Prevalence, Causes and Management of Neovascular Glaucoma: A 5 Year Review

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Introduction: Neovascular glaucoma (NVG) also called 100 day or congestive glaucoma, is a severe form of secondary glaucoma that occurs with blockage of aqueous outflow due to development of new vessels on the iris (NVI) and iridocorneal angle (NVA) as a result of anterior segment ischaemia.[1,2] It was first described by Weiss et al in 1963.[3]

Anterior segment neovascularisation is believed to result from posterior segment ischaemia arising from ocular disorders such as retinal venous occlusion(RVO) and arterial occlusions, proliferative diabetic retinopathy (PDR), carotid artery obstructive disease and uveitis.[4,5,6,7] Glaucoma develops through secondary open angle or secondary closed angle mechanisms.[8]

Patients usually present with complaints of redness, photophobia, pain and loss of vision.[1] Other findings include, elevated IOP, conjunctival congestion, corneal edema, hyphaema and posterior segment ischaemia.[1]

Anti- VEGFs are now being used with other modalities of treatment such as transscleral cyclophotocoagulation(TSCPC), incisional glaucoma surgery and medical therapy in the management of NVG[9,10]. Successful visual outcome is reduced once NVG is established, timely detection of the risk factors and their control are key in preventing visual loss.

Methods: Case records of patients attending the retina clinic of the University of Port Harcourt Teaching Hospital between January 2015 to December 2017 were reviewed. Parameters evaluated included patients' demographics, visual acuity, cause of neovascular glaucoma and treatment modalities.

Results: Seventeen eyes (53%) had Retinal vein occlusion, followed by eyes with proliferative diabetic retinopathy 13 (41%) and only 2(6%) eyes had uveitis (Figure 1).

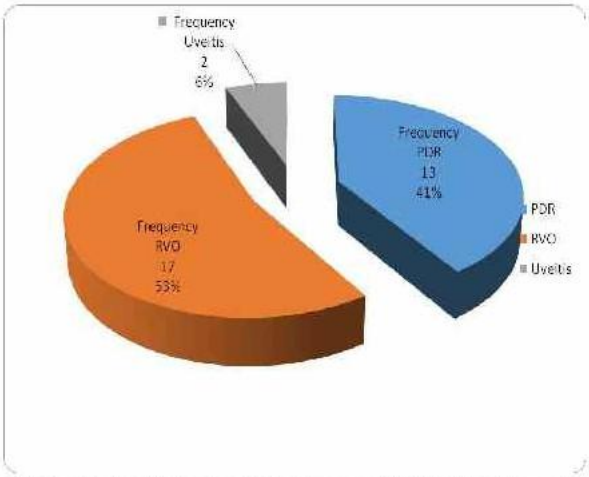

Fig. 1: Aetiological factors of NVG in the study population

The distribution of the types of intervention administered is shown in Table 1 while Table 2 shows the clinical outcome of treatment on intraocular pressure. The reduction in IOP after intervention was statistically significant (Table 2).

Table 1: Types of intervention administered

Intervention	Number (Percentages)
Medical Treatment	12 (37.5)
Medical Treatment + Anti- VEGF	13 (40.6)
Medical Treatment + Anti- VEGF+ TSCPC	5 (15.6)
Medical Treatment +TSCPC	2 (6.3)

KEY:
Anti – VEGF- Anti vascular endothelial growth factor
TSCPC – Trans scleral cyclophotocoagulation

Table 2: Clinical outcome of treatment on intraocular pressure (IOP)

IOP	IOP on Presentation Number	Percentage	IOP Post intervention Number	Percentage
11-22	-	0	16	50
23-30	4	12.5	7	21.9
31 and above	28	87.5	9	28.1
TOTAL	**32**	**100**	**32**	**100**
Chi Square= 23.649 p=0.000				

Discussion: The hospital prevalence of neovascular glaucoma in this study was 0.3%, slightly higher than reported in a tertiary hospital in Thailand.[11] It is similar to that reported in population based studies, 0.20%- 0.55% [12-14].
NVG is a sequeale of vascular and non vascular disorders of the eye that result from retinal ischemia. The commonest cause of NVG in this study was RVO followed by PDR. Most studies reported a similar trend where RVO and PDR were the two leading causes of NVG [5,7,15,16]. Uveitis 2(6%) was a rare causative factor similar to other studies.[16]

A combination of several modalities of treatment have been shown to be more effective in IOP reduction, than medical management alone in NVG.[9,10] In this study the most common form of treatment used was a combination of medical therapy with IOP lowering drugs and anti-VEGFs 13(40.6%). Looking at the IOP response in the study group overall, there was a statistically significant reduction in IOP (P= 0.000).

Conclusion: Neovascular glaucoma is a blinding condition with challenges in control of IOP and preservation of vision. Medical therapy with intraocular pressure lowering drugs in combination with anti-Vegf are effective in IOP control.

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