Review Article

Corneal edema after phacoemulsification

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Phacoemulsification is the most commonly performed cataract surgery in this era. With all the recent advances in investigations and management of cataract through phacoemulsification, most of the patients are able to achieve excellent visual outcome. Corneal edema after phacoemulsification in the immediate postoperative period often leads to patient dissatisfaction and worsening of outcome. Delayed onset corneal edema often warrants endothelial keratoplasty. This review highlights the etiopathogenesis, risk factors, and management of corneal edema in the acute phase including descemet's membrane detachment (DMD) and toxic anterior segment syndrome. Various investigative modalities such as pachymetry, specular microscopy, anterior segment optical coherence tomography, and confocal microscopy have been discussed briefly.

Key words: Corneal edema, descemet membrane detachment, phacoemulsification, toxin anterior segment syndrome

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The art of phacoemulsification surgery has evolved over aeon 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. Many a mishaps that happen in the course of surgery can lead to multiple postoperative complications. In the age where cataract surgery is deemed to have vision par perfection, the slightest error can leave a patient in misery. Even immediate incoherent vision, however temporary, is accountable for. Postoperative corneal edema, hence, can plague even the most proficient of surgeons.

Method of Literature Search

A Medline search was carried out for articles in the English language, with the keywords, corneal edema; cataract surgery; phacoemulsification; bullous keratopathy; endothelial disease, endothelial dystrophy, endothelial dysfunction, keratoplasty, endothelial keratoplasty, descemet's stripping automated endothelial keratoplasty (DSAEK), and descemet's membrane (DM) endothelial keratoplasty. All relevant articles were included in this review. Priority was given to prospective studies and randomized clinical trials. However, retrospective studies and case reports were included if important.

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.[1,2]

Among the several myriad complications that might ensue a cataract surgery, corneal edema is frequently encountered on. 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.[3]

Risk factors

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

- 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. Overall, Fuchs endothelial corneal dystrophy accounts for about 10.8%–23.8% of all penetrating keratoplasty done[4]
- Iridocorneal endothelial (ICE) syndrome is an endothelial disorder characterized by abnormal endothelial cell formation and formation of a proliferating membrane over the angle and iris. Three clinical forms of ICE syndrome described are iris nodules (iris nevus and Cogan-Reese syndromes), corneal endothelial abnormality (Chandler

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Table 1: Risk factors for postcataract surgery corneal edema

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

CHED: Congenital hereditary endothelial dystrophy, FECD: Fuchs endothelial corneal dystrophy, ICE: Iridocorneal endothelial, PPMD: Posterior polymorphous endothelial dystrophy, IOL: Intraocular lens

syndrome), and essential (progressive) iris atrophy. 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[1,3]

- Glaucoma: Concurrent glaucoma may be associated with significantly lower endothelial cell counts. 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.[5,6] In eyes that experienced an acute attack of angle closure, mean endothelial cells count was 2,106 cells/mm² if the attack lasted <72 h and 759 cells/mm² if the attack lasted >72 h.[7] The magnitude of the cell loss is usually found to correlate with the duration of rise in IOP and combination with corneal guttata leads to corneal decompensation following cataract extraction.[8] Further, 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 inflammation is associated with decreased central endothelial cell density, which correlates with the duration of active uveitis, high intraocular pressure during disease and high laser flare photometry value.[9] Moreover, a further insult in the form of surgery may ignite further attacks terminating in acute decompensation 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
- Trauma: An erstwhile history of trauma may again point to an endotheial insult and cell paucity that may exacerbate following a subsequent surgery.[11] 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.[12] 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 vitreous loss can lead to corneal edema. Studies have shown that phacoemulsification leads to corneal endothelial cells damage by generation of free radicals during surgery. Several studies have emphasized the protective role of various free radical scavengers such as reduced glutathione, calcium, adenosine, sodium hyaluronate, superoxide dismutase, and ascorbic acid. 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. Thus, several authors have recommended the use of irrigating solutions with the above substances such as BSS plus and viscoelastics containing sodium hyaluronate to reduce the chances of postoperative corneal edema.[13] 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.

