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Glaucoma management after corneal transplantation surgeries

Helen L. Kornmann and **Steven J. Gedde**

Bascom Palmer Eye Institute, University of Miami, Miller School of Medicine, Miami, Florida, USA

Abstract

Purpose of review—Intraocular pressure (IOP) elevation and glaucoma progression following corneal transplantation, specifically, penetrating keratoplasty, Descemet's stripping endothelial keratoplasty, and Boston keratoprosthesis, are well described causes of ocular morbidity. Depending on the procedure performed, the incidence of glaucoma is highly variable. Several etiologic factors have been identified, the most common being synechial angle closure and corticosteroid-induced IOP elevation. The purpose of this review is to describe the various treatment strategies for glaucoma following corneal transplantation.

Recent findings—Medications and laser treatments are usually first-line therapies for postoperative IOP elevation. Surgical intervention, including filtering surgery and glaucoma drainage devices, may be necessary to control IOP and prevent progressive glaucomatous damage.

Summary—Glaucoma is a common complication of corneal transplantation, and the degree of aggressiveness is often related to the indication for corneal surgery. Although postoperative IOP elevation may be controlled with medical therapy alone, refractory cases may require glaucoma surgery. In all cases, early detection and intervention are necessary to optimize patient outcomes.

Keywords

corneal transplant; glaucoma; medical and surgical management

INTRODUCTION

Glaucoma is a well known complication following various corneal transplantation surgeries. Diagnosis can be difficult, particularly when accurate intraocular pressure (IOP) measurements cannot be obtained and assessments of the optic nerve and/or visual field testing are not possible. Multiple studies have documented that preoperative glaucoma is a major risk factor for the development of postkeratoplasty pressure elevation [1–4]. Patients with peripheral anterior synechiae preoperatively are also at a higher risk of developing glaucoma after penetrating keratoplasty [5], suggesting that preexisting angle distortion and compression could contribute to elevated IOP after corneal transplant [6]. Uncontrolled IOP is also an important risk factor for endothelial cell loss, and postkeratoplasty glaucoma represents the second leading cause of graft failure after rejection [7–10]. Detecting

Correspondence to Steven J. Gedde, MD, Bascom Palmer Eye Institute, University of Miami, Miller School of Medicine, 900 NW 17th Street, Miami, FL 33136, USA. Tel: +1 305 326 6435; fax: +1 305 326 6478; sgedde@med.miami.edu.

Conflicts of interest

There are no conflicts of interest.

glaucoma early in these patients can lead to fewer complications and improved patient outcomes.

GLAUCOMA AFTER PENETRATING KERATOPLASTY

Incidence, risk factors, and pathogenesis

The incidence of glaucoma after penetrating keratoplasty reportedly ranges from 5.5 to 31% in the early postoperative period [1,11–13] and from 17 to 35% in the late postoperative period [1,5,6,14]. The presence of preoperative glaucoma is the strongest risk factor for the development of postkeratoplasty glaucoma [1,2,5]. However, the indication for corneal transplant also affects the incidence of post-penetrating keratoplasty glaucoma, which occurs more frequently in eyes with aphakic bullous keratopathy and less frequently in Fuchs' corneal dystrophy and keratoconus [2,4,11,14]. Keratoplasties combined with other surgical procedures have also been associated with the need for surgical glaucoma intervention [15].

The leading causes of glaucoma after penetrating keratoplasty are synechial angle closure, with the degree of closure strongly correlated with subsequent glaucoma surgery [16], and corticosteroid-induced elevation in IOP [4,11,17]. Other factors, include iridocorneal angle collapse, postoperative inflammation, iatrogenic injury to the angle, and vitreous prolapse into the angle [6,11,18]. The cause of corneal disease also affects glaucoma progression, with penetrating keratoplasty performed for Fuchs' corneal dystrophy and keratoconus associated with a lower risk of progression than those indicated for herpetic infection, corneal ulcers, or corneal perforation [11,19,20].

Intraocular pressure measurement after penetrating keratoplasty

Following penetrating keratoplasty, surface irregularities, scarring, and increased corneal thickness often prevent precise IOP measurements [21]. Early in the postoperative period, IOP can be measured by the Mackay-Marg electronic applanation tonometer, pneumatic applanation tonometer, Tono-Pen, or dynamic contour tonometer (DCT), as these instruments measure IOP independent of corneal thickness within certain ranges of IOP [22]. If the corneal graft surface is smooth with an intact epithelium and regular mires can be obtained, Goldmann applanation tonometry (GAT) can be used. The iCare, a handheld rebound tonometer, has shown good correlation with GAT and other tonometers in healthy and glaucomatous eyes [23–26] but significantly underestimates GAT IOP in postpenetrating keratoplasty eyes [27].

