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Clinical profile and outcomes of neovascular glaucoma in the era of anti-vascular endothelial growth factor

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Purpose: To report the clinical profile and visual impairment in various stages of neovascular glaucoma (NVG) at a tertiary eye center in East India. **Methods:** The electronic medical records of the hospital database of patients with neovascular glaucoma seen between 2013 and 2020 were reviewed. Gonioscopic details were used to stratify patients into nonspecified NVG (Group 1), open-angle NVG (Group 2), and closed-angle NVG (Group 3). The clinical profile, angle features, cause of NVG, systemic associations, visual impairment, and blindness (defined as logarithm of the minimum angle of resolution, LogMar >1.3 at baseline and at final follow-up), and outcomes of medical/surgical interventions were compared between the three groups. **Results:** Of 846 eyes of 810 patients with NVG (Group 1, n = 564 eyes, Group 2, n = 61 eyes, and Group 3, n = 220 eyes), at baseline, the blindness rates in Groups 3 and 2 were 90 and 75%, respectively. The time from a previous intervention to the onset of NVG ranged from 3 to 5 months, while the median duration of NVG was about 4–4.5 months (0.03–120 months). Multivariate regression identified a longer duration of NVG as the only variable associated with poor final visual acuity. **Conclusion:** Visual morbidity by NVG remains as high as 75–90% in developing countries, even with the availability of anti-VEGFs and after improved management/investigative at all stages.

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Neovascular glaucoma (NVG) represents a severe form of secondary glaucoma and accounts for 5–7% of all glaucoma worldwide.[1-3] This is often refractory to conventional treatment and is associated with high rates of visual morbidity.[4-7] Population studies and hospital-based studies report a prevalence of 0.5–9% visual morbidity across different parts of the world.[2,8-10] This results from ocular ischemia triggering neovascularization of the iris (NVI), angle (NVA), or the posterior segment (neovascularization elsewhere, NVE) with consequent obstruction of outflow channels in the eye. Raised intraocular pressure (IOP) and refractory glaucoma in NVG often require multiple interventions and a multidisciplinary approach. Antivascular endothelial growth factor (anti-VEGF) injections have significantly changed the treatment regimen and the visual outcome of NVG caused by retinal neovascular, and ischemic pathologies.[5,6,7,11,12] The significant causes of NVG reported across studies include diabetic retinopathy (DR), central retinal vein occlusions (CRVO), and ocular ischemic syndrome (OIS).[2,3] Earlier studies have reported the demographic profile of NVG caused by various etiologies with scanty reference to the stage of the disease or the rates of blindness in different disease stages.[8-10] Gomez *et al*.[9] reported 14% blindness in 350 NVG Mexican patients, with half of the eyes being blind at presentation. This study stratified NVG eyes into three stages while including eyes without raised IOP. Yet, this study did not analyze the differences in visual profiles in the various stages of the disease.

Glaucoma usually develops from a prerubeotic stage, wherein neovascularization remains quiescent until NVI/NVA make their appearance with eventual raised IOP and optic nerve damage.[2,3,13,14] Early and aggressive interventions in the earlier stages are likely to help prevent visual loss and disease progression. Yet, there is a lack of literature on the differences in the clinical profile of NVG, and the effects of interventions in eyes that present with open or closed-angle stages of NVG. This information is essential to identify and prognosticate NVG eyes at presentation. This is important to determine the crucial window period for timely management directed toward reducing preventable blindness in this entity. This study evaluates the clinical profile and visual impairment in the various stages of NVG.

Methods

The hospital database of electronic medical records of patients with NVG seen in the glaucoma clinic at a tertiary eye center in East India between 2013 and 2020 was reviewed. The institutional review board of LV Prasad Eye Institute, MTC campus, Bhubaneswar, approved this retrospective study that adhered to the tenets of Helsinki's declaration. Clinical details that were collected at presentation and follow-up visits included age, gender, best-corrected visual acuity (BCVA), IOP, number of medicines, associated systemic and ocular comorbidities, gonioscopic details of the angle, lens status, fundus examination (when possible), type and outcome of medical/surgical interventions, history of previous surgery,

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duration from the last diagnosis to first intervention for NVG, and time of onset of NVG after diagnosis of primary disease (like DR, CRVO). Snellen visual acuity in the affected eye was converted to logarithm of the minimal angle of resolution (LogMar) scale for analysis and categorized into various levels as follows:

Good vision – LogMar BCVA > 0.5, poor vision – 0.5–1.3, and severe visual impairment/blindness – >1.3 (blindness here refers to the affected eye as an independent unit).

