Treatment of Malignant Glaucoma With Contact Transscleral Cyclophotocoagulation

Five cases of pseudophakic malignant glaucoma were successfully treated with a single session of contact transscleral cyclophotocoagulation (CTCP) with diode laser (twenty 4-J spots over 360°, 1.5 mm posterior to the limbus) after failure of Nd:YAG laser hyaloidotomy and vitreolysis. All patients except for case 2 had a preoperative diagnosis of chronic angle-closure glaucoma. At the end of the follow-up, all the eyes had a well-controlled intraocular pressure (IOP), with no medications required in 3 cases and topical B-blocker prescribed in 2. No major side effects of laser treatment were observed.

Malignant glaucoma is a severe complication of anterior segment surgery mainly associated with eyes with angle-closure glaucoma. The term “malignant glaucoma” refers to a condition characterized by ocular hypertension with shallow or flat anterior chamber despite a patent iridotomy and a normal posterior segment anatomy. The condition is typically unresponsive to treatment with miotics, but it can be relieved by cycloplegics. Different pathogenic mechanisms may be involved, such as ciliovitreal or cilio-lenticular block, but there is general agreement about the presence of posterior diversion of aqueous flow, leading to anterior displacement of the hyaloid and secondary angle closure.1,2

The treatment of choice for malignant glaucoma is Nd:YAG laser anterior hyaloidotomy (through the pupil, in aphakic or pseudophakic eyes, after posterior capsulotomy, or through a peripheral iridectomy), possibly followed by Nd:YAG laser vitreolysis.3,4 When laser treatment is not possible or is ineffective, anterior pars plana vitrectomy is usually required.5,6

We report 5 cases of pseudophakic malignant glaucoma, defined as ocular hypertension with a shallow central anterior chamber, patent peripheral iridotomy, and no evidence of choroidal effusion or other uveal abnormalities on B-scan echographic examination.7,8 All patients were successfully treated with CTCP and diode laser after failure of Nd:YAG laser hyaloidotomy and vitreolysis.

Report of Cases. Case 1. A 79-year-old woman with nuclear cataract and chronic angle-closure glaucoma secondary to plateau iris, already treated with peripheral laser iridotomy, underwent extracapsular cataract extraction plus trabeculectomy with placement of a posterior chamber 7-mm intraocular lens (IOL) in the left eye. Preoperative axial length was 21.60 mm.

The first day after surgery her IOP was 37 mm Hg and the anterior chamber was shallow, with peripheral iridolenticular contact. Therapy with 1% atropine sulfate and 0.2% dexamethasone sodium phosphate 6 times a day, 0.5% timolol maleate twice daily, and 0.5% apraclonidine hydrochloride and 125 mg of acetazolamide 3 times a day was started, but her IOP was 40 mm Hg on the next day and the anterior chamber was more shallow. The Nd:YAG laser iridotomy was repeated and a posterior capsulotomy was done. On day 4 (after cataract surgery), the IOP was 40 mm Hg and the iridoendothelial touch was complete, with a thin prelenticular aqueous layer and corneal edema. After unsuccessful Nd:YAG hyaloidotomy, the patient refused consent to vitrectomy. Contact transscleral cyclophotocoagulation was performed on day 7 (after the initial surgery), sparing the upper quadrant to allow restoration of filtration, although no bleb was visible. We used a diode laser (Optikon EOS 3000, Rome, Italy) with 2.6-W maximum nominal output power coupled with a 400-µm diameter optic fiber ending with a 3-mm focusing tip. After retrobulbar anesthesia, twenty 4-J spots (exposure time was set according to the fiber transmission measured with the built-in laser meter) were delivered holding the probe perpendicular to the scleral surface and placing its center 1.5 mm posterior to the limbus with firm indentation.9

On day 8 after the initial surgery, the IOP was 8 mm Hg, the anterior chamber was shallow, but the iridocorneal touch had disappeared, and a filtering bleb had formed. Her medications were gradually tapered. One month postoperatively after the initial surgery, the IOP was 12 mm Hg, the anterior chamber was deep, and the filtering bleb was evident. When last examined, 22 months after CTCP, the IOP was 12 mm Hg with no medications.

Case 2. A 67-year-old woman underwent bilateral extracapsular cataract extraction with peripheral iridectomy and placement of a 7-mm posterior chamber IOL at another hospital. Two weeks after surgery she developed a bilateral flat anterior chamber that was diagnosed as malignant glaucoma. Nd:YAG laser hyaloidotomy was unsuccessful and 6 weeks later she was referred to our center.

Best-corrected visual acuity was 4/200 OU. The IOP was 36 mm Hg in the right eye and 33 mm Hg in the left eye while receiving 0.5% timolol maleate twice daily, 0.5% apraclonidine hydrochloride 3 times a day, 250 mg of acetazolamide 3 times a day, 1% atropine sulfate three times a day, and 0.2% dexamethasone sodium phosphate 4 times a day. The irides were touching the corneas with a thin prelenticular aqueous layer. Early bullous keratopathy was present. Bilateral vitrectomy was judged hazardous. Because the left eye previously had better vision, we performed vitrectomy in the left eye and CTCP in the right eye.

