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Intravitreal Faricimab for Uveitic Macular Edema in Vogt-Koyanagi-Harada Syndrome: A Case Report

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Abstract

Vogt-Koyanagi-Harada (VKH) syndrome is a multisystem autoimmune disease in which uveitic macular edema (UME) represents a major cause of vision loss during chronic stages. We present the case of a 32-year-old Hispanic woman with a 10-year history of chronic VKH who presented with acute blurred vision, tinnitus, and headaches. Clinical evaluation revealed localized cystoid macular edema in the left eye with a central subfield thickness (CST) of 325 μm, loss of foveal depression, and best-corrected visual acuity (BCVA) of 20/150 in the left eye. Fluorescein angiography and anterior segment examination confirmed active inflammation localized strictly to the left macula without acute panuveitic flare. A single intravitreal injection of Faricimab (6.0 mg/0.05 mL) was administered on November 4, 2025, concurrently with a low-frequency topical corticosteroid regimen (prednisolone acetate 1.0%/phenylephrine 0.12%). One month post-injection, complete resolution of cystoid macular edema was observed, with CST decreasing to 163 μm, restoration of the normal foveal contour, and BCVA improvement to 20/50 in the left eye. No intraocular inflammation, intraocular pressure elevation, or adverse events occurred. While rapid anatomical and functional improvement was observed following faricimab administration with concurrent topical corticosteroid therapy, this uncontrolled observation cannot establish faricimab's independent efficacy or steroid-sparing capacity. Dual VEGF/Ang-2 inhibition represents a promising adjunct for localized uveitic macular edema, but prospective controlled studies are required to define its independent role.

Keywords

Vogt-Koyanagi-Harada Syndrome, Faricimab, Uveitic Macular Edema, Angiopoietin-2, Corticosteroids, Dual Inhibition

1. Introduction

Vogt-Koyanagi-Harada (VKH) syndrome is a chronic, multisystemic autoimmune disorder characterized by bilateral granulomatous panuveitis directed against melanocyte-associated antigens in the eye, inner ear, meninges, and skin [1] [2]. Predominantly affecting pigmented populations, the clinical course typically evolves through four distinct phases: prodromal, acute uveitic, convalescent, and chronic recurrent [3] [4]. During chronic stages, persistent or recurrent uveitic macular edema (UME) remains one of the leading causes of irreversible central visual impairment, driven by sustained breakdown of the blood-retinal barrier [3] [4].

The pathophysiology of UME involves hyperpermeability of retinal capillaries mediated by vascular endothelial growth factor A (VEGF-A) and destabilization of endothelial cell-cell junctions by angiopoietin-2 (Ang-2) [5] [6]. Faricimab is a novel bispecific monoclonal antibody designed for simultaneous, high-affinity dual inhibition of both VEGF-A and Ang-2, offering synergistic vascular stabilization and anti-inflammatory properties [6] [7]. While intravitreal faricimab is well established for neovascular age-related macular degeneration (nAMD) and diabetic macular edema (DME) [8] [9], and has recently been investigated for retinal vein occlusion (RVO) [7], clinical evidence regarding its application in uveitic populations remains limited [6].

In localized reactivations of chronic VKH, systemic immunosuppression with high-dose corticosteroids or disease-modifying antirheumatic drugs (DMARDs) may carry significant systemic toxicity risks [2] [3]. Targeted local therapies offer an attractive alternative; however, evaluating novel local agents requires careful interpretation when topical anti-inflammatory treatments are administered concurrently. Herein, we report the clinical course of a patient with chronic VKH who experienced rapid anatomical clearance of localized UME following intravitreal faricimab administration alongside topical corticosteroid therapy, and we discuss the diagnostic, therapeutic, and methodological considerations surrounding dual-pathway inhibition in uveitis.

2. Case Report

2.1. Patient History and Clinical Presentation

A 32-year-old Hispanic woman with a 10-year history of bilateral chronic VKH syndrome presented in September 2025 complaining of acute bilateral blurred vision, persistent daily headaches, and tinnitus following a period of severe work-related psychological stress. Her family history was negative for autoimmune, rheumatologic, or ocular diseases.

