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Effect of Faricimab on Visual Acuity and Retinal Structure in Neovascular Age-Related Macular Degeneration Previously Treated With Anti-VEGF Therapy

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Abstract

Purpose: To evaluate the effects of switching from traditional anti-vascular endothelial growth factor (anti-VEGF) therapies to faricimab, a bispecific monoclonal antibody that targets both VEGF and angiopoietin-2, on eyes with neovascular age-related macular degeneration (nAMD). **Methods:** This study retrospectively reviewed patients with nAMD who were previously treated with bevacizumab, ranibizumab, or aflibercept and then switched to faricimab. We compared injection frequency and visual acuity (VA) during the time period before faricimab initiation (1 year prior) and after initiation (6-12 months after). Optical coherence tomography images were analyzed from initiation to final follow-up (6-12 months after initiation). **Results:** We evaluated 84 eyes of 68 patients. Following faricimab initiation, eyes had a reduced mean ± SE central macular thickness (CMT) (282.3 ± 16.2 μm preinitiation vs 244.8 ± 14.3 μm postinitiation; $P < .01$). Annual injection frequency increased from 7.73 ± 0.33 to 8.66 ± 0.28 injections ($P < .001$). VA did not change significantly during the year before faricimab initiation ($P = .539$) but decreased after initiation (from 0.56 ± 0.05 logMAR to 0.66 ± 0.06 logMAR; $P < .01$). Four eyes developed macular atrophy following faricimab initiation ($P < .01$). **Conclusions:** Eyes with nAMD that were previously treated with anti-VEGF therapy and later switched to faricimab showed reduced CMT; however, some patients had increased injection frequency, decreased VA, and macular atrophy. These findings should be explored further using larger datasets.

Keywords

neovascular age-related macular degeneration, faricimab, anti-VEGF

Introduction

Age-related macular degeneration (AMD) is the leading cause of central vision loss in patients older than 55 years in developed countries, and its incidence is projected to rise by 2040.[1,2] The development of AMD is thought to be multifactorial, involving a complex interplay of age and genetic, environmental, and lifestyle factors,[2–4] and manifests in 1 of 2 forms: dry and wet (neovascular; nAMD). Although nAMD accounts for only about 10% of all AMD cases, it is responsible for 90% of the severe vision loss associated with the disease.[5]

Therapies for nAMD have traditionally focused on the inhibition of anti-vascular endothelial growth factor (anti-VEGF), a key driver of angiogenesis and increased vascular permeability.[2] Agents such as bevacizumab, ranibizumab, and aflibercept have become a cornerstone of therapy, with clinical trials and real-world evidence demonstrating significant improvements in morphology and visual acuity (VA).[2]

Faricimab is a bispecific monoclonal antibody that targets both VEGF and angiopoietin-2 (Ang2) and was approved by the US Food and Drug Administration for the treatment of nAMD in 2023.[6] In clinical trials, faricimab was shown to be noninferior to other anti-VEGF agents for both VA and central macular thickness (CMT).[6] However, to the best of our knowledge, few studies have explored outcomes in patients who transitioned from an anti-VEGF medication to faricimab.

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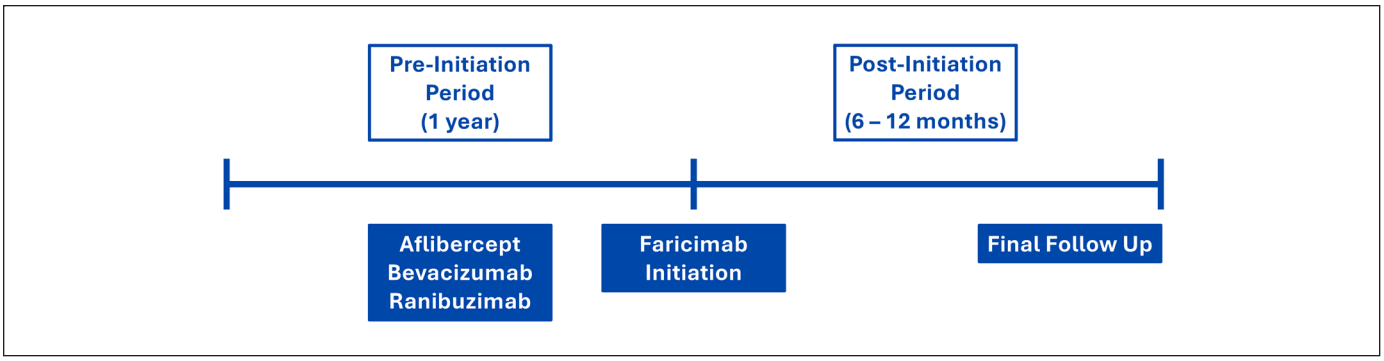

Figure 1. Study design. Patients included in this study received at least 1 year of aflibercept, bevacizumab, or ranibizumab during the preinitiation period. Patients switched to faricimab and had final follow-up at 6 to 12 months after faricimab initiation.

