Contents lists available at ScienceDirect



American Journal of Ophthalmology Case Reports

journal homepage: www.elsevier.com/locate/ajoc



Monocular hemianopia secondary to stroke

Samuel K Lee^a, Yao Wang^a, Laura B Asplund^b, Collin M McClelland^a, Michael S Lee^{a,*}

^a Department of Ophthalmology and Visual Neurosciences, University of Minnesota, Minneapolis, MN, USA
^b Isight Family Eye Care, Roseville, MN, USA

ARTICLE INFO

ABSTRACT

Keywords: Purpose: As a general rule, homonymous hemianopic defects localize to the retrochiasmal visual pathway and a Unilateral monocular defect localizes at or anterior to the chiasm. We report three patients with a monocular hemianopia Monocular on automated static perimetry following cerebral stroke. Hemianopsia Observations: In this retrospective, consecutive case series, the charts of individuals presenting with stroke and Hemianopia monocular hemianopia were reviewed. Three individuals suffered cerebral stroke. Automated, static perimetry Retrochiasmal revealed a normal visual field in one eye and a monocular hemianopia in the other eye. No other neurologic, Visual field orbital or ocular causes were found. Conclusions and Importance: To our knowledge, this is the first report of this pattern of visual field loss following stroke, and we hypothesize that this phenomenon may be a unique feature of automated perimetry. Magnetic resonance imaging of the brain could be considered in patients with a monocular hemianopia on static perimetry.

1. Introduction

Retrochiasmal lesions typically result in homonymous hemianopic defects, where a visual field defect that respects the vertical meridian is seen in the same hemifield of each eye.¹ Although lesions of the anterior occipital lobe can cause monocular crescent defects located $60-90^{\circ}$ temporally,² to our knowledge, monocular hemianopic defects from retrochiasmal stroke have not been reported.

2. Findings

2.1. Case 1

A 51-year-old woman awoke with severe retrobulbar pain and described vision loss in the right eye only. Neuroimaging showed acute ischemia along the distribution of the right middle cerebral artery and an abrupt focal segment of critical stenosis at the origin of the right internal carotid artery. She underwent internal carotid artery stenting and mechanical thrombectomy. Afterwards, she felt that the right eye had a persistent field defect and the left eye was normal throughout her clinical course.

Seven weeks after the stroke, her visual acuities were 20/20, both eyes. The pupils and color vision were normal. The remainder of the ophthalmic and neurologic examination was unremarkable except for confrontation visual fields, which showed a nasal hemianopia right eye and normal results left eye. A 24–2 threshold frequency doubling technology visual field showed a left hemianopic defect right eye (0% false positive (FP), 0% false negative (FN) errors) and a normal result left eye (0% FP, 0% FN) (Fig. 1A). Seven months later, repeat testing with a 24-2 Octopus (Haag-Streit, Koniz, Switzerland) automated visual field using the glaucoma tendency oriented program (GTOP) demonstrated a persistent left hemianopic defect right eye (25% FP, 0% FN) and normal results left eye (0% FP, 0% FN) (Fig. 1B).

2.2. Case 2

An 80-year-old man developed cognitive deficits, vertical binocular diplopia, right sided weakness and blurred vision after waking up from colectomy. Brain magnetic resonance imaging (MRI) showed moderate areas of restricted diffusion in the bilateral frontal, parietal, and occipital regions consistent with stroke.

Three months later, his acuities were 20/50 right eye, 20/20 left eye. Besides a comitant 6 prism diopter esotropia and epiretinal membrane right eye, the rest of his ophthalmic examination was unremarkable including normal pupils and color vision. Neurologic examination revealed right-sided hemiplegia and bilateral ataxia. The visual field using the GTOP program showed normal results right eye and a right hemianopic defect left eye with perfect reliability in both

https://doi.org/10.1016/j.ajoc.2020.100758

Received 20 June 2019; Received in revised form 19 March 2020; Accepted 23 May 2020 Available online 28 May 2020

2451-9936/ © 2020 The Authors. Published by Elsevier Inc. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/BY-NC-ND/4.0/).

