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EEG changes during left- and right-sided weakness in patient with sporadic hemiplegic migraine

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To the Editors

Migraine is a common neurological disorder, with a prevalence of 15-20% in the general population. Hemiplegic migraine is a rare subtype of migraine with aura. The characteristic feature of hemiplegic migraine is the presence of fully reversible unilateral motor weakness and fully reversible visual, sensory and/or speech/language symptoms. Hemiplegic migraine can occur as a sporadic or a familiar disorder [1–3]. The latter has an autosomal dominant mode of inheritance. Weakness is a manifestation of motor aura, and is believed to be caused by cortical spreading depression. Hemiplegic migraine can mimic several other neurological disorders, i.e. stroke/transient ischaemic attack, encephalitis/meningitis, and seizures [4, 5].

Migraine and epilepsy are both frequent episodic neurological disorders that share many clinical features and underlying pathophysiological mechanisms. Hemiplegic migraine is often misdiagnosed as epilepsy, especially in children or in adults who have attacks without headaches.

Furthermore, somnolence, decreased consciousness, confusion, and even episodes of coma can occur during hemiplegic migraine attacks, leading to an incorrect diagnosis of epilepsy and consequently unsuccessful treatment [6]. Hemiplegic migraine can be caused by a mutation in the *CACNA1A*, *ATP1A2* and *SCN1A* genes. The *SCN1A* gene has frequently been linked to epilepsy. What's more, seizures can occur frequently in patients with familial hemiplegic migraine [7].

Electroencephalography (EEG) is not a standard examination for the diagnosis of migraine. Nevertheless, it is useful for making a differential diagnosis with seizures [5]. Different types of EEG abnormalities, most commonly unilateral or bilateral delta activity, have been reported during hemiplegic migraine attacks with aura. However, only a few cases of EEG performed during an episode have been published [7, 8]. Epileptiform discharges can be recorded in patients having epilepsy independently of hemiplegic migraine attacks [5]. Of course, in the case of plegic/paretic aura of migraine and/or focal EEG recording, it is necessary to perform neuroimaging i.e. magnetic resonance imaging of the brain [3].

We here present the case of a man with left- and right-sided hemiplegic migraine attacks along with EEG recordings obtained during episodes.

A 24-year-old man was referred to the epilepsy clinic with multiple episodes of impaired responsiveness and weakness of the right arm since the age of 15. He had initially been diagnosed with epilepsy and had been treated with carbamazepine 800/d without improvement.

The medical history revealed episodes of numbness and paresis that began in the right hand and gradually spread to the arm and face, accompanied by aphasia and drowsiness. These symptoms usually lasted 40-90 minutes and were followed by a severe, throbbing left-sided headache with photophobia and phonophobia, nausea, and vomiting. These symptoms resolved completely between attacks. By the date of the first evaluation, the patient had experienced 11 stereotypical episodes.

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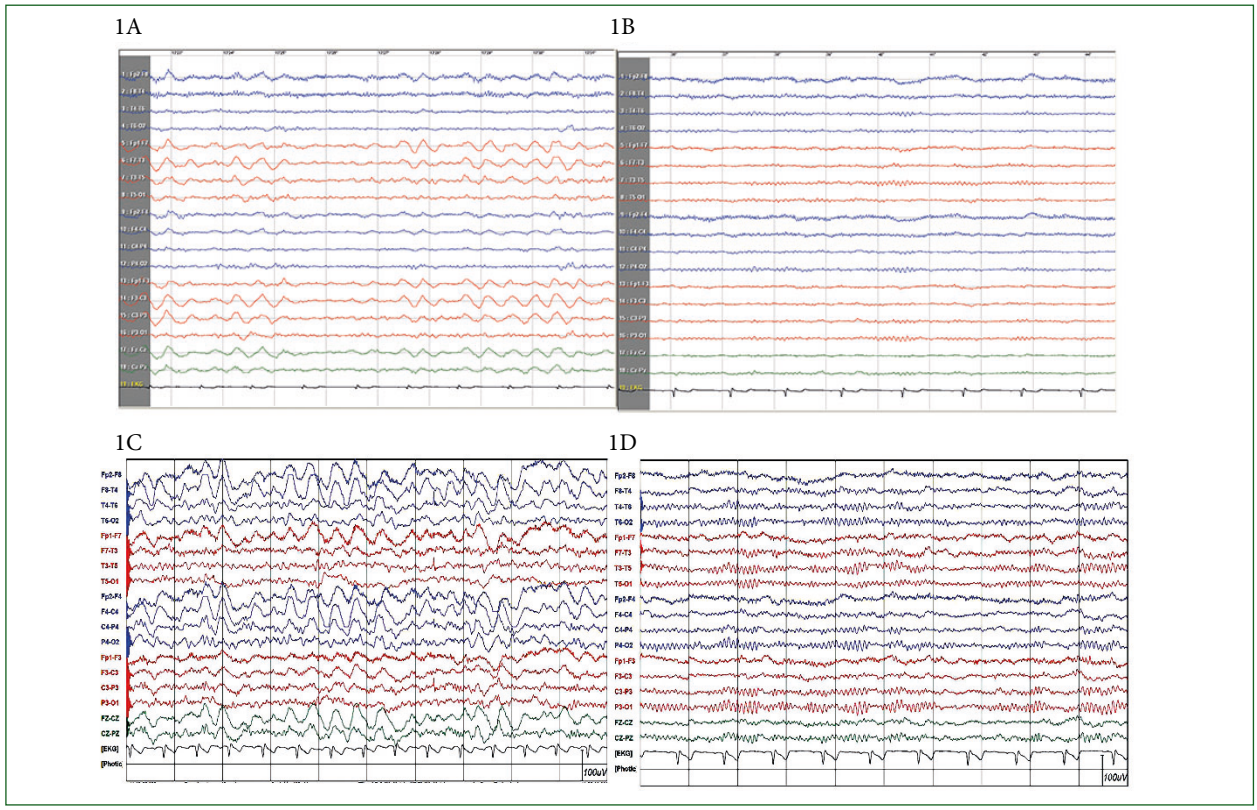

