

Is YAG capsulotomy safe? Medically refractory intraocular pressure elevation after laser treatment of an aphakic eye

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Abstract

We report on a patient with traumatic angle recession who developed persistently high intraocular pressure after YAG posterior capsulotomy. The increased intraocular pressure failed to respond to medical treatment and required glaucoma surgery with implantation of an ExPress shunt drainage device. The surgery was augmented with intraoperative mitomycin C (0.4 mg/mL). Despite early postoperative hypotony, the patient's visual acuity was 0.5 and intraocular pressure was 6 to 13 mm Hg 5 months after the glaucoma surgery.

Key words: Intraocular pressure; Laser therapy; Lens implantation, intraocular

Introduction

Intraocular pressure (IOP) spike is known to be a major complication of YAG capsulotomy.¹ However, persistent IOP rise that is refractory to medical treatment is rare. We report on a patient who developed persistent rise in IOP after YAG capsulotomy and was successfully managed with Ex-PRESS Miniature Glaucoma Device (Alcon, Neve Ilan, Israel) filtration surgery.

Case report

A 73-year-old man with a history of post-traumatic right eye phthisis and left eye aphakia for more than 10 years was listed for left YAG laser capsulotomy for posterior capsule opacification. The trauma had occurred in childhood, and was vaguely remembered as a blast injury. It was believed to have involved both eyes. The patient could not recall if an operation was done at that time.

Best-corrected visual acuity (VA) for the left eye on presentation was 0.6 (Snellen decimal), with refraction of -2.50D myopia and -0.50D astigmatism with axis at 60 degrees. There was residual cortical remnant at the inferonasal quadrant of the capsular bag and intact posterior capsule with capsular opacification, suggesting a resorbed crystalline lens. The fundus was myopic with myopic maculopathy. There was an episode of ocular hypertension of 34 mm Hg in the left eye in the same year and IOP was controlled with timolol 0.5% eyedrops. Gonioscopy revealed open angles with angle recession in the inferior 180 degrees. The optic disc was pink with a cup:disc ratio of 0.4.

Nd:YAG laser capsulotomy was offered, consented for, and performed. Power used was 3.0 mJ with 22 shots. A few hours later, the patient presented to the accident and emergency department with blurring and headache. On examination, VA was count fingers and IOP was 50 mm Hg. There was corneal

edema and inflammatory cells in the anterior chamber (2+). The anterior chamber was markedly deep, with a complete capsulotomy opening and small amount of vitreous humor at the superior pupil rim. There was no pupil block and the pupil was reactive to light. Gonioscopy finding was the same as that in the previous examination.

Despite maximal medical treatment including topical eyedrops (timolol 0.5% twice daily, dorzolamide combined with timolol twice daily, brimonidine 3 times a day, latanoprost daily), systemic acetazolamide (500 mg intravenous injection given once and 250 mg orally 4 times a day) and 20% mannitol infusion, together with 1% atropine eyedrops twice daily and topical steroid (prednisolone acetate 1% starting at hourly dose round the clock, and later tapered to 4 times a day), the pressure remained high at 29 to 53 mm Hg in the following 2 weeks. Multiple anterior chamber paracenteses were performed, and resulted in only transient reduction in IOP. Due to the recalcitrant nature of the increased IOP, anterior vitrectomy and implantation of Ex-PRESS Miniature Glaucoma Device (P-50; Alcon, Neve-Ilan, Israel) with 0.4 mg/mL mitomycin C application were discussed, consented for, and performed.

On postoperative day 1, IOP was 6 mm Hg. The bleb was diffuse and the anterior chamber was deep. However, hypotony (IOP, 1 to 3 mm Hg) and choroidal effusions developed in the following 2 weeks secondary to suspected ciliary body shut down (**Figure 1**). A blood streak near the optic disc was also noted. Topical atropine, hourly prednisolone acetate 1% eyedrops, and a course of oral steroid at 15 mg 3 times a day for 10 days, then 15 mg twice daily for 2 days were given. Choroidal effusion gradually resolved 3 weeks postoperatively, with IOP maintained at low teens (6 to 13 mm Hg) till the latest follow-up at 5 months postoperatively. The best-corrected VA was 0.5 (Snellen decimal) and the blood streak near the optic disc also resolved (**Figures 2 and 3**).

