

Clinical Outcomes of Ranibizumab, Aflibercept, and Bevacizumab in the Treatment of Macular Edema Secondary To Branch Retinal Vein Occlusion

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ABSTRACT

Purpose: To evaluate and compare the efficacy of three intravitreal anti-VEGF agents (Bevacizumab, Aflibercept, and Ranibizumab) in the treatment of macular edema (ME) secondary to branch retinal vein occlusion (BRVO).

Materials and Methods: A retrospective study was conducted on treatment-naïve patients diagnosed with BRVO-related ME between 2022 and 2024. All patients received three consecutive monthly intravitreal injections of either Bevacizumab (IVB, n=29), Aflibercept (IVA, n=19), or Ranibizumab (IVR, n=30). Best-corrected visual acuity (BCVA) and central macular thickness (CMT) were evaluated at baseline and after the third injection.

Results: Mean baseline BCVA (logMAR) was similar across groups (IVB: 0.87 ± 0.46 , IVA: 0.91 ± 0.70 , IVR: 0.96 ± 0.41 ; $p=0.23$), as were baseline CMT values (IVB: 616.9 ± 569.0 μm , IVA: 609.3 ± 560.5 μm , IVR: 537.3 ± 504.0 μm ; $p=0.258$). All three groups showed significant improvement in BCVA and CMT following treatment. Final BCVA improved to 0.57 ± 0.37 in IVB, 0.45 ± 0.29 in IVA, and 0.43 ± 0.29 in IVR. Final CMT reduced to 393.2 ± 343.0 μm (IVB), 308.0 ± 295.0 μm (IVA), and 320.5 ± 280.0 μm (IVR), all with $p < 0.0001$. No statistically significant differences were observed between groups in terms of BCVA or CMT changes ($p=0.30$ and $p=0.36$, respectively).

Conclusion: All three anti-VEGF agents were similarly effective in improving visual acuity and reducing macular thickness after three monthly injections. Treatment selection can be guided by availability, cost, and clinical context, as no significant efficacy differences were found in the short term.

Keywords: Anti-VEGF, Intravitreal injection, Macular edema, Retinal vein occlusion

INTRODUCTION

Retinal vein occlusion (RVO) is the second most common retinal vascular disorder after diabetic retinopathy and is a significant cause of visual impairment. The incidence of RVO ranges from 0.5% to 1.8% in the general population, with branch retinal vein occlusion (BRVO) occurring more frequently than central retinal vein occlusion (CRVO).¹ The pathogenesis of retinal vein occlusion macular edema (RVO-ME) is complex and multifactorial, involving fac-

tors such as vascular occlusion, retinal ischemia, and local hypoxia.² Retinal ischemia caused by venous obstruction and circulatory stasis triggers the release of VEGF-A, leading to increased vascular permeability, subsequent macular edema, and retinal neovascularization.³

Over the years, various therapeutic options have been developed to treat RVO-ME, including surgical interventions, laser therapy, corticosteroid injections, and, more recently,

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Received: 19.08.2025

Accepted: 19.10.2025

J Ret-Vit 2025; 34: 303-309

DOI:10.37845/ret.vit.2025.34.42

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intravitreal anti-VEGF agents. Laser photocoagulation, once the primary treatment for RVO, has shown limited efficacy in improving visual outcomes in several studies, prompting a shift toward anti-VEGF therapies.⁴ Intravitreal anti-VEGF therapy has become the standard treatment for ME secondary to RVO, and the most commonly used anti-VEGF agents are ranibizumab, a humanized antibody fragment that targets all VEGF-A isoforms; bevacizumab, a full-length humanized antibody; and aflibercept, a fusion protein consisting of VEGF receptors 1 and 2 linked to a monoclonal antibody backbone.⁵⁻⁷

All three anti-VEGF agents have been shown to significantly improve visual acuity and reduce ME in patients with BRVO and CRVO when compared to sham injections or laser photocoagulation. Multicenter, randomized controlled trials, such as BRAVO and VIBRANT, have demonstrated the efficacy and safety of intravitreal injections of ranibizumab (0.5 mg) and aflibercept (2.0 mg) in patients with ME associated with BRVO.^{8,9} Studies in the literature have shown that bevacizumab may be frequently used as an off-label alternative to the more expensive FDA-approved anti-VEGF agents ranibizumab and aflibercept for the treatment of ME secondary to RVO.¹⁰⁻¹²

