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INTRAVITREAL FARICIMAB (VABYSMO) IN NEOVASCULAR AGE-RELATED MACULAR DEGENERATION (NAMD) AND DIABETIC MACULAR EDEMA (DME): STRUCTURED EVIDENCE REVIEW

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ABSTRACT

Background: Anti-VEGF intravitreal therapy has transformed the treatment of neovascular age-related macular degeneration and diabetic macular edema; however, issues like treatment burden and incomplete anatomic responses persist. Faricimab (Vabysmo) is a humanized bispecific antibody that inhibits both VEGF-A and stabilizes vessels, addressing these challenges through its dual action. Objective: To assess the effectiveness, durability, safety, pharmacodynamics and feasibility of intravitreal faricimab in adults with nAMD or DME. **Methods:** Structured literature search was performed, using peer-reviewed pivotal randomized trials, two-year follow-up studies, pharmacodynamic studies, regulatory prescribing information, systematic reviews/network meta-analyses, trial registries and real-world observational evidence. The core evidence base included the TENAYA/LUCERNE trials in nAMD, the YOSEMITE/RHINE trials in DME, and some comparative and real-life studies. This is a structured evidence synthesis and not a prospective, registered systematic review, because it lacked duplicate, independent screening and a new quantitative meta-analysis. **Results:** At one year, faricimab showed non-inferior visual outcomes compared to aflibercept for treatment-naïve nAMD, with sustained results at two years. A significant percentage of patients achieved Q16W dosing by week 112. There were no notable differences between fixed DME Q8W and personalized DME T&E faricimab regimens in visual outcomes one year post-treatment. Faricimab demonstrated similar visual outcomes with fewer injections and favored anatomic results for two-year DME when compared to aflibercept. Safety profiles were similar; however, specific risks such as intraocular inflammation and arterial thromboembolic events require monitoring. Real-world data indicate that visual improvements may be less durable in previously treated nAMD eyes. **Conclusions:** Faricimab demonstrates effective visual outcomes, strong anatomical control, and a prolonged dosing schedule in treating nAMD and DME. However, its primary benefit is not necessarily a consistent improvement in visual acuity. Analysis must consider trial populations, aflibercept Q8W comparator treatments, sponsor involvement in pivotal trials, and the absence of long-term independent comparative data.

KEYWORDS: Faricimab; Vabysmo; Intravitreal Injection; Neovascular Age-Related Macular Degeneration.

INTRODUCTION

The most common preventable and treatable causes of sight loss in older age and in people with diabetes are neovascular AMD and DME. nAMD involves vascular leakage, hemorrhage, retinal damage to the retinal pigment epithelium and photoreceptors. DME is caused by damage to the inner blood–retinal barrier, the build-up of fluid in the inner layer of the retina or the outer layer of the retina and neurovascular dysfunction in the retina associated with diabetes. While the initiating causes may

vary, both disorders share the common features of retinal vascular permeability, inflammation and tissue remodeling. These mechanisms have contributed to the use of anti-VEGF therapy as a cornerstone of modern-day retinal care.[1,2] Intravitreal anti-VEGF drugs greatly improved visual function in both diseases. However, success in clinical trials cannot necessarily be replicated in clinical practice. Multiple injections may be needed over many years, visits for monitoring are needed on a regular basis, and interval adjustments are needed on a per-patient basis. Repeated injections over many years, frequent monitoring visits and individual interval adjustments are all common. Treatment burden impacts patient, caregiver and health system adherence. If injections are not given on time or if visits are missed, this can allow the fluid, irreversible photoreceptor damage, fibrosis, or macular ischemic damage to return. The intensity of treatment is often reduced from protocols in nAMD, responses in DME are variable, and factors such as glycemic control, renal disease, macular ischemia, epiretinal membranes, and outer retinal layer integrity may all play a role. A key therapeutic goal is not only to control VEGF, but to control the disease for extended periods of time without compromising the vision.[3] The currently available traditional agents are mainly VEGF inhibitors (ranibizumab, aflibercept and bevacizumab). In pathological angiogenesis, VEGF-A plays a key role in enhancing endothelial permeability, but is not the only factor that regulates vascular stability. The angiopoietin–Tie2 pathway is also important. Angiopoietin-1 can promote the integrity of endothelial junctions and vascular quiescence via Tie2 activation, while Ang-2 may be able to block Ang-1/Tie2 signalling and make vessels more responsive to VEGF-A. In disease conditions, Ang-2 and VEGF-A could act together to allow for leakage, inflammation, neovascularization and destabilization of the retinal microvasculature. The rationale for dual pathway inhibition, versus VEGF blockade alone, is provided by this biological model.[1,4] Faricimab is a humanized bispecific immunoglobulin G1 (bIgG1) antibody with dual specificities against VEGF-A and Ang-2. The Fc region has been engineered to decrease binding to Fc receptors and neonatal Fc receptor to decrease systemic uptake and Fc-mediated inflammatory activity upon intraocular administration. The molecule is thus a pharmacological attempt to have both direct VEGF-A suppression and stabilization of the vessels via the Ang-2 inhibition. Pharmacodynamic modeling of the patients with nAMD or DME indicated that there would be > 50% Ang-2 suppression for a minimum of 12 weeks at the 12W and 16W dosage regimens, and 9–10 week VEGF-A suppression was anticipated.[5] These data do not conclusively prove, but are consistent with, longer dosing duration reported in clinical trials.

