

Case Report

A rare case report on 90 day glaucoma due to branched retinal vein occlusion

Jawahar Jyoti Kuli, Utkarsh Rai*

Department of Ophthalmology, Assam Medical College, Dibrugarh, Assam, India

Received: 12 June 2021

Accepted: 08 July 2021

*Correspondence:

Dr. Utkarsh Rai,

E-mail: utkarshphoenix@gmail.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

A 19 year old female, presented to the ophthalmology OPD with pain and redness in her left eye since 30 days. Upon examination, there was no perception of light in left eye, both the pupils were mid-dilated and non-reactive to light stimulation. The left eye intra-ocular pressure (ATn) was 60 mm of Hg. On slit lamp examination, 3600 iris neovascularisation, ciliary congestion and corneal oedema were noted. Indirect gonioscopy revealed grade I angle closure in the left eye. Fundus examination of the affected eye revealed pale optic disc, CDR of 0.9:1, multiple dot and blot hemorrhages, suggestive of supero-temporal BRVO (branched retinal vein occlusion). Fundus fluorescein angiography revealed blocked fluorescence and leakage from neovascularization in the left eye. Conservative management with anti-glaucoma drugs failed to lower the IOP. Subsequently, pan-retinal photocoagulation (PRP) with intravitreal anti-VEGF (vascular endothelial growth factor) in the left eye followed by cyclo-cryoablation helped her symptoms to subside.

Keywords: Neovascular Glaucoma, BRVO, Iris Neovascularisation, Bevacizumab, Pan-Retinal Photocoagulation, Cyclocryoablation

INTRODUCTION

Neovascular glaucoma (NVG), also called as 90 day glaucoma, is a potentially blinding secondary glaucoma, characterized by development of neovascularisation of iris and angle of anterior chamber and elevated intra-ocular pressure (IOP) and in many instances, poor visual prognosis.^{1,2} For a logical understanding and scientific rationale for management of any disease, one first has to know the basic issues involved and the scientifically valid information available on the disease. To prevent or reduce the visual loss caused by NVG, the first essential is to have a high index of suspicion of its development, that is, to be aware of the various ocular diseases in which it can develop. Once it develops, early diagnosis and rational management are important to minimize the

visual loss. Therefore, the objective of this review on NVG was to discuss its causes, pathogenesis and pathology, methods of early diagnosis and finally management. The most common diseases majorly responsible for development of NVG are ischemic CRVO, proliferative diabetic retinopathy and ocular ischemic syndrome. In the management strategy, the first priority should be to try to prevent its development by appropriate management of the causative diseases. If NVG develops, early diagnosis is crucial to reduce the extent of visual loss. Management of NVG primarily consists of controlling the high IOP by medical and/or surgical means to minimize the visual loss. Currently there is still debate over satisfactory means of treating NVG and preventing visual loss in the majority, in spite

of multiple modes of medical and surgical options advocated over the years and claims made.

CASE REPORT

A 19 year old female reported to the department of ophthalmology, Assam medical college and hospital, Dibrugarh, Assam, with complaint of pain, dull aching in nature and radiating to the forehead and redness in her left eye for 30 days. She also reported diminution of vision, which was sudden in onset and gradually progressive in nature since 4 months. There was no significant medical or surgical history in the past. Her family history was insignificant.



Figure 1: Under diffuse illumination.



Figure 2: Rubeosis iridis.

Patient's examination revealed best corrected visual acuity (BCVA) OD 6/6, Negative for Perception of Light (PL) negative. External ocular findings were normal on the right side, while the left eye had circum-corneal congestion, hazy cornea and decreased corneal sensation. Pupil was vertically oval, mid-dilated and non-reactive to light stimulation, with iris neovascularisation. Anterior chamber depth was within normal limits with no cells or flare. IOP on applanation tonometry showed 26 mm Hg OD and 60 mm Hg OS.



Figure 3: Supero-temporal BRVO on funduscopy.



Figure 4: FFA showed blocked fluorescence and non-perfusion areas (NPA).

Indirect gonioscopy with 4-mirror goniolens showed normal angle structures on the right side and 360-degree neovascularisation with grade I angle closure on the left side. Funduscopy revealed a pale disc, CDR of 0.9:1, thinned out NRR in the supero-temporal part, bayoneting and nasally shifted optic disc vessels. The left eye foveal reflex was dull, multiple blot haemorrhages and ghost vessels were noted supero-temporally. Fundus fluorescein angiography of left eye revealed blocked fluorescence in supero-temporal peripheral fundus, areas of non-perfusion, vascular sheathing and leakage from neovascularization.

Treatment

The patient was started on anti-glaucoma medications, topical, periocular and intravitreal steroids, topical atropine and intravenous mannitol, but the IOP failed to subside. Subsequently, laser PRP with intra-vitreal anti-VEGF bevacizumab (avastin) followed by cyclo-cryoablation of the left eye showed improved IOP results.

