Corneal Edema after Cataract Surgery: An Overview

Dr. Rakesh Kumar, S.R.(EYE), S.K.M.C.H, Muzaffarpur
Co-Author: Dr. Kanchan Mala, Doms, Rims, Ranchi

I. Introduction

The art of phacoemulsification surgery has evolved over a period of time and perfecting itself in every aspect from the construction of incisions to the intricacies of intraocular lenses (IOLs). Corneal edema following phacoemulsification is a postoperative complication that may occur in some cases. In the age where cataract surgery is deemed to have vision par perfection, the slightest error can leave a patient in misery. Even immediate blurry vision, however temporary, is accountable for. Postoperative corneal edema, hence, can plague even the most proficient of surgeons.

II. Etiopathogenesis

The light transmissibility of human cornea has been accredited to the lattice arrangement of collagen fibrils, with consequent minimization of light scattering and destructive interference at the stromal level in combination with corneal crystallins; and the relative state of dehydration maintained at the endothelial level by an array of molecular pumps, chemical modulators and cell junctional properties. Among the several myriad complications that might ensue a cataract surgery, corneal edema is frequently encountered. The post-phacoemulsification corneal edema may occur due to endothelial pump failure following surgery, which may be due to mechanical injury, chemical injury, subsequent infection/inflammation, or concurrent/preexisting endothelial compromise.

RISK FACTORS

The risk factors [Table 1] for postoperative phacoemulsification corneal edema include the following:

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Table 1: Risk Factors for Post-cataract Surgery Corneal Edema

CHED: Congenital hereditary endothelial dystrophy, FECD: Fuchs endothelial corneal dystrophy, ICE: Iridoendothelial, PPMD: Posterior polymorphous endothelial dystrophy, IOL: Intraocular lens
Preoperative Risk Factors

- **Preexistent corneal endothelial dystrophies:** In the backdrop of an endothelial dystrophy, the mechanical stress of a complicated surgery can accelerate the cell loss from the endothelial cell layer that may result in late-onset corneal edema following surgery.
- **Iridocorneal endothelial (ICE) syndrome:** Chandler syndrome is often associated with early corneal decompensation with a hammered silver appearance on slit-lamp examination. This endothelial abnormality may often develop postcataract surgery corneal edema.2
- **Glaucoma:** Gagnon et al. observed that the endothelial cell counts were inversely proportional to the intraocular pressure and eyes receiving three or four glaucoma medications had lower cell counts than those receiving one or two medications.3 The magnitude of the cell loss is usually found to correlate with the duration of rise in IOP and combination with corneal guttae leads to corneal decompensation following cataract extraction.4 An eye with shallow anterior chamber (AC) is more likely to encounter multiple intraoperative complications in terms of space for manipulation and recurrent corneolenticular touch.
- **Uveitis:** Chronic antecedent anterior segment inflammations associated with decreased central endothelial cell density, which correlates with the duration of active uveitis, high intraocular pressure during disease and high laser flarephotometry value.5 Moreover, a further insult in the form of surgery may ignite further attacks terminating in acutedecompensation of the cornea.
- **Pseudoexfoliation syndrome (PXF):** PXF may be associated with large clumps of typical pseudoexfoliation material, which may be adhered to the corneal endothelium so that the endothelial layer appears irregular and discontinuous.10 These pathological changes, along with the inherent complications that may possibly occur during the surgery may potentiate acute decompensation in the early as well as late postoperative period.6
- **Trauma:** Endothelial cells around the traumatic lesion undergo the greatest torsion and energy absorption as the cornea moves axially posterior and then relaxes to its original position.7 The injury may also be due to a direct contact between the corneal endothelium and a lens or iris. Dysfunction of damaged endothelial cells is replaced with circumferential normal endothelial cells.

