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Intravitreal Ranibizumab for Macular Oedema secondary to BRVO

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KEYWORDS

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ABSTRACT:

Retinal vein occlusion is a common retinal vascular disorder and an important cause of unilateral, painless visual impairment, with branch retinal vein occlusion (BRVO) frequently complicated by cystoid macular oedema (CMO), the principal driver of reduced central vision. We report a case series from Aarupadai Veedu Medical College (AVMC) describing functional and OCT-guided anatomical outcomes following intravitreal ranibizumab (0.5 mg/0.05 mL) for BRVO-associated macular oedema. Case 1 involved a 50-year-old hypertensive woman with superotemporal BRVO and centre-involving CMO (central macular thickness [CMT] 477 µm) with baseline BCVA 6/18 in the affected eye; after two injections, BCVA improved to 6/9 with CMT reducing to 320 µm after the first dose. Case 2 involved a 32-year-old man without known systemic risk factors who presented late (7 months) with signs of old inferotemporal BRVO and poor baseline BCVA (4/60); following two injections, BCVA improved sequentially to 6/24 and then 6/12. Case 3 involved a 61-year-old woman with poorly controlled hypertension and inferior quadrant BRVO with marked CMO (CMT 584 µm) and severe baseline visual loss (BCVA 1/60); BCVA improved to 6/36 after the first injection and to 6/12 after the second dose administered four weeks later. Overall, intravitreal ranibizumab was associated with rapid symptomatic relief and meaningful visual recovery across varying ages and disease chronicity, supported by OCT-documented reduction in macular oedema in acute presentations.

Introduction

Retinal vein occlusion (RVO) is a common retinal vascular disorder and an important cause of unilateral, painless visual impairment, second only to diabetic retinopathy among retinal vascular diseases.(1) Branch retinal vein occlusion (BRVO) was first described ophthalmoscopically by Theodor von Leber in 1877, and it remains frequently encountered in day-to-day ophthalmic practice. BRVO most often involves the superotemporal quadrant (attributed to a higher density of arteriovenous crossings in that region), followed by the inferotemporal quadrant, with nasal quadrant involvement being relatively uncommon. Clinically, BRVO typically presents with sectoral retinal haemorrhages, venous dilatation and tortuosity, cotton-wool spots, and retinal edema within the distribution of the affected venous branch, and visual loss is largely determined by the presence and severity of macular involvement.(2, 3) The widely accepted pathophysiology involves compression of the retinal vein at arteriovenous crossings by an arteriosclerotic arteriole, leading to venous stasis, thrombosis, and downstream retinal hypoxia; this stimulates vascular endothelial growth factor (VEGF)–driven vascular leakage and cystoid

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macular oedema (CMO), the principal cause of reduced central vision in BRVO.(1) Systemic vascular risk factors—especially hypertension and increasing age—are consistently associated with BRVO, underscoring the importance of systemic evaluation and risk-factor optimisation alongside ocular therapy.(4)

Over the past three decades, the diagnostic and therapeutic landscape of BRVO has evolved substantially. Optical coherence tomography (OCT) has become central for confirming macular oedema, quantifying central macular thickness, and objectively monitoring treatment response, thereby enabling timely retreatment decisions in routine practice.(5) In parallel, intravitreal anti-VEGF agents have become the recommended first-line treatment for BRVO-related macular oedema, with pivotal randomized trials (e.g., BRAVO) demonstrating rapid anatomical improvement and clinically meaningful gains in visual acuity with ranibizumab (0.3–0.5 mg) and a favourable safety profile.(6) Newer intravitreal agents and strategies aimed at improving durability and reducing treatment burden (including dual-pathway biologics such as faricimab) are also being evaluated in RVO.(7, 8) Additionally, longer-acting delivery platforms and gene-therapy approaches targeting VEGF pathways are being explored to potentially provide sustained intraocular VEGF suppression and reduce injection frequency.(9) In this context, we report three BRVO cases with macular oedema managed with intravitreal ranibizumab at Aarupadai Veedu Medical College (AVMC), highlighting OCT-guided anatomical outcomes and functional recovery across differing ages and disease chronicity.

