
Migraine-like Symptoms Triggered by Occipital Lobe Seizures: Response to Sumatriptan

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ABSTRACT: Background: The relationship of ictal and post-ictal cephalic pain to migraine remains debatable. We hereby report 2 patients whose post-ictal migraine-like symptoms ameliorated in response to oral sumatriptan. **Methods:** Two patients, a 27-year-old woman and 41-year-old man were referred for assessment of migraine of 10 years and 4 years duration, respectively. The man described a recent episode of "vacant stare" and the woman has sensitivity to bright colours and light. Neurological examination, visual field testing, CAT scan, MRI scan, and prolonged EEG recordings were accomplished for each patient. **Results:** The man had a spontaneous seizure with ictal discharge arising from the right occipital lobe. Bilateral occipital spikes, associated with palinopsia was triggered in the woman by photic stimulation. Both patients developed post-ictal headaches with symptoms similar to their migraines. The symptoms subsided following treatment with oral sumatriptan. **Conclusion:** Migraine-like symptoms occurring following occipital lobe seizures may share similar pathophysiological substrates with idiopathic migraine. Serotonergic mechanisms may be implicated.

RÉSUMÉ: Manifestations d'allure migraineuse déclenchées par de l'épilepsie occipitale: réponse au sumatriptan. Introduction: La relation entre la céphalée ictale et post-ictale et la migraine demeure controversée. Nous rapportons le cas de 2 patients dont les symptômes d'allure migraineuse ont bien répondu à l'administration orale de sumatriptan. **Méthodes:** Deux patients, une femme de 27 ans et un homme de 41 ans, ont été référés pour évaluation de migraines présentes depuis 10 ans et 4 ans respectivement. L'homme a décrit un épisode récent de regard fixe et vague et la femme avait une hypersensibilité aux couleurs vives et à la lumière. On a procédé à un examen neurologique, des champs visuels, un CAT scan, une RMN et des enregistrements EEG prolongés chez chaque patient. **Résultats:** L'homme a eu une crise spontanée avec une décharge ictale provenant du lobe occipital droit. Des pointes occipitales bilatérales associées à une palinopsie ont été déclenchées chez la femme par stimulation photique. Les deux patients ont développé une céphalée post-ictale accompagnée de symptômes semblables à leurs migraines. Les symptômes ont régressé avec l'administration orale de sumatriptan. **Conclusions:** Les symptômes d'allure migraineuse survenant suite à de l'épilepsie occipitale peuvent avoir des bases physiopathologiques semblables à celles de la migraine idiopathique. Des mécanismes sérotoninergiques pourraient être impliqués.

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That a link exists between epilepsy and migraine has been debated for over a century. Liveing sparked the controversy by describing migraine as resulting from "nerve storm" in the brain, akin to epilepsy.¹ Gowers also supported the contention that migraine and epilepsy originated from disturbances of the cerebral cortex and included migraine in the borderline of epilepsy.²

In the last 20 years, interest in the relationship between epilepsy and migraine has been rekindled by reports of children having the syndrome of "epilepsy of childhood with occipital paroxysms."³⁻⁸ Camfield et al.⁹ described 4 patients with migraine-like symptoms, focal-onset seizures and striking epileptiform paroxysms in the posterior temporal-occipital regions. They postulated that their patients had basilar migraine and attributed the epileptiform disturbances to ischemia induced by migrainous vasoconstriction in the basilar artery territory.

More recent experience indicates that patients with clinical and EEG findings similar to those of Camfield et al.⁹ have epilepsy of occipital lobe origin and may experience post-ictal migraine-like

symptoms.^{7,8,10} The conflicting conclusions underline the difficulties in distinguishing between migraine with visual aura and simple partial occipital lobe seizures with post-ictal migraine-like symptoms. The distinction can only be incontrovertibly made by obtaining EEG recordings at the time of the attacks.

We describe two adult patients who we investigated for recurrent episodes of visual hallucinations followed by headache, nausea and photophobia. Both of them were originally diagnosed to have "migraine with visual aura". Both had relief of their post-ictal migraine-like symptoms using the antimigraine medication, sumatriptan.

