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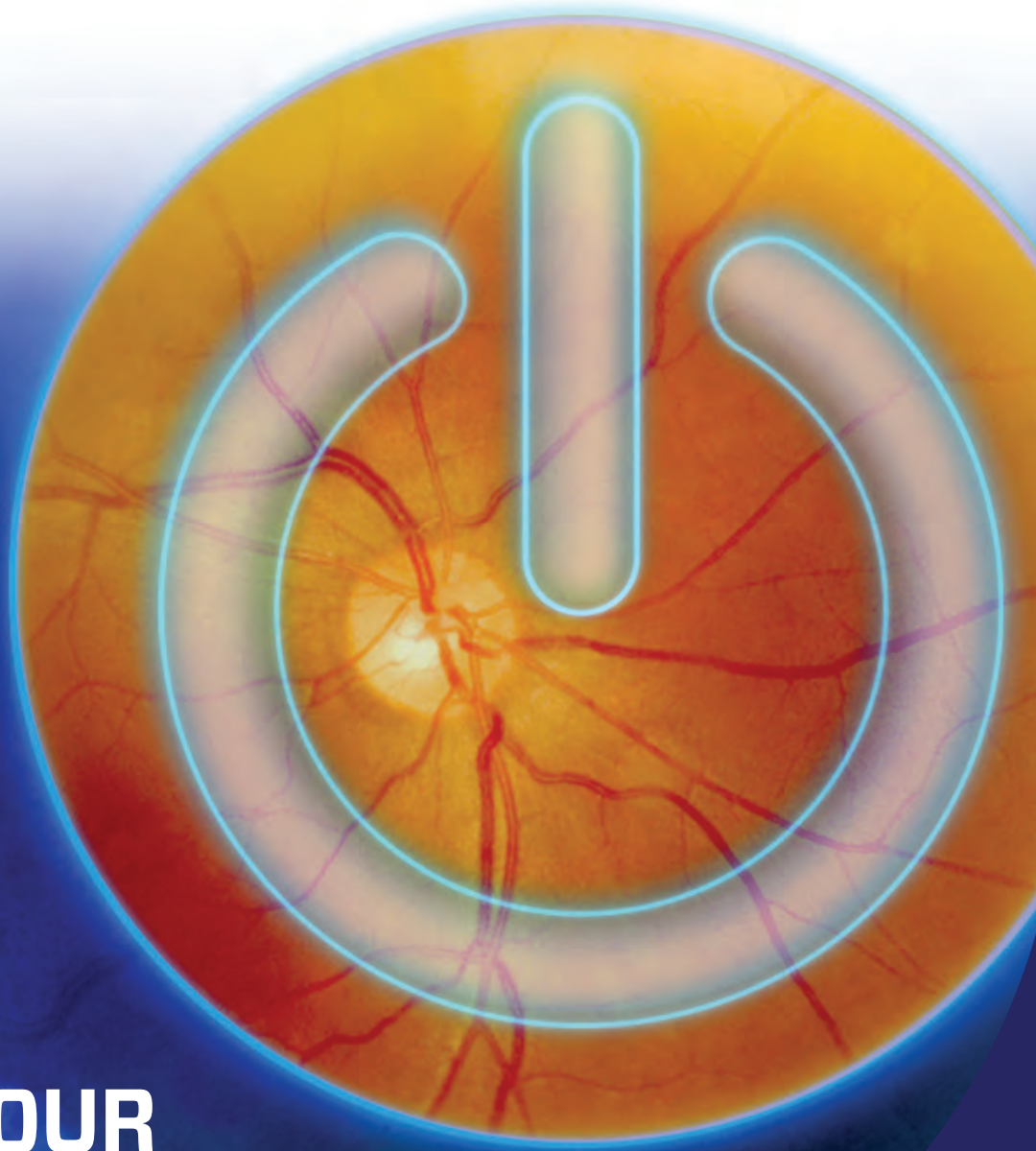

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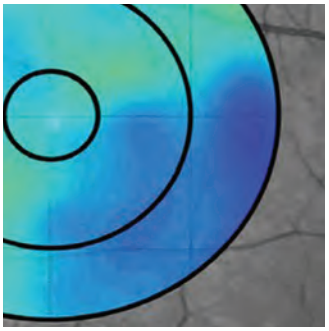

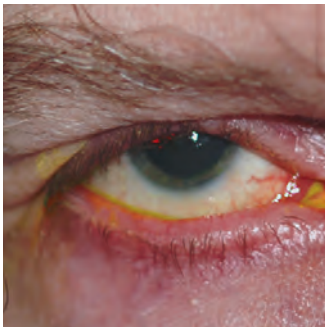

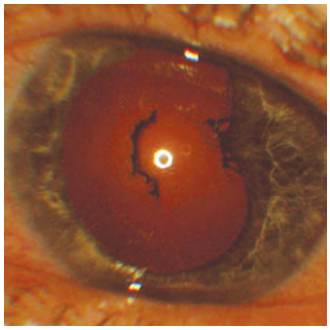

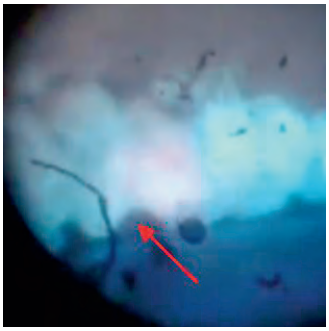

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Evidence-based lifestyle modifications for progressing myopes

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MYOPIA is widely considered to result from a combination of genetic and environmental influences.[1,2] While little can currently be done in regard to one's genetic susceptibility to myopia, it is possible that modification of environmental contributors may help to control the development and progression of myopia before it advances to pathological levels.

Two of the major environmental factors considered to play a role in myopia include time outdoors and near-work activities. Recent developments in technology to quantify the visual environment and measure ocular parameters have provided novel insights regarding these factors and provide an evidence base for lifestyle modifications to potentially reduce myopia risk.

In recent years, an important role for outdoor activity in myopia has emerged, and while the majority of studies indicate that spending more time outdoors protects against the development of myopia, the exact mechanism underlying these effects is not as clear.[3,4]

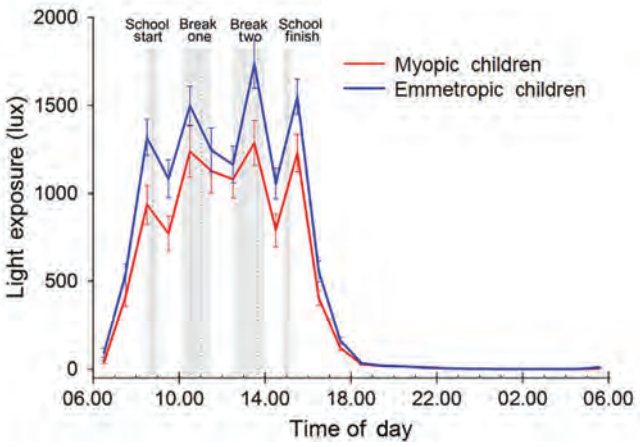
ROAM study results

In the role of outdoor activity in myopia (ROAM) study, our research team utilised wearable sensor technology to examine for the first time the relationship between myopia (and axial eye growth) in childhood and objective measures of ambient light exposure and physical activity, providing new insights into the mechanisms underlying the protective effects of spending more time outdoors.[5,6]

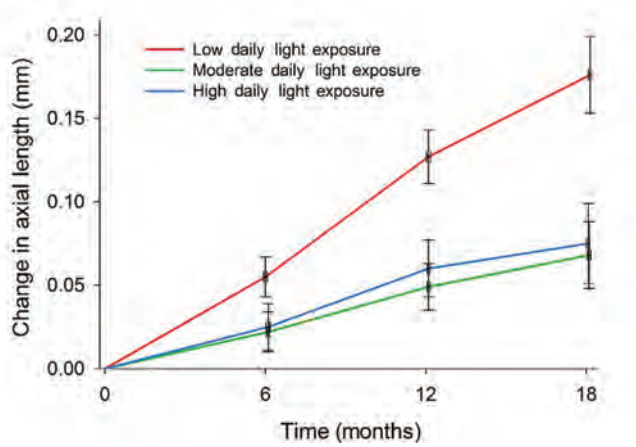
This study of myopic and emmetropic Brisbane school children revealed no significant differences in the daily physical activity of myopes and emmetropes but did find that myopic children spent significantly less time each day exposed to bright outdoor light levels (Figure 1). This suggests that it is the effect of bright light itself, rather than being active outdoors per se, that appears to impart the protective effect against myopia. Analysis of axial eye growth in this population revealed that irrespective of refractive group, those children who experienced the lowest average daily light exposure had the fastest rate of eye growth (Figure 2).[6] Children who spent less than 60 minutes per day exposed to bright light (> 1000 lux) were found to exhibit significantly faster axial eye growth, and therefore were at greater at risk of myopia development and progression. In contrast, children spending on average 120 minutes per day in bright outdoor light levels were found to show slower axial eye growth.

Near work and myopia

The duration and intensity of near work activities have also often been linked to myopia, with a number of



▲ Figure 1. The average daily light exposure of myopic and emmetropic children in the ROAM study, measured with wrist-watch light sensors worn by each child in the study for two 14-day periods. Note the significantly greater light exposure for emmetropic children during the day.[5]



▲ Figure 2. The average axial eye growth over 18 months for the children in the ROAM study stratified according to their average daily light exposure, irrespective of refractive group. Children habitually exposed to low daily light exposure exhibited significantly faster axial eye growth.[6]

studies finding myopia development associated with greater amounts of near work.[7-10] These findings have prompted numerous investigations into the influence of accommodation on ocular parameters. Recent studies from the QUT Contact Lens and Visual Optics Laboratory, utilising highly precise measures of eye length and ocular parameters such as choroidal thickness, have revealed that accommodation results in a small, transient increase in eye length and a thinning of the choroid (Figure 3).[11,12]

Interestingly, in emmetropic eyes, these changes are found to return to normal immediately after the near task is ceased, but in myopes this increased axial length appears to linger following task cessation, taking up to 10 minutes after accommodation is relaxed to return to baseline.[12,13] Higher accommodative demands (6D) are accompanied by larger magnitudes of axial elongation,[11,14] choroidal thinning[14] and anterior scleral thinning[15] in the eyes of young adults. This may indicate that in individuals who perform large amounts of intensive near work, particularly those who don't take sufficient visual breaks, these temporary structural changes will occur more frequently and for longer periods, which may predispose the eye to longer-term structural changes, such as choroidal thinning which is known to be associated with myopia (Figure 4).[16]

These studies provide some theory for evidence-based guidelines to provide to myopic children and their parents regarding lifestyle interventions for myopia control. Children should be encouraged to increase outdoor light exposure to 120 minutes or more per day, regardless of whether this is associated with being physically active. The continued use of sun protection in the form of hats and sunglasses is recommended. In terms of near-work behaviours, because myopic eyes appear to have prolonged recovery times from the ocular effects of intensive near work, frequent visual breaks (a 10-minute break after every 30 minutes of near work) are recommended when performing prolonged near-work tasks. ▲

