Correspondence

Comment on: Characterization of In Vivo Biomechanical Properties in Macular Corneal Dystrophy

EDITOR:

WE READ WITH INTEREST THE STUDY BY FEIZI AND ASSOCIates[1] elucidating the corneal biomechanics in patients with macular corneal dystrophy (MCD).

The authors evaluated corneal hysteresis (CH) and corneal resistance factor using the Ocular Response Analyzer (Reichert Ophthalmic Instruments, Buffalo, New York, USA) in 3 groups: cases with MCD, cases that underwent penetrating keratoplasty (PK) for MCD, and normal control subjects.[1]

Did the authors observe any case in the post-PK group with a recurrence of MCD over the follow-up period of ≤264 months? If so, it would be interesting to know the corneal biomechanics in recurrent MCD cases after PK.

An interesting finding brought to light by the authors was that the mean CH did not differ significantly between the MCD eyes and the normal eyes, in spite of reduced central corneal thickness and structural damage caused by the corneal dystrophy. Fahnehjelm and associates[2] reported an increased CH in patients with mucopolysaccharidosis I and VI and attributed it to the stromal deposition of glycosaminoglycans. MCD, like mucopolysaccharidosis, is associated with an accumulation of glycosaminoglycans in the corneal stroma; however, the resultant incremental effect on CH may have been negated by the disruption of the Descemet membrane, Bowman layer, and altered collagen fibril spacing in MCD. Future studies in this arena may help us further understand the impact of corneal structural alterations on its biomechanics.

Intraocular pressure (IOP) was evaluated using Goldmann applanation tonometry (GAT). The authors remarked that GAT IOP might be underestimated in patients with MCD or those who have undergone PK and commented on the significance of accurate IOP assessment in these patients. Cornea-compensated IOP and Goldmann-correlated IOP measured with the ocular response analyzer have been shown to be higher than GAT IOP in post-PK patients.[3,4] The authors have not commented on these IOP values obtained in their cases. A comparative evaluation of cornea-compensated IOP among the 3 groups and its correlation with conventional GAT IOP readings will help further our understanding regarding accuracy of IOP estimation by different means.

Lastly, there is no mention of corneal curvature and astigmatism in post-PK or the control group. It is as yet not clear if the corneal curvature and astigmatism have a definite impact on the measured corneal biomechanical parameters and IOP.[5,6] A discussion of this aspect would help readers further understand the impact of corneal curvature on the corneal biomechanics in post-PK patients.

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FUNDING/SUPPORT: NO FUNDING OR GRANT SUPPORT. Financial disclosures: The following authors have no financial disclosures: Sridevi Nair, Manpreet Kaur, and Jeewan Singh Titiyal. The authors attest that they meet the current ICMJE criteria for authorship.

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Reply to Comment on: Characterization of In Vivo Biomechanical Properties in Macular Corneal Dystrophy

EDITOR:

WE ARE GLAD THAT OUR ARTICLE[1] WAS OF GREAT INTEREST to Nair and associates and thank them for their in-depth insight and constructive comments. Their correspondence

0002-9394/$36.00
https://doi.org/10.1016/j.ajo.2020.07.042

gives us a chance to elaborate on different aspects of our article. The results of our study showed that corneal resistance factor was significantly reduced in eyes with macular corneal dystrophy (MCD) compared with normal corneas.[1] This parameter did not return to the normal value after penetrating keratoplasty (PK), which may suggest a decrease in the rigidity of the sclera, in addition to the cornea, in patients with MCD.[1] Reduced corneal rigidity can lead to underestimating intraocular pressure (IOP) in eyes with MCD and those undergoing corneal transplantation for this condition.[1] Furthermore, decreased corneal and scleral rigidity explains why corneal transplantation is more challenging technically in MCD compared with other stromal dystrophies (eg, lattice and granular). It is a common observation by corneal transplant surgeons that the anterior chamber collapses easily and that the lens–iris diaphragm displaces forward during PK for MCD, increasing the incidence of iris prolapse and iridocorneal adhesions. The following are answers to questions raised by Nair and associates.

