pharma
June 2019
Optometry AUSTRALIA
ADVANCING OPTOMETRY

SUPPORTED BY NOVARTIS

AMD
The standard of care—
from detection to treatment

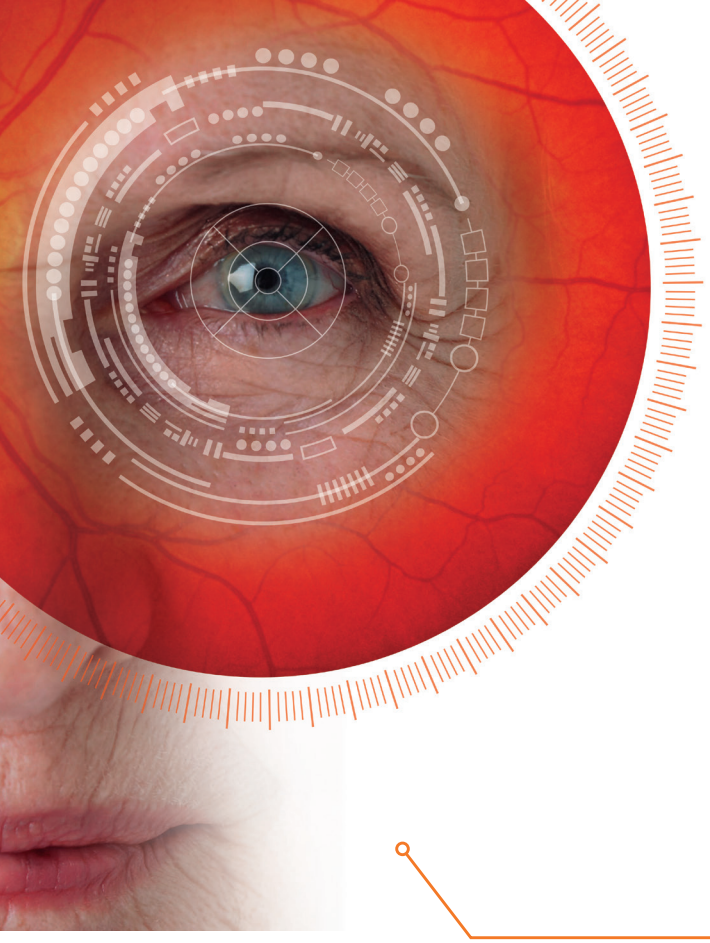
Domiciliary eye care
Common ocular co-morbidities

Anti-VEGF treatment
The optometrist's role in treatment compliance

Accredited CPD points available with this issue of *Pharma*.

N·Z·A·O CPD

Optometry Board of Australia
6
1T



POWERFUL†

TO IMPROVE PATIENTS' LIVES[1–6]

†As measured by mean change in vrQoL.[1–6]

Lucentis® – the only TGA-approved and PBS-listed anti-VEGF available in a pre-filled syringe.[7,8]

nAMD · DME · BRVO · CRVO · other CNV (NEW) · PM (NEW)

BRVO, branch retinal vein occlusion; CNV, choroidal neovascularisation; CRVO, central retinal vein occlusion; DME, diabetic macular oedema; nAMD, neovascular age-related macular degeneration; PBS, Pharmaceutical Benefits Scheme; PM, pathological myopia; TGA, Therapeutic Goods Administration; VEGF, vascular endothelial growth factor; vrQoL, vision-related quality of life.

PBS Information: Authority required for the treatment of wet AMD, DME, BRVO, CRVO, PM or for the treatment of CNV secondary to causes other than wet AMD and PM. Refer to PBS Schedule for full Authority information.

Before prescribing, please review full Product Information available from www.novartis.com.au/products/healthcare-professionals

Indication: Treatment of neovascular (wet) age-related macular degeneration (AMD). *The treatment of visual impairment due to choroidal neovascularisation.* Treatment of visual impairment due to choroidal neovascularisation (CNV) secondary to pathologic myopia (PM). Treatment of visual impairment due to diabetic macular oedema (DME). Treatment of visual impairment due to macular oedema secondary to retinal vein occlusion (RVO). **Dosage and administration:** Complex dosage and administration – see full PI before prescribing. **Contraindications:** Hypersensitivity to product components, active or suspected ocular or periocular infections active intraocular inflammation. **Precautions:** Intravitreal injections have been associated with endophthalmitis, intraocular inflammation, rhegmatogenous retinal detachment, retinal tear, iatrogenic traumatic cataract and increased intraocular pressure. Proper aseptic injection techniques must be used. Review patients during the week following injection to permit early treatment if an infection occurs. Transient increases in intraocular pressure (IOP) have been seen within 60 minutes of injection of Lucentis. Sustained IOP increases have also been reported. Intraocular pressure and perfusion of the optic nerve head must be monitored and managed appropriately. Patients should be reviewed for IOP rise preinjection and 60 minutes post-injection. *The dose should be withheld and treatment should not be resumed earlier than the next scheduled treatment in the event of an intraocular pressure of ≥30 mmHg.* Bilateral use: limited data do not suggest an increased risk of systemic adverse events compared with unilateral treatment. There is a potential risk of arterial thromboembolic events following intravitreal use of VEGF inhibitors. A numerically higher stroke rate was observed in patients treated with ranibizumab 0.5mg compared to ranibizumab 0.3mg or control, however, the differences were not statistically significant. Patients with known risk factors for stroke, including history of prior stroke or transient ischaemic attack, should be carefully evaluated by their physicians as to whether Lucentis treatment is appropriate and the benefit outweighs the potential risk. As with all therapeutic proteins, there is a potential for immunogenicity with Lucentis. Lucentis has not been studied in patients with concurrent eye conditions such as retinal detachment or macular hole. No formal interaction studies have been performed. Limited experience with treatment of patients with prior episodes of RVO and of patients with ischemic branch RVO (BRVO) and central RVO (CRVO). In patients with RVO presenting with clinical signs of irreversible ischemic visual function loss, treatment is not recommended. Should be used with caution in women of child bearing potential in general, and during pregnancy in particular. For women who wish to become pregnant and have been treated with ranibizumab, it is recommended to wait at least 3 months after the last dose of ranibizumab before conceiving a child; use of effective contraception is recommended for women of childbearing potential; breastfeeding is not recommended. Lucentis is not recommended for use in children and adolescents Patients who experience temporary visual disturbances following treatment must not drive or use machines until these subside. **Adverse effects:** Very common (≥10%): Intraocular inflammation, vitritis, vitreous detachment, retinal haemorrhage, visual disturbance, eye pain, vitreous floaters, conjunctival haemorrhage, eye irritation, foreign body sensation in eyes, lacrimation increased, blepharitis, dry eye, ocular hyperaemia, eye pruritus, intraocular pressure increased, nasopharyngitis, headache, arthralgia. Common (1 to 10%): Retinal degeneration, retinal disorder, retinal detachment, retinal tear, detachment of the retinal pigment epithelium, retinal pigment epithelium tear, visual acuity reduced, vitreous haemorrhage, vitreous disorder, uveitis, iritis, iridocyclitis, cataract, cataract subcapsular, posterior capsule opacification, punctuate keratitis, corneal abrasion, anterior chamber flare, vision blurred, injection site haemorrhage, eye haemorrhage, conjunctivitis, conjunctivitis allergic, eye discharge, photopsia, photophobia, ocular discomfort, eyelid edema, eyelid pain, conjunctival hyperaemia, stroke, influenza, urinary tract infection*, anaemia, anxiety, cough, nausea, allergic reactions (rash, pruritus, urticaria, erythema). Uncommon (0.1 to 1%): Blindness, endophthalmitis, hypopyon, hyphaema, keratopathy, iris adhesions, corneal deposits, corneal oedema, corneal striae, injection site pain, injection site irritation, abnormal sensation in eye, eyelid irritation. Serious adverse events related to intravitreal injections include endophthalmitis, rhegmatogenous retinal detachment, retinal tear and iatrogenic traumatic cataract. *Observed only in the DME population. Based on TGA approved Product Information dated 18 December 2018 *(luc181218m). *Please note changes to Product Information in italics.*

References: 1. Hernando M et al. Eur J Hosp Pharm 2018; 25 (Suppl 1): A129–A130. Poster 4CPS-187. **2.** Turkoglu EB et al. J Diabetes Complications 2015; 29 (4): 540–543. **3.** Inoue M et al. Clin Ophthalmol 2014; 8: 1711–1716. **4.** Finger RP et al. Ophthalmology 2014; 121 (6): 1246–1251. **5.** Symeonidis C et al. Ophthalmologica 2014; 232 (Suppl 2): 9. Abstract 169. **6.** Charonis A et al. Ophthalmologica 2014; 232 (Suppl 2): 9. Abstract 165. **7.** Lucentis® Product Information. December 2018. **8.** Pharmaceutical Benefits Scheme. Ranibizumab syringe. www.pbs.gov.au/medicine/item/10138N-10374B (accessed 8 April 2019).

Novartis Pharmaceuticals Australia Pty Limited. ABN 18 004 244 160. 54 Waterloo Road, Macquarie Park, NSW 2113. ®Registered Trademark. AU-7315. McCann Health NOLU15419M. April 2019..

NOVARTIS

LUCENTIS® RANIBIZUMAB

pharma | Optometry AUSTRALIA

June 2019 AMD and aged care

This issue of *Pharma* is supported by NOVARTIS

From the Editors...

According to the Australian Institute of Health and Welfare, the life expectancy in Australia—82.5 years—is among the highest in the world. There are 5.5 million baby boomers in Australia, ranging from 54 to 73 years old and the Australian Bureau of Statistics projects that the number of people over 85 will increase from 0.4 million today to 1.7 million in 2050.

Given the link between ageing and the incidence of eye diseases such as AMD, optometrists are increasingly being called upon to play a key role in the provision of collaborative health care.

Research undertaken by Optometry Australia in 2018 suggests that only two per cent of practitioners 'often' or 'always' provide home visits, and fewer than five per cent regularly provide care in an aged-care facility.

Have you thought about what you can do to improve eye health outcomes for this population?

In this issue, we explore some of the conditions that optometrists are called upon to diagnose, treat and refer in their elderly patients. Highlights include Clinical Editor Kerryn Hart's interview with Dr Simon Chen about anti-VEGF therapy and the role that optometrists can play in patient compliance; a summary of the ocular co-morbidities associated with domiciliary care by Mae Chong and Lesley Dacion; and a discussion of the LEAD study on laser treatment for AMD by Professors Erica Fletcher and Robyn Guymer.

As a featured member resource, we've provided an AMD classification table, drawn from Optometry Australia's 2019 Clinical Practice Guide for the diagnosis, treatment and management of age-related macular degeneration.

The editors would like to thank Novartis for partnering with us to bring you this issue of *Pharma.*

02
Understanding patients with ring scotomas
Paul Graveson

04
Beyond the macula
Dr Lisa Nivison-Smith

07
Ocular co-morbidities in domiciliary eye care
Mae FA Chong and Lesley Dacion

10
Anti-VEGF injections and the prevention of irreversible visual loss
Dr Simon Chen and Kerryn Hart

12
Evidence-based advice for AMD
Dr Laura Downie

14
FEATURE
Clinical classification for aged-related macular degeneration (AMD)
Optometry Australia

17
Nanosecond laser treatment for age-related macular degeneration
Professor Erica L Fletcher and Professor Robyn H Guymer

20
CXO featured article:
OCT in the investigation of systemic neurologic disease
Dr Sangeetha Srinivasan and Emeritus Professor Nathan Efron

22
Not quite as it seems
Pauline Xu

25
OCT-A and delayed-onset traumatic macular oedema
Joe Wang, Jessie Tan and Dr Kwang Meng Cham

27
Low vision referral
Nabill Jacob

This issue of *Pharma* offers 6 (1T) CPD points.

Access the online CPD modules at www.optometry.org.au

Editor JEFF MEGAHAN
j.megahan@optometry.org.au

Clinical Editor KERRYN HART
BOptom GCertOcTher MPH
Teaching Scholar, Deakin University

Publications Manager JESSICA DONALD

Cover 'Boomer optometry' by Lachlan Hessing. Image Karen Wilson Photography.

Optometry Australia ABN 17 004 622 431
Level 1, 201 Clarendon Street
South Melbourne VIC 3205
Ph 03 9668 8500
www.optometry.org.au

Pharma is distributed in Australia and New Zealand. All references to pharmaceutical preparations in *Pharma* are applicable within Australia.

Comments made in *Pharma* are of a general nature and intended for guidance only.

Optometry Australia and the individual contributors expressly disclaim all liability and responsibility to any person in respect of, and for the consequences of, anything done or omitted to be done in reliance wholly or partly on anything in this publication.

Understanding patients with ring scotomas

Paul Graveson
BOptom BA GradDip(Optom) PGCOT

Low Vision Consultant
The Royal Hobart Hospital Eye Clinic

Principal Optometrist
Hobart Optometry

All optometrists encounter patients who, despite having very advanced macular degeneration, retain surprisingly good visual acuity (VA). However, they may complain of difficulty with tasks we usually associate with much greater levels of vision loss, such as difficulty recognising faces even at close range.

CASE REPORT

Patient A was an 89-year-old Caucasian female with age-related macular degeneration (AMD). The maculae appear reasonably similar (Figure 1 and 2) with large areas of photoreceptor loss, but surprisingly the VA was markedly different: R 6/12, L 6/240.

I asked her to look away from the VA chart, towards a more 'real-world' view (my desk and bookshelves across the room), and tell me which eye she saw better with. She covered the right, then the left, then the right and the left again, and finally answered 'I think the right is just a touch better.' Many optometrists would find that perplexing. 'Surely,' you would think, 'the difference between an eye with 6/12 and an eye with 6/240 should be easily apparent?'

The explanation is that, somewhere within the large area of macular atrophy, the right eye had a tiny area of foveal tissue remaining, with which she could read small, isolated high-contrast letters. But her difficulty in answering told me that the foveal remnant was too small to make much of a practical difference to her. Functionally, she was only a whisker away from end-stage AMD in both eyes. She found it impossible to read any text comfortably, even newspaper headlines. And she could not recognise faces even when they were as close as 50 cm.

The ring of blindness

Although AMD may affect the fovea even in the early stages, in geographic atrophy (GA) it's very common for the fovea to be spared until the very last. The effect is to have the macula eaten away by small scotomas which gradually become confluent, creating a ring of blindness around a foveal remnant. That foveal remnant then shrinks, until it finally disappears. Figure 3 shows autofluorescence scans of a typical progression over several years.

The severe loss of functional vision caused by ring scotomas often goes unrecognised, because the patient retains both good VA and good mobility vision until the very last stage of the disease. In the meantime, the patient (and their family) may suffer a great deal of confusion and distress, so it's important for all optometrists to be familiar with this type of presentation.

On the VA chart, such patients may find very large letters even harder to read than very small letters, since only part of each large letter fits within their foveal remnant field. Similarly, they may have the confusing symptom of finding newspaper headlines harder to read than the body of the article. They are often unable to recognise faces and their reading fluency is frustratingly poor. But preservation of the foveal field lets them see tiny, isolated details at times (for instance,

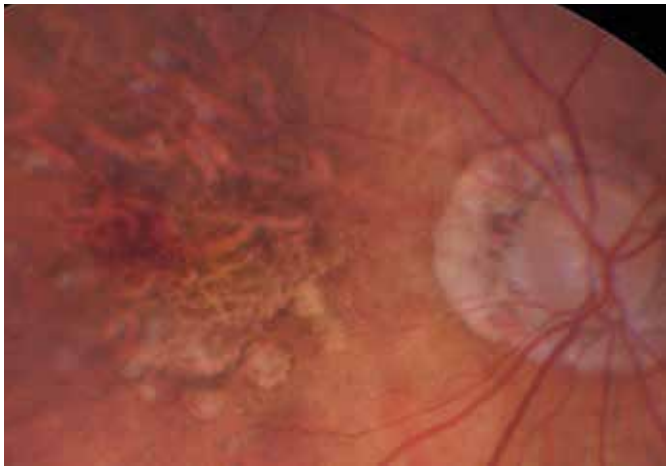


Figure 1. Patient A, right eye. Picture supplied by author.

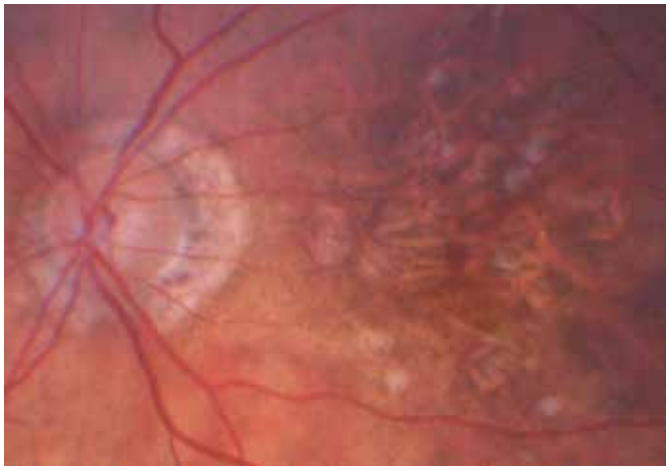


Figure 2. Patient A, left eye. Picture supplied by author.

noticing that someone has a little bit of spinach stuck in their teeth, or noticing a fly in their food). The patient can't understand how they can have so many problems if they can see such tiny things, and their family may even start suspecting that the person is malingering. It's very powerful if you can explain it to them. You can see the comprehension dawning as they finally understand what's been going on.

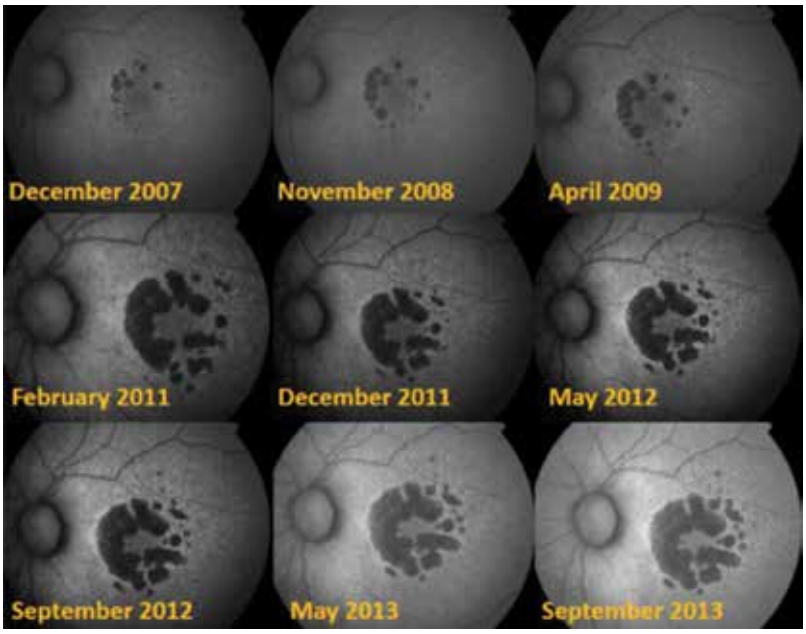


Figure 3. Progression of geographic atrophy showing foveal sparing. Courtesy of J. Monés, MD, PhD. Institut de la Màcula.

