

Clinical & Refractive Optometry is pleased to present this continuing education (CE) article by Dr. Leonid Skorin Jr. and Jordan F. Keith entitled **Management of Elevated Intraocular Pressure Following Cataract Surgery**. In order to obtain a 1-hour Council of Optometric Practitioner Education (COPE) approved CE credit, please refer to page 292 for complete instructions.

Management of Elevated Intraocular Pressure Following Cataract Surgery

Leonid Skorin Jr., OD, DO, FAAO, FAOCO;

Jordan F. Keith, BA

ABSTRACT

Elevated intraocular pressure (IOP) is a common problem following cataract surgery. For most patients it is transient and harmless. However, some patients may experience ocular discomfort, increased corneal edema, and even sight-threatening complications demanding further intervention. Physicians involved in cataract postoperative care should be aware of these high-risk patients and know how to manage them appropriately. Decompression of the anterior chamber through the side-port incision (paracentesis site) is a simple and effective way to quickly lower IOP, relieve patient symptoms, and prevent further complications.

INTRODUCTION

Elevated intraocular pressure (IOP) is a common problem following cataract surgery. The increase may be marked and is the most frequent postoperative complication requiring specific treatment.¹⁻³ Increased IOP following cataract surgery has been recognized since the 1960s. The first report of ocular hypertension one day after cataract surgery was documented by Gormaz in 1962.⁴ Since then, numerous studies have evaluated the risks of an increase in IOP following cataract surgery, the demographics, the etiology of the increase, and the most effective way to treat it both short and long term. Treatment options include preoperative and postoperative use of IOP lowering agents, surgical techniques to ensure open outflow channels, and decompression of the anterior chamber. Although nearly all patients' IOPs will return to baseline with or without treatment, some individuals with

IOP spikes may experience ocular pain, corneal edema and even sight-threatening complications such as retinal vascular occlusion, progressive field loss in advanced glaucoma, and anterior ischemic optic neuropathy (AION) in susceptible patients.⁵⁻⁷ For this reason, practitioners involved in cataract postoperative care should be aware of the high-risk patients and know how to manage them appropriately. Decompression of the anterior chamber through the side-port incision (paracentesis site), which will be the focus of this article, is a simple and effective way to quickly lower IOP.

In most patients, postoperative increase in IOP following cataract surgery is transient and benign.⁸ As many as 25% of patients experience an IOP spike >30 mmHg, 4 to 6 hours after uncomplicated phacoemulsification according to a recent study.⁹ At 24 hours postoperatively, the incidence of IOP spikes decreased significantly to 10% and in all cases IOP was within normal limits (≤ 21 mmHg) three weeks later.⁹ Another study found >18% of nonglaucoma patients had an IOP of >28 mmHg, 3 to 7 hours postoperatively, which decreased to below preoperative levels by four days in most individuals.¹⁰ The clinical data suggests that as a general rule, patients with healthy eyes can tolerate a transient postoperative rise in IOP with no detectable effect on visual function.⁸

PATIENTS AT RISK

Although it is not always possible to predict who will have a pressure rise, there is a discernible group of patients at higher risk. Several studies have revealed that patients with or without glaucoma often have a difference in their postoperative IOPs. Shingleton et al¹¹ found a maximum increase in IOP to be 44 mmHg for patients with glaucoma and 32 mmHg in patients without glaucoma. A different study evaluated successive IOP changes for 24 hours following cataract surgery in glaucomatous and nonglaucomatous patients. Patients with glaucoma had a mean IOP of approximately 30 mmHg, 8 hours postoperatively with 7 out of the 13 patients showing an IOP >35 mmHg. In patients without glaucoma, the mean IOP rose to approximately 22 mmHg at 12 hours.¹²

L. Skorin, Jr. — Staff Ophthalmic Surgeon, Albert Lea Eye Clinic, Mayo Health System, Albert Lea, MN; J.F. Keith — 4th Year Optometry Student, Pacific University College of Optometry, Forest Grove, OR

Correspondence to: Dr. Leonid Skorin, Jr., Albert Lea Eye Clinic, Mayo Health System, 1206 West Front Street, Albert Lea, MN 56007; E-mail: skorin.leonid@mayo.edu