DM is the basement membrane of the corneal endothelium, which is essential for the maintenance of corneal transparency. DMD after cataract extraction was first described by Scheie as a vision-threatening complication.[14] The various risk factors of DMD can be categorized into pre-, intra-, and post-operative. Preoperative causes include preexisting endothelial dysfunction, age >65 years and dense cataract. Intraoperative risk factors include the use of blunt instruments, inadvertent insertion of instruments between the corneal stroma and DM, inappropriate incisions, tight main incision, damage of DM during irrigation/aspiration, IOL or phaco probe insertion, and surgeon inexperience.[15]

DMD can be detected on slit-lamp biomicroscopy in cases with clear cornea however, in cases with severe corneal edema, anterior segment optical coherence tomography (ASOCT) remains the best imaging tool to conclusively diagnose its occurrence.[15,16] Various classification systems have been proposed for DMD such as planar (separation between the stroma and DM is <1 mm) versus nonplanar (>1 mm), scrolled versus nonscrolled, and peripheral versus peripheral with central involvement. DMDs that exhibit a rolled edge are described as scrolled, and those without a rolled edge are described as nonscrolled.[15,16] However, with the introduction of ASOCT in the evaluation of DMD, Jacob *et al*.[17] proposed a classification according to etiology, clinical features, ASOCT, intraoperative features, and management protocol. They classified DMDs into rhegmatogenous, tractional, bullous, and complex types. Rhegmatogenous DMDs are secondary to tear, hole, or dialysis of DM at Schwalbe's line. Tractional DMDs are due to traction of DM secondary to inflammation, fibrosis, or incarceration in any of several locations: graft–host junction, peripheral anterior synechiae, or suture with subsequent contraction. Bullous DMDs present as a smooth bulge of DM into the AC in the absence of any DM break or with a small needle puncture. Complex DMDs include DM macrofolds, rolls, or scrolled edges as well as combinations of other variants of DMD.

TASS is one of the important causes of postcataract surgery corneal edema. It is important to differentiate it from infectious endophthalmitis. TASS is characterized by sterile postoperative anterior segment inflammation within 12–48 h of surgery. It is commonly observed after uneventful anterior segment surgeries, specifically after cataract surgery and is associated with minimal pain, diffuse limbus-to-limbus corneal edema, fibrinous AC reaction, iris atrophy, trabecular meshwork damage but the absence of vitritis.[18]

TASS is quite rare with an incidence of 0.22%– 0.80% described in literature. Severe cases are often prone to develop endothelial failure and secondary glaucoma.[19,20] Endothelial decompensation requiring keratoplasty has been reported in 0%–83.3% cases and secondary glaucoma has been reported in 0%–33.3% cases of TASS in different case series.[19-23]

IOL-related factors: Various factors, which can often cause 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. Few authors have reported corneal decompensation due to dislocated IOL fragments, retained fragments after IOL exchange, and damage of the IOL during implantation.[24-26] Capitena *et al*. reported a case of delayed presentation of a severed acrylic IOL haptic fragment causing corneal edema after uneventful surgery. A distal haptic fragment was seen in the inferior AC angle which originated from the temporal haptic and had a clean, flat surface, suggesting it was severed by a sharp object due to improper implantation.[27] These cases are often managed in two stages. First stage includes the explanation of the unstable IOL or retained IOL fragments with or without IOL implantation in sulcus if adequate or intrascleral haptic fixation. In the second stage, endothelial keratoplasty can be planned for decompensation.

Various risk factors that have been described in the etiopathogenesis of TASS include contaminants in intraocular irrigating solutions, intraocular medications and ointments, preservatives in ophthalmic solutions, poor technique in sterilization of instruments and tubing, retained viscoelastic in instruments, etc.[19-23,28,29]

