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Pathophysiology of Transient Corneal Edema and Pseudophakic Cystoid Macular Edema

Dear Editor,

We read with interest the article titled "Transient corneal edema is a predictive factor for pseudophakic cystoid macular edema after uncomplicated cataract surgery" [1], in which the authors found that transient corneal edema (TCE) following cataract surgery is a predictive factor of pseudophakic cystoid macular edema (PCME). The study was well designed and conducted and answered a question that we thought we might know but never really studied.

However, although the authors suggested that TCE and PCME may partly share the same etiologies of inflammation, we would like to point out that TCE and PCME may have different pathophysiologies. As the authors described, PCME may be caused by disruption of the blood-aqueous and blood-retinal barriers due to postoperative inflammation [2]. Postoperative anterior chamber (AC) reaction is also caused by destruction of the blood-aqueous barrier and reflects the degree of inflammation; thus, increased AC reaction is associated with development of PCME [3]. However, TCE is caused by endothelial cell damage due to ultrasound energy, direct mechanical trauma or toxicity of the irrigating solution [4], rather than by disruption of the blood barriers. TCE tends to occur more frequently in cases with increased surgical difficulty, such as advanced age, increased nuclear density, narrower AC, small pupil, floppy iris syndrome and pseudoexfoliation syndrome, in which a larger amount of ultrasound energy is used, mechanical trauma to corneal endothelial cells is frequent and the toxic effect of the irrigation solution increases with operation time. The authors also demonstrated that patients with TCE had significantly smaller AC volume compared to those without TCE and also tended to have older age, shorter AC depth and longer surgical duration, although the differences were not statistically significant [1]. These conditions also tend to increase the severity of postoperative inflammation as higher ultrasound energy, increased manipulation of surgical instruments and longer surgical duration result in increased damage to tissues including iris, ciliary body and trabecular meshwork. This exacerbates the breakdown of blood-aqueous and blood-retinal barriers, consequently leading to PCME and AC reaction. Taken together, therefore, although TCE and PCME may have basically the same cause, the mechanisms leading to the two conditions appear to be different. Nevertheless, we agree with the authors that TCE in an early postoperative period can be a valuable predictor of later development of PCME because they successfully showed the significant association between the two conditions.

In addition, we suggest that the results would be even more interesting if the authors included information about the degree of AC reaction. As AC reaction is a marker of postoperative inflammation and does share, at least in part, the pathogenesis of CME [3], we expect that there is a close relationship among TCE, PCME and AC reaction. We understand that the authors had difficulty obtaining data on AC reaction, as well as nuclear density and cumulative ultrasound energy [1]. The authors previously showed that TCE and PCME can be quantified using spectral domain optical coherence tomography, and AC reaction can be quantified using laser photometry [3]. We believe analyses of the quantified data on TCE, PCME and AC reaction in further studies will provide more detailed information that will be helpful in clinical practice.

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Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Author reply

Dear Editor,

Thank you very much for this opportunity to discuss our recent paper titled "Transient corneal edema is a predictive factor for pseudophakic cystoid macular edema after uncomplicated cataract surgery" [1]. In this paper, we concentrated on very subtle transient corneal edema (TCE) after routine uncomplicated cataract surgery. Such subtle TCE is occasionally overlooked on slit-lamp examination (4 of 17 eyes in our study). Although TCE occurs with relatively high frequency (11.3% in our paper and 50% in Tao et al. [2]), little is known about the etiology and clinical relevance of subtle TCE. TCE in our study should be differentiated from prominent corneal edema caused by mechanical endothelial damage during difficult or inadvertent phacoemulsification. In the setting of mechanical damage, serial tracing of endothelial cell count shows a marked decrease in final cell density. In our study, although preoperative endothelial density in TCE eyes was slightly lower than that in control eyes, the serial tracing and comparison of endothelial cell density did not show any significantly increased loss compared to that in control eyes. Because postoperative endothelial count was not performed in all study eyes, this result was omitted from the original paper. Therefore, we suggest inflammation and transient functional dysfunction as the main etiology of TCE, although mechanical effects cannot be excluded. The finding that diabetes mellitus increased the risk of TCE also supports the greater role of dysfunction compared to mechanical cell death. Inflammatory material released from damaged tissue during cataract surgery can fill the anterior chamber during the immediate postoperative period. The small volume of the anterior chamber can greatly increase the concentration of inflammatory mediators and result in frequent TCE, as shown in our paper. The lack of quantitative analysis of anterior chamber reaction during the postoperative course and then cumulative ultrasound energy delivered are the major limitations of our study. We agree that including these two parameters in the analysis will provide better understanding of TCE and pseudophakic cystoid macular edema in future study.

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A Case of Sympathetic Ophthalmia after 23-Gauge Transconjunctival Sutureless Vitrectomy

Dear Editor,

Sympathetic ophthalmia (SO), a bilateral granulomatous panuveitis, is a rare condition that can occur after a penetrating eye injury or intraocular surgery [1]. Although the risk of SO following conventional 20-gauge vitrectomy has previously been suggested [1], there are only two reported cases of SO following transconjunctival sutureless vitrectomy (TSV) [2,3]. We present a case of SO after 23-gauge TSV.

A 45-year-old Korean man presented with blurred vision in his left eye. His best-corrected visual acuity was 0.8 in the right eye and 0.4 in the left eye, and the corresponding intraocular pressures were 13 and 15 mmHg, respectively. Anterior segment slit-lamp examination showed tobacco dust in the anterior vitreous cavity of the left eye. Fundoscopic examination revealed a superotemporal macula-off retinal detachment in the left eye. A large horseshoe tear and several small retinal tears were observed in the area of the detached retina. The patient had no history of ocular trauma or surgery.

The patient underwent 23-gauge TSV under local anesthesia. Endolaser photocoagulation and gas (sulphur hexafluoride) tamponade were used to treat the retinal tears. Postoperative complications, including intraocular pressure increase or decrease, were not observed. One month after surgery, his best-corrected visual acuity in the left eye was 0.6 and the retina was reattached in the left eye. Two months after surgery, the patient returned with decreased vision and metamorphopsia in his right eye. His best-corrected visual acuity was 0.7 in the right eye and 0.6 in the left eye. Slit-lamp examination showed a moderate inflammatory reaction in the anterior chamber and anterior vitreous of both eyes. Both fundoscopic examination and optical coherence tomography revealed bilateral subretinal fluid, choroidal thickening, and choroidal folds in both eyes (Fig. 1A-1D). Fluorescein angiography showed multiple pinpoint leakages at the level of retinal pigment epithelium in the late phase (Fig. 1E and 1F).

As the patient had bilateral panuveitis, subretinal fluid, and multiple leakages in angiography, we considered either Vogt-Koyanagi-Harada disease (VKH) or SO as a diagnosis. However, he had no systemic symptoms. Furthermore, according to the diagnostic criteria of VKH, it could not be VKH because he had a recent history of ocular surgery. In addition, we did not find any evidence of infection in this patient. As a result, we diagnosed him with SO. The patient was treated with 90 mg of oral prednisolone per day. The subretinal fluid cleared in both eyes following treatment (Fig. 1G and 1H). Oral prednisolone was gradually tapered and continued at a low dose. Nine months after surgery, his best-corrected visual acuity was 0.9 in the right eye and 0.6 in the left eye while taking oral prednisolone 10 mg per day.

SO is a rare bilateral diffuse granulomatous uveitis that occurs a few days to several decades after penetrating accidental or surgical trauma to the eye. Pars plana vitrectomy is a surgical procedure associated with SO [1]. The exact mechanism of SO is not clear, but it is hypothesized that SO