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Faricimab in the Management of Pseudophakic Cystoid Macular Edema (Irvine-Gass Syndrome): A Case Report

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Abstract

Background: Pseudophakic cystoid macular edema (CME), or Irvine-Gass syndrome, is a recognized complication of cataract surgery. While most cases respond to topical anti-inflammatory therapy, a subset develops persistent or late-onset edema requiring intravitreal intervention. **Case Presentation**: We report a 74-year-old female who developed CME in the left eye following phacoemulsification, with a postoperative course complicated by retained cataract fragments requiring surgical removal. Following an inadequate response to topical bromfenac sodium therapy, OCT confirmed clinically significant macular edema with a central macular thickness (CMT) of 620 μm and best-corrected visual acuity (BCVA) of 20/60, consistent with Irvine-Gass syndrome. The patient received a single intravitreal injection of faricimab (6.0 mg/0.05 mL). **Intervention and Outcome**: At six-week follow-up, CMT decreased to 249 μm (≈60% reduction) and BCVA improved to 20/30, with marked reduction in intraretinal and subretinal fluid and no treatment-related adverse events. Observation was maintained given the favorable anatomical response. **Conclusion**: This case suggests that faricimab, through its dual inhibition of VEGF-A and angiopoietin-2, may represent a potential therapeutic option in selected cases of late-onset or persistent pseudophakic CME, particularly where conventional topical anti-inflammatory therapy has proven insufficient.

Keywords

Faricimab, Cystoid Macular Edema, Irvine-Gass Syndrome, Pseudophakic Macular Edema, Optical Coherence Tomography, Phacoemulsification, Intravitreal Injection, Case Report

1. Introduction

Pseudophakic cystoid macular edema (CME), also known as Irvine-Gass syndrome, is a recognized postoperative complication of phacoemulsification, with a clinical incidence of approximately 1% - 2% and subclinical rates detectable by optical coherence tomography (OCT) of up to 20% [1] [2]. Its pathogenesis is driven primarily by a prostaglandin-mediated inflammatory cascade that disrupts the blood-retinal barrier (BRB), leading to fluid accumulation in the outer plexiform and inner nuclear layers of the macula [1]. Beyond prostaglandins, vascular endothelial growth factor A (VEGF-A) and angiopoietin-2 (Ang-2) have been identified as complementary molecular mediators of BRB breakdown and macular edema perpetuation [1] [3]. Most cases resolve with topical nonsteroidal anti-inflammatory drugs (NSAIDs) and corticosteroids; however, a clinically meaningful subset develops persistent or late-onset CME that requires escalation to intravitreal therapy [2] [4]. Available second-line options include intravitreal corticosteroids and anti-VEGF agents, yet a proportion of patients may not achieve sustained resolution with monotherapy alone [4]. Faricimab (Genentech/Roche, South San Francisco, CA) is a first-in-class bispecific monoclonal antibody that simultaneously inhibits VEGF-A and Ang-2, offering a dual-pathway approach to retinal vascular stabilization [5] [6]. Herein, we report its off-label use in a patient with late-onset pseudophakic CME and a complex postoperative course, and discuss the mechanistic rationale and emerging evidence supporting this approach.

2. Case Presentation

Clinical Timeline:

- **July 2, 2025:** Cataract surgery (phacoemulsification with intraocular lens implantation), left eye.
- **October 22, 2025:** Surgical removal of retained lens fragments.
- **Pre-diagnosis period:** Topical bromfenac sodium (OS), one drop every 12 hours for two months, with an inadequate anatomical response.
- **January 13, 2026:** OCT-confirmed pseudophakic cystoid macular edema (Irvine-Gass syndrome).
- **January 13, 2026:** Intravitreal faricimab (6.0 mg/0.05mL) administered.
- **March 5, 2026:** Follow-up OCT demonstrated significant anatomical improvement.
- **Follow-up plan:** Observation with follow-up in approximately four months.