Morphological Changes

Careful examination may reveal a DMD that has stripped off the endothelium from the posterior corneal surface with a surrounding localized or generalized corneal edema depending on the size of the DMD. Small incidental DMDs, near the surgical wound may be as high as 47%, however, those of clinical significance vary between 0.044% and 0.5% in phacoemulsification.[30,31] The risk factors predisposing a patient to develop an intraoperative DMD are shallow AC, suboptimal quality of the operating instruments, hard nuclear cataracts, misdirected stream of fluid, or viscoelastic that gains entry into the space between the stroma and DM.[14] Fashioning an anterior or shelved incision for AC entry can also add to the risk of creating a DMD.[32] An abnormal tenuous attachment of the DM to the posterior corneal stroma has been postulated in those patients who have been reported to have bilateral DMD following intracapsular cataract extraction or uncomplicated extracapsular cataract extraction surgery. However, it must be taken into account that the endothelial sheet on the detached DM can maintain glycolysis in the endothelium and even late management of the DM may salvage the cornea from irreversible edema.[33] The amount of ultrasonic energy required for emulsification is strongly dependent on the density of the cataract, despite that fact the consumed energy is not the determining factor for the corresponding severity of corneal edema, which is more a function of the endothelial cell viability.[34,35]

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.[36,37] The irrigating solutions or drugs used intraoperatively may be a 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).

The aforementioned mechanisms in addition to the altered level of integrins leads to upregulation of inflammatory chemical mediators such as insulin-like growth factor 1, transforming growth factor-beta, 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.

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

A thorough examination can reveal the extent and severity of corneal involvement as well as point toward the etiology of the same. The presence of corneal edema makes the cornea lusterless and hazy and there may be the concurrent presence of increased corneal thickness on slit lamp biomicroscopy.

The incidence of unobvious DMD in a seemingly uncomplicated cataract surgery is approximately 47%.[16,30] On careful evaluation, if the haziness does not preclude it 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 or 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. 1-5].

Differential diagnosis

The occurrence of TASS is associated with endothelial failure which heralds diffuse limbus-to-limbus corneal edema, fibrinous AC reaction, iris atrophy, and trabecular meshwork damage.[23] The same has to be differentiated from *endophthalmtis* where corneal edema is often associated with vitreous exudates, loss of red glow, ciliary congestion, and severe AC reaction. Previous episodes of angle closure attacks may reveal iris and pupillary ruff atrophy with goniosynechiae and a pigmented trabecular meshwork. Signs of *herpetic endotheilitis* such as 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. 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 in particular will reveal 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. Indirectly, the surgical planning will vary in accordance with the amount of edema that has to be resolved by the endothelial graft. The corneal thickness can be measured with *in vivo* imaging using OCT, which shows excellent correlation to values obtained by ultrasound pachymetry.[38]

Specular microscopy

The normal healthy endothelial cell sheet is a quasiregular array of hexagonal cells. Morphometrical analysis can be done preoperatively in suspect patients such as endothelial dystrophies or with history of multiple precedent surgeries. The mean cell area and mean cell density are increased and