Case Series

Case 1: A 50-year-old woman presented to the Ophthalmology OPD with sudden-onset, painless diminution of vision in the right eye for 15 days that was progressive in nature; she was a known hypertensive for 7 years and was compliant with antihypertensive therapy, which is relevant because advancing age and systemic vascular risk factors—particularly hypertension—are well-recognized associations for retinal vein occlusion. On ocular evaluation, BCVA was 6/18 in the RE and 6/12 in the LE with a refractive correction of +0.75 DS/+0.50 DC in both eyes; the anterior segments were unremarkable. Dilated fundus examination by binocular indirect ophthalmoscopy was consistent with a superotemporal BRVO in the RE, a condition classically attributed to venous compression at arteriovenous crossings with subsequent venous stasis and thrombosis, leading to retinal ischemia and upregulation of vascular endothelial growth factor (VEGF), thereby increasing vascular permeability and precipitating cystoid macular oedema. OCT of the RE macula confirmed cystoid macular oedema with a CMT of 477 µm (Fig. 1a). In view of centre-involving macular oedema with visual impairment, intravitreal anti-VEGF therapy was planned, consistent with current guidance that anti-VEGF agents are first-line treatment for macular oedema secondary to retinal vein occlusion; accordingly, the patient received intravitreal ranibizumab 0.5 mg/0.05 mL, a commonly recommended dose administered approximately monthly for macular oedema due to RVO. At review 20 days after the first injection, she reported symptomatic improvement with BCVA improving to 6/12 in both eyes, and OCT demonstrated reduction of macular oedema with CMT decreasing to 320 µm in the RE. A second intravitreal ranibizumab injection was scheduled, and at follow-up 20 days later the patient noted marked improvement with BCVA of 6/9 in the RE and 6/12 in the LE.

Case 2: A 32-year-old man presented to the Ophthalmology OPD with a 7-month history of sudden-onset, painless, progressively worsening visual impairment in the RE, with no known diabetes mellitus, hypertension, preceding trauma, or prior ocular disease; such presentations are uncommon but recognised in younger adults, in whom the aetiology is often unclear and warrants targeted systemic assessment for vascular and hematologic risk factors. On examination, BCVA was 4/60 in the RE and 6/6 in the LE, the anterior segments were normal, and dilated fundus evaluation by binocular indirect ophthalmoscopy showed a sclerosed inferotemporal retinal venule in the RE, consistent with sequelae of an old BRVO. In line with recommended work-up for retinal vein occlusion, baseline evaluation was directed toward identifying modifiable systemic associations (including blood pressure and glucose assessment, and basic laboratory screening such as full blood count and ESR), with further investigations guided by history and initial results; routine thrombophilia testing is not universally recommended, although selected testing may be considered when clinical

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suspicion is high (e.g., atypical presentations or suggestive personal/family history). Given the marked reduction in visual acuity and the chronic post-occlusive appearance, macular involvement was considered likely, and OCT—recommended for diagnosing and monitoring macular oedema and response to therapy in RVO—was used to guide management. As intravitreal anti-VEGF therapy is the first-line treatment for centre-involving macular oedema secondary to BRVO, the patient was planned for intravitreal ranibizumab 0.5 mg/0.05 mL. Following the first injection, his RE visual acuity improved to 6/24 at follow-up, and a second dose was administered; at subsequent review, RE visual acuity further improved to 6/12.