This classification was used to identify eyes that had no vision at presentation or at final follow-up. The gonioscopic details retrieved for analysis included the structures seen in each quadrant, presence, or absence of NVI/NVA. This was used to stratify eyes into Group 1 with a nonspecified status of the angle (in eyes with corneal edema/opacification/disfigured atrophic globe or other causes precluding angle examination at any visit and not just end-stage disease), Group 2 with open angles, and Group 3 with closed angles.

We also noted the number of patients who had a follow-up <6 months, 1 year, 2 years, 3 years, and >5 years and compared variables in those with longer follow-up. The duration of NVG was defined as the time from onset of symptoms of reduced vision, redness, pain with or without raised IOP to the first diagnosis, and treatment of NVG. The time from diagnosis of primary disease to time of diagnosis of NVG and the time between last retinal interventions for the primary disease to the diagnosis of NVG were also retrieved from the database.

As per institutional protocol, standard care for each primary disease (cause for NVG like DR, CRVO, etc.) is given by the respective departments (retina, glaucoma services). Medical therapy for raised IOP was undertaken as per standard guidelines, with surgery advocated for uncontrolled IOP despite medical treatment or intolerance to antiglaucoma medications. The glaucoma surgeries done for such cases included glaucoma drainage devices, trabeculectomy, and diode laser trans-scleral cylcophotocoagulation (TSCPC). Success was defined as an IOP between 6 and 21 mmHg with or without topical or systemic glaucoma medications after surgery, while failure was defined as IOP <6 mm or >21 mm Hg despite medicines after 6 weeks of surgery, or decrease of ≥2 lines on LogMar, or loss of vision.

All statistical analyses were done using Stata with $P < 0.05$ considered as statistically significant. Descriptive statistics are represented as Mean + standard deviation (or median/range for nonparametric) continuous variables or proportions for categorical variables. Normality was analyzed using the Kolmogorov–Smirnov test with non-normal variables being depicted as median (interquartile range). One-way ANOVA or Kruskal–Wallis test with posthoc Bonferroni, or Dunn's test, was used to compare variables between the groups. A univariate and multivariate regression model was used to evaluate the association of clinical prognostic factors or the outcome in each eye with the multivariate regression model, including only those variables with $P < 0.2$ in the univariate analysis. For bilateral disease, each eye was taken as a separate entity. Since both eyes can be correlated to each other, the results were repeated after excluding the better eye in 18 bilateral NVG cases to see if the correlation caused any bias in results.

Results

We reviewed 846 eyes of 810 patients with NVG seen during the period, which included 18 patients with bilateral NVG, 220 eyes with closed angles, 61 eyes with open angles, and 564 eyes with nonspecified angle status.

The mean age at presentation was not significantly different in the three groups [Table 1]. Eyes with closed angles and nonspecified angle status presented with worse presenting BCVA and higher presenting IOP, $P = 0.001$ for each. A systemic association like diabetes mellitus and hypertension were seen in 40–50% of eyes, with the maximum associations seen in Group 2. Preexisting glaucoma was seen in 15–20% of eyes in all three groups, with bilaterally blind eyes seen in three of the patients. The blindness rates at baseline (as defined earlier as LogMar VA >1.3) were 90% of Group 3 eyes and 75% in Group 2, reflecting the massive burden of visual morbidity in this cohort [Fig. 1].

The causes for NVG included CRVO/BRVO seen in around 40%, while a third of NVG were caused due to DR. OIS was seen in only 21 eyes, with no OIS eyes seen in Group 2 [Table 2]. The other causes of NVG included tumors, old retinal detachments, vasculitis, Vogt-Koyanagi-Harada, uveitis, pseudoexfoliation (PXF), retinitis pigmentosa, microbial keratitis, and endophthalmitis.

