Glaucoma following Corneal Transplant Surgery

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ABSTRACT

Post-PK glaucoma is an important cause for irreversible visual loss and graft failure after penetrating keratoplasty. The etiology for this disorder is multifactorial and with the use of new diagnostic equipment it is now possible to elucidate the exact pathophysiology of this condition. A clear understanding of the various mechanisms which operate during different time frames following keratoplasty is essential to chalk out the appropriate management algorithms. The various issues with regard to its management, including the putative risk factors, IOP assessment post-PK, difficulties in monitoring with regard to the glaucoma visual fields and optic nerve evaluation are discussed. A stepwise approach to management starting from the medical management to surgery with and without metabolites and the various cycloablative procedures in cases of failed filtering procedures and excessive perilimbal scarring is presented.

Keywords: Glaucoma, Penetrating keratoplasty, Iridoplasty, Trabeculectomy.

INTRODUCTION

Glaucoma following penetrating keratoplasty (PK) is one of the most common causes of irreversible visual loss and the second leading causes of graft failure after rejection. Postkeratoplasty glaucoma is a significant clinical problem because of its frequency of occurrence, difficulty in diagnosis and monitoring, and complexity of management. An increase in intraocular pressure at any time after penetrating keratoplasty (PK) leads to a significant endothelial cell loss with dire consequences as the endothelial reserve is already low. Timely diagnosis of post-PK glaucoma with initiation of appropriate treatment is mandatory to preserve optimal graft clarity and optic nerve head function.

Post-PK glaucoma is defined as an elevated intraocular pressure greater than 21 mm Hg after penetrating keratoplasty, with or without associated visual field loss or optic nerve head changes. Diagnostic difficulty arises due to errors in tonometry recordings of a thick or astigmatic corneal graft. In addition, it is often not possible to assess adequately the optic nerve and visual field before surgery or in the immediate postoperative period because of preoperative media opacification and postoperative corneal distortion with high astigmatism.

It was only in the 1960s, with the development of instruments that allowed routine monitoring of IOP after keratoplasty, that the problem of post-PK glaucoma was recognized. In 1969, Irvine and Kaufman first reported the high incidence of increased intraocular pressure (IOP) following PK. They reported a mean maximum pressure of 40 mm Hg in aphakic transplants and 50 mm Hg in combined transplants and cataract extraction in the immediate postoperative period. Since then various authors have reported the incidence of glaucoma following keratoplasty to be from 9 to 31% in the early postoperative period and from 18 to 35% in the late postoperative period.

ETIOLOGY

Risk factors for glaucoma in patients undergoing PK include:
- Aphakic and pseudophakic bullous keratopathy
- Mesodermal dysgenesis
- Irido-corneal-endothelial syndrome
- Pre-existing glaucoma
- PK for perforated corneal ulcer
- Adherent leucoma
- Previous keratoplasty
- Post-traumatic cases
- Combined PK and cataract extraction
- Performance of vitrectomy during PK.

Indications for Penetrating Keratoplasty and associated Rates of Chronic Post-PK Glaucoma

Goldberg et al reported 29% incidence of increased IOP in the early postoperative period and 30% in the late postoperative period in patients with aphakic bullous keratopathy. They reported that the incidence of increased IOP is higher in patients with repeat grafts, both in the early (45%) and late postoperative phase (52%), and those with pre-existing glaucoma (71% of all patients with pre-existing glaucoma developed elevated IOP in the early postoperative course). Kirkness et al reported a higher incidence of glaucoma in patients undergoing keratoplasty following corneal perforation, especially that following suppurrative keratitis, due to peripheral anterior synechias.
formation and secondary angle closure. The rates of chronic glaucoma after keratoplasty differ significantly based on the indication for keratoplasty (from a low of 0 to 12% for keratoconus to a high of 75% after infectious keratitis).