While the results from clinical trials have established the efficacy of anti-VEGF agents for treating RVO-ME, data on the outcomes in real-world clinical settings remain limited. Real-world patient populations are often more heterogeneous than those in clinical trials, and treatment practices can vary significantly based on local regulations, access to medications, and clinical experience.¹³

In our study, we aimed to evaluate the functional and anatomical clinical outcomes of bevacizumab, ranibizumab, and aflibercept in macular edema secondary to retinal vein occlusion.

MATERIALS- METHODS

Medical records of treatment-naïve patients with BRVO who were examined in the retina department between 2022 and 2024 were retrospectively reviewed. The study adhered to the principles of the Declaration of Helsinki, and informed consent was obtained from all participants. Approval for the study was granted by the local ethics committee.

Only treatment-naïve patients diagnosed with macular edema secondary to BRVO who had not received any prior intravitreal injections were included in the study. Each patient was initially treated with a single anti-VEGF agent (intravitreal Bevacizumab (IVB), Ranibizumab (IVR), or Aflibercept (IVA)) and no agent switches were made during the follow-up period. According to the treatment protocol, patients received three consecutive monthly injections of the same anti-VEGF drug. Group assignments were made retrospectively based on the anti-VEGF agent each patient received. Exclusion criteria included prior ocular surgery, laser photocoagulation and/or intravitreal injection, other retinal pathologies, except RVO, previous history of autoimmune disorders, liver and kidney dysfunction and current use of systemic steroids or immunomodulatory medications.

All patients received a comprehensive ophthalmic evaluation, which included best corrected visual acuity (BCVA) assessment using the Snellen chart (with conversion to logMAR), anterior segment examination via slit-lamp biomicroscopy, intraocular pressure measurement with a non-contact tonometer (Topcon CT-80, Topcon Medical Systems, Paramus, New Jersey, USA), and dilated fundus examination employing a 90-diopter lens. Additionally, fundus color photography, fundus fluorescein angiography (FFA) and spectral-domain optical coherence tomography (OCT) (Cirrus, Carl Zeiss Meditec Inc, Dublin, CA) were performed.

The diagnosis of BRVO and ME was confirmed through fundus examination, OCT, and fundus fluorescein angiography (FFA). The diagnosis of ischemic BRVO was made based on standard FFA findings. Ischemia was defined as the presence of capillary non-perfusion areas greater than 5 disc diameters on FA. Central macular Thickness (CMT) was measured by OCT device using the 512 × 128 macular cube protocol, both before and after intravitreal treatment.

Intravitreal anti-VEGF injections were administered to treatment-naïve patients with ME who had a BCVA below 0.5 and a CMT greater than 250 microns. Patients received three consecutive doses of intravitreal anti-VEGF injections and were divided into three groups according to the anti-VEGF agent administered. Group 1 consisted of patients treated with IVB (1.25 mg/0.05 mL, Altuzan 100 mg/4 mL, Genentech, Roche, Switzerland), Group 2 with

IVA (2 mg/0.05 mL, Eylea, Bayer Hispania, S.L., Barcelona, Spain), and Group 3 with IVR (0.5 mg/0.05 mL, Lucentis; Novartis Pharma AG, Basel, Switzerland). Intravitreal injections were performed in the operating room under sterile conditions with a 30-gauge needle under topical anesthesia. Following the injections, patients were treated with topical moxifloxacin for one week.

BCVA and CMT were evaluated before the first injection and one month after the third injection and the results were compared between the groups.

Statistical analysis was performed using SPSS version 21.0 (SPSS Inc., Chicago, IL, USA). Differences in categorical variables between groups were assessed using the chi-square test. Given the small sample size (<30 patients) and the non-normal distribution of the data, nonparametric tests were used. The Wilcoxon signed-rank test was used to compare pre- and post-treatment mean values within groups, while the Kruskal Wallis test was applied to compare variables between different groups. A p-value of <0.05 was considered statistically significant.

