Review Article

Anti-VEGF injections in retina practice: where do we stand today?

Abstract

The advent of intravitreal anti-vascular endothelial growth factor (anti-VEGF) therapy has revolutionized the management of retinal vascular and neovascular diseases over the past two decades. Diseases that once led inevitably to severe visual loss, including neovascular age-related macular degeneration (nAMD), diabetic macular edema (DME), retinal vein occlusion (RVO), myopic choroidal neovascularization (mCNV), and retinopathy of prematurity (ROP), can now be effectively controlled with repeated intravitreal pharmacotherapy. The evolution from first-generation agents such as pegaptanib and bevacizumab to newer molecules including aflibercept, brolucizumab, faricimab, and port delivery systems has transformed treatment paradigms toward longer durability and personalized regimens. Despite remarkable success, challenges remain regarding treatment burden, real-world adherence, tachyphylaxis, cost, systemic safety, and inequitable access in developing countries. Emerging innovations including bispecific antibodies, gene therapy, sustained-release implants, biosimilars, artificial intelligence-guided retreatment, and home monitoring technologies are reshaping the future landscape of retinal therapeutics.

Keywords: Anti-VEGF, intravitreal injections, age-related macular degeneration, diabetic macular edema, retinal vein occlusion, faricimab, aflibercept, retina

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Abbreviations: anti-VEGF, anti-vascular endothelial growth factor; nAMD, neovascular age-related macular degeneration; PlGF, placental growth factor; DME, diabetic macular edema; RVO, retinal vein occlusion; mCNV, myopic choroidal neovascularization.

Introduction

Retinal vascular and neovascular disorders remain among the leading causes of irreversible visual impairment worldwide.[1,2] Conditions such as Age-related Macular Degeneration, Diabetic Macular Edema, Retinal Vein Occlusion, myopic choroidal neovascularization, and proliferative diabetic retinopathy collectively contribute to a major global burden of blindness, particularly in the aging and diabetic populations.[1,2] Before the advent of intravitreal pharmacotherapy, therapeutic strategies for these disorders were largely limited to thermal laser photocoagulation, transpupillary thermotherapy, photodynamic therapy, intravitreal corticosteroids, and vitreoretinal surgery.[3] Although these modalities occasionally stabilized disease progression, meaningful visual recovery was uncommon, and many patients continued to suffer progressive central vision loss despite treatment.

The discovery of vascular endothelial growth factor (VEGF) as a pivotal mediator of ocular angiogenesis and vascular permeability marked a turning point in retinal therapeutics.[4,5] VEGF is a signaling glycoprotein that plays an essential physiological role in embryogenesis, wound healing, endothelial cell survival, and maintenance of normal vascular function. However, pathological overexpression of VEGF within the retina and choroid results in abnormal neovascularization, increased vascular permeability, breakdown of the blood-retinal barrier, inflammatory cascade activation, and subsequent macular edema. Elevated intraocular VEGF levels have been demonstrated in several retinal diseases, establishing VEGF inhibition as a rational and highly targeted therapeutic approach.[4–6]

The development of anti-VEGF agents revolutionized ophthalmology in a manner rarely witnessed in any medical specialty. The approval of pegaptanib sodium in 2004 initiated the anti-VEGF era,[7] but the subsequent introduction of intravitreal bevacizumab, ranibizumab, and aflibercept transformed the prognosis of retinal diseases that were once considered inevitably blinding.[8–10] Large multicentric clinical trials demonstrated unprecedented visual gains, with many patients not only maintaining but also improving visual acuity after treatment.[8–10] Anti-VEGF therapy rapidly became the gold standard for neovascular retinal diseases and remains the cornerstone of modern retina practice today.

Over the past two decades, the field of retinal therapeutics has evolved from simple VEGF blockade toward increasingly sophisticated and individualized treatment strategies. Newer agents such as brolucizumab and faricimab have focused on improving durability, reducing treatment burden, and targeting additional angiogenic pathways beyond VEGF alone.[11–13] Simultaneously, advances in retinal imaging, especially Optical Coherence Tomography and OCT angiography, have enabled precise disease monitoring and personalized retreatment protocols.[14]

Despite remarkable therapeutic success, several challenges continue to influence real-world outcomes. The requirement for frequent intravitreal injections imposes a substantial burden on patients, caregivers, and healthcare systems. Poor adherence, undertreatment, socioeconomic disparities, cost considerations, tachyphylaxis, and concerns regarding long-term ocular and systemic safety remain important limitations of current anti-VEGF therapy. Furthermore, real-world studies consistently reveal visual outcomes inferior to those achieved in controlled clinical trials, emphasizing the gap between efficacy and effectiveness in routine clinical practice.[14]

In developing countries such as India, the anti-VEGF revolution has been both transformative and challenging. While off-label bevacizumab has significantly improved accessibility and affordability, issues related to repeated treatment costs, healthcare infrastructure, follow-up compliance, and unequal distribution of retina services continue to affect patient care.[9] At the same time, the emergence of biosimilars and cost-effective therapeutic alternatives may help bridge these gaps in the near future.