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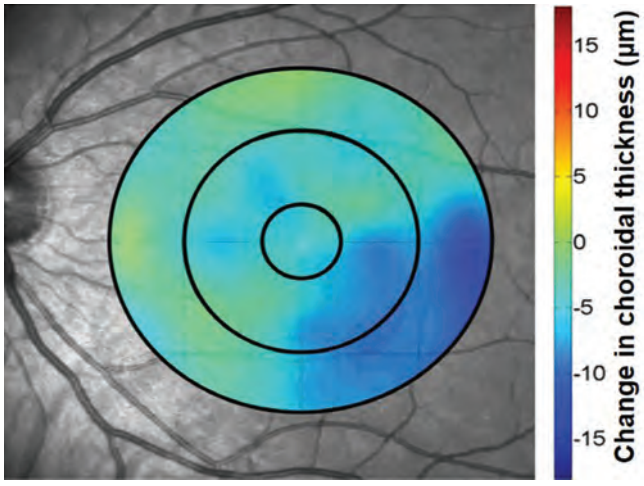
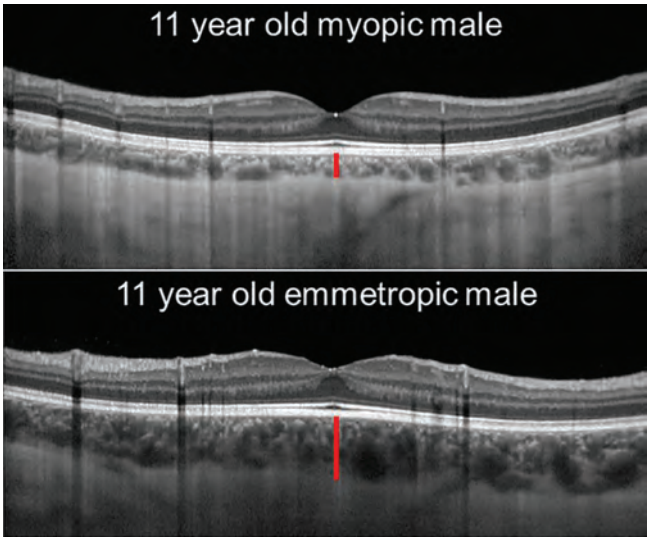


▲ Figure 3. The average change in choroidal thickness (derived from OCT imaging) associated with a 10-minute period of near work (6D accommodation). On average a thinning of the choroid up to ~10 microns was observed.[14]



▲ Figure 4. Example of the longer-term changes in choroidal thickness (denoted by red line in OCT scans) associated with myopia. Note the significant thinning of the choroid evident in the 11-year-old myopic child.

0.01% atropine for myopia control

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Childhood myopia

BROTHERS A and B have a strong family history of myopia. While their mother's myopia is moderate (-3.50 DS), their father's myopia is very high (-17.00 D). Both boys had onset of myopia initially identified at approximately seven years of age and were prescribed dual focus soft contact lenses as myopic progression treatment.

In 2014, the older brother's myopia showed a marked progression over a six-month period, prompting the prescribing of 0.01% atropine, one drop, both eyes, at night. Before commencing 0.01% atropine, brother A's myopic refractive error had increased from -0.50 DS to -4.75 DS over four years, with rate of progression approximately -1.00 D/year. Progression rate slowed to -0.38 D/year following commencement of 0.01% atropine treatment (-4.75 DS to -5.50 DS over two-year interval).

Younger brother B was showing a comparable myopia progression profile, with onset at a similar age to that of his brother who preceded him by three years. Prior to commencing 0.01% atropine, brother B was becoming more myopic at a rate of -1.25 D/year in the right eye and -0.25 D/year in the left eye. Following commencement of atropine 0.01% treatment, progression rate reduced to -0.25 D/year in both eyes, with current myopic refractive error R -2.25/-0.50x90 L -1.50 DS.

The prevalence of myopia globally has rapidly increased to disturbing levels in recent decades, a finding that is concerning due to the increased risk of associated ocular pathology such as cataracts, glaucoma and retinal detachment.[1,2] The prevalence of myopia is alarmingly high in various Asian countries, where 70-80 per cent of young adults are significantly myopic.[4] This trend is also apparent in Australia with a recent report that 31 per cent of 17-year-olds are myopic.[3]

Risk factors for the development of myopia have been identified, which are useful tools in clinical practice for identifying at-risk patients. While the most significant predictor of myopia is a lower hyperopic refraction at baseline, less time spent outdoors, increased near work, family history and East-Asian ethnicity are all identified as risk factors for incident myopia.[2,3]

In recent years, there has been a shift in clinical management, especially of young progressing myopes with a strong family history of myopia. Previously, mainstay treatment for myopia was aimed at correcting myopic refractive error to maximise distance visual acuity without controlling progression. Now, treatment has shifted to other forms of correction or management protocols that are designed to not only correct visual acuity but also control or minimise myopic progression.[2]

Treatment options that have been shown to slow continued myopia development include progressive addition lenses, dual focus contact lenses, orthokeratology and low dose atropine.[5,6] Topical antimuscarinic medication such as atropine and pirenzepine appears to be the most effective treatment for controlling myopia.[5,6]

Eyes treated with atropine have statistically significant reduced myopia progression and less axial elongation compared to controls.[7] Initial studies reported the mean myopic progression after one year in the 1% atropine treatment group was significantly less than in controls (+0.3 D ± 0.50 D compared to -0.76 D ± 0.44 D [$p < 0.0001$]);[7] after two years the mean myopic progression in the 1% atropine treatment group was -0.25 D ± 0.92 D compared to -1.20 D ± 0.69 D in controls.

Further studies to investigate the minimum dose required to effectively minimise myopic progression over five years demonstrated that 0.01% atropine was the most effective concentration in slowing myopia progression with fewer side-effects and less rebound progression compared to higher doses.[8] Over five years the mean myopic progression was -1.38 ± 0.98 D in the 0.01% atropine group compared to -1.83 ± 1.16 D in the 0.1% group and -1.98 ± 1.10 D in the 0.5% group. Over five years, the mean change in axial length was least in the 0.01% group (+0.19 ± 0.18 mm) compared to other concentrations.[8]

Overall, it appears that 0.01% atropine slowed myopia progression by at least 50 per cent.

Pharmacology

Atropine is a belladonna alkaloid that blocks the action of acetylcholine. As a non-selective muscarinic antagonist, atropine blocks the M1-M5 receptors found in the eye.[9] Low dose 0.01% atropine appears to have minimal effect on the M3 receptor, which is related to pupil size and accommodation.[10] Animal studies suggest that the M1 and M4 receptors are involved with myopic progression.[11]

In a study that involved deprivation-induced myopia in tree shrews, it was shown that eyes that were injected with M1 and M4 blocking compounds displayed a marked reduction in myopic progression compared to controls.[11] The site of action within the eye for myopic progression inhibition is still unknown but studies have postulated that it could be within the retina or sclera.[9,11]

Currently, 0.01% atropine solution is not commercially available in

Australia so it must be compounded by a pharmacy that has access to a sterile room. The compounded 0.01% atropine is diluted from a higher concentrated stock solution of atropine sulphate and then combined with BZA 0.1mg/mL. Non-active ingredients include disodium edetate, benzalkonium chloride and hypromellose, boric acid and distilled water.

To ensure you have a comprehensive understanding of the solution that you are prescribing, it is important to talk to the compounding pharmacy about any other non-active ingredients that are added to the compound. Compounded 0.01% atropine solution has a 30-day life after opening. A non-preserved solution is available, depending on the pharmacy, but it has a very short shelf-life of seven days.

Use of 0.01% atropine for myopia control treatment

0.01% atropine is intended to be used in conjunction with spectacle or contact lens correction. One drop of atropine 0.01% should be instilled in the specified eye(s) at night before bed. While shown to be effective in slowing myopia progression in the short term, the long-term use of 0.01% atropine is still not completely understood. After ceasing atropine following 24 months of use, some patients demonstrated rebound myopia, with the rebound effect more pronounced in patients who had been treated with higher initial concentrations of atropine (0.5% and 0.1%).[8] However, the rebound effect was linked to the dose of atropine with minimal rebound in the 0.01% group.[8]

Clinical implications

- 0.01% atropine is an effective way to control myopia and is a good alternative to dual focus contact lenses or orthokeratology lenses, especially in young patients.[5]
- Daily administration of 0.01% atropine is required for myopia control. One drop of 0.01% atropine should be instilled at night before bed in the specified eye(s).
- If atropine treatment is ceased, some patients will demonstrate a rebound effect so they must be monitored for signs of progression.[8]
- Atropine can be used in conjunction with other myopia control techniques such as dual focus contact lenses. The combined effect is unknown.
- 0.01% atropine is a compounded solution. For a patient to obtain the medication, a valid prescription must be sent to the compounding pharmacy. The compounding process is initiated after the parent or carer contacts the pharmacy to verify the order, and confirm payment and delivery.
- 0.01% atropine has a use-by date of 90 days as an unopened product or 30 days after opening the bottle. Different compounding pharmacies may have different recommendations: verify this with the compounding pharmacist.
- Approximate cost is $130-$150 for three bottles, plus courier fee. The cost of the product will vary depending on the compounding pharmacy and the collection/delivery options.
- To increase compliance, discuss and provide a written instruction sheet that includes ordering, administration, storage and potential adverse effects.

Ocular and systemic side-effects

0.01% atropine appears to be very well tolerated with no known serious adverse effects.[10] Ocular side-effects of increase in pupil size and responsiveness have been reported.[10] While glare was reported during trials, there was no impact on overall quality of life with the use of the medication.[10]

At slightly higher doses of atropine, some patients have developed associated ocular allergies.[9] Systemic side-effects of atropine include mucous membrane dryness, flushing of skin, confusion, unusual behaviour and irritability. These side-effects are rare and related to higher doses of the medication than the dose prescribed for myopia control.

Current legislation

The current legislation in Queensland, the Health (Drugs and Poisons) Regulations 1996 (QLD), stipulates that therapeutically endorsed optometrists can prescribe only preparations listed on the Australian Register of Therapeutic Goods. The Department of Health in Queensland has indicated that optometrists cannot prescribe compounded medications. This legislation is in the process of being reviewed.

Summary

Given the increasing prevalence of myopia and the associated ocular complications, strategies must be implemented to reduce myopic progression. 0.01% atropine is an effective way to control myopia and is a good alternative to other treatment options. Low dose atropine appears to be very well tolerated without serious adverse effects.[10] ▲

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Red eye acute anterior segment presentation

Paige Lynch

BVisSc MOptom

UVEITIS is characterised by inflammation of the uveal tract, which comprises the iris, ciliary body and choroid.[1,2] It can be classified based on the predominant anatomical site of inflammation into variously anterior, intermediate, posterior or panuveitis.[1,3,4] A prevalence of 115 uveitis cases per 100,000 persons has been reported.[5]

Anterior uveitis (AU) accounts for 60-90 per cent of reported uveitis episodes, making it the most common type.[6] Highlighting the potential severity of the disease is the fact that uveitis accounts for 10-15 per cent of visual disability in working-age persons.[7]

AU can be acute (sudden onset and limited), chronic (relapse in less than three months of treatment) or recurrent (repeated episodes; inactive periods on or after three months of treatment).[3] Of these, acute anterior uveitis (AAU) is the most common form.[6] AAU is a frequent cause of a unilateral red eye with presenting symptoms of photophobia, lacrimation, possible blurring of vision and a dull ocular pain enriched by accommodative effort.[1,2,6,8,9] Signs vary depending on the degree of inflammation and are listed in Table 1.

