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December 2019

ADVANCING THERAPEUTIC OPTOMETRY

Christmas Eye

It's not what you think

Member-submitted case reports

OCT for VMT by Rose Huang

Thygeson's superficial punctate keratopathy by Anna Delmadoros

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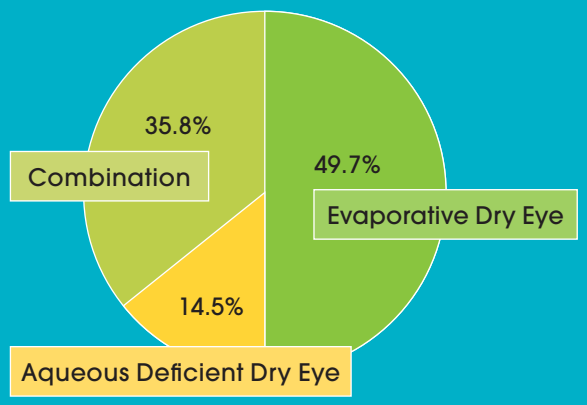

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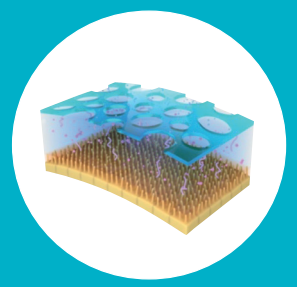

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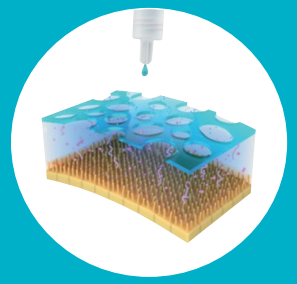

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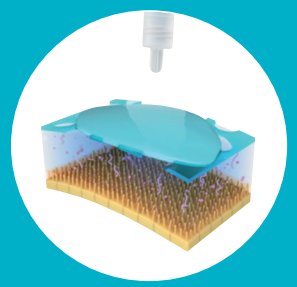

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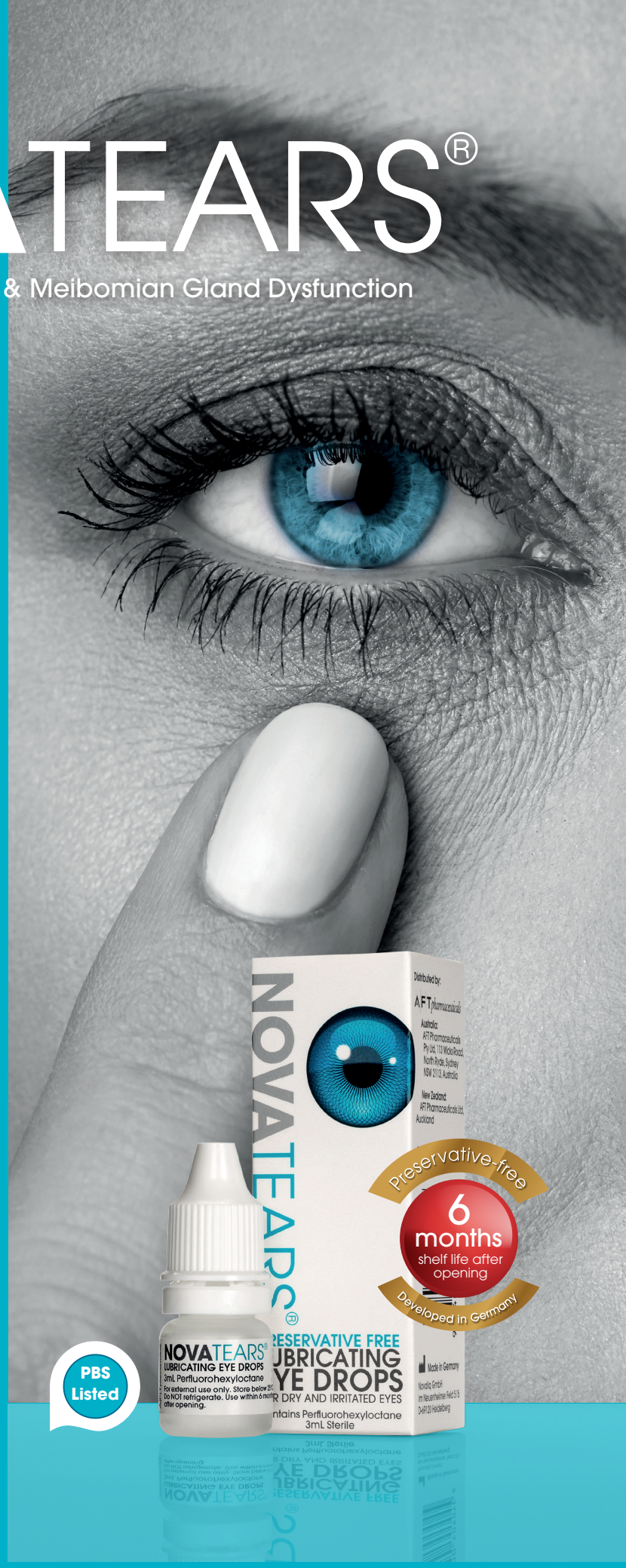

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December 2019 Anterior eye

From the Editors

As the diverse range of topics covered in this issue of *Pharma* plainly shows, the practice of optometry is full of surprises.

Consider Rob Holloway's article on 'Christmas Eye,' probably one of the most unusual corneal conditions in the field of optometric health. A form of acute toxic keratitis that is found primarily in Australia's south east from November to February, Christmas Eye, as Rob Holloway explains, is associated with pain that has reached 'folkloric levels.'

Another surprising article, by Debra Gleeson, traces the increase in reported incidents of tattoo-related uveitis (TAU) and may lead you to take a professional interest in your patients' tattoos. It may also provide an opportunity to call upon your local tattoo parlour for a professional consultation.

In this issue, we also commemorate a milestone with the publication of the 20th annual contact lens prescribing trends survey results by Efron, Morgan and Woods.

Finally, this issue marks the first time that we have published member-submitted case reports. In response to our call for papers at the beginning of the year, Rose Huang presents an informative review of the use of OCT to diagnose bilateral dynamic vitreomacular traction. And Anna Delmadoros shares an illuminating case report on Thygeson's superficial punctate keratopathy.

The editors strongly believe that *Pharma* serves the members of Optometry Australia best when it offers a place for them to exchange ideas and to pursue learning opportunities at a pace conducive to their busy schedules.

We invite all the members of Optometry Australia and the New Zealand Association of Optometry to submit their own case reports for consideration in 2020.

Send a 200-word explanatory summary of your case report to pharma@optometry.org.au.

This issue of Pharma offers 6 (4T) CPD points.

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Non-arteritic anterior ischemic optic neuropathy

An atypical presentation

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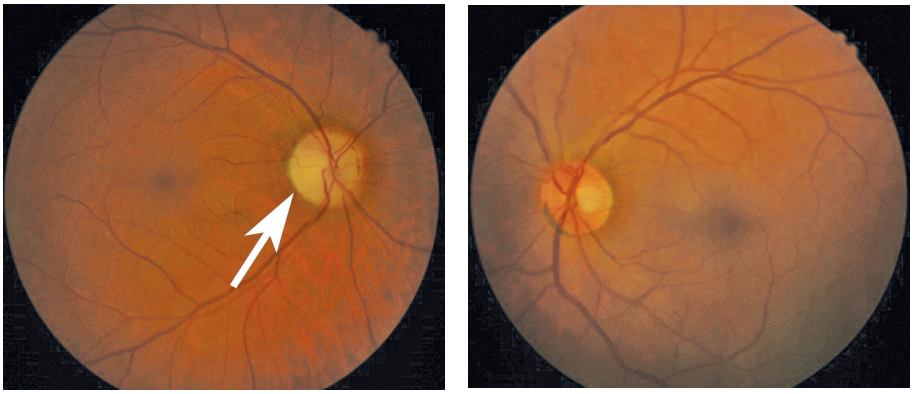

Figure 1. Note the inferior-temporal optic nerve pallor in the right eye (arrow). The left optic nerve was normal.

Non-arteritic anterior ischemic optic neuropathy (NAION) is the most common type of acute optic neuropathy in people over 50 years of age, and it affects every two to 10 individuals per 100,000.[1] Approximately 95 per cent of all ischemic optic neuropathy cases are NAION.[1] NAION results from an ischemic damage of the anterior portion of the optic nerve head secondary to infarction of the short posterior ciliary artery, leading to axonal oedema and consequently painless vision loss.[2]

CASE REPORT

A 56-year-old male patient attended the practice in October 2017 with a two-week onset of blurry vision in his right eye. The patient described his vision as 'only bottom-half visible.' He reported no flashes of light, floaters, or any change in his physical or mental health status. He did not take regular medications and was a non-smoker who drunk alcohol occasionally.

On examination, his vision was 6/24 in the right eye with no improvement with pinhole testing. The left eye achieved 6/6. A right relative afferent pupillary defect was detected; red cap testing showed desaturation in the same eye. Anterior slitlamp biomicroscopy evaluation was unremarkable. A dilated fundus examination revealed right optic disc pallor inferior-temporally while the left optic nerve appeared normal (Figure 1). The maculae and retinal periphery examination was normal in both eyes.

The patient was urgently referred to a medical retinal specialist for assessment and evaluation of the possible optic nerve neuropathy. Optical coherence tomography (OCT) (Figure 2) showed diffuse retinal nerve fibre layer thinning in the right eye, with a right superior altitudinal visual field defect on central 24-2 threshold testing. An MRI scan of the brain with angiography and venography demonstrated no intracranial or orbital space occupying lesion, aneurysm or demyelination. A blood work-up did not detect any systemic infection or inflammation.

The patient was subsequently referred to a neuro-ophthalmologist who performed antibody testing for myelin oligodendrocyte glycoprotein (MOG) and neuromyelitis optica (NMO-IgG) to rule out autoimmune conditions. On the basis of difficulty in confirming diagnosis retrospectively, a provisional diagnosis of non-arteritic anterior ischemic optic neuropathy (NAION) was made and a follow-up in eight weeks was scheduled.

At the follow-up appointment, the neuro-ophthalmologist confirmed negative NMO-IgG and MOG antibodies. The vision in the right eye had improved to 6/18 and the visual field defect showed mild improvement. After considering the patient profile and all clinical findings, the patient was diagnosed with NAION. He was discharged from the neuro-ophthalmologist and a 12-month review with the optometrist was scheduled.

The patient returned to the practice in March 2019. His vision in the right eye remained at 6/18 with stable optic nerve head appearance (Figure 3). The left eye remained normal. The patient was advised to seek prompt assessment and treatment if any form of vision loss or visual disturbance is observed in either eye.

Discussion

In this case report, the patient described his vision loss as a 'blur of the top half of his vision,' which is a typical way of describing the symptoms of NAION.[3] Findings of a relative afferent pupillary defect and red cap desaturation, though non-specific, can indicate unilateral or bilateral asymmetric optic neuropathy. NAION typically causes (inferior) altitudinal and arcuate visual field defects and does not lead to dyschromatopsia due to sparing of central nerve fibres.[3,4]

Diffuse or sector optic disc oedema is observed during the acute phases of

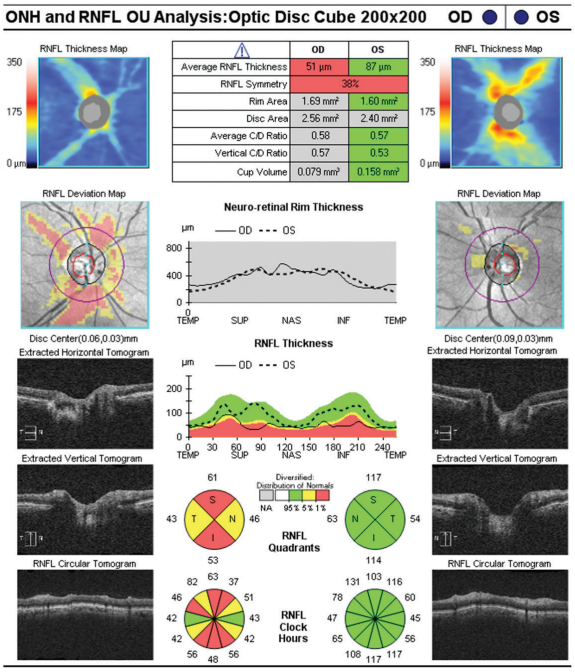

Figure 2. Diffuse retinal nerve fibre layer thinning in the right eye. The left eye appeared normal.

NAION, which is important for clinical diagnosis. Within the next two to three months, optic atrophy occurs.[3] In our case, the patient waited too long to seek help since early intervention using steroids or neuroprotective agents may improve visual performance,[5] and it also made the diagnosis challenging.

The exact cause of the ischemic event in NAION remains unknown. Risk factors include a small/'crowded disc' with small cup-to-disc ratio, which has been referred as 'structural disc at risk' for NAION (the patient presented here does not have the typical small optic disc/disc at risk); optic disc drusen which may disrupt blood flow causing ischemia;[6] and other systemic conditions such as nocturnal hypotension, hypertension, diabetes, hypercholesterolemia, sleep apnoea and smoking may increase the risk of optic nerve head hypoperfusion leading to NAION.[2,6]

The most critically important differential diagnosis is arteritic anterior ischemic optic neuropathy secondary to giant cell arteritis, which is life threatening as well as the significant risk of visual loss to the other eye. The other main differential diagnosis is optic neuritis secondary to multiple sclerosis. In these instances, a comprehensive systemic evaluation, including neuroimaging, is essential to make an accurate diagnosis.

NAION stabilises within two to three months. The visual prognosis of NAION is mostly guarded, with studies reporting vision improvement by three lines in more than one-third of patients.[3,7] The visual field defect is unlikely to show major improvement. The rate of recurrence in the same eye is less than five per cent,[3] and the possibility of fellow eye involvement is approximately 15 per cent over five years.[8] When the fellow eye develops NAION, the impact of vision and visual field loss cannot be predicted from the prognosis of the previously affected eye.[9]

NAION is a common sight-threatening disease with unknown pathophysiology. When a patient presents with atypical visual complaints, optometrists need to be proficient and proactive in conducting a targeted history, performing relevant entrance tests, and be aware of the life-threatening differentials. This case report has illustrated the importance of multi-disciplinary collaborative assessment and management in providing best patient care and delivery. This patient will require an ongoing 12-month review in the future due to significant risk to the fellow eye.