Table I Risk factors for vision loss secondary to IOP spikes

- Advanced glaucomatous cupping
- Patients prone to AION
 - Disc at risk
 - Systemic hypertension
 - Diabetes mellitus
 - Dyslipidemia
 - Collagen vascular disease
 - Nocturnal hypotension
 - Anemia
 - Sleep apnea syndrome

Patients with healthy optic nerves seem to tolerate transient increases in IOP without any significant permanent optic nerve damage. However, some patients are at risk for vision loss (Table I). Glaucoma patients may experience further visual field loss and/or loss of fixation.^{13,14} Significant visual changes as a result of an IOP elevation may also occur in eyes prone to AION. Hayreh⁶ reported 13 cases of AION developing immediately following uncomplicated cataract extractions. This postcataract extraction AION appears to be linked to the IOP spikes. The increase in IOP causes a reduction of the perfusion pressure [mean arterial pressure (MAP) – IOP] resulting in significant compromise of the blood supply in the optic disc, especially in eyes with a vulnerable optic nerve head circulation.⁶ Patients susceptible to AION include individuals with structural crowding of the optic nerve head, otherwise known as a “disc at risk”, and/or systemic conditions such as arterial hypertension, diabetes mellitus, dyslipidemia, collagen vascular disease, nocturnal hypotension, anemia, and sleep apnea syndrome.¹⁵⁻¹⁸

CAUSES OF IOP SPIKES

The causes of elevated IOP are numerous (Table II). It has been well accepted that retained viscoelastic material inhibits aqueous outflow through the trabecular meshwork resulting in a higher incidence and degree of postoperative increase in IOP.¹⁹⁻²² This risk can be reduced, but not entirely prevented, by meticulous removal of all visible viscoelastic by irrigation and aspiration (I/A). Rocking the IOL with the I/A tip to promote release of viscoelastic from behind the implant and several sweeps of the angle will assure maximal removal.

When considering the various causes of pressure elevation, topical steroids are often suspected. However, steroids in the immediate postoperative period are rarely a cause of pressure rise. Typically, a steroid-induced glaucoma requires 3 to 6 weeks of continuous use to elicit an IOP response.²³ Other risk factors for an increased IOP include a watertight wound closure, decreased outflow facility, degree of surgical trauma,

Table II Causes of increased IOP following cataract surgery

- Retained viscoelastic
- Watertight wound closure
- Use of corneoscleral sutures vs. non-sutured incision
- Retained lens debris
- Iris pigment release
- Pre-existing outflow compromise
- Pre-existing glaucoma
- Degree of surgical trauma
- Secluded/occluded pupil with iris bombe
- Uveitis or inflammatory cells
- Hyphema or red blood cells
- Expulsive hemorrhage
- Aqueous misdirection
- Epithelial down growth
- Steroid response (3 to 6 weeks after surgery)

retained lens debris, iris pigment release, and hyphema.²⁴⁻²⁶

The skill of the surgeon has been evaluated as well. These risk factors may explain why increased surgical experience is correlated with a decreased risk for postoperative ocular hypertension.²⁴

TREATMENT

No clear guidelines exist for who should be treated following cataract surgery, when they should be treated, and with what methods. Patients at risk for a known increase in IOP and vision loss should be pretreated. Even healthy eyes may have an IOP spike as high as 68 mmHg after uneventful phacoemulsification.²⁴ Several physicians use a one-day postoperative IOP of ≥ 30 mmHg as a risk for further complications and indication for treatment.^{23,27,28} In those patients with glaucomatous cupping and visual field loss, or any patient who cannot tolerate a marked postoperative IOP spike, combining cataract and glaucoma surgery has been recommended.²³

There are several classes of drugs used to treat postoperative increases in IOP including: oral and topical carbonic anhydrase inhibitors (acetazolamide, brinzolamide, dorzolamide, and methazolamide), alpha agonists (apraclonidine and brimonidine), prostaglandin analogs (latanoprost, bimatoprost, and travoprost), beta blockers (timolol and levobunolol), and miotics (intracameral carbachol, pilocarpine, and intracameral acetylcholine). Although several drugs lower IOP after cataract surgery, none of them consistently prevents pressure increases from occurring.²⁹ Numerous studies have reported on the efficiency of these IOP lowering drugs. Several of these studies were recently evaluated and compared.²⁹ For patients at high risk, and in those who tolerate the medications, it is recommended that a fixed combination of timolol and dorzolamide along with brimonidine be used at the end of the surgery, and that the patients should continue to instill these drugs at their usual scheduled time.²⁹ Depending on the patients' risk for postoperative

