Effect of 0.5 mm larger donor corneal size on intraocular pressure following penetrating keratoplasty

Col Sagarika Patyal*, Brig Ajay Banarji*, Col VS Gurunadh#, Col Madhu Bhadauria (Retd)***, Col Poninder Kumar*, Col AK Upadhyay##

ABSTRACT

BACKGROUND

Glaucoma can develop after penetrating keratoplasty resulting in irreversible loss of vision. The incidence of post-penetrating keratoplasty glaucoma varies from 31% in the early postoperative period to 29% after three months. Various factors are responsible for the rise of intraocular pressure (IOP). This study was carried out to evaluate the effect of 0.5 mm larger donor corneal size on IOP following penetrating keratoplasty.

METHODS

Patients were divided into two groups: group I was phakic and group II consisted of aphakic and pseudophakic patients. The same surgical technique was used for obtaining and suturing the donor graft. The viscoelastics and the postoperative regime also remained the same in all cases. IOP was measured by the rebound tonometer.

RESULTS

The incidence of postoperative raised IOP in the first seven days in the phakic group was found to range from 16 mmHg to 25 mmHg and IOP in the aphakic/pseudophakic group ranged from 16 mmHg to 42 mmHg. The IOP in the phakic group after three weeks of surgery was around 12.3 mmHg and that in the pseudophakic/aphakic group was 14.8 mmHg–16.2 mmHg. In aphakic patients, IOP was controlled in the first six months with eyedrops Timolol 0.5% and tablet acetazolamide which was given only for short periods. IOP settled to < 12 mmHg with timolol 0.5% after six months. In the pseudophakic patients, IOP became normal by six months. These were inclusive of patients who had undergone anterior reconstruction and/or vitrectomy.

CONCLUSION

The study proves that keeping the donor corneal size 0.5 mm larger does not affect IOP and that aphakia itself is a factor responsible for rise of IOP due to anterior chamber angle compression.

MJAFI 2011;67:113–116

Key Words: intraocular pressure; penetrating keratoplasty; rebound tonometer

*Assoc Prof, Department of Ophthalmology, AFMC, Pune – 40,

*Consultant, #Senior Advisor, CH (EC) Kolkata, **CMO, Sitapur Eye Hospital, ##Senior Advisor, AFMC, Subroto Park, Delhi Cantt.

Correspondence: Col Sagarika Patyal, Assoc Prof, Department of Ophthalmology, AFMC, Pune – 40.

E-mail: sagarikapatyal@gmail.com

Received: 13.03.2010; Accepted: 09.07.2010

INTRODUCTION

Post-penetrating keratoplasty glaucoma is defined as elevated intraocular pressure (IOP) greater than 21 mmHg after penetrating keratoplasty (PK) with or without associated visual field loss or optic nerve changes necessitating the addition of medication to reduce IOP.¹ Intractable glaucoma can develop after PK causing irreversible loss of vision. The leading cause of enucleation after corneal transplantation is secondary glaucoma amounting to 46%.² The incidence of early increase of IOP after PK is 31% and rise of IOP after three months of PK is 29%.³ This is often refractory to medical management and surgical treatment of the same may lead to graft failure.

The cause of rise of IOP could be due to inflammatory sequelae such as fibrinous iritis, peripheral anterior synechiae, and posterior synechiae. It may also be due to suturing technique that may cause compression of anterior chamber angle, trabecular meshwork collapse, and wound gape. The use of corticosteroids and viscoelastics, ghost cell glaucoma, ciliary block glaucoma, pre-operative angle recession, and pre-operative glaucoma are all known causes of rise of IOP after keratoplasty.⁴ The two potential intra-operative factors that may increase the risk of developing glaucoma after PK are the size of the grafts used and use of viscoelastics Zimmerman et al⁵ have suggested that the size of the graft may be an important factor in causing glaucoma in aphakia because of the following factors:

