

Neo-Vascular Glaucoma: Etiology and Outcome of Treatment in Lagos

Adekunle Rotimi-Samule, Adeola O. Onakoya, Karim O Musa, Olufisayo T Aribaba, Folasade B. Akinsola

Department of Ophthalmology, Lagos University Teaching Hospital, Idi-Araba, Lagos, Nigeria

ABSTRACT

Objective: To determine the causes and the intraocular pressure (IOP) outcome of neo-vascular glaucoma at the Guinness Eye Centre of the Lagos University Teaching Hospital, Lagos, Nigeria. **Materials and Methods:** The case records of all patients with the diagnosis of neo-vascular glaucoma who presented at Guinness Eye Centre from January 1st, 2008 to December 31st, 2012 were reviewed. Information extracted from their records included the bio-data, eye affected, best corrected visual acuity, intra-ocular pressure, etiology of neo-vascular glaucoma, ocular co-morbidity, systemic co-morbidity and forms of treatment received. Cases of retinoblastoma were excluded from the study. Data were analyzed using the SPSS V.17.1 **Results:** A total of 31 cases of neovascular glaucoma were reviewed. The male to female ratio was 1.8:1 and the age range was from 26 to 83 years. Patients aged 51 years or older were 22 (70.4%). The presenting best corrected visual acuity in the affected eye or in the worse affected eye was counting fingers in 30 (96%) patients. The presenting IOP was 30 mmHg or higher in 26 (83.2%). Both eyes were affected in three (9.6%) patients. Patients with diabetes mellitus, hypertension or both constituted 57.8% of the total number of patients. Primary open angle glaucoma and retina vein occlusion were the etiologies of neo-vascular glaucoma in 14 (45.2%) patients. A total of 15 (48.4) patients failed to turn up for repeat appointments. Only 5 (16.0%) patients (who received ocular medications, intravitreal bevacizumab and pan-retina photocoagulation) showed a significant IOP reduction. **Conclusion:** Late presentation, loss to follow up and lack of full and complete treatment regime were important findings in this study. A significant IOP reduction occurred with only in the patients who had a combination therapy of IOP lowering medications, intravitreal bevacizumab and pan-retina photocoagulation. A routine eye examination for patients at risk is essential for early detection and treatment in order to minimize visual loss in neo-vascular glaucoma.

Keywords: Combination therapy, early detection, late presentation, neo-vascular glaucoma

INTRODUCTION

Neo-vascular glaucoma (NVG) is a secondary glaucoma occurring with the formation of a fibrovascular membrane on the anterior surface of the iris (rubeosis iridis) which extends to the anterior chamber angle.^[1] An initial open angle glaucoma then progresses to angle closure as the fibrovascular tissue contracts to produce

synechiae angle closure glaucoma, often resulting in loss of vision.^[2,3] New blood vessel formation on the iris is triggered by vascular endothelial growth factors diffusing anteriorly from a hypoxic retina.^[4,5] It has been suggested that iris neovascularization is never primary but secondary to other ocular disorders.^[6] Hypoxic retinopathy may result from diabetic retinopathy, central retina vein occlusion (CRVO), branch retina vein occlusion (BRVO), central retina artery occlusion (CRAO), uveitis, longstanding retinal detachment, intraocular tumors, carotid obstructive disease and carotico-cavernous fistulae, all of which may lead to NVG.^[2,6]

In Ibadan, Nigeria, NVG constituted 0.4% of new patients attending an eye clinic with retinal vein occlusion (RVO) being the etiological factor in the

Access this article online

Quick Response Code	Website: www.nigerianjournalofophthalmology.com
	DOI: ***

Address for correspondence

Dr. Rotimi-Samuel A., Department of Ophthalmology, Lagos University Teaching Hospital, Idi-Araba, Lagos, Nigeria.
E-mail: kunleogbe@yahoo.com

majority (78.7%) of subjects.^[7] A study in Cameroon showed that NVG constituted 47.7% of all secondary glaucomas.^[8] The most common causes of NVG in Romania are diabetic retinopathy, ischemic CRVO and ocular ischemic syndromes.^[9] NVG accounted for 16 (22%) of all secondary glaucomas seen in a tertiary eye centre in Paraguay.^[10] In Thailand, the most common causes of NVG were CRVO (47%), proliferative diabetic retinopathy (42%), and ocular ischemic syndromes (5%).^[11] In Saudi Arabia, the common causes of neovascular glaucoma were diabetic retinopathy (56.0%), retina venous obstruction 26.4% and chronic retinal detachment (3.56%).^[12]

The treatment of NVG may be pharmacological or surgical such as trabeculectomy, aqueous drainage implants, cryotherapy or laser partial destruction of the ciliary body, photocoagulation of the hypoxic retina or a combination of any of these. The use of anti vascular endothelial growth factors is a significant milestone in the management of NVG as they block the formation of new blood vessels in the eye. Bevacizumab, for example binds to the VEGF-A, thereby inhibiting angiogenesis.^[13] The methods of treatment employed in a particular case can be individualized based on the available options, severity of disease and potential for vision.