Medical therapy

Most eyes that develop postpenetrating keratoplasty glaucoma respond positively to medical therapy [5,13,14,28], and it remains the first-line treatment for elevated IOP after penetrating keratoplasty. The effect of topical glaucoma therapy on graft survival remains unclear. Some studies suggest that pilocarpine, topical carbonic anhydrase inhibitors, prostaglandin analogs, and adrenergic agents cause higher rates of graft failure [29], whereas other studies report no difference with respect to medication used [5].

In cases of corticosteroid-responsive glaucoma, the dose of steroids may be tapered to the minimum required. Alternatively, potent IOP elevating agents, such as prednisolone or dexamethasone, can be replaced by corticosteroids that have a decreased tendency to increase IOP, such as fluorometholone, loteprednol, and rimexolone [30].

Laser therapy

In some instances, laser trabeculoplasty may be beneficial for treating elevated IOP after penetrating keratoplasty. Van Meter *et al.* [31] reported a 29.7% mean reduction in IOP 2 years after argon laser trabeculoplasty after penetrating keratoplasty, and Nakakura *et al.* [32] reported a case of selective laser trabeculoplasty after penetrating keratoplasty resulting in an IOP of 18 mmHg 6 months after surgery with adjunctive use of latanoprost (0.005%). However, the long-term effects of laser trabeculoplasty tend to diminish over time, and may therefore have limited utility.

Cyclodestructive procedures

Cyclodestructive procedures used to control IOP after penetrating keratoplasty, include cyclocryotherapy, Nd:YAG laser cyclophotocoagulation (CPC), diode laser CPC, transpupillary argon laser photocoagulation, and endoscopic CPC. CPC is especially useful for eyes that have undergone multiple ocular procedures with severe conjunctival scarring and poor visual potential. However, cyclodestructive procedures are also associated with a higher incidence of graft failure, hypotony, and visual loss [33,34] and may also produce phthisis bulbi [22]. Therefore, these procedures are often reserved for patients who have failed all other interventions.

Surgical management

Trabeculectomy has been performed in patients with previous penetrating keratoplasty; however success rates are highly variable, ranging from 20 to 80% [35,36]. Conventional trabeculectomy is usually not effective if there is perilimbal conjunctival scarring, and trabeculectomy without antimetabolites after penetrating keratoplasty has been reported to have up to a 50% increased risk of failure at 3 years, with 90% of these occurring within the first 6 months of surgery [35]. Adjunctive antimetabolite use has significantly improved success. Ayyala *et al.* [37] reported a 76.5% (13 of 17 eyes) success rate for trabeculectomy with mitomycin C (MMC) after penetrating keratoplasty during a mean follow-up of 23 months.

Kirkness *et al.* [38] was the first to report the use of glaucoma drainage devices (GDDs) in patients with glaucoma following penetrating keratoplasty. GDDs have become important methods for controlling IOP in refractory cases [39–41], and the reported success rates up to 1 year after GDD implantation range from 74 to 92% [40–42]. Almousa *et al.* [43] described Ahmed glaucoma valve (New World Medical Inc., Rancho Cucamonga, California, USA) implantation in 59 eyes with high-risk penetrating keratoplasty, including corneal neovascularization, repeat grafts, large grafts, bilateral grafts, past or present inflammatory eye disease, past or present elevated IOP, history of anterior segment surgery, younger age, ocular surface disease, and many quadrants of anterior synechiae. Success was achieved in

44 eyes (75.8%) during an average follow-up of 78 months. Corneal graft survival was 87% at 1 year, but declined to 47% by 5 years of follow-up.

Graft survival after glaucoma surgery

Glaucoma surgery is associated with a shorter time to graft rejection, a greater likelihood of multiple rejection episodes, and a greater chance of graft failure when compared with nonglaucomatous and medically treated eyes [10,44]. The use of antimetabolites that are toxic to the corneal endothelium [45] combined with a decrease in endothelial cell density after intraocular surgery may lead to endothelial failure [36]. Glaucomatous eyes also have higher levels of proinflammatory cytokines in the aqueous humor compared with nonglaucomatous eyes [46], and the aqueous inflammatory response in primary open angle glaucoma (POAG) is elevated in eyes with previous cataract or glaucoma surgery compared with eyes treated medically [47■■]. A breach of the blood-aqueous barrier would be expected to result in a similar rate of graft failure in eyes with previous trabeculectomy and GDD.

