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Case Report

90-DAY GLAUCOMA: A RARE CASE REPORT

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ABSTRACT

Neovascular glaucoma (NVG) also called as 90-day glaucoma, is a potentially devastating sequela of serious underlying ocular and/or systemic diseases. Anterior segment neovascularization involving the iris, the angle or both is accompanied by the formation of a fibrovascular membrane that obstructs the aqueous outflow through the trabecular meshwork and results in rise of IOP. Here we present a case of a fifty seven year old male who presented with diminution of vision in left eye since 45 days associated with pain and redness L/E since 15 days. On examination visual acquity of R/E was 6/6,N_6 and in L/E patient denied perception of light. Our case presented with almost all features suggestive of NVG except that of rubeosis iridis which makes it unique, as to the best of our knowledge this has been very rarely reported anywhere in literature before.

KEY WORDS: Neovascular Glaucoma, Congestive Glaucoma, Gender, Gonioscopy, Unilateral.

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INTRODUCTION

Neovascular glaucoma is defined as increased intraocular pressure associated with iris and/or angle neovascularization [1]. In 1963 Weiss and colleagues, proposed the term NVG [2]. It is also known as hemorrhagic glaucoma (referring to hyphema that is present in some patients), thrombotic glaucoma (underlying vascular thrombotic etiology) or rubeotic glaucoma [3].

Risk factors: The most common causes of NVG are central vein retinal occlusion, proliferative diabetic retinopathy and ocular ischemic syndrome, and central retinal artery obstruction [4].

Epidemiology: The incidence of NVG was similar between genders, with slight higher prevalence of men. It more commonly affects the elderly. It was observed that 46.16 % of the patients were between 60 and 79 years of age at onset and 30.68 % were over the age of 80 [5].

Clinical feature: The diagnosis of NVG is clinical and requires detailed patient's history and a complete ophthalmological examination. Case history is important to determine the origin of ischemia. Patients may be asymptomatic, especially when the IOP rise occurs slowly, or they can present with symptoms such as low vision, ocular pain and photophobia. In the early stages, exam findings can be subtle, requiring the ophthalmologist to maintain high index of suspicion in face of conditions that are commonly associated with the above mentioned etiology [6].

In rare cases, there may be neovascularization of angle without neovascularization of the pupillary border, especially after ischemic central retinal vein occlusion. Therefore, it is important to perform gonioscopy even when the border of the pupil is not involved.

Management: One of the strategies of medical management of NVG consists of IOP-lowering agents. Panretinal photocoagulation remains the mainstay in controlling the neovascular drive and should be considered in all cases of NVG when retinal ischemia is present [7]. Recently, use of anti-VEGF in the management of NVG has been extensively investigated [8]. Surgical interventions for NVG include: trabeculectomy with antimetabolites, glaucoma drainage devices, cyclophotocoagulation, among others.

CASE REPORT

A 57 year old hindu male, social worker by profession presented with the chief complain of diminution of vision L/E since 45 days associated with pain and redness in the same eye since 15 days(figure 1). The diminution of vision was gradual in onset and progressive in nature associated with pain which was dull aching in nature, radiating towards the left side of the forehead. Patient complained of seeing coloured halos on and off with one episode of vomiting.

Patient gave no previous history of taking medication for hypertension and diabetes mellitus. He gave history of drinking alchohol occasionally, regular intake of betel leaf and no history of smoking. There was no significant positive family history.

Examination revealed the right eye to have a normal vision with normal anterior and posterior segments. However, the left eye vision was reduced to just perception of light.

In left eye palpebral aperture was reduced as compared to the right eye. On slit lamp examination (figure 2) of L/E conjuctiva had circumcorneal and conjuctival congestion. Cornea was mildly hazy and corneal sensation was diminished; anterior chamber depth was within normal limit with no flare/cells. Pupil was vertically oval, mid dilated, non-reacting to light. Iris was normal in pattern; lens was within normal limit and Intra ocular pressure was 58 mmHg.

On gonioscopy of left eye by goldman single mirror gonioscope there was neovascularization around 360 degree, more at 6 o'clock. Angle structure could not be visualized.(figure 3). There was no new vascularization of iris seen.