This study seeks to address this gap by evaluating the clinical outcomes of patients with nAMD who were previously treated with bevacizumab, ranibizumab, or aflibercept and, due to limited treatment response, were switched to faricimab therapy. We assessed changes in injection burden (frequency), VA, and macular structural changes. By exploring the outcomes of patients who transitioned to faricimab, the study aims to inform clinical decision-making and highlight areas for future research.

Methods

Study Population

Because this was a retrospective study, the Duke University Health System Institutional Review Board deemed it exempt from review, and informed consent was not obtained. Patients diagnosed with nAMD who were previously treated with bevacizumab, ranibizumab, or aflibercept, and then switched to treatment with faricimab, were identified via electronic medical records.

Epic SlicerDicer (PIXOTEC) was used to identify patients seen at Duke Eye Center between January 2012 and April 2023 given the following search criteria: "age-related exudative macular degeneration of left eye," "age-related exudative macular degeneration of right eye," "age-related macular degeneration," "aflibercept" followed by "faricimab," "bevacizumab" followed by "faricimab," and "ranibizumab" followed by "faricimab."

Retrospective Chart Review

All patients identified through Epic SlicerDicer were manually reviewed individually by 4 authors (N.S., W.Z., A.C., M.C.). Demographic information, injection data, and VA were obtained from patient charts.

Demographic data included age, sex, and race or ethnicity. The number of bevacizumab, ranibizumab, and aflibercept injections during the preinitiation period, defined as 1 year leading up to faricimab initiation, was obtained (Figure 1). Patients with less than 1 year of anti-VEGF therapy prior to faricimab initiation were excluded. The number of faricimab injections during the postinitiation period, including the initiation dose, was also obtained. This postinitiation period was defined as the 6 to 12 months following faricimab initiation. The study excluded eyes that received less than 6 months of faricimab therapy or less than 1 year of another anti-VEGF injection medication, as well as any eyes that were switched back to an injection medication other than faricimab.

To characterize the injection intervals during the preinitiation and postinitiation periods, all injection dates were obtained from electronic medical records. The number of weeks between injection dates was obtained, and a mean injection interval was calculated for the preinitiation and postinitiation periods for each eye by summing the interval durations and dividing that number by the number of injections. The number of patients who received a loading dose of faricimab was assessed by evaluating the first 3 injection intervals. Patients were deemed to have had loading doses if all 3 of these intervals were 4 or 5 weeks long and were followed by increases in injection interval for subsequent doses. Furthermore, the injection frequency was calculated for the preinitiation and postinitiation periods by multiplying the total number of injections by 12 (for total number of months) and dividing this number by the follow-up time (in months).

Snellen VA measurements were collected at 3 timepoints: at the start of the preinitiation period, time of faricimab initiation, and final follow-up. Eyes without VA measurements within 1 month of each of these timepoints and eyes with inconsistent methods of VA measurement (with or without correction) across timepoints were excluded. Snellen measurements were converted to logMAR units for analysis.

Optical Coherence Tomography Analysis

Optical coherence tomography (OCT) images (Heidelberg Engineering) for each eye were obtained at faricimab initiation and final follow-up. One author, a retina specialist (K.S.), assessed the images for the presence of, and changes in, intraretinal fluid (IRF), subretinal fluid (SRF), and subretinal hyperreflective material (SHRM), and for macular atrophy. Macular

Table 1. Patient Demographics.

Characteristic	Value
Total eyes (n)	84
Total patients (n)	68
Mean age (y) ± SD	81.9 ± 7.8
Female sex, n (%)	39 (57.4)
Right eye, n (%)	43 (51.2)
Race, n (%)	
White	64 (94.1)
Black/African American	1 (1.5)
Asian	3 (4.4)
Mean time to final follow-up after faricimab initiation (mo) ± SD	10.98 ± 1.6

Table 2. Anti-VEGF Medications Prior to Faricimab Initiation.

