CONFLICT OF INTEREST DISCLOSURES: SEE THE ORIGINAL article for any disclosures of the authors.

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The Risk of Primary Open-Angle Glaucoma Following Vitreoretinal Surgery—A Population-based Study

EDITOR:

WE CONGRATULATE AND HIGHLY APPRECIATE MANSUKHANI and associates for their article.[1] However, we would like to mention a few points and seek the authors' kind attention.

Firstly, it is not very clear to us why the cases in this study were designated as primary open-angle glaucoma. In previous studies, it was already established that raised intraocular pressure (IOP) was a known sequela of vitreoretinal surgery,[2,3] and in this present study also, raised IOP was following vitreoretinal surgeries and therefore secondary glaucomas.

Secondly, the authors have mentioned that high baseline IOP was associated with increased risk of primary open-angle glaucoma following vitreoretinal surgery. We are interested to know the baseline IOP of those who developed open-angle glaucoma postoperatively.

Thirdly, in this present study, subjects using steroid for more than 2 months were excluded. Whereas previous studies reported IOP rise within 3–6 weeks of steroid use and few studies reported early rise of IOP within first or second week after initiation of steroid use.[4,5]

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The Risk of Primary Open-Angle Glaucoma Following Vitreoretinal Surgery—A Population-based Study

REPLY

WE THANK BORGOHAIN AND ASSOCIATES FOR THEIR COMments on our previously published paper, "The risk of primary open-angle glaucoma following vitreoretinal surgery—a population-based study."[1] We agree that raised intraocular pressure (IOP) is a known sequela of vitreoretinal surgery owing to multiple causes. However, elevation of IOP is not synonymous with glaucoma, and is in fact not part of the current definition of glaucoma.[2] Most patients with elevated IOP never develop glaucoma, while a large proportion of glaucoma patients (27% in our study) do not have recorded IOP elevations. In the Discussion section of our paper, we listed reasons why the glaucoma in our patients could be considered secondary. However, we were unable to determine phenotypic differences from primary open-angle glaucoma (POAG). As well, there are clear secondary causes of glaucoma after vitreoretinal surgery, and our cases were designated as POAG to distinguish them from cases where a clear etiology was present.

The paper that Borgohain and associates referenced to support their argument reported outcomes of secondary glaucoma following vitrectomy.[3] Thirty-nine percent of their cohort had intravitreal silicone oil and 11% of eyes had extensive peripheral anterior synechiae or neovascularization. In other words, their cohort represented the types of patients we meticulously excluded. As we stated in the Discussion section of our paper, a secondary cause of glaucoma could not be attributed to our cohort based on existing knowledge. However, we postulated that, with increased understanding of the pathophysiology in these patients, "glaucoma following vitrectomy" will become a recognized entity among the causes of secondary glaucoma.

Concerning the issue of mean baseline IOP being a risk factor for the development of glaucoma, it is important to note that the patients included in the study did not have elevated baseline pressures. The mean baseline IOP among the cohort developing POAG, or POAG and POAG suspect, was 16.9 ± 2.5 mm Hg and 16.3 ± 2.7 mm Hg (mean ± standard deviation), respectively. The mean baseline IOP of the operative cohort as a whole was 15.2 ± 3.2 mm Hg.

With respect to the issue of steroid use, we agree that IOP rise can occur within 1–2 weeks of initiating therapy, and therefore all patients developing elevated IOP while on steroids were excluded from the study. Out of an abundance of caution, we also excluded patients who used steroids for an extended period of time, defined as 2 or more months (18 patients overall, Figure 1[1]). Prolonged steroid use after vitreoretinal surgery is often recommended when there is ongoing inflammation, and the intent was to minimize confounding from glaucoma associated with inflammation or its treatment. However, excluding all patients receiving steroid eye drops for 1–2 weeks would be impractical, as this would include most postoperative patients, and a cut-off of 2 months was felt to be more reasonable.

We thank Borgohain and associates for their interest in our paper and the opportunity to clarify some key points.

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Predictive Factors of Response to Mineralocorticoid Receptor Antagonists in Nonresolving Central Serous Chorioretinopathy

REPLY

WE THANK DRS DAN AND MIHAI CĂLUGĂRU FOR THEIR interest in our work.[1] However, we would like to clarify some of the points they raised.

We agree that our study has a retrospective design with missing data. Nevertheless, it is incorrect to state that "34% of eyes were lost to follow-up." As described in the methods section, patients with complete resolution of serous retinal detachment at 3 months were treated for only 3 months and were not included in the analysis at 6 months. All missing data and number of studied eyes are precisely written in each table.

However, we acknowledge the high rate of missing data for optical coherence tomography (OCT) angiography. Indeed, we included patients from 2012 to 2016 and the OCT angiography machine was available in our hospital in 2015. All choroidal neovascularization (CNV) detected in our study were type 1 CNV. Our results are in line with the study by Sacconi and associates[2] showing that the presence of CNV is a factor of poor response to treatment. Thus, we agree that the difference between both groups in our study is not statistically significant, probably because of the small sample size. This limitation is identified in the discussion section of our paper.

We also agree that corticosteroid intake could be a factor of poor response to treatment, but it did not reach statistical significance in the multivariate analysis. We thank the authors for their insight into the possible role of corticosteroids related to the mineralocorticoid receptor pathway hypothesis.

Furthermore, the authors focus on the lack of extensive characterization of retinal imaging. We agree that analyzing OCT parameters such as the retinal pigment epithelium status or the line of photoreceptor cells would have been interesting for predicting treatment response and may be a suitable outcome for future analyses. We analyzed several established parameters including central macular thickness, subretinal detachment height, and