Table 2—Surgical and postoperative data

Variable	Case 1	Case 2	Case 3
Surgical data			
Anesthesia	General anesthesia	General anesthesia	General anesthesia
Additional surgical procedures	Phacoemulsification, placement of in-the-bag IOL, and vitrector-assisted peripheral iridectomy	Epithelial debridement and vitrector-assisted peripheral iridectomy	Epithelial debridement, phacoemulsification, placement of in-the-bag IOL, and vitrector-assisted peripheral iridectomy
Tamponade	Air	Sulfur hexafluoride (SF_6) 20%	Sulfur hexafluoride (SF_6) 20%
Donor diameter (mm)	8.0	8.0	8.0
Donor cell count (cells/mm$^{2)}$	2736	3039	2855
Time from donor death to preservation in Optisol (h)	11	36	7
Donor death to use time (days)	3	5	18
Postoperative data			
Follow-up (mo)	20	10	6
UCVA (logMAR)	0.14	0.00	0.38
BSCVA (logMAR)	0.10	−0.10	0.30
Manifest refraction	+1.50/−1.00 × 180	+1.00/−1.50 × 40	+0.50/−1.50 × 170
CCT (μm)	541	462	454
Endothelial cell count (cells/mm$^{2)}$	1658	1936	2004
Cell loss	39.4%	36.2%	29.8%

IOL, intraocular lens; UCVA, uncorrected visual acuity; BSCVA, best spectacle-corrected visual acuity; CCT, central corneal thickness.

In conclusion, our results indicate that DMEK in ICE syndrome is a safe and successful procedure, but further studies are required to compare the long-term outcomes of DMEK with that of DSEK and PK, for treatment of ICE syndrome.

Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.jcjo.2018.02.011.

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Homonymous hemianopsia as the presenting sign of migrainous infarction

Migrainous infarction is a rare complication of migraine headaches that accounts for 0.2%−0.5% of ischemic strokes.[1,2] This condition frequently occurs in the posterior cerebral circulation and typically affects a single vascular territory.[1,2] The most common symptom preceding an acute migrainous infarction is a visual aura (82.3%) that resolves within minutes to hours.[1] Visual field defects like partial or complete homonymous hemianopsia (HH) after a migrainous infarction have been reported, but the prevalence is unknown.[3–7] An Amsler grid can serve as a rapid screen for a homonymous visual field defect. We report a case of migrainous infarction presenting as acute intractable cephalgia with associated left HH whose delay in diagnosis may have been prevented with use of an Amsler grid.

Case Report

A 47-year-old nonsmoking African American female had a history of migraine with visual aura per International Headache Society (IHS) criteria,[8] diabetes mellitus type II (on glimepiride), hyperlipidemia, and hypertension (on losartan-hydrochlorothiazide). Her hemoglobin A1C was 6.9% and left arm blood pressure was 136/80 mm Hg. She received contraceptive depo-medroxyprogesterone acetate (DMPA) every 3 months. She reported compliance with medications and denied recreational drug use.

She awakened with her typical migraine headache accompanied by photophobia, phonophobia, and nausea. After no improvement with oral medications, she went to the emergency room (ER), reporting headache and "bright pinkish spots" in the upper left visual field of both eyes that had not been previously experienced with prior headaches. She was diagnosed with a migraine, treated symptomatically, and discharged. She went to the ER twice more that month with similar results. Head computed tomography (CT) and confrontation visual fields in the ER were normal.

She was evaluated by neuro-ophthalmology 3 weeks later. Visual acuity was 20/20 OU, colour vision normal, and pupils round, equal, and reactive to light with no relative afferent pupil defect. External, motility, slit-lamp, and pressure examinations were within normal limits. Ophthalmoscopy and optical coherence tomography were unremarkable. Automated perimetry revealed a left, denser superiorly, macular sparing, congruous HH (Fig. 1). Amsler grid testing revealed similar findings (Fig. 2).