RESULTS

A total of 78 treatment-naïve patients with BRVO-related ME were included in the study, with 29 receiving IVB (Group 1), 19 receiving IVA (Group 2), and 30 receiving IVR (Group 3). The mean age was 62.3 ± 11.4 years in Group 1, 61.1 ± 7.1 years in Group 2, and 61.0 ± 9.4 years in Group 3, with no statistically significant difference among the groups ($p = 0.909$).

Gender distribution was also similar across the groups ($p = 0.452$).

Ischemia detected on fluorescein angiography was observed in 7 patients (24.1%) in Group 1, 11 patients (57.8%) in Group 2, and 10 patients (33.3%) in Group 3 ($p = 0.06$).

The prevalence of diabetes mellitus was 8 patients (27.6%) in the IVB group, 4 patients (21.1%) in the IVA group, and 6 patients (20.0%) in the IVR group. No statistically significant difference was observed between the groups ($p=0.765$).

The prevalence of hypertension was 19 patients (65.5%) in the IVB group, 10 patients (52.6%) in the IVA group, and

20 patients (66.7%) in the IVR group. This distribution also showed no statistically significant difference ($p=0.570$).

To evaluate macular edema profiles, OCT images were retrospectively reviewed. The presence of subretinal fluid (SRF) was detected in 11 patients (37.9%) in the IVB group, 7 patients (36.8%) in the IVA group, and 11 patients (36.7%) in the IVR group, with no significant difference between groups ($p=0.994$). Cystoid macular edema (CME) was observed in 15 (51.7%), 10 (52.6%), and 16 (53.3%) patients, while diffuse macular edema (DME) was found in 3 (10.3%), 2 (10.5%), and 3 (10.0%) patients in the IVB, IVA, and IVR groups, respectively. No statistically significant differences were found between the groups for either edema type ($p_1=0.768$, $p_2=0.978$).

Following three consecutive doses of anti-VEGF therapy, mean logMAR BCVA significantly improved in all groups: from 0.87 ± 0.46 to 0.57 ± 0.37 in the IVB group ($p = 0.002$), from 0.91 ± 0.74 to 0.45 ± 0.29 in the IVA group ($p = 0.002$), and from 0.96 ± 0.41 to 0.43 ± 0.29 in the IVR group ($p < 0.001$) (Figure 1). However, there was no statistically significant difference in visual improvement between the groups ($p = 0.300$).

CMT significantly decreased after treatment in all groups: from 616.93 ± 206.83 μm to 393.21 ± 139.03 μm in the IVB group, from 609.33 ± 233.11 μm to 308.0 ± 93.17 μm in the IVA group, and from 537.38 ± 135.25 μm to 320.48 ± 280.0 μm in the IVR group ($p < 0.0001$ for all) (Figure 2). However, the difference in the amount of CMT reduction among the three groups was not statistically significant ($p = 0.360$). No complications related to intravitreal injections were observed in any of the groups.

DISCUSSION

In this study, we assessed the real-world anatomical and functional outcomes of intravitreal ranibizumab, bevacizumab, and aflibercept in patients with ME secondary to BRVO. Our findings showed that there were no statistically significant differences in visual or anatomical improvements among the treatment groups, and no injection-related adverse events were observed. These results support the real-world safety and efficacy of all three agents in the short-term management of BRVO-associated macular edema.

The role of VEGF in the pathogenesis of RVO-related macular edema is well established. VEGF promotes increased vascular permeability and intraretinal fluid accumulation, leading to edema and vision loss.¹ Anti-VEGF agents directly target this process and have replaced older treatment modalities such as grid laser photocoagulation, intravitreal corticosteroids, and observation as the mainstay of therapy.

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The efficacy and safety of anti-VEGF agents have been demonstrated in randomized controlled trials (RCTs).¹⁹⁻²¹ While ranibizumab and aflibercept are FDA-approved for the treatment of various retinal diseases with associated costs, bevacizumab is frequently used off-label as a cost-effective alternative. Limited evidence comparing the effectiveness of available anti-VEGF agents influences treatment decisions and reimbursement policies for patients with RVO.