Number of Anti-VEGF Medications	Number of Eyes (%)
One	76 (90.5)
Aflibercept	54 (71.1)
Bevacizumab	9 (11.8)
Ranibizumab	13 (17.1)
Two	6 (7.1)
Aflibercept and bevacizumab	6 (100)
Aflibercept and ranibizumab	0 (0)
Bevacizumab and ranibizumab	0 (0)
Three	2 (2.4)

Abbreviation: anti-VEGF, anti-vascular endothelial growth factor.

atrophy is defined as complete retinal pigment epithelium and outer retinal atrophy as outlined by the Classification of Atrophy Meetings.[7] CMT was measured by 1 author (K.S.) using the Spectralis system caliper (Heidelberg Engineering).

Data Analysis

Data analysis was performed using SPSS Statistics for Windows (version 29.0, IBM). Paired sample *t* tests and Wilcoxon signed rank tests were used to assess changes in injection frequency, VA, and CMT, with statistical significance defined as $P < .05$. Eyes that received loading doses of faricimab and 1 eye that received multiple aflibercept and ranibizumab doses at very short intervals (2-week intervals, sometimes 2 injections in the same week) during the preinitiation period were excluded from analysis of injection frequency. A Pearson χ^2 test was performed to evaluate changes in the frequency of macular atrophy in the study cohort. Mean values are ± SE.

Results

In total, 84 eyes of 68 patients met the inclusion criteria for this study, and their demographic data are presented in Table 1. The mean patient age was 81.9 years, and the mean time to final follow-up after faricimab initiation was 11 months. During the preinitiation period, 73.8% (62/84) had received aflibercept injections, 13.1% (11/84) received bevacizumab injections, and 17.9% (15/84) received ranibizumab injections. Most eyes received only 1 type of anti-VEGF medication prior to faricimab (n = 76 [90.5%]), with the majority of eyes receiving only aflibercept (n = 54 [71.1%]) (Table 2). Six eyes received aflibercept and bevacizumab (7.1%), and 2 eyes received all 3 medications prior to faricimab initiation (2.4%).

The distribution of mean injection intervals for the preinitiation and postinitiation periods is shown in Figure 2. Nine eyes (10.7%) received loading doses upon faricimab initiation. These eyes, along with 1 eye that had short injection intervals (mean, 2.23 weeks) and received multiple injections within the same week, were excluded from injection interval and frequency analysis. Most eyes during the preinitiation period had

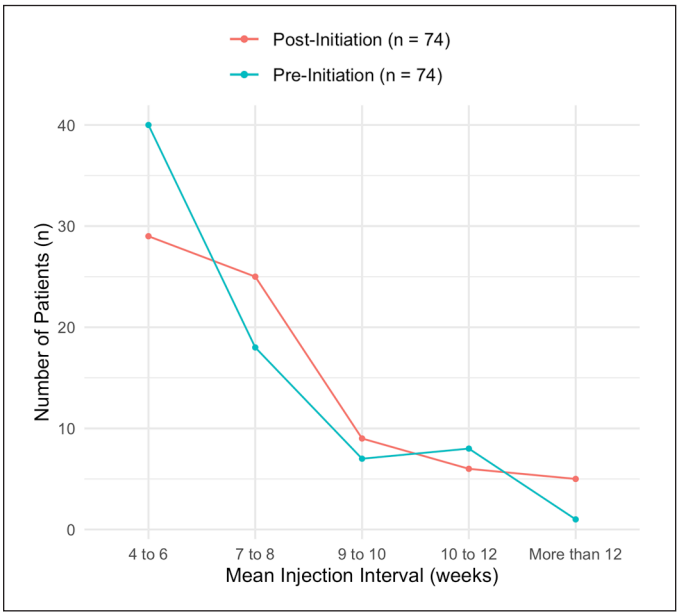

Figure 2. Frequency of mean injection intervals during the preinitiation and postinitiation periods. Loading dose eyes (n = 9) and 1 eye with a mean injection interval of 2.23 weeks with multiple injections per week were excluded from this analysis.