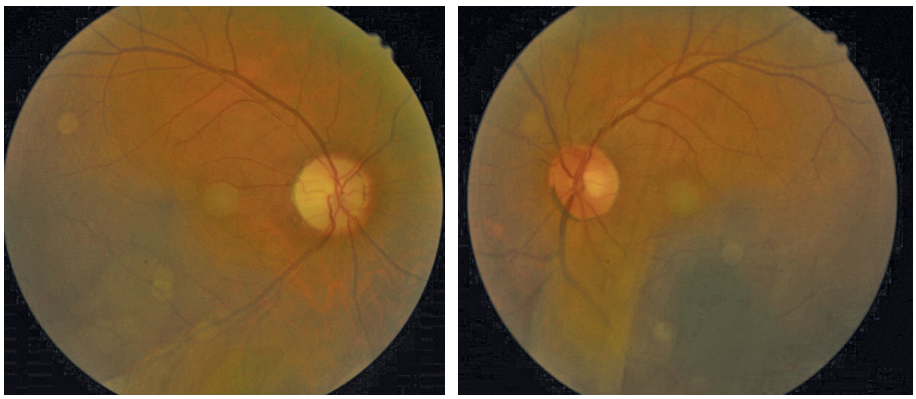

Figure 3. Optic atrophy in the right eye. The left optic nerve remained normal.

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OCT for VMT

Ocular coherence tomography for management of vitreomacular traction

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This original case report was submitted by fellow Optometry Australia member Rose Huang in response to our nation-wide call for papers.

With the introduction of optical coherence tomography (OCT) and recent studies, we have been able to better visualise and understand the vitreomacular interface and therefore provide a better diagnosis, management and prognosis for our patients.[1]

When the anterior vitreous pulls, it can cause asynchronous weakening in areas of attachments which may ultimately lead to anomalous posterior vitreous detachments. These can often result in vitreomacular traction (VMT) and other vitreoretinal diseases. VMT is characterised by the incomplete vitreous detachment and persistent macular adherence.[2]

Static anterior traction can result in foveal elevation or distortion, yet patients are often asymptomatic. It is the dynamic traction which is associated with ocular rotations and eye movements which ultimately lead to the most pronounced symptoms. These eye movements are often related to accommodation and downward head posture.[3]

It is interesting to note that the tractional effects are largely determined by the strength and size of the residual vitreoretinal adhesion, with a smaller diameter associated with higher tractional stress and consequently greater foveal involvement and retardation. Generally, a vitreofoveal adhesion ≤ 500 µm is more symptomatic and commonly associated with a microhole, lamellar hole and full-thickness macular hole.[4] In patients with concurrent diseases such as wet age-related macular degeneration (AMD) and diabetic clinically-significant macular oedema (CSME), VMT may be exacerbated.[5]

A 66-year-old Caucasian female presented complaining of discomfort and an intermittent bilateral central scotoma blur with and without glasses. The symptoms had started months ago, however had gradually worsened, and consequently she was now having difficulty with crocheting, knitting and reading.

Examination through a dilated pupil revealed pattern macular dystrophy. Otherwise posterior health was unremarkable. Pattern macular dystrophy presents as bilateral, multiple, yellow vitelliform lesions and are autosomal dominant in nature. It typically presents between the ages of 40–60.[6]

Generally, there is minimal effect on visual acuity with most patients able to attain 6/6–6/12 vision, however in certain cases central metamorphopsia may be observed on the Amsler Grid, however this is more commonly associated with non-multifocal pattern dystrophies.[7] Therefore symptoms were not associated with underlying pattern dystrophy.

A symptom diary was administered so she could better identify the nature of her problems. Upon review, she could confidently pinpoint that accommodation and near-tasks were the causative factors. She graded her symptoms as severe and the only management which worked was rest breaks or sleep.

As her symptoms were elicited by near-based work, an OCT was performed prior to 30 minutes of crocheting (Figures 1A, 2A, 3A and

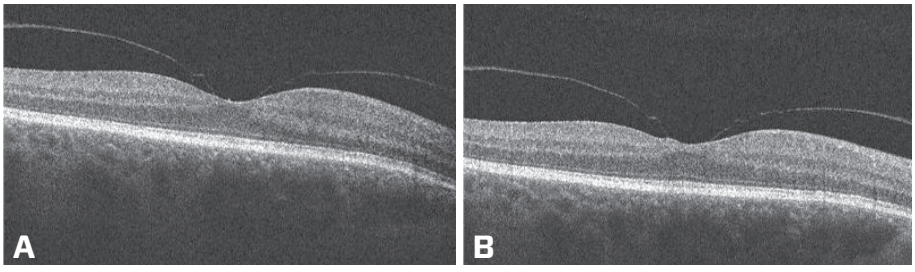

Figure 1. A: Right eye before near work. B: Right eye 30 minutes post near work.

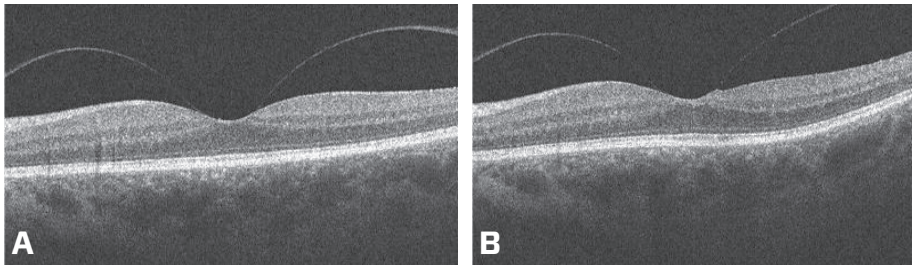

Figure 2. A: Left eye before near work. B: Left eye 30 minutes post near work.

4A) and then repeated post crocheting (Figures 1B, 2B, 3B and 4B). Although the patient was told to crochet for 30 minutes, within 10–15 minutes she had largely given up.

In early stages, it is not unusual for the outer retina to appear intact. Figures 1B and 2B show moderate inner retinal layer distortion, however the outer retina is largely preserved.

The OCT scans show subtle changes in the macular thickness profile and foveal elevation following near work (Figures 3B and 4B). It can be argued that there are already early changes in the outer retina morphology, which increases the risk for a microhole, lamellar hole or macular hole.

Diagnosis

The patient was diagnosed with bilateral dynamic VMT induced by accommodation.

The prevalence of VMT without the incidence of a macular hole has been estimated as approximately 22.5 cases per 100,000, with an incidence of 0.6/100 000 persons per year. The mean age of patients is estimated around 65–70 years, with a predominance of females.[8]

Once a mydriatic has been instilled for dilation, accommodation will be blocked and therefore symptoms cannot be detected. A 10-2 was performed, however as expected, results were within normal limits, as it is difficult to induce symptoms in an upright situation. There was, however, distortion on the Amsler Grid in both eyes.

Management

As visual acuity was excellent, an initial conservative approach was chosen. Ultimately as the anterior interface detaches from the site of the vitreofoveal attachment, symptoms should resolve. Not surprisingly, the patient returned six weeks later and requested to go ahead with the vitrectomy and consequent cataract surgery. Post-operation, she was able to conduct near-based work again with ease.

In a recent study, spontaneous resolution of VMT occurred in 21.4 per cent of patients while 7.7 per cent of subjects developed a lamellar or full-thickness macular hole after a mean follow-up of 11.4 ± 12.6 months.

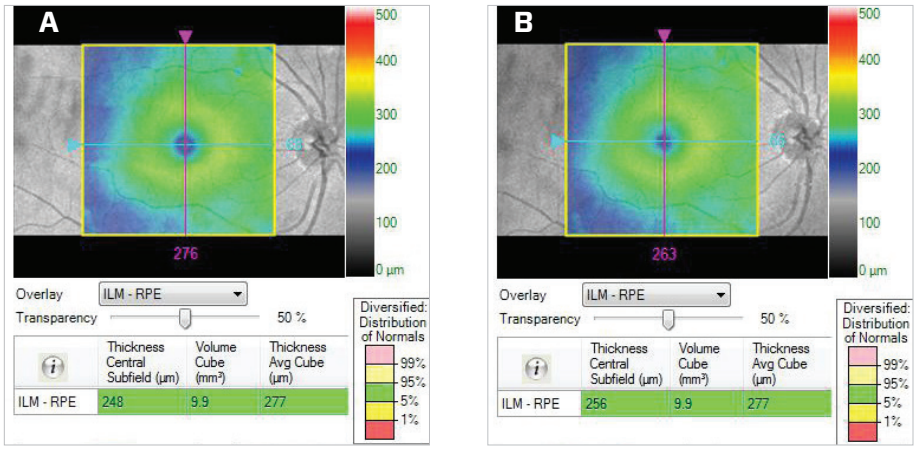

Figure 3. A: Right eye macular thickness analysis *before* near work. B: Right eye macular thickness analysis *after* near work.

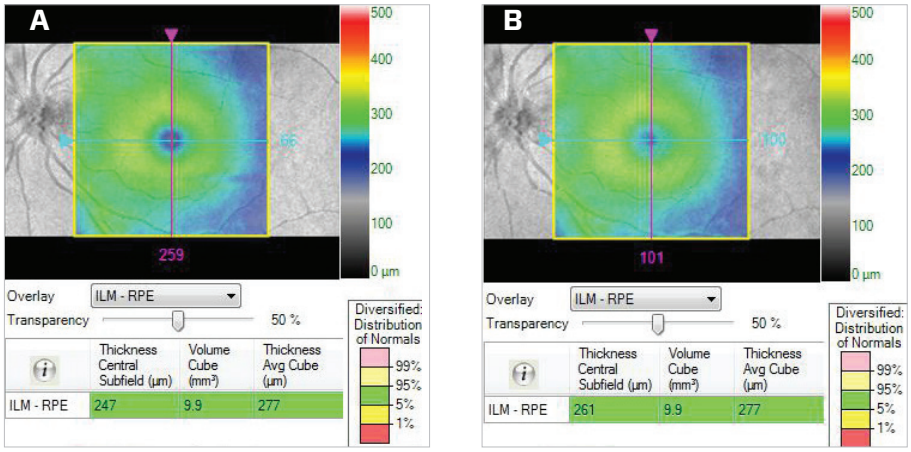

Figure 4. A: Left eye macular thickness analysis *before* near work. B: Left eye macular thickness before *after* near work.

The baseline OCT may predict progression to a full-thickness macular hole, with patients presenting with intraretinal cysts, clefts and foveal detachment more likely to advance.[9] In these patients, the foveal detachment secondary to severe traction would cause a central scotoma and unfortunately lead to a drop in visual acuity and therefore surgical options of intravitreal injections or vitrectomy may be the more viable option.[10]

In conclusion, cases of transient blur after near-work should be further investigated for potential dynamic vitreomacular traction syndrome.

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Contact lens prescribing trends 2019

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20th ANNUAL SURVEY

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Efron, Morgan and Woods report on their 20th annual survey of Australian contact lens prescribing habits

The 20th annual survey of Australian contact lens prescribing was conducted during the first three months of 2019. The same format as in previous years was employed. An email was sent to all members of Optometry Australia with a link to a questionnaire, and a request that this be downloaded, printed and completed to provide details of the first ten patients fitted with contact lenses after receipt of the questionnaire. The survey was specifically designed to be straightforward to complete while capturing key patient information.

Practitioners were asked general questions about themselves. For each contact lens fitting, they were requested to complete the following details: date of fitting, new fitting or refitting, age and sex of patient, lens material, lens design, frequency of replacement, times per week of wear, modality (daily or extended wear) and care system. Practitioners were asked to return the photographed or scanned copies of the questionnaire by email.

Completed questionnaires relating to 502 contact lens fits were returned, which provides a sound basis for a meaningful analysis. Each fitting was given an annualised weighting based on the number of lenses fitted during the survey period and the time taken to complete the fits. This means that data generated by practitioners with a higher frequency of fitting contact lenses were afforded a higher weighting than those with a lower frequency of fits.

The discussion below will concentrate primarily on data relating to new lens fits, as opposed to refits. We believe that new fits are a more sensitive barometer of current patterns and future trends, whereas refits are more indicative of previous fitting behaviours.

In keeping with other markets around the world,[1] a majority of lenses (65

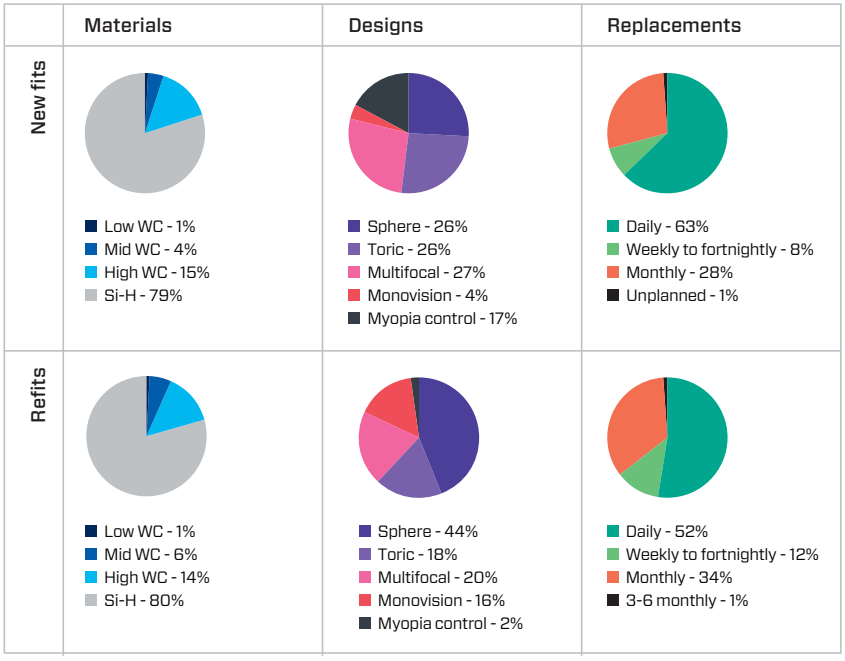

Figure 1. Detailed results for soft contact lens prescribing in the 2019 Australian survey (Si-H: silicone hydrogel; WC: water content).

per cent) were fitted to females. The average age of contact lens wearers at the time of fitting has increased over the past two decades, from 32 in 2000 to 37.0 ± 17.4 years this year. The age at fitting ranged from 1 to 89 years.

