
Expert Opinion

Postictal Headache and Migraine

Case History submitted by Randolph W. Evans, MD

Expert Opinion by Jack Gladstein, MD

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The borderland of epilepsy is as fascinating today as it was to Gowers in 1907.¹ Is a postictal headache (PIH) a migraine? Would the headache respond to a triptan?

CLINICAL HISTORY

A 40-year-old man was seen for evaluation of spells. In 1985, he sustained a gunshot wound to the head and underwent 2 craniotomies. Since then, he has had spells occurring about once every 1 to 2 months. He sees a bubble in the left visual field for about 2 to 3 minutes. His wife stated that he then stares and has semipurposive movements with his hands for about 2 minutes and is then confused. After the event, he always has a severe right-sided throbbing headache associated with nausea, vomiting, and light and noise sensitivity. He goes to bed and the headache lasts about 1 day. He has no other headaches or history of headaches before the trauma. He took phenobarbital for several years after the surgery without a reduction in the seizures and stopped the medication himself. He has not had any further evaluation or treatment for the seizures since and, in fact, he and his wife did not know that they were seizures. Neurological examination demonstrated a right-sided craniotomy defect, a

minimal left facial paresis, left hemiparesis, left hyperreflexia, and circumduction of the left leg with walking. He was started on phenytoin.

Questions.— Is the headache a migraine triggered by the partial complex seizure? Would the headache respond to a triptan? What types of headaches can occur postictally? Can a headache be the only manifestation of a seizure?

EXPERT COMMENTARY

In this case, a 40-year-old man had no history of migraines or seizures before being shot in the head. He is left with deficits consistent with a right-sided lesion affecting his motor abilities. He now has “spells” once every month or 2. These spells start with 2 minutes of visual symptoms, followed by an alteration of consciousness and weird semipurposive arm movements. He then gets a nasty headache with autonomic features. Diagnosis is clear here. His aura is not migraine, but part of his partial complex seizures. The aura is short-lived (2 minutes), and its shape is not characteristic of migraine visual aura (bubble vs. wavy lines). This visual aura is then followed by the next phase of a partial complex seizure. As you know, partial complex seizures are characterized by alterations of consciousness, along with some focal findings. These can be motor, somatosensory, autonomic, or psychic in nature. The seizure can arise with changes in mental status from the beginning, or evolve from a simple partial

Address all correspondence to Dr. Randolph W. Evans, 1200 Binz #1370, Houston, TX 77004; or Dr. Jack Gladstein, University of Maryland School of Medicine, 655 West Baltimore Street, M-004, Bressler, Baltimore, MD 21201.

seizure, where there is no change in mental status at the onset.² As with our patient, there are automatisms, described here as “semipurposeful movements.” His partial complex seizure is followed by a headache that would easily fit International Headache Society (IHS) criteria.³ He has severe, throbbing 1-sided headache with autonomic symptoms that last until the next day.

This patient’s migraine should respond to a triptan. Jacob describes 2 patients with migraine after seizure who responded well after receiving 100 mg of sumatriptan orally.⁴ Although the seizure was his migraine precipitant, his migraine pathophysiology should not be different. Therefore, the headache should be treated with good antimigraine drugs. This man should receive either nasal spray or injectable triptan, since his nausea and vomiting might limit the effectiveness of an oral preparation.

The relationship between migraine and epilepsy has been discussed for over a century, and is based upon prevailing theories of disease pathogenesis. Around the turn of the century, when the art of medicine was in the description and association of findings, Gowers wrote “Some surprise may be felt that migraine is given a place in the borderland of epilepsy, but the position is justified by many relations.” This theory of “nerve storms”⁵ was replaced by the vascular hypothesis.⁶ Since ergotamine, a potent vasoconstrictor, worked in aborting migraine attack, it was theorized that the cerebral vasoconstriction and vasodilation of a seizure could be involved in the genesis of a migraine. As Leao’s model of spreading depression took hold, authors theorized that depression of brain function in both conditions may link them to a common pathogenesis.⁷ We now know that migraine can lower seizure threshold by increasing potassium and glutamate release and disturbing gamma-aminobutyric acid (GABA) function.^{8,9} This explains how a migraine can trigger a seizure. Interictally, migraineurs may have a defect in mitochondrial oxidative phosphorylation and have a low intracellular magnesium.¹⁰ The resultant “excitable brain” may account for the propensity to both migraine and seizures in the same individual.

After a partial complex seizure, one can have headaches with a predominance of migrainous features or not. The incidence of PIH varies by population studied. D’allesandro described 240 patients

with seizures who were over age 16. He found that 174 had partial complex seizures. Out of these, 23 (13%) had PIH, and 10 (6%) had migraine.¹¹ Schon and Blau interviewed 100 patients with both “major and minor” epilepsies. They found that 51% of those questioned had PIHs. They claim that almost all of these headaches had migrainous features. However, they did not use strict IHS criteria. As expected, patients with grand mal seizures had a much higher incidence of headaches. Eight of 9 patients with interictal migraine had migraine immediately after their seizure.¹² Ito distinguished between patients with occipital lobe epilepsy (OLE) from those with temporal lobe epilepsy (TLE). They found that 62% of patients with OLE had PIH, but only 23% of TLE patients had PIH. In sharp contrast to Schon and Blau, they state that very few patients had migraine. Again, no IHS criteria were applied.¹³ In another study by Ito et al, PIH occurred in 41% of TLE patients, 40% of frontal lobe epilepsy patients, and 59% of OLE patients.¹⁴ PIH occurred significantly more frequently in subjects with generalized tonic-clonic seizures. In a study of patients with refractory partial complex seizures, seen for neurosurgical intervention, Bernasconi found 50% of patients with TLE had PIHs, of which 60% were migraines. This study did use IHS criteria.¹⁵ Finally, Velioglu studied 412 adults with various seizure disorders and found that 14% had seizure-induced migraine.¹⁶

Karaali-Savrun and colleagues¹⁷ reported the presence of interictal headaches in 50 (40.7%) of 135 seizure patients (109 with partial onset and 26 with generalized onset). Seventy-nine (58.51%) patients had periictal headache. Eleven of these patients had preictal headache, 3 had ictal headache, and 56 of these had PIH. Preictal headache and PIH were more frequently encountered before and after secondary generalized tonic-clonic seizures compared to other seizure groups. The type of pain in PIH was “throbbing” in complex partial seizures and “steady” in generalized tonic-clonic seizures.

We are taught not to order an electroencephalogram (EEG) for the routine patient with migraine, since the implications of an abnormal EEG in clinical management is difficult. However, the “never say never” rule does apply. There have been reports of patients with headache as the sole manifestation of their

seizure. Pain can be 1 of several types of somatic sensations, with partial complex epilepsy. Other sensations include tingling paresthesias, thermal sensations, and sexual sensations. These rarely occur alone. Siegel described 8 cases of partial complex seizures whose main complaints were of pain. In all of the cases, the pain did not hint of run of the mill migraine.¹⁸ It is easier to confuse the visual auras of migraine and partial complex seizures. In migraine, the aura is gradual in onset, monochromatic, and wavy. In partial complex seizures, it is more rapid in onset, with complex, round colorful shapes.¹⁹

This patient would certainly benefit from a triptan to treat his acute headaches, and may benefit from topiramate for both seizure and migraine prophylaxis.

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