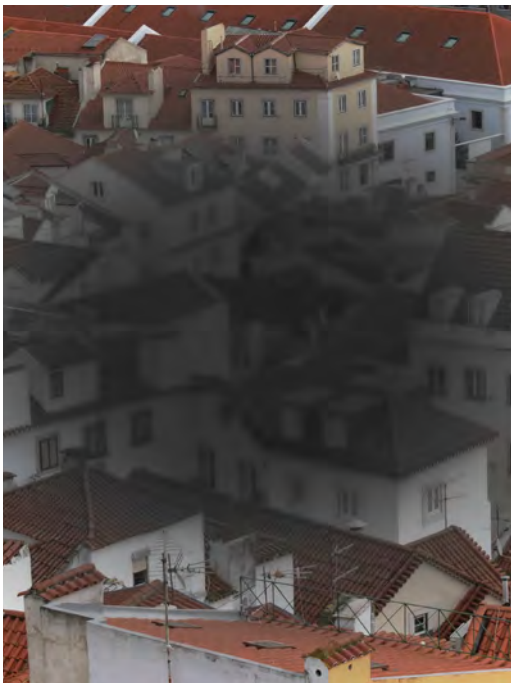

Age-related macular degeneration: Updates for general practitioners

Carl Eiselen, Marita Long, Elaine W Chong

Background
Age-related macular degeneration (AMD) is the leading cause of legal blindness in Australians over 50 years, accounting for 60% of all blindness. AMD progresses from asymptomatic early and intermediate stages to late neovascular ('wet') or atrophic ('dry') forms that cause irreversible central vision loss. Age and family history are the strongest risk factors, while smoking, poor diet and cardiovascular disease are key modifiable contributors.

Objective
To raise awareness of AMD in general practice and provide some advice on what general practitioners (GPs) can do to assist in early detection and prevention with an ageing population. GPs can examine the macula in at-risk adults, as well as encourage smoking cessation, a Mediterranean-style diet, regular exercise, and Age-Related Eye Disease Study 2 (AREDS2) supplementation for intermediate disease. Sudden distortion or vision loss requires urgent ophthalmology referral for possible neovascular AMD.

Discussion
Intravitreal anti-VEGF (vascular endothelial growth factor) therapy remains the mainstay for wet AMD, while complement inhibitors show promise for geographic atrophy. Emerging gene, sustained-delivery and laser therapies might soon reduce treatment burden and improve adherence.

AGE-RELATED MACULAR DEGENERATION (AMD) is the leading cause of legal blindness in people over 50 years, with AMD contributing to 60% of all blindness in Australia.[1] AMD predominantly affects the macula, this often results in significant central vision loss, which has consequences for driving, reading and detailed tasks. Clinically, AMD progresses from early to intermediate stages characterised by the accumulation of drusen (yellow extracellular deposits beneath the retinal pigment epithelium) to late-stage disease. Late AMD has two forms that often coexist: neovascular ('wet') AMD, defined by choroidal neovascularisation which leads to haemorrhage and fluid exudation, and atrophic ('dry') AMD featuring progressive geographic atrophy of the retinal pigment epithelium and photoreceptors (Figure 1). Early and intermediate AMD might be asymptomatic or cause only mild visual disturbances, whereas late AMD leads to significant central vision loss. Given the growing elderly population and lack of cure for advanced stages, AMD poses an expanding public health and socioeconomic burden.

Objective

The general practitioner's (GP's) role encompasses detecting risk factors, recognising early symptoms, coordinating timely referral, and reinforcing lifestyle modifications and treatment adherence. While diagnosis confirmation and treatment remain specialist-led, GPs provide critical first-line assessment and ongoing support to patients.

Risk factors

Risk of AMD is multifactorial with non-modifiable and modifiable risk factors intertwined. Increasing age is the strongest and most consistent risk factor for AMD. A recent meta-analysis of 18 studies confirmed that the likelihood of developing AMD increases by approximately 11% per year of age, with prevalence rising sharply after 60 years.[2] Given Australia's ageing population, this age-related effect underpins the growing burden of AMD in general practice and highlights the importance of proactive screening in older adults.