Our results are consistent with previous clinical trials such as the BRAVO and VIBRANT studies, which demonstrated significant anatomical and functional improvements with ranibizumab and aflibercept in patients with BRVO-related macular edema.^{8,9} In the BRAVO study, patients with BRVO who received monthly ranibizumab for six months followed by as-needed dosing showed promising results: 64.9% achieved a BCVA of 20/40 or better at six months, and 86.3% reached normal central macular thickness (CMT) by 12 months.⁸ Similarly, in the CRUISE study, which focused on CRVO, 46.9% of patients reached 20/40 or better BCVA at six months, and 77.7% had normalized CMT at one year.¹⁰

The VIBRANT study evaluated aflibercept in BRVO and found that patients gained an average of 17 letters in visual acuity and had a 281 μ m reduction in CMT, with these benefits maintained through week 52.⁹ For CRVO, the COPERNICUS and GALILEO trials demonstrated that aflibercept treatment led to improvements of 16.2 to 18.0 letters in BCVA and reductions in CMT ranging from 413 to 457 μ m. All of these studies also reported favorable safety profiles for the anti-VEGF agents used.^{11,12}

In a meta-analysis involving 18 studies, bevacizumab, ranibizumab, and aflibercept were found to be significantly superior to sham injections in terms of BCVA improvement and CMT reduction, with no statistically significant differ-

ence observed among the anti-VEGF agents.¹³ Similarly, a retrospective study of real-world data examined 52 patients with untreated macular edema due to BRVO. Twenty-seven patients were treated with intravitreal bevacizumab and 25 with intravitreal aflibercept on an as-needed (PRN) basis, with monthly follow-up for 12 months. Both bevacizumab and aflibercept were found to be similarly effective in reducing central macular thickness and improving visual acuity.²² In another study involving RVO patients treated with either ranibizumab or aflibercept and followed for at least 6 months, the improvement in visual acuity and the reduction in CMT.²³

The LEAVO study, a three-arm, double-blind, randomized trial, compared the clinical and cost-effectiveness of ranibizumab, aflibercept, and bevacizumab in the treatment of macular edema due to CRVO. A total of 463 patients were followed for 100 weeks. The mean visual gain at week 100 was +15.1 letters in the aflibercept group, +12.5 letters in the ranibizumab group, and +9.8 letters in the bevacizumab group. Aflibercept was found to be non-inferior to ranibizumab, but bevacizumab was not shown to be non-inferior to either drug. Bevacizumab stood out as the most economical option due to its lower cost; however, it should be noted that its efficacy may be somewhat lower than that of other agents.²⁴

Treatment outcomes may be influenced by individual patient characteristics, including age, systemic comorbidities, genetic predispositions, and the baseline severity of the disease. In our cohort, baseline BCVA and CMT values were statistically similar among the groups, which strengthens the validity of our outcome comparisons. This suggests that the observed treatment effects were not confounded by baseline disease severity. Nonetheless, it should be acknowledged that additional factors such as ischemic status may influence treatment response. The proportion of patients with ischemic BRVO in our study was not significantly different between groups. While other studies have reported slight differences in injection frequency or response durability among the three agents, especially in longer follow-up periods, these differences were not evident within the 3-month observation window of our study.

In many real-world settings, especially those with limited healthcare resources, treatment choice may be influenced more by economic and logistical factors than by efficacy

concerns. Our findings reinforce the notion that bevacizumab is a viable and effective option when access to on-label agents is limited, provided that appropriate quality control measures are in place for compounding and storage.

In this study, the similar visual and anatomical improvements observed across the three treatment groups can be attributed to the shared mechanism of action of the anti-VEGF agents in reducing vascular permeability and controlling edema. Additionally, the comparable baseline visual acuity and central macular thickness among groups provided a balanced foundation for outcome comparisons, minimizing confounding effects due to disease severity. The lack of significant differences may also be influenced by the retrospective design and similar patient selection criteria. These findings emphasize that while clinical efficacy appears comparable in the short term, treatment decisions should also consider factors such as cost, availability, and patient-specific circumstances.