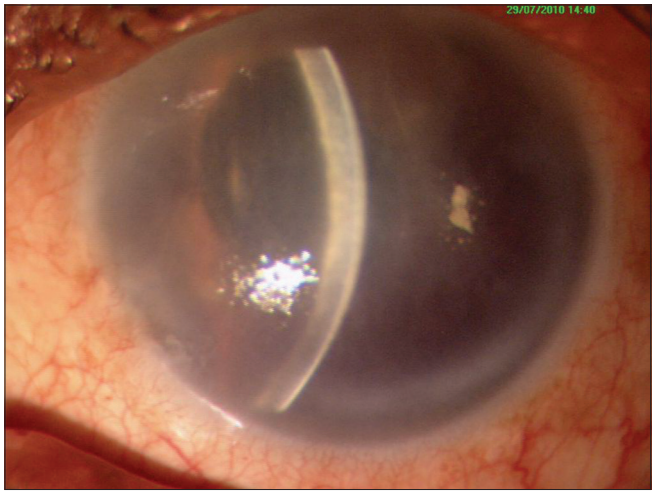

Figure 1: Corneal edema with increased corneal thickness

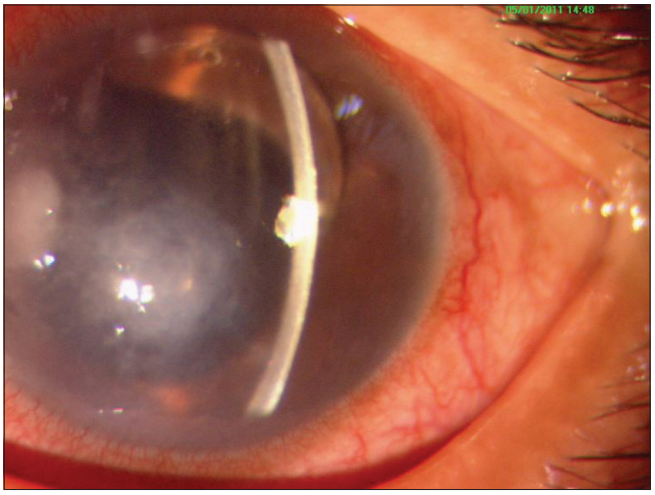

Figure 2: Localized Corneal Edema with intraocular lense in anterior chamber

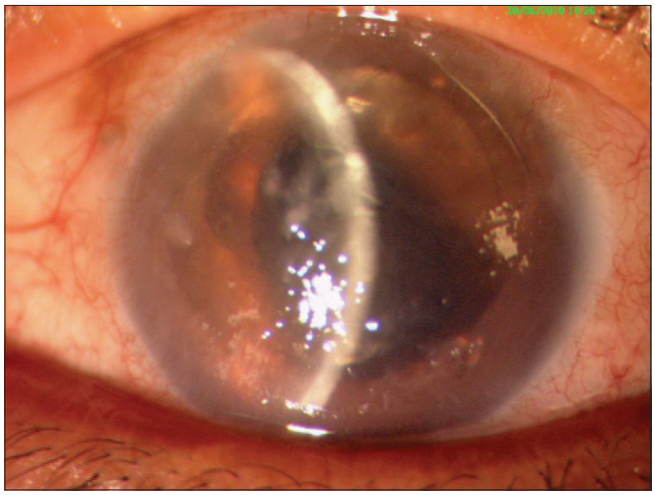

Figure 3: Anterior chamber intraocular lense with Corneal edema

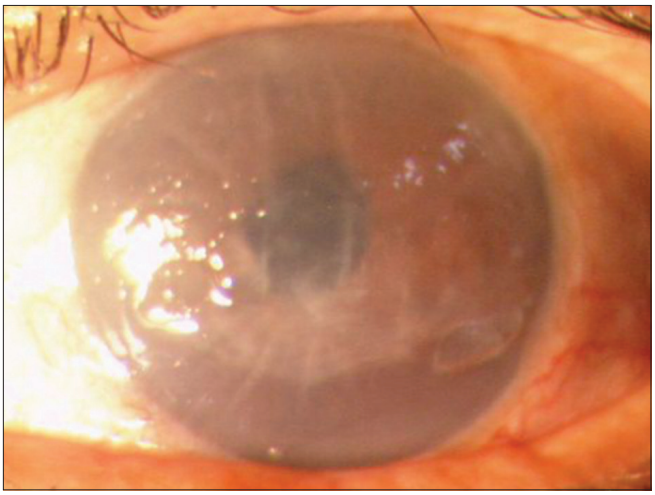

Figure 4: Corneal edema with descemet's membrane detachment

decreased respectively in endothelial loss. Quantitative indices such as coefficient of variation, percentages of hexagonality and mean cell area with standard deviation are abnormal depending on the extent of endothelial damage incurred and the duration of corneal edema.

Anterior segment optical coherence tomography

Optical coherence tomography (OCT) is a noncontact technology that produces high-resolution cross-sectional images of ocular tissues. ASOCT enables the precise visualization of anterior segment structure; thus, it can be used in various corneal and ocular surface disorders. Incision architecture and pachymetry at the wound level can be measured by ASOCT. In eyes with extensive edema that precludes clinical examination in detail, the areas and size of DMDs, corneal thickness and levels of scarring can be determined[16,39] [Fig. 6].

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

The management of corneal edema following phacoemulsification surgery depends upon the underlying cause and visual potential. For treatment purpose, it is better to classify it into categories as described in Table 2.