Currently, retina specialists stand at a critical juncture where the goals of therapy are expanding beyond short-term visual improvement toward long-term disease modulation, reduced treatment burden, enhanced durability, and precision medicine. Innovations including sustained-release drug delivery systems, gene therapy, artificial intelligence-assisted monitoring, home-based OCT technologies, and novel molecular targets are rapidly reshaping the future landscape of retinal therapeutics.[12,13]

Biology of VEGF

The VEGF family consists of several related proteins, including VEGF-A, VEGF-B, VEGF-C, VEGF-D, VEGF-E, and placental growth factor (PlGF), among which VEGF-A is the principal isoform implicated in ocular diseases.[4,5] VEGF-A itself undergoes alternative mRNA splicing, producing multiple isoforms such as VEGF121, VEGF145, VEGF165, VEGF189, and VEGF206. Among these, VEGF165 is considered the most biologically active and pathogenic isoform in retinal vascular diseases because of its potent angiogenic and vascular permeability-enhancing properties.[4,5] These isoforms differ in tissue diffusibility and extracellular matrix binding, thereby influencing their biological behavior within ocular tissues.

VEGF exerts its actions through interaction with specific transmembrane tyrosine kinase receptors located predominantly on vascular endothelial cells, namely VEGFR-1, VEGFR-2, and VEGFR-3.[4,5] VEGFR-2 is regarded as the principal mediator of pathological angiogenesis and vascular leakage in retinal diseases.[4,5] Activation of VEGFR-2 stimulates endothelial cell proliferation, migration, nitric oxide production, increased vascular permeability, and endothelial survival through several intracellular signalling pathways including the MAPK/ERK, PI3K/Akt, and phospholipase C pathways. VEGFR-1 contributes to inflammatory regulation, monocyte recruitment, and modulation of angiogenesis, whereas VEGFR-3 is mainly associated with lymph angiogenesis and has a comparatively limited role in retinal vascular pathology.

Under physiological conditions, VEGF performs several critical functions within the eye. Retinal pigment epithelial cells constitutively secrete VEGF to maintain the integrity and survival of the choriocapillaris.[15] VEGF also exhibits neuroprotective properties for retinal neurons and contributes to wound healing and vascular repair following tissue injury.

In pathological states, retinal hypoxia and ischemia serve as the most important stimuli for VEGF overexpression. Hypoxic retinal tissue activates hypoxia-inducible factor-1 alpha (HIF-1α), which subsequently enhances VEGF gene transcription.[16] Elevated intraocular VEGF levels have been demonstrated in diseases such as Age-related Macular Degeneration, Diabetic Macular Edema, proliferative diabetic retinopathy, Retinal Vein Occlusion, myopic choroidal neovascularization, and Retinopathy of Prematurity.[4–6] VEGF-induced endothelial dysfunction disrupts tight junction proteins such as occludin and claudin within the blood-retinal barrier, resulting in increased vascular permeability, plasma leakage, and accumulation of intraretinal and subretinal fluid, ultimately causing macular edema. Simultaneously, VEGF promotes endothelial proliferation and migration, leading to the formation of fragile abnormal neovascular complexes that are prone to leakage, hemorrhage, fibrosis, and tractional complications.

In addition to its angiogenic role, VEGF also acts as a pro-inflammatory mediator.[5] It enhances leukocyte adhesion, cytokine release, endothelial dysfunction, and inflammatory cascade activation, thereby linking inflammation with angiogenesis in retinal diseases. This evolving understanding of retinal vascular biology has led to the development of newer therapeutic strategies targeting multiple pathways beyond VEGF alone. The angiopoietin-Tie2 pathway, particularly angiopoietin-2 (Ang-2), has emerged as an important contributor to vascular instability and inflammation.[12,13] Elevated Ang-2 levels destabilize retinal vasculature and amplify VEGF-mediated leakage and neovascularization. This concept formed the basis for the development of faricimab, the first bispecific antibody targeting both VEGF-A and Ang-2 pathways simultaneously.[12,13]

The molecular understanding of VEGF biology paved the way for anti-VEGF therapeutics, which act either by directly binding VEGF molecules, blocking receptor activation, or functioning as decoy receptors (Tables 1–3).

