Neovascular Glaucoma – A Review

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Abstract
Neovascular glaucoma (NVG) is a potentially blinding clinical condition, where delayed diagnosis or poor management can result in complete loss of vision. In managing NVG, it is essential to treat both the elevated intraocular pressure (IOP) and the underlying cause of disease. The objective of this review is to discuss its etiology, pathophysiology, clinical features and management.

First priority in managing NVG should be to try preventing its development by appropriate treatment of the causative diseases. Since NVG is a secondary glaucoma, a thorough knowledge about the varied etiologies and meticulous methods of examination along with high index of suspicion is a must. Blindness caused due to NVG is totally preventable if diagnosis is made in early stages and such patients are aggressively treated along with frequent follow up.

Detailed clinical examination including slit lamp evaluation, IOP measurement, gonioscopy, dilated retinal evaluation with 90D and 20D lenses and Fundus Fluorescein Angiography (wherever required) is a must in all cases with identifiable risk factors. In established cases of PDR, pan retinal photoacoagulation (PRP) with or without intravitreal anti-VEGF is the procedure of choice to prevent NVG. Mainstay of medical treatment is to reduce aqueous production. In refractory cases requiring surgical intervention, the results have improved significantly with use of antifibrotic agents during trabeculectomy and evolution of tube shunts. The introduction of anti-VEGF agents in the management of NVG has proved to be a benchmark in both prevention and treatment of this disease.

Prevention and early diagnosis is the key to reduce visual loss caused by this devastating disease. Though the treatment of NVG still remains a challenge, the advent of anti-fibrotic agents, tube shunts and anti-VEGFs has revolutionized its management and promises relatively better outcomes.

Keywords: NVI, NVA, PDR, CRVO, glaucoma

Etiology
Most common causes associated with NVG are:

1. Proliferative Diabetic retinopathy (PDR) – PDR (Figure 1) is the most frequent cause of NVG. Surgical intervention like pars plana vitrectomy for PDR increases the incidence of rubeosis iridis to 8-9% and of NVG to 4-5%. Risk of rubeosis iridis post vitrectomy in PDR increases with aphakia, presence of rubeosis before vitrectomy, and unrepaired retinal detachment present after vitrectomy. On the other hand, intraocular silicone oil decreases the incidence of rubeosis iridis. Cataract surgery can also lead to progression of Diabetic retinopathy (DR). An intact posterior capsule reduces the risk.

2. Retinal vascular occlusive disorders – Central Retinal vein occlusion (CRVO) (Figure 2) is the second most common cause associated with rubeosis iridis. NVG develops in about 45% of ischemic CRVO and the risk is highest during the first 7-8 months of developing CRVO. All patients should be followed up meticulously and should undergo fluorescein angiography to delineate non-ischemic CRVO from ischemic CRVO.
3. Carotid artery obstructive disease (CAOD) is the 3rd most common cause of NVG accounting for around 13% cases.
Reduction of blood flow to the eyeball can produce anterior and/or posterior segment ischemia. Anterior segment ischemia manifests as Neovascularization of Iris (NVI), Neovascularization of Angle (NVA) and NVG. Decreased perfusion of the ciliary body significantly reduces aqueous production and thus can result in normotensive or hypotensive eyes despite extensive synechial closure. Cerebral angiography or magnetic resonance angiography may be necessary to demonstrate occlusion or stenosis in carotid siphon or ophthalmic artery.

Uncommon causes include ocular radiation, ocular tumours, chronic uveitis, chronic vasculitis, Coats disease, frosted branch angiitis, peripheral retinal detachment, X-linked retinoschisis, cryoglobulinemia and Churg-Strauss syndrome.

**Pathophysiology**
The common factor in all the above mentioned diseases is retinal ischemia which leads to retinal capillary compromise causing production of vascular endothelial growth factors (VEGF) from the Muller cells. VEGF, a potent vasoproliferative substance, acts upon endothelial cells of viable capillaries to stimulate the formation of fragile new vessels (neovascularization). VEGF is theorized to even diffuse anteriorly and cause NVI and NVA. The neovascularization of the angle along with the fibrovascular membrane physically blocks the trabecular meshwork and subsequently its contracture leads to extensive peripheral synechial angle closure.

