CASE REPORT

Hemifield Slide Phenomenon as a Result of Heteronymous Hemianopia

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ABSTRACT

Heteronymous hemianopic defects may lead to the hemifield slide phenomenon. We report one case of binasal and two of bitemporal visual field defects causing hemifield slide. Both patients with bitemporal visual field defects underwent strabismus surgery. One with adjustable sutures reported omission or duplication of a central strip of vision dependent on alignment during adjustment, but did not have long-term improvement of hemifield slide in two patients. Visual field improvement resolved hemifield in two patients, one of whom had strabismus surgery. Strabismus surgery to restore binocular visual function can be performed in hemifield slide patients. Patients with improving visual fields may have a better sensory prognosis.

Keywords: Binocular vision, diplopia, hemifield slide, strabismus surgery, visual field

INTRODUCTION

Hemifield slide describes the phenomenon of inability to stabilise and fuse visual hemifields in the setting of heteronymous visual field loss.^{1,2} This presumably results from loss of corresponding retinal points in the two eyes, allowing an underlying phoria to manifest as a tropia, or instability of ocular alignment.^{1,2} For example, a patient with an exophoria may report doubling of a central strip of vision, whereas an absence of a central strip of vision may occur with an esophoria.^{3,4} A vertical misalignment of the eyes leads to vertical dissociation of the hemifields.⁴

Hemifield slide phenomenon is most commonly due to bitemporal hemianopia, but has been reported with heteronymous altitudinal defects as well.² Beckmann and Kubie reported the theoretical possibility of binasal field defects leading to diplopia without frank ophthalmoparesis.⁵ We present three patients with hemifield slide phenomenon, including

one case of binasal hemianopia in whom symptoms improved with expansion of the visual fields.

SUBJECTS AND METHODS

This study was approved by the Institutional Review Board of the University of California, Los Angeles, and was determined to be exempt from review by the Institutional Review Board of Emory University. This study complied with all relevant privacy laws and the Health Insurance Portability and Accountability Act. The medical records of patients with diplopia in the setting of heteronymous hemianopia who were identified at the Emory Eye Clinic and Jules Stein Eye Institute were retrospectively reviewed.

For all patients, review of records included full ophthalmic examination, including medical history, visual acuity, ocular deviations at distance (20 feet) and near (14 inches), pupil examination, ocular

ductions, fundusoscopic examination, visual fields, and ophthalmic surgical reports.

REPORT OF CASES

Case 1

A 26-year-old obese man with idiopathic intracranial hypertension eventually had a lumboperitoneal (LP) shunt performed with residual optic atrophy and mildly constricted visual fields. After 15 years of stability, he developed shunt malfunction, manifesting with headaches, vomiting, and ultimately further bilateral visual loss and binocular horizontal diplopia. Neuro-ophthalmic examination showed visual acuity of finger counting at 6 inches OD and hand motion OS, and recurrence of papilloedema. His LP shunt was replaced, with resolution of his headaches and improvement of his vision, but he continued to complain of intermittent binocular horizontal diplopia.

On post-operative examination, visual acuity was 20/25 OD and 20/30 OS, and there was a left relative afferent pupillary defect. Extraocular motility was full in both eyes. He had a flick exotropia on alternate

cover testing for distance. Fundusoscopic examination showed bilateral optic atrophy. Goldmann visual fields showed a superotemporal island of vision OD and a temporal island of vision OS (Figure 1a).

On follow-up 3 months later, he reported resolution of his diplopia. His visual acuity and fundusoscopic examination were unchanged. He was orthophoric on cross-cover testing for distance and had a 6-prism-dioptre exophoria for near. Goldmann visual fields again showed binasal visual field defects, but with significant expansion of his temporal visual fields in both eyes to the vertical midline (Figure 1b).

