

Case Series

Optimizing Outcome with Intravitreal Faricimab in Case of Diabetic Macular Odema and Refractory Macular Brvo with CME

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Abstract

Objective: To evaluate the clinical efficacy, safety, novelty and patient-reported outcomes of intravitreal faricimab in Indian patients presenting with diabetic macular edema (DME) and refractory cystoid macular edema (CME) secondary to branch retinal vein occlusion (BRVO).

Methods: This case series documents two distinct adult patients with macular edema who received intravitreal faricimab injections. Key outcome measures evaluated over a 3-month follow-up period included best-corrected visual acuity (BCVA), central macular thickness, ocular and systemic adverse events, and subjective patient satisfaction measured on a standardized scale (ranging from "not satisfied" to "very satisfied").

Results: Both eyes demonstrated substantial anatomical resolution and functional visual improvement. Better visual recovery correlated with shorter diabetes duration and better baseline BCVA. No intraocular inflammation, intraocular pressure spikes, or other ocular complications were reported. Subjective patient satisfaction shifted from "moderate" at baseline to "very satisfied" post-treatment.

Conclusion: Intravitreal faricimab demonstrates promising therapeutic potential for managing DME and treatment-resistant CME secondary to BRVO in an Indian demographic. Dual-pathway targeting offers strong anatomical stabilization and visual gains, warranting broader clinical evaluation.

Keywords: Angiopoietin-2 (Ang-2), CrossMAb (Cross Over Monoclonal Antibody), DME-Diabetic Macular Edema, Macular BRVO (Macular Branch Retinal Vein Occlusion).

INTRODUCTION

Retinal vascular disorders—specifically diabetic macular edema (DME) and neovascular age-related macular degeneration (nAMD)—remain major causes of vision loss among working-age populations and older adults worldwide. A shared pathological hallmark of these conditions is the upregulation of vascular endothelial growth factor-A (VEGF-A), a powerful pro-angiogenic cytokine that triggers microvascular hyperpermeability, breakdown of the blood-retinal barrier, and abnormal angiogenesis. This results in pathological neovascularization, fluid accumulation, and progressive visual decline [1]. Anti-VEGF therapy has traditionally served as the mainstay of treatment, aiming to mitigate vessel leakage and suppress neovascular proliferation to stabilize visual function.

Despite the widespread adoption of anti-VEGF agents, the global human and economic burden of retinal diseases continues to surge. Diabetic retinopathy (DR) currently affects roughly 30% to 40% of individuals diagnosed with diabetes mellitus and is a leading contributor to preventable blindness [3]. Epidemiological projections estimate that the global prevalence of DR will escalate from 103 million individuals in 2020 to more than 161 million by 2045 [4].

Because many patients display sub-optimal responses or treatment fatigue under conventional anti-VEGF regimens, addressing complementary biological pathways—such as Angiopoietin-2 (Ang-2) inhibition to enhance vascular stability—has emerged as a priority. Faricimab, a novel bispecific antibody targeting both VEGF-A and Ang-2, represents an innovative approach to managing resistant macular edema and optimized long-term disease control.

Purpose: To report case series of 2 patients treated with INTRAVITREAL FARICIMAB.

CASE 1

A 64-year-old male presented in the outpatient department with a history of gradual painless diminution of vision in both eye for 2 years, which was gradual in onset, progressive in nature and not associated with pain. He is known case of hypertension since 6 months and known case of diabetes mellitus since 1.5 years. He had history of laser (panretinal photocoagulation) done elsewhere 6 months back.

On ocular examination of the right eye, the patient has a vision of counting finger 1 metre not improving with pinhole and 6/36 vision in left eye. Best corrected visual acuity was not improving in both eyes and on anterior segment evaluation revealed nuclear sclerosis grade 3

with posterior subcapsular cataract grade II in the right eye and nuclear sclerosis grade 1 in left eye.

On fundus examination of right eye revealed cup disc ratio of 0.3, multiple hard exudate in circinate pattern in macular area with few dot blot haemorrhages in all quadrant with laser marks.