a mean injection interval of 4 to 6 weeks (n = 40 [54.1%]), with 18 eyes (24.3%) in the 7- to 8-week range, 7 eyes (9.46%) in the 9- to 10-week range, 8 eyes (10.8%) in the 10- to 12-week range, and 1 eye (1.35%) with a mean interval of more than 12 weeks. In the postinitiation period, most eyes also had a mean injection interval of 4 to 6 weeks (n = 29 [39.2%]), with 25 eyes (33.8%) in the 7- to 8-week range, 9 eyes (12.2%) in the 9- to 10-week range, 6 eyes (8.11%) in the 10- to 12-week range, and 5 eyes (6.76%) with a mean interval of more than 12 weeks. Fifty eyes (67.6%) had an increase in their mean injection interval from the preinitiation to postinitiation periods, while 23 eyes (31.1%) had a decrease in their mean interval, and 1 eye (1.35%) had no change in mean interval. Mean injection frequency increased from 7.73 ± 0.33 injections per year to 8.66 ± 0.28 injections per follow-up period after faricimab initiation ($P < .01$) (Figure 3).

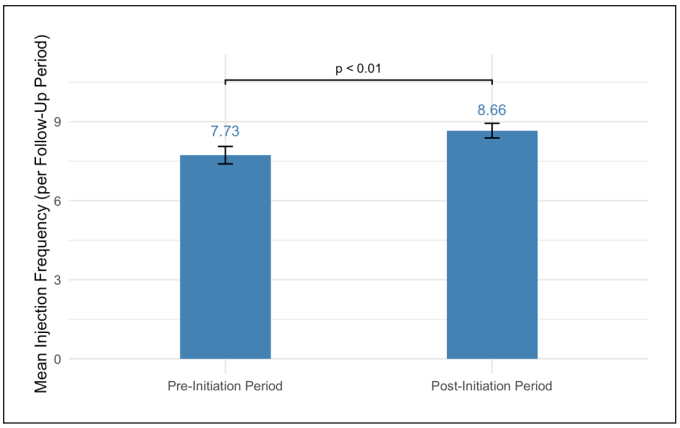

Figure 3. Mean injection frequency per follow-up period, during the preinitiation period compared with the postinitiation period. Loading dose eyes (n = 9) and 1 eye with a mean injection interval of 2.23 weeks with multiple injections per week were excluded from this analysis. Statistical significance was determined using a paired sample *t* test. Error bars represent SEs.

Mean VA marginally but significantly worsened during the postinitiation period, from 0.56 ± 0.05 logMAR to 0.66 ± 0.06 logMAR ($P < .01$), and during the entire study period, from 0.53 ± 0.06 logMAR to 0.66 ± 0.06 logMAR ($P < .05$) (Figure 4). The mean change in VA from 0.53 ± 0.06 logMAR at the beginning of preinitiation to 0.56 ± 0.05 logMAR at the time of faricimab initiation was not statistically significant ($P = .539$).

Of the 84 eyes, 67 had OCT images available through electronic medical records. At faricimab initiation, 34.3% (23/67) had IRF only, 28.4% (19/67) had SRF only, and 19.4% (13/67) had both IRF and SRF (Table 3); 26 (38.8%) of 67 patients had SHRM. Of the eyes with IRF at faricimab initiation, 50% (18/36) had complete resolution of their IRF, 30.6% (11/36) had decreased IRF, and 13.9% (5/36) had increased IRF at final follow-up. For more than 80% of eyes (26/32) with SRF at initiation, their SRF resolved by final follow-up. Only 3.1% (1/32) had decreased SRF, and 12.5% (4/32) had increased SRF at final follow-up. In 38.5% of eyes with SHRM (10/26), the case resolved postinitiation, and in 34.6% (9/26), SHRM decreased after initiation, with only 7.7% (2/26) developing increased SHRM.

Mean CMT significantly decreased from 282.3 ± 16.2 μm at faricimab initiation to 244.8 ± 14.3 μm at final follow-up ($P < .01$) (Figure 5). Furthermore, 15 eyes of 15 separate patients were found to have macular atrophy at faricimab initiation (Figure 6). By final follow-up, 19 eyes of 18 patients had macular atrophy ($P < .01$). A representative OCT image for those patients who developed atrophy is shown in Figure 7.

Conclusions

This study provides insights into the real-world effects of faricimab on patients with nAMD who were previously treated with other available anti-VEGF therapies. Our findings demonstrate that switching to faricimab resulted in a significant reduction of CMT, with many patients having a resolution or improvement of IRF, SRF, and SHRM following initiation. However, the increased injection frequency, worsened VA, and development of macular atrophy in a portion of patients raise important clinical considerations.