The median time from a previous intervention to the onset of NVG ranged from 3 to 5 months in the three groups (range 0.03–192 months). Seventeen eyes with CRVO presented < 3months after the initial interventions such as laser panretinal photocoagulation (PRP), while the median duration of NVG was about 4–4.5 months (range 0.03–120 months). The time of onset to initiation of treatment after diagnosis of NVG ranged from 2 TO 3 months (range 0.2–240 months) with the lowest duration of NVG seen in Group 2. This again reflects that the eyes of Group 2 patients represent an earlier form of the disease that requires aggressive treatment.

Clinical features like AC flare were seen in only 11–23% cases across the groups, while NVA, neovascularization of the disc, and NVE were seen in < 25% of eyes in Groups 1 and 3, and 70–80% of the eyes in Group 2 [Table 2]. This reflects the poor visibility of the angle in these groups compared to the open-angle eyes with a clear cornea. Previous intravitreal anti-VEGF injections (before NVG diagnosis) had been given for 13–22% of eyes across the groups. More than 50% of the eyes in Groups 1 and 2 required retinal interventions (PRP, anti-VEGF injections, or vitreoretinal surgery), which may reflect a larger number of Group 3 eyes with poor prognosis that were not amenable to any treatment. Glaucoma interventions like TSCPC were done maximally in the eyes of the patients in Groups 1 and 3 with limited TSCPC (parts of one quadrant) done in 10–12% of eyes in the two groups. The final IOP was significantly lower in eyes with open-angle, $P = 0.001$ [Table 2 and Fig. 2]. Despite interventions, improvement of visual acuity was seen in only 7–20% eyes, while the majority had no change in BCVA or deteriorated [Table 2].

The final visual acuity and blindness rates were not different between the groups, with an increase in the number of eyes with a poor vision seen at final follow-up in all the groups [Tables 1 and 2].

Table 1: Clinical profile of patients with different stages of neovascular glaucoma (NVG)

	Group 1* *n*=564	Group 2* *n*=61	Group 3* *n*=220	*P*#
Age	60±15.03	58±13.6	61±14.7	0.4
Male:female	430 :134	52:9	152:68	0.7
Bilateral	6	1	11	0.8
Systemic associations				
DM	229 (40%)	46 (75%)	106 (48%)	0.06
HYT	217 (38%)	38 (62%)	110 (50%)	0.001
CVA	8 (0.01%)	0	0	0.002
Others	92 (16.3%)	9 (14.7%)	4 (1.8%)	0.03
Lens status				
Phakic	437 (78%)	52 (85%)	188 (85%)	0.7
Aphakic	10 (1.7%)	9 (15%)	0	0.01
Pseudophakic	117 (20.3%)	0	32 (15%)	0.001
BCVA criteria	2±0.4	2±3.05	2±0.4	0.0001
Final VA	2±0.4	2±0.7	3±0.6	0.001
Baseline IOP	39±13.4	32±13.09	36±14.03	0.003
Final IOP	33±15.8	28±14.5	36±20.8	0.001
Follow-up duration (months, range)	12±22.8 (0-144)	6±5.9 (0-31)	35±15.3 (0.5-143)	0.001

*See the text for detailed description of groups/stages of NVG; #*Post hoc* test reveal maximal difference between Groups 1 versus 2 and Group 3 versus 2; IOP - Intraoccualr pressure in mm Hg; BCVA - Best-corrected visual acuity, VA - Visual acuity; IOP - intraocular pressure; DM - Diabetes mellitus; HYT - hypertension; CVA - cardioavascular abnormalities