At initial presentation in 2015 (age 22), she had experienced bilateral diffuse granulomatous panuveitis with extensive serous retinal detachments, severe hypoacusis, and neurological symptoms. During the subsequent convalescent phase, she developed documented integumentary manifestations, including poliosis, vitiligo, and areas of cutaneous depigmentation with subsequent partial repigmen-

tation. Under the Revised Diagnostic Criteria (2001), her clinical course fulfilled the diagnostic criteria for Complete VKH Syndrome based on bilateral ocular involvement, neurological/auditory signs, and documented integumentary manifestations without prior ocular trauma [3] [4]. Her ocular history was notable for secondary complicated cataracts requiring bilateral phacoemulsification with intraocular lens (IOL) implantation (right eye in 2018; left eye in 2021). She subsequently underwent Neodymium-doped Yttrium Aluminum Garnet (Nd:YAG) laser capsulotomy for posterior capsule opacification in the right eye in August 2018 and in the left eye in October 2023 under intensive topical corticosteroid cover.

2.2. Baseline Diagnostic Assessment and Localized Disease Activity

At the baseline examination on October 29, 2025, best-corrected visual acuity (BCVA) was 20/20 in the right eye and 20/150 in the left eye. Intraocular pressure (IOP) measured 12 mmHg in the right eye and 14 mmHg in the left eye. Slit-lamp biomicroscopy demonstrated quiet anterior chambers (0 cells, 0 flare; SUN grade 0) and vitreous cavities (grade 0 haze) in both eyes. Fundus examination revealed a classic "sunset glow" fundus bilaterally with diffuse depigmentation and peripapillary atrophy (**Figure 1**). In the left eye, macular evaluation demonstrated loss of foveal depression and cystoid macular edema. Spectral-domain optical coherence