The clinical development program included four phase 3 trials of faricimab. The primary endpoint of TENAYA and LUCERNE were treatment-naïve nAMD, treated with faricimab based on disease-activity criteria up to Q16W versus aflibercept Q8W.[6] YOSEMITE and RHINE compared a fixed Q8W dose of faricimab to personalized T&E faricimab through Q16W and aflibercept Q8W.[7] The primary purpose of these trials was to prove non-inferiority with respect to visual acuity, and durability, retinal thickness, fluid status and safety were important secondary evidence. Two-year publications later looked at whether both visual and anatomic benefits were preserved when the dosing of faricimab was personalized.[8,9] The extrapolation of faricimab involves separating out the concepts of visual efficacy, anatomic efficacy and durability of therapy. Even if the mean difference in visual acuity between a two therapies is minimal, one therapy may result in the same visual outcome as the other therapy treatment with fewer injections. On the other hand, an increase in retinal drying is not necessarily accompanied by a corresponding improvement in visual function, especially if accompanied by chronic retinal edema, retinal atrophy, fibrosis or retinal ischemia limiting photoreceptor recovery. The most clinically relevant question is then whether the number of injections needed to maintain vision and disease control is reduced in populations that are similar to the clinical routine. Randomized trials are not the only type of evidence that is relevant since registrational studies often recruit relative or treatment-naïve patients. The TRUCKEE study, for instance, had a fair number of previously treated eyes with nAMD, and offers data on switching, short term anatomic response and early safety data in routine practice.[10] But there is a risk of selection bias, regression to the mean, variable follow-up, unequal treatment interval and confounding by indication in observational studies. Likewise, there are limitations in the ability of network meta-analysis to compare regimens indirectly without completely removing differences in eligibility, loading schedules, outcome definition, or follow-up. The focus of this review will thus be to offer a balanced academic review of intravitreal faricimab in the treatment of nAMD and DME. It emphasizes mechanism of action, key randomized trials, durability of response, safety, comparative interpretation, real-world applicability and gaps in the research. The main clinical interest of faricimab is not universally, but more likely, the reduction in visual acuity in comparison to existing anti-VEGF treatments, but rather its ability to preserve visual outcomes while extending treatment intervals.

METHODOLOGY

Discuss the design and the question.

The question addressed by this article was: What is the comparative efficacy of intravitreal faricimab with established anti-VEGF therapy in terms of visual acuity, retinal structure, treatment durability, total number of intravitreal injections, and safety, among adults with nAMD or DME? Evidence identification Medline and PubMed records, open access full-text journal articles, large ophthalmology and medical journals, ClinicalTrials.gov records, regulatory prescribing information, and systematic reviews or network meta-

analyses were used to find evidence. Search terms were faricimab, Vabysmo, intravitreal, neovascular age-related macular degeneration, diabetic macular edema, TENAYA, LUCERNE, YOSEMITE, RHINE, treat-and-extend, safety, and real-world. The search was geared towards primary trial publications and regulatory documents of authority; secondary reviews were not intended to supplant primary reports, but were utilized to give context to comparative evidence.

Eligibility & selection principles

Randomized controlled trials, prespecified long-term extensions, pharmacodynamic studies, systematic reviews/network meta-analyses, regulatory documents, and observational studies in adult populations of nAMD or DME were eligible for inclusion. Studies were prioritised if they reported best corrected visual acuity (BCVA), central subfield thickness (CST), fluid status, dosing interval, number of injections administered, adverse events or treatment discontinuation. The principal scope excluded studies which were solely on other retinal diseases. RVO, pediatrics and non-ocular indications were not thoroughly reviewed.