Reductions in CMT with faricimab therapy are consistent with prior trials.[8–10] However, this retrospective study showed an increase in injection frequency, which differs from a 2022 study by Rush and Rush,[8] which found a decrease in injection frequency when patients were switched from aflibercept to faricimab. Rush and Rush performed their study at the patient level, while the present study was performed at the eye level; the Rush and Rush sample size was comparable to that of our study.[8] Leung et al[11] demonstrated improved VA and central subfield thickness (CST) after switching to faricimab for treatment-resistant AMD, but had only a 3-month follow-up period. Their study also reported an improvement in mean VA of approximately 0.06 logMAR, compared with an approximate 0.1 logMAR decline in VA in the present study. Tamiya et al[12] found no significant change in VA in patients with aflibercept-refractory AMD 2 months after switching to faricimab. Moreover, 2-year results from the TENAYA and LUCERNE trials showed successful extension of faricimab dosing and improved VA, and other studies have corroborated these findings.[13,14]

Potential explanations for these discrepancies, besides the larger sample sizes in the case of LUCERNE and TENAYA, include patient disease severity (in our study, more severe and refractory cases were switched to faricimab), the need for more aggressive management to stabilize or improve retinal pathology, and time to follow-up. The clinical significance of the increased injection frequency and slight decline in VA is multifaceted. Further research and real-world studies should explore the cohort of patients receiving faricimab at the authors' institution to elucidate potential barriers to therapy while maximizing faricimab's therapeutic potential.

This study also noted the development of macular atrophy in 4 eyes. To the authors' knowledge, there is no evidence or literature suggesting a causal relationship between faricimab and macular atrophy development. Macular atrophy has been reported as a complication of anti-VEGF therapy in patients with nAMD and is correlated with declines in VA.[15,16] Clinical trials have reported incidences of macular atrophy between 30% and 40% in eyes treated with anti-VEGF medications, with 1 study reporting a prevalence of 74% in their cohort.[16]Although only 5.88% of eyes (4/68) in this study developed atrophy, macular atrophy typically develops with long-term use of anti-VEGF injections, with trials reporting data from at least 5 years of therapy.[16] Although the mechanism of atrophy development with anti-VEGF therapy is unclear, risk factors such as increased age, specific nAMD phenotype, and choroidal thinning have been reported.[16] A larger study with an extended follow-up period and greater number of patients is needed to better understand the relationship between faricimab (and other injectables) and macular atrophy given its role as a cause of irreversible vision loss.[16]

