralepsy [39]. Blood–brain barrier damage and consequent edema have been suggested as explanations for reversible radiological alterations in these cases.

By contrast, MRI in the "ictal epileptic headache" cases published in the literature revealed secondary brain lesions in the right temporo-parieto-occipital region, with limited spreading to the right occipital region [32,33] or enlarged sulci in the right parietal region [25]. In addition, a child affected by "Rasmussen encephalitis" whose clinical EEG picture perfectly fits our definition of "ictal epileptic headache" was described in a recent case report [34]. "Ictal epileptic headache" has, however, also been reported in patients with idiopathic epilepsy [13,28].

## 3. Multicenter retrospective "migralepsy" study: Methods and results

### 3.1. Inclusion criteria

We reviewed the records of approximately 4600 children with epilepsy (diagnosed according to the ILAE classification criteria) examined in five "child neurology and epilepsy centers" in Rome, Chieti, L'Aquila, and Naples between 1990 and 2010. We retrospectively selected 16 (4 males and 12 females, aged between 5 and 18 years) of these children (0.3%) on the basis of the following inclusion criteria: (1) close temporal association between MA or MO attacks and the onset of their epileptic seizures, which had to have occurred within 1 hour of the migraine attack; (2) at least one available ictal EEG recording during a migraine attack followed by a seizure within 1 hour. See Table 2 for the details and clinical/EEG pictures of the sample.

**Table 1**
Data from the migralepsy cases published in the literature.
*Source.* Modified from Sances G. et al. [31].

| | | |
|---|---|---|
| Migraine type | Migraine with aura | 62% |
| | Migraine without aura | 12% |
| | Basilar-type migraine | 14% |
| | Probable migraine with aura | 12% |
| Seizure type | Simple partial (SP) | 8% |
| | Complex partial (CP) | 16% |
| | Generalized tonic–clonic (GTC) | 50% |
| | SP + GTC | 12% |
| | CP + GTC | 6% |
| | SP + CP + GTC | 2% |
| | SP + epilepsia partialis continua | 6% |
| Ictal EEG | Not available | 68% |
| | Normal | 4% |
| | Abnormalities | 28% |
| Interictal EEG | Not available | 6% |
| | Normal | 32% |
| | Localized abnormalities | 34% |
| | Generalized abnormalities | 28% |
| Brain imaging (X-ray, CT, MRI) | Not available | 32% |
| | Normal | 48% |
| | Structural alterations | 14% |
| | Transient alteration | 6% |
| Diagnosis | Migraine-triggered seizures | 4% |
| | Does not fulfill ICHD-II criteria | 30% |
| | Questionable (epileptic seizure) | 28% |
| | Uncertain | 38% |
| Comorbidities | | 22% |

**Table 2**
Clinical and EEG data related to our "migralepsy" multicenter case series.

| Case | Sex/age | Migraine type | Seizure type | Interictal EEG | Ictal EEG (during migraine) | MRI | Symptoms (during migraine) | Other visual symptoms (during migraine) | Familiarity |
|---|---|---|---|---|---|---|---|---|---|
| 1 | M/7 | MO[a] | Partial T-O | T-O spike–waves | T-O spike–waves | Neg | Vomiting | Absent | Yes |
| 2 | F/8 | MA | Partial P-O | Negative | Left P-O spike–waves | Neg | Absent | Micropsia | No |
| 3 | F/7 | MO | Partial C-O | Negative | Right T-O spike–waves | Neg | Absent | Absent | No |
| 4 | M/11 | MO | GTC | Generalized spike–waves | Generalized spikes–waves | Neg | Absent | Absent | Yes |
| 5 | F/10 | MO | Partial P-O | Right P-O spike–waves | Right P-O spike–waves | Neg | Absent | Absent | Yes |
| 6 | F/12 | MO | Partial O | Right O spikes | Right O spikes | Neg | Absent | Scotoma + amaurosis | No |
| 7 | F/10 | MO | Generalized | Generalized spike–waves | Generalized spike–waves | Neg | Dizziness | Absent | No |
| 8 | F/18 | MA | Partial C-O | Right C-O spike–waves | Right C-O spike–waves | Leucoencephalopathy | Nausea + photophobia | Disorders of the visual field | Yes |
| 9 | F/18 | MA | GTC | Generalized spike–waves | Generalized theta | Neg | Photophobia | Scotoma | No |
| 10 | M/18 | MA | Partial C-T | Right C-T spikes | Right C-T theta | Altered neuronal migration | Nausea | Loss of the visual field | No |
| 11 | F/18 | MA | Partial O | Right O spike–waves | Right O spike–waves | Neg | Photophobia | Absent | No |
| 12 | F/18 | MA | Partial C-T | Left T-C spikes | Left T-C spikes | Hydrocephalus and stenosis | Vomiting | Scotoma | Yes |
| 13 | M/7 | MO | Partial O | Left O spikes | Left O spikes | Neg | Vomiting and photophobia | Absent | Yes |
| 14 | F/7 | MA | Partial T-C | Left C-T spike–waves | Left C-T spike–waves | Neg | Vomiting | Scotoma | Yes |
| 15 | F/5 | MA + MO | GTC | Left T-C theta | Left T-C theta | Periventricular gliosis | Vomiting | Micropsia + complex hallucinations | No |
| 16 | F/17 | MA + MO | GTC | Left T-C theta | Bilateral frontal theta with spikes | Neg | Confusional status | Scotoma | Yes |