AU aetiology may be infectious, autoimmune, malignant, traumatic or idiopathic.[1,2,4,6] Approximately 50 per cent of AU cases have an idiopathic mechanism, which forms the most common aetiology.[5,6] The propensity for AAU to have an immune origin is reflected in the high occurrence of systemic conditions among AAU sufferers.[10,11]

Both genetic predisposition and environmental factors are thought to be involved in AAU pathogenesis.[12]

The goals of AAU treatment are to provide symptomatic relief, promptly eliminate inflammation and prevent the development of sight-threatening ocular sequelae.[6,9] This is typically achieved by use of a topical corticosteroid and cycloplegic/mydriatic agent;[6,12] however, it is imperative to rule out an infectious or neoplastic cause, as corticosteroid therapy may have deleterious effects in such cases.[12]

CASE REPORT

A 56-year-old Caucasian male presented to a rural optometry practice with a five-day history of an irritated, sore and red left eye. He had no significant ocular history, no general health issues and was not taking any systemic medications. The patient denied back pain, joint pain or bowel issues and was unaware of his HLA-B27 status.

INITIAL PRESENTATION DAY 1

Ophthalmic examination

The patient's BCVA was OD 6/6, OS 6/9.5. His intraocular pressure (IOP) at 1:00 pm was OD 21 mmHg and OS 16 mmHg. A slitlamp examination of his left eye revealed moderate circumlimbal injection and moderate to severe diffuse bulbar conjunctival hyperaemia (Efron Grading Scale), Grade 4+ anterior chamber cells and 1+ anterior chamber flare, as per SUN grading.[3]

The pupil margin was irregular and demonstrated posterior synechiae at 2-3 and 7-10 o'clock. A few non-granulomatous keratic precipitates were present. The anterior chamber angles were open on Van Herick OU and no corneal NaFl staining was present. No discharge, hypopyon, iris nodules or iris trans-illumination defects were noted.

Bulbar conjunctiva	Circumlimbal injection
Cornea	Keratic precipitates (lymphocyte accumulations on the endothelium)
	Oedema
Aqueous	Cells (inflammatory cellular infiltration) and flare (protein exudation)
	Hypopyon
Pupils	Myotic, irregular pupil
Iris	Posterior synechiae
	Peripheral anterior synechiae
	Iris atrophy
	Iris nodules
Lens	Inflammatory debris on the anterior lens capsule
	Cataract
Intraocular pressure	Generally reduced (due to ciliary body inflammation and subsequent diminished aqueous production)
	Occasionally may be elevated

Adapted from Gutteridge et al[6] and Agrawal et al[9]

▲ Table 1. AAU clinical signs

Slitlamp examination of the right eye was unremarkable. No posterior involvement was observed on dilated fundus examination.

Diagnosis

Given the presentation of an acute, unilateral, sore and red left eye and clinical findings of anterior chamber cells and flare, circumlimbal injection, posterior synechiae and reduced OS IOP, a diagnosis of OS AAU was provided. Because the patient had no systemic symptoms (back or joint pain, bowel issues, skin rashes), no known systemic conditions, his HLA-B27 status was unknown, and he reported no history or signs suggestive of an infectious cause, an idiopathic mechanism was presumed.

Management

The patient was prescribed Prednefrin Forte eye-drops (prednisolone acetate 1%, phenylephrine 0.12% suspension); loading dose every 20 minutes for one hour, then every 30 minutes for four hours, then every hour during waking hours. As well, he was prescribed Isopto Homatropine 2% every 30 minutes for first hour, then four times a day and one drop phenylephrine 2.5%.

After one hour of therapy, the 7-10 o'clock posterior synechiae had broken (Figure 1). The patient's condition was scheduled to be reviewed on the following day.

Differential diagnoses

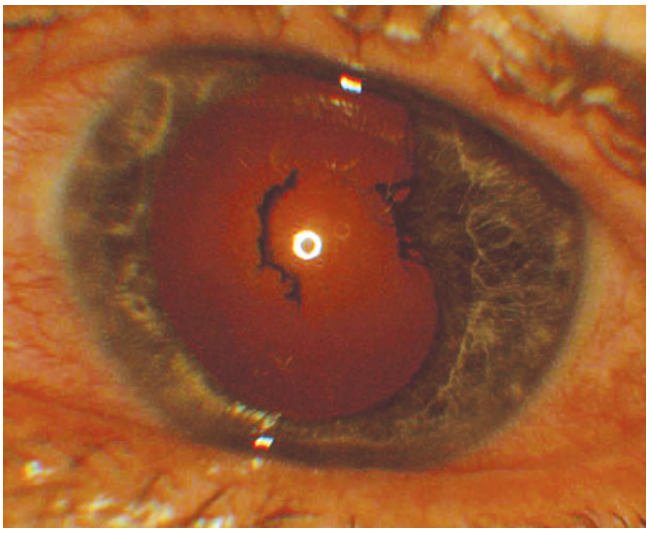
- AAU of infectious origin, such as herpetic uveitis (herpes simplex virus or varicella zoster virus. However, IOP was not raised, iris transillumination defects were absent, there was no previous history of an ocular infection and no skin rashes or vesicles were present. Reconsider if worsening with therapy.

- HLA-B27-associated AAU, given that the patient was male and the AAU demonstrated significant anterior chamber cells and flare. However, HLA-B27 status was unknown, there was no known underling seronegative spondyloarthropathy, no symptoms suggestive of back or joint pain, or skin rashes or plaques, and no history of a previous AAU attack.

- Microbial keratitis, due to the unilateral red eye and anterior chamber reaction. However, no epithelial defect or corneal ulcer, infiltrate, hypopyon or mucopurulent discharge was present and there was no associated contact lens wear or other predisposing factor(s).

- Acute angle closure, given the acute unilateral red eye, circumlimbal injection and posterior synechiae. However, IOP in the affected eye was reduced.

- Scleritis, due to the symptom of ocular pain, vascular congestion and the anterior chamber reaction. However, the vascular injection was superficial and lacked a violaceous hue, IOP was reduced and there was no history or symptoms suggestive of a systemic connective tissue disease.



▲ Figure 1. OS anterior eye, demonstrating circumlimbal injection, posterior synechiae at 2-3 o'clock and resolved posterior synechiae at 7-10 o'clock

OS REVIEW, DAY 2

The patient reported symptomatic improvement. His IOP was OD 20 mmHg and OS 23 mmHg. The patient had a slight reduction in OS circumlimbal injection and bulbar conjunctival hyperaemia, resolution of the 2-3 o'clock posterior synechiae, reduction in anterior chamber cells to Grade 3+ and resolution of the fibrin (Grade 0) (SUN grading).[3] There was no posterior involvement.

Management

The patient was prescribed Prednefrin Forte 1% every hour until the end of day, then reduced to every two hours as well as Homatropine 2% four times a day. The patient was scheduled for review in two days.

OS REVIEW, DAY 4

On the four-day follow-up, the patient's IOP was: OD 20 mmHg and OS 27 mmHg. There was a resolution of OS ciliary injection and bulbar conjunctival hyperaemia, Grade 1+ anterior chamber cells, Grade 0 fibrin (SUN grading).[3] There was no posterior involvement.

Management

The patient was prescribed Prednefrin Forte 1% every three hours, Homatropine 2% four times a day until end of day, then cease. If IOP reached 30 mmHg, add a topical ocular hypotensive agent. The patient was scheduled for review in two days.

OS REVIEW, DAY 6

On the six-day follow-up, the patient's BCVA: was OD 6/6; OS 6/7.5; IOP was: OD 19 mmHg; OS 20 mmHg (non-contact tonometer) and OS grade 0.5+ anterior chamber cells, Grade 0 fibrin (SUN grading).[3]

Management

The patient was prescribed Prednefrin Forte 1% four times a day, and scheduled for review in one week.

Continued page 8

Red eye acute anterior segment presentation

From page 7

DISCUSSION

Corticosteroids in AAU treatment

As AAU is an inflammatory process for which the mainstay of treatment is topical corticosteroids.[6,13,15] Corticosteroids increase the synthesis of lipocortins, which inhibit phospholipase A2. This prevents the production of arachidonic acid from phospholipids. Consequently, there is inhibition of both the cyclo-oxygenase and the lipoxygenase pathways and ultimately, the inflammatory cascade.[9,16]

Corticosteroid efficacy in AAU is based on its corneal penetration and anterior chamber potency.[6] This depends on the chemical composition of the corticosteroid and the dosing frequency.[6] The corneal epithelium is lipoidal and provides the greatest barrier to drug penetration.[15,17] Therefore, hydrophilic derivatives (prednisolone phosphate) are significantly retarded in comparison to lipophilic derivatives (dexmethasnone alcohol and prednisolone acetate).[18]

Essentially, all data relating to corticosteroid efficacy in anterior segment inflammation are extrapolated from its corneal effects.[14] It can be seen in Table 2 that among the topical corticosteroids available to Australian optometrists with a scheduled medicines endorsement, prednisolone acetate 1% yields the greatest corneal anti-inflammatory effect.

Both prednisolone acetate and dexamethasone alcohol demonstrate sufficient aqueous penetration and therefore are appropriate agents to be used for AAU treatment (Table 2).[15] It can be seen that prednisolone acetate 1% achieves the highest concentration in the anterior chamber.[15] This is attributable to its high corneal penetration. Indeed, prednisolone acetate is from 3.5 to six times more potent than dexamethasone in the anterior chamber.[14] Furthermore,

Corticosteroid	Mean % decrease in corneal inflammation*	Mean peak aqueous concentration (ng/ml)	Time to peak (min)
Prednisolone acetate 1%	51%	669.9 1130	120 30-45
Fluorometholone acetate 0.1%	48%	–	–
Dexamethasone alcohol 0.1%	40%	31	90-120
Fluorometholone alcohol 0.1%	31%	5.1	31-60
Prednisolone sodium phosphate 1%	28%	25.6#	90-240#

* Intact corneal epithelium, # Prednisolone sodium phosphate 0.5%
Adapted from Leibowitz et al,[14] Awan et al[15] and Bartlett et al[19]

▲ Table 2. Reduction in corneal inflammation and comparison of human aqueous humour concentration of corticosteroids following topical application

Prednefrin Forte also contains 0.12% phenylephrine, which is an α-adrenoreceptor agonist.[20] Therefore, it can cause vasoconstriction by stimulating α-adrenoreceptors on the conjunctival blood vessels, thereby blanching these vessels to improve cosmesis in AAU.

The frequency of corticosteroid administration depends on the degree of inflammation, with higher drop instillation frequency demonstrating greater anti-inflammatory activity. A 'loading dose' was implemented in the reported case to achieve a therapeutic concentration quicker. This is sometimes recommended;[21] however, more frequent dosing can increase the rate of significant IOP increase.[22-25]

The patient's OS IOP spiked to 27 mmHg at the second review. This IOP spike could be a result of the inflammation or it could be corticosteroid-induced (see 'AAU sequelae' below). Corticosteroids can raise IOP by increasing aqueous outflow resistance and consequently, can lead to secondary OAG.[13,15,26] However, corticosteroid-induced IOP rise typically occurs after two to eight weeks of steroid use.[19] Given the spike occurred after only three days of corticosteroid use, in addition to the fact that IOP subsequently reduced at the following review, an inflammatory mechanism was most likely (see 'AAU ocular sequelae' below).

