Sir,

Cataract presenting as manifest refractive astigmatism: case study

Refractive errors have been well described in association with cataract, most commonly in the form of myopic progression with nuclear sclerosis.¹ We present the following cases of patients with cataracts presenting as manifest refractive astigmatism secondary to lenticular astigmatism, in turn due to posterior subcapsular and nuclear sclerotic cataracts.

Case 1

A 46-year-old man presented to his optician complaining of deterioration in binocular vision quality over the previous 10 months, worse in the right eye. He was referred to our department with a provisional diagnosis of cataracts. He was myopic and had had retinal detachment surgery in the right eye 36 years earlier.

On examination, best-corrected visual acuities were 6/12 (right) and 6/6 (left) with the following refraction: right eye $-11.00/+7.00 \times 100$; left eye -6.50 DS. A dense posterior subcapsular cataract was noted in the right eye and cortical lens opacities were noted in the left eye. Dilated funduscopy showed evidence of previous retinal surgery in the right eye consistent with treatment for a retinal dialysis.

Reproducible corneal topography (Alcon, Eyemap) showed only 1.56 DC at 100° of corneal astigmatism in the right eye (Figure 1a), suggesting the presence of significant lenticular astigmatism. Data from previous refractions showed that as lenticular opacities developed over the period 1990 to November 2003 the manifest refractive astigmatism in the patient's right eye increased from $+2.50$ to $+7.00$ DC (Table 1).

Corneal topography for the left eye indicated 0.85 D of with-the-rule astigmatism, similar to that of the right cornea.

The patient was prepared for bilateral phacoemulsification with primary implantation of intraocular lens (SI40 Clariflex, Advanced Medical Optics Inc.), and contact ultrasound biometry was performed (OcuScan, Alcon). An A-constant of 118.00 was used with the SRK-T formula to calculate the required IOL powers. The lens powers selected for implantations were 13.50 D (right eye) and 11.50 D (left eye), predicting refractive errors of $+0.13$ DS (spherical equivalent) and -0.12 DS for right and left eyes, respectively.

The corneal astigmatism in the right eye was also corrected at the time of surgery with arcuate keratotomy into clear cornea just inside the limbus.² Data from the corneal topography plots were used with the nomogram 'Kill-the-Cyl 2003' software (PB Chell, personal data) to plan the location and length of correcting incisions. The desired 100° meridian was located with a Mendez degree gauge (Duckworth & Kent) and the cornea was marked with ink (skin marker pen, Universal Hospital Supplies) on a Thornton lowest profile eight-blade radial marker (Duckworth & Kent). A Langerman Hinge Flap incision³ was made on the steep meridian of the cornea. The arcuate keratotomy incision was made with a guarded blade set at a depth of $550 \mu\text{m}$ (Micro-Feather, Wilson Ophthalmics) and extended to a length of 4 mm. The desired wound gape was observed and a self-sealing three-plane phacoemulsification wound was completed within the anti-astigmatic incision, using a 2.85 mm

keratome (LaserEdge, Bausch & Lomb).

Phacoemulsification was then performed routinely with two paracenteses for bimanual aspiration. The IOL was placed into the posterior chamber and the wounds were sealed by stromal hydration. The surgery was uncomplicated, and the post-operative corneal topography is shown in Figure 1b. Phacoemulsification and intraocular lens implant were also performed on the left eye 4 weeks later following the same practice. No anti-astigmatic procedure was used and the surgery was also uncomplicated.

The refraction and visual acuities 2 months after surgery are shown in Table 1.

Case 2

A 69-year-old lady was referred by her optician after he found a dramatic change in her astigmatism previously which he could not correct with glasses. The optician noting a rise in astigmatism subsequently referred the patient with a provisional diagnosis of keratoconus (Table 2).

On examination, visual acuity fully corrected was 6/12 right and left. Significant findings were limited to her bilateral nuclear sclerotic cataract.

Corneal topography (Figure 2) (Alcon, Eyemap) produced results markedly different from those found on refraction, namely an astigmatism of 1.14 D at 090° left eye. The difference was attributable to the cataract that had formed over the years. The patient underwent uncomplicated phacoemulsification using the same SRK/T formula a 21.5 D was inserted into the left with a post-operative predictive refraction of -0.66 DS. The surgery was performed by KB according to the same technique as described for case 1. For this patient, however, no arcuate keratotomy was performed. Post-operative refraction demonstrated a much reduced astigmatism (Table 2).

Comment

Development of cataract is recognised to be associated with progression of myopia, especially for nuclear sclerotic cataracts.¹ This case illustrates the importance of breaking down astigmatism into its main vector components of lenticular and corneal astigmatism, particularly when manifest refractive astigmatism increases in a patient with cataract. In case 1, the cataract was mainly posterior subcapsular although there was some cortical opacity present in both eyes.

The arcuate keratotomy was calculated to correct the corneal astigmatism of 1.50 D, implying that surgical removal of the lens had eliminated a further $+7.10$ D at 37° in the right eye calculated using the mathematics of obliquely crossed cylinders.⁴ This is an example of patients presenting with the majority of the pre-operative astigmatism being lenticular in origin and developing as the lenticular opacities progressed. Such a large amount of lenticular astigmatism due to cataract is not infrequently seen, even in our experience, in the absence of vast amounts of nuclear sclerosis.