This study aims to determine the common causes and outcome of treatment of NVG in patients attending the retina clinic of the Lagos University Teaching Hospital.

MATERIALS AND METHODS

This is a retrospective study of the causes of and the outcome of treatment for NVG at the Guinness Eye Centre, Lagos University Teaching Hospital, Lagos, Nigeria. Ethical approval was obtained from the Lagos University Teaching Hospital Research and Ethics Committee. All new cases attending the eye clinic from January 2008 to December 2012 with the diagnosis of ‘NVG’ had their records retrieved manually by the medical records officers. Information extracted from the records included bio-data, eye affected, best corrected visual acuity, intraocular pressure (IOP), etiology of NVG, ocular co-morbidity, systemic co-morbidity, and forms of treatment received. All cases of retinoblastoma were excluded as they are not normally labeled as cases of NVG in our centre. Data were analyzed using the SPSS V.17. 1

RESULTS

A total of 31 patients had NVG out of the total number of 52,335 new patients seen at the Guinness Eye Centre during the period of review. This gives a clinic incidence of 0.06%. There were 20 males and 11 females with a male to female ratio of 1.8:1. The mean age at

presentation was 58.9 years and the age range was from 26 to 83 years. Patients aged 51 years and above were 22 (70.4%) as shown in Figure 1.

The right eye alone was involved in 15 (48.0%) patients and the left eye alone in 13 (41.6%). Both eyes were involved in NVG in 3 (9.6%) patients. The presenting best corrected visual acuity (BCVA) in the affected eye, or in the better eye where both eyes were affected, was counting fingers (CF) or worse in 30 (96%) patients. This is shown in Table 1.

In the non-affected eye, 6 (19.4%) patients had visual acuity of 3/60 or worse. Twelve patients (38.7%) had no associated systemic disorders, while 10 patients (32%) and 1 patient (3.2%), respectively, had hypertension and diabetes mellitus (DM) only. However, seven (22.6%) patients had both hypertension and DM concurrently [Table 2].

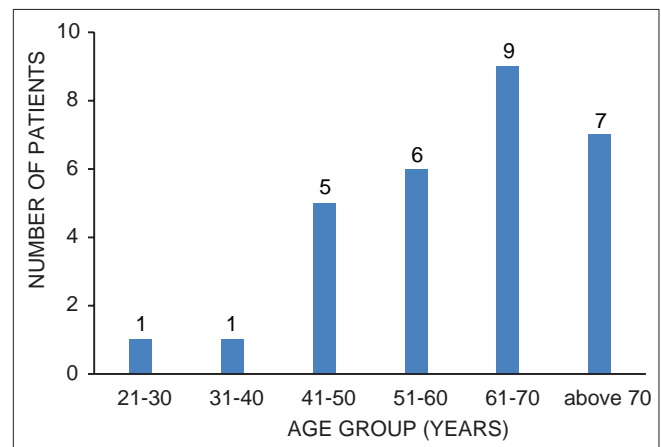


Figure 1: Age groups

Table 1: The BCVA at presentation in the affected (or worse affected) eye

BCVA	Frequency	Percent
6/24-6/36	1	3.2
Counting fingers	2	6.5
Hand movement/perception of light	8	25.8
No perception of light	20	64.5
Total	31	100.0

BCVA: Best corrected visual acuity

Table 2: Associated systemic disorders

Associated systemic disorders	Frequency	Percent
Hypertension alone	10	32.0
Diabetes mellitus alone	1	3.2
Hypertension and diabetes mellitus	7	22.6
Sickle cell disease	1	3.2
No known systemic association	12	38.7
Total	31	100

Three (9.3%) patients had associated ocular trauma (couching). Ten (32%) patients had retinal vascular occlusion either without a known etiology or secondary to glaucoma or diabetic retinopathy [Table 3].

Ocular medications alone (atropine, steroid eye drops and anti-glaucoma tablets and drops) represented the form of treatment most solely utilized by majority of the patients. Twenty-four (76.8%) patients took up this form of treatment alone, whereas, only 5 (16.0%) patients had a combination therapy consisting of ocular medications, intravitreal bevacizumab injections and pan-retina photocoagulation as shown in Table 4.