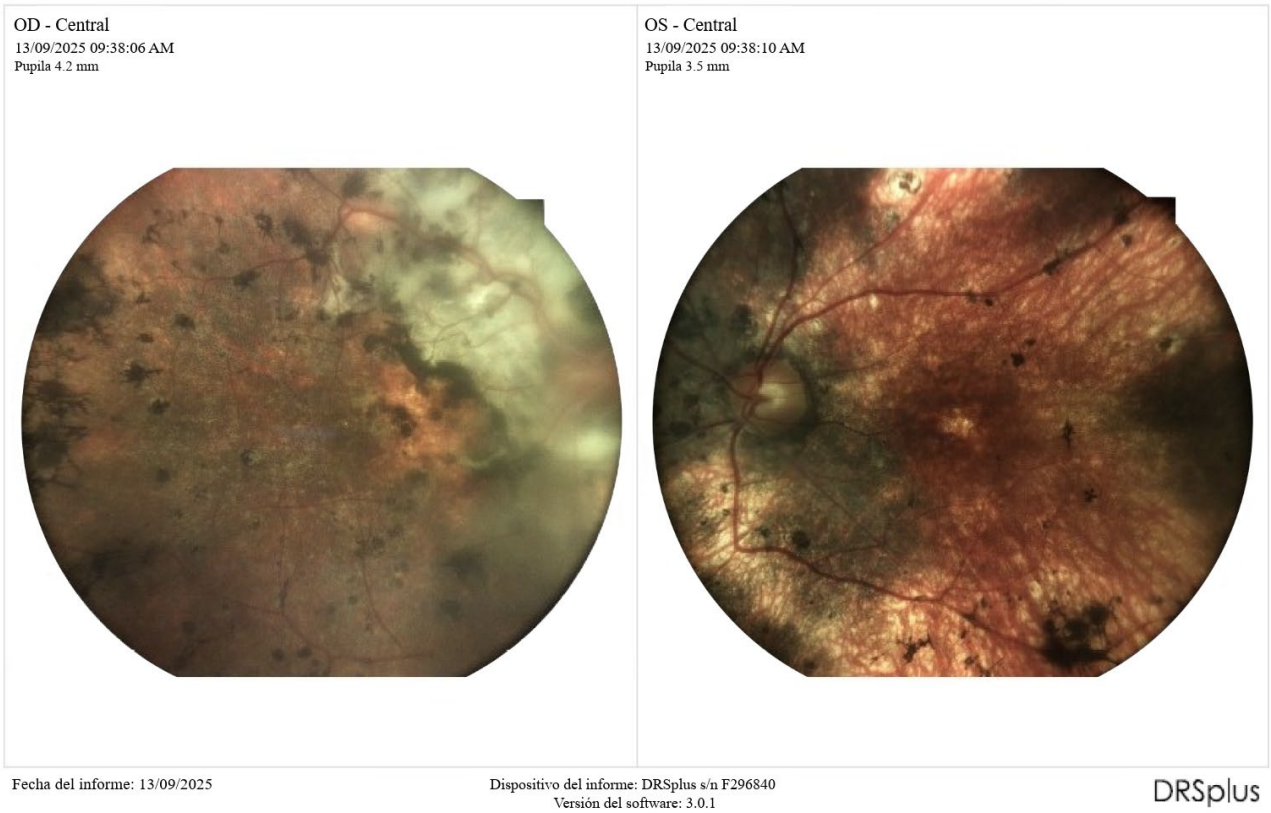


Figure 1. Wide-field fundus photography demonstrating bilateral "sunset glow" fundus appearance with diffuse depigmentation and peripapillary atrophy characteristic of chronic convalescent VKH syndrome.

tomography (SD-OCT) of the left eye confirmed cystoid intraretinal fluid accumulation, foveal thickening, and a central subfield thickness (CST) of 325 μm (Figure 2(A)). The right eye maintained a normal foveal architecture with a CST of 185 μm. Fluorescein angiography (FA) confirmed absence of active multifocal pinpoint leakage, hot discs, or exudative retinal detachments, establishing that inflammatory reactivation was localized strictly to the left macula (Figure 3). Laboratory evaluation demonstrated a mildly elevated C-reactive protein (CRP, 7.7 mg/L) with normal erythrocyte sedimentation rate (ESR, 11 mm/h) and normal white blood cell count (7.3 × 10^9/L), supporting localized retinal vascular leakage rather than an acute systemic panuveitic flare.

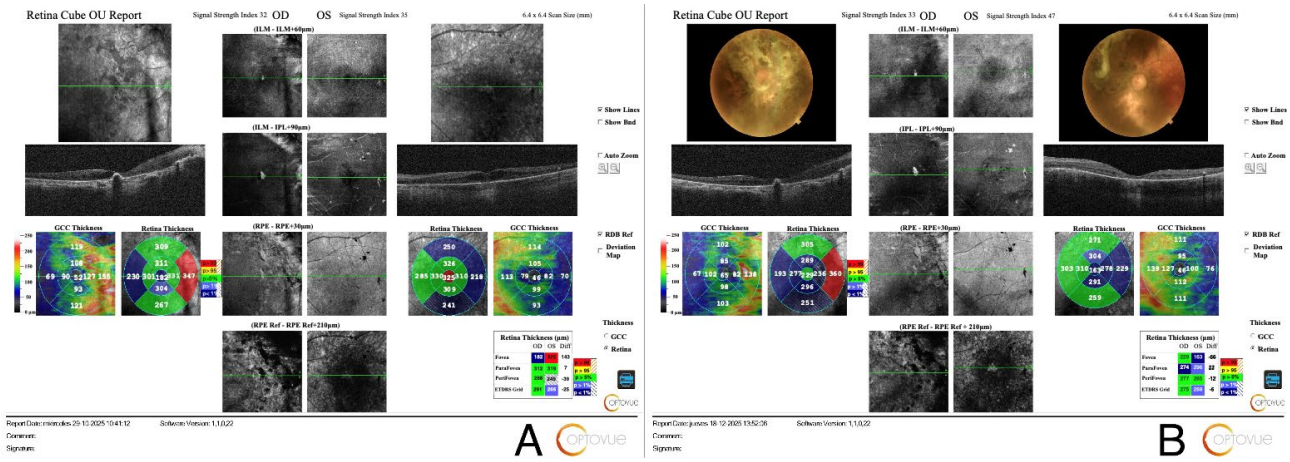


Figure 2. Optical coherence tomography (OCT) of the left eye. (A) Baseline OCT demonstrating cystoid macular edema with loss of foveal depression and a central subfield thickness (CST) of 325 μm. (B) One month post-faricimab injection demonstrating complete anatomical resolution of intraretinal fluid, restoration of foveal contour, and CST reduction to 163 μm.