Cycloplegic/mydriatic agents in AAU

Cycloplegic/mydriatic agents are cholinergic antagonists. The goals of cycloplegic/mydriatic use are to provide symptomatic relief and photophobia by immobilising the inflamed iris and ciliary body.[6,12,29] They are also used to break previously-formed posterior synechiae, prevent future posterior synechiae from forming and to stabilise the blood aqueous barrier.[6,12,19] Potential agents include atropine, homatropine, cyclopentolate and tropicamide.[9,12,28]

Phenylephrine is an α1-adrenergic agonist that binds α1-adrenergic receptors on the iris dilator muscle to cause mydriasis.[20] It may be used in the acute phase to exert a synergistic effect with an anti-cholinergic agent to cause maximal pupillary dilation in order to break existing posterior synechiae.[9]

It is thought that to reduce the risk of synechiae formation in the fixed dilated pupil position, a shorter-acting anti-cholinergic agent is preferred to allow a degree of pupil mobility.[2,29] Homatropine encompasses the most desirable effects. It has a duration of mydriasis and cycloplegia from five hours to four days, and from 10 hours to two days, respectively.[19,28] Recently, homatropine was discontinued. This calls for use of an alternative agent. Cyclopentolate has been reported to induce a chemokinetic neutrophil response and therefore is not recommended in active inflammatory processes.[6,30] Given this, along with its short duration of action (mydriasis: one day, cycloplegia: six to 24 hours), atropine appears to be the next best candidate for future AAU management.[28]

Cycloplegic agents may be withdrawn when flare is absent, minimal cellular reaction is present, and a low risk of synechiae forming exists.[6,9] This is documented to be typically within two weeks; however, the reported case showed a much shorter duration.[6]

AAU ocular sequelae

With appropriate treatment, the prognosis of AAU is favourable.[6]

The most common sequelae are posterior synechiae, secondary cataract, cystoid macular oedema (CMO), peripheral anterior synechiae (PAS), ocular hypertension, secondary glaucoma, band keratopathy, epiretinal membrane formation, hypotony and most devastatingly, phthisis bulbi.[1,2,6,7]

Many mechanisms exist for inducing ocular hypertension in AAU. These include trabeculitis, trabeculosclerosis, inflammatory cellular/debris accumulation in the trabecular meshwork, PAS, posterior synechiae, or corticosteroids.

It is important to monitor and treat any significantly elevated IOP. The risk of glaucomatous damage and other complications, such as retinal vein occlusion, increases at higher IOPs.[3,32,33] Practitioners in the SUN working group suggest an IOP above 30 mmHg warrants initiation of hypotensive therapy.[3] Topical hypotensive agents aiming to reduce aqueous production are typically used.[6] β-adrenoreceptor antagonists, α2-adrenoreceptor agonists and topical carbonic anhydrase inhibitors are alternative options.[6] Prostaglandin analogues pose a theoretical risk of amplifying inflammation and therefore, should typically be avoided.[6] Likewise, miotics should be used with caution as they may exacerbate blood-aqueous barrier breakdown and increase the risk of inducing posterior synechiae in the miosed position.[6,19]

HLA-B27 typing in AAU

HLA-B27 is present in approximately 50 per cent of AAU patients.[1,33,34] It also significantly increases the chance of developing a seronegative spondyloarthropathy.[34] It has been suggested that an undiagnosed spondyloarthropathy could be overlooked in about 40 per cent of patients if only recurrent AAU cases are provided onward referral.[33] Therefore, key questioning regarding HLA-B27 typing, back or joint pain, and skin rashes was appropriate to determine if onward referral was required. ▲

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Prognostic biomarkers in

Advanced imaging yields a growing

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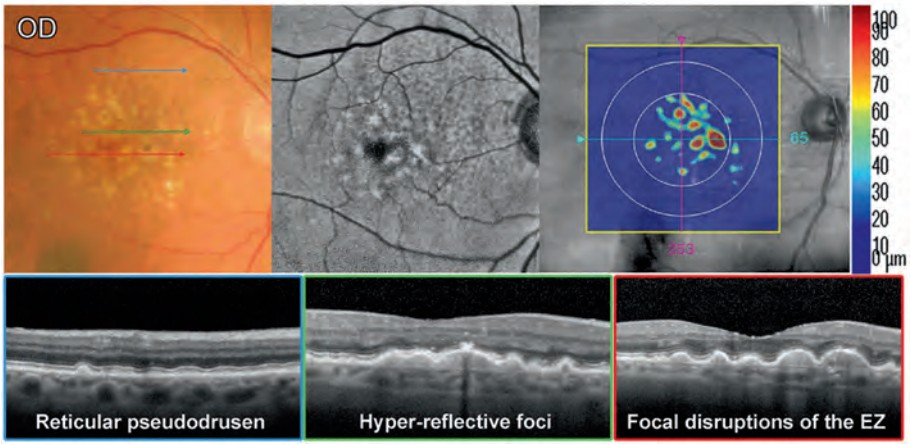


▲ Figure 1. Imaging results from the patient's baseline visit in the right eye. Top left: Colour fundus photograph, Top middle: Fundus autofluorescence image, Top right: Cirrus OCT advanced RPE analysis result, Bottom: OCT B-scans extracted from a Spectralis OCT macular cube scan illustrating additional high risk signs for progression.

AGE-RELATED macular degeneration (AMD) is a leading cause of blindness in Australia.[1] As practising clinicians, we face the challenge of accurately identifying and managing as early as possible patients with or at risk of vision loss. While neovascular AMD mandates immediate referral to an ophthalmologist, determining risk of progression in earlier stages is fundamental to appropriate management.

In the past few years phenotyping of the disease has become highly refined, staging and nomenclature of the disease have been clarified and treatment options have expanded. The 2013 Beckman initiative for macular research clinical classification scale[2] recommends subdividing the disease into early, intermediate and late stages.

Late AMD represents the most advanced form associated with visual impairment due to geographic atrophy (GA) or choroidal neovascularisation (CNV). It has an estimated incidence of 6.8 per cent over 15 years in an Australian population.[3] Intermediate AMD is typically asymptomatic and may vary dramatically in presentation from one eye to the next. The hallmark signs are large drusen greater than 125 µm in diameter, or pigmentary changes within the macular area. Patients with intermediate AMD in one or both eyes hold a wide-ranging three per cent to 53 per cent risk of progression to advanced AMD in five years, depending on the combination of clinical signs present.[4]

With the advent of advanced imaging, in particular optical coherence tomography (OCT), a growing number of indicators have been associated with an increased risk of AMD progression. Practitioners should be aware of these indicators and incorporate them into routine clinical practice.

CASE REPORT

An 85-year-old Caucasian male was referred to Centre for Eye Health (CFEH) for a macular assessment due to bilateral drusen. He reported a history of bilateral cataract surgery one year prior to presentation. General health was remarkable for hypertension, managed using ramipril. He was a non-smoker and there was no known family history of AMD.

Entering unaided acuities were 6/9.5-1 OD and 6/9.5-2 OS, improving to 6/6-2 OD and 6/7.6-1 OS with pinhole. Amsler grid was unremarkable in each eye. Contrast sensitivity was within normal limits at 1.56 units in each eye tested monocularly using the MARS test (normal range 1.52 to 1.76 log units for patients older than 60 years).

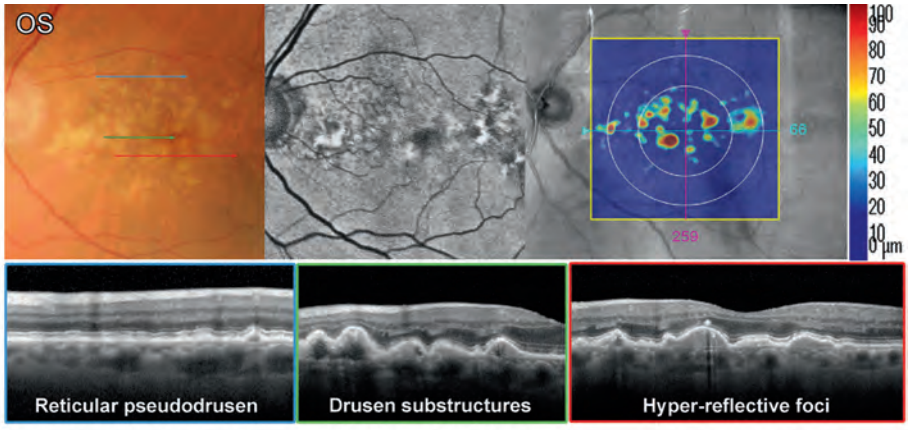
Funduscopy, retinal photography and Spectralis OCT revealed extensive, small to large drusen OU. Hyperpigmentary changes at the macula were also present OU. There was no evidence in either eye of any geographic atrophy, choroidal neovascularisation or exudative changes, including intraretinal, subretinal or sub-RPE fluid.

Additional, structural signs that may portend the development of late AMD were also identified including:

1. reticular pseudodrusen, also known as subretinal drusenoid deposits,[5-7] at the superonasal macula OU
2. hyper-reflective foci[8-10] using OCT corresponding with hyperpigmentary changes OU

intermediate AMD

number of indicators



▲ Figure 2. Corresponding imaging results from the patient's baseline visit in the left eye. The results are presented in the same order as described in Figure 1.

3. L-type or low reflective core OCT reflective drusen substructures[11] OU
4. focal apposition and disruption of the ellipsoid zone and external limiting membrane overlying a relatively high drusen load OU.[8,9,12] (Figures 1 and 2)

Fundus autofluorescence imaging also revealed a variety of patterns OU. In particular, alterations associated with a higher risk of conversion to late AMD including the patchy and reticular patterns were noted OU.[13] Drusen volume measured 0.12 mm^3 OD and OS in the central 3 mm using Cirrus OCT advanced RPE analysis (drusen volumes greater than 0.03 mm^3 have been associated with a four-fold higher risk of progression to late AMD).[14]

In summary, these baseline findings are consistent with intermediate AMD in both eyes and a 53 per cent probability of progression to late AMD within the next five years according to the AREDS simplified severity scale.[4] Structural changes including high drusen volume, reticular pseudodrusen, hyper-reflective foci, OCT reflective drusen substructures and abnormal FAF patterns were also identified, which may suggest a relatively greater risk of progressing to late AMD. His additional historical risk factors for progression include age and a medical history of hypertension.[15]

Current clinical guidelines recommended a review period in intermediate AMD between six and 24 months.[16,17] In this instance, due to the additional risk factors identified, the patient was reviewed in conjunction with the referring optometrist every six months. Follow-up assessments facilitated by advanced imaging revealed evidence of drusen progression, followed by regression, indicative of overall disease progression.[18] Progression to presumed late AMD was identified 28 months later OS (Figure 3) at which point the patient was referred promptly for ophthalmological care.

Early detection of progression to neovascular AMD is critical to maximising visual and functional outcomes.[19] The progression to CNV may have been missed or the diagnosis delayed had it not been for several factors: the utility of ocular imaging, evidence-based practice and the CFEH collaborative care model.