© The Royal Australian College of General Practitioners 2026

Genetic predisposition also plays a critical part. Common polymorphisms in the complement factor H (CFH), age-related maculopathy susceptibility 2 (ARMS2) and high temperature requirement factor A1 (HTRA1) genes confer approximately two- to three-fold higher odds of AMD.[2] A positive family history (first-degree relative with AMD) roughly triples a person's risk, reflecting the contribution of these heritable factors.[2] Male sex is associated with higher AMD odds (odds ratio [OR] 1.63 vs females) despite the disease's overall higher prevalence in women due to their longevity.[2] Lastly, the strongest prognostic factor for an individual eye is the status of the fellow eye. The presence of any AMD in one eye carries a 19–28% chance of bilateral involvement within 5 years, and late-stage AMD in one eye confers up to a 27–68% risk of late AMD developing in the fellow eye over 5 years.[3] This underscores the need for vigilant monitoring of the contralateral eye in patients with unilateral disease.

Smoking is one of the strongest and important modifiable risk factors (overall pooled OR of 1.86 for smokers vs non-smokers) that also accelerates progression from unilateral to bilateral AMD, with heavy cigarette exposure conferring nearly a three-fold increased risk of both geographic atrophy and neovascular disease.[2–4] Systemic vascular risk factors have been implicated in AMD. Hypertension (24%), cardiovascular disease (44%) and diabetes (44%) significantly increase AMD risk.[2] Given these associations, comprehensive cardiovascular risk management benefits macular health. Optimising blood pressure and lipid profiles according to guidelines is advisable in AMD patients, not only for general health but potentially to mitigate progression.

