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Recurrent occipital seizures misdiagnosed as status migrainosus

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ABSTRACT – Periictal headache is commonly reported in patients with epilepsy and often exhibits migraine features. Misdiagnosis is frequent since visual seizures may often be misinterpreted as visual aura of migraine. We herein describe a 35-year-old woman with recurrent occipital seizures, clinically presenting with intractable headache. EEG monitoring was crucial in order to reach the correct diagnosis.

Key words: occipital seizure, differential diagnosis, status migrainosus, headache, migraine

The association between epilepsy and headache is well known (Andermann and Lugaresi, 1987). Epilepsy and migraine may coexist independently in the same individual or may be causally related, with one leading to the other. Seizure-associated headache may occur in preictal, ictal or postictal periods. Postictal headache (PIH) occurs in ~50% of patients after focal (mainly occipital or temporal) or generalised tonic-clonic seizures (Schon and Blau, 1987). PIH has been described in one to two thirds of patients with idiopathic or symptomatic occipital lobe epilepsy (OLE) (Panayiotopoulos, 1999a). Misdiagnosis is frequent since visual seizures may often be misinterpreted as visual aura of migraine, and PIH often exhibits migraine features (Panayiotopoulos, 1999b). A migraine attack lasting for more than 72 hours is defined as status migrainosus (The International Classification of Headache Disorders, 2005 [ICHD-II]). We herein describe an anecdotal case of recurrent occipital seizures misdiagnosed as status migrainosus.

Case report

A 35-year-old woman with a seven-day history of headaches, which did not respond to common non-steroidal anti-inflammatory drugs, was admitted to our department.

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At the age of 16 years, the patient presented an episode characterised by vision of coloured round circles in the right hemifield, lasting for about two minutes, and followed by fronto-temporal, stubbing headache associated with nausea and vomiting. Two hours later, a generalised tonic-clonic seizure (GTCS) occurred. A second episode with similar features occurred one year later. Brain CT scan was normal. Phenobarbital (PB) at 100 mg/day was then prescribed by her general practitioner, and she remained seizure-free for seven years. PB was then gradually discontinued. Thereafter, the above mentioned episodes of headache preceded by visual symptoms reoccurred at a yearly frequency without any apparent precipitating factor. Diagnosis of migraine with aura and migraine-triggered seizures was then proposed by a specialist.

When the patient came to our attention, she complained of severe and stubbing left fronto-temporal headache associated with nausea, along with vision of coloured round circles in the right hemifield lasting for about two minutes and occurring several times a day. Neurological examination showed right hemianopia. She was administered 500 mg *e.v.* lysine acetylsalicylate without any improvement. The following day, a standard EEG was performed and two occipital seizures were recorded, occurring 11 minutes apart and lasting ~90 and ~70 seconds, respectively. Clinically, the patient presented only the aforementioned visual symptoms. Ictal EEG showed a rhythmic fast activity with phase-reversal at O1, with a recruiting-derecruiting pattern, gradually replaced by polyspike and polyspike-and-wave activity *(figures 1, 2)*. A bolus of 10 mg *i.v.* diazepam was administered with complete resolution of the electroclinical pattern *(figure 3)*. Dexamethasone at 8 mg/day (which was gradually discontinued after five days) and 100 mg/day PB were also started, in view of the previous efficacy of PB and because of the patient's distrust towards new drugs. Brain MRI revealed hyperintense signal area on T2-FLAIR weighted images in left occipital cortico-subcortical regions, in keeping with postictal vasogenic oedema *(figure 4)*. Headache and hemianopia gradually resolved in the following 24-48 hours. Control brain MRI performed nine days later showed almost complete resolution of the left occipital oedema *(figure 5)*. The patient remained seizure-free at one-year of follow-up. Standard and sleep EEG performed afterwards did not show any abnormality including photoparoxysmal response.