She was admitted to the hospital for stroke workup. Brain magnetic resonance imaging (MRI) revealed a lesion in the right occipital cortex extending into the calcarine fissure consistent with an ischemic cerebrovascular accident (Fig. 3). Head and neck imaging showed no stenosis, dissection, or fetal posterior cerebral artery (PCA). Vasculitis workup, including 4-vessel cerebral angiogram, was negative. The angiogram showed patent PCAs but revealed multifocal areas of mild intracranial atherosclerotic disease in the right M1 and left M2 segments of the middle cerebral arteries (MCA), so dual antiplatelet therapy (DAPT) was initiated. Electrocardiogram was normal, and transesophageal echocardiogram showed a normal ejection fraction and no endocarditis, thrombus, or patent foramen ovale. Hemoglobin electrophoresis was normal. Factor VIII and anticardiolipin immunoglobulin M antibodies were mildly elevated to 180% and 24 MPL, respectively, but both were normal on repeated testing. Anticardiolipin immunoglobulin G antibodies were negative. Glycoprotein 2B and dilute Russell venom viper time were normal. The remainder of the hypercoagulable workup was entirely negative.

She was diagnosed with a migrainous infarction in accordance with the IHS criteria in the absence of another more accountable diagnosis.[8]

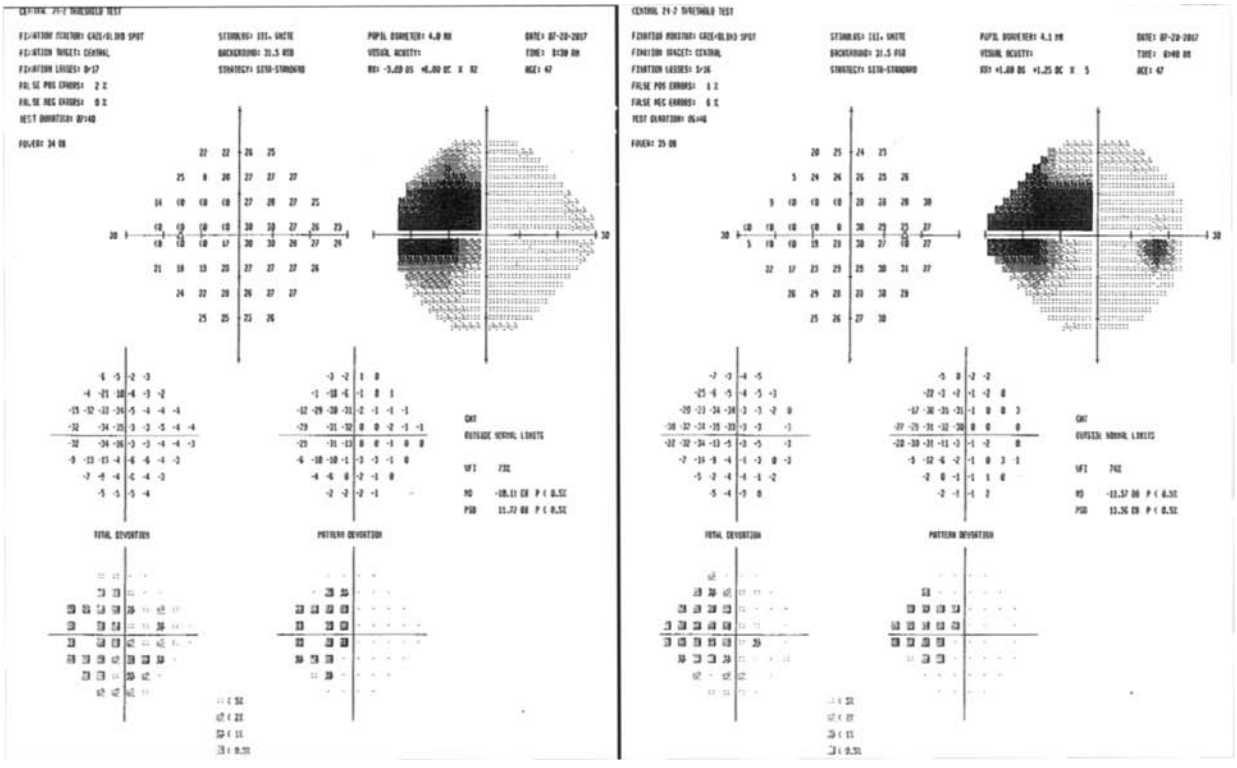

Fig. 1—Humphrey visual field analyzer (HVF 24-2) results demonstrating a left, denser superiorly, homonymous, macular sparing, congruous hemianopsia.