[a] MO, migraine without aura; MA, migraine with aura; C, central; O, occipital; T, temporal; P, parietal.

Symptoms/signs associated with ictal and interictal migraine attacks (especially visual symptoms) were collected from the patients' clinical records. The migraine attacks of all 16 patients were classified as migraine without aura (MO) or migraine with aura (MA) according to the ICHD-II criteria, even in patients whose records dated from before 2004 .

We reviewed the temporal relationship between the migraine and seizure events on the basis of the clinical descriptions provided by the patient on admission, as reported in the clinical record.

All brain MRI scans and ictal (during migraine) and interictal EEGs obtained (with electrodes placed according to the International 10–20 System) were analyzed jointly by all authors involved in this retrospective study during three "ad hoc" meetings held in Rome.

Table 2 summarizes the characteristics of all 16 patients, 4 (25%) of whom were male and 12 (75%) female. Eleven patients (69%) had complex partial seizures: 2 (18%) with centro-occipital lobe focus, 3 (27%) with occipital lobe focus, 1 (9%) with temporo-occipital lobe focus, 2 (18%) with parieto-occipital lobe focus, and 3 (27%) with centrotemporal lobe focus. Five (31%) patients had primary generalized epilepsy; 4 (80%) of these 5 patients had generalized tonic–clonic seizures. Four patients had partial epilepsy with secondarily generalized seizures.

The MRI was normal in all except 4 cases (cases 8, 10, 12, and 15 in Table 2), who had the following lesions: altered neuronal migration, hydrocephalus, leukoencephalopathy, and periventricular gliosis.

As stated above, all 16 patients were selected on the basis of the close temporal association described in the ICHD-II between their migraine attacks and the occurrence of an ILAE-classified seizure; 7 (44%) of the 16 patients had MOs, 7 (44%) had MAs, and the remaining 2 patients (12%) displayed features typical of both types of migraine.

The interictal EEG recordings revealed unilateral or bilateral epileptiform complexes in 14 (87%) patients, whereas the interictal EEG recording was normal in the remaining 2 (12%) patients, with partial epilepsy, who had complex seizures during the migraine attacks.

Nine (56%) patients showed spike-and-wave complexes during the migraine ictal EEG recording; 2 of these 9 patients had generalized epilepsy (associated with interictal generalized EEG discharges), whereas the remaining 7 had partial epilepsy.

Visual symptoms (e.g., amaurosis, elementary, or complex visual hallucinations) were present in 9 (56%) patients. The interictal EEG showed spike or spike-and-wave complexes in 6 (67%) of these 9 patients; theta activity was observed in two (22%, cases 9 and 15) (see Table 2). The MRI was abnormal in 4 patients. Others symptoms, which consisted of nausea, abdominal pain, photophobia and dizziness, were present in 8 (50%) patients. Nine patients reported elementary or complex visual hallucinations.

