

Surgical Management of Neovascular Glaucoma

Robert F. Haverly, MD, FACS

Abstract: Neovascular glaucoma is a vision- and eye-threatening disease that carries a poor prognosis. Understanding the cause of neovascular glaucoma with early diagnosis and treatment is important for a positive outcome. There are numerous surgical treatment options available, but to have the best outcome, deciding which surgical technique to use is important. Some patients are better candidates than others for the various surgical techniques. An early- or late-stage clinical presentation, as well as the other ocular conditions associated with neovascular glaucoma, will dictate the appropriate surgical plan. This article provides a step-by-step approach to surgically managing neovascular glaucoma and explains new surgical techniques to help with the best chance of a positive patient outcome.

(*Tech Ophthalmology* 2010;8: 87–93)

Neovascular glaucoma is a serious complication of systemic and retinal disease. The prognosis is poor, and early, aggressive treatment is a must to achieve the best outcome. Many eyes with neovascular glaucoma eventually need an enucleation, but the incidence of enucleation for neovascular glaucoma has gradually been on the decline.¹ Early detection and better medical and surgical techniques are most likely the reason for improved outcomes. Unfortunately, it is all too common to first see a patient with a red, painful eye, with vision loss, and a pressure in the 60- or 70-mm Hg range. Although, the prognosis is generally poor, some cases can have a surprisingly good outcome. To help in the diagnosis of neovascular glaucoma, early screening for patients at high risk is important. Examining the iris for rubeosis and the angle and the retina for neovascularization is important in the screening process. Understanding the systemic diseases associated with glaucoma is a must to have a heightened suspicion for neovascular glaucoma. Vascular diseases such as diabetes, hypertension, heart disease, and peripheral vascular disease are common underlying causes of neovascular glaucoma. Ischemia to ocular tissue is theorized to be the genesis of neovascular glaucoma.² The most common causes of neovascular glaucoma related to the systemic diseases include ischemic central retinal vein occlusion, proliferative diabetic retinopathy, and carotid artery disease associated with ocular ischemic syndrome. Other causes of neovascular glaucoma include hemiretinal and branch retinal vein occlusions, branch or central retinal artery occlusion, and giant cell arteritis. Treatment of the ocular ischemia associated with these conditions can reduce the incidence of neovascular glaucoma.²

Neovascular glaucoma develops from the induced hypoxia from ischemic retinal disease, which releases vascular endothelial growth factor (VEGF), a vasoproliferative substance that acts upon healthy endothelial cells of viable capillaries to stimulate the formation of a fragile meshwork of vessels (neo-

vascularization). In extreme retinal hypoxia, there are few viable retinal capillaries. In this instance, VEGF is theorized to diffuse to the nearest area of viable capillaries, which in many cases is the posterior iris. Neovascularization develops from these capillaries and progresses along the posterior iris, into the pupil, anterior iris, and also into the angle of the eye.³ Once in the angle, the neovascularization develops a fibrovascular support membrane that acts to both physically block the angle as well as bridge the angle that blocks the trabecular meshwork. Peripheral anterior synechiae with permanent angle closure can happen quickly. This results in secondary angle closure glaucoma without papillary block. Blood in the angle and a hyphema are common at this stage. The intraocular pressure can increase to extreme levels greater than 50 mm Hg. At this stage, the diagnosis is readily apparent, but the clinical presentation may be more subtle in other cases.⁴

CLINICAL PRESENTATION

Early Stage

Early signs and symptoms of neovascular glaucoma may only include rubeosis and/or mild vascularization of the iris without vision loss or pain. There may be minimal visual symptoms associated with the presentation at this time, so other common causes of neovascular glaucoma such as chronic carotid artery occlusive disease,^{5,6} sickle cell disease,⁷ neoplasias,^{8–11} Fuchs heterochromic iridocyclitis,¹² and pseudoexfoliation syndrome¹³ must be ruled out. It is difficult to predict how long it takes to progress from the early signs of neovascular glaucoma to the advanced stage.

Advanced Stage

Advanced stage of neovascular glaucoma can occur rapidly from the early stage. Once neovascularization of the angle occurs, the fibrovascular meshwork prevents aqueous drainage, which causes a rapid and profound increase in intraocular pressure.^{2,4} The patient's vision blurs; conjunctival injection and pain ensue. At this stage, there are corneal edema and anterior chamber reaction with or without red blood cells in the anterior chamber (Figs. 1 and 2). Anterior and posterior synechiae develop. If the patient is phakic, a cataract may form or progress. A vitreous hemorrhage may develop, which makes visualization of the retina and optic nerve difficult, but ischemia of the retina and optic nerve will be present.

TREATMENT AND SURGICAL TECHNIQUES

Initially, when a diagnosis of neovascular glaucoma is made, a treatment plan and prognosis need to be discussed with the patient and family. The patient's overall health and underlying cause of neovascular glaucoma need to be under consideration while developing a treatment plan. The eye may have no hope of visual recovery and become blind and painful. The treatment plan in this case will be different than a patient in fairly good health, with the ability to perform laser ablation early. The entire clinical picture needs to be viewed from a "top-down" approach.