Data extraction and synthesis

Extracted variables were study design, size of the study population, disease population, comparator, dosing schedule, follow-up, efficacy endpoints, durability measures, anatomic outcomes, and safety outcomes. Due to the varied backgrounds, disease populations, loading schedules, endpoint timing and control regimens for the studies, results were not pooled in a new meta-analysis but instead were summarized narratively and also in evidence tables. Published values are reported numerically; if a range or if the value was reported for two trials, this will be explicitly stated. Randomized phase 3 evidence was considered the strongest source of information for comparative efficacy, regulatory information was considered the strongest for labeled administration and warnings, and retrospective observational data were considered the lower level of certainty for effectiveness and safety.

Methodological limitations

This was not a prospectively registered systematic review. No de novo PRISMA flow diagram was created, no dual independent screening was done and no formal risk-of-bias scoring nor independent statistical re-analysis was performed. There is also the possibility of publication and source-availability bias should be noted as the review also covers industry-sponsored pivotal trials and selected open-access sources. These restrictions are mentioned to avoid misinterpretation of this article as a formal quantitative systematic review or a clinical practice guideline.

RESULTS

Pivotal randomized evidence

The pivotal evidence consisted of two phase 3 trials in nAMD and two phase 3 trials in DME. TENAYA and LUCERNE enrolled treatment-naïve patients aged at least 50 years with active nAMD. Patients received four initial monthly faricimab doses followed by protocol-defined dosing up to Q16W, whereas the comparator received aflibercept 2.0 mg after loading and then Q8W. At one year, both trials met the prespecified non-inferiority objective for BCVA. At two years, visual gains remained comparable, despite faricimab treatment being individualized in year two.[6,8]

YOSEMITE and RHINE enrolled adults with center-involving DME and randomized them to faricimab Q8W, faricimab personalized T&E up to Q16W, or aflibercept Q8W. All faricimab regimens met the non-inferiority objective for one-year visual outcomes. At two years, visual gains remained clinically meaningful and comparable with aflibercept, while the T&E arm used fewer injections. The most consistent advantage of faricimab in DME was anatomic: CST reductions and absence of intraretinal fluid were generally more frequent than with aflibercept Q8W.[7,9]

Table 1: Pivotal randomized trial outcomes for faricimab in nAMD and DME.

Trial and population	Design and comparator	Faricimab visual outcome	Comparator visual outcome	Principal anatomic or interpretive finding
TENAYA, nAMD	Phase 3; faricimab up to Q16W vs aflibercept Q8W	Week 48: +5.8 ETDRS letters	Week 48: +5.1 letters	Non-inferior vision; greater early CST reduction reported during matched dosing
LUCERNE, nAMD	Phase 3; faricimab up to Q16W vs aflibercept Q8W	Week 48: +6.6 letters	Week 48: +6.6 letters	Non-inferior vision; early anatomic outcomes favored faricimab numerically
TENAYA, nAMD	Two-year follow-up	Week 112: +3.7 letters	Week 112: +3.3 letters	CST change at year 2: −146.5 vs −146.2 µm; broadly comparable
LUCERNE, nAMD	Two-year follow-up	Week 112: +5.0 letters	Week 112: +5.2 letters	CST change at year 2: −150.3 vs −141.6 µm
YOSEMITE, DME	Phase 3; faricimab Q8W, faricimab PTI, or aflibercept Q8W	Week 52: +10.7 Q8W; +11.6 PTI	+10.9 letters	Both faricimab regimens non-inferior; PTI permitted extension up to Q16W
RHINE, DME	Phase 3; faricimab Q8W,	Week 52: +11.8	+10.3 letters	Both faricimab regimens non-

	faricimab PTI, or aflibercept Q8W	Q8W; +10.8 PTI		inferior; PTI permitted extension up to Q16W
YOSEMITE, DME	Two-year follow-up	Year 2: +10.7 Q8W; +10.7 T&E	+11.4 letters	CST reduction: −216.0 Q8W; −204.5 T&E; −196.3 aflibercept
RHINE, DME	Two-year follow-up	Year 2: +10.9 Q8W; +10.1 T&E	+9.4 letters	CST reduction: −202.6 Q8W; −197.1 T&E; −185.6 aflibercept

