



Case report

Kahook Dual Blade goniotomy in post penetrating keratoplasty steroid-induced ocular hypertension

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ABSTRACT

Purpose: Steroid-induced ocular hypertension (OHTN) after penetrating keratoplasty (PKP) may cause irreversible damage to the optic nerve and graft failure. The purpose of this study is to report the first case of a post PKP patient with poorly controlled IOP, successfully treated with Kahook Dual Blade (KDB) goniotomy in both eyes. **Observations:** The patient was a 62-year old male with prior PKP in both eyes for lattice corneal degeneration. After an uncomplicated phacoemulsification in the left eye, his IOP increased to 32 mmHg on maximum tolerated IOP lowering therapy, including oral acetazolamide. This patient was dependent on scleral contact lenses for his irregular astigmatism post PKP to achieve his best-corrected visual acuity. Thus, we needed to consider a conjunctival sparing procedure and decided to proceed with performing a KDB goniotomy in the left eye. At 29 months follow up the visual acuity (VA) remained at 20/20 and IOP 13 mmHg on dorzolamide/timolol combination drop. A year following, his right eye also required KDB goniotomy combined with cataract surgery to treat his cataract and elevated IOP of 28 mm Hg. At 18 months post KDB goniotomy, the right eye VA was 20/50 and IOP 13 mmHg on dorzolamide/timolol combination drop. **Conclusions:** This case demonstrates KDB goniotomy may be a good surgical alternative for post PKP steroid-induced OHTN or glaucoma, especially in patients requiring scleral contact lens for their visual rehabilitation.

1. Introduction

Ocular hypertension (OHTN) is a common complication following corneal transplantation surgery and can contribute to the development of glaucoma. Additionally, the accelerated chronic endothelial cell loss following penetrating keratoplasty (PKP) in eyes with poorly controlled intraocular pressure (IOP) may result in graft failure.¹ Thus, elevated IOP is a common cause of vision loss in post PKP patients.² Many studies have tried to determine the incidence of OHTN or glaucoma following penetrating keratoplasty (PKP).^{3,4} The mechanism for intraocular pressure (IOP) elevation following PKP is thought to be multifactorial and includes the need for chronic topical steroid use, synechial angle closure, anatomical distortion of the angle, collapse of the trabecular meshwork, and postoperative inflammation.⁵ Medical therapy remains the key for management of post PKP OHTN and glaucoma. Medically refractory cases are traditionally treated with trabeculectomy, cyclodestructive procedures, or glaucoma drainage devices.^{6–8} These surgical techniques effectively lower the IOP but carry a high-risk of complications, including hypotony, blebitis, tube exposure, endothelial cell loss, and

graft failure.^{9–11} Novel minimally invasive glaucoma procedures have become commercially available in recent years. They offer an improved safety profile and faster postoperative recovery due to smaller incisions and quicker operative times.¹² Little is known about the efficacy of these devices in the IOP management in post PKP patients. The Kahook dual blade (New World Medical Inc., Rancho Cucamonga, California, USA) was launched in the United States in 2015 and was designed to achieve near complete removal of TM with minimal, if any, surrounding tissue damage. This is in contrast to other ab interno trabeculectomy techniques (Gonioscopy-Assisted Transluminal Trabeculectomy (GATT), Trabectome, and traditional trabeculectomy), where residual TM leaflets remain post-procedure.¹³ There is no data on the efficacy of KDB goniotomy in post PKP patients with steroid-induced OHTN or glaucoma. We report a case of a patient with post PKP steroid-induced OHTN successfully treated with KDB goniotomy.

2. Case report

A 62 year-old male with a history of bilateral PKP for lattice corneal

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dystrophy presented with elevated IOP after undergoing uncomplicated cataract surgery in his left eye. He had a known history of steroid-induced OHTN with well-controlled IOP on topical beta-blocker daily prior to his cataract phacoemulsification. He was referred to the glaucoma clinic with IOP of 32 mmHg OS on maximum topical IOP lowering therapy. A slit lamp exam revealed clear PKP in both eyes with arcuate cuts in left cornea. The anterior chamber was deep and quiet in the right eye and deep with trace cell in the left eye given his recent cataract surgery. The left eye had posterior chamber intraocular lens and the right eye had a mild to moderate nuclear sclerotic cataract. He had open angles bilaterally with no synechiae and 2+ pigment in the trabecular meshwork. The optic nerve exam showed cup-to-disc ratio of 0.4 in both eyes and normal appearance of both maculas. Previous Humphrey 24-2 visual fields demonstrated nonspecific scattered depressions in both eyes. His optic nerve optical coherence tomographies (OCT) had low signal strengths, but were within normal limits. Oral acetazolamide extended release twice daily was added but his IOP remained uncontrolled at 28 mmHg OS. The patient had irregular astigmatism following his corneal transplants and was dependent on scleral contact lenses to achieve his best-corrected visual acuity. Therefore a procedure that required a conjunctival bleb was deemed high risk for failure, complications and a poor choice for his visual rehabilitation. The decision was made to proceed with a KDB goniotomy in the left eye given that his IOP remained elevated with maximum IOP lowering therapy, including oral carbonic anhydrase inhibitor for four weeks prior to surgery. The KDB goniotomy was performed according to a standard protocol as described by Sieck et al.¹⁴ A paracentesis was made and viscoelastic was injected into the anterior chamber. A 2.4 mm clear corneal incision was made avoiding the donor tissue. The patient's head was rotated 30–45° away from the surgeon and the microscope was tilted 30–45° toward the surgeon. A gonioscope was placed on the cornea and the KDB was introduced and advanced along Schlemm's canal in the nasal quadrant. Approximately 3–4 clock hours of TM was excised. Viscoelastic was irrigated and aspirated out of the anterior chamber, and the clear corneal wound was hydrated.

