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Case Report

Monocular Visual Field Defect on Humphrey 24-2 SITA-Fast Testing Later Identified as a Highly Incongruous Homonymous Defect on Humphrey 30-2 SITA-Fast Testing

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Abstract
Monocular visual field defects generally localize at or anterior to the optic chiasm, while homonymous hemianopias localize to the retrochiasmal visual pathway. Highly incongruous visual field defects may be difficult to identify on 24-2 Humphrey visual field testing, and this case demonstrates the value of optical coherence tomography (OCT) ganglion cell-inner plexiform layer (GCIPL) in rapidly localizing the lesion. A 54-year-old woman was found on routine examination to have an isolated superonasal quadrant visual field defect respecting the vertical meridian in the left eye only on Humphrey 24-2 SITA-Fast testing. She had a remote history of significant head trauma. Visual acuity, anterior segment, and fundus examination were normal. OCT revealed a bow-tie atrophy of the retinal nerve fiber layer in the right eye (OD), and binocular homonymous hemi-macular atrophy of OCT GCIPL, confirming the localization was the left retrochiasmal visual pathway. A repeat Humphrey 30-2 SITA-Fast visual field demonstrated that the visual field defect was also present in the OD in a highly incongruous manner. Magnetic resonance imaging of the brain with contrast showed mild atrophy of the left optic tract. This case demonstrates that highly incongruous visual field defects may be difficult to identify on Humphrey 24-2 SITA-Fast visual fields, and OCT GCIPL serves as a rapid way to localize the lesion. More detailed visual field testing including 30-2 programs should be considered in these cases.

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Introduction

Homonymous hemianopias are caused by lesions in the contralateral retrochiasmal visual pathways, which most commonly involve the occipital lobe, optic radiations, or optic tract [1]. The most common etiologies are stroke, trauma, and brain tumors [1]. They are often associated with homonymous hemi-macular atrophy of the ganglion cell-inner plexiform layer (GCIPL) on optical coherence tomography (OCT). A homonymous visual field defect is considered congruous when it appears similar in both eyes and incongruous when it differs between eyes. The "rule of congruity" states that the more posterior a lesion is within the retrochiasmal visual pathways, the more congruous the defect [2]. We report a case of binocular homonymous hemi-macular atrophy of the GCIPL with only a repeatable monocular visual field defect on Humphrey 24-2 SITA-Fast but a highly incongruous visual field defect on Humphrey 30-2 SITA-Fast visual fields.

Case Presentation

A 54-year-old asymptomatic woman was noted to have a visual field defect respecting the vertical meridian in the left eye (OS) on a routine optometry examination. She had no known medical conditions but reported a history of a motor vehicle accident resulting in significant head trauma requiring hospitalization at age 5. There was an associated right zygomatic bone fracture that was conservatively managed. No further details regarding the admission were available. Neuro-ophthalmic examination revealed a visual acuity of 20/20 in both eyes, no relative afferent pupillary defect, normal color vision, and normal-appearing optic nerves without obvious pallor (online suppl. Figure 1; for all online suppl. material, see www.karger.com/doi/10.1159/000516663). Confrontation visual fields revealed a superonasal visual field defect in the OS and full visual fields in all other quadrants in both eyes. Humphrey 24-2 SITA-Fast visual fields showed a visual field defect only in the superonasal quadrant of the OS, similar to what was seen on the optometry exam (Fig. 1a). OCT retinal nerve fiber layer (RNFL) showed bow-tie atrophy in the right eye (OD) and diffuse thinning in the OS with an average RNFL thickness of 67 and 64 μm, respectively (Fig. 2a). OCT GCIPL showed inferior homonymous hemi-macular atrophy of the nasal retina in the OD and the temporal retina in the OS, with an average thickness of 71 and 67 μm, respectively (Fig. 2b). Magnetic resonance imaging (MRI) of the brain and orbits with contrast showed subtle atrophy of the left optic tract (Fig. 3). Follow-up examination including a repeat visual field (24-2 SITA-Fast) at 6 months was unchanged. However, an additional Humphrey 30-2 SITA-Fast visual field was performed twice at the follow-up visit and revealed a highly incongruous visual field defect as there was small superotemporal defect in the OD (Fig. 1b).