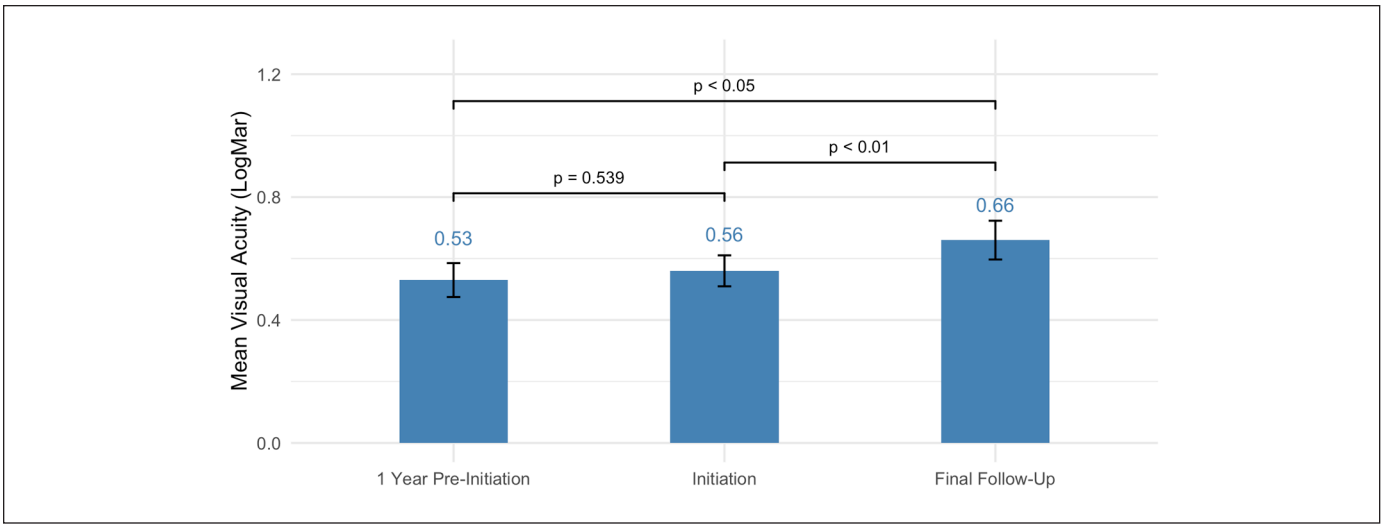

Figure 4. Bar graph showing the mean visual acuity during the preinitiation period compared with the postinitiation period. Statistical significance was determined using a paired sample *t* test. Error bars represent SEs.

Table 3. Frequencies of Retinal Features Detected on Optical Coherence Tomography Analysis.

Structural Pathology	Number (%)
At faricimab initiation	
IRF only	23 (34.3)
SRF only	19 (28.4)
IRF and SRF	13 (19.4)
SHRM	26 (38.8)
Pathology at final follow-up	
IRF	
Decreased IRF	11 (30.6)
Resolution of IRF	18 (50.0)
Increased IRF	5 (13.9)
SRF	1 (3.1)
Decreased SRF	
Resolution of SRF	26 (81.3)
Increased SRF	4 (12.5)
SHRM	
Decreased SHRM	9 (34.6)
Resolution of SHRM	10 (38.5)
Increased SHRM	2 (7.7)
Qualitative final evaluation	
Total resolution	26 (11.5)
Partial improvement	13 (5.7)
No change	7 (3.1)
Worsening	7 (3.1)

Abbreviations: IRF, intraretinal fluid; SRF, subretinal fluid; SHRM, subretinal hyperreflective material.

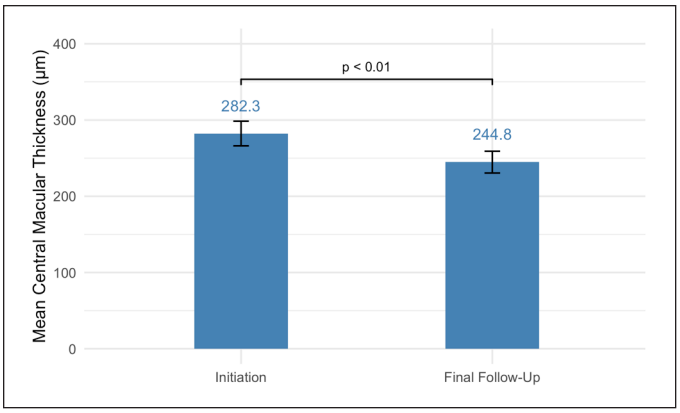

Figure 5. Mean central macular thickness at faricimab initiation compared with final follow-up. Statistical significance was determined using a paired sample *t* test. Error bars represent SEs.

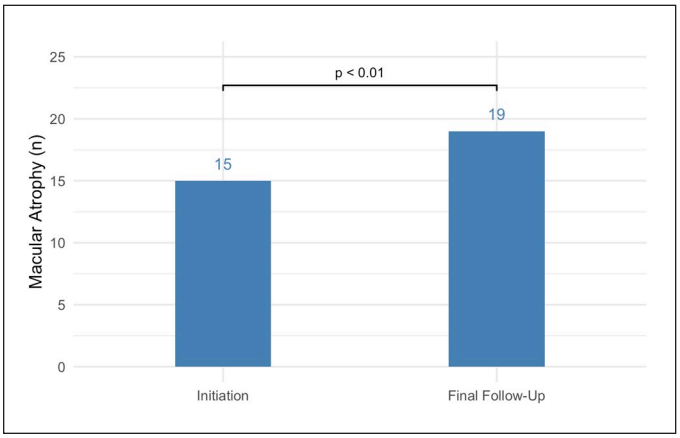

Figure 6. Number of eyes with macular atrophy at faricimab initiation compared with final follow-up. Statistical significance was determined using a Pearson χ^2 test. Error bars represent SEs.