A 74-year-old female was referred for evaluation of possible ocular inflammation. Her relevant medical history included hypertension, hypothyroidism, and arthritis, managed with levothyroxine, propranolol, valsartan, and naproxen sodium as needed. She had previously undergone uncomplicated phacoemulsification with IOL implantation in the right eye (January 2019). She subsequently underwent phacoemulsification with IOL implantation in the left eye (July 2, 2025), with postoperative topical therapy including bromfenac sodium, travoprost, and preservative-free lubricating drops. At a scheduled follow-up on October 14, 2025,

slit-lamp examination revealed retained cataract/lens fragments in the inferior anterior chamber of the left eye, while fundus photography (**Figure 1**) demonstrated postoperative inflammatory changes without direct visualization of the fragments. Surgical removal was performed on October 22, 2025 under standard sterile conditions via a 2.4 mm corneal incision, without intraoperative complications. Postoperatively, a prednisolone acetate taper was prescribed along with short courses of topical antibiotic and hypotensive therapy. At follow-up on December 11, 2025, the left eye was free of clinically detectable edema and the suture was removed. During subsequent follow-up, the patient reported blurred vision in the left eye. At presentation on January 13, 2026—approximately 12 weeks after the retained fragment removal—BCVA in the left eye was 20/60. OCT demonstrated significant macular thickening with a CMT of 620 μm, and intraretinal cystic spaces in the outer plexiform and inner nuclear layers consistent with Irvine-Gass syndrome (**Figure 2** and **Figure 3**). OCT of the right eye was within normal limits. In addition, OCT angiography/non-contrast angiographic imaging did not demonstrate findings suggestive of microvascular or ischemic retinal disease. Alternative causes of macular edema, including diabetic macular edema, retinal vein occlusion, uveitic macular edema, and tractional pathology, were therefore considered less likely based on the absence of supporting clinical, tomographic, or angiographic findings and the postoperative timing of edema onset [1] [2]. Fluorescein angiography was not performed at initial diagnosis, as OCT, OCT-A/non-contrast angiography, and the clinical context were considered sufficient to support the diagnosis. Because fluorescein angiography requires intravenous dye administration and severe hypersensitivity reactions, including anaphylaxis, have been reported [7], it was reserved for diagnostically uncertain cases or cases with inadequate response to initial management. In addition, OCT provided a noninvasive and more readily accessible initial assessment compared with fluorescein angiography. Intraocular pressure on the date of CME diagnosis was not documented in the available clinical record. Anterior chamber and vitreous inflammation grading was not available in the clinical record. While a direct causal relationship between the retained fragments and the subsequent CME cannot be established with certainty, the associated postoperative inflammatory response may have contributed to blood-retinal barrier disruption. Before escalation to intravitreal therapy, the patient had received topical bromfenac sodium in the left eye, one drop every 12 hours for approximately two months, with an inadequate anatomical response, and was no longer receiving topical corticosteroid therapy at the time CME was diagnosed. Given persistent pseudophakic CME and the absence of prior intravitreal or periocular corticosteroid therapy, a decision was made to proceed with an intravitreal injection of faricimab (6.0 mg/0.05 mL), administered at 4 mm from the corneoscleral limbus under topical anesthesia, without complications. At follow-up on March 5, 2026, OCT demonstrated marked improvement: CMT decreased to 249 μm (≈60% reduction) and BCVA improved to 20/30, with a substantial reduction in intraretinal and subretinal fluid (**Figure 4**). Minimal residual fluid persisted, consistent with ongoing anatomical resolution. At this follow-up,

intraocular pressure measured 17 mmHg in the right eye and 20 mmHg in the left eye, with no intraocular inflammation and no injection-related complications. Given the favorable anatomical response and the estimated duration of faricimab activity, observation was maintained pending further evaluation [5]. It should be noted that the six-week follow-up interval is insufficient to assess long-term durability or recurrence risk after biologic clearance.

Figure 1. Color fundus photography (October 14, 2025) of the left eye (OS) showing mild vascular engorgement and diffuse haze consistent with postoperative inflammatory changes, obtained prior to the diagnosis of CME.

