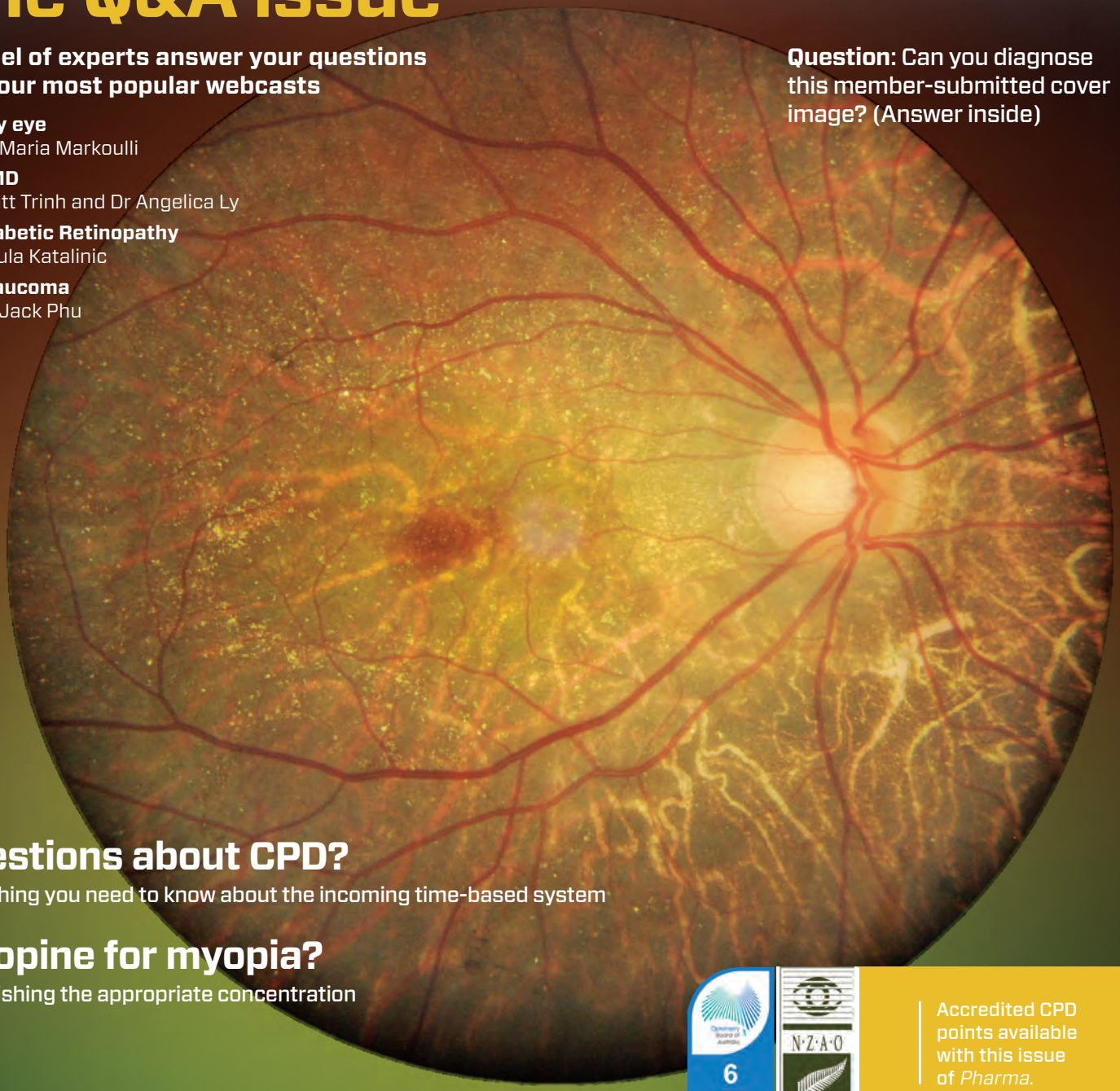
pharma
September 2020
Optometry AUSTRALIA
ADVANCING OPTOMETRY

The Q&A issue

A panel of experts answer your questions from our most popular webcasts

- **Dry eye**
  Dr Maria Markoulli
- **AMD**
  Matt Trinh and Dr Angelica Ly
- **Diabetic Retinopathy**
  Paula Katalinic
- **Glaucoma**
  Dr Jack Phu

Question: Can you diagnose this member-submitted cover image? (Answer inside)



Questions about CPD?

Everything you need to know about the incoming time-based system

Atropine for myopia?

Establishing the appropriate concentration

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3T

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September 2020 Q&A

From the Editors

It would be hard to think of a time more appropriate to publish a Q&A issue. In so many facets of life right now, there are a lot more Q's than A's. Against this backdrop, it has been gratifying to provide answers to at least some of your questions.

Working with the Institute of Excellence, we researched some of the most frequently-asked member queries from their most popular webcasts and invited the original presenters to respond. Unanimously, our contributors report that the process has helped them expand on their original talks and round out their discussions. Their answers, on the topics of dry eye, AMD, diabetic retinopathy and glaucoma are presented in this issue alongside articles on other topics, inspired by more general, but no less relevant, questions.

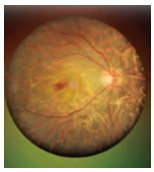
The changing CPD requirements have inspired quite a few questions as well. In response, Optometry Australia's Member Services team has provided a helpful Q&A explaining the shift from a points-based system to a time-based system.

Finally, there are questions about *Pharma*. After more than 10 years, Optometry Australia has made the decision to cease its production and launch a new publication to align with the sector's change to time-based CPD requirements.

Debuting in December, *Optometry Connection* reflects Optometry Australia's strategic imperative to continue to evolve and adapt within a fast-changing world. We are confident that *Optometry Connection* will inspire and assist you in your day-to-day patient and clinical care decision making.

As we farewell *Pharma*, we'd like to thank the long list of contributors who, for over a decade, have helped to establish *Pharma* as a veritable anthology of clinically-relevant case studies and articles on the subject of optometric care. We'd also like to thank you, our loyal readers, for the support you have shown and the guidance you have offered.

Ultimately, the key question of 2020 is: 'what's next?' And the answer for our readers (and, we hope, for all of us) is: 'something better.'



COVER
Answer: Bietti crystalline dystrophy. Pauline Xu submitted this image of the rare condition that features crystalline deposits in the retina. Associated with atrophy of the retinal pigment epithelium, pigment clumping and choroidal sclerosis, Bietti crystalline dystrophy can result in progressive vision loss.

To submit your photo for the cover, email publications@optometry.org.au

This issue of *Pharma* offers 6 (3T) CPD points.

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What lies beneath

The role of systemic conditions in normal tension glaucoma

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This original case report was submitted by Optometry Australia member Steven Lam in response to our ongoing call for member papers.

Normal tension glaucoma (NTG) is a sub-category of glaucoma, a term used to describe a group of diseases that causes optic neuropathy. Glaucoma is characterised by the progressive loss of the retinal nerve fibre layer (RNFL), ganglion cell layer (GCL) and functional visual field.[1] Although the exact cause is unknown, common risk factors identified are people of Japanese ethnicity, an increase in age, raised intra-ocular pressure (IOP), decreased diastolic ocular perfusion pressure (DOPP) and obstructive sleep apnoea (OSA).[2,3] Glaucoma can be classified as 'open angle' which includes primary open angle glaucoma (POAG) where IOP is > 21mmHg or NTG with IOP < 21mmHg.[3,4] Other classifications include narrow angle and secondary glaucoma.[5]

Depending on the severity, patient symptoms can range from being asymptomatic to severe paracentral vision loss. Visual acuity (VA) can also decrease if macula fibres are affected.[4] The rate of peripheral vision loss in untreated NTG can vary from -0.2 decibel (dB) to > -2.0dB per year on a total deviation plot of a standard automated perimeter (SAP).[6] Clinically, NTG may present with optic disc haemorrhages, optic nerve rim thinning accompanied by peripapillary atrophy and IOP less than 21 mmHg. Asymmetry of IOP can also be seen with the more severely affected eye usually 1-2 mmHg greater than the other eye.[4] Medical imaging with optical coherence tomography (OCT) can show RNFL thinning at the optic nerve (ON) and GCL thinning at the macula region. Visual field (VF) examinations usually show arcuate defects respecting the horizontal mid line and will correspond to the region of RNFL/GCL loss.[4,7]

Management of NTG is targeted at lowering IOP to slow the progression of VF and RNFL/GCL loss. Current therapies include topical medication, selective laser trabeculoplasty (SLT), stent insertions and trabeculectomy. The decision to start management will depend on the rate of glaucoma progression and potential impact on the patient's quality of life.[4]

CASE REPORT

Mrs X, a 50-year-old female with a family history of glaucoma, presented for examination reporting gradual blurred vision over the last year.

Her best corrected distance VA was 6/6 OU and near vision was N5. Anterior chamber angle assessment revealed an open angle and no signs of secondary glaucoma. Assessment of the optic nerve through a Volk super 66 lens revealed large optic discs of R 1.9 mm and L 1.8 mm. The neural retinal rim (NRR) of the RE showed superior nerve excavation and an inferior notch (Figure 1) was found in the LE. Both NRRs had relative concentric thinning with a cup-to-disc ratio of R 0.7 and L 0.8. The patient's IOP was 21 mmHg bilaterally measured with a Perkins tonometer at 12:33pm and central corneal thickness was R 505 µm and L 499 µm.

Medical imaging results with an OCT confirmed that there was RNFL thinning of the superior rim in the RE and inferior rim in the LE. Ganglion cell complex analysis showed bilateral inferior thinning. Further investigation with a Zeiss Humphrey Field Analyzer 3 (24-2) showed a normal RE and central defects in the LE. Analysis with a 10-2 (Figure 2) revealed significant superior arcuate defects

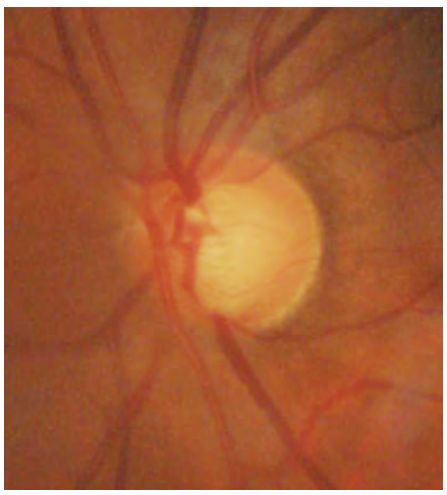

Figure 1. Inferior notch seen on left optic nerve on initial presentation

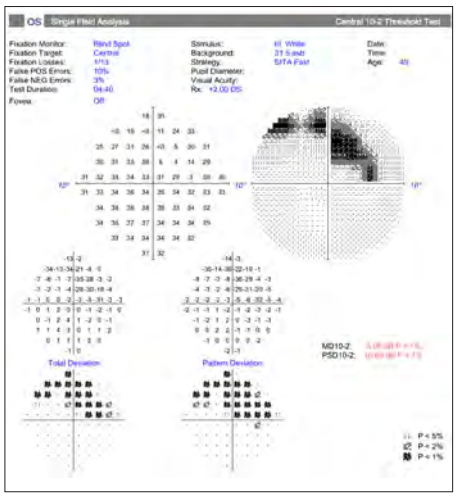

Figure 2. HVFA3 (10-2) showing a superior arcuate defect on initial presentation

in the LE only. These results were successfully repeated one week later.

Mrs X was diagnosed with NTG and a treatment plan was developed to reduce her IOP by 30 per cent. After a diurnal IOP was established at 21 mmHg with a maximum of 22 mmHg, a target pressure of 15 mmHg was set. She was prescribed Xalatan eye drops once a night in the LE, the RE was used as a control. After three weeks, target pressure was achieved and bilateral treatment initiated. She was scheduled to be reviewed in three months and was also referred to the local ophthalmologist for confirmation of the treatment plan and consideration for SLT. The ophthalmologist confirmed the plan and Mrs X was happy to continue Xalatan rather than SLT.

