

Short-Term Anatomical and Functional Outcomes After Switching to Faricimab in Therapy-Resistant Retinal Vein Occlusion–Related Macular Edema: A Real-World Study

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Research Article

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Abstract

Background

This study evaluated the real-world anatomical and functional outcomes of faricimab in patients with macular edema secondary to retinal vein occlusion (RVO) who showed insufficient response to prior intravitreal therapies.

Methods

This retrospective, observational single-center study included consecutive patients with macular edema secondary to branch (BRVO) or central retinal vein occlusion (CRVO) who were switched to faricimab after inadequate response to prior intravitreal therapy, from January 2024 to October 2025. Persistent macular edema was defined as refractory intraretinal and/or subretinal fluid on optical coherence tomography (OCT) and/or lack of functional improvement despite previous intravitreal treatment with at least five intravitreal injections of anti-vascular endothelial growth factor (VEGF), and/or dexamethasone implant. Eligible eyes received at least three faricimab injections after the switch and had at least three months of follow-up. Best-corrected visual acuity (BCVA) and central subfield thickness (CST) were assessed before and after switching. Secondary outcomes included macular volume analysis and change in treatment intervals.

Results

Twenty-six eyes (25 patients; mean age $\pm$ SD 70.8 ± 10.7 years) were included; 53.8% ($n = 14$) BRVO and 46.2% ($n = 12$) CRVO. All eyes had received extensive prior intravitreal treatment: 16.6 ± 15.5 (20 eyes, 76.9%) mean previous ranibizumab injections; 33.0 ± 31.4 (23 eyes, 88.5%) mean previous aflibercept 2 mg injections; 3 eyes (11.5%) received at least 1 prior dexamethasone implant. Overall, the cumulative anti-VEGF burden before switching was 42.0 ± 37.6 injections per eye. After the first faricimab injection, mean CST decreased significantly from $307.4 \pm 80.0 \mu\text{m}$ to $269.5 \pm 48.2 \mu\text{m}$ ($\Delta - 37.9 \mu\text{m}$, mean paired change $- 35.5 \mu\text{m}$, $p = 0.001$). Mean macular volume also showed a reduction, most notably in the 6-mm foveal-centered ETDRS area ($p = 0.019$). BCVA remained stable, with a non-significant trend toward improvement ($p = 0.063$). Treatment intervals were maintained compared with pre-switch regimens ($p = 0.572$). After 3 injections, anatomical benefits were maintained, with persistent CST reduction ($p = 0.034$). No serious ocular or systemic adverse events were observed.

Conclusion

In this real-world cohort of therapy-resistant RVO-related macular edema, switching to faricimab produced rapid anatomical improvement while maintaining visual acuity and treatment intervals,

supporting its use as an effective, well-tolerated switch therapy in difficult-to-treat RVO.

Background

Retinal vein occlusion (RVO) is a common cause of visual impairment and is hallmarked by cystoid macular edema (CME) and retinal ischemia.¹ Depending on the extent of vascular involvement, RVO can be classified as central retinal vein occlusion (CRVO), hemiretinal vein occlusion (HRVO), and branch retinal vein occlusion (BRVO), with BRVO being the most common clinical subtype.¹ Although the exact pathophysiological mechanism remains under investigation, it is widely accepted that RVO-associated macular edema results from a disruption of vascular integrity and increased vascular permeability, driven by inflammatory processes.²

Intravitreal injections of anti-vascular endothelial growth factor (VEGF) agents are usually the first-line treatment for RVO-associated ME, with corticosteroid injections usually serving as the second-line therapy.³ Pharmacological advancements have enriched anti-VEGF treatment for RVO over the years. Specifically, after the first off-label use of bevacizumab,⁴ ranibizumab and aflibercept were approved in large clinical trials.^{5,6} However, the burden of patients requiring frequent injections, hinted to the need for broader-acting agents for the treatment of this complex disease.

Extensive research suggests the overexpression of angiopoietin-2 (Ang-2) in retinal vascular diseases, as well as its functional interconnection with VEGF-A in the exacerbation of the disease.^{7,8} In line with these findings, the recent approval of faricimab (Vabysmo, Roche/Genentech) for the treatment of RVO represents an improvement for targeting sustained disease control.⁹ The phase III BALATON and COMINO trials demonstrated similar clinical outcomes of faricimab compared to aflibercept 2mg in patients with RVO-associated CME.^{10,11} The dual inhibition of VEGF-A and Ang-2, differentiates the bispecific monoclonal antibody activity of faricimab from other VEGF-only targeting agents, potentially improving patient management and extending treatment intervals.⁹

Beyond the positive results of registration trials, real-world evidence on the effects of faricimab in patients with RVO is beginning to emerge, but remains largely confined to small, short-term cohorts, with limited data on switch outcomes such as those reported here.^{12–17} Thus, this study aims to investigate short-term treatment outcomes of faricimab in routine clinical practice for chronic, treatment-resistant RVO-associated CME in patients who previously received other intravitreal therapies.

