development of this disease. Although the disease is associated with the HLA-B51 locus, not all patients have this genotype.[18] Furthermore, considerable individual variation in disease course and severity is commonly observed. The study by Kitaichi *et al* contributes to our understanding of this complex disease, and we predict it will form the basis of further large-scale international collaborations in the future, including ones involving the development of new therapies.

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Aphakic glaucoma

Aphakic glaucoma: a never-ending story?

A V Levin

Regular screening and continuing research are important in the fight against aphakic glaucoma

Aphakic glaucoma has plagued those who provide ophthalmic care to children ever since paediatric cataract surgery became a reality. It is the second most common cause of paediatric glaucoma,[1] and all children who have cataract surgery remain at risk for life. Its pathophysiology is largely not understood, and there appears to be little if any end in sight on the near horizon. There are many theories as to its causation.

BAROTRAUMA TO THE IMMATURE ANGLE

In support of this theory is the higher frequency of aphakic glaucoma in children who have their surgery at younger ages. Arguing against this theory is the seemingly equal rate of aphakic glaucoma following pars plana lensectomy.

STRUCTURAL CHANGES TO THE ANGLE DRAINAGE COMPLEX

Certainly this makes sense in the ''soft'' paediatric eye and may be supported by a possible lower incidence of glaucoma in pseudophakic eyes, although the latter factor requires many more years of study, and the incidence of glaucoma in pseudophakia seems to rise as the years pass: a rise far above the initial cautious expectations.[2]

GONIODYSGENESIS

Some eyes with aphakic glaucoma do have an angle appearance on gonioscopy very reminiscent of the angle in congenital/infantile glaucoma. Such eyes may even respond to goniotomy. This suggests that there is more that is wrong with the eye than the cataract. If this is true, then perhaps we should see more patients with isolated cataract plus glaucoma before surgery. Such patients may exist but have their ''natural'' glaucoma ''prevented'' by cataract surgery, which is needed promptly to obtain a better vision outcome.

ROLE OF THE VITREOUS

For those who frequently do cataract surgery with primary posterior capsulotomy, especially in younger eyes left aphakic, there is a familiarity with the high frequency of vitreous entry into the anterior chamber. Perhaps the presence of vitreous, even when temporary, can cause a more permanent alteration of aqueous drainage. Gonioscopy techniques do not allow us to assess this possibility well. Again, time will be the judge as we follow the course of pseudophakic patients who had their capsulotomy done after IOL implantation via the pars plana. But these patients are on average older and therefore less at risk for glaucoma.

CHEMICAL FACTORS

Even in adults, when the posterior capsule ruptures during cataract surgery even without vitreous entering the anterior chamber, there is an increase in later glaucoma. Is it the mere exposure of the posterior chamber to the anterior chamber that causes some chemical factor to ''poison'' the angle? Although vitreous excitatory amino acids and other factors have been speculated to have a role in glaucoma, there is much work to be done in this area, and the consistency of the paediatric vitreous is very different from that of adults.

GENETIC FACTORS

Last, and certainly not least, is the unknown role of genetics in determining which child will or will not get aphakic or pseudophakic glaucoma. The aphakic glaucoma does not always seem to segregate in families with heritable cataract, but sometimes it does. The fact that

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