IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 18, Issue 1 Ser. 19 (January. 2019), PP 60-64
www.iosrjournals.org

Corneal Edema after Cataract Surgery: An Overview

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Date of Submission: 19-01-2019 Date of acceptance: 04-02-2019

I. Introduction

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

II. Etiopathogenesis

The light transmissibility of human cornea has been accreditedto the lattice arrangement of collagen fibrils, with consequentminimization of light scattering and destructive interference atthe stromal level in combination with corneal crystallins; and the relative state of dehydration maintained at the endotheliallevel by an array of molecular pumps, chemical modulators and cell junctional properties.[1]

Among the several myriad complications that might ensuea cataract surgery, corneal edema is frequently encounteredon. The post-phacoemulsification corneal edema may occurdue to endothelial pump failure following surgery, whichmay be due to mechanical injury, chemical injury, subsequentinfection/inflammation, or concurrent/preexisting endothelial compromise.[2]

RISK FACTORS

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

Table1: Risk Factors for Post-cataract Surgery Corneal Edema[10]

Preoperative	Intra-operative	Postoperative
Endothelial dystrophy	Instrument	Vitreous in anterior
CHED	trauma	chamber
FECD, ICE syndrome	Irrigating	IOL - endothelial touch
PPMD	solutions toxicity	Toxic anterior
Pseudoexfoliation	Excessive use	segment syndrome
Hard cataract	of phaco power	Raised intraocular
Chronic uveitis	Intracameral	pressure
Chronic angle-closure	drugs toxicity	Chronic inflammation
glaucoma	DMD	Brown McLean
Trauma to corneal	IOL-related	syndrome
endothelium	factors	Wound leak/shallow
		chamber/hypotony

CHED: Congenital hereditary endothelial dystrophy, FECD: Fuchs endothelial corneal dystrophy, ICE: Iridocorneal endothelial, 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 acomplicated surgery can accelerate the cell loss from theendothelial cell layer that may result in late‐ onset cornealedema following surgery.

• ***Iridocorneal endothelial (ICE) syndrome:*** Chandler syndrome is often associated with early cornealdecompensation with a hammered silver appearance on slitlamp examination. This endothelial abnormality may oftendevelop postcataract surgery corneal edema.[2]

• ***Glaucoma:***Gagnon et al.observed that the endothelial cell counts were inverselyproportional to the intraocular pressure and eyes receivingthree or four glaucoma medications had lower cell countsthan those receiving one or two medications.[3] The magnitude of the cell loss is usually found to correlatewith the duration of rise in IOP and combination withcorneal guttata leads to corneal decompensation followingcataract extraction.[4]An eye with shallow anteriorchamber (AC) is more likely to encounter multipleintraoperative complications in terms of space for manipulation and recurrent corneolenticular touch.

• ***Uveitis:*** Chronic antecedent anterior segment inflammationis associated with decreased central endothelial cell density,which correlates with the duration of active uveitis, highintraocular pressure during disease and high laser flarephotometry value.[5] Moreover, a further insult in the formof surgery may ignite further attacks terminating in acutedecompensation of the cornea.

• ***Pseudoexfoliation syndrome (PXF):*** PXF may be associatedwith large clumps of typical pseudoexfoliation material,which may be adhered to the corneal endotheliumso that the endothelial layer appears irregular anddiscontinuous.[10] These pathological changes, along withthe inherent complications that may possibly occur duringthe surgery may potentiate acute decompensation in theearly as well as late postoperative period.[6]

• ***Trauma:***Endothelial cells aroundthe traumatic lesion undergo the greatest torsion and energyabsorption as the cornea moves axially posterior and thenrelaxes to its original position.[7] The injury may also be due to a direct contact between the corneal endothelium anda lens or iris. Dysfunction of damaged endothelial cells isreplaced with circumferential normal endothelial cells.

Intraoperative Risk Factors

The various intraoperative factorssuch as surgeon's experience, instrument trauma, irrigatingsolutions, duration of surgery, and complications such asvitreous loss can lead to corneal edema. phacoemulsification leads to corneal endothelial cellsdamage by generation of free radicals during surgery. Endothelial cells are attached to eachother by discontinuous tight junctions that are mainlycalcium‐ dependent. So, the use of calcium‐ free irrigatingsolutions during surgery can reduce the barrier function leading to corneal edema.[8]

Postoperative Risk Factors

Acute corneal edema immediately aftersurgery can be because of endothelial damage by ultrasoundenergy, inadvertent DM stripping that is DMD, due to theinfusion of toxic substances into the AC that is the toxicanterior 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 endothelialtouch, dislocated or retained IOL fragments, posteriorchamber intraocular lense in AC and unstable anteriorchamber intraocular lense.[10]

