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Original Research Article

A study on the effect of Anti- VEGF on retinal venous occlusive disease with macular edema in a tertiary health care centre of southern Assam

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ABSTRACT

Background: Macular oedema is considered to be the primary reason for significant visual loss and impaired visual recovery in patients with retinal venous occlusion and rise in VEGF is strongly implicated in macular edema and as such there is a good rationale in treating these conditions with intravitreal anti-vascular endothelial growth factors. Aim of this study was to assess the effect of anti-VEGF (Bevacizumab) on retinal venous occlusive disease with macular edema.

Materials and Methods: This was prospective open label hospital based interventional study done from June 2021 to May 2022 in fifty eyes of fifty patients diagnosed with macular edema secondary to retinal venous occlusion and Snellen's visual acuity at presentation of less than 6/12 were included in accordance with the inclusion and exclusion criteria.

Results: The results shows statistically significant improvement in mean central macular thickness and mean Snellen's visual acuity at the end of 12 months and factors like Hypertension, Diabetes mellitus, Dyslipidemia and smoking are found to be highly associated with retinal venous occlusive diseases.

Conclusion: The intravitreal anti-VEGF (Bevacizumab) has been found to be effective in improving the macular edema and visual acuity in the study population with retinal venous occlusive diseases to a statistically significant level and that it was more pronounced in cases of BRVO, at the completion of the study period.

1. Introduction

Retinal vein occlusion is one of the most common vascular diseases of the retina and is the second most prevalent cause after diabetic retinopathy.[1] Retinal vein occlusion affects around 180 000 eyes per year in the United States, with branch retinal vein occlusion being more prevalent than central retinal vein occlusion accounting for about 80% of RVO cases.[2] In a population-based study of 4711 subjects in central India, RVO was detected in 0.8 per cent of the population. The prevalence of BRVO was estimated to be seven times that of CRVO.[3] Visual recovery in RVO depends on the extent of ischemic damage and on the development of macular edema during the early stages. Macular oedema is considered to be the primary reason for significant visual loss and impaired visual recovery in patients with RVO. This edema is caused by hypoxia-induced upregulation of vascular endothelial growth factor (VEGF), which loosens endothelial tight junctions, increasing vascular permeability and exudative material deposition.[4] The loss of vision in the eye can be severe or mild, but it usually occurs suddenly and painlessly. Recent years have seen the successful application

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of antiangiogenic therapy with anti-vascular endothelial growth factor (anti-VEGF) to treat macular edema due to a wide range of aetiologies. Because patients with retinal vein occlusions have been shown to have elevated intraocular levels of VEGF, there is a solid foundation for the hypothesis that anti-VEGF may be helpful in the treatment of vascular leakage and macular edema.[5,6] Several multicentric studies have shown the efficacy of anti-VEGF (bevacizumab) treatment in reducing intraretinal fluid and improvement in visual acuity (VA) in about 60% of the patients after treatment with anti-VEGF. However, only few studies have evaluated the role of anti-VEGF in macular edema associated with RVO in the Indian population and none to our knowledge in Southern Assam.

2. Materials and Methods

The study was conducted for a period of one year starting from June 2021 to May 2022.

All the patients fulfilling the inclusion and exclusion criteria of the study were included. Patients aged > 30 years of both sexes, diagnosed to have macular edema secondary to retinal vein occlusion, confirmed with fundoscopy, 90 Dioptre lens, and Spectral Domain Ocular Coherence Tomography (SD-OCT) showing central macular thickness >250 microns and Snellen's visual acuity <6/12 were included in the study. Additional eye diseases that could compromise visual acuity, ocular inflammation, and intraocular surgery ≤1 month before presentation, uncontrolled glaucoma, prior treatments with laser photocoagulation or other intervention for macular edema due to retinal vein occlusion and pregnant subjects were excluded from the study. A total of 50 patients fulfilled the criteria and total 50 eyes were included in the study. Our study was a drug interventional, open-label, prospective study to collect the efficacy of anti VEGF (Bevacizumab) in patients with visual impairment due to macular oedema secondary to retinal vein occlusion. Eligible subjects were given 3 doses of intravitreal Bevacizumab 1.25 mg (0.05 ml volume) at 1 month interval following immediate diagnosis/ initial presentation. All participants were followed up at 1st, 2nd, 3rd, 6th, 9th and 12th months with IDO, 90dioptre lens and SD-OCT. The study was cleared by the Institutional ethical committee and written informed consent was obtained from all participants prior to enrolment.

