THE GAME PLAN

FIVE POINTERS ON NEOVASCULAR GLAUCOMA WITH ACTIVE NEOVASCULARIZATION

Strategies for detection, intervention, and prevention of rapid damage from IOP elevation.


BY JAKE RADELL, MA;
AND JOSEPH F. PANARELLI, MD

Neovascular glaucoma (NVG) is a severe form of glaucoma that occurs secondary to vascular proliferation in the anterior chamber of the eye. Like other glaucomatous disease, NVG is characterized by increased IOP, which can lead to optic nerve damage and ultimately loss of vision. Unlike many other forms of glaucoma, NVG can cause rapid damage due to the severe IOP elevation that results.

In the vast majority of cases (~95%), NVG occurs secondary to other conditions that cause retinal ischemia, such as proliferative diabetic retinopathy (PDR), central retinal vein occlusion (CRVO), and carotid artery occlusive disease.[1] These conditions may lead to retinal ischemia, which stimulates the release of proangiogenic growth factors such as vascular endothelial growth factor (VEGF). The increase in angiogenic growth factors causes neovascularization to begin at the pupillary border and later invade the iridocorneal angle, where it disrupts aqueous outflow through the trabecular meshwork, thereby increasing IOP.

1. SUSPECT NVG IN ALL PATIENTS AT RISK FOR RETINAL ISCHEMIA

Typically, retinal ischemia is the precipitating factor for neovascularization of the angle (NVA) and for NVG. Early NVG is often asymptomatic, with patients developing ocular pain or vision loss only in later disease stages. In order to prevent NVG from silently progressing to vision loss, it is extremely important to perform gonioscopy and a careful slit-lamp examination of the iris in all patients at significant risk for ischemia, including those with poorly controlled diabetes, hypertension, arteriosclerosis, and especially active PDR or prior CRVO.

In early disease stages, tufted vessels may be visible at the pupillary margin. These vessels can be distinguished from the normal vasculature of the iris by their meandering growth pattern, which differs from the classically described radial distribution of iris vessels. NVA can be visualized as vessels that cross the scleral spur. As NVA progresses, new vessels become more prominent and fibrovascular membranes pull the angle closed.

2. TREAT ANTERIOR CHAMBER NEOVASCULARIZATION EARLY

If neovascularization is seen on the iris or in the angle, it is best to begin

AT A GLANCE

- Neovascular glaucoma (NVG) is a severe form of glaucoma that occurs secondary to vascular proliferation in the anterior chamber of the eye.
- Unlike many other forms of glaucoma, NVG can cause rapid damage due to the severe IOP elevation that results.
- Patients with NVG must be treated not only for glaucoma but also for the retinal ischemia precipitating the neovascularization.

"THE OPHTHALMOLOGIST SHOULD COLLABORATE WITH OTHER MEMBERS OF THE PATIENT'S CARE TEAM, SUCH AS THE PRIMARY CARE PHYSICIAN, CARDIOLOGIST, OR VASCULAR SURGEON, IN ORDER TO PREVENT ONGOING RETINAL ISCHEMIA OR NEOVASCULARIZATION."

treatment as quickly as possible. Neovascularization of the iris and of the angle should be treated with panretinal photocoagulation (PRP) and possibly injection of anti-VEGF agents. Ablation of the ischemic regions of the retina with PRP can reduce or eliminate the stimulus for angiogenesis, thereby attenuating neovascularization and potentially halting the development of NVG.[2]

Although PRP is the gold standard treatment in these cases, it is often supplemented with intravitreal injection of anti-VEGF agents such as bevacizumab (Avastin, Genentech), ranibizumab (Lucentis, Genentech), and aflibercept (Eylea, Regeneron). These drugs have been shown to decrease angiogenesis and prevent further neovascularization[3]; however, they are contraindicated in certain patients, so proper precautions must be taken.

If early and aggressive treatment is undertaken, progressive synechial closure of the angle can be prevented. If significant scarring of the angle is already evident at the time of presentation, treatment with PRP and anti-VEGF agents is still helpful but often cannot help restore normal aqueous outflow, and ultimately traditional glaucoma surgery is needed.

3. BEGIN TREATMENT WITH SMALL-VALVED GLAUCOMA DRAINAGE IMPLANTS

In our opinion, the most effective way to treat NVG is via surgical placement of a glaucoma drainage implant. Valved devices such as the Ahmed Glaucoma Valve (New World Medical) are preferable to nonvalved implants because valved implants are functional upon implantation and may be less prone to long-term hypotony in these rather ischemic eyes.[4] If the patient is a poor surgical candidate or if the eye has minimal visual potential, transscleral cyclophotocoagulation can be considered.

4. DON'T BE A HERO IN THE OR

Many patients with NVG have preexisting hyphema or may have an intraoperative bleed that can vary in severity. For the most part, the goal of surgery is to get in and get out quickly. Trying to evacuate the blood and/or clot often makes the situation worse. If bleeding is encountered during the procedure, the best course of action is to quickly tamponade the hemorrhage by injecting a cohesive OVD into the anterior chamber.

With a valved implant in place, the eye can be left at a relatively firm pressure to ensure that the bleeding stops. The pressure will lower overnight, and patients typically have an IOP in the teens on postoperative day 1.

5. ALWAYS ADDRESS THE ROOT CAUSE OF NEOVASCULARIZATION

Patients with NVG must be treated not only for glaucoma but also for the retinal ischemia precipitating the neovascularization. Although retinal ischemia can be primary, it is far more likely to be the result of other health conditions and systemic diseases. For example, in NVG secondary to PDR, diabetes must be controlled in order to prevent further retinopathy. In NVG secondary to CRVO or carotid artery occlusive disease, systemic cardiovascular health must be addressed.

The ophthalmologist should collaborate with other members of the patient's care team, such as the primary care physician, cardiologist, or vascular surgeon, in order to prevent ongoing retinal ischemia or neovascularization. This collaboration may even help save the patient's life, as he or she may be at high risk for more serious complications, such as a cerebrovascular accident or myocardial infarction. ■

1. Sivak-Callcott JA, O'Day DM, Gass JDM, Tsai JC. Evidence-based recommendations for the diagnosis and treatment of neovascular glaucoma. *Ophthalmology*. 2001;108(10):1767-1776.
2. Olmos LC, Lee RK. Medical and surgical treatment of neovascular glaucoma. Int Ophthalmol Clin. 2011;51(3):27-36.
3. SooHoo JR, Seibold LK, Kahook MY. Recent advances in the management of neovascular glaucoma. *Semin Ophthalmol*. 2013;28(3):165-172.
4. Ayyala RS, Zurakowski D, Smith JA, et al. A clinical study of the Ahmed glaucoma valve implant in advanced glaucoma. *Ophthalmology*. 1998;105(10):1968-1976.

JOSEPH F. PANARELLI, MD
- Glaucoma Fellowship Director and Chief of Glaucoma Service, Department of Ophthalmology, NYU Langone Health, New York
- joseph.panarelli@nyulangone.org
- Financial disclosure: None

JAKE RADELL, MA
- Icahn School of Medicine at Mt. Sinai, New York
- jake.radell@icahn.mssm.edu
- Financial disclosure: None