aphakic glaucoma is usually bilateral also supports a genetic theory. Many genes are known to be involved in both cataract and glaucoma, PAX6 being perhaps a paradigm. The complex interaction of gene mutations and polymorphisms continues to be unravelled.

Many of the answers to these questions will lie in research. Some of this work will occur at the basic science bench and the ocular histopathology laboratory. Some will look like that done by Swamy and colleagues ***(see page 1627)***.[3] There is value to retrospective case series. For example, their 20-year study adds to our recognition that younger age and smaller eyes are at greater risk for the development of glaucoma following cataract surgery. Studies like this will be strongest if the definitions are rigorous. For example. Swamy and co-workers did not measure corneal diameter and did not measure central corneal thickness, the latter being elevated in many cases of aphakia.[4]

Ultimately, prospective comparative studies would yield the most power, but they are very hard to do and quite expensive. The challenges are many. Not all aphakic glaucoma is the same; there are early closed angle forms, those induced by postoperative inflammation or steroid use, and the more classic ''open angle'' late-onset variety. The late average age of onset requires that the studies be conducted over years, if not decades. The variable onset of cataract, the different morphologies, and the variation of surgical technique (which also evolve over time) necessitate large sample sizes, long-term funding, and above all investigator persistence and patience.

So what is a paediatric ophthalmologist/cataract surgeon to do? First and foremost, screening must be part of the long term follow-up care of aphakic children. Their risk for glaucoma appears to be lifelong. I recommend that intraocular pressure be measured at least annually even if sedation or anaesthesia is required. Such procedures also allow for careful measurements, corneal pachymetry and photodocumentation of the optic nerve. Portable techniques will eventually be available to include nerve-fibre analysis on the supine patient. Normal controls will be essential. If a child is not compliant with awake tonometry, virtually every outpatient visit should include proxy measurements for glaucoma such as refraction (or over refraction) to look for increasing axial length, slit-lamp examination looking for corneal oedema and, if possible, a view of the optic nerve. These can often be accomplished outside the academic centres with a bit of patience and without pharmacological dilation of the pupil. I recommend outpatient visits no less than every 6 months.

Second, we must continue research not only into the pathophysiological basis of aphakic glaucoma but also into the most effective ways to screen, measure, and treat the disease. Although we should continue retrospective studies, prospective randomised trials with rigorous inclusion criteria and detailed data collection offer the best hope but at the highest cost. Multicentre collaborative approaches in partnership with academic facilities where these children tend to congregate and countries such as India and Saudi Arabia, where the incidence may be higher due to the higher incidence of cataract, will be essential. It may be many years before this story has an ending, but the wait will well be worth it all.

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Photoreceptor layer integrity

Can the integrity of the photoreceptor layer explain visual acuity in branch retinal vein occlusion?

Naoichi Horio

A possible new preoperative indicator for postoperative visual acuity via optical coherence tomography

In this issue of the *British Journal of Ophthalmology*, Ota and his associates (***see page 1644***)[1] report that the presence of the third high reflectance band (HRB) in images obtained by optical coherence tomography (OCT) postoperatively is correlated with the visual outcome after different treatments for macular oedema secondary to branch retinal vein occlusion (BRVO). They also investigated whether the appearance of the preoperative OCT images could predict the final visual acuity. Their results indicated that the presence of the third HRB in the parafoveal area preoperatively could indeed be a predictor of the postoperative visual acuity.

Grid laser photocoagulation has been the standard treatment for macular oedema secondary to BRVO.[2] However, over the past decade, more interventional therapeutic options have emerged. Vitrectomy with or without arteriovenous sheathotomy,[3–5] intravitreal triamcinolone injection[6] and intravitreal injection of tissue plasminogen activator[7] have been used to treat macular oedema. The efficacies of these interventions, however, are controversial because of the lack of a randomised controlled studies.[8] In a previous study, the authors reported the efficacy of intravitreal tissue plasminogen activator injection,[7] and also demonstrated a correlation between the presence of the third HRB and visual outcome.[9] The current report includes 46 eyes treated by the other interventions, with 19 of the 46 eyes treated by two or more interventions. Even after the resolution of macular oedema, some of the eyes did not achieve a significant improvement in visual acuity. Therefore, their retrospective study focused on eyes in which macular oedema was resolved, and the foveal thickness in the 46 eyes studied was <250 μm after the treatments. The significant correlation between the absence of the third HRB and poor postoperative visual acuity identified one of the factors accounting for the poor visual outcomes after successful anatomical results.