Case 3: A 61-year-old woman presented to the Ophthalmology OPD with sudden-onset, painless diminution of vision in the RE for 4 days, with no antecedent trauma; she was a known hypertensive for 7 years and admitted to inconsistent adherence to antihypertensive medication. BCVA was 1/60 in the RE and 6/12 in the LE. Anterior segment evaluation was essentially normal except for early lenticular changes in both eyes. Dilated fundus examination by binocular indirect ophthalmoscopy revealed a branch retinal vein occlusion involving the inferior quadrant of the RE. The presentation was consistent with the established pathophysiologic concept that BRVO commonly occurs at arteriovenous crossings where a thickened arteriole can compress the adjacent venule, precipitating venous stasis and thrombosis; downstream retinal hypoxia drives VEGF upregulation, increasing vascular permeability and leading to CMO. OCT of the macula confirmed cystoid macular oedema in the RE with a markedly increased CMT of 584 µm. In view of centre-involving macular oedema with significant visual loss, intravitreal anti-VEGF therapy was planned. The patient received intravitreal ranibizumab 0.5 mg/0.05 mL (the standard adult dose, typically administered at approximately monthly intervals), alongside counselling for strict systemic blood pressure control and cardiovascular risk-factor optimisation. After the first injection, BCVA improved to 6/36 in the RE, and a second dose of intravitreal ranibizumab was administered 4 weeks later; following the second injection, the patient reported substantial symptomatic improvement with BCVA improving to 6/12 in the RE.

Discussion

Retinal vein occlusions are among the most common retinal vascular disorders and are an important cause of unilateral, sudden or subacute, painless visual loss, with macular oedema being the principal mechanism for reduced central vision in BRVO.(5) Across all three cases, the clinical picture—sectoral venous involvement with corresponding retinal signs and OCT-confirmed cystoid macular oedema—fits established descriptions of BRVO, where vision is primarily affected when the occlusion involves drainage of the macular region or triggers centre-involving oedema.(1) The prompt anatomical improvement noted on OCT after ranibizumab in the first and third cases (CMT reductions from 477→320 µm and 584 µm downward) mirrors the expected treatment-responsive component of VEGF-mediated vascular leakage that drives much of the oedema burden in RVO.(6, 10)

The pathogenesis of BRVO is classically linked to arteriovenous crossing changes; arteriosclerotic thickening of the retinal arteriole and a shared adventitial sheath can compress the adjacent vein, leading to disturbed flow, endothelial damage, and thrombus formation.(2, 11) Downstream venous congestion and capillary nonperfusion contribute to retinal hypoxia, which upregulates VEGF and other inflammatory mediators, increasing vascular permeability and producing cystoid macular oedema. Case 1 (superotemporal BRVO) and Case 3 (inferior quadrant BRVO) highlight the typical “sectoral” nature of BRVO, where the location of the occluded venous branch determines the distribution of haemorrhages, edema, and ischemia, while central vision depends largely on whether the macula is involved.(12) Systemic risk factors strongly shape both susceptibility and prognosis in BRVO, and hypertension remains one of the most consistently reported associations.(13) Epidemiologic data (including Beaver Dam Eye Study findings) emphasizes hypertension and related arteriolar changes (e.g., arteriovenous nicking, focal arteriolar narrowing) as key correlates of BRVO.(14, 15) This aligns closely with Case 1 and Case 3, where long-standing hypertension was present, and is particularly instructive in Case 3, where poor adherence plausibly contributed to ongoing vascular injury and arteriolosclerosis that predispose to occlusion at vulnerable crossings.(16)

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Case 2 illustrates a clinically important variant. BRVO in a younger adult without known diabetes or hypertension and with a chronic history (7 months) and fundus evidence of a sclerosed vessel suggestive of an old event.(17) While BRVO is less common in younger patients, literature recognizes that "atypical" presentations should prompt a targeted search for secondary contributors such as undiagnosed hypertension, metabolic risk, inflammatory disease, hyperhomocysteinemia, or other prothrombotic states—guided by history and baseline testing rather than indiscriminate panels.(18) Evidence also cautions against routine thrombophilia screening in all RVO patients, as systematic testing has not shown consistent clinical utility; instead, selective testing is generally reserved for those with strong suspicion (very young age, bilateral/recurrent events, personal/family thrombosis history, or absence of conventional risk factors after basic evaluation).(18, 19) This framework supports the approach described in Case 2—checking modifiable systemic associations first (BP, glucose, CBC/ESR) and escalating investigations only when indicated by clinical context.(1)