From the Laser Eye Surgery of Erie, Erie, PA.
Address correspondence and reprint requests to Robert F. Haverly, MD,
FACS, Laser Eye Surgery of Erie, 311 W 24th St, Suite 401, Erie, PA
16502 (e-mail: rhaverly@erielasereye.com).
Copyright © 2010 by Lippincott Williams & Wilkins

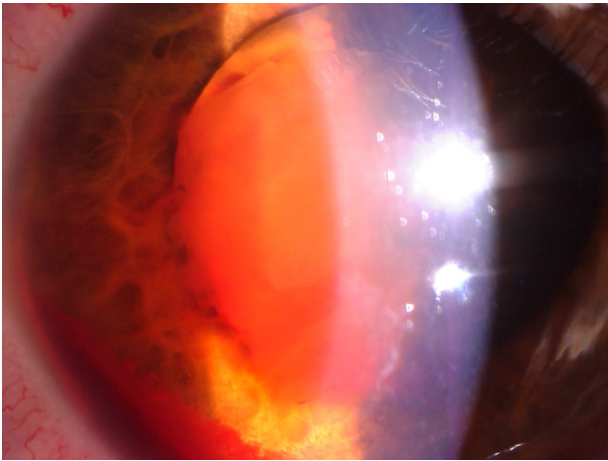


FIGURE 1. Slit-lamp photograph of an eye with neovascular glaucoma showing significant corneal edema, a hyphema, anterior chamber fibrin, and rubeosis.

Ophthalmologists and their patients are fortunate to have many different treatment modalities at their disposal. In deciding an initial treatment, the first order is to determine if the retina can be visualized, and if there is a clear view for pan retinal photocoagulation. If the answer is yes, this should be done as soon as possible.^{14,15} If this cannot be performed, which happens very often in the advanced stage, it is important to know the underlying cause of the neovascular glaucoma. If it is from an underlying tumor, treatment will be different. In cases of a uveal melanoma, retinoblastoma, or metastatic disease, an enucleation may be the best choice, but radiation, chemotherapy, and/or a radioactive plaque may also be an option. If there is no visual potential and no tumor is present, transscleral diode laser, transscleral cryotherapy, enucleation, and retrobulbar injection of alcohol are all possible options.

If the retina is visible, perform panretinal photocoagulation. I find it best to give a retrobulbar block and perform panretinal photocoagulation 360 degrees from the arcades and as far into the periphery as possible with a Rodenstock, gonioscopes, or other wide-field lenses. Many times, the pupil will not dilate well, and the peripheral retina is difficult to ablate, but do as much as possible. I prefer to administer an intravitreal injection of an anti-

VEGF agent (bevacizumab [Avastin] or ranibizumab [Lucentis]; Genentech) after the retinal ablation. The patient is given a β -blocker, acetazolamide, α -blockers, and prostaglandin drops. If possible, oral carbonic anhydrase inhibitor is started. In some cases, this may be all the treatment necessary to reduce the intraocular pressure and treat the neovascular glaucoma.

Follow up on the patient closely, and if the intraocular pressure is still elevated, a trabeculectomy with mitomycin C, minishunt, glaucoma implant, or endocyclophotocoagulation should be performed. If possible, stop anticoagulant therapy before surgery. Neovascular eyes have a great propensity for intraocular bleeding during and after surgery, which will negatively affect the outcome. It is important to note not to perform intraocular surgery before panretinal photocoagulation, unless one can perform a panretinal ablation at the time of surgery. The sudden drop in intraocular pressure will cause the fragile neovascular vessels to bleed severely. It is also important to avoid iris manipulation as much as possible and not to perform a peripheral iridotomy with a laser to treat the angle closure. It is acceptable to perform an iridectomy with the trabeculectomy once there is adequate panretinal photocoagulation or retinal ablation. The iris associated with neovascular glaucoma is very vascular and thickened as shown in Figure 3. If intraocular surgery is needed to lower the intraocular pressure, I prefer the Ahmed valve (New World Medical, Rancho Cucamonga, Calif), because there is no iris manipulation, and the postoperative intraocular pressure usually does not drop as low as other glaucoma implants, minishunts, or trabeculectomies. A low postoperative pressure (<10 mm Hg) usually is associated with more extensive intraocular hemorrhage. The valve mechanism within the Ahmed valve helps prevent hypotony. Keeping viscoelastic in the eye during surgery and not removing the viscoelastic material from the anterior chamber at the end of the surgery help prevent postoperative hypotony. Figures 4 to 8 display a step-by-step placement of the Ahmed valve.

Another option is endoscopic cyclophotocoagulation (ECP) of the ciliary body (EndoOptiks, Little Silver, NJ). There are numerous advantages to this, including minimal manipulation of the conjunctiva, which can be very difficult to work with when performing a trabeculectomy or placement of a glaucoma implant in a patient with active neovascular glaucoma. There is no manipulation of the iris with ECP, and ECP can be performed with an undilated pupil. With the exception of

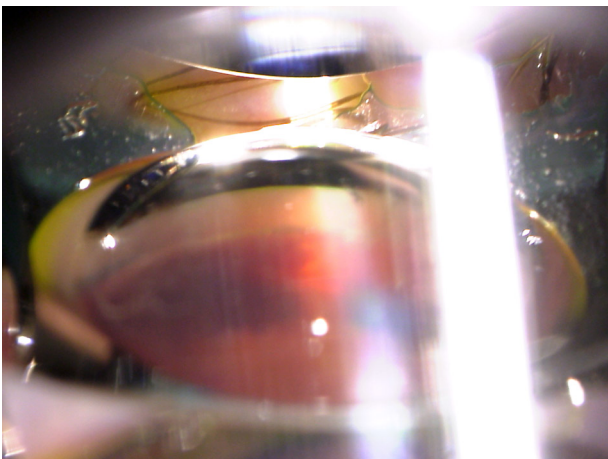


FIGURE 2. Gonioscopic view of blood and neovascularization of the angle of the same eye as in Figure 1.