ETDRS, Early Treatment Diabetic Retinopathy Study; CST, central subfield thickness; DME, diabetic macular edema; nAMD, neovascular age-related macular degeneration; PTI, personalized treatment interval; Q8W, every 8 weeks; Q16W, every 16 weeks; T&E, treat-and-extend. Values are adjusted mean changes as reported in the cited trial publications.[6-9]

Durability and treatment burden

Durability was the defining differentiating feature of the faricimab program. In TENAYA and LUCERNE, approximately 45% of faricimab-treated participants were receiving Q16W treatment at week 48. By week 112, Q16W dosing was achieved by 59.0% of patients in TENAYA and 66.9% in LUCERNE; 74.1% and 81.2%, respectively, were receiving Q12W or longer intervals. These proportions demonstrate that a substantial subset of treatment-naïve nAMD patients can remain controlled on extended intervals, although they do not imply that every patient can safely receive Q16W treatment.[8]

The DME findings were similar but were expressed through the PTI/T&E algorithm. At week 96, more than 60% of patients in the faricimab T&E arms were receiving Q16W dosing and approximately 80% were receiving Q12W or longer dosing. The median number of study-drug injections over the full two-year study was 10 in YOSEMITE and 11 in RHINE for faricimab T&E, compared with 15 injections for faricimab Q8W and 14 injections for aflibercept Q8W. Importantly, the reduction in injections was achieved without loss of mean visual acuity.[9]

Table (2): Faricimab durability and treatment-burden outcomes.

Disease and study	Follow-up point	Faricimab durability finding	Injection-burden implication	Clinical interpretation
nAMD, TENAYA	Week 48	Approximately 45% achieved Q16W	Fewer injections than fixed Q8W for eligible patients	Early durability was demonstrated in a selected treatment-naïve population
nAMD, LUCERNE	Week 48	Approximately 45% achieved Q16W	Same general implication as TENAYA	Durability was reproducible across two trials
nAMD, TENAYA	Week 112	59.0% achieved Q16W; 74.1% achieved Q12W or longer	Sustained interval extension in most patients	Q16W is feasible for a substantial, not universal, subgroup
nAMD, LUCERNE	Week 112	66.9% achieved Q16W; 81.2% achieved Q12W or longer	Sustained interval extension in most patients	Results support disease-activity-guided individualized dosing
DME, YOSEMITE	Week 96	>60% of T&E patients achieved Q16W	Median 10 T&E injections vs 15 Q8W injections	T&E reduced treatment burden while preserving vision
DME, RHINE	Week 96	>60% of T&E patients achieved Q16W	Median 11 T&E injections vs 15 Q8W injections	Durability was consistent across the two DME trials
DME, pooled trial interpretation	Week 96	Approximately 80% achieved Q12W or longer in T&E arms	Fewer visits/injections for responders	Interval should remain contingent on OCT and visual response

DME, diabetic macular edema; nAMD, neovascular age-related macular degeneration; OCT, optical coherence tomography; Q8W, every 8 weeks; Q12W, every 12 weeks; Q16W, every 16 weeks; T&E, treat-and-extend.[8,9]

Safety, pharmacodynamics, comparative evidence, and real-world findings

The FDA prescribing information specifies a 6-mg dose in 0.05 mL of a 120-mg/mL solution. For nAMD, four initial monthly doses are followed by disease-activity assessment and dosing at Q8W, Q12W, or Q16W according to the labeled algorithm. For DME, the label permits either a personalized interval strategy after initial monthly treatment or a fixed schedule involving monthly loading followed by Q8W treatment. Contraindications include ocular or periocular infection, active intraocular inflammation, and known hypersensitivity. The label warns about endophthalmitis, retinal detachment, transient IOP elevation, and the potential for arterial thromboembolic events associated with VEGF inhibition.[11]

In the pivotal studies, ocular adverse events were broadly comparable between faricimab and aflibercept. Intraocular inflammation was uncommon, generally

around 1–2% in the reported trial populations, and no new safety signal emerged in the two-year nAMD or DME reports. The absence of significant difference in common adverse events should not be interpreted as proof that risks are identical in every patient or every practice setting. Intravitreal injection itself carries risks, and rare events may only become apparent after wider post-marketing exposure. Pharmacodynamic data support the biological plausibility of durability. In patients receiving Q12W or Q16W regimens, more than 50% Ang-2 suppression was predicted to persist for at least 12 weeks, while VEGF-A suppression was predicted to last approximately 9–10 weeks. This finding supports, but does not replace, OCT- and vision-based treatment decisions.[5]

A 2024 network meta-analysis of nAMD trials concluded that faricimab provided comparable efficacy and safety to other anti-VEGF agents while reducing annual injections relative to fixed or flexible regimens. A DME network meta-analysis similarly suggested favorable anatomic and durability outcomes for faricimab T&E, but indirect comparisons are limited by cross-trial heterogeneity and differences in dosing protocols.[12,13] The strongest comparative inference therefore remains the direct non-inferiority evidence against aflibercept 2.0 mg Q8W in the pivotal trials.