2.1. Sample size

$$\frac{2\sigma(Z_{\beta}+Z_{\alpha/2})^2}{d^2}$$

$Z_{\alpha\ 2} = Z_{0.05/2} = Z_{0.025} = 1.96$ (From Z table) at type 1 error of 5%

$Z_{\beta} = Z_{0.20} = 0.842$ (From Z table) at 80% power

d = effect size = difference between mean values

Considering a minimum difference of 5 points with pooled SD 7.5 a minimum of 47 samples required to detect 80% power at a 95% confidence interval. On rounding up The sample size in our study is taken as 50.

3. Results

3.1. Baseline characteristics

A total of 50 patients fulfilled the criteria and 50 eyes were included in the study. The mean age was 58.54 ± 8.70 years, and the median age was 60 years, ranging from 30 to 80 years (Table 1). The mean Snellen's visual acuity at presentation (baseline) was found to be 0.1806 ± 0.06729 (approximately 6/36). Of the total 50 cases of RVO, 34 cases (68%) were BRVO, 12 cases (24%) were Non-ischemic CRVO and 4 cases (8%) were ischemic CRVO.

Table 1: Baseline characteristics

Sex	Frequency	Percent
M	27	54
F	23	46
Age		
30-40	3	6
41-50	3	6
51-60	24	48
61-70	18	36
71-80	2	4

3.2. Associated systemic disease

The cases were associated with systemic diseases such as diabetes mellitus- 2%, diabetes mellitus with dyslipidaemia-6%, hypertension-44%, hypertension with diabetes mellitus-10%, hypertension with diabetes mellitus and smoking- 10%, hypertension with dyslipidaemia 10%, hypertension with smoking-16%, smoking- 2% (Table 2).

Table 2: Associated systemic disease

Associated Systemic Disease	Number	Percentage
Diabetes mellitus	1	2
Diabetes mellitus / dyslipidemia	3	6
Hypertension	22	44
Diabetes mellitus/ Hypertension	5	10
Hypertension/smoking/ Diabetes mellitus	5	10
Hypertension/ dyslipidemia	5	10
Hypertension/ smoking	8	16
Smoking	1	2
Total	50	100

3.3. Visual acuity

At the end of one month, the mean Snellen's visual acuity was found to be 0.2558 ± 0.08564 (approximately 6/24)

with a mean difference of 0.752 (41.6%) from baseline Snellen's visual acuity (p <0.001).

At the end of two months, the mean Snellen's visual acuity was 0.3256 ± 0.11 (approximately between 6/24 and 6/18) with a mean difference of 0.145 (80.3%) from baseline Snellen's visual acuity (P <0.001) and mean difference of 0.0698 (p<0.001) from Snellen's visual acuity at the end of one month.

At the end of three months, the mean Snellen's visual acuity was found to be 0.3446± 0.12 (approximately 6/18) with a mean difference of 0.164(90.8%) from baseline Snellen's visual acuity (p<0.001) and mean difference of 0.019 (p=0.005) from Snellen's visual acuity at the end of two months.

At the end of six months, the mean Snellen's visual acuity was found to be 0.3398± 0.12 (approximately 6/18) with a mean difference of 0.1592 (88.2%) from baseline Snellen's visual acuity (p<0.001) and mean difference of 0.0048 (p=0.083) from Snellen's visual acuity at the end of three months.