On postoperative day 1, the vision in the left eye was 20/300 without correction, 20/60 through pinhole. The intraocular pressure was 7 mmHg, a microhyphema was noted and the patient was told to stop all IOP lowering medications and to start a regimen of pilocarpine 1%, moxifloxacin, and prednisolone acetate 4 times daily in his left eye. The pilocarpine use was part of standard post goniotomy regimen to prevent formation of peripheral anterior synechiae and potential failure of the procedure. At 1 week the IOP was 11 mmHg and the moxifloxacin was discontinued. The patient continued a weekly taper of the prednisolone and pilocarpine. At 1 month, his IOP was 8 mmHg in the left eye. His prednisolone acetate was changed to loteprednol 3 times daily, and the pilocarpine was discontinued. Six months postoperatively, his visual acuity was 20/25-2 with his scleral contact lens in place and IOP remained at 10 mmHg off all IOP lowering medications. A gradual IOP elevation to 21 mmHg was noted over time and at 15 months dorzolamide-timolol topical combination drop was started twice daily to assure further IOP control. At his most recent visit, 29 months after the KDB goniotomy, his best corrected visual acuity was 20/20-1 and his IOP was 13 mmHg on dorzolamide-timolol and loteprednol 0.5% twice daily.

Eleven months following his glaucoma procedure in the left eye, his right eye had IOP elevation to 28 mmHg refractory to maximum topical IOP lowering therapy and required an oral carbonic anhydrase inhibitor. The decision was made to proceed with KDB goniotomy combined with cataract phacoemulsification in the right eye. On postoperative day 1, the VA in the right eye was 20/300 without correction and 20/100 through pinhole with IOP of 17 mmHg with microhyphema. At six months postoperatively his right eye had best corrected visual acuity of 20/25 + 1 with a scleral contact lens and IOP 14 mmHg on dorzolamide-timolol twice daily. The most recent exam was 18 months after the surgical intervention in the right eye and his IOP remains well controlled

at 13 mm Hg on dorzolamide-timolol and loteprednol twice daily. His vision has decreased to 20/50 in the right eye due to lattice degeneration recurrence in the corneal graft. He continues to have a healthy cup-to-disc ratio and stable visual fields and retinal nerve fiber thickness.

3. Discussion

There are many potential complications that can occur in post PKP patients with steroid-induced OHTN or glaucoma being one of the more serious and vision threatening problems. If not well controlled the elevation in IOP can also lead to graft failure. Consequently, it is imperative to have good IOP control following PKP and to monitor these patients closely for optic nerve damage to prevent future vision loss.

The mechanism for developing glaucoma is believed to be multifactorial; however, the prolonged use of topical steroids after PKP is thought to be a significant cause of IOP elevation.⁵ In patients with keratoconus, Pramanik et al. estimated the incidence of steroid-induced glaucoma to be 3.6% (4/112 eyes).¹⁵ Erdurmus et al. found the incidence of steroid-induced OHTN or glaucoma to be 73% in 100 patients with keratoconus who underwent PKP.¹⁶ Medical management and laser trabeculoplasty¹⁷ are common initial nonsurgical treatment options for post PKP steroid-induced OHTN or glaucoma. If IOP remains poorly controlled despite maximum tolerated medical therapy and laser trabeculoplasty, surgical intervention must be considered. Traditionally, medically refractory cases are treated with trabeculectomy, cyclodestructive procedures, or glaucoma drainage devices.^{6–8} There is a greater risk of graft failure with these surgical procedures. In filtering procedures, the risk of endothelial cell loss is due to the use of antimetabolites and their toxicity.¹⁸ With glaucoma drainage devices the cause of endothelial cell loss is due to the tube's proximity to the graft.¹⁹ Some recent studies suggest that this risk of cell loss could be minimized with ciliary sulcus tube placement.²⁰ Minimally invasive glaucoma surgeries could potentially provide a safer surgical option in these patients if they can be shown to be effective. A recent case report by Nazarali et al. demonstrated the efficacy of gonioscopy-assisted transluminal trabeculotomy (GATT) in a post PKP patient with steroid-induced glaucoma.²¹

In this report the KDB goniotomy effectively reduced the high IOP present in both eyes of our patient after failed maximum tolerated medical therapy. Importantly, he was able to continue to use scleral contact lenses after the procedure to achieve his best-corrected visual acuity. The effect of the KDB goniotomy was sustained for more than 2 years postoperatively in the left eye, and 1.5 years postoperatively in the right eye. He required the addition of combination dorzolamide-timolol in the left eye at 15 months and was kept on this medication in the right eye after his KDB goniotomy.,

Complications with the KDB goniotomy are typically mild. The most common complications that have been reported include IOP spike and hyphema due to blood reflux from Schlemm's canal which have an approximately 6% and 40% occurrence respectively.^{22,23}

4. Conclusions

We report the first case of post PKP steroid-induced OHTN effectively treated with KDB goniotomy. This procedure may be especially beneficial for patients needing to continue their contact lens wear to achieve best-corrected vision. The risk for endothelial cell loss is expected to be less as there is no device left behind in the angle and no antimetabolite use. Further prospective studies of larger patient populations are needed to elucidate the role of KDB goniotomy in post PKP patients.

Patient consent

The patient provided written consent for his case to be published.

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Authorship

All authors meet the current ICMJE criteria for authorship.

Declaration of competing interest

The authors have no financial conflicts of interest to disclose.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.ajoc.2020.100826>.

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