Pathophysiology

The pathophysiology of post-PK glaucoma is multifactorial and may be related to distortion of the angle with collapse of the trabecular meshwork, suturing technique, postoperative inflammation, use of corticosteroids, peripheral anterior synechiae formation, etc. Olson and Kaufman,9 using a mathematical model, proposed that the elevated IOP following keratoplasty in an aphakic patient might be the result of angle distortion secondary to a compressed tissue in the angle. Edema and inflammation after surgery lead to a further compromise in the trabecular meshwork function and the situation is further aggravated by angle distortion. Factors that contribute to angle distortion include tight suturing, long bites (more compressed tissue), larger trephine sizes, smaller recipient corneal diameter and increased peripheral corneal thickness. The following surgical variables reduce angle compression:

- Less tight sutures
- Deep sutures
- Short sutures
- Suture bites equal on either side of wound
- Smaller sized grafts
- Donor corneas larger than the recipient
- Thinner recipient corneas
- Larger overall corneal diameter.

On the other hand, Zimmerman et al10 proposed that mechanical collapse of the trabecular meshwork in aphakic grafts is the main problem leading to glaucoma. They postulated that the trabeculum needs posterior fixation afforded by the ciliary body—lens support system and an anterior support afforded by the Descemet’s membrane. In aphakia, the posterior support is relaxed with the removal of the lens. After keratoplasty, the Descemet’s membrane is incised, which leads to relaxation of the anterior support. Both these factors lead to partial trabecular collapse and obstruction of aqueous outflow. Supporting this theory, Zimmerman et al11 reported that oversized donor buttons (0.5 mm larger than the host bed) in aphakic patients reduced the incidence of glaucoma. The effect was more obvious when an 8 mm donor button was used in a 7.5 mm host bed. Retained viscoelastic is another important cause for increased IOP in the early postoperative period, especially with the use of sodium hyaluronate 2.3% (Healon 5) or Healon GV (1.4%) and its combination with chondroitin sulfate (Viscoat) used during keratoplasty. One has to be cautious when these high viscosity agents are used because they can clog the trabecular meshwork, leading to a severe postoperative IOP spike. Complete removal must be done at the end of surgery.

The main cause for late post-PK glaucoma is synechial angle closure with the degree of synechial closure strongly correlated with the need for surgery.12 A floppy atrophic iris may also lead to a higher incidence of peripheral anterior synechiae (PAS) formation, which can be prevented by iris suturing or iridoplasty.13

Diagnosis

Accurate measurement of intraocular pressure, assessment of visual fields and neuroretinal structures are often not possible before keratoplasty due to the primary corneal disease. This often leads to an inability to diagnose pre-existing glaucomatous optic neuropathy. Following PK, changes in corneal thickness, postoperative astigmatism and refractive changes often preclude reliable postoperative assessment of IOP, disk and visual field. Extra time needs to be devoted for such patients with the main emphasis on stereoscopic optic nerve head evaluation under mydriasis. Gonioscopy may be performed to view the site and extent of peripheral anterior synechiae or PAS. The ideal way is to take disk photographs at the first examination and serially repeat them at least once a year to detect if there is any progression of the glaucomatous optic neuropathy.

The diagnosis of post-PK glaucoma is primarily based on IOP measurements in the early postoperative period and, on IOP, optic disk changes, and progressive visual field changes in the late postoperative period. IOP in the early postoperative period, when the corneal surface is irregular, can be measured with the Mackay-Marg electronic applanation tonometer,14 the pneumatic applanation tonometer, the Tono-Pen, or recently, the Dynamic Contour tonometer which measures IOP, independent of the corneal thickness. If the graft surface is smooth with an intact epithelium and regular mires can be obtained, then Goldmann applanation can be used to measure the IOP. Marked corneal astigmatism causes an elliptical fluorescein pattern. To obtain an accurate reading with the Goldmann applanation tonometer, the clinician should rotate the prism so that the red mark on the prism holder is set at the least curved meridian of the cornea (along the negative axis). Alternately, two pressure readings, taken 90° apart, can be averaged. The accuracy of applanation tonometry is reduced in certain situations, such as corneal edema, scars, blood staining, or any condition that thickens or alters the corneal elasticity.15 Corneal epithelial edema and stromal edema predispose to inaccurately low readings, whereas pressure measurements taken over a corneal scar will be falsely high. While measuring IOP with Goldmann applanation tonometer, which is standardized for a corneal thickness of 520 microns, overestimation of IOP may occur due to an increase in the corneal thickness.

