

CASE REPORT

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Rapid resolution of submacular hemorrhage in neovascular age-related macular degeneration using pneumatic displacement and faricimab

Patrick Xiang Ji¹, Joshua E. Herman² and Nirojini Sivachandran^{2,3,4,5,6*}

Abstract

Background Submacular hemorrhage (SMH) is a rare but visually devastating complication of neovascular age-related macular degeneration (nAMD) associated with poor long-term prognosis due to photoreceptor iron toxicity, subretinal fibrosis, and retinal pigment epithelial (RPE) damage. Pneumatic displacement (PD) combined with anti-vascular endothelial growth factor (anti-VEGF) therapy is an established approach for managing acute SMH; however, the role of faricimab, a novel dual inhibitor of VEGF-A and angiopoietin-2 (Ang-2), in this context has not been previously evaluated.

Case presentation This study reports the outcomes of PD combined with faricimab injection in a case of a 75-year-old female with acute SMH secondary to nAMD. The patient with dense SMH and a large PED secondary to presumed polypoidal choroidal vasculopathy (PCV) underwent PD with 0.40 mL of sulfur hexafluoride and intravitreal faricimab injection (50 µL, 6 mg) on the same day as presentation, followed by 5 days of face-down positioning with excellent patient compliance confirmed by verbal verification. Two additional faricimab injections were administered at 4 and 8 weeks, followed by treat-and-extend protocol.

Conclusions The use of timely PD with appropriate head positioning can effectively manage patients with acute large SMH and improve visual outcomes. While the combined approach with serial faricimab injections showed longer-term benefits, the immediate improvement was likely due to mechanical techniques. The potential added benefit of faricimab's dual inhibition mechanism over other anti-VEGF therapies in SMH remains to be investigated through larger, comparative trials.

Summary statement

This reports shows the successful management of submacular hemorrhage in presumed polypoidal choroidal vasculopathy using pneumatic displacement. The timely displacement of subretinal hemorrhage observed within 24 h was likely because of the mechanical technique with good head positioning, whereas longer-term anatomical stabilization can be attributed to continued anti-VEGF injections with faricimab.

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Significant SMH displacement was seen within 24 h post-procedure, with visual acuity improving from counting fingers to 20/100. After 3 intravitreal faricimab injections over 3 months, complete resolution of SMH and serous PED was achieved with visual acuity improving to 20/50. A mild self-resolving vitreous hemorrhage was seen after 1 month.

Keywords Submacular hemorrhage, Neovascular age-related macular degeneration, Polypoidal choroidal vasculopathy pneumatic displacement, Faricimab, Anti-VEGF agents, Angiopoietin-2

Background

Submacular hemorrhage (SMH) is a rare complication of neovascular age-related macular degeneration (nAMD) that causes sudden vision loss and long-term visual disruption through photoreceptor iron toxicity, subretinal fibrosis, atrophic scarring and retinal pigment epithelial (RPE) tears [1]. Depending on the SMH size, vitrectomy or pneumatic displacement (PD) may be used to displace the hemorrhage away from the fovea and macula while anti-vascular endothelial growth factor (VEGF) agents are used to decrease hemorrhage by reducing vascular hyperpermeability and neovascularization to allow for more rapid resorption [2, 3].

Although anti-VEGF monotherapy (such as aflibercept and ranibizumab) have revolutionized the treatment of nAMD, management of large SMH remains complex. Emerging evidence has identified angiopoietin-2 (Ang-2) as an important ligand in a distinct biochemical pathway that modulates retinal neovascularization, inflammation and fibrosis [4]. Its synergistic action with VEGF-A plays a critical role in vascular permeability and stability [5]. Ang-2 has also been shown to localize to highly vascularized choroidal neovascular complexes, which have a higher propensity to present with hemorrhage [6]. In this regard, faricimab – the first bispecific antibody independently inhibiting both VEGF-A and Ang-2 – may offer a theoretical and clinical advantage by stabilizing neovascular complex or polyps more effectively, whilst reducing the likelihood of recurrent hemorrhage and facilitating intraretinal and subretinal fluid resolution. We now present our experience using faricimab in combination with PD to treat SMH and a large serous pigment epithelial detachment (PED) in a patient with the polypoidal choroidal vasculopathy (PCV) subtype of nAMD. The CARE Checklist has been completed by the authors for this case report, attached as online supplementary material.