At the end of nine months, the mean Snellen's visual acuity was found to be 0.3377 ± 0.11 with a mean difference of 0.1571 (86.98%) from baseline Snellen's visual acuity (p<0.001) and mean difference of -0.0021 (p=0.083) from Snellen's visual acuity at the end of six months.

At the end of twelve months, the mean Snellen's visual acuity was found to be 0.3343 ± 0.11 with a mean difference of 0.1537 (86.98%) from baseline Snellen's visual acuity (p<0.001) and mean difference of -0.0034 (p=0.083) from Snellen's visual acuity at the end of the ninth month (Tables 3 and 4).

3.4. Central macular thickness

We also evaluated the central macular thickness (CMT) at baseline and at presentation, first, second, third, sixth, ninth- and twelfth-month using SD-Optical Coherence Tomography. Mean central macular thickness at presentation (baseline) was found to be 534.2±85.622 microns. At the end of one month, the mean CMT was found to be 421.3±81.407 microns with a mean difference of -112.9 micron (-21.1%) from baseline (at presentation) CMT (p<0.001). At the end of two months, the mean CMT was found to be 338.58±70.13 micron with a mean difference of -195.64 micron (-36.6%) from baseline (at presentation) CMT (p <0.001). At third months, the CMT was found to be 307.3±58.382 micron with a mean difference of -226.92 micron (-42.5%) from the baseline (at presentation) CMT (p<0.001) At six months, the mean CMT was found to be 297.04 ±68.42 microns with a mean difference of -237.18 microns (- 44.4%) from baseline (at presentation) CMT (p<0.001). At ninth month, the mean CMT was found to be300.04 ±68.40 microns with a mean difference of -234.18 microns (- 43.83%) from baseline (at presentation) CMT (p<0.001). At twelfth months, the mean CMT was found to be 304.09 ±67.82 microns with a mean difference of -230.13 microns (- 43.07%) from baseline (at presentation) CMT (p<0.001) (Table 5).

3.5. Visual acuity in ischemic CRVO, non- ischemic CRVO and BRVO

In BRVO patients the mean Snellen's visual acuity at presentation (baseline) was found to be 0.2056± 0.0513 (approximately between 6/36 and 6/24).

At the end of one month, the mean Snellen's visual acuity was found to be 0.2879 ± 0.06089. At the end of two months, three months, six months, nine months and twelve months the mean Snellen's visual acuity was found to be 0.3729 ± 0.08625, 0.3853 ± 0.08802, 0.3829± 0.09057, 0.3799± 0.09047 and 0.3789 respectively (Table 6).

In ischemic CRVO patients the mean Snellen's visual acuity at presentation was found to be 0.0475±0.00957 (approximately between 3/60 and 2/60).

At the end of one month, two months, three months, six months, nine months and twelve months the mean Snellen's visual acuity was found to be 0.06 ± 0.01414, 0.07 ± 0.02, 0.08 ± 0.01633, 0.08± 0.01633, 0.079 ± 0.01633 and 0.072 ± 0.01633 respectively (Table 6).

3.6. Non-ischemic CRVO

In non-ischemic patients the mean Snellen's visual acuity at presentation (baseline) was found to be 0.1542±0.05583 (approximately 6/36).

At the end of one month, two months, three months, six months, nine months, and twelve months the mean Snellen's visual acuity was found to be 0.23 ± 0.0603, 0.2767 ± 0.05211, 0.3175 ± 0.06917, 0.3042± 0.08096, 0.3033± 0.08085 and 0.3022 ± 0.3022 respectively (Table 6).

3.7. Time of presentation

The number of patients in the BRVO group presenting at less than 1 week, 1-2 weeks, 2-3 weeks, 3-4 weeks and more than 4 weeks were 1,3,4,10 and 16 respectively. Similarly, in the non-ischemic CRVO group it was 3, 2, 1, 6 respectively. In the ischemic CRVO group 2 patients presented within 1 week and 2 between 1 and 2 weeks (Table 7).