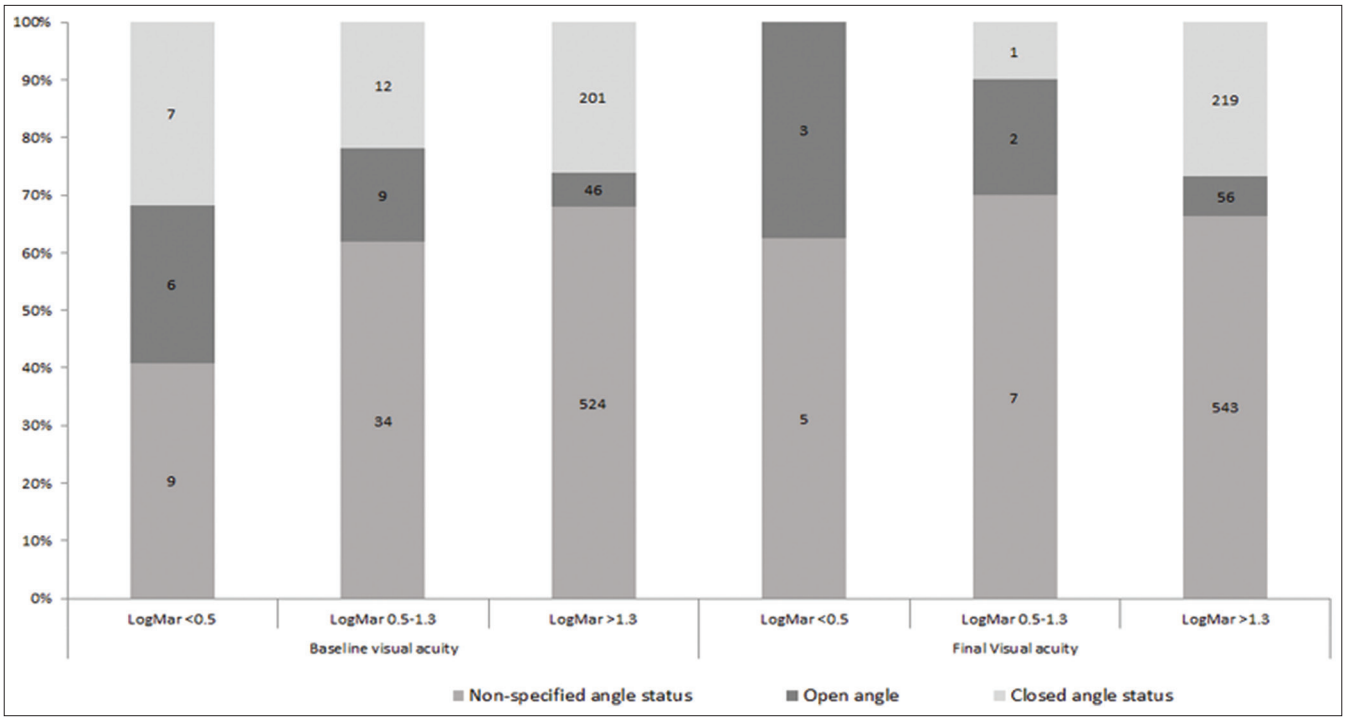

Figure 1: Stacked percentage bar showing percentage of eyes with different visual acuity (labels indicate actual number of eyes) in eyes with different stages of neovascular glaucoma at baseline and final visit

Comparing the difference in eyes with different causes of NVG, those with OIS presented early compared to other causes, Table 3, though this difference was not statistically different. The baseline IOP or BCVA did not differ between the different causes, while OIS again presented with the least duration of NVG. The final IOP also did not differ in these eyes [Table 3].

We also compared eyes with different follow-up periods and did not find any significant difference in the final IOP or final vision in eyes that followed-up later versus those that -p 6–12months [Table S1]. To see the bias caused by the correlation between two eyes of the same patient, we repeated the analysis after excluding the better eye in 18

Table 2: Causes and clinical profile of neovascular glaucoma

	Group 1 *n*=564	Group 2 *n*=61	Group 3 *n*=220	*P*
Preexisting glaucoma *n*, %	115 (20.3%)	10 (16%)	35 (15.9%)	0.4
Cause for NVG (*n*)				
VO	249	29	96	0.8
DR	179	25	85	0.4
OIS	15	0	6	0.06
Blindness in affected eye (*n*)				
Baseline	524	46	201	<0.001
Final	543	56	219	<0.001
AC flare (*n*)				
Present	133	7	25	0.054
Absent	291	54	195	0.2
Not visualized	140	0	0	0.01
NVA (*n*)				
Present	118	38	53	0.02
Absent	139	14	101	0.3
Not visualized	297	9	66	0.3
NVD (*n*)				
Present	132	48	45	0.4
Absent	72	2	4	0.09
Not visualized	360	11	170	0.5
NVE (*n*)				
Present	161	48	49	0.3
Absent	36	2	1	0.8
Not visualized	365	170	11	0.2
Time from previous intervention to NVG diagnosis (months, median, and range)	5 (0.03-192)	3 (0.5-8)	5.5 (0.25-84)	0.08
NVG duration (Months)	9±20.9 3 (0.2-240)	4±5.6 2 (0.2-26)	3±5 3 (0.025-48)	0.002
No of eyes that had previous anti-VEGF *n*, %	74 (13%)	16 (22%)	36 (16%)	0.1
No of eyes that underwent glaucoma surgery (*n*)				
TSCPC	137	5	33	0.02
Glaucoma Surgery	2	5	9	0.4
Anti-VEGF	51	14	15	0.1
PRP	49	11	16	0.8
VR surgery	24	5	4	0.07
Anti-VEGF + ARC	11	0	5	0.3
Others	33	6	23	0.2
Visual Outcome (*n*)				
No change	226	31	75	0.2
Improved	43	8	44	0.1
Deteriorated	120	9	37	0.2