## 4. Discussion

The ICHD-II includes "migraine-triggered seizure" (i.e., migralepsy) among the complications of migraine coded at "1.5.5" (as a rare event in which a seizure occurs during migraine aura), whereas hemicrania epileptica (coded at "7.6.1" as an "ictal headache," ipsilateral to the ictal EEG discharge and occurring "synchronously" with a partial seizure recognized by the ILAE classification) and postictal headache (coded at "7.6.2") are described among the headaches that may be attributed to an epileptic seizure. The terms migralepsy, hemicrania epileptica, and postictal headache do not appear in the currently used ILAE classification. What makes things even more confusing is that the ILAE classification defines, among the terms used to describe epileptic seizure semeiology, a "headache" as a cephalic (not autonomic) (2.2.1.7) sensation in the head (as well as lightheadedness or tingling); other types of "pain" are listed among the "somatosensory" (at 2.2.1.1 of ILAE "glossary" [36,40]) perceptions, which include tingling, numbness, electric shock sensation, sense of movement, and desire to move; by contrast, an "autonomic seizure" is described as "an objectively documented and distinct alteration of the autonomic nervous system function involving cardiovascular, pupillary, gastrointestinal, sudomotor, vasomotor and thermoregulatory functions."

The 16 new retrospective cases described here (Table 2) and the previously published migralepsy cases (Table 1) illustrate the difficulties that may be encountered when attempting to make a diagnosis, as well as the inadequacy of the recent ICHD-II "migralepsy" diagnostic criteria [31]. Moreover, a recent review of the literature (Table 1) identified a remarkably large number of purely epileptic disorders among cases reported as migralepsy [31]. Indeed, one patient described in a case report we published previously [13] responded (immediately from both the clinical and EEG points of view) to intravenous anticonvulsant (diazepam) administration, as did other cases (three responded to diazepam and one to phenytoin) [28,32–34].

Moreover, Maggioni et al. [30], when discussing two of their "migralepsy" cases, stressed the role of a preexisting low epileptic threshold, suggesting that further studies be designed to assess the need to include MO-triggered epileptic seizures in the diagnostic criteria for migralepsy. They also suggested that migralepsy might occur more frequently than is reported because the epileptic event overshadows the MO [30].

With respect to our "migralepsy" cases (Table 2), we found, on the basis of the afore-described temporal "inclusion criteria," only 16 of approximately 4600 epileptic children whose seizure "occurred within 1 hour of a MA or MO attack"; we should also point out that we even included (in accordance with Maggioni et al. [30], though in contrast to the current ICHD-II "migralepsy" criteria, point 1), patients manifesting MO as the sole migraine type associated with "epileptic seizures" (patients 1, 3–7, and 13), thus not adopting MA as a mandatory condition (the "sine qua non" condition requested by the current ICHD-II classification to make a diagnosis of "migraine-triggered seizure" or "migralepsy").

With respect to the EEG findings, we decided to select (see Inclusion Criteria) only subjects with an ictal EEG recorded during the "migraine phase." Interestingly, all 16 patients displayed focal or generalized "ictal EEG abnormalities" during the migraine attacks. Moreover, it is worth bearing in mind, as mentioned above, that only 4% of previous reports of "migralepsy" cases fulfill the current ICHD-II diagnostic criteria, and that a migraine-phase EEG was available in only 32% of those cases [31]. There seems to be no correlation between specific EEG abnormalities and MA or MO in the children we identified (see Table 2); indeed, the spike or spike-and-wave pattern, which is the most commonly observed EEG pattern, was associated with both MA and MO (Table 2), whereas EEG theta activity was surprisingly associated exclusively with MA or a "double migraine pattern," in which MA and MO coexisted (patients 9, 10, 15, and 16). Fourteen of 16 children (the exceptions were cases 2 and 3) displayed interictal EEG abnormalities. Our sample (Table 2) exhibited a marked prevalence of "migralepsy" in females (8 F vs 4 M), whereas the "migralepsy association" with MO (as the sole migraine type) prevailed in males (75%).

Another intriguing finding in our patients with "migralepsy" (Table 2) is the lack of a correlation between specific cortical localization of the EEG abnormalities and a synchronous headache onset. Indeed, whereas the "occipital localization" has previously been reported to prevail, or even be exclusive, in migralepsy, we did not observe any specific cortical localization in our 16 patients; for example, we recorded occipital (Fig. 1), centrotemporal (Fig. 2), and even frontal (Fig. 3) ictal EEG abnormalities, respectively, in patients 6, 14, and 16, all of whom presented with an ictal headache (see Table 2).