Case presentation

Clinical presentation

A 75-year-old female with hypertension and dyslipidemia presented with a 2-week history of sudden painless vision loss in the left eye. She has an unremarkable ocular history. On initial presentation, her best corrected visual acuity (BVCA) was 20/25 in the right eye and counting fingers (CF) in the left eye, phakic, and her intraocular pressures were within normal limits. On examination,

she had dense extra-foveal SMH extending from the peripapillary area along the superior and inferior arcades, measuring greater than 4 disc diameters (DD), height less than 400 µm, with a large serous PED and subretinal fluid (Figs. 1a, b and 2a). The right eye had peripapillary pigmentary changes with pachychoroid and pachyvesels. Given her demographics, multilobulated PED, and absence of choroidal neovascular membrane on multimodal imaging, and the presence of pachychoroid in the fellow eye the presentation was consistent with the PCV subtype of nAMD [7].

Treatment protocol

Written informed consent was obtained from the patient prior to the procedure. On the same day of presentation to the retina clinic, using sterile technique (speculum and topical povidone-iodine 5%) and appropriate anaesthesia (topical and subconjunctival lidocaine), anterior chamber paracentesis was performed using a 30G needle on a TB syringe, followed by superotemporal injection of 0.40 mL of sulfur hexafluoride (SF₆) gas 4.0 mm from the limbus. After normalization of intraocular pressure, an inferotemporal intravitreal injection of faricimab (50 µL, 6 mg) was performed 4.0 mm from the limbus using aspheric technique. The patient was counselled extensively regarding the importance of face-down positioning for effective hemorrhage displacement, and was instructed to maintain this positioning for five days. Compliance was verified at each follow-up visit through verbal confirmation; the patient successfully maintained face-down positioning for the entire prescribed period, despite occasional musculoskeletal discomfort in the neck and shoulder region.

Following the initial treatment, the patient received two additional monthly intravitreal faricimab injections, completing a total of three loading doses, after which a treat-and-extend regimen was implemented. The patient was assessed on post-procedure day 1, day 5, and monthly thereafter, for a total follow-up period of five months.

Clinical outcomes

On post-procedure day 1, significant displacement of the SMH to outside the superior and inferior arcades was observed, with improvement in serous PED (Figs. 1c and d and 2b). BCVA improved from CF to 20/100 in the left eye. Table 1 summarizes the timeline of visual and

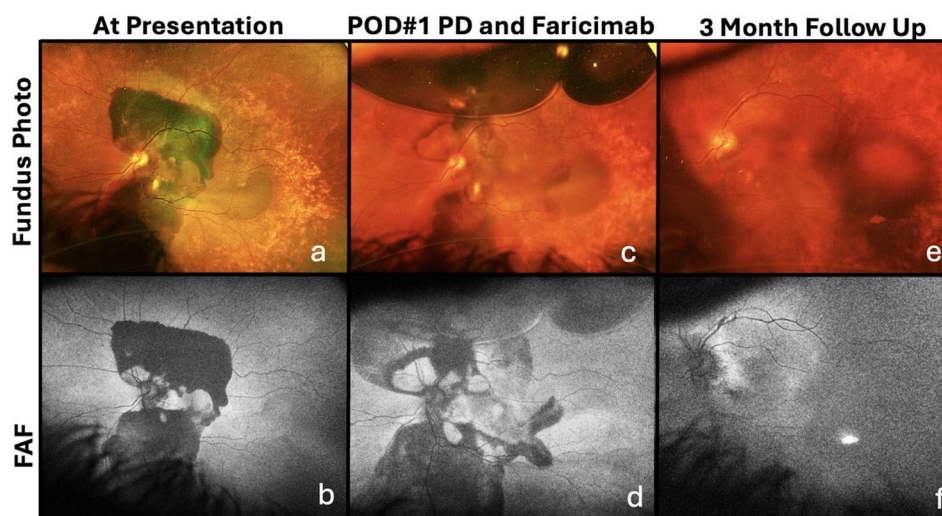


Fig. 1 Widefield color fundus and autofluorescence photos of SMH resolution Ultra-widefield fundus imaging using Optos and fundus autofluorescence (FAF) shown at presentation (**a** and **b**). The dark areas of hypoautofluorescence in **b** clearly demarcates the area of submacular hemorrhage. Postprocedure day 1 of pneumatic displacement (POD#1 PD), the SF₆ gas bubbles can be noted superiorly (**c**). The submacular hemorrhage is displaced outside of the arcade (**c** and **d**). Resolution of submacular hemorrhage noted post three faricimab treatments at 3 months from presentation (**e** and **f**). An area of hyper-autofluorescence is noted in (**f**), likely calcified drusen