Intraoperative Risk Factors

The various intraoperative factors such as surgeon’s experience, instrument trauma, irrigating solutions, duration of surgery, and complications such as avitrous loss can lead to corneal edema. Phacoemulsification leads to corneal endothelial cell damage by generation of free radicals during surgery. Endothelial cells are attached to each other by discontinuous tight junctions that are mainly calcium-dependent. So, the use of calcium-free irrigating solutions during surgery can reduce the barrier function leading to corneal edema.8

Postoperative Risk Factors

Acute corneal edema immediately after surgery can be because of endothelial damage by ultrasound energy, inadvertent DM stripping that is DMD, due to the infusion of toxic substances into the AC that is the toxic anterior segment syndrome (TASS) and IOL endothelial touch.9

IOL-related factors, which can oftencause corneal decompensation in the postoperative period, include IOL decentration and instability with endothelial touch, dislocated or retained IOL fragments, posterior chamber intraocular lense in AC and unstable anterior chamber intraocular lense.10

Biochemical Alterations in Corneal Edema

The endothelial tight junctions on the lateral membranes function in a delicate balance of the biochemical microenvironment. Both calcium and adenosine are required for the barrier function of the endothelium. Paucity of calcium ion or antioxidant glutathione and adenosine contribute to corneal edema.11 The irrigating solutions or drugs used intraoperatively may bea source of insult to the pump functioning of the endothelium. The differential distribution of molecular channels regulating water influx and efflux called aquaporins (AQP) are implicated in affecting the water transport mechanisms within the cornea. AQP abnormalities have been found in pseudophakic corneal edema (PCE) corneas (decreased AQP1 and increased AQP3 and AQP4) and Fuchs endothelial corneal dystrophy corneas (decreased AQP).10

The altered level of integrins leads to upregulation of inflammatory chemical mediators such as insulin-like growth factor 1, transforming growth factor-β, bone morphogenetic protein-4 (BMP-4), interleukin-1 leading to progressive loss of stromal keratocytes, and formation of a posterior collagenous layer. In these long-standing cases, the corneal epithelium accumulates anti-adhesive proteins with simultaneous loss of adhesive proteins leading to the formation of fluid-filled bullae.10
CLINICAL FEATURES

Symptoms
The patients usually present with diminution of vision in the immediate postoperative period with a lack of expected gain in quality of vision. This may be associated with raised IOP and hence symptoms pertaining to the same may be present.

In addition, pain, photophobia, watering, congestion may be present to variable degree due to corneal edema and associated inflammation. A long-standing corneal edema may be associated in the initial stages with bullae and the rupture of the same may lead to severe photophobia and pain.

Clinical examination
The presence of corneal edema makes the cornea hazy and and there may be the concurrent presence of increased corneal thickness on slit lamp biomicroscopy.

On careful evaluation, if the haziness does not preclude otherwise, a DMD may very well be manifested. A complicated surgery that yielded no particular gain in vision will again be distinguished by the presence of a posterior capsular defect, a poorly stable IOL, with possible vitreous in the AC and possibly multiple sutures.

As the disease reaches chronicity, in addition to the overlap with aforementioned features, can reveal evidence of scarring, microcysts, severe stromal edema with or without scarring bullae [Figs. 1A-C].

DIFFERENTIAL DIAGNOSIS

- **Toxic Anterior Segment Syndrome**: TASS is associated with endothelial failure which heralds diffuse limbus-to-limbus corneal edema, fibrinous AC reaction, iris atrophy, and trabecular meshwork damage.

- **Endophthalmitis**: Corneal edema is often associated with vitreous exudates, loss of red glow, ciliary congestion, and severe AC reaction.

- **Previous episodes of angle closure attacks**: It may reveal iris and pupillary ruff atrophy with goniosynechia and a pigmented trabecular meshwork.

- **Herpetic endotheliitis**: It is associated with KP’s, loss of corneal sensation, localized corneal edema, presence of herpetic footprints/nebulo macular corneal opacity, patchy iris atrophy, and history of recurrent attacks in the past must be looked for.

- **Pseudoexfoliation**: PXF is distinguished by collection of exfoliated material in the angles with iris atrophy and a poorly dilating pupil that may be detected in the fellow eye.

- **Endothelial dystrophies**: Multiple central as well as peripheral guttae in the other eye aiding diagnosis.