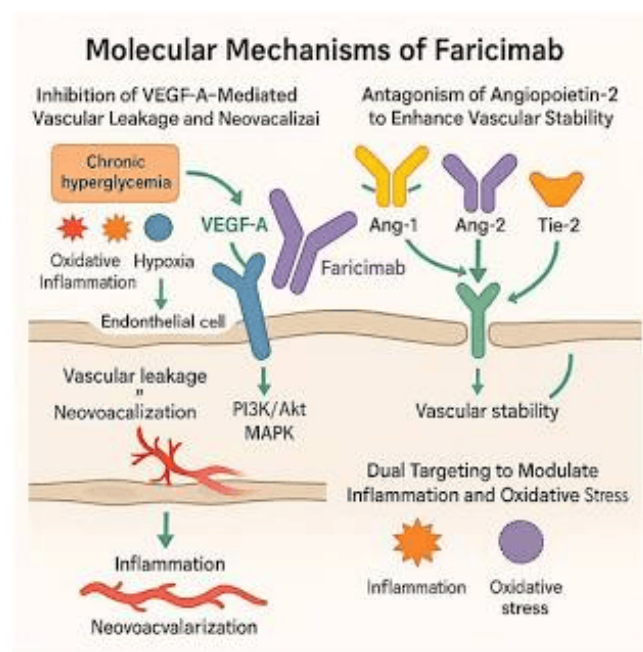


Figure 1. Molecular mechanisms of Faricimab

On fundus examination of left eye revealed, cup disc ratio of 0.3, multiple hard exudate in circinate pattern in macular area with few dot blot haemorrhages in all quadrant with laser marks suggestive of Both eye immature senile cataract with proliferative diabetic retinopathy S/P panretinal photocoagulation with diabetic macular edema.

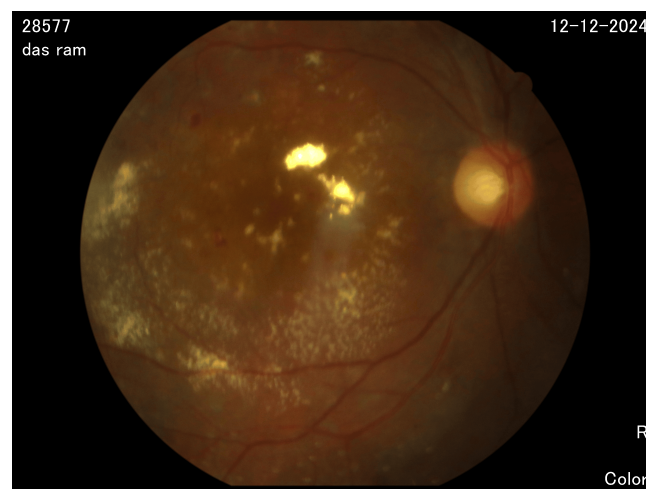


Figure 2. The fundus photo of the right eye was suggestive of proliferative diabetic retinopathy S/P panretinal photocoagulation with diabetic macular edema

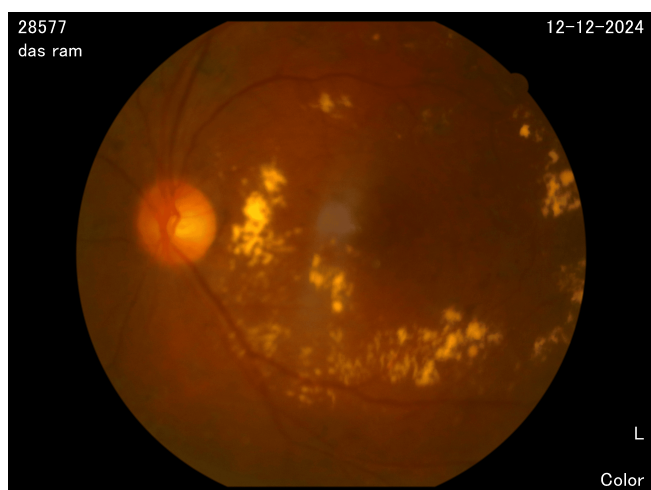


Figure 3. Fundus photo LE-shows multiple hard exudate in Circinate pattern, FR was dull, Macular edema present, suggestive of LE-Proliferative diabetic retinopathy S/P panretinal photocoagulation with diabetic macular edema