Discussion

This report highlights the complexity of diagnostic assessment of patients with focal seizures mainly manifesting with periictal headache. Indeed, at admission, our patient mainly complained of severe, intractable headache with migrainous features, along with short-lasting, recurrent visual symptoms. An accurate medical history may help in discerning

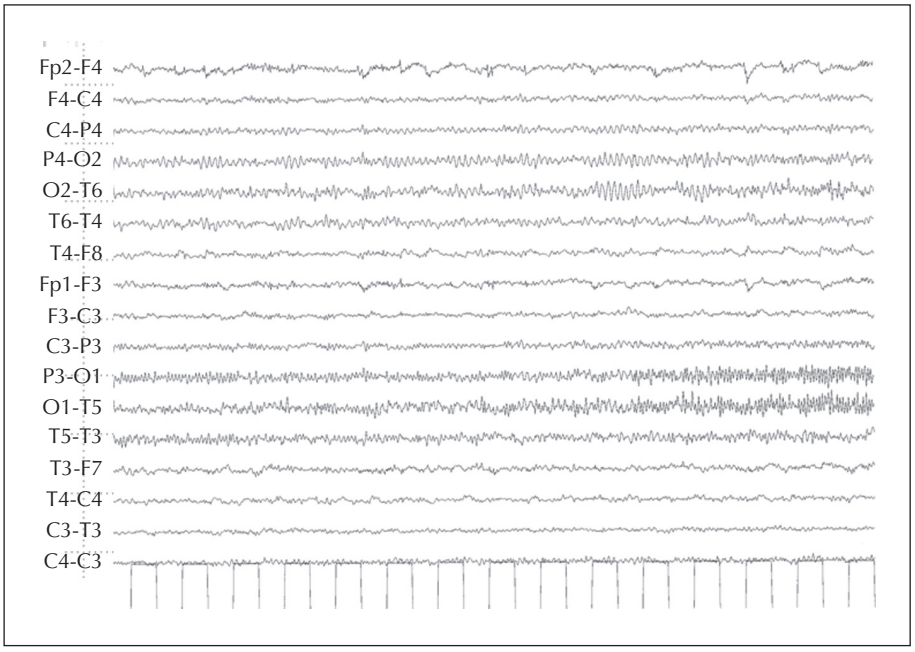

Figure 1. Ictal EEG recording of first seizure. Note a rhythmic polyspike activity over the left occipito-temporal regions, with phase-reversal at O1. Low- and high-frequency filters were set at 0.53 Hz and 50.0 Hz, respectively.

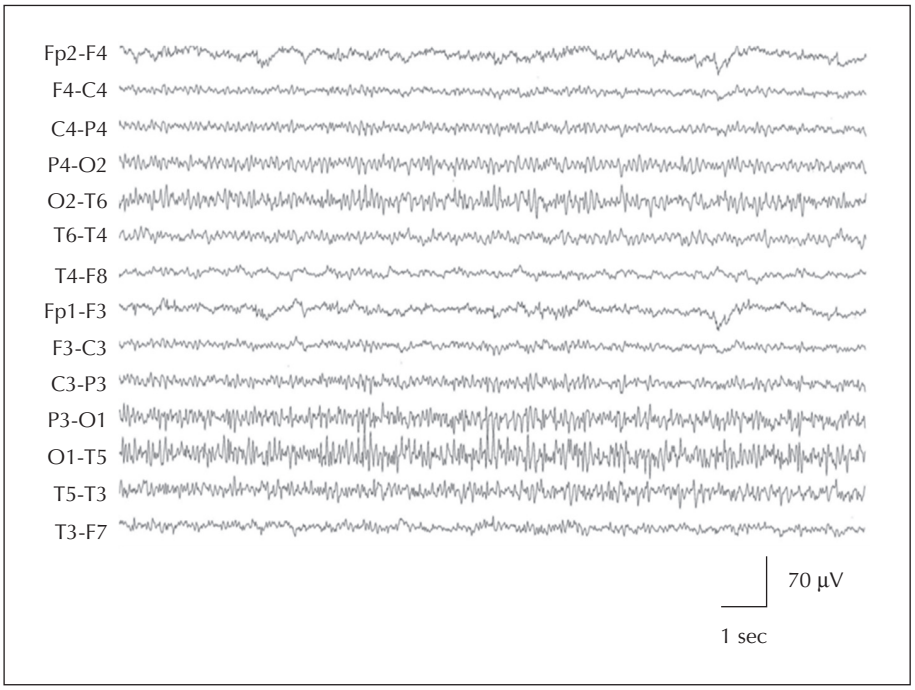

Figure 2. The same ictal EEG recording as in *Figure 1*, 30 seconds later. Polyspike and polyspike-and-wave activity with phase-reversal at O1. Low- and high-frequency filters were set at 0.53 Hz and 50.0 Hz, respectively.

