combined dexamethasone intravitreal implant and glaucoma drainage device placement for uveitic glaucoma

To the Editor:

Uveitic glaucoma is a clinician’s gray area of interest where the best treatment is still not clear. We read with great interest the recently published article by Nguyen et al1 “Combined Dexamethasone Intravitreal Implant and Glaucoma Drainage Device Placement for Uveitic Glaucoma” and we would like to appreciate the work of authors.

The cause of raised intraocular pressure in these cases may be the blockage of the trabecular meshwork by the inflammatory cells/pigments, trabeculitis, peripheral anterior synechiae, and which may get exaggerated by steroid responsiveness.2 Gonioscopy is an important tool to determine the angle status but the authors have not mentioned about the angle status in this study cohort. Uveitic episodes of hypotony due to ciliary body shutdown at postoperative month 9 from a vitreous hemorrhage following a repeat intraocular pressure building up once the inflammation resolves3,4.

We made a few more queries and observations and would like to share with the authors.

First, the mean preoperative logMAR best-corrected visual acuity was 0.55 ± 0.40 which deteriorated over the 9 months of follow-up as illustrated in the Box plot. We want to know whether the cause for decreased vision preoperatively was the presence of cataract, uveitis or any posterior segment pathology. As mentioned by the authors there was a decrease in number of episodes of hypotony due to ciliary body inflammation followed by intraocular pressure building up once the inflammation resolves3,4.

Second, preoperative finding like anterior chamber depth, lens status (phakic or pseudophakic), degree of synechiae in each eye has not been mentioned. Bayer and Onol5 and our own clinical experience show that above factors play a determinant role in deciding the site and position (ie, anterior chamber or ciliary sulcus) of tube placement of Ahmed glaucoma valve. A sulcus placement may be desirable in case of progressing synechiae. In case the tubes were placed in the anterior chamber, we wanted to know if there was an increase noted in degree of synechiae postsurgery as this may affect the tube position. We also want to know if these eyes had undergone previously failed filtering surgeries or were virgin eyes with no previous surgical intervention.

In results, it was found that 1 eye had hypotony on postoperative 1-month upto 3-month; however, the cause for hypotony was not mentioned. Also we want to know if there was corneal-tube touch or lens-tube touch noted in this case.

Third, most of the patients in this study cohort were on topical and systemic immunosuppressant preoperatively and postoperatively, so to what extent the dexamethasone implant was beneficial in reducing the frequency of uveitic episodes or increasing the success of the tube implant is not clear. A control group comprising of similar patients on preoperative topical and systemic corticosteroids/immunosuppressant undergoing tube implant without the dexamethasone implant should have been included. So, we agree with the authors that a long-term prospective study with a control group would be needed to give a better understanding of the role of combined placement of intravitreal dexamethasone implant with glaucoma drainage device in eyes with uveitic glaucoma.

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In Reply:

We thank Bansal and colleagues for highlighting some of the challenges in managing uveitic glaucoma and for their correspondence regarding our study.

While there was no statistically significant change in visual acuity throughout the 1-year postoperative period, the study’s small sample size led to greater data spread at postoperative months 1 and 9 (Fig. 1). The box plots demonstrate how mean logMAR visual acuity (crosshairs) was more susceptible to skew from the larger data spread, while the median logMAR visual acuity (line) was more stable.

Causes of decreased vision in the postoperative period included one eye with hypotony maculopathy due to ciliary body shutdown at postoperative months 1 and 3, which resolved by postoperative month 6 (without corneal-tube or lens-tube touch). Another eye had transiently-reduced visual acuity at postoperative month 9 from a vitreous hemorrhage following a repeat intravitreal dexamethasone implant injection performed in the retina clinic, which

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resolved by month 12. Furthermore, all eyes had cataract surgery either before (n = 6) or in conjunction (n = 2) with combined dexamethasone intravitreal implant and glaucoma drainage device placement. Thus, cataract was not a cause of visual acuity changes in the postoperative period.

With regards to the status of the angle, preoperative gonioscopy demonstrated either open angles without synechiae (n = 6) or few scattered peripheral anterior synechiae (n = 2). Systematic measurement of anterior chamber depth and postoperative gonioscopy were not performed. In addition, all eyes (n = 8) had no previous glaucoma filtering surgeries and had the glaucoma drainage device placed in the anterior chamber.

As noted by Bansal and colleagues, future prospective studies comparing the effectiveness of combined dexamethasone intravitreal implant with glaucoma drainage device versus glaucoma drainage device alone will help further elucidate the risks and benefits of this surgical approach for managing uveitic glaucoma.

**Effects of Intravitreal Anti-VEGF Therapy on Glaucoma-like Progression in Susceptible Eyes**

**To the Editor:**

We read with great interest the recently published article “Effects of intravitreal anti-vascular endothelial growth factor (VEGF) therapy on glaucoma-like progression in susceptible eyes” by Du and colleagues and would like to appreciate the work of the authors for the same. It highlights well the need for a reliable preinjection visual field analysis/optical coherence tomography (OCT) and the need for constant intraocular pressure monitoring and regular glaucoma follow-up in patients with coexisting glaucoma and retinal disease.

We would like to highlight a few points in the study that require some clarification.

First, in the inclusion criteria, it is mentioned that a minimum of 2 consecutive Humphrey visual field or retinal nerve fibre layer (RNFL) thickness by OCT have been considered for chart analysis (minimum duration between baseline and second test being > 12 mo). However, it is not very clear how they have assessed progression and what were the number of fields studied for glaucoma progression analysis. It would be of value to mention the average number of fields/OCT per patient studied in both the groups (injected and noninjected eyes) because 2 to 3 fields/OCT might not be enough to accurately study progression in such eyes. Moreover, patients of retinal diseases can have fallacious results of perimetry/RNFL due to progression of retinal disease itself. Monitoring of fundus photographs to look for disc changes might be a better option in such cases.

Second, retinal lasers have not been excluded from the study. An article published in October 2019 by Wadhwani et al concludes that PRP can cause RNFL thinning on long-term follow-up. Hence, it is not clear whether any of the patients underwent retinal laser treatment because that can confound the perimetry and OCT findings.

Another point to be looked into is the study of the noninjected eyes as the control group to study the natural progression of pre-existing glaucoma in these patients. Although, at baseline, both the groups were almost similar in severity of glaucoma in terms of baseline intraocular pressure, number of prescribed anti-glaucoma drops, number of prior invasive glaucoma interventions, mean deviation and pattern standard deviation on visual field analysis, and RNFL thickness on OCT, it is well known that bilateral eyes of primary open angle glaucoma can show asymmetrical progression and hence, just by comparing with the noninjected fellow eye, we cannot truly comment whether the progression in injected eyes could be attributed to pre-existing glaucoma or due to the anti-VEGF injections.

Last, there is no mention about the number of injections in the eyes that progressed and required further glaucoma surgery or laser. It would be worthwhile to study any co-relation between the number of anti-VEGF injections and progression.

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