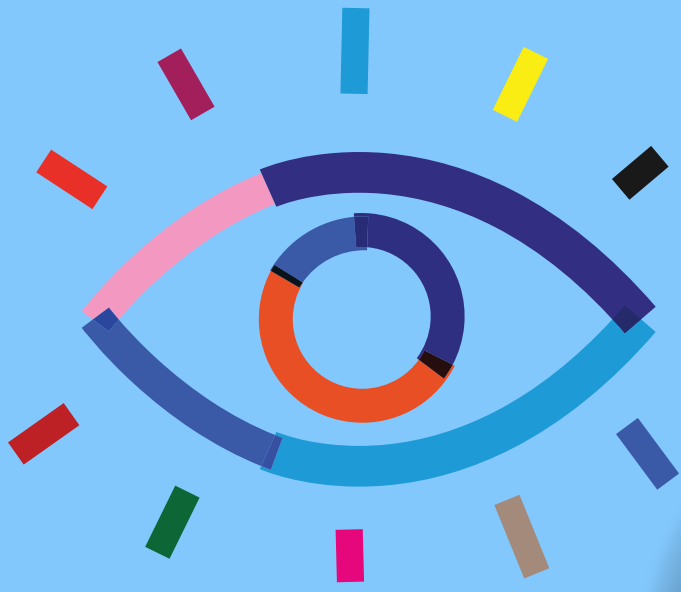

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MANAGING Persistent Postoperative CORNEAL EDEMA

Tips for cataract surgery in patients who have a compromised cornea.

By Zeba A. Syed, MD

Considerable advances in phaco techniques and technologies have reduced trauma to the corneal endothelium. Postoperative persistent corneal edema can still occur, however, especially in an eye with a compromised cornea. The edema may be transient and resolve after the immediate postoperative period, or it may be chronic and lead to progressively worsening vision and patient frustration.

The focus of this article is fourfold:

- No. 1: The pre- and intraoperative risk factors for persistent corneal edema after cataract surgery in patients with a compromised cornea;
- No. 2: Key components of the evaluation;
- No. 3: Strategies for medical management; and
- No. 4: Strategies for surgical management.

FOCUS NO. 1: RISK FACTORS

Preoperative. The most common risk factor for persistent postoperative corneal edema is Fuchs endothelial corneal dystrophy (FECD), a condition involving the development of guttae in the Descemet membrane and subsequent endothelial dysfunction. The resulting corneal edema is associated with decreased vision and sometimes pain in advanced cases where superficial bullae have formed. In one study, approximately 10% of patients with FECD who underwent cataract surgery later required endothelial keratoplasty.[1]

A history of endothelial trauma is another risk factor. Patients who have undergone glaucoma surgery (eg, trabeculectomy or the implantation of a drainage device) may have preexisting endothelial compromise. Potential mechanisms of cell loss include alterations in the circulation patterns of aqueous humor, intermittent tube-cornea touch, and elevated oxidative and inflammatory markers in the anterior chamber.[2] Chronic anterior segment inflammation in patients with a history of uveitis can affect endothelial cell health. These individuals are also at risk of postoperative corneal decompensation.[3]

During the preoperative evaluation, several findings suggest that further endothelial decompensation may occur postoperatively. Microcystic edema, clinical stromal thickening observed at the slit lamp, a low central endothelial cell density (< 1,000 cells/mm^2) on specular microscopy, and/or increased central corneal thickness (> 640 µm) on ultrasound pachymetry may be prognostic of deteriorating endothelial function postoperatively.[4]

Intraoperative. Factors that may aggravate endothelial compromise include surgeon inexperience, duration of surgery, trauma from the inappropriate use of instruments, the use of elevated amounts of phaco energy (as occurs in eyes with advanced cataracts), toxicity from medications injected intracamerally, and complications such as vitreous loss and IOL-endothelial touch.

Phacoemulsification damages corneal endothelial cells by producing free radicals.[5] Patients with compromised corneas are therefore particularly vulnerable to the potentially detrimental effects of cataract surgery.

FOCUS NO. 2: CLINICAL EVALUATION

I typically measure central corneal thickness with an ultrasound pachymeter at the first postoperative visit for patients with persistent corneal edema to act as a baseline against which to compare subsequent measurements. Specular microscopy can help verify endothelial cell loss, but I do not find it particularly useful in eyes with significant corneal edema because the measurements are often unreliable.

Patients have been referred to me for persistent postoperative corneal edema that turned out to be Descemet membrane detachments identified with anterior segment OCT, which can be a helpful diagnostic tool especially if the area of edema is localized or well demarcated. Identification of this underlying diagnosis can save a patient from undergoing an unnecessary corneal transplant. The detachments usually resolve with the placement of an air bubble in the anterior chamber and face-up positioning.

FOCUS NO. 3: MEDICAL MANAGEMENT

The medical management of postoperative corneal edema has two primary goals: to eliminate the aggravators of endothelial dysfunction and to treat the corneal edema.