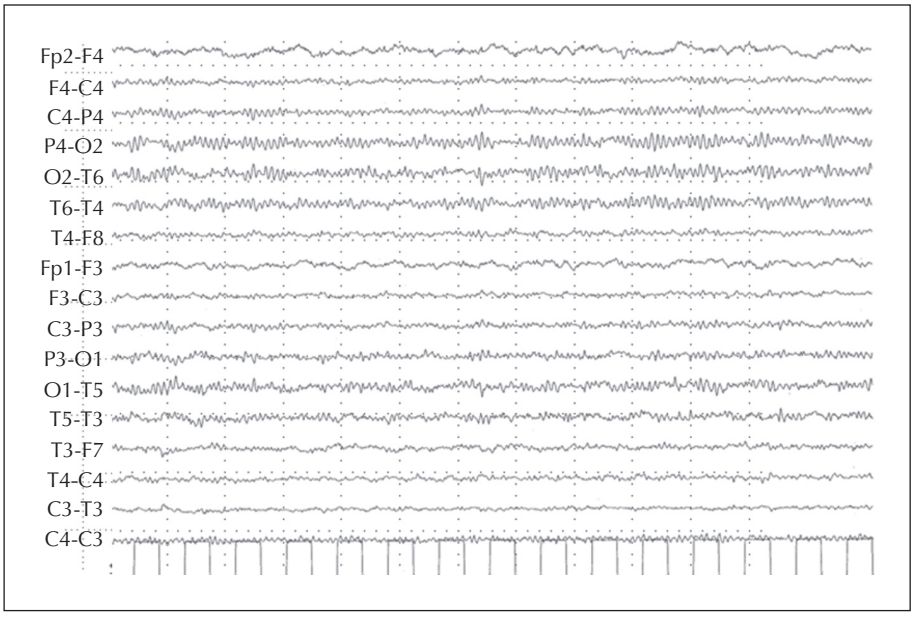

Figure 3. EEG normalization after administration of *i.v.* diazepam.

migraine with aura from occipital seizures with periictal headache. Long-lasting severe intractable headache is more commonly reported in migraine without aura. In addition, the duration and features of visual symptoms (a coloured, geometric pattern, lasting for between seconds and three minutes) may suggest the diagnosis of occipital seizures. Conversely, visual aura of migraine is usually characterised by

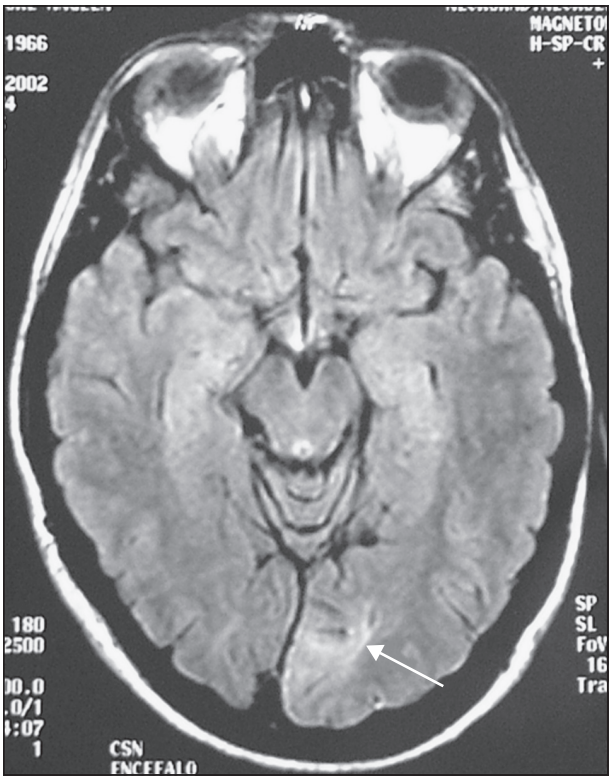

Figure 4. Brain MRI, axial FLAIR image: hyperintense signal area in left occipital cortico-subcortical regions (see arrow).