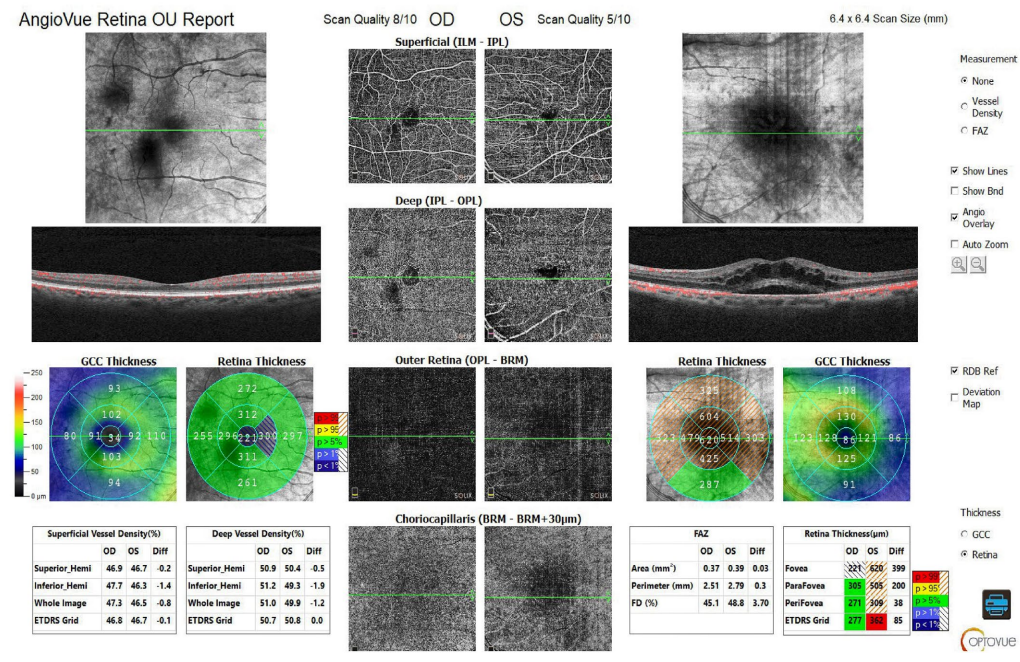

Figure 2. Baseline OCT-A (January 13, 2026) demonstrates significant macular thickening in the left eye (OS) with a central macular thickness of 620 µm, consistent with pseudophakic cystoid macular edema (Irvine-Gass syndrome).

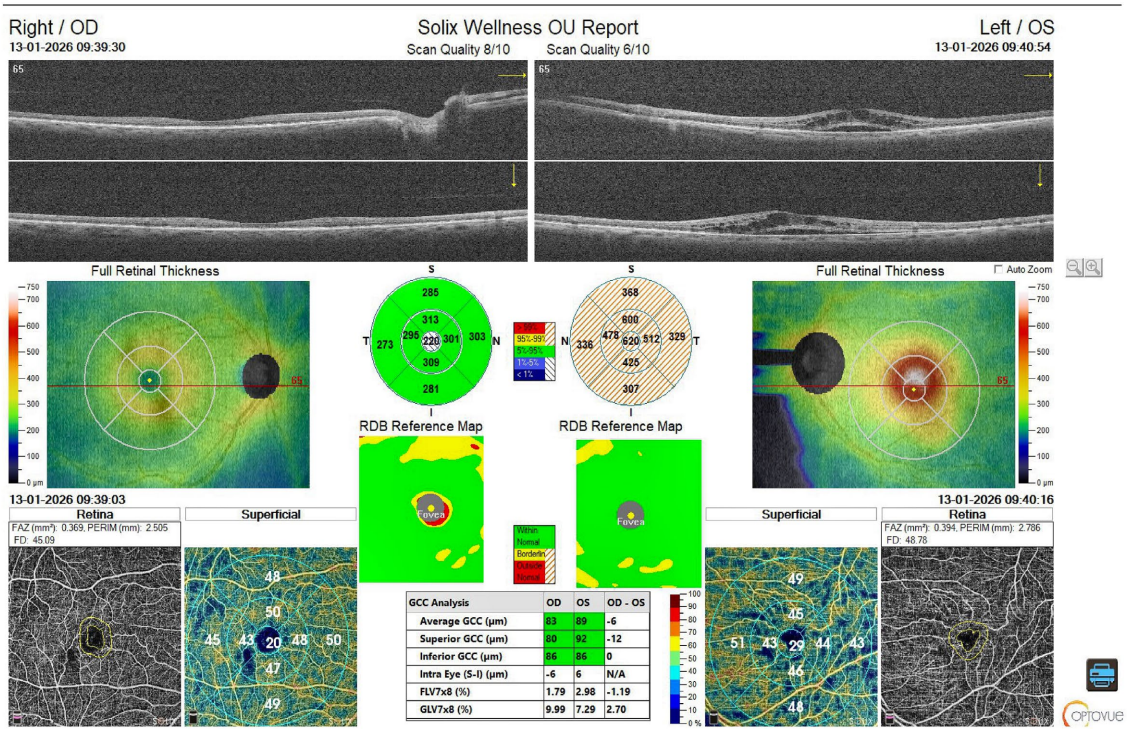

Figure 3. Structural OCT (January 13, 2026, baseline) of the left eye (OS) showing intraretinal cystic hyporeflective spaces with a central macular thickness of 620 µm, consistent with pseudophakic cystoid macular edema.