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REPORT OF PATIENTS

Patient One

This 41-year-old man has a 4 year history of episodic visual disturbances and throbbing headaches. He usually had the attacks about once every 2 weeks although on occasions, he has had as many as 3 attacks within 24 hours. They always began with hallucination of flashing, bright circle or line of light in his left visual field. The visual hallucination often lasted 2 minutes or longer. Whenever the hallucination lasted for more than 5 minutes, a throbbing fronto-temporal headache associated with nausea and photophobia invariably developed. There was relief of his headache and nausea using subcutaneous sumatriptan. Propranolol was also effective in reducing the frequency of headaches but was not helpful in decreasing attacks of visual hallucinations. Sibelium and lorazepam had no effect in preventing the visual disturbances with or without the headaches.

On one occasion, he experienced an episode of palinopsia; while driving through the town, he began to see the houses which he had seen about 5 minutes before. He stopped driving for a few minutes and the episode subsided. Again, he had throbbing bilateral fronto-temporal headache after the visual disturbance was over.

His physical examination, neurological examination, formal visual field testing and CT scan of the brain were normal. MRI scan revealed an area in the inferomedial part of the right occipital lobe (anterior calcarine cortex) which was hypo-intense on T1 and hyper-intense on T2 weighted images. The lesion was interpreted as focal cortical dysplasia. It showed no change in size with repeated MRI scans over 3 years (Figure 1, see Arrow).

A spontaneous attack was recorded using long-term video-EEG. Flashing light suddenly began in his left visual field gradually enlarging to involve his entire field of vision. About 20 minutes later, he complained of throbbing bifrontal headache, nausea and photophobia. Associated with the visual hallucination, the EEG showed rhythmic theta activities and several seconds later, high-amplitude spikes in the right parietal-occipital region (Figure 2). The headache, nausea and photophobia commenced about 12 minutes after the termination of the ictal discharge. Oral sumatriptan produced complete resolution of the post-ictal headache, nausea and photophobia within 30 minutes.

Patient Two

A 27-year-old woman has had attacks of throbbing headaches for 10 years. She related the symptoms to exposure to bright lights and shiny colours. Visits to shopping malls or offices with fluorescent lights often precipitated headaches. Other occasional precipitants included viewing floors, walls or objects with lime green, orange or red colours. She has had isolated, occasional brief palpitations since age 16 years.

The general medical examination was normal except for a grade 2/6

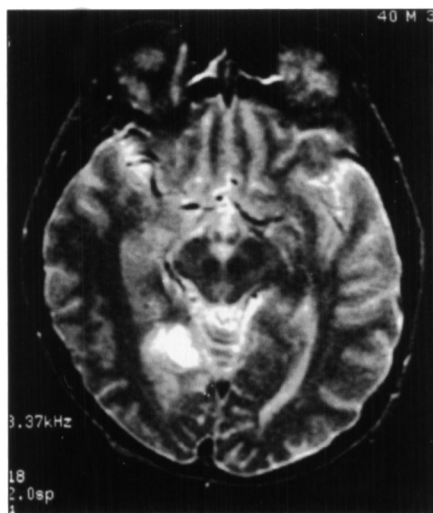


Figure 1: MRI scan of the brain showing an area of cortical dysplasia in the inferomedial portion of the right occipital lobe.

mid-systolic murmur. The neurological examination was normal. ECG, echocardiogram, CT scan and MRI scan of the brain were normal. Mitral valve prolapse was diagnosed by a cardiologist and a neurologist diagnosed migraine.

EEG recording was obtained with 2 sessions of photic stimulation. The EEG remained normal until the second session of photic stimulation when repetitive spikes were triggered in the right occipital region. The ictal discharge (Figure 3), lasted about 5 minutes and was associated with elementary visual hallucination (flashing light). She complained of severe throbbing pain in the right eye and nausea, when the EEG showed post-ictal delta activities in the right posterior head region (Figure 4). The attack precipitated by the photic stimulation was quite similar to her episodes of "migraine". Treatment with oral sumatriptan completely suppressed the post-ictal symptoms.

DISCUSSION

Headache is the dominant symptom of migraine but is also commonly associated with epileptic seizures. Headache may rarely be an ictal manifestation^{12,13} but is more commonly a post-ictal phenomenon.^{3,4,8,11,14} The symptoms often associated with the headache of migraine such as nausea, vomiting, photophobia and irritability may also attend post-ictal headache.¹⁵