Biochemical Alterations in Corneal Edema

The endothelial tight junctions on the lateral membranes functionin a delicate balance of the biochemical microenvironment. Bothcalcium and adenosine are required for the barrier functionof the endothelium. Paucity of calcium ion or antioxidantglutathione 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 channelsregulating water influx and efflux called aquaporins (AQP)are implicated in affecting the water transport mechanismswithin the cornea. AQP abnormalities have been found inPseudophakic corneal edema (PCE) corneas (decreased AQP1and increased AQP3 and AQP4) and Fuchs endothelial cornealdystrophy corneas (decreased AQP).[10]

The alteredlevel of integrins leads to upregulation of inflammatorychemical mediators such as insulin‐ like growth factor 1,transforming growth factor‐ beta, bone morphogenetic protein4 (BMP‐ 4), interleukin‐ 1 leading to progressive loss of stromalkeratocytes, and formation of a posterior collagenous layer. Inthese long‐ standing cases, the corneal epithelium accumulatesanti‐ adhesive proteins with simultaneous loss of adhesiveproteins 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 gainin quality of vision. This may be associated with raised IOPand hence symptoms pertaining to the same may be present.

In addition, pain, photophobia, watering, congestion may bepresent to variable degree due to corneal edema and associatedinflammation.A long- standing corneal edema may be associated in theinitial stages with bullae and the rupture of the same may leadto severe photophobia and pain.

Clinical examination

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

On careful evaluation, if the haziness does not preclude itotherwise, a DMD may very well be manifested. A complicatedsurgery that yielded no particular gain in vision will again be distinguished by the presence of a posterior capsular defector a poorly stable IOL, with possible vitreous in the AC andpossibly 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].

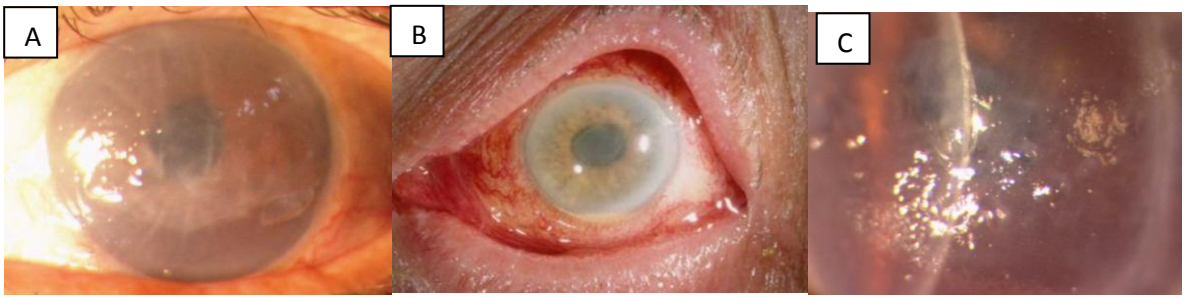

FIG 1: A- Corneal oedema in DMD; B- Limbus –to limbus corneal edema in TAAS, C- Cornea epithelial bullae formation.

DIFFERENTIAL DIAGNOSIS

- ***Toxic Anterior Segment Syndrome*:**TASS is associated with endothelialfailure which heralds diffuse limbus- to- limbus cornealedema, fibrinous AC reaction, iris atrophy, and trabecular meshwork damage.
- **Endophthalmtis:** corneal edema is often associated withvitreous exudates, loss of red glow, ciliary congestion, andsevere AC reaction.
- ***Previous episodes of angle closure attacks:***It may reveal iris and pupillary ruff atrophy with goniosynechiaeand a pigmented trabecular meshwork.
- ***Herpetic endotheliitis***: It is associated with KP's, loss of corneal sensation, localizedcorneal edema, presence of herpetic footprints/nebulo macularcorneal opacity, patchy iris atrophy, and history of recurrentattacks in the past must be looked for.
- ***Pseudoexfoliation***: PXF is distinguished by collection of exfoliated material in the angles with irisatrophy and a poorly dilating pupil that may be detected inthe fellow eye.
- ***Endothelial dystrophies***: multiple central as well as peripheral guttae in the other eyeaiding diagnosis.[10]

INVESTIGATIONS

Pachymetry

Optical or ultrasonic measurements of corneal pachymetryestimate the severity of the corneal edema. Thecorneal thickness can be measured with in vivo imaging usingOCT, which shows excellent correlation to values obtained byultrasound pachymetry.[12]

Specular microscopy

Morphometrical analysis can bedone preoperatively in suspect patients such as endothelialdystrophies or with history of multiple precedent surgeries.

Anterior segment optical coherence tomography (ASOCT)

In eyes withextensive edema that precludes clinical examination in detail,the areas and size of DMDs, corneal thickness and levels ofscarring can be determined.[13]

Confocal microscopy

Confocal microscopy is useful in detecting corneal endothelialstatus in the presence of corneal edema. It provides high- qualitylayer- by- layer analysis of the edematous cornea therebyproviding a clue towards the probable diagnosis.