Methods

Study design and cohort

This retrospective, observational, single-center study included consecutive patients with macular edema secondary to retinal vein occlusion (central or branch RVO) treated with faricimab at a tertiary referral center (Jules-Gonin Eye Hospital, Lausanne, Switzerland) between January 2024 and October 2025.

Eligible patients were required to have received a minimum of three intravitreal faricimab injections and to have been switched to faricimab for persistent macular edema following prior intravitreal therapy (anti-VEGF or corticosteroids). Persistent macular edema was defined as the persistence of intraretinal and/or subretinal fluid on spectral-domain optical coherence tomography (SD-OCT) and/or failure to achieve meaningful visual improvement despite at least five consecutive injections of another agent administered at standard dosing intervals. Exclusion criteria comprised fewer than three faricimab injections, treatment-naïve patients, macular edema secondary to other disease than RVO, absence of informed consent, and inadequate clinical and imaging data before and after the switch. The study was conducted in accordance with the principles of the declaration of Helsinki. The study protocol was reviewed and approved by the local ethics committee of the Canton of Vaud, Switzerland (CER-VD, Protocol Number: 2017 – 00493). All patients included had provided written general consent allowing the use of their coded health-related data for research purposes.

Treatment and imaging protocol

All patients underwent routine ophthalmic assessment and macular SD-OCT at each visit (Heidelberg Spectralis, Heidelberg Engineering, Germany). BCVA was measured as the number of letters read on standardized Early Treatment Diabetic Retinopathy Study (ETDRS) charts and converted to logMAR for statistical analysis. After switching, faricimab was administered without reinitiating a loading phase; instead, the treatment interval from the prior anti-VEGF agent was maintained or adjusted according to standard treat-and-extend principles, with extension or reduction in increments of 2 weeks at the discretion of the treating physician. Given the limited follow-up duration of this study, the full treat-and-extend protocol could not be completed or fully evaluated within this cohort. Clinical data were collected from electronic medical records and included patient demographics, RVO subtype, prior intravitreal therapies, and functional outcomes before and after switching to faricimab. Anatomical outcomes were derived from SD-OCT and included central subfield thickness (CST), presence or absence of intraretinal and/or subretinal fluid, and ETDRS macular volume metrics. Macular volumes were exported for the 1 mm (central subfield) and 6 mm ETDRS grids centered on the fovea using the Heidelberg Spectralis automated segmentation software and are presented in mm³. All segmentations were reviewed by a single grader (D.K.) and manually corrected where errors were identified prior to data export.

Statistical analysis

The sample size was determined by the number of eligible patients presenting between January 2024 and October 2025. Descriptive statistics were reported as mean $\pm$ standard deviation. The primary outcome measures were mean changes in BCVA and CST. Secondary outcomes included change in ETDRS macular volume, presence or absence of cystoid macular edema, treatment interval, and ocular or systemic adverse events. Pre- and post-switch continuous outcomes were compared using the Wilcoxon signed-rank test, as the data were not normally distributed. Categorical paired outcomes, including macular edema status, were compared using exact McNemar tests. Where multiple

comparisons were performed across time points or anatomical endpoints, p values were adjusted using the Holm method. A two-sided p value < 0.05 was considered statistically significant.

Results

Study population and baseline characteristics

A total of 26 eyes from 25 patients were included in the analysis. The mean age at the time of switching was 70.8 ± 10.7 years. The cohort comprised 15 male (60.0%) and 10 female patients (40.0%), with 15 left eyes (57.7%) and 11 right eyes (42.3%). Of the 26 included eyes, 53.8% (n = 14) had BRVO and 46.2% (n = 12) had CRVO. Cystoid macular edema was present in all 26 eyes (100%) at the time of switch, confirming active disease in the entire cohort.