Drainage tubes can also damage the corneal endothelium via direct contact [48] or by acting as a conduit for the retrograde passage of inflammatory cells into the anterior chamber [38]. Arroyave *et al.* [41] identified placement of the drainage tube in the anterior chamber as being 12 times more likely to result in earlier graft failure, although Almousa *et al.* [43] did not find this association. The risk of rejection appears to be similar between valved and nonvalved devices [22]. The timing of GDD implantation on corneal graft survival has also been evaluated, although the results are inconclusive. Some studies have shown improved graft survival when the implant is placed prior to or during penetrating keratoplasty [49,50], whereas others have shown no association [41,42]. The 10-year results of the Cornea Donor Study [51] reported that a history of glaucoma, particularly when prior glaucoma surgery had been performed, was also associated with graft failure.

GLAUCOMA AFTER DESCEMET'S STRIPPING ENDOTHELIAL KERATOPLASTY

Incidence, risk factors, and pathogenesis

Descemet's stripping endothelial keratoplasty (DSEK) has been performed with increasing frequency in recent years [52] by virtue of its advantages over full–thickness keratoplasty, including accelerated healing, more predictable refractive outcomes, improved corneal integrity, and rapid visual recovery [53–56]. Although the initial expectations were that DSEK would also offer lower risks of postoperative ocular hypertension, the factors that generate high IOP after penetrating keratoplasty may also apply to DSEK. The incidence of post-DSEK ocular hypertension is reportedly between 29 and 47% [56–63], and the incidence is even higher in patients with preexisting glaucoma, with estimates ranging from 43 to 54% [56,57]. Indeed, ocular hypertension is the most frequently reported complication following DSEK, exceeding graft dislocation [64–66], rejection [67,68], and endothelial failure [52].

The most common cause of glaucoma after DSEK is corticosteroid-induced elevation in IOP [56,63], although chronic angle closure, inflammation, and graft rejection are other potential causes [13,56,69]. A retrospective study by Allen *et al.* [57] reported that a previous history of glaucoma was associated with a 2.4 times greater chance of developing post-DSEK IOP elevation. Additionally, eyes with preoperative glaucoma or undergoing additional intraoperative procedures during DSEK, such as phacoemulsification, synechiolysis, anterior chamber intraocular lens exchange, or vitrectomy, were more likely to require an escalation of glaucoma therapy [61]. Chan *et al.* [62] identified glaucoma in the fellow eye, postoperative complications or procedures, and age less than 60 years as additional risk factors for the development of post-DSEK ocular hypertension. These risk factors may reflect more severe angle damage and compromised trabecular outflow, increased intraocular inflammation altering trabecular function, and a tendency for corticosteroid responsiveness in younger individuals. Unlike penetrating keratoplasty, the surgical indication for DSEK does not appear to influence the risk of postoperative ocular hypertension [62].

In the early postoperative period, air trapped behind the pupil may lead to pupillary block and a significant elevation in IOP. Although pupillary block is an uncommon occurrence, it can lead to significant complications, including graft failure and peripheral anterior synechiae with subsequent chronic angle closure glaucoma [70,71].

Intraocular pressure measurement after Descemet's stripping endothelial keratoplasty

One factor to consider in postoperative DSEK management is the accuracy of IOP measurements following surgery. The average preoperative thickness of a DSEK graft is approximately 100–200 μm, and most eyes with DSEK grafts have a central corneal thickness near 700 μm [71–73]. Although corneal thickness affects the accuracy of GAT, DCT has been shown to be less dependent on corneal thickness and shape. Two studies [72,73] assessing the effect of increased corneal thickness on IOP measurements from GAT and DCT suggest good correlation between both methods following DSEK. Increased corneal thickness did not appear to result in falsely elevated IOP measurements, and IOPs measured by GAT should not be corrected for a postoperative increase in corneal thickness, as this could miss a truly elevated IOP.

Medical therapy

Topical glaucoma medications are usually the initial therapy for elevated IOP in post-DSEK eyes. Vajaranant *et al.* [56] reported that after DSEK, only one out of 315 (0.3%) patients without and seven of 85 (8%) patients with preexisting glaucoma required glaucoma surgery; most patients were treated medically. Maier *et al.* [63] also found that medical therapy sufficiently controlled IOP elevation after DSEK, which occurred in 23 eyes (39%). In this study, however, the criterion for IOP success was not defined and progression of glaucoma by optic nerve head changes or visual field loss was not evaluated.