Faricimab-treated eye (Left eye)

On ocular examination, the patient had 6/36 vision in left eye. Best corrected visual acuity was not improving in both eyes. OPTICAL COHERENCE TOMOGRAPHY (OCT). OCT of left eye also showed cystoid macular edema with hard exudates in outer plexiform layer; CMT (central macular thickness) in the left eye was 572 μm .

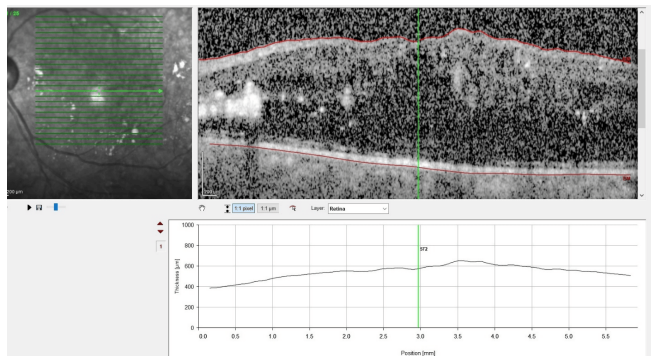


Figure 4. OCT examination left eye revealed cystoid and serous macular odema, CMT was 572 μm . This is preop picture.

Informed consent was obtained from the patient after discussing risk benefits. Patient was advised left eye intravitreal faricimab in dose of 6 mg in 0.05 mL, 8 days after intravitreal injection in right eye and was treated with the same. After 1 month 14 days of follow up, patient had 6/18 aided vision in left eye. On OCT examination left eye again revealed cystoid and serous macular edema. Patient was advised 2nd dose of left eye intravitreal faricimab in dose of 6 mg in 0.05 mL, the interval of second dose was 1 month 14 days. On follow-up after 1 month patient had 6/18 aided vision in the left eye, OCT examination revealed resolving macular edema in left eye. Patient was monitored for adverse event in

every follow up and no adverse events were observed. Patient satisfaction improved from moderate at early follow-ups to very satisfied by 3 months.

Visual outcome (Right eye)

On ocular examination of the right eye, the patient has a vision of counting fingers at 1 metre not improving with pinhole. Best corrected visual acuity was not improving in both eyes. OPTICAL COHERENCE TOMOGRAPHY (OCT) of the right eye showed cystoid macular edema with hard exudates in outer plexiform layer, I was 599 μm .

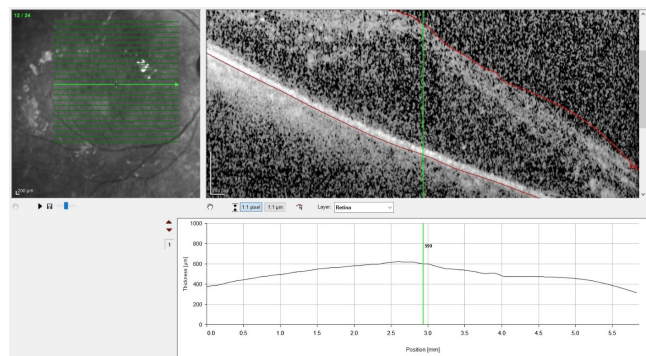


Figure 5. On OCT examination right eye revealed cystoid and serous macular odema, CMT was 599 μm . This is preop picture

Patient was advised Right eye intravitreal ranibizumab and was treated with the same. Vision in right eye improved to counting finger 2 meter on day 1 post intravitreal injection. After 1 month 14 days of follow up, patient had 6/36 aided vision in right eye. On OCT examination right eye again revealed cystoid macular edema.