Discussion

This case report highlights foremost the utility of ocular imaging in clinical practice. On the whole, imaging technologies have empowered eye-care professionals by allowing us to deliver more individualised care. Several evidence-based practice guidelines,[16,17,20] including the most recent iteration edited by the Royal Australian and New Zealand College of Ophthalmologists,[21] outline the importance of early detection using imaging and the role of optometrists in AMD screening, stratification and management. As illustrated in the case study, 'full phenotyping' of each AMD case is now possible with the widespread clinical availability of ocular imaging, especially OCT.[21-24] (**CFEH Chair-side Reference, pages 12-13**)

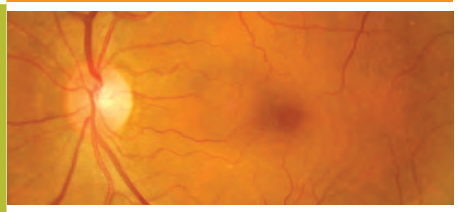
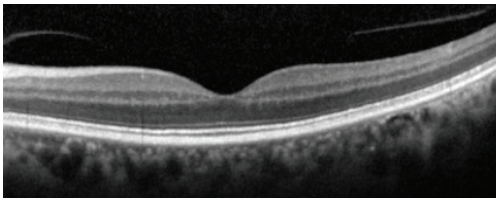
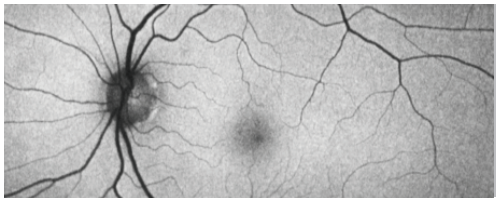
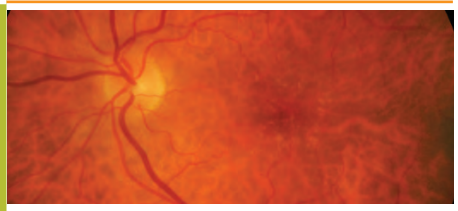
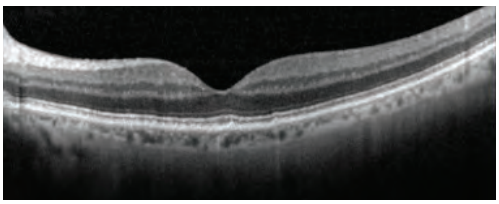
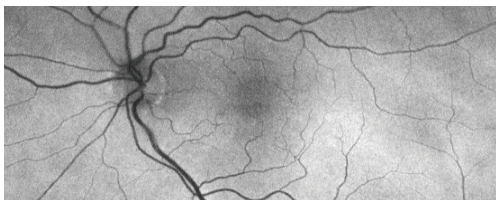
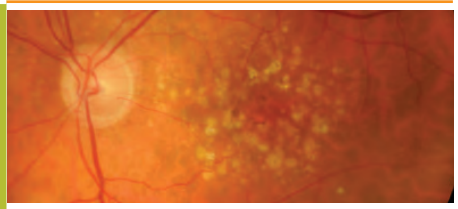
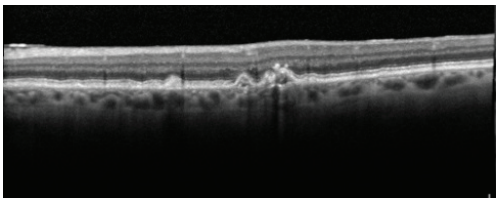
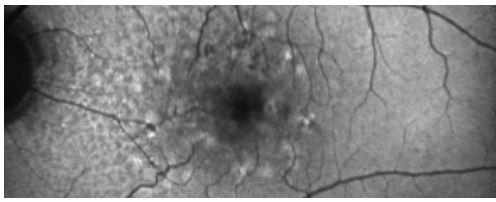
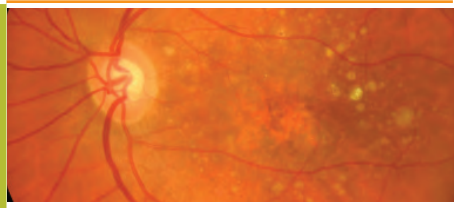
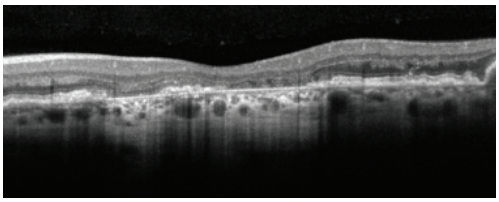
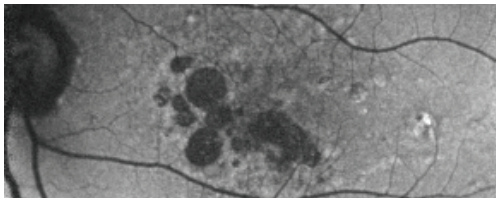
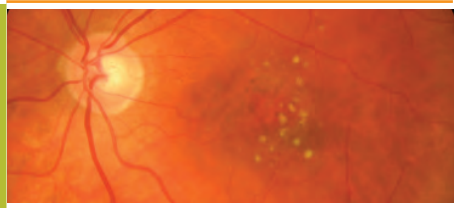
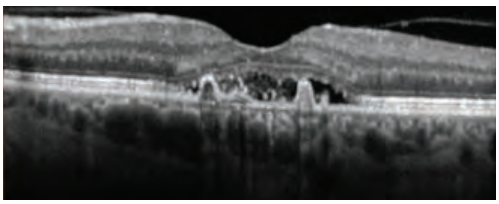
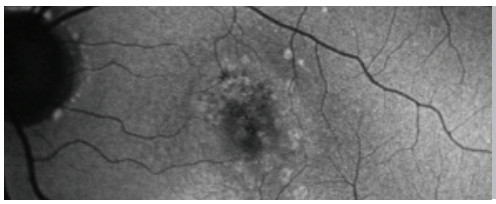
There is an onus on us as clinicians to systematically diagnose and stage ocular disease in order to determine a patient's risk of vision loss and to ensure timely referral for treatment. Once a diagnosis has been made, we can then tailor the management plan according to the patient's prognosis or risk of progression. This will require identifying the subtler disease-specific signs that herald a negative prognosis, many of which have been characterised only recently in the literature. Other signs relevant to AMD not described in the case study above include: nascent geographic atrophy,[8] sub-RPE hyper-reflective columns[25] and small pockets of subretinal fluid located in the depression between confluent drusen in the absence of CNV.[26]

With the incidence of diseases such as AMD rising, it is now more imperative than ever before that eye-care professionals collaborate to meet the demands of an ageing population. To deliver the best possible care to our patients, it is critical for practitioners to keep pace with the

Continued page 14

Centre for Eye Health

Chair-side reference

	AMD-related phenotype and distinguishing clinical features	Optical coherence tomography (OCT)	Fundus autofluorescence (FAF)
NORMAL AGEING CHANGES			
	• Drupelets (≤63 µm diameter) only • Should not show any characteristics of early, intermediate or advanced AMD	• Drupelets appear as just detectable discrete irregularities or elevations of the RPE • Some drupelets may be too small for OCT to resolve	• Drupelets may colocalise with punctate spots of normal, hyper- or hypo-fluorescence • FAF may also appear normal with the central macula showing diffuse, homogeneous autofluorescence and a gradual reduction in signal approaching the fovea centre
EARLY AMD			
	• Medium drusen (between 63 and 125 µm in diameter) • Should not have any of the characteristics of intermediate or advanced AMD • May present with a different stage of AMD in the fellow eye	• Medium drusen typically appear as discrete elevations of the RPE with variable internal reflectivity (similar to small drusen)	• Similar to drupelets, medium drusen may display a variable normal, hyper- or hypo-fluorescence pattern • A range of patterns may be observed in early and intermediate AMD including: normal, minimal change, focal increased, patchy, linear, lacelike, reticular, speckled, focal confluent, focal plaque-like or scattered
INTERMEDIATE AMD			
	• Large drusen (>125 µm in diameter) and/or pigmentary abnormalities (hyper- or hypo-pigmentary changes associated with at least medium-sized drusen) • No characteristics of late AMD	• Large drusen may appear as dome-shaped, occasionally confluent, elevations of the RPE, with a visible underlying BM • May be associated with overlying disruption of the EZ and ELM, thinning of the ONL and/or enhanced visibility of HFL • Increased reflectivity of underlying choroidal vessels due to focal RPE depigmentation • Hyper-pigmentary changes may be seen as discrete hyper-reflective foci (in the ONL or attached to drusen) with posterior shadowing	• May reveal any of the patterns described under early AMD • Predominantly reveals spots or punctate changes of hyper-fluorescence • Less commonly, spots of hypo-fluorescence and lines of hyper-fluorescence may also be observed • Patchy, linear and reticular FAF patterns are associated with a higher risk of conversion to neovascular AMD
ADVANCED AMD (Geographic atrophy)			
	• Any sharply delineated round or oval hypopigmented areas at least 175 µm in diameter that feature apparent absence of the RPE and increased visibility of choroidal vessels • Areas may coalesce to form a ring-type configuration, eventually involving the fovea • May be preceded by calcification of large drusen • Typically also displays signs of other AMD stages	• Loss of RPE causes sharply demarcated areas of increased signal penetration into the choroid • Associated outer retinal atrophy is seen as thinning or loss of the ONL, ELM and EZ, which may extend or taper beyond the margins of the GA lesions themselves	• Single or multiple areas of well-demarcated marked hypo-fluorescence • Foveal sparing is characterised by irregular hypo-fluorescence at the residual foveal island (such as in this example) or a symmetrical and gradual reduction in FAF approaching the fovea • The 'diffuse trickling' pattern in this image is associated with a significantly higher rate of progression • FAF may enable better detection of discrete/small areas of GA compared to other modalities
ADVANCED AMD (Neovascular AMD)			
	• May be characterised by any of: RPE detachment(s), neurosensory retinal detachment, subretinal or sub-RPE neovascular membrane(s), epiretinal, intraretinal, subretinal or sub-pigment epithelial scar/glial tissue or fibrin-like deposits (excluding idiopathic epiretinal membrane), subretinal haemorrhages and/or hard exudates • Neovascular lesions may appear subtle, green-grey or pink-yellow often complicated by the secondary signs above	• PEDs present as broad elevations of the RPE band anterior to BM. • Fibrovascular PEDs show medium internal reflectivity • Serous PEDs are well demarcated, dome-shaped and smooth with internal homogeneous hyporeflectivity • Haemorrhagic PEDs may also occur and are further described under polypoidal choroidal vasculopathy • Sub-RPE, subretinal or intra-retinal fluid may be present and indicative of AMD related choroidal neovascularisation (confirmed using FA)	• FAF changes corresponding with areas of choroidal neovascularisation may be characterised by its inherent features as follows: • Subretinal fluid corresponds with increased FAF in approximately 56.5% of cases • Haemorrhages, exudate and fibrovascular membranes are also likely to cause hypo-autofluorescence patterns • Can also present with normal or near normal FAF imaging results