Treatment options

How can we help the patient with a ring scotoma? It's tough, really tough. Their expectations tend to be high; their function is low. They may present expecting an easy fix, because they have been getting a consistent message from their ophthalmologist that their vision is 'still quite good' (based on their VA alone), and so they are hopeful that you will be able to give them the 'right' pair of glasses that so many others have failed to deliver. Finding out that they actually have quite advanced vision loss can be quite a shock (although at the same time, deep down they know it to be true, since they've been experiencing such difficulties).

In practical terms, helping such patients achieve 'spot reading' of short sections of text is not too difficult. Often the best intervention is simply better illumination, perhaps with a little magnification. Bear in mind though, their VA is usually still good, and whatever you give them with magnification they lose in field of view. The effect of optimal light is to expand the foveal remnant a little. A small pocket torch can be useful to provide a small spot of very good illumination on price tags, etc. Low-powered illuminated stand magnifiers are sometimes helpful for spot reading of smaller print.

Beyond that, the best interventions involve using high magnification, large field of view and contrast enhancement, just as if they had a more conventional form of end-stage AMD. That means using a closed-circuit television (CCTV) electronic magnifier to give high magnification so the patient can use their intact paramacular retina.

The bad news is, even CCTVs don't tend to work well with these patients. Using the paramacular area requires that the patient learn to use eccentric fixation and very high (10x or more) magnification, but the fact that the fovea is intact means there is a powerful stimulus to continue with direct fixation.

Eventually, the fovea will be snuffed out. If you're the optometrist who sees them after foveal extinction, it can be disconcerting. A typical scenario is that you last saw the patient six months ago, at which time they had 6/12 vision in their better eye, but today that eye sees only 6/240. Alarm bells go off in your mind—what's happened? When you ask how long ago the vision dropped, the patient is often quite vague, which seems strange—how could they fail to notice such a dramatic drop in vision?

The thing to remember is: it wasn't actually a dramatic drop in vision. The vision went from being severely impaired but with a tiny, not-very-useful foveal remnant to being just severely impaired across the entire macula. Sometimes patients might remember that their magnifier seemed to 'stop working' a month or so ago, which would probably be the time of foveal dropout.

The funny thing is, many patients seem happier at this point—or at least more settled, less frustrated with their vision loss. It seems counter-intuitive, but it makes sense. Near the end, the foveal area is too tiny to be helpful, but large enough to cause frustration, so the loss of the fovea means a loss of the source of frustration.

Once the fovea has been lost, patients often spontaneously develop eccentric fixation (and start coping with some tasks much better), and they no longer spend their time laboriously using their magnifiers to painstakingly read text a few letters at a time. If they've tried and failed with a CCTV before, now might be a good time to refer them to a low vision clinic to try again. ▲

This article is adapted from a page on the author's website Understanding Low Vision. Visit understandinglowvision.com for more practical tips on helping patients with low vision.

Beyond the macula

Peripheral changes in patients with AMD

Dr Lisa Nivison-Smith
PhD BSc (Hons)

Research Associate
Centre for Eye Health

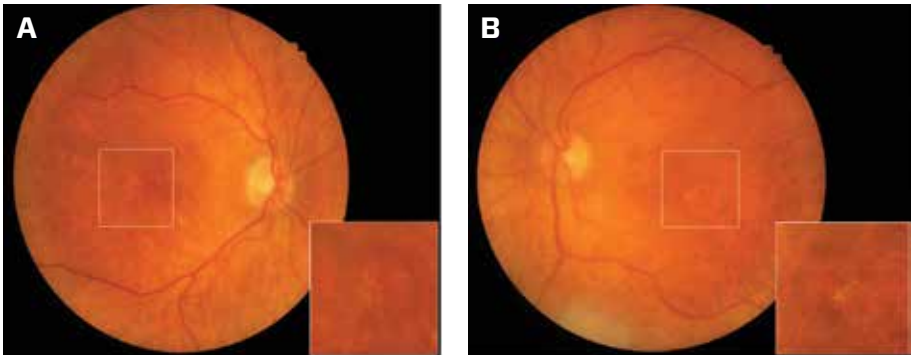


Figure 1. Retinal photograph of A: right and B: left eye of an 82-year-old AMD patient with drusen at the macula.

Mounting evidence suggests that the term 'macular' in age-related macular degeneration (AMD) may be a misnomer. As the following case study demonstrates, a number of degenerative changes are often evident in the periphery, including drusen, pigmentary abnormalities and pavingstone degeneration. Such peripheral AMD findings have also been associated with underlying genetic causes of AMD (that is: specific AMD gene variants) and functional deficits, suggesting that assessment of the peripheral retina may play an increasingly important role in the future management of AMD.

The advent of ultra-widefield imaging such as Optos Optomap means imaging of the peripheral retina can be done quickly in routine clinical practice. As such, a number of studies have assessed the prevalence of peripheral retinal findings in AMD. Drusen have been reported in the mid to far periphery in 49 per cent to 97 per cent of early/intermediate AMD patients using ultra-widefield imaging.[1-3] Domalpally et al[3] also reported pigmentary changes and reticular pseudodrusen in the mid-periphery of 46 per cent and 15 per cent respectively of study eyes in the AREDS2 study and senile reticular pigmentary changes in 72 per cent of study eyes.

Altered peripheral autofluorescence (FAF) is also a common theme, observed in 64 per cent of AMD eyes with descriptions of granular and mottled FAF patterns; more common in neovascular AMD than non-neovascular AMD[4] or early AMD.[5] Abnormal peripheral FAF also appears to be unaltered in neovascular AMD following treatment (50 per cent in untreated vs 52 per cent in treated eyes).[6]

Note that these results should be considered relative to the usual prevalence of peripheral findings in an age-matched population of normal eyes. For example, peripheral drusen have been reported in 36 per cent to 48 per cent of healthy control eyes[2,3] and abnormal peripheral FAF has been reported in 18 per cent to 36 per cent of healthy eyes.[1,4,5] A recent study in Australia also suggested the prevalence of most peripheral findings in AMD patients is similar to that of healthy age-matched eyes.[7]

Despite this controversy, evidence suggests that documentation of these findings may become important as part of the clinical management of AMD, as certain peripheral changes have been associated with poorer visual performance and genetic risk factors. Lains et al[8] found AMD eyes with peripheral reticular pigmentary changes or decreased mid-peripheral FAF had greater impairments in dark adaptation than those without peripheral changes.[8] Peripheral drusen and pigmentary changes have also been associated with greater AMD severity and known AMD genotypes.[9-11]

The following case study demonstrates an individual with AMD who was found to also have peripheral changes following ultra-widefield imaging. Based on current research, these findings suggest assessing the patient's dark adaptation function and closely examining their family history of AMD to help determine their risk of AMD progression.

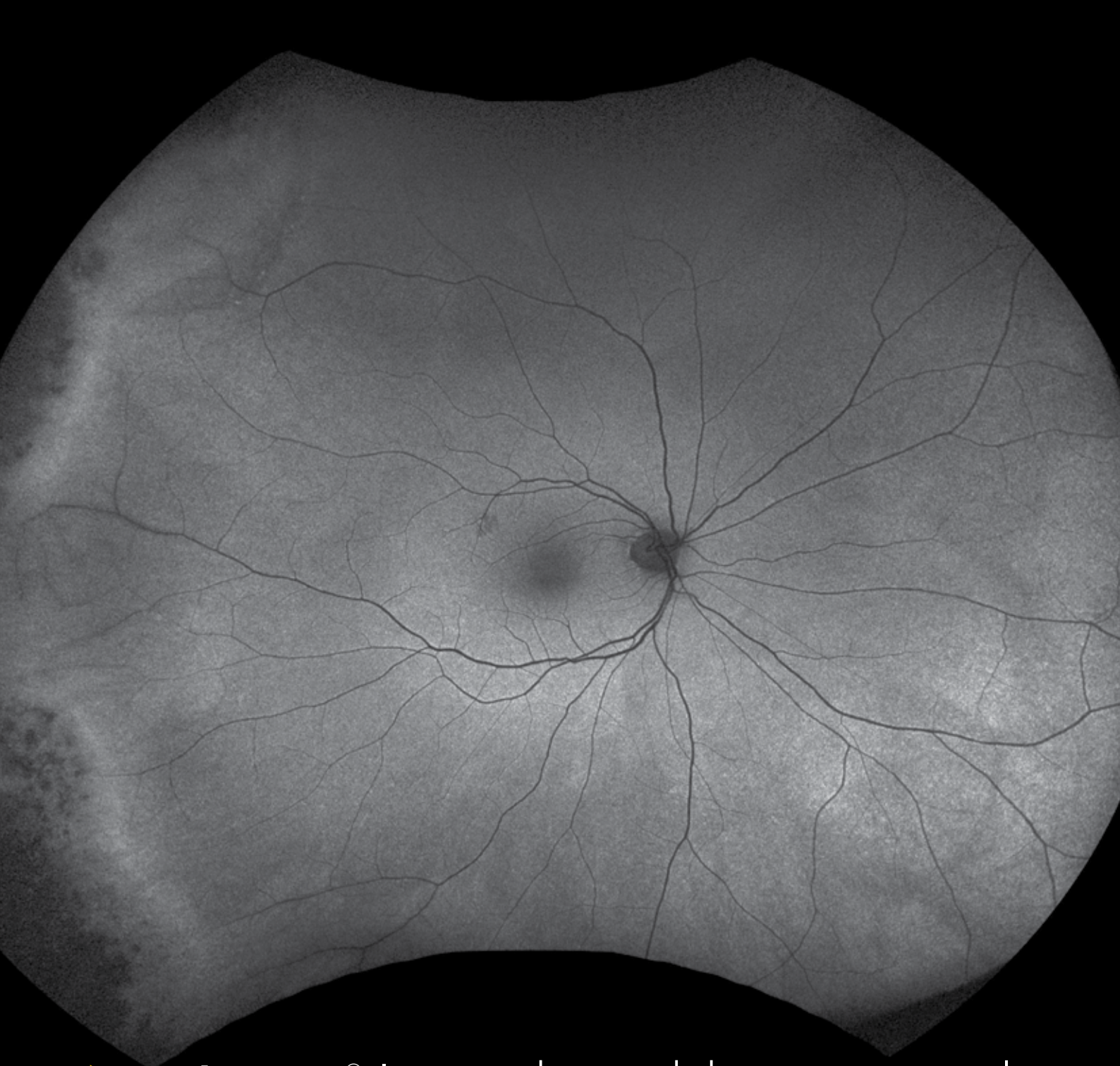
CASE REPORT

An 82-year-old Caucasian male was referred to the Centre for Eye Health, UNSW Sydney for a macular assessment based on evidence of drusen in both maculae. The patient reported a medical history of hypertension, gout, hypercholesterolaemia and was taking anti-platelet, blood pressure lowering and cholesterol lowering medication. He also reported having a pacemaker and a mini-stroke several years ago.

His entering unaided acuities were 6/9.5 (NIPH) in the right eye and 6/12 (NIPH) in the left eye with no distortions on Amsler grid and normal Mars contrast sensitivity values for each eye. Fundoscopy revealed medium and large drusen at the right temporal fovea and left central fovea with no evidence of macular pigmentary abnormalities (Figure 1A, B). These findings are consistent with intermediate AMD in both eyes.[12]

Dilated peripheral retinal assessment and ultra-widefield imaging revealed extensive mid-peripheral drusen in both eyes. Reticular pigmentary degeneration was also observed in the

Continued page 6



optosmap® Images beyond the vortex vessels
in less than ½ second

the ONLY ultra-widefield, single-capture *af* image

Find out more at www.optos.com
Contact us to put optosmap **in your practice, call 08 8444 6500 or email auinfo@optos.com**

Nikon

optos®

AMD in the periphery

From page 4

temporal periphery (Figures 2A-F). An area of localised hyperfluorescence adjacent to the inferior optic disc in the right eye (Figure 2G) which corresponded to an isolated drusen on OCT was also observed. Peripheral changes were not observable on the fundus image alone.

Consequently, the patient was diagnosed with intermediate AMD in both eyes.[12] This AMD stage confers approximately a 12 per cent probability of progression to late AMD within the next five years according to the AREDS Simplified Scale.[13] It was recommended that the patient monitor his vision at home using an Amsler grid and to consult his GP regarding an AREDS-based vitamin supplementation for his condition in conjunction with his existing medication. A repeat eye examination was recommended in 12 months to assess for progression. ▲

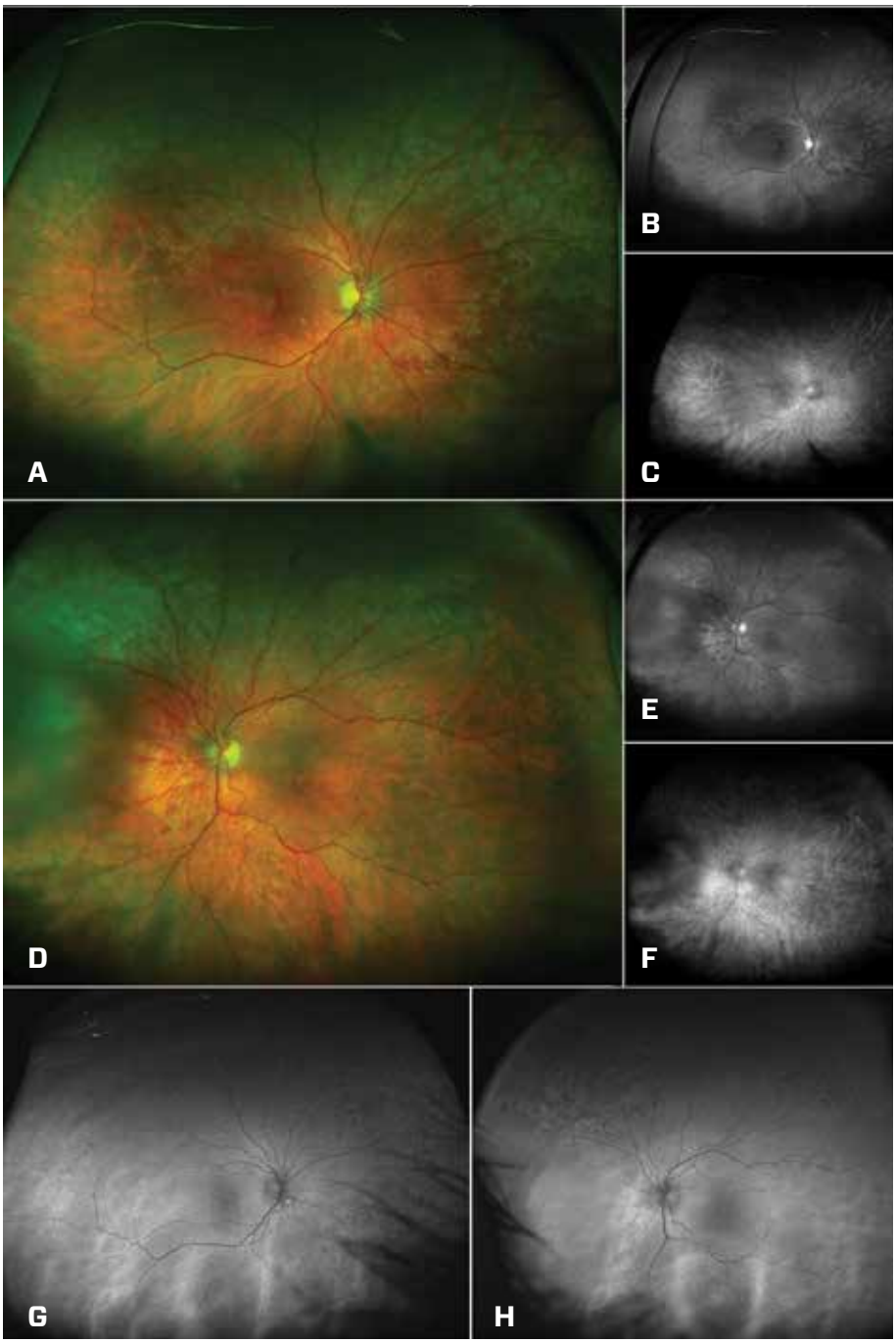


Figure 2. Ultra-widefield imaging (colour, red free, green free) of A-C: right and D-F: left eye indicating peripheral drusen and reticular pigmentary changes and G-H: associated FAF imaging.

1. Lengyel I, Csutak A, Florea D et al. A Population-Based Ultra-Widefield Digital Image Grading Study for Age-Related Macular Degeneration Like Lesions at the Peripheral Retina. *Ophthalmology* 2015; 122: 1340-1347.
2. Vatavuk Z, Andrijevi Derk B, Kneževi T et al. Morphological and Angiographic Peripheral Retinal Changes in Patients with Age-Related Macular Degeneration. *Ophthalmology Retina* 2018; 2: 201-208.
3. Writing Committee for the OPRs, Domalpally A, Clemons TE et al. Peripheral Retinal Changes Associated with Age-Related Macular Degeneration in the Age-Related Eye Disease Study 2: Age-Related Eye Disease Study 2 Report Number 12 by the Age-Related Eye Disease Study 2 Optos PEripheral RetinA (OPERA) Study Research Group. *Ophthalmology* 2017; 124: 479-487.
4. Tan CS, Heussen F, Sadda SR. Peripheral autofluorescence and clinical findings in neovascular and non-neovascular age-related macular degeneration. *Ophthalmology* 2013; 120: 1271-1277.
5. Witmer MT, Kozbial A, Daniel S et al. Peripheral autofluorescence findings in age-related macular degeneration. *Acta Ophthalmol* 2012; 90: e428-433.
6. Suetsugu T, Kato A, Yoshida M et al. Evaluation of peripheral fundus autofluorescence in eyes with wet age-related macular degeneration. *Clin Ophthalmol* 2016; 10: 2497-2503.
7. Nivison-Smith L, Milston R, Chiang J et al. Peripheral retinal findings in populations with macular disease are similar to healthy eyes. *Ophthalmic Physiol Opt* 2018; 38: 584-595.
8. Lains I, Park DH, Mukai R et al. Peripheral Changes Associated With Delayed Dark Adaptation in Age-related Macular Degeneration. *Am J Ophthalmol* 2018; 190: 113-124.
9. Seddon JM, Reynolds R, Rosner B. Peripheral retinal drusen and reticular pigment: association with CFHY402H and CFHrs1410996 genotypes in family and twin studies. *Invest Ophthalmol Vis Sci* 2009; 50 :586-591.
10. Shuler RK, Jr., Schmidt S, Gallins P et al. Peripheral reticular pigmentary change is associated with complement factor H polymorphism (Y402H) in age-related macular degeneration. *Ophthalmology* 2008; 115: 520-524.
11. Munch IC, Ek J, Kessel L et al. Small, hard macular drusen and peripheral drusen: associations with AMD genotypes in the Inter99 Eye Study. *Invest Ophthalmol Vis Sci* 2010; 51: 2317-2321.
12. Ferris FL, 3rd, Wilkinson CP, Bird A et al. Clinical classification of age-related macular degeneration. *Ophthalmology* 2013; 120: 844-851.
13. Ferris FL, Davis MD, Clemons TE et al. A simplified severity scale for age-related macular degeneration: AREDS Report No. 18. *Arch Ophthalmol* 2005; 123: 1570-1574.