Real-world data are more heterogeneous. In the six-month TRUCKEE study, 376 nAMD eyes were evaluated after one faricimab injection, including 337 previously treated eyes and 39 treatment-naïve eyes. Mean BCVA increased by 1.1 letters overall and CST decreased by 31.3 µm. Previously treated eyes had a smaller mean visual change (+0.7 letters) but a significant CST reduction of 25.3 µm. After three injections, 94 eyes showed a mean gain of 3.4 letters and a mean CST reduction of 43.4 µm. One case of intraocular inflammation and one case of infectious endophthalmitis were reported, with resolution after treatment.[10] These findings suggest that switching may improve anatomy and stabilize vision, but they do not establish superiority over continued anti-VEGF therapy and cannot be extrapolated to long-term durability without caution.

Table 3: Safety and applicability of intravitreal faricimab.

Evidence domain	Main finding	Certainty	Practical implication or limitation
Regulatory dose	6 mg/0.05 mL intravitreally	High	Administration should follow the current local product label and qualified-physician technique
Contraindications	Ocular/periocular infection, active intraocular inflammation, hypersensitivity	High	Treatment should be deferred or avoided when contraindications are present
Common adverse reaction	Conjunctival hemorrhage reported in approximately 7% in the original label safety population	High	Usually mild, but incidence varies by study and comparator
Intraocular inflammation	Generally uncommon; approximately 1–2% in pivotal trial reports	Moderate to high	Patients require evaluation for inflammation and other post-injection complications
Arterial thromboembolic events	Low and comparable with aflibercept in the original label data	High for trial populations	Systemic vascular history should be considered clinically; the label warning remains relevant
Pharmacodynamic durability	Predicted >50% Ang-2 suppression for ≥12 weeks in Q12W/Q16W groups	Moderate	Supports, but does not determine, interval extension
nAMD randomized efficacy	Non-inferior BCVA with extended dosing; comparable two-year anatomy	High	Best evidence applies to treatment-naïve trial-like nAMD patients
DME randomized efficacy	Non-inferior BCVA; greater CST and fluid control in several comparisons	High	Anatomic superiority does not guarantee a proportional visual advantage
Previously treated nAMD	Stable or modestly improved vision with CST reduction after switching in TRUCKEE	Low to moderate	Retrospective design and short follow-up limit causal inference
Generalizability	Real-world populations may be more treatment-resistant and less adherent	Moderate	Trial durability may overestimate routine-practice durability

Ang-2, angiopoietin-2; BCVA, best-corrected visual acuity; CST, central subfield thickness; DME, diabetic macular edema; nAMD, neovascular age-related macular degeneration; Q12W, every 12 weeks; Q16W, every 16 weeks.[5,10,11]