Prior to switching to faricimab, all eyes had received prior intravitreal therapy with anti-VEGF agents and/or corticosteroids. Among included eyes, the mean number of prior ranibizumab injections was 16.6 ± 15.5 (20 eyes, 76.9%), and the mean number of prior aflibercept 2 mg injections was 33.0 ± 31.4 (23 eyes, 88.5%), yielding a mean cumulative anti-VEGF treatment burden of 42.0 ± 37.6 injections per eye. This reflects a highly pretreated cohort, with some eyes having received more than 100 prior anti-VEGF injections before the switch. Additionally, 3 eyes (11.5%) had received at least one prior dexamethasone implant. Prior retinal laser treatment had been performed in a subset of eyes, with 6 eyes (23.1%) having undergone previous panretinal photocoagulation (PRP) and 5 eyes (19.2%) having undergone previous focal laser treatment. Baseline characteristics of the study population, defined as the examination preceding the first faricimab injection (i.e., at the time of switch), are summarized in Table 1.

Table 1
Baseline characteristics of the study population (26 eyes, 25 patients).

Characteristic	Value
Eyes / Patients, n	26 / 25
Age at switch, mean $\pm$ SD (years) ¹	70.8 $\pm$ 10.7
Sex ¹	
Male, n (%)	15 (60.0%)
Female, n (%)	10 (40.0%)
Laterality ²	
Left eye, n (%)	15 (57.7%)
Right eye, n (%)	11 (42.3%)
RVO subtype ²	
BRVO, n (%)	14 (53.8%)
CRVO, n (%)	12 (46.2%)
Cystoid macular edema at switch, n (%) ²	26 (100%)
Prior intravitreal treatment²	
Ranibizumab, mean $\pm$ SD (n eyes)	16.6 $\pm$ 15.5 (n = 20)
Aflibercept 2 mg, mean $\pm$ SD (n eyes)	33.0 $\pm$ 31.4 (n = 23)
Total anti-VEGF injections, mean $\pm$ SD	42.0 $\pm$ 37.6
Prior dexamethasone implant, n (%)	3 (11.5%)
Prior retinal laser treatment²	
Panretinal photocoagulation (PRP), n (%)	6 (23.1%)
Focal laser treatment, n (%)	5 (19.2%)

BRVO, branch retinal vein occlusion; CRVO, central retinal vein occlusion; PRP, panretinal photocoagulation; SD, standard deviation; VEGF, vascular endothelial growth factor.

¹ Calculation per patient, ² calculation per eye.

Functional and anatomical outcomes

Mean BCVA remained stable throughout follow-up, measuring 0.18 ± 0.40 logMAR at baseline and 0.15 ± 0.42 , 0.19 ± 0.27 , 0.20 ± 0.49 , and 0.24 ± 0.58 logMAR after the first, second, third, and fourth faricimab injections, respectively. Although there was a trend towards improvement after the first injection, this did not reach statistical significance (mean change -0.028 logMAR; $p = 0.063$).

CST decreased significantly after the first faricimab injection, from 307.4 ± 80.0 μm at baseline to 269.5 ± 48.2 μm , corresponding to a mean between-group difference of -37.9 μm and a mean paired change of -35.5 μm ($p = 0.001$). Anatomical improvements were maintained during follow-up, with persistent CST reduction after 3 injections (mean paired change -30.9 μm ; $p = 0.034$), although this did not remain statistically significant after adjustment for multiple comparisons. The longitudinal evolution of CST after switching to faricimab is illustrated in Fig. 1.

CME was present in all eyes at baseline. All 26 eyes had a documented first, second, and third faricimab injection. However, BCVA and OCT assessments were not consistently performed or recorded at each injection visit. Outcome data after one injection, corresponding to the second-injection visit, were available for 22 eyes, with BCVA available for 23 eyes. After two injections, corresponding to the third-injection visit at approximately 3 months, complete BCVA and OCT data were available for 14 eyes (53.8%), despite all 26 eyes having received a third injection. After three injections, assessed at the fourth-injection visit, outcome data were available for 17 eyes (65.4%). After four injections, assessed at the fifth-injection visit, outcome data were available for 12 eyes (46.2%). This variation reflects the retrospective real-world design and the non-uniform timing and availability of clinical assessments relative to injection visits.

After the first faricimab injection, assessed at the second-injection visit, CME was absent in 7 of 22 evaluable eyes (31.8%), representing a significant reduction ($p = 0.016$), although this did not remain significant after correction for multiple comparisons (Holm-adjusted $p = 0.062$). CME absence was observed in 1 of 14 eyes (7.1%) after two injections, 3 of 17 eyes (17.6%) after three injections, and 4 of 12 eyes (33.3%) after four injections; however, these later changes were not statistically significant.