Three days later, the anterior chamber was formed and the IOP was 10 mm Hg in the right eye and 8 mm Hg in the left. Her medications were tapered. Three weeks later, her IOP was 14 mm Hg in the right eye and 18 mm Hg in the left while receiving 0.5% timolol maleate twice daily, 0.2% dexamethasone sodium...
phosphate twice daily, and 1% atropine sulfate 3 times a day. Three months after CTCP, her best-corrected visual acuity was 20/100 OD and 20/150 OS. The IOP was 14 mm Hg in the right eye and 26 mm Hg in the left, while receiving 0.5% timolol maleate twice daily, 1% atropine sulfate twice daily, and 0.1% clobetasone butyrate twice daily. The anterior chamber was shallow in both eyes in the periphery, due to peripheral anterior synechiae, more evident in the left eye, but its central depth was 1.3 mm in the right eye and 1.2 mm in the left according to ultrasound biomicroscopy measurement. Her left eye was treated with CTCP. Her medications were tapered and 1 month later the visual acuity was unchanged and the IOP was 13 mm Hg in both eyes while receiving 0.5% timolol twice daily only.

Case 3. A 74-year-old man with chronic angle-closure glaucoma previously treated with laser iridotomy and compensated with 0.5% betaxolol hydrochloride twice-daily therapy underwent peripheral superior iridectomy and extracapsular cataract extraction with implantation of a 7-mm posterior chamber IOL in the left eye. Preoperative axial length was 20.74 mm. On day 2, the IOP was 35 mm Hg and the anterior chamber was flat. Since initial posterior subcapsular lens opacity was already present, and corneal touch was likely to precipitate cataract and corneal decompensation, we performed phacoemulsification and 5-mm posterior chamber IOL implant.

Preoperative axial length was 21.08 mm. On postoperative day 7, the IOP was 22 mm Hg with peripheral iridoendothelial contact impairing filtration (Figure 4). Treatment with 1% atropine sulfate and 0.2% dexamethasone sodium phosphate 6 times a day, Nd:YAG laser capsulotomy, and 2 sessions of Nd:YAG laser anterior hyaloidotomy was unsuccessful. On day 12, CTCP was performed, sparing the upper quadrant to maintain filtration. By day 14, the iridocorneal touch had resolved and a small filtering bleb was present. The IOP was 17 mm Hg. His medications were gradually tapered over the following weeks. Five months after the initial surgery, his IOP was 11 mm Hg with no medications.

Case 5. A 64-year-old woman with chronic angle-closure glaucoma underwent iridectomy and phacotrabeculectomy in the right eye with implantation of a 5-mm posterior chamber IOL. Preoperative axial length was 21.48 mm. On postoperative day 2, her best-corrected visual acuity was 20/30 with −0.75 diopters (D) and the IOP was 8 mm Hg. On day 7, the IOP was 12 mm Hg, the anterior chamber was shallow and the refraction had shifted to −3.50 D. Treatment with 1% atropine sulfate 6 times a day was prescribed. Despite a wide patent iridectomy, the iris was bombé in the lateral and inferior sectors. Two additional peripheral Nd:YAG iridotomies were done, following which vitreous oozed from the new openings, forming 2 small collections in the newly deepened anterior chamber. The refraction was −0.25 D, the IOP was 13 mm Hg, and a small filtering bleb was present. The patient continued therapy with 1% atropine sulfate 3 times a day.
Ten months later, the patient developed marked allergy to atropine and was switched to treatment with 1% cyclopentolate hydrochloride 3 times a day. One week later, the IOP was 38 mm Hg, the bleb was absent, and the best-corrected visual acuity had worsened to 20/200 with −4.00 D. Ultrasound biomicroscopy showed marked flattening of all ciliary structures and a shallow anterior chamber (Figure 5). Posterior Nd:YAG capsulotomy and 2 sessions of hyaloidotomy were unsuccessful. Three days later the patient underwent CTCP in the inferior sectors. One week after CTCP, the IOP was 18 mm Hg, a small bleb was present again and vision was restored. Five months later, the IOP was 20 mm Hg with a deep anterior chamber while receiving 0.5% timolol twice daily (Figure 6).

Comment. We successfully used diode laser CTCP to treat 5 patients with malignant glaucoma in whom anterior hyaloidotomy had failed. In case 1 we used CTCP as a final alternative when the patient refused to consent to vitrectomy. In case 2 we preferred not to attempt bilateral intracocular surgery; this case allowed us to compare the outcome of diode laser CTCP with anterior vitrectomy. Encouraged by these 2 successes we managed 3 more cases of malignant glaucoma refractory to hyaloidotomy, and all were resolved by CTCP.