Table 1 Evolution of anti-VEGF therapy

Anti-VEGF agent	Evolution / generation	Mechanism & role	Major ophthalmic indications
Pegaptanib[7]	First FDA-approved anti-VEGF agent (2004); first-generation aptamer	Selectively binds VEGF-165 isoform; reduces neovascularization and vascular leakage	Neovascular AMD
Bevacizumab[9]	Full-length monoclonal antibody initially developed for colorectal carcinoma; off-label ophthalmic use	Binds all VEGF-A isoforms; decreases vascular permeability and neovascularization	Neovascular AMD, DME, RVO, myopic CNV, proliferative diabetic retinopathy, neovascular glaucoma, ROP
Ranibizumab[8]	Designed specifically for intraocular use; second-generation anti-VEGF	Humanized monoclonal antibody fragment targeting all VEGF-A isoforms	Neovascular AMD, DME, RVO, myopic CNV, diabetic retinopathy
Aflibercept[10]	Third-generation anti-VEGF; introduced for longer durability	Recombinant fusion protein acting as decoy receptor for VEGF-A, VEGF-B, and PlGF	Neovascular AMD, DME, RVO, diabetic retinopathy, myopic CNV
Brolucizumab[11]	Newer-generation single-chain antibody fragment designed for extended dosing intervals	Potent VEGF-A inhibition with high molar concentration and better tissue penetration	Neovascular AMD
Faricimab[12,13]	Latest generation bispecific antibody targeting dual pathways	Simultaneously inhibits VEGF-A and angiopoietin-2, improving vascular stability	Neovascular AMD, DME

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Table 1 Continued...

Conbercept[10]	Developed mainly in China as modified VEGF trap	Binds VEGF-A, VEGF-B, VEGF-C, and PlGF	AMD, DME, RVO
Abicipar Pegol[11]	Designed as long-acting DARPin molecule	Potent VEGF-A inhibition with prolonged intraocular durability	Investigational for neovascular AMD
Anti-VEGF Biosimilars[20]	Recent evolution to improve affordability and accessibility	Similar mechanism to parent molecule; VEGF inhibition	Same as parent compounds depending on approval

Table 2 Advantages and limitations

Anti-VEGF agent	Advantages	Disadvantages / limitations
Pegaptanib[7]	Historically important; selective inhibition theoretically preserves physiological VEGF activity	Inferior efficacy compared with pan-VEGF inhibitors; rarely used currently
Bevacizumab[9]	Very low cost; widely available; effective in multiple retinal diseases	Off-label use; compounding-related contamination risk; shorter durability
Ranibizumab[8]	Excellent safety profile; high retinal penetration; extensive evidence	Expensive; frequent injections required
Aflibercept[10]	Longer duration; stronger binding affinity; better drying effect	Expensive; repeated injections still needed
Brolucizumab[11]	Longer dosing intervals; superior retinal drying in some cases	Concerns regarding retinal vasculitis and vascular occlusion
Faricimab[12,13]	Longer durability up to 16 weeks; dual pathway inhibition	Long-term real-world data still evolving; expensive
Conbercept[10]	Longer half-life; broad VEGF blockade	Limited global availability; fewer international trials
Abicipar Pegol[11]	Longer dosing intervals	High intraocular inflammation rates
Anti-VEGF Biosimilars[20]	Lower cost; improved accessibility	Limited long-term data for some molecules