Soft lens materials and designs

Soft lenses are still the main type of contact lens fitted, accounting for 90 per cent of new fits; soft lenses have represented the vast majority of contact lens fits since our survey began two decades ago.[2]

Figure 1 is a composite of pie charts detailing the key findings of the 2019 survey in relation to soft lenses. Silicone hydrogels are still the dominant material, representing 79 and 80 per cent of materials prescribed as new fits and refits, respectively, with the balance comprising mainly of mid- and high-water content hydrogel materials.

The key categories of lens designs are spherical, toric, multifocal, monovision, coloured (tinted) and myopia control. Spherical and toric designs each represented 26 per cent of new fits (Figure 1).

Figure 2 shows trends in contact lens materials prescribed over the past two decades. It can be seen that there was a gradual increase in silicone hydrogel prescribing from 2000 to 2017, which has remained steady since then. The reason for the popularity of this highly oxygen-permeable material type is that it essentially eliminates hypoxic complications such as limbal and conjunctival redness, stromal oedema, corneal neovascularisation and epithelial microcysts.[3]

Multifocal designs (27 per cent of new fits) continue to be preferred to monovision (four per cent) for the correction of presbyopia. This trend, which has been evident since the turn of the century, largely can be attributed to improved multifocal lens designs. The fact that almost one-third of soft lens fits are for the correction of presbyopia highlights the importance of this growing demographic in modern day contact lens practice.

Coloured (tinted) lenses do not seem to be popular in Australia; in fact, no coloured lens fits were recorded in our 2019 survey.

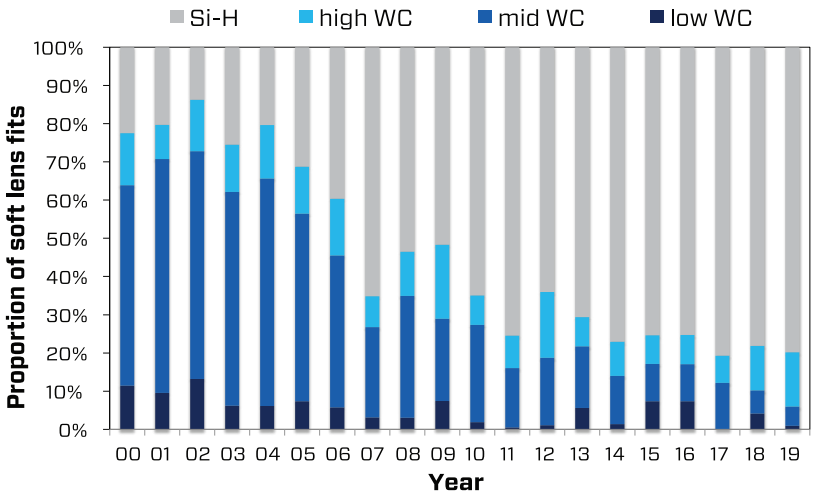

Figure 2. Proportion of all fits of various soft lens material types in Australia between 2000 and 2019 (Si-H: silicone hydrogel; WC: water content)

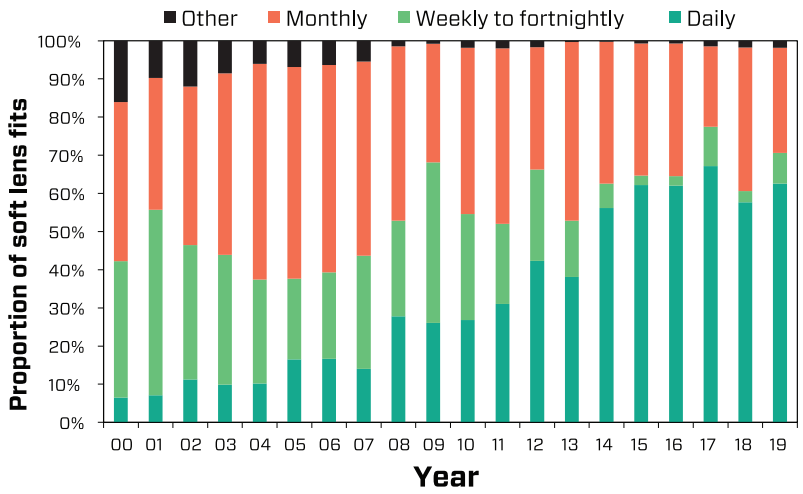

Figure 3. Proportion of all fits of soft lens lenses according to replacement frequency in Australia between 2000 and 2019

Myopia control lenses incorporate special designs for arresting the rate of progression of myopia.[4] This year saw a sudden surge of interest in prescribing for myopia control, with such lenses representing 17 per cent of new fits. This sudden increase may be related to the recent introduction into Australia of the MiSight lens (CooperVision), which is specifically designed for myopia control. As well, the considerable discussion and debate in the literature, at conferences and in online forums has undoubtedly fuelled interest in this modality of lens correction.

Soft lens replacement and wearing modality

Virtually all soft lenses are replaced daily (63 per cent) or monthly (28 per cent). Trends since 2000 in soft lens fitting according to replacement frequency are shown in Figure 3. Daily disposable lenses now dominate the Australian market, with the level of prescribing of this modality remaining relatively constant at above 60 per cent since 2015.

Extended wear lens fitting, almost exclusively with silicone hydrogel materials, has remained constant at under 10 per cent of all lens fits over the past decade, and has dropped to a low point of five per cent of all lens fits in 2019.

Multi-purpose solutions are now used almost exclusively by those wearing

Continued page 8

CL trends 2019

From page 7

reusable lenses, with this solution type representing 98 per cent of prescribed care regimens. The balance is peroxide systems.

Australia versus the world

We conduct annual contact lens fitting surveys in about 40 countries each year.[1] This provides an opportunity to benchmark against international colleagues, and this year we compare contact lens prescribing in Australian against world trends (the latter derived from 2018 data[1]) (Figure 4). Seven key categories of lens type are represented. The outer and inner rings display the Australian and world-wide fitting data,[1] respectively.

Perhaps the greatest difference revealed in Figure 4 is that daily disposable silicone hydrogel lenses – widely believed to be the most advanced lens type in terms of eye health – are prescribed at more than twice the rate in Australia (35 per cent) compared with the rest of the world (17 per cent). Australia has always been recognised as being at the forefront of contact lens prescribing, with Australian optometrists being 'early adopters' of new technologies. As well, the rate of rigid lens prescribing is higher in Australia than world averages.

Conclusions

The results of our 2019 survey confirm the ongoing high rate of prescribing of silicone hydrogel materials and daily disposable lenses in Australia. The sudden spike in prescribing of lenses for myopia control is perhaps the stand-out highlight of our 20th anniversary report; it will be interesting to see if this very high rate of prescribing of this lens type is sustained into the future.

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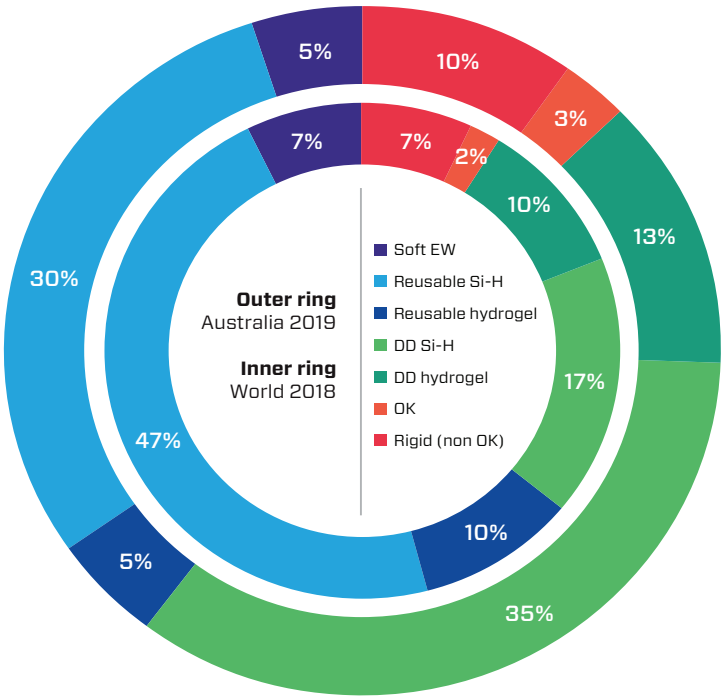

Figure 4. Percentage of all contact lenses prescribed in Australia (2019, outer ring) compared with the world (2018, inner ring). (EW: extended wear; Si-H: silicone hydrogel; DD: daily disposable; OK: orthokeratology).

CLINICAL AND EXPERIMENTAL
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Brimonidine is a drug that most optometrists would associate with treatment for glaucoma due to its action as an alpha 2 agonist. In Australia, it is available in both 0.2 and 0.15% concentrations and is branded as Alphagan. At these concentrations, brimonidine acts to increase aqueous outflow and decrease aqueous production. In a recent paper published in *Clinical and Experimental Optometry* authors Ackerman, Torkildsen, McLaurin and Vittitow present an alternate use of low-dose brimonidine: as a topical vasoconstrictor for the relief of ocular redness.

A red eye can be a result of a multitude of causes including allergy, infection, dry eye, contact-lens wear and exposure to environmental stimulants. In clinical practice, in order to treat a red eye, the cause should first be identified so that management can be appropriately targeted. When ocular redness, with no identifiable cause, becomes part of a person's baseline characteristics, over-the-counter

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*.

Low-dose brimonidine for relief of ocular redness: integrated analysis of four clinical trials

Summary and comment provided by Maria Markoulli
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vasoconstrictors or decongestants may be recommended in order to temporarily manage the appearance.

Vasoconstriction is achieved with drugs with alpha-adrenoceptor agonist activity (α-agonists) by binding to α-receptors on vascular smooth muscle. This acts to induce vasoconstriction, hence reducing the appearance of ocular redness. A limitation of this class of drugs is that their continued use results in tachyphylaxis, or tolerance, so that rebound redness results, particularly when the treatment is discontinued.

Vascular smooth muscle has two types of alpha-adrenoceptors: alpha1 (α1) and alpha2 (α2). While α1-adrenoceptors are the predominant α-receptor located on vascular smooth muscle, depending on the tissue and type of vessel, α2-adrenoceptors can also be found on smooth muscle. Different vasoconstrictors have differing affinity for each of these receptors. Phenylephrine, for example, is selective for α1 receptors, while naphazoline binds to both α1 and α2 receptors.

It is thought that tachyphylaxis relates to a reduction in the α1 receptor response as a result of chronic exposure to α1-agonists, with a subsequent rebound in redness due to loss of vascular tone. Brimonidine is a highly selective α2-receptor agonist, meaning that it is not susceptible to a reduction in α1 receptor response.

The authors of this study hypothesised that, given its affinity for α2 receptors which are expressed primarily in veins, brimonidine would be less likely to result in rebound redness and tachyphylaxis. In the USA, a low-dose version (0.025%) of brimonidine has received approval for ocular redness by the Food and Drugs Authority (FDA). The authors of this study therefore set out to report on the efficacy and safety profile of low dose brimonidine for idiopathic ocular redness.

The authors collated the data of four studies that included participants who were prescribed low-dose brimonidine four times a day for 28 days (three studies) or a single dose followed by four times a day for five days. The two studies that explored the efficacy of brimonidine were randomised and double-masked, with participants randomised to the brimonidine or the vehicle. The potential for tachyphylaxis was determined by evaluating the change from pre-instillation to five-minutes post-instillation on day 15 compared to day 29 of the study. Safety was evaluated with plasma collections at various time points post-instillation in order to establish brimonidine plasma concentrations. Safety was also determined by monitoring visual acuity, the ocular surface, fundus, intraocular pressure and vital signs. Rebound redness was also measured as part of the safety assessment, as was comfort.

When the investigators assessed ocular redness, it was found to be significantly lower to that of the vehicle alone at all post-instillation time-points and that was the case both when the clinician assessed the ocular redness and when the participants assessed their own ocular redness.

Tachyphylaxis was not apparent and rebound redness was rare. Adverse events reported were low and included a reduction in visual acuity in four per cent of cases on brimonidine and 4.3 per cent of cases on the vehicle and conjunctival hyperaemia in 2.6 and 2.9 per cent, respectively. There was no difference in the reported comfort between the drops or between other measures. Analysis of plasma samples in the pharmacokinetic study showed concentrations of brimonidine were below the lower limit of detection at all time points.

The authors concluded from this work that low-dose brimonidine reduces ocular redness without tachyphylaxis over a 28-day instillation process, and with a low risk of both ocular and non-ocular adverse events. Brimonidine 0.025% is currently not available in Australian pharmacies. Until it is commercially available, most ophthalmic compounding pharmacies in Australia make the 0.025% mixture. (See list of ophthalmic compounding pharmacists on page 28 of this issue).

Ackerman, Torkildsen, McLaurin et al. Low-dose brimonidine for relief of ocular redness: integrated analysis of four clinical trials. *Clin Exp Optom* 2019; 102: 131–139

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Thygeson's superficial punctate keratopathy

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This original case report was submitted by fellow Optometry Australia member Anna Delmadoros in response to our nation-wide call for papers.