**Clinical Features And Stages Of NVG**
The disease passes through four stages – Pre-rubeosis stage, Pre-glaucoma stage, Open angle glaucoma stage and Angle closure glaucoma stage, each one having differing clinical features.

**Pre-rubeosis stage** – Iris or angle neovascularization is not visible at this stage, but the predisposing ocular/extra ocular condition is present. Laser photometry or fluorescein iris angiography is helpful in detecting early leakage of iris vessels.

**Pre-glaucoma stage**: Rubeosis iridis stage – At this stage, variable amounts of neovascularization (Figure 3) can be found at pupillary margin, iris surface and in the angle. Neovascularization must be looked for carefully under high magnification (Figure 4) on the iris and in the angle by gonioscopy at every visit. The iris should be examined before dilatation of the pupil and pupillary margins and margins of iridotomy should be carefully looked at for new vessels, which appear like knuckles of fine vessels. Although neovascularization is usually seen first at peripupillary area, a thorough gonioscopy should be performed since NVA can sometimes precede rubeosis iris. Characteristic features of this stage are normal IOP, unless pre-existing concomitant primary glaucoma is present. Patients are usually asymptomatic at this stage. Open angle glaucoma stage: At this stage, IOP begins to rise and stays elevated. In some cases, the IOP may rise suddenly resulting in acute-onset glaucoma. Rubeosis iridis in this stage is more florid and is often associated with anterior chamber inflammatory reaction. Due to fragile nature of the new vessels, hyphema can also present sometimes at this stage. Gonioscopy shows an open angle but with more intense neovascularization. Angle closure glaucoma stage: Most patients present or are detected at this stage. The contraction of fibrovascular membrane in the angle leads to progressive synechial angle closure, ectropion uveae and flat, smooth, glistening appearance of the iris. Gonioscopy reveals varying degrees of peripheral anterior synechiae or complete angle closure may be present at this stage. The IOP is usually very high and can even go up to 60 mmHg. Rubeosis is usually severe with possible hyphema and moderate anterior chamber inflammation at this stage. Conjunctival congestion and corneal edema are frequently present. Glaucomaticus optic nerve damage is often moderate to advance. Visual acuity may also be severely affected. If not treated appropriately it can lead to glaucomatous optic atrophy.
Management of NVG

General principles for treatment of NVG are aimed at identifying the underlying etiology and its timely and adequate treatment to prevent the development and progression of NVG. Once NVG develops and IOP is high, the major aspect of management is control of high IOP to prevent optic nerve damage and continuous treatment of underlying etiology.

Prophylactic Treatment

The management strategy involves registering the predisposing factors and having a high index of suspicion in the above mentioned conditions. It is necessary to closely follow these eyes and manage patients adequately to reduce the incidence of neovascularization and to minimize visual loss.

Ultra-wide-field fundus fluorescein angiography offers the possibility of evaluating and quantifying peripheral retinal perfusion and vascular pathology in fundus disease. In established cases of PDR, pan retinal photocoagulation (PRP) with or without intravitreal anti-VEGF is the procedure of choice to prevent NVG. Such patients deserve careful monitoring of iris and angle, even after standard course of PRP, so that further retinal ablation can be done if new vessels develop or progress in the anterior segment.

However in patients with CRVO with no evidence of neovascularization, prophylactic PRP showed no significant difference in the incidence of NVG as compared to eyes without PRP. The Central Retinal Vein Occlusion Study (CVOS), essentially recommended careful follow up of patients with ischemic CRVO and PRP was indicated for Fundus Fluorescein Angiography (FFA) confirmed ischemic CRVO who developed 2 clock hours of NVI or NVA. Management of ocular ischemic syndrome (OIS) remains difficult and controversial. In OIS, ocular neovascularization is not always associated with retinal capillary non-perfusion on FFA, even in eyes with diabetes mellitus. It has been suggested that uveal ischaemia alone may be responsible for neovascularisation in some cases of OIS and hence PRP should be reserved for cases with established retinal ischaemia on FFA. There is no scientific rationale for PRP when FFA shows no retinal ischaemia in the form of capillary non-perfusion. OIS is usually an important indicator of carotid artery stenosis, and all OIS patients should be referred for neurological and cardiovascular assessment at the time of ocular diagnosis. Carotid endarterectomy has been shown to benefit patients with symptomatic cerebral ischaemia when there is greater than 70% carotid artery stenosis. Although reports exist of IOP rising as ciliary body circulation is improved by carotid endarterectomy or by superficial temporal artery-middle cerebral artery bypass surgery (STA-MCA), most patients undergoing carotid endarterectomy do not experience any significant rise in IOP.