Discussion

Monocular visual field defects localize to the pre-chiasmatic optic nerve or globe, and investigations are guided by the clinical history and examination. In this case, a monocular defect seen on Humphrey 24-2 visual field testing in the right nasal hemifield of the OS was the result of a previous insult to the left retrochiasmal visual pathways. OCT RNFL and GCIPL were instrumental in localizing the lesion and prompted more detailed visual field testing with the Humphrey 30-2 program. On OCT, there was right bow-tie atrophy of the RNFL and homonymous hemi-macular atrophy of the GCIPL in both eyes. There was a functional-anatomical correlation as the left optic tract appeared atrophied on MRI. There were no other

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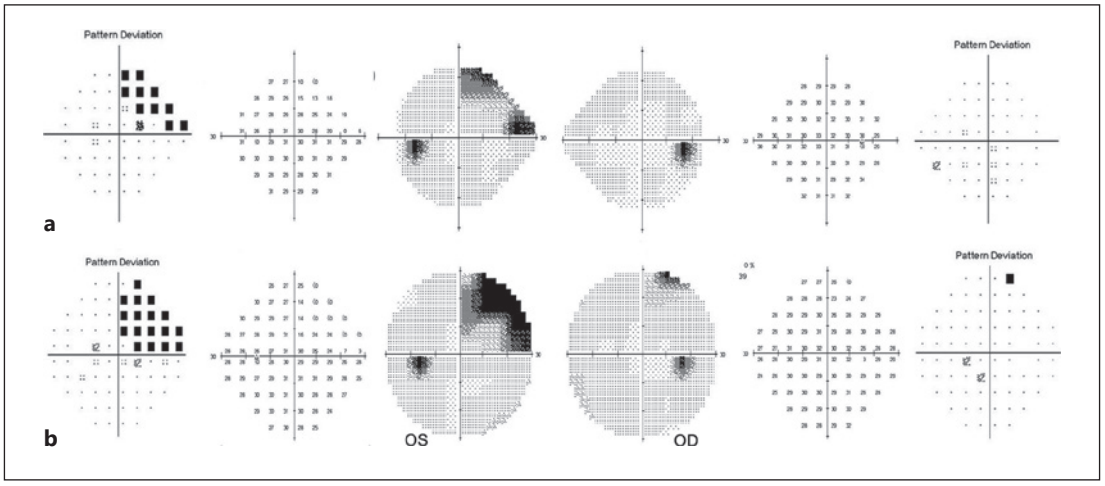

Fig. 1. a Humphrey 24-2 SITA-Fast visual fields of the OS and OD showed defect in the superonasal quadrant of the left eye with mean deviation of −0.79 dB OD and −4.11 dB OS. The pattern deviation, numerical sensitivity plot, and greyscale map are shown for each eye. **b** Humphrey 30-2 SITA-Fast visual fields show a highly incongruous hemianopia with a mean deviation of −1.28 dB OD and −5.72 dB OS. OS, left eye; OD, right eye.