Ocular co-morbidities in domiciliary eye care

Mae FA Chong
BOptom PGDipAdvClinOptom PGCertOcTher FACO

Lead Optometrist Low Vision Services, Clinical Services Division

Lesley Dacion
BOptom, PGCertOcTher

Staff Optometrist, Clinical Services Division

Australian College of Optometry

The Australian College of Optometry (ACO) provides domiciliary care in a number of settings, including aged care facilities, supported residential services and crisis accommodation centres with a significant proportion of these services provided to older Australians (aged over 65 years).

The ocular conditions that an optometrist can expect to encounter in these settings have been summarised by the 2016 National Eye Health Survey (NEHS),[1] which investigated the causes of vision impairment in the Aboriginal and/or Torres Strait Islander (40 years and over) and non-Aboriginal and/or Torres Strait Islander (50 years and over) populations.

It was established that in non-Aboriginal and/or Torres Strait Islander Australians, the leading causes of vision loss were uncorrected refractive error (61.3 per cent), cataract (13.2 per cent) and age-related macular degeneration (AMD) (1.3 per cent), compared to uncorrected refractive error (60.8 per cent), cataract (20.1 per cent) and diabetic retinopathy (5.2 per cent) in Aboriginal and/or Torres Strait Islander peoples.

Uncorrected Refractive error

Uncorrected refractive error accounts for almost two-thirds of all cases of vision impairment in Australia in all groups. Unfortunately, this has not reduced in the last 20 years, with similar findings (62 per cent) in the Blue Mountains Eye Study and Melbourne Vision Impairment Project of the 1990s.[2] A major focus of optometry care, addressing refractive error remains an important part of service provision both in clinic and on domiciliary visits.

Cataract

With increasing age comes the likelihood of patients experiencing systemic co-morbidities rendering cataract surgery more challenging or not possible. Additionally, there are patients for whom cataract surgery is deemed to be of limited benefit due to the presence of compounding visually-limiting conditions, including AMD.

Cataract referral may also be delayed in older Australians due to a less visually-demanding lifestyle, for example: a patient who is no longer driving. It is well to remember that more indirect methods of examining the ocular fundus (for example binocular indirect ophthalmoscopy) or certain ocular coherence tomography (OCT) imaging systems incorporating a scanning laser ophthalmoscope (SLO) will be better at penetrating the opacified lens than direct methods (such as direct ophthalmoscopy).

Age-related macular degeneration

AMD is the predominant cause of vision impairment in optometry low vision clinics at Kooyong (49.0 per cent)[3] and the Australian College of Optometry (39.6 per cent).[4] Patients who require domiciliary optometry services may also be affected by limited access to anti-VEGF treatment due to financial or transport difficulties.

A difficulty with AMD in domiciliary care is the added challenge of not always having access to OCT and limited availability of a clear, highly magnified, binocular view of the ocular fundus to assess the profile of the macula. It is likely that patients with reduced vision and AMD will require earlier ophthalmology referral (or attendance at an optometry clinical practice) to investigate for the presence of a choroidal neovascular membrane.

Diabetic retinopathy

Patients with a diagnosis of diabetes mellitus can be well-cared for in domiciliary practice with portable diagnostic equipment to assess visual, cranial nerve function, anterior segment and dilated posterior ocular health. However, patients with sudden or unexplained visual acuity loss require careful attention, as they may benefit from early ophthalmology referral or attendance at an optometry clinical practice to confirm the presence or absence of macular oedema.

Low vision

The NEHS established the overall prevalence of vision loss in Australia as 6.5 per cent, with increasing rates with age (5.0 per cent in the 50–59-year cohort vs 37.3 per cent in the 90+ years cohort) and Aboriginal and/or Torres Strait Islander status (11.2 per cent overall). Many rehabilitation organisations, including Guide Dogs Australia and Vision Australia, provide domiciliary assessments and support; inability to attend a clinic should not be a reason to delay referral for such care. To provide introductory low vision services to patients as needed, a small selection of basic optical magnifiers is a worthwhile addition to a domiciliary visit kit.

DOMICILIARY CARE CASE REPORT 1

Mrs A was seen as part of an annual visiting optometry service to a large aged care facility situated in a small town about five hours drive from Melbourne, three hours from the closest regional centre with an

Continued page 8

Domiciliary eye care

From page 7

ophthalmology service.

Mrs A did not report any knowledge of past ocular health problems, nor any concerns with her vision. She reported undergoing right eye cataract surgery and reduced left vision (possibly since childhood). Best corrected visual acuities were R 6/9.5 L 6/38, with no improvement with pinhole. Portable slitlamp examination revealed bilateral posterior chamber intraocular lenses (IOLs), despite Mrs A being quite insistent of only ever having surgery to her right eye. Intraocular pressures were consistently asymmetric with repeated measurements (R 13 mmHg L 20 mmHg with Perkins applanation tonometry).

Mydriatic ocular fundus examination revealed a large area of macular swelling in the left eye, along with some fine new vessels on the superior margin of the left disc. Scattered small haemorrhages were also noted in the midperiphery of the left fundus.

After lengthy discussion with the patient, site staff and a later phone call to Mrs A's daughter who did not live locally, the decision was made to refer Mrs A to an ophthalmologist (three hours away), which required ambulance transport. A diagnosis of neovascular AMD was confirmed (Figure 1), however no further treatment was initiated due to difficulties with access and follow-up, and Mrs A's declining treatment.

This case highlights some of the additional challenges faced in domiciliary eye care, namely poor health literacy, limited continuity of care, access to ophthalmology care, providing care to patients with cognitive decline and consent regarding treatments.

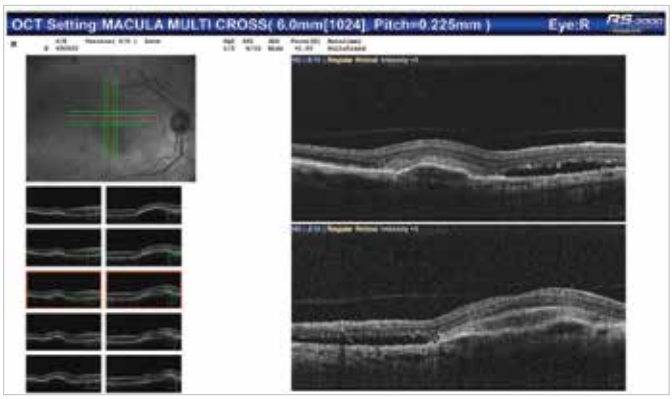


Figure 1. Nidek OCT scan (Macula Multi) demonstrating neovascular AMD in another ACO patient

DOMICILIARY CARE CASE REPORT 2

Mr B was provided optometry care at his residence in an aged care facility in metropolitan Melbourne. He reported a known history of 'dry' AMD, having previously been under the care of a private ophthalmologist and undergoing bilateral cataract surgery some years ago. He had not attended for ophthalmology review for some time, as he had been told, 'Nothing more could be done.' Mr B's main concern was being able to read books and the newspaper a little better.

Best corrected visual acuities were R 6/18 L 6/30. Appropriate near addition enabled N8 print to be achieved with limited fluency. Dilated ocular health examination revealed bilateral clear and stable IOLs, and confirmed intermediate AMD at both maculae.

Several hand-held magnifiers and stand magnifiers were trialled; as optometry assessment took place at Mr B's own residence, he was able to trial magnifiers at his own desk and with his own reading material. Lighting in his room was assessed, with an existing floor lamp relocated to provide extra focal lighting. In this way, Mr B was able to ascertain that a 3x illuminated stand magnifier was the most appropriate magnifier for his needs; enabling N4 print to be read fluently.

This case demonstrates the benefit of being able to conduct a low vision examination in a patient's own setting, with the practitioner able to provide practical advice regarding the setup of lighting and visual ergonomics to enable the comfortable use of magnifiers as low vision aids.

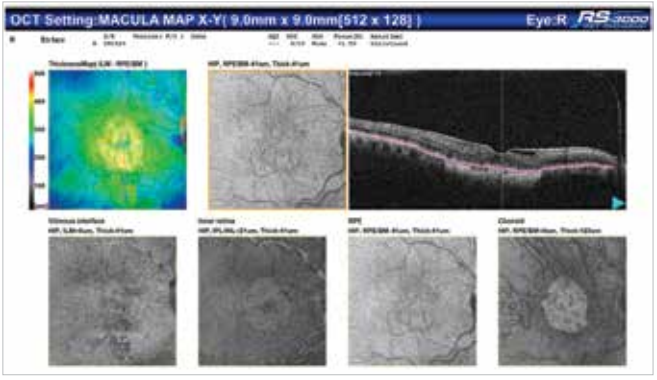


Figure 2. Nidek OCT (Macula Map) demonstrating late AMD (geographic atrophy) in the right eye

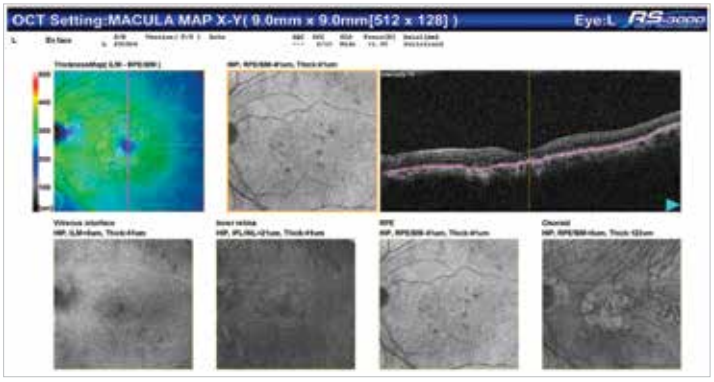


Figure 3. Nidek OCT (Macula Map) demonstrating late AMD (geographic atrophy) in the left eye

CLINIC-BASED CARE CASE REPORT 3

A contrasting case is Mrs C, a long-standing patient at the ACO Carlton clinic who resides in a nursing home and self-arranges transport for any required appointments. Having been monitored for many years with intermediate AMD, Mrs C presented for routine review, reporting a decline in her vision. She was found to have visual acuity R & L 6/120 reduced from 6/24 (six months prior) with a commensurate reduction in her near acuity. The anterior eye was unremarkable with posterior chamber IOLs noted to be clear and well positioned. Dilated ocular fundus examination revealed atrophic pigmentary changes at both maculae, which appeared to be flat. OCT confirmed that there was no choroidal neovascularisation and the maculae were flat with no subretinal fluid (Figures 2 and 3). Mrs C's ability to regularly attend a fully-equipped clinic for her care enabled a more conclusive diagnosis of intermediate AMD progressing to late AMD (geographic atrophy).

Conclusion

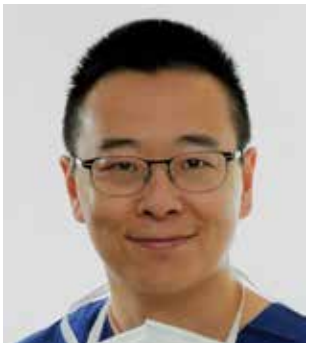
The ocular co-morbidities associated with domiciliary care are not dissimilar to those found in routine clinical practice; once the older population group is taken into consideration. The most frequently-expected finding is uncorrected refractive error and most conditions can be adequately managed with the portable diagnostic equipment that is the mainstay of domiciliary care. It is worth noting, however, that conditions such as AMD and diabetic retinopathy may require earlier referral to ophthalmology due to the lack of detailed ocular fundus examination and/or ocular diagnostic imaging in the domiciliary setting.

Additional challenges of delivering optometry care in an aged-care domiciliary setting include: limitations in health literacy, reduced continuity of care, limited access to ophthalmology, care provision with the overlay of cognitive decline, lack of health information or awareness and the difficulties in communicating with patients from culturally and linguistically diverse backgrounds. In these cases, it is important to liaise and engage with health workers and others involved in a patient's care to ensure that the most appropriate and timely attention is provided to patients in optometry domiciliary care. ▲

1. Foreman J, Xie J, Keel S et al. The prevalence and causes of vision loss in Indigenous and Non-Indigenous Australians: The National Eye Health Survey. *Ophthalmology* 2017; 124: 1743-1752.
2. Taylor HR, Keeffe JE, Vu HT et al. Vision loss in Australia. *Med J Aust* 2005; 182: 565-568.
3. Chong MFA, Jackson AJ, Wolffsohn JS et al. An update on the characteristics of patients attending the Kooyong Low Vision Clinic. *Clin Exp Optom* 2016; 99: 555-558.
4. Chong MFA, Cho HHI, Jackson AJ et al. Profile of the Australian College of Optometry Low Vision Clinic. C*lin Exp Optom* 2018; 101: 793-798

Anti-VEGF injections and irreversible visual loss

The role of the optometrist in



Dr Simon Chen
MBBS (London), BSc (Hons)
FRCOphth FRANZCO

Retinal & Cataract Surgeon

Conjoint Senior Lecturer
University of NSW

Anti-vascular endothelial growth factor (anti-VEGF) drugs have revolutionised the treatment of age-related macular degeneration (AMD) and have preserved the vision of patients throughout the world. Still, many millions remain at risk of severe AMD-related vision loss, and perhaps most disconcertingly, it is because they have chosen to discontinue their treatment.

Pharma's Clinical Editor Kerryn Hart recently conducted an interview with retinal surgeon Simon Chen to discuss the burdens and benefits of intravitreal injections and the role of the optometrist in improving patient adherence to anti-VEGF therapy. An edited version of the interview appears below.

KH: Dr Chen, anti-VEGF therapy has been an established treatment for neovascular AMD (nAMD) since 2006. Has anything changed since its initial appearance?

SC: The treatment itself has not fundamentally changed, the anti-VEGF agent is administered into the eye via an intravitreal injection on a regular basis. Beyond that, there have been three fundamental changes: 1) there has been a movement toward customised treat-and-extend protocols; 2) a growing understanding of the importance of aggressive and long-term treatment of nAMD; and 3) since 2006, a wider range of anti-VEGF agents have become available.

Undeniably, we now have a much greater understanding of important aspects of anti-VEGF therapy such as the pros and cons of different treatment regimens, the importance of life-long treatment for most patients, the risks and benefits of treatment and the long term visual outcomes of therapy.

Treat and extend protocols

Retinal specialists are shifting away from pro re data (PRN) treatment protocols (where injections are only administered when signs of disease activity recur) and regular fixed interval treatment protocols (where all patients are injected every four weeks) and towards customised 'treat and extend' protocols whereby the frequency of injections is tailored to the individual patient according to their own response to treatment.

Treat and extend protocols have been shown to provide better visual results than PRN protocols while reducing the number of injections needed compared to regular fixed interval treatment protocols.[1]

Importance of aggressive and long-term treatment

It's now well-established that patients need to be treated early in the disease process (that is, as soon as signs of nAMD become apparent). Treatment also needs to be administered frequently, especially in the early stages, when the disease is most active. We now know that for the majority of patients with nAMD, long term, potentially life long, anti-VEGF treatment is required because high rates of disease reactivation and permanent visual loss have been reported in patients that cease therapy.

KH: I've read that patients are often lost to follow-up with their anti-VEGF regime. Does your experience show this?

SC: Recent studies in the United States have indicated that up to 25 per cent of patients that start anti-VEGF treatment are eventually lost to follow-up.[2] This figure, of course, varies significantly between ophthalmic practices due to differences in patient demographics (older patients may find it more difficult logistically to attend for treatment and may be limited by other co-morbidities), patient socio-economic status and geographic location of the clinic.

KH: What are the key factors for patient drop out?

SC: For new patients, fear about possible pain or complications associated with the injection procedure are a common and natural reaction, especially in patients with a tendency towards anxiety.

Logistic barriers are a common cause for patients to drop out of treatment. (Difficulties travelling to and from a clinic due to factors such as inability to drive, difficulties accessing public transport, limited wheel chair access, poor memory causing patients to forget appointments, the need to arrange carers who may need to take time off work for injection visits). Patients may experience guilt about the ongoing burden on carers which in turn may makes them less likely to attend for treatment.

In older patients, co-morbidities may limit their access to care. For example: a patient suffering a hip fracture may be hospitalised and require long-periods of post-op rehabilitation causing them to miss out on scheduled anti-VEGF injections.

For patients with financial difficulties, concerns about out-of-pocket-costs associated with treatment may impact their treatment decision.

the prevention of

improving patient adherence to therapy

Kerryn Hart
BOptom GCertOcTher MPH

Clinical Skills Teacher In Optometry, Deakin University

Optometry Australia Policy and Standards Advisor

Clinical Editor of *Pharma*

Over time, 'injection fatigue' can set in among patients as they become frustrated with the ongoing need for treatment causing their commitment to treatment to wane.

For some patients there may be a lack of perceived benefit especially if treatment has not led to an improvement in their vision or their vision has continued to deteriorate despite treatment.

KH: What role does the optometrist play in improving compliance with anti-VEGF injections?

SC: Optometrists have a vital role to play in optimising patients' compliance with anti-VEGF therapy and therefore maximising their visual function and quality of life.

They can do this at an early stage by educating their patients with early-to-intermediate AMD about the long term risk of developing the neovascular form of AMD and making them aware of the potential vision-saving benefits of anti-VEGF injections before they have even developed nAMD. By exposing patients to the notion that anti-VEGF injections are an important treatment at an early stage, they may be better primed to accept the need for treatment when the time comes.

When optometrists refer patients with neovascular AMD to an ophthalmologist for anti-VEGF treatment, they can facilitate patient compliance by stressing the importance of attending the appointment without unnecessary delay. This should involve explicitly telling the patient that they are at risk of permanent visual loss if their condition is not assessed and managed in an appropriately urgent time frame. Without this information being provided to them, patients may not always appreciate the urgency of initiating treatment and so may delay seeing an ophthalmologist, potentially leading to irreversible visual loss due to progression of the neovascular process.

Optometrists also have an important continuing role in supporting patient compliance throughout the course of ongoing anti-VEGF therapy by providing ongoing educational and sometimes even emotional support. Patients should be reminded about the importance of ongoing treatment at every eye examination.

Many patients and their carers do not realise that nAMD is a chronic disease which is likely to require life-long treatment and unless patients are treated aggressively, they will generally lose vision. Real-world evidence has shown that anti-VEGF injection frequency is an important factor in achieving optimal gains in vision. In Australia, the Fight Retinal Blindness Study Group reported that visual acuity was maintained throughout five years of anti-VEGF treatment.[3] On average patients receive about five injections per year. Despite this, relative under-treatment is common.