Thygeson's superficial punctuate keratitis (TSPK), first described by Phillips Thygeson in 1950,[1] is a relatively uncommon, recurrent and chronic non-infectious corneal condition that is typically bilateral and asymmetric.[1-5] It has no sex predilection and no age bias.[2,6] The prevalence of the condition is unknown and the underlying aetiology remains controversial.[1-5]

Diagnostic features of TSPK are multiple, mildly elevated, whitish/grey granular or stellate intraepithelial opacities, occurring predominantly in the central cornea, in the absence of accompanying oedema or conjunctival involvement.[1-4] Symptoms vary in degree and include photophobia, foreign body sensation and lacrimation.[2] Visual acuity may be slightly reduced depending on the density and location of the opacities.[2,3]

A hallmark feature of TSPK is the presence of active and quiescent phases, which vary in duration and frequency.[1-3] Symptomatic exacerbations lasting weeks to months appear to be sporadic and the triggers unknown.[1,2] During periods of remission (months to years), the cornea is essentially void of any signs and the patient is completely asymptomatic.[1,3] TSPK is a self-limiting condition that is reported to resolve spontaneously on average 3–7.5 years after the first presentation,[2-5] however course durations of up to 41 years have been reported.[3,5]

Therapeutic intervention with low-dose topical corticosteroids is the mainstay of treatment during the active phases of the condition for symptomatic relief.[2,6,7] Ocular lubricants, therapeutic soft contact lenses (SCLs) and cyclosporin A (CsA) have also been recommended in the literature.[2,6] There is no cure for TSPK.

While TSPK is an uncommon condition, it should be considered as a differential in cases of chronic non-specific ocular discomfort and is therefore an important condition for eye-care practitioners to recognise and manage. A case is presented where, after 11 months of misdiagnosis, the condition was eventually diagnosed as TSPK and successfully managed.

A 22-year-old Caucasian female first presented to the clinic reporting an 11-month history of recurrent episodes of moderate-to-severe light sensitivity, watery, stinging eyes and gritty sensation that lasted 5–7 days, occurring approximately every 4–5 weeks. The ocular discomfort on the day was subjectively graded as 3 out of 10, with reports of grade 8 discomfort in prior instances. She denied ocular redness, discharge and/or contact lens wear and medical history was unremarkable.

She reported consulting several eye-care practitioners, each with conflicting diagnoses. Various topical preparations were prescribed including ocular lubricants, Chloramphenicol 0.5% and Aciclovir 3% w/w ointment, all with minimal alleviation of symptoms despite strict compliance.

Best corrected acuities were 6/6+2 in each eye. Both corneas exhibited multiple, scattered whitish-grey round/oval and stellate lesions (twelve in the right and seven in the left), confined within the superficial epithelium (Figure 1) that stained with sodium fluorescein (Figure 2). Some lesions were essentially flat and others minimally elevated. The eyes were otherwise white and quiet, and corneal sensitivity intact.

A provisional diagnosis of TSPK was made, with herpes simplex keratitis (HSK) still a differential, although unlikely.

Management approach was conservative given the mild symptoms and corticosteroids possibly contraindicated; existing drops were discontinued, and non-preserved artificial tears were recommended every 1–2 hours and ointment before bed; advice was to return immediately if symptoms worsened. Review at 24 hours revealed an unchanged appearance of the corneal lesions.

Significant improvement in symptoms was reported at the follow-up visit three days later, although the patient reported some impracticalities in maintaining 1–2 hourly dosing and an aversion to the ointment. Acuities remained unchanged and besides a reduction in the number of lesions, eyes continued to be white and quiet. The marked corneal improvement and absence of any dendritic lesions solidified the diagnosis and led to management with preservative-free ocular lubricants three times a day and to return for review at the next flare up.

Three weeks later, the patient presented with symptoms identical to previous occurrences, this time with acuity mildly reduced to 6/6-2 in the right eye and 6/6-1 in the left. There were numerous grouped corneal intraepithelial lesions, indistinguishable in appearance to

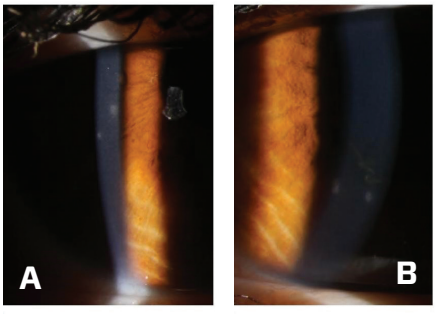

Figure 1. TSPK corneal lesions. A: right eye. B: left eye.

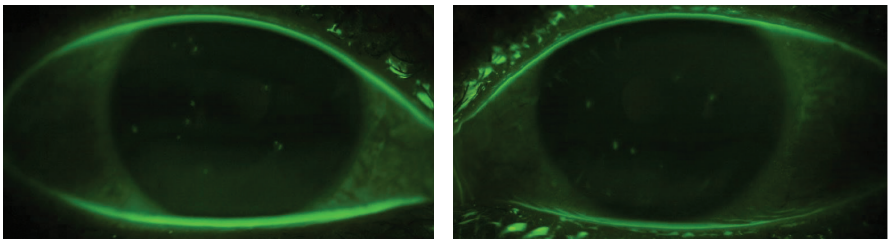

Figure 2. Fluorescein staining of corneal lesions. Figure A: fight eye. Figure B: left eye

previous, with greater than twenty lesions in each eye, worse in the right.

Active TSPK was diagnosed and patient commenced on fluorometholone (FML) 0.1% four times a day with continued use of 1–2 hourly non-preserved lubricating eye drops in between steroid dosing, at least 15 minutes either side of steroid use. There was a marked improvement in symptoms at the 24-hour follow-up visit.

Five days later, there was complete resolution of symptoms with all except one corneal lesion remaining in the right eye. The corneas did not stain with fluorescein; intraocular pressures were 12 mmHg in each eye. FML 0.1% use was tapered and a two-week review advised. An essentially identical treatment protocol was implemented for each of the subsequent flare ups.

The patient was unfortunately lost to follow-up due to a move overseas. Subsequent correspondence revealed she was self-managing flare ups with FML and ocular lubricants. Two years from onset, exacerbations were reportedly milder, less frequent (every 3–4 months) and shorter in duration (1–2 days). Despite the improvement, the importance of regular ophthalmic reviews was stressed with regards to the possible side effects of long-term topical corticosteroid use; and the opportunity to discuss and consider alternate management therapies with an enhanced safety profile.

Discussion

The clinical picture of TSPK is characterised by recurrent, bilateral epithelial keratitis, with an essentially white and quiet eye in an otherwise healthy patient. The keratitis is variable in its presentation and follows a relapsing course with remissions and exacerbations over several years, until spontaneous resolution. There is generally an excellent long-term visual outcome, although there have been reports of some patients developing permanent faint sub-epithelial opacities.[6,8]

TSPK is commonly confused with other conditions, and patients frequently report conflicting diagnoses from different practitioners and minimal success in treating their symptoms. Differential diagnoses and key differential features are outlined in Table 1.

Although there is no cure, therapeutic intervention is focused on symptomatic relief in the active phase of TSPK, as the presenting symptoms can be debilitating for some patients.

Treatment varies depending on the case severity. Tolerable symptoms in mild cases may be relieved by regular use of non-preserved ocular lubricants with an ointment at bedtime.[6] Otherwise the mainstay of treatment is a mild topical corticosteroid (FML 0.1%, Loteprednol or similar), although a more potent steroid such as fluorometholone acetate may be required in more severe cases, with minimal strength and dose to control symptoms.[2,3,5,6] A gradual taper of the corticosteroid is essential to prevent recurrence, however, it can prove challenging given the variable presentation of TSPK, with some patients requiring a tapering schedule over the course of weeks to months to avoid a recurrence.[6] Other treatment options such as therapeutic SCLs or topical CsA where available can be considered, the latter indicated in recalcitrant cases or where topical steroid treatment is contraindicated.[2,10,11]

The risks of extended corticosteroid use in an otherwise benign condition must be considered, bearing in mind that TSPK can take many years to resolve.[2,3,5] Although low potency steroids have less likelihood of side effects,[9] there are still concerns associated with long term topical corticosteroid use, including steroid-induced glaucoma, cataracts and

Continued page 12

Differential	Unilateral/bi-lateral	Lesion appearance, associated features	Vital staining	Additional features
Early herpes simplex keratitis	Usually unilateral	Fine/course SPK. Small bullous epithelial vesicles.	+ Fluorescein Sodium + Rose Bengal	SPK coalesce to dendritic lesions. Heaped margins and terminal bulbs. Reduced corneal sensitivity
TSPK	Typically bilateral	Multiple, mildly elevated, whitish central intraepithelial corneal opacities	+ Fluorescein Sodium	Recurrent keratitis in a white and quiet eye
Adenoviral keratopathy	Bilateral	Nummular sub-epithelial corneal opacities	-	Swollen lymph nodes. Follicles
Keratoconjunctivitis sicca (KCS)	Usually bilateral	Small, finer SPK	+Fluorescein Sodium + Lissamine Green	
Sterile sub-epithelial infiltrates	Usually unilateral	Usually peripheral corneal distribution	-	
Microcystic epithelial oedema	Usually unilateral	Small raised bullae. Corneal oedema	-	Blurred vision, halos
Acanthamoeba keratitis	Mostly bilateral	Raised subepithelial opacities	-	History of CL wear. Blurred vision. Symptoms disproportionate to signs.
Toxic epitheliopathy	Usually bilateral	Widespread superficial punctate epitheliopathy (SPE)	+ Fluorescein Sodium	Stinging, burning sensation. Conjunctival injection. History multiple/chronic use of topical drugs/CL and/or solution use
Neurotrophic keratopathy (mild/early)	Usually unilateral	SPE	+ Fluorescein Sodium	Red eye. Blurred vision. No irritation/pain.

Table 1. Differential diagnoses and key differential features

TSPK

From page 11

increased susceptibility to corneal infections; there's also speculation that corticosteroids may potentially prolong the chronic nature of the disease.[2,3,5,6,8]

Those receiving topical corticosteroid treatment should have their intraocular pressures monitored closely, especially if they are at risk of a steroid response.

Finally, the chronic and recurrent course of TSPK may lead patients to self-medicate, putting themselves at higher risk for steroid related ocular complications. It is therefore imperative that all patients be acutely aware of the potential side effects of topical steroid use and the importance of regular reviews. In addition, the practitioner should be astute in limiting the number of repeats when prescribing topical steroids. With compliance, patients can be reassured that TSPK usually resolves without any long-term effects on vision.

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Contact lenses: beyond

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The use of contact lenses continues to grow, with an estimated 140 million wearers globally. While the majority of wearers use contact lenses for the correction of refractive error, there is growing interest in their use for 'non-standard' concepts. As we approach the magical year (for optics) of 2020, we ask: if we glimpse into the future, what will the contact lenses of (say) 2035 look like? What options will they provide to our patients that will differ from the lenses of today?[1]

Myopia control

Myopia is a significant public health problem. In 2010 it was estimated that 28 per cent of the world's population was myopic, but it is predicted that by 2050, 50 per cent of the global population could be myopic.[2] As discussed in the September issue of *Pharma*, the most rapid increase has been in East Asian countries, where it has already reached epidemic proportions, affecting over 90 per cent of adults in some regions such as Korea, Taiwan and Singapore.[3] Myopia is not merely an inconvenience resulting in the patient requiring an optical correction. The myopic eye, particularly those with high myopia of > 6.00 D, has an increased risk of developing ocular pathology that may lead to vision loss, in particular retinal detachment and myopic macular degeneration. Thus, methods to slow or prevent myopia progression (and the ocular axial elongation that accompanies it) are extremely important if myopia-induced pathologies are to be avoided. Recent estimates suggest that slowing myopia progression by one dioptre should reduce the likelihood of a patient developing myopic maculopathy by 40 per cent.[4]

Several treatment paradigms for slowing myopia progression have been evaluated in intervention studies, largely encompassing the use of either pharmacological means (most commonly through the use of the topical anti-muscarinic drug atropine) or various optical interventions.[5-13] These optical interventions include progressive addition spectacle lenses, bifocal spectacles, orthokeratology rigid contact lenses and multifocal soft contact lenses.[8,14-19]

To date, many of the contact lens methods used remain 'off-label,' meaning that while studies would suggest that such technologies do indeed appear to show a slowing of myopia progression, regulatory approvals for these products do not yet exist to support the product being used in such a manner.[20] One such example is orthokeratology, which is approved for use in the reduction of refractive error, but not approved for slowing the progression of myopia, despite many studies supporting this to be the case.

However, as the interest in using contact lenses for slowing myopia progression increases, more products will gain regulatory approval for this indication, resulting in this being a significant growth area for contact lenses over the next decade. Two commercially available daily disposable soft lens products that have gained regulatory approval for slowing the progression of myopia in various countries following successful clinical trials are MiSight (CooperVision)[21] and NaturalVue Multifocal (Visioneering Technologies).[22]

Drug delivery

One of the biggest opportunities for the development of 'specialised' contact lenses relates to their use as drug delivery devices. A lens that would

20/20

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release therapeutically-relevant doses of a topical drug for five-to-seven days would likely find an immediate place in clinical practice and there is great interest in this concept among clinicians.[23] The interest in this topic is evidenced by the fact that over 350 peer-reviewed publications have addressed this issue, with 25 per cent of them being published within the last five years. This is particularly relevant for diseases which require consistent dosing over many weeks or months, in which compliance with instilling drops becomes lower over time, for example in the management of glaucoma.[24]

Several research groups around the globe are actively looking at developing such technologies. There are various methods proposed to deliver ocular medications, with many employing novel approaches based on nanotechnology.[25] Recent publications on this topic include those describing the extended release of anti-glaucoma medications,[26] antibiotics,[27-29] antifungal agents,[30,31] drugs to treat dry eye and surface inflammation,[32] drugs to slow the progression of myopia[33] and anti-inflammatory drugs.[34,35] Contact lenses have already been used in full-scale clinical trials that release an anti-allergic agent (ketotifen).[36]

Based upon the rapidly expanding literature in this field and the level of interest, it appears to be just a matter of time before such devices become commercially available. However, concerns relating to regulatory approvals, how to control the leaching of drugs into the surrounding blister pack solution and which practitioners are licensed to dispense such products will likely delay their introduction for some time while these issues are addressed.

Detection and monitoring of disease

The ability of contact lenses to monitor ocular and systemic diseases such as diabetes and glaucoma would appear to be something approaching science fiction. However, there is growing interest in the use of wearable sensors for many aspects of health monitoring.[37-39] There is already a commercially-available contact lens device (Figure 1) that uses sophisticated strain-gauge technology to continuously measure intraocular pressure over a 24-hour time period,[40,41] and several groups have published work on the development of materials that can monitor glucose levels within the tear film.[42-44] Published work has also looked at using the tear film to monitor signs of cancer[45,46] and contact lenses that could detect such small levels of biomarkers within the tear film would be invaluable.[47] Thus, the continued expansion of interest in the development of such devices seems inevitable, as developments in miniaturisation of batteries and electrical components improves.[48,49]

Figure 1. The Sensimed Triggerfish continuous ocular monitoring system

Advanced optical designs

The final area of interest relates to the manufacture of lenses with novel optics. Spectacle-mounted head-up displays with the ability to access the internet, display websites and email, stream video or take photographs have been under development for several years, with the most well-known being the Google Glass concept, launched in 2013.