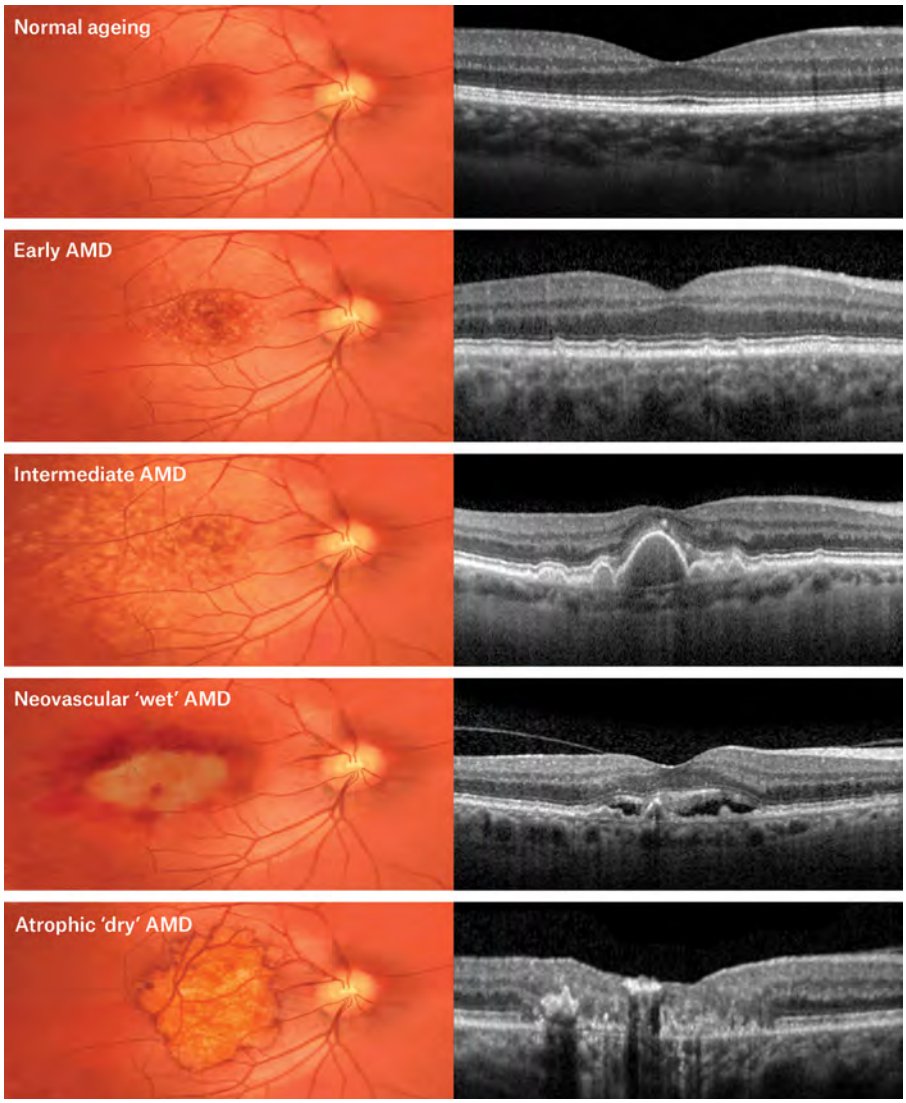

Figure 1. Clinical spectrum of AMD. Colour fundus photographs (left) and corresponding OCT scans (right) demonstrating the progressive structural changes in AMD. Normal ageing shows preserved retinal layers. Early AMD presents with small drusen and mild RPE irregularities. Intermediate AMD exhibits larger drusen with early outer retinal disruption. Neovascular ('wet') AMD is characterised by subretinal haemorrhage and exudation with RPE elevation. Atrophic ('dry') AMD demonstrates confluent geographic atrophy with loss of outer retina and RPE.

Adapted from One Right Eye. One Right Eye, updated 2021 (www.onerighteye.com), with permission from One Right Eye.

AMD, age-related macular degeneration; OCT, optical coherence tomography; RPE, retinal pigment epithelium.

Clinical presentation

Early AMD is typically asymptomatic. Functional impairment begins with delayed dark adaptation, linked to early rod dysfunction, often preceding visible drusen or pigmentary change on exam.[5] Patients might report difficulty seeing in dim light or slower adaptation after glare. Mild metamorphopsia (straight lines or objects appear distorted or wavy) or blurred central vision might develop in intermediate stages but are often subtle.

Routine dilated eye exams are essential in at-risk individuals. While Amsler grid testing and digital versions might detect central distortion, it has limited sensitivity (~50%).[6] Fundoscopy in general practice is challenging and can be limited by time, access to equipment and imaging where early signs might be subtle. Optical coherence tomography (OCT) provides high sensitivity for drusen, subretinal fluid or atrophy and should be arranged via optometry or ophthalmology if AMD is suspected.

© The Royal Australian College of General Practitioners 2026

Timely referral

Suspected wet AMD should be treated as an ophthalmic emergency. In general practice, wet AMD is suspected (rather than diagnosed) by acute visual symptoms – particularly acute metamorphopsia, new central blur, scotoma or rapid vision loss (Figure 2, Red flags). These symptoms warrant urgent referral (within 24 hours) for retinal assessment.[7]

Where immediate ophthalmology access is limited, same-day OCT via optometry can expedite triage. Definitive distinction between wet and dry AMD requires retinal imaging and is not expected to be made in general practice.

Dry AMD does not require urgent referral and is usually managed in the community. Dry AMD progresses with gradual visual decline and might be asymptomatic.

Monitoring can be done by an optometrist with periodic fundus examination and OCT, typically every 6–12 months. Referral to ophthalmology is non-urgent and appropriate for diagnostic uncertainty, unexplained visual loss, or discussing emerging therapies. In Australia, optometrists can also refer directly to ophthalmologists, though GPs commonly coordinate care.