Consistent messaging from different health care providers, including ophthalmologists, optometrists and general practitioners, helps to maintain patient compliance with treatment.

Patients should be encouraged to continue having regular optometric reviews even when they are having regular anti-VEGF injections as it is important that patients have the best optical correction possible in order to maximise their limited vision. Optometrists are well positioned to detect interval ocular pathology such as cataracts and assess the potential need for low vision services in patients with reduced vision.

Some patients are reluctant to voice concerns with their ophthalmologist and feel more comfortable discussing them with their optometrist. Concerns about injection-related discomfort, clinic waiting times and out-of-pocket costs are examples of issues that patients may find easier discussing with their optometrist. By encouraging the patient to talk to their ophthalmologist or liaising with the ophthalmologist on behalf of the patient, these issues can be addressed and optometrists may potentially prevent patients being lost to follow-up.

KH: Can you give any examples of where the optometrist has played an important role in improving/maintaining compliance with anti-VEGF injections?

SC: One example I am often reminded of is that of an 85 year-old woman that I am still seeing for regular anti-VEGF injections. She is an avid reader and passionate about painting. She is fiercely independent. Approximately five years ago, she developed loss of central vision in her only seeing eye. The fellow eye had become blind following complications of cataract surgery. Her vision had deteriorated rapidly to 6/60 over the course of a week due to nAMD. She had lost the ability to read, paint and drive. She was distressed about the possibility of losing her independence as she lived alone. She was referred to me for assessment by her optometrist whom she had been seeing for approximately 20 years.

At the initial consultation, I confirmed

Continued page 12

Anti-VEGF

From page 11

a diagnosis of nAMD and urged her to have treatment with anti-VEGF injections the same day. Despite my best efforts, she refused treatment because of a fear of complications that might lead to her losing more vision, as had happened when she had previously had cataract surgery. She promised to return the next day for treatment after she had time to collect her thoughts. The next day she failed to attend our arranged appointment and treatment. I spoke with her on the phone and she stated that she had determined that she did not want treatment and would just take her chances, letting nature take its course.

I called to explain the situation to her optometrist who took quick action in calling her and managed to convince her to attend for treatment the same day. It was the long-term rapport that she had built up with her optometrist over the years that provided the confidence to trust his advice. He explained that we had shared numerous patients with nAMD together and that the results had been very positive. He reiterated the potential consequences on her quality of life of not having treatment. She responded extremely well to treatment, eventually regaining a visual acuity of 6/9 which has been maintained to this day. She remains fully independent and her passion for reading and painting is unchanged. ▲

1. Mantel I. Optimizing the Anti-VEGF Treatment Strategy for Neovascular Age-Related Macular Degeneration: From Clinical Trials to Real-Life Requirements. *Transl Vis Sci Technol.* 2015; 4: 6.
2. Weiss M, Sim D, Herold T et al. Compliance and adherence of patients with diabetic macular edema to intravitreal anti-vascular endothelial growth factor therapy in daily practice. *Retina* 2018; 38: 2293–300.
3. Gillies MC, Campain A, Barthelmes D et al. Long-Term Outcomes of Treatment of Neovascular Age-Related Macular Degeneration: Data from an Observational Study. *Ophthalmology.* 2015; 122: 1837-1845

An extended version of this interview, including a step-by-step explanation of the anti-VEGF procedure, appears online in the open access version of this article on the *Pharma* page of the Optometry Australia website.

Evidence-based advice for

Clinical classifications, modifiable lifestyle

Dr Laura Downie
BOptom PhD(Melb) PGCertOcTher FAAO FACO DipMus(Prac) AMusA

Senior Lecturer, Department of Optometry and Vision Sciences, Faculty of Medicine, Dentistry and Health Sciences

The University of Melbourne

While therapeutic interventions exist for late-stage neovascular age-related macular degeneration (AMD), currently there are no approved medical therapies for earlier stages of the disease or late-stage geographic atrophy (GA). Given its association with sight-threatening retinal pathology, reducing progression to late-stage AMD is vital for decreasing vision loss and the associated individual and community burden of AMD.

AMD clinical classification

AMD is characterised by retinal changes that occur in a two-disc diameter radius of the fovea in people aged 55 years or older. The early stages of AMD are indicated by the appearance of drusen, comprising accumulations of lipoproteineous substance between the retinal pigment epithelium (RPE) and Bruch's membrane. Drusen can also be associated with disruptions to the RPE, evident clinically as areas of relative hyper- or hypo-pigmentation. AMD can then progress to late-stage GA of the RPE and/or choroidal neovascularisation (CNV).

In 2013, the Beckman Initiative for Macular Research Classification Committee, a panel of international experts in the field, published a key paper defining an AMD clinical classification for implementation in clinical research and practice.[1] This classification system (summarised on pages 14 and 15) defines five categories, based on retinal features, and is of value for predicting an individual's risk of developing late-stage AMD.[1]

There are several key points worth noting about this AMD classification scheme,[2] as follows:

- AMD severity is described in three stages: 'early,' 'intermediate' and 'late'
- the terms 'wet AMD' and 'dry AMD' are not used in this classification. These descriptors were judged to be potentially confusing, as 'dry AMD' has historically been used to describe a spectrum of AMD-related changes, ranging from isolated drusen to GA
- drusen are defined by their size (at their smallest diameter); subjective descriptors such as 'soft' and 'hard,' are not used
- the presence of only drupelets (a small druse of less than 63 µm in diameter) within two-disc diameters of the fovea defines a pre-AMD category of 'normal ageing changes,' which is distinct from 'early AMD.'

RISK FACTORS FOR AMD

There are several risk factors for AMD. In terms of non-modifiable risk, advancing age is the strongest factor. The risk of developing AMD is three times higher in individuals older than 75 years, compared to those between 65 and 74 years of age.[3] A family history of AMD, particularly having a first-degree family member affected by the condition, also confers a significantly-elevated risk of developing the disease.[4] Of major clinical importance is identifying—and if possible modifying—lifestyle risk factors that can also influence the development and/or progression of AMD.

Tobacco smoking

Cigarette smoking is the single most important modifiable AMD risk factor;[5] smoking at least doubles a person's risk of developing the condition.[6] Furthermore, a direct association has been identified between the number of cigarettes smoked over time and the risk of developing late-stage AMD.[7]

AMD

factors and the quality of eye care

Despite these known links between tobacco smoking and sight-threatening ocular disease, studies suggest that primary eye care providers may not be routinely asking their patients about smoking or providing advice about the benefits of smoking cessation. Research undertaken in several developed countries,[8-11] including Australia,[12] involving surveys of eye care clinicians, identifies scope for optometrists to be more proactive in discussing tobacco smoking as a modifiable risk factor for eye disease with their patients. Eye care clinicians have identified a range of potential barriers to undertaking smoking counselling with their patients; these include a perceived lack of sufficient consulting time to perform this task, a perception that there should be sufficient public awareness about the health risks of smoking and/or considering discussing smoking habits with their patients to be too intrusive.[12]

With the intent of overcoming these barriers, and to assist eye care clinicians with capturing key information about a patient's smoking behaviours and how these relate to AMD risk, my colleague Associate Professor Peter Keller and I developed a 'Quantitative Clinical Smoking Behaviour Tool.'[9] This tool, the first of its kind for eye-care clinicians, comprises 10 questions that capture information about a person's smoking behaviours, across three main areas:

1. Current and former smoking status (using a validated classification system), and how this informs a person's risk of developing AMD or having progressive disease.
2. Degree of smoking dependence, which relates to a current smoker's level of nicotine dependence.
3. Level of motivation to cease smoking, to ascertain a current smoker's readiness to consider smoking cessation, quantified on a validated behavioural scale.

For each of these three key areas, the tool provides an evidence-based summary (including a comprehensive list of relevant citations) that eye care clinicians can use to provide evidence-based advice to patients about the benefits of smoking cessation for their eye health.

Diet

Diet is another key area for potential AMD risk modification. Multiple epidemiological studies have reported the potential benefits of a healthy diet, rich in the macular carotenoids (zeaxanthin and lutein) and omega-3 fatty acids, for lowering the risk of developing AMD. A meta-analysis that included several observational studies reported that the consumption of two or more servings of oily fish per week was beneficial in the primary prevention of AMD.[13]

High glycaemic index diets and alcohol consumption (in excess of two drinks per day) may also increase the risk of AMD, although further studies are required to confirm these associations.[14]

A recent systematic review concluded that high consumption of vegetables rich in carotenoids and oily fish containing omega-3 fatty acids was beneficial for people at risk of AMD.[14] However, emphasising the need to differentiate between nutritional components derived from whole foods and supplementation, consuming anti-oxidant supplements does not prevent the development of AMD.[15]

In terms of modifying AMD progression, a Mediterranean diet (rich in foods such as fruits, vegetables, legumes, and fish) has been linked to a reduced risk of AMD progression.[16] Epidemiology studies suggest that a high dietary intake of omega-3 fatty acids is associated with a significant reduction in the risk of both intermediate[17,18] and late-stage AMD.[19,20] It has also been recommended that vegetable oils and animal fats (which contain high levels of omega-6 fatty acids) and red/processed meat should be consumed minimally to reduce the risk of AMD progression.[14]

Supplements

With respect to supplementation, the Age Related Eye Disease Study (AREDS) showed that daily, long-term, high-dose supplementation with vitamin C (500 mg), vitamin E (400 IU), beta-carotene (15 mg), zinc (80 mg, as zinc oxide), and copper (2 mg, as cupric oxide) in people with at least intermediate-stage AMD reduced the relative risk of progression to late AMD from 28 per cent to 20 per cent at five years.[21] As such, it may be relevant to consider the potential benefit of a high-dose anti-oxidant vitamin and mineral supplement in individuals with intermediate-stage AMD. The decision to recommend such formulations to patients requires consideration of the patient's systemic health, as well as the relative benefits versus risks of supplementation. For example, there is evidence that the risk of lung cancer is significantly increased with high-dose beta-carotene supplementation in current and former smokers.[22,23]

A quantitative clinical diet and nutritional supplement tool[9] is also now available for optometrists to use in their practice. This simple survey, developed as a companion to the smoking behaviour tool, supports the capture of key clinical information relating to an individual's diet that are relevant to the risk of AMD, as follows:

1. Omega-3 fatty acid intake.
2. Lutein and zeaxanthin intake.
3. Nutritional supplement consumption.

CLINICAL AUDIT

The MaD-CCAT tool

Through a collaborative project with Professor Robyn Guymer, Associate Professor Peter Keller, Dr Lauren Ayton, Professor Algis Vingrys and Ms Ji-hyun (Anna) Lee, funded by the Macular Disease Foundation Australia, my research team has developed an optometric clinical audit tool for assessing the quality of eye care provided to people with AMD.

The MaD-CCAT tool enables optometrists to evaluate their practices with respect to the clinical care provided to their AMD patients, relative to current evidence-based standards. This process enables the identification of potential areas for practice improvement, to enhance the quality and outcomes of optometric patient care to people with AMD.

Continued page 16

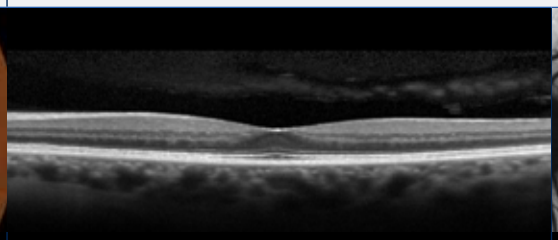
Clinical classification for Aged-Related Macular

From the 2019 Optometry Australia Clinical Practice Guide for the diagnosis, treatment and management

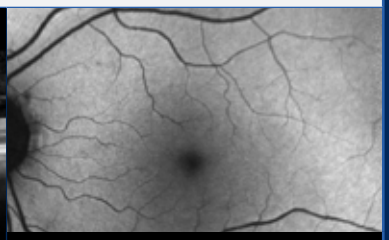
The most current clinical classification scheme for AMD is the Beckman classification.[1] The classifications are determined based on clinical examination (using common ophthalmoscopy equipment, such as an ophthalmoscope or slitlamp with accessory lenses) or evaluation of a fundus photo. Classification is based on fundus lesions within two disc diameters of the fovea in patients older than 55 years of age.

AMD classification	Definition
No apparent ageing changes	No drusen and no AMD pigmentary abnormalities†

retinal fundus photo



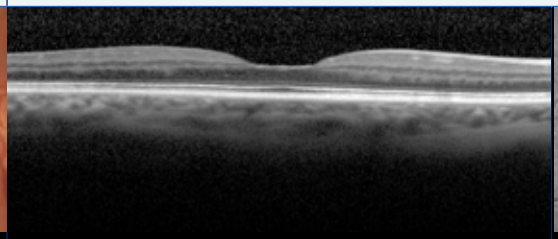
optical coherence tomography



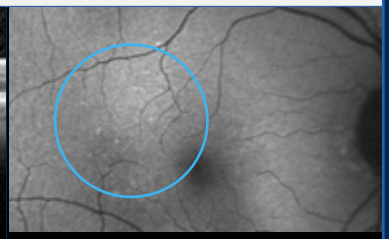
fundus autofluorescence

AMD classification	Definition
Normal ageing changes	Only drupelets (small drusen ≤ 63µm) and no AMD pigmentary abnormalities†

retinal fundus photo

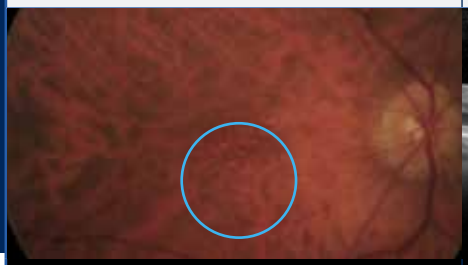


optical coherence tomography

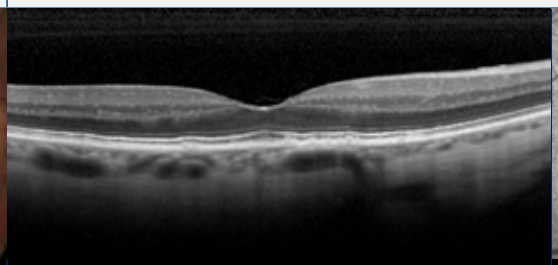


fundus autofluorescence

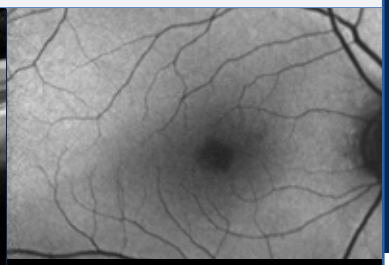
AMD classification	Definition
Early AMD	Medium drusen (> 63µm and ≤ 125µm) and no AMD pigmentary abnormalities†



retinal fundus photo

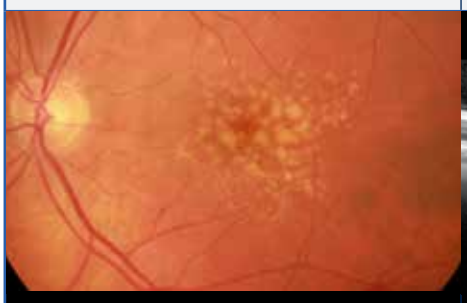


optical coherence tomography

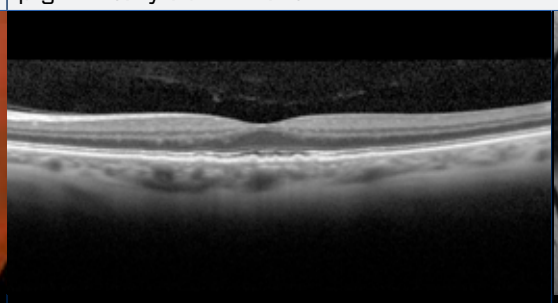


fundus autofluorescence

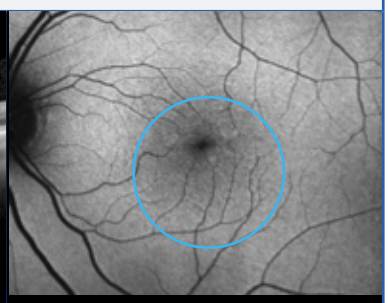
AMD classification	Definition
Intermediate AMD	Large drusen (> 125µm)‡ or medium drusen (> 63µm) in addition to AMD pigmentary abnormalities



retinal fundus photo



optical coherence tomography



fundus autofluorescence

1. Ferris FL, 3rd, Wilkinson CP, Bird A et al. Clinical classification of age-related macular degeneration. *Ophthalmology* 2013; 120: 844-851.
2. Ferris FL, Davis MD, Clemons TE et al. A simplified severity scale for age-related macular degeneration: AREDS Report No. 18. *Arch Ophthalmol* 2005; 123: 1570-1574.

Degeneration (AMD)

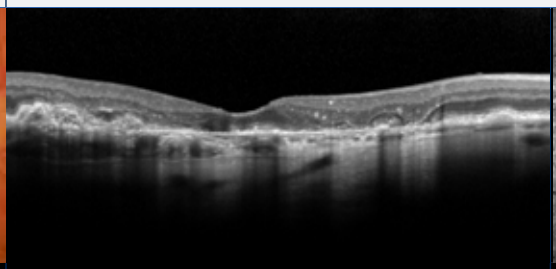
of Age-Related Macular Degeneration.

Optometry AUSTRALIA

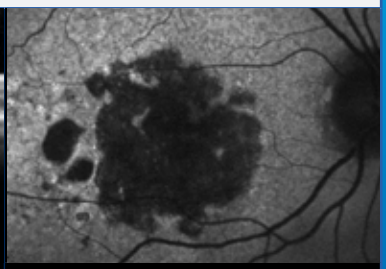
The Beckman classification is based on fundus lesions within two disc diameters of the fovea in patients older than 55 years of age.