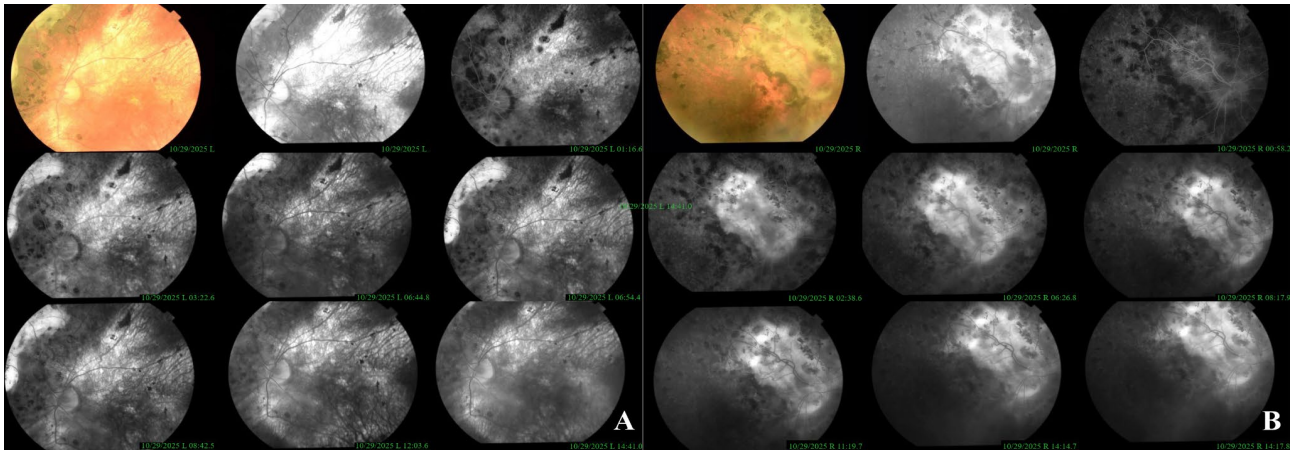


Figure 3. Fluorescein angiography (FA) at baseline. (A) Left eye early-phase angiogram showing localized macular hyperfluorescence. (B) Late-phase angiogram demonstrating focal cystoid macular leakage restricted to the left macula without peripheral vasculitis or optic disc hyperfluorescence.

2.3. Therapeutic Intervention and Concurrent Steroid Regimen

Given that inflammatory reactivation was localized to the left macula without

acute panuveitic activity, targeted intravitreal therapy was selected to achieve rapid anatomical stabilization while avoiding the systemic risks of high-dose corticosteroids or DMARD escalation. To address active ocular inflammation, the patient was prescribed concurrent topical corticosteroid drops consisting of Prednisolone acetate 1.0%/Phenylephrine hydrochloride 0.12% (Prednefrin Forte) eye drops on an intensive 6-week tapering schedule (1 drop q1h for 7 days, q3h for 7 days, q6h for 7 days, q8h for 7 days, q12h for 7 days, and q24h for 7 days before discontinuation). At the time of faricimab administration, the patient was on a maintenance taper of 1 drop daily. Following detailed discussion regarding off-label status, potential risks, and alternative treatments (including intravitreal triamcinolone acetonide), written informed consent was obtained. A single intravitreal injection of Faricimab (6.0 mg/0.05 mL; Vabysmo®, Genentech/Roche) was administered into the left eye under sterile operating room conditions on November 4, 2025.

