

Interestingly Kim and associates presented that the rate of VF progression was higher in the follow-up period after cataract surgery than in the period before the surgery. Furthermore, eyes with worse baseline VF defect and those without prior trabeculectomy were concluded to be more vulnerable to intraoperative and early postoperative stress caused by cataract surgery. The postoperative peak of IOP was associated with postoperative rates of VF decay.

In this study, the baseline IOP was relatively low both in mild and moderate-to-severe primary open-angle glaucoma (POAG) patients. It should be noted that intraoperative and early postoperative IOP spikes may outweigh statistically significant but clinically marginal long-term IOP reduction in terms of glaucoma progression.² In light of the findings, proper phaco settings and the type of ocular viscosurgical device (OVD) and its careful removal at the end of the surgery, as well as efficient postoperative anti-glaucoma medication, are of special importance. These findings also raise the question whether prostaglandin analogs are advisable to pause before and after cataract surgery in glaucoma patients (especially in ones with VF defects), or not.³

Given the long pre- and postoperative follow-up time (median 6.5 and 5.3 years, respectively) a retrospective study design predisposes the data for statistical challenges and bias when the results are not properly adjusted for confounders. First, the long follow-up predisposes the risk of changing habits not recognized. For instance, there was more than 1 visual field device. Was one of the devices used toward the starting date of the study rather than at the end of the study period, and were the settings similar over the follow-up? The same comes with IOP measurements. Were the IOP measurements based on applanation or rebound tonometry, or both? And were some of the devices used toward the starting date of the study? Interestingly, during the follow-up, increased rates of glaucoma progression according to visual fields were noted, but the mean number of glaucoma meds were reduced. Were the treatment decisions based on the IOP readings, or is the discrepancy explained by something else, such as compliance problems when patients are getting older? This in turn raises a question: Did automated imaging analysis of the glaucoma (OCT, GDX, etc) support the findings regarding VF decay? Second, considering the long follow-up time, the results should be adjusted with ocular comorbidities such as posterior capsule opacification (PCO) in pseudophakic eyes. Considering the potential differences in the IOL optics (eg, light-filtering qualities), the analysis between clear and blue light-filtering intraocular lenses would have been informative. Third, although the associations between the baseline patient age and visual field rates after the surgery were nonsignificant, the long follow-up periods pre- and postoperatively and nonlinear glaucoma progression should be incorporated in the statistical analysis to avoid the overestimation of VF decay post-pseudophakia.

Taken together, the findings presented by Kim and associates need to be confirmed in a controlled prospective study setting. To evaluate the role of intraoperative and early postoperative stress on VF decay, one could randomize the patients to different peri- and early postoperative glaucoma treatment protocols, phaco settings and fluidics, as well as conduct a subgroup analysis according to the presence of early IOP spikes. Polyclinical evaluations could be supplemented by the IOP home device measurements.

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Risk Factors Associated with Persistent Anterior Uveitis After Cataract Surgery



EDITOR:

THE UVEITIS OF CATARACT SURGERY HAS RECENTLY BEEN studied by Reddy and colleagues.¹ They report that the uveitis is typically of a longer duration in African Americans than in whites. It is also stated that any anterior uveitis that persists “may not be detrimental to long-term visual outcomes.”

Persisting uveitis after cataract emulsification merits some thought. While such uveitis may not adversely affect persons of African ancestry, in older whites it may trigger a neovascular effect at the macula. Degeneration of the aged macula is common in white populations. It has been debated whether cataract surgery can convert age-related macular degeneration (AMD) from the dry to the wet

neovascular type.² Postoperative uveitis is vital to the discussion. Surgical leakage from the blood-aqueous barrier sends cytokines into the vitreous. The efflux includes growth factors. Conversely, molecules can drift from the posterior to the anterior segment. A notable example is vascular endothelial growth factor. Exuded by ischemic retina, it flows anteriorly to induce rubeosis iridis. After ultrasonic dismantling of the lens, angiogenic cytokines leak towards the macula. If the macular target is vulnerable there may plausibly arise a neovascular membrane. Thus, the strength of the angiogenic stimulus and the sensitivity of the macula are key.³

Regarding the “chemical stimulus,” consider the case of postoperative macular edema. Certain cytokines in the aqueous are raised in eyes with edema when compared to eyes without edema.⁴ Vascular endothelial growth factor is part of this gradient from the aqueous to the macula. The emulsification of a denser cataract (and related iris trauma) means more cytokines⁵ and the sending of a stronger angiogenic signal towards the macula.

The other side of the mechanism is the capacity of the AMD macula to grow a neovascular membrane. Concerning macular response, let us refer to the extraction of cataract from the diabetic eye. Maculae with microangiopathy—a vulnerable substrate—are more prone to developing edema after operation. In AMD, a spectrum of risk again exists in terms of neovascular potential. Maculae with a few fine drusen are a low-risk phenotype. Maculae with larger drusen, atrophic patches, or pigmentary change have more disordered tissue. These eyes carry a risk of growing a neovascular membrane.⁶ Also, if the fellow eye has neovascular AMD the risk of new vessels at the macula is greater still.

On noting flow dynamics, it is seen that anterior uveitis feeds an angiogenic stimulus to the macula. In white populations, it is important to assess the macula judiciously when planning a cataract extraction. If a macula has neovascular potential then it can develop angiogenesis as a result of surgery. Beside these maculae—where denser cataracts are emulsified—it is valid to tightly control the uveitis to lessen the chance of neovascular sequelae. Generally, I suppress post-phaco uveitis over 2 months for the at-risk macula. Unless the course of surgical uveitis is well controlled, the macula receives from the anterior segment a flow of cytokines that favor neovascular AMD, a lesion that imposes lasting deficits on central vision.

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EDITOR:

WE ARE GRATEFUL TO DR. GANDHI FOR THE OPPORTUNITY to provide further comments regarding our recently published article.¹

We agree with Dr. Gandhi that postcataract surgery may exacerbate the rate of age-related macular degeneration (AMD) progression as demonstrated by Donoso and associates.² Cataract surgery is hypothesized to physically predispose the eye to AMD, perhaps through inflammatory mechanisms as suggested by van der Schaft and associates.³ In addition, the study that Dr. Gandhi cited by Ho and associates⁴ demonstrated epidemiologic evidence of a link between cataract surgery and neovascular AMD over 5-year follow-up using a population-based claims data set from Taiwan.

The scope of our study was to look for the postphacoemulsification incidence of persistent anterior uveitis as designated by the Standardization of Uveitis Nomenclature Working Group.⁵ No patients in our study population developed neovascular AMD during the limited follow-up period. Interestingly, while African-Americans were at higher risk for developing persistent anterior uveitis, a separate observational study conducted at our institution