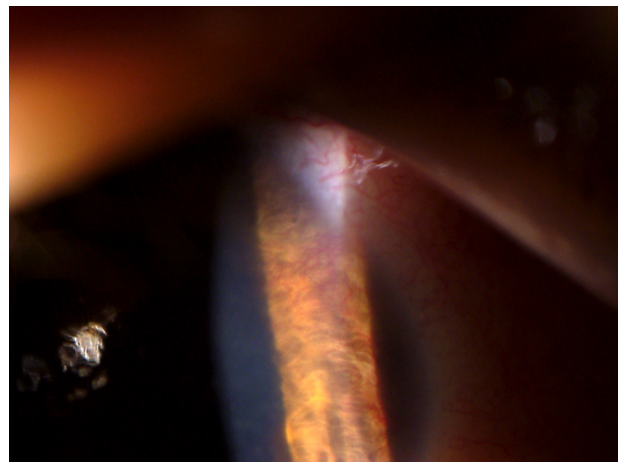


FIGURE 3. Biomicroscopic view of an eye with neovascular glaucoma displaying a very vascular and thickened iris. Surgical manipulation of this iris will usually cause bleeding.

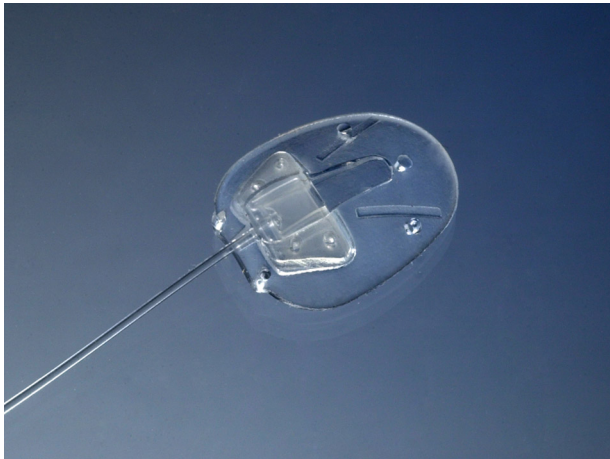


FIGURE 4. Photograph of a flexible plate (FP7) Ahmed valve.

making the incisions during surgery, there is no abrupt drop in intraocular pressure.

If panretinal photocoagulation is not possible because of a limited view, there are several options. Transscleral cycloablation can be performed with or without a glaucoma implant. However, transscleral diode laser cyclophotocoagulation and transscleral cryotherapy are very destructive to the eye and vision.^{16,17} Usually, vision is lost after this type of treatment, and hypotony and phthisis can ensue. Another option is removing the opacity that is preventing panretinal photocoagulation such as cataract extraction and/or vitreous hemorrhage and then performing panretinal photocoagulation as well as placement of a glaucoma implant or a trabeculectomy. However, in many cases, the pupil will still need to be surgically dilated, and this leads to additional intraocular hemorrhaging. If the cornea is clouded or scarred, this approach is even more difficult. It is not unusual in

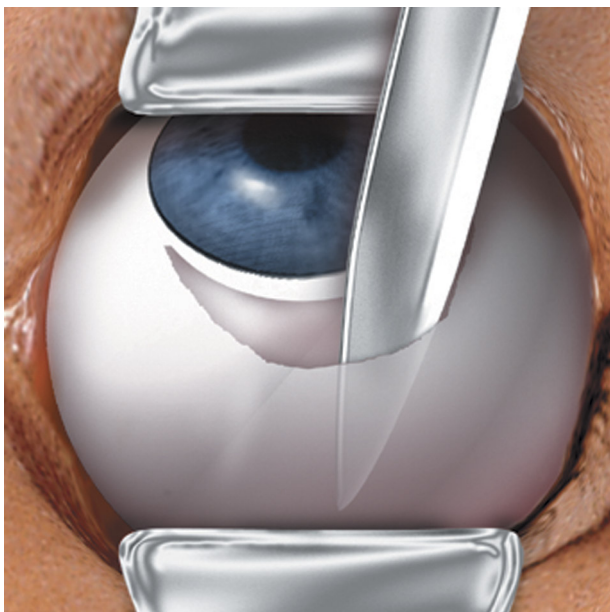


FIGURE 5. A preferred location for the seton is in the superior, temporal quadrant where a large peritomy is created using blunt Wescott scissors. The conjunctival pocket is deepened with tenotomy scissors in a reverse cutting fashion.