In cases with complete tarsorrhaphy, the IOP can be digitally estimated by palpation16 although new tonometers which measure IOP through the lid (proview phosphene tonometer) are now available. In such cases, measuring the IOP in the normal eye using the Goldmann applanation tonometer and then performing digital palpation gives a better estimate of the IOP.
Ultrasound Biomicroscopy

Ultrasound biomicroscopy is useful to assess the angle and establish the cause for post-PK glaucoma, especially in eyes with a failed graft where anterior segment details are not clearly visible. The extent of iridocorneal adhesions, location of IOL, phakic/aphakic status, anterior chamber depth, angle width and corneal thickness can be determined with this technology. Moreover, UBM can be of use to the anterior segment surgeon by providing preoperative data regarding the anterior segment anatomy, especially with regard to the presence of iridocorneal adhesions and to guide the surgeon regarding extraprecautions to be taken in such areas to prevent or minimize inadvertent iris damage during penetrating keratoplasty. It also aids the glaucoma surgeon in planning the site for trabeculectomy or a glaucoma drainage device.

In a ultrasound biomicroscopic study, the anterior segment anatomy was assessed with the use of UBM in 26 opaque grafts with post-PK glaucoma. The mean CCT was 936.3 μm (range: 573-1574 μm). One out of 26 (3.8%) patient had peripheral anterior synechias up to 90°. 6/26 (23.08%) had synechias from 90 to 180°, 9/26 (34.61%) had synechias from 180 to 270°, and 10/26 (38.46%) had synechias from 270 to 360°. The following types of synechiae were noted on the UBM—PAS in 26/26 eyes (100%), synechiae at the graft host junction (GH Jn) in 3/26 eyes (11.53%), both PAS and GH junction synechiae in 8/26 eyes (30.77%), central iris corneal synechia in 4/26 eyes (15.38%), and IOL iris synechia in 2/26 eyes (7.6%). Additionally, AC IOL tilt was seen in 3/5 eyes (60%) with AC IOLs, PC IOL tilt was seen in 1/8 eyes (12.5%), AC IOL haptic touching the endothelium in 3/5 eyes (60%), and AC IOL haptic buried deeply in the angle in 1/5 eyes (20%).

MANAGEMENT

Management of post-PK glaucoma is a complex issue and requires an insight into the pathophysiology leading to elevated IOP. It is important to take appropriate steps during surgery for a primary prevention of this blinding disorder.

Prophylaxis

Preoperative Factors

Pre-existing glaucoma is a major risk factor for postoperative glaucoma and graft failure and is especially difficult to treat in aphakic and pseudophakic eyes. Reinhard et al estimated the 3-year graft survival rate in patients with a preoperative history of glaucoma to be 71%, in contrast to 89% without such a history. A high incidence of graft failure has been reported if a glaucoma operation is performed following the penetrating keratoplasty. Some studies, therefore, suggest treating the glaucoma with mitomycin C trabeculectomy or with a glaucoma drainage device combined with the penetrating keratoplasty surgery.

Intraoperative Factors

During surgery, the use of following procedures may reduce the risk for postpenetrating glaucoma: 1 mm oversize donor button, deep bites, goniosynechiolysis, iridoplasty (iris tightening procedure in cases of a floppy iris, viscoelastic removal at the end of surgery and careful wound closure to prevent postoperative wound leaks.

Postoperative Factors

Postoperatively judicious use of topical steroids controls the inflammation and prevents peripheral anterior synechiae formation. Cycloplegics keep the pupil mobile and prevent pupillary block glaucoma. The IOP must be monitored as long term use of steroid therapy can lead to secondary open angle glaucoma.