4. Discussion

We prospectively studied the effects of three doses of anti-VEGF (Bevacizumab) intravitreal injection at regular interval of one month, in retinal venous occlusive disease with macular edema. The intravitreal injection used was Bevacizumab. Our study found significant improvement in visual acuity and central macular thickness over the course of 12 months of follow-up.

Retinal vein occlusion (RVO) remains one of the most common retinal vascular disorders and branch retinal vein

Table 3: Snellen's visual acuity at presentation and monthly interval

	Mean	SD	Mean Diff from Baseline	% Change from Baseline	p value
VA0	0.1806	0.06729			
VA1	0.2558	0.08564	0.752	41.6%	<0.001
VA2	0.3256	0.11454	0.145	80.3%	<0.001
VA3	0.3446	0.11555	0.164	90.8%	<0.001
VA6	0.3398	0.11885	0.1592	88.2%	<0.001
VA9	0.3377	0.11675	0.1571	86.98%	<0.001
VA12	0.3343	0.11565	0.1537	85.1%	<0.001

Table 4: Mean difference in Snellen's visual acuity at monthly intervals

	Mean Difference	p value
VA0 - VA1	0.0752	<0.001
VA0 - VA2	0.145	<0.001
VA0 - VA3	0.164	<0.001
VA0 - VA6	0.1592	<0.001
VA1 - VA2	0.0698	<0.001
VA1 - VA3	0.0888	<0.001
VA1 - VA6	0.084	<0.001
VA2 - VA3	0.019	0.005
VA2 - VA6	0.0142	0.056
VA3 - VA6	-0.0048	0.083
VA9 - VA6	-.0021	0.09
VA12 – VA9	-.0034	0.13

Table 5: Central macular thickness at presentation and monthly intervals

	Mean	SD	Mean change from Baseline	% Change from Baseline	p value
CMT0	534.22	85.622			
CMT1	421.3	81.407	-112.9	-21.1%	<0.001
CMT2	338.58	70.13	-195.64	-36.6%	<0.001
CMT3	307.3	58.382	-226.92	-42.5%	<0.001
CMT6	297.04	68.42	-237.18	-44.4%	<0.001
CMT9	300.04	68.40	-234.18	-43.83%	<0.001
CMT12	304.09	67.82	-230.13	-43.07%	<0.001

Table 6: Comparison of Snellen's visual acuity in BRVO, I-CRVO and N-CRVO

	BRVO		I-CRVO		N-CRVO		
	Mean	SD	Mean	SD	Mean	SD	p value
VA0	0.2056	0.0513	0.0l475	0.00957	0.1542	0.05583	
VA1	0.2879	0.06089	0.06	0.01414	0.23	0.0603	<0.001
VA2	0.3729	0.08625	0.07	0.02	0.2767	0.05211	<0.001
VA3	0.3853	0.08802	0.08	0.01633	0.3175	0.06917	<0.001
VA6	0.3829	0.09057	0.08	0.01633	0.3042	0.08096	<0.001
VA9	0.3799	0.09057	0.079	0.01633	0.3033	0.08085	<0.001
VA12	0.3789	0.09057	0.072	0.01633	0.3022	0.08076	<0.001

Table 7: Time of presentation

Time of Presentation	BRVO	N-CRVO	I-CRVO	Percentage
< 1 week	1(2.94%)	3(25%)	2(50%)	12%
1 week to 2 week	3(8.82%)	2(16.67%)	2(50%)	14%
2 week to 3 week	4(11.76%)	1(8.33%)	-	10%
3 week to 4 week	10(29.41%)	6(50%)		32%
>4 week	16(47.06%)	-		32%

occlusion is second only to diabetic retinopathy.[7] The most common attributable risk factors are Age, Hypertension, Diabetes mellitus, Dyslipidemia and Glaucoma.[8] Macular edema secondary to these conditions is the most common cause of the diminution of vision.[9] The elevated intraocular levels of vascular endothelial growth factors can be attributed to vascular leakage resulting in macular edema.[4] Thus, there is a strong rationale for using VEGF antagonists in eyes with macular edema following retinal venous occlusion.