anatomical outcomes at each key assessment point, differentiating the immediate effects of mechanical PD from the longer-term pharmacological effects of serial faricimab injections.

At four weeks post-treatment, there was a significant reduction in PED height from 1,137 μm to 184 μm (Fig. 2c); however BCVA temporarily decreased to 20/250 due to a mild vitreous hemorrhage. The patient continued to receive faricimab injections monthly, and her retinal anatomy continued to improve after the second treatment (Fig. 2d). After three loading doses, complete resolution of serous PED, SMH and vitreous hemorrhage was achieved (Figs. 1e, f and 2e), with BCVA improving to 20/50 – a gain of six lines – and no evidence of submacular fibrosis.

Safety and adverse events

Aside from a minor self-resolving vitreous hemorrhage at one month, no other adverse events were observed, including RPE tears, anterior or posterior segment inflammation, retinal vasculitis, or endophthalmitis.

Discussion and conclusions

This case describes a successful treatment approach for acute large SMH with serous PED secondary to presumed PCV, using PD with SF₆ and concurrent intravitreal injection of faricimab. The rationale behind this treatment is to mechanically displace blood away from the fovea, minimizing toxicity to photoreceptors and scarring in the region of high-acuity central vision [3]. The anti-VEGF and anti-Ang-2 properties of faricimab address the underlying pathologic process by reducing

vascular permeability and neovascularization, thereby facilitating rapid fluid resorption [3, 5, 8].

While extensive hemorrhage was observed in the temporal region (Fig. 2), optical coherence tomography (OCT) imaging demonstrated preserved subfoveal RPE reflectivity, suggesting minimal subfoveal heme thickness, which may explain the favorable visual outcome in this patient. The shape and size of early reductions in PED height within the first 24 h was unexpected and likely reflects the combined effects of mechanical displacement of the gas bubble followed by early fluid mobilization of faricimab. The resolution of serous PED from 1,137 μm to 184 μm within the first four weeks is attributed to the dual inhibition of VEGF-A and Ang-2. In this case, adequate mechanical displacement of the SMH with expansile gas was achieved, corresponding to a rapid gain in visual acuity from CF to 20/100 within the first 24 h. These immediate improvements were primarily due to mechanical displacement by the expanding SF₆ gas bubble, rather than the pharmacological drug effects of faricimab alone, as the timeframe was too short for meaningful drug effect and Ang-2 inhibition typically requires multiple injections to demonstrate full efficacy [9, 10]. Nevertheless, continued faricimab injections were effective in rapidly clearing subretinal hemorrhage, subretinal and sub-RPE fluid, leading to a 953 μm reduction in PED height at one month. After vitreous hemorrhage (VH) resolution, the patient further improved her visual acuity to 20/50 at three months for a total gain of six lines.

Regarding the choice to use faricimab, the LUCERNE and TENAYA phase III non-inferiority randomized