Age-related macular degeneration

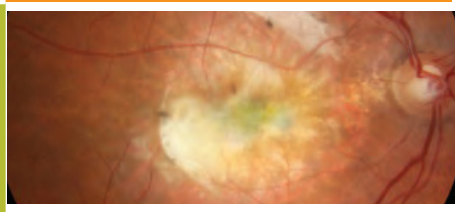
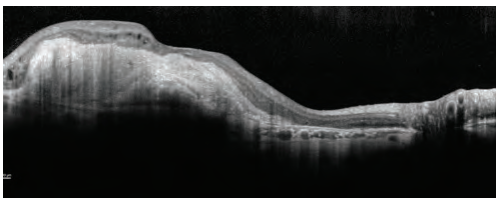
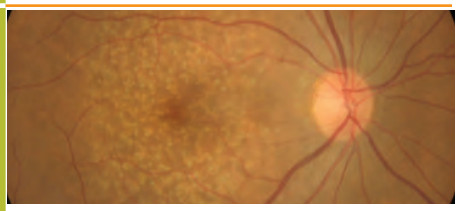
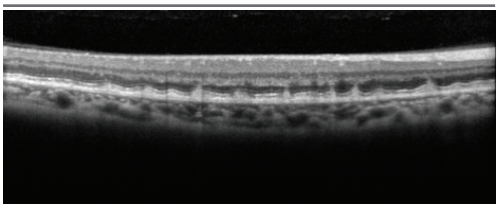
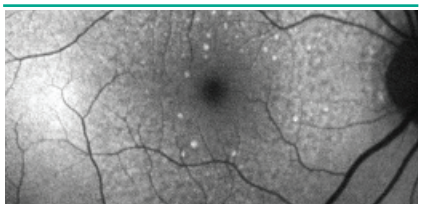
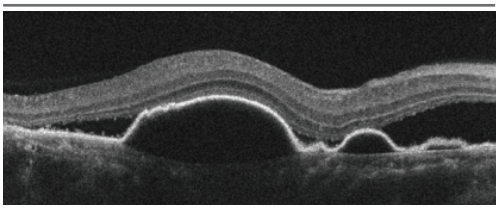
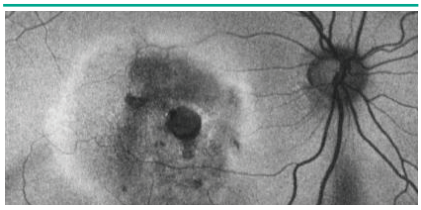
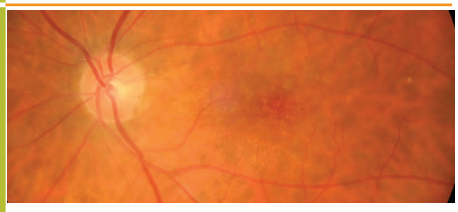
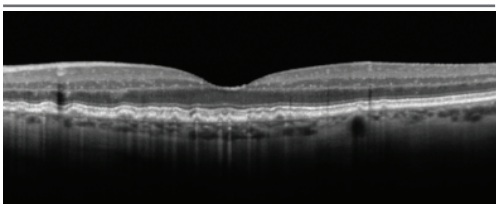
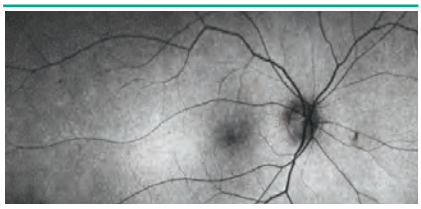
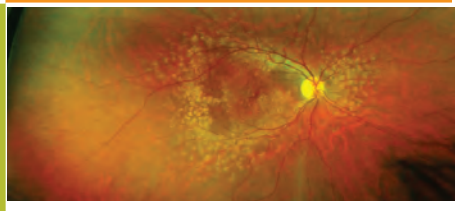
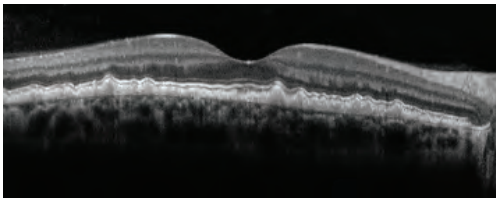
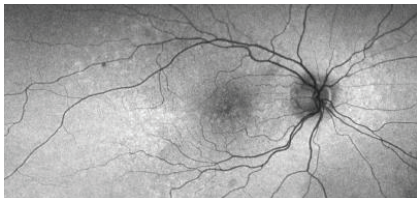
Optometry
NEW SOUTH WALES
AUSTRALIAN CAPITAL TERRITORY

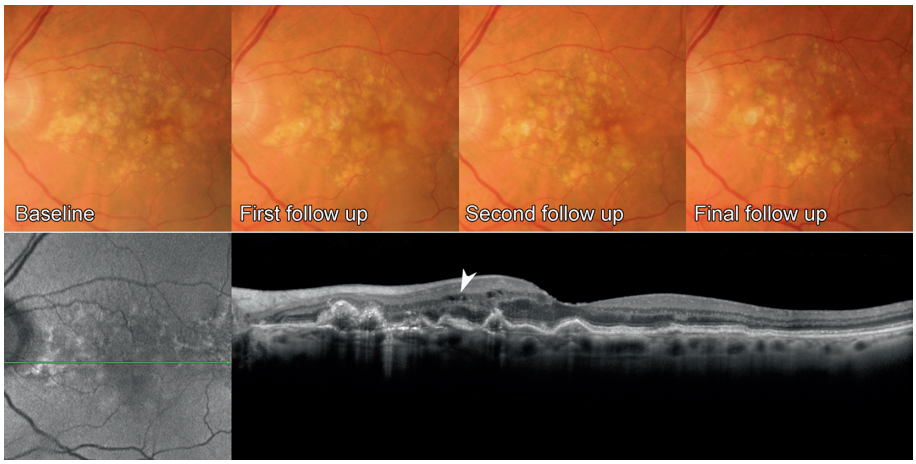
This chair-side reference was designed to assist optometrists in private practice when distinguishing between the different stages and phenotypes of age-related macular degeneration.

It provides general information only and may not be applicable to atypical cases. Fluorescein angiography and indocyanine green angiography are gold standard procedures for the diagnosis of neovascular AMD and polypoidal choroidal vasculopathy, respectively. However, they are outside the scope of the optometric practice and so are not included. It remains controversial whether polypoidal choroidal vasculopathy, cuticular drusen and familial dominant drusen represent distinct clinical entities or subtypes of AMD.

KEY

AMD: age-related macular degeneration; OCT: optical coherence tomography; FAF: fundus autofluorescence; RPE: retinal pigment epithelium; BM: Bruch's membrane; EZ: inner segment ellipsoid zone; ELM: external limiting membrane; ONL: outer nuclear layer; HFL: Henle's fibre layer; GA: geographic atrophy; FA: fluorescein angiography; PED: pigment epithelial detachment

AMD-related phenotype and distinguishing clinical features	Optical coherence tomography (OCT)	Fundus autofluorescence (FAF)
ADVANCED AMD (Neovascular AMD)		
		FAF not available
• End-stage presentations of neovascular AMD are characterised by regression of the vascular component and an increase in the fibrous component, and may appear as a disciform scar	• Well-demarcated, highly hyper-reflective lesions associated with loss and dysplasia of the overlying retinal layers	• Discifom scarring consistently demonstrates uneven hypo-autofluorescence of the lesion, surrounded by marked hyper-autofluorescence
RETICULAR MACULAR DISEASE		
		
• Indistinct, interlacing, yellow-white, round or oval lesions ranging from 125 - 250 µm in diameter • Most visible in red-free or blue light	• Reticular pseudodrusen may colocalise with subretinal drusenoid deposits which appear as conical deposits above the RPE	• Appear as low contrast hypo-fluorescent, circular, networked deposits • Individual lesions may also have a 'target-like' appearance (iso fluorescent core and surrounding hypo-fluorescent halo)
POLYPOIDAL CHOROIDAL VASCULOPATHY / POSTERIOR UVEAL BLEEDING SYNDROME		
		
• Multiple and recurrent serous and haemorrhagic RPE detachments • Orange-red subretinal nodules • Spontaneous, recurrent subretinal/vitreous haemorrhage • Drusen are atypical • Minimal fibrous scarring. Typically presents as a serosanguineous maculopathy in middle-aged (50-65 years) African or Asian women • Often bilateral but asymmetric	• Haemorrhagic PED appears as a hyper-reflective elevation of the RPE line with no reflectivity within or under the PED (the large, central PED pictured) • In contrast, a reflective line representing BM /choriocapillaris can be observed beneath the fluid of a serous PED (smaller PED pictured).	• Confluent hypo-fluorescence at the sites of polypoidal lesions and granular hypo-fluorescence at the branching choroidal vascular networks
CUTICULAR DRUSEN		
		
• Numerous, densely packed, relatively uniform, small drusen, better seen using FA, described as a 'starry-sky' pattern • 50 to 75 µm in diameter	• Blunted triangular appearance with a saw tooth pattern	• May reveal numerous hypo-fluorescent 'dots'
FAMILIAL DOMINANT DRUSEN / DOYNE'S HONEYCOMB DYSTROPHY / MALATTIA LEVENTINESE		
		
• Numerous drusen that extend beyond the vascular arcades and often nasal to the optic disc • Age of presentation is typically between 20 and 30 years (younger than of typical AMD) • Bilateral and relatively symmetrical	• Drusen characteristics are similar to those seen in AMD • Larger, round drusen are typified by confluence and more diffuse deposition between the RPE and BM	• Reveals marked hyper-fluorescence of large drusen • Smaller radially distributed drusen appear more faint



▲ Figure 3. Top: Follow-up findings using colour fundus photography, Bottom: Imaging results from the final follow-up visit including fundus autofluorescence (left) and an OCT line scan through the central macula (right). Both show exudative changes, including intraretinal fluid (arrowhead), signifying the likely conversion to neovascular AMD.

Prognostic biomarkers

From page 11

literature, apply and interpret ocular imaging appropriately, and refer for management advice and treatment in a judicious and timely manner. For challenging cases, patients can be referred to organisations with a special interest in ocular disease, such as CFEH. Such organisations are available to aid practising professionals by providing additional patient management advice.

Acknowledgements

The author thanks Michael Yapp, Professor Michael Kalloniatis and Paula Katalinic for reviewing the manuscript and Tyson Xu for his assistance in the literature search and for identifying the case images. ▲

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A week in neuro-ophthalmology—where the rare is routine

Perspectives for the optometrist

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Dr Leonid Skorin Jr

OD DO MS FAAO FAOCO

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OUR PATIENTS might come to us with no specific complaints, or they may walk in reporting complete, sudden vision loss. Usually it's somewhere in between but it's up to us to determine how urgent the problem is. A quick way to become more comfortable with making decisions regarding vision-threatening situations is to spend time with those at the end of the referral line. For them, the rare is routine and managing seemingly complex and serious issues is the norm.

We hope that sharing some of the cases we see during a typical week in our neuro-ophthalmology clinic can help as a refresher for primary care clinicians who are often faced with making initial treatment or referral decisions. We will provide a brief overview of relevant case information, current facts about each condition and finally, a description of how that information was used to manage the patients.

Monday: Temporal arteritis

The case

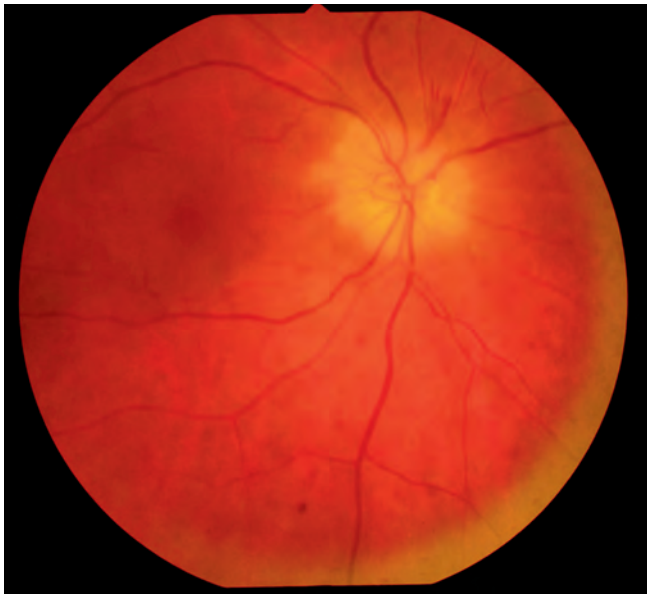
A 62-year-old white male was referred to the neuro-ophthalmology clinic by his comanaging optometrist for evaluation. The patient reported that he had experienced severe vision loss in the right eye on waking about five weeks previously. He said that '99 per cent of the vision was gone in his right eye.' He denied any headaches, jaw claudication or scalp tenderness associated with the vision loss. His medical history was positive for suspected temporal arteritis (TA) eight years earlier; however, he had not experienced any vision changes at that time. He was also an insulin-dependent diabetic patient.

