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 cytokines[5] 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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Risk Factors Associated with Persistent Anterior Uveitis After Cataract Surgery

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 associates[4] 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

showed that African-American race was associated with a reduced risk of early/intermediate AMD (adjusted odds ratio [AOR] = 0.08, confidence interval [CI] 0.01–0.67) and neovascular AMD (AOR = 0.15, CI = 0.03–0.72).[6] In order to elucidate any postcataract surgery uveitis as a primary etiology for conversion to neovascular AMD, it would require a separate prospective well-designed longitudinal study.

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Predictors of Neovascular Glaucoma in Central Retinal Vein Occlusion

EDITOR:

WE WOULD LIKE TO ADDRESS SEVERAL ISSUES WITH THE study of Rong and associates.[1]

The study had a retrospective design and a relatively short-term follow-up (17 months), with a pretty high proportion of patients (13.26%) who developed neovascular glaucoma (NVG). It is assumed that the proportion of patients with NVG would have been greater if the gonioscopy had been uniformly performed by clinicians at each visit for detecting patients with early NVG (neovascularization of the angle and normal intraocular pressure [IOP]).

There was a selection bias attributable to inclusion in the study of patients with 2 types of central retinal vein occlusions (CRVOs) (ischemic and nonischemic forms) having definitely different pathogenesis, clinical features, prognoses, and management. Likewise, 2 completely different etiologic subgroups of patients have been encompassed, namely, patients older than 50 years who usually have common systemic conditions such as hypertension and diabetes, and patients less than 50 years of age, where other mechanisms, such as the hyperviscosity syndrome or inflammatory condition should be specifically considered and accounted for. Taken together, these findings may have confounded the results.

The diagnosis of CRVO in this series was based on acute vision loss, diffuse intraretinal hemorrhages, and venous tortuosity. Taking into account these findings as well as the fact that only 14 patients (14.28%) had relative afferent pupillary defect (RAPD), we inferred that the vast majority of the patients included in this study experienced nonischemic CRVO. Nothing was stated referring to the diagnostic criteria for the ischemic type of acute CRVO, when marked and extensive intraretinal hemorrhages prevented a clear angiographic evaluation of the retinal capillary nonperfusion zones. Accordingly, we suggested[2,3] the presence of at least 4 of the 5 following criteria: the visual acuity ≤20/400 Snellen equivalent; the ability to see ≤V/4e isopter based on the Goldmann perimeter; the presence of the RAPD in patients with 1 normal eye; the extensive ocular fundus changes (striking amount of hemorrhages, venous tortuosity, cotton-wool spots [>5], and disc and macular edema); and the intraocular pressure reduction in the occluded eye of ≥4 mmHg compared with the congener eye.

The following relevant data are missing in the study: the stratification of the CRVOs (ischemic/nonischemic forms); the type of anti–vascular endothelial growth factor (anti-VEGF) agent used and the schedule of treatment; the assessment of the macular ischemia by quantification of the diameters and area of the foveal avascular zone; the existence or not of the disorganization of the retinal inner layers and its severity; the optical coherence tomography patterns of the macular edema (diffuse/subretinal fluid/cystic changes/mixed type) and the location of the intraretinal cystoid fluid (ganglion cell layer/inner or outer nuclear layers); the damages of the photoreceptor cell layer (thinning of the outer nuclear layer/external limiting membrane band defects/ellipsoid zone disruption, interdigitation zone loss); the changes of the retinal pigment epithelial band-Bruch membrane complex (pigment migration within the neurosensory retina, retinal pigment epithelium [RPE] porosity, microrips or blowouts in the RPE, focal RPE atrophy, RPE thickening); and the proportion of the patients with ocular hypertension, cardiovascular and cerebrovascular diseases, obesity, hyperviscosity syndromes, and inflammatory conditions.