Management

Medical management

Hypertonic agents (sodium chloride 5% eye drops or 6% ointment): Thesedrugs create a hypertonic tear film that draws water outof the edematous cornea. Hypertonic saline can lead to resolution of cornealedema in almost one- third of patients, especially in early cases,but the treatment may have to be continued for 3 months.[14] Hypertonic saline is useful forsymptomatic improvement in all cases of corneal edema. Itmust be remembered that it does not have any effect on thecause of the disease that must be identified and treated.[10]

Bandaged contact lenses (BCL) : Extended- wearhydrophilic contact lenses are useful in reducing painassociated with epithelial bullae. It must be remembered that it does notlead to any reduction in epithelial or stromal edema. They actby creating an effective precorneal protective layer that shieldsthe swollen epithelium from the lid movement and preventsthe rupture of bullae.[14]

Hydrophilic extended- wear contact lenses along withhypertonic saline drops can be used to create a hypertonicreservoir. This reservoir continuously bathes the cornea,providing corneal deturgescence for a relatively longerperiod.

Antiglaucoma drugs: Raised IOP can be managed with topical antiglaucomamedications or surgical options such as trabeculectomy withmitomycin C or a glaucoma drainage implant in cases notcontrolled with topical therapy. Lowering the IOP not onlyimproves the corneal edema but also prevents further damageto endothelium.

Topical Steroids:Associated inflammation must be treated with topicalsteroids. It is better to avoid using steroids at a higher frequencysince associated problems like raise in IOP may further delayclearing of corneal edema.

Management of TASS, in the acute phase, includes theintensive use of topical corticosteroids and systemic steroids(in severe cases) along with the use of antiglaucoma drugs.In late phase, development of endothelial decompensationrequires keratoplasty.

Surgical management

Spontaneous reattachment is commonly seen in planar andnonscrolled DMD's within days after surgery.[14]It has beenreported by Mackool and Holtz that intervention is oftenrequired in cases of extensive, central, nonplanar DMD withscrolled or torn edges.[15]

Descemetopexy: Sparks first described this procedure in three eyes with extensive DMDs.[16] Three major tamponade agents used are air, 15%–20% SF6 and12%–14% C3F8. Due to the fast absorption of air, SF6and C3F8 are the preferred agents [Fig 2].

The major complication reported is pupillary block (7.7%),which can be prevented and managed with the use ofcycloplegics, prophylactic laser iridotomy, oral and topicalantiglaucoma drugs, or a partial fill of AC with air or gas.

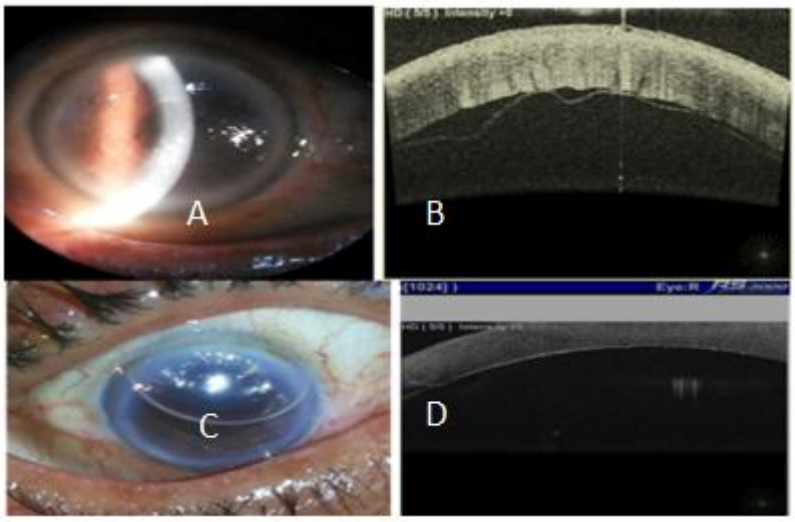

Fig 2: A&B- DMD after cataract surgery, which is shown in ASOCT. C&D- after descemetopexy, DM attached to stroma and corneal edema started decreasing, which is shown in ASOCT.

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 cornealdecompensation.[10]

The time interval between cataract surgery and DSEK is a critical factor in determining long- term success and aminimum 3–6- month waiting period after cataract is essential foroptimal outcomes. Surgery if performed within 3 months wasseen to be associated with dismal visual outcome and often arepeat graft is required.[17]

III. Conclusion

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

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Dr. Rakesh Kumar. "Corneal Edema after Cataract Surgery: An Overview." IOSR Journal of Dental and Medical Sciences (IOSR-JDMS), vol. 18, no. 1, 2019, pp 60-64.