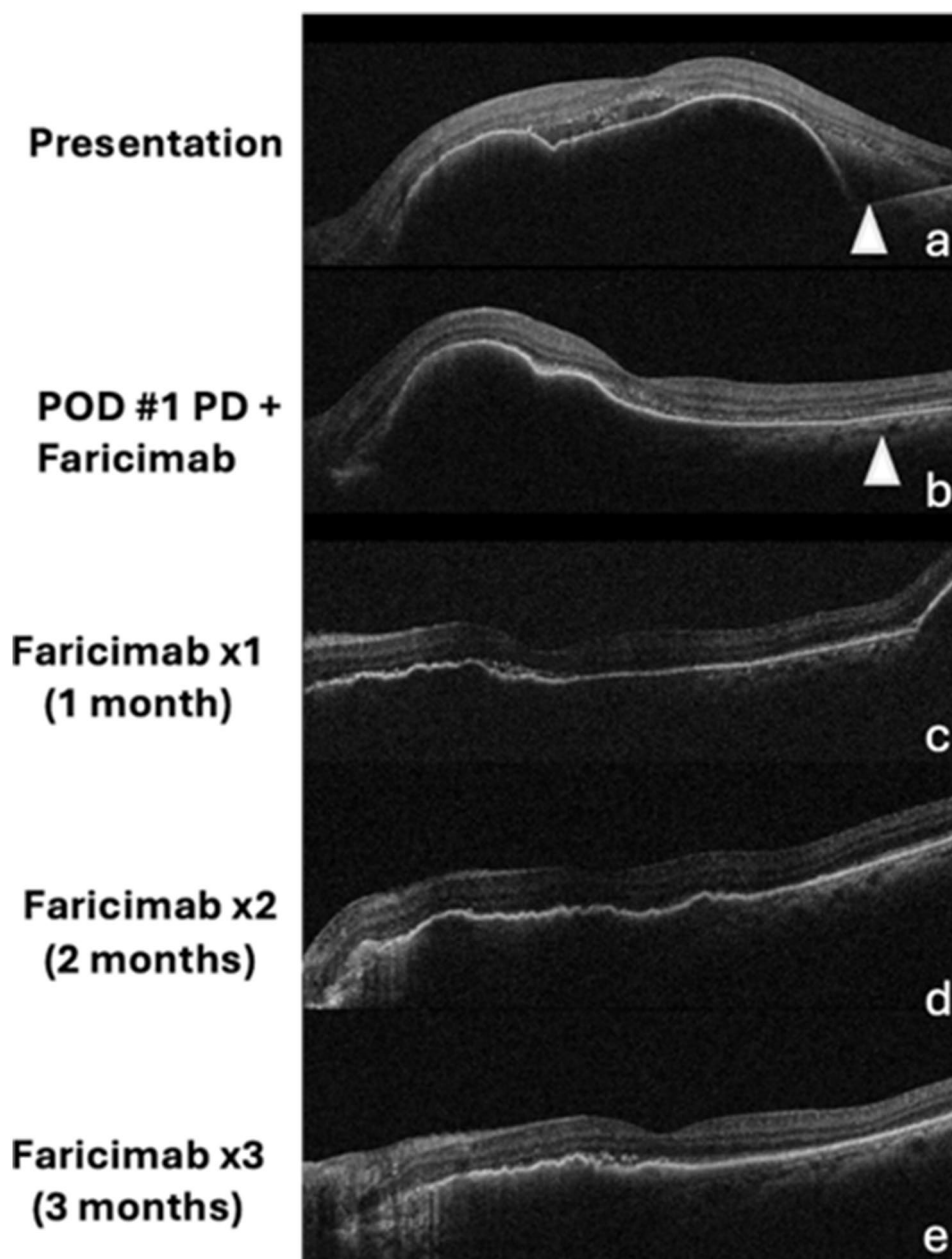


Fig. 2 Spectral domain OCT of SMH and PED resolution. Spectral domain OCT of the left eye at baseline (a). Post-operative day 1 of pneumatic displacement and faricimab injection (POD #1 PD + Faricimab) there was displacement of subretinal hemorrhage (arrowhead) (b). At one month follow-up post a single treatment of faricimab there was complete resolution of PED and subretinal fluid with mild vitreous hemorrhage (c). Retinal anatomy continued to improve after the second treatment with faricimab (d) and vitreous hemorrhage resolved after the third treatment of faricimab (e)

controlled trials (RCTs) comparing aflibercept to faricimab in nAMD demonstrated that faricimab was associated with faster resolution of intraretinal and subretinal fluid compared to aflibercept [11]. In a post-hoc analysis by Lim and colleagues, at week 12, only 3.9% of faricimab-treated eyes versus 12.0% aflibercept-treated eyes had serous PED [12]. Furthermore, Ang-2 compared to Ang-1 has been shown to localize to highly vascularized choroidal neovascular membranes (CNV) in post mortem specimens [6], and vascularized CNV are more

likely to present with SMH compared to immature CNV [13]. Hence, this led us to believe that Ang-2 inhibition should provide additional benefit in patients presenting with SMH. Given the need for rapid and stable clearance of hemorrhage to avoid long-term complications, faricimab was chosen.