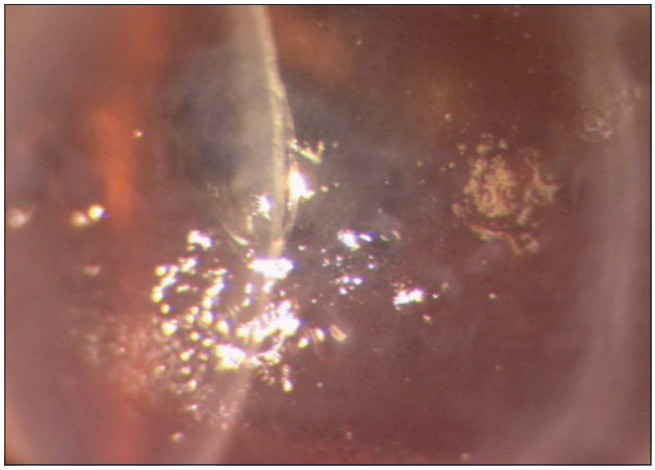

Figure 5: Corneal epithelial bullae formation

Medical management

Medical management includes the use of hypertonic agents, such as sodium chloride 5% eye drops or 6% ointment. These drugs create a hypertonic tear film that draws water out of the edematous cornea. In author's experience, ointment formulation is more effective than the solution form. Applying ointment at night may significantly reduce the early morning symptoms. Hypertonic agents may lead to irritation in some cases, and they may not be that effective in reducing the stromal edema. 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.[40] Knezović *et al*.[41] in a prospective comparative trial including 70 eyes of bullous keratopathy reported the outcomes of hypertonic saline 5% in early and late cases of corneal edema and they found that hypertonic saline 5% was significantly useful in the initial stages only (stromal edema with 613–694 μm [in the central corneal area] and 633–728 μm [at corneal periphery]) of bullous keratopathy for improving visual acuity and reducing the corneal thickness, but was not useful in the advanced disease (epithelial with stromal edema).[41] However, in our experience 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), especially extended-wear hydrophilic contact lenses are useful in reducing pain associated with epithelial bullae.[40] A thin, high water content lens is suitable in these cases as the oxygen permeability is better in these lenses. 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.[42,43]

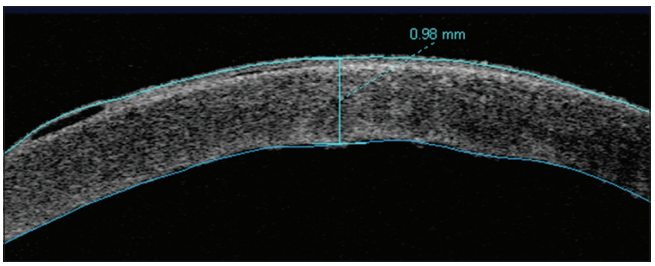

Figure 6: Anterior segment optical coherence tomography showing increased thickness with bullae formation

Table 2: Types of corneal edema and its management approach following cataract surgery

Onset	Description	Underlying cause	Examples	Management
Immediate	Edema present since day one of the surgery	Endothelial trauma with functional failure	Excessive phaco energy Long duration surgery Raised IOP	Medical management If no improvement endothelial transplant
		Structural lesions	DMD Endothelial/DM tear due to instrument trauma	Medical management with specific surgery such as intracameral C3F8
Delayed onset	Edema develops after a period of corneal clarity following surgery	Endothelial cell loss	ABK PBK	Endothelial transplant or symptomatic therapy in cases of poor visual function

DMD: Descemet's membrane detachment, DM: Descemet's membrane, ABK: Aphakic bullous keratopathy, PBK: Pseudophakic bullous keratopathy, IOP: Intraocular pressure

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. BCL is especially useful in cases where the patient is symptomatic owing to rupture of bullae.[40] Considering the potential risk of infection, prolonged use of BCL is discouraged, and a broad-spectrum antibiotic must be used along with these lenses.[40]

One important thing to remember is that associated abnormalities such as raised IOP or inflammation should be looked for and managed simultaneously.