Other studies have observed a higher failure rate of medical therapy alone to control IOP after DSEK. Chan *et al.* [62] reported that two years after DSEK, 15 of 51 eyes (29.4%) required trabeculectomy with MMC. The higher rates of surgical intervention reported in

this study may relate to the fact that high-potency corticosteroids were continued in the face of elevated IOP to reduce the risk of graft rejection and failure.

Laser therapy

Early in the postoperative period, IOP elevation may occur because of pupillary block via air migration behind the iris [74,75]. An inferior iridotomy preoperatively, or an iridectomy performed intraoperatively, can avoid this potential complication.

Surgical management

Studies evaluating outcomes for glaucoma surgery after DSEK are limited. The surgical options for glaucoma after DSEK are similar to those for penetrating keratoplasty and include trabeculectomy with MMC and GDD implantation. Quek *et al.* [61] investigated the effect of DSEK on IOP control in Asian eyes with preexisting glaucoma or ocular hypertension and found that 62% of eyes required an increase in IOP lowering medical therapy and 28% needed filtration surgery. Eyes with no history of filtration surgery or that underwent additional intraoperative procedures during DSEK were 10 to 18 times more likely to require IOP-lowering treatment after DSEK ($P = 0.002$, $P = 0.005$), respectively.

Surgical outcomes following glaucoma surgery may be more favorable in those who have had previous DSEK compared with penetrating keratoplasty. Boey *et al.* [76] compared outcomes of trabeculectomy with MMC after DSEK versus penetrating keratoplasty. The DSEK group achieved a 70.1% mean IOP reduction compared with 55.6% in the penetrating keratoplasty group. The proportion of patients who achieved an IOP less than 12 mmHg was also significantly higher in the DSEK group (80.0 versus 43.9%, $P = 0.03$). The rates of graft failure arising after trabeculectomy with MMC were similar between the two groups at approximately 10%.

There are concerns about performing DSEK after previous glaucoma surgery centered on the difficulty in obtaining a complete anterior chamber air fill. Phillips *et al.* [58] reported a 14% (5 of 34 eyes) dislocation rate in patients undergoing DSEK after glaucoma surgery; four of the five dislocations occurred in eyes with hypotony. On the other hand, Wiaux *et al.* [59] found a slightly lower dislocation rate (7.1%) in eyes with previous glaucoma surgery. A similar dislocation rate of 7.8% (36 of 462 procedures) was reported by Aldave *et al.* [77■■] with no significant difference between the groups with and without previous glaucoma surgery. These results suggest that occluding the lumen of a tube shunt, as has been suggested previously [78], might unnecessarily place an eye with advanced glaucoma at increased risk for intra or postoperative pressure spikes. Moreover, a relatively low dislocation rate also argues against the need for suture closure of an iridectomy to obtain a sufficiently tense air fill and ensure attachment of the donor DSEK button.

GLAUCOMA AFTER BOSTON KERATOPROSTHESIS

Incidence, risk factors, and pathogenesis

Indications for the Boston type I keratoprosthesis (B-KPro; Massachusetts Eye and Ear Infirmary, Boston, Massachusetts, USA) have expanded rapidly over the last decade [79],

but the high incidence and fast progression of glaucoma after B-KPro implantation presents an ongoing challenge. The incidence of glaucoma prior to B-KPro is reportedly between 36 and 76% [79,80■,81–88], and many of these eyes are already receiving maximal medical therapy or have had glaucoma surgical intervention. Progression of preexisting glaucoma after B-KPro implantation has been estimated to be as high as 25% [83] with the fastest rates of progression occurring in patients requiring keratoprosthesis for corneal burns, and the slowest in those with no history of prior corneal surgery [80■]. Additionally, *de novo* glaucoma occurring after B-KPro has been reported to range from 2 to 28% [79,80■, 81,82,84, 85,89,90].

The mechanisms of glaucoma after B-KPro implantation are multifactorial and related to damage to the anterior chamber angle. Iridocorneal adhesions with progressive shallowing of the angle [91] and inflammatory debris obstructing the trabecular meshwork [80■] have been demonstrated. Crowding of the anterior segment by the large B-Kpro backplate can also compromise the angle, and if a patient is left aphakic or if the iris is removed, the trabecular meshwork scaffold can collapse and become distorted [92]. The chronic use of topical corticosteroids prior to placement of the B-KPro may also lead to corticosteroid-induced glaucoma [93].