3. Discussion

The rationale for faricimab in pseudophakic CME rests on its bispecific design simultaneously targeting VEGF-A and Ang-2—the two principal molecular axes of retinal vascular permeability and instability. VEGF-A drives BRB leakage, while Ang-2 destabilizes the retinal vasculature and amplifies the postoperative inflammatory milieu that underlies Irvine-Gass syndrome [3] [6]. By neutralizing both mediators, faricimab addresses mechanisms not fully resolved by VEGF monotherapy or topical anti-inflammatory therapy alone [5] [6]. Although pivotal trial evidence for faricimab derives from RVO and nAMD cohorts—settings mechanistically distinct from pseudophakic CME—the underlying molecular effectors of vascular permeability share substantial overlap, providing an indirect mechanistic rationale for its off-label use in this context [5].

3.1. Epidemiology

Irvine-Gass syndrome is among the most clinically significant causes of suboptimal visual outcomes following cataract surgery. Clinical CME occurs in approximately 1% - 2% of uncomplicated phacoemulsification procedures, while subclinical CME detectable by OCT may affect up to 20% of patients [1] [2]. Most cases resolve spontaneously within three months; however, a clinically meaningful subset progresses to persistent or late-onset CME. Recognized risk factors include posterior capsule rupture, epiretinal membrane, uveitis, diabetic retinopathy, retinal vein occlusion, a history of CME in the contralateral eye, and systemic conditions

such as diabetes mellitus, hypertension, and prostaglandin analog use [1] [2].

3.2. Pathophysiology

The pathogenesis of pseudophakic CME is multifactorial and has been attributed primarily to a prostaglandin-mediated inflammatory cascade initiated by surgical trauma. Phacoemulsification disrupts the integrity of the anterior segment, triggering the release of arachidonic acid from cell membrane phospholipids. The subsequent enzymatic conversion of arachidonic acid via the cyclooxygenase (COX) pathway generates prostaglandins, which diffuse posteriorly and disrupt the inner and outer blood-retinal barriers (BRBs), culminating in the transudation of plasma constituents into the outer plexiform and inner nuclear layers of the macula. The resulting fluid accumulation produces the characteristic petaloid pattern of cystoid spaces observed on fluorescein angiography and OCT imaging [1].

Beyond the prostaglandin axis, VEGF-A and Ang-2 have been implicated as complementary molecular mediators of edema perpetuation. VEGF-A increases BRB leakage through disruption of tight junctions and endothelial architecture, while Ang-2 antagonizes Tie2 receptor signaling, destabilizing the retinal vasculature and amplifying the inflammatory response beyond what VEGF-A alone produces [3] [6].

Although pseudophakic CME is mechanistically distinct from DME, nAMD, and RVO-associated macular edema insofar as it is primarily surgery-induced, the downstream molecular effectors of vascular permeability share substantial overlap [5] [6] [8]. This convergence provides the pathophysiological rationale for dual-pathway inhibition strategies in persistent pseudophakic CME, particularly where prostaglandin-mediated mechanisms alone are insufficient to explain the magnitude or persistence of edema [1] [8].

3.3. Diagnostic Criteria

The diagnosis of pseudophakic CME is clinical and imaging-based. Symptomatic patients typically present with painless, progressive reduction in BCVA within the first four to twelve weeks postoperatively, though late-onset presentations may be subtle, as illustrated here [1]. OCT constitutes the gold standard, demonstrating intraretinal hyporeflective cystic spaces in the outer plexiform and inner nuclear layers, increased CMT, foveal contour disruption, and, in advanced cases, subretinal fluid—arranged in the characteristic petaloid configuration of Henle's fiber layer [1] [2]. Fluorescein angiography may be employed in diagnostically ambiguous cases, characteristically revealing progressive petaloid hyperfluorescence in late frames. The absence of acute visual symptoms does not preclude clinically significant CME, underscoring the value of systematic OCT surveillance after phacoemulsification [1].