AMD classification	Definition
Late AMD	Geographic atrophy (GA)

retinal fundus photo

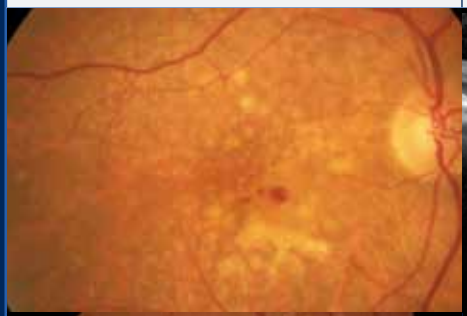


optical coherence tomography

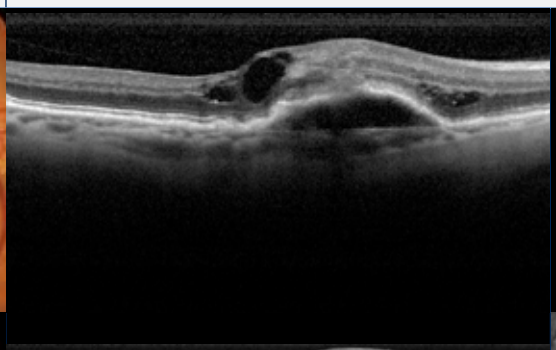


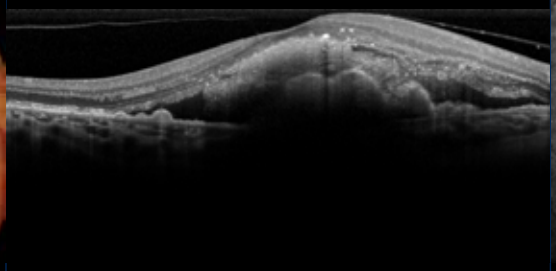
fundus autofluorescence

AMD classification	Definition
Late AMD	Neovascular AMD (nAMD)

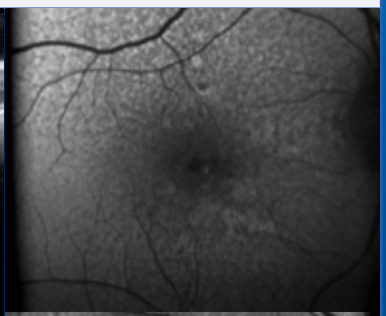


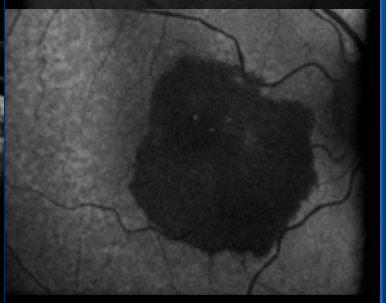
retinal fundus photo





optical coherence tomography





fundus autofluorescence

†AMD pigmentary abnormalities are defined as any definite hyper-pigmentary or hypo-pigmentary abnormalities associated with medium or large drusen, but not associated with known disease entities.

‡125µm is the approximate width of the major retinal venule as it crosses the optic disc margin.

Five-year risk of progression to late AMD[2]

Risk factors	Risk of progression for patients without late AMD in either eye at baseline*	Risk of progression for patients with late AMD in one eye at baseline^
0	0.4%	
1	3.1%	
2	11.8%	14.8%
3	25.9%	35.4%
4	47.3%	53.1%

*** Assign one risk factor: for each eye with large drusen**

- for each eye with pigment abnormalities
- if neither eye has large drusen and both eyes have medium drusen (early AMD)

^ Assign two risk factors for the eye that has late AMD.

- Assign an additional risk factor if the eye at risk has large drusen and an additional risk factor if the eye at risk also has pigmentary abnormalities.

Table 1. The Beckman classification scheme was designed to reflect the fact that risk profiles are linked to the clinical signs of drusen and pigmentary abnormalities. In early AMD (medium drusen only), people have a 3.1 per cent chance of progressing to late AMD within five years.[2] However, once a person has large drusen and pigmentary abnormalities in both eyes (intermediate AMD), this risk increases to around 47.3%.[2] If a patient presents with late AMD in one eye at baseline, the risk of progression in the other eye is slightly higher.[2]

EVIDENCE

From page 13

The MaD-CCAT supports streamlined auditing of multiple aspects of AMD clinical care, including: the identification of modifiable risk factors, diagnostic accuracy (including AMD severity classification), rate/timeliness and appropriateness of referrals for ophthalmologic evaluation, and the quality of clinical record keeping.

Data are captured using a check-box system, for ease of entry. A summary statistics worksheet then automatically populates information comparing an optometrist's practices with current best-practice guidelines for diagnosing and managing AMD.

A representative snapshot of the data analytics 'Overview' page is provided in Figure 1. As audit data are progressively added, the summary statistics highlight areas of relative strength and potential areas for practice improvement. Clinicians can then self-identify practice areas for continuous improvement.

It is of vital importance that primary eye-care providers identify and provide evidence-based advice to their patients in relation to modifiable risk factors for AMD. The availability of new clinical tools to enable clinicians to undertake these assessments, and self-evaluate their own clinical practices, provides a basis for ongoing practice improvement to optimise the delivery of primary eye care to people with AMD. ▲

1. Ferris FI, Wilkinson CP, Bird A et al. Clinical Classification of Age-related Macular Degeneration. *Ophthalmology* 2013; 120: 844-851.
2. Downie LE, Keller PR. Nutrition and age-related macular degeneration: research evidence in practice. *Optom Vis Sci* 2014; 91: 821-831.
3. Klein R KB, Knudtson MD, Meuer SM et al. Fifteen-year cumulative incidence of age-related macular degeneration: the Beaver Dam Eye Study. *Ophthalmology* 2007; 114: 253-262.
4. Klein ML, Francis PJ, Ferris FL, 3rd et al. Risk assessment model for development of advanced age-related macular degeneration. *Arch Ophthalmol* 2011; 129: 1543-1550.
5. Thornton J, Edwards R, Mitchell P et al. Smoking and age-related macular degeneration: a review of association. *Eye (Lond)* 2005; 19: 935-944.
6. Evans JA, Fletcher AE, Wormald RP. 28,000 Cases of age related macular degeneration causing visual loss in people aged 75 years and above in the United Kingdom may be attributable to smoking. *Br J Ophthalmol* 2005; 89: 550-553.
7. Khan JC, Thurlby DA et al. 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: 75-80.
8. Lawrenson JG, Evans JR. Advice about diet and smoking for people with or at risk of age-related macular degeneration: a cross-sectional survey of eye care professionals in the UK. *BMC Public Health* 2013; 13: 564.
9. Thompson C, Harrison RA, Wilkinson SC et al. Attitudes of community optometrists to smoking cessation: an untapped opportunity overlooked? *Ophthal Physiol Opt* 2007; 27: 389-393.
10. Caban-Martinez AJ, Davila EP, Lam BL et al. Age-Related Macular Degeneration and Smoking Cessation Advice by Eye Care Providers: A Pilot Study. *Prev Chron Dis* 2011; 8: A147.
11. Brûlé J, Abboud C, Deschambault E. Smoking cessation counselling practices among Québec optometrists: evaluating beliefs, practices, barriers and needs. *Clin Exp Optom* 2012; 95: 599-605.
12. Downie LE, Keller PR. The self-reported clinical practice behaviors of Australian optometrists as related to smoking, diet and nutritional supplementation. PLOS One 2015.
13. Chong EW, Wong TY, Kreis AJ et al. Dietary antioxidants and primary prevention of age related macular degeneration: systematic review and meta-analysis. *BMJ* 2007; 335: 755.
14. Chapman NA, Jacobs RJ, Braakhuis AJ. Role of diet and food intake in age-related macular degeneration: a systematic review. *Clin Exp Ophthalmol* 2019; 47: 106-127.
15. Evans JR, Lawrenson JG. Antioxidant vitamin and mineral supplements for preventing age-related macular degeneration. *Cochrane Database Syst Rev* 2017; 7: Cd000253.
16. Merle BMJ, Colijn JM, Cougnard-Gregoire A et al. Mediterranean Diet and Incidence of Advanced Age-Related Macular Degeneration: The EYE-RISK Consortium. *Ophthalmology* 2019; 126: 381-390.
17. Christen WG, Schaumberg DA, Glynn RJ et al. Dietary omega-3 fatty acid and fish intake and incident age-related macular degeneration in women. *Arch Ophthalmol* 2011; 129: 921-929.
18. Seddon JM, George S, Rosner B. Cigarette smoking, fish consumption, omega-3 fatty acid intake, and associations with age-related macular degeneration: the US Twin Study of Age-Related Macular Degeneration. *Arch Ophthalmol* 2006; 124: 995-1001.
19. Chong EW, Kreis AJ, Wong TY et al. Dietary omega-3 fatty acid and fish intake in the primary prevention of age-related macular degeneration: a systematic review and meta-analysis. *Arch Ophthalmol* 2008; 126: 826-833.
20. Sangiovanni JP, Agron E, Meleth AD et al. {omega}-3 Long-chain polyunsaturated fatty acid intake and 12-y incidence of neovascular age-related macular degeneration and central geographic atrophy: AREDS report 30, a prospective cohort study from the Age-Related Eye Disease Study. *Am J Clin Nutr* 2009; 90: 1601-1607.
21. Age-related eye disease study research group. A randomized, placebo-controlled, clinical trial of high-dose supplementation with vitamins C and E, beta carotene, and zinc for age-related macular degeneration and vision loss: AREDS report no. 8. *Arch Ophthalmol* 2001; 119: 1417-1436.
22. The Alpha-Tocopherol, Beta Carotene Cancer Prevention Study Group. The effect of vitamin E and beta carotene on the incidence of lung cancer and other cancers in male smokers. *N Engl J Med* 1994; 330: 1029-1035.
23. Omenn GS, Goodman GE, Thornquist MD et al. Effects of a combination of beta carotene and vitamin A on lung cancer and cardiovascular disease. *N Engl J Med* 1996; 334: 1150-1155.

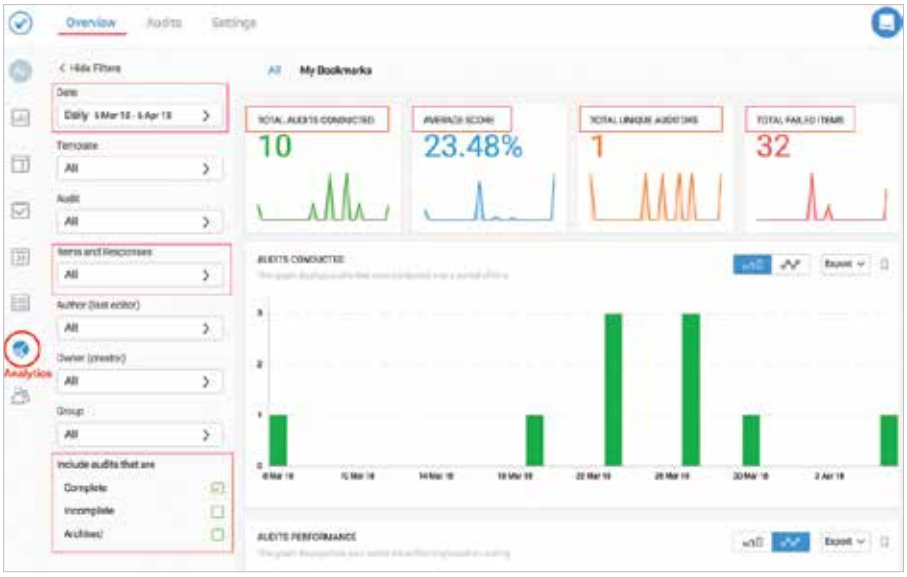


Figure 1. Snapshot of the MaD-CCAT analytics page, which provides clinicians with summarised information about their clinical audit contributions.

§ Optometrists interested in accessing the MaD-CCAT tool, the diet and nutritional supplement tool and the quantitative clinical smoking behaviour tool can email the author directly at ldownie@unimelb.edu.au.

Nanosecond laser treatment for age-related macular degeneration

Professor Erica L Fletcher
MScOptom PhD

Department of Anatomy and Neuroscience, University of Melbourne

Professor Robyn H Guymer
AM FRANZCO PhD

Centre for Eye Research Australia

Royal Victorian Eye and Ear Hospital

University of Melbourne, Department of Surgery (Ophthalmology)

Could the results from a recent clinical trial LEAD the way to a new AMD treatment?

Age-related macular degeneration (AMD) is the leading cause of irreversible vision loss in industrialised nations, and costs the Australian economy in excess of $2.5 billion per year.[1,2]

Treatment in the form of anti-VEGF agents can reduce vision loss in advanced 'wet' AMD. And although there is evidence-based advice available on modifying lifestyle risk factors that can influence the development/progression of AMD, there is currently no definitive way of reducing progression to advanced disease.

In addition, many people treated with anti-VEGF agents experience ongoing vision loss to less than 6/60 after seven years of anti-VEGF therapy.[3] For improved care, effective treatments for early-stage disease, or that reduce progression, are required.

The recent development of the nanosecond laser (2RT, Ellex, Pty Ltd) offers great promise as a treatment that may reduce progression of disease.[4] Below, we have summarised the rationale for this trial and its main outcomes. It's important to point out that, although promising, further work is required before nanosecond laser treatment can become a part of the mainstream standard of care for those with intermediate AMD.

Continuous-wave laser

Drusen are an important early feature of AMD, whose size is predictive of the risk of progression. Importantly, large drusen > 125 µm in diameter together with pigmentary changes is known to lead to advanced vision-threatening disease in approximately 50 per cent of patients within five years.[5]

Methods of treating eyes with drusen as a means for reducing progression of AMD have been investigated since the initial observation by Gass that laser photocoagulation was associated with drusen regression in some patients.[6] Consequently, a series of large, multicentre trials investigating the potential of continuous-wave lasers to reduce progression of disease were conducted in the 1980s, with mixed results.[7] In particular, although many studies showed reduction in drusen in response to laser therapy, some studies reported an acceleration of neovascular complications in the early period after treatment. Nine multicentre clinical trials have recently been evaluated in a Cochrane study and demonstrated that, although drusen regression was reduced nine-fold (odds ratio > 9), laser treatment with continuous-wave lasers neither slowed, nor accelerated disease progression.[8] Nevertheless, in view of the concerns for disease progression, laser treatment for drusen was largely abandoned.

Nanosecond laser

The nanosecond laser is a recently-developed unique laser that is quite unlike its continuous-wave predecessors.[9] It is a 532 nm pulsed laser, that delivers energy in a speckled pattern and selectively targets the retinal pigment epithelium (RPE) with single 3 ns pulses.[10] Owing to its short pulse length, the amount of energy absorbed by melanin within the RPE is 1/500th of that of a continuous-wave laser, and thus there is little if any 'thermal' damage of cellular tissue, including the adjacent retina or choroid.[10] Preclinical studies show that the nanosecond laser selectively ablates small areas of the RPE, inducing a healing response that is accompanied by drusen regression in some people.[10,11] Importantly, Bruch's membrane thins in response to a single laser treatment, suggesting an improvement in posterior eye health. Moreover, this change occurred in the absence of any noticeable damage in the neural retina.[10] A re-examination of the potential for laser treatment to reduce progression of AMD was, therefore, warranted.

LEAD trial

The Laser intervention in Early stages of Age Related Macular Degeneration (LEAD) trial was a multicentre clinical trial that evaluated whether nanosecond laser treatment of patients with intermediate AMD could reduce progression to advanced disease (either signs of choroidal neovascularisation, or atrophy, which, for the first time in a trial, was defined using multi modal imaging).[4] In total, 292 participants across five sites in Australia, and one in Northern Ireland were enrolled and treated with nanosecond laser (n = 147) or sham (n = 145) every six months for 36 months. Participants enrolled were those with intermediate AMD; they had large (> 125 µm) drusen in both eyes and had been carefully screened by

Continued page 18

LEAD trial

From page 17

OCT to be free of any sign of atrophy (nascent geographic atrophy). Every six months, patients received 12 laser or sham laser spots around the macula in a manner that was not targeted to the drusen directly. Advancing disease was defined as the presence of any sign of atrophy on OCT, geographic atrophy (GA) or choroidal neovascularisation (CNV).

The main (primary) outcome of the LEAD trial—that nanosecond laser reduced progression of AMD—was not achieved.[4] That is, in the 292 patients with intermediate AMD, there was neither an acceleration nor a reduction in the progression of AMD to vision-threatening advanced disease over the three year follow-up period. Forty-five participants developed late AMD, including 20 in the laser group and 25 in the sham group. However, an important observation was made that suggested that not all patients responded in the same way to the laser treatment.

Reticular pseudodrusen

Using a post-hoc analysis, patients with conventional (large) drusen showed a four-fold reduction in progression, whereas those with reticular pseudodrusen (RPD) potentially showed an acceleration (worsening) in disease (Table 1). Reticular pseudodrusen are a recently-described deposit that form in the subretinal space, between photoreceptor outer-segments and the RPE (Figure 1).

Treatment	With RPD	Without RPD
Sham (n = 145)	6/35 (17%)	22/110 (20%)
Laser (n = 147)	13/35 (37%)	15/112 (13.4%)

Table 1. Summary of the number of participants receiving sham or nanosecond laser treatment that showed advancement of disease, stratified by the presence of reticular pseudodrusen. Each part of the table shows the number of affected individuals compared with the total, with the percentage in parentheses. (Table republished with permission from Guymer et al.)[4]

Reticular pseudodrusen have a different composition to conventional drusen that form beneath the RPE between the RPE and Bruch's membrane, and are likely to be of different aetiology.[12] It is possible that RPD develop as a consequence of an unhealthy RPE and represent a further advancement in RPE pathology beyond what is associated with conventional drusen. It is possible that in those with RPD, the RPE is so unhealthy that a laser treatment that selectively ablates RPE simply adds to the already sick and dying RPE, hastening the progression of disease.

Why the LEAD trial matters

The outcomes of the LEAD trial are important for several reasons. First, it was the first clinical trial that used OCT-defined atrophy as a way to demonstrate advancement of disease. Importantly, areas of atrophy can be identified by OCT, and is termed nascent geographic atrophy (nGA) and is visible on OCT prior to the development of GA. The LEAD trial is the first trial to use this novel early atrophic change as part of a combined atrophic endpoint with GA, paving the way for other early interventional trials to use similar design. Secondly, the LEAD trial showed that those with RPD may respond in a different way to therapy to those with conventional drusen. Finally, the results of this trial provide valuable information about laser treatment as a means for reducing progression.

It's essential to point out that, when all participants are considered together, progression of AMD was not reduced. This means that further studies are required to determine the effect that drusen type has on treatment outcome. A clearer recommendation is not possible at this time, due to the design of the trial.

Importantly, the treatment effect on different AMD phenotypes was done in a post-hoc manner which means it was not planned at the beginning of the trial and, as such, the interpretation of the results needs to be considered as 'exploratory and hypothesis-generating,' not 'definitive proof' of a treatment effect.