Corticosteroids. When corneal edema occurs immediately postoperatively, inflammation from surgical trauma often contributes to the endothelial dysfunction. In this situation, topical corticosteroids can reduce inflammation and corneal edema. The agents should be tapered once the inflammation subsides. Patients have been referred to me who were treated for corneal edema with high-dose topical corticosteroids for months after cataract surgery. In the presence of a quiet eye, the topical corticosteroid does not provide a direct benefit but can cause undue side effects such as elevated IOP.

Hypertonic saline. Both hypertonic saline drops and ointment can accelerate corneal deturgescence and provide symptomatic relief. Neither medication directly promotes endothelial viability. Hypertonic saline tends to work better in cases of mild edema. Importantly, corneal edema sometimes recurs after hypertonic saline is discontinued. Patients who wish to avoid endothelial keratoplasty should therefore be counseled that long-term hypertonic saline therapy may be required.

Rho kinase inhibitors. The Rho kinase (ROCK) pathway plays a role in regulating endothelial cell migration, proliferation, and adhesion. ROCK inhibitors have been shown to support endothelial wound healing and accelerate corneal deturgescence after surgery.[6] Options for ROCK inhibition include netarsudil ophthalmic solution 0.02% (Rhopressa, Alcon) and ripasudil ophthalmic solution 0.4% (Glanatec, Kowa). Both formulations may improve endothelial cell viability after surgical trauma. I typically prescribe a ROCK inhibitor two to four times daily for 4 to 6 weeks postoperatively or until corneal deturgescence is noted. I titrate therapy based on tolerability (eg, reduced frequency in cases of hyperemia).

Fibroblast growth factors. Trefoil Therapeutics is engineering a variant of fibroblast growth factor to promote endothelial cell proliferation and migration. Early data are promising,[7] but further studies are needed to assess the drug's clinical utility for treating postoperative corneal edema.

FOCUS NO. 4: SURGICAL MANAGEMENT

Descemet stripping only (DSO) has emerged as an option to treat patients with mild FECD and guttae involving the central approximately 4.5 mm of the cornea. A central area of guttae is stripped with a Descemetorhexis technique. Topical ROCK inhibiters are initiated to promote the migration of peripheral endothelial cells onto the denuded area (Figure). Large guttae have been shown to block endothelial monolayer formation, and they are detrimental to endothelial cell survival.[8]

DSO removes guttae in visually significant areas of the cornea to allow healthy endothelial cell migration. The procedure is not effective for the treatment of postoperative secondary corneal edema associated with diffuse endothelial cell loss because there are insufficient peripheral endothelial cells to repopulate the stripped area.

Some patients whose corneal edema has resolved may have symptomatic guttae that cause glare, halos, and decreased contrast sensitivity. DSO may have clinical value for these individuals even if they do not present with frank edema.

If DSO is not an option, either Descemet membrane endothelial keratoplasty or Descemet stripping endothelial keratoplasty may be effective. The former provides more rapid visual rehabilitation. The presence of glaucoma tubes, large iridotomies, or an otherwise unstable anterior chamber, however, may make Descemet membrane endothelial keratoplasty more challenging to perform.

CONCLUSION

FECD and iatrogenic endothelial cell loss predispose patients to persistent corneal edema after cataract surgery. Preoperative counseling that communicates the risk of endothelial decompensation after surgery can help reduce patients' postoperative anxiety and prepare them for the steps necessary to achieve successful outcomes. ■

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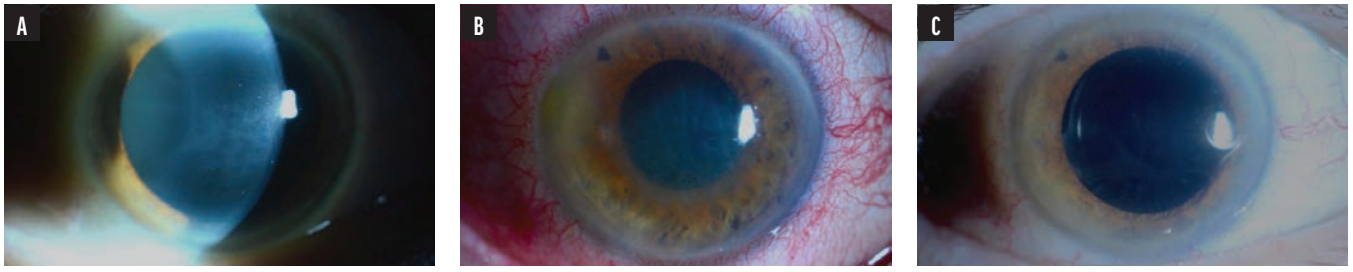

Figure. An eye with FECD and guttae involving the central 4.5 mm of the cornea (A). One day after DSO, a central 4.5-mm area of stromal and epithelial edema was noted (B). By 8 weeks postoperatively, the edema had resolved clinically (C).