2.4. Treatment Outcomes and Safety Follow-Up

At the 1-month post-injection follow-up (December 9, 2025), visual acuity in the left eye improved significantly to BCVA 20/50. Repeat SD-OCT demonstrated complete anatomical resolution of cystoid intraretinal fluid, restoration of normal foveal contour, and a reduction in CST from 325 μm to 163 μm (a net decrease of 162 μm) (**Figure 2(B)**). Ocular safety evaluation at 1 day, 1 week, and 1 month post-injection demonstrated quiet anterior chambers and vitreous cavities (0 cells, 0 flare), stable intraocular pressure (10 - 14 mmHg), and complete absence of sterile endophthalmitis, intraocular infection, cataract progression, retinal vasculitis, or retinal tears/detachments. Optic nerve head (ONH) OCT analysis in the right eye showed a decrease in disc area from 3.80 mm^2 at baseline to 1.49 mm^2 at 1 month, which was confirmed upon image verification to represent resolution of baseline peripapillary edema and automated boundary segmentation error rather than structural tissue loss (**Figure 4**).

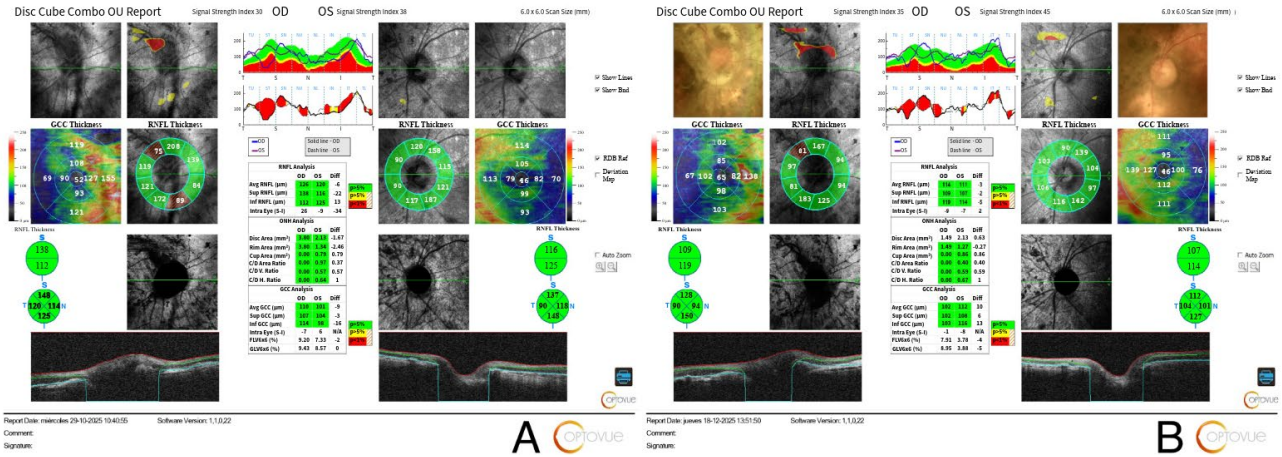


Figure 4. Optic nerve head (ONH) OCT analysis and boundary segmentation. Right eye baseline scan demonstrated a disc area of 3.80 mm^2 due to acute peripapillary edema and automated boundary segmentation error. At 1 month follow-up, resolution of swelling restored accurate boundary detection, yielding a true structural disc area of 1.49 mm^2.

2.5. Ethics and Informed Consent

Written informed consent was obtained from the patient for off-label intravitreal faricimab administration following comprehensive counseling regarding treatment alternatives, potential risks, and lack of formal FDA approval for uveitis. Written informed consent was also obtained for publication of anonymized clinical data and images. Institutional Ethics Committee approval was reviewed by Santa Lucía Hospital, which granted an exemption for single-case report publications involving anonymized patient data.

3. Discussion

In chronic VKH syndrome, reactivations frequently manifest as localized macular edema secondary to persistent breakdown of the inner and outer blood-retinal barriers. Managing UME in chronic VKH presents a therapeutic dilemma: while systemic corticosteroids and DMARDs remain the mainstay for systemic or panuveitic flares [1] [2], localized macular reactivations may be managed with targeted intravitreal agents to achieve prompt fluid clearance without systemic adverse effects.