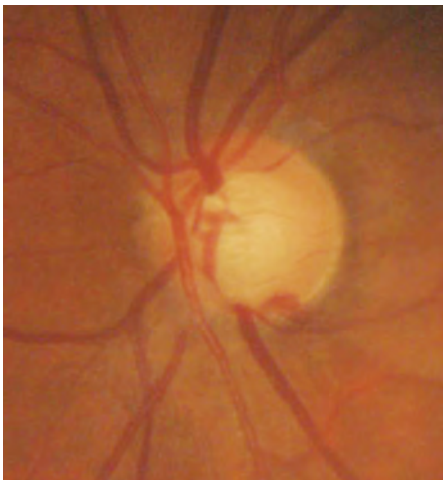

Figure 3. Left optic nerve showing an inferior Drance haemorrhage seen after treatment initiated

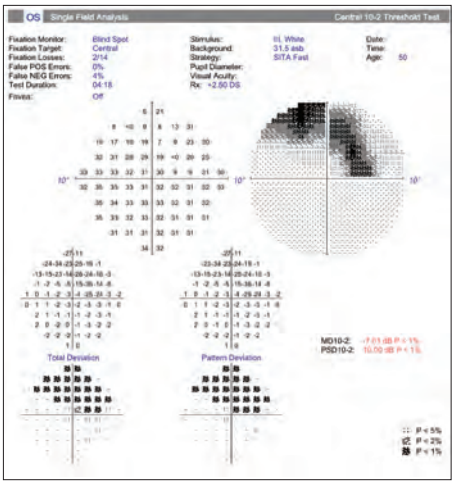

Figure 4. HVFA3 (10-2) showing progression of VF defect in the LE three months after initial treatment

At her three month review, an inferior Drance haemorrhage (Figure 3) was seen on the L ON. VF (10-2) results revealed significant progression of the superior defects of the LE (Figure 4). A new target pressure of < 12 mmHg was set and timolol was added to her treatment with a three-week review scheduled. During this review, Mrs X also stated that she was tired, felt dizzy when sitting up and normally found it hard to sleep as she would wake up in the middle of the night. Our optometrist then referred her to her general practitioner (GP) for investigations into low blood pressure (BP) and OSA.

Mrs X's BP was confirmed to be low at 90/50 mmHg and she was diagnosed with moderate OSA. She was then referred to her pharmacist for a continuous positive airway pressure machine (CPAP) and a dentist to create a mandibular advancement device (MAD) for treatment of OSA.

Her IOP at the three-week review was R 10 mmHg L 11 mmHg, reaching target pressures, and no progression was seen three months later.

Discussion

The interactions between BP and IOP have been shown to influence glaucoma as it can affect DOPP (Diastolic BP – IOP).[3] Low BP can occur spontaneously or secondary to anti-hypertensive medication. Dips in BP have been shown to occur more commonly at night and patients can be classified as normal-dippers (< 10 per cent decline) or extreme dippers (> 20 per cent decline).[2,3] Diurnal variation in IOP is influenced by body position, with IOP likely to rise nocturnally when patients are sleeping in the supine position.[8] Low BP and increased IOP can decrease DOPP resulting in ischemic damage to the ON. A DOPP of < 55 mmHg has shown to increase the risk of glaucoma by 3.2 times and in regards to Mrs X, her DOPP before treatment was 30 mmHg (50 mmHg-20 mmHg).[8]

OSA is a significant risk factor in developing glaucoma. It is characterised by repetitive episodes of complete or partial obstruction to the upper airway during sleep leading to apnoea, hypopnea and hypoxia. This causes a reduction in oxygenation to the ON which can eventually lead to glaucoma.[7,9,10] Clinical symptoms include loud snoring, nocturnal gasping, lack of energy, reduced concentration, memory impairment, dry throat upon waking and morning headaches. Systemic implications of OSA include impeded neurocognitive behaviour and cardiovascular disease. First line therapy is usually with a CPAP machine which has shown positive effects on glaucoma by preventing a drop in arterial oxygen.[7,10] In regards to Mrs X, her symptoms matched that of OSA and her diagnosis was confirmed by a sleep specialist. ▲

Conclusion

Mrs X's risk for glaucoma was increased by her low DOPP and OSA. Although there has been some evidence that managing OSA may slow glaucoma progression, there is currently no research to show that CPAP therapy alone is enough to consider withdrawal of glaucoma medication. This case highlights the importance of collaborative health care and for optometrists to consider the patient's systemic status in NTG. ▲

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Dry eye disease

Causes, symptoms and treatment options

Q A

From the 'Taking the dryness out of dry eye' webcast, available on the Institute of Excellence.

Maria Markoulli
PhD MOptom GradCertOcTher
FBCLA FAAO

Department of Optometry and Vision Science, UNSW

Q: Can you talk about the osmolarity test unit and its predictability/ specificity to dry eye disease (DED), as well as the cost per test?

A: Osmolarity plays a key role in the pathophysiology of dry eye disease and forms part of the 2017 Tear Film and Ocular Surface Dry Eye Workshop II (TFOS DEWS II) diagnostic criteria. According to TFOS DEWS II, people with dry eye disease show more variability in their osmolarity measurements than their non-dry eye counterparts, and this is considered diagnostic.

The current guidelines include a difference of ≥ 8 mOsm/L between eyes, or a measurement of ≥ 308 mOsm/L for mild to moderate dry eye and ≥ 316 mOsm/L for moderate to severe dry eye.[1]

One challenge with the use of osmolarity has been the reported variability, for example, in people with Sjögren Syndrome and blepharitis, potentially complicating clinical interpretation.[2] In a group of healthy controls, a clinically-relevant difference of 34 mOsm/L was found.[2] What this means is: when a single tear osmolarity measurement is taken, such as before and after treatment, the measurement error and the variability between visits needs to be taken into account before considering the change with treatment to be clinically-relevant. This variability also means that a single measurement is not enough to distinguish between those with and those without dry eye. This large variability could explain the reported lack of association between tear osmolarity and clinical signs and symptoms.[3]

There are currently two devices on the market for the measurement of osmolarity: TearLab (TearLab Cooperation, California, US) and the iPen (iMedPharma, Quebec City, Canada). The TearLab equipment can be purchased outright or leased for a more affordable option. The iPen is a portable device and can also be purchased. Clinicians can contact the companies directly for a quote.

Q: Does chemotherapy or radiation therapy cause DED?

A: The antineoplastic mechanisms of chemotherapy can lead to undesirable systemic and ocular side effects resulting from cytotoxicity, inflammation and neurotoxicity.[4]

The ocular surface is particularly susceptible to toxicity with reported conditions including meibomian gland dysfunction, epiphora, dry eye, conjunctivitis, keratitis and ocular discomfort.[4] An important factor to also consider is the interaction of ocular therapeutics with concurrent anticancer drugs. For example, oral dexamethasone can potentially decrease the concentration of anti-cancer drugs, as can certain antibiotics such as clarithromycin, and oral antifungals such as fluconazole.[4]

There have also been reports about radiotherapy impacting the ocular

Working in tandem with the Institute of Excellence, we have collated some of the most frequently-asked and relevant questions submitted by members on dry eye, AMD, diabetic retinopathy and glaucoma. The guest speakers were invited to revisit their popular presentations and provide their answers in this issue of *Pharma* magazine.

A comprehensive array of online CPD offerings (including webcasts, online courses, *Pharma* and *Clinical & Experimental Optometry*) are available for all members on the Institute of Excellence learning platform.

surface. One study has reported that periocular radiotherapy contributes to tear film instability as a result of meibomian gland damage.[5]

Q: Do we know why 'screen time' makes meibomian gland dysfunction (MGD) symptoms worse? Is it just from evaporative dry eye?

A: Two main theories exist – first: screen time results in a reduced blink rate,[6] or a greater number of partial, rather than complete, blinks,[7] and that this, in turn, impacts on the spread of the tear film, resulting in corneal desiccation.

Second: the decreased blink rate means that there is less expression of meibum, since meibum is largely expelled onto the lid margin with each blink due to the action by the muscle of Riolan within the lids.

In a study by Wang et al.,[8] incomplete blinking was associated with a two-fold increased risk of dry eye disease. Hence, it is important to remind our patients about 'blink hygiene,' particularly when using their devices.

Q: Do you use a numbing agent prior to gland expression? And how do you know when to stop expressing each gland?

A: I use the Blephasteam for 10 minutes to heat up the lids to facilitate expression, and then instil a drop of anaesthetic into each eye.

I follow that up with a cotton bud soaked in anaesthetic that I use to run along the meibomian gland orifices to loosen up any keratinised material obstructing the orifices.

I then use lissamine green to delineate Marx's line and debride along the meibomian gland orifices with a golf spud. Finally, I use forceps to express the glands, typically making two-to-three passes. I will often instil a corticosteroid post-expression and advise patients that they may experience some redness and discomfort post-procedure.

Q: What is the most popular/ recommended heat therapy method used in retail optometry practices for in-room meibomian gland expression?

A: The Blephasteam is quite straight forward to use. Otherwise, use any heat pack like the Bruder Moist Heat Eye Compress, which is washable, or the EyeEco Derm mask, which has disposable liners.

Q: Are there any studies showing that dry eye treatment slows MG drop out? And how do you manage MG drop out?

A: Not many studies have looked at this. In a retrospective review of patients who have undergone intense pulsed light, an improvement in meibomian gland dropout was noted at three months in people with mild-to-moderate gland atrophy.[9]

A recent Cochrane review, however, showed that there is a current scarcity of evidence that this form of treatment has any effect on meibomian gland dropout.[10] This suggests that we need more randomised, controlled, clinical studies to be conducted that include meibomian gland dropout as an endpoint.

Q: Which steroid is your drug of choice for DED?

A: The two main steroids of choice would be either Flarex (flurometholone acetate) or FML (fluorometholone alcohol). In the case where preserved drops are not an option, I opt for preservative-free prednisolone sodium phosphate 0.5% minims. ▲

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AMD Masterclass:

From the AMD webcast, available on the Institute of Excellence.

Your reticular pseudodrusen questions answered

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Angelica Ly
PhD GradCertOcTher BOptom (Hons) FAAO

Centre for Eye Health and the School of Optometry and Vision Science UNSW

The topic of reticular pseudodrusen (RPD) has seen a surge in research and clinical relevance since its first descriptions in the 1990s.[1] We now understand that RPD confer a much higher risk of progression to late age-related macular degeneration (AMD) compared to conventional drusen.[2]

With the widespread dissemination of multimodal imaging, our definitions of RPD have evolved to incorporate its combined appearance via optical coherence tomography (OCT), near-infrared (NIR), and fundus autofluorescence (FAF). Nevertheless, a universal definition and comprehensive management protocol still evade us. In this Q&A session, we will answer some of the most pertinent questions relating to the diagnosis and management of RPD (with a focus on the application of the Age-Related Eye Disease Study [AREDS] type supplementation).

Q: Can you tell the difference between RPD and conventional drusen by looking at the fundus photo alone?

A: Yes, but don't do it – you will miss a lot of RPD!

Using coloured fundus photography (CFP) to look for RPD, we'd look for lesions that are typically: 150-250 µm in size; 'yellowish' but whiter than conventional soft drusen; flatter and more regular than conventional soft drusen; more visible using the blue-channel; in an interlacing network (although they can occur in isolation).[3] However, how many of the above criteria exactly match the appearance of RPD seen in Figure 1?

To accurately diagnose RPD, studies recommend using two or more imaging modalities.

OCT and NIR consistently show the greatest overall sensitivity and specificity for diagnosing RPD (> 90 per cent), followed by fundus autofluorescence (70-90 per cent sensitivity, > 90 per cent specificity).[4–7]

Overall, OCT and/or NIR should thus be used as the primary modalities to detect RPD. FAF and/or CFP may then be used as supplementary tools to confirm diagnosis.[4–8]

With further multi-modal imaging, our criteria for defining RPD has evolved significantly beyond what was originally established using CFP:

- Using OCT and confirmed through histological studies, RPD are hyperreflective lesions, existing above the RPE, directly beneath the photoreceptors;[9–11]
- Using NIR, RPD are hyporeflective with a mild hyperreflective background, and can be 50-400 µm in size as opposed to earlier descriptions of 125-250 µm;[8]
- Using FAF, RPD typically have a hypo- or iso-fluorescent centre with mild hyperfluorescent borders amongst a reticular, interlacing network.[3,12,13]
- Overall, using a relatively broader scanning area, for example 30 degrees × 25 degrees, will help maximise detection as RPD typically present at the vascular arcades.[5]

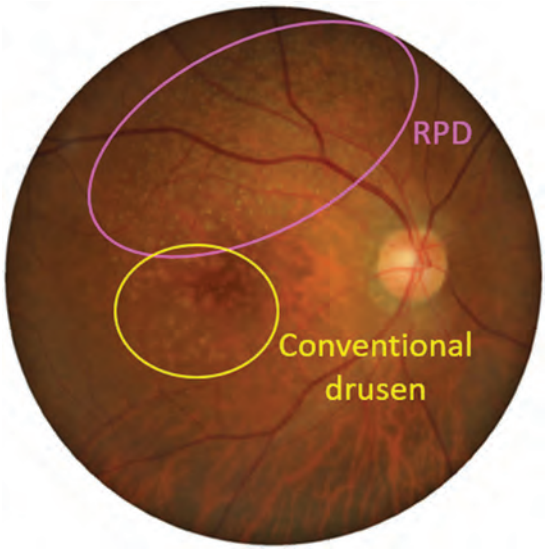

Figure 1. An 81-year-old female with RPD and conventional drusen, revealing a subjective component to interpretation when imaged through CFP alone

Q: Are RPD exclusive to AMD?