Medical Management

Use of topical medications to control IOP is still the first line treatment of postpenetrating glaucoma. The following medications can be used:

- Beta adrenergic blockers (timolol, betaxolol), alpha 2 adrenergic agonists (brimonidine), miotics (pilocarpine), prostaglandin analogs (latanoprost, travoprost, bimatoprost) and carbonic anhydrase inhibitors (acetazolamide, methazolamide).

- Beta adrenergic blocking agents and adrenergic agonists act by decreasing aqueous humor production having no effect on outflow pathways. Adrenergic agents are rarely used in current practice as they are not very effective and cause chronic conjunctival inflammation. Brimonidine tartarate 0.2% or 0.15% with purife preservative can be used (TDS as monotherapy and BD as combination therapy). Apraclonidine 0.5% is a potent vasoconstrictor and is useful both to prevent anterior chamber bleeding during the operation and to treat resultant IOP spikes postoperatively from such a bleed. Miotics have little effect in the presence of angle closure caused by peripheral anterior synechiae and are no longer recommended.

- Systemic carbonic anhydrase inhibitors are very useful as a short-term therapy in the early postoperative period to control the intraocular pressure. Long-term is limited by serious side effects such as tinnitus, nausea, gastrointestinal disturbances, paresthesias, fatigue, depression, anorexia, weight loss, nephrolithiasis and blood dyscrasias. Topical carbonic anhydrase inhibitors ( dorzolamide, brinzolamide) should be used with caution as they also suppress the carbonic anhydrase enzyme in the corneal endothelium and long-term use can lead to graft decompensation. Though these group of drugs can be safely used in healthy grafts with good endothelial reserve, their use for a long period of time should be avoided in cases with marginal endothelial reserve.

- Prostaglandin analogs can also be used to decrease IOP by increasing uveoscleral outflow and can also be used in conjunction with beta blockers. Preservatives, such as benzalkonium chloride (BAC 0.01%) used in majority of topical antiglaucoma
Laser trabeculoplasty may also be used.

When using the topical drugs to lower the IOP, one has to keep in mind the side effects that are peculiar with them in the setting of postkeratoplasty glaucoma. Beta adrenergic blockers can lead to superficial punctate keratopathy, exacerbation of dry eye, and corneal anesthesia all of which can themselves be detrimental to the state of the graft. Alpha 2 adrenergic agonist drugs can lead to allergic periocular reactions, besides superficial punctate keratopathy and dry eyes. The use of miotics in this setting is discouraged because they promote breakdown of blood aqueous barrier, thus stimulating graft rejection and increase the risk of retinal detachment particularly in aphakics. The use of prolonged topical carbonic anhydrase inhibitors can lead to graft decompensation in presence of borderline corneal endothelial status. Finally, the prostaglandin analogs should also be used with caution as they may lead to uveitis, cystoid macular edema (CME) in aphakia and pseudophakia, and reactivation of herpes simplex keratitis in patients grafted with a previous history of healed herpetic keratitis. The use of adrenergic agents like epinephrine, dipivefrin is also discouraged in the modern day management of these patients because of their potential corneal epithelial toxicity, exacerbation of CME in aphakics, pseudophakes and promotion of conjunctival inflammation thereby making future surgical intervention all the more difficult.

In cases of steroid responsive glaucoma, the dose of steroid drops may be tapered to the minimum required. Alternatively, stronger steroid drops can be replaced by steroids that have less tendency to increase IOP, e.g. topical fluorometholone, loteprednol and rimexolone. Cyclosporine A, 0.5 to 2.0% topical drops QID can be substituted for the steroids and this can also help to control the intraocular pressure.

**Surgical Management**

The following options are available:

- Laser trabeculectomy
- Laser iridoplasty/iridotomy
- Trabeculectomy with antimetabolites
- Glaucoma drainage devices.