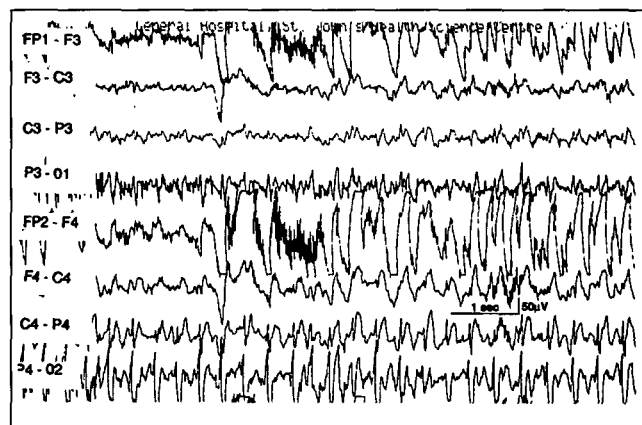


Figure 2: EEG of Patient 1 shows ictal discharge in the right parietal-occipital region, during elementary visual hallucination.

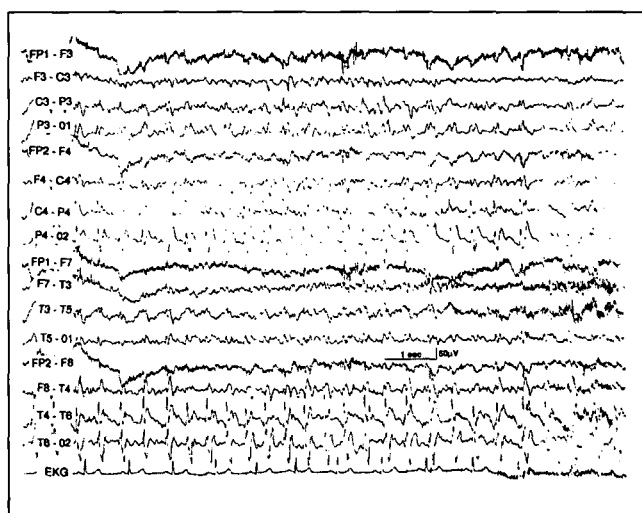


Figure 3: EEG of Patient 2 showing ictal discharge in the right occipital region. The focal discharge was triggered by photic stimulation.

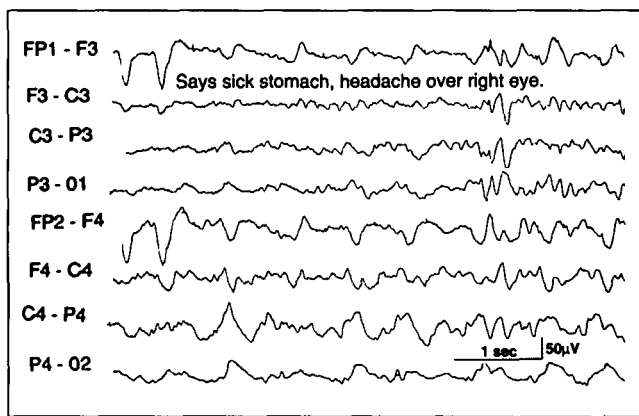


Figure 4: EEG of Patient 2 at the onset of nausea and pulsating pain in the right eye. Note the delta slow waves.

The Sequence of Symptoms and EEG Findings

The attacks recorded in our patients indicated clearly that the visual hallucinations were ictal in origin. The EEG showed concomitant focal ictal discharges in the posterior head regions (Figures 2 and 3). In contrast, both patients began to experience throbbing headaches only after the termination of the ictal discharges and when post-ictal delta waves prevailed in the posterior head regions. This relationship between the EEG findings and the symptomatology provides direct evidence that headache with migraine-like features may be triggered by occipital lobe seizures. It does not however exclude the possibility that the patients had coincidental migraine and epilepsy.^{16,17}

The Effect of Sumatriptan

We elected to treat our patients with sumatriptan for a number of reasons: (i) the patients' head pain had qualities similar to those of migraine, (ii) they had previously experienced relief of their symptoms with the medication and (iii) there was a possibility that the associated symptoms of nausea, photophobia and irritability may be aborted by using sumatriptan rather than analgesics which may require co-medication with antiemetic or sedative drugs.

It is of interest that both patients were relieved of their migraine-like symptoms after oral sumatriptan. Since they were quite aware that they were taking a medication which had previously proved beneficial, we cannot clearly ascribe the suppression of symptoms as a specific reaction to the sumatriptan. A placebo-controlled treatment in a much larger number of patients would be more definitive. Nevertheless, our observation suggests that a trial of sumatriptan or other serotonin receptor agonists may be warranted for patients having severe post-ictal migraine-like symptoms.