A: Definitely not; up to 35 per cent of RPD occur in aged eyes with no AMD.[1,5,14]

RPD has been associated with acquired vitelliform lesions (Figure 2A), pseudoxanthoma elasticum, and Sorsby's fundus dystrophy.[3]

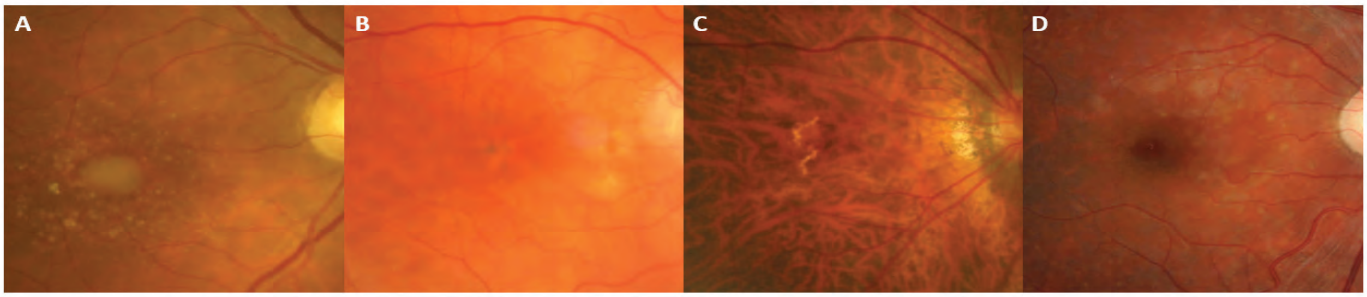

Figure 2. CFP images of yellowish fleck-like deposits at the macula. A: Large acquired central vitelliform lesion with associated RPD superiorly. B: Acquired vitelliform lesion at the fovea. C: Lacquer cracks in high myopia. D: Stargardt's disease. Note the difficulty in diagnosis without any contextual clues, or the use of other imaging modalities beyond CFP. Further guidance on the differentiation of these lesions is available elsewhere.[15]

Additionally, RPD need to be differentiated from similar yellowish fleck-like deposits at the macula such as other types of drusen, Stargardt's disease, retinitis punctata albescens and fundus albipunctatus. Differentiating between these lesions can be difficult using CFP alone (Figures 2B-D), so again, we recommend using other imaging modalities as well to supplement the diagnosis.

Q: Would you recommend use of nutritional supplements for patients with RPD, even if they only have early or no AMD?

A: No. There is currently no evidence available that confirms nutritional supplements help with RPD lesions. The AREDS formula has only been proven to be effective in reducing the risk of progression to neovascular AMD in eyes with at least intermediate AMD.[16]

Thus, the best management for RPD is to manage the underlying condition and in the case of AMD, to be aware that there is increased risk of progression to late AMD[2] and greater loss of functional vision[17] when compared to conventional drusen. It is interesting to note that the AREDS studies classified AMD based off fundus photos, and thus RPD were not clearly distinguished as a unique entity from conventional drusen.[16]

For further controversy around the use of AREDS type supplements, see 'bonus question.' ▲

BONUS Q: Should patients on AREDS-type supplements be genetically tested?

A: Time will tell. Pharmacogenetic testing for AREDS supplementation has the potential to become a lot more prevalent in the future, although clinical trials are still underway and the implementation of necessary supportive resources is ongoing.

In a recent statistically-robust study regarding pharmacogenetic testing with AREDS supplementation for AMD, it was identified that: 'individuals with high CFH and no ARMS2 risk alleles and taking the AREDS formulation had increased progression to NV (neovascularisation) compared to placebo. Those with low CFH risk and high ARMS2 risk had decreased progression risk.'[18]

The risk for progression to NV in the former genotype group taking AREDS supplementation was almost 300 per cent, versus just 50 per cent in the latter group.

Of note, this study confirmed previous reports that AREDS supplementation is only effective in reducing risk of progression to neovascular AMD, and not central geographic atrophy.[16]

If the individual results of taking supplementation are so variable, then why isn't pharmacogenetic testing more prevalent? On the one hand, as clinicians, we should ensure that we are not harming the patient by suggesting use of the AREDS supplementation.

A recent study has also shown that a majority of participants were interested in undergoing AMD genetic testing regardless of having no signs or symptoms of AMD, as they had a family history of AMD or another genetic disorder. Results of testing being relayed to participants also subsequently led to modified behaviours to reduce the risk of AMD.[19]

However, on the other hand: there is the possibility of inducing anxiety and financial burden; concerns regarding the security and privacy of health data may arise, particularly if tests are available online and not properly regulated; genetic typing may lead to discrimination (by affecting eligibility for particular health insurance policies). Also: clinicians will need to be trained in interpreting and relaying all manner of results and management plans to patients.

Overall, while the evidence for pharmacogenetic testing for AREDS supplementation appears promising thus far, further trials are needed to (1) validate results particularly in different cohorts, and (2) ensure proper resources are in place before the implementation of routine pharmacogenetic testing for AMD patients.

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AMD Q&A

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Diabetic Retinopathy

Risks, detection and signs of DR

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Q: Optometry Australia's clinical guideline 'Examination and management of patients with diabetes' mentions that people at higher risk of diabetic retinopathy (DR) include those with a longer duration of diabetes. What is considered a 'longer duration'?

A: While the 'duration of diabetes' is clearly defined as the length of time since diagnosis, the definition of the term 'longer duration' isn't so straightforward, but it reflects an increasing risk of developing DR the longer a person has diabetes.

The bad news...

The META-EYE study pooled the data from 35 studies conducted from 1980 to 2008 of patients with both type 1 and type 2 diabetes mellitus (DM). It reported an overall prevalence of any DR of 21.1 per cent at 10 years after diagnosis, increasing to 76.3 per cent at 20 years. The risk of vision-threatening DR (macular oedema and/or proliferative DR) was also shown to increase with diabetes duration.[1]

In the Wisconsin Epidemiologic Study of Diabetic Retinopathy (WESDR), the cohort with type 1 DM of 0-2 years duration demonstrated a prevalence of any DR of less than two per cent; however, this rose dramatically to 44 per cent at 5-6 years, 75 per cent at 9-10 years and 97 per cent at 15-16 years (see Figure 1). In the same cohort, the prevalence of proliferative DR increased markedly after the first decade (three per cent at 5-6 years rising to 41 per cent at 19-21 years).[2]

In patients with type 2 DM, it is important to keep in mind that there is often a delay from onset to diagnosis which increases the risk of having DR present at the time of diagnosis. The Atherosclerosis Risk in Communities (ARIC) study estimated a median delay from onset of DM to diagnosis of 2.4 years with more than seven per cent of incident cases undiagnosed for at least 7.5 years.[3]

At the time of diagnosis, it is estimated that 20-30 per cent of patients with type 2 DM will have DR and up to three per cent will have vision threatening DR (macular oedema or proliferative DR).[2,4] The Japanese Diabetes Complications Study showed more rapid progression of DR between five- and 10-years duration, compared with before or after that period, in a cohort with type 2 DM.

The good news...

Longitudinal data from WESDR suggests that increasingly effective treatments for hyperglycaemia and more timely interventions for vision-threatening DR have resulted in a relatively lower prevalence of vision impairment over time.[5] As a result, prevalence data from earlier studies may overestimate the current risk of a patient's DR developing at different time points following diagnosis of DM.

Q: Would you recommend checking for neovascularisation of the angle in patients with diabetes?

Detecting angle neovascularisation (NV) in patients with DM is important in preventing progression to sight-threatening neovascular glaucoma. But when is gonioscopy necessary?

In most cases, if NV develops in the angle in a patient with DM, it will also be present on the pupillary margin of the iris.[6,7] Browning performed high magnification slitlamp examination on 310 eyes in 155 patients with DM and found that no eyes had NV in the angle alone; 20 eyes (31 per cent) had NV at both the pupil margin and in the angle whereas 44 eyes (69 per cent) had NV at the pupil margin only.[7]

If IOP is found to be elevated in a

From the DR webcast, available on the Institute of Excellence.

patient with DM, gonioscopy should also be performed. While uncommon, there are isolated case reports in the literature of patients with a history of DR who demonstrated elevated IOP in combination with angle neovascularisation neovascularisation of the iris (NVI).[8]

The verdict?

Assessing for NVI with high-magnification slitlamp examination is essential in all patients with diabetes, however, performing gonioscopy to assess for angle neovascularisation is only necessary if there is NVI present or IOP is elevated.

Q: Do I still need to dilate my patient with diabetes if I have performed ultra-wide field retinal imaging?

A: Ultra-wide field (UWF) imaging is a undoubtedly a valuable supplementary test for our patients with diabetes, however it is not currently considered a substitute for a dilated eye examination in clinical practice.

UWF imaging provides a number of advantages over standard digital retinal photography including: an expanded field of view providing a more 'global' view of the eye and allowing detection and documentation of DR occurring in the retinal periphery; the ability to capture images through small and/or poorly dilating pupils; and image quality that is less impacted by media opacities. In addition, it can be very useful for detecting progression between visits; visualising the retina in patients who cannot be dilated; for effective patient education; and to supplement clinical examination in patients with photophobia, media opacities or poor fixation.

In a 2018 study, Aiello et al. compared UWF imaging to Early Treatment Diabetic Retinopathy Study (ETDRS) 7-field imaging in 742 eyes of 385 participants.[9] Exact agreement in the level of DR, using an 8-point scale, was found in 48.4 per cent of eyes and agreement was within one step in 88 per cent of eyes. Seventeen per cent of eyes initially showed a difference of two or more steps and were adjudicated by a senior retinal specialist ophthalmologist.

Following adjudication, the two imaging modalities gave exact agreement in 59 per cent and within one level in 96.9 per cent: ETDRS 7-field imaging was considered more accurate in 22 eyes and UWF imaging was considered more accurate in 31 eyes, suggesting benefits to both imaging modalities. Importantly, UWF imaging revealed DR lesions located outside the 7-field area including 15 per cent of haemorrhages or microaneurysms, 13 per cent of intraretinal microvascular abnormalities (IRMA), 12 per cent of venous beading, and 53 per cent of new vessels elsewhere. This reinforces the need to examine the retinal periphery in patients with DM particularly as previous studies have demonstrated that the presence of peripheral DR increases the risk of future progression.[10]

In our clinical practice at the Centre for Eye Health, we find that subtle retinopathy signs that can be visualised with dilated slitlamp fundoscopy (such as small microaneurysms, IRMA and early NV) can sometimes be difficult to see on UWF images. In our experience, detection of DR with UWF imaging improves with experience as well as methodical use of the zoom, gamma and green-separation filters to optimise the retinal view.

Finally, it is worth noting that Medicare does not cover retinal imaging by optometrists and MBS item 10915 for examining the eyes of a patient with DM requires instillation of a mydriatic and a comprehensive examination to meet the item descriptor for billing. ▲

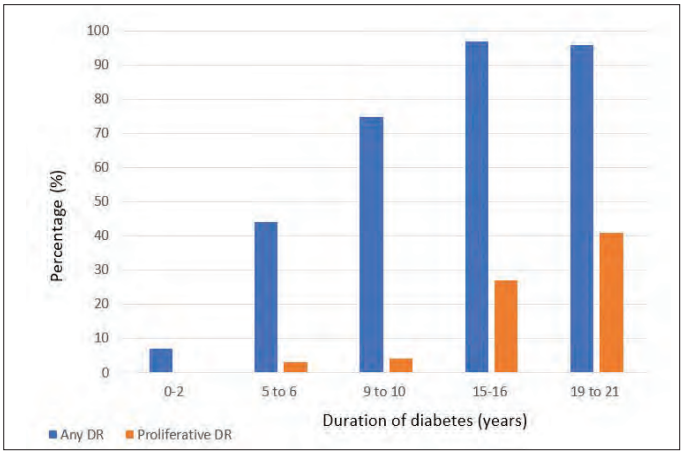

Figure 1. Prevalence of DR with duration of diabetes in patients with type 1 diabetes (data from the Wisconsin Epidemiological Study of Diabetic Retinopathy adapted from Klein et. al. 1984)[2]

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Low-dose atropine for myopia control

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Globally, the use of low-dose atropine eye drops (0.01% - 0.05%) for controlling myopia progression is becoming increasingly common.[1] However, important clinical questions remain regarding efficacy, the appropriate concentration to use, and when and how to discontinue treatment. Providing evidence-based answers to these questions is problematic, as the increased prescribing of atropine has outstripped the available evidence from well-controlled clinical trials. This short article answers some of the questions; it reveals that starting children on atropine is one thing – how to manage them afterwards may be quite another.

