

LETTER TO THE EDITOR

High myopia following excessive occlusion therapy in the first year of life

EDITOR,—One of the major obstacles in the management of monocular aphakia in an infant is the occlusion therapy. Usually the problem is poor compliance; however, excessive patching of the phakic eye can lead to reverse amblyopia. In our case patching may also have contributed to the development of high myopia in the occluded eye.

CASE REPORT

The male child born at full term with a birth weight of 7 lb 13 oz (3.54 kg), was noted to have leukocoria immediately after birth caused by a dense nuclear cataract in the left eye. Family history was negative for congenital cataracts or high myopia. Corneal diameters were right eye 10.0 mm and left eye 9.5 mm. Cycloplegic retinoscopy was right eye +1.00+1.00×90 and left eye poor reflex.

Funduscopy examination showed the absence of retinal pathology. Axial lengths, measured by A scan ultrasonography with a contact probe, were right eye 17.4 mm and left eye 16.5 mm (average axial length for the age group is 17.0 mm). A lensectomy with anterior vitrectomy was performed at 15 days of age. The eye was fitted with a silicone contact lens, and the right eye was occluded 75% of the waking hours, with periodic examinations. An esotropia was noted shortly after the cataract surgery and strabismus surgery was performed at 1 year of age.

When we saw the patient at the age of 14 months he was still being patched 75% of his waking hours with excellent compliance. His psychomotor development was proceeding normally and he had no dysmorphic features. He had no fixation with the right eye but was able to fix and follow well with his left eye. Teller acuity card testing at 38 cm showed right eye no response to even the broadest gratings and left eye 6.5 cycles/cm, which is normal for the age group. Slit-lamp examination disclosed no corneal or lenticular abnormalities in the right eye. He was orthotropic by the Hirschberg test. A

cycloplegic retinoscopy revealed right eye $-11.50+0.50\times 90$ and left eye $+13.50$ spheres. Fundus examination showed a prominent choroidal pattern (as commonly seen in myopic eyes) in the right eye but not in the left eye, otherwise the retina was normal in both eyes. The intraocular pressure, measured under sedation with chloral hydrate, was right eye 14 mm Hg and left eye 12 mm Hg. Axial lengths, measured by A scan ultrasonography with a contact probe, were right eye 23.5 mm and left eye 22.0 mm (average for the age group is 22.0). Once the right eye was fitted with a soft contact lens, Teller acuity card testing was repeated, with no change from the initial examination. The left eye was patched 4 hours a day with careful monitoring of the occlusion therapy. Five months later Teller acuity card testing at 38 cm showed right eye 6.5 cycles/cm and left eye 6.5 cycles/cm, normal for the age group.

COMMENT

This patient developed high myopia and reverse amblyopia, possibly both deprivation and anisometropic, in the phakic eye following excessive occlusion therapy in the management of monocular aphakia. Excessive occlusion in the first year of life can account for the development of myopia because of its interference with patterned visual stimulation during the most active stage of growth of the eye. It appears that there is a biofeedback mechanism which relies on normal visual stimulation for normal growth of the eye and emmetropisation.¹ This has been demonstrated in animal models, which develop high axial myopia following suturing of the eyelids or corneal opacification in the first year of life.^{2,3} The relation between eyelid closure and media opacities in the first year of life and the development of myopia is well documented in humans.^{1,4,5} The degree of myopia appears to correlate with the age of onset and the duration of the visual deprivation.⁴ However, the effect of visual deprivation on axial elongation has been reported to be less predictable in humans than in

animals.^{4,6} Though our patient demonstrated axial elongation, it is not sufficient to account for the magnitude of the myopia observed. It is possible that the myopic shift has a corneal/lenticular component but it cannot be corroborated because of the absence of keratometric readings. Additionally, the echographic measurements with a contact probe, rather than by immersion, do not allow us to ascertain the lens dimensions. Other conceivable causes of acquired high myopia in infancy such as glaucoma, lens dislocation, and nuclear cataract were excluded by clinical examination.

To our knowledge the development of high myopia in the phakic eye is a previously unreported possible complication of excessive occlusion therapy in the management of monocular aphakia during the first year of life. It stresses the need for careful monitoring of visual acuity and periodic cycloplegic refractions while patching infants to treat amblyopia.

MICHELLE MUÑOZ
HILDA CAPO
*Department of Ophthalmology,
Bascom Palmer Eye Institute,
Miami, Florida, USA*

Correspondence to: Michelle Muñoz, MD,
PO Box 016880, Miami, FL 33101, USA.

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