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Fixation Switch Diplopia

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New-onset diplopia in an adult patient can be a highly concerning symptom that may trigger a neurologic or neuro-ophthalmic evaluation. Certainly, well-known causes of diplopia initially must be considered including ocular motor cranial nerve palsies, myasthenia gravis, and thyroid eye disease. However, when the diplopic patient has a history of childhood strabismus or amblyopia, the evaluation and management is different. Patients whose strabismic angle is unchanged or minimally changed, despite the new onset of diplopia, are particularly challenging. The differential diagnosis for new-onset diplopia in a patient with a history of childhood eye disease includes elimination of suppression in the amblyopic eye, a change in the angle of strabismus causing a drift outside the patient's suppression scotoma, unmasking of diplopia that was previously masked by a cataract or ptosis, central disruption of fusion, or fixation switch diplopia. The focus of this editorial will be on the topic of fixation switch diplopia.

Fixation switch diplopia occurs in patients with long-standing strabismus, who partially lose visual acuity in the fixating dominant eye, often as a result of cataract or refractive error (1). Because of their change in vision, such patients do not perceive diplopia, due to a suppression scotoma in the non-dominant eye that developed during childhood. However, when there is a change in vision in the dominant eye, fixation may switch to the nondominant eye, effectively removing the suppression scotoma, causing new-onset diplopia without a change in the strabismic angle. To help illustrate this phenomenon, consider the following case.

A 66-year-old man reported the onset of diplopia immediately after cataract surgery in his left eye. Medical history was unremarkable, but his ophthalmic history was significant for childhood strabismus and amblyopia. He recalled patching his right eye, as a child, but vision in his left eye was always somewhat "weaker." Preoperatively, his best-corrected visual acuity was 20/40, right eye and 20/60, left eye. His manifest refraction was +2.50 sphere in each eye. Slit-lamp examination revealed nuclear sclerotic cataracts, left greater than the right. He had a 6 prism diopter left esotropia that was comitant in all directions. He had never noticed diplopia. After uneventful left cataract surgery, the patient began to note binocular horizontal diplopia. His visual acuity postoperatively was 20/40, right eye, and 20/25, left eye. On motility examination, he had full ductions, and a comitant 6 prism diopter esotropia; however, his left eye was now fixating, and he had a manifest right esotropia. The differential diagnosis for this patient's new onset of diplopia after cataract

surgery included myotoxicity from a retrobulbar block, change in strabismic angle, preexisting diplopia that was masked by the diminished vision due to a cataract in his left eye, central disruption of fusion, anisophoria, and fixation switch diplopia. The most likely diagnosis was fixation switch diplopia because the patient did not have a change in his angle of strabismus, there was no restriction in his motility to indicate anesthetic myotoxicity, and his fixation was switched from his dominant right eye to the nondominant left eye due to an improvement in the visual acuity of the left eye. In addition, since he was able to fuse with the prism, central fusion disruption and optical aberrations were much less likely. A decompensation of a previous monofixation syndrome was considered, but unlikely given the small, unchanged esotropia (2). Following successful right cataract surgery (acuity: 20/20), the patient's fixation switched back to the right eye, and his diplopia was eliminated.

Other common scenarios in which fixation switch diplopia occurs include the following: 1) the development of myopia in a previously preferred eye in patients with mild contralateral amblyopia, 2) induced "monovision" either by optical or refractive surgical means, and 3) when patients with mild amblyopia receive an unbalanced refraction that leads to fixation with their nondominant eye (3). In the largest study of patients with fixation switch diplopia, Kushner (3) reviewed the records of 16 patients with a history of childhood strabismus and who were later diagnosed with fixation switch diplopia due to one of these 3 causes. Of the 16 patients, 4 developed a switch in fixation and subsequent diplopia due to the development of myopia in the previously preferred eye. As individuals grow into adulthood, it is normal for the eye to become longer and more myopic. If patients are anisometropic, it is possible for the eye that is more hyperopic during childhood (and therefore more likely to become amblyopic) to become the less myopic eye during adulthood. An example of this would be a child who develops amblyopia and a small angle strabismus secondary to anisometropia. This may occur if right eye refractive error is plano, while the left eye refractive error is +2.50 sphere. The child may develop amblyopia in the left eye with a small angle esotropia, and a suppression scotoma. However, over time, as both eyes shift toward myopia, the patient could develop a myopic refractive error in the right eye and a plano or mildly hyperopic refraction in the left eye. If this occurs, fixation may shift to the left eye and cause diplopia. Treatment would be to prescribe the appropriate refractive correction for the right eye allowing that eye to regain fixation. A second cause of fixation switch diplopia is the use of "monovision," which was the underlying etiology in 6 of Kushner's patients. The use of "monovision" as a treatment for presbyopia, in which one eye is corrected for distance and another eye is corrected for near, may cause fixation with the nondominant eye at distance or near, and result in diplopia. For this reason, the clinician should ask all patients if they have a history of childhood strabismus or amblyopia when considering presbyopia management with "monovision" either optically or by refractive surgery. A third common cause of fixation switch diplopia, occurring in 6 of the patients in Kushner's series, was dispensing an incorrect refraction. In these cases, incorrect spectacle correction resulted in fixation with the nondominant eye. Other published causes of fixation switch diplopia include a corneal ulcer and resultant scar in the dominant eye of a patient with monofixation syndrome and amblyopia (4), cataract surgery (5), and retinal detachment in the dominant eye of patients with strabismus and amblyopia (1).

Fixation switch diplopia is not commonly considered in the differential diagnosis of adult-onset diplopia. In the United Kingdom, the avoidance of fixation switch diplopia after cataract surgery was the subject of a review article by Hale et al (6). These authors determined that approximately 9,000 cataract extractions per year are performed on patients with amblyopia, all of whom were at risk for fixation switch diplopia. Hale et al recommended that cataract surgery initially be performed on the nonamblyopic eye in cases of bilateral visually significant cataracts. Similarly, before cataract surgery, Kushner and Kowal (7) recommended a patient screening procedure consisting of history of childhood eye disease or treatment such as patching or eye muscle surgery, checking spectacles for prism, cycloplegic refraction and cover testing for all patients undergoing refractive surgery, especially for those planning for “monovision.” A 4 base-out prism test can be performed to quickly identify a monofixation syndrome in patients with postoperative diplopia, as can blurring the fixating eye. Monofixation syndrome may go undiagnosed during the course of a patient’s lifetime due to the lack of a visible strabismus angle and the mild level of amblyopia. It is essential to ask about a family history of strabismus (8) and by using synoptophore testing to demonstrate peripheral fusion with a lack of bifoveal fusion.

Patients who present with fixation switch diplopia have an excellent prognosis. Typically, if fixation can be “switched” back to the dominant eye, by pursuing either refractive or surgical correction, patients will gradually experience less diplopia until fixation is maintained by the dominant eye. However, in cases where the fixation cannot be switched back due to permanent and untreatable vision loss, then optimization of the newly dominant eye’s vision with refractive correction, and the use of prisms or strabismus surgery should be considered. Rarely, fixation switch diplopia cannot be alleviated, and in these patients occlusion of 1 eye is required.

Given that 3%–5% of children in the United States have a diagnosis of strabismus or amblyopia (9,10), there are a large number of individuals at risk for fixation switch diplopia. As our general population ages, fixation switch diplopia may become a more common diagnosis. Therefore, it should be considered in any adult with new onset of diplopia and a history of strabismus, especially if there has been no change in the magnitude or type of strabismus detected on clinical examination.

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