Other manufacturers have worked on various versions of these 'smart spectacle' platforms and the development of newer technologies have enabled companies to consider incorporating this technology into a contact lens platform. This concept could be used to develop, for example, a multifocal contact lens that changes power depending upon the distance at which the wearer is viewing an item of interest, a magnifying contact lens for people with low vision and even opens up the potential for almost invisible virtual reality systems that offer great improvements over the current bulky, head-mounted versions.[50-54]

The future for contact lenses remains bright, with many new and exciting developments ahead. Contact lenses to control myopia progression are already here and the number of options will rapidly expand; in the near future, lenses to detect and treat ocular disease will be available and in the more distant future, we are likely to see the availability of lenses with highly sophisticated optics for unique optical applications.

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A complete list of references for this article is available on the Pharma *page of the Optometry Australia website. Additionally, a complete copy of the references is available by contacting the editor at pharma@optometry.org.au.*

Red eye conditions

From the 2019 Optometry Australia Anterior Eye Clinical Practice Guide

	Common symptoms	Clinical presentations	Risk factors	Differential diagnoses
Bacterial Keratitis	• Redness • Pain • Photophobia • Reduced vision • Lid Swelling • Mucopurulent discharge • "White spot on eye"	• Irregular focal lesion, may be >1 mm in size • Epithelial defect • Discharge • Anterior chamber reaction – cells & flare • Lid swelling • Infiltrate • Posterior synechiae • Conjunctival injection	**Age** • 15-64 years (Trauma and Contact Lenses) • > 60 years – Previous ocular surgery **External** • Contact lenses (e.g. extended wear, poor hygiene, inadequate disinfection, sharing of lenses, use of tap water) • Trauma • Previous ocular surgery • Immunosuppression • Substance abuse **Internal** • Tear-film deficiencies • Viral keratitis • Recurrent corneal erosion **Systemic conditions** • Diabetes • Atopic dermatitis • Blepharoconjunctivitis • Gonococcal infection • Vitamin A deficiency	• Sterile peripheral infiltrate • Marginal keratitis • Fungal keratitis • Herpes simplex keratitis • Exposure keratopathy • Neurotrophic • Acanthamoeba keratitis • Shield ulcer • Dellen • Phlyctenular keratitis
Herpes Simplex Keratitis	• Redness • Pain/Discomfort • Photophobia • Reduced vision • Lid swelling • Mild watery discharge • Reduced corneal sensitivity	• Epithelial disease (dendritic or geographic ulcers) • Stromal disease • Neurotrophic keratitis • Endotheliitis • Conjunctivitis (mild) • Skin lesions • Anterior chamber reaction • Conjunctival injection • Preauricular node	• Long-term corticosteroid inhalers • Long-term corticosteroid creams • Asthmatic patients • Cardiovascular disease • Immunosuppressed patients • Atopic patients • Multiple previous episodes	• Acanthamoeba keratitis • Herpes Zoster Ophthalmicus • Recurrent corneal erosion • Healing abrasion
Acute Anterior Uveitis	• Redness • Pain • Photophobia • Reduced vision • Copious watery discharge	• Circumlimbal flush • Anterior chamber reaction – cells and flare • Miotic pupil • Keratic precipitate • Hypopyon • Abnormal IOP • Corneal oedema • Posterior synechia	• HLA-B27 positive • Rheumatoid conditions • Inflammatory bowel conditions • Trauma • Keratitis • Idiopathic • Ulcerative colitis • Crohn's disease • Syphilis • Behcet's disease • Sarcoidosis • Tuberculosis • Multiple Sclerosis	• Glaucoma (acute angle closure) • Fuchs Heterochromic iridocyclitis • Endophthalmitis • Posner-Schlossman Syndrome • Lens induced uveitis • Intraocular foreign body

Triggers for referral & appropriate timing	Pharmacological management	Review
Same day/within 24 hours • Larger (> 2 mm), more central or deeper lesions – risk of scarring and/or perforation • Consider referral for culture/corneal scrape to identify causative organism • Non-responding cases: be aware of bacterial resistance to antibiotic treatment • Consider non-bacterial causes **Within 72 hours** • Cases that do not respond to initial treatment or slow/inadequate healing	**Topical ciprofloxacin or ofloxacin** **Loading dose:** Q1h for 2 days then (if good response) QID until completely resolved. **Considerations:** • Fluoroquinolones (ciprofloxacin and ofloxacin) cover both gram positive and gram negative pathogens • Ciprofloxacin has enhanced activity towards gram positive – may be preferred in hot climates in contact lens microbial keratitis • Ofloxacin in cooler climates for Staph species • Atropine – (prevent ciliary spasm) if significant pain and oral analgesia insufficient. • Corticosteroids – limit scarring during healing • Steroid treatment should be introduced only after 2-3 days of progressive improvement of the ulcer	**Daily until ulcer shows improvement.** **Weekly until complete resolution.** Clinical discretion should be applied. Review schedule should be considered on a case by case basis. Factors to consider include: • Severity of infection • Risk of side effects • Reliability of patients to comply with instructions
Same day/within 24 hours • Stromal and endothelial involvement • Bilateral cases • Large geographic ulcers **Within a week** • Cases that do not respond to initial treatment	**Epithelial and Geographic** 3% Acyclovir* ointment - 5 times/day for 7 days then 3 times/day for next 7 days. (*Can be toxic to ocular surface. Cease 1-2 days after resolution and consider non-preserved lubricants to help with ocular surface toxicity) Consider cycloplegic agent with anterior chamber reaction **Stromal Keratitis** Topical corticosteroids with oral prophylactic antivirals **Considerations** Topical steroids will worsen herpes simples keratitis HSK epithelial disease Oral antivirals may be indicated in patients with many recurrences, e.g. • Valacyclovir 500mg 1xday • Acyclovir 400mg 2x/day Consider referral for medical opinion	**1-2 days until HSK is improving.** **Weekly until complete resolution.** Clinical discretion should be applied. Review schedule should be considered on a case by case basis. Factors to consider include: • Severity of infection • Risk of side effects • Reliability of patients to comply with instructions
Same day/within 24 hours • Severe cases e.g. significant posterior synechiae, poor view of posterior pole, atypical inflammation • Hypopyon • Bilateral • Posterior segment involvement • Recent surgery • Presence of drainage bleb • IOP > 30 mmHg **Within 72 hours** • Cases that do not respond to initial treatment • Refer to medical practitioners (GP, ophthalmologist) following 2nd episode	Topical Steroids with good intraocular penetration: Predforte or Maxidex. **May require loading dose:** • Q1h waking hours (consider overnight based on severity) for 2 days, then (if improvement) Q2h for 2 days, then (if improving) • Qid for 1 week, then • Tid for 1 week, then • Bid for 1 week, then • Qd for 1 week, then stop. Monitor IOP while treating with topical steroids to identify steroid responders Atropine (bid – tid) until anterior chamber reaction under control.	**Review on first or second day after commencing treatment.** Clinical discretion should be applied. Review schedule should be considered on a case by case basis. Factors to consider include: • Severity of inflammation • Risk of side effects • Reliability of patients to comply with instructions

Gut flora: why optometrists should be paying attention

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Our bodies are mostly microbes (micro-organisms such as bacteria, viruses or amoebae). For every human cell we have, there is at least one microbial cell in our microbiome.[1] The microbiome is the collective genome of all micro-organisms living in us and on us, and although the number of organisms in the average microbiome is approximately equal to our own cells, the number of their genes outnumbers our own by many orders of magnitude.[2]

In the language of the ads, if our 'good' bacteria are outgunned by too many 'bad' bacteria, our health can suffer. This imbalance is called dysbiosis,[3] and has been linked to many systemic conditions[4] that are associated with eye disease, including type 1 diabetes, type 2 diabetes, inflammatory bowel diseases such as Crohn's, and cardiovascular disease.

Most of the bacteria that call us home are beneficial to our health. They produce essential amino acids, proteins and vitamins, they help us to extract nutrients from our food,[5] and they produce anti- inflammatory substances such as butyrate. Butyrate is a short-chain fatty acid which protects against bowel cancer, protects the lining of the gut and can down-regulate the vascular endothelial growth factor (VEGF) gene.[6]

In a healthy individual, the total number and biodiversity of beneficial bacteria in the gut help to stop any pathogenic organisms from becoming too numerous. This is both through simply competing for food resources, and through more active competition. For example, some Bacteroides species kill off *Candida albicans* by injecting the yeast cells with hydrogen peroxide.[7]

However, in an individual whose diversity or total population of beneficial bacteria is limited, dysbiosis results, causing the bad bacteria to take over. Pathogenic bacteria, such as *Clostridium difficile* and some species of *E. coli*, will trigger inflammation in the lining of the gut, which loosens the tight junctions between the cells of the submucosa.[7] Inflammatory mediators, bacterial toxins and bacteria themselves then have a direct route to the circulation and therefore the rest of the body, and inflammation follows. In this way, dysbiosis can lead to inflammation of other tissues such as skin, lungs, joints and eyes.

Another route for inflammation in the gut to reach other tissues is along the vagus nerve, part of the gut-brain axis.[8] Material from the gut can pass directly along this route to the brain, and researchers are now asking serious questions as to whether this is how Parkinson's disease starts.[9] Neurones of the central nervous system can express inflammatory cytokines, such as IL-1 and TNF, and it's thought that these cytokines play a role in inter-neuronal communication.[9]

Either way, we have at least two routes through which inflammation in the gut can lead to inflammation and disease elsewhere, including the eye. This is great news. It means that not only can we help our dry eye patients with traditional and topical approaches, such as lubricating drops, steroids and oral omega-3s, it also means that we can provide more information to help our patients to help themselves.

Implications for optometrists

We don't have to be experts on the microbiome. Although we're not expert dieticians, it is within our scope to provide lifestyle information on reducing the risk of vision loss from macular degeneration.

An appropriate step, as health care professionals wanting the best outcomes for our patients, is to help steer them in the right direction. By giving them relevant information and nudging them towards further reading, as well as collaborating with dieticians, we can potentially achieve

better results for many of our more challenging dry-eye patients.

Supplements

A simple way to help rebalance a dysbiotic large intestine is to take probiotics.[10] A probiotic supplement typically contains billions of bacteria from the species known to be beneficial to our overall health. These are readily available in powder or capsule form, as well as in food products such as various yoghurts and drinks.

Diet

Other lifestyle changes are also potentially beneficial to a healthy microbiome. These include a healthy diet, resplendent in brightly-coloured vegetables, particularly those that contain polyphenols.[11] Among other things, polyphenols nourish Akkermansia bacteria,[12] one of the good guys. Akkermansia have the attention of the research community because they seem to play a role in battling insulin resistance and obesity,[12] and are associated with better control of blood glucose after a meal.[13]

You'll also find polyphenols in coffee, tea, red wine, dark chocolate, dried herbs, olives and oily fish.[14]

Exercise

Exercise has been linked to a greater diversity of healthy species in the microbiome.[15] So has exposure to the great outdoors,[7] whether it's getting out and about or simply opening a window to your home or office. Similarly, people who expose themselves to dirt in their garden are increasing their exposure to (mostly) good bacteria.[7]

The list goes on

Maintaining regular sleep patterns and reducing cortisol levels by reducing stress, where possible, are good for a healthy, diverse microbiome.[7] Fasting from time to time allows Akkermansia bacteria access to one of their favourite foods – the mucus lining of our large intestine.[7] The sugars found in fast foods and alcohol will (unfortunately) mostly feed the pathogens,[7] so red wine in moderation is the recommendation. Fast food also contains emulsifiers, which alter microbiome diversity in ways that promote inflammation.[16]

Inappropriate use of systemic antibiotics will annihilate many of our healthy species.[17] In the dry eye arena, this might make us think twice before referring a patient for doxycycline therapy. It also might explain why many patients taking doxycycline will suffer side effects such as nausea, and thrush, caused by *Candida albicans*.[18]

Another step, for those who are particularly interested, is to advise patients they can now have their intestinal microbiome genetically sequenced. Professor Ian Frazer and the University of Queensland have launched a company (www.microba.com) which offers this service, including bespoke dietary and lifestyle advice.

Dry eye

Perhaps when patients complain of dry eyes, we could gain some insights into whether their condition might be more internally driven before making exclusively topical recommendations. For example, if a patient complains of frequent dryness, we could simply ask if they also suffer from problems with their lungs, joints or skin, or whether they have gastrointestinal problems. This could at least open the door to discussion as to how best to manage the condition in the longer term, rather than potentially just 'window-dressing' by concentrating only on the ocular surface.

Dry-eye care is the tip of the iceberg when considering how we, as optometrists, can steer patients in the direction of better eye health through better gut health. There are now clear links between gut inflammation and uveitis,[19,20] between gut inflammation and retinal inflammation,[20,21] (for example in Crohn's patients) and between gut inflammation and ARMD.[20, 21]

Systemic conditions

Finally, many of our patients are affected by systemic conditions, such as diabetes and hypertension, which not only have the potential to affect vision adversely, but which may often be better controlled through attention to the intestinal microbiome. Even just a gentle nudge from us, in the direction of a Google search—linking dysbiosis to their disease—might make the world of difference to their overall health and the protection of their sight.

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Revisiting ‘Christmas Eye’

'Tis the season for acute toxic keratopathy

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Figure 1. Orthoperus releases a blistering agent when crushed.

In 2008, I wrote a case study for this publication regarding the condition of ‘Christmas Eye.’ Eleven years later, I have been asked to update our understanding and management of this peculiar seasonal condition.