**Laser Trabeculectomy**

Argon laser trabeculectomy (ALT) can be used if the angle is open. Recommended settings are a 50 micron spot size, 0.1 second duration and 600 to 900 mW of power. The laser is aimed at the junction of the pigmented and nonpigmented trabecular meshwork and the end point is minimal blanching or bubble formation. Complications are postoperative IOP spikes and uveitis which can trigger graft rejection. It is imperative to give postlaser topical steroids at an increased frequency (2-4 hourly) to minimize the chances of triggering graft rejection. ALT may be indicated in patients with open angles, clear grafts and moderately elevated IOP (20-25 mm Hg) on glaucoma medications. Diode laser trabeculectomy and selective laser trabeculectomy may also be used.

Laser iridotomy may be performed with a Nd:YAG laser, if a pupillary block is suspected. Gonioplasty can be done for peripheral anterior synechiae, which have been present for less than one year.

**Trabeculectomy**

Conventional trabeculectomy is usually not effective due to dense perilimbal scarring and fibrosis with an increased risk of failure. The failure rate is further increased in aphakic eyes where it is mandatory to do a vitrectomy to prevent any vitreous from blocking the trabeculectomy ostium. Antimetabolites (5-fluouracil and mitomycin-C) must be used in these patients to inhibit the fibroblastic response. 5 mg of 5 FU in 0.1 cc is given daily as a subconjunctival injection in the immediate postoperative period for 7 to 10 days. Apart from the inconvenience of daily injections, 5-FU injections are associated with a high rate of corneal epithelial toxicity and therefore should be used with caution in patients with postkeratoplasty glaucoma.

Mitomycin-C application (0.2-0.4 mg applied for 1-4 min subconjunctival or sub scleral) has significantly improved the success rate of filtering surgery for glaucoma. The reported success rate in IOP control with mitomycin trabeculectomy in patients with postkeratoplasty glaucoma is 67 to 91% and that of graft failure is 12 to 18%. Intraoperative care must be taken to prevent shallowing of anterior chamber to minimize endothelial loss and mitomycin-C should be thoroughly washed off prior to entry into the anterior chamber.

**Glaucoma Drainage Devices**

Glaucoma drainage devices (GDDs) create an alternate aqueous pathway by channeling aqueous from the anterior chamber through a long tube to an equatorial plate that promotes bleb formation. In 1987, Kirkness was the first to report the use of glaucoma drainage devices in postkeratoplasty glaucoma. Even though the use of glaucoma drainage devices appears to control glaucoma in a high percentage of patients in all published series (71-96%, with an average of 84.8%), it appears to be associated with a high incidence of graft failure in the range of 10 to 51% (average 36.2%). The risk of graft rejection may be increased after glaucoma shunt surgery because the drainage tube may provide a conduit for retrograde passage of inflammatory cells into the anterior chamber. It is important to note that the risk of graft rejection following the insertion of any GDD whether valved or nonvalved is similar. This implies that other mechanisms besides passage of inflammatory cells into the anterior chamber through the tube may play a role. Inflammation caused by the surgical procedure itself may lead to post surgical uveitis and breakdown of blood aqueous barrier, triggering graft rejection.

The etiology of graft failure probably is multifactorial. The presence of underlying chronic inflammation, extensive peripheral synechiae, and multiple previous surgeries may
compromise the graft. It has been seen that the most corneal transplants undergo a 60% reduction in the central endothelial cell count during the first two postoperative years after implant surgery. The complication associated with glaucoma drainage devices, like shallow anterior chamber with iris/tube graft endothelial touch might accelerate the process leading to graft failure. Meticulous surgery should be done to avoid the complication of flat anterior chamber and tube endothelial touch. The use of high-dose steroids for 3 to 6 months in the postoperative phase may help in controlling and suppressing inflammation. Other complications include conjunctival erosion, prolonged hypotony, tube endothelial touch, tube obstruction, tube failure, retinal detachment, tube plate extrusion, epithelial downgrowth and infection.