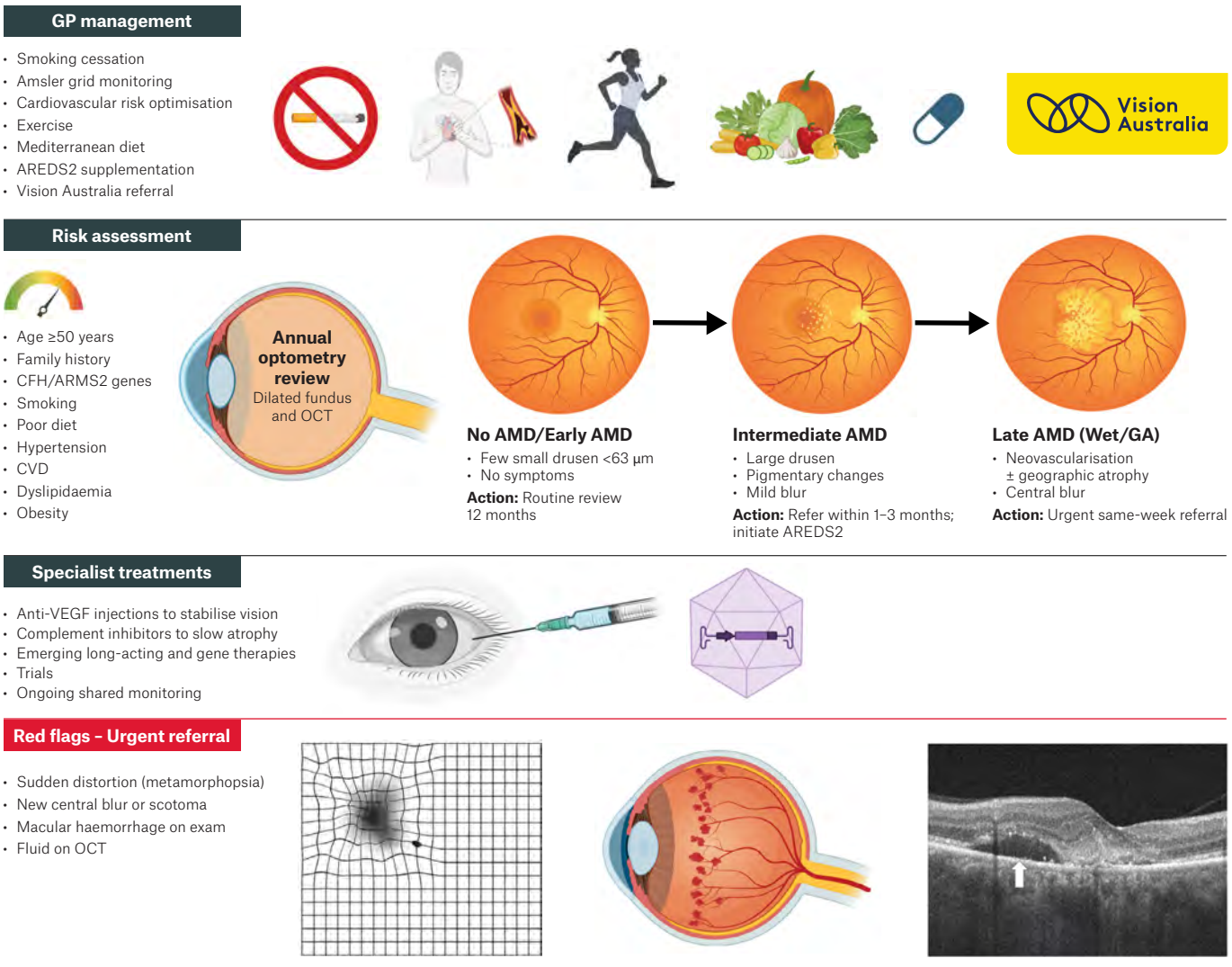

Figure 2. GP referral and management pathway for AMD.

Initial assessment should include history of visual symptoms, smoking, family history, and cardiovascular risk factors. Early and intermediate AMD require optometry review and lifestyle modification, with semi-urgent ophthalmology referral if progression is suspected. Sudden distortion or central vision loss warrants urgent same-week referral for possible neovascular AMD.

AMD, age-related macular degeneration; AREDS2, Age-Related Eye Disease Study 2; ARMS2, age-related maculopathy susceptibility 2; CFH, complement factor H; CVD, cardiovascular disease; GA, geographic atrophy; GP, general practitioner; OCT, optical coherence tomography; VEGF, vascular endothelial growth factor.

© The Royal Australian College of General Practitioners 2026

Box 1. Digital tools and resources for patient education and monitoring

OneRightEye.com – Free interactive 3D animations explaining AMD. GPs can register and use during consultations for visual education.

Macular Disease Foundation Australia – Comprehensive resources, support groups, free Amsler grids (1800 111 709).

Vision Australia – Practical support, technology training, NDIS/My Aged Care funding assistance (1300 847 466).

AMD, age-related macular degeneration; 3D, three-dimensional; GP, general practitioner; NDIS, National Disability Insurance Scheme.

GPs also have a key role in coordinating low-vision support, assessing driving safety and reinforcing symptom vigilance (Box 1). In older patients, any new central visual change should be treated as urgent until neovascular AMD is excluded.