Moreover, no specific EEG pattern emerges from either the "migralepsy" cases listed in Table 2 or the ictal epileptic headache cases that have recently been published [13,16–22,32–34], with a wide range of patterns and localizations (occipital, temporoparietal, and frontal) being recorded during migraine-like complaints. These findings suggest, as we have previously hypothesized [15], that the epileptic focus that activates the trigeminovascular system might remain purely autonomic (it being associated exclusively with migraine complaints), without ictal neuronal activation of nonauto-nomic cortical areas (different neuronal network thresholds appear to be involved); in this case, the focus fails to reach the symptomato-genic threshold required to induce sensory–motor manifestations, as has been described for other ictal autonomic manifestations in Panayiotopoulos syndrome [41].

The diagnosis in these rare cases is complicated even further by the fact that the epileptic headache may be isolated; that is, it is not associated with any other ictal epileptic manifestations or EEG abnormalities that can be detected by scalp EEG recording [2]. In this regard, it is often impossible (in 20 to 40% of patients) to detect ictal epileptic activity by means of scalp EEG recording in other types of epileptic manifestations, such as in frontal lobe epilepsy [37].

Another noteworthy issue that causes some confusion is the common "epilepsy–migraine" sequence, which has widely been ignored despite its high prevalence in occipital epilepsy, in which "postictal" headache occurs in 50% of patients. Indeed, although a migraine-type headache is a common postictal phenomenon that occurs in approximately 50% of patients with epilepsy, it is often neglected because of the dramatic manifestation of the seizure. This condition has been included in the recent ICHD-II [35] and is diagnosed on the basis of the following criteria: (1) headache has tension-type headache features or, if in a patient with migraine, migraine headache features and fulfills criteria 2–4; (2) the patient has had a partial or generalized seizure; (3) headache develops within 3 hours of the seizure; (4) headache resolves within 72 hours of the seizure. In this regard, Schön and Blau [42] described a series of 100 patients with epilepsy, 51 of whom had postictal headache, which has been reported particularly in idiopathic occipital seizures [43]. It is likely that the seizure discharges in the occipital lobes trigger a genuine migraine headache through cortical spreading depression (CSD) and trigeminovascular or brainstem mechanisms, as previously suggested by our group [15–20]. Indeed, the onset of epileptic seizures may facilitate that of CSD to a greater degree than the onset of CSD facilitates that of epileptic seizures. This may explain why, in the clinical context, patients with epilepsy with postictal headache (51% according to Schön and Blau) [42] are more likely to be observed than subjects with migraine with epileptic seizures [15–20].

Yet another source of confusion is the lack of an accurate description of symptoms during "ictal epileptic headache"; indeed, an "ictal epileptic headache" is usually orbital or manifests itself as painful discomfort that does not have a clear localization within the head and, thus, displays a "pattern" different from that of "migraine-associated" pain. By contrast, "postictal headache" is more character-istic of migraine symptoms; a slight impairment in consciousness may be associated with "ictal epileptic headache" more often than it is with postictal headache conditions, during which patients are more likely to be able to clearly describe their "postictal" symptoms (severity, type, and localization of pain).

A possible partial overlap between headache and epilepsy, which on rare occasions may even be a complete overlap (e.g., in patients with "ictal epileptic headache"), is supported by clinical/EEG and genetic studies on familial hemiplegic migraine (FHM) [44–46]. In FHM, errors in the same gene may be associated with migraine in some cases and with epilepsy in others [46].

To sum up, the authors of most of the "migralepsy" cases previously published in the literature have highlighted the fact that the EEG and clinical features of such subjects may be suggestive of occipital lobe seizures, although the information currently available is insufficient to confirm this hypothesis. In this regard, it should be borne in mind that ictal EEG recordings during the migraine phase are available in only 32% of previously published "migralepsy" cases, which prevents any firm conclusions from being drawn regarding "migralepsy" EEG-associated patterns. Indeed, Sances et al. suggested that ICHD-II 1.5.5 (the paragraph defining migralepsy) be relocated to the ICHD-II Appendix until a larger number of migralepsy cases are reported or the condition is better characterized [31].

**A**

**B**

**Fig. 1.** Case 6 in Table 2. (A) Right occipital theta activity with sporadic spikes during migraine. (B) Normal EEG some months later.