OCT is central to modern BRVO care because it objectively quantifies macular edema (e.g., CMT) and helps track treatment response over time.(5) The observed parallel between decreasing CMT and improving BCVA in Cases 1 and 3 is directionally consistent with studies showing that reductions in retinal thickness/macular volume often correlate with visual improvement, even though structure–function relationships can be imperfect due to ischemia, photoreceptor damage, and chronicity.(10, 20) In clinical practice, OCT findings are often complemented by fluorescein angiography or OCT angiography when ischemia is suspected, because the extent of capillary nonperfusion is a key determinant of complications such as retinal/iris neovascularization.(21) More severe retinal ischemia increases the likelihood of ocular neovascularization, reinforcing the need for follow-up tailored to ischemic risk rather than symptoms alone.(21)

Therapeutically, all three cases reflect contemporary first-line management. Intravitreal anti-VEGF agents are recommended as the initial treatment for macular edema secondary to RVO because VEGF is a major driver of vascular leakage in this condition.(5, 22) Ranibizumab has robust evidence in BRVO-related macular oedema, most notably from the BRAVO trial, which demonstrated rapid and meaningful improvements in visual acuity and macular thickness with monthly 0.3 mg or 0.5 mg ranibizumab injections, with a favourable safety profile.(6, 23) The early functional gains in these patients—Case 1 improving from 6/18 to 6/9 after two injections, and Case 3 improving from 1/60 to 6/12—are clinically plausible within the known response pattern of anti-VEGF therapy, where significant improvement can occur within weeks as edema resolves.(24) Timing and disease chronicity are critical when interpreting the differences across cases.(25) Case 3 received treatment within days of symptom onset and showed marked recovery after two injections, whereas Case 2 presented after 7 months with poor baseline acuity, suggesting chronic structural damage or persistent edema that can limit maximal recovery despite treatment. Literature indicate that delaying initiation of anti-VEGF therapy may reduce the magnitude of visual recovery in RVO, supporting early diagnosis and prompt treatment for centre-involving macular oedema.(25) Nevertheless, the meaningful improvement in Case 2 (4/60 to 6/12) underscores that even eyes with poor presenting vision can derive anatomical and functional benefit from anti-VEGF therapy, particularly if a reversible edema component remains.

Finally, these cases reinforce that BRVO management is not solely injection-based. It includes monitoring for recurrence of edema and for ischemic complications (retinal neovascularization, vitreous haemorrhage, neovascular glaucoma), while concurrently addressing systemic vascular health. Ranibizumab trials report low rates of serious ocular and systemic adverse events, but standard intravitreal injection risks (endophthalmitis, intraocular pressure rise, inflammation) necessitate counselling and post-injection surveillance.(6) In hypertensive patients (Cases 1 and 3), consistent blood pressure control is especially important because hypertension is a key risk factor for BRVO and may influence the broader vascular milieu in which retinal occlusion occurs.

Conclusion

Intravitreal ranibizumab (0.5 mg/0.05 mL) produced rapid and clinically meaningful improvement in visual acuity in all three patients with BRVO-associated macular oedema, with parallel anatomical recovery

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evidenced by reduction in OCT-measured central macular thickness in the acute presentations. These cases reinforce that macular oedema is the principal cause of visual impairment in BRVO and that timely initiation of anti-VEGF therapy can yield early functional gains, particularly when treatment is instituted soon after symptom onset. The series also highlights the importance of OCT-based monitoring to document response and guide repeat dosing, and it underscores the need for comprehensive systemic evaluation and optimisation of vascular risk factors—especially hypertension—to reduce ongoing microvascular injury and potentially mitigate recurrence or progression. Overall, ranibizumab was an effective and practical first-line option for BRVO-related macular oedema across differing ages and stages of presentation in routine clinical care.