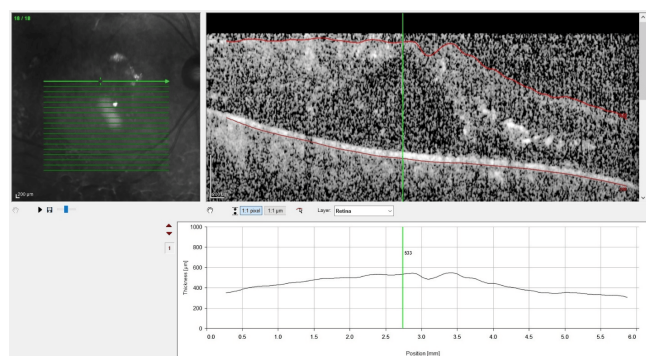


Figure 6. On OCT examination right eye again revealed cystoid macular odema, CMT was 533 μm . This is preop picture.

Patient was planned for right eye cataract surgery with IOL with intravitreal triamcinolone acetonide and was treated with same. Vision in right eye was 6/36 on 1st post operative day. On follow up after 1 month, OCT examination in right eye again revealed resolving diabetic macular edema, CMT was 245 μm .

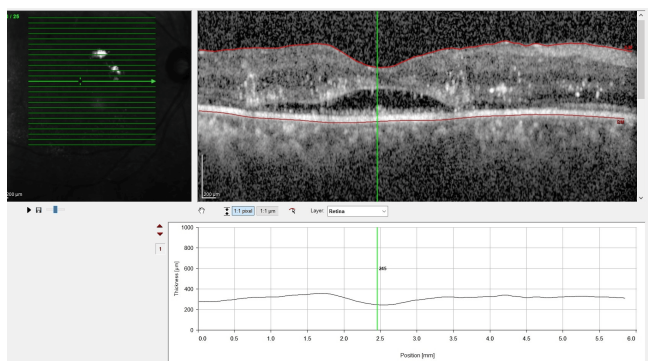


Figure 7. OCT examination right eye again revealed resolving diabetic macular edema, CMT was 245 μm . This is postop picture

CASE 2

A 55-year-old female presented in the outpatient department with a history of gradual painless diminution of vision in left eye since 4 months. She is known case of hypertension since 3 years on regular treatment and recently diagnosed case of diabetes mellitus since 2 months.

On ocular examination of the right eye, the patient has a vision best corrected visual acuity of 6/18 and 6/36 vision in left eye and on anterior segment evaluation revealed pseudophakia in both eyes.

On fundus examination of right eye revealed cup disc ratio of 0.3, multiple hypopigmented lesion in inferior quadrant. On fundus examination of left eye revealed, cup disc ratio of 0.3, multiple dot blot and flame shaped haemorrhages and hard exudate in macular area, FR (Foveal reflex)-was dull, cystoid macular edema was present, multiple hypopigmented lesion was present in inferior quadrant.



Figure 8. Fundus Photo of LE shows-Macular BRVO with CME, multiple dot blot and flame shaped hemorrhages and hard exudate in macular area.

OPTICAL COHERENCE TOMOGRAPHY (OCT) of the left eye showed cystoid macular edema with hard exudates in outer plexiform layer.

Patient was advised left eye intravitreal ranibizumab and was treated with the same. In the next followup

after 2 months vision in right eye was 6/18 and 6/24 vision in left eye. On OCT examination left eye again revealed cystoid macular edema, CMT was 564 μm .

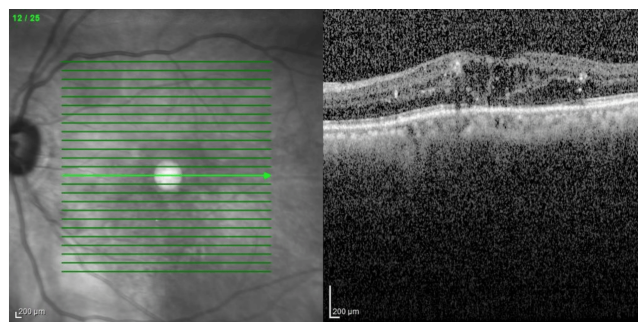


Figure 9. OCT examination of left eye revealed cystoid macular edema.