DISCUSSION

NVG is defined as increased IOP associated with iris and/or angle neovascularization.² Coined by Weiss et al the anterior segment neovascularization in NVG is accompanied by the formation of a fibrovascular membrane that obstructs the aqueous outflow through the trabecular meshwork and results in rise of IOP.³ It is difficult to manage and often results in disastrous visual loss. NVG, with slightly higher prevalence in men, more commonly (75%) affects the elderly, in their 7-8th decade of life, with cardiovascular risk factors such as hypertension and diabetes and obstructive sleep apnea syndrome.^{4,5} NVG arise from retinal vascular occlusion in 36% cases, 32% from proliferative diabetic retinopathy and 13% from carotid artery occlusive disease.⁶ The case reported here was a female in her 2nd decade of life with no previous history of cardiovascular disorder. Neovascularization was rare in a truly non-ischemic CRVO and common in ischemic CRVO, rarely present in BRVO. The 3 year cumulative incidence of NVI was 8.5%.⁷ The case reported here was diagnosed with NVG secondary to BRVO. Neovascularization of iris (NVI) in most NVG cases is first seen on the peri-pupillary iris.^{8,9} Gonioscopy may show neovascularization of the angle (NVA) with angle anatomy ranging from completely open to focal or complete synechial closure.^{10,11} In our case there was neovascularization over the iris (rubeosis iridis) and angle. VEGF plays a major part in mediating active neovascularization in patients with ischaemic retinal disease. High levels of VEGF are found in the anterior chamber of patients with ischemic CRVO and PDR. Studies have demonstrated a close temporal correlation between aqueous VEGF levels and the degree of iris neovascularization.¹² Ocular hypertension and glaucomatous optic nerve cupping has been shown to be associated with BRVO occurring at the optic nerve head or optic cup without optic nerve head edema which was consistent with the optic disc architecture observed in the case reported.¹³ Management of NVG is still highly challenging and usually involves laser PRP as well as VEGF and corticosteroids to reduce the ocular ischemia causing the regression of NVI and reducing the IOP.¹⁴ Medical management with IOP lowering agents often yields intractable results. Trabeculectomy with anti-metabolite, although achieves greater lowering of IOP, is undesirable in presence of florid neovascularization, due to bleb failure through conjunctival scarring at the filtration site.¹⁵

CONCLUSION

NVG is a severe form of secondary glaucoma characterized by fibro vascular proliferation on iris and/or in the anterior chamber angle. Synonyms are hemorrhagic glaucoma, congestive glaucoma, thrombotic glaucoma, rubeotic glaucoma.¹⁶ Ischemic CRVO and diabetes are the most common causes, although very rarely, may be due to BRVO. There is a high likelihood of profound

vision loss by increased IOP, making early diagnosis key to preserving ocular function.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: Not required

REFERENCES

- Rodrigues GB. Neovascular glaucoma: a review. *Int J Retin Vit.* 2016;2:26.
- Călugăru D, Călugăru M. Neovascular glaucoma-etipathogeny and diagnosis. *Oftalmologia.* 2012;56(2):3-14.
- Albert DM, Jakobiec FA. Neovascular Glaucoma. In: Albert DM, Jakobiec FA, eds. *Principles and practice of ophthalmology.* 3rd ed. Philadelphia: WB Saunders Publishers; 1999.
- Pedroso L, Junior EDSC, PARanhos A, Junior JAP, Mello PAA. The real cost of glaucoma treatment for an outpatient. *Arq Bras Oftalmol.* 1999;62(6):677-82.
- Chen KH, Wu CC, Roy S, Lee SM, Liu JH. Increased interleukin-6 in aqueous humor of neovascular glaucoma. *Invest Ophthalmol Vis Sci.* 1999;40(11):2627-32.
- Brown GC, Magargal LE, Schachat A, Shah H. Neovascular glaucoma etiologic considerations. *Ophthalmol.* 1984;91(4):315-20.
- Chan CK, Ip MS, Vanveldhuisen PC, et al. SCORE Study report #11: incidences of neovascular events in eyes with retinal vein occlusion. *Ophthalmology.* 2011;118(7):1364-72.
- Madsen PH. Haemorrhagic glaucoma. Comparative study in diabetic and non-diabetic patients. *Br J Ophthalmol.* 1971;55(7):444-50.
- Ohrt V. The frequency of rubeosis iridis in diabetic patients. *Acta Ophthalmol (Copenh).* 1971;49(2):301-7.
- Browning DJ, Scott AQ, Peterson CB, Warnock J, Zhang Z. The risk of missing angle neovascularization by omitting screening gonioscopy in acute central retinal vein occlusion. *Ophthalmology.* 1998;105(5):776-84.
- Havens SJ, Gulati V. Neovascular glaucoma. *Retinal Pharmacotherapeutics.* Basel: Karger Publishers; 2016: 196-204.
- Shiba T, Takahashi M, Hori Y, Saishin Y, Sato Y, Maeno T. Relationship between sleep-disordered breathing and iris and/or angle neovascularization in proliferative diabetic retinopathy cases. *Am J Ophthalmol.* 2011;151(4):604-9.
- Klein BE, Meuer SM, Knudtson MD, Klein R. The relationship of optic disk cupping to retinal vein occlusion: Beaver dam eye study. *Am J Ophthalmol.* 2006;141(5):859-62.
- Ogino K, Tsujikawa A, Murakami T, Muraoka Y, Kurashige Y, Yoshimura N. Grid photocoagulation combined with intravitreal bevacizumab for

- recurrent macular edema associated with retinal vein occlusion. Clin Ophthalmol. 2011;5:1031-6.
15. Shen CC, Salim S, Du H, Netland PA. Trabeculectomy versus Ahmed glaucoma valve implantation in neovascular glaucoma. Clin Ophthalmol. 2011;5:281-6.
16. Mocanu C, Barascu D, Marinescu F. Neovascular glaucoma-Retrospective study. Ophthalmologica. 2005;49(4):58-65.

Cite this article as: Kuli JJ, Rai U. A rare case report on 90 day glaucoma due to branched retinal vein occlusion. Int J Res Med Sci 2021;9:2485-8.