INVESTIGATIONS

**Pachymetry**
Optical or ultrasonic measurements of corneal pachymetry estimate the severity of the corneal edema. The corneal thickness can be measured with in vivo imaging using OCT, which shows excellent correlation to values obtained by ultrasound pachymetry.

**Specular microscopy**
Morphometrical analysis can be done preoperatively in suspect patients such as endothelial dystrophies or with history of multiple precedent surgeries.

**Anterior segment optical coherence tomography (ASOCT)**
In eyes with extensive edema that precludes clinical examination in detail, the area of size of DMDs, corneal thickness and levels of scarring can be determined.

**Confocal microscopy**
Confocal microscopy is useful in detecting corneal endothelial status in the presence of corneal edema. It provides high-quality layer-by-layer analysis of the edematous cornea thereby providing a clue towards the probable diagnosis.
Management

Medical management

**Hypertonic agents (sodium chloride 5% eye drops or 6% ointment):** These drugs create a hypertonic tear film that draws water out of the edematous cornea. Hypertonic saline can lead to resolution of corneal edema in almost one-third of patients, especially in early cases, but the treatment may have to be continued for 3 months. Hypertonic saline is useful for symptomatic improvement in all cases of corneal edema. It must be remembered that it does not have any effect on the cause of the disease that must be identified and treated. Hypertonic saline is useful for symptomatic improvement in all cases of corneal edema. It must be remembered that it does not have any effect on the cause of the disease that must be identified and treated.

**Bandaged contact lenses (BCL):** Extended-wear hydrophilic contact lenses are useful in reducing pain associated with epithelial bullae. It must be remembered that it does not lead to any reduction in epithelial or stromal edema. They act by creating an effective precorneal protective layer that shields the swollen epithelium from the lid movement and prevents the rupture of bullae. Hydrophilic extended-wear contact lenses along with hypertonic saline drops can be used to create a hypertonic reservoir. This reservoir continuously bathes the cornea, providing corneal deturgescence for a relatively longer period.

**Antiglaucoma drugs:** Raised IOP can be managed with topical antiglaucoma medications or surgical options such as trabeculectomy with mitomycin C or a glaucoma drainage implant in cases not controlled with topical therapy. Lowering the IOP not only improves the corneal edema but also prevents further damage to the endothelium.

**Topical Steroids:** Associated inflammation must be treated with topical steroids. It is better to avoid using steroids at a higher frequency since associated problems like raise in IOP may further delay clearing of corneal edema. Management of TASS, in the acute phase, includes the intensive use of topical corticosteroids and systemic steroids (in severe cases) along with the use of antiglaucoma drugs. In late phase, development of endothelial decompensation requires keratoplasty.

**Surgical management**

Spontaneous reattachment is commonly seen in planar and nonscrolled DMD’s within days after surgery. It has been reported by Mackool and Holtz that intervention is often required in cases of extensive, central, nonplanar DMD with scrolled or torn edges. Descemetopexy: Sparks first described this procedure in three eyes with extensive DMDs. Three major tamponade agents used are air, 15%–20% SF6 and 12%–14% C3F8. Due to the fast absorption of air, SF6 and C3F8 are the preferred agents. The major complication reported is pupillary block (7.7%), which can be prevented and managed with the use of cycloplegics, prophylactic laser iridotomy, oral and topical antiglaucoma drugs, or a partial fill of AC with air or gas.

![Fig 2: A&B - DMD after cataract surgery, which is shown in ASOCT. C&D- after descemetopexy, DM attached to stroma and corneal edema started decreasing, which is shown in ASOCT.](image)

Thus, the standard treatment option remains descemetopexy. However, other surgical options are viscoelastic injection, suture fixation, and endothelial or penetrating keratoplasty in cases where DMD progress to a stage of corneal decompensation.
The time interval between cataract surgery and DSEK is a critical factor in determining long-term success and a minimum 3–6-month waiting period after cataract is essential for optimal outcomes. Surgery if performed within 3 months was seen to be associated with dismal visual outcome and often are repeat graft is required.

III. Conclusion

Corneal edema following cataract surgery is an untoward but avoidable complication in most of the cases. A careful preoperative workup, intraoperative precautions and vigilant postoperative care can avoid this complication.

References