Patient was switched to faricimab after inadequate ranibizumab response. Informed consent was obtained from patient after discussing risk benefits. Patient was advised left eye intravitreal faricimab in dose of 6 mg in 0.05 mL, the interval between second dose was 2 months. After one month follow up vision improved to 6/18 in left eye. On OCT there was resolution of cystoid macular edema and CMT was 180 μm .

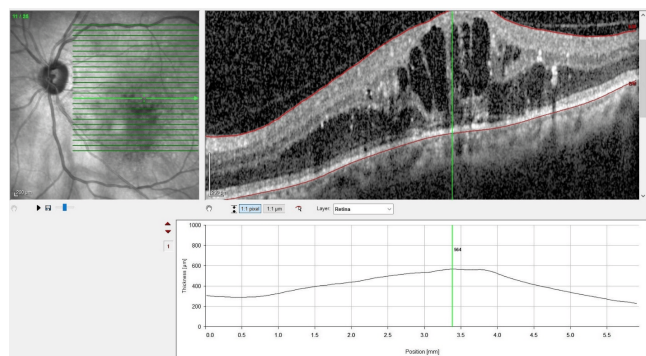


Figure 10. OCT examination of left eye revealed cystoid macular edema, CMT is 564 μm . This is the preop picture

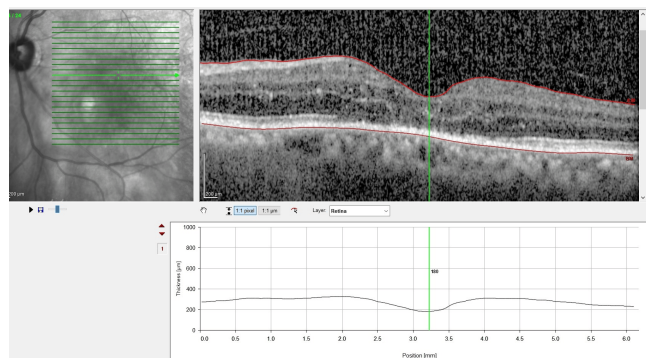


Figure 11. OCT examination of left eye after 1 month follow up with resolution of macular edema, CMT was 180 μm .

Patient was monitored for adverse event in every follow up and no adverse events were observed. Patient sat-

isfaction improved from moderate at early follow-ups to very satisfied by 3 months.

FARICIMAB

Faricimab is a novel bispecific monoclonal antibody composed of two heavy chains and two light chains. It is designed to target two pathways simultaneously: one ligand-binding arm neutralizes VEGF-A, while the other binds angiopoietin-2 (Ang-2). Faricimab targets two key pathways involved in DME pathogenesis [5].

Faricimab is a fully human 150-kDa monoclonal antibody of bispecific design. Its unique architecture is born from the CROSSMAB (CROSS OVER MONOCLONAL ANTIBODY) technology, which allows for the development of bispecific antibodies that closely resemble the natural immunoglobulin, featuring two separate antigen-binding domains [6,7].

Recent studies have demonstrated that the tyrosine kinase with immunoglobulin-like and epidermal growth factor-like domains 2 (Tie2) plays a critical role in angiogenesis as well as vascular stability, and inhibiting Ang2 leads to Tie2 activation. Tie2 is a transmembrane receptor expressed on endothelial cells and it serves as a binding site for the angiopoietin family of ligands, which includes angiopoietin-1 (Ang1) and Ang2 [8,9].

Once bound to Tie2, Ang1 functions to phosphorylate the receptor and then activates the downstream pathways that ultimately suppress vascular permeability and maintain vascular stability [10,11]. Ang2 is an endogenous, partial agonist of Tie2 that competes with Ang1 to downregulate the phosphorylation and activation of Tie2.

