Neovascular Glaucoma Treatment With Extraction of Anterior Chamber Fibrovascular Tissue

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Compared with other types of glaucoma, neovascular glaucoma (NVG) has poor visual field prognosis. Its pathogenesis involves retinal ischemia, which induces the formation of new vessels and fibrous tissue in the root and stroma of the iris; these act mechanically on the angle, causing its closure and subsequent ocular hypertension.1,2

In the initial stages, intraocular pressure (IOP) is usually normal, so therapeutic measures are primarily directed at treating the cause of ischemia.3,4 Panretinal ablation and, more recently, administration of antibody to vascular endothelial growth factor (anti-VEGF),5-9 are the most widely accepted techniques used to induce neovascular regression.

However, after the angle closure phase, IOP elevation requires immediate treatment (with corticosteroids or cycloplegic or hypotensive agents). This is followed by treatment of the pathogenic causes to control the process of neovascularization, and finally by treatment of residual glaucoma (with filtering surgery, drainage devices, or cyclodestructive procedures).3,4

Etiological treatment is intended to resolve the causes of retinal ischemia that lead to the formation and progression of neovessels, in combination with anti-VEGF treatment.5-9 However, various studies have reported IOP decompensation after the administration of anti-VEGF secondary to angle closure.10,11

The surgical technique described here involves removal of fibrovascular tissue at the level of the iris and angle in order to release the angle and facilitate drainage of aqueous humor, thereby controlling IOP.

Surgical Technique

The first step is to inject 0.05 mL (1.25 mg) of bevacizumab (Avastin; Genentech/Roche) into the anterior chamber 48 to 72 hours before surgery, to avascularize the neovessels, thus preventing massive bleeding during surgery and facilitating the removal of fibrotic tissue covering the chamber angle.12

Corneal paracentesis is performed with 0.5-mm incisions at the 10 and 2 o’clock positions, immediately followed by anterior chamber washout to remove any residual blood from severed vessels. Air and Trypan blue are then injected into the anterior chamber to stain the fibrovascular tissue. This is followed by viscosurgery with 10 mg/mL viscoelastic (Biolon; Cryopharma S.A.) to mechanically separate the fibrotic tissue from the iris stroma. With alternating unimanual and bimanual techniques, de Smet forceps and scissors are used to extract fibrovascular tissue from the surface of the iris, the angle, and the pupil area (Figure 1). These maneuvers must be performed with extreme caution because of the risk of damaging or dislodging the iris root. When part of the fibrotic tissue is freed, it is extracted via the incisions or with a 23-gauge vitreotome. Finally, the viscoelastic is removed from the anterior chamber and another 0.05 mL of bevacizumab is injected (see the Video).

We then proceed to treat the posterior pole disorder causing the ischemia with vitrectomy when necessary.

This technique was applied in 2 cases of NVG with extensive macroscopic fibrovascular proliferation that covered the entire surface of the iris and complete chamber angle closure. The first case (Figure 1 and Video) was secondary to Coats disease, with an IOP of 46 mm Hg unresponsive to pharmacological control. The second case

Figure 1. Photograph Taken From the Eye Surgery Video

Single-frame photograph taken from the eye surgery video showing unimanual fibrovascular tissue dissection with de Smet forceps on the surface of the iris.
The use of anti-VEGF has been shown to promote regression of neovessels, visualized with indocyanine green angiography. In cases in which the angle is still open, before synechiae formation and angle closure, IOP control can be achieved with anti-VEGF administration and without the need for additional surgical procedures. In cases with angle closure, the IOP response is lower and additional surgery is needed.

At the same time as our observations, there have been case reports describing fibrovascular tissue contraction after the injection of bevacizumab, inducing angle closure and a consequent increase in IOP. In a study of 5 cases of NVG treated with bevacizumab, our group found regression of neovascularization and IOP control. However, 3 of these cases showed increased IOP 48 hours after injection, which required cycloablation for control. Fibrovascular tissue contraction after injection of anti-VEGF caused displacement of the iris–zonules–ciliary body complex, decreasing iridocorneal angle amplitude, as shown by anterior segment optical coherence tomography, thus inducing IOP depensation. We have therefore opted to treat the underlying cause using fibrovascular membrane dissection to facilitate aqueous humor drainage, because release of the material causing the blockage favors IOP control after anti-VEGF administration. In patients undergoing this procedure, we have noted good postoperative IOP results and good angular amplitude, as shown by anterior segment optical coherence tomography (Figure 2).

In addition, the use of anti-VEGF 48 hours before surgery resulted in initial nonperfusion of the neovessels rather than elimination of them; this greatly facilitates surgery because it prevents massive bleeding and it improves postoperative results.

In conclusion, in selected cases of NVG with IOP depensation, before anti-VEGF treatment, we recommend studying the extent of fibrovascular tissue and performing gonioscopy to assess the degree of synechiae. If fibrovascular tissue is visible, the combination of tissue dissection and anti-VEGF injection may help decrease postoperative IOP. However, randomized clinical trials would be needed to assess the efficacy of this approach.


OPHTHALMIC IMAGES

Leiomyoma of the Lower Eyelid

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Figure 1. A 55-year-old woman presented with an ill-defined mass (arrows) of the left lower eyelid fornix associated with excessive tearing and mattering for 5 weeks. Magnetic resonance imaging of the orbits revealed soft tissue swelling. The mass was excised and histologically diagnosed as a lower eyelid leiomyoma.

Figure 2. A. Microscopic examination demonstrates conjunctival tissue with an area of haphazard smooth muscle bundles (hematoxylin-eosin, original magnification ×40). B. Higher magnification reveals fascicles composed of fusiform cells with cigar-shaped end nuclei (arrow) (hematoxylin-eosin, original magnification ×400). C. Cytoplasmic staining with muscle-specific actin immunohistochemistry stain is observed (original magnification ×400).