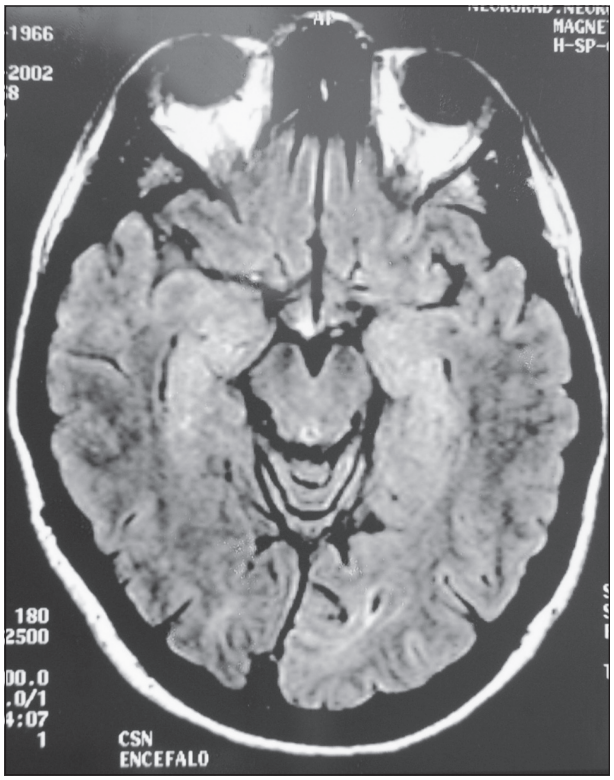

Figure 5. Control MRI performed nine days later, showing the almost complete resolution of oedema.

monochromatic linear zig-zag patterns or loss of vision, usually has a longer duration (5-60 minutes), and may be followed by sensory symptoms or dysphasic speech disturbances.

According to ICHD-II, migraine-triggered seizures (also named migralepsy) may occur during or within one hour after a migraine aura, but they are extremely rare and their existence is still controversial (Panayiotopoulos, 1999a; Sances *et al*., 2009). Indeed, most of reported patients had clinical features strongly suggestive of occipital lobe seizures. Our patient had been misdiagnosed with migralepsy, and only the EEG recording of ictal events eventually facilitated the correct diagnosis. In a systematic prospective study of occipital seizures with elementary visual hallucinations (Panayiotopoulos, 1999a), Panayiotopoulos showed that postictal headache is often severe and indistinguishable from migraine, even after brief visual seizures without convulsions. In most patients, headache lasts for between two and 24 hours, often contralateral to the side of visual hallucinations. Postictal migraine symptoms are equally frequent and share the same features in idiopathic and symptomatic occipital lobe epilepsies, and may be associated with vomiting, photophobia and phonophobia (Panayiotopoulos, 1999a). In contrast, postictal hemianopia may be found in a minority of patients, usually lasting up to three hours. Interestingly, in our patient both postictal headache and hemianopia were of a prolonged duration, probably due to the recurrence of seizures.

Pathophysiological mechanisms underlying periictal headache have not yet been clarified. A trigeminovascular mechanism has been proposed to explain the physiopathology of migraine and aura. According to this model, neurogenic inflammation of pain-sensitive cranial structures may be mediated by the release of peptides from sensory axons of the trigeminal nerve, determining inflammatory response and local vasodilatation. Moreover, activation of a "headache generator", located in pain-involved brainstem areas (*i.e*. dorsal raphe nuclei and locus coeruleus) has also been hypothesized (Welch, 1997). A local vasodilatation of the meningeal vessel provoked by ictal discharge leading to activation of trigeminal pain pathways in predisposed patients has been postulated (Penfield and Jasper, 1954; Bernasconi *et al*., 2001).

Finally, this report strongly highlights the importance of performing emergency EEG in patients with intractable headache, especially when visual symptoms are not typical of migraine aura. □

Disclosure.
None of the authors has any conflict of interest or financial support to disclose.

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