At the time that the LEAD trial was initiated, very little was known about RPD, so participants were not

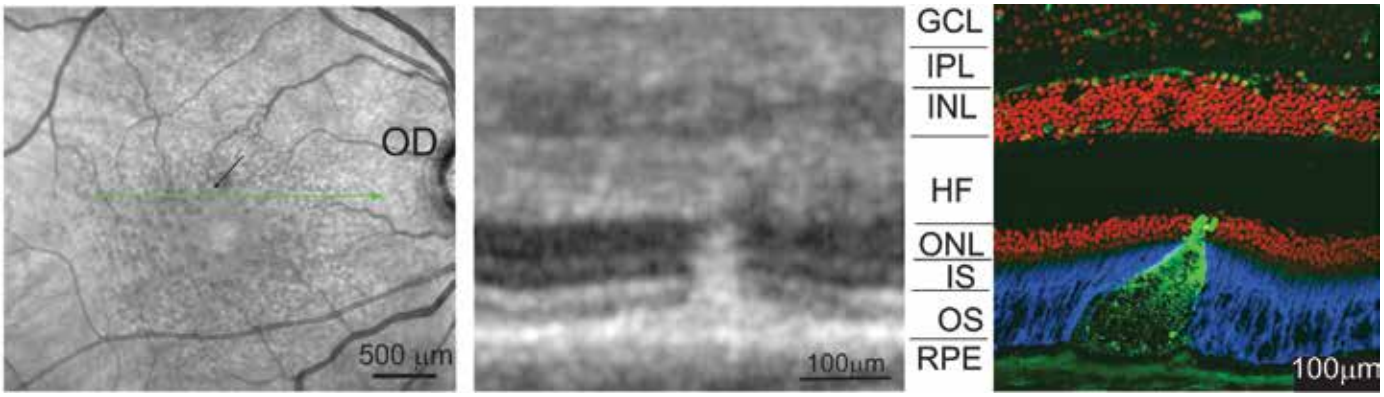


Figure 1. Fundus image and companion OCT of a person with reticular pseudodrusen. The posterior eye for the same person was processed for immunocytochemical labelling for vitronectin (green; a plasma protein that labels retinal deposits), peanut agglutinin that labels photoreceptor outer-segments (blue) and a nuclear stain (red). Histological analysis demonstrates that reticular pseudodrusen are deposits that form within the subretinal space between the photoreceptor outer segments and retinal pigment epithelium. (Image republished with permission from Greferath et al.)[12]

randomised into groups with and without RPD. It has only been since then that the role of RPD in disease progression has been determined. Only 70 participants in the LEAD trial had RPD at baseline, making definitive conclusions about laser treatment in those with RPD impossible.

In summary, the use of nanosecond laser for treating patients with intermediate AMD shows some promising results for reducing progression of disease, especially in those with conventional drusen. However, as the results of the LEAD trial were not definitive, more work is needed before it can be recommended for broad use in the community. Follow-up studies are required to validate the LEAD results and to evaluate how laser treatment effects disease progression in those with different forms of drusen.

If the reductions in progression in those with conventional drusen are replicated, it would represent a major advance in how we manage and treat those with AMD. ▲

1. Lim LS, Mitchell P, Seddon JM et al. Age-related macular degeneration. *Lancet* 2012; 379: 1728-1738.
2. Rein DB, Wittenborn JS, Zhang X et al. Forecasting age-related macular degeneration through the year 2050: the potential impact of new treatments. *Arch Ophthalmol* 2009; 127: 533-540.
3. Rofagha S, Bhisitkul RB, Boyer DS et al. Seven-year outcomes in ranibizumab-treated patients in ANCHOR, MARINA, and HORIZON: a multicenter cohort study (SEVEN-UP). *Ophthalmology* 2013; 120: 2292-2299.
4. Guymer RH, Wu Z, Hodgson LAB et al. Subthreshold Nanosecond Laser Intervention in Age-Related Macular Degeneration: The LEAD Randomized Controlled Clinical Trial. *Ophthalmology* 2018 doi: 10.1016/j.ophtha.2018.09.015. [Epub ahead of print]
5. Ferris FL, 3rd, Wilkinson CP, Bird A et al. Clinical classification of age-related macular degeneration. *Ophthalmology* 2013; 120: 844-851.
6. Gass JD. Drusen and disciform macular detachment and degeneration. *Arch Ophthalmol* 1973; 90: 206-217.
7. Findlay Q, Jobling AI, Vessey KA et al. Prophylactic laser in age-related macular degeneration: the past, the present and the future. *Eye (Lond)* 2018; 32: 972-980.
8. Virgili G, Michelessi M, Parodi MB et al. Laser treatment of drusen to prevent progression to advanced age-related macular degeneration. *Cochrane Database Syst Rev* 2015: CD006537.
9. Wood JP, Plunkett M, Previn V et al. Nanosecond pulse lasers for retinal applications. *Lasers Surg Med* 2011; 43: 499-510.
10. Jobling AI, Guymer RH, Vessey KA et al. Nanosecond laser therapy reverses pathologic and molecular changes in age-related macular degeneration without retinal damage. *FASEB J* 2015; 29: 696-710.
11. Guymer RH, Brassington KH, Dimitrov P et al. Nanosecond-laser application in intermediate AMD: 12-month results of fundus appearance and macular function. *Clin Experiment Ophthalmol* 2014; 42: 466-479.
12. Greferath U, Guymer RH, Vessey KA et al. Correlation of Histologic Features with In Vivo Imaging of Reticular Pseudodrusen. *Ophthalmology* 2016; 123: 1320-1331.

Professor Erica Fletcher presents 'What's new in management and treatment of age-related macular degeneration' on Sunday, July 21 as part of Optometry Victoria's O=MEGA19 event at the Melbourne Convention and Exhibition Centre.

OCT in the investigation of systemic neurologic disease

Summary and comment provided by
Maria Markoulli PhD MOptom GradCertOcTher FBCLA FAAO
Deputy Editor, *Clinical and Experimental Optometry*

Senior Lecturer Postgraduate Research Coordinator
School of Optometry and Vision Science, UNSW Sydney

Dr Sangeetha Srinivasan
PhD

Emeritus Professor Nathan Efron
AC DSc PhD FAAO

Institute of Health and Biomedical Innovation, Queensland University of Technology

In this issue, Clinical and Experimental Optometry *Deputy Editor Maria Markoulli offers a look at a review published in* Clinical and Experimental Optometry *that may herald a new frontier in optometry: the standard use of an OCT to identify a range of neurodegenerative disorders.*

In practice, we will frequently encounter patients with neurodegenerative conditions such as age-related Parkinson's and Alzheimer's disease, as well as multiple sclerosis and diabetic peripheral neuropathy. The prevalence of these conditions is on the rise, courtesy of the ageing population in Australia. Having a means by which to detect these conditions accurately and prior to the onset of symptoms will enable multidisciplinary teams of clinicians to better manage the sequelae of these diseases at an earlier stage.

Optometrists are in a key position to facilitate this early detection. As optometrists, we know only too well that the eye is the window to the rest of the body. The transparency of the ocular media makes the eye the perfect site for direct and non-invasive examination of vascular and neural tissue. With the majority of optometrists either owning an optical coherence tomographer (OCT), or having easy access to one, the imaging of retinal neuronal and vascular structures is now routine and is being recommended as a first-line of screening for neurodegenerative systemic disease. In their recent publication, authors Sangeetha Srinivasan and Nathan Efron from the Queensland University of Technology review the literature and present an update of where we currently stand with the use of this technology in investigating systemic neurologic disease.

The authors explain that the layers that have received considerable attention are the retinal nerve fibre layer (NFL) and the ganglion cell complex (GCC). The ganglion cell complex comprises three layers: the nerve fibre layer, made up of axons of ganglion cells; the ganglion cell layer, made up of cell bodies of ganglion cells; and the inner plexiform layer, made up of dendrites of ganglion cells. Changes to the NFL and GCC as well as macular thickness have been advocated as potential disease biomarkers (Figure 1).

Parkinson's disease

Parkinson's disease is a progressive neurodegenerative disorder that results from a loss of nerve cells in the substantia nigra of the brain and accumulation of Lewy bodises. This causes a downstream loss of dopaminergic cells and a reduction in the neurotransmitter dopamine which regulates body movement. This results in the clinical signs we are familiar with: involuntary shaking of the body, stiffness of skeletal muscles and loss of balance. Using OCT, a reduction in retinal nerve fibre layer thickness, especially inferiorly, has been found in individuals with Parkinson's disease compared to healthy age-matched individuals. In an animal study, these retinal changes have been found to precede dopaminergic cell

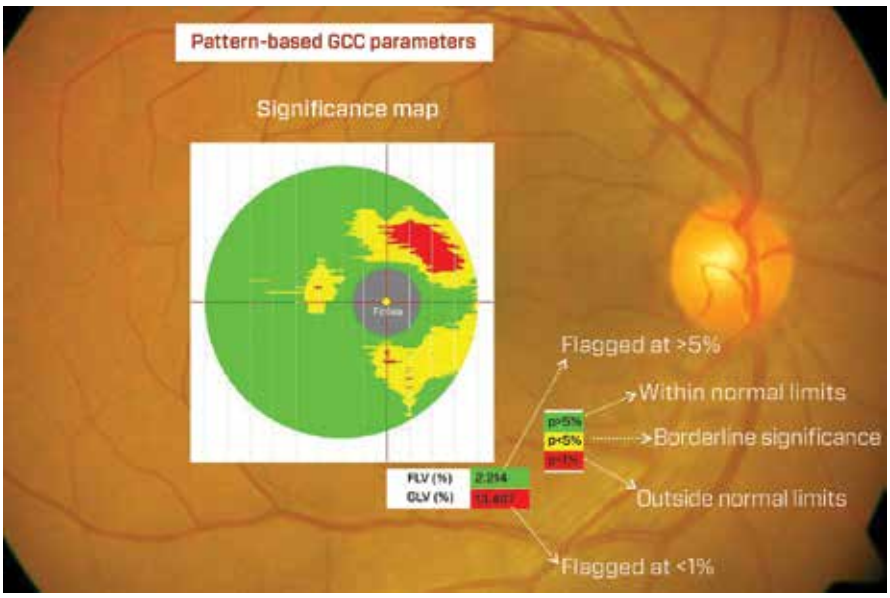


Figure 1. Pattern-based ganglion cell complex parameters as assessed by OCT. (FLV (%) = focal loss volume percentage. GLV (%) = global loss volume percentage).

CLINICAL AND EXPERIMENTAL OPTOMETRY

Pharma and Optometry Australia's official journal *Clinical and Experimental Optometry (CXO)* are collaborating to bring our readers up to date with some of the most interesting articles, reviews and original research available in the latest issues of *CXO*.

loss, suggesting that retinal changes may be predictive of the disease.

A thinner macular has also been reported, while an increase in outer plexiform layer thickness and volume has been shown. Interestingly, a thicker choroid has been reported in Parkinson's disease, possibly related to the changes in the density of connective tissue surrounding the vasculature in Parkinson's disease. The recommendations from the review by Srinivasan and Efron are that when a patient presents to a neurology or eye clinic, if mild tremors are noted, OCT can be used as a screening tool. If retinal structural abnormalities are detected, it is possible that the patient has Parkinson's disease.

In the case that such retinal abnormalities are not detected, functional testing such as colour vision and contrast sensitivity may reveal functional compromise. These ocular assessment tools can also give a measure of disease progression and can lead to appropriate and timely referral for symptom management.

Multiple sclerosis

Multiple sclerosis is a demyelinating disease that affects the central nervous system, and hence cranial nerve 2 (the optic nerve). The underlying mechanisms are thought to be an autoimmune response to self-antigens that work against myelin, cellular and axonal components. The optic nerve is therefore affected with potential irreversible damage to retinal structure and function. Multiple systems are involved, leading to loss of balance, muscle stiffness, fatigue and poor coordination. Retinal nerve layer measures have been found to correlate with visual functional and cognitive measures and optic neuritis is the first clinical presentation in 20 per cent of patients with the disease, with 70 per cent developing optic neuritis at some stage of the disease.

The recommendation from this review is that optometrists could play a role in the diagnosis or management of individuals with multiple sclerosis.

Someone presenting to the clinic with blurred vision that cannot be explained by refraction, symptoms of fatigue, poor balance and coordination should be assessed for their extraocular movements and signs of a relative afferent pupillary defect.

The optic nerve head may appear normal in case of lesions beyond the optic nerve head. In this case, an OCT examination can be combined with visual field or colour vision tests, which may reveal retinal structural or functional compromise, or both, even in the absence of clinically visible optic neuropathy. The individual may then be referred to a neurologist for further investigation.

Alzheimer's disease

Alzheimer's disease is associated with memory loss and cognitive impairment. In the brain, structural changes include deposition of amyloid plaques, hippocampal atrophy and the presence of neurofibrillary tangles. These changes can only be detected by cerebrospinal analysis, imaging or post-mortem examination. Due to the embryogenic similarities between brain tissue and the eye, ocular changes can give a sense of the neurological changes in the disease.

OCT examination reveals a reduced macular volume and a thinner retinal nerve fibre layer by an average of 12 µm. The retinal ganglion cell complex thickness has also been reported to be reduced in patients with Alzheimer's disease, the hypothesis being that this is a result of deposition of amyloid-B plaques in the retina, which in many cases occurs prior to cognitive decline. This places optometrists in a unique position to use OCT for the detection of Alzheimer's disease in its early stages and in monitoring the effect of treatment.

Diabetic peripheral neuropathy

Diabetic peripheral neuropathy affects up to 50 per cent of individuals with diabetes and can lead to painful symptoms, foot ulceration and in severe instances, amputation.

Traditional investigations of neuropathy include vibration threshold tests or electrophysiology, which predominantly explore the function of large nerve fibres. However, it is the small nerve fibres that are affected first and the techniques available for testing these nerves are either invasive or unreliable.

The vascular and neural nature of the retina makes it the perfect environment for *in vivo* assessment in diabetes. Ganglion cells have been shown to be lost early in diabetes irrespective of the presence of diabetic retinopathy.

When the diagnostic capability of OCT-derived parameters was explored, of all the retinal parameters, the focal loss volume had 53 per cent sensitivity and 80 per cent specificity, with an area under the receiver operating characteristic curve of 0.829 in differentiating moderate or severe neuropathy, indicating that OCT may detect a previously undiagnosed neuropathy.

The authors conclude from their review that OCT, along with many other diagnostic instruments available to optometrists, can assist in our understanding, diagnosis and management of systemic neurodegenerative diseases. Future work needs to confirm the role of these retinal markers so that optometrists can assist multidisciplinary teams in the co-management and timely referrals of such patients. ▲

Srinivasan S, Efron N. Optical coherence tomography in the investigation of systemic neurologic disease *Clinical and Experimental Optometry* 2018 Dec 11. doi: 10.1111/cxo.12858. [Epub ahead of print]

Not quite as it seems

How to differentiate macular dystrophy from AMD

Pauline Xu
BOptom (Hons) MOptom GradCertOcTher

Lead Clinician - Retinal Dystrophies
Centre for Eye Health

Macular dystrophies refer to a heterogeneous group of inherited disorders characterised by bilateral central vision loss and generally symmetric fundoscopic presentation of macular abnormalities.[1] Due to its overlapping clinical features with age-related macular degeneration (AMD), it can be easily misdiagnosed. Although electrophysiology and genetic testing can provide valuable information, this additional workup is not readily available for the everyday clinician. A thorough understanding of mimicking and differentiating signs and some general rules of thumb can be your guide.

CASE REPORT

A 70-year-old male was referred to the Centre for Eye Health for a macular workup. He was previously seen by an ophthalmologist several years ago. He reported a gradual deterioration of vision over the last few years which was not relieved with spectacles. His medical history included hypertension and hypercholesterolemia controlled by Karvezide and Crestor. His family's ocular health was unremarkable.

He was moderately myopic in both eyes and best corrected visual acuities were 6/15^{+2} OD and 6/9.5^{+2} OS with no improvement with pinhole. Amsler grid showed mild waviness four degrees temporal to fixation OD as well as superior and nasal to fixation OS. Mars contrast sensitivity was 1.40 units OD and 1.60 units OS (normal range for people over the age of 60 are 1.52 to 1.76 log units).

Colour fundus photographs (Figure 1A, B) showed irregular hypo and hyper-pigmentation in the central macula with yellow deposits OU. Optomap fundus autofluorescence (FAF) (Figure 1C, D) revealed asymmetric, mixed hyper and hypo autofluorescence of the macula OU with a linear pattern to the hyper-autofluorescence OS. Spectralis optical coherence tomography (OCT) (Figure 1E) across the right fovea showed subretinal hyper-reflective material corresponding to the intense hyper-AF, consistent with a vitelliform lesion. Adjacent to the lesion, there was complete retinal pigment epithelium (RPE) and outer retinal atrophy (cRORA) which exhibited profound hypo-AF. In the left eye, OCT (Figure 1F) showed disruption of the ellipsoid zone, RPE irregularities and pigment migration.

DIFFERENTIAL DIAGNOSIS

Acquired disorders

Vitelliform lesions can be present in a broad range of acquired conditions including AMD, pseudoxanthoma

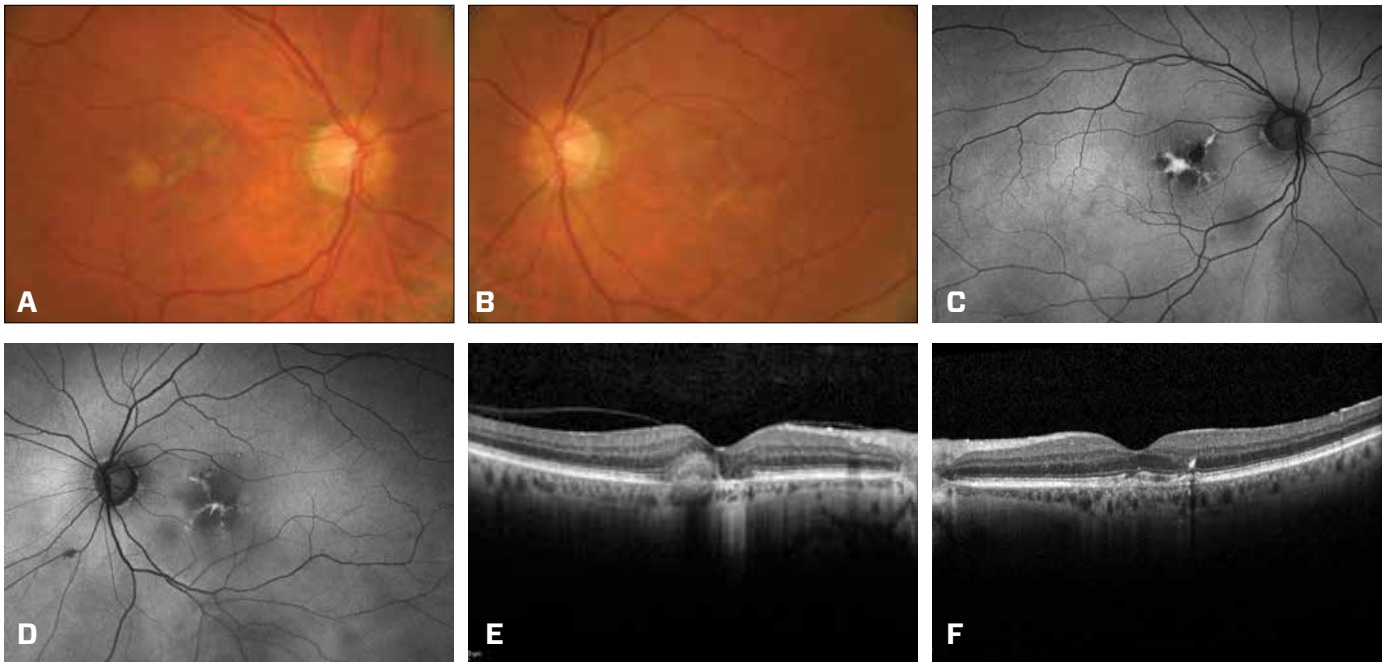


Figure 1. Imaging results from a 70-year-old male who was referred for a macular workup at the Centre for Eye Health. 1A, C and E: colour fundus photograph, fundus autofluorescence and OCT of the right eye; 1B, D and F: colour fundus photograph, fundus autofluorescence and OCT of the left eye.

elasticum (PXE) and angioid streaks, vitreomacular traction and central serous chorioretinopathy (CSCR).[2,3]

In this case, the patient's age, presence of pigmentary abnormalities at the central macula, a vitelliform lesion, and in particular the chorioretinal atrophy in the right eye could be consistent with a diagnosis of AMD. Critically however, there was an absence of drusen, the hallmark feature of AMD, resulting in this being excluded as a diagnosis.