Why prescribe atropine?

The primary reason for prescribing atropine is to reduce the abnormal rate of eye growth associated with myopia progression. Excessive enlargement of the myopic eye stretches the retina and choroid and increases the risk of serious ocular pathologies[2] later in life. Thus, to be effective, myopia control interventions such as atropine must slow the rate of abnormal eye elongation.

Furthermore, all myopia, not just high myopia, is associated with considerable risk of later pathology,[3] implying that almost all children with progressing myopia are potential candidates for myopia control.

Low-dose atropine eye drops are a safe and well-tried method for controlling myopia progression in Asian[4-7] and Western[8,9] settings. They are moderately effective, in a dose-dependent manner, and have much milder side-effects and less rebound on cessation, than higher concentrations (0.1% to 1%).[10]

Atropine as monotherapy is an appropriate alternative to orthokeratology or other contact lens methods of control, and it has the advantage that it can be administered to children by parents at home. In addition, increasing evidence suggests that, when used in conjunction with optical methods such as orthokeratology,[11] atropine can enhance myopia control efficacy.

When to prescribe?

Progression is typically fastest in the early stages of childhood myopia development,[12] so atropine treatment will have maximum benefit when administered early. This is particularly relevant in children at risk of developing significant degrees of myopia: those with rapid progression, myopia at a young age, parental myopia, and predisposing lifestyle with little outdoor time.[13]

Which concentration?

Early treatment with 0.025% or 0.05% atropine will be more effective in slowing eye growth than the more commonly used 0.01%. Recent, well-controlled (but short) studies show that low-dose atropine reduces refractive and axial length changes in a dose-dependent manner.[4,6,7] However, most evidence indicates that it is less effective at inhibiting eye growth than in reducing refractive progression, and that the most commonly employed concentration (0.01%) has debatable clinical efficacy in retarding eye growth.[14] Consequently, it has been proposed that treatment should be initiated with higher concentrations (0.025% or even 0.05%).[15]

Clearly, the choice of which concentration to use for an individual case will depend on many factors, including the side-effects (for example: the degree of mydriasis) that the patient will tolerate and the degree of risk for rapid progression.

Although efficacy may depend on circumstances (ethnicity, environment and so on), even with 0.05%, 45 per cent of children in a long-term Taiwanese study[5] had the atropine concentration increased to 0.1% because they were progressing more than 0.50D/year when using 0.05%. Beneficial effects may take some months to develop, and it has been suggested that loading doses may hasten a therapeutic response.

However, whether loading should be in the form of more frequent dosing with low concentrations, or by increasing the once-a-day concentration to start with is not known.

What should be monitored?

Regular monitoring of progression is needed so that adjustments to management (increasing concentration) can be made in a timely manner. Ideally, both refractive changes and axial eye length changes should be monitored to assess progression, but both have drawbacks. Measurement of axial length is typically a more repeatable measure than refraction, but changes in eye length with myopia progression will be partially confounded by natural eye

growth in children up to approximately 13 years of age.[16]

Refractive changes are affected by atropine via its action on the ciliary muscle. The effects of chronic atropine instillation on ciliary muscle action are not well understood, but it is possible that the ciliary muscle continues to relax over time, slowly flattening the crystalline lens and thus masking the effects of increased eye length on refraction. This may account for the observation that low-dose atropine appears more effective at slowing refractive changes than at slowing eye growth. Nevertheless, lack of access to eye length measurement should not preclude practitioners from using atropine for myopia control–with the appreciation that refractive changes may not be telling the whole story.

When and how to discontinue?

This important issue has received little attention in the literature. A joint report by The World Health Organization and the Brien Holden Vision Institute in 2015[17] included very brief potential clinical guidelines relating to the use of 0.01% atropine for myopia control in children six to 10 years of age. The guidelines suggest treatment with 0.01% for two years initially. For those progressing between 0.50D and -1.00D in the second year, treatment is continued for a further one to two years. For children progressing either less than -0.50D or more than 1.00D in the second year, tapering and stopping atropine is recommended, on the grounds of no further need (for slow progressors) and likely non-responders (for fast progressors). These tentative guidelines are clearly incomplete; in particular, they provide no suggestions for proactively managing fast progressors.

Recently, more complete guidelines for controlling progression have been published,[18] which acknowledge the benefits of integrating other strategies such as adjusting atropine concentration, increasing outdoor time, the inclusion of alternative optical treatments, and so on into clinical myopia control.

These more complete guidelines recommend treating with 0.01% initially and monitoring progression with cycloplegic refraction every six months. If progression remains below -0.50D/year, then 0.01% atropine is continued for two years, at which point treatment is stopped for one year to assess untreated progression. If, in that year, progression greater than 0.50D/year occurs, then treatment is resumed. Alternatively, if at one of the initial six-month follow-ups, progression is greater than 0.50D/year, then other options are considered, such as increasing atropine concentration or switching to optical methods. Thereafter, two alternative strategies are proposed: one is treating for two years followed by no treatment for one year to assess untreated progression.

Another strategy is simply to continue treatment and monitoring and to stop treatment in late adolescence (16-18 years of age), when myopia progression typically slows naturally. There is little published information on either of these strategies. Stopping treatment after approximately 18 years of age on the basis that no significant myopia progression occurs after late adolescence may be a wishful assumption. If an individual's myopia progression has been successfully controlled in childhood, stopping treatment, even after 18 years of age, may possibly result in rebound, as axial myopia progression is not uncommon in adults.[19] ▲

Disclosures: The author is named inventor on patents relating to contact lens designs for slowing myopia progression in children.

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ADDENDUM: study suggests 0.05% might be the way to go

The randomised, double-masked trial extended from the Low-Concentration Atropine for Myopia Progression (LAMP) study showed that those treated with 0.05% atropine (compared to lower concentrations) had the least change in mean spherical error and axial length.

This was also seen in the placebo group that was commenced on this treatment. The side-effect profile was well tolerated while the visual acuity and vision-related quality of life remained unaffected.

Yam JC, Li FF, Zhang X et al. Two-Year Clinical Trial of the Low-Concentration Atropine for Myopia Progression (LAMP) Study: Phase 2 Report. *Ophthalmology* 2020; 127: 910-919.

The Black Triangle scheme

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As new medicines are developed and come onto the Australian market, it is important to understand all the side effects and adverse reactions that can occur when they are used by the public.

Started by the Therapeutic Goods Administration (TGA) in January 2018, the Black Triangle Scheme was implemented to monitor new medicines.

What is the Black Triangle Scheme?

All medications that enter the market have undergone clinical trials to ensure appropriate safety and efficacy, however, it's important that these medicines continue to be monitored for adverse effects as they become widely used outside of the strict inclusion and exclusion criteria of clinical trials.

The Black Triangle Scheme was introduced as a simple way for practitioners and patients to identify all new medicines, or medicines that are being used in significantly different ways. An example of when a currently-available medicine would be added to the black triangle scheme is if it was previously used systemically and is reformulated for topical use in the eye. When practitioners and patients are using medications indicated as part of the Black Triangle Scheme, they are encouraged to report any adverse effects that are associated with the medication.

Which ophthalmic preparations are currently on the Black Triangle Scheme?

Cequa (ciclosporin 900 microgram/mL) is a commercially-available formulation of ciclosporin eye drops. It is available for prescribing by therapeutically endorsed optometrists and is on the Black Triangle Scheme. Ciclosporin has been used for many years to prevent graft rejection following kidney, liver and heart allogeneic transplantation. However, because it is only now being used for the ocular surface and dry eye it has been put on the Black Triangle Scheme.

Xiidra (lifitegrast 50mg/ml) is a novel treatment for moderate-to-severe dry eye. As this is a new medication to the Australian context, it is not currently listed on the schedule of medicines that optometrists can prescribe. It is indicated when use of conventional lubricants are insufficient to manage the disease.

Which medications are put on the Black Triangle Scheme?

All newly-registered prescription medications will be included in the scheme. It does not apply to biosimilar medicines or generic versions of previously-approved medications. Seasonal influenza vaccinations have a different monitoring system and are also not included in the Black Triangle Scheme.

Medications will generally stay on the Black Triangle Scheme for five years. If concerns are raised, the TGA can extend the timeframe a specific medicine stays on the Black Triangle Scheme.

Medications that are being used for a different condition, or for a significantly different population will also be added to the scheme.

Does this mean the medication is more dangerous?

No. All newly-registered medications will be added, and it does not indicate there is evidence that there is any increased risk of adverse reaction.

How do I know which medications are on the Black Triangle Scheme?

The Black Triangle symbol will appear

Where and how do I report adverse reactions?

Health professionals and consumers can report adverse reactions online at aems.tga.gov.au. This process collects information about the practitioner, the medicine and the details of the adverse reaction.

Australian Register of Therapeutic Goods (ARTG) search: www.tga.gov.au/australian-register-therapeutic-goods

For more information on the scheme visit: www.tga.gov.au/black-triangle-scheme

To report an adverse event visit: aems.tga.gov.au.

on Product information (PI) and consumer product information (CMI) along with the following information encouraging health professionals and consumers to report adverse effects:

For the PI: 'This medicinal product is subject to additional monitoring. This will allow quick identification of new safety information in Australia. Healthcare professionals are asked to report any suspected adverse events at Reporting problems' (www.tga.gov.au/reporting-problems).

For the CMI: 'This medicine is subject to additional monitoring in Australia. This will allow quick identification of new safety information. You can help by reporting any side effects you may get. You can report side effects to your doctor, or by visiting Reporting problems' (www.tga.gov.au/reporting-problems).

Also you can search the Australian Register of Therapeutic Goods (ARTG) at www.tga.gov.au/australian-register-therapeutic-goods and by utilising the advance search you can specify inclusion or exclusion of Black Triangle Scheme medications.

What adverse reactions should be reported?

Any unfavourable or unintended sign, symptom or disease entity associated with the use of the medicine should be reported. Particular attention to serious complications or complications that do not appear as a known side effect should be reported. It is important to recognise you do not need to be certain that it is associated with the medication, just suspicious.

Each adverse event report aids the TGA in understanding the medication's safety, and health practitioners are encouraged to help by reporting adverse events.

The Black Triangle Scheme is aimed to provide a clear pathway for practitioners and patients to report adverse events when using new medications. The information gathered is used to inform practitioners and improve safety for the public. Optometrists often identify adverse reactions to medications, and this provides a mechanism to proactively participate in keeping the public safe. ▼

Glaucoma

Q A

From the Glaucoma webcast, available on the Institute of Excellence.

Progression analysis and the evidence on intervention

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Q: What is the minimum time required between optical coherence tomography (OCT) scans to detect progression? For example, if you take a baseline for a patient with a suspicious optic nerve head (ONH), in the interest of minimising patient costs, how long should you wait before taking a repeat OCT to detect change?

A: Currently, OCT devices do not account for age-related change when performing the change analysis.

When a change is identified as significant, it only signifies that there is a significant difference to 0. In other words, it's a statistical test, but not a clinical indicator.

This is different to visual field testing, where indices like the mean deviation score is corrected for age. Furthermore, some progression analyses include confidence intervals or error bars for the slope, which accounts for the variability in the measurement. It is essential for clinicians to account for test-retest variability–which may differ across instruments–to make accurate judgements on progression.

Studies examining visual field changes typically use an interval of two years for progression analysis. Fortunately, disease progression in glaucoma tends to occur slowly, and significant vision loss is unlikely to occur within two years.[1]

This is something for the clinician to bear in mind in terms of the urgency at which progression needs to be detected.

As a rule-of-thumb then, a clinician following the manufacturer's recommendations should use a minimum of five test results over a period of two years (assuming two baseline scans).

It is important to remember that follow-up should occur in the interim, with repeat testing indicated and titrated based on suspicious findings, such as patients in whom there are other risk factors such as pseudoexfoliation. In the case of glaucoma, signs such

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Glaucoma Q&A

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as intraocular pressure fluctuations or elevations, or disc haemorrhages should signal the need to reassess.

Q: Is race/ethnicity a variable in OCT analysis and if yes, what is the basis for this?

A: Race and ethnicity have been comprehensively demonstrated to affect relevant ocular biometric parameters and may play roles in the epidemiology of disease.[2] The basis of this is biological.

For example, the work of Girkin et al.[3] showed that European patients have smaller optic disc areas compared to other races, Indian patients have smaller rim area, Indian and Hispanic patients have thicker global retinal nerve fibre layer measurements, and African patients have thinner inner retinal thickness at the macula.