Existing observational evidence indicates that approximately 40–60% of patients with SMH in nAMD treated with PD and anti-VEGF (with or without tissue plasminogen activator [tPA]) will have improved vision at

Table 1 Timeline of visual acuity and anatomical outcomes following pneumatic displacement and serial intravitreal faricimab injections

Timepoint	Procedure / Treatment	BCVA (Left Eye)	Key Anatomical Finding
Day 0 (Baseline)	PD: SF ₆ 0.40 mL (superotemporal) + Faricimab 6 mg (inferotemporal) Anterior chamber paracentesis	CF (left eye) 20/25 (right eye)	Dense extra-foveal SMH > 4 DD, height < 400 µm Serous PED height 1,137 µm Subretinal fluid present
Post-procedure Day 1	Face-down positioning (Day 1 of 5)	20/100 (↑ from CF)	SMH displaced outside superior and inferior arcades Early improvement in serous PED
Day 5	Completion of face-down positioning	—	Displacement maintained; clinical assessment
Week 4 (1 month)	Faricimab injection #2	20/250* (*temporary)	PED height: 1,137 µm → 184 µm (184%) Mild vitreous hemorrhage onset
Week 8 (2 months)	Faricimab injection #3	Improving	Continued retinal anatomy improvement VH resolving
Month 3	Treat-and-extend initiated	20/50 (+ 6 lines from baseline)	Complete resolution of SMH, serous PED, and VH No submacular fibrosis
Month 5	Treat-and-extend follow-up	20/50 (maintained)	Sustained anatomical and functional stability

*Temporary decrease due to mild self-resolving vitreous hemorrhage. BCVA = best-corrected visual acuity; CF = counting fingers; DD = disc diameters; PD = pneumatic displacement; PED = pigment epithelial detachment; SF₆ = sulfur hexafluoride; SMH = submacular hemorrhage; VH = vitreous hemorrhage

three-six months, with an average gain of around three lines [3, 14–16]. Therefore, a gain of six lines such as in this case exceeds what have been shown in the literature, and would be considered an excellent result. For comparison, a retrospective study from Japan using aflibercept or ranibizumab combined with PD reported visual acuity changes from 0.88 logMAR at baseline to 0.80 logMAR at three months ($p = 0.40$), from 0.88 logMAR at baseline to 0.76 logMAR at six months ($p = 0.24$), and from 0.92 logMAR at baseline to 0.56 logMAR at twelve months ($p = 0.01$) [17]. Our case demonstrated quicker and a considerable improvement in visual outcomes at three months, with sustained visual gains at six months and twelve months with a treat and extend protocol. Our patient's age of 75 years was consistent with their reported mean age of 75.5 and 74 years for six-months and twelve-months follow-up, respectively. The mild adverse event in our case represented by minor self-resolving vitreous hemorrhage at 1 month was consistent with 31.8% of eyes examined by Ota et al., occurring at an average of 21.8 days (range 1–33 days) post-procedure. Notably, 9.1% of their patients also developed more serious adverse events such as rhegmatogenous retinal detachment and recurrence of SMH post treatment.

More recently, emerging literature evidence from real-world studies has specifically examined faricimab's role in nAMD complicated by subretinal hemorrhage (SRH). A multi-centre retrospective analysis of 63 treatment-naïve nAMD patients with SRH by Sivachandran et al., demonstrated that 42.6% gained ≥ 3 lines of vision at last follow-up, with complete SRH resolution achieved with a median of only two faricimab injections and a mean central subfield thickness (CST) reduction from 391.8 µm to 249.4 µm [18]. Separately, Lim et al., reported that six patients with nAMD and large SMH (mean SMH coverage 73.8% of CNV lesion area) managed with

faricimab monotherapy – without pneumatic displacement – achieved a mean visual acuity gain of 16.4 ETDRS letters (from 20/138 to 20/50) over one year, with resolution occurring in a mean of 2.7 injections [19]. This case series is particularly relevant as it suggests faricimab monotherapy may be sufficient for SMH below approximately 750 µm in thickness, whereas cases with thicker hemorrhage may benefit from combined mechanical displacement. Furthermore, the J-CREST multicentre cohort study by Yanai et al., with $n = 186$ eyes demonstrated that faricimab-treated eyes with pretreatment SMH achieved a significantly higher dry macula rate (93.9%) compared to eyes without SMH (78.4%, $p = 0.048$) [20], supporting the hypothesis that faricimab's Ang-2 inhibition may provide specific vascular stabilizing advantages in the presence of hemorrhage.