^{*} Corresponding author. 420 Delaware Street SE, MMC 493, Minneapolis, MN, 55455, USA. *E-mail address:* mikelee@umn.edu (Michael S Lee).

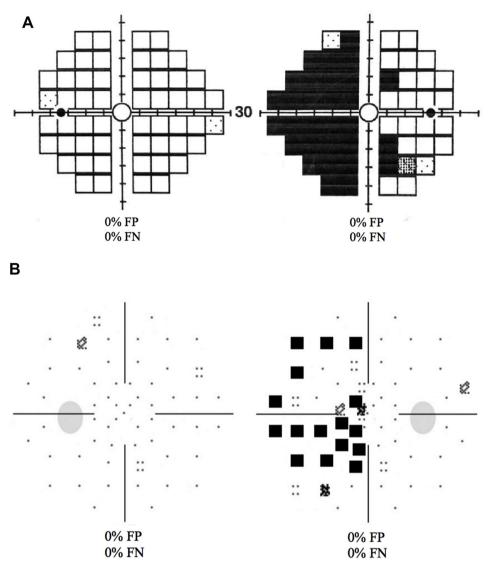


Fig. 1. (A) Pattern deviation plots from frequency doubling technology visual fields from case 1 show a left hemianopia right eye (RE) [0% false positive (FP), 0% false negative (FN) errors] and a normal visual field left eye (LE) [(0% FP, 0% FN errors)]. (B) Pattern standard deviation plots from repeat Octopus visual fields show (A) left hemianopia RE (25% FP, 0% FN errors) and normal field LE (0% FP, 0% FN errors).

eyes (0% FP, 0% FN errors) (Fig. 2). He did not return for follow up.

2.3. Case 3

A 49-year-old man suffered a tonic-clonic seizure. Past medical history was significant for hypertension, diabetes mellitus type 2, dyslipidemia, and diastolic heart failure. In the emergency department, brain computed tomography and MRI revealed a parenchymal hemorrhage in the left occipital lobe measuring $3.1 \times 2.5 \times 3.4$ cm. Perfusion imaging demonstrated matching cerebral blood flow and volume defects in the left occipital, parietal and temporal lobes. He was managed medically and discharged 11 days later. His expressive aphasia resolved over one month, but his visual field defect persisted.

Two months after the stroke, his visual acuities were 20/20, both eyes. His neurologic and ophthalmic examinations were unremarkable including pupils and color vision. The visual fields using the GTOP program revealed normal results right eye (22% FP, 11% FN errors) and a left hemianopic defect left eye (0% FP, 11% FN errors) (Fig. 3). The neurologic examination showed normal strength and sensation throughout, and grossly normal coordination. The patient did not return for follow up.

3. Discussion

We report three patients with a monocular hemianopia secondary to retrochiasmal, contralateral stroke. Monocular hemianopias represent uncommon visual field defects.^{1–3} They have been described with nonorganic disease and various prechiasmal or chiasmal³ defects including cataract, sphenoid wing meningioma, and optic nerve hypoplasia.^{4–6} Monocular temporal hemianopias can also occur with chiasmal compression.³ To our knowledge, monocular hemianopia from retrochiasmal stroke has not been reported. Recently, Zaslavsky and Margolin7 reported a monocular hemianopia from contralateral optic tract compression. They showed corresponding macular GCL loss in the affected eye.

