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Case Report

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Bispecific Antibody Faricimab in the Treatment of AMD: Case Report

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Introduction

Age-related macular degeneration (AMD) is one of the leading causes of central visual loss in adults over 55 years old, with a substantial impact on quality of life due to its increasing prevalence in aging populations. The condition is classified into two main forms: non-exudative ("dry"), characterized by the presence of drusen and eventual geographic atrophy, and exudative ("wet"), marked by pathological choroidal neovascularization that leads to the formation of fragile vessels beneath the retinal pigment epithelium, resulting in exudation, subretinal hemorrhage, and subfoveal fibrosis. Although the dry form accounts for approximately 80–85% of cases, the wet form causes most cases of legal blindness due to its aggressive course and rapid visual acuity deterioration. In 2020, it was estimated that about 190 million individuals worldwide were affected by AMD, with projections reaching 288 million by 2040, particularly due to global population aging [1,2].

In Brazil, epidemiological data indicate that approximately 1% of individuals over 60 years have exudative AMD, representing a significant cause of legal blindness in this age group [3]. Established risk factors for AMD development and progression include advanced age, genetic predisposition (polymorphisms in complement pathway genes such as *CFH* and *ARMS2*), active smoking, diets high in saturated fats, systemic hypertension, obesity, and chronic exposure to ultraviolet light.

The pathogenesis of wet AMD involves dysregulation of the angiogenic pathway, especially due to the overexpression of vascular endothelial growth factor A (VEGF-A), which promotes the proliferation of abnormal choroidal vessels. These neovessels invade the subretinal space, leading to exudation and hemorrhages that compromise foveal structure. Furthermore, angiopoietin-2 (Ang-2) plays a critical role in vascular instability; its elevation correlates with increased permeability and inflammatory cell recruitment, exacerbating macular edema and favoring subfoveal hemorrhage. Thus, exclusive VEGF-A blockade, although effective, leaves the Ang-2 pathway uncontrolled, which may explain the need for frequent maintenance and recurrences in some patients even after intensive anti-VEGF regimens.

Keywords: Age-related macular degeneration; Faricimab; VEGF-A; Angiopoietin-2; Choroidal neovascularization

Objectives

To describe a case report and the clinical, anatomical, and functional evolution of a patient with exudative age-related macular degeneration refractory to conventional treatment with aflibercept, after therapeutic switch to faricimab, emphasizing the impact of bispecific VEGF-A and Ang-2 blockade on intraretinal fluid and subfoveal hemorrhage resolution, as well as improvements in visual acuity and treatment tolerability.

Case Report

A 61-year-old male engineer, undergoing treatment for AMD for three years with aflibercept, presented in October 2023 with complaints of visual distortion and difficulty reading in the left eye (LE), which progressed to sudden vision loss within seven days. Uncorrected visual acuity (VA) was 20/20 in the right eye (RE) and 20/80 in the LE. Biomicroscopy revealed calm conjunctiva, clear cornea and lens in both eyes. Fundoscopy revealed drusen in the RE and significant subretinal hemorrhage in the LE. Intraocular pressure was 14 mmHg in both eyes. Optical coherence tomography (OCT) revealed massive subretinal hemorrhage in the LE. Given this scenario, a therapeutic switch was made to intravitreal Faricimab 6 mg, with six monthly injections.

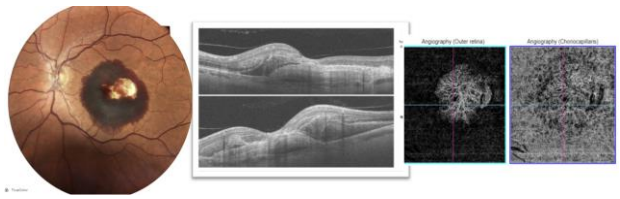

Figure 1: OCT angiography of the LE showing BCVA 20/800 and CST 425 µm.

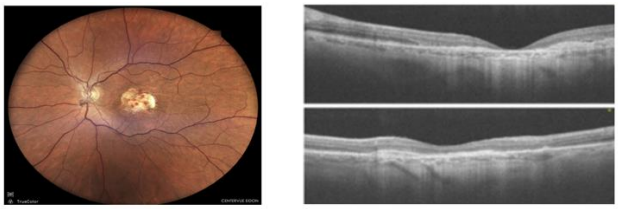

Figure 2: OCT angiography of the LE showing BCVA 20/100 and CST 125 µm.

Discussion

Refractory exudative AMD to conventional monotherapy with anti-VEGF represents a significant clinical challenge. Although VEGF-A blockade is effective for most patients, approximately 15–20% maintain persistent intraretinal or subretinal fluid, and some develop recurrent subfoveal hemorrhages even under intensive monthly regimens. In such cases, Ang-2 an endothelial junction destabilizer and pro-inflammatory mediator may remain active when using VEGF-A inhibitors alone, explaining incomplete macular edema control and a higher risk of recurrence.

Faricimab, a bispecific antibody targeting both VEGF-A and Ang-2, has shown in clinical trials that, in addition to visual gains comparable to those of anti-VEGF monotherapies, it allows extended injection intervals, reducing treatment burden. The TENAYA and LUCERNE trials demonstrated that up to 80% of patients could be treated every 12–16 weeks, while only 45% required 8-week intervals, without a significant increase in adverse events [2,4].

In this case, the switch to faricimab after aflibercept refractoriness resulted in gradual resolution of fluid and hemorrhage, with functional improvement from 20/80 to 20/40 over six months, suggesting that additional Ang-2 inhibition was crucial for anatomical stabilization. Although the initial 2mune2t dosing deviated from the extended regimen typically recommended for treatment-naïve patients, it allowed a faster response in the 2mune2te of active hemorrhage. After fluid and hemorrhage control, injection intervals may be gradually extended, reducing treatment burden. In terms of safety, there were no episodes of endophthalmitis, significant intraocular inflammation, or 2mune2tente intraocular hypertension. These findings are 2 mune 2 tente with previous reports indicating similar safety profiles between faricimab and anti-VEGF monotherapies, although clinical vigilance for potential inflammatory responses remains recommended as the 2mune implications of dual blockade are still under evaluation.

Conclusion

The clinical progression of this patient refractory to aflibercept, with gradual resolution of intraretinal fluid and subfoveal hemorrhage, and functional improvement from 20/80 to 20/40 over six months, demonstrates that bispecific blockade of VEGF-A and Ang-2 by faricimab may overcome limitations of anti-VEGF monotherapy. By stabilizing endothelial junctions and reducing vascular permeability more comprehensively, faricimab not only effectively controls edema and hemorrhage but also allows for intravitreal injection spacing in suitable cases, reducing treatment burden and improving adherence among elderly patients. Despite the high unit cost, the prospect of lower injection frequency and decreased recurrence risk suggests a favorable long-term cost-effectiveness. Further studies, especially in refractory patients with different neovascular profiles, as well as cost-effectiveness analyses in regional healthcare systems, are necessary to consolidate its role as a first-line therapeutic alternative in exudative AMD.

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