The IOP at presentation was 30 mmHg or higher in 26 (83.2%) patients. In all, 15 (48.4%) patients failed to turn up for follow-up appointments after the first visit. The remaining patients who turned up for appointments had varying IOP reductions depending on treatments received. Five (16%) patients who had a combination therapy of ocular medications, intraocular bevacizumab injections and retinal laser photocoagulation treatments showed an average IOP reduction of 19 mmHg [Table 5]. However, the remaining 11 patients who came for follow-up but did not receive either intravitreal bevacizumab or PRP showed different pattern of IOP outcomes. While 3 eyes had a further elevation of IOP, one eye became phthisical [Table 6].

DISCUSSION

With a male to female ratio of 1.8:1, male patients in this study were almost twice as many as females. This may be a reflection of unequal access to eye care services rather than male susceptibility to NVG.

The mean age at presentation of 58.9 years is comparable to the mean age at presentation in the Ibadan study.^[7] Twenty-two (70.4%) patients were aged 51 years and above with the age range 61–70 years having the highest frequency [Figure 1]. This is to be expected as the diseases likely to cause NVG such as glaucoma, DM, and hypertension become more chronic with advancing age.

The BCVA at presentation was very poor. Thirty (96.8%) patients presented with BCVA of counting fingers or worse in the affected eye (or in the better eye where both eyes were involved) suggesting that patients in this study generally presented late. This is underscored by the finding that no patients showed any improvement in vision and two of them actually deteriorated from BCVA of hand movement/perception of light to no perception of light.

Table 3: Neo vascular glaucoma and associated ocular disorders

Associated ocular disorders	Frequency	Percent
Primary open angle glaucoma	7	22.6
Nil association	6	19.4
CRVO	4	12.9
Trauma (couching)	3	9.7
BRVO+glaucoma	3	9.7
BRVO+dr*	1	3.2
BRVO+hypertension	1	3.2
CRAO	1	3.2
Retinal detachment	1	3.2
Sickle cell retinopathy	1	3.2
Diabetic retinopathy alone	1	3.2
Angle closure glaucoma	1	3.2
Uveitis	1	3.2
Total	31	100.0

*DR: Diabetic retinopathy, CRVO: Central retina vein occlusion, BRVO: Branch retina vein occlusion, CRAO: Central retina artery occlusion

Table 4: Forms of treatment uptake

Treatment modality	Frequency	Percent
Ocular medications alone	24	76.8
Ocular medications, bevacizumab and PRP	5	16.0
Ocular medications and bevacizumab alone	2	6.4
Total	31	100.0

PRP: Pan retinal photocoagulation

Table 5: IOP readings in patients who came for follow-up (PRP, intravitreal bevacizumab, medications)

Case number	Entry IOP (mm Hg)	Last IOP reading (mm Hg)	IOP reduction (mmHg)
5	19	10	10
12	30	18	12
15	43	18	25
23	38	29	9
24	52	15	37
Average	36.4	18	19

PRP: Pan retinal photocoagulation, IOP: Intraocular pressure

Table 6: IOP readings in patients who came for follow-up (medications alone)

Case number	Entry IOP (mm Hg)	Last IOP reading (mm Hg)	IOP change (mmHg)
4	19	14	-5
13	35	54	+19
14	15	48	+33
17	Not available	Not available	0
18	16	12	-2
26	42	34	-8
27	40	34	-6
28	Not available	15	0
29	52	52	0
30	46	0	-46
31	22	40	+22

-: Means IOP reduction, +: Means IOP increase, IOP: Intraocular pressure

Systemic hypertension and DM, either alone or in combination, were the most important systemic disorders associated with NVG in this study. Both of these disorders may lead to retina ischemia and consequent neovascularization of the anterior segment of the eyes. Six (19.4%) patients were blind (cause of blindness not stated in the charts) in the contralateral eye at presentation and this underscores the importance of comprehensive eye examination for diabetic and hypertensive patients at regular patients. At the very least, each patient should have visual acuity and a direct funduscopy at every clinic visit.

Couching was the etiology of NVG in three patients in this study as was the case in seven cases in the Ibadan study.^[7] Couching, as a form of treatment for cataract, is still performed in Nigeria despite the availability of modern methods of cataract surgery. In the Nigerian National Blindness and Visual Impairment Survey, 46.1% of all operated cataracts was by couching.^[14] The finding of couching as an etiology of NVG in this study suggests a persistent gap between availability of and accessibility to modern cataract surgery in Nigeria. In Lagos alone there are at least two tertiary eye centers, six secondary eye centers and numerous private ophthalmologists' clinics and yet patients still choose to patronize couching practitioners.