On fundus examination optic disc margin was well defined. Colour of optic disc was pale. Cup:Disc $\geq$ 0.9; NRR : small rim in superior and nasal part; Bayoneting sign was present with presence of α and ß zone. There was nasal shifting of blood vessels and no disc haemorrhages seen.(figure 4)

Foveal reflex was dull with presence of multiple blot haemorrhages present superotemporally and above the macula. There was presence of ghost vessels inferotemporally and superotemporally.

Fig. 1: Patient On First Day.

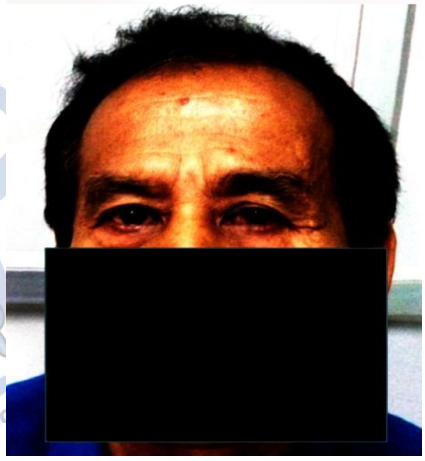


Fig. 2: Slit Lamp Examination.

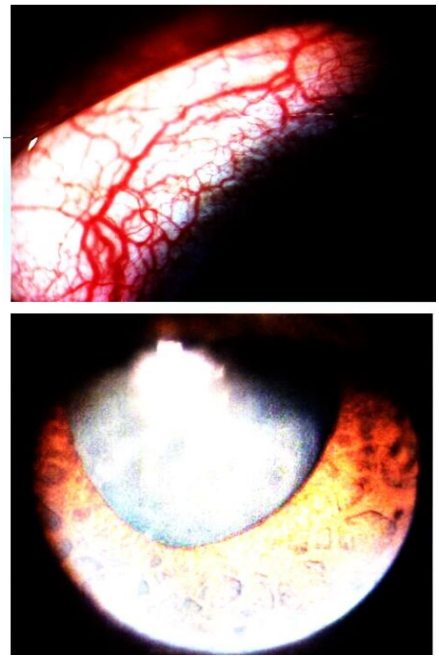


Fig. 3: Gonioscopy.

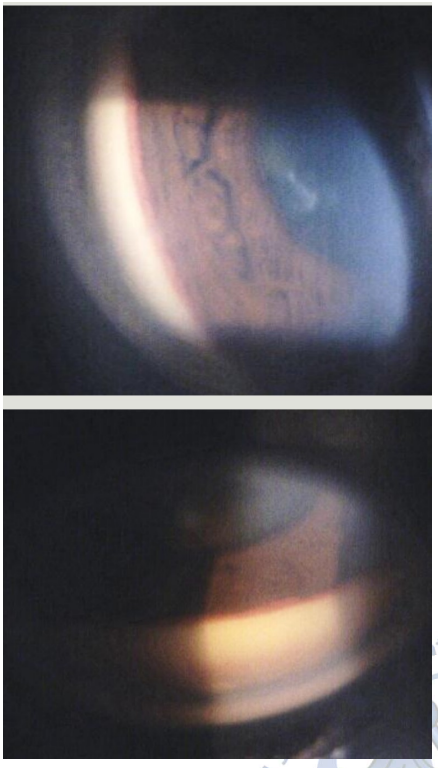


Fig. 4: Fundus Photograph.