3.4. Differential Diagnosis

Pseudophakic CME must be differentiated from other causes of postoperative

macular thickening. Diabetic macular edema (DME) may mimic pseudophakic CME on OCT; the distinction requires evaluation of the clinical context, diabetic retinopathy findings on fundoscopy, and the temporal relationship to surgery [1] [2]. Macular edema secondary to retinal vein occlusion (RVO) is typically accompanied by characteristic fundoscopic signs (flame hemorrhages, dilated tortuous veins, disc edema) and can be further characterized by FA and OCTA [5] [6] [8]. Epiretinal membrane and vitreomacular traction may produce similar foveal distortion; en face OCT facilitates their distinction and these conditions may coexist with pseudophakic CME [1]. Uveitic macular edema may be clinically indistinguishable from Irvine-Gass syndrome on OCT alone; anterior chamber cells and flare, vitreous cells, and a history of uveitis are key discriminating features [9]. Additional entities including central serous chorioretinopathy and choroidal neovascularization may be evaluated with multimodal imaging as indicated [1] [9].

3.5. Therapeutic Strategy

The management of pseudophakic CME follows a stepwise approach. Topical NSAIDs and corticosteroids constitute established first-line therapy, and the majority of acute cases resolve with this regimen alone [2] [4]. In persistent cases, escalation to intravitreal therapy may be considered. Intravitreal corticosteroids—including triamcinolone acetonide and the dexamethasone intravitreal implant—have been used in this context; however, their use carries a risk of corticosteroid-induced ocular hypertension, particularly relevant in patients with pre-existing glaucoma [2] [4]. In the present case, faricimab was selected as the first intravitreal agent as part of a steroid-sparing strategy, considering the potential risk of corticosteroid-associated intraocular pressure elevation and the mechanistic rationale for dual VEGF-A/Ang-2 inhibition in a postoperative inflammatory and vascular permeability-driven setting; its use in this indication remains off-label. In the local clinical context, limited access to the dexamethasone intravitreal implant in the Honduran market also influenced the decision to consider a steroid-sparing intravitreal approach. Anti-VEGF agents have also been employed as an off-label second-line option in selected cases, though a subset of patients may fail to achieve sustained resolution, possibly reflecting the contribution of Ang-2-mediated vascular destabilization beyond the VEGF-A pathway alone [4] [8]. Although faricimab is currently approved for neovascular age-related macular degeneration, diabetic macular edema, and macular edema secondary to retinal vein occlusion, its use in pseudophakic cystoid macular edema remains off-label [5] [10]. Faricimab simultaneously targets VEGF-A and Ang-2, addressing both principal axes of retinal vascular permeability and instability in a single injection [5]. Its steroid-free mechanism avoids the risk of corticosteroid-induced IOP elevation, a clinically relevant consideration in this patient given her concurrent use of prostaglandin analogs [2] [5]. While direct evidence for faricimab in pseudophakic CME remains limited, real-world studies in macular edema secondary to retinal vein occlusion have demonstrated meaningful CMT and BCVA improvements, provid-

ing indirect mechanistic support for its potential utility in other forms of vascular permeability-driven macular edema [6] [8] [11]. Emerging case reports have additionally documented beneficial outcomes in recalcitrant multifactorial and uveitic macular edema [3] [9]. In this case, the complex postoperative context—retained fragments preceding the CME diagnosis by approximately 12 weeks—may have amplified the inflammatory burden through both VEGF-A- and Ang-2-mediated pathways, providing additional mechanistic rationale for dual inhibition. The anatomical and functional improvement observed was associated with this therapeutic approach, though a causal relationship cannot be definitively established from a single case report [3].