AC - anterior chamber; NVD - neovascularization of the disc; NVE - neovascularisation elsewhere; NVA - neovascularization of the angle; ARC - anterior retinal cryopexy; TSCPC - trans-scleral cyclophotocoagulation; PRP - pan retinal photocoagulation; VEGF - Vascular endothelial growth factor; VR - vitreo-retinal; DR - diabetic retinopathy; DM - Diabetes mellitus; HYT - hypertension; OIS - Ocular ischemic syndrome; VO - vein occlusions

Table 3: Comparison of clinical profile of patients with different etiology of neovascular glaucoma

	Vascular occlusions *n*=307	Proliferative DR *n*=285	OIS *n*=21	Others *n*=234	*P**
Age (years)	61±14.4	61±14.7	58±14.9	59±14.9	0.3
BIOP (mm Hg)	39±13.2	37±14.4	38±12.1	36±13.2	0.3
BCVA	2±0.4	2±1.5	2±0.5	2±0.3	0.09
Duration of NVG (months)	9±19.03 0.03-120	11±23.1 0.25-192	1±0.5 1-2	11±14.1 0.1-48	<0.001

*Kruskal-Wallis test; BCVA - Best-corrected visual acuity; NVG - neovascular glaucoma; BIOP - baseline Intraocular pressure; DR - Diabetic retinopathy; OIS - ocular ischemic syndrome

bilateral NVG eyes and found no significant difference in the results.

Univariate analysis regression showed a significant association of blindness (LogMar >1.3) in NVG to presenting

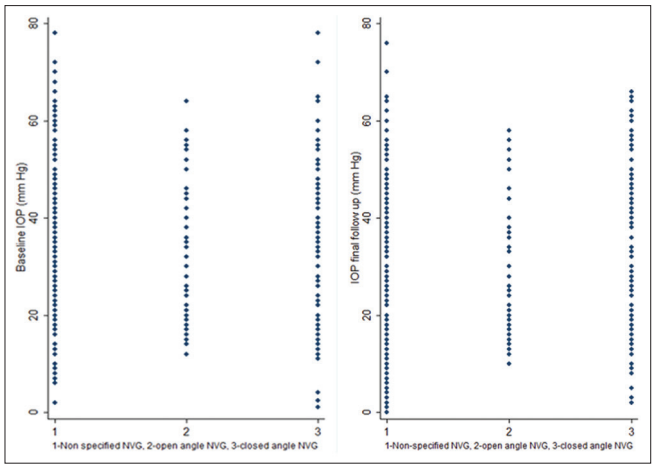

Figure 2: Baseline and final intraocular pressure in eyes with different stages of neovascular glaucoma

visual acuity ($r = 0.4$, $P = 0.04$), duration of NVG ($r = 0.7$, $P = 0.01$), and closed-angle status ($r = 0.3$, $P = 0.045$). Multivariate regression, however, showed a longer duration of NVG before initiation of treatment as the only variable associated with poor final visual acuity. No association was found between blindness and the number of previous injections or interventions, baseline or final IOP, presence of NVA/NVE, or any other clinical variable.