This is a favorable outcome especially given that large hemorrhage size, and treatment delay of greater than 14 days have been associated with worse outcomes irrespective of treatment modality [14, 21]. It is notable that our patient received therapy during the same day of her presentation, and this may have potentially contributed to the favorable visual outcomes through minimizing photoreceptor toxicity from prolonged exposure to blood. Regarding decision-making surrounding our intervention, existing observational evidence supports anti-VEGF monotherapy as an effective first-line treatment for small (1–4 DD) and thin (< 450–500 microns tall) SMH in nAMD and PCV [3, 11, 14, 15, 22]. However, superior anatomical and functional outcomes have been demonstrated in medium sized (4 DD to arcades) and thick (> 450–500 microns tall) SMH when vitrectomy or PD is combined with anti-VEGF, with or without tPA [3, 14, 22].

While faricimab was selected based on aforementioned mechanistic rationale, it likely played a minimal role in

the early days where mechanical displacement was the primary mechanism of action. Although it was likely that continued faricimab injections contributed to the longer-term visual improvements and anatomical stabilization, its superiority in dual Ang-2/VEGF inhibition over other anti-VEGF treatments remains to be elucidated in SMH.

This case report has several limitations. First, we could not draw conclusions about the independent treatment effect of PD versus faricimab injections based on a single case. Second, despite the overall favourable visual outcome, the 2-week symptom duration before clinical presentation may indicate a treatment delay, which may have influenced retinal structural integrity, although this likely aided in liquefaction of the hemorrhage and facilitated displacement away from the fovea and macula. Third, the diagnosis of PCV remains presumptive based on OCT morphological criteria and not based on confirmation through ICGA given the density of the hemorrhage. Fourth, we could not reliably assess proper face-down positioning, as only verbal confirmation from the patient was provided during clinical visit. These factors may limit the overall generalizability of the results.

In conclusion, we have presented a case in which a combination of same-day PD with appropriate face-down positioning and intravitreal faricimab successfully managed acute large SMH (>4DD) with a large serous PED in a patient with presumed PCV. The minimal subfoveal heme thickness and rapid PED resolution provide evidence of anatomical improvement and support the combined benefits of mechanical and pharmacological treatments. This case presents a scenario where rapid fluid resolution is particularly significant for prognosis, and highlights the importance of understanding how quickly various mechanical displacement techniques or intravitreal agents can “dry” subretinal, intraretinal or sub-RPE fluid. We acknowledge that this case report represents only a single patient, and the favorable visual outcomes reflect the optimal anatomical factors and timing of treatment. Future prospective observational or controlled trials should aim to stratify outcomes by SMH size, thickness to clarify which patients would benefit most from combined treatment versus faricimab monotherapy. Age- and comorbidity-stratified analyses would help to identify patient subgroups most responsive to Ang-2 inhibition. Direct comparative studies of PD and faricimab versus PD combined with other anti-VEGF agents are needed, and would provide greater insight into the comparative efficacy of faricimab’s dual inhibition compared to other traditional anti-VEGF agents in treating SMH.

Abbreviations

Ang-2	Angiopoietin-2
BCVA	Best-corrected visual acuity
CF	Counting fingers

CNV	Choroidal neovascular membrane
DD	Disc diameters
nAMD	Neovascular age-related macular degeneration
OCT	Optical coherence tomography
PCV	Polypoidal choroidal vasculopathy
PD	Pneumatic displacement
PED	Pigment epithelial detachment
RCT	Randomized controlled trial
RPE	Retinal pigment epithelium
SF ₆	Sulfur hexafluoride
SMH	Submacular hemorrhage
tPA	Tissue plasminogen activator
VEGF	Vascular endothelial growth factor
VH	Vitreous hemorrhage

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Author contributions

All listed authors meet the International Committee of Medical Journal Editors criteria. PXJ: Writing – original draft, data analysis, review and editing, conceptualization. JEH: Writing – original draft, data analysis, review and editing, conceptualization. NS: Writing – original draft, data analysis, review and editing, conceptualization, supervision, methodology.