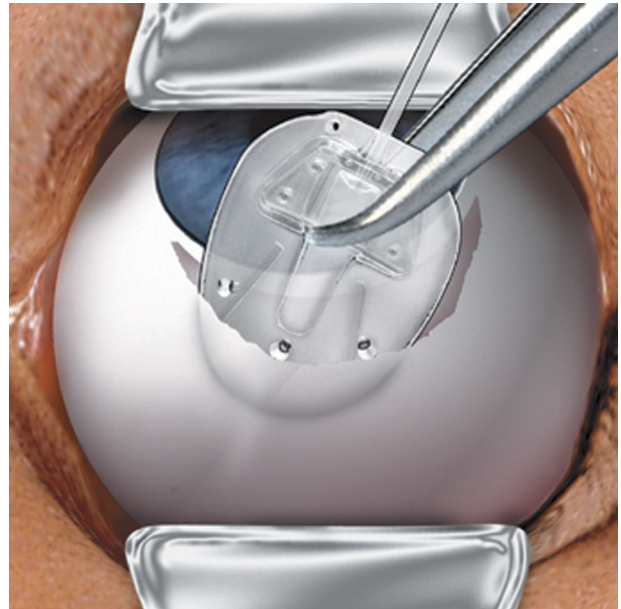


FIGURE 6. The seton is placed into the conjunctival pocket and sutured with 8-0, 9-0, 10-0 Nylon between 8 and 10 mm from the limbus between the rectus muscles. A lateral relaxing incision of the conjunctiva helps to provide visualization for suturing. It is very important to prime the implant with balance salt solution or sterile water through the drainage tube with a blunt 26 to 30 gauge cannula before implantation of the seton.

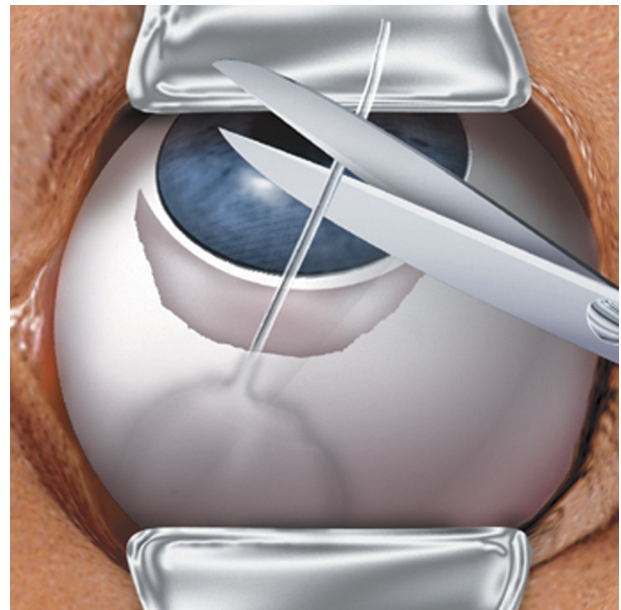


FIGURE 7. The tube is cut to the appropriate length so that it does not block the visual axis but is far enough into the eye to remain in the anterior chamber. A scleral tunnel approximately 2.0 mm from the limbus with a 23-gauge needle creates the track for the tube. The tube can be anchored with 8-0 or 9-0 Nylon at its scleral insertion. Viscoelastic placed in the anterior chamber through a paracentesis site helps with tube placement.

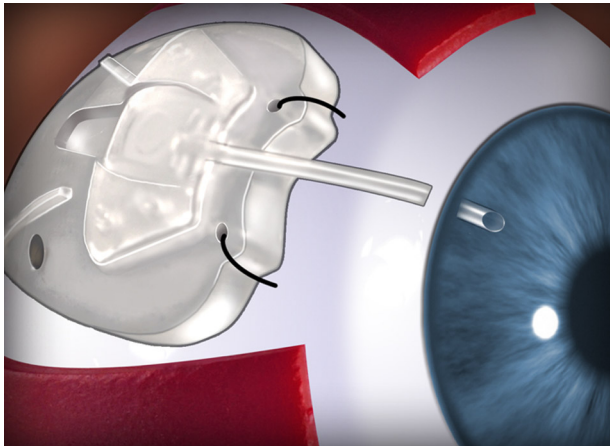


FIGURE 8. This shows the final placement of the seton. A scleral patch graft is anchored over the tube with 10-0 or 9-0 nylon, and the conjunctiva is then sutured to the limbal conjunctiva to completely cover the scleral patch and seton usually with 7-0 or 8-0 Vicryl sutures.

patients with advanced neovascular glaucoma to have several of these conditions preventing adequate panretinal ablation. Endoscopic cyclophotocoagulation may be a better choice.