Furthermore, the patient denied a medical history of PXE and there were no radiating lines emanating from the optic discs suggestive of angioid streaks. OCT did not show signs of vitreomacular traction. The patient's profile and history did not fit typical CSCR, and there were no signs of neurosensory detachment, subretinal fluid, pigment epithelial detachment, or abnormal choroidal features typically seen in the pachychoroid spectrum.[4]

Macular dystrophies

Vitelliform lesions can also occur in a number of macular dystrophies such as Best vitelliform macular dystrophy (BVMD) and adult-onset foveomacular vitelliform dystrophy (AFVD).

Best vitelliform macular dystrophy

BVMD (Best disease) is an autosomal dominant dystrophy characterised by a single, yellow 'egg-yolk' like lesion at the macula.[5] Best disease is caused by a mutation in the *BEST1* gene,[6,7] which encodes a transmembrane protein named bestrophin-1, located in the RPE. Mutation of this gene ultimately leads to accumulation of fluid and debris separating neurosensory retina and RPE, overload of lipofuscin in the RPE and secondary photoreceptor degeneration.[8]

BVMD typically starts in childhood, although it is usually not detected until later stages as the visual acuity remains good for many years.[9] The median age at the onset of the visual symptoms was 33 years (range: 2–78).[10] There is gradual decrease in vision with age, generally. One large case series showed on average, a VA less than 6/12 was reached by age 55 years, a VA of less than 6/19 was reached by age 66 years, and five per cent of patients progressed to legal blindness.[10]

BVMD presents over a wide clinical spectrum, represented by five stages: subclinical, vitelliform, pseudohypopyon, vitelliruptive and atrophic/cicatricial.[5] The original classification was based on fundus appearance alone and more recent studies with FAF and OCT have further characterised them[11-14] (Table 1). It should be noted that these stages do not always occur consecutively nor do they occur inevitably in all patients,[15] and choroidal neovascularisation (CNV) is a complication which can occur in virtually any stage.[14]

Diagnosis of BVMD is based on clinical findings and can be confirmed by an electro-oculogram (EOG). The EOG utilises the RPE's response to changing illumination to assess the function of the outer retina and RPE.[16] A light peak to dark trough ratio (previously known as Arden ratio) of 1.5 or lower is typically the threshold for diagnosis of BVMD.[14]

BVMD can be differentiated from AMD by early onset before 50, central vitelliform lesions without surrounding drusen; marked autofluorescence changes within the vitelliform lesion; and location of the lesion above the RPE; and a markedly abnormal EOG.[17]

Adult-onset foveomacular vitelliform dystrophy

AFVD was first described by Gass in 1974[18] as a macular disorder sharing phenotypical features with BVMD. Mutation in several genes have been identified to be associated with AFVD including *PRPH2*, *BEST1*, *IMPG1* and *IMPG2*,[3] although most cases are sporadic.[3] *PRPH2* encodes peripherin-2 which is located in the photoreceptors and has an important structural role in the photoreceptor outer segments.[19]

The onset of adult-onset foveomacular vitelliform dystrophy (AFVD) is typically after 40 years as opposed to Best disease which manifests in the first decade.[17] The size of the vitelliform lesion varies, from 1/3 to up to 1 disc-diameter.[14,18]

Continued page 24

Stages	Fundoscopy	FAF	OCT
Subclinical	Normal or subtle RPE alteration	Absent or only slight autofluorescence	Bilateral thicker and more reflective appearance of the interdigitation zone (Verhoeff's membrane) between the RPE and the ellipsoid zone (EZ)
Vitelliform	A well circumscribed, yellow yolk-like lesion of 0.5 to 2-disc-diameters centred in the macula	Intense hyper-AF	Subretinal hyper-reflective materials
Pseudohypopyon	Yellow material accumulates inferiorly	Well-circumscribed hyper-AF in the inferior macula	Subretinal hyper-reflectivity in the inferior macula
Vitelliruptive	Borders and yellow colour of the lesion become irregular reminiscent of a 'scrambled egg'	Typically no increased autofluorescence	Optically empty lesion between the RPE and the EZ with clumping of hyper-reflective materials on the posterior retinal surface
Atrophic/cicatricial	Macular atrophy and scarring	Atrophy shows hypo-AF. Fibrotic lesions show inhomogeneous mixed area of absolute hypo-AF and hyper-AF	Atrophy shows loss of outer retina and RPE. Fibrosis shows highly hyper-reflective thickening at the RPE level

Table 1. Imaging characteristics of five stages of BVMD

Differentiating MD from AMD

From page 23

Vitelliform lesions in AFVD can demonstrate features and stages, such as pseudohypopyon, vitelliruptive and atrophy, seen in Best disease.[14] Multiple vitelliform lesions have also been reported.[3] The natural course of the AFVD is thought to be slowly progressive, and vision loss occurs when atrophy and/or CNV develops. AFVD can be differentiated from Best disease by later age of onset as well as EOG, where the light rise on EOG is normal or only slightly abnormal in AFVD; it is virtually absent in Best disease.[17]

AFVD has been classified as one of the heterogeneous groups of progressive RPE alterations, collectively labelled pattern dystrophy, which are characterised by deposition of yellow-dark pigment involving the macula and posterior pole.[5] Based on the pattern of pigment distribution, pattern dystrophy can be divided into five groups: AFVD, butterfly pattern dystrophy (yellow deposits consists of three to five linear lines, resembling the rings of a butterfly), multifocal pattern dystrophy stimulating fundus flavimaculatus (irregular yellow flecks in the posterior pole and vascular arcade), reticular dystrophy of the RPE (clearly defined network of hyperpigmented lines that resemble a fishnet with knots) and fundus pulverulentus (punctiform mottling of the RPE).[5] Different types of pattern dystrophy can occur in two eyes of a patient and in different members of the same family carrying the identical mutation.[17] The common OCT signs of pattern dystrophy are hyper-reflectivity between the RPE/Bruch's complex and the outer retina, with some disruption in the ellipsoid zone.[20]

Diagnosis

The presence of vitelliform lesion in conjunction with the rest of the clinical picture is consistent with adult-onset foveomacular vitelliform dystrophy OD and butterfly pattern dystrophy OS, which both belong to the category of pattern dystrophy. The reduced visual acuity OD was at least partially attributed to the subfoveal atrophy.

Management

There is currently no treatment available to prevent the development of vitelliform lesions or delay the atrophic process. Photodynamic therapy was found to cause a significant decrease in vision in those with vitelliform lesions.[21] In the present case, following electronic review by the centre's consultant retinal specialist, the patient was given an Amsler grid to self-monitor at home and ongoing review with their previous ophthalmologist was recommended. Optometrists can also co-manage these cases through imaging such as OCT to monitor closely for exudative changes; OCT angiography for signs of CNV; and FAF to evaluate the vitelliform material and measure progression of macula atrophy. When AFVD is complicated by CNV, anti-vascular endothelial growth factor therapy may be used to manage the CNV with guarded visual outcomes.[3]

Summary

Differentiating macular dystrophy and AMD can be challenging, particularly for those with late onset and no distinct familial involvement. As highlighted in this case, some general rules of thumb can guide the clinician through the initial workup:

- Drusen are hallmark features of AMD.
- Absence of drusen can exclude AMD.

Drusen and pigmentary changes in AMD are predominately found at the central macula. If the deposits involve the entire posterior pole, vascular arcade and/or beyond, a dystrophy should be suspected.

FAF is one of the most valuable tools for diagnosing a dystrophy and the FAF presentations are often more excessive than fundoscopic signs. Lesions such as the flecks in Stargardt disease and vitelliform materials in BVMD are highly visible on FAF. ▲

1. Michaelides M, Hunt DM, Moore AT. The genetics of inherited macular dystrophies. *J Med Genet* 2003; 40: 641-650.
2. Freund KB, Laud K, Lima LH et al. Acquired Vitelliform Lesions: correlation of clinical findings and multiple imaging analyses. *Retina* 2011; 31: 13-25.
3. Chowers I, Tiosano L, Audo I et al. Adult-onset foveomacular vitelliform dystrophy: A fresh perspective. *Prog Retin Eye Res* 2015; 47: 64-85.
4. Cheung CMG, Lee WK, Koizumi H et al. Pachychoroid disease. *Eye* (Lond) 2019; 33: 14-33.
5. Agarwal A. Gass' *Atlas of Macular Diseases,* 5th Edition ed. Philadelphia: Elsevier; 2011.
6. Bitner H, Schatz P, Mizrahi-Meissonnier L et al. Frequency, genotype, and clinical spectrum of best vitelliform macular dystrophy: data from a national center in Denmark. *Am J Ophthalmol* 2012; 154: 403-412 e404.
7. Petrukhin K, Koisti MJ, Bakall B et al. Identification of the gene responsible for Best macular dystrophy. *Nat Genet* 1998; 19: 241-247.
8. Boon CJ, Klevering BJ, Leroy BP et al. The spectrum of ocular phenotypes caused by mutations in the BEST1 gene. *Prog Retin Eye Res* 2009; 28: 187-205.
9. Budiene B, Liutkeviciene R, Zaliuniene D. Best vitelliform macular dystrophy: literature review. *Cent Eur J Med* 2014; 9: 784-795.
10. Booij JC, Boon CJ, van Schooneveld MJ et al. Course of visual decline in relation to the Best1 genotype in vitelliform macular dystrophy. *Ophthalmology* 2010; 117: 1415-1422.
11. Querques G, Zerbib J, Georges A et al. Multimodal analysis of the progression of Best vitelliform macular dystrophy. *Mol Vis* 2014; 20: 575-592.
12. Pichi F, Abboud EB, Ghazi NG et al. Fundus autofluorescence imaging in hereditary retinal diseases. *Acta Ophthalmol* 2018; 96: e549-e561.
13. Querques G, Zerbib J, Santacroce R et al. The spectrum of subclinical Best vitelliform macular dystrophy in subjects with mutations in BEST1 gene. *Invest Ophthalmol Vis Sci* 2011; 52: 4678-4684.
14. Querques G, Souied EH. Macular dystrophies.London: Springer; 2016.
15. Marmorstein AD, Cross HE, Peachey NS. Functional roles of bestrophins in ocular epithelia. *Prog Retin Eye Res* 2009; 28: 206-226.
16. Constable PA, Bach M, Frishman LJ et al. International Society for Clinical Electrophysiology of V. ISCEV Standard for clinical electro-oculography (2017 update). *Doc Ophthalmol* 2017; 134: 1-9.
17. Saksens NT, Fleckenstein M, Schmitz-Valckenberg S et al. Macular dystrophies mimicking age-related macular degeneration. *Prog Retin Eye Res* 2014; 39: 23-57.
18. Gass JD. A clinicopathologic study of a peculiar foveomacular dystrophy. *Trans Am Ophthalmol Soc* 1974; 72: 139-156.
19. Boon CJ, den Hollander AI, Hoyng CB et al. The spectrum of retinal dystrophies caused by mutations in the peripherin/RDS gene. *Prog Retin Eye Res* 2008; 27: 213-235.
20. Hannan SR, de Salvo G, Stinghe A et al. Common spectral domain OCT and electrophysiological findings in different pattern dystrophies. *Br J Ophthalmol* 2013; 97: 605-610.
21. Ergun E, Costa D, Slakter J et al. Photodynamic therapy and vitelliform lesions. *Retina* 2004; 24: 399-406.

The author would like to thank Angelica Ly and Michael Yapp for reviewing and input into this article.

OCT-A and delayed-onset traumatic macular oedema

Joe Wang
BOptom
University of Melbourne Eyecare

Jessie Tan
OD BBiomed LMusA AmusA*

Dr Kwang Meng Cham
PhD BOptom GCertUniTeach PGCertOcTher*

*Department of Optometry and Vision Sciences, The University of Melbourne

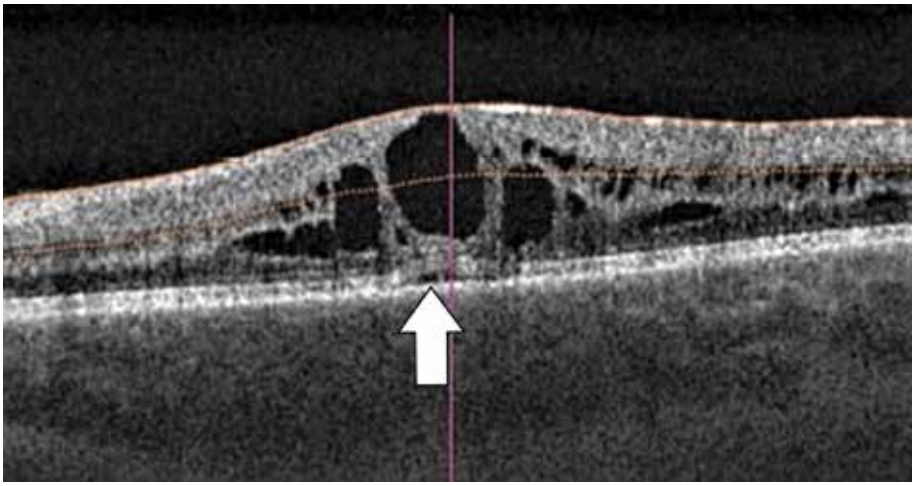


Figure 1. Spectral domain optical coherence tomography of the macula. The white arrow indicates cystoid macula oedema with subretinal fluid.

In this case, we discuss the characteristics and results of optical coherence tomography angiography (OCT-A) in a patient with cystoid macula oedema with subretinal fluid, three years after an episode of commotio retinae.

CASE REPORT

A 32-year-old Caucasian woman presented to the University of Melbourne Eyecare Clinic in 2015 following a champagne cork injury to the left eye.[1] Her visual acuity in the left eye was 6/38, which corrected to 6/12 with a low myopic correction. Trace anterior chamber reaction, mild vitreous haemorrhage and commotio retinae were noted. Optical coherence tomography (OCT) examination revealed a small partial macular hole with ellipsoid zone disruption. These changes resolved uneventfully four months later under ophthalmology observation.

In 2018, the patient presented with painless and gradually worsening vision in the left eye. Her visual acuity was 6/15 and a moderate hyperopic shift (approximately -1.75 DS in 2015 to +1.25 DS in 2018) was observed. The anterior segment was clear and quiet, and significant cystoid macular oedema with subretinal fluid was observed at the left macula (Figure 1). OCT-A (OCT, Topcon DRI Triton OCT, Topcon Corporation, Tokyo, Japan)[2] showed irregularity of the foveal avascular zone (Figure 2A), along with disruption of inner retinal blood flow at the level of the superficial capillary layer (Figure 2B). No obvious involvement of the deep capillary layer (Figure 2C) or choriocapillaris (Figure 2D) were observed. The patient was referred to her previous ophthalmologist and was subsequently treated with topical prednisolone acetate 1%. The macular

Continued page 26

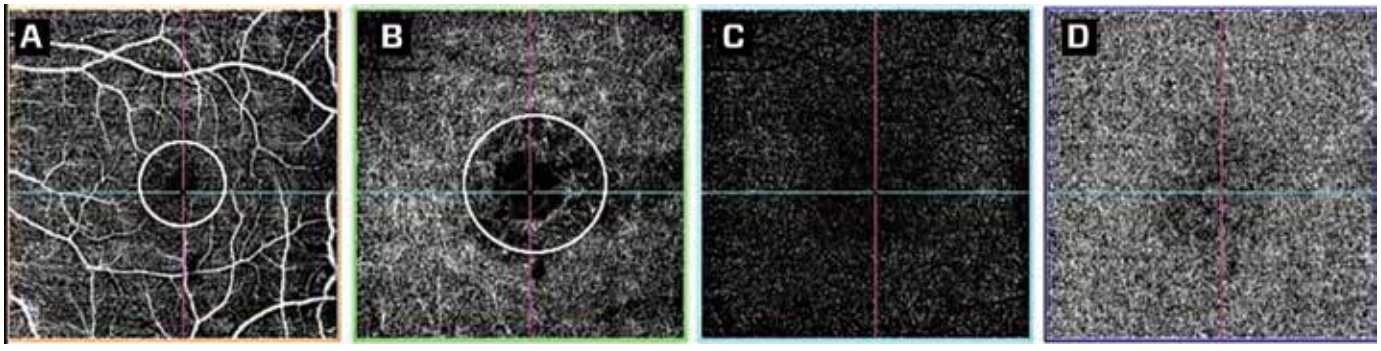


Figure 2. Topcon OCT-A analysis identifying areas of disruption to blood flow (white circle) in the A: foveal avascular zone and B: superficial capillary layer. C: The deep capillary plexus and D: choriocapillaris does not appear to show involvement.

OCT-A

From page 25

oedema and subretinal fluid eventually resolved and her left eye remained at 6/12 without any correction.

DISCUSSION

Hyperopic shift is a commonly reported finding in central serous chorioretinopathy and cystoid macula oedema, possibly due to an anterior shift of retinal plane with oedema.[3,4]

In the absence of active uveitis and as the patient did not have any other significant systemic or medical history (taking any long-term medications, vitamins or supplements), we postulated that her macular abnormalities had arisen from the initial traumatic event three years prior. Intraocular and posterior segment complications of blunt trauma such as cataract, commotio retinae, retinodialysis and optic neuropathy have been well documented in the literature.[5-7] To the best of our knowledge, there have been no reports of delayed-onset traumatic macular oedema.

The patient initially presented with a small partial macular hole in 2015, with some ellipsoid zone disruption observed at the time. Although 'cystic spaces' have been described as a hallmark feature of vitreomacular traction[8] in OCT, we are unaware of any association between macular holes and cystoid macular oedema. Future reports of OCT-A findings in cystoid macular oedema and vitreomacular traction will hopefully help to differentiate more esoteric macular presentations. ▲

1. Cham KM, Di Pasquale DN, Jaworski A. A case of commotio retinae following champagne cork injury. *Clin Exp Optom.* 2018; 101: 140-142.
2. Topcon-medical.eu [Internet] The Netherlands: DRI OCT Triton series swept source optical coherence tomography; [cited 2018 May 30]. Available from http://www.topcon-medical.eu/files/EU_Downloads/Products/DRI_OCT_Triton/DRI_OCT_Triton_Brochure_EN.pdf
3. Wang M, Munch IC, Hasler PW et al. Central Serous Chorioretinopathy. *Acta Ophthalmol* 2008; 86: 126-145.
4. Tasman W, Jaeger E. *The Wills Eye Hospital Atlas of Clinical Ophthalmology*, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2001. p88.
5. Archer D, Galloway N. Champagne-cork injury to the eye. *The Lancet* 1967; 290: 487-489.
6. Johnston P B. Traumatic retinal detachment. *Br J Ophthalmol* 1991; 75: 18-21.
7. Samardzic K, Samardzic J, Janjetovic A et al. Traumatic optic neuropathy – to treat or to observe? *Acta Inform Med* 2012; 20:131-132.
8. Trichonas G, Kaiser PK. Optical coherence tomography imaging of macular edema. *Br J Ophthalmol* 2014; 98: 24-29.