These findings are largely corroborated by Knight et al.,[4] who highlighted that people of African descent have large disc size, cup-disc ratio and cup volume compared to people of other races.

A question remains regarding individuals of mixed race. This has not been studied in the literature.

While race and ethnicity have been acknowledged to be important in the interpretation of OCT results, many instruments do not have normative databases of sufficient ethnic diversity to perform race-specific analyses. Indeed, there are comments that other forms of biometric diversity such as refractive error[5] should be considered.

Q: What advice do you generally tell patients regarding diet, supplements and lifestyle?

A: Glaucoma is a multifactorial disease and risk factors–individually or in combination–contribute to the overall course of the disease in a complex manner. There are no robust evidence-based guidelines to support significant modifications to diet, supplementation and lifestyle specifically for glaucoma risk.

Reports in the literature are largely limited to observational studies, far from the expected standard of a randomised clinical trial. Clinicians should remain wary and sceptical, as observational studies have a high risk of biases including selection bias. See Al Owaifeer and Al Taisan for a review.[6]

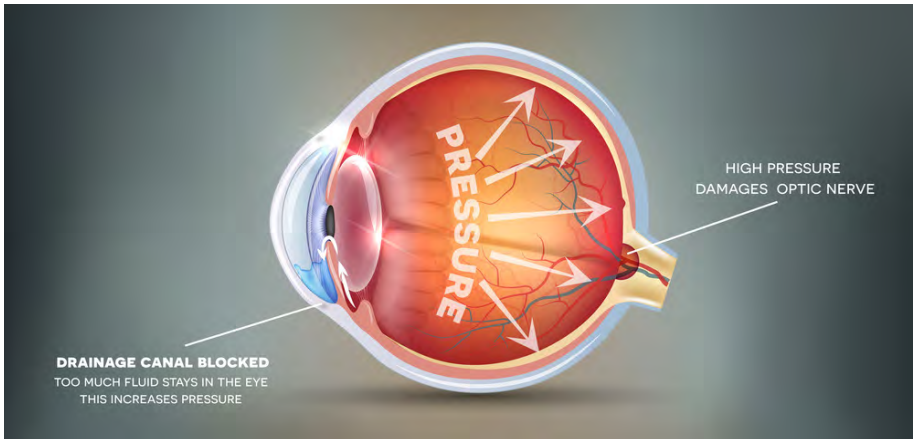
The clinician should bear in mind though that this kind of advice would be specific to the individual and their own circumstances.

Furthermore, there is evidence to show that effects from any of these interventions are likely transient (for example, intraocular pressure reductions lasting in the order of minutes) and are unlikely to significantly affect the course of a chronic disease.[7] Thus, no specific interventions are currently supported by the literature.[8]

Q: Is there any association between the gut microbiome and glaucoma?

A: The link between gut microbiome and glaucoma has been hypothesised to arise from the microbiota-gut-retina axis:[9] the resultant autoantibodies and auto-reactive T cells lead to autoimmunity and hence damage to the optic nerve.

Analogous neurodegenerative diseases such as Alzheimer's disease and Parkinson's disease have also been linked to gut microbiome. As evidence is still emerging, it may be better to regard gut microbiome as an emerging risk factor for glaucoma, in the same manner as other systemic vascular or ischaemic disease. ▲



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Adenoviral conjunctivitis

A challenging condition in a challenging time

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Human adenovirus is the most common cause of infection in the conjunctiva, accounting for up to 75 per cent of all conjunctivitis cases, affecting people of all ages and demographics worldwide.[1] The most frequent presentation of adenovirus conjunctivitis is epidemic keratoconjunctivitis (EKC), typically caused by serotypes 8, 9, 19, 37, 53 and 54, followed by pharyngoconjunctival fever (PCF), usually caused by serotypes 1-5, 7 and 11.[1-3] Non-specific follicular conjunctivitis is another manifestation, primarily due to serotypes 1-11 and 19.[1,2]

The prevalence and incidence of adenoviral conjunctivitis is unknown as many affected people do not seek medical care.[1] Fortunately, most infections are mild and self-limiting, however there can be serious repercussions if complications are not recognised.

Patients with EKC usually present with a red, watery eye with possible foreign body sensation and photophobia. There may be ocular or periorbital pain and decreased vision in more severe cases. They may report flu-like symptoms, such as fever, malaise, myalgia and respiratory symptoms or a recent history of a family member being affected.

The common ocular signs include bulbar conjunctival redness, chemosis of the eyelid and conjunctiva, tarsal follicular reaction and petechiae.[1,4]

Pseudomembranes and true conjunctival membranes may form in EKC, ultimately causing subepithelial fibrosis and the formation of a symblepharon and punctal occlusion, which can lead to diplopia and ongoing epiphora.[1,4-6]

Corneal involvement distinguishes EKC from other adenoviral infections. Multifocal subepithelial infiltrates (SEIs) may typically develop seven to 10 days after the initial signs of infection, possibly reducing acuity and may persist for weeks to years.[1,4]

CASE REPORT

A 35-year-old Caucasian male presented to the Royal Victorian Eye and Ear Hospital with a unilateral red eye. He reported the conjunctiva in his left eye had been red accompanied by mild watery discharge for the previous three days.

He denied experiencing any cold or flu-like symptoms and was not in close contact with any other person with conjunctivitis.

He was not a contact lens wearer, denied taking any medication currently and was not aware of having any allergies.

On examination, visual acuities were R 6/6 and L 6/6. The bulbar conjunctiva was moderately hyperemic with prominent follicles present on the inferior palpebral conjunctiva of the left eye (Figure 1). His right eye was normal. Intraocular pressures were measured as R and L 13 mmHg with an iCare tonometer. All other ocular findings were unremarkable.

A diagnosis of possible adenoviral conjunctivitis was made and

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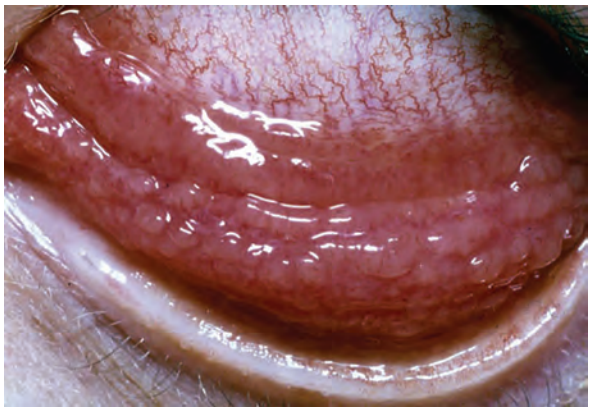

Figure 1. Inferior palpebral conjunctiva showing prominent follicles. Image: Royal Victoria Eye and Ear Hospital.

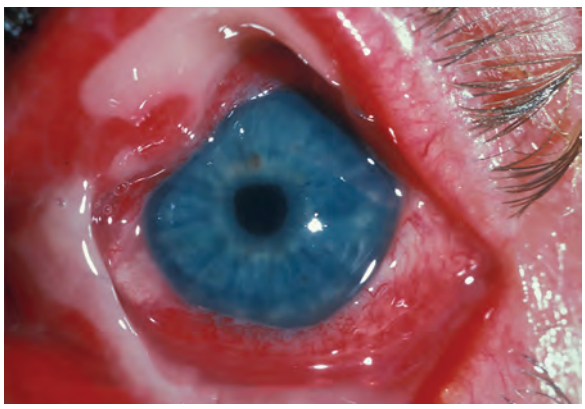

Figure 2. Inferior and superior palpebral conjunctivae with pseudomembranes. Image: Royal Victorian Eye and Ear Hospital.

Adenoviral conjunctivitiis

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the patient was advised to use preservative-free lubricants every two hours for the next two weeks or until symptoms resolve. He was also cautioned about being careful not to spread the infection to his right eye and to other members of his family. He was advised to take time off work as he was likely infectious. He was told to return if his left eye worsened, in particular to monitor for declining visual acuity and increasing discomfort over the next week.

Ten days later, the patient returned with bilateral swollen eyes complaining of increasing stringy and watery discharge. He had noticed that his symptoms were worsening.

On examination, pseudomembranes were present in the superior and inferior palpebral conjunctivae of the left eye, with marked conjunctival hyperemia and inflammation (Figure 2). There was an area of symblepharon which fortunately had not affected his eye movements, with no diplopia reported. His right eye had developed follicles only and there was no corneal involvement in either eye.

Visual acuity in the left eye dropped to 6/12, mainly due to the discomfort and discharge.

Debridement of the pseudomembranes was painstakingly performed, every two days, with Flarex cover to control the inflammation, until no further psuedomembranes formed. Careful use of steroid drops aims to limit the development of further symblepharon. Lubricants were continued regularly for relief of symptoms.

The patient's conjunctivitis resolved two weeks later, but the symblepharon remained permanently as an undesirable complication.

Discussion

The diagnosis of an adenovirus infection is typically made based on the history, symptoms and clinical findings. Laboratory diagnostic testing with polymerase chain reaction (PCR) is usually not performed due to the costs and time delay. The rapid antigen detection immunoassay may be a better alternative, carried out in-office with a result in 10 minutes.[1-4] Testing is done if there is uncertainty with the diagnosis as it is vital that the correct diagnosis is made before deciding on the management. Differential diagnoses of the more common forms of conjunctivitis with their key features are outlined in Table 1.[1,7,8]

Another differential diagnosis to consider, especially during the current novel coronavirus pandemic (COVID-19), is the SARS-CoV-2 virus.

There have been reported cases of acute viral conjunctivitis in patients with confirmed SARS-CoV-2 infection, often describing a sore throat, foreign body sensation, conjunctival redness, watery discharge, with the presence of palpebral conjunctival follicles and pre-auricular lymphadenopathy.[9] All patients with this presentation, seen during the pandemic, should be referred for COVID-19 PCR nasopharyngeal and throat swabs.

The symptoms and duration of adenoviral conjunctivitis can vary widely, with most resolving completely within three weeks. Supportive treatment, such as preservative-free artificial lubricants and cool compresses, can provide satisfactory symptomatic relief.[1,2,4,7]

Steroids should be restricted to cases of EKC with complications involving pseudomembranes or persistent subepithelial infiltrates (SEI) (Figure 3) which may reduce vision.[1,4,11] Ophthalmological referral may be necessary in these cases. Steroids reduce conjunctival and corneal inflammation, but may actually enhance adenoviral replication and increase the period of viral shedding, prolonging the entire clinical course of EKC.[4,9,12]

Before prescribing a steroid, it is important to rule out acute herpes simplex virus (HSV) conjunctivitis. Without accompanying skin or corneal involvement, herpetic clinical presentation may be very similar to adenoviral conjunctivitis,[2] comparative symptoms and signs shown in Table 1. Similarly, steroids drops in bacterial or Acanthamoeba infections can result in rapid deterioration, with severe corneal injury including corneal melts and perforation possible.[2]

Povidone-iodine (PVI), commercially available as Betadine, is a broad-spectrum antiseptic ophthalmic solution which has been used for many years to prophylactically reduce microbial flora prior to ocular surgery. PVI has been reported to

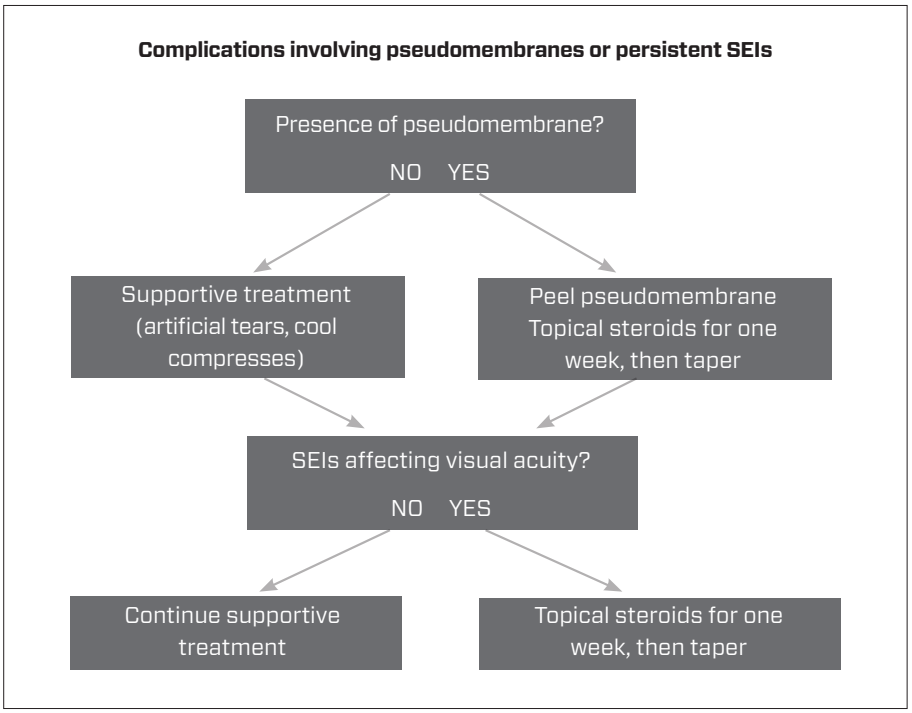

Figure 3. Treatment options for adenoviral conjunctivitis (Adapted from Pihos[4])

	Adenoviral	Herpetic	Bacterial	Allergic	Chlamydial
Unilateral/Bilateral	Begins unilateral, may become bilateral	Unilateral	Unilateral	Bilateral	Unilateral
Hyperemia	Generalised	Generalised	Generalised	Generalised	Generalised
Discharge	Watery	Thin, watery	Mucopurulent	Watery	Mucopurulent
Itching	Minimal	None	Minimal	Severe	Minimal
Palpebral involvement	Follicles Pseudomembranes	Follicles	Papillae	Papillae	Follicles
Pre-auricular lymphadenopathy	Common	Common	Uncommon	None	Common
Sore throat/fever	Occasional	Occasional	Occasional	Never	Never

Table 1. Differential diagnoses and key features of conjunctivitis

reduce the viral load and severity of symptoms *in vitro* and *in vivo* studies for the treatment of EKC, but there are no controlled trials supporting this treatment option,[11] so its use currently remains off-label.[12] After topical anaesthetic, a pre-irrigation non-steroidal anti-inflammatory drug (NSAID) drop is instilled followed by five drops of 5% povidone-iodine for 60 seconds. The lid margins are swabbed with 5% povidone-iodine and the ocular surface rinsed with sterile normal saline.[12] (That is: saline solution with 0.9% sodium chloride, as opposed to hypotonic or hypertonic saline solution).