Ang2 levels are elevated in retinal vascular diseases, including nAMD, DR, proliferative DR, and RVO. Ang-1 ensures vascular stability in healthy tissues, Ang-1 activates the Tie2 receptor. Ang-2 is upregulated in retinal diseases. Ang-2 and VEGF-A are upregulated in the vitreous of people with nAMD, DR, and RVO, supporting a role for the Ang-2/Tie2 pathway in these pathologies.

Ang-2 and VEGF-A both act synergistically. Ang-2 inhibits the Tie2 receptor, promotes inflammation, and sensitises blood vessels to the activity of VEGF-A, leading to leakage and neovascularization.

Discussion

DIABETIC MACULAR EDEMA (DME) - PIVOTAL TRIALS

YOSEMITE and RHINE

1891 patients (YOSEMITE n=940, RHINE n=951) were randomized to intravitreal faricimab 6.0 mg every 8 weeks, faricimab 6.0 mg per personalized treatment

interval (PTI), or aflibercept 2.0 mg every 8 weeks up to week 100.

PTI is based on a protocol-driven dosing regimen, similarly to the so-called treat-and-extend regimen, wherein patients receive faricimab 6.0 mg every 4 weeks until CENTRAL SUBFIELD THICKNESS (CST) is less than 325 μm at/or after week 12, after which treatment intervals could be increased by 4 weeks (up to 16 weeks) or reduced by 4 or 8 weeks (down to 4 weeks) based on CST and best corrected visual acuity (BVCA). Across both trials, Faricimab showed non-inferior vision gains and improved anatomical outcomes when compared to Aflibercept [15].

The TAHOE trial noted improvements in visual acuity (as measured in ETDRS letters) and central subfield thickness parameters for both treatment-naïve and previously treated patients with DME who received at least one intravitreal injection in a real-world setting [19].

Long-term hyperglycemia contributes to breakdown of the blood–retina barrier with an increase in vascular permeability and a macular exudative fluid accumulation—hallmarks of DIABETIC MACULAR EDEMA (DME). Since the advent of intravitreal ANTI-VASCULAR ENDOTHELIAL GROWTH FACTOR (ANTI-VEGF) therapies in 2010, visual outcomes in patients with DME have significantly improved, establishing these agents as the standard first-line treatment for center-involved DME [16,17]. However, a substantial proportion of patients—over 30%—continue to exhibit persistent edema despite one year of appropriate therapy [18].

Repeated intravitreal injections are often required for management, which can prolong the treatment course and create significant financial and emotional stress for patients. In addition, repeated injections may increase the risk of ocular complications, including persistent elevation of intraocular pressure, retinal pigment epithelium tears, and geographic atrophy [20].

Biomarker of inflammation

HYPERREFLECTIVE FOCI (HRF) are identified on OCT as discrete, well-circumscribed, dot-shaped lesions up to 50 μm in diameter showing reflectivity equal to or greater than that of the retinal pigment epithelium band [21]. HRF are believed to be representative of activated microglia and may be a biomarker of an inflammatory microenvironment in retinal diseases [21,22].

OCT and OCTA Analysis

OCT serves to assess CRT (μm) in the central 1 mm circle of the ETDRS grid. SUPERFICIAL CAPILLARY PLEXUS (SCP) AND DEEP CAPILLARY PLEXUS (DCP), FOVEAL