Case 2

A 28-year-old woman with a history of amblyopia (likely anisometropic) in the left eye and bipolar disorder suffered a fall resulting in traumatic brain injury, subdural haematoma, epidural haemorrhage, intraventricular haemorrhage, left orbital fractures, multiple facial fractures, and a 22-day coma. Upon awakening she noted binocular diplopia and blurry vision. She was evaluated by an ophthalmologist while hospitalised and was diagnosed with bilateral third nerve palsies, bilateral trochlear nerve palsies,

FIGURE 1 Case 1. (a) Goldmann visual fields at initial postoperative evaluation. (b) Goldmann visual fields at follow-up examination showing significant expansion of the visual field in each eye.

and left optic neuropathy. Examination 5 months after injury revealed visual acuities of 20/30 OD and 20/150 OS, 12/15 Ishihara colour plates correct OD, and 3/15 plates correct OS, no ptosis, and poorly reactive pupils with a trace left relative afferent pupillary defect and light-near dissociation. Adduction was mildly reduced in both eyes, and she had otherwise full extraocular motility. She had a 50-prism-dioptre exotropia and a 20-prism-dioptre left hypertropia in primary position at distance, decreasing to a 40-prism-dioptre exotropia at near. She was able to fuse with 30 to 50 prism dioptres base-in OS; no vertical correction was needed to relieve her diplopia. There was trace optic nerve pallor OS. Confrontation visual fields showed bitemporal depression.

A left medial rectus muscle resection and a left lateral rectus muscle recession with a one-half-tendon width inferior transposition of both muscles were performed. She was evaluated for adjustment on the day of her surgery. She immediately noted that she no longer had diplopia, but complained of having a missing section of images in front of her. Adjustment

attempts either created a doubled central strip of vision, or caused a missing strip in images, dependent upon whether her eyes were exotropic or esotropic, respectively. For example, although slightly esotropic or orthotropic, the image of the surgeon as seen in profile was described as having her "ponytail attached to her ear," with a missing section of the surgeon's face (Figure 2). In slight exotropia, she noted duplication of a central vertical strip of vision. Suture adjustment was performed, leaving her slightly esotropic to orthotropic, although she continued to have a missing central vertical strip of vision.

By her 9-month follow-up visit, she no longer complained of a missing area of central visual field. She had a small exotropia by Krimsky light reflex.

Case 3

A 15-year-old boy with no past medical history was found to have a Rathke cleft cyst during the workup of delayed puberty and short stature. Immediately after resection of his Rathke cleft cyst,

FIGURE 2 Case 2. (a) A normal image as seen by a patient. (b) Image as seen by the patient in case 2 when esotropic during adjustment. (c) Image as seen by the patient in case 2 when exotropic during adjustment. (d) Image as seen by a patient with hemifield slide with a hypertropia.

he noticed constant binocular horizontal diplopia. Neuro-ophthalmic examination revealed visual acuities of 20/25 OD and 20/30 OS. Pupils were equal and there was no relative afferent pupillary defect. He was orthotropic with no detectable ocular motility abnormalities. There was bitemporal optic disc pallor. Goldmann visual fields showed a bitemporal hemianopia (Figure 3a). One month postoperatively, he developed a frank exotropia. Bagolini striated lenses demonstrated crossed diplopia. He was treated with botulinum toxin to the left lateral rectus muscle. He had residual exotropia, and a bilateral lateral rectus muscle recession was performed. Postoperatively, he developed a V-pattern exotropia and a right hypertropia, which was worse on left gaze and with right head tilt. Subsequent bilateral lateral rectus

re-recession with left inferior rectus muscle recession was performed. Postoperatively images were nearly superimposed, but he was unable to maintain fusion. He developed a recurrent exotropia and a left hypertropia. Prismatic correction was recommended, and he had occasional single vision with this treatment. At follow-up 6 months later (1.5 years after Rathke cleft cyst resection), he no longer had diplopia. Goldmann visual fields showed significant improvement from prior examination (Figure 3b).