Numerous trials have been underway worldwide to develop a safe and effective antiviral drug for ocular adenoviral infections,[3,12] including a topical treatment aimed at reducing symptom duration, currently recruiting at the Royal Victorian Eye and Ear Hospital.

EKC is highly contagious and easily transmitted through hand to eye contact or respiratory droplets and commonly from exposure to infective ophthalmic clinics or family members.[2,14] Adenoviruses, in their desiccated form, may remain viable and can be recovered at a clinically infectious concentration up to 28 days on dried plastic or metal surfaces.[15] Patient education is vital to minimise spread of the infection particularly in the two week period from when symptoms begin. Contact lens wearers should dispose of their lenses as the virus can survive in both chemical and hydrogen peroxide disinfection systems.[16]

Eye-care practitioners should also take additional precautions when examining patients with known or suspected adenoviral infections. Single-use instruments and equipment should be employed, such as disposable gloves, single-dose eye drops and disposable tonometer prisms or shields.[17,18] Adenoviruses can be resistant to many disinfectants, with recent data suggesting that 70 per cent isopropyl alcohol is ineffective.[19] Surfaces in the consulting and waiting rooms as well as frequently touched objects, such as door knobs and handrails, must be cleaned and disinfected regularly with a bleach-based solution.[19]

Although adenoviral conjunctivitis can be a common condition, its presentation and treatment may be quite variable and challenging to manage, with particular vigilance required during the COVID-19 pandemic. ▲

The authors would like to acknowledge the RVEEH for providing the photographic images.

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COVID-19 and retinal OCT

An Australian case study

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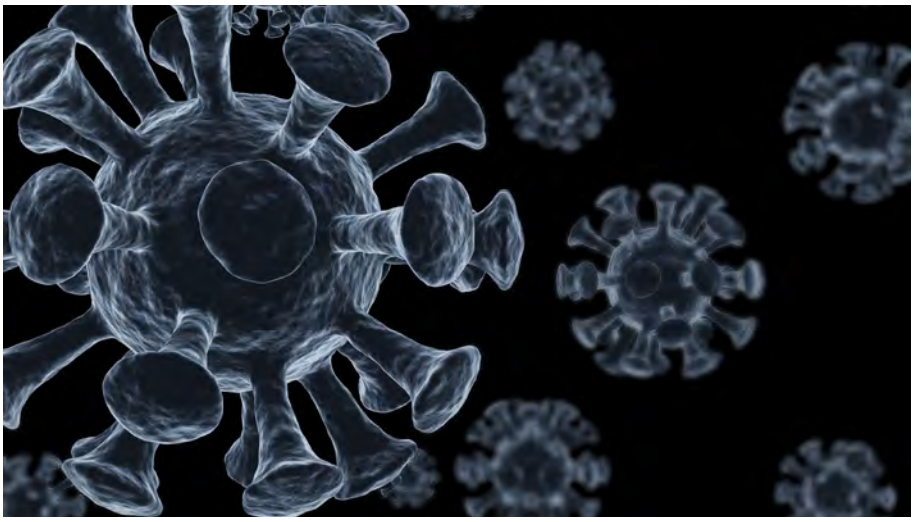

The following presents a primary care optometry encounter with a patient who has recovered from COVID-19. This article aims to highlight the potential for optometry to contribute to a better understanding of the visual and retinal manifestations of patients diagnosed with COVID-19.

Background

Coronaviridae affect different parts of the body, including the nervous system and ocular tissues.[1] Further, coronaviridae are often highly contagious[2] with SARS-CoV-2, in particular, presenting a challenge in containment and treatment.[3]

Infection control and personal protective equipment (PPE) use have long been an important part of clinical competency in order to safely examine patients.[4] The use of PPE has, of course, become more pertinent recently since the COVID-19 outbreak. Providers of primary eye care have ongoing contact with patients,[5] some of whom may be asymptomatic, pre-symptomatic and otherwise undiagnosed, or who have recovered from COVID-19.[6]

A comprehensive eye examination requires close contact which can put both clinician and patient at risk.[7] Practising optometrists should not only have an adequate understanding of infection control, but should also have an understanding of the potential for clinically-significant and subclinical vision and retinal changes in COVID-19 in those who have recovered from the virus.

Recent correspondence in *The Lancet* reported retinal changes in a group of adults with symptomatic COVID-19 infection.[8] All 12 had fever, lethargy and breathing problems and 11 out of 12 also presented with anosmia ('smell blindness'). All 12 showed hyper-reflective lesions at the level of the ganglion cell layer and inner plexiform layers that were most prominent in the papillomacular bundle in both eyes.[8] Four of these patients showed

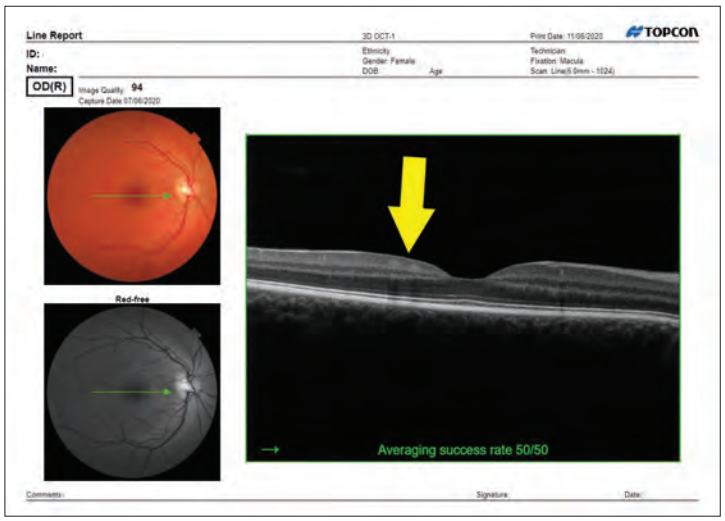

Figure 1. No cotton wool spots or retinal bleeds detected, only mild hyper-reflective lesions detected (Topcon 3D OCT-1).

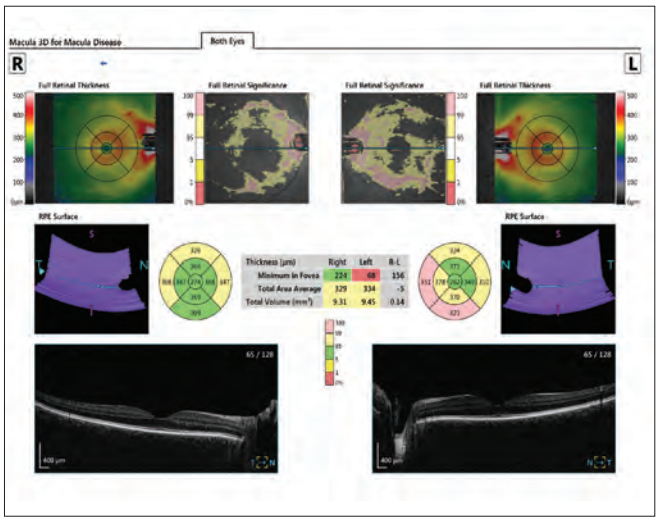

Figure 2. Repeat analysis found similar subclinical results (Canon OCT HS100).

subtle cotton wool spots and micro-haemorrhages in the retinal arcades. Visual acuity and pupil reflexes were normal for this group and there were no visual field test results published. Marinho et al. contend that ganglion cell and plexiform layer changes could be associated with central nervous system (CNS) manifestations that have also been described in animal studies[9] and in COVID-19-related neurological events.[10]

CASE REPORT

The following case study involves a 31-year-old female, Mrs CV, who reported testing positive for COVID-19 in March after flying to Brisbane from London. She had some difficulty smelling and tasting at the time. She reported no vision changes, no blink disturbances, and no other neurological symptoms and was able to exercise rigorously on her treadmill throughout the COVID-19 infection. Mrs CV provided consent to appear in this case study.

Mrs CV had slightly worse central vision in the RE compared with her LE, but as a first presentation to this practice there were no previous records to confirm that this was a reduction in central vision of the RE. Unaided vision: RE 6/6 compared to LE 6/5, with no improvement with pinhole or refraction. She had worse low-contrast sensitivity in the RE compared with her LE. She passed monocular Ishihara with zero errors each eye but could report the RE had duller colour saturation compared with her LE. No symptoms or signs of intraocular inflammation were detected. Binocular vision was normal. Stereopsis was normal. Amsler grid was normal and given for home monitoring. Pupillary reflexes were normal. Wide view retinal scan and corneal topography results were normal for both right and left eye.

Optical coherence tomography (OCT) scans and fundus photography were generally normal, with no cotton wool spots and micro-haemorrhages observed. Mild hyper-reflective lesions at the level of ganglion cell and inner plexiform layers were found in both eyes (Figure 1). These were subtle in comparison to the hyper-reflective lesions described by Marinho et al. There was some slight vascular distension of the inferior vascular arcades observed in the LE with OCT.

Both of these signs were so slight as to be in the order of magnitude of a possible artefact. Repeat OCT (as per Marinho et al.) conducted with another OCT instrument found similar subclinical results (Figure 2).

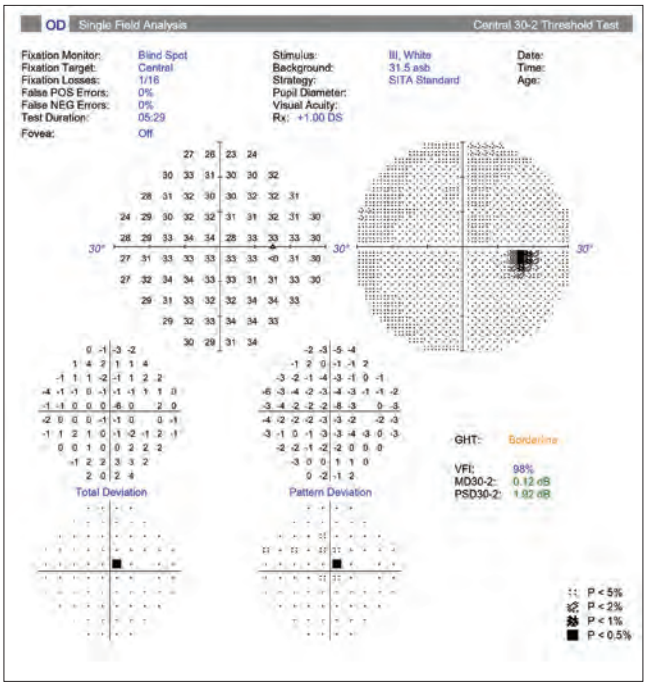

Figure 3. Visual field test found a mild repeatable central relative scotoma in the right eye

Mrs CV completed a Humphrey 30-2 SITA Standard test which found a small central defect in the right eye, confirmed with repeat 10-2 SITA Standard test. This may be associated with mild dysfunction of the inferior papillomacular ganglion cell layer (GCL) bundle (Figure 3).