Macular volume decreased after switching to faricimab, with the most pronounced reduction observed in the 6-mm foveal-centered ETDRS area. Mean 6-mm foveal-centered ETDRS volume decreased from 9.00 ± 1.14 mm^3 at baseline ($n = 26$) to 8.51 ± 0.79 mm^3 after the first faricimab injection ($n = 22$; $p = 0.019$). This reduction was maintained after three injections, when data were available for 17 eyes (mean paired change -1.125 mm^3 ; $p = 0.012$; Holm-adjusted $p = 0.048$). The 1-mm ETDRS volume also decreased from 0.25 ± 0.08 mm^3 at baseline to 0.21 ± 0.04 mm^3 after the first injection ($n = 22$; $p = 0.013$), although this was only borderline significant after correction for multiple comparisons (Holm-adjusted $p = 0.050$). The longitudinal evolution of 6-mm foveal-centered ETDRS macular volume is illustrated in Fig. 2.

A representative case illustrating the clinical benefit of switching to faricimab is shown in Fig. 3. This patient with CRVO had received extensive prior intravitreal therapy, with persistently recurrent macular edema and inability to extend the treatment interval beyond four weeks. Following the switch to

faricimab, the macula remained dry at five weeks after the first injection, demonstrating both anatomical improvement and early evidence of improved durability.

The treatment interval used with the previous anti-VEGF agent was initially maintained after switching and was subsequently adjusted according to standard treat-and-extend principles, with extension or shortening in 2-week increments at the discretion of the treating physician. Treatment intervals remained unchanged after switching to faricimab. The mean pre-switch treatment interval was 6.2 ± 2.6 weeks, compared with a mean post-switch interval of 6.4 ± 2.6 weeks, with no statistically significant difference compared with pre-switch regimens ($p = 0.572$). On a per-eye basis, treatment intervals were not significantly extended after switching to faricimab. Only 2 of 26 eyes (7.7%) achieved a mean interval extension of at least 2 weeks.

No cases of intraocular inflammation, retinal vasculitis, or endophthalmitis were observed during the study period. No serious systemic adverse events related to faricimab were reported.

Subgroup analysis

In an exploratory subgroup analysis, CRVO eyes were significantly more heavily pretreated than BRVO eyes before switching to faricimab, with a higher number of prior anti-VEGF injections (68.3 ± 40.2 vs 19.5 ± 13.2 ; $p = 0.001$; Holm-adjusted $p = 0.013$). Baseline BCVA, CST, and 6-mm foveal-centered ETDRS volume did not differ significantly between BRVO and CRVO eyes. No statistically significant differences were observed between subgroups in CST reduction, BCVA change, or 6-mm foveal-centered ETDRS volume reduction during follow-up. Within the BRVO subgroup, CST decreased significantly after 1 and 3 faricimab injections, and 6-mm foveal-centered ETDRS volume was significantly reduced after 3 injections; however, these findings should be interpreted cautiously given the small sample size.

Discussion

In this retrospective single-center Swiss study, we evaluated the functional and anatomical outcomes of switching to faricimab in a real-world cohort of 26 eyes with ME secondary to RVO that had shown insufficient response to prior intravitreal therapies.

Our results demonstrate meaningful anatomical improvement following the switch to faricimab, and are broadly consistent with registration trials on faricimab for RVO-associated macular edema. BALATON and COMINO demonstrated non-inferior visual outcomes compared with aflibercept at 24 weeks in treatment-naïve RVO eyes, with sustained visual and anatomical outcomes and extended dosing durability reported through 72 weeks.^{10,11} These observations also align with published real-world data from recent international studies, which have demonstrated the efficacy of faricimab in ME secondary to RVO, particularly in terms of anatomical control. Studies switching therapy-resistant eyes to faricimab have consistently reported significant BCVA and CST/central foveal thickness (CFT) improvements at 3–9 months, including cohorts of 19–42 eyes from single centers in Germany and Japan.^{12–15} In a study

comparing naïve and switched eyes treated under a 1 + Pro Re Nata (PRN) regimen (30 eyes of 30 patients), anatomical gains were more pronounced than functional gains in switched eyes.¹⁶ Similarly, in a Chinese cohort of 52 pre-treated eyes (mean 9.16 ± 4.26 prior anti-VEGF injections), faricimab significantly improved visual acuity and reduced central macular thickness, with a favorable safety profile.¹⁷ Hikichi et al. reported in 42 eyes switched after aflibercept relapse that faricimab improved anatomical and functional outcomes while extending treatment intervals, though larger prospective studies with longer follow-up remain needed.¹⁵ Our cohort, albeit limited in size, is distinguished by particularly chronic, therapy-resistant cases and a comparatively short follow-up, which should be considered when interpreting the magnitude of functional versus anatomical response observed.