The recently developed high resolution OCT allows not only accurate quantitative

measurements of macular thickness but also qualitative analysis of the different retinal layers. The third HRB is reported to represent the junction of the inner and outer segments of the photoreceptor cells,[10 11] and thus the absence of the third HRB was suggested to indicate a degeneration or disorganisation of the photoreceptor cells. To explain the mechanisms leading to the absence of the third HRB, Ota *et al* argue that severe ischaemia or swelling of the foveal photoreceptor layer could lead to significant disorganisation of the photoreceptors. Although there were no angiographic data to confirm this in the current study, fluorescein angiography can show macular ischaemia and vascular leakage, which are widely accepted as signs of the severity of macular oedema. Severe ischaemia often reduces the macular oedema without improving the visual function. By contrast, it has been reported that eyes with incomplete macular perfusion, the ischaemic type, had a higher incidence of an improvement in visual acuity than eyes with macular perfusion, the nonischaemic type.[12] Therefore, fluorescein angiography could have provided additional information to explain the mechanism determining the visual outcome.

Investigations that attempt to determine whether the presence of the third HRB is a prognostic factor of the postoperative visual acuity are of great value. Thus, it would be of interest to use OCT images of the parafoveal area because retinal haemorrhages and severe thickening of the retina often prevent the acquisition of a clear image of the outer retina at the fovea before treatment. Indeed, the third HRB at the fovea could be detected in only 4 of the 29 eyes in the study by Ota *et al*, and the findings in these eyes were not correlated with the postoperative visual acuity. One of the reasons for this might be that the other 25 eyes included eyes with an intact photoreceptor layer but without the third HRB in their images due to retinal haemorrhage or severe swelling of the inner retinal layers. By contrast, the presence of the third HRB in the parafoveal (eg, at 500–1000 μm) unaffected area superior to the fovea, was well correlated with the postoperative visual acuity. The findings in this area might reflect the severity of the macular oedema because the association between the absence of the third HRB at the unaffected area and the distance from the fovea indicated the extent of macular oedema or retinal haemorrhage. It is reasonable to assume that extensive macular oedema or retinal haemorrhage would be associated with poor visual improvement.

The preoperative visual acuity is a very important prognostic factor in eyes with clear media.[5 13 14] It is well known that good preoperative visual acuity results in a better improvement of visual acuity not only in the natural course of the disease process but also after treatment. In the 46 eyes in the Ota *et al* study, there was no difference in the initial visual acuity between the groups with or without the third HRB after treatment. For their second investigation of prognostic factors, 29 of 46 eyes were examined using OCT3, which resulted in a different grouping. These eyes were divided into subgroups determined by the absence or presence of the third HRB. Therefore, the initial visual acuity in each group, with or without the third HRB, should be shown at each point from the fovea. The authors, however, did not mention the association between the preoperative visual acuity and the preoperative third HRB at 500 μm and 1000 μm from the fovea where the absence of the third HRB is well correlated with poor visual outcome. These findings would be more valuable as prognostic factors if there had been no difference in the initial visual acuity between the groups with or without the third HRB around the fovea.

The presence or absence of the third HRB around the fovea determined from the OCT image is supposed to be a predictor of the visual prognosis in eyes with macular oedema secondary to BRVO. The inclusion criterion of this study was eyes with absorbed macular oedema, and the retrospective style of the study could limit the conclusions. Because the unresolved macular oedema can also lead to poor prognosis, it is necessary to evaluate if the third HRB around the fovea can also predict refractory macular oedema. Prospective studies are needed to determine whether the presence of the third HRB around the fovea can be widely accepted as a prognostic factor after treatment for macular oedema secondary to BRVO.

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