Both of these subclinical observations point to the need for further research into the form and function of the papillomacular bundle and the way it responds to neuro invasive viral infection. However, despite these interesting VF and retinal signs, in the absence of cotton wool spots and micro-haemorrhages, there is insufficient evidence of retinopathy, optic nerve or CNS disease and she remains subclinical with further review examinations pending.

> Even though non-sight threatening, these findings are important and can contribute to a better understanding of the ocular manifestation of COVID-19.

Conclusion

OCT has become an important diagnostic tool with a range of retinal conditions. Some sight-threatening retinal change is linked to systemic disease, such as diabetes (DM), or even demyelinating neurological conditions such as multiple sclerosis (MS). In these conditions the retinal changes

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with even early disease are easily detectable with OCT.

There has been some published, on-going research into the potential for CNS damage from coronaviridae and similar infections.[1]

SARS CoV-2 showed potential as a highly neuro-invasive and neurotropic disease, and has been demonstrated to be so on occasion during this pandemic.[13,14] Further, the virus has been detected in the human retina of deceased COVID-19 patients.[15]

To date, published clinical ocular manifestations of COVID-19 have focused on ocular tropism and viral conjunctivitis, which are both important considerations when ensuring COVID safe practice. However, recently-published clinical observations have returned the focus to subtle retinal changes.[8]

Pre-COVID-19, there has been a great deal of literature discussing the significance of non-sight-threatening OCT changes and early detection of changes in populations with Alzheimer's and cognitive decline.[11] The elderly are particularly prone to glaucoma and this has been a confounding factor in the construction of a definitive OCT screener for Alzheimer's as the retinal changes with glaucoma are often more obvious than those attributable to potential dementia.[12]

Here also with post COVID-19 examination, the subtle retinal and vision signs detected in this case study are so slight that COVID-19 cannot easily be attributed as their cause and other differential diagnosis such as idiopathic or early MS can be as important. It is important to balance the possibilities of both under-diagnosis and over-diagnosis.

Even though non-sight threatening, these findings are important and can contribute to a better understanding of the ocular manifestation of COVID-19. Australian eye-care professionals are well placed to contribute to these current gaps in knowledge regarding COVID-19. ▲

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CLINICAL AND EXPERIMENTAL

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Advances in the clinical tools that we as optometrists use to diagnose and manage our patients come in leaps and bounds. When it comes to the assessment of the retina, previous generations of optometrists relied on their assessment with direct and indirect ophthalmoscopy, with the possibility of referral to ophthalmology for fluorescein angiography. In the early 1990s we saw the introduction of optical coherence tomography (OCT) which revolutionised the way we assess the retina, enabling a layer-by-layer structural assessment. Since then, OCT has become an integral part of clinical practice, particularly in the monitoring of conditions such as glaucoma and macular degeneration.

One limitation is that OCT did not contribute to our understanding of retinal vasculature. However, in the decades since, OCT has been adapted to enable imaging of the retinal vasculature and is now available in clinical practice as optical coherence tomography angiography, or OCT-A.

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.*

Optical coherence tomography angiography in primary eye care

Summary and comment provided by Maria Markoulli
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OCT-A is non-invasive and relatively inexpensive compared to fluorescein angiography, without the risk of allergy to fluorescein, and without the need for special training. The instrument captures 3D images of the retinal vasculature through motion contrast of moving red blood cells. Blood flow is detected with repeated B-scans at the same retinal cross-section within a specified period.

Volumetric data is produced by combining B-scans at different retinal locations to visualise the retinal vasculature of a region. In the case of disease, the instrument can erroneously segment, resulting in an incorrect representation, so careful attention of the clinician is required.

Unlike fluorescein angiography, OCT-A does not quantify areas of vascular leakage and does not provide temporal information, and it has a limited field of view, with a decrease in image quality in the periphery. Montaging algorithms are being explored to enable increased field of view without compromising resolution.

The parameters that can be evaluated include: acircularity index (the degree to which the foveal avascular zone is different from a perfect circle), flow index, flow velocity, foveal avascular zone area, foveal avascular zone diameter, fractal dimension, percentage area of non-perfusion, vessel density, vessel length density.

While it is in its infancy in optometric practice – the first commercially available OCT-A outside the USA came to market in 2014 – it is important to be aware of the capabilities of this technique over existing tools. To that end, researchers from Flinders University in Adelaide evaluated the evidence available for the role of OCT-A in the diagnosis and prognosis of ocular disease in optometric practice – an important and useful read for anyone contemplating the purchase of such a device.

Alexandra Coffey and her team from Flinders University evaluated 78 randomised, controlled human studies which had used OCT-A in the diagnosis or prognosis of ocular pathology. They report that few of these studies compared against the gold standard, fluorescein angiography, and few studies included appropriate, age-matched controls, further limiting the interpretation of the data. Also, seven different OCT-A devices were evaluated between these 78 studies, further limiting the comparison between studies.

OCT-A was found to be helpful in differentiating adult-onset vitelliform macular dystrophy from age-related macular degeneration as it revealed an increase in vessel density early on in adult-onset vitelliform macular dystrophy. When OCT-A was compared to fluorescein angiography in age-related macular degeneration, it was found to be both sensitive and specific to the detection of neovascularisation. Vessel density was found to be altered in fellow eyes with OCT-A, suggesting that it could improve the detection of subclinical vasculature changes well ahead of what is currently achieved clinically, which would enable earlier intervention. In diabetic retinopathy, OCT-A demonstrated good diagnostic accuracy compared to fluorescein angiography, with a sharper demarcation of the ischaemic vascular areas. In pathological myopia, vessel density as detected with OCT-A was decreased at the radial peripapillary capillary and at the optic nerve. In glaucoma, OCT-A was found to be useful in the non-invasive assessment of microvascular changes. Both optic nerve head and macular vessel density has been found to be reduced, with this correlating with both structural and functional changes. In diagnosing optic neuritis due to multiple sclerosis, OCT-A was found to enhance diagnostic accuracy when combined with OCT image analysis of the nerve fibre layer and ganglion cell complex.

This analysis highlights that OCT-A has the ability to non-invasively detect changes to blood flow within the retina across a range of conditions, although more research needs to be undertaken comparing OCT-A to the gold standard of fluorescein angiography. Over time, advancements to the software and hardware to improve the scan area and resolution will further improve its utility in clinical practice. OCT-A is likely to be the next leap and bound in our clinical toolset. ▲

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Collaborative care of patients with cataracts

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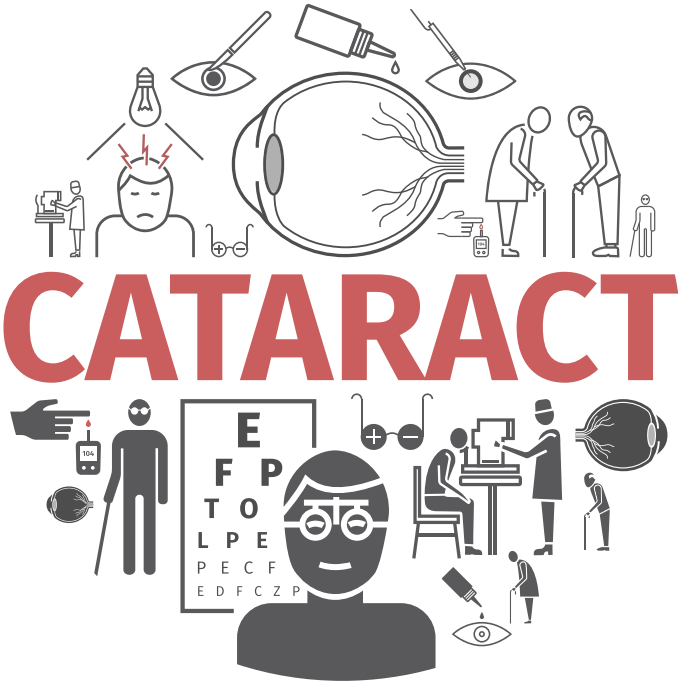

The public health system for eye care in Australia is under stress by increased demand which is resulting in long delays for non-urgent appointments in some areas. Patients seeking cataract surgery in particular can face long waiting times for assessment and subsequent treatment. There are two key factors contributing to this: 1) inadequate collaborative processes between the respective professions, and 2) the fact that referral triage is limited by the lack of clinical information supplied.

Cataract surgery volume in Australia

Cataract surgery was the most common procedure for admissions from all elective surgery waiting lists in 2017–18,[1] increasing by around 20 per cent since 2010-11. Medicare data for 2018-2019 shows 180,669 cataract surgeries in Australia and this figure does not include most procedures performed through public hospitals.

The reported national median waiting time for cataract surgery in 2018 in the public system was 85 days,[1] however there is marked regional variations, with waiting times reported to be as long as 301 days in Tasmania. This wait time data does not take into account the waiting time for a public hospital clinic appointment for clinical assessment before a patient is wait-listed for surgery (the 'wait for the wait'). For example, one study involving two metropolitan hospitals in Sydney in 2017 showed that two thirds of patients referred for cataract surgery were yet to have their initial hospital appointment one year after the referral was sent.[2]

Deciding when to refer for surgery: Is it 6/12 acuity?

Cataract assessment and referral is a daily part of optometric clinical practice, yet clearly defined state or national referral guidelines are currently lacking for the public system. The vision standards for driving are a de facto standard, however it is important to note that the emergence of manifest hyperopia can coincide with the development of early cataract. This has been shown to result in the need to wear distance glasses for the first time for up to 30 per cent of people older than 60 years.[3] Thus, while minimal use of glasses is the usual goal of cataract surgery, the need to wear glasses that was not present previously is not the primary reason a person should be referred to the public system.

While the timing of surgical intervention itself is frequently not crucial to a successful outcome (apart from very advanced cataracts) other factors in addition to best corrected visual acuity (BCVA) need to be considered when deciding when to refer. In particular: the patient's reported symptoms of reduced contrast sensitivity, impaired binocular function and glare sensitivity need to be evaluated, and put in context with the consequent impact on everyday visual function, such as the ability to drive both legally and safely.[4] The patient's ability to perform other activities of daily living, with the resultant impact on quality of life, mood and—importantly—the risk of falls are also key factors that need to be evaluated and considered.[5]

A study of referral characteristics of Australian optometrists for cataracts in 2013[6] indicated that the vast majority reported use of visual acuity, glare, driving and patient-centred hobbies as criteria for referral. An ongoing study of referrals to a public hospital eye clinic in Sydney suggests that while this information may be collected, it is not included in referrals (see below).

As well as these factors, the patients desire to undergo surgical intervention as well as the need to manage ocular comorbidities needs to be considered. This includes angle closure risk as well as improving fundus assessment for ocular conditions such as age related macular degeneration, as well as to address the risk of falls. The endpoint of this consultation process is to support the patient in reaching a decision to seek advice about surgical treatment at the appropriate time.

Deciding where to have surgery: public or private

In the Australian public health system, cataract surgery is provided at no cost to the patient. Factors valued by patients electing to have the surgery performed in the private sector include: selecting the desired specialist, less waiting times for appointments and surgery and additional options with IOLs, particularly extended depth of focus IOLs or lower thresholds for astigmatism correction.

A 'shared decision-making approach' should be adopted[7] when supporting the patient making this decision. This involves the primary health care professional being able to introduce the two options succinctly and accurately and conveying information that compares the differences between the two options. As a result, it is important that optometrists work closely with their local GPs to ensure that this information is consistent and readily available.

Research suggests that while cost is the primary consideration, waiting time is also a key factor, with the choice of doctor, or the involvement of training registrars of less concern.[8,9]

Reducing waiting times could prevent vision loss

A recent study from the UK suggested that around 22 patients per month permanently lose vision while on hospital wait lists.[10] The initial data from the Centre for Eye Health (CFEH) and Prince of Wales Hospital (POWH) collaborative clinic suggests that this same risk exists in Australia. Around three per cent of patients triaged as 'non-urgent' based on the supplied referral information needed prompt further treatment or assessment, following their eventual initial consultation at CFEH.

Current state of public hospital referrals

To facilitate the setting of appointments within an appropriate time frame, referrals need to contain adequate clinical information to enable accurate triage.

The CFEH and POWH eye clinic's ongoing study has so far reviewed over 500 prospective, sequential referrals to the POWH eye clinic. Forty per cent of all referrals to the eye clinic were from optometrists with a similar number from GPs. Only eight per cent of all referrals specified the degree of urgency, and over 40 per cent of all referrals were for cataract assessment.

While the ocular assessment details from optometry referrals were better than those from GPs, in total 34 per cent of referrals for cataract assessment did not report the patient's best corrected visual acuity (BCVA) and very few reported any information about the presence of glare symptoms, or the impact on driving or hobbies. One referral from an optometrist contained only: 'Dear Doctor, please assess for cataract surgery' with no other information provided.