The dual mechanism of action of faricimab – targeting both VEGF-A and angiopoietin-2 – may explain these findings, acting not only on vascular permeability but also on inflammation and vascular stabilization, mechanisms particularly relevant to the pathophysiology of RVO. The present cohort adds complementary data from a Swiss tertiary-care real-world setting. A notable strength of this study is that all included eyes had already received extensive prior intravitreal treatment – with some having received more than 100 anti-VEGF injections – yet still demonstrated significant anatomical improvement after switching to faricimab. These observations are particularly relevant given the refractory nature of the population studied.

While the anatomical response was robust, the functional outcomes deserve a more nuanced interpretation. The absence of significant BCVA improvement should be interpreted in context: baseline BCVA was relatively good on average, leaving limited room for functional gain. In addition, chronic macular edema, ischemic damage, disorganization of the retinal inner layers, or photoreceptor disruption may limit visual recovery despite anatomical drying. Therefore, the main clinical message of this study should be anatomical improvement with visual stabilization, rather than visual acuity gain.

Beyond efficacy, an important practical question concerns whether faricimab allowed for a less demanding treatment regimen. On a per-eye basis, treatment intervals were not significantly extended after switching to faricimab: only 2 of 26 eyes (7.7%) achieved a mean interval extension of at least 2 weeks. Despite the treat-and-extend design, intervals were thus largely maintained rather than extended during the study period, likely reflecting the short follow-up and the severity of the treatment-resistant population studied. This is nonetheless clinically relevant, as anatomical gains were achieved without intensifying the injection schedule. However, the results should not be presented as evidence of improved durability or reduced treatment burden in this cohort. Longer follow-up and a prespecified treat-and-extend protocol would be needed to evaluate whether faricimab can extend intervals in this specific refractory population.

With regards to the exploratory subgroup analyses, CRVO eyes were significantly more heavily pretreated than BRVO eyes, yet no significant differences in treatment outcomes were observed between subtypes, suggesting that faricimab may be effective regardless of RVO subtype in this refractory setting.

Finally, in terms of safety and tolerability, none of our patients developed any serious adverse events, ocular or systemic, during follow-up. This suggests a favorable safety profile for faricimab in everyday clinical use. However, rare adverse effects, such as intraocular inflammation or thromboembolic events, have been reported in some studies and in post-marketing pharmacovigilance.¹⁸ Close clinical monitoring is therefore still recommended, and the use of faricimab should be interrupted in case of any complication.

Our study has several limitations. Its retrospective nature exposes it to selection bias and heterogeneity in follow-up and treatment protocols. In addition, the limited sample size restricts the statistical power and generalizability of the results. The absence of a control group and the variability of previous treatment regimens also constitute methodological limitations. A larger cohort of patients with longer follow-up would be needed to confirm these observations in real-world settings.

Conclusion

In this real-world cohort of therapy-resistant RVO-related macular edema, switching to faricimab resulted in rapid anatomical improvement, including significant macular volume reduction, and macular edema resolution in a subset of eyes, while visual acuity and treatment intervals remained stable. These findings suggest that faricimab may be an effective switch option in difficult-to-treat RVO eyes with persistent edema despite prior intravitreal therapy. Larger prospective controlled studies with longer follow-up are needed to confirm durability, functional outcomes, and safety.

Abbreviations

Ang-2	angiopoietin-2
BCVA	best-corrected visual acuity
BRVO	branch retinal vein occlusion
CER-VD	Commission cantonale d'éthique de la recherche sur l'être humain, Canton of Vaud
CFT	central foveal thickness
CME	cystoid macular edema
CRVO	central retinal vein occlusion
CST	central subfield thickness
ETDRS	Early Treatment Diabetic Retinopathy Study
FAERS	FDA Adverse Event Reporting System

FDA	Food and Drug Administration
HRVO	hemiretinal vein occlusion
logMAR	logarithm of the minimum angle of resolution
ME	macular edema
OCT	optical coherence tomography
PRN	Pro Re Nata
PRP	panretinal photocoagulation
RVO	retinal vein occlusion
SD	standard deviation
SD-OCT	spectral-domain optical coherence tomography
VEGF	vascular endothelial growth factor

Declarations

Ethics approval and consent to participate

This retrospective study was conducted in accordance with the principles of the Declaration of Helsinki. The study protocol was reviewed and approved by the Cantonal Ethics Committee of Vaud, Switzerland (Commission cantonale d'éthique de la recherche sur l'être humain, CER-VD; Protocol Number: 2017-00493). The requirement for individual written study-specific consent was waived by the ethics committee given the retrospective design; all patients had provided prior written general consent authorizing the use of their coded health-related data for research purposes, in accordance with Swiss law.

Clinical trial number: not applicable

Consent for publication

Not applicable — the imaging data presented in Figure 3 are fully anonymized and do not contain any information that could identify the individual patient.