Table 3 Contraindications and adverse effects

Anti-VEGF agent	Contraindications / precautions	Ocular side effects	Systemic adverse effects
Pegaptanib[7,17]	Ocular infection, active inflammation, hypersensitivity	Endophthalmitis, transient IOP rise, vitreous floaters	Rare thromboembolic events
Bevacizumab[9,17,18]	Active ocular/periocular infection, severe ocular inflammation	Endophthalmitis, sterile inflammation, retinal detachment, traumatic cataract	Hypertension, stroke, myocardial infarction
Ranibizumab[8,17]	Active ocular infection, intraocular inflammation	Endophthalmitis, vitreous hemorrhage, ocular pain	Small risk of arterial thromboembolic events
Aflibercept[10,17]	Ocular infection, inflammation, hypersensitivity	RPE tear, increased IOP, intraocular inflammation	Rare hypertension and thromboembolic phenomena
Brolucizumab[11]	History of retinal vasculitis or active inflammation	Severe intraocular inflammation, retinal vascular occlusion	Rare systemic thromboembolic risk
Faricimab[12,13]	Active ocular infection, inflammation, hypersensitivity	Conjunctival hemorrhage, blurred vision, endophthalmitis	Potential arterial thromboembolic events
Conbercept[10]	Similar to other anti-VEGF agents	Similar injection-related complications	Similar systemic vascular risks
Anti-VEGF Biosimilars[20]	Same as parent drugs	Similar injection-related adverse effects	Similar systemic risks

Real-world challenges of anti-VEGF therapy

Despite the extraordinary success of intravitreal anti-vascular endothelial growth factor (anti-VEGF) therapy in transforming the prognosis of retinal diseases, several real-world challenges continue to limit optimal outcomes in routine clinical practice. Although randomized controlled trials have consistently demonstrated impressive visual gains in conditions such as Age-related Macular Degeneration, Diabetic Macular Edema, and Retinal Vein Occlusion, real-world studies frequently report inferior visual outcomes compared with those achieved in clinical trials.[14] One of the most significant reasons for this discrepancy is undertreatment. In controlled studies, patients undergo strict monthly monitoring and receive protocol-driven injections, whereas in real-life practice many patients fail to maintain the required treatment frequency because of financial constraints, travel difficulties, limited caregiver support, systemic illness, or poor understanding of the chronic nature of retinal diseases.

The burden associated with repeated intravitreal injections remains one of the greatest limitations of current anti-VEGF therapy.[14] Most retinal diseases require long-term, often lifelong treatment with frequent follow-up visits and serial imaging using Optical Coherence Tomography. This imposes considerable psychological, social, logistical, and economic stress on patients and caregivers. Elderly patients with neovascular AMD often have multiple systemic comorbidities and mobility limitations, making frequent hospital visits difficult. Similarly, diabetic patients frequently struggle with multiple medical appointments and systemic complications, further affecting adherence to retinal treatment schedules. As treatment intervals increase or appointments are missed, recurrent fluid

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accumulation and irreversible photoreceptor damage may occur, ultimately compromising long-term visual outcomes.

Economic burden represents another major challenge, particularly in low- and middle-income countries such as India.[14] Although off-label bevacizumab has significantly improved affordability and accessibility, repeated injections over many years still create substantial financial strain for patients and healthcare systems. Branded agents such as ranibizumab, aflibercept, and faricimab may be prohibitively expensive for many patients without adequate insurance coverage. In addition, disparities in healthcare infrastructure, limited availability of retina specialists, and inadequate access to advanced retinal imaging further widen the gap between ideal and achievable care in resource-constrained regions.

Another important concern is variability in treatment response. While many patients demonstrate excellent anatomical and functional improvement, others exhibit incomplete response, persistent intraretinal or subretinal fluid, recurrent edema, or progressive fibrosis despite regular therapy. Tachyphylaxis and tolerance to anti-VEGF agents have also been described, wherein the therapeutic response diminishes over time.[18] In such cases, switching between different anti-VEGF molecules or combining therapies with corticosteroids, laser photocoagulation, or surgical intervention may be necessary. Furthermore, chronic retinal diseases often involve multiple pathogenic mechanisms beyond VEGF alone, including inflammation, fibrosis, oxidative stress, and vascular instability, which may explain incomplete responses in some patients.

Long-term safety concerns also continue to generate discussion.[17,19] Although intravitreal anti-VEGF injections are generally safe, cumulative exposure over several years raises concerns regarding sustained elevation of intraocular pressure, retinal pigment epithelial atrophy, geographic atrophy progression in AMD, and potential systemic vascular effects. Repeated injections also increase the cumulative lifetime risk of injection-related complications such as endophthalmitis, retinal tears, and intraocular inflammation. In addition, newer agents such as brolucizumab highlighted the importance of post-marketing surveillance after reports of retinal vasculitis and occlusive retinal vasculopathy emerged following widespread clinical use.[11]

Another real-world challenge involves patient counselling and expectation management. Anti-VEGF therapy controls disease activity but does not cure the underlying pathology. Many patients expect complete restoration of vision after a few injections and may become discouraged when long-term treatment is required. Maintaining adherence over several years therefore requires extensive patient education, counselling, and continuous physician-patient communication.