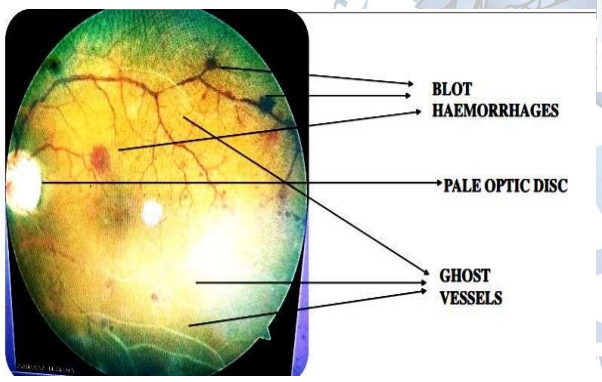
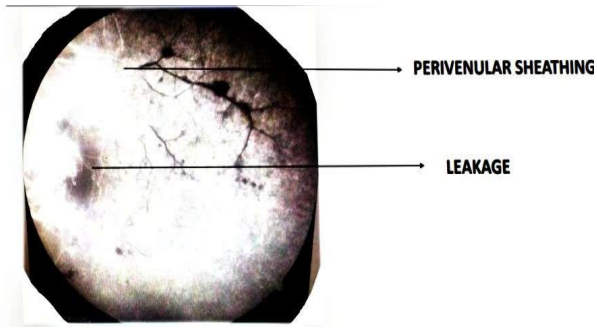
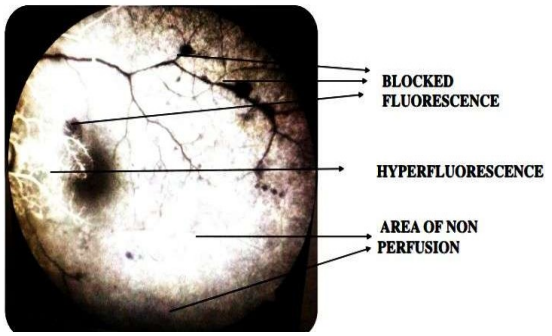


Fig. 5: Fundus Fluorescein Angiography.





Investigation

Routine Blood Investigations were done. **Hb %** - 14.9gm%; **ESR** – 15mm at the end of 1 hour; **Total leucocyte count** – 10,200 per cubic mm; **Differential leucocyte count (%)** – P- 65, E- 09, L- 19, B- 00, M- 07; **Prothrombin time** – 11.4sec (normal 10.5-12.5); **Activated partial thromboplastin time** – 22.4sec (normal 24-32sec); **INR** – 0.99 (normal); **Serum Na+** - 137.1 m mol/l (normal 135-148 m mol/l); **Serum K+** - 4.32 m mol/l (normal 3.5-5.3 m mol/l); **HbA1c**- 8.2%; **urine sugar**- 1+

Investigation	Observed value	Reference value
Fasting Blood Sugar	**173 mg/dl**	70-110 mg/dl
Post Prandial Blood Sugar	**243 mg/dl**	up to 140 mg/dl
Serum Cholesterol	162 mg/dl	150-250 mg/dl
Serum LDL Cholesterol	81 mg/dl	60-200 mg/dl
Serum HDL Cholesterol	**35 mg/dl**	40-60 mg/dl
Serum Triglyceride	**230 mg/dl**	30-150 mg/dl
Blood Urea	26 mg/dl	15-39 mg/dl
Serum Creatinine	1.07 mg/dl	0.55-1.30 mg/dl

Fundus fluorescein angiography showed blocked fluorescence; area of non perfusion; perivenular sheathing and leakage.(figure 5)

Hence the final diagnosis was made as Neovascular glaucoma with glaucomatous optic atrophy of left eye.

Treatment

· Inj. Mannitol (20%) 1 gm/Kg IV stat 200 ml was given.

· E/D Dorzolamide Hydrochloride(2%) + Timolol maleate (0.5%) -1 drop 3 times daily in L/E to continue

· E/D Brimonidine tartarate(0.2%) – 1 drop 2 times daily in L/E to continue

· E/D Atropine sulphate (1%) – 1 drop 3 times daily in L/E to continue

· E/D Prednisolone acetate(1%) – 1 drop 4 times daily in L/E to continue

· Tab Acetazolamide (250mg) – 1 tab 3 times daily after food to continue

· Tab Analgesic- 1 tab SOS

· Medicine consultation was taken for the raised blood sugar level and deranged lipid profile

Follow up: Patient's IOP was not lowered satisfactorily after two weeks in left eye and he was further planned for anti-VEGF followed by trabeculectomy with anti-metabolite. The IOP was stabilized below 24 mm of Hg and the patient is still in follow up. The further treatment modality is to place a ahmed glaucoma implant or cycloabalation. However his vision did not improve due to the already severely damaged optic nerve. The right eye was however normal.

DISCUSSION

The first report of neovascular glaucoma was made in 1871. In 1928, Salus, described new vessels on the iris of diabetic patients [9]. Rubeosis iridis and NVG has been associated with a wide range of conditions. Of these conditions, retinal ischemia accounts for the majority of the causes, with CRVO and diabetes retinopathy producing nearly two thirds of all cases of NVG [10].