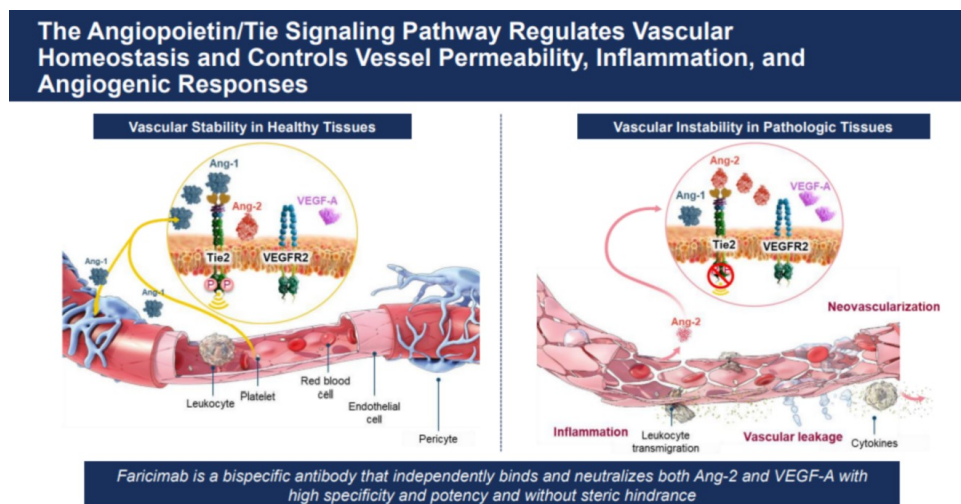


Figure 12. Molecular Mechanisms of Faricimab. The Angiopoietin/Tie Signaling Pathway Regulates Vascular Homeostasis and Controls Vessel Permeability, Inflammation, and Angiogenic Responses.

AVASCULAR ZONE (FAZ), OUTER RETINAL FLOW AREA (ORFA) AND CHORIOCAPILLARIS FLOW AREA (CCFA) are measured automatically using OCTA software.

In study by Dorota et al and Spiewak OCT and OCTA were performed before the first injection and one month after each subsequent administration of 6 mg faricimab. NON-PERFUSION AREAS (NPA) were measured manually but by examining ophthalmologist. Automatic measurements of ORFA and CCFA demonstrates the generation of vessel density maps covering a 6.4×6.4 mm area.

SCP and DCP vessel density is measured across the ETDRS grid with concentric circles at 1, 3, and 6 mm centred at the fovea. Pre-treatment scans reveal numerous NPA foci measured in the superficial layers of the retina; the software sums their area and displays the result in mm². In Optovue Solix, NPA areas are marked in yellow.

Following treatment, these areas showed noticeable improvement in capillary perfusion is seen as a reduction in the area of NPA zones marked in yellow. Improved retinal vascularization protects against the development of pathological retinal neovascularization with its numerous consequences, including blindness.

However, the SCP showed significant increase by the four month, indicating a delayed but notable therapeutic effect on perfusion status. The linear mixed-effects model for the DCP demonstrated a clear and statistically robust increase in DCP density from baseline over the four-month therapy period.

In most patients with DME, anti-VEGF therapy improves vascular perfusion. Additionally intravitreal faricimab led to a meaningful reduction in FAZ, which was more pronounced at two, three and four months. An improvement in the FAZ circularity was also observed,

which may indicate the restoration of parafoveal perfusion. Literature reports also indicate an increase in FAZ circularity within the superficial retinal vasculature after anti-VEGF therapy. Thus, the loss of FAZ circularity might be consistent with capillary atrophy at the FAZ border [23].

Result

Traditional first-line anti-VEGF monotherapies (such as ranibizumab or bevacizumab) exclusively target VASCULAR ENDOTHELIAL GROWTH FACTOR A (VEGF-A). However, as demonstrated in Case 2, a significant subset of patients exhibit anatomical non-responsiveness or early recalcitrance to selective VEGF-A blockade.

The case report is use about intravitreal Faricimab, in second case of refractory BRVO with cystoid macular edema as it was inadequate response to ranibizumab, so the Patient was switched to faricimab. There have been very fewer case reports on intravitreal faricimab, so this case report highlights the importance of novel drug intravitreal faricimab in DME and refractory BRVO with cystoid macular edema.

Importance of Intravitreal Faricimab in Reconstructive & Anti-VEGF Resistant DME

Faricimab represents a structural shift in treating refractory DME through its design as a bispecific antibody. It simultaneously targets two distinct disease pathways.