Trabecular meshwork collapse due to retraction of Descemet's membrane towards the iridocorneal angle thereby reducing anterior support to the angle structures. Loss of zonular tension by lens extraction may reduce the posterior support of the angle. Bourne et al⁶ have also corroborated the findings of Zimmerman et al⁵ by showing that using a 0.5 mm larger donor cornea resulted in significant decrease in the incidence of postoperative glaucoma in aphakia. However, in two different studies by Nissenkorn et al⁷ and Perl et al⁸ there was no significant elevation of IOP using shallow suturing techniques in same sized grafts or disparate size grafting. Studies done by Foulks GN, Perry HD, Dohlman CH, Heideman et al have also stated that keeping the donor corneal size 0.5 mm larger does not affect IOP.

Keeping the donor cornea size larger than the recipient is one factor that may prevent anterior chamber angle compression there by preventing the rise of IOP. However, there is paucity in literature of studies that have assessed by this factor for causing post-PK glaucoma.

A study was conducted therefore to determine the effect of 0.5 mm larger corneal graft on postoperative IOP and also the effectivity of rebound tonometer in the measurement of IOP in post-PK eyes.

MATERIALS AND METHODS

Patients with corneal opacities registered at the Eye Bank for keratoplasty comprised the study group. The study was conducted over a period of two years.

The following patients were excluded from the study: pre-existing glaucoma, complications during surgery.

Patients were divided into two groups depending on their lens status.

Group I: Those who were phakic before surgery

Group II: Those who were aphakic/pseudophakic before surgery

A total of 30 cases were studied. The results were tabulated and subjected to statistical analyses.

All patients were evaluated pre-operatively with detailed history to ascertain the cause of the opacity, any other ocular illness, past ocular surgery, and systemic diseases. Complete general and systemic examination was done.

Ocular examination included: best corrected visual acuity, ocular surface and the integrity of lids and adnexa, tear film by Schirmers' test, type and nature of corneal opacity, presence of any structural abnormalities of the iris, status of lens, IOP measurement by rebound tonometer, USG-A/B scan for status of posterior segment.

Selected patients were then taken up for PK.

Routine workup for surgery was done and intraocular lens (IOL) power was calculated where a combined procedure was planned. Power of IOL to be implanted was calculated by SRK-2 formula. Axial length and keratometer readings of the diseased eye were used wherever feasible. If keratometry reading from diseased eye could not be obtained then the readings from other fellow eye were used.

All patients were started on tablets ciprofloxacin 500 mg twice a day orally 12 hours prior to surgery along with topical ciprofloxacin six hourly 48 hour prior to surgery. Two hundred millilitre of 20% mannitol was given one hour prior to surgery and one drop of 5% betadine was put into every eye just before surgery.

All surgeries were performed by surgeons of equal expertise using standard surgical techniques. Peribulbar anaesthesia was used in all cases of this study. The recipient bed was made of 7 mm and donor button was 0.5 mm larger, i.e. 7.5 mm.

A uniform donor graft of 7.5 mm was taken. Donor corneal button graft was prepared from the endothelial side prior to cutting the recipient cornea.

Recipient cornea was trephined using a hand held hollow disposable trephine up to 80% of thickness. Anterior chamber was entered using 26 G needle and viscoelastics were injected to form the anterior chamber. The viscoelastics used in the entire surgery was 2% hydroxypropyl methyl cellulose in all

cases. The ocular architecture was restored in a few cases by reconstruction of their anterior segment by synechiolysis, anterior vitrectomy in aphakic eyes, iridoplasty, and pupilloplasty. Meticulous anterior vitrectomy was done when required. In cases with cataractous lens, anterior capsulorhexis or capsulotomy was done and nucleus delivered, cortical matter was washed using a two-way cannula. A 6.5-mm optic-sized single-piece polymethyl methacrylate (PMMA) posterior chamber IOL was implanted in the bag. The donor graft was sutured with 16 interrupted 10-0 nylon sutures in between four cardinal sutures. At the end of the procedure, all patients were given 2 mg of dexamethasone and 125 mg of amikacin subconjunctivally.