Lifestyle modifications and treatment

Lifestyle modifications form the cornerstone of secondary prevention, which aims to slow progression in patients with early or intermediate AMD by addressing key modifiable risks. Smoking cessation is essential, as cigarette smoking is the strongest modifiable risk factor for AMD: smokers have a two- to four-fold higher risk of neovascular AMD.[2,4] Adherence to a Mediterranean-style diet is also protective: pooled data from European cohorts showed high Mediterranean-diet scores corresponded to ~41% lower incidence of advanced (late) AMD.[8] Leafy greens provide macular carotenoids (lutein and zeaxanthin) that accumulate as protective pigments, filtering blue light and reducing oxidative stress.

Randomised trial data support high-dose supplements in at-risk eyes: the Age-Related Eye Disease Study (AREDS1) (vitamins C, E, β-carotene, zinc, copper) and Age-Related Eye Disease Study 2 (AREDS2) formulations (vitamins C, E, zinc, copper plus lutein/zeaxanthin replacing β-carotene) reduced the 5-year risk of progressing from intermediate to late AMD by ~25%.[9] Importantly, the AREDS2 formula is recommended for smokers and former smokers, since β-carotene formulations significantly raised lung cancer risk in this group.[10] Emerging evidence suggests regular exercise might also be protective. A meta-analysis reported that higher levels of physical activity are associated with lower odds of developing AMD by 8% for early AMD and 41% for late AMD.[11] In summary, evidence-based lifestyle measures to slow AMD progression include smoking cessation, exercise, adherence to a Mediterranean style diet and AREDS2 supplementation (Figure 3).

Treatment strategies for wet and dry AMD is specialist led, with ongoing shared care support from general practice. Wet AMD is treated with regular intravitreal anti-VEGF (vascular endothelial growth factor) injections (such as aflibercept, ranibizumab, faricimab and bevacizumab), which improve or stabilise vision and are listed on the Pharmaceutical Benefits Scheme (PBS).[12] Despite efficacy, adherence is a major limitation, with systematic review data demonstrating 17.5–30% patient-led non-adherence.[13] Leading barriers include travel, carer burden, cost, and dissatisfaction or discomfort with treatment. Newer agents such as faricimab and higher dose aflibercept aim for longer treatment intervals to reduce burden.[14] For dry AMD, intravitreal complement inhibitors reduce lesion growth in real world data. Pegcetacoplan (C3 inhibition) has Therapeutic Goods Administration approval, with avacincaptad pegol (C5 inhibition) awaiting approval, however both are not PBS-listed.[15]

Both AMD subtypes might co-exist, and some patients require both treatments. GP support through monitoring for conversion to wet AMD, reinforcing adherence to treatment (where prescribed), and coordinating ongoing ophthalmic follow-up are all useful in detection and prevention of AMD.

AREDS2 daily formula

Vitamin C	500 mg
Vitamin E	400 IU
Zinc	25–80 mg[A]
Copper	2 mg
Lutein	10 mg
Zeaxanthin	2 mg

Evidence summary

- 25% risk reduction in progression to late AMD
- 10-year follow-up shows sustained benefit
- Level 1 RCT evidence (AREDS & AREDS2)
- β-carotene excluded (due to lung-cancer risk in smokers)

When to use (secondary prevention)[B]

- Intermediate AMD (large drusen ≥125 µm, pigment changes)
- Late AMD in one eye
- High-risk phenotypes (family history, CFH/ARMS2)

Australian TGA note

- 80 mg zinc formulation not permitted (TGA limit = 50 mg/day)
- Low-zinc formulas are clinically effective and recommended

Common Australian brands[C]

- MD Eyes: closest AREDS2 match (low-zinc 25–50 mg)
- Macula Guard: AREDS2-equivalent (low-zinc version)
- MacuVision: half-dose, no lutein/zeaxanthin
- MacuVision Plus: ≈ ½ lutein/zeaxanthin
- PreserVision AREDS2: Full formula (import only)

Figure 3. Secondary prevention in AMD.

Lifestyle and nutritional interventions that reduce progression risk in patients with early or intermediate AMD. Secondary prevention refers to measures that slow disease progression and prevent conversion to late AMD, rather than preventing disease onset. Evidence-based strategies include smoking cessation, adherence to a Mediterranean-style diet rich in antioxidants and omega-3 fatty acids, regular exercise, and AREDS2 supplementation.

[A]Low-zinc (25–50 mg) variants are approved and clinically equivalent.
[B]Not to be used when no AMD/early AMD/for primary prevention.
[C]Australian pharmacy supplements often provide only half the AREDS2 dose; doubling daily tablets or adding lutein/zeaxanthin might be required for equivalence.