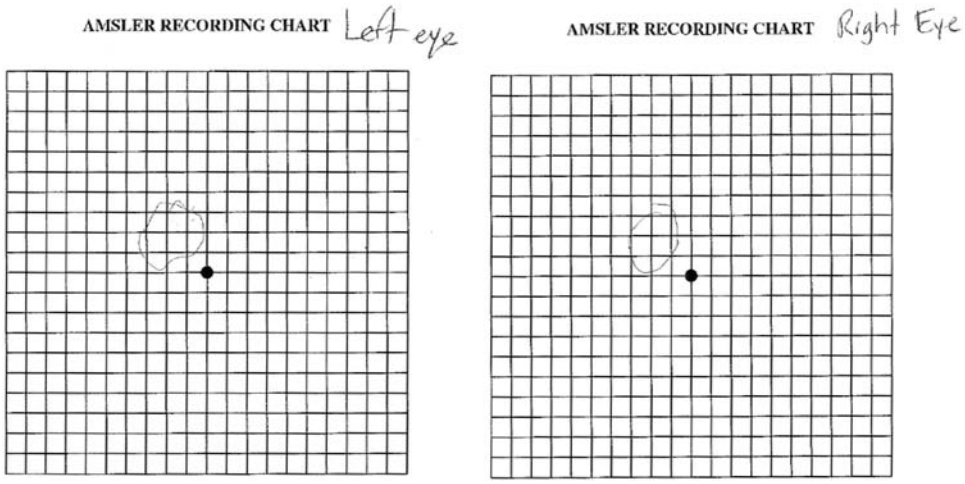

Fig. 2—Amsler grids of the patient's left eye and right eye.

Discussion

Migrainous infarction is a rare complication of migraine headaches found in 3.36 per 100 000 people annually.[8] The likely pathogenic mechanism of migrainous infarction starts with a short-lasting wave of depolarization, known as cortical spread depression