**A**
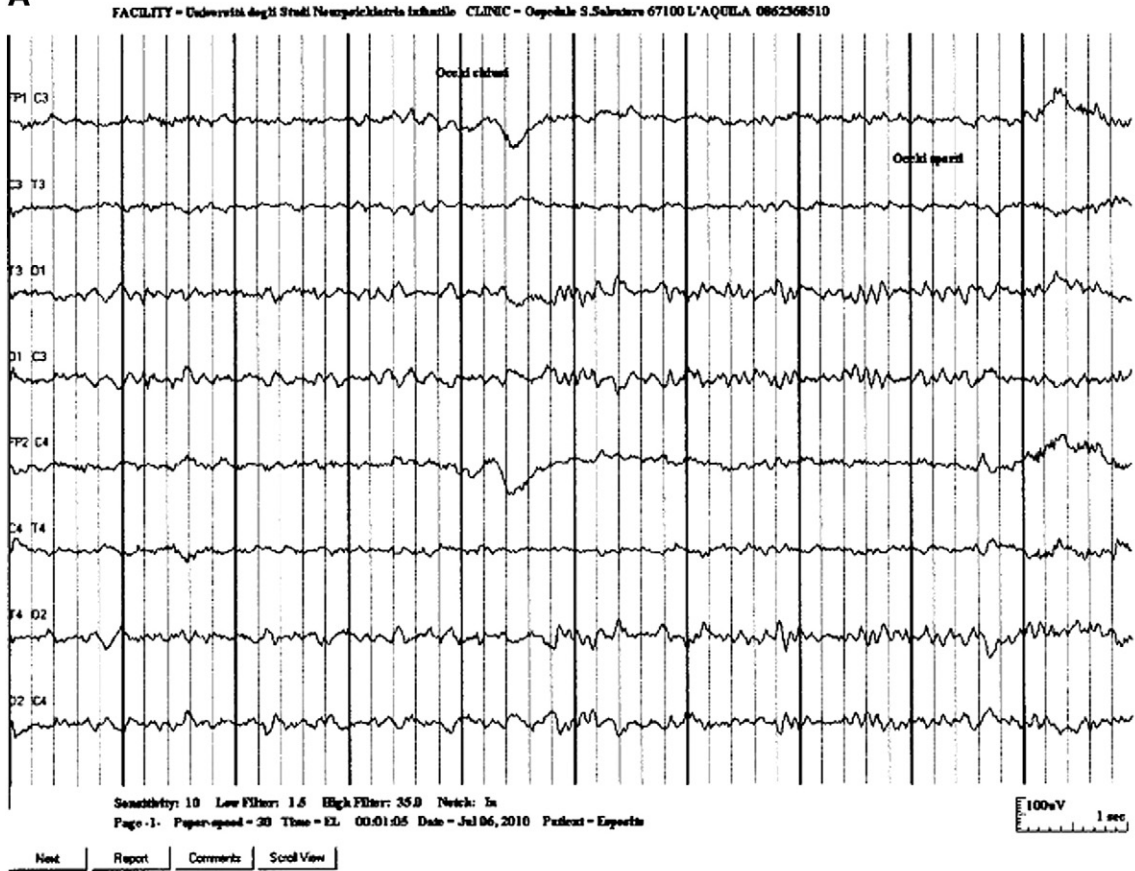


**B**



**Fig. 2.** Case 14 in Table 2. (A) EEG 15 minutes before migraine onset. (B) Spikes over the left centrotemporal region during the migraine phase.
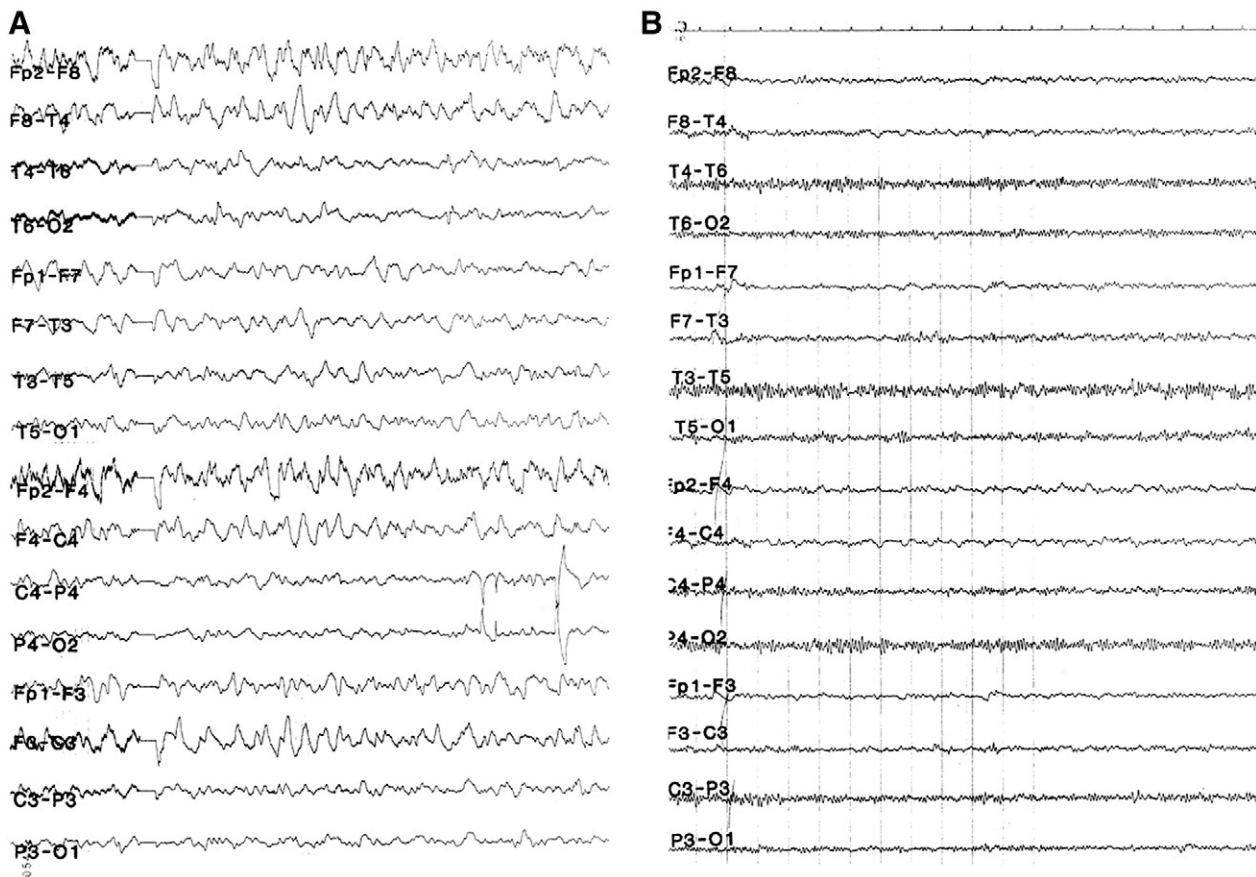
**Fig. 3.** Case 16 in Table 2. (A) Theta activity with intermingled spikes over the frontal bilateral regions during migraine. (B) Normal EEG 6 hours later.

Moreover, we believe, on the basis of the data in the literature as well as our own experiences described here, that the "migralepsy concept" might not even exist at all and that headache is merely the first ictal epileptic symptom in most "migralepsy" cases. In other words, seizures associated with "migraine-like" manifestations probably represent an epileptic event that starts with an "ictal epileptic headache" (followed by other sensory/motor/autonomic ictal epileptic signs/symptoms) as opposed to episodes characterized by both migraine and epileptic mechanisms. As far as the "ictal epileptic headache" classification is concerned, we have suggested [17,19,20] that it be included under "autonomic epilepsy," as has recently been suggested for Panayiotopoulos syndrome; in cases with long-lasting episodes, this rare event could even fulfill an "autonomic status epilepticus" condition [47].

Lastly, we recommend that any patients displaying features of both migraine and epilepsy undergo an ictal EEG recording during the migraine attacks to demonstrate (even though it is not always possible [2,37]) the underlying pathogenic mechanism of these episodes and to identify those rare cases of "ictal epileptic headache." Moreover, by adopting this approach (ictal EEG recording during headache) in previously diagnosed subjects with epilepsy, we will have the possibility to more reliably define whether the "migralepsy concept" deserves its "nosologic dignity" and should consequently be included in the ILAE and ICHD-II classifications.

In conclusion, we believe that further research is warranted to shed light on the complex relationships that link headache/migraine and epilepsy.

## Conflict of interest statement

None of the authors has any conflict of interest to disclose in publishing this article.

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