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Migraine with aura complicated by "migraine triggered seizures" and "occipital lobe infarction": A case report

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Abstract

Migraine with aura is one of the major subtypes of migraine, and can be associated with ischaemic brain infarction. Use of oral contraceptive pills (OCPs) increases the risk of infarction in this type of migraine. Seizures and migraine also have a complex relationship, one element of which is migraine-triggered seizures. We report a case of bilateral occipital lobe infarction and migraine-triggered seizures, most likely precipitated by oral contraceptive pills (OCPs) in a patient with migraine with visual aura. OCPs, triptans and ergotamines should be used cautiously in these patients. Methods of birth control other than OCPs should be considered.

INTRODUCTION

Ischemic stroke and migraine are known to have a complex relationship. Many studies have shown that there is an increased risk of ischemic stroke in patients who have migraine with aura compared with patients without aura.[1] The risk is further magnified by the combination of smoking and/or the use of oral contraceptive pills (OCPs), in the absence of other vasculopathic risk factors.[1] The involvement of the posterior circulation territory in migrainous stroke is a disastrous complication of migraine that can affect the vision. We report a case of migraine- triggered seizures and bilateral occipital lobe infarction in a patient with migraine with visual aura.

CASE REPORT

A 32-year-old school teacher had been experiencing episodes of scintillating scotoma 3-4 times per year since the age of 18 years old. These were characterized by stereotyped repetitive geometric patterns of multicolored lights lasting 5-10 minutes with no succeeding headache. After the delivery of her first child, she began to sporadically experience a left temporo-occipital throbbing headache following her visual aura lasting a few hours. At the age of 30-year old, after her second child was born, she began taking OCPs for a total course of one year, with a resultant increase in the frequency and severity of the visual aura and headaches that started at 3 months while she was on OCPs. After 8 months on OCPs, she had 3 hospital admissions of one month interval in between hospitalisation. During the first admission, she developed intense scintillations aura with a typical migrainous headache, that lasted for 9 hours, and experienced a generalized tonic-clonic seizure during the peak of the headache. Upon recovery, her scintillations scotoma persisted without any other neurological deficits. A month later, she developed a nearly identical presentation of headache with visual aura and seizure. During the second event, she also had sudden, severe bilateral vision loss to the extent of only being able to perceive hand movements. Her vision recovered within 2 days, but the scintillating scotomas persisted. There were no other neurological deficits. On her third hospital admission, she again developed an intense visual aura with the sudden onset of reduced vision bilaterally followed by a migrainous headache. This was illustrated by her subsequently in Figure 1. She again perceived only hand movements with greater involvement of her central vision than the peripheral vision.

The patient had a history of recurrent miscarriages, including two miscarriages at the age of 25-year old. Her father had also experienced similar symptoms of migraine with visual aura.

On assessment of her higher cortical function, speech and orientation were normal. She had

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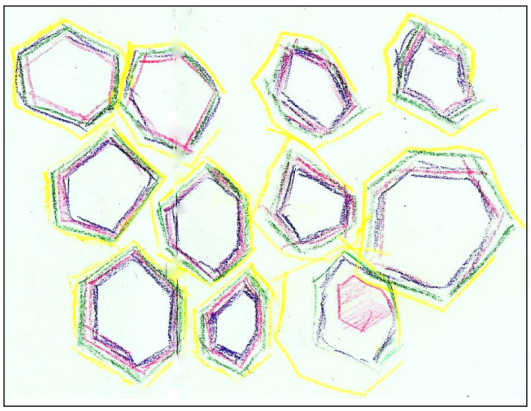

Figure 1. This picture (which was drawn by the patient) shows multicolored, geometric patterns of the patient's scintillating scotoma at her initial presentation.

cerebral achromatopsia and Riddoch phenomenon, in which a visual stimulus was perceived during movement but not with static presentation. Further ophthalmic examination showed vision sufficient for only hand movements bilaterally with central scotoma. Funduscopy showed a healthy disc and retina. Other cranial nerve examinations were normal. The motor and sensory systems were unremarkable.

Blood investigations revealed a normal platelet count, ESR, C-reactive protein, antithrombin III, protein C and protein S levels; and negativity for antinuclear antibody (ANA), rheumatoid factor (RF) and anticardiolipin antibody. Magnetic resonance imaging (MRI) showed bilateral occipital infarcts and diffusion weighted MRI Imaging (DWI) showed an area of restricted diffusion in both occipital lobes predominantly over the right side, indicating an edematous area of recent infarct. (Figure 2, 3) Magnetic resonance angiography (MRA) and venography (MRV) were normal, with no evidence of arterial or venous thrombosis. Electroencephalography (EEG) showed frequent inter-ictal epileptiform discharges from the right occipital lobe and infrequent discharges from the left occipital lobe. Visual evoked potential (VEP) was normal. Echocardiography showed no evidence of patent foramen ovale (PFO).