IOP spike and the condition of their optic nerve at the time of surgery, physicians may want to see them later on the operative day to perform serial paracentesis. If this procedure is required, it has been recommended that physicians prescribe prostaglandins q.d. and/or cholinergics q.i.d. for 2 days after surgery.²⁹

There is a dispute in the literature as to whether medications which reduce aqueous secretion are more effective in controlling postoperative IOP spikes or those that increase aqueous outflow. Arshinoff²⁸ found that aqueous secretion inhibitors, such as alpha agonists and beta blockers, are ineffective in treating postoperative IOP spikes because they delay washout of residual viscoelastic from the eye; miotics and prostaglandin analogs increase aqueous washout, effectively lowering postoperative IOP spikes. Conversely, Lewis²³ found that prostaglandin analogs were less effective in the immediate postoperative setting because they required a more prolonged onset of action and additionally may induce anterior segment inflammation. Besides possibly worsening the postoperative anterior segment inflammation, prostaglandin analogs may cause or exacerbate cystoid macular edema, which is a well known potential postoperative complication following uncomplicated or complicated cataract extraction. Both sides appear to have strong arguments and until a study proves which drugs definitively work the best, the choice of IOP lowering medication postoperatively is left to patient contraindications and clinician preference.

ANTERIOR CHAMBER DECOMPRESSION

One of the fastest ways to lower the IOP following phacoemulsification is by anterior chamber decompression through the side-port incision. There are several variations of this procedure reported in the literature.^{3,23,28}

Lewis²³ recommends using a 30-gauge needle to apply pressure on the side-port incision to release aqueous and lower the IOP. Hildebrand et al³ recommended gently applying pressure on the external lip of the side-port incision using a 24-gauge needle to allow controlled release of aqueous to a level <10 mmHg. This method of decompression was used on 11 patients without significant ocular complications. Pressure prior to decompression ranged from 40 to 68 mmHg and decreased to a mean of 4.73 +/-3.00 mmHg immediately following decompression. The IOP, however, rapidly rose to >30 mmHg at 30 minutes and 38.5 mmHg at 60 minutes after decompression. Hildebrand et al³ concluded that this procedure provides only transient relief and that additional treatment is needed to protect high-risk individuals. In follow-up to this study, Arshinoff²⁸ advised repeated attempts be made at the side-port drainage. If the IOP is >30 mmHg the morning after cataract surgery, he suggested draining aqueous from

the paracentesis site, instilling 1 gtt of pilocarpine 2% and 1 gtt of latanoprost, and have the patient return in 45 minutes to 1 hour. Decompression is repeated hourly for 3 hours and the patient is instructed to instill one drop each of pilocarpine 2% q.i.d. and latanoprost q.d. for 2 days.²⁸

OUR PROCEDURE

Although studies suggest that aqueous release in the management of a postoperative IOP spike may be temporary and limited to <1 hour, we perform decompression of the anterior chamber on every patient presenting with a one-day postoperative IOP >30 mmHg.³⁰ At these elevated pressures, we find patients often experience more corneal edema and ocular discomfort. Following the procedure, the patient often notices an improvement in acuity, comfort, and leaves the office more confident about the postoperative outcome.

Before the procedure, instill one drop of topical anesthetic and one drop of moxifloxacin in the eye to be decompressed. A lid speculum is usually unnecessary, but may be required if the patient is unable to keep their eye open. Seated comfortably behind the biomicroscope, the side-port incision is identified. Using a jewelers forceps, pressure is applied to the scleral side (external lip) of the incision at the limbus until an egress of fluid is noted. Release of fluids is titrated based on measured elevated IOP. It is a good idea to warn the patient that they may feel a sharp sensation for a brief moment. Most patients, however, do not report any discomfort. Remind them that they should remain perfectly aligned behind the biomicroscope and to fixate on a target straight ahead.