We postulate that the patients herein could have suffered highly incongruous homonymous hemianopias. Since the static stimuli on automated perimetry are six degrees apart, the hemianopic defect could fit between stimuli in the "normal" field. We recognize that manual, kinetic perimetry with Goldmann visual field testing may have unveiled a small hemianopia in the normal eye. However, we believe our findings are extremely relevant to the practicing ophthalmologist, because the vast majority of providers utilize automated perimetry without

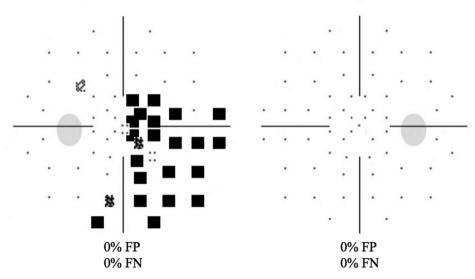


Fig. 2. Pattern standard deviation plots from Octopus visual fields show normal visual field right eye [0% false positive (FP), 0% false negative (FN) errors] and right hemianopic defect left eye (0% FP, 0% FN errors) in case 2.

access to manual perimetry. Alternatively, these patients may have experienced asymmetric improvement in their hemianopia since they were seen several weeks to months after the initial insult. The initial hemianopia may have improved in the normal eye but persisted in the hemianopic eye. The hemianopic defect in the contralateral eye could also have fallen outside the confines of the 24–2 perimetry testing protocol. We acknowledge that patient error could also have resulted in the normal fields. However, case 1 also had similar fields on a second, repeat visual field and the perfect reliability in the normal eye in cases 1 and 2 argue against patient error. Finally, the absence of an afferent pupillary defect paired with a monocular hemianopia could suggest functional vision loss.⁸ Our patients had no apparent secondary gain and the timing of their visual field loss onset coincided with the onset of stroke affecting the visual pathways.

4. Conclusions

In summary, while almost all cases of monocular hemianopia occur from prechiasmatic lesions or nonorganic vision loss, our patients demonstrate that retrochiasmal lesions could be added to the differential diagnosis. Clinicians may want to consider neuroimaging among patients demonstrating a monocular hemianopia on automated, static perimetry.

Funding

No funding was received for this work.

Support

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Authorship

All authors attest that they meet the current ICMJE criteria for Authorship.

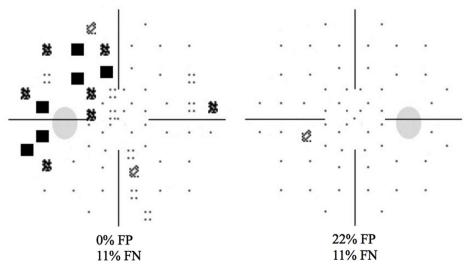


Fig. 3. Pattern standard deviation plots from Octopus visual fields show normal visual field right eye [22% false positive (FP), 11% false negative (FN) errors] and a left hemianopic defect left eye (0% FP, 11% FN errors) in case 3.

Patient consent

The patients consented to publication of the case reports in writing. This report does not contain any personal information that could lead to the identification of the patient.

Disclosures

The following authors have no financial disclosures: SL, YW, LA, CM, ML.

Declaration of competing interest

No conflict of interest exists.

Acknowledgments

None.

References

- Harrington DO. Visual field character in temporal and occipital lobe lesions. Arch Ophthalmol. 1961;66:778–792.
- 2. Ali K. The temporal crescent syndrome. Practical Neurol. 2015;15:53–55.
- Hershenfeld SA, Sharpe JA. Monocular temporal hemianopia. Br J Ophthalmol. 1993;77:424–427.
- Rahman I, Nambiar A, Spencer AF. Unilateral nasal hemianopsia secondary to posterior subcapsular cataract. Br J Ophthalmol. 2003;87:1045–1046.
- Stacy RC, Jakobiec FA, Lessell SM, Cestari DM. Monocular nasal hemianopia from atypical sphenoid wing meningioma. J Neuro Ophthalmol. 2010;30:160–163.
- Brooks DB, Subramanian PS. Monocular temporal hemianopia with septo-optic dysplasia. J Neuro Ophthalmol. 2006;26:195–196.
- Zaslavsky K, Margolin E. Unilateral temporal hemianopsia and nasal ganglion cell loss secondary to optic tract compression. *Ophthalmology*. 2020;127:176.
- Kosmorsky GS, Tomsak RL, Diskin DK. Absence of the relative afferent pupillary defect with monocular temporal visual field loss. J Clin Neuro Ophthalmol. 1992;12:181–191.