This study had several limitations. First, the retrospective design and relatively small sample size may limit the generalizability of the findings. Second, the follow-up period

was limited to three months, which precludes conclusions regarding long-term efficacy, need for retreatment, or development of treatment resistance. Third, the lack of randomization introduces potential selection bias. Fourth, the presence of macular ischemia was not statistically analyzed between groups due to insufficient image quality in some patients, limiting the generalizability of these data. Lastly, we did not incorporate additional structural and functional markers such as OCT angiography, which could have provided a more comprehensive assessment of retinal function.

In conclusion, this study demonstrated that intravitreal bevacizumab, ranibizumab, and aflibercept provided similar short-term anatomical and functional benefits in patients with BRVO-associated ME. The absence of statistically significant differences among the agents suggests that treatment choice may be based on cost, availability, and clinician preference. However, future prospective randomized trials with longer follow-up and larger sample sizes are warranted to evaluate long-term efficacy, durability, and possible differences in retreatment needs or safety profiles.

Table 1: Demographic data of patients

	Group 1 (IVB) n (%)	Group 2 (IVA) n (%)	Group 3 (IVR) n (%)	p
n	29	19	30	
Gender				
Male	19 (65.5%)	10 (52.6%)	15 (50%)	0.452
Female	10 (34.5%)	9 (47.4%)	15 (50%)	
Age (years)	62.34±11.42	61.11±7.14	61.00±9.42	0.909
Lateralite				
Right	18 (62.1%)	10 (52.6%)	18 (60%)	0.801
Left	11 (37.9%)	9 (47.4%)	12 (40%)	
Type				
Superior temporal BRVO	18 (62.1%)	11 (57.9%)	17 (56.7%)	0.902
Inferior temporal BRVO	11 (37.9%)	8 (42.1%)	13 (43.3%)	
Ischaemic type	7 (24.1%)	11 (57.8%)	10 (33.3%)	0.064
Diabetes mellitus	8 (27.6%)	4 (21.1%)	6 (20%)	0.765
Hypertension	19 (65.5%)	10 (52.6%)	20 (66.7%)	0.570
IVB: Intravitreal bevacizumab, IVA: Intravitreal aflibercept, IVR: Intravitreal ranibizumab, BRVO: Branch retinal vein occlusion				

Table 2: BCVA and CMT changes of patients before injection and after 3 consecutive doses of anti-VEGF injection

	Group 1 (IVB) n=29	Group 2 (IVA) n=19	Group 3 (IVR) n=30	p ^a
Pre-treatment BCVA (logMAR)	0.87±0.46	0.91±0.74	0.96±0.41	0.438
Post-treatment BCVA (logMAR)	0.57±0.37	0.45±0.29	0.43±0.29	0.806
Difference	0.29±0.55	0.49±0.64	0.54±0.42	0.300
p ^b	0.002	0.002	0.0001	
Pre-treatment CMT (µm)	616.93±206.83	609.33±233.11	537.38±135.25	0.258
Post-treatment CMT (µm)	393.21±139.03	308.0±93.17	320.48±280.0	0.030
Difference	223.72±258.41	301.33±258.74	216.89±131.28	0.360
p ^b	0.0001	0.0001	0.0001	
Pre-treatment IOP (mmHg)	15.24±3.11	17.11±2.94	16.31±2.63	0.089
Post-treatment IOP (mmHg)	15.28±3.59	16.17±3.03	16.69±2.98	0.176
Difference	0.03±3.24	-1.00±2.90	0.43±3.37	0.116
p ^b	0.879	0.122	0.361	
IVB: Intravitreal bevacizumab, IVA: Intravitreal aflibercept, IVR: Intravitreal ranibizumab, BCVA: best-corrected visual acuity, CMT: Central macular thickness, IOP: Intraocular pressure p ^a : Kruskal Wallis test (Group1-2-3), p ^b : Wilcoxon test (pre-post treatment)				

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