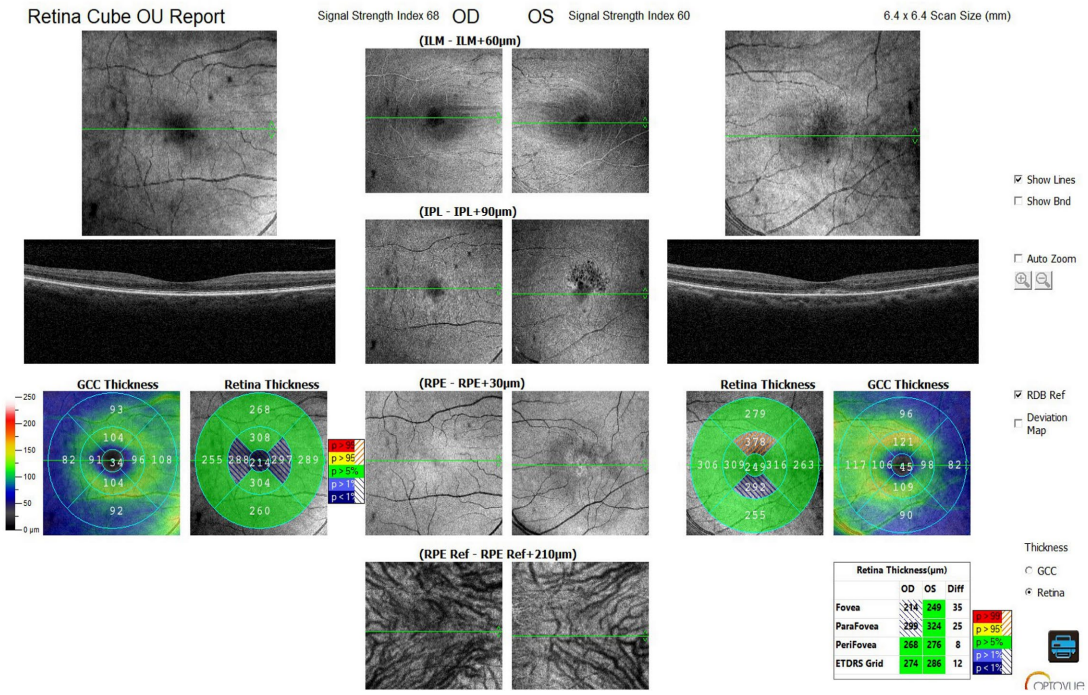

Figure 4. Follow-up OCT (March 5, 2026) of the left eye (OS) demonstrating anatomical improvement after intravitreal faricimab, with central macular thickness reduced from 620 µm to 249 µm and partial resolution of the perifoveal cystic pattern.

4. Conclusion

This case report describes the off-label use of intravitreal faricimab in a patient with late-onset pseudophakic cystoid macular edema occurring in the context of a complex postoperative course that included retained cataract fragment removal. The anatomical and functional improvements observed—CMT reduction from 620 µm to 249 µm and BCVA improvement from 20/60 to 20/30 at six weeks, with marked diminution of intraretinal and subretinal fluid—support the mechanistic rationale for dual VEGF-A/Ang-2 inhibition in persistent pseudophakic CME, particularly in patients with a complex postoperative inflammatory course. The postoperative inflammatory burden associated with retained lens material may represent an underappreciated contributor to the molecular pathways that farici-

mab simultaneously targets, providing additional rationale for its use in patients with a surgically complicated postoperative course. Of particular note, faricimab's steroid-free mechanism may offer a clinically meaningful advantage in patients at risk for corticosteroid-induced ocular hypertension or glaucoma, for whom intravitreal corticosteroids are relatively contraindicated. Several limitations of this report warrant emphasis. The patient did not undergo a trial of conventional second-line therapies—such as periocular or intravitreal corticosteroids—prior to faricimab, limiting the ability to characterize the case as strictly refractory to established therapies. Additionally, the complex postoperative history of retained cataract fragment removal represents a confounding factor that may have heightened the inflammatory environment, and these findings may not be fully generalizable to uncomplicated Irvine-Gass syndrome. The six-week follow-up interval is insufficient to establish long-term anatomical durability or assess recurrence risk after biologic clearance. Conclusions derived from a single case must therefore be interpreted with appropriate caution. Prospective, controlled studies with longer follow-up are necessary to establish standardized treatment protocols, optimal dosing intervals, and the long-term efficacy and safety of faricimab in post-cataract CME cohorts.

Ethical Approval and Informed Consent

Written informed consent was obtained from the patient for the publication of this case report and any accompanying images, in accordance with the ethical standards of the institution. Formal ethics committee approval was not required for this case report under the applicable institutional guidelines; however, the study adhered to the principles of the Declaration of Helsinki.

Conflicts of Interest

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

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