There is ongoing discussion on whether SRF may protect against the development of macular atrophy in eyes with nAMD treated with anti-VEGF therapy.[17–20] One study observed a low incidence of macular atrophy in eyes with nAMD and SRF that were treated with anti-VEGF therapy.[20] Another study showed

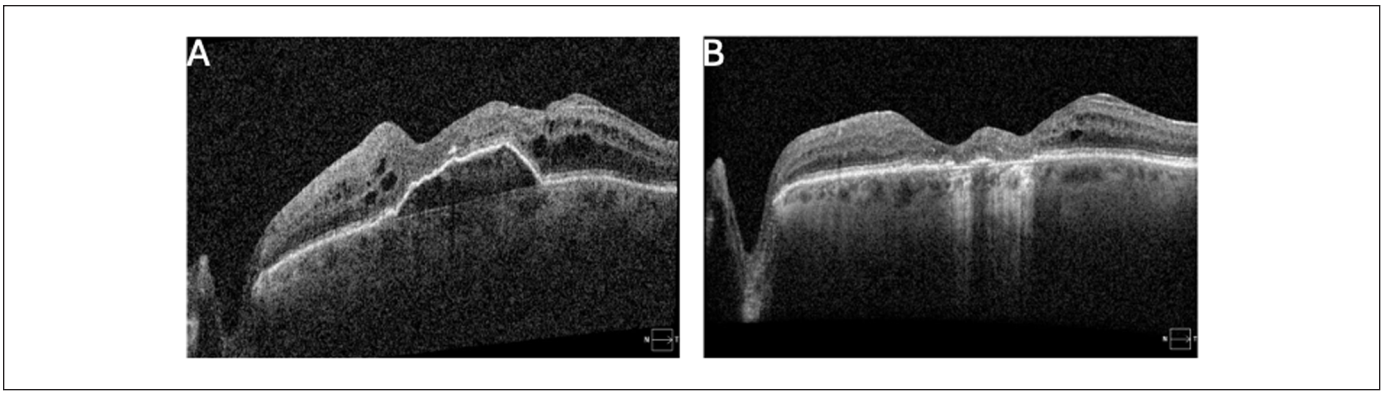

Figure 7. Representative optical coherence tomography image of a retina at (A) faricimab initiation and (B) final follow-up for patients who developed macular atrophy.

that best-corrected VA outcomes showed the greatest improvement when retinal fluid was completely resolved.[21] A study by Tamiya et al[12] found that more than 50% of aflibercept-refractory patients experienced SRF resolution after just 1 dose of faricimab, but they do not comment on the development of macular atrophy in their cohort. In the present study, more than 80% of patients with SRF at initiation had their fluid resolved by final follow-up, with 4 eyes developing macular atrophy. Only 1 patient who developed macular atrophy was observed to have SRF on OCT imaging at baseline. It is currently unclear what degree of macular atrophy in the 4 eyes can be attributed to the initiation of faricimab treatment. More work is needed to define the role of SRF in the development of macular atrophy in the setting of nAMD treated with faricimab.

This study has several limitations. As it is a retrospective study, there was heterogeneity in the preinitiation anti-VEGF treatment course and treatment time intervals. Further work should be done to understand how changes in VA, CMT, and OCT structural findings correlate with the type of preinitiation anti-VEGF treatment as well as preinitiation injection frequency. For instance, 1 study showed a greater effect power in improvement of morphological changes after faricimab in patients who previously received ranibizumab vs aflibercept for recalcitrant nAMD.[22] The heterogeneity in the type of anti-VEGF agents administered for varying durations before switching to faricimab may influence observed outcomes, making it challenging to attribute changes solely to faricimab. Additionally, 1 study observed that injection frequency did not influence the rate of change in distance VA in patients undergoing long-term anti-VEGF therapy.[23] While this study focused on patients who had undergone faricimab treatment over 6 to 12 months, more work is needed to understand the long-term effects of faricimab. Further investigation can be done to understand how changes in VA, CMT, and OCT structural findings correlate with the type of preinitiation anti-VEGF treatment, as well as preinitiation injection frequency.

In summary, this study examines retinal structural and functional outcomes, highlighting faricimab's potential as an injection therapy for nAMD. While this study does not fully characterize the long-term effects on VA and OCT structural findings, further investigations will help elucidate the full spectrum of safety and adverse events associated with faricimab's use.

Ethical Approval

This study was deemed exempt from review by the Duke University Health System Institutional Review Board because it was categorized as secondary research.

Statement of Informed Consent

No human subjects were involved; therefore, institutional review board approval was not required.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of the article.

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Data Availability Statement

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

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