Case

Figure 9 shows the eye of a 70-year-old woman with neovascular glaucoma. She had a central retinal vein occlusion, and the referring ophthalmologist could perform only a partial panretinal photocoagulation because of a poor view of the retina. The patient was in pain, and the intraocular pressure was 68 mm Hg. The cornea, iris, and angle had vascularized, and there was blood in the angle. There was a very poor view into the eye due to corneal scarring, edema, poor pupillary dilation, and a vitreous hemorrhage. There was a lens implant with some posterior capsular opacification. It would be difficult and may be impossible to surgically dilate the pupil, perform a pars plana vitrectomy with endolaser, and place a glaucoma implant. My

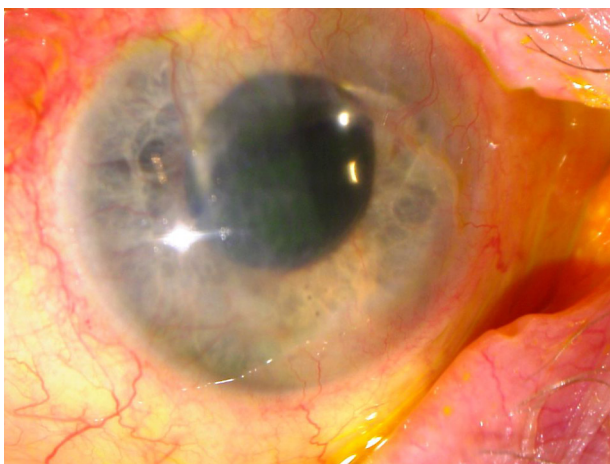


FIGURE 9. Photograph of a patient's eye with neovascular glaucoma showing significant corneal scarring and vascularization. Phenylephrine 2.5% and tropicamide 1% were placed before the photograph. Note the poor pupillary dilation secondary to the lens-iris synechiae and the large vessels associated with rubeosis.

preferred approach in cases with compromised view is to perform an endoscopic pars plana vitrectomy with endolaser of the retina and of the ciliary body. This eliminates the need for a clear view into the eye through the pupil. The endoscope provides a clear view around the opacities and avoids having to stretch and dilate a rubeotic pupil. I use the 23-gauge vitrectomy system (Alcon). The conjunctiva is rotated anteriorly with serrated-tip forceps, and the 23-gauge trocar and sleeve are inserted at a 10-degree angle initially and halfway into the sclera turned 90 degrees. These techniques are used to help create a self-sealing entry. A 23-gauge infusion port is placed in the inferior temporal quadrant, and a 23-gauge sleeve is placed in the superior temporal quadrant. They are placed 3.5 mm from the limbus if the patient is pseudophakic and 4.0 mm from the limbus if the patient is phakic. A larger 18- to 19-gauge port is necessary to insert the endoscopic laser. With the infusion on in the inferior port, I usually start with the straight endoscope placed into 18-gauge opening. Place the Occutome (Alcon) in the superior 23-gauge sleeve and perform an endoscopic vitrectomy (Figs. 10–12). Once a good core endoscopic vitrectomy is complete, the same endoscopic probe is used to perform the retinal ablation. The settings on the laser usually range from 600 to 800 mW. The size and the intensity of the laser spot are also dependent on the distance the probe is from the retina. The size of the laser spot is adjusted by bringing the probe closer or farther away from the retina. The spot size is larger than the usual endolasers fitted for the 23-gauge sleeve, so there are not as many laser spots needed. However, the same concept of treatment applies. Treat the retina until it lightly whitens, and then move to the next area. When the panretinal photocoagulation is complete, the curved endoscopic tip is used to perform treatment of the ciliary body and the processes to the pars plana (Figs. 13–16). The power settings are set between 200 and 300 mW of laser power. The intensity and size of the spot are dependent on the distance from the ciliary body as well. It is not necessary to press up and down on the foot peddle to create a new spot. By pressing continuously on the foot peddle and moving the probe closer and farther away from the ciliary processes or retina, 1 laser spot after another is created. A 180-degree ablation of the ciliary body is usually enough to control the intraocular pressure without creating hypotony. In neovascular

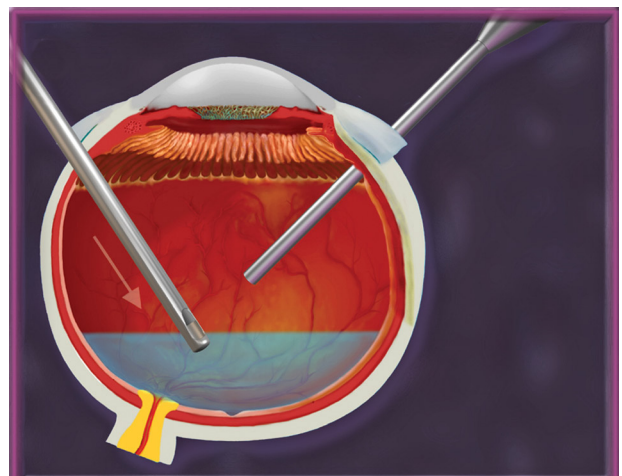


FIGURE 10. The endoscopic probe is on the right through the pars plana providing visualization of the vitrector on the left. While doing the vitrectomy, the monitor connected to the endoscopic system, not the microscope, is used to view the procedure.