Low vision referral

Meeting the demand for support and services

Nabill Jacob
DOBA BAppSc-Orthoptics (Syd)
MCommHlth (Syd) MIP (ACHSM)

Clinical Relationship Manager
Vision Australia

As the clinical face of eye health, every day optometrists review and help different people get the most out of their vision and often refer them on to other specialists in the circle of care. Most of the time this is routine. However, sometimes a simple conversation with your patient can dramatically alter the routine.

Some patients with low vision are reluctant to admit they are struggling with the most basic tasks, such as cooking, grooming, shopping or socialising.

CASE REPORT

Mr B was a 70-year-old retired sailor, who lived alone as his wife, who has dementia, lives in a nursing home. He has one son who lives in a different state. Mr B reported slightly elevated cholesterol for which he takes one Crestor PO tablet daily, but was otherwise in good health.

Mr B was diagnosed with age-related macular degeneration (AMD) 10 years ago. He has a shared care arrangement between his optometrist and ophthalmologist.

Upon examination by his optometrist, Mr B's prescription was R -0.50 (6/18 part), L -1.00 (6/24 part). His intraocular pressure (IOP) was R 16 mmHg and L 18 mmHg.

During examination, Mr B seemed a little apprehensive and admitted that he was having a few problems which had been getting worse, namely:

- Reading the newspaper
- Watching TV
- Making a simple meal
- Shaving
- Shopping and socialising
- He was feeling isolated and depressed as he couldn't go out on a boat or to the boat club, which are his passions
- Playing lawn bowls
- Mobility (He is scared of bumping into things or falling)

Mr B told his optometrist that he was afraid of further losing his sight. He explained he wanted to stay in his own home and stay independent but was also frightened he'd end up in a nursing home.

By thinking of the patient's case from a holistic perspective, the consulting optometrist's primary concerns for Mr B were:

- Assessing falls risk/mobility issues. The risk of falls doubles with visual impairment; the risk of hip fractures increases four to eight times.[1]
- Identifying mental health issues – there is a three-fold increase in the risk for depression.[1]

Action to take

A referral to Vision Australia was made, offering Mr B reassurance that the organisation could help and potentially address his concerns with their full suite of low vision services available.

Vision Australia assessment

At his initial assessment with Vision Australia, Mr B was asked what he would like to be able to achieve and to list some of the tasks he'd like to be able to do.

The Vision Australia orthoptist performed a functional vision assessment to help clarify which services and aids may be of most benefit and to ensure no significant changes in vision had occurred since the referring optometrist's last examination. (Should any changes or concerns be noted, Mr B would be immediately referred back to the optometrist.)

Subsequently, Mr B identified the following goals he would like to achieve:

- To have a guide dog or white cane to be able to go out into the community and get on his – or any – boat again
- To be able to keep reading novels and newspapers
- To feel safe at home and to continue to cook and take care of himself
- To see the price, ingredients and use-by date of items at the supermarket
- To socialise at his local boat club and learn new skills, and not to feel isolated and useless

Post-referral to Vision Australia

A few months after the optometrist's referral to Vision Australia for assessment and intervention, the following outcomes were successfully achieved:

- A Seeing Eye Dog was allocated and orientation and mobility training with the use of a white cane was provided
- Mr B was now able to walk to shops and the boat club safely as well as use public transport safely and confidently

Continued page 28

Low vision

From page 27

- He could now visit his son in Perth with confidence while also knowing that Vision Australia has an office there should he need help

He joined the Vision Australia Library and immediately had access to more than 45,000 publications in audio book and large print formats. He was also introduced to Vision Australia Radio.

An occupational therapist assessed his home and re-arranged his kitchen and bathroom as well as organised slight home modifications. He was introduced to aids and equipment to suit people with low vision. Mr B was now able to successfully self-manage, and has even been able to sleep a few nights in his moored sail boat.

The orthoptist prescribed and instructed him on the use of a hand held as well as portable electronic magnifier. His home lighting was also adjusted.

Mr B was introduced to Vision Australia's woodwork and day programs to ensure socialisation and to introduce Mr B to other people in the community with low vision.

Discussion

It has been estimated that there are over 575,000 people who are currently blind or have vision loss living in Australia, and that number is projected to grow to over 800,000 by 2020.[2]

The expanding role of optometry means patient welfare sometimes extends beyond clinical and optical care, requiring a holistic view of each patient and their individual needs.

The comprehensive services offered by Vision Australia aim to meet the needs of low vision and blind patients, and are also a natural extension of the collaborative and continuum of care model practiced by optometrists.

When to refer:

Upon diagnosis of a permanent, non-correctable or progressive eye condition. Referring at < 6/12 BCVA or < 30 degrees of visual field (both eyes open) is strongly recommended.

- When vision loss starts to impact daily activities such as getting around safely or reading
- When glasses no longer correct vision or when they need support adjusting to vision loss
- For children, early intervention is key

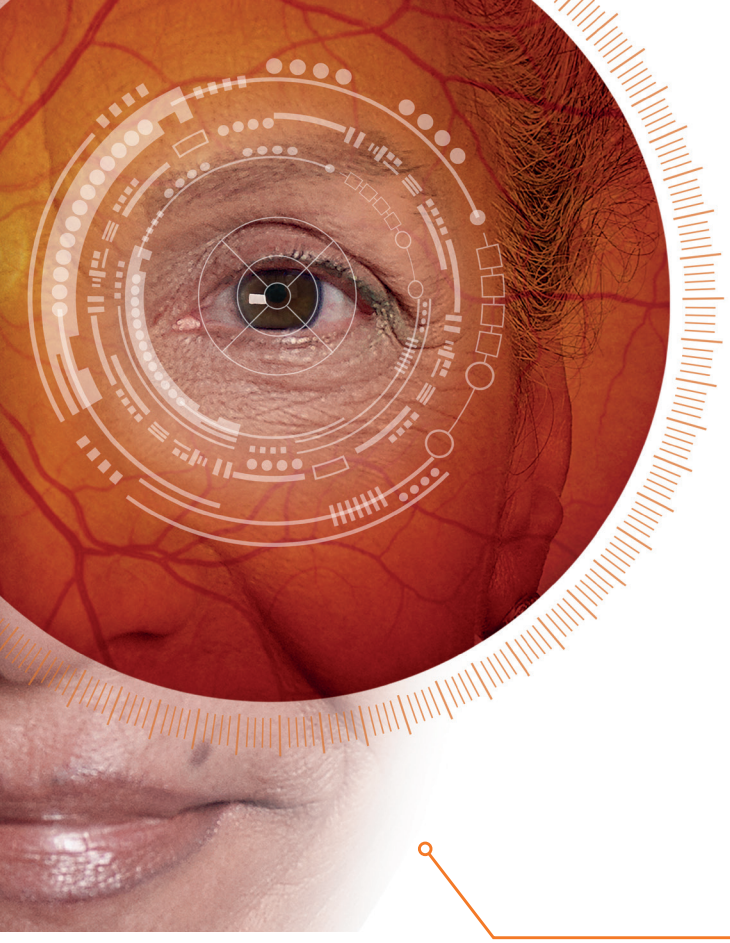
There are no barriers to referral; to refer a patient to Vision Australia nationally, use the 'Refer my patient here' tab on the Vision Australia homepage. Full support in securing NDIS and My Aged Care funding for services and equipment/aids is available through Vision Australia. Additionally, mobile assessment services to home, nursing home, TAFE/university/school/pre-school and workplace are also available.

As experience with Mr B shows, low vision services are vital. They reduce the barriers that stand in the way of those who are still capable of experiencing a full and active life, and they fundamentally improve the quality of these lives through training, support and a range of adaptive technologies and tools. ▲

1. Access Economics Pty Ltd. Clear Insight: The Economic Impact and Cost of Vision Loss in Australia in 2004 [Internet]. Melbourne: Centre for Eye Research Australia; 2004 [cited 2019 March 22]. Available from: https://www.cera.org.au/wp-content/uploads/2013/12/CERA_clearinsight_overview.pdf
2. Access Economics Pty Ltd. Clear Focus: The Economic Impact of Vision Loss in Australia in 2009 [Internet]. Melbourne: Vision 2020 Australia; 2010 [cited 2019 March 15]. Available from: http://www.vision2020australia.org.au/uploads/resource/85/v2020aus_report_clear_focus_overview_jun10.pdf

Vision Australia
Blindness. Low Vision. Opportunity.

Contact Vision Australia for more information: www.visionaustralia.org or 1300 84 74 66.



PRECISE†

DESIGNED FOR THE EYE[1–5]

†Selectively targets VEGF-A isoforms.[1] Non-clinical description of mode of action. Not intended to imply a clinical benefit.

Selectively target VEGF-A with Lucentis® – specifically developed for ocular use.[1,3]

nAMD | DME | BRVO | CRVO | other CNV (NEW) | PM (NEW)

BRVO, branch retinal vein occlusion; CNV, choroidal neovascularisation; CRVO, central retinal vein occlusion; DME, diabetic macular oedema; nAMD, neovascular age-related macular degeneration; PM, pathological myopia; VEGF, vascular endothelial growth factor.

PBS Information: Authority required for the treatment of wet AMD, DME, BRVO, CRVO, PM or for the treatment of CNV secondary to causes other than wet AMD and PM. Refer to PBS Schedule for full Authority information.

Before prescribing, please review full Product Information available from www.novartis.com.au/products/healthcare-professionals

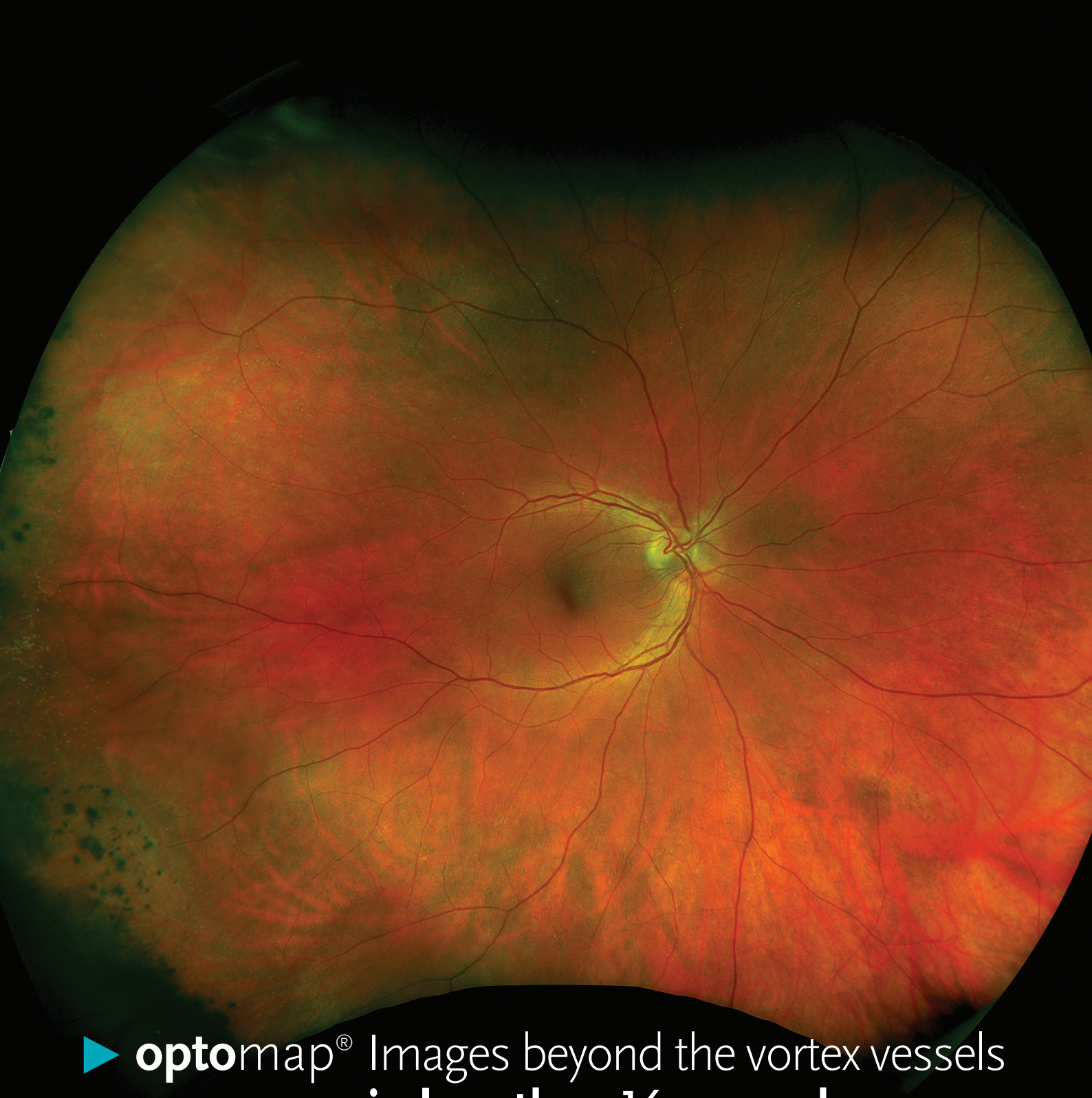
Indication: Treatment of neovascular (wet) age-related macular degeneration (AMD). *The treatment of visual impairment due to choroidal neovascularisation.* Treatment of visual impairment due to choroidal neovascularisation (CNV) secondary to pathologic myopia (PM). Treatment of visual impairment due to diabetic macular oedema (DME). Treatment of visual impairment due to macular oedema secondary to retinal vein occlusion (RVO). **Dosage and administration:** Complex dosage and administration – see full PI before prescribing. **Contraindications:** Hypersensitivity to product components, active or suspected ocular or periocular infections active intraocular inflammation. **Precautions:** Intravitreal injections have been associated with endophthalmitis, intraocular inflammation, rhegmatogenous retinal detachment, retinal tear, iatrogenic traumatic cataract and increased intraocular pressure. Proper aseptic injection techniques must be used. Review patients during the week following injection to permit early treatment if an infection occurs. Transient increases in intraocular pressure (IOP) have been seen within 60 minutes of injection of Lucentis. Sustained IOP increases have also been reported. Intraocular pressure and perfusion of the optic nerve head must be monitored and managed appropriately. Patients should be reviewed for IOP rise preinjection and 60 minutes post-injection. *The dose should be withheld and treatment should not be resumed earlier than the next scheduled treatment in the event of an intraocular pressure of ≥30 mmHg.* Bilateral use: limited data do not suggest an increased risk of systemic adverse events compared with unilateral treatment. There is a potential risk of arterial thromboembolic events following intravitreal use of VEGF inhibitors. A numerically higher stroke rate was observed in patients treated with ranibizumab 0.5mg compared to ranibizumab 0.3mg or control, however, the differences were not statistically significant. Patients with known risk factors for stroke, including history of prior stroke or transient ischaemic attack, should be carefully evaluated by their physicians as to whether Lucentis treatment is appropriate and the benefit outweighs the potential risk. As with all therapeutic proteins, there is a potential for immunogenicity with Lucentis. Lucentis has not been studied in patients with concurrent eye conditions such as retinal detachment or macular hole. No formal interaction studies have been performed. Limited experience with treatment of patients with prior episodes of RVO and of patients with ischemic branch RVO (BRVO) and central RVO (CRVO). In patients with RVO presenting with clinical signs of irreversible ischemic visual function loss, treatment is not recommended. Should be used with caution in women of child bearing potential in general, and during pregnancy in particular. For women who wish to become pregnant and have been treated with ranibizumab, it is recommended to wait at least 3 months after the last dose of ranibizumab before conceiving a child; use of effective contraception is recommended for women of childbearing potential; breastfeeding is not recommended. Lucentis is not recommended for use in children and adolescents Patients who experience temporary visual disturbances following treatment must not drive or use machines until these subside. **Adverse effects:** Very common (≥10%): Intraocular inflammation, vitritis, vitreous detachment, retinal haemorrhage, visual disturbance, eye pain, vitreous floaters, conjunctival haemorrhage, eye irritation, foreign body sensation in eyes, lacrimation increased, blepharitis, dry eye, ocular hyperaemia, eye pruritus, intraocular pressure increased, nasopharyngitis, headache, arthralgia. Common (1 to 10%): Retinal degeneration, retinal disorder, retinal detachment, retinal tear, detachment of the retinal pigment epithelium, retinal pigment epithelium tear, visual acuity reduced, vitreous haemorrhage, vitreous disorder, uveitis, iritis, iridocyclitis, cataract, cataract subcapsular, posterior capsule opacification, punctuate keratitis, corneal abrasion, anterior chamber flare, vision blurred, injection site haemorrhage, eye haemorrhage, conjunctivitis, conjunctivitis allergic, eye discharge, photopsia, photophobia, ocular discomfort, eyelid edema, eyelid pain, conjunctival hyperaemia, stroke, influenza, urinary tract infection*, anaemia, anxiety, cough, nausea, allergic reactions (rash, pruritus, urticaria, erythema). Uncommon (0.1 to 1%): Blindness, endophthalmitis, hypopyon, hyphaema, keratopathy, iris adhesions, corneal deposits, corneal oedema, corneal striae, injection site pain, injection site irritation, abnormal sensation in eye, eyelid irritation. Serious adverse events related to intravitreal injections include endophthalmitis, rhegmatogenous retinal detachment, retinal tear and iatrogenic traumatic cataract. *Observed only in the DME population. Based on TGA approved Product Information dated 18 December 2018 *(luc181218m). *Please note changes to Product Information in italics.*

References: 1. Lucentis® Product Information. December 2018. **2.** Ferrara N, Adamis AP. Nat Rev Drug Discov 2016; 15 (6): 385–403. **3.** Steinbrook R. N Engl J Med 2006; 355: 1409–1412. **4.** Mordenti J et al. Toxicol Pathol 1999; 27 (5): 536–544. **5.** Gaudreault J et al. Retina 2007; 27 (9): 1260–1266.

Novartis Pharmaceuticals Australia Pty Limited. ABN 18 004 244 160.54 Waterloo Road, Macquarie Park, NSW 2113. ®Registered Trademark. AU-7316. McCann Health NOLU15419M. April 2019.

NOVARTIS

LUCENTIS® RANIBIZUMAB



optomap® Images beyond the vortex vessels in less than ½ second

the ONLY ultra-widefield, single-capture *colour* image

Find out more at www.optos.com

Contact us to put optomap in your practice, call 08 8444 6500 or email auinfo@optos.com

Nikon

optos®