Patients were examined on day 1, 2, 3, 7, 14, 28 and there after every four weeks for the first six months and then every two months. Apart from routine examination at every visit, the IOP was recorded with rebound tonometer (RBT). All patients were advised to report immediately in case of development of any redness, pain, or blurring of vision.

The postoperative treatment consisted of: dexamethasone 0.1% and chloramphenicol 1% eyedrops and ciprofloxacin eyedrops every two hours for the first 14 days after which the drops were tapered depending upon the response, 2% homatropine hydrobromide eyedrop was given thrice daily for the first 14 days and then tapered, 0.8% methyl cellulose eyedrops every six hours for 28 days, raised IOP was managed with topical 5% timolol twice daily if the IOP could not be controlled by timolol, tablet acetazolamide 250 mg was given orally for 3-5 days.

RESULTS

Indications for keratoplasty are given in Table 1.

Pre-operative visual acuity ranged from PL+/PR accurate to 6/60 in all cases. Final visual recovery was best corrected visual acuity at the time of last follow-up (Table 2).

Table 1 Indications for penetrating keratoplasty.

Indications	No. of cases	% age
Corneal opacities	10	33.33
Pseudophakic bullous keratopathy	10	33.33
Aphakic bullous keratopathy	7	23.34
Regrafts	3	10
Total	30	100

Table 2 Visual recovery.

Visual acuity	No. of cases	% age
>6/18	3	10.0
6/24-6/36	8	26.67
6/60-1/60	12	46.0
HMCF	4	13.34
PL/PR acc	3	10.0
Total	30	100

Table 3 Intraocular pressure following penetrating keratoplasty.

Study groups	Mean IOP (in mmHg using rebound tonometer)								P
	Day 1	Day 2	Day 3	Day 5	Day 7	Day 21	6 months	2 years	
Group I: Phakic SD	18.1 (2.66)	17.4 (4.74)	14.6 (2.56)	12.6 (2.87)	11.6 (2.06)	12.3 (1.65)	9 (1.28)	8.6 (1.16)	P=0.007
Group II: Aphakic SD	34.4 (3.28)	26.1 [#] (7.06)	22.4 [#] (3.67)	17 [#] (3.73)	20.2 [#] (2.64)	14.8 (3.23)	12.4* (2.69)	11.7* (1.00)	P=-3.018
Pseudophakic SD	20.6 (3.02)	18* (2.50)	14.2* (2.63)	10.9* (1.59)	11.2* (1.13)	16.1 (1.44)	12.3* (1.56)	11.3* (2.04)	

[#]With tablet acetazolamide (250 mg 8 hourly) and eyedrop timolol (0.5%) 12 hourly.

*With eyedrop timolol (0.5%) 12 hourly. IOP: intraocular pressure.

Table 4 Surgical procedure.

Surgical procedure	No. of cases	Percentage
PK	9	30.0
PK with cataract extraction and PCIOL	4	13.33
PK with anterior chamber Reconstruction and anterior vitrectomy	8	26.67
PK with anterior vitrectomy only	9	30.0
Total	30	100

PK: penetrating keratoplasty.

The incidence of postoperative raised IOP (Table 3) in phakic group in the first seven days postoperative period was found to range from 16 mmHg to 25 mmHg with a mean of 18.1 mmHg. On the other hand, during the first postoperative week, IOP in the aphakic/pseudophakic group was found to range from 16 mmHg to 42 mmHg with a mean of 28 mmHg. The IOP after three weeks of surgery in the phakic group was around 12.3 mmHg while in the pseudophakic/aphakic group was 14.8–16.2 mmHg. In aphakic patients, IOP was controlled in the first six months with eyedrops timolol 0.5% and tablet acetazolamide given only for short intervals. IOP settled to <12 mmHg with timolol 0.5% after six months. In the pseudophakic patients, IOP became normal by six months. These were inclusive of patients who had undergone anterior reconstruction and/or vitrectomy. Surgical procedure done has been shown in Table 4.