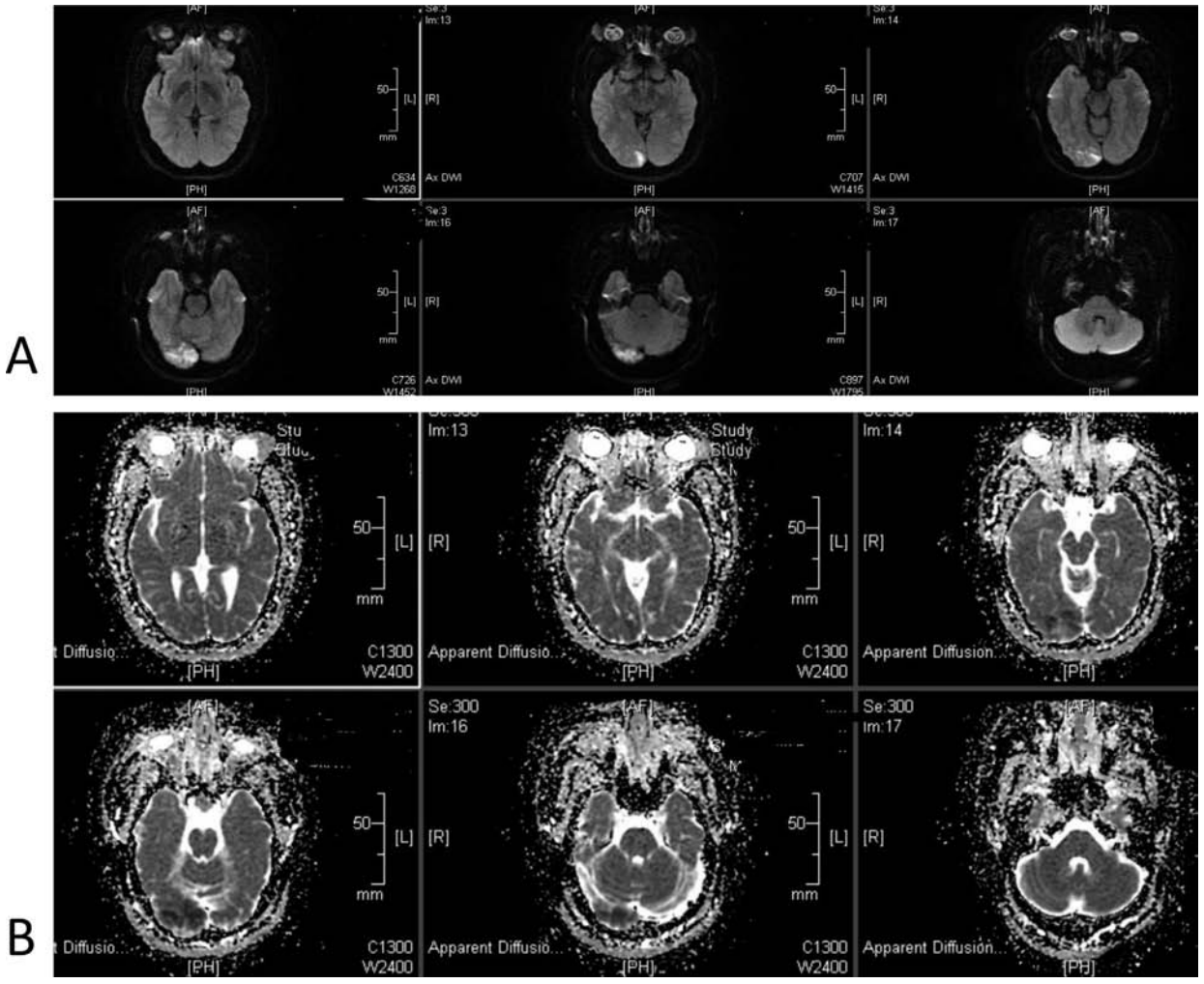

Fig. 3—Axial diffusion weighted magnetic resonance images demonstrating restricted diffusion in the right occipital cortex extending into the calcarine fissure (A) corresponding to hypointensity on apparent diffusion coefficient images (B).

(CSD), that moves from the occipital region to the frontal region at 3–5 mm/min and irritates the trigeminal nerve by releasing inflammatory mediators.[9,10] In a migrainous attack, cerebral hypoperfusion occurs, and the pathologic response to CSD is severe vasoconstriction, leading to infarction.

Further workup is necessary when migrainous infarction is suspected. Our patient had elevated cholesterol controlled with statin therapy, and DAPT was recommended in accordance with the Stenting and Aggressive Medical Management for Preventing Recurrent Stroke in Intracranial Stenosis regimen.[11] A calcium channel blocker was not added to her regimen because angiotensin-converting-enzyme inhibitors are also associated with reduced frequency and severity of migraines.[1] Progestin-only contraceptives (POCs) have been associated with increased risk for blood clots in some studies.[12] However, the U.S. Medical Eligibility Criteria for Contraceptive Use concluded that there is an absence of accurate data on the absolute risk of venous thromboembolism in users of DMPA.[13] Some studies found increases in lipids after starting POCs.[14] The clinical significance of this, however, is unknown. The patient's race is a stroke risk factor. Most importantly, the posterior cerebral arteries were patent. The only findings of atherosclerosis were in MCA regions, but cortical damage was found in the PCA region. Factor VIII and anticardiolipin antibodies are known acute phase reactants,[15] and their normalization within a few days in tandem with her well-controlled medical conditions supports the notion that the single best unifying diagnosis for this patient is a migrainous infarction.

Migrainous infarction presents a diagnostic challenge for clinicians, and initial evaluation lacks nuance. This case is unique because infarction occurred in the context of her typical migraine with aura, and this report contributes to the limited but growing body of literature surrounding the diagnosis and management of migrainous infarction with persistent visual field defects. In some reports, the visual defect is transient.[6] However, these patients were younger, and migrainous infarct was recognized earlier, reinforcing the importance of considering this rare condition in patients with visual defects and migraine history. The Amsler grid, an easy-to-use and effective tool for visual field defects, can accommodate this need especially when small and paracentral homonymous hemianopic visual loss is present. Clinicians should be aware that migrainous infarction can present as the typical migraine headache with persistent "atypical" migraine aura with otherwise normal neurologic and eye examinations. Diagnosis should follow IHS criteria. This is challenging in the ER setting because there is limited, if any, access to automated perimetry. To this end, we advocate for greater use of Amsler grids in emergency settings because confrontational visual fields may miss small defects. Furthermore, MRI should be performed in patients with persistent migraine-related visual loss because CT results can be negative in acute migrainous infarctions.

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