Dual Pathway Synergism (VEGF-A & Ang-2)

While VEGF-A drives endothelial cell proliferation and initial leakage, ANGIOPOIETIN-2 (Ang-2) acts synergistically to destabilize the vascular endothelium, increase sensitivity to VEGF, and promote structural inflammation.

Table 1. Clinical progression, treatments, and visual outcomes of both patients

Case	Patient Profile	Initial Ocular Status & Baseline CMT	Therapeutic Intervention(s)	Anatomical & Visual Outcome
Case 1	64M, HTN (6 mon), DM (1.5 yr), S/P PRP (6 mon)	RE: Vision - CF 1m; NS3 + PSC2 Cataract; CMT 599 μ m (DME present). LE: Vision 6/36; NS1 Cataract; CMT 572 μ m (DME present).	RE: IV Ranibizumab right eye; Phacoemulsification + IOL + IV Triamcinolone Acetonide (IVTA). LE: 2 doses of IV Faricimab 6 mg in 0.05 mL.	RE: Vision improved to 6/36; CMT resolved to 245 μ m. LE: Vision improved to 6/18; resolving macular edema; very high patient satisfaction by 3 months.
Case 2	55F, HTN (3yr), recently diagnosed DM (2 mon)	RE: Vision 6/18; Pseudophakic. LE: Vision 6/36; Pseudophakic; CMT 564 μ m (CME present with haemorrhages, hard exudates in macular area).	LE: Initial IV Ranibizumab. Switched to IV Faricimab 6 mg in 0.05 mL due to inadequate response to ranibizumab.	LE: Vision improved to 6/18; resolution of resistant cystoid macular edema; high patient satisfaction by 3 months.

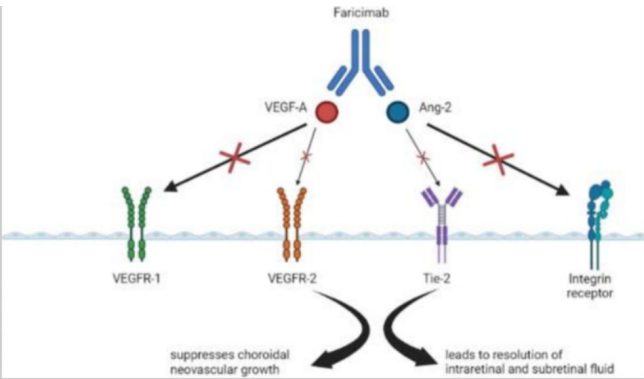


Figure 13. Dual pathway mechanism of action of Faricimab.

By inhibiting both ligands, Faricimab:

- Restores endothelial junction stability (via the Tie-2 receptor pathway).
- Decreases vascular permeability more effectively than VEGF-A inhibition alone.
- Suppresses underlying inflammatory cascades that contribute to chronic cystoid spaces.

Management of Refractory/Non-Responsive DME

As seen in the pseudophakic eye of Case 2, structural fluid persistence (CMT 564 μ m) despite ranibizumab therapy highlights the limitations of solo VEGF inhibition. Switching to faricimab yields secondary visual improvements to 6/18 within a single month. This underscores its role for eyes showing suboptimal response to traditional molecules.

Faricimab’s stabilizing effect on the capillary beds results in extended fluid control. In both clinical scenarios, stabilizing the macula led to a marked pivot in patient experience.

Conclusion

We report case series of 2 patients treated with Intravitreal FARICIMAB. FARICIMAB is the first intraocular bispecific antibody uniquely designed to simultaneously inhibit both Ang-2 and VEGF-A. Anti-VEGF-A reduce neovascularisation and vascular leakage. Anti-Ang-2 Reduce inflammation and vascular leakage and stabilise vessels. Additionally, the modified Fc region minimizes

systemic exposure and lowers inflammatory potential.

The visual and anatomical improvements observed with faricimab highlight its key important role in DME and refractory macular BRVO with CME. The limitation were very small sample size, short follow-up, heterogeneous diagnoses, and confounding by other ocular treatments in the fellow eye.

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