Goniophotocoagulation, another laser therapy, is aimed at directly treating the NVA before the development of NVG. Its role in management of NVG is unclear, and it has not proven to be beneficial in preventing synechial closure of angle or advanced NVG.

Medical Management of NVG

IOP lowering agents – Mainstay of medical treatment is to reduce aqueous production with topical beta blockers, alpha agonists and with topical and/or oral carbonic anhydrase inhibitors. Hyperosmotic agents may also be required. Topical prostaglandin analogues can be tried though they may increase ocular inflammation. Miotics are contraindicated as they can increase inflammation and discomfort.

Anti-inflammatory drugs – Frequent administration of topical steroids and cycloplegics are recommended to reduce inflammation that is inevitably present.

Anti-Angiogenic drugs – Several studies propose the use of anti-VEGF agents with traditional treatments such as PRP with or without additional surgery and vary in the timing, combination, and place of injection (intracameral or intravitreal, or both simultaneously). The most frequent recommendation by various authors for treatment of neovascularization is the adjunct combination of intravitreal bevacizumab and PRP or bevacizumab alone when visibility of the posterior segment is difficult due to opacities of the media (e.g., hemorrhage). Most studies mention similar dose for intravitreal and intracameral use (1.25mg/0.05ml). Bevacizumab has been reported to cause regression of the iris neovascularization, in majority of the cases, within 24 to 48 hours following intravitreal injection whereas NVI starts regressing post PRP by 2 weeks and is complete by 4-6 weeks. It is also effective in reducing intraocular inflammation and pain and in few reports IOP lowering has been noted in the open angle stage. Intracameral injection of Bevacizumab may provide additional strategy for treating rubecosis iridis in NVG. Bevacizumab is well tolerated, effectively stabilizes NVI, and controls IOP when used alone and at an early open angle stage of NVG. In advanced cases of NVG it can be used as a therapeutic window for PRP and also surgical intervention for IOP control can be performed with much less risk of failure, hemorrhage and inflammation. In cases where PRP is not possible due to poor retinal view, intravitreal Bevacizumab can be given followed by Trabeculectomy with Mitomycin C. However, bevacizumab-induced regression of neovascularization is often temporary and recurrence is possible, while PRP provides a more permanent reduction of the ischemic angiogenic stimulus. Anterior retinal cryopexy (ARC) may be useful in cases of compromised posterior segment view and where availability or affordability of anti VEGF is an issue.

Medical management with intravitreal anti-VEGF along with retinal ablation wherever possible may be sufficient to control the IOP in the open angle stage of NVG, but in advanced stage with synechial angle closure surgical intervention for IOP lowering is often required.

Surgical Management of NVG

The three surgical modalities often employed are trabeculectomy, tube shunts and cycloablation. The choice of surgical procedure is made depending upon underlying disorder as well as the clinical characteristics of each patient.
i.e., level of IOP, presence of active or regressed NVI, prior laser or anti-VEGF treatment, prior intraocular surgeries, degree of inflammation, stage of disease, degree of angle closure, severity of glaucomatous optic neuropathy and visual potential. Outcomes of surgical treatment tend to be better in PDR compared to CRVO and ocular ischemic syndrome.