AMD, age-related macular degeneration; AREDS, Age-Related Eye Disease Study; AREDS2, Age-Related Eye Disease Study 2; ARMS2, age-related maculopathy susceptibility 2; CFH, complement factor H; RCT, randomised controlled trials; TGA, Therapeutic Goods Administration.

© The Royal Australian College of General Practitioners 2026

Emerging therapies aim to reduce treatment burden while maintaining vision. Gene therapy trials underway for both wet and dry AMD demonstrate a 97% reduction in injection burden at 9 months.[16] Oral or long-acting tyrosine kinase inhibitors also show encouraging early data of up to 90% reduction in treatment frequency with non-inferior visual outcomes.[17] Similarly, nanosecond laser shows reduced progression of intermediate disease in an early Australian trial.[18] Overall, these advances might lessen treatment burden and improve real-world adherence (Figure 4).

Conclusion

AMD is a common and progressive condition with significant implications for vision and independence in older adults. GPs and optometrists play a central part in risk factor modification, early symptom recognition, timely referral for suspected wet AMD, and ongoing support for treatment adherence.

Even as therapeutic options continue to expand, particularly for advanced disease, primary care remains an essential part of the multidisciplinary team, especially in the areas of primary and secondary prevention and early detection.

Key points

- AMD is the leading cause of legal blindness in people aged over 50 years in Australia.
- AMD risk factors matter: increasing age and family history are major risks, while smoking, poor diet and cardiovascular disease are modifiable contributors.
- Monitoring is essential to detect change early: encourage regular Amsler grid testing (weekly for at-risk, daily for diagnosed AMD) and prompt review for any new sudden distortion, blur or central scotoma seen on the grid. Regular/yearly optometry assessments are recommended for at risk persons of AMD progression.
- New treatments are in the pipeline, potentially reducing the burden of care: advances include longer-acting intravitreal injections, sustained-delivery gene and tyrosine kinase inhibitor therapies, and investigational nanosecond laser approaches designed to preserve vision with fewer injections.

Authors

Carl Eiselen BSc, MD, GDipSurgAnat, Clinical Research Fellow, Nuffield Department of Clinical Neurosciences, University of Oxford, Oxford, UK; Honorary Clinical Research Fellow, Oxford Eye Hospital, Oxford University Hospitals NHS Foundation Trust, Oxford, UK

Marita Long MBBS, FRACGP, DCH, Cert S & RH, General Practitioner and Lecturer in Primary Care, Department of General Practice and Primary Care, Faculty of Medicine, Dentistry and Health Sciences, The University of Melbourne, Melbourne, Vic

Elaine W Chong MBBS, MEpi, PhD, FRANZCO, FAMS, Head of Ophthalmology, Department of Ophthalmology, The Royal Melbourne Hospital, Melbourne, Vic; Senior Research Fellow, Centre for Eye Research Australia, Melbourne, Vic; Deputy Head of the Corneal Unit and Consultant Ophthalmologist in Medical Retina, Department of Ophthalmology,