Emerging advances in anti-VEGF therapy

The limitations associated with conventional anti-VEGF therapy have driven intense research toward developing more durable, effective, and personalized retinal treatment strategies. One of the most important recent advances is the development of longer-acting anti-VEGF molecules designed to reduce injection frequency while maintaining efficacy. Faricimab represents a major innovation in this regard because it simultaneously targets VEGF-A and angiopoietin-2 (Ang-2), thereby addressing both angiogenesis and vascular instability.[12,13] Clinical trials such as TENAYA, LUCERNE, YOSEMITE, and RHINE demonstrated that many patients could be maintained on treatment intervals of up to 16 weeks, significantly reducing treatment burden.

Sustained drug-delivery systems are another promising advancement aimed at minimizing the need for repeated intravitreal injections. The port delivery system (PDS) with ranibizumab was developed as an implantable intraocular reservoir capable of continuously releasing medication over extended periods.[20] Although challenges related to surgical implantation and device-related complications remain, such technologies represent an important step toward long-term retinal drug delivery. Biodegradable implants, nanoparticle-based delivery systems, hydrogels, and refillable reservoirs are also under active investigation.

Gene therapy is emerging as one of the most revolutionary future approaches in retinal therapeutics.[20] Instead of repeated injections, gene therapy aims to enable retinal cells to continuously produce anti-VEGF proteins after a single administration. Viral vector-mediated delivery systems using adeno-associated viruses have shown encouraging early results in clinical trials. If proven safe and effective, gene therapy may fundamentally alter the management paradigm of chronic retinal diseases by potentially providing sustained therapeutic effects with minimal retreatment requirements.

Another important advancement is the rise of biosimilars, which are expected to improve accessibility and reduce healthcare expenditure globally. Biosimilar versions of ranibizumab and aflibercept are increasingly entering the market, especially in developing nations where treatment affordability remains a major barrier. Wider availability of cost-effective biosimilars may significantly enhance long-term adherence and visual outcomes in resource-limited settings.

Advances in retinal imaging and artificial intelligence (AI) are also reshaping the future of anti-VEGF therapy.[16,20] Modern imaging modalities such as OCT angiography allow detailed visualization of retinal and choroidal vasculature without dye injection, enabling earlier diagnosis and precise monitoring of disease activity. Artificial intelligence algorithms are being developed to analyse retinal imaging, predict disease progression, determine optimal retreatment intervals, and identify early recurrence. AI-assisted decision-making may eventually facilitate personalized treatment strategies and reduce unnecessary clinic visits.

Home-based retinal monitoring technologies are another emerging innovation.[20] Portable home OCT devices and smartphone-based visual monitoring systems may enable early detection of recurrence and improve long-term disease surveillance, especially for elderly patients with limited mobility. Teleophthalmology platforms may further improve access to retinal care in underserved areas.

Research is also expanding beyond VEGF inhibition toward targeting additional molecular pathways involved in retinal disease pathogenesis. New therapeutic strategies focusing on complement inhibition, inflammation modulation, fibrosis prevention, and neuroprotection are currently under evaluation. Combination therapies targeting multiple pathways simultaneously may eventually overcome limitations associated with VEGF monotherapy and improve outcomes in refractory or chronic disease.[12,13,20]

Thus, the field of retinal therapeutics is rapidly transitioning from conventional repetitive injection-based therapy toward a future characterized by longer durability, molecular precision, personalized medicine, advanced imaging integration, and potentially curative approaches. These emerging advances hold immense promise for reducing treatment burden, improving patient adherence, expanding accessibility, and ultimately enhancing long-term visual outcomes worldwide.

Conclusion

Anti-VEGF therapy remains the gold standard for managing retinal vascular and neovascular diseases.[7–13] Tremendous progress has occurred from first-generation agents to sophisticated bispecific antibodies and sustained-release systems.[7–13,20] Current retina practice emphasizes individualized treatment protocols that balance efficacy, durability, cost, and safety.

While anti-VEGF therapy has dramatically improved visual prognosis worldwide, significant unmet needs persist. The future lies in reducing treatment burden, enhancing durability, integrating artificial intelligence, and expanding global accessibility.[12,13,16,20] The journey of anti-VEGF therapy reflects not only scientific innovation but also the evolving philosophy of retinal care—from disease control to precision vision preservation.

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Conflict of interests

The authors declare that they have no known competing financial interests or personal relationships that appeared to influence the work reported in this study.