DISCUSSION

Corneal transplantation has the highest rate of success amongst all organ transplants because of the avascularity of the cornea.¹ Endothelial decompensation, allograft rejection, dry eye, postoperative uveitis, recurrence of original disease or infection, and secondary glaucoma can lead to graft failure. Raised IOP causes irreversible damage to the optic nerve head and also

damages the endothelial cells thereby affecting graft clarity and ultimately resulting in poor visual gain following PK. Therefore determination of IOP and its management are imperative to maintain graft clarity.

In the prospective study, 30 cases of PK were studied for the incidence of post-PK glaucoma while standardizing the following parameters: 0.5 mm larger size of the donor graft, similar surgical procedure, 2% methyl cellulose as the viscoelastic agent, using dexamethasone–chloramphenicol eyedrops.

The overall incidence of glaucoma on the first postoperative day was 30.77% in phakic eyes and 70% in the aphakic pseudophakic group with an overall incidence of 53.34%, being significantly higher in group II as compared to group I. In 21 patients who underwent PK combined with other procedures such as anterior vitrectomy, IOL removal, cataract surgery, and exchange of IOL, IOP was significantly higher in the postoperative period both in the immediate and late postoperative period. Among the aphakic and pseudophakic group the aphakic group showed higher postoperative IOP.

A 7.5-mm donor graft was chosen because grafts smaller than 6.5 mm can cause a high postoperative astigmatism and a large donor cornea may induce an immunological reaction. The graft size was kept 7.5 mm, 0.5 mm larger than the recipient cornea without compromising on the site of placement of the graft or allowing overly thick areas of recipient cornea. Interrupted sutures were applied with 10/0 monofilament nylon after placing of four cardinal sutures in all cases. Sutures were of two-thirds depth in host and recipient corneas and short bites were taken on either end. In spite of keeping all these factors constant, it was found that patients of aphakic bullous keratopathy had a rise of IOP postoperatively. This was possibly due to damage to the angle structures due to crowding of the tissues of the iridocorneal angle.

The tonometer used for measuring IOP pre- and postoperatively was the rebound tonometer. This was found to be well tolerated by patients and gave consistent readings. Each IOP was measured at least twice and only when two consistent readings were obtained, was the IOP taken. No case of infection was found after tonometry. Postoperative early raise of IOP was seen in both the groups during the first week. The rise was significantly higher in group II as compared to group I, IOP

stabilised within a week in group I without any medication. However, patients in group II required putting one to two drugs to lower the IOP. For seven patients tablet acetazolamide had to be given for five days after which IOP settled. This was possibly due to inflammation in these eyes and difficulty in egress of methylcellulose due to compromised anterior chamber angles.

Patients of group II also showed a rise in IOP eight weeks postoperatively and IOP was maintained in the lower teens with use of timolol eyedrops and tablet acetazolamide which was given for only short intervals when IOP was >30 mmHg. This pressure rise was higher in aphakes and in those pseudophakes where anterior segment reconstruction was done. Irvin et al⁹ found that aphakic patients undergoing keratoplasty and those patient having combined procedures often (70%) have increased IOP postoperatively. A retrospective analysis done by Karesh et al³ revealed that of the 80 eyes who underwent PK, aphakic eyes had a significantly greater incidence of post-operative glaucoma than eyes with lenses both in the early and late postoperative periods.

Our results are consistent with the above two studies. The study proves that keeping the donor corneal size 0.5 mm larger does not affect IOP and that aphakia itself is a factor responsible for rise of IOP due to anterior chamber angle compression.