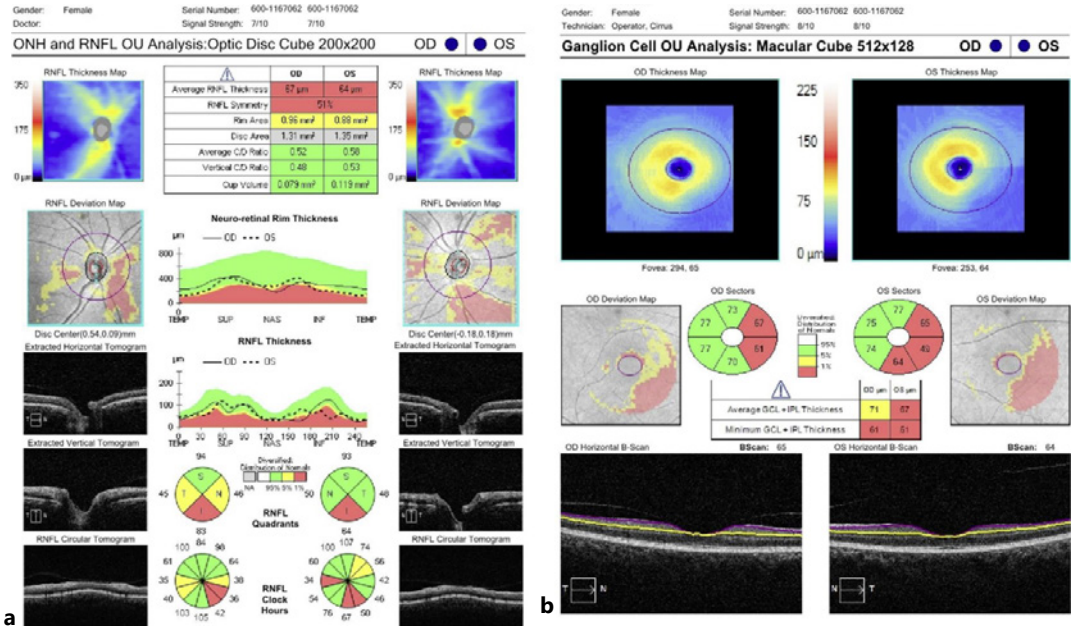

Fig. 2. OCT. **a** RNFL showed bow-tie atrophy OD and diffuse thinning OS. **b** Macular GCIPL showed congruent thinning nasally in the OD and temporally in the OS. OCT, optical coherence tomography; GCPIL, ganglion cell-inner plexiform layer; RNFL, retinal nerve fiber layer; OS, left eye; OD, right eye.

radiological findings in the left retrogeniculate visual pathways. The incongruous nature of the visual field and OCT findings suggests this was a result of primary involvement of the left optic tract, but it may also have been a result of involvement of the left retrogeniculate pathways and secondary retrograde transsynaptic degeneration [3].

Although rare, monocular visual field defects have been reported in the setting of retrochiasmal pathology. A 65-year-old woman was found to have a right temporal visual field

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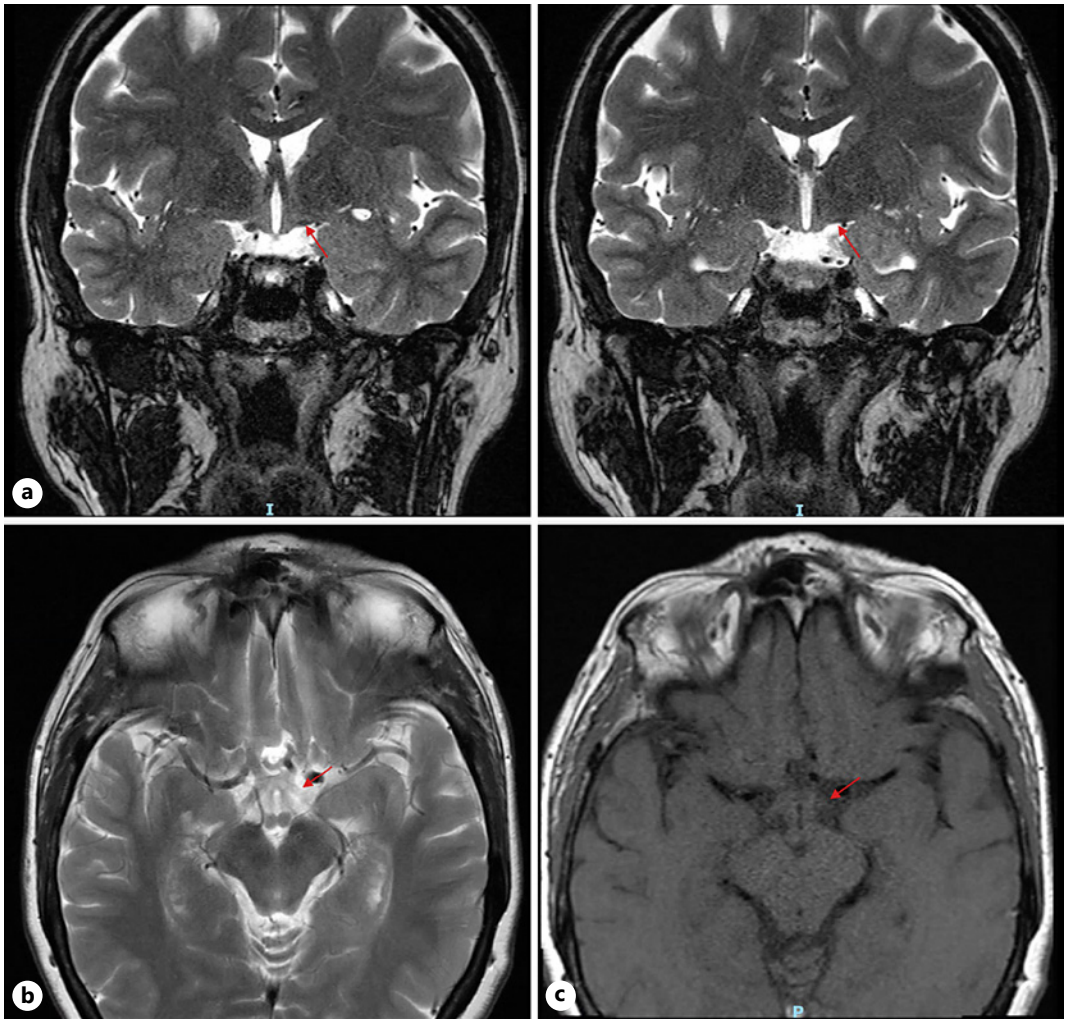

