

LETTERS TO THE EDITOR

Axial length measurement discrepancies in asteroid hyalosis

SIR,—Axial length (AL) measurement is the most important factor affecting intraocular lens (IOL) power calculations. A difference of 1 mm in axial length will affect the postoperative refraction by approximately 2.5 dioptres. The ophthalmologist should therefore be aware of factors which will yield spurious measurements. These factors include posterior pole colobomas, posterior vitreous detachments, macular oedema, and others.¹ Asteroid hyalosis (AH) is generally not expected to affect AL measurements. A review of the literature provided only one case report of falsely decreased AL measurement secondary to AH necessitating a second IOL implantation.²

Asteroid hyalosis is an uncommon condition occurring most frequently in elderly individuals in the 7th and 8th decades. It appears as a myriad of tiny spherules containing calcium and phospholipids suspended throughout the vitreous. It is usually unilateral, has no sex predilection, and has not been definitively associated with systemic or ocular disease.³

We present a case in which asteroid hyalosis in a cataractous eye falsely lowered the axial length measurement, yielding an incorrect IOL power calculation. Implantation of this lens would have resulted in a significant refractive error. We studied the axial length measurements in five additional cases with asteroid hyalosis to compare measurements with predicted estimates.

A 52-year-old black diabetic female had been followed up by her retinal specialist for macular oedema OS. Examination of the left eye was remarkable for a visual acuity of 20/400 with a -1.00 sphere, a moderate nuclear sclerotic cataract, severe macular oedema, and asteroid hyalosis. The right eye had a posterior chamber IOL and was emmetropic, with a visual acuity of 20/25. Asteroid hyalosis was present OD, but ophthalmoscopic examination gave otherwise normal results.

An A scan was performed with the Cooper ABX 1000 and yielded AL measurements of 22.00 mm OD and 22.10 mm OS. The measurements were reproducible and resulted in an IOL power calculation of +24 D (SRK II regression formula) for both eyes. Since the average AL is 23.5 mm, these readings would be consistent with a hyperopia of 4 dioptres. However, the patient's elicited refractive history, current visual acuity, and results on ophthalmic examination were not consistent with such a degree of hyperopia. In addition, the pseudophakic eye had received a +20.50 dioptres IOL (not 24.0 D) and had achieved emmetropia. We concluded that the AL measurement in both eyes was spuriously decreased secondarily to AH. Based on our clinical impression a +21.50 IOL was implanted OS instead of the calculated +24.0, with a resultant postoperative refraction of plano.

Our case report describes artefactual lowering of the AL measurement in a patient with AH. Except for the above mentioned report¹

AH has not previously been known to affect AL measurements. In our case macular oedema may have been a contributing factor in spuriously decreasing AL in the left eye. The right eye, however, had asteroid hyalosis without macular oedema but still had artefactual lowering of the AL measurement.

Owing to these findings we examined five patients who had a history of unicolor AH without any other ocular abnormality. Retinoscopic readings, keratometry, current visual acuity, and refractive history were all studied. An A scan was performed with the Cooper ABX 1000. Four of the five patients had predictable AL measurements consistent with their history and findings on examination. One patient, however, had an AL which measured 2.5 mm shorter than predicted in the eye with AH. A difference of this magnitude from the expected AL is extremely unusual.

Although no formal study has been undertaken on this issue, we believe strongly that asteroid hyalosis can cause artefactual lowering of axial length measurements. Careful consideration of the refractive history and clinical examination may avert implantation of an erroneous lens.

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Diabetes and retinal function

SIR,—We were interested in the recent article by Bek and Lund-Andersen¹ and in your accompanying editorial.² It is true that much has been published in recent years concerning the vascular aspects of diabetic retinopathy. The earliest changes in diabetic retinopathy need not, however, be vascular. Thus, Bek and Lund-Andersen could not demonstrate areas of visual field loss corresponding to retinal hard exudates or fluorescein leakage, but this may be because their test of retinal function was not sufficiently sensitive. A similar study using a more sensitive test of retinal function such as S cone pathway sensitivity³ or luminance or chromatic threshold measurement⁴ might increase the likelihood of detecting differences in function between an area of apparently normal retina and an adjacent area where a breakdown in the blood-retinal barrier is obvious.

Although a breakdown in the blood-retinal barrier may precede neurosensory dysfunction as suggested by Bek and Lund-Andersen, recent work in our laboratory suggests the opposite may be true. We compared 36 patients with insulin-dependent diabetes with 36 age-matched controls. Funduscopy, fundal photography, and fluorescein angiography confirmed that none of the diabetics had breakdown of the blood-retinal barrier, but colour vision assessed by the Farnsworth-Munsell 100-hue test was markedly abnormal in the diabetic group compared with normal controls (mean FM 100-hue error score for diabetics

85.2 (SEM 7.6) v 29.5 (SEM 3.3) for controls, $p < 0.001$).

We believe the question of whether vascular or neurosensory dysfunction occurs first in diabetic retinopathy remains open.

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Reply

SIR,—The question of which measure of retinal function is the best of course depends on what one wishes to study. What I think is important, however, is that perimetric light sensitivity in practice appears to reflect clinically significant visual loss. Therefore the relation between perimetric results and extrafoveal morphology may help us to learn which morphological lesions in the foveolar area may lead to lowered visual acuity. In this context the most important finding described in the paper might perhaps be the lack of correlation between fluorescein leakage and loss of retinal light sensitivity. The reason why some hard exudates do not produce visual field scotomata is probably that this lesion causes considerable light scattering, a phenomenon that can be directly observed with the scanning laser ophthalmoscope.

I agree that some subclinical measure of retinal neurosensory impairment may perhaps be the initial sign of retinopathy. However, I don't think that the reduced blue-sensitivity of diabetic patients that has been known since the 1960s¹ is necessarily an argument in favour of this hypothesis. It has been shown that the lens browning (nuclear sclerosis) of diabetic lenses is higher than in normals, increasing with age² and with poor metabolic control.³ This lens browning causes increased lens autofluorescence, absorption, and light scattering, and thereby less light transmission, especially in the blue-green area, an effect that can account for the decreased blue sensitivity of these patients (Larsen *et al*, in preparation: (personal communication). Therefore I think that an evaluation of colour vision anomalies of diabetic patients at least requires a proper correction for the individual wavelength-dependent light loss in the refractive media (notably the lens) due to autofluorescence, absorption, and scatter.

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