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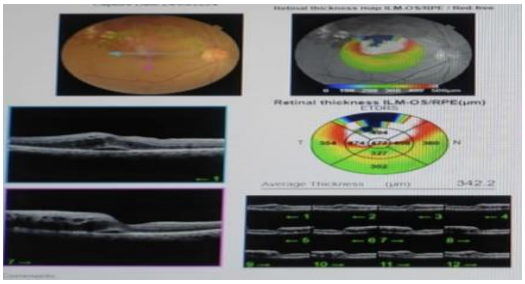
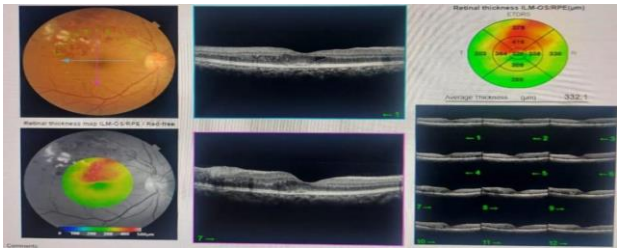
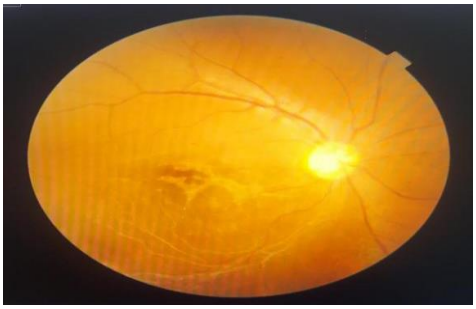
1a. RE BRVO with cystoid macular oedema



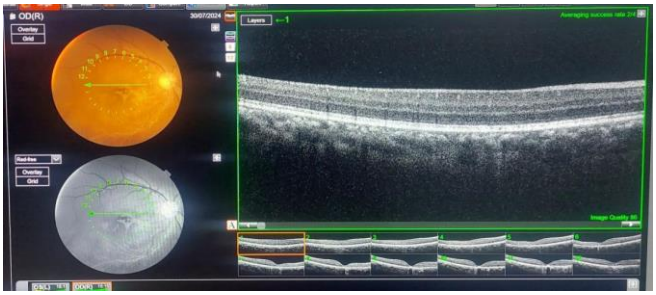
1b. Post Ranibizumab injection shows reduction in CMT and CMO



2a. RE fundus showing inferior quadrant sclerosed vessels



2b. RE fundus showing persistance of sclerosed vessel following intravitreal injection



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3a. RE cystoid macular oedema with central macular thickness of 584

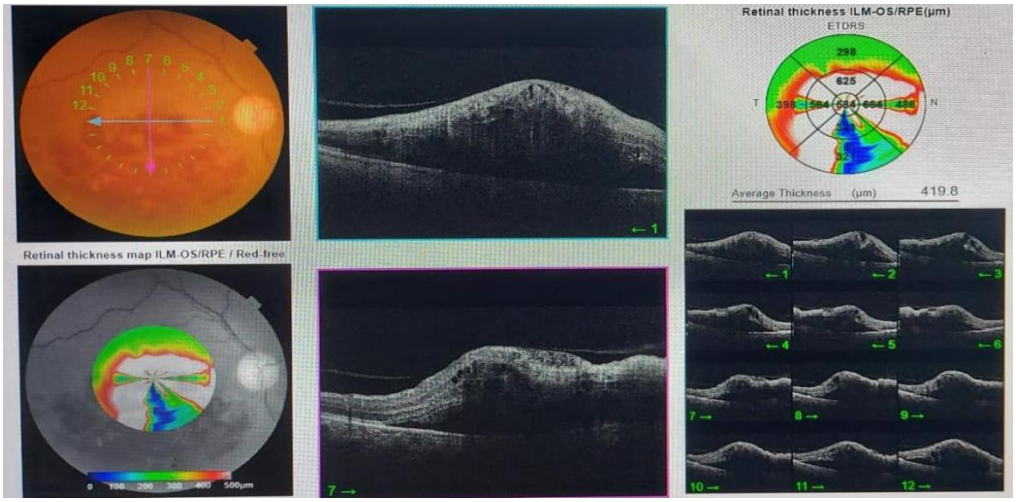

Figure 1: Clinical images