Availability of data and materials

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Competing interests

CE has participated in advisory boards and served as a lecturer for AbbVie, Apellis, Astellas, Bayer, and Roche. AM has received educational grants from Roche Pharma (Switzerland) Ltd, and speaker fees from F. Hoffmann-La Roche Ltd. SV, DK and EDOF declare that they have no competing interests.

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Authors' contributions

SV, DK, AM and CE conceived and designed the study. DK collected the clinical data. SV and DK performed the statistical analysis. SV and DK interpreted the results and drafted the manuscript. All authors critically revised the manuscript for important intellectual content, approved the final version, and agree to be accountable for all aspects of the work.

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Not applicable.

References

1. Laouri M, Chen E, Looman M, Gallagher M. The burden of disease of retinal vein occlusion: review of the literature. *Eye (Lond)*. 2011;25(8):981–8. 10.1038/eye.2011.92.
2. Dinah C, Dodds M, Lotery A, et al. Treatment patterns and long-term outcomes in anti-VEGF-treated macular oedema secondary to retinal vein occlusion: a retrospective observational study. *Eye (Lond)*. 2025;40(1):107–16. 10.1038/s41433-025-04089-2.
3. Modi YS, Klufas MA, Sridhar J, Singh RP, Yonekawa Y, Pecun P. Current best clinical practices—management of retinal vein occlusion. *J Vitreoretin Dis*. 2020;4(3):214–9. 10.1177/2474126420906395.
4. Dascalu AM, Popa-Cherecheanu A, Popa-Cherecheanu M, Nica A, Serban D. Off-label use of intravitreal bevacizumab in non-ischemic macular edema secondary to retinal vein obstructions. *Rom J Ophthalmol*. 2016;60(2):90. 10.22336/rjo.2016.15.
5. Heier JS, Campochiaro PA, Yau L, et al. Ranibizumab for macular edema due to retinal vein occlusions: long-term follow-up in the HORIZON trial. *Ophthalmology*. 2012;119(4):802–9. 10.1016/j.opthta.2011.12.005.
6. Pielen A, Clark WL, Boyer DS, et al. Integrated results from the COPERNICUS and GALILEO studies. *Clin Ophthalmol*. 2017;11:1533–40. 10.2147/OPTH.S140665.

7. Regula JT, von Lundh P, Foxton R, et al. Targeting key angiogenic pathways with a bispecific CrossMAb optimized for neovascular eye diseases. *EMBO Mol Med*. 2016;8(11):1265–88. 10.15252/emmm.201505889.
8. Kuriyama A, Nakamura S, Inokuchi Y, et al. The protective effect of anti-VEGF-A/Ang-2 bispecific antibody on retinal vein occlusion model mice. *Eur J Pharmacol*. 2024;976:176691. 10.1016/j.ejphar.2024.176691.
9. Chen KY, Chan HC, Chan CM. Effectiveness and safety of anti-vascular endothelial growth factor therapies for macular edema in retinal vein occlusion: a systematic review and network meta-analysis of randomized controlled trials. *Surv Ophthalmol*. 2025;70(6):1067–89. 10.1016/j.survophthal.2025.05.008.
10. Tadayoni R, Paris LP, Danzig CJ, et al. Efficacy and safety of faricimab for macular edema due to retinal vein occlusion: 24-week results from the BALATON and COMINO trials. *Ophthalmology*. 2024;131(8):950–60. 10.1016/j.opthta.2024.01.029.
11. Danzig CJ, Dinah C, Ghanchi F, et al. Faricimab treat-and-extend dosing for macular edema due to retinal vein occlusion: 72-week results from the BALATON and COMINO trials. *Ophthalmol Retina*. 2025;9(9):848–59. 10.1016/j.oret.2025.03.005.
12. Inokuchi S, Mizuki Y, Kamata A, Onishi J, Hayashi T, Mizuki N. Real-world efficacy of intravitreal faricimab for macular edema secondary to retinal vein occlusion: short-term outcomes and optical coherence tomography biomarker analysis. *Clin Ophthalmol*. 2025;19:3669.
13. Hafner M, Herold TR, Kufner A, Asani B, Anschütz A, Eckardt F, et al. Switching to faricimab in therapy-resistant macular edema due to retinal vein occlusion: initial real-world efficacy outcomes. *J Clin Med*. 2025;14(7):2454. 10.3390/jcm14072454.
14. Hafner M, Herold TR, Kufner A, et al. Extended real-world efficacy of faricimab in therapy-resistant macular edema due to retinal vein occlusion: 9-month follow-up results. *J Clin Med*. 2025;14(20):7197. 10.3390/jcm14207197.
15. Hikichi T, Kurabe H, Notoya A, et al. Short-term outcomes of switching to faricimab for macular edema secondary to retinal vein occlusion. *Jpn J Ophthalmol*. 2025;69:918–25.
16. Hirakata T, Toride A, Ashikaga K, et al. Short-term real-world effectiveness of faricimab on macular edema due to retinal vein occlusion. *Int J Retina Vitreous*. 2025;11(1):703. 10.1186/s40942-025-00703-3.
17. Sun Y, Zhao M, Miao H, et al. Real-life short-term experience of switching from other anti-VEGF therapy to faricimab in patients with refractory macular edema secondary to retinal vein occlusion in China. *Int Ophthalmol*. 2025;45:463.
18. He CZ, Qiu Q, Lu SJ, Xue FL, Liu JQ, He Y. Adverse event reporting of faricimab: a disproportionality analysis of FDA adverse event reporting system (FAERS) database. *Front Pharmacol*. 2025;16:1521358. 10.3389/fphar.2025.1521358.