The condition

‘Christmas Eye’ is an acute toxic keratitis that occurs during the hot, dry summer months in south eastern Australia. It has also been referred to as Albury-Wodonga syndrome, harvester’s eye or seasonal corneal ulcer.[1] Typically, we expect presentations from mid-November until late February. The cases often occur in clusters and the patient history consistently involves some outdoor activity (gardening, mowing, etc.) the previous afternoon or evening.

Prior to the use of the bandage contact lenses, the effects of Christmas Eye were debilitating in the short term. The patient was unable to work and suffered extreme pain until the cornea had recovered. Typically, up to a week of employment or useful activity was lost.

The level of pain associated with Christmas Eye has achieved folkloric status. The hardy farming types, who make up a sizeable portion of the victims, shake their heads with sympathy when they hear of a friend or colleague who has been affected.

One of our patients has been affected three times in ten years.

Pederin and Orthoperus

For many years, the cause of the condition has been frustrated by a lack of physical evidence. But further research has supplied the reason for this lack of evidence. The causative

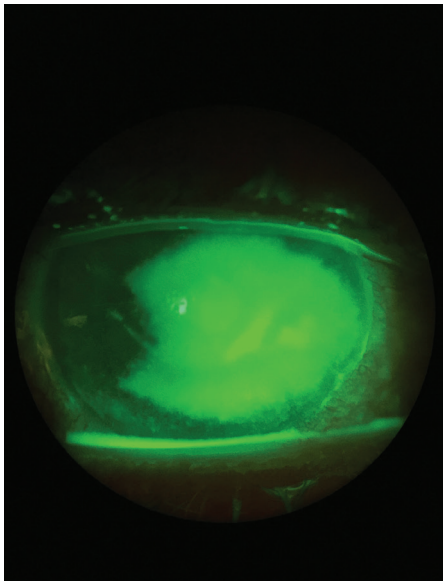

Figure 2. Typical NaFL corneal staining associated with Christmas Eye

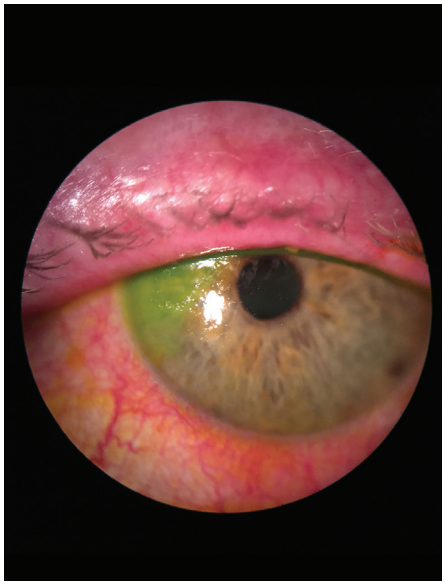

Figure 3. Subject 1 LE white light

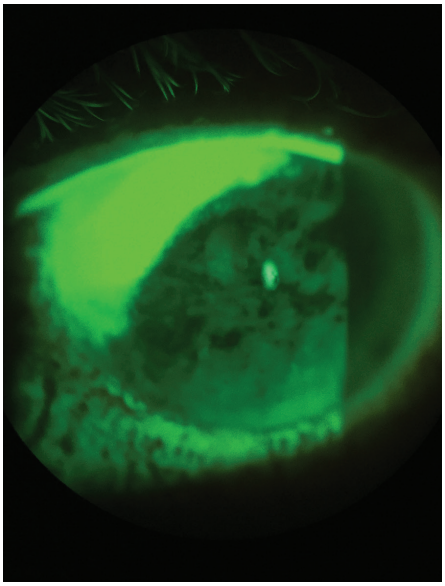

Figure 4. Subject 1 LE cobalt blue/Wrattan filter

agent is thought to be a small beetle of the genus Orthoperus.[2]

Orthoperus is a genus of minute hooded beetles in the family *Corylophidae*. Their size is of the order 0.5–0.7mm (Figure 1). Orthoperus are known to carry the compound Pederin[3] in their haemolymph which is released when the insect is crushed on the skin or eye. Pederin is a powerful inhibitor of protein biosynthesis and mitosis and is a known vesicant (blistering agent). With these properties, it is unsurprising that it has such a dramatic effect on the corneal epithelium.

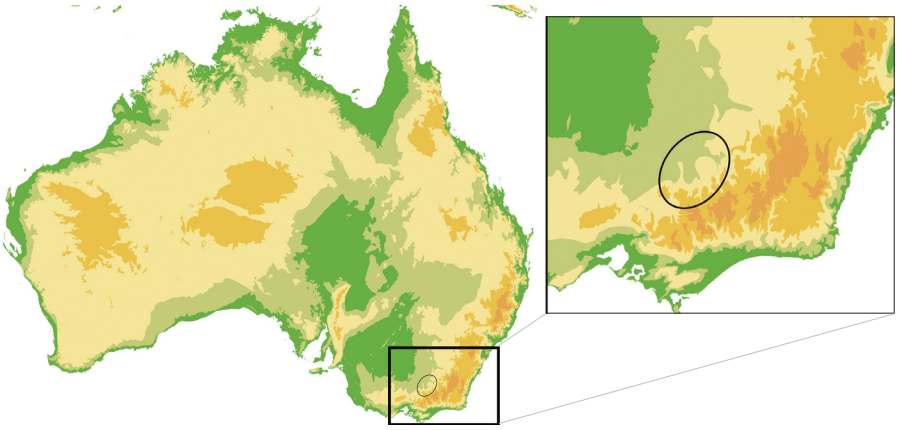

Figure 5. Geographic distribution of Christmas Eye

Signs and symptoms

Patients will often present in the early hours of the morning at the local Emergency Department suffering extreme pain.

Clinical signs

- Mottled corneal epithelium disturbance progressing to extensive full thickness epithelial loss involving up to 90 per cent of the cornea (Figure 2).
- Corneal oedema increasing corneal thickness up to 30 per cent with accompanying endothelial wrinkling
- Extensive bulbar conjunctival injection
- Bulbar conjunctival chaemosis and oedema
- Mild anterior chamber reaction with cells and flare
- Moderate lid and periorbital oedema
- Decreased vision (6/15 to 6/24) (Figures 3 and 4)

Clinical symptoms: PAIN

The pain level is extreme with a pain score of 8-9/10 and, in the early stages, is often disproportionate to the degree of corneal disruption.

- Excessive lacrimation
- Marked photosensitivity
- Headache
- Nausea

Geographic distribution

Traditionally, 'Christmas Eye' in Australia was thought to be limited to north east Victoria and southern NSW. Further enquiries have shown it is distributed further afield with occasional cases being reported in western Victoria, Gippsland and central NSW. Statistically however, the vast majority of cases occur in north east Victoria and southern NSW (Figure 5).

Differential diagnosis

Conditions with potentially similar presentations include:

- Herpes keratitis
- Corneal abrasion
- Infectious corneal ulcer

Taking a careful history, paying particular attention to the timeline of pain and discomfort, will generally provide the diagnosis. Biomicroscopy will show the extent of corneal damage, the integrity of the remaining epithelium and provide a confirmation of the diagnosis.

An essential fact in determining your differential diagnosis is the time of year. Christmas Eye only seems to occur between late October and early March. A clinical presentation outside of this time of year makes the diagnosis extremely unlikely.

The higher the pain level, the more likely the Christmas Eye presentation. Patients will often arrive cradling their head with their hand cupped over the affected eye. They are miserable and have often attended following an initial presentation at the local hospital emergency department.

Details regarding the timeframe of the pain onset will also give key clues as to the diagnosis.

A corneal abrasion will cause instant pain and the offending object can often be readily identified. Herpes has a more gradual build-up of discomfort over a day or so. Christmas Eye generally wakes a person in the early hours of the morning with increasing eye pain that continues to build despite the patient's best efforts to reduce the aggravation.

Corneal observation with fluorescein will provide further information to assist with the diagnosis.

Herpes will commonly present with its tell-tale linear and lobular staining, quite different to the lesions found in Christmas Eye. The epithelium in the surrounding area with Christmas Eye is often disrupted and can be dislodged with ease. This is quite different to the other differentials where the surrounding tissue is still intact and looks 'normal.'

Management

The management of Christmas Eye is quite straight-forward once the diagnosis has been made.

It is well known that the corneal epithelium heals rapidly and that pain levels diminish as the epithelium recovers. The control and reduction of pain is the key tenant of the management strategy.

In 2008, treatment involved the fitting of a bandage silicone hydrogel contact lens and intensive use of a topical nonsteroidal anti-inflammatory such as diclofenac sodium (Voltaren).

Continued page 20

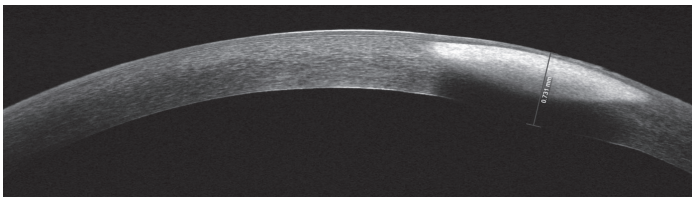

Figure 6. Marked localised corneal swelling

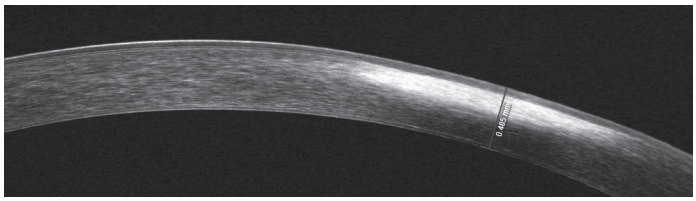

Figure 7. Localised corneal thinning

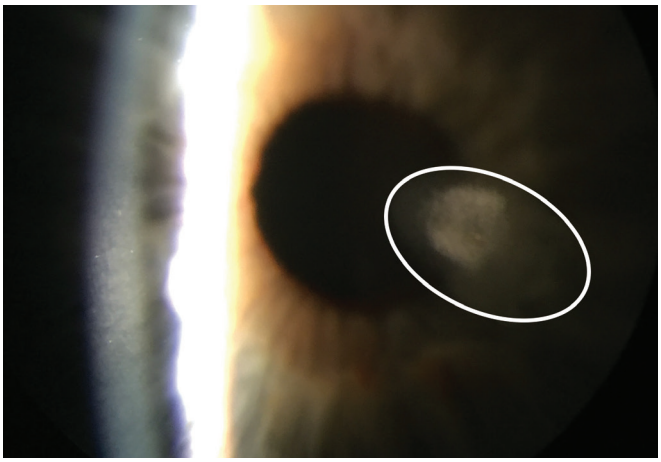

Figure 8. Corneal scarring

Christmas Eye

From page 19

In 2019, our management has been modified but the key principle of pain relief has remained. My current regime is as follows:

At initial presentation

- Topical anaesthesia to alleviate pain and allow examination of the eye
- Photo document the extent of epithelial loss
- Record corneal thickness (anterior OCT)
- Insert bandage silicone hydrogel contact lens (B&L Ultra)
- Review every two to three days until corneal epithelium is healed
- Provide patient with after-hours contact number

Prescribe

- Chloramphenicol eye drops, four times a day
- Oral non-steroidal anti-inflammatory medication, for example: Nurofen or Voltaren – maximum daily dose

When the corneal epithelium has healed

- Remove bandage silicone hydrogel contact lens
- Stop antibiotics
- Stop oral pain relief
- Commence non-preserved lubricants (AFT Hyloforte) four times daily for two weeks
- Review in two weeks and then discharge

Adverse events

The vast majority of patients will heal perfectly without any adverse effects.

Visual acuity and corneal structure return to pre-incident levels and there are no residual signs of corneal insult.

Over twenty years, I have dealt with hundreds of cases of Christmas Eye without any problems using the regime outlined in 2008. However, two years ago, for the first time, we experienced a cluster of adverse events involving the cornea.

Sub-epithelial corneal haze

Looking similar to pronounced sub-epithelial haze seen in surface photorefractive keratectomy (PRK) refractive surgery, the haze was present in the anterior stroma and contributed to a slight reduction in acuity and increased glare. This gradually resolved over 12 months with a slow return to the original corneal topography and thickness.

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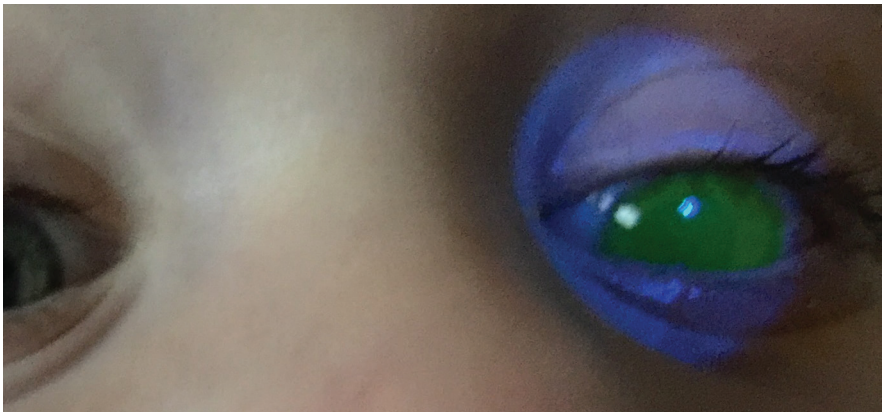

Figure 9. Child's cornea completely stained with NaFL

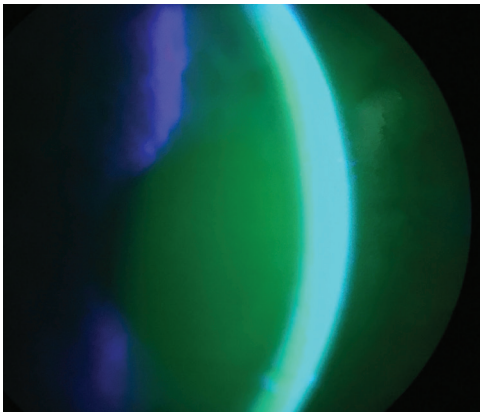

Figure 10. Cornea and aqueous humour stained with NaFL

DON'T LOSE SIGHT OF WHAT'S IMPORTANT

Age-related macular degeneration is the leading cause of legal blindness in Australia. Yet one in three Australians over 50 hasn't had their macula checked in the last two years.[2,3]

That's why Novartis and Macular Disease Foundation Australia are encouraging people to visit their optometrist for an eye exam.[1]

Find out more at:
seewhatsnext.com.au

SEE WHAT'S NEXT[1]

Macular Disease Foundation AUSTRALIA

NOVARTIS

The Macular Disease Foundation Australia (MDFA) and Novartis Pharmaceuticals Pty Ltd are working together on the common goal to raise awareness of age-related macular degeneration in the community. See What's Next is an awareness campaign developed by Novartis. By supporting this campaign the MDFA is not endorsing any specific treatment or therapy. **References: 1.** Optometry Australia. 2019 Clinical Practice Guide for the diagnosis, treatment and management of Age-Related Macular Degeneration. **2.** Macular Disease Foundation Australia. The Journey to See: A Model for Success. **3.** Awareness of macular disease' survey conducted by YouGov Galaxy, commissioned by Macular Disease Foundation Australia, 2018. Novartis Pharmaceuticals Australia Pty Limited ABN 18 004 244 160. 54 Waterloo Road, Macquarie Park NSW 2113. Ph (02) 9805 3555. August 2019. AU-9993. NOBR16943WP. Ward6

Christmas Eye

From page 20

Localised corneal thinning

I have seen three cases where there has been a localised area of stromal thinning. The corneal response appears as expected until the four-day mark where marked corneal inflammation and oedema seems to remain. As it subsides, the stroma appears to thin and the corneal topography changes to show an area of depression. There is associated vision disruption due to the altered topography.