Discussion

One-third of patients in all groups had received previous anti-VEGF injections before the diagnosis of NVG in this study. Eyes with OIS presented earlier than other DR or CRVO though this difference was not statistically significant. While vascular occlusions were the predominant cause for NVG in this cohort, several eyes presented in less than three months duration with NVG. Visual impairment and blindness ranged from 75-90% in this cohort, reflecting a huge visual burden and morbidity attributed to NVG. Improvement of vision was seen in an exceedingly small proportion of cases (7–20%) despite adequate and prompt standard of care. The duration of NVG was the only predictor of a poor visual outcome at all stages.

NVG represents an aggressive form of ocular disorder caused by secondary insults like DR, OIS, or vascular occlusions. It constitutes one of the aggressive and refractory forms of secondary glaucoma, with the need for surgeries for IOP control and control of ischemia. Earlier studies have reported poor surgical outcomes in eyes with NVG and have also reported low success rates of different glaucoma filtering surgeries in NVG eyes. The prevalence of NVG across studies has been reported to range between 7 and 10%. Indian and global studies report similar prevalence rates of NVG with a high rate of visual impairment.[1-3] We found 75–90% eyes to be blind at presentation in this cohort, with little improvement in vision despite aggressive interventions, which is higher than in previously reported studies.[2,3,8-10]

The duration of NVG was the only predictor of a poor visual outcome in this study. A worse visual acuity at baseline and history of systemic hypertension has been reported to predict NVG in CRVO eyes.[4] Preoperative parameters such as elevated IOP, iris/angle neovascularization, fasting blood sugar and HbA1c discrepancies, administration of insulin, and use of retinal tamponade during retinal surgery are also identified as risk factors for developing NVG in eyes undergoing vitrectomy in proliferative DR.[2,4,5-14] The risk of NVG increases with the length of time between the onset of symptoms and diagnosis, as well as with the severity of ipsilateral carotid artery stenosis in eyes with OIS.[5,6,15-21] Our study did not find baseline VA, IOP, or systemic association as a predictor for a poor visual outcome. This could be attributed to the retrospective study design and advanced disease at presentation, which is typical in a developing country.

A closed-angle represents a clinically advanced stage of NVG disease with progressive closure of the angle by the neovascular membrane.[15,17-21] This study concurred with the traditional wisdom with a poorer prognosis seen in eyes with closed angles. Yet, presenting clinical features and duration of NVG were not significantly different in these eyes, suggesting that other subclinical factors like VEGF load may be responsible for driving the disease toward an aggravated course compared to other eyes. The natural course of CRVO reports the onset of glaucoma after 3 months after an episode of vasoocclusive disease.[2,3,13,14] Yet, we had several eyes which presented within one month of CRVO diagnosis. It may be possible that these eyes represent a worse disease at presentation, a higher VEGF load, or simply represent an underreported duration of symptoms.

This was a retrospective study with its inherent limitations. We did not study fluorescein angiography features, which also may be presumably different in different etiologies and eyes with different stages. Being a retrospective study, the follow-up was variable. It may be possible that patients with initial improvement after interventions can again show a decline owing to recurrence or disease progression; yet, we do not believe the visual morbidity rates would be very different (least expected to be better) if we had patients with longer follow-up.

Conclusion

We believe that our study supports the traditional knowledge of early intervention in the open-angle NVG stage while reflecting the considerable burden of baseline visual impairment in all stages of NVG in developing countries, even in the era of the improved availability of anti-VEGF agents and improved treatment algorithms.

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Conflicts of interest

There are no conflicts of interest.

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Table S1: Comparison of clinical profile of patients with neovascular glaucoma with varying duration of follow up

	>6 m *n*=153	>1 year *n*=66	>2 year *n*=44	>3 year *n*=44	>5 years *n*=143
Age	60±14.9	59±11.4	54±14.7	60±12.3	62±14.2
Baseline IOP	37±14.4	40±13.1	35±14.1	36±13.8	38±13.6
Final IOP	35±23.03	32±16.8	28±15.9	28±7.4	29±15.4
BCVA	2±0.5	2±0.5	1±0.7	2±0.6	2±0.4
Number of previous anti-Vegf	21	17	15	5	21
Retinal sx	61	24	21	23	31

BCVA - Best corrected visual acuity; NVG - neovascular glaucoma; DR-Diabetic retinopathy; OIS - ocular ischemic syndrome; VEGF - vascular endothelial growth factor; IOP - intraocular pressure