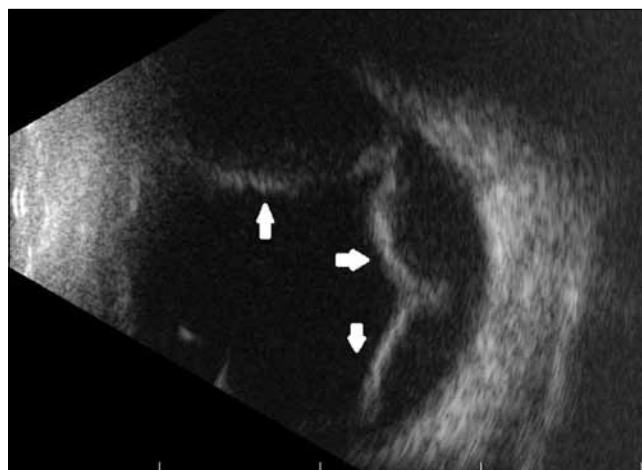


Figure 1. Transverse cut on B-scan ultrasonography of the temporal aspect of the left eye showing multiple choroidal effusions (arrows) secondary to hypotony on day 2 postoperation.

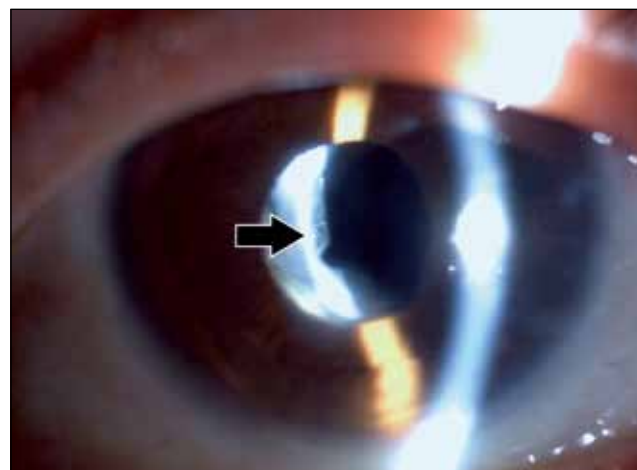


Figure 2. Slitlamp photo of posterior capsulotomy (arrow) in the left eye 5 months post laser treatment.

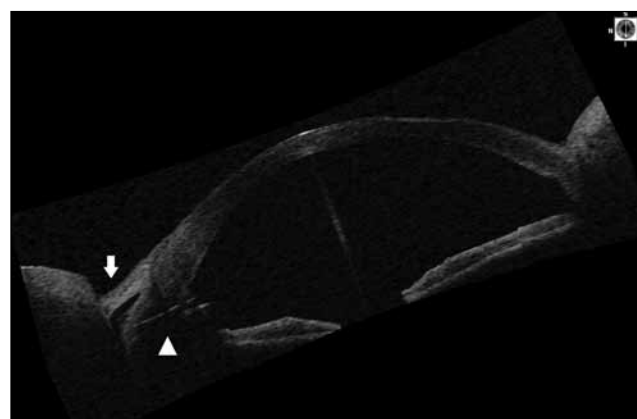


Figure 3. Anterior segment optical coherence tomography showing Ex-PRESS shunt in-situ (arrowhead) and bleb (arrow) from the shunt 5 weeks post filtration surgery.