Intraocular pressure measurement after Boston keratoprosthesis

Globe palpation is the most commonly employed method of estimating IOP after keratoprosthesis implantation because the rigidity of the large backplate precludes an accurate measurement of IOP by standard tonometers [93]. Finger tension has been established as an accurate method for detecting IOPs of 30 mmHg or more, particularly when performed by practiced observers [94–96]. However, the intrinsic inaccuracy and lack of interexaminer reliability are additional challenges in the management of these patients.

Cyclodestructive procedures

CPC can be performed at the time of, or subsequent to, B-KPro placement [84,89,97,98]. Rivier *et al.* [89] reported the results of 18 eyes with B-KPro undergoing diode transscleral CPC, 13 of which were performed after previously implanted GDDs failed to adequately control IOP. In this study, normal IOP (estimated 10–21 mmHg) was achieved in 12 eyes (67%) with or without the use of adjunctive medications. The utility of CPC may be particularly applicable to B-KPro eyes that may have a higher risk of complications from GDD or that have uncontrolled IOP despite prior GDD placement.

Surgical management

The high incidence and severity of glaucoma has prompted a low threshold for performing glaucoma surgery either prior to or simultaneously with B-KPro implantation [90,93]. However, there is no consensus regarding the timing or type of glaucoma surgery. In a study by Kamyar *et al.* [83], 93% of eyes receiving B-KPro with no previous glaucoma surgery developed postoperative IOP elevation, whereas 47% with prior glaucoma surgery did not. This suggested a reduced risk of progression in those with aggressive perioperative management of glaucoma. Panarelli *et al.* [99] reported prophylactic pars plana vitrectomy and GDD placement prior to B-KPro implantation in eyes not already diagnosed with

glaucoma. Postoperatively, these eyes maintained open angles and a normal IOP without adverse outcomes up to 18 months after surgery. More recently, Huh *et al.* [100■] evaluated outcomes of combined pars plana GDD and B-KPro, and 65% of patients achieved an IOP less than or equal to 20 mmHg by scleral pneumotonometry after 31.6 months of follow-up. The authors also reported a lower risk of GDD-related complications with pars plana tube placement. Crnej *et al.* [80■] also found that eyes with glaucoma surgery performed before or during B-KPro implantation developed disc cupping at a significantly lower rate than those with glaucoma surgery performed after B-KPro implantation ($P = 0.013$; $P = 0.001$; Wilcoxon test).

However, the judiciousness of empiric GDD placement is not without debate. Robert *et al.* [88] compared the loss of best-corrected visual acuity in patients receiving B-KPro alone versus combined with GDD implantation. In the combined group, there was an increased risk of glaucoma progression (22%), tube occlusion resulting in IOP spikes (22%), or choroidal hemorrhage (17%), and best-corrected visual acuity loss was nearly double the rate of vision loss in the B-KPro alone group. Li *et al.* [101] also reported a positive correlation of GDD-related complications and vision loss, including GDD erosions in 10 of 17 eyes (58.8%). The postoperative use of a bandage contact lens to prevent desiccation of donor corneal tissues was also found to heighten the risk of GDD erosions, likely as the result of mechanical contact [101].

The above data may suggest that in patients without signs or a history of glaucoma prior to B-KPro, GDD implantation could be deferred until definitive evidence of glaucomatous damage develops, given the significant attendant risks. It should be noted, however, that glaucoma surgery following B-KPro implantation can be more challenging, since ensuring an adequate shave of the vitreous base for pars plana tube placement is difficult when the optical viewing area is limited to 3 mm. Conversely, in patients already diagnosed with glaucoma and in whom glaucoma surgery has not yet been performed, glaucoma surgery may best be performed prior to or simultaneously with B-KPro implantation, since progression can occur rapidly in these very damaged eyes and further compromise long-term visual rehabilitation.

CONCLUSION

Glaucoma is a well known complication following corneal transplantation. Depending on the underlying corneal disorder and surgery performed, glaucoma can be both aggressive and refractory to medical and surgical treatment. Irregularities in the corneal surface and media opacities often prevent accurate IOP measurements and/or reliable assessments of the optic nerve and visual field. Although these cases can be challenging for clinicians, early detection and a low threshold for glaucoma treatment can ultimately lead to improved patient outcomes.

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KEY POINTS

- Elevations in intraocular pressure and glaucoma progression are significant causes of ocular morbidity following corneal transplant surgery.
- Although medications and laser therapy are usually first-line treatments, surgical therapy may be necessary to prevent glaucomatous damage.
- Glaucoma following corneal transplant surgery may be difficult to detect and monitor, but having a low threshold for treatment may ultimately lead to improved patient outcomes.