Figures

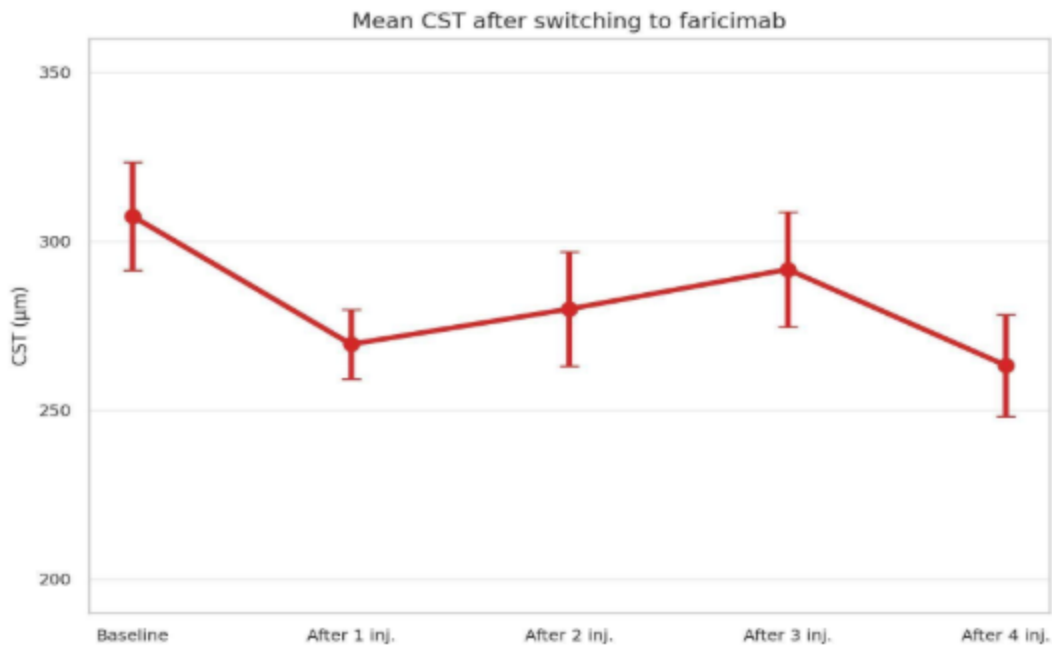


Figure 1

Mean central subfield thickness (CST) before and after switching to faricimab. Values are shown at baseline and after each of the first four faricimab injections. Error bars represent standard error of the mean