Figure 1. **A.** EEG during episode with right-sided paresis: delta waves dominating over left hemisphere; **B.** EEG one week after episode: alpha rhythm 11 Hz; **C.** EEG during episode with left-sided paresis: delta waves dominating over right hemisphere; **D.** EEG four days after episode: alpha rhythm 11 Hz.

Magnetic resonance imaging (MRI) and angio-MRI performed between episodes were normal. An EEG taken on the day of the first visit to our clinic revealed background activity consisting of alpha rhythm in the occipital regions and beta waves in the frontal regions, with normal reactivity to eyes opening and symmetric photic driving during photostimulation.

He had no family history of epilepsy or migraine.

Three months later, the patient came to the clinic 40 minutes after the onset of a stereotypical event. He was drowsy, had a slight right arm paresis, and mixed aphasia, followed by a severe pulsatile headache with nausea, photophobia, and phonophobia. Scalp EEG obtained during this attack revealed slowing over the left hemisphere (Figure 1A); a follow-up EEG was normal (Fig. 1B). The total duration of aphasia and right arm paresis was 80 minutes. Hemiplegic migraine was diagnosed, and carbamazepine was withdrawn.

Four years later, he experienced an episode of paresis of the left arm and hemiface followed by a right-sided headache. The total duration of the paresis was 60 minutes. EEG performed during that episode showed slowing over the right hemisphere (Fig. 1C), which resolved within one week (Fig. 1D).

The diagnosis of migraine without aura is usually straightforward, although migraine with visual aura or hemiplegic migraine can mimic a seizure disorder. The following points help to distinguish a migraine attack from a focal seizure. Most seizures last from a few seconds up to three minutes, whereas migraine aura last more than five, and up to 60, minutes. If three symptoms (i.e. visual disturbances, aphasia, and paresis) occur during an aura, the acceptable maximal duration is three times 60 minutes (i.e. 180 min) [1]. Black and white, brilliant scotoma, or linear zig-zag patterns or negative symptoms (visual field defect, blindness) suggest migraine, while multicolour circular patterns or complex hallucinations suggest epilepsy. Where alteration of consciousness, automatisms, or tonic clonic-seizures occur, these suggest epilepsy [9].

EEG is not recommended for routine evaluation of patients with migraine, but it may be helpful in cases that raise the suspicion of a seizure disorder. EEG abnormalities in patients with migraine have been reported in several studies. In most cases they have been nonspecific and included focal, diffuse or hemispheric slowing of the background rhythm, plus abnormalities during activation procedures such as hyperventilation or photostimulation. During attacks of migraine with aura, hemiplegic migraine or migraine with brainstem aura, unilateral or bilateral slowing has been reported. Epileptiform discharges, such as spikes or sharp waves, have been less commonly reported [4,10]. In migraine aura-triggered seizure, also

known as migralepsy, EEG abnormalities have included high voltage, rhythmic 11–12 Hz activity with spikes, high voltage theta activity with sharp waves, and bilateral continuous spike-wave and slow-wave discharges [11].

No epileptiform patterns, including di- or tri-phasic waves with sharp or spiky morphology or seizure patterns, such as repetitive epileptiform EEG discharges at > 2 Hz and/or characteristic pattern with quasi-rhythmic spatio-temporal evolution, lasting ≥ 10 s, electrodecrement, and low voltage fast activity have been recorded during our patient's episodes [12]. Transient and fully reversible lateralised slowing in the delta range was seen in our patient.

These EEG abnormalities contradict the previous diagnosis of epilepsy, and support our diagnosis of hemiplegic migraine. The diagnosis of hemiplegic migraine in our patient was based on the clinical picture, albeit this was supported by the capture of two episodes on EEG.

To the best of our knowledge, this is the first case of EEG recordings obtained in a patient during episodes of left-sided and right-sided hemiplegic migraine. We had the opportunity to document that an EEG pattern of unilateral slowing, contralateral to the affected body side, characterises the acute episodes, with complete resolution of abnormalities in an asymptomatic state. Slowing during the acute episode can reflect depression of cortical reactivity or vasoconstriction and normal follow-up EEG resolution of cortical changes.

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