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References

1. Bourne RRA, Steinmetz JD, Flaxman S, et al. Trends in prevalence of blindness and distance and near vision impairment over 30 years: an analysis for the global burden of disease study. *Lancet Glob Health*. 2021;9(2):e130–e143.
2. Wong TY, Cheung CMG, et al. Diabetic retinopathy. *Nat Rev Dis Primers*. 2016;2:16012.
3. Treatment of age-related macular degeneration with photodynamic therapy (TAP) Study Group. Photodynamic therapy of subfoveal choroidal neovascularization in age-related macular degeneration with verteporfin: one-year results of 2 randomized clinical trials—TAP report 1. *Arch Ophthalmol*. 1999;117(10):1329–1345.
4. Ferrara N. Vascular endothelial growth factor: basic science and clinical progress. *Endocr Rev*. 2004;25(4):581–611.
5. Ferrara N, Adamis AP. Ten years of anti-vascular endothelial growth factor therapy. *Nat Rev Drug Discov*. 2016;15(6):385–403.
6. Aiello LP, Avery RL, Arrigg PG, et al. Vascular endothelial growth factor in ocular fluid of patients with diabetic retinopathy and other retinal disorders. *N Engl J Med*. 1994;331(22):1480–1487.
7. Gragoudas ES, Adamis AP, Cunningham ET Jr, et al; VEGF Inhibition Study in Ocular Neovascularization Clinical Trial Group. Pegaptanib for neovascular age-related macular degeneration. *N Engl J Med*. 2004;351(27):2805–2816.
8. Brown DM, Kaiser PK, Michels M, et al. Ranibizumab versus verteporfin for neovascular age-related macular degeneration. *N Engl J Med*. 2006;355(14):1432–1444.
9. Comparison of Age-related Macular Degeneration Treatments Trials (CATT) Research Group, Martin DF, Maguire MG, Ying GS, et al. Ranibizumab and bevacizumab for neovascular age-related macular degeneration. *N Engl J Med*. 2011;364(20):1897–1908.
10. Heier JS, Brown DM, Chong V, et al. Intravitreal aflibercept (VEGF trap-eye) in wet age-related macular degeneration. *Ophthalmology*. 2012;119(12):2537–2548.
11. Dugel PU, Koh A, Ogura Y, et al. HAWK and HARRIER: phase 3, multicenter, randomized, double-masked trials of brolucizumab for neovascular age-related macular degeneration. *Ophthalmology*. 2020;127(1):72–784.
12. Khanani AM, Guymer RH, Basu K, 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(10326):729–740.
13. Wykoff CC, Abreu F, Adamis AP, et al. Efficacy, durability, and safety of intravitreal faricimab with extended dosing up to every 16 weeks in diabetic macular oedema (YOSEMITE and RHINE): two randomised, double-masked, phase 3 trials. *Lancet*. 2022;399(10326):741–755.
14. Ciulla TA, Hussain RM, Pollack JS, et al. Visual acuity outcomes and anti-vascular endothelial growth factor therapy intensity in real-world neovascular age-related macular degeneration patients: a systematic review and meta-analysis. *Ophthalmol Retina*. 2020;4(1):1–10.
15. Saint-Geniez M, Kurihara T, Sekiyama E, et al. An essential role for RPE-derived soluble VEGF in the maintenance of the choriocapillaris. *Proc Natl Acad Sci U S A*. 2009;106(44):18751–18756.
16. Semenza GL. Hypoxia-inducible factor 1 and disease pathophysiology. *IUBMB Life*. 2007;59(10):591–597.
17. Avery RL, Gordon GM. Systemic safety of prolonged monthly anti-vascular endothelial growth factor therapy for diabetic macular edema: a systematic review and meta-analysis. *Retina*. 2016;36(1):1–9.
18. Forooghian F, Cukras C, Meyerle CB, et al. Tachyphylaxis after intravitreal bevacizumab for exudative age-related macular degeneration. *Retina*. 2009;29(6):723–731.
19. Grunwald JE, Pistilli M, Ying GS, et al. Growth of geographic atrophy in the comparison of age-related macular degeneration treatments trials. *Ophthalmology*. 2015;122(4):809–816.
20. Campochiaro PA, Wykoff CC, Singer M, et al. Port delivery system with ranibizumab for neovascular age-related macular degeneration (ARCHWAY): a phase 3, multicentre, randomised, open-label trial. *Lancet*. 2021;398(10312):1795–1806.

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