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 endothelium. Although any class of anti-glaucoma can be useful two groups of drugs, carbonic anhydrase inhibitors and Prostaglandin analogues (PGA) must be avoided.[44] Inhibition of corneal endothelial carbonic anhydrase pumps can lead to decreased fluid flow from stroma to aqueous, resulting in precipitation of corneal edema. PGA, theoretically, can flare up the intraocular inflammation.[44]

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.[45]

Surgical management

The presence of DMD in the immediate postoperative phase requires surgical intervention in the form of intracameral air or gas injection.[16] Most of the other surgical options such as anterior stromal puncture, amniotic membrane, conjunctival flap, phototherapeutic keratectomy, corneal collagen cross-linking, endothelial keratoplasty [Fig. 7], and penetrating keratoplasty are reserved for delayed onset or persistent corneal edema due to irreversible corneal endothelial damage.[41]

Spontaneous reattachment is commonly seen in planar and nonscrolled DMD's within days after surgery.[14] It has been reported by Mackool and Holtz[46] that intervention is often required in cases of extensive, central, nonplanar DMD with scrolled or torn edges. Assia *et al*.[47] suggested that nonplanar and nonscrolled DMDs could reattach even if the separation between the DM and stroma was >1 mm.

Kumar *et al*.[48] proposed HELP (height-, extent-, length-, and pupil-based) treatment protocol for DMD. DMD <1.0 mm long and <100 μm high in any zone, medical management was considered. DMD 1–2 mm in length and 100–300 μm high in zone 2 (paracentral 5–8 mm) and 3 (periphery >8 mm) were managed medically. DMD >2 mm in length and >300 μm high in zone 1 (central 5 mm) and 2 (paracentral 5–8 mm) were managed surgically and in zone 3 (periphery >8 mm) were managed medically. Surgical intervention included intracameral gas (14% perfluoropropane [C3F8] or 20% sulfur hexafluoride [SF6]) or sterile air injection. The authors compared group 1 (managed surgically, *n*=96) with group 2 (managed medically, *n*=65). Group 1 (eyes managed medically) was compared with group 2 (managed surgically). The complete reattachment rate was 95.8% in Group 1 and 96.9% in Group 2. The corrected distance visual acuity was 20/40 or better in 83.3% of eyes in Group 1 and 92.3% of eyes in Group 2. Various options for management of DMD include observation, topical treatment with steroids and hyperosmotic agents, intracameral air or expandable gases injection descemetopexy, viscoelastic injection, transcorneal suturing, endothelial keratoplasty, and conventional penetrating keratoplasty.[15]

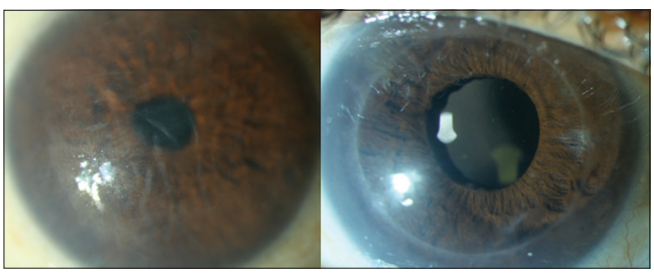

Figure 7: A case of pseudophakic corneal edema managed with Descemet's stripping automated endothelial keratoplasty

Descemetopexy

Sparks[49] first described this procedure in three eyes with extensive DMDs. Several studies have reported successful anatomical and visual outcomes with this procedure.[50-54] Three major tamponade agents used are air, 15%–20% SF6 and 12%–14% C3F8 [Table 3]. Due to the fast absorption of air, SF6 and C3F8 are the preferred agents.[55-57]

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.[48,56]

Another concern with the use of long-acting gases is the corneal endothelial toxicity, which has been reported, in animal studies.[58-62]

The authors have described an algorithm to decide how to approach a case of DMD using ASOCT in cases of nonresolving postoperative corneal edema. DMDs that were in the superior half of the cornea with a planar configuration alone were managed using intracameral air, and those with scrolled edges were managed using intracameral 14% perfluoropropane (C3F8). DMDs that were in the inferior half of the cornea with planar or scrolled edges were managed using intracameral C3F8 injection. The mean time of resolution with this approach, with intracameral air or 14% C3F8 gas, has been observed to be 16 ± 7.1 days.[16]

Thus, the standard treatment option remains descemetopexy [Fig. 8]. However, other surgical options are viscoelastic injection,[63] suture fixation, and endothelial or penetrating keratoplasty in cases where DMD progress to a stage of corneal decompensation.