Detailed anterior OCT scans confirm the stromal thickness loss as the epithelium regrows with normal thickness.

Visual recovery from this is very slow and can be incomplete. OCT scans and topography over the past three years show both a localised thickening of the overlying epithelium and an improvement in the corneal regularity.

Dense corneal scarring

Unfortunately, I have one patient who experienced the development of a disc-shape intrastromal corneal scar. His incident history was identical until the day-five mark, when he developed a disc shaped sub-epithelial lesion with marked corneal thickening. His corneal epithelium was nearly fully healed (Figure 6).

Over the next six months the cornea involved proceeded to flatten and thin. The density of the corneal scarring also reduced. The original size of the scar is indicated by the white outline in Figure 8. At its peak, the corneal thickness through the lesion was 721microns. Four weeks later, the thickness had reduced to 489 microns (Figures 7 and 8).

A year later, the lesion is still present but less dense. The topography and corneal thickness changes have stabilised. Fortunately, the lesion was off axis and is now only causing slight blur and mild flare at night while driving.

Discussion

Why there was a cluster of these adverse events in the summer of 2017–18 is unknown. Last summer (2018–19) I experienced no complications.

It may be the causative agent was slightly different, creating a greater inflammatory response or that these individuals happened to be more susceptible to corneal damage from inflammation.

The events have led me to remove the topical NSAIDS from the management plan. There have been literature reports of corneal damage related to topical NSAID use.[4] I am unsure why this would become an issue in one particular year and it may be completely unrelated.

These events appear to be individual reactions to the extreme corneal inflammation that occurs with Christmas Eye. They are a cautionary reminder that unusual events can occur when treating eye conditions.

Imaging

The development of imaging systems such as topography, anterior OCT and digital photography allows the practitioner to capture some of very strange images that can be associated with Christmas Eye. This technology allows us to monitor the effects on the eye as never before (Figures 9 and 10).

Conclusion

Christmas Eye can be a confronting condition for those who are unaware of its signs and symptoms. Any condition that causes extreme pain is stressful for both the practitioner and particularly the patient. Care, reassurance and pain control are the keys in managing this unusual condition.

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TAU: Tattoo-

Cutaneous

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As a cosmetic and decorative body art, tattooing has dramatically increased particularly among young adults. A survey of 1,013 Australians by market researcher McCrindle in April 2018[1] showed that the number of people getting tattooed had hit a record high with one in five people having one or more tattoos.

The majority have more than one tattoo (61 per cent) and around 14 per cent have six or more. Fifty-one per cent had obtained their first tattoo between the ages of 18 and 25, and thirty-six per cent at 26 or older. Australian women with tattoos (20 per cent) outnumber men (19 per cent).

Given these figures, we need to be aware of a possible increase in presentations of tattoo-associated uveitis (TAU).

CASE REPORT

A 25-year-old male presented to the emergency department in 2017 with decreased visual acuity, intermittent redness and a feeling of 'pressure' in both eyes (OU). These symptoms had been intermittent for approximately one year.

The patient's medical history was unremarkable; he denied any past ocular history but was diagnosed with allergic conjunctivitis in 2015. Old keratic precipitates (KP) OU were also noted at this time, indicating previous inflammation. His vision was 6/36 OU with no pinhole improvement. Bilateral anterior uveitis and posterior synechiae were noted, intraocular pressures (IOP) were RE 9 mmHg and LE 10 mmHg.

-associated uveitis

reactions and ocular problems

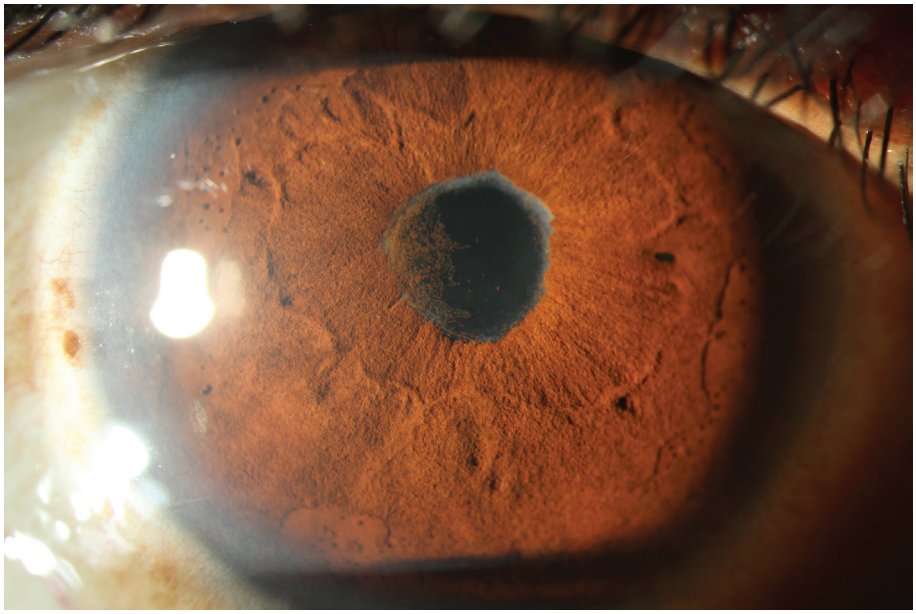

Figure 1. Posterior synechiae (adhesion of the iris to the capsule of the lens) due to inflammation.

He was diagnosed with bilateral acute anterior uveitis (AAU) and cystoid macular oedema (CMO), and commenced on topical medications: prednefrin forte (PF) hourly, and atropine twice daily in both eyes for pain management and to reduce further posterior synechiae formation.

Blood tests were taken to rule out sarcoidosis, syphilis, HLA-B27 positivity and various infectious and inflammatory aetiologies (repeated throughout his follow-up).

On review 10 days later, it has been noted that compliance had been poor with the topical medications. His vision had deteriorated further (6/60 OU), the uveitis persisted (+2 cells) and his optic nerves were swollen and hyperaemic.

He was commenced on systemic prednisolone (PNL) 50 mg daily. With improved compliance of all medications, his vision improved to 6/9 OU due to decreased CMO and inflammation. Intraocular pressures (IOP) were RE 19mmHg and LE 16 mm Hg. He was advised to reduce his PNL to 37.5 mg and atropine was replaced with tropicamide. Vision and IOP remained stable and further weaning of PNL was suggested.

He missed his next review, and on presentation seven weeks later there was a recurrence of bilateral uveitis (2+ cells, SUN grading), disc swelling and left CMO. Compliance was stressed but it was thought that a left orbital floor injection of triamcinolone may be needed in the future.

Having missed an appointment, he was reviewed five weeks later. He had lost his prescription and had only instilled topical medications on a few occasions. His left CMO had reduced, and an indurated red, raised left tricep tattoo was noted.

In retrospect, he felt that his ocular problems commenced around the time that he got the tattoo. In light of previous negative investigations and the presence of inflamed tattoos, a diagnosis of tattoo-associated uveitis (TAU) with CMO OU was made.

He was lost to follow-up for almost five months after which bilateral recurrent anterior uveitis (3+ cells) and CMO (LE > RE) had recurred. Bilateral posterior synechiae and a left iris nodule were noted. The retina could not be visualised. Due to severe eye-threatening uveitis, he was given a left orbital floor injection and was advised to reduce his PNL by half and continue with PF hourly OU.

At his next visit he complained that his tattoos were feeling 'lumpy' again. Control of his ocular inflammation was difficult, exacerbated by his poor attendance and treatment compliance, so he was commenced on a weekly dose of both immunosuppressant drug methotrexate (MTX) and folic acid. He was to continue with 5 mg PNL and PF four times each day. Monthly blood tests were initiated to monitor for dosage and side effects of the MTX.

Improvement was noted in his vision and ocular inflammation, however, a right IOP of 32 mm Hg was noted (left 12 mm Hg). His topical and systemic medications were reduced by half and he was sent to the Glaucoma Unit due to development of right ocular hypertension (OHT) caused by iris bombe (synechial closure R > L). He underwent a right Yag laser peripheral iridotomy and his IOP was noted to be 8 mmHg after this. His cup-to-disc ratios were 0.2 OU and his visual field tests were essentially normal.

When last seen, his vision was 6/9 OU; both eyes were quiescent and there was minimal CMO. IOPs were RE 8 mmHg and LE 8 mmHg. He had been weaned off the PF but was to continue with the immunosuppressant drug MTX to curtail the cutaneous reaction and reduce the risk of recurrent uveitis.

Discussion

There have been an increasing number of cases in literature[2] of TAU since Rorsman et al.[3] described three cases with light blue tattoo granuloma and anterior uveitis with no features of systemic disease in 1969. In 2014 Ostheimer et al.[4] submitted the largest study which followed seven patients with various sequelae of uveitis with simultaneous tattoo induration over 20 months.

TAU presents with bilateral recurrent/chronic uveitis, though in one case the second eye became involved one month later.[5] Cases cited have

Continued page 24

Tattoo uveitis

From page 23

ranged from having anterior uveitis (predominately non-granulomatous) to chronic pan uveitis and hypopyon.

Tattoo swelling has been said to precede uveitis for a week on recurrent episodes.[6]

The time frame of onset can occur from at least six months after tattoo placement and up to 13 years which possibly presents a specific granulomatous-delayed allergic response to ink containing metal compounds.[7] Obtaining a number of tattoos over a short period of time possibly increases the toxic load.[4] Most had extensive tattoos and were predominately male.

The majority of cases have been in the USA and many of the inks used were industrial grade colours suitable for printers and automobile ink. The induration appeared in more extensively tattooed areas that contained or consisted entirely of black ink. The black ink possibly contained toxic, mutagenic or carcinogenic compounds.[4] Two articles cite skin reaction to light blue[3] and red[8] ink.

In Australia, state and territory authorities are responsible for regulating the safety of tattoo inks including product labelling and restrictions on their use in tattooing. Chemicals used in tattoo and permanent makeup (PMU) inks are classified as industrial chemicals in Australia.[9] These regulations may not be adhered to, particularly if used outside of a registered tattoo parlour or in a country without a regulatory body.

The cutaneous reactions of erythema, pruritis, indurated papules or nodules can occur on the border or within the tattoo area (Figure 2). Similar cutaneous reactions may arise in patients with sarcoidosis (33 per cent), as can granulomatous uveitis (80 per cent).[9] There have been a number of cases where TAU has been the presenting feature of sarcoidosis. It has been postulated that sarcoidosis may be diagnosed in these cases in the long term[10] or that this entity is perhaps a subset of sarcoidosis.[7] It was noted that when there was complete excision of the affected tattoo, ocular symptoms completely resolved without medication,[4] an option not possible where large areas are affected.

Figure 2. Induration (cutaneous reaction) of a black chest tattoo.

The inflammatory response can be difficult to control and many patients have suffered potentially vision-threatening ocular complications such as seclusio pupillae, iris bombe, OHT (often refractory), uveitic glaucoma, pupillary membranes, severe CMO, elevated and hyperaemic optic nerves with papillomacular exudates and retinal detachment.[4]

As shown, in addition to topical, periocular and systemic steroids, systemic immunosuppressants may be required. Reactivation is common on tapering steroids. Regular ophthalmic review and blood tests to watch for serious side effects are required. Raised IOP due to uveitic mechanisms and/or topical steroid use may require topical glaucoma drops. Traditional glaucoma surgery can have poor outcomes in inflamed eyes. Drainage tubes have been required due to refractory OHT or secondary glaucoma.

The following questions need to be asked of anybody presenting with uveitis: do you have tattoos and if so, are they inflamed, red or lumpy? Those presenting with old KPs need to be asked in regard to tattoos and previous ocular/cutaneous reactions.

Tattoo parlours should make their customers aware of cutaneous reactions which may be a precursor to ocular problems and that an urgent eye check is required with symptoms such as ocular pain, photophobia, redness and blurred vision.

Immediate treatment may reduce the severity and sequelae of uveitis which may require laser, surgery and medications with possible serious side effects.

The author would like to acknowledge Dr Catrin Bertalot (Medical Retinal Fellow, RVEEH) for her advice and Matthew Ayres (Medical Photographer, RVEEH) for the photographic images.

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Envisioning the future with Novartis

Novartis is reimagining the treatment and prevention of visual impairment and blindness. By working to push the boundaries of medicine and technology, we aim to develop life-changing gene therapies, next-generation pharmaceuticals, and transformative technologies for diseases and conditions spanning every area of eye disease, from the front to the back of the eye.