The baseline BCVA findings of the Sapphire and the Topaz study was 0.2, ranging from 0.2 to 0.05 (approximately 6/36)[10] and the central macular thickness ranging from 234 to 1676 (mean 661.5) micron in Sapphire study and 220 to 1527 (mean 642) micron in Topaz study. The findings of these studies are comparable to our findings.

In the study by Florian Rensch et al,[11] they found the baseline BCVA to be 0.15±0.13 Snellen acuity and baseline CMT to be 530 ± 152 microns, this is similar to the findings in the non-ischaemic CRVO cases of our study.

In another study by Leangelo Hall et al,[12] the baseline BCVA was Log MAR 1.3 and CMT as 858 ± 311 microns comparable to the findings in ischaemic CRVO in our study.

In the study "Treatment of Branch Retinal Vein Occlusion induced Macular Edema with Bevacizumab"[13] Mathias Abegg et al. also found that Visual acuity was significantly better 4 to 6 weeks after Bevacizumab treatment compared to visual acuity prior to treatment (0.68 ± 0.3 and 0.5 ± 0.35 log MAR, before and after injection respectively, mean ± standard deviation; $p < 0.01$, paired t-test). The gain in visual acuity was accompanied by a significant decrease in retinal thickness (454± 117 to 305 ± 129 μm, $p < 0.01$, paired t-test). Follow-up shows that improvement in both visual acuity and retinal thickness lasts for several months after Bevacizumab use. The result of this study is comparable to our study as we found similar improvements in BCVA and CMT after intervention at the end of 2 months and six months.

In the study of "Bevacizumab for macular edema in central retinal vein occlusion: a prospective, randomized, double-masked clinical study"[14] by David L J Epstein et al they found that 60.0% in the study group had gained ≥15 letters compared with 20.0% in the control group (P=0.003). The BCVA improved by 14.1 letters at 24 weeks compared with a decrease of 2.0 letters in the control group ($P < 0.003$). The mean decrease in central retinal thickness (CRT) was significantly greater in the study group (426 μm) than in the control group (102 μm) at all time points up to week 24 ($P < 0.001$). Though we did not have a control group, the findings of this study with respect to study group are similar and supportive of the findings of our study.

In the study "Two-year outcomes of intravitreal bevacizumab therapy for macular oedema secondary to branch retinal vein occlusion"[14] by Taiichi Hikichi et al. The baseline logarithm of the minimum angle of resolution visual acuity (VA) was 0.64±0.24 (mean±SD), which significantly (p=0.001) improved 1 month after the first injection to 0.39±0.22. One year after the first injection, VA improved significantly (p=0.001) to 0.33±0.21 and remained 0.34±0.21 until 2 years after the first injection (p=0.001). The changes in foveal thickness were correlated with those of VA during the 2-year follow-up period with a mean of 3.8±1.5 injections (including the first injection). In this study, the result of mean Snellen's visual acuity is comparable to the findings of our study.

In the study "Early intravitreal bevacizumab for non-ischaemic central retinal vein occlusion"[11] by Florian Rensch et al, it was found that at baseline, mean visual acuity measured 0.15±0.13 Snellen acuity, and mean central retinal thickness was 531±152 μm. At all examinations during follow-up visual acuity was significantly higher than at baseline, with an improvement to 0.70±0.42 log MAR one month after the first injection; to 0.69±0.46, three months after the first injection; and to 0.69±0.52, six months after the first injection. The improvements with respect to Snellen's visual acuity and CMT in the non- ischemic CRVO cases in our study are similar to the findings of Florian Rensch et al.[15]