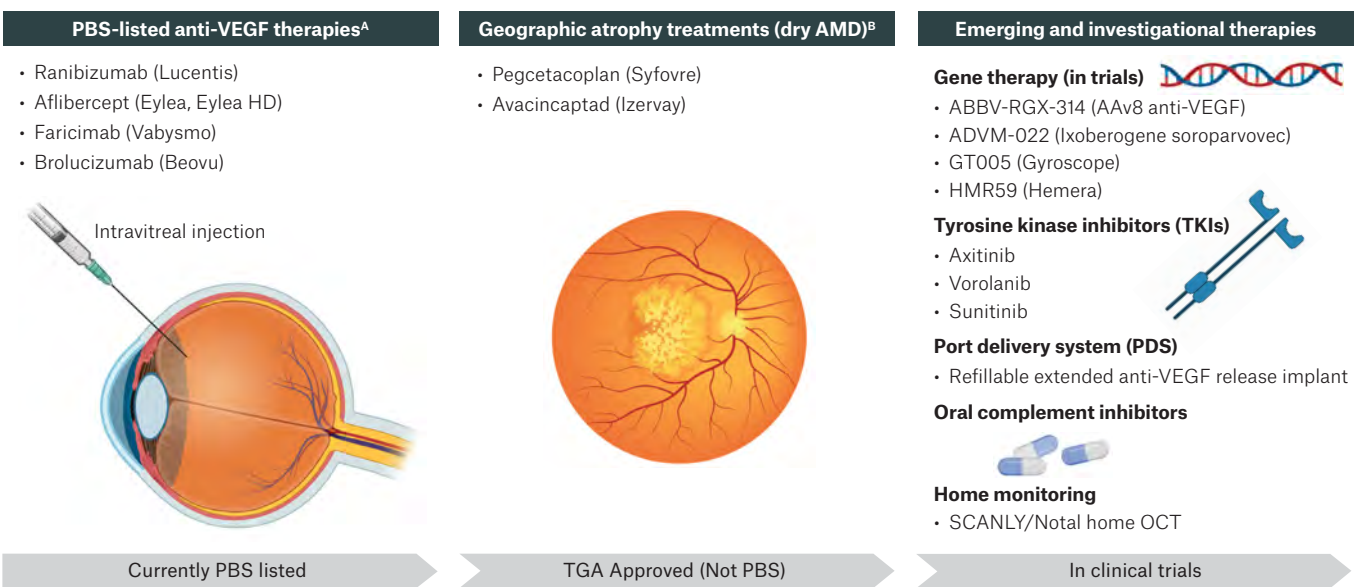

Figure 4. Current and emerging therapies for AMD categorised by regulatory status.

[A]All fully PBS funded for neovascular AMD.
[B]Complement inhibitors slow atrophy progression but are not PBS listed.

AAV, adeno-associated virus; ABBV-RGX-314, AbbVie/REGENXBIO investigational gene therapy (anti-VEGF Fab); ADVM-022, Adverum investigational gene therapy (aflibercept-encoding); AMD, age-related macular degeneration; C3, complement component 3; C5, complement component 5; GT005, Gyroscope/Novartis investigational complement factor I gene therapy; HMR59, Hemera Biosciences investigational CD59 gene therapy; OCT, optical coherence tomography; PBS, Pharmaceutical Benefits Scheme; TGA, Therapeutic Goods Administration; TKI, tyrosine kinase inhibitor; VEGF, vascular endothelial growth factor.

© The Royal Australian College of General Practitioners 2026

The Royal Victorian Eye and Ear Hospital, Melbourne, Vic

Competing interests: None.

Funding: None.

Provenance and peer review: Commissioned, externally peer reviewed.

AI declaration: The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

Correspondence to:
doctoreiselen@gmail.com

Acknowledgements

Dr Eiselen thanks Dr Marita Long and A/Prof Elaine W Chong for their editorial guidance and collaboration on this clinical update.

Dr Carl Eiselen would like to acknowledge his father, Dr Ernst Eiselen, for his lifelong example as an inspirational general practitioner, and his mother, Estelle Eiselen, whose work as a practice manager embodies compassion and dedication to patient care. He also acknowledges Dr David Homewood, whose vision and leadership helped inspire this special edition of the *Australian Journal of General Practice*.

The authors thank OneRightEye for providing open-access educational resources that informed the figures.

References

References are available online only.