Measuring IOP after PK is also an important issue. The gold standard of measuring IOP is applanation tonometry which is difficult to use in recently grafted eyes because of surface irregularity, fluorescein pooling around corneal sutures and corneal oedema which may render IOP reading unreliable.¹ Schiotz tonometer readings are also inaccurate after keratoplasty.¹⁰ NCT can transfer infection from one patient to another and should not be advocated in the immediate postoperative period.¹¹ Rebound tonometer (RBT), a newer tonometer that does not require an anaesthetic or fluorescein dye for measurement, may be a good alternative to non-contact tonometer for measuring IOP postoperatively in cases following PK. In our study IOP was measured by rebound tonometer made by Tiolat, Helsinki, Finland. This tonometer called I-Care, is hand-held and anaesthetic free. It features a small single-use disposable probe of 0.9 mm radius held in position by an electromagnetic field. The probe collides with the central cornea while the instrument is aligned 4–8 mm from the patient's eye. The movement of the probe induces a small induction current, allowing the impact duration to be measured. Measurements are taken within 0.1 seconds. The probe is changed for every patient. The force applied is so minimal that it does not even elicit a blink reflex. Good correlation has been found between rebound tonometer and Goldmann applanation tonometer.¹²

Keratoplasty remains an excellent modality for restoring vision in the corneal blind provided good pre-operating assessment is done for determining the aetiology of the opacity, absence of corneal vascularisation, adequacy to tear film, and

maintaining IOP within normal limits especially after PK has been done.

Intellectual Contributions of Authors

Study concept: Col AK Upadhyay, Col Sagarika Patyal

Drafting and manuscript revision: Col VS Gurunadh, Col Poninder Kumar

Statistical analysis: DR Bassanar

Study supervision: Brig Ajay Banarji, Col Madhu Bhadauria (Retd)

CONFLICTS OF INTEREST

This study has been funded by research grants from the O/o DGAFMS, New Delhi.

REFERENCES

1. Brandt JD, Lim M, O'Day DG. Glaucoma after penetrating keratoplasty. In: *Cornea*, 2nd ed, Krachmer JH, Mannis MJ, Holland EJ, eds. Elsevier Mosby, 2005:1575–1589.
2. Lee PP, McDonnell PJ. Penetrating keratoplasty and glaucoma. In: *Principles and Practice of Ophthalmology*, 2nd ed, Albert DM, Jackobiec FM, eds. Philadelphia: WB Saunders Company, 2000:2860–2890.
3. Karesh JW, Nirankari VS. Factors associated with glaucoma after penetrating keratoplasty. *Am J Ophthalmol* 1983;96:160–164.
4. Newton C, Burk LL. Glaucoma. In: *Corneal Surgery Theory Technique Tissue*, 3rd ed, Brightbill FS, ed. Mosby 1999:387–393.
5. Zimmerman TJ, Krupin T, Brodski W, Waltman SR. The effect of suture depth on outflow facility in penetrating keratoplasty. *Arch Ophthalmol* 1978;96:505–506.
6. Bourne W M, Davison MA, O' Fallon W M. The effect of over size donor buttons on post-operative intra ocular pressure and corneal curvature in aphakic penetrating keratoplasty. *Ophthalmology* 1982;89: 242–246.
7. Nissenkorn I, Wood TO. Intraocular pressure following aphakic transplants. *Ann Ophthalmol* 1983;15:1168–1171.
8. Perl T, Charlton KH, Binder PS. Disparate diameter grafting: astigmatism, intraocular pressure and visual acuity. *Ophthalmology* 1981; 88:774–781.
9. Irvine AR, Kauffmann HE. Intraocular pressure following penetrating keratoplasty. *Am J Ophthalmol* 1969;68:835–844.
10. Kauffman HE, West CE, Wood TO, Wind CA. Measurement and control of intraocular pressure in corneal disease. *Int Ophthalmol Clin* 1970; 10:387–401.
11. Britt JM, Clifton BC, Barnebey HS, Mills RP. Microaerosol formation in non-contact 'air-puff' tonometry. *Arch Ophthalmol* 1991;109:1346.
12. López-Caballero C, Contreras I, Muñoz-Negrete FJ, Rebolledo G, Cabrejas L, Marcelo P. Tonometría de rebote en la práctica clínica. Comparación con tonometría de aplanación. *Arch Soc Esp Oftalmol* 2007;82:273–278.