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Data availability

All data generated or analyzed during this study are included in this article and/or its online supplementary material. Further inquiries can be directed to the corresponding author.

Declarations

Ethics approval and consent to participate

This study was performed in accordance with the Declaration of Helsinki. Ethics approval for this human study was waived by University of Toronto Research Ethics Board. Written informed consent was obtained from the individual for publication of the details of their medical case and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief.

Consent for publication

Written informed consent was obtained from the individual for publication of the details of their medical case and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief.

Competing interests

The authors declare no competing interests.

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References

1. Avery RL, Fekrat S, Hawkins BS, Bressler NM. Natural history of subfoveal, sub-retinal hemorrhage in age-related macular degeneration. *Retina*. 1996;16:183–9. <https://doi.org/10.1097/00006982-199616030-00001>
2. Khanna S, Komati R, Eichenbaum DA, Hariprasad I, Ciulla TA, Hariprasad SM. Current and upcoming anti-VEGF therapies and dosing strategies for the treatment of neovascular AMD: a comparative review. *BMJ Open Ophthalmol*. 2019;4:e000398. <https://doi.org/10.1136/bmjophth-2019-000398>.
3. Gabrielle P-H, Delyfer M-N, Glacet-Bernard A, Conart JB, Uzzan J, Kodjikian L, et al. Surgery, Tissue Plasminogen Activator, Antiangiogenic Agents, and Age-Related Macular Degeneration Study. *Ophthalmology*. 2023;130:947–57. <https://doi.org/10.1016/j.ophtha.2023.04.014>.
4. Chaudhary V, Mar F, Amador MJ, Chang A, Gibson K, Jousseaume AM, et al. Emerging clinical evidence of a dual role for Ang-2 and VEGF-A blockade with faricimab in retinal diseases. *Graefes Arch Clin Exp Ophthalmol*. 2025;263:1239–47. <https://doi.org/10.1007/s00417-024-06695-4>.
5. Jousseaume AM, Ricci F, Paris LP, Korn C, Quezada-Ruiz C, Zarbin M. Angiopoietin/Tie2 signalling and its role in retinal and choroidal vascular diseases: a review of preclinical data. *Eye*. 2021;35:1305–16. <https://doi.org/10.1038/s41433-020-01377-x>.
6. Otani A, Takagi H, Oh H, Koyama S, Matsumura M, Honda Y. Expressions of angiopoietins and Tie2 in human choroidal neovascular membranes. *Invest Ophthalmol Vis Sci*. 1999;40:1912–20.
7. Cheung CMG, Lai TTY, Teo K, Ruamviboonsuk P, Chen S-J, Kim JE, et al. Polypoidal Choroidal Vasculopathy. *Ophthalmol*. 2021;128:443–52. <https://doi.org/10.1016/j.ophtha.2020.08.006>.
8. Heier JS, Khanani AM, Quezada Ruiz C, Basu K, Ferrone PJ, Brittain C, et al. Efficacy, durability, and safety of intravitreal faricimab up to every 16 weeks for neovascular age-related macular degeneration (TENAYA and LUCERNE): two randomised, double-masked, phase 3, non-inferiority trials. *Lancet*. 2022;399:729–40. [https://doi.org/10.1016/S0140-6736\(22\)00010-1](https://doi.org/10.1016/S0140-6736(22)00010-1).
9. Diack C, Avery RL, Cheung CMG, Csaky KG, Gibiansky L, Jaminion F, et al. Ocular Pharmacodynamics of Intravitreal Faricimab in Patients With Neovascular Age-Related Macular Degeneration or Diabetic Macular Edema. *Transl Vis Sci Technol*. 2024;13:13. <https://doi.org/10.1167/tvst.13.11.13>.