As mentioned in our article, all PK eyes had clear grafts at the time of enrollment, and the presence of any graft opacities, including recurrence of dystrophy in the graft, led to patient exclusion.

Evaluation of the accuracy of different tonometers for measuring IOP in patients with MCD was not in the scope of our study, which aimed to evaluate corneal biomechanics in this stromal dystrophy. It can be a subject for future investigations to compare IOP measurements by different tonometers in MCD. Per the recommendation of Nair and associates, we opted to compare IOP measured by the ocular response analyzer, including Goldmann-related IOP (IOPg) and cornea-compensated IOP (IOPcc), with that measured by the Goldmann applanation tonometer (GAT). IOP_{GAT}, IOPg, and IOPcc were 11.25 ± 1.69 mm Hg, 12.06 ± 4.03 mm Hg, and 14.37 ± 3.41 mmHg in the MCD group, respectively ($P < .001$). These measurements were 12.0 ± 2.67 mm Hg, 11.44 ± 3.04 mm Hg, and 13.90 ± 2.90 mm Hg in the PK group ($P = .01$) and 13.46 ± 2.17 mm Hg, 14.02 ± 2.01 mm Hg, and 13.63 ± 2.70 mm Hg in the control group ($P = .42$), respectively. Intragroup analyses revealed that IOP_{GAT} and IOPg were significantly lower than IOPcc in the MCD and PK groups, whereas these 3 IOP measurements were comparable in control subjects. Intergroup comparisons showed that IOP_{GAT} and IOPg were significantly lower in the MCD and PK groups than the corresponding measurements in the control group. IOPcc, however, was comparable among the 3 groups.

Mean keratometry and keratometric astigmatism were 46.35 ± 2.91 diopters (D) and 6.28 ± 2.80 D after PK, respectively. These readings were 42.81 ± 1.19 D and 1.74 ± 0.61 D in the control group, respectively. Keratometry readings had no significant correlation with corneal hysteresis, corneal resistance factor, IOP_{GAT}, IOPg, or IOPcc in PK or control group. Nonsignificant association of astigmatism with IOP_{GAT} readings in the PK group is attributable to the method we used to reduce the effect of corneal astigmatism on readings by GAT, which was rotation of the tonometer prism 43° to the least-curved meridian.

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FUNDING/SUPPORT: NO FUNDING OR GRANT SUPPORT. Financial disclosures: The following authors have no financial disclosures: Sepehr Feizi, Zahra Karjou, Hamed Abbasi, Mohammad Ali Javadi, and Amir A. Azari. The authors attest that they meet the current ICMJE criteria for authorship.

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Comment on: Long-term Results of Trimethoprim Sulfamethoxazole Versus Placebo to Reduce the Risk of Recurrent *Toxoplasma gondii* Retinochoroiditis

EDITOR:

WE READ WITH GREAT INTEREST THE ARTICLE TITLED "Long-term results of trimethoprim sulfamethoxazole versus placebo to reduce the risk of recurrent *Toxoplasma gondii* retinochoroiditis" by Fernandes Felix and associates.[1] This prospective double-masked clinical trial found superior effects of 1 year of treatment with trimethoprim-sulfamethoxazole (TMP-SMZ) vs placebo in reducing the risk of recurrence during a 6-year follow-up period. The authors concluded that TMP-SMZ may be used safely for prophylaxis of recurrent toxoplasmic retinochoroiditis.[1]

Visual acuity loss in toxoplasma retinochoroiditis occurs because of the involvement of the macula or optic nerve head. In healthy individuals, toxoplasmic retinochoroiditis is a self-limited disease. Small peripheral lesions may be observed for spontaneous resolution. The majority of lesions in this study were peripheral (around 70% in the TMP-SMZ arm and 65% in the placebo arm).[1] There was no benefit in terms of visual acuity improvement with drug therapy over placebo treatment. Even with multiple recurrences, the visual acuity improvement and final visual acuity were not inferior as compared to cases with no/single recurrence. Perhaps such cases with peripheral lesions could have been observed.