In Australia, Novartis offers one of the industry's largest and most diverse ophthalmology pharmaceuticals portfolios,[2] and is the industry's largest investor in clinical trials across Australia:[3]

- 14 ophthalmology trials sponsored in the last 3 years alone[4]
- 100,000 blind or vision-impaired Australians' lives touched by Novartis in 2017–18[4]
- Proud hosts of the Australian annual *Future Directions in Ophthalmology* symposium since 2014

Envisioning improved diagnosis for people with wet AMD

Anti-VEGF injections have revolutionised the treatment of wet (neovascular) age-related macular degeneration (AMD).[5] Australian wet AMD patients treated with anti-VEGF maintain their starting level of vision for an average of 6 years.[6]

However, there's still more to do. More than 1 in 2 patients with wet AMD did not receive anti-VEGF treatment in 2017, putting them at serious risk of losing their vision.[7,8]

This undertreatment is largely driven by:

- 1 in 4 people with AMD undiagnosed[9]
- 1 in 3 people discontinuing anti-VEGF treatment in the first year[8]

At Novartis, we're working to eliminate wet AMD-related vision impairment and blindness

Novartis has partnered with Macular Disease Foundation Australia to launch *See What's Next,* a public awareness campaign to support patients with wet AMD.[1]

Over the past few weeks, we've been running an awareness and education campaign to encourage people at risk of AMD to see their optometrist for an eye exam.

Together, we can help improve diagnosis of AMD and ensure patients receive the care they need to maintain their vision.

If you'd like to order free materials to help increase awareness of wet AMD in your practice, scan the QR code or **visit medhub.com.au/see-whats-next**

SEE WHAT'S NEXT[1]

Macular Disease Foundation Australia (MDFA) and Novartis Pharmaceuticals Pty Ltd are working together on the common goal to raise awareness of age-related macular degeneration in the community. See What's Next is an awareness campaign developed by Novartis. By supporting this campaign MDFA is not endorsing any specific treatment or therapy. **Abbreviations:** AMD: age-related macular degeneration; VEGF: vascular endothelial growth factor. **References: 1.** Optometry Australia. 2019 Clinical Practice Guide for the diagnosis, treatment and management of Age-Related Macular Degeneration. **2.** Novartis data on file. IMS ophthalmology market sales, May 2019. **3.** Austrade. Clinical Trials Capability Report 2018. **4.** Novartis data on file. **5.** Al-Zamil W, Yassin S. Recent developments in age-related macular degeneration: a review. *Clin Interv Aging* 2017;12:1313–30. **6.** Gillies M *et al.* Long-term outcomes of treatment of neovascular age-related macular degeneration: data from an observational study. *Ophthalmology* 2015;122:1837–45. **7.** Macular Disease Foundation Australia. Macular degeneration research update. Dec 2017. **8.** Drug Utilisation Sub-Committee (DUSC). Ranibizumab and aflibercept: analysis of use for AMD, DMO, BRVO and CRVO. May 2018. **9.** Neely DC *et al.* Prevalence of Undiagnosed Age-Related Macular Degeneration in Primary Eye Care. *JAMA Ophthalmol* 2017;135:570–75. Novartis Pharmaceuticals Australia Pty Limited ABN 18 004 244 160. 54 Waterloo Road, Macquarie Park NSW 2113. Ph (02) 9805 3555. September 2019. AU-10075 NOBR17308WP. Ward6.

NOVARTIS

PBS list of medicines prescribed by optometrists

Revised November 2019

Note: To satisfy PBS criteria for combination antiglaucoma agent, patient must have been inadequately controlled with monotherapy

	Product	Max qty	Repeats
ANTI-GLAUCOMA PREPARATIONS			
Betaxolol eye-drops, solution, 5 mg (as hydrochloride)/mL, (0.5%), 5 mL	Betoptic, BetoQuin	1	5
Bimatoprost eye-drops 300 mcg/mL (0.03%), 3 mL	Lumigan, Bimatoprost Sandoz, Bimtop, APO-Bimatoprost	1	5
Bimatoprost eye-drops 300 mcg/mL (0.03%) 30 x 0.4 mL unit doses	Lumigan PF*	1	5
Bimatoprost with timolol eye-drops containing bimatoprost 300 mcg/mL (0.03%) with timolol 5 mg (as maleate)/mL (0.5%), 3 mL	Ganfort 0.3/5	1	5
Bimatoprost with timolol eye-drops containing bimatoprost 300 mcg/mL (0.03%) with timolol 5 mg (as maleate)/mL (0.5%), 30 x 0.4 mL unit doses	Ganfort PF 0.3/5*	1	5
Brimonidine tartrate eye-drops 1.5 mg/mL (0.15%), 5 mL	Alphagan P 1.5	1	5
Brimonidine tartrate eye-drops 2.0 mg/mL (0.2%), 5 mL	Alphagan, Enidin	1	5
Brimonidine with timolol eye-drops containing brimonidine tartrate 2 mg/mL (0.2%) with timolol 5 mg (as maleate)/mL (0.5%), 5 mL	Combigan	1	5
Brinzolamide eye-drops 10 mg/mL (1%), 5 mL	Azopt, BrinzoQuin	1	5
Brinzolamide 10 mg/mL (1%) eye-drops containing brimonidine tartrate 2 mg/mL (0.2%), 5 mL	Simbrinza	1	5
Brinzolamide with timolol eye-drops containing brinzolamide 10 mg/mL (1%) with timolol 5 mg (as maleate)/mL (0.5%) 5 mL	Azarga	1	5
Dorzolamide eye-drops 20 mg (as hydrochloride)/mL (2%), 5 mL	Trusopt, Trusamide, APO-Dorzalamide	1	5
Dorzolamide with timolol eye-drops containing dorzolamide 20 mg (as hydrochloride)/mL (2%) with timolol 5 mg (as maleate)/mL (0.5%), 5 mL	Cosopt, Cosdor, Dorzolamide/ Timolol 20/5 (AN, APO)	1	5
Latanoprost eye-drops 50 mcg/mL (0.005%), 2.5 mL	Lanpro, Latanoprost (APO, Actavis, Sandoz), Xalaprost, Xalatan	1	5
Latanoprost with timolol eye-drops containing latanoprost 50 mcg/mL (0.005%) with timolol 5 mg (as maleate)/mL (0.5%), 2.5 mL	Xalacom, Xalamol 50/5, Lantim, Latanaprost/Timolol (AN, APO, Sandoz)	1	5
Pilocarpine eye-drops containing pilocarpine hydrochloride 10 mg/mL (1%), 15 mL	Isopto Carpine	1	5
Pilocarpine eye-drops containing pilocarpine hydrochloride 20 mg/mL (2%), 15 mL	Isopto Carpine	1	5
Pilocarpine eye-drops containing pilocarpine hydrochloride 40 mg/mL (4%), 15 mL	Isopto Carpine	1	5
Tafluprost eye-drops 15 mcg/ml (0.0015%) 30 x 0.3mL unit doses	Saflutan*	1	5
Timolol eye-drops 5 mg (as maleate)/mL (0.5%), 5 mL	Timoptol	1	5
Timolol eye-drops (gellan gum solution) 5 mg (as maleate)/mL (0.5%), 2.5 mL	Timoptol XE	1	5
Travoprost eye-drops 40 mcg/mL (0.004%), 2.5 mL	Travatan	1	5
Travoprost with timolol eye-drops containing travoprost 40 mcg/mL (0.004%) with timolol 5 mg (as maleate)/mL (0.5%), 2.5 mL	Duotrav	1	5

* Unit doses

	Product	Restriction	Max qty	Repeats
ANTI-VIRAL EYE PREPARATIONS				
Aciclovir eye ointment 30 mg/g (3%), 4.5 g	AciVision	Restricted: Herpes simplex keratitis	1	0

PBS list of medicines prescribed by optometrists

Revised November 2019

	Product	Restriction	Max qty	Repeats
ANTIBIOTICS				
Chloramphenicol eye-drops 5 mg/mL (0.5%), 10 mL	Chlorsig	Restricted: For treatment of patients identifying as Aboriginal or Torres Strait Islander	1	2
Ciprofloxacin† eye-drops 3 mg /mL (0.3%), 5 mL	CiloQuin, Ciloxan	Authority required: bacterial keratitis	2	0
Framycetin sulfate eye-drops 5 mg/mL (0.5%), 8 mL	Soframycin		1	2
Gentamicin eye-drops 3 mg/mL (0.3%), 5 mL	Genoptic	Restricted: Suspected pseudomonal eye infection	1	2
Ofloxacin† eye-drops 3 mg/mL (0.3%), 5 mL	Ocuflox	Authority required: bacterial keratitis	2	0
Tobramycin eye-drops 3 mg/mL (0.3%), 5 mL	Tobrex	Restricted: Suspected pseudomonal eye infection	1	2
Tobramycin eye ointment 3 mg/g (0.3%), 3.5 g	Tobrex	Restricted: Suspected pseudomonal eye infection	1	0
†NOTE: must be in consultation with an ophthalmologist				
ANTI-INFLAMMATORY AGENTS				
Dexamethasone eye-drops 1 mg /mL (0.1%), 5 mL	Maxidex		1	0
Fluorometholone eye-drops 1 mg/mL (0.1%), 5 mL	FML Liquifilm		1	0
Fluorometholone acetate eye-drops 1 mg/mL (0.1%), 5 mL	Flarex		1	0
Hydrocortisone acetate eye ointment 10 mg/g (1%), 5 g	Hycor		1	0
Prednisolone acetate with phenylephrine hydrochloride eye-drops 10 mg-1.2 mg/mL (1%-0.12%), 10 mL	Prednefrin Forte	Restriction: Uveitis	1	0
TEAR SUPPLEMENTS		Restricted: Severe dry eye including Sjögren's syndrome		
Carbomer 980 eye gel 2 mg/g (0.2%), 10 g	Optifresh eye gel	As above	1	5
	PAA	As above	1	5
	Viscotears	As above	1	5
Carmellose sodium eye-drops 5mg/mL (0.5%) with glycerol 9 mg/mL (0.9%), 15ml	Optive	As above	1	3
Carmellose sodium eye-drops 10 mg/mL (1%), 15 mL	Refresh Liquigel	As above	1	5
Carmellose sodium eye-drops 5 mg/mL (0.5%), 15 ml	Refresh Tears plus	As above	1	5
Hypromellose eye-drops 3 mg/mL (0.3%), 15 mL (contains sodium perborate)	In a Wink Genteal	As above	1	5
Hypromellose eye-drops 5 mg/mL (0.5%), 15 mL	Methopt	As above	1	5
Hypromellose 3 mg/mL (0.3%) with carbomer 980 2 mg/g (0.2%) ocular lubricating gel, 10 g	HPMC PAA Genteal Gel	As above	1	5
Hypromellose 3 mg/mL (0.3%) with dextran eye-drops 1 mg/mL (0.1%), 15 mL	Poly-Tears, Tears Naturale	As above	1	5
Polyethylene glycol 400 mg/mL (0.4%) with propylene glycol 3 mg/mL (0.3%) eye-drops, 15 mL	Systane	As above	1	5
Polyvinyl alcohol eye-drops 14 mg/mL (1.4%), 15 mL	PVA Tears, Liquifilm Tears	As above	1	5

PBS list of medicines prescribed by optometrists

Revised November 2019

	Product	Restriction	Max qty	Repeats
UNPRESERVED TEAR SUPPLEMENTS**		Authority required: streamlined		
Carbomer 974 ocular lubricating gel 3 mg/g (0.3%), single dose units 0.5 g x 30	Poly Gel	Severe dry eye syndrome in patients sensitive to preservatives in multi-dose eye-drops	3	5
Carbomer 980 eye gel 2 mg/g (0.2%), single dose units 0.6 mL x 30	Viscotears Gel PF	As above	3	5
Carmellose sodium eye-drops 5 mg/mL (0.5%), single dose units 0.4 mL x 30	Cellufresh Optifresh Tears	As above	3	5
Carmellose sodium eye-drops 10 mg/mL (1%) , single dose units 0.4 mL x 30	Celluvisc Optifresh Plus	As above	3	5
Hypromellose 3 mg/ mL (0.3%) with dextran eye-drops 1 mg/mL (0.1%), single dose units 0.4 mL x 28	Bion Tears	As above	3	5
Perfluorohexyloctane eye-drops (100%), 3mL	NovaTears	As above	1	5
Polyethylene glycol 400, 4 mg/mL (0.4%) with propylene glycol 3 mg/mL (0.3%) eye-drops, single dose units 0.8 mL x 28	Systane	As above	2	5
Sodium Hyaluronate sodium hyaluronate eye-drops 1 mg/mL (0.1%), 10 mL	Hylo-Fresh	As above	1	5
Sodium Hyaluronate sodium hyaluronate eye-drops 2 mg/mL (0.2%), 10 mL	Hylo-Forte	As above	1	5
Soy Lecithin 1% + tocopherol 0.002% + vitamin A palmitate 0.025% eye spray, 100 actuations	Tears again	As above	2	5
TOPICAL OCULAR LUBRICANT OINTMENTS				
Paraffin 1 g/g compound eye ointment 3.5 g	Polyvisc		2	5
Paraffin 1 g/g pack containing 2 tubes eye ointment 3.5 g	Polyvisc (2 pack), Ircal (2 pack), Refresh Night Time (2 pack)		1	5
Paraffin paraffin + retinol palmitate 138 mcg/g (0.0138%) (equivalent to 250 units/g vitamin A) eye ointment, 5 g	VitA-POS		2	5

Optometrists have two **Streamlined Authority Codes for unpreserved tear supplements:
4105 Hylo-Fresh and Hylo-Forte, and **6172** all other unit-dose ocular lubricants

Ophthalmic compounding pharmacists

This list is intended to assist the members of Optometry Australia and is not an endorsement of any of the pharmacists listed below. If you know a certified ophthalmic compounding pharmacist, please email us at pharma@optometry.org.au and we will add it to future published lists.

NEW SOUTH WALES

CustomCare Compounding Pharmacy
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Email: info@customcarepharmacy.com.au
www.customcarepharmacy.com.au

Stenlake Compounding Chemist
Bondi Junction, NSW
Tel: (02) 9387 3205
Email: info@stenlake.com.au
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QUEENSLAND

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Email: info@yoursolutioncompounding.com.au
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Email: compounding@greendispensary.com.au
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If you have CONTENTS or STOCK	✓
If you could not maintain normal business operation if you were to suffer a major loss at your business	✓
If you are RESPONSIBLE to insure LEASED equipment	✓
If you have staff who visit worksites with expensive equipment	✓

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