During our evaluation, best corrected vision in his right eye was light perception and 6/7.5 in his left eye. He had a marked relative afferent pupillary defect and severe swelling of the optic disc in the right eye (Figure 1). The optic disc of the left eye was unremarkable. Laboratory testing revealed an elevated C-reactive protein (CRP) level of 4.7 mg/L, an elevated erythrocyte sedimentation rate (ESR) of 102 mm/hr and a platelet count of 145,000 mm^3. We performed a temporal artery biopsy on the patient's right side three days after the consultation. A 1.3 mm specimen was excised and processed by the pathology laboratory (Figure 2). The results came back negative for inflammation that would indicate TA.

The facts

Temporal arteritis, also known as giant cell arteritis, is a chronic autoimmune disorder characterised by inflammation of the small- and medium-sized arteries throughout the body. Unilateral vision loss is a common presenting symptom of TA due to arteritic anterior ischaemic optic neuropathy (AAION). Inflammation and hyperplasia of the intimal layers of the arteries causes occlusion of the short posterior ciliary arteries. This occlusion causes the optic disc to become ischaemic in patients with AAION, leading to vision loss. Patients with TA may also experience severe headaches, scalp tenderness, jaw claudication and general malaise. The peak age of incidence is between 60 and 75 years and it occurs very rarely under the age

Continued page 16



▲ Figure 1. Fundus photo of the swollen optic disc in the right eye

A week in neuro-ophthalmology

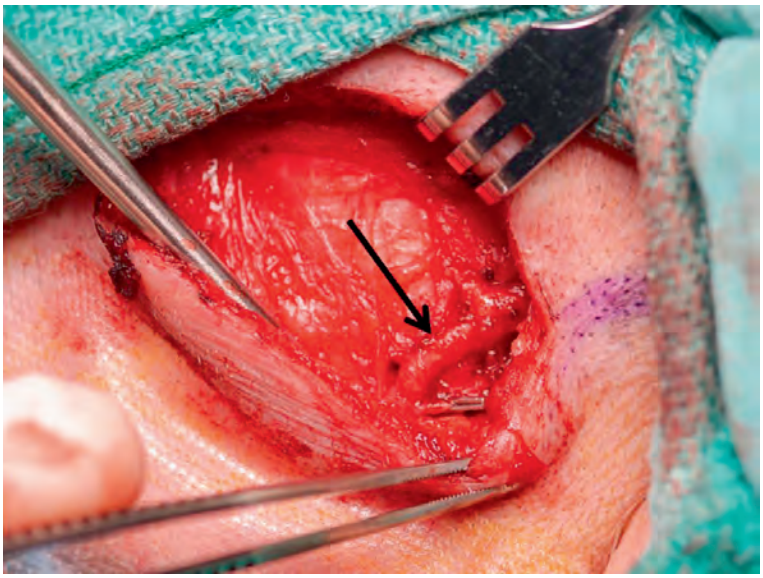
From page 15

of 50 years. A patient presenting with vision loss and two or more of the other characteristics is highly suspect for temporal arteritis with AAION.

Differentiating AAION from NAION

The primary differential for AAION is non-arteritic anterior ischaemic optic neuropathy (NAION). Unilateral vision loss in NAION is, on average, less severe (6/30 or better) than is seen with AAION. Patients with NAION are often younger (< 60 years of age) and do not complain of headaches or other associated symptoms. Objectively, the optic disc of the affected eye appears less swollen in NAION compared to AAION. Additionally, the disc of the non-affected eye in a patient with NAION may be classified as a 'disc at risk' characterised as a cup/disc ratio less than 0.3. AAION is not associated with any specific cup/disc ratio measurements. While NAION has no standard treatment and often resolves on its own, AAION associated with TA is much more serious and may result in severe vision loss in the fellow eye within a week if treatment is not initiated quickly. The risk of bilateral blindness makes differentiation of the two diseases critical.

The most reliable differentiating tests are the erythrocyte sedimentation rate (ESR) test, C-reactive protein (CEP) test and complete blood count (specifically, platelet count). CRP values over 2.5 mg/L, ESR values over 50 mm/hr and a platelet count of greater than 400,000 mm^3 are all indicative of TA/AAION.[1] Laboratory tests for NAION generally come back normal. If a patient's laboratory results are elevated, the next step is to perform a temporal artery biopsy, which is the gold standard for TA diagnosis. A ≥ 1.0 cm specimen is excised from the frontal branch of the superficial temporal artery and submitted to a pathology laboratory for evaluation. Positive tests are characterised by the presence of near complete lumen occlusion, 'giant' multi-nucleated cells and granulomatous inflammation. A negative specimen will not exhibit any of these characteristics.



▲ Figure 2. Isolated temporal artery specimen prior to excision. Arrow indicates vessel.

Corticosteroids are the treatment of choice in those with TA.[1] Treatment should be initiated immediately if TA is suspected, even if a temporal artery biopsy has not been performed. Treatment will not skew the results of the biopsy until two to four weeks after treatment has been initiated.[2,3]

The best treatment for those patients with severe vision loss in one eye is one gram of intravenous methylprednisone for three days followed by oral steroids (1 mg/kg of body weight/day).[4] If this option is not available, 40 to 100 mg of oral prednisone (about 1 mg/kg of body weight/day) should be started and increased (maximum of 100 mg/day) until symptomatic improvement is reached.[5] ESR and CRP should be checked weekly to measure improvement. Once an effective dose is reached, it should be continued for four to six weeks. The steroid should then be tapered very slowly, reducing the dosage by 5-10 mg per month. As a result, treatment regimens can last from one to two years.

Putting it all together

Our case is a good example of the ambiguity associated with suspected temporal arteritis. Our patient exhibited severe vision loss but no other symptoms. His ESR and CRP were elevated, but the results may have been complicated due to concurrent end-stage renal disease from his long-standing diabetes which may also increase inflammatory markers.[6] His history was positive for suspected TA in the past; however, a temporal artery biopsy was never performed. When the procedure was performed, the results were negative. In the end, half the diagnostic data pointed toward TA, while the other half was not consistent with the typical disease characteristics.

Ultimately, he was treated with oral steroids. Although he did not present to his optometrist until two weeks after he had first noticed his vision loss, he was immediately started on 60 mg of oral prednisone daily. Two weeks later, the medication was stopped by his general practitioner due to a rapid elevation in his blood glucose levels (245 mg/dL). It was not until after treatment was discontinued that he was referred to our office.

The patient reported that his vision had improved when the therapy was initiated, but deteriorated rapidly when it was stopped. We cannot definitely say the discontinuation of the treatment caused further vision impairment because vision loss can continue in up to 13 per cent of patients receiving standard treatment.[7] Fortunately, his left eye was not affected at the time, though it is at risk. The temporal artery biopsy was performed about five weeks

after the onset of symptoms as well as after the steroid treatment. Only 60 per cent of biopsies come back positive under these circumstances.[2] The recommendation was made to perform the procedure on the other side as 23 per cent of bilateral temporal artery biopsies are positive on one side and negative on the other. Results of the second biopsy were negative as well. We classified his condition as NAION. Regardless of the diagnosis and despite all of the interventions, the patient's vision in his right eye is not likely to improve.

We believe our case exemplifies the seriousness of this condition and the potentially detrimental effects of delayed treatment and testing. Patients who are at most at risk should be educated about returning to the clinic immediately if they experience any of the symptoms described above. Additionally, communication between practitioners is paramount to preventing vision loss through early treatment and laboratory testing. Treatment protocol should be well understood by all parties to prevent relapse and further complications.

Tuesday: Pseudotumour cerebri

The case

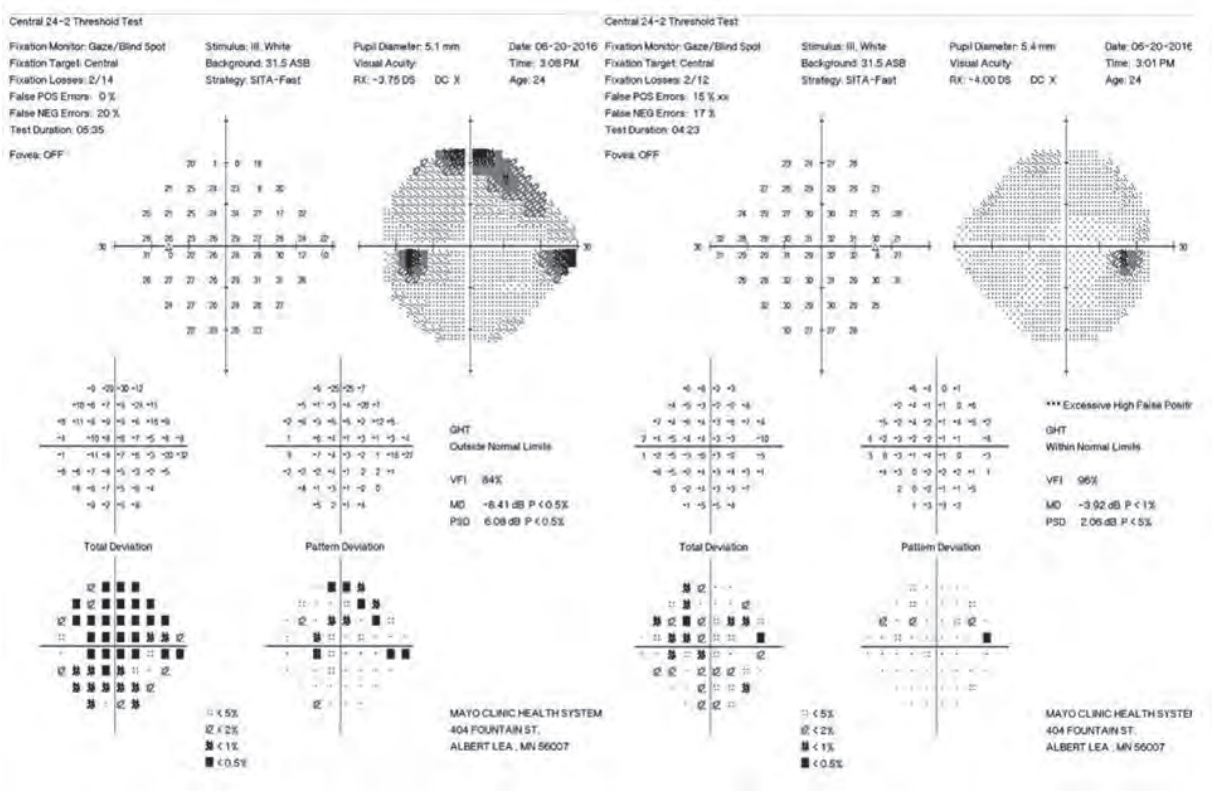
A 24-year-old white female was referred to the neuro-ophthalmology clinic by the emergency department for evaluation. The patient reported 'spots in her vision', primarily her left eye, which had started five days before presenting to the emergency department. She was experiencing severe headaches across the frontal portion of her head and they were progressively getting worse. Her medical history was positive for pseudotumour cerebri two years prior. The patient had also been taking oral tetracycline medication to treat her acne.

During our evaluation, her corrected visual acuities were 6/9 in each eye. She subjectively reported 'grey spots' both superior temporally and superior nasally during Amsler grid testing of the left eye. Amsler grid testing was unremarkable for the right eye. Visual field testing was performed for each eye (Figure 3). Enlargement of the blind spot was noted in the right eye and a superior arcuate defect extending from an enlarged blind spot to the nasal field, crossing the horizontal midline was noted in the left eye. Both optic discs were elevated and indistinct, consistent with papilloedema. Other findings during the dilated fundus examination were unremarkable. The patient returned to the emergency department and a lumber puncture was performed. Opening pressure was measured to be 34 cm.

