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CASE REPORT

Iatrogenic visual aura: a case report and a brief review of the literature

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Abstract: Iatrogenic migraine aura following transseptal catheterization has only rarely been reported in the literature. We report the case of a 60-year-old female who presented with new onset of migraine with visual aura 1 day after transseptal cryoballoon catheter ablation for atrial fibrillation. The patient had a 5-year history of typical migraine without aura and had never experienced visual aura before the cardiac intervention. The neurological examination, fundoscopy, and blood tests were normal. The magnetic resonance imaging of the brain showed small vessel ischemia without evidence of vessel ischemic changes in the occipital lobes and large blood vessel disease. A change in the characteristics of existing migraine could occur following an iatrogenic episode, which in this case was catheter ablation for atrial fibrillation. A new onset of aura is considered an indication for a brain scan as it may signify underlying new pathology.

Keywords: migraine aura, iatrogenic, transseptal catheterization

Introduction

Migraine aura consists of focal neurological symptoms comprising unilateral fully reversible visual, sensory, or other central nervous system symptoms.[1] The migraine aura usually develops gradually over 5 to 20 minutes and lasts less than 60 minutes and is usually followed by headache and associated migraine symptoms.[1] Visual aura is the most common type of aura, occurring in >90% of patients with migraine with aura.[1]

The diagnostic criteria of migraine with aura in International Classification of Headache Disorders (ICHD)-3 beta include:

A. At least two attacks fulfilling criteria B and C.
B. One or more of the following fully reversible aura symptoms:
    1. visual,
    2. sensory,
    3. speech and/or language,
    4. motor,
    5. brain stem,
    6. retinal.
C. At least two of the following four characteristics:
    1. at least one aura symptom spreads gradually over 5 minutes and/or two or more symptoms occur in succession,
    2. each individual aura symptom lasts 5–60 minutes,
    3. at least one aura symptom is unilateral,
    4. the aura is accompanied, or followed within 60 minutes, by headache.
D. Not better accounted for by another ICHD-3 diagnosis, and transient ischemic attack has been excluded.

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Case presentation

A 60-year-old right-handed nonsmoker female was seen in the Hull Headache Clinic for recurrent episodes of flashing lights, which started in the left visual field and progressed over 1 hour to cover most of the visual field. This lasted for 10–15 minutes and was followed by a severe left-sided headache associated with nausea, photophobia, and phonophobia but without autonomic features or other neurological deficits. The attacks occurred 1 day after she had endocardial pulmonary vein cryoballoon ablation for paroxysmal atrial fibrillation using an interatrial septal puncture with inguinal approach. The venous access during the procedure was difficult and eventually gained with two left femoral vein punctures in the left and a single right femoral vein puncture in the right. SL-1 long sheath was used with BRK transseptal needle to perform the puncture. Once access was gained into the left atrium, 10,000 units of heparin was administered. The activated clotting time (ACT) was kept above 350 seconds. The venogram demonstrated four separate pulmonary veins, and all veins were isolated using a 28 mm cryoballoon.

The patient had daily attacks for 2 weeks, which slowly improved, and eventually, she was left with five to six episodes of migraine with aura per month, lasting for 2 hours when taking paracetamol. In the last 5 years, the patient experienced similar left periocular and temporal headaches once a month lasting for 3–4 days, associated with nausea, photophobia, and phonophobia (migraine without aura). Before that, she used to get the attacks during menstrual cycles only. There was a family history of migraines (father). The patient has a history of paroxysmal atrial fibrillation and hypertension. Her regular medications are rivaroxiban, propafenone, and ramipril. On examination, her weight was 114 kg, and the blood pressure was 136/69 mmHg. The neurological examination and fundoscopy were normal. Her plasma viscosity, C-reactive protein, complete blood count, serum electrophoresis, and kidney and liver functions were within normal limits. Her autoimmune screen showed increased anti-ds-DNA to 9 IU/L (range 0–8) without clinical significance. The MRI head showed minimal T2/FLAIR hyperintensities related to small vessel ischemia without evidence of large blood vessel disease and vessel ischemic changes in the occipital lobes. The MRI diffusion-weighted imaging (DWI) and apparent diffusion coefficient (ADC) did not show abnormalities. The MRI head has been performed 5 months after the onset of migraine aura. Our patient was not keen on migraine prophylaxis but opted to optimize her acute attack analgesic treatment while avoiding analgesic overuse. Written informed consent was obtained from the patient for publication of her medical data for this case report.