As well as the quality of cataract referrals, the nature of other conditions being referred to the public eye service also needs to be refined. The main outcome of the POWH/CFEH clinic was reduced waiting times, however, results also showed that over 40 per cent of patients could be managed without subsequent referral to the hospital clinic though either CFEH/optometrist collaboration, or through follow-up in the community.

A surprising finding was that 10 per cent of patients referred specifically for cataract assessment, did not either want or require surgery at the time of CFEH assessment. Even so, this is a lower rate than has been previously published where one half of referrals for cataract assessment were not listed for surgery.[2]

Proper cataract referrals

Appropriate referral refinement can be expected to substantially reduce the waiting time for public hospital eye clinic appointments, with the consequent reduction in the total time waiting for cataract surgery, when it is required.

Approaches to improve public eye clinic cataract referrals

1) Establish clear guidelines as to when and how to refer a patient to the public system.

Some public eye clinics such as at Westmead and the Royal Victorian Eye and Ear Hospitals (RVEEH) have published defined criteria in their referral guidelines. These also include associated internal recommended triage time frames.[11,12]

One referral from an optometrist contained only: 'Dear Doctor, please assess for cataract surgery' with no other information provided.

The use of standardised referral forms will also assist with this process. A trial utilising a proposed state-wide referral form template is currently underway in NSW.[13] This includes guidelines that encourages that both a GP and optometric assessment report are supplied with a referral, and in the case of the optometrist referral, that recent best corrected acuity and refraction are supplied, if possible in conjunction with the previous spectacle correction.

A cataract clinical care standard from the Australian Commission on Safety and Quality in Health Care will be finalised soon, and this also addressed these issues. The current version, available for comment, is available at the time of writing at https://www.safetyandquality.gov.au/our-work/clinical-care-standards/cataract-clinical-care-standard.

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Cataract collaboration

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2. Rejecting referrals that do not meet referral criteria

Recent NSW Ministry of Health policy supports public hospital eye clinics that return referrals to the referrer when they do not contain adequate information to enable appropriate triage.

3. Intermediate-tier assessment of patients referred for consideration of cataract surgery incorporating telehealth

An alternative option to rejecting inadequate referrals involves initial patient assessment at an associated optometric clinic with tele-ophthalmology oversight. This model enables a uniform approach to assessment and referral for surgery based on defined protocols. It is also likely to assist in detecting other causes of vision loss, especially when referrals do not come from optometrists, thereby mitigating the potential for vision loss through the worsening impact of ocular co-morbidities during the wait for assessment within the public hospital system. Variations of this model are currently being investigated in a number of different locations in Australia, including at CFEH.

Conclusion

The Optometry Board of Australia defines collaborative care as 'when the care of a patient is provided by two or more health practitioners, each practising within their sphere of expertise in consultation with the patient.' With regards to patients with cataracts in particular, there is a pressing need for improved collaboration between optometrists, GPs and public eye clinics to achieve better access, quality, satisfaction and efficiency for patients. It is recommended that optometrists review how and what they are currently referring to public eye clinics to ensure that they are providing adequate information and that they work more closely with their local GPs to ensure the right patient is seen by the right practitioner at the right time. ▲

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Following the

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The Australian Government through the Department of Human Services presents items listed on the Pharmaceutical Benefits Scheme (PBS) for statistical analysis. Item codes corresponding to those drugs supplied by optometrists can be entered into the Medicare Australia website.[1] Statistics are generated for both the PBS and Repatriation PBS (RPBS–items supplied to war veterans). The statistics can be generated as a volume of items as services or as a value of benefit in a dollar amount that has been processed by Medicare Australia. The statistics presented by Medicare Australia refer only to paid services that are processed from claims presented by approved pharmacies.

The statistics presented are from the most current reporting period which is July 2018 through June 2019. Table 1 (page 28) is a list of drugs prescribed by optometrists and dispensed by pharmacy in decreasing order of amount dispensed.*

What do these statistics mean to Australian optometrists?

Australian optometrists began therapeutic medication prescribing in the year 2000 and yet little information is available about optometric pharmaceutical trends. Year-on-year, there will be an increase in optometrists who are eligible to prescribe therapeutic medications. This is primarily due to all optometrists now graduating with optometry degrees in Australia who are eligible to prescribe therapeutic medications.

According to the Optometry Board of Australia there are 5,781 registered optometrists for the 2018/19 year and of those registered, 62.8 per cent

script Optometric trends in prescribing therapeutic medications

are endorsed to prescribe therapeutic medications.[2] On average, each therapeutically-endorsed optometrist writes 31 prescriptions each year not including those written for dry eye treatments or for chloramphenicol. This suggests that the core function of optometrists would still seem to be as prescribers of refractive corrections.

Glaucoma preparations, especially latanoprost, dominate the prescriptions written by optometrists. One could argue that optometrists are increasing their co-management of patients with glaucoma with ophthalmology.[3] There is also the possibility that glaucoma patients are being monitored solely by optometrists due to the availability of optometrists in the community and the difference in cost to attend an optometrist versus an ophthalmologist.[4]

A literature review searching for articles with glaucoma and co-management or shared care in the title or abstract, provided evidence of an equal quality of care by optometrists compared to ophthalmologists[5] as provided by hospital-based optometrists on those patients with stable glaucoma.

According to Department of Health (DOH) Australia's Future Health Workforce – Ophthalmology study of 2018, the demand for ophthalmology services is estimated to grow at 2.8 per cent per year to 2030.[6] The results of the projections reveal an undersupply of ophthalmology specialists throughout the entire projection period. The study also found that the shared-care models developed between ophthalmology and optometry reduced waiting times for new patients.

Optometrists can assess vision, manage refractive errors through issuing glasses and screen for, diagnose and treat serious eye diseases; allowing ophthalmologists to focus on surgical management of eye disease. In some situations, optometrists provide routine follow-up for stable eye disease, referring back to ophthalmologists when needed. Patients benefit through accessible, high-quality eye care and timely follow-up, thereby reducing the risk of adverse consequences of chronic diseases.[7]

RANZCO asked the DOH to analyse these models of care to determine the effects of the transfer of work to optometrists, and implications of demand for ophthalmology services. Upon investigation of the Medicare data, while there is much higher growth in optometry services, there has been no noticeable halting or decline in growth of ophthalmology services – possibly due to taking on the more complex cases, improved patient accessibility and an increased aging population with an attendant increased incidence of eye disease.

While the DOH also found optometry items billed to Medicare are growing rapidly, there is no evidence of task

Continued page 28

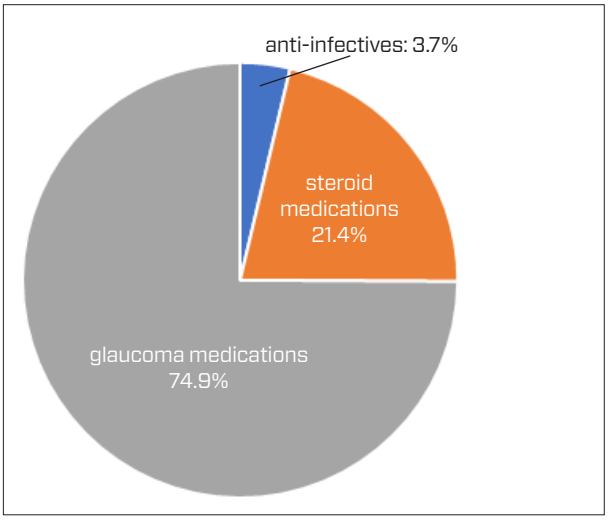

Figure 1. A comparison of therapeutic medications prescribed by Australian optometrists by medication action for the period July 2018 through June 2019

Prescribing trends

From page 27

substitution as there is no recent or noticeable decline in the growth rate for ophthalmology services. In addition, the selected Medicare items related to glaucoma services are only a small percentage of overall ophthalmology services. From this information there is certainly more scope for optometry to provide nearly all of the care for stable glaucoma patients.[8,9]

Conclusion

There is certainly more opportunity for optometrists to use their therapeutic skills in the diagnosis and management of eye disease. The fact that Australian optometrists prescribe mostly anti-glaucoma treatments demonstrates the amount of glaucoma management they are undertaking possibly reflecting a confidence in their management skills of this chronic disease. The key to providing more therapeutic eye care to Australians may well be in the promotion of the skills of optometry. The skill set of optometrists could be promoted to both medical practitioners and the public alike to present the changing face of optometry as a therapeutic profession. More articles on the diagnosis and management of therapeutic conditions by optometrists would also provide an insight into this changing pathway of the profession.

*Chloramphenicol is excluded as the item code is not exclusive to optometrists. Chloramphenicol may also be prescribed by nurse practitioners, midwives and medical practitioners. Dry eye therapies are also excluded because prescriptions for these therapies are usually written for those on income assistance and therefore not truly reflecting the whole dry eye therapy market. ▲

Drug	Number of prescriptions dispensed
Latanoprost	30,223
Latanoprost + Timolol	9,762
Fluorometholone	9,678
Fluorometholone Acetate	7,028
Brimatoprost + Timolol	6,708
Brimatoprost	6,666
Brinzolamide	5,679
Prednisolone Acetate + Phenylephrine	5,476
Travaprost + Timolol	4,807
Brimonidine	4,506
Timolol	4,485
Dexamethasone	3,222
Travaprost	3,371
Brinzolamide + Brimonidine	3,101
Tobramycin	2,645
Dorzolamide + timolol	2,330
Brimonidine + timolol	2,008
Brinzolamide +Timolol	1,795
Tafluprost	1,255
Dorzolamide	1,016
Aciclovir	1,143
Betaxalol	529
Pilocarpine	452
Ciprofloxacin	270
Ofloxacin	235
Framycetin Sulfate	48
Gentamicin	39
Total prescriptions dispensed	113,001

Table 1. Drug and the amount prescribed by Australian optometrists for the period July 2018 through June 2019

1. Australian Government Department of Health. Pharmaceutical Benefits Scheme 2019 [PBS Expenditure and Prescriptions Report 1 July 2018 to 30 June 2019]. Available from: https://www.pbs.gov.au/info/statistics/expenditure-prescriptions/pbs-expenditure-and-prescriptions-report.
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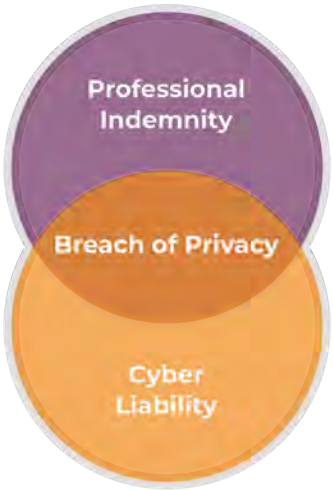

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CEQUA™ ciclosporin 900 microgram/mL eye drops ampoule. **Indications:** Increases tear production in patients with moderate to severe keratoconjunctivitis sicca (dry eye) where prior use of artificial tears has not been sufficient. **Contraindications:** Hypersensitivity to the active substance or excipients. Active or suspected ocular or peri-ocular infection, malignancies or premalignant conditions **Precautions:** *Potential for eye injury and contamination:* avoided by not touching the eye or other surfaces with the ampoule tip. *Contact Lenses:* remove contact lenses prior and reinsert 15 minutes after administration. Careful monitoring of patients with severe keratitis is recommended. *Infections:* resolve existing or suspected ocular or peri-ocular infections before initiating treatment and if an infection occurs during treatment, withhold temporarily until infection resolves. *Effects on the immune system:* may affect host defenses against local infections and malignancies so regular examination of the eye(s) is recommended, e.g. at least every 6 months, when used for long periods. *Paediatric use:* safety and efficacy is not established below the age of 18. **Pregnancy:** Category C **Adverse effects:** *Very Common and Common:* instillation site pain, conjunctival hyperemia and punctate keratitis. **Dosage and administration:** one drop twice daily (approximately 12 hours apart) into the affected eye(s). Response to treatment should be reassessed at least every 6 months. Can be used concomitantly with artificial tears, with a 15-minute interval between products. **Storage:** Store below 25°C. Do not freeze. Store the ampoules in the original foil pouch. Protect from light. **Date of preparation:** May 2020.

▼ This medicinal product is subject to additional monitoring in Australia. This will allow quick identification of new safety information. Healthcare professionals are asked to report any suspected adverse events at https://www.tga.gov.au/reporting-problems.

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Adverse events may be reported to Sun Pharma by either email: adverse.events.aus@sunpharma.com or phone: 1800 726 229.
Date of preparation: May 2020. CEQ05/2020opa1

Reference: 1. CEQUA™ Product Information. 2020 Feb, 2020; Available from: Sun Pharma by calling 1800 726 229.