Remeasure the IOP after release of aqueous. If there is an insufficient drop in the IOP, repeat the decompression. At the completion of the procedure, check for Seidel's sign. Usually it will be negative but if it is present you may want to instill additional fluoroquinolone to prevent an infection. We then instill a drop of an alpha-2 adrenergic agonist to help lower the pressure. We do not use prostaglandin analogs because of the potential risk of increased anterior segment inflammation and cystoid macular edema. We also do not use miotics because of the potential risk of exacerbating any anterior segment inflammation. Rarely, we have the patient back for a repeat of the procedure which may be required for high-risk eyes. Most patients return on their one-week postoperative visit with normalized pressure.

We did a retrospective review of 90 uneventful cataract surgeries performed by a single surgeon on nonglaucomatous eyes over a two-and-a-half month period. The average one-day postoperative IOP was 22.97 mmHg with a range of 8 mmHg to 44 mmHg. A total of seven patients (7.7%) had an IOP >30 mmHg. All seven patients had aqueous released from the side-port

| Table III Summary of four patients who had aqueous drained through the side-port incision at the one-day postoperative visit | | | | |
|--|--------|-------------------|----------------------------|-----------------------------|
| Age | Gender | Pre-op IOP (mmHg) | One-Day Post-op IOP (mmHg) | One-Week Post-op IOP (mmHg) |
| 86 | F | 16 | 38 | 22 |
| 82 | F | 16 | 40 | 20 |
| 68 | M | 14 | 41 | 17 |
| 79 | M | 25 | 44 | 24 |

incision as described previously. Each patient was also administered one drop of 0.5% apraclonidine. Three patients whose pressures were >30 mmHg had a follow-up that was not included into our study criteria. These three patients could not return within the one-week follow-up time period and therefore were excluded from our analysis. The IOP of the remaining four patients essentially returned to baseline IOP at their one-week follow-up (Table III).

There are risks associated with aqueous release. They include a flat chamber (hypotony), persistent leakage, and endophthalmitis.

CONCLUSION

Elevated IOP following cataract surgery is not uncommon. Based on the literature, it appears that IOP spikes in healthy eyes are transient and probably harmless, but there is evidence to suggest that similar IOP spikes may be detrimental to patients with compromised optic nerves. For healthy or compromised eyes with one-day postoperative IOP spikes, regardless of their long-term visual outcome, patients may experience ocular pain and corneal edema causing decreased visual acuity leading to disappointment and even consternation. Decompression of the anterior chamber is a quick and safe method to lower IOP, relieve patient symptoms, and help avoid potential long-term visual complications. □

REFERENCES

- Cohen VML, Demetria H, Jordan K, et al. First day postoperative review following uncomplicated phacoemulsification. *Eye* 1998; 12: 634-636.
- Dinakaran S, Desai SP, Raj PS. Is the first postoperative day review necessary following uncomplicated phacoemulsification surgery? *Eye* 2000; 14: 364-366.
- Hildebrand GD, Wickremasinghe SS, Tranos PG, et al. Efficacy of anterior chamber decompression in controlling early intraocular pressure spikes after uneventful phaco-emulsification. *J Cataract Refract Surg*. 2003; 29: 1087-1092.
- Gormaz A. Ocular tension after cataract surgery. *Am J Ophthalmol* 1962; 53: 832-841.
- Vannas S, Tarkkanen A. Retinal vein occlusion and glaucoma. Tonographic study of the incidence of glaucoma and of its prognostic significance. *Br J Ophthalmol* 1960; 44: 583-589.
- Hayreh SS. Anterior ischemic optic neuropathy. IV. Occurrence after cataract extraction. *Arch Ophthalmol* 1980; 98: 1410-1416.
- Savage JA, Thomas JV, Belcher CD III, Simmons RJ. Extracapsular cataract extraction and posterior chamber intraocular lens implantation in glaucomatous eyes. *Ophthalmology* 1985; 92: 1506-1516.