Discussion

Iatrogenic migraine aura is an uncommon phenomenon.[2] The most common visual symptoms of migraine aura are the positive ones such as photopsia (unformed flashes of light), teichopsia or fortification spectrum, scotoma (partial loss of sight or a "blind spot" that is often crescent shaped), visual distortion, "heat waves," blurring, and hemianopsia.[3]

Migraine aura can be induced by intravascular procedures such as transseptal catheterization for atrial fibrillation ablation,[4] catheter ablation for Wolff-Parkinson-White (WPW) syndrome,[5,6] percutaneous foramen ovale (PFO) closure,[7] endovascular balloon dilation and stenting procedures,[8] cerebral angiography by carotid puncture,[9] vertebral angiography,[10] and sclerotherapy for varicous veins.[11] A systematic review of potential methods for the provocation of migraine has been conducted by Lindblad et al in 2016.[12] The systematic review has identified studies of experimental migraine aura provocation by administration of sublingual glyceryl trinitrate,[13] calcitonin gene-related peptide infusion,[14] intravenous injection of insulin,[15] visual stimulation,[16] chocolate,[17] or physical activity.[18]

Only a few cases of isolated migraine aura in patients undergoing transseptal catheterization (TSC) have been described before.[4] The exact mechanism of this phenomenon is not well understood. A study conducted by Chilukuri et al[4] in 2009 included 571 patients who underwent TSC over a 3-year duration. Of these, three patients presented with visual symptoms in the first month after the procedure; two patients with a history of atrial fibrillation and no prior history of headaches or visual disturbance developed visual symptoms after catheter ablation for atrial fibrillation.[4] However, the third patient had a history of migraine with aura and paroxysmal ventricular tachycardia and developed sudden onset of visual symptoms with no associated headache next day after she underwent catheter ablation of left-sided accessory pathway.[4] Iatrogenic visual aura has also been described in patients after catheter ablation for WPW syndrome.[5,6] Saravanan et al[5] reported the case of a 55-year-old woman with a history of migraine with aura who underwent ablation procedure for WPW syndrome and developed visual symptoms during the procedure. Other invasive cardiac interventions have been reported to influence migraine.[7] A study conducted by Mortelmans et al[7] in 2005 included 114 patients who underwent PFO closure. In 12 patients who suffered from migraine before closure,

the migraine disappeared. New onset migraine was noted in 10 patients.[7] However, a recent study conducted by Larrosa et al[19] in 2016 has shown that PFO is not more common or larger in chronic migraine than in episodic migraine patients. The study findings do not support a relationship between PFO and migraine frequency.[19]

In recent times, the pathophysiology of migraine has been focused more as a neurovascular disorder rather than purely vascular. The trigeminovascular system, neurogenic inflammation, and activation of certain areas such as the brain stem may play an important role in the pathogenesis of migraine. Although the pathophysiology of the migrainous aura is poorly understood, cortical spreading depression (CSD) is considered to be the cause of migraine aura. The slowly propagating wave of depolarization across the cortex, especially in the occipital lobes, accounts for the progression of visual symptoms typical of migraine aura.[20] Although the exact mechanism behind migraine aura induced by intravascular procedures is not known, there are possible theories that could explain the phenomenon. The mechanism could be related to changes in the regional brain blood flow,[21] possible toxicity of contrast substances,[22] or release of endothelin-1 from the carotid endothelium that possibly induces CSD, as it has been shown in the experimental studies on rats.[23]

Intravascular procedures should be performed with periprocedural anticoagulation and therapeutic international normalized ratio to avoid silent or symptomatic brain embolization. A prospective study conducted by Mohanty et al[24] in 2015 evaluated the effect of catheter ablation and periprocedural anticoagulation regimen on the clinical course of migraine in atrial fibrillation patients with or without pre-existent migraine. In most patients with a known history of migraine, the symptoms have improved substantially after catheter ablation. Interestingly, the only cases of new migraine and aggravation of pre-existent headache had subtherapeutic international normalized ratio during the procedure and new cerebral infarcts.[24] New onset of visual aura will require brain imaging to rule out new underlying pathology such as cerebral vascular infarcts.

Conclusion

We have hereby reported the case of a new onset typical visual aura 1 day after transseptal catheter ablation for atrial fibrillation, followed by the patient's usual headaches. Although migraine and migraine aura have been reported in association with invasive cardiac interventions, the exact relationship between these migraine phenomena and cardiac procedures is poorly understood. A possible mechanism could be related to the release of endothelin-1 from the vessel wall that could induce CSD as it has been shown experimentally on rats.[23] More research in this field will contribute to a better understanding of the underlying mechanisms. Brain scan is recommended in a new onset of visual aura to rule out new underlying pathology.

Author contributions

All authors contributed toward data analysis, drafting and critically revising the paper and agree to be accountable for all aspects of the work.

Disclosure

Fayyaz Ahmed has received honorarium to deliver training workshops for Allergan paid to the British Association for the Study of Headache (BASH) and has received honorarium to attend Allergan Advisory Board meetings. He is on the standing committee of the headache guidelines (CG150 Revision) for the National Institute of Clinical Excellence, he is a trustee for the migraine trust, and is an educational officer for BASH. The authors report no other conflicts of interest in this work.

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