The patient was started on intravenous methylprednisolone 1gram daily for 3 days, and carbamazepine CR 200mg twice per day. Soon, the visual acuity in both eyes improved such that she was able to count fingers. The visual aura also changed its pattern and quality 5 days after admission. This was illustrated by the patient in Figure 4. She was hospitalized for 11 days. Upon discharge, the right eye improved to 6/45, while the left eye vision remained at the ability to count fingers. She was discharged on oral aspirin and carbamazepine. During follow-up at one month, the visual acuity in both of her eyes improved to 6/45. There was no recurrence of her severe migrainous headache. At follow up, 12 months

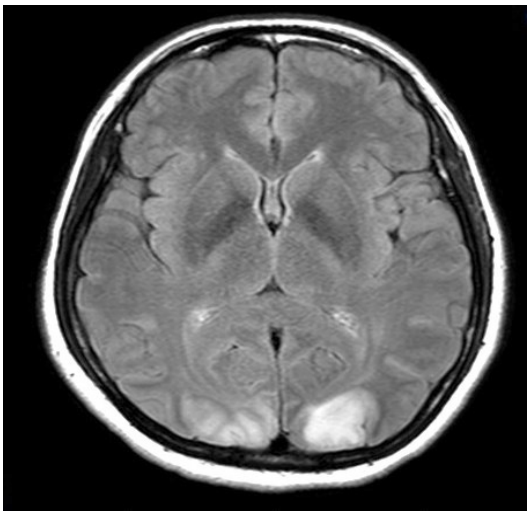

Figure 2. Magnetic resonance imaging (MRI) FLAIR showing hyperintensity over the occipital lobes bilaterally, suggesting infarct.

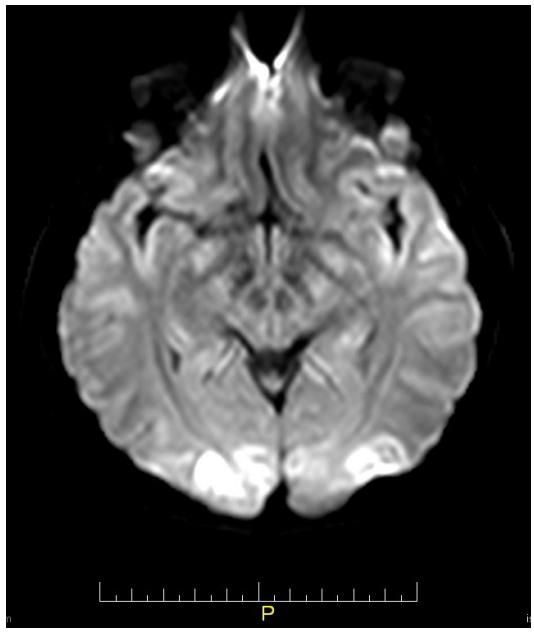

Figure 3. Magnetic resonance imaging diffusion-weighted image (DWI) revealing restricted diffusion predominantly over the right occipital lobe, suggesting edema and recent ischemia.

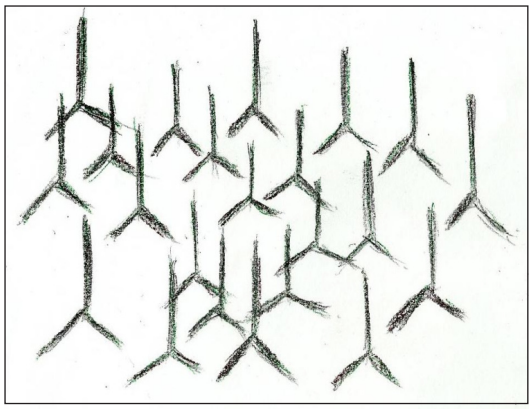

Figure 4. This picture (which was drawn by the patient) shows the persistence of the patient's visual aura after occipital lobe infarction, which was a dark, repetitive, unformed geometric pattern.

after the last admission, visual acuity both eyes had improved to 6/24.