- Tranos P, Bhar G, Little B. Postoperative intraocular pressure spikes: the need to treat. *Eye*. 2004; 18: 673-679.
- Tranos P, Wickremasinghe S, Hildebrand D, Asaria R, Mearza A, Nouri S et al. Same vs first postoperative day review after uncomplicated phacoemulsification. Are we overtreating early intraocular pressure spikes? *J Cataract Refract Surg* 2003; 29(3): 508-512.
- Ahmed IK, Kranemann C, Chipman M, Malam F. Revisiting early postoperative follow-up after phacoemulsification. *J Cataract Refract Surg* 2002; 28(1): 100-108.
- Shingleton BJ, Garnell LS, O'Donoghue MW, et al. Long-term changes in intraocular pressure after clear corneal phacoemulsification: normal patients versus glaucoma suspect and glaucoma patients. *J Cataract Refract Surg* 1999; 25: 885-890.
- Barak A, Desatnik, A, Ma-Naim T, Ashenkasi I, Neufeld A, Melamed S. Early postoperative intraocular pressure pattern in glaucomatous and nonglaucomatous patients. *J Cataract Refract Surg* 1996; 22: 607-611.
- Savage JA, Thomas JV, Belcher III CD, Simmons RJ. Extracapsular cataract extraction and posterior chamber intraocular lens implantation in glaucomatous eyes. *Ophthalmology* 1985; 92: 1506-1516.
- Kolker AE. Visual prognosis in advanced glaucoma: a comparison of medical and surgical therapy for retention of vision in 101 eyes with advanced glaucoma. *Trans Am Ophthalmol Soc* 1977; 75: 539-555.
- Buono LM, Foroozan R, Sergott RC, Savino PJ. Nonarteritic anterior ischemic optic neuropathy. *Curr Opin Ophthalmol* 2002; 13(6): 357-361.
- Basile C, Addabbo G, Montanaro A. Anterior ischemic optic neuropathy and dialysis: role of hypotension and anemia. *J Nephrol* 2001; 14(5): 420-423.
- Kanski JJ. *Clinical Ophthalmology: A Systemic Approach*. Fifth Edition. Oxford, Butterworth Heinemann, 2003; 603-605.
- Palombi K, Renard E, Levy P, et al. Non-arteritic anterior ischemic optic neuropathy is nearly systemically associated with obstructive sleep apnea. *Br J Ophthalmol* 2006; 90: 879-822.
- Berson FG, Patterson MM, Epstein DL. Obstruction of aqueous outflow by sodium hyaluronate in enucleated human eyes. *Am J Ophthalmol* 1983; 95: 668-672.
- Passo MS, Ernest JT, Goldstick TK. Hyaluronate increases intraocular pressure when used in cataract extraction. *Br J Ophthalmol* 1985; 69: 572-575.
- Rainer G, Menapace R, Findl O, et al. Intraocular pressure after small incision cataract surgery with Healon5 and Viscoat. *J Cataract Refract Surg* 2000; 26: 277-281.
- Tanaka T, Inoue H, Kudo S, Ogawa T. Relationship between postoperative intraocular pressure elevation and residual sodium hyaluronate following phacoemulsification and aspiration. *J Cataract Refract Surg* 1997; 23: 284-288.
- Lewis RA. What is the best way to prevent and manage postoperative intraocular pressure spikes? In: Chang DF (ed). *Curbside Consultation in Cataract Surgery*. Thorofare, NJ, Slack, 2007; 187-189.
- Bomer TG, Lagreze W-DA, Funk J. Intraocular pressure rise after phacoemulsification with posterior chamber lens implantation: effect of prophylactic medication, wound closure, and surgeon's experience. *Br J Ophthalmol* 1995; 79: 809-813.
- Meyer MA, Savitt ML, Kopitas E. The effect of phacoemulsification on aqueous outflow facility. *Ophthalmology* 1997; 104: 1221-1227.
- Fang EN, Kass MA. Increased intraocular pressure after cataract surgery. *Semin Ophthalmol* 1994; 9: 235-242.
- Noble BA, Simmons IG. *Complications of Cataract Surgery*. Oxford, Butterworth Heinemann, 2001; 75-76.
- Arshinoff S. Postoperative intraocular pressure spikes. *J Cataract Refract Surg* 2004; 30: 733-734.
- Gokhale PA, Patterson E. Elevated IOP after cataract surgery. *Glaucoma Today* 2007; 5(3): 19-22.
- Bahadur GG, Sinsky RM. *Manual of Cataract Surgery*, 2nd ed. Boston, Butterworth-Heinemann, 2000; 71.