Discussion

Spiking of IOP has been reported to occur in 24% to 54% of patients receiving YAG capsulotomy.¹ According to Stark et al's cohort,¹ IOP usually peaks within 1.5 to 4 hours post laser treatment and, occasionally, may rise to a level higher than 30 mm Hg. In 60% of the eyes, IOP returned to normal within 24 hours, and in 90% of the eyes within a week.¹

Certain risk factors have been reported for the rise in IOP after YAG capsulotomy, including pre-existing glaucoma, use of high laser power,¹ aphakia,² as well as high myopia.³ It has been postulated that inflammatory debris and cells cause obstruction of trabecular meshwork and, hence, reduce drainage capacity.⁴ Another explanation may be shockwave damage to trabecular endothelial cells. The presence of capsular intraocular lens provides barrier against the cellular debris reaching the trabeculum and dampens the shockwave to the trabecular meshwork.⁵ A retrospective review of 69 glaucoma patients by Lin et al⁶ showed that loss of baseline

IOP control or need for more aggressive therapy was common among glaucoma patients after undergoing YAG capsulotomy, possibly due to reduced physiological reserve to handle any further resistance to outflow in these patients. However, no case required emergent surgery in the case series.⁶

In our patient, even with maximal medical treatment as well as multiple anterior chamber paracentesis, IOP was persistently high at over 30 mm Hg for over 2 weeks, making filtration surgery necessary as a definitive treatment. The severity and persistence of the raised IOP in this case was also exceptional compared with that in other reported cases, where the pressure rise was usually transient and amenable to medical control. We postulate that this rise in IOP was contributed by multiple risk factors, namely aphakia, presence of compromised angle as evidenced by gonioscopy and history of raised IOP, high myopia and residual cortical material.

Ex-PRESS implantation was chosen instead of trabeculectomy for several reasons. While the former

shares similar success rate with trabeculectomy, it has also demonstrated a lower rate of early postoperative hypotony,⁷ which potentially reduces the risk of suprachoroidal hemorrhage in this susceptible patient with high myopia and aphakia.⁸ Furthermore, postoperative inflammation may have been reduced in our patient with the omission of trabeculectomy and iridotomy in the procedure.⁷ We also observed that, even after resolution of hypotony, the bleb formed again relatively quickly, without much adhesion to the underlying sclera, as in trabeculectomy.

In conclusion, attention to the presence of multiple risk factors for IOP spike after YAG capsulotomy is warranted, as medically recalcitrant increase in IOP may require surgical intervention. Ex-PRESS shunt is a treatment option that may have potential advantages in surgical management as in this case, where intraocular inflammation needed to be minimized and hypotony was anticipated.

Declaration

No conflicts of interests were declared by authors.

References

1. Stark WJ, Worthen D, Holladay JT, Murray G. Neodymium: YAG lasers. An FDA report. *Ophthalmology*. 1985;92:209-12.
2. Kraff MC, Sanders DR, Lieberman HL. Intraocular pressure and the corneal endothelium after neodymium-YAG laser posterior capsulotomy. Relative effects of aphakia and pseudophakia. *Arch Ophthalmol*. 1985;103:511-4.
3. Parrish RK 2nd, Slomovic A. Myopia and unexpected intraocular pressure elevations after neodymium-YAG capsulotomy. *Arch Ophthalmol*. 1985;103:1277.
4. Lynch MG, Quigley HA, Green WR, Pollack IP, Robin AL. The effect of neodymium: YAG laser capsulotomy on aqueous humor dynamics in the monkey eye. *Ophthalmology*. 1986;93:1270-5.
5. Gimbel HV, Van Westenbrugge JA, Sanders DR, Raanan MG. Effect of sulcus vs capsular fixation on YAG-induced pressure rises following posterior capsulotomy. *Arch Ophthalmol*. 1990;108:1126-9.
6. Lin JC, Katz LJ, Spaeth GL, Klancnik JM Jr. Intraocular pressure control after Nd:YAG laser posterior capsulotomy in eyes with glaucoma. *Br J Ophthalmol*. 2008;92:337-9.
7. Maris PJ Jr, Ishida K, Netland PA. Comparison of trabeculectomy with Ex-PRESS miniature glaucoma device implanted under scleral flap. *J Glaucoma*. 2007;16:14-9.
8. Chu TG, Green RL. Suprachoroidal hemorrhage. *Surv Ophthalmol*. 1999;43:471-86.