There are two types of glaucoma drainage devices which can be used. (1) Valved—offer resistance to outflow (Ahmed valve, Krupin implant), (2) Valveless—no resistance to outflow (Molteno implant, Baerveldt implant). The advantages of the valved implants, especially that of the Ahmed glaucoma valve, is the ease of insertion and low incidence of hypotony in the immediate postoperative phase. However, the Ahmed valve is associated with a high incidence of increased IOP—hypertensive phase (as much as 80%), 1 to 3 months after the operation, which may need needling and 5-FU injections. On the other hand, glaucoma drainage devices with a larger surface area, such as the double-plate Molteno and Baerveldt appear to exhibit a lesser incidence of the hypertensive phase and may achieve slightly lower IOPs in the long-term. The overall success rate and other complications, including corneal decompensation, appear to be similar among all glaucoma drainage devices.

It is preferred to use the Ahmed glaucoma valve in patients with mild-to-moderate glaucomatous optic nerve damage and larger surface area implants, such as the double-plate Molteno and the Baerveldt implant, which are associated with more significant pressure reduction in patients with more advanced optic nerve damage.

**Cyclodestructive Procedures**

Cyclodestructive procedures aim to control the IOP by decreasing aqueous humor production by destroying part of the ciliary body. Cyclocryotherapy, transsceral cyclophotocoagulation with Nd:YAG, diode or krypton laser are the various procedures that can be performed on patients with intractable postkeratoplasty glaucoma. Since conventional filtering surgeries and implants have a high failure rate, cyclophotocoagulation is a widely adopted procedure, since it is noninvasive and can be done as a low cost outpatient procedure.

Cyclocryotherapy has been a principal mode of treatment of postkeratoplasty glaucoma. The glaucoma cryoprobe is placed for 1 minute, 1.5 mm behind the corneoscleral limbus. Total 68 applications are made with equidistant spots involving the inferior 180° or 270° circumference of the globe at a temperature of −60°C to −80°C. About 23 clock hours of the superior quadrant of the globe should be left untreated to allow for a future filtering surgery procedure. The treatment can be repeated if indicated. Complications of this procedure include uveitis, immediate rise in IOP, graft failure, corneal decompensation, macular edema, phthisis bulbi, etc.

**Nd:YAG laser cyclophotocoagulation** involves the use of 15 evenly spaced burns, placed 1 to 1.5 mm from the limbus for 180° with a Nd:YAG laser. The recommended mean energy level is 4.0 to 9.0 Joules. Postoperative pain medication and topical steroids are indicated. Low energy settings are preferred in patients previously treated with cyclocryotherapy or filtering procedure to avoid hypotony. Repeated applications may be necessary before adequate control is achieved.

**Diode laser cyclophotocoagulation** utilizes a semiconductor diode laser with a wavelength of 810 nm, has low scleral transmission than the Nd:YAG laser (1064 nm) but greater absorption by melanin. As semiconductor diodes have solid-state construction, they have the advantage of portability, durability and smaller size as compared with Nd:YAG lasers. The recommended power settings with the diode laser are 1250-2000 mW, with a 2-second exposure time. An initial power setting of 1500 mW is increased or decreased by 250 mW increments until it is 250 mW below that producing an audible popping sound. Complications include decrease in the Snellen visual acuity (22-56%), graft failure (11-65%), persistent hypotony (5-10%), anterior uveitis, epithelial defects, and loss of vision, severe pain, phthisis bulbi, hyphema, hypotony, intractable pain, sympathetic ophthalmia, scleral thinning and vitreous hemorrhage. It is recommended to avoid the 3 and 9 o’clock positions to spare the ciliary nerves.

**Transpupillary argon laser photocoagulation** is another modality which can be used. It has been seen that transpupillary argon laser photocoagulation of the ciliary process can reduce IOP in direct proportion to the number of ciliary processes ablated. The technique requires a Goldmann three-mirror lens. The laser is set at 50 to 100 μm spot size for duration of 0.1 to 0.2 second and with a power of 1000 mW. Hemostasis is obtained with repeated application of the laser with larger (200 μm) spot size, 0.2 seconds duration and lower power (250 mW). Ciliary processes are ablated one at a time. A limiting factor of transpupillary argon laser cyclophotocoagulation is the visualization of the processes requiring widely dilated pupil and specialized contact lens. **Endoscopic cyclophotocoagulation** is another technique which can be used.