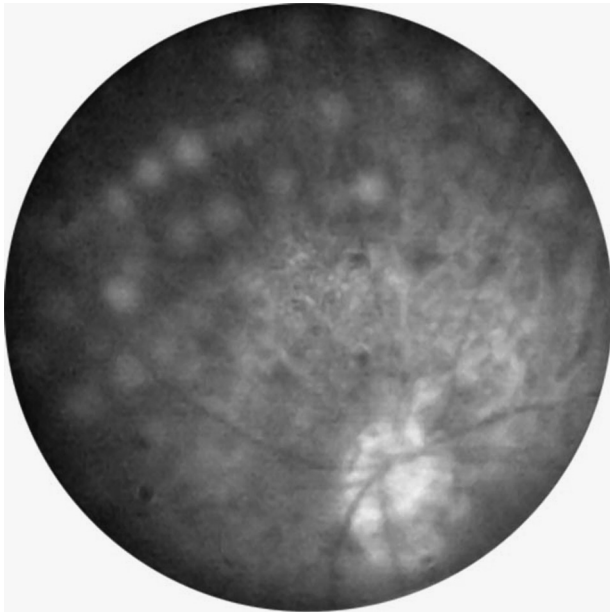


FIGURE 11. The image through the endoscope showing panretinal photocoagulation of the posterior retina with the optic nerve visible.

glaucoma, the ciliary processes are already damaged, and 180-degree treatment is usually all that is needed for adequate intraocular pressure control.^{18,19} Other glaucomas do not respond as well to ECP and usually require more than 270 degrees of treatment. When ablating the ciliary body, treat until it whitens, shrinks, and contracts, but avoid gas bubble formation and audible “popping”. I like to also treat the ciliary processes to the pars plana because these structures also produce aqueous fluid. When finished with the laser ablation, use the endoscope to check for any breaks in the retina and treat if necessary. The

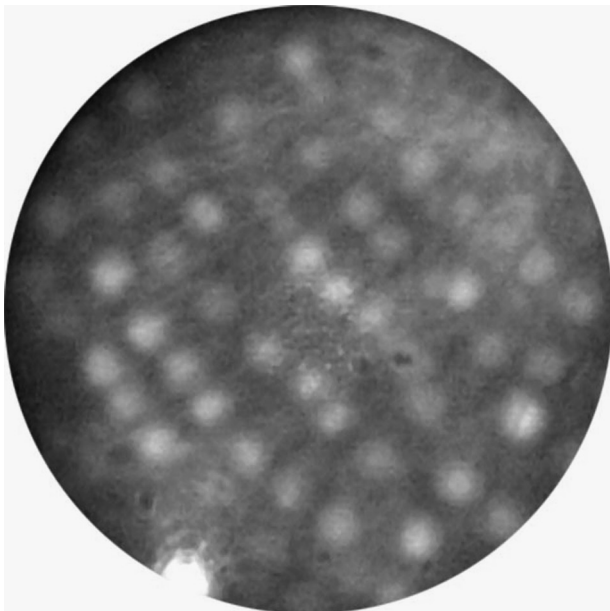


FIGURE 12. The image through the endoscope showing the completion of the panretinal photocoagulation with the tip of the Occutome (Alcon) visible.

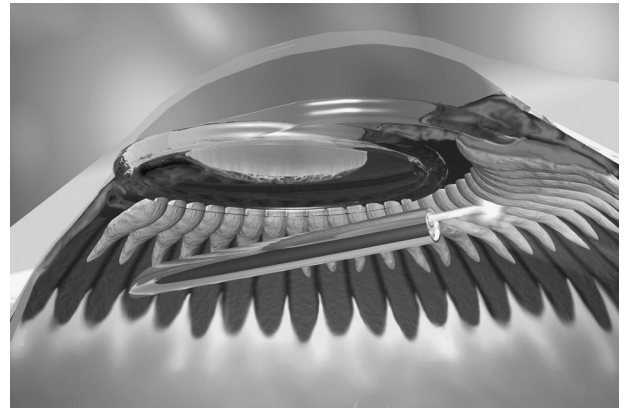


FIGURE 13. Schematic representation of the endoscopic probe ablating the ciliary processes through the pars plana. This is done while looking at the monitor connected to the endoscopic system. The intraocular lens is shown above the endoscopic probe.

endoscope can allow visualization 360 degrees and is quite helpful. Suture the port through which the endoscope was placed. I usually use 7-0 Vicryl. All wounds should be tested for vitreous and leakage. The 23-gauge sleeve ports may be self-sealing and do not require a suture to close the wound. An intravitreal injection of dexamethasone at the end of the case is useful to reduce postoperative inflammation. I find that it is not necessary to perform a trabeculectomy or place a seton valve. The postoperative intraocular pressure is usually well controlled with the ECP laser.

SURGICAL PEARLS

In neovascular glaucoma, the neovascularization needs to be treated first. Currently, there is no substitute for panretinal photocoagulation. Intravitreal

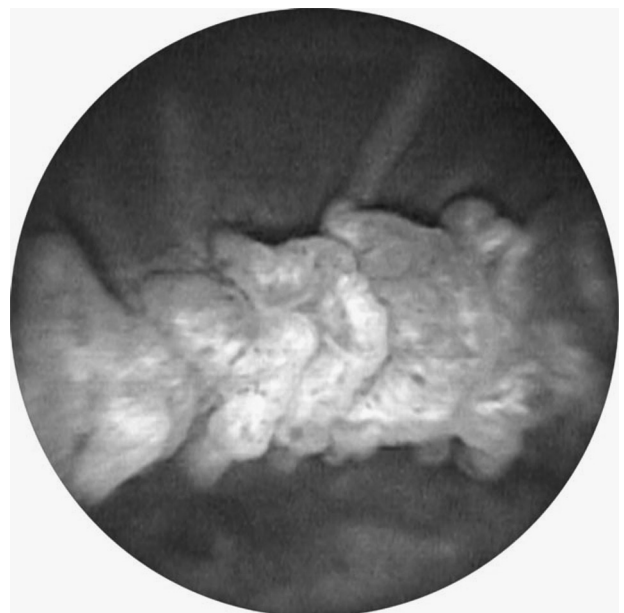


FIGURE 14. The endoscopic view of ciliary processes with hemorrhage near the ora serrata.

injections of anti-VEGF may help reduce the vascular response, but the results have been mixed.²⁰⁻²³ I do give an intravitreal injection of an anti-VEGF (Avastin or Lucentis) immediately after panretinal photocoagulation in many cases. Do not administer the intravitreal injection before panretinal photocoagulation because it may induce a vitreous hemorrhage, and it will make the retinal ablation difficult to perform.