10. Cheng AM, Joshi S, Banoub RG, Saddemi J, Chalam KV. Faricimab Effectively Resolves Intraretinal Fluid and Preserves Vision in Refractory, Recalcitrant, and Nonresponsive Neovascular Age-Related Macular Degeneration. *Cureus*. 2023;15:e40100. <https://doi.org/10.7759/cureus.40100>.
11. Schmidt-Erfurth U, Chong V, Loewenstein A, Larsen M, Souied E, Schlingemann R, et al. Guidelines for the management of neovascular age-related macular degeneration by the European Society of Retina Specialists (EURETINA). *Br J Ophthalmol*. 2014;98:1144–67. <https://doi.org/10.1136/bjophthalmol-2014-305702>.
12. Iapoco C, Jennifer I, Lim MD. Effect of faricimab on nAMD with pigment epithelial detachments | HCPLive 2025. <https://www.hcplive.com/view/jennifer-i-lim-md-effect-faricimab-namd-persistent-epithelial-detachments>. (Accessed September 14, 2025).
13. Kovacs KD, Gonzalez LA, Mahrous MA, Orlin A, Papakostas TD, D'Amico DJ, et al. Choroidal Neovascularization and Macular Hemorrhage: Real-World Experience During the New York City COVID-19 Lockdown. *J Vitreoretin Dis*. 2021;5:525–30. <https://doi.org/10.1177/2474126421993664>.
14. Jeong S, Park D-G, Sagong M. Management of a Submacular Hemorrhage Secondary to Age-Related Macular Degeneration: A Comparison of Three Treatment Modalities. *JCM*. 2020;9:3088. <https://doi.org/10.3390/jcm9103088>.
15. Kitahashi M, Sakurai M, Yokouchi H, Kubota-Tani M, Mitamura Y, Yamamoto S, et al. Pneumatic displacement with intravitreal bevacizumab for massive submacular hemorrhage due to polypoidal choroidal vasculopathy. *OPHTH*. 2014;485. <https://doi.org/10.2147/OPHTH.S55413>.
16. Höhn F, Mirshahi A, Hattenbach L-O. Kombinierte intravitreale Injektion von Bevacizumab und SF6-Gas bei AMD-assoziiierter, submakulärer Hämorrhagie. *Ophthalmologie*. 2010;107:328–32. <https://doi.org/10.1007/s00347-009-2004-3>.
17. Ota H, Takeuchi J, Nonogaki R, Tamura K, Kominami T. Pneumatic displacement and anti-VEGF therapy for submacular hemorrhage in neovascular age-related macular degeneration: a retrospective study. *JCM*. 2025;14(3154). <https://doi.org/10.3390/jcm14093154>.
18. Sivachandran N, Ji PX, Khan H, Pickel L, Mojumder O, Aziz AA, et al. Visual and Anatomic Outcomes of Faricimab in Naïve Neovascular Age-Related Macular Degeneration with Subretinal Hemorrhage: A Multi-Centre Retrospective Analysis. *Clin Ophthalmol*. 2026;20:589703. <https://doi.org/10.2147/OPHTH.S589703>.
19. Lim J, Cho H, Ramkumar H, Adrean SD. Neovascular age-related macular degeneration complicated by large submacular hemorrhage managed with faricimab monotherapy. *Int J Retina Vitreous*. 2025;11:142. <https://doi.org/10.1186/s40942-025-00771-5>.
20. Yanai R, Murao F, Miki A, Terasaki H, Chujo S, Sakaeda Y, et al. 6-month outcomes of intravitreal faricimab injection for neovascular age-related macular degeneration and their relationships with clinical findings: a multicentre cohort study from the J-CREST. *BMJ Open Ophthalmol*. 2025;10:e002415. <https://doi.org/10.1136/bmjophth-2025-002415>.
21. Hattenbach L-O, Klais C, Koch FHJ, Gümbel HOC. Intravitreal injection of tissue plasminogen activator and gas in the treatment of submacular hemorrhage under various conditions. *Ophthalmology*. 2001;108:1485–92. [https://doi.org/10.1016/S0161-6420\(01\)00648-0](https://doi.org/10.1016/S0161-6420(01)00648-0).
22. Shin JY, Lee J, Byeon SH. Anti-Vascular Endothelial Growth Factor With or Without Pneumatic Displacement for Submacular Hemorrhage. *Am J Ophthalmol*. 2015;159:904–e9141. <https://doi.org/10.1016/j.ajo.2015.01.024>.

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