DISCUSSION

The data presented evidence of the efficacy and longevity of faricimab in the treatment of neovascular age-related macular degeneration (nAMD) and diabetic macular edema (DME) intravitreal therapy. It has the most notable clinical claim for non-inferior visual efficacy possibly with longer treatment intervals. In TENAYA and LUCERNE, the vision-preserving efficacy of faricimab was similar to aflibercept with up to 16-week protocol-defined treatment intervals and then treatment and extend dosing. The same trend was seen with the DME treatment in YOSEMITE and RHINE where the number of injections was decreased, but the visual outcome was preserved. This difference is important because any treatment that results in fewer injections and a relatively small difference in BCVA (BCVA - MAV) has the potential to be of significant value even though the difference in mean VA is near zero.[6-9] Biological explanation is logical. Both VEGF-A inhibition and angiopoietin-2 inhibition can decrease vascular permeability and angiogenic signaling, and the latter can also stabilize endothelial cells via the Tie2 pathway. Based on pharmacodynamic modelling, there is an extended period of suppression of angiopoietin-2, and the clinical trials demonstrate that this pharmacology can equate to an extended dosing period for many patients. It is not appropriate to assume that the dual mechanism always results in better vision in patients, however. Irreversible photoreceptor loss and fibrosis, atrophy, macular ischemia, cataract and other comorbidities are factors that affect visual acuity. Therefore, anatomic drying is an important, but incomplete surrogate for functional benefit.[4-7] The DME results demonstrate the best anatomic signal. The mean reductions in central subfield thickness were more and intraretinal fluid more commonly absent with faricimab than with aflibercept in the reported analyses at 2 years. This information could potentially be clinically beneficial in eyes with ongoing fluid but should be considered when making treatment decisions along with visual function, chronicity of fluid, location of fluid, diabetic control, ischemia, and overtreatment risk. The presence of a few, stable subretinal fluid pockets may not be as clinically relevant as the intraretinal cysts highlighted by trial-specified thresholds, and thresholds cannot be applied mechanistically to all patients.[7,9] Clinically meaningful treatment burden benefit too. Multiple injections are logistically, financially and psychologically burdensome and can decrease adherence in elderly or those with mobility issues. The number of injections required was about 10–11 in the DME arm, compared with 14–15 in the fixed arms every 8 weeks over the 2-year duration of the trials. At 2 years, over half of the patients in nAMD receiving faricimab treatment had received the drug in every 16 weeks. However, the numbers are those that are protocol-based trials. In practice, there are many different treat and extend comparator regimens that are started every 8 weeks, as was the case for the aflibercept comparator in the trial. So, the benefit of the injection over the new real-world aflibercept treat and extend therapy cannot be assumed based on these trials.[3,8,9] The safety profile is generally similar to that of aflibercept in controlled studies and is generally good. The key risks of intravitreal therapy in general are endophthalmitis, retinal detachment, transient intraocular-pressure elevation, inflammation and possible arterial thromboembolic events. Randomized trials are under-powered when looking for very rare complications because of the low frequency of occurrence. Further pharmacovigilance is therefore required, especially when used in previously treated eyes, in patients with a history of inflammation and different indications with different risk profiles.[6-9,11] Scale up to the real world and get a little nuance. TRUCKEE comprised a high percentage of previously treated nAMD eyes, and showed a high degree of anatomic improvement and a stable or slightly better vision. This implies that faricimab could be beneficial as a treatment option where there is still a persisting fluid problem and/or a short treatment interval. However, switching studies are observational studies and are susceptible to regression to the mean – when patients are switched, they may be so because they are experiencing disease activity, and subsequent improvement may be due to the natural response to any effective injection. Furthermore, a lack of central subfield-thickness reduction in the early stages of the trial does not mean faricimab will do the same in the long term or improve visual outcomes.[10] There are a number of caveats which limit the confidence in the overall conclusions. First, there were many investigators who had financial relationships and the pivotal trials had been sponsored by the manufacturer. This doesn't refute the findings, but it's a reminder of the need for independent replication. Second, the principal nAMD trials included treatment-naïve patients, which decreases the applicability to eyes that fail to respond to or have had prior injections. Third, the evidence against aflibercept 8 mg and the evidence against the contemporary flexible treat and extend strategies are not as extensive as the evidence against aflibercept 2 mg every 8 weeks. Fourth, with network meta-analyses, the assumptions of transitivity for the network should be considered and might not be fully validated in retinal trials. Lastly, the treatment interval is not a predetermined characteristic of the molecule as it will be affected by the disease activity, interpretation of imaging, adherence, clinician practice and patient-specific anatomy.[6,8,12,13] Independent comparative-effectiveness studies, long-term outcome beyond extension periods, patient reported treatment burden, cost-effectiveness and biomarkers predicting every-16-week responses should all be a priority area for future research. Treatment-naïve and switch populations should also be separated in studies and outcomes should be reported according to standardised definitions of fluid, disease recurrence and interval failure and discontinuation. Comparisons with more recent anti-VEGF agents, such as newer high dose or long-acting anti-VEGF agents would further elucidate its role in the evolving treatment paradigm.[8,9,12,13]

CONCLUSION

Intravitreal faricimab Is a science-driven and clinically proven treatment for nAMD and DME. The pivotal evidence shows the visual efficacy at least non-inferior to aflibercept 2.0 mg Q8W and offers significant anatomic advantages for DME, along with a large treatment potential for Q12W or Q16W dosing for patients that are well-suited. It is durable and reduces the burden of treatment which is its primary benefit, rather than improving vision in everyone. Safety is similar to aflibercept in clinical trials, with similar, but more uncommon, inflammatory and vascular risks that need to be monitored. In practice, faricimab should be considered a disease-activity guided treatment for which the benefits are likely to be highest when there is visual or anatomic control that allows for extended dosing without compromising.

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