It is very important not to perform an aqueous shunting procedure before adequate retinal ablation. The extreme drop in intraocular pressure will usually cause intraoperative and postoperative intraocular hemorrhaging that will complicate the condition further.

While doing the vitrectomy, a 23-gauge microincision vitrectomy with ECP works quite well to treat neovascular glaucoma. Smaller or larger endoscopic vitrectomy systems may also be used. While doing the endoscopic vitrectomy, different visual clues need to be used. One cannot rely on the usual shadowing effect with the light probe or chandelier lights to judge the distance of the Occutome from the retina. The smaller the shadow between the Occutome (Alcon) and the retina, the closer the retina is to the tip of the Occutome (Alcon). When doing an endoscopic vitrectomy, the surgery is performed while looking at the video monitor. The distance is judged based on the size of the image on the screen. The larger the image, the closer the probe tip is to the object. The distance of the Occutome (Alcon) from the retina is directly visualized on the video monitor not with shadowing. It

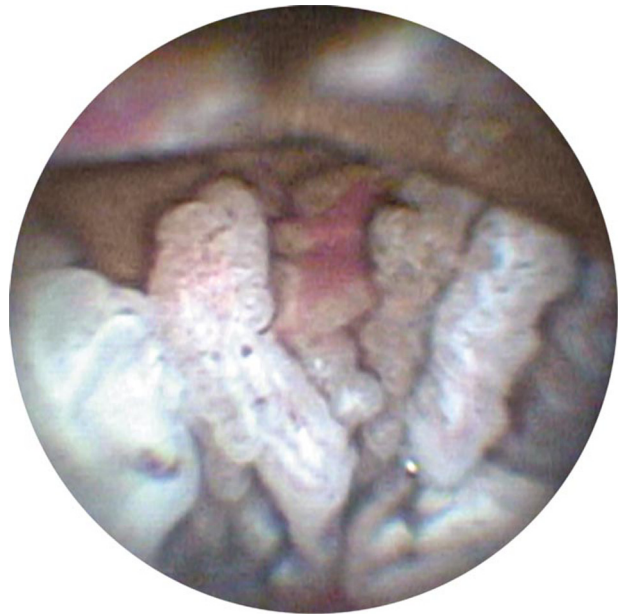


FIGURE 16. The endoscopic view of hemorrhage on the ciliary process surface migrating anteriorly from the vitreous cavity. This is the right distance from the probe to treat the processes.

is important to have the endoscope always pointing toward the Occutome (Alcon) while doing the vitrectomy portion of the surgery to be sure that the cutter is in a safe position.

The straight probe is initially easier to use than the curved endoscopic probe. The curve adds another variable that changes the orientation of the image. The image moves and rotates with clockwise or counterclockwise rotation of the probe as well as X-Y and Z movement of the tip of the probe.

DISCUSSION

Neovascular glaucoma is a vascular disease that usually results in secondary glaucoma. Treatment of neovascular glaucoma requires a blend of glaucoma and retinal specialties. The retinal vascular disease needs to be treated first followed by a glaucoma procedure. The prognosis is poor, and prompt diagnosis and treatment are needed to achieve the best outcome. The underlying cause of neovascular glaucoma is important to know because this will help direct appropriate treatment. Treatment of neovascular glaucoma secondary to a tumor is different than treatment for other causes. The most common causes of neovascular glaucoma that most ophthalmologists will encounter are retinal vascular disorders, diabetic retinopathy, and carotid artery occlusive disease.²⁴

Neovascular glaucoma responds poorly to standard glaucoma procedures, but the success rate can be improved by choosing the most appropriate surgical treatment for each individual case. However, on average, a trabeculectomy with mitomycin C for neovascular glaucoma has a success rate of about 50%,²⁵ and for a glaucoma implant in a similar setting has a success rate of about 60%.²⁶ There are some reports that intravitreal anti-VEGF may improve this success rate.²⁶ There are reports that the success rate of ECP in the setting of neovascular



FIGURE 15. The endoscopic view of treated and untreated ciliary processes. The treated ciliary processes are on the left, which appear white.

glaucoma is near 90%,¹⁷ but in most settings, it is probably lower. I have found that all of the treatments can be used successfully to treat neovascular glaucoma, and results vary based on the underlying condition and severity of the disease. However, in patients who have a conjunctiva that is scarred and vascularized, a trabeculectomy or placement of a glaucoma implant may be difficult. Such cases may occur in patient who had previous scleral buckles, multiple eye surgeries, trauma, or chemical burns. Patients with neovascular glaucoma usually have similar conjunctival tissue. The best choice in many of these cases would be ECP.