Fig. 3. MRI of the brain showing subtle thinning of the left optic tract on coronal T2 (**a**), axial T2 (**b**), and axial T1 images (**c**). MRI, magnetic resonance imaging.

defect and corresponding nasal hemi-macular OCT GCIPL atrophy due to a left carotid aneurysm compressing the left optic tract [4]. This case utilized Humphrey 24-2 testing, although the unilateral nature of OCT GCIPL defect argues that this may have been truly unilateral. However, the timing of the visual field and OCT in relation to the insult were not reported. With longer term follow-up, it may be possible that the OS manifests OCT GCIPL or 30-2 visual field changes. Lee et al. [5] reported 3 individuals who suffered cerebral stroke but had detectable visual field defects by automated perimetry in only one eye. No OCT data was available in these cases. Only 24-2 programs were utilized in these cases, and it is possible that a more detailed visual field program may show highly incongruous defects. Our case adds to the literature on monocular defects detected on 24-2 visual field programs related to retrochiasmal pathology; uniquely, the defect was later revealed to be highly incongruous on Humphrey 30-2 testing. OCT GCIPL was instrumental in this case as it was able to rapidly demonstrate that a monocular defect was the result of retrochiasmal pathology. There was excellent anatomical-functional correlation between the visual fields and the OCT GCIPL as the inferior quadrants of the macula were most affected and this correlated to the superior visual field, which was affected. The OS also had more significant atrophy which correlated with its more dense visual field defect.

The differences between 24-2 and 30-2 Humphrey visual fields have been studied previously for neuro-ophthalmology patients. Khoury et al. [6] analyzed 187 Humphrey visual fields from neuro-ophthalmology patients with nonglaucomatous optic neuropathies and 206 Humphrey visual fields from patients with glaucoma. An occluder device was designed to cover the additional outer 22 points tested in the 30-2 strategy. Ninety-five percent of the fields in neuro-ophthalmology patients were read similarly with the 24-2 and 30-2 strategies. In the few cases where there was discrepancy, appropriate clinical management would not have been compromised by using the 24-2 strategy. The authors concluded that 24-2 testing strategy provides information comparable to that provided by the 30-2 strategy. The 24-2 strategy has the advantage of reduced testing time. In our patient, the duration of the Humphrey 24-2 test was 5 min and 34 s, whereas the duration for the 30-2 test was 7 min and 44 s. This has implications for busy clinics where visual field testing can be in short supply. The additional use of OCT, which captures an image in seconds, is an important adjunct as retrochiasmal pathology often manifests as homonymous hemi-macular atrophy. The 30-2 testing program can be reserved for cases such as ours where a homonymous defect is suspected, but only manifests in 1 eye.

The importance of OCT in localizing retrochiasmal visual field defects has been previously reported as the 24-2 visual field may be normal in both eyes. Lukewich et al. [7] described 7 patients with homonymous OCT GCIPL hemi-macular atrophy related to demyelination or traumatic brain injury. Other pathologies may also produce such a change. Momen et al. [8] reported a patient with an asymptomatic optic tract glioma from neurofibromatosis type 1 who was found to have homonymous hemi-macular thinning on OCT GCIPL. More recently, Zaslavsky et al. [9] reported a case of congenital porencephalic cyst resulting in an OCT GCIPL homonymous quadrantic defect without detectable visual field change. Traumatic brain injury has been reported to result in homonymous visual field defects, and it may be seen on OCT in the absence of any significant visual field defect [6]. Decramer et al. [10] described the use of diffuse tensor imaging to provide a morphometric analysis of the primary visual cortex and emphasized the importance of early imaging with this modality as the process of Wallerian degeneration could affect the results. Our patient did have subtle thinning of the left optic tract noted, and it was possible that there was primary traumatic damage to this area or trauma to the retrogeniculate pathway with secondary transsynaptic degeneration. Bow-tie atrophy on OCT RNFL is classically seen in contralateral optic tract lesions but can also be the result of contralateral retrogeniculate lesions after retrograde transsynaptic degeneration [11].