In the second case, the development of nuclear sclerotic cataract induced the astigmatism. As no arcuate keratotomy was performed, it can be seen that removing the offending cataract significantly reduced the patient's astigmatism. Again using similar vector analysis, it can

Figure 1 (a) Pre-operative corneal topography, right eye, showing $K_{\text{steep}} = 45.97$ D, $K_{\text{flat}} = 44.41$ D, and $KD = 1.56$ D. (b) Corneal topography, right eye, at 6 weeks after surgery showing $K_{\text{steep}} = 45.89$ D, $K_{\text{flat}} = 44.57$ D, and $KD = 1.32$ D.

Table 1 Changes in manifest refraction for patient 1 before and after cataract surgery

	Right eye	Left eye
<i>Pre-operative</i>		
1/1990	-5.00/+2.00 × 100	-7.00/+0.50 × 90
4/2002	-5.50 DS	
2/2003	-9.00/+6.00 × 125	
11/2003	-11.00/+7.00 × 125	-6.50 DS
<i>Post-operative</i>		
2/2004	-0.50/+0.50 × 75 VA 6/12	-1.50/+0.50 × 45 VA 6/4

Table 2 Changes in manifest refraction for patient 2 before and after cataract surgery

	Left eye
<i>Pre-operative</i>	
6/1999	+0.50/-1.00 × 20
11/2002	+0.75/-5.50 × 13
<i>Post-operative</i>	-0.25/-0.75 × 145 VA 6/6

be seen that a change of +5.42 D at 9° change is seen in the post-operative refraction in the left eye.

Ludwig *et al*⁵ described the development of lenticular astigmatism in a 6-year-old patient following repair of an open globe injury. This was attributed to anterior vitreous fibres incarcerated in the inner scleral wound, which contracted and caused distortion of the posterior lens capsule. Release of the tension on these fibres via anterior vitrectomy resulted in rapid reduction of the

Figure 2 Pre-operative corneal topography, left eye, $K_{\text{steep}} = 42.60$ D, $K_{\text{flat}} = 41.46$ D, and $KD = 1.14$ D.

patient's astigmatism, implying that distortion of the posterior lens and capsule can cause significant lenticular astigmatism. Hence, a posterior subcapsular lens opacity, as we have demonstrated, can also cause significant lenticular astigmatism and hence can increase manifest astigmatism.

It is important to consider the origin of the astigmatism when planning cataract surgery in order to optimise the visual outcome. The greater component of astigmatism usually resides in the cornea,^{6,7} and nomograms are readily available^{8,9} for correction of corneal astigmatism via arcuate incisions that may be placed on meridian (steep) and be coincident or non-coincident with the meridian of the primary incision or side ports. However, it is essential that the astigmatism is fully evaluated with corneal topography, as a large difference between the cylindrical component of the manifest refraction and corneal astigmatism demonstrated on topography implies the presence of significant lenticular astigmatism. The magnitude of the lenticular astigmatism may be derived by vector analysis¹⁰ from the total minus the corneal astigmatic components. Ninomiya *et al*¹¹ describe an elegant method for indirect evaluation of lenticular astigmatism using wavefront analysis, using traditional methods SIRC Koch and Holladay.

Patients may also present for cataract surgery with pre-existing conditions that place them at high risk for lenticular astigmatism. Tilted disc syndrome (TDS) is present in 1–3% of the population¹² and is readily diagnosed at a pre-operative examination. Gündüz *et al* found lenticular astigmatism to be significantly more common and of greater magnitude in patients with TDS than in controls matched by age and refractive error and suggested that tilting of the crystalline lens was responsible for induced astigmatism.

Whether the lenticular astigmatism is due to the morphology or position of the crystalline lens, it will be eliminated by cataract surgery. This must therefore always be considered when pre-operative assessment indicates the presence of astigmatism. The corneal and lenticular components must be clearly identified. This will also present a common cause of refractive surprise 'unmasking of corneal astigmatism' whereby pre-existing corneal astigmatism that is neutralised by opposite lenticular astigmatism suddenly emerges as a refractive astigmatic surprise. This will allow a bespoke surgical plan to be formulated in order to address it in an optimal manner and avoid leaving or even creating significant residual astigmatic error.

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Eye (2007) **21**, 1239–1241; doi:10.1038/sj.eye.6702752;
published online 20 July 2007

Sir, Intraoperative floppy-iris syndrome associated with chronic use of chlorpromazine

Intraoperative floppy-iris syndrome (IFIS) during phacoemulsification has been described in patients who are using α_1 -adrenoceptor (AR)-blocking agents such as tamsulosin, terazosin, alfuzosin, and doxazosin.¹ Recently, labetalol and mianserin which are antihypertensive and antidepressant agents, respectively have also been reported to be associated with this syndrome.^{2,3} We report a new case in which IFIS developed in a patient owing to chronic use of chlorpromazine.

A 48-year-old man with schizophrenia had bilateral nuclear cataract. Despite standard pharmacologic dilation (topical cyclopentolate 1%, tropicamide 1%, phenylephrine 10%) for fundus examination, the patient's pupils did not dilate well. He had no posterior synechia, pseudoexfoliation, history of miotic use, or diabetes mellitus. His medical history revealed that he has been using chlorpromazine 50–200 mg (average 100 mg) for 29 years for schizophrenia.

Right cataract operation was planned under general anaesthesia. A relatively small capsulorhexis was performed. During the phacoemulsification procedure, characteristics of IFIS developed: there was a flaccid iris stroma, which undulated, billowed, and prolapsed to the main and side incisions, and progressive miosis