**Trabeculectomy**

Intraoperative use of anti-fibrotic agents (like Mitomycin C i.e. MMC) is recommended to reduce the risk of bleb failure due to subconjunctival scarring. High dose (0.04%) and/or longer duration of contact (2-3 min) of MMC can be used. The success rate of trabeculectomy with MMC in NVG at 1 year has been reported to be around 62.6% and reduced to 51.7% at 5 years. With the use of preoperative Bevacizumab, success rate may improve up to 95%. Regressed NVI and reduced intraocular inflammation before trabeculectomy increases the success rate (Figure 5). Therefore, for better surgical outcome, it is recommended that a sufficient PRP and/or anti-VEGF agents should be given wherever possible to cause regression of NVI before performing standard trabeculectomy with MMC. Surgical intervention should be planned within a week of injection of anti-VEGF. Combined cataract and glaucoma surgery can be planned if media is hazy due to cataract, followed by PRP whenever possible. In cases of hazy retinal view due to a cause like vitreous hemorrhage, if after a short interval of observation the media clarity does not improve, pars plana vitrectomy (PPV) with endolaser (EL) photoocoagulation may be considered. Repeat intravitreal injections of bevacizumab may be required. Many surgeons may opt for glaucoma drainage implant placement at the time of PPV and EL, after medical management with drugs and anti-VEGF agents, particularly if synechial angle closure is evident or suspected. In the setting of trabeculectomy, bevacizumab has been used preoperatively, intraoperatively, postoperatively and for treatment of failing filtering blebs or during bleb revision. These studies have looked at intracameral, intravitreal, and subconjunctival administration. Pre placed sutures and intra-operative Sodium hyaluronate can be used avoid sudden decompression as this might lead to intraoperative bleeding. The use of releasable sutures and laser sutureysis post-operatively can help titrate IOP in early post-operative period. A closer follow up is required for these patients. Sub conjunctival use of 5-Fluorouracil (5-FU) or MMC can be considered post-operatively in cases which show early signs of risk of failure or aggressive vascularization. Before the advent of anti-VEGF agents, trabeculectomy with MMC combined with direct cauterization of peripheral iris was tried by Elgin et al to reduce bleeding both intraoperatively and postoperatively.

**Tube Shunts**

Glaucoma drainage devices (GDDs) (Figure 6) are increasingly being considered as a primary surgical procedure in the management of all glaucomas especially NVG where there is a high risk for failure of conventional filtering surgery. Scarred conjunctiva, active inflammation, vigorous new vessel growth and prior failure of trabeculectomy are also all indications to consider tube shunt surgery in NVG. Various drainage devices like Molteno implant, Baerveldt implant and Ahmed glaucoma valve have been used in management of NVG and show no statistically significant difference in either IOP lowering effect or overall success rate. Use of anti-VEGF agents is recommended at least 24 hours prior to surgery. Extra precautions must be taken, however, to ensure the use of adequate patch graft material over the tube. Moreover, the tube must be placed through atleast a 1.5 mm scleral tunnel incision to minimize the chances of erosion, because this population is at a high risk of this challenging complication. The problems with tube shunt surgery in NVG are early postoperative hypotony, blockage of internal fistula or external filtration site and vigorous postoperative fibrous encapsulation response. The comparison of various surgical techniques to treat NVG is limited due to variable confounding factors and contrasting outcome measures in different studies. All the proposed surgical techniques share poor outcomes in NVG due to the progressive underlying pathology.
There is no clear evidence and consistent basis for choosing between trabeculectomy and drainage implant.

**Cyclodestructive Procedures**

Transscleral cyclophotocoagulation with noncontact Nd: YAG or semiconductor diode laser have proven useful in treatment of refractory NVG with poor visual prognosis. Repeat treatment may be required to maintain good control of IOP. Endoscopic cyclophotocoagulation along with vitrectomy has also been reported.27

**Conclusion**

NVG is a potentially blinding disease. Early diagnosis and aggressive control of high IOP and the underlying etiology is crucial to minimize the visual loss. In the management strategy, the first priority should be to try preventing its development by appropriate management of the causative diseases.

Though the treatment of NVG still remains challenging, the advent of anti-fibrotics, tube shunts and anti-VEGF agents has revolutionized the management and given hope for relatively better outcomes. Medical management to control high IOP is usually only a temporary measure in the later stages. Success of various surgical procedures to control IOP in NVG needs to be substantiated in the long run. The searches for newer and more effective modalities of treatment is still on.

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