ACKNOWLEDGMENTS

Figures 3 to 8 are courtesy of New World Medical (Rancho Cucamonga, Calif). Figures 10 to 16 are courtesy of EndoOptiks.

REFERENCES

- Setlur VJ, Parikh JG, Rao NA. Changing causes of enucleation over the past 60 years. *Graefes Arch Clin Exp Ophthalmol*. 2010;248(4):593–597.
- Sivak-Callcott JA, O'Day DM, Gass JD, et al. Evidence-based recommendations for the diagnosis and treatment of neovascular glaucoma. *Ophthalmol*. 1980;87(5):400–402.
- Tripathi RC, Li J, Tripathi BJ. Increased level of vascular endothelial growth factor in aqueous humor of patients with neovascular glaucoma. *Ophthalmology*. 1998;105(2):232–237.
- Albert DM, Jackobeic FA. *Neovascular glaucoma. Principles and Practice of Ophthalmology, Clinical Ophthalmology* [book on CD-ROM]. Chapter 215.
- Abedin S, Simmons RJ. Neovascular glaucoma in systemic occlusive vascular disease. *Ann Ophthalmol*. 1982;14(3):284–287.
- Coppeto J, Wand M. Neovascular glaucoma and carotid vascular occlusion. *Am J Ophthalmol*. 1985;99(5):567–570.
- Galinos S, Rabb MF, Goldberg MF, et al. Hemoglobin SC disease and iris atrophy. *Am J Ophthalmol*. 1973;75(3):421–425.
- Ellett EC. Metastatic carcinoma of choroid. *Am J Ophthalmol*. 1946;29(12):1585.
- Sullivan ST, Dallow RL. Intraocular reticulum cell sarcoma. *Ann Ophthalmol*. 1977;9(4):401–406.
- Ferry AP, Font RL. Carcinoma metastatic to the eye and orbit. *Arch Ophthalmol*. 1975;93(7):472–482.
- Shields MB, Proia AD. Neovascular glaucoma associated with an iris melanoma: a clinicopathologic report. *Arch Ophthalmol*. 1987;105:672–674.
- Perry HD, Yanoff M, Scheie HG. Rubeosis in Fuchs heterochromic iridocyclitis. *Arch Ophthalmol*. 1975;93:337–339.
- Ringvold A, Davanger M. Iris neovascularization in eyes with pseudoexfoliation syndrome. *Br J Ophthalmol*. 1981;65:138–141.
- Laatikainen L. A prospective follow-up study of panretinal photocoagulation in preventing neovascular glaucoma following ischaemic central retinal vein occlusion. *Graefes Arch Clin Exp Ophthalmol*. 1983;220(5):236–239.
- Wand M, Dueker DK, Aiello LM, et al. Effects of panretinal photocoagulation on rubeosis iridis, angle neovascularization, and neovascular glaucoma. *Am J Ophthalmol*. 1978;86(3):332–339.
- Yildirim N, Yalvac IS, Sahin A, et al. A comparative study between diode laser cyclophotocoagulation and the Ahmed glaucoma valve implant in neovascular glaucoma: a long-term follow up. *J Glaucoma*. 2009;18(3):192–196.
- Uram M. Endoscopic cyclophotocoagulation in glaucoma management: indications, results and complications. *Curr Opin Ophthalmol*. 1995;6(2):19–29.
- Berke SJ. Endolaser cyclophotocoagulation in glaucoma management. *Techniques Ophthalmol*. 2006;4(2):74–81.
- Uram M. Ophthalmic laser microendoscope ciliary process ablation in the management of neovascular glaucoma. *Ophthalmology*. 1992;99(12):1823–1828.
- Yazdani S, Hendi K, Pakravan M, et al. Intravitreal bevacizumab for neovascular glaucoma: a randomized controlled trial. *J Glaucoma*. 2009;18(8):632–637.
- Avery RL. Regression of retinal and iris neovascularization after bevacizumab (Avastin) treatment. *Retina*. 2006;26(3):352–354.
- Iliev ME, Domig D, Wolf-Schnurrbursch U, et al. Intravitreal bevacizumab (Avastin) in the treatment of neovascular glaucoma. *Am J Ophthalmol*. 2006;142(6):1054–1056.
- Wakabayashi T, Oshima Y, Sakaguchi H, et al. Intravitreal bevacizumab to treat iris neovascularization and neovascular glaucoma secondary to ischemic retinal diseases in 41 consecutive cases. *Ophthalmol*. 2008;115(9):1571–1580.
- Al-Shamsi HN, Dueker DK, Nowilaty SR, et al. Neovascular glaucoma at King Khaled Eye Specialist Hospital—etiologic considerations. *Middle East Afr J Ophthalmol*. 2009;16(1):15–19.
- Takahara Y, Inatania M, Fuckushima M, et al. Trabeculectomy with mitomycin C for neovascular glaucoma: prognostic factors for surgical failure. *Am J Ophthalmol*. 2009;147(5):912–918.
- Eid TM, Radwan A, el-Manawy W, et al. Intravitreal bevacizumab and aqueous shunting surgery for neovascular glaucoma: safety and efficacy. *Can J Ophthalmol*. 2009;44(4):451–456.