DISCUSSION

Isolated migraine aura per se without headache typically affects older people who have had aura with migraine headache at younger ages.[2] However, it can also occur in younger people as an initial symptom of migraine. Based on the International Classification of Headache Disorders (ICHD), the initial presentation in this patient fit the diagnosis of migraine with typical aura without headache.[3] She had a typical visual aura, which was described as dynamic and multicolored adopting a geometric pattern. The aura developed gradually, lasted less than 1 hour and was completely reversible.[4] In contrast, aura in ischemic attack is static, dark and dimmed in quality, lasts for a shorter time than migraine aura.[4]

Adolescent onset of visual aura can also be a manifestation of occipital lobe seizure. The stereotyped and repetitive patterns of visual aura in migraine resemble visual hallucinations in partial seizure. However, ictal symptoms are usually brief, only lasting for seconds and consists of mainly of multiple unformed, brightly-coloured, small circular spots or circles. It may cause blindness in the early course of the disease, and post-ictal headache is a common symptom.[5]

Evidence from the literature has shown that migraine with visual aura is a significant risk factor for ischemic stroke.[1] The use of OCPs not only causes worsening of pre-existing aura but also further multiplies the risk of ischemic stroke. Migrainous infarction is a rare complications following migraine with visual aura. The incidence has been estimated at 3.36 per 100,000 persons, and it occurs more commonly in women younger than 45 years old.[6] According to the ICHD, migrainous infarction is defined as cerebral infarction occurring during a typical attack of migraine with visual aura.[3] Although there is a known association between migraine with aura and ischemic stroke, the underlying pathogenesis remains unknown. Purported mechanisms include vasospasm, arterial dissection, small vessels arteriopathy, hypercoagulability and cardioembolism (such as in patent foramen ovale).[7] In addition, alterations in the cerebral blood flow and volume, combined with activation of the clotting system can results in arterial or venous thrombosis or ischemia.[8] Cortical spreading depression (CSD) as a result of neuronal excitability has been suggested to generate migraine with visual aura.[9] This CSD is associated with characteristic cerebral blood flow changes and can cause tissue hypoxia and infarction.[9]

Reversible cerebral vasoconstriction syndrome (RCVS) can present in the same manner as migrainous infarction. Typically, the headache in RCVS is severe and excruciating, with evidence of multiple vasoconstrictions in the cerebral arteries on angiography. The CT and MRI is normal in most cases.[10] Another possible differential diagnosis is posterior reversible encephalopathy syndrome (PRES). Patients with PRES can present with headache, visual loss and seizures, similar to the symptoms found in the patient. The imaging findings show vasogenic edema, particularly in the occipital-parietal region. PRES usually occurs following an acute rise in the blood pressure such as in eclampsia, infection, autoimmune disorder or with the use of immunosuppressive drugs.[11] None of these factors were found in our patient, although the symptoms and imaging might have indicated PRES.

In patients with migraine, MRI of the brain may show increased frequency of white matter hyperintensities.[12] In migraine patients with aura, a higher prevalence of subclinical infarcts can be found in the posterior circulation territory. The CAMERA study showed an association between migrainous aura and infarct-like lesions in the cerebellum and brainstem.[13] Impairment of the adaptive cerebral hemodynamic mechanism in the posterior circulation during the attack makes this site susceptible to infarction.[14]

This patient also fulfilled the ICHD criteria for migraine-triggered seizure.[3] The seizure

occurred within an hour following the episode of migraine with aura. This phenomenon is a rare and is sometimes referred to as migralepsy. However, migraine-like headaches are frequently observed during post-ictal period. Migraine and epilepsy share several common pathogenic mechanisms. Cortical spreading depression (CSD), imbalance between excitatory-inhibitory glutamate-mediated transmission and abnormal activation of voltage-operated ionic channels, has been implicated in both migraine and epilepsy.[15] Thus, it is not surprising that anti-epileptic drugs are also effective as anti-migraine agents which could treat both of our patient's problems.

The management of migrainous infarction currently based on physicians' clinical experience, case reports and the pathogenesis of the disease.[6] Vasoconstrictive drugs such as ergotamines and triptans should be used with caution. Various prophylactic treatments had been tried in order to prevent ischemia and infarction, which include nitrates, nifedipine, furosemide, valproate, acetazolamide, ketamine, prochlorperazine and magnesium. An anti-epileptic drug, lamotrigine, which blocks sodium channels and glutamate release can reduce migraine aura. Low-dose aspirin (75-150 mg daily) is often prescribed as secondary prophylaxis after the infarction. Persistence of visual aura is a rare complication of migraine with aura, and it may possibly reflect bilateral infarction in the occipital lobes.

In conclusion, although migraine with aura is a benign condition with a low-risk of ischemic infarct, the identification of patients who are at higher risk for developing cerebral infarction is essential in managing the patient . The use of oral contraceptives, triptans and ergotamines should be used with caution. Women of reproductive age should consider methods of birth control other than using OCPs.

DISCLOSURE

Conflicts of interest: None

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