The pathogenesis of malignant glaucoma is not clear, but posterior aqueous diversion seems involved, either with or without ciliolenticular (or ciliovitreal) block from apposition of the ciliary processes to the lens equator (or anterior hyaloid). Treatment normally includes aqueous suppressants, cycloplegics, and corticosteroids to break ciliary block, hyperosmotic to reduce vitreous volume, anterior Nd:YAG hyaloidotomy and vitrectomy to evacuate vitreous pockets, and anterior vitrectomy. Laser photocoagulation of the ciliary processes has been proposed in cases where a wide basal iridectomy is present. Other suggested treatments include posterior sclerotomy with fluid aspiration from the vitreous and air injection in the anterior chamber, lens extraction, and pars plana tube insertion following vitrectomy. The events leading to resolution of malignant glaucoma appear to involve (1) reduction of aqeous production (via medical therapy), (2) posterior rotation of ciliary processes (as a result of cycloplegia), and (3) restoration of normal aqueous flow patterns (by Nd:YAG laser hyaloidotomy or anterior vitrectomy). In understanding how CTCP may help in the resolution of malignant glaucoma, it is unlikely that CTCP may act on the vitreous. The mechanism of action could involve the reduction of aqueous production secondary to ciliary body ablation, but this is unlikely to be the sole reason, since even maximal aqueous suppression was ineffective. The success of CTCP may therefore be attributed to the posterior rotation of ciliary processes secondary to coagulative shrinkage, which also eliminates the abnormal vitreociliary relationship. Transscleral cyclophotocoagulation has been reported to cause malignant glaucoma in 1 case, however, in that case no Nd:YAG hyaloidotomy had been performed beforehand, and the treatment had been performed with a noncontact Nd:YAG technique. The results from our 5 cases raise the possibility that if normal aqeous flow cannot be restored by breaking the anterior hyaloid or presumed vitreous pockets with Nd:YAG laser application(s) in malignant glaucoma, diode laser CTCP could be considered before vitrectomy. Further investigation will help establish the mechanism of this treatment modality and provide more data on its efficacy.

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A Retained Intraocular Surgical Needle 2 Years After Cataract Extraction

Cystoid macular edema (CME) occurs not infrequently after cataract extraction. Various theories exist as to the cause of this phenomenon, including vitreous traction on the macula and uveal inflammation causing a disruption of the blood-retina barrier. Persistent irritation to the iris and anterior uvea may serve to promote or exacerbate this condition, and in part may help explain the observation that CME occurs more frequently in patients with anterior chamber intraocular lenses (IOLs) as compared with patients with posterior chamber IOLs. In this report we describe a patient with a retained intraocular suture needle after cataract extraction, in association with chronic CME unresponsive to medical therapy.

Report of a Case. A 90-year-old patient was seen at our service 2 years after a cataract extraction and IOL placement. He had had poor vision since his surgery. His ocular history was otherwise unremarkable, and his medical history was significant for diet-controlled diabetes mellitus.

His best-corrected visual acuity was 20/400 OD and 20/50 OS. His pupils, extraocular motility, and ocular adnexa were normal. Slitlamp examination of his right eye showed scattered deposits on the corneal endothelium consistent with previous inflammation, a well-placed anterior chamber lens, and a metallic foreign body between the IOL and the iris at the 3-o’clock position (Figure 1). Examination results of his left eye were only remarkable for a moderate nuclear sclerotic cataract. Intraocular pressures were normal in both eyes. Dilated fundus examination showed severe chronic CME in his right eye, and this was confirmed with fluorescein angiography.

Given the long-standing CME and its poor response to topical steroid and nonsteroidal anti-inflammatory agents, the patient was offered pars plana vitrectomy as well as removal of the foreign body. A standard 3-port pars plana vitrectomy was performed. The superotemporal sclerotomy was then enlarged slightly with a microvitreoretinal blade. An intraocular forceps was then inserted through the superotemporal sclerotomy and passed anteriorly through the pupil. With upward pressure on the posterior surface of the IOL, the foreign body was grasped and withdrawn. Further inspection of the foreign body showed it to be a needle to a 10-0 suture (Figure 2). Postoperatively, the patient was prescribed topical steroids and antibiotic drops and cycloplegia was maintained. The macular edema improved and he had a visual acuity of 20/160 OD after 4 weeks of follow-up.

Comment. While the patient was at risk for CME given his complicated cataract surgery and anterior chamber IOL, chronic uveal irritation from an intraocular needle may have served as an aggravating factor. Fortunately, retained needles occur uncommonly in surgical practice and, to our knowledge, are unreported after intraocular surgery. Metal fragments, however, have been found in the anterior chamber after phacoemulsification, presumably left behind by the phaco tip. The physiological consequence of these retained particles is not known.

In summary, retained surgical needles may occur after intraocular surgery and may be associated with a poor visual outcome. Removal of retained surgical material may be indicated in selected cases.

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