NVG is more prevalent in elderly patients who have cardiovascular risk factors such as hypertension and diabetes, and may be more aggressive in those with obstructive sleep apnea syndrome [11]. In our case, patient is 57 years old with no previous history of cardiovascular disorder and hypertension. However patient's investigation revealed him with poor glycemic control.

The incidence of NVI among diabetic patients ranges from 1 to 17%[12]. Diabetics with NVG in one eye have a 33% risk of developing NVG in their other eye [13]. However in our case patient's other eye was within normal limit.

There is a high level of VEGF in the anterior chamber of patients with ischemic CRVO and PDR. A close temporal correlation between aqueous VEGF levels and the degree of iris neovascularization has been demonstrated [11] In our case however there was no neovascularization in iris which has been also shown by Browning DJ et al [12-14] in his study where he stated that Neovascularization in most cases is first seen on the peripupillary iris, although it may be first seen in the anterior chamber angle.

Vision is commonly at the level of counting fingers to hand motions, and IOP ranges from 40 to 60 mm Hg or higher. Gonioscopy demonstrates neovascularization of the angle (NVA) with angle anatomy ranging from completely open to focal or complete synechial closure [15] which is consistent with our case findings in which patient denied perception of light.

Ocular hypertension and glaucomatous optic nerve cupping has been shown to be associated with branch RVO occurring at the optic nerve head or optic cup without optic nerve head edema [16] which was consistent with our case's Optic disc architecture findings and fundus findings gave a picture of old CRVO.

Though NVG is the most dreaded and blinding complication of ischemic CRVO its management is still highly challenging, unpredictable, difficult and controversial. The initial management usually involves laser pan retinal photocoagulation (PRP) as well as intravitreal anti-vascular endothelial growth factor (VEGF) and corticosteroids to reduce the ocular ischemic thus in turn causing the regression of NVI and finally reducing the IOP [17].

However in a study conducted by Hayreh SS et al showed that if NVG is diagnosed, the medical treatment is aimed at reducing IOP. Common medications include agents that decrease aqueous humor production. Corticosteroids have been shown to lower IOP by increasing aqueous humor outflow in patients with NVG secondary to ocular inflammation [18] which was initially followed in our case too but with intractable result.

VEGF inhibitors such as bevacizumab target the primary pathway of neovascularization triggered by VEGF. In a recent systematic review and meta-analysis, Zhou M, *et al.* reported the use of intravitreal bevacizumab injection (IVB) pretreatment in NVG to be a safe and effective additional step during Ahmed glaucoma valve implantation [19].

Trabeculectomy with anti-metabolite offers the advantage of achieving lower post-operative IOP compared to shunts but is undesirable in the presence of florid neovascularization, which often results in bleb failure through conjunctival scarring at the filtration site [20]. In our case however the procedure was uneventful with good post-operative outcome and the patient is still in follow up.

PRP aims to reduce further neovascularization

and should be considered in cases of retinal ischemia-induced NVG, especially in the setting of PDR. In the setting of active hyphema and inflammation, an aqueous shunt is recommended. Immediate IOP control and subsequently better visual results have made the Ahmed valve glaucoma drainage device the implant of choice for NVG [21].

For non-clearing or recurrent VH, pars plana vitrectomy with intraoperative endolaser should be considered. Interestingly, studies have shown that a vitrectomy elevates oxygen tension for many months and, thus, decreases the risk of future neovascularization [20]. Anti-VEGF injections with or without PRP should be performed prior to any intraocular surgery in the setting of NVG. Cycloablative diode therapy (*e.g.,* CPC) may ultimately be necessary if filtering procedures, injections, and laser are unsuccessful in halting NVG progression.

To summarize, Our case presented with almost all features suggestive of NVG except that of rubeosis iridis which makes it unique as to the best of our knowledge this has been very rarely reported anywhere in literature before.

CONCLUSION

Early diagnosis is critical in the prognostication and initiation of prompt appropriate treatment. Every patient with NVG should undergo a comprehensive medical and ocular evaluation. Attention should be paid to pupillary responses (looking for an afferent pupillary defect), and detailed slit lamp, gonioscopic and dilated fundus examinations are required. Early undilated gonioscopy is essential in identifying early NVA and PAS.

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