In the study by Siegfried G. Priglinger et al. "Intravitreal bevacizumab injections for treatment of central retinal vein occlusion"[16] the findings of Non-ischemic Central Retinal Vein occlusion at baseline, 2 weeks, 6 weeks, 3 months, 6 months of BCVA are 0.91 ± 0.34, 0.66 ± 0.34, 0.62 ± 0.36, 0.58 ± 0.35, 0.51 ± 0.36 Log MAR respectively and the CMT are 535 ± 124, 359 ± 103, 371 ± 110, 359 ± 106, 350 ± 104 microns respectively. The findings of ischemic Central Retinal Vein Occlusion at baseline, 2 weeks, 6 weeks, 3 months, 6 months of BCVA are 1.52±0.34, 1.19±0.46, 1.15± 0.39, 1.26 ±0.46, 1.06± 0.45 Log MAR respectively and the CMT are 534 ±193, 297± 153, 295±124, 301± 122, 279± 127 microns respectively. In our study the improvements in mean snellens visual acuity and CMT in the CRVO (non-ischemic and ischemic CRVO) cases is comparable to the study results of Siegfried G. Priglinger et al.[17]

Two patients in our study did not maintain the mean central macular thickness at the end of six months and were thus given the fourth dose of Anti – VEGF at six months. Both the patients had initial CMT of more than 570 microns.

The findings of the study "Risk factors of recurrence of macular oedema associated with branch retinal vein occlusion after intravitreal bevacizumab injection" by Jun Ho Yoo et al[18] is supportive of the result of our study in relation to recurrence of macular edema.

In the current study, the cases with visual acuity of 0.1 and less in Snellen's decimal acuity (equivalent to 6/60 and less) showed improvement of mean Snellen's visual acuity only upto 0.19545 (approximately 6/36) as compared

to 0.3806 (approximately between 6/18 and 6/12) in cases with initial visual acuity of more than 0.1 Snellen's decimal acuity (equivalent to 6/60).

The finding of the study "Results of bevacizumab as the primary treatment for retinal vein occlusions" by M S Figueroa et al[19] is comparable with the result of our study as they also concluded that patients with poor initial visual acuity at presentation had poor final visual acuity in spite of the reduction of macular oedema.

5. Conclusion

The most recent treatment modality of macular edema due to retinal vaso-occlusive disease is the intravitreal injection of Anti-Vascular Endothelial Growth Factors.

In our study "Anti-vascular endothelial growth factor (Bevacizumab) in macular edema due to retinal vascular occlusive disease" the following conclusions can be drawn:

1. It reduced the Central Macular Thickness significantly and maintained it through twelve months and beyond.
2. The early use of intravitreal injection of Anti-Vascular Endothelial Growth Factors (Bevacizumab) is useful in improving the Snellen's visual Acuity to a statistically significant level.
3. Three doses of intravitreal injection of Anti Vascular Endothelial Growth Factors (Bevacizumab) was sufficient enough to bring about the significant statistical difference in Central Macular Thickness.
4. In our study it was also found that most of the patients could maintain the visual acuity and the Central Macular Thickness at the end of 12 months and beyond.
5. The study also establishes that Hypertension, Diabetes mellitus, Dyslipidemia and smoking are associated with retinal venous occlusive diseases.
6. Our study also reflects that there was a delay in presentation in the cases of branch retinal vein occlusion than the cases of central retinal vein occlusion.
7. The improvements in the macular thickness and visual acuity in the cases of branch retinal vein occlusion was better than the cases central retinal vein occlusion.
8. The Study population was small in our study with a follow up period was only twelve months and hence sweeping conclusion could not be derived on the disease profile in the whole population. Furthermore, poor metabolic control due to lack of awareness in the study population did not reflect the sole effect of Anti-VEGF on the study outcomes.

However, sweeping generalizations cannot be made about the intervention and doses as the number of patients studied were small.

6. Source of Funding

None.

7. Conflict of Interest

The authors have no conflicts of interest to declare.

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