The majority of patients 24 (76.8%) received ocular medications alone as a form of treatment. This may be due to lack of funds to access other forms of treatment which are considerably more expensive. A dose of bevacizumab injection and a session of retina laser photocoagulation treatment, for example, may cost up to 35,000:00 naira (about 200 US dollars) each, a sum which is about twice the minimum wage in Nigeria. On the other hand it may be that patients did not wish to have any further treatments once the immediate concerns of pain have been alleviated by the initial ocular medications. Nonetheless, the result suggests a failure to deliver a particular form of eye care service to those who need it.

Most patients in this study presented with IOP of 30 mmHg or more, indicating a greater possibility of optic nerve damage and consequent loss of vision. The average IOP reduction of 19 mmHg occurred in five patients who came for follow up and received a combination therapy of ocular medications (pressure lowering and anti-inflammatory medications), intravitreal bevacizumab and pan-retina laser photocoagulation. This suggests that a combination therapy may reduce IOP and, perhaps, preserve vision in NVG if the patient presents early. None of the patients in this study received such forms of treatment as valve drainage implants or cyclo-destruction by laser or cryo-therapy. This represents a limitation of the options

of treatment for NVG in our center but their impact on outcome of treatment in our environment needs to be studied as most of the patients presented rather late.

CONCLUSION

Late presentation, loss to follow up and lack of full and complete treatment regime were important findings in this study. A routine eye examination for patients at risk is essential for early detection and prompt treatment for better visual outcome. Such patients should at least include all diabetics and hypertensives.

ACKNOWLEDGEMENTS

The authors wish to acknowledge with appreciation the encouragements of Prof. Adefule-Ositelu O.A towards the completion of this paper. We also give our sincere thanks to Femi-Soetan O.I (Mrs) and Olusope-Ogunye T.O (Mrs) both of them staffs of the medical records unit of Guinness Eye Centre, LUTH for retrieving the patients' records.

REFERENCES

1. Leffler KU. Neovascular Glaucoma. Aetiology, pathogenesis and treatment. *Ophthalmology* 2006;103:1057-63.
2. Kanski JJ, Bowling B. Glaucoma. In: *Clinical Ophthalmology: A systematic approach*. 7th ed. Edinburgh: Elsevier Saunders; 2011.
3. Sivak-Callot JA, O'Day DM, Gass JD, Tsai JC. Evidence-based recommendations for the diagnosis and treatment of neovascular glaucoma. *Ophthalmology* 2001;108:1767-76.
4. Fanous MM. Neovascular glaucoma; *Ophthalmology*. In: Yanoff M, Duker JS, editors. 2nd ed. St Louis: Mosby Publishers; 2004.
5. Garnish KS, Ramamurthi S, Saidkasimova S, Kamaesh K. Intravascular bevacizumab and augmented Trabeculectomy for neovascular glaucoma in young diabetics. *Eye* 2009;23:979-81.
6. Gartner S, Henkind P. Neovascularization of the iris (rubeosis iridis). *Surv Ophthalmol* 1978;22:291-312.
7. Ashaye AO, Adeoti CO. Neovascular glaucoma in a Nigerian African population. *East Afr Med J* 2006;83:559-64.
8. Ellong A, Mvogo CE, Bella-Haig AL, Mouney EN, Ngosso A, Litumbe CN. Prevalence of glaucoma in a black Camroonian population. *Sante* 2006;16:83-8.
9. Calugaru D, Calugaru M. Neovascular glaucoma: Etiopathogeny and diagnosis. *Oftalmologia* 2012;56:3-14.
10. Strohl A, Pozzi S, Wattiez R, Roesen B, Miño de Kaspar H, Klaus V. Secondary glaucoma in Paraguay. Etiology and incidence. *Ophthalmologie* 1999;96:359-63.
11. Kiddee W, Tantisasart T, Wangsupadilok B. Neovascular glaucoma: A retrospective review of 5 year experience in Songkalangarin Hospital. *J Med Assoc Thai* 2012;19:S36-42.
12. Al-Shamsi HN, Dueker DK, Al-Shahan SA. Neovascular glaucoma at King Khaled Eye Specialist Hospital-Etiologic Considerations. *Middle East Afr J Ophthalmol* 2009;16:15-9.
13. Murkheji SK. Bevacizumab (avastin). *AJNR Am J Neuroradiol* 2010;31:235-6.
14. The Nigerian national blindness and visual impairment survey 2005-2007, page 3. Federal Ministry of Health, Abuja, Nigeria.

How to cite this article:***

Source of Support: Nil, Conflict of Interest: None declared