DISCUSSION

Hemifield slide describes a horizontal or vertical binocular dissociation of remaining visual fields

FIGURE 3 Case 3. (a) Goldmann visual fields at initial postoperative evaluation. (b) Goldmann visual fields at follow-up examination showing significant expansion of the visual field in each eye.

secondary to loss of visual field overlap between the two eyes, which can lead to diplopia in the absence of a muscle paresis.¹ Beckmann and Kubie first discussed the phenomenon in 1929, although it was attributed to decreased macular visual acuity and attempted monocular eccentric fixation.⁵ In 1949, Bardram called the phenomenon "non-paretic diplopia" and attributed the development of diplopia in the setting of bitemporal visual field defects to decreased visual acuity with an associated slight divergence of the eyes.⁶ Stone described a vertical median strip of the retina, 0.2 mm wide, from which retinal ganglion cells projected to both optic tracts.⁷ This overlap of the receptive fields of ganglion cells in the median strip presumably allows the visual fields of both eyes to appear uniform without interruption in the midline.⁸ Patients with complete bitemporal hemianopia (whose visual fields represent only the temporal projection of each eye) lose the median strip of overlap, and therefore also the physiological linkage between the half fields. When the eyes are moving, the loss of overlap can result in difficulty maintaining the two half fields juxtaposed, perhaps because the eyes do not move at exactly the same rate.⁹ Nachtigäller and Hoyt described the sensory effects of ocular misalignment with complete bitemporal hemianopia in detail in 1970.¹⁰ An underlying exo-deviation can result in overlap of a central strip of vision; an eso-deviation in separation of hemifields; and a vertical deviation in vertical dissociation of the hemifields.^{3,4} Kirkham coined the term "hemifield slide" in 1972 to describe this phenomenon.¹

Hemifield slide phenomenon has been most commonly associated with bitemporal hemianopia, usually secondary to chiasmal compression by a pituitary adenoma.^{5,6,11,12} Elkington described the preoperative symptomatology of patients with pituitary adenoma, and was the first to attribute the symptom to his patients' inability to correctly register an image in each eye.¹³ In his series, 170 of 260 patients had bitemporal visual field loss and 84 of those (49%) complained of diplopia without having ocular palsy.¹³

Hemifield slide is also possible with other heteronymous hemianopias. Borchert *et al.* reported two patients in whom hemifield slide was attributed to heteronymous altitudinal visual field defects (those respecting the horizontal midline) from bilateral ischaemic optic neuropathy.² They concluded that the overlap of preserved visual fields was responsible for duplication of images.² Interestingly, Beckmann and Kubie recognised the possibility of binasal field defects without ophthalmoparesis leading to diplopia, although no cases were described.⁵

Strabismus surgery in patients with hemianopic defects, including two patients with bitemporal defects and hemifield slide, has recently been described.¹⁴ One patient suppressed one eye, and

therefore avoided symptoms from hemifield slide, and a second patient had significantly improved binocular visual fields following strabismus surgery, but still experienced hemifield slide due to a vertical deviation.¹⁴

The three cases presented here demonstrate two of the possible manifestations of hemifield slide. In cases 1 and 3, intermittent diplopia was the chief complaint, whereas in case 2, the patient reported a missing central strip in the binocular visual field. Two of our patients had a measurable ocular misalignment. In all three cases, due to lack of corresponding retinal points, fusional mechanisms were unable to maintain stable ocular alignment. Cases 2 and 3 underwent strabismus surgery. In case 2, her description of her field of vision before, during, and after her adjustment were classic for hemifield slide phenomenon. It is possible that ultimately, with an exotropic drift, she had realignment of her visual fields, leading to resolution of her symptoms from hemifield slide. However, it is more probable that she learned to suppress her amblyopic eye. Case 1 had relief of diplopia only after expansion of the visual fields to midline. Case 3 had relief of diplopia following botulinum toxin injection, strabismus surgery, prism spectacles, and expansion of the visual fields to midline. It is possible that expansion of the visual fields led to regained binocular retinal correspondence along the vertical midline. This is in accordance with Fisher and colleague's hypothesis of eye alignment instability, in which eyes without visual field overlap cannot easily overcome a strabismus.⁹

In conclusion, hemifield slide phenomenon can manifest as binocular diplopia, central strip "scotoma," or vertical misalignment when both eyes are open. Although a bitemporal visual field defect underlies most cases of hemifield slide, it can result from any heteronymous hemianopia that causes lack of retinal correspondence between the two eyes. Finally, symptoms secondary to hemifield slide phenomenon may be difficult to correct with strabismus treatment, and sensory abnormalities may persist despite apparent elimination of ocular misalignment.

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Note: Figures 2 and 3 of this article are available in colour online at www.informahealthcare.com/oph

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