correspondence **ajgp@racgp.org.au**

© The Royal Australian College of General Practitioners 2026

References

1. Heath Jeffery RC, Mukhtar SA, Lopez D, et al. Incidence of newly registered blindness from age-related macular degeneration in Australia over a 21-year period: 1996–2016. Asia Pac J Ophthalmol (Phila) 2021;10(5):442–49. doi: 10.1097/APO.0000000000000415.
2. Babaker R, Alzimami L, Al Ameer A, et al. Risk factors for age-related macular degeneration: Updated systematic review and meta-analysis. Medicine (Baltimore) 2025;104(8):e41599. doi: 10.1097/MD.0000000000041599.
3. Joachim N, Colijn JM, Kifley A, et al. Five-year progression of unilateral age-related macular degeneration to bilateral involvement: The Three Continent AMD Consortium report. Br J Ophthalmol 2017;101(9):1185–92. doi: 10.1136/bjophthalmol-2016-309729.
4. Khan JC, Thurlby DA, Shahid H, et al; Genetic Factors in AMD Study. Smoking and age related macular degeneration: The number of pack years of cigarette smoking is a major determinant of risk for both geographic atrophy and choroidal neovascularisation. Br J Ophthalmol 2006;90(1):75–80. doi: 10.1136/bjo.2005.073643.
5. Owsley C, Swain TA, McGwin G Jr, et al. How vision is impaired from aging to early and intermediate age-related macular degeneration: Insights from ALSTAR2 baseline. Transl Vis Sci Technol 2022;11(7):17. doi: 10.1167/tvst.11.7.17.
6. Boon J, Rojas-Carabali W, Asad Y, Lim JTY, Rajagopalan R, Agrawal R. Evaluation of a Digital Amsler Grid (PocDoc) for macular disease screening: A comparative analysis with the conventional method. Ophthalmol Ther 2024;13(5):1289–301. doi: 10.1007/s40123-024-00910-5.
7. National Institute for Health and Care Excellence (NICE). Age-related macular degeneration. NICE, 2018. Available at www.nice.org.uk/guidance/ng82/chapter/recommendations [Accessed 24 October 2025].
8. Merle BMJ, Colijn JM, Cougnard-Grégoire A, et al; EYE-RISK Consortium. Mediterranean diet and incidence of advanced age-related macular degeneration: The EYE-RISK Consortium. Ophthalmology 2019;126(3):381–90. doi: 10.1016/j.ophtha.2018.08.006.
9. Age-Related Eye Disease Study Research Group. The Age-Related Eye Disease Study (AREDS): Design implications. AREDS report no. 1. Control Clin Trials 1999;20(6):573–600. doi: 10.1016/S0197-2456(99)00031-8.
10. Chew EY, Clemons T, SanGiovanni JP, et al; AREDS2 Research Group. The Age-Related Eye Disease Study 2 (AREDS2): Study design and baseline characteristics (AREDS2 report number 1). Ophthalmology 2012;119(11):2282–89. doi: 10.1016/j.ophtha.2012.05.027.
11. McGuinness MB, Le J, Mitchell P, et al. Physical activity and age-related macular degeneration: A systematic literature review and meta-analysis. Am J Ophthalmol 2017;180:29–38. doi: 10.1016/j.ajo.2017.05.016.
12. Fragiotta S, Bassis L, Abdolrahimzadeh B, Marino A, Sepe M, Abdolrahimzadeh S. Exploring current molecular targets in the treatment of neovascular age-related macular degeneration toward the perspective of long-term agents. Int J Mol Sci 2024;25(8):4433. doi: 10.3390/ijms25084433.
13. Shahzad H, Mahmood S, McGee S, et al. Non-adherence and non-persistence to intravitreal anti-vascular endothelial growth factor (anti-VEGF) therapy: A systematic review and meta-analysis. Syst Rev 2023;12(1):92. doi: 10.1186/s13643-023-02261-x.
14. London NJS, Cheung CMG, Michels S, et al. Outcomes by faricimab treatment interval at week 48 of TENAYA-LUCERNE phase III trials in neovascular age-related macular degeneration. Ophthalmol Retina 2025;9(11):1081–89. doi: 10.1016/j.oret.2025.05.004.
15. Rush RB, Klein W, Reinauer RM. Real-world outcomes with complement inhibitors for geographic atrophy: A comparative study of pegacetacoplan versus avacincaptad pegol. Clin Ophthalmol 2025;19:1167–74. doi: 10.2147/OPTH.S518398.
16. Campochiaro PA, Avery R, Brown DM, et al. Gene therapy for neovascular age-related macular degeneration by subretinal delivery of RGX-314: A phase 1/2a dose-escalation study. Lancet 2024;403(10436):1563–73. doi: 10.1016/S0140-6736(24)00310-6.
17. Das N, Talcott KE. Tyrosine kinase inhibitors for wet AMD and diabetic retinopathy. Retin Physician 2024;21(April):14–17. Available at https://retinalphysician.com/issues/2024/april/tyrosine-kinase-inhibitors-for-wet-amd-and-diabetic-retinopathy [Accessed 25 October 2025].
18. Guymer RH, Wu Z, Hodgson LAB, et al. Subthreshold nanosecond laser intervention in age-related macular degeneration: The LEAD randomized controlled clinical trial. Ophthalmology 2019;126(6):829–38. doi: 10.1016/j.ophtha.2018.09.015.

© The Royal Australian College of General Practitioners 2026