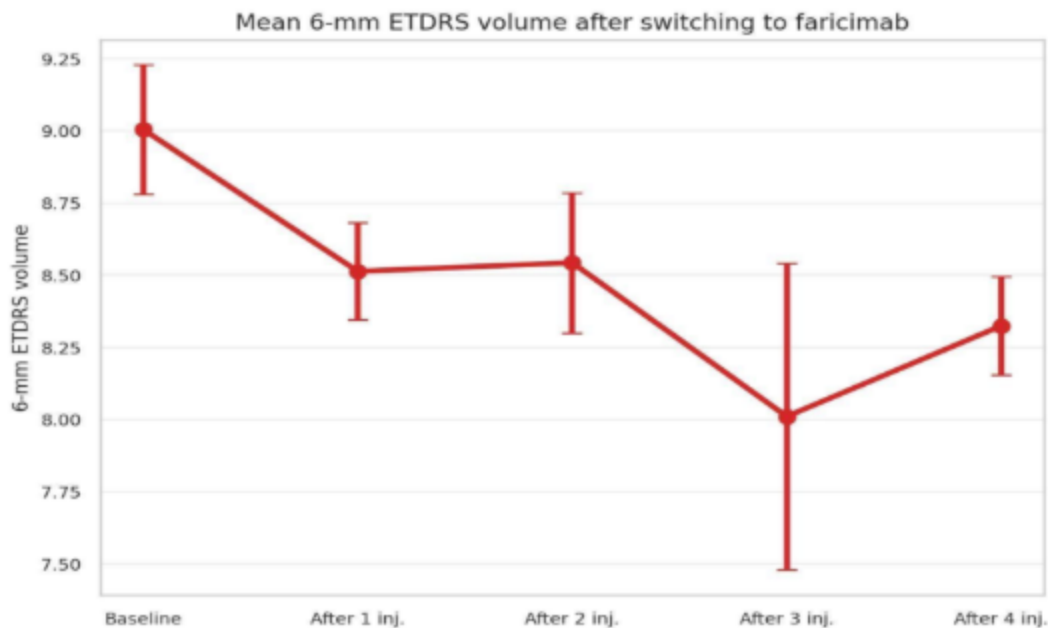


Figure 2

Mean 6-mm foveal-centered ETDRS macular volume before and after switching to faricimab. Values are shown at baseline and after each of the first four faricimab injections. Error bars represent standard error of the mean.

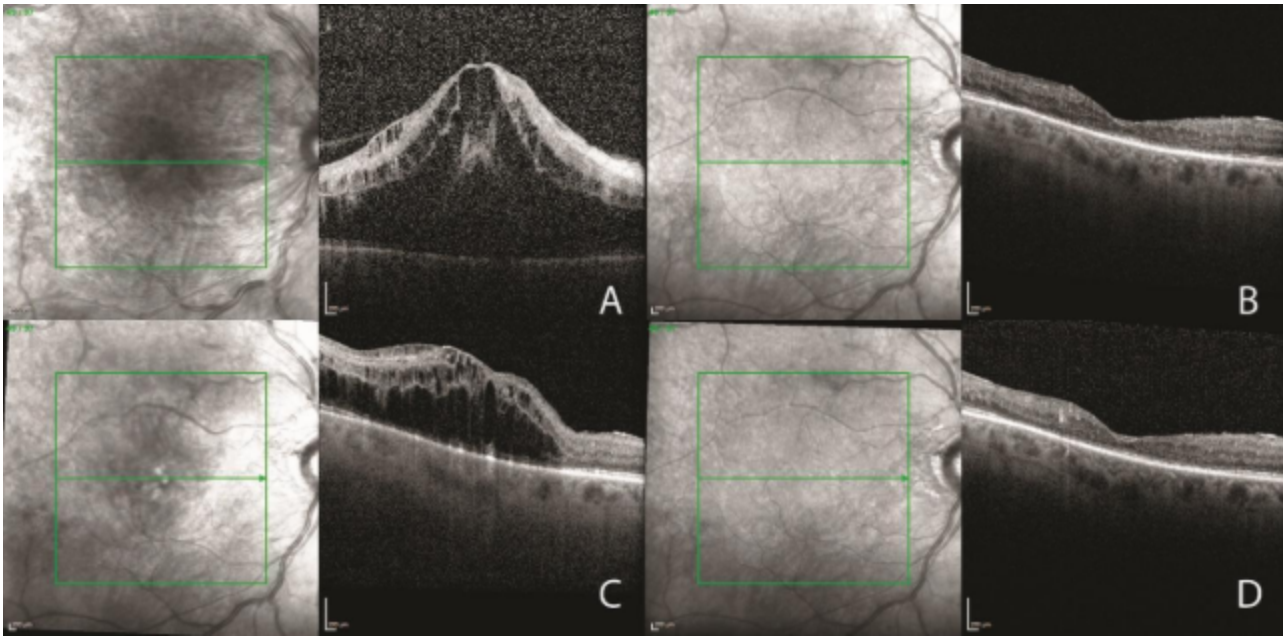


Figure 3

Representative treatment course of a single patient from the cohort. (A) Baseline macular OCT of the right eye demonstrating central retinal vein occlusion with CME. Over a 7-year follow-up under intravitreal therapy, the patient received a total of 42 ranibizumab injections, 4 dexamethasone intravitreal implants (Ozurdex) and 5 aflibercept 2 mg injections, with persistently recurrent macular edema. (B) Disease control achieved on a fixed 4-week ranibizumab regimen. (C) Recurrence of macular edema upon attempted treatment extension to a 5-week interval. (D) Following the switch to faricimab, the macula remained dry at 5 weeks after the first injection.