Post-Ictal Headache and Serotonergic Mechanisms

Sumatriptan is an antimigraine drug with a specific mechanism of action. It is a serotonin (5-hydroxytryptamine, 5-HT_{1D}) receptor agonist.¹⁸ The favourable response of our patients post-ictal migraine-like symptoms to sumatriptan points to the possibility that serotonergic mechanisms may be implicated in the pathophysiology of post-ictal headache. It is unclear whether the salutary effect is mediated through vasoconstriction in trigeminal-innervated vasculature¹⁹ or by blockade of neural transmission in trigeminovascular axon terminals.²⁰

Long Term Treatment

Both patients were started on carbamazepine monotherapy because the video-EEG recordings indicated that epileptic seizures of occipital lobe origin were likely the underlying reason for their recurring symptoms. During 15 months' follow-up, both of them have remained free from visual symptoms and headache.

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REFERENCES

1. Liveing E. "On Megrin, Sick-Headache and Some Allied Disorders." London, J. & Churchill, 1873.
2. Gowers WR. "The Borderland of Epilepsy." London, J. & A. Churchill, 1907.
3. Commission on Classification and Terminology of the International League Against Epilepsy. Proposal for revised classification of epilepsies and epileptic syndromes. *Epilepsia* 1985; 26: 268-278.
4. Gastaut H. A new type of epilepsy: benign partial epilepsy of children with occipital spike-waves. *Clin Electroencephalogr* 1982; 13: 13-22.
5. Gastaut H, Zifkin BG. Benign epilepsy of childhood with occipital spike and wave complexes. In: Andermann F, Lugaresi E, eds. *Migraine and Epilepsy*. Boston: Butterworths, 1987; 37-81.
6. Panayiotopoulos CP. Basilar migraine? Seizures and severe epileptiform EEG abnormalities. *Neurology* 1980; 30: 1122-1125.
7. Panayiotopoulos CP. Benign childhood epilepsy with occipital paroxysms: a 15-year prospective study. *Ann Neurol* 1989; 26: 51-56.
8. Maher J, Ronen GM, Ogunyemi AO, Goulden KJ. Occipital paroxysmal discharges suppressed by eye opening: variability in clinical and seizure manifestations in childhood. *Epilepsia* 1995; 36: 52-57.
9. Camfield PR, Metrakos K, Andermann F. Basilar migraine, seizures and severe epileptiform EEG abnormalities: a relatively benign syndrome in adolescents. *Neurology* 1978; 28: 584-588.
10. Aicardi J, Newton R. Clinical findings in children with occipital spike wave complexes suppressed by eye opening. In: Andermann F, Lugaresi E, eds. *Migraine and Epilepsy*. Boston: Butterworths, 1987; 111-124.
11. Walker MC, Smith SJM, Sisodiya SM, Shorvon SD. Case of simple partial status epilepticus in occipital lobe epilepsy misdiagnosed as migraine: clinical, electrophysiological and magnetic resonance imaging characteristics. *Epilepsia* 1995; 36: 1233-1236.
12. Laplante P, Saint-Hilaire JM, Bouvier G. Headache as an epileptic manifestation. *Neurology* 1983; 33: 1493-1495.
13. Young GB, Blume WT. Painful epileptic seizures. *Brain* 1983; 106: 537-554.
14. Schon F, Blau JN. Post-epileptic headache and migraine. *J Neurosurg Psychiatry* 1987; 50: 1148-1152.
15. Guerrini R, Dravet C, Genton P et al. Idiopathic photosensitive occipital lobe epilepsy. *Epilepsia* 1995; 36: 883-891.
16. Andermann E, Andermann FA. Migraine-epilepsy relationships: Epidemiological and genetic aspects. In: Andermann FA, Lugaresi E, eds. *Migraine and Epilepsy*. Boston: Butterworths, 1987; 281-291.
17. Ottman R, Lipton RB. Comorbidity of migraine and epilepsy. *Neurology* 1994; 44: 2105-2110.
18. Hoyer D, Clarke DE, Forzard JR et al. International Union of Pharmacology classification of receptors for 5-hydroxy tryptamine (serotonin). *Pharmacol Rev* 1994; 46: 157-203.
19. Humphrey PPA, Feniuk W. Mode of action of the anti-migraine drug sumatriptan. *Trends Pharmacol Sci* 1991; 12: 444-446.
20. Moskowitz MA. Neurogenic versus vascular mechanisms of sumatriptan and ergot alkaloids in migraine. *Trends Pharmacol Sci* 1992; 13: 307-311.