**Prognosis of Postkeratoplasty Glaucoma**

The two things which are important in managing a case of postkeratoplasty glaucoma are—influence of glaucoma on graft survival and the effect of glaucoma in causing permanent irreversible visual loss. There is uniform agreement in the literature regarding adverse outcomes on both of the above accounts. A study from our center, based on a retrospective
review of 747 penetrating keratoplasties reported only 50% graft clarity over the study period\textsuperscript{18} and further worse only 19% of eyes could retain a visual acuity of 6/18 or better despite medical and surgical measures to control the glaucoma. One thing that needs to be kept in the mind while managing a case of postkeratoplasty glaucoma is that the synechiae formation is progressive over time and that IOP control might worsen over long-term follow-up. Therefore, long-term vigilance is required in taking care of such eyes. Another important issue is the effect of any intervention on triggering the graft rejection and subsequently graft failure. Kwon et al\textsuperscript{33} in their 3-year retrospective study documented 44% graft rejection and nonimmunologic causes of graft failure following insertion of glaucoma drainage device in such eyes. Shah P et al\textsuperscript{38} determined the visual outcome after diode laser cyclophotocoagulation in 28 eyes with atleast 6 months follow-up. They documented stable baseline visual acuity in 17 of the 28 (61%) patients and worsening in eight of the 28 (28%) patients.

**SUMMARY**

Uncontrolled IOP after penetrating keratoplasty is one of the leading causes of graft failure and visual loss in this patient population. It is mandatory that the IOP is monitored on a regular basis after corneal transplantation and aggressively treated if found high. Any patient with pre-existing glaucoma must be carefully evaluated prior to the corneal transplant.

Patients with uncontrolled IOP or patients with borderline IOP control on two or more medications may be treated with mitomycin-C trabeculectomy or glaucoma drainage implant surgery\textsuperscript{41,42} prior to or combined with the planned corneal transplant. Preoperative glaucoma is an important risk factor for the development of postkeratoplasty glaucoma and must be treated prior to the keratoplasty or during the procedure because of a high incidence of graft failure has been reported after glaucoma operations in eyes that have undergone penetrating keratoplasty.

Postkeratoplasty glaucoma that does not respond to medications should be treated surgically. Mitomycin-C trabeculectomy appears to be the safest operations in terms of IOP control and graft survival. Published results support a combined mitomycin-C trabeculectomy with penetrating keratoplasty for patients with pre-existing glaucoma and corneal decompensation. Additional surgical procedures should be avoided, if possible, at the time of trabeculectomy, as this is associated with higher incidence of trabeculectomy failure.

Implantation of a glaucoma drainage device appears to be preferred over other surgical options for patients with postkeratopasty glaucoma and extensive limbal conjunctival scarring, shallow anterior chamber, extensive peripheral synechiae, and failed trabeculectomy. It appears to be superior to cyclodestructive procedures in cases that have failed mitomycin-C trabeculectomy or in those in which mitomycin-C trabeculectomy is contraindicated (i.e. contact lens wearing patients). The graft failure rate following glaucoma drainage implant surgery and cyclodestructive procedures may be the same, but there appears to be a higher incidence of permanent visual loss and hypotony following cyclodestructive procedures.

Cyclodestructive measures should be reserved for patients who have failed all other interventions. In a retrospective study, that compared the surgical outcomes of mitomycin-C trabeculectomy, glaucoma drainage implant surgery,\textsuperscript{34} and Nd:YAG cyclophotocoagulation in the management of intractable glaucoma after penetrating keratoplasty, Ayyala et al\textsuperscript{33} found no differences among the three procedures with respect to controlling IOP and graft failure. However, patients treated with Nd:YAG cyclophotocoagulation tend to have a higher incidence of graft failure, glaucoma failure, hypotony, and visual loss by more than one line, although this was not statistically significant. With development of safer glaucoma procedures and glaucoma drainage implants, early surgery may become the mainstay of treatment in this patient population.

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