The highly incongruous nature of the visual field defect was likely a result of involvement of the left optic tract from the patient's remote traumatic brain injury. This is because fibers just posterior to the chiasm (crossed and uncrossed fibers) remain spatially distant, which is the basis for the "rule of congruity" [2]. These fibers then run in closer proximity in the occipital lobe, which is why lesions in this area are highly congruent. The incongruency of the OCT GCIPL lesion also supports this notion. It is also possible that this patient had a more congruent defect with asymmetric recovery. The OCT findings argue against 2 distinct lesions in each eye, and the OCT findings argue against individual optic neuropathies.

In summary, this is a rare case of a patient who had a monocular visual field defect on 24-2 Humphrey SITA-Fast visual fields, right bow-tie atrophy on OCT RNFL, and right homonymous hemi-macular atrophy on GCIPL with a normal MRI brain due to presumed remote traumatic brain injury. This case highlights that OCT is extremely helpful in localizing the lesion to the retrochiasmal visual pathways and that monocular defects on 24-2 SITA-FAST visual fields may be highly incongruous defects on 30-2 programs.

Statement of Ethics

This study was carried out in accordance with the World Medical Association Declaration of Helsinki, and the need for approval was waived as per University of Toronto Research Ethics Board guidelines. Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

C.W.Y. prepared the manuscript and gave final approval. J.A.M. conceived and designed the study, prepared the manuscript, and gave final approval.

References

1 Zhang X, Kedar S, Lynn MJ, Newman NJ, Biousse V. Homonymous hemianopias: clinical-anatomic correlations in 904 cases. Neurology. 2006;66(6):906–10.
2 Fraser JA, Newman NJ, Biousse V. Disorders of the optic tract, radiation, and occipital lobe. Handb Clin Neurol. 2011;102:205–221.
3 Tong KA, Ashwal S, Holshouser BA, Shutter LA, Herigault G, Haacke EM, et al. Hemorrhagic shearing lesions in children and adolescents with posttraumatic diffuse axonal injury: improved detection and initial results. Radiology. 2003 May 1;227(2):332–9.
4 Zaslavsky K, Margolin E. Unilateral temporal hemianopsia and nasal ganglion cell loss secondary to optic tract compression. Ophthalmology. 2020 Feb;127(2):176.
5 Lee SK, Wang Y, LBA, McClelland CM, Lee MS. Monocular hemianopia secondary to stroke. Am J Ophthalmol Case Reports. 2020 Sep 1;19:100758.
6 Khoury JM, Donahue SP, Lavin PJ, Tsai JC. Comparison of 24-2 and 30-2 perimetry in glaucomatous and nonglaucomatous optic neuropathies. J Neuroophthalmol. 1999 Jun 1;19(2):100–8.
7 Lukewich MK, Schlenker MB, Micieli JA. Homonymous hemi-macular atrophy of the ganglion cell-inner plexiform layer with preserved visual function. J Neurol Sci. 2020 Oct 15;417:117072.
8 Momen AI, Muir RT, Barnett C, Sundaram ANE. Homonymous retinal ganglion cell layer atrophy with asymptomatic optic tract glioma in neurofibromatosis type I. Front Neurol. 2020 Apr 15;11:256.
9 Zaslavsky K, Margolin E, Micieli J. Homonymous OCT-GCIPL quadrantanopia without a visual field defect. Ophthalmology. 2021;128:728.
10 Decramer T, Van Keer K, Stalmans P, Dupont P, Sunaert S, Theys T. Tracking posttraumatic hemianopia. J Neurol. 2018 Jan 1;265(1):41–5.
11 Pellegrini F, Interlandi E, Pichi F, Lee AG. Retrogeniculate lesion of the visual pathways: retinal optical coherence tomography angiography shows evidence of transsynaptic retrograde degeneration. Neuroophthalmology. 2020 Mar 3;44(2):114–7.