Symptomatic Occipital Epilepsy Misdiagnosed as Migraine

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Two young patients with symptomatic occipital lobe epilepsy due to discrete lesions of cysticercosis were misdiagnosed and treated for 2 years as migraine with visual aura. The patients suffered from frequent visual seizures often followed by migraine-like headache. Seizures manifested with colored and mainly circular elementary visual hallucinations of up to 1 minute duration. Headache, often severe and of long duration, was frequently associated with nausea, photophobia, and phonophobia. Both patients became seizure-free with appropriate treatment of the underlying disease and epileptic seizures.

Key words: migraine, epilepsy, occipital epilepsy

Episodic elementary visual hallucinations followed by headache are common and with the same sequence in migraine with aura\(^1\) and visual occipital lobe seizures.\(^2\) However, elementary visual hallucinations of visual seizures are markedly different in quality, location, and chronological development in comparison with the visual aura of migraine.\(^3,4\)

Elementary visual hallucinations of occipital seizures are brief for seconds to 3 minutes, develop fast within seconds and are predominantly colored and circular.\(^3,4\) They usually start in the periphery of a hemifield and often march to other seizure symptoms or convulsions. Conversely, visual aura of migraine consists mainly of achromatic zigzag linear patterns, starts in the center of the visual field, it gradually progresses over \(>4\) min usually lasting \(<30\) min toward the periphery of one hemifield and often leaves a scotoma.\(^5\)

Postictal headache, often indistinguishable for migraine as defined by the International Classification of Headache Disorders (ICHD-2),\(^1\) occurs in more than half of patients with visual seizures.\(^4\)

Misdiagnosis of visual occipital seizures as migraine appears to be common with significant repercussion on management issues.\(^1,4\)

We report 2 patients with symptomatic occipital epilepsy who were misdiagnosed as migraine with aura though ictal clinical symptoms were typical of visual epileptic seizures despite severe postictal migraine like headache.

CASE REPORTS

First Case.—A 14-year-old boy started having brief attacks of elementary visual hallucinations followed by headache at age of 11 years. These consisted of flashes of light simulating a yellow bulb being turned on and off intermittently in his right visual hemifield. They lasted for 30 seconds to 1 minute and were strictly unilateral. These episodes were often followed by left-sided headache associated with nausea, vomiting, photophobia, and phonophobia. The headache started immediately after the end of the visual
seizures, lasted for 1 to 2 hours and was minimally relieved with analgesics and sleep. The visual seizures with or without headache were diurnal and mainly in the morning hours with no apparent triggering factors.

There was no family history of migraine. His father had a single episode of afebrile convulsions in his childhood.

The diagnosis of migraine with visual aura was made and he was treated with β-blockers and flunarazine with no improvement. On the contrary, his attacks became more frequent and at the age of 13 years one of his habitual seizures progressed to a generalized tonic clonic convulsion.

Routine biochemical investigation was normal. Brain X-ray computerized tomography scan (CT) showed a ring-enhancing lesion in the left occipital lobe with minimal edema suggestive of neurocysticercosis (Fig. 1). EEG was normal. The diagnosis of symptomatic occipital epilepsy was made and the patient was treated with albendazole for 21 days and phenytoin 200 mg daily. There were no further epileptic seizures or significant headaches of any type in 1-year follow-up.

**Second Case.**—A 14-year-old girl had a 2-year history of colored, rainbow-like flashes of light in the left visual hemifield lasting less than 1 minute and followed by headache. The headache started 1 to 2 minutes after the end of the visual symptoms, was right-sided, and often associated with nausea, occasional vomiting, photophobia, and phonophobia. The duration of headache was 4 to 5 hours, which significantly restricted her daily activities.

All headaches were preceded by the visual symptoms. Some of the symptoms were precipitated by stress, strong smells, hunger, and lack of sleep. Their frequency was 3 to 4 episodes in a month. She was diagnosed as having migraine with visual aura and was unsuccessfully treated with β-blockers. The attacks became more frequent in the last 6 months and on 2 occasions they progressed to generalized tonic clonic seizures. There was no family history of migraine or epilepsy.

Routine biochemical investigations were normal. Her EEG had mild and nonspecific abnormalities. Brain CT scan showed a ring-enhancing lesion in the right parieto-occipital area with minimal edema suggestive of neurocysticercosis (Fig. 2). Patient was treated with albendazole for 21 days and carbamazepine. No further seizures or headache occurred in 2 years of follow-up and brain CT scan normalized.

**COMMENTS**

Both patients of this report had symptomatic occipital lobe epilepsy as documented with brain imaging, demonstrating discrete occipital lesions of cysticercosis. They were misdiagnosed and treated as migraine with visual aura for 2 years thus depriving them from appropriate diagnostic procedures and management; cysticercosis is a common, treatable, and reversible cause of epilepsy in developing countries. In both patients, the correct diagnosis was established only after the onset of generalized convulsions.
The reason for such misdiagnosis is mainly 2-fold:

1. Elementary visual hallucinations of occipital seizures are inappropriately equated with visual aura of migraine an error which could be avoided if visual hallucinations were assessed in a qualified and systematic manner.

2. Postictal headache that frequently (in more than half of patients) occurs after occipital seizures is often indistinguishable from migraine as defined by the ICHD-2.1

In addition, these cases confirm the emergence of migraine headache after brief nonconvulsive visual epileptic seizures in an epilepsy-migraine sequence, which appears to be more common than the prevailing view of a migraine-epilepsy succession in occipital lobe and epilepsies in general. It is possible that occipital seizures activate trigemino-vascular or brain stem mechanisms responsible for migraine headache.8

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REFERENCES


A Migraine-Like Headache Induced by Carotid-Cavernous Fistula

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Carotid-cavernous fistula (CCF) generally causes periorbital aching with ocular symptoms due to high venous pressure in the cavernous sinus, while migraine is caused by arterial dilatation-stimulating trigeminal nerves around the vessels. The authors present a case of 47-year-old woman with a 4-month history of a temporal throbbing headache. As her symptoms were well controlled by triptans, her headache was considered to be migraine in type. However, a Barrow’s type-D CCF was revealed by radiological examinations. Self-compression of common carotid artery method was initially tried for therapy of the CCF, but endovascular embolization was finally necessary due to intractable headache. Although the headache was considered arterial in origin, transvenous embolization of the left cavernous sinus successfully ameliorated the patient’s symptoms. CCF should be considered as an unusual etiology of headaches that appear arterial in origin.

Key words: carotid-cavernous fistula, endovascular embolization, migraine, transvenous, triptans

Abbreviations: CCF carotid-cavernous fistula, NSAIDs nonsteroidal anti-inflammatory drugs, MRI magnetic resonance imaging, MRA magnetic resonance angiography, AV arterio-venous, T2WI T2-weighted imaging, AP anterior-posterior

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A periorbital headache is one of the common symptoms in carotid-cavernous fistula (CCF), and the headache is caused by high venous pressure of cavernous sinus stimulating the peripheral trigeminal nerve. Therefore, most CCF cases present with complaints of ocular symptoms such as proptosis, diplopia, subconjunctival hemorrhage, chemosis,