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. The most commonly described is the penetrating keratoplasty which is the last resort in cases with corneal opacification, however, failure of these grafts is quite

Table 3: Role of descemetopexy in post-phacoemulsification descemet's membrane detachment

Author	Type of study	Sample size	Method	Results
Sparks, 1967[49]	Case series, outcomes of descemetopexy in extensive DMDs	3 eyes	Withdrawing aqueous and injecting air into the anterior chamber of three eyes with straightening of the membrane with a cyclodialysis spatula in two of the eyes with extensive DMDs	Successful outcome in two out of three eyes
Wylegała and Nowińska, 2009[51]	Retrospective	14 eyes	Eight eyes treated conservatively and 6 eyes underwent intracameral air injection with additional ab externo stab incisions. In 12 eyes, DM reattached successfully	In 12/14 eyes, DM reattached successfully
Huang *et al*., 2016[52]	Case report	3 cases	ASOCT-guided aqueous fluid drainage and intracameral air injection used	Complete attachment with clear cornea noted in 2 cases
Chaurasia *et al*., 2012[54]	Interventional case series	14 cases	Intracameral (100%) air injection	Successful reattachment of DM and resolution of corneal edema occurred in all except 1 patient
Ti *et al*., 2013[50]	Retrospective analysis	16 cases	Air was injected into the anterior chamber until full air fill was obtained. Tamponade was maintained for 8-10 min, with concurrent massage of the corneal surface with Weck-Cel sponge from an area of greatest loculation toward the tear, followed by air fluid exchange to retain approximately two-thirds air fill	AB tamponade for DMD effectively restored corneal clarity in 87.5% of cases (14 of 16 eyes). Significant risk factors included endothelial disease and first postoperative day corneal edema
Kumar *et al*., 2015[48]	Prospective comparative case series	161 eyes	HELP treatment protocol used Surgical intervention included intracameral gas (14% perfluoropropane [C3F8] or 20% SF6) or sterile air injection under peribulbar anesthesia	The complete reattachment rate was 95.8% in Group 1 (managed medically) and 96.9% in Group 2 (managed surgically). The CDVA was 20/40 or better in 83.3% of eyes in Group 1 and 92.3% of eyes in Group 2

DMDs: Descemet's membrane detachments, DM: Descemet's membrane, HELP: Height-, extent-, length-, and pupil-based, AB: Air bubble, CDVA: Corrected distance visual acuity, SF6: Sulfur hexafluoride, ASOCT: Anterior segment optical coherence tomography

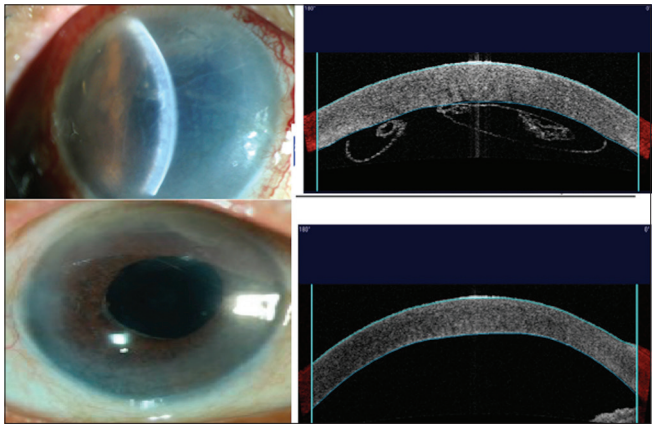

Figure 8: A case of descemet's membrane detachment managed with descemetopexy with C3F8

common due to the persistent AC inflammation and fibrous membranes.[19-23,28]

Endothelial keratoplasty has been reported to have good visual and anatomical outcomes in cases of post-TASS corneal decompensation.[48,49,64,65] Kaur *et al*.[66] evaluated the visual and anatomical outcomes of DSAEK in patients with TASS. They showed that the time interval between TASS and DSAEK is a critical factor in determining long-term success and a minimum 3–6-month waiting period after TASS is essential for optimal outcomes. Surgery if performed within 3 months was seen to be associated with dismal visual outcome and often a repeat graft is required. This is due to various intraoperative problems such as friable DM leading to difficult scoring, and a floppy iris with extensive pigment release. In addition, an intense postoperative inflammation associated with secondary glaucoma (57.1%) further contributes to failure of keratoplasty.[66]

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 and save the operating surgeon from patients ire as well as medicolegal implications.

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Conflicts of interest

There are no conflicts of interest.

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