A critical consideration in evaluating this case is the concurrent administration of topical corticosteroids. Because the patient received topical prednisolone acetate 1.0% alongside intravitreal faricimab, this uncontrolled single-case observation cannot isolate faricimab's independent therapeutic contribution from corticosteroid-mediated anti-inflammatory effects or spontaneous disease resolution. While topical corticosteroids exhibit limited posterior segment penetration in phakic eyes, pseudophakic status and Nd:YAG capsulotomy state enhance posterior drug delivery. Consequently, the observed anatomical resolution must be described as improvement following combined faricimab and topical corticosteroid therapy rather than definitive evidence of faricimab efficacy or steroid-sparing capacity.

Diagnostic classification in VKH relies on the 2001 Revised Diagnostic Criteria [3] [4]. In this patient, documented history of bilateral granulomatous panuveitis, neurological/auditory symptoms, and integumentary signs (poliosis and vitiligo) satisfied the criteria for Complete VKH Syndrome. Establishing robust diagnostic categorization is essential when evaluating novel therapeutic responses in autoimmune uveitis to ensure homogeneous patient selection.

Multimodal imaging played a central role in guiding treatment and monitoring response. Fluorescein angiography confirmed that active inflammation was restricted strictly to the left macula, justifying localized intravitreal therapy over systemic immunosuppression. Furthermore, longitudinal ONH OCT analysis highlighted the importance of verifying automated segmentation parameters: the observed reduction in right disc area (3.80 to 1.49 mm^2) reflected resolution of acute peripapillary swelling rather than true anatomical disc remodeling.

Dual VEGF-A and Ang-2 inhibition with faricimab offers theoretical advantages over selective anti-VEGF mono-inhibition in inflammatory retinal diseases [5] [6]. Ang-2 upregulation under inflammatory conditions promotes vas-

cular permeability, leukocyte extravasation, and pericyte loss; simultaneous blockade of Ang-2 and VEGF-A restores vascular stability and reduces vascular leakage [6] [7]. Recent clinical reports have demonstrated short-term anatomical clearance with faricimab in non-infectious uveitic macular edema, including recalcitrant cases [6]. Safety reporting in uveitic cohorts remains paramount; in line with broader safety analyses [5], our patient exhibited no intraocular inflammation, sterile endophthalmitis, or IOP spikes.

While faricimab is approved for nAMD based on the TENAYA and LUCERNE trials [8] and for DME based on the YOSEMITE and RHINE trials [9], its application in uveitis remains off-label. The rapid CST reduction (325 to 163 μm) and BCVA recovery (20/150 to 20/50) observed here support further investigation. However, well-designed, prospective, controlled trials are imperative to isolate faricimab's independent efficacy and evaluate its steroid-sparing potential in uveitic macular edema.

4. Conclusion

In summary, a patient with chronic VKH syndrome experienced rapid anatomical clearance of localized UME and functional visual recovery following a single intravitreal faricimab injection within a regimen containing concurrent topical corticosteroids. While dual VEGF-A/Ang-2 inhibition represents a pathophysiologically sound modality for inflammatory macular edema, uncontrolled case reports cannot establish independent efficacy or steroid-sparing capacity. Controlled prospective studies are required to define faricimab's role in uveitis management.

Author Contributions

The authors confirm their contribution to the paper as follows: F.D., J.P.H: Data collection and clinical management; A.N., D.R.: Drafting, writing, review, and editing of the manuscript. All authors reviewed the results and approved the final version.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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List of Abbreviations

Ang-2:	Angiopoietin-2
BsAb:	Bispecific Monoclonal Antibody
CME:	Cystoid Macular Edema
CST:	Central Subfield Thickness
FA:	Fluorescein Angiography
OCT:	Optical Coherence Tomography
PCR/CRP:	C-Reactive Protein
UME:	Uveitic Macular Edema
VEGF:	Vascular Endothelial Growth Factor
VKH:	Vogt-Koyanagi-Harada
RE:	Right Eye
LE:	Left Eye
YAG:	Yttrium Aluminum Garnet
BCVA:	Best-corrected Visual Acuity