The facts

Pseudotumour cerebri is also known as idiopathic intracranial hypertension (IIH). It is characterised by increased intracranial pressure due to an unknown cause. Vision disturbance or obscuration is generally transient and visual acuity is not drastically reduced as the macula is not usually involved.[8] The most common area of visual field loss in patients with IIH

Continued page 18



▲ Figure 3. 24-2 Humphrey visual field of the right and left eye in a patient with pseudotumour cerebri

A week in neuro-ophthalmology

From page 17

is inferonasal.[9] Associated symptoms include severe headache with or without nausea and pulsate tinnitus (pulse-synchronous whooshing sound in ears). The condition is most commonly seen in overweight women of child-bearing age. Medications that are commonly used by this population may increase the risk of developing IIH including oral contraceptives and oral tetracyclines.

A patient with these signs and symptoms should be referred, specifically for neuro-imaging. IIH is a diagnosis of exclusion and magnetic resonance imaging is indicated to rule out an increase in intracranial pressure due to a mass or hydrocephalus and a magnetic resonance venography should be done to rule out dural sinus thrombosis. A lumbar puncture is then performed to measure the intracranial pressure. Normal opening pressure is between 10 and 20 cm. A reading over 25 cm is considered diagnostic for IIH provided imaging is negative for any other pathology.

In addition to being a diagnostic test, lumbar puncture usually provides some immediate, short-term relief of symptoms to patients with severe headaches. Long-term treatment includes weight loss and medications to reduce intracranial pressure. A six per cent reduction in body weight has been shown to improve signs and symptoms indefinitely if the weight is maintained.[10] In cases where weight loss is not enough, oral carbonic anhydrase inhibitors (CAI) are indicated.

Oral acetazolamide (Diamox) is the first-line medical treatment for IIH and acts by reducing the production of cerebral spinal fluid. Alternatively, topiramate (Topamax) which is a weak CAI and aids in weight loss may also be a good option for some patients. In cases of more severe visual field loss, a surgical shunt to drain the cerebral sinus fluid or optic nerve sheath fenestration may be appropriate.

A surgical referral should be made if a new visual field defect is noted or a previously noted defect gets worse even with medical treatment.

Putting it all together

Our patient's magnetic resonance imaging and magnetic resonance venography scans were negative for any pathology when she was initially diagnosed with IIH two years prior to the referral. At that time, she had been prescribed two, 500 mg Diamox tablets daily and reported improvement of symptoms. She continued to take her medication for over 18 months but stopped because she felt her symptoms had completely resolved. Over the next four months, her symptoms returned. She restarted the Diamox; however, it did not provide headache relief and prompted her to present to the emergency department.

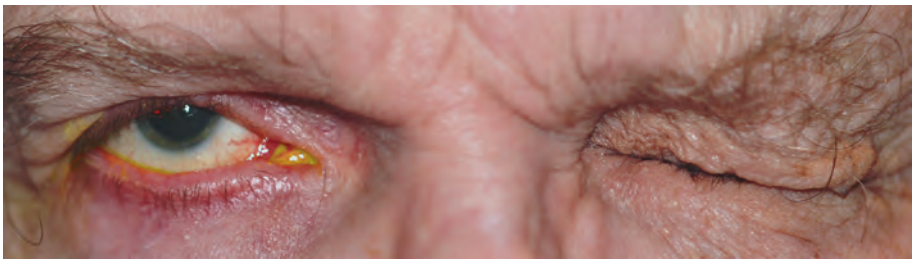
Computed tomography (CT) of the brain was done in the emergency department the day of our consultation and was found to be negative for a mass. The patient reported relief after a lumbar puncture was performed and was started back on the same Diamox dosing. Additionally, 25 mg of topiramate daily by mouth was prescribed to aid in weight loss and reduce intracranial pressure. She was scheduled for a follow-up visual field test in one month.

This case is more straightforward than the last but it illustrates an important point. Making the appropriate referral is crucial even if the answers seem obvious. Findings were consistent with a previous diagnosis and it may have been tempting to observe the patient to see if restarting the Diamox medication would eventually alleviate her symptoms. However, the referral served two important purposes: any serious pathology was ruled out with CT scanning and the lumbar puncture provided instant relief for the patient's headache symptoms.

Thursday: Bell's palsy

The case

A 72-year-old white male was referred for consultation by his ophthalmologist to the neuro-ophthalmology clinic with complaints that the right side of his face and mouth were 'droopy' and that he was unable to close his right eye. After further questioning, the patient reported the onset was sudden and painless; however, he did not know when the problem had started. The patient's wife had first noticed the problem 10 days previously.



▲ Figure 4. Lack of Bell's phenomenon in a patient with Bell's palsy attempting to close both eyes

During our evaluation, entering corrected acuities were 6/30 in the right eye and 6/7.5 in the left eye with no improvement on pinhole. The anterior segment examination revealed mild, diffuse injection of the bulbar conjunctiva and areas of superficial punctate keratitis covering the entire cornea of the right eye. During auxiliary cranial nerve testing, the patient was unable to wrinkle the right side of his forehead or smile with the right side of his mouth. There was blunting of the nasolacrimal fold on the right side compared to the left side. Examination of the right ear canal for herpetic lesions to rule out herpes zoster oticus (Ramsay-Hunt syndrome) was unremarkable. When asked to close his eyes, the right eye remained open and did not properly elevate while the left closed normally (Figure 4).

We rated his Bell's phenomenon (elevation of the eyes when the lids are closed) as poor. Tear break up time was four seconds in both eyes and the Schirmer-2 test was 8 mm in the right eye and 6 mm in the left. Corneal sensitivity was tested with a cotton swab and rated as very poor.

The facts

Bell's palsy is characterised by hemi-facial paresis and inhibited eyelid closure. The pathophysiology of Bell's palsy involves inflammation of cranial nerve seven (CN VII). Infection of the nerve ganglion by the herpes simplex virus (HSV-1) in the most commonly accepted cause of the condition. Vision may be affected due to ocular surface decompensation as a result of blink inhibition. The lack of consistent blinking also causes tearing, burning or general ocular irritation.

Vision-care providers should be familiar with eye and facial signs that may aid in differentiating between stroke and Bell's palsy. Specifically, it is not likely that eyelid closure and frontalis function (furrowing of the forehead) would be affected in a patient who suffered a stroke; however, they may exhibit other neurological signs. Conversely, patients with Bell's palsy will have a complete unilateral facial palsy but no other signs. If a stroke is suspected, patients require immediate referral so that patients who require anti-clotting treatment can be treated in a timely manner. Prognosis is most favourable if treatment is started within the first three hours after onset of a stroke.

Bell's palsy is significantly more benign and the treatment window is much broader. The discoveries of the inflammatory and viral nature of the condition led to treatment strategies combining both corticosteroids and antivirals. Administration of oral steroids within 72 hours of onset has been shown to significantly increase the rate of recovery.[11]

Putting it all together

We presented the information to our patient and his wife, explaining that because the onset had been 10 days prior, treatment might not be as effective as if it had been provided sooner. The patient and his wife elected to do combination therapy. We prescribed oral acyclovir 400 mg twice-daily and oral prednisone 10 mg twice-daily, both for one week.

We then explained treatment options to heal the ocular surface. Patients who develop corneal damage despite appropriate conservative therapy need surgery to reanimate their paralysed eyelids. The most vulnerable patient is one who manifests the 'BAD' syndrome, that is, lacks the Bell's phenomenon, and has corneal anaesthesia and dry eye.[12] The patient was completely unable to close his eye and did not blink once during the examination. His Bell's phenomenon was reduced when he attempted to close his eyes. Our testing also confirmed that he had corneal anaesthesia and dry eye, putting him at high risk for corneal damage.

We chose to apply 3M surgical tape horizontally from the medial to lateral aspect of the upper lid to act as a temporary weight. This was enough to induce a partial ptosis in primary gaze and close the eye when the patient tried to shut his eyes and the levator relaxed. Gel drop artificial tears were prescribed every two hours and preservative-free ointment was prescribed for overnight coverage.

After two weeks, his condition had greatly improved. He and his wife reported that he had completed the oral antiviral and steroid treatment as directed. The visual acuity in his right eye improved to 20/25+ and he had no other visual complaints. His corneal epithelium was intact and did not stain with sodium fluorescein. He was able to shut both his eyes tightly and did not display any residual lagophthalmos. A referral may be appropriate if full function of the orbicularis oculi muscle does not return and the cornea remains exposed. Lid weights can be surgically implanted into the upper eyelid to induce ptosis and protect the ocular surface.

Conclusion

The question of when to refer, to whom and how quickly can be tricky. Often, it depends on the comfort of the primary care clinician in making a diagnosis and initiating treatment. Regardless of the ultimate decision, precise documentation of all examination information is crucial. It allows optometrists to track improvement if they are managing the patient and it also allows the physicians receiving a referral to cross-check information and make sure they have the facts straight. A thorough initial examination, accurate assessment and differential diagnosis and good communication between practitioners are the keys to success and may save a patient's vision in an urgent or emergent situation.

Disclosures

Turpin and Skorin report no relevant financial disclosures. ▲

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Therapeutic NEWS of note

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Efficacy of corneal collagen cross-linking for keratoconus in children

A study published in the February issue of the journal *Cornea* confirms that corneal cross-linking (CXL) is effective in stabilising keratoconus for more than two years in most paediatric eyes.

To report the long-term outcome of corneal collagen cross-linking for progressive keratoconus in paediatric patients, researchers conducted epithelium-off CXL in paediatric eyes with progressive keratoconus. Spectacle-corrected distance visual acuity (CDVA), retinoscopy, topography and tomography were documented preoperatively and postoperatively at three months, six months, one year and annually thereafter.

A total of 377 eyes of 336 paediatric patients aged eight to 18 years with progressive keratoconus underwent CXL. Of these, 194 eyes had a follow-up beyond two years and up to 6.7 years. At last follow-up, there was significant improvement in mean CDVA from 0.33 +/- 0.22 to 0.27 +/- 0.19 logMAR ($p <= 0.0001$), reduction in mean topographic astigmatism from 7.22 +/- 3.55 to 6.13 +/- 3.28 D ($p = 0.0001$), mean flattening of 1.20 +/- 3.55 dioptres in maximum keratometry (Kmax) ($p = 0.0002$), and mean corneal thinning of 31.1 +/- 36.0 µm ($p < 0.0001$) after CXL.

The mean change in Kmax was most significant in moderately advanced keratoconus (average keratometry 48-53 dioptres). Central cones showed more corneal flattening than peripheral cones. Stabilisation or flattening of Kmax was seen in 85 per cent of eyes at two years and in 76 per cent after four years. Stabilisation or improvement of CDVA was seen in 80.1 per cent of eyes at two years and in 69.1 per cent after four years.

The authors concluded that CXL remains effective in stabilising keratoconus for longer than two years in a majority of paediatric eyes. Flattening of Kmax was greater in moderately advanced keratoconus and central cones; however, long-term follow-up beyond four years revealed that a few eyes showed features suggestive of a reversal of the effect of CXL.

Cornea 2017 Feb; 36: 2: 138-143; doi: 10.1097/ICO.0000000000001102

How often should the Goldmann Applanation tonometer be checked for calibration error?

According to a study published in the *Journal of Glaucoma*, twice a year, unless it's more than one year old, then it should be checked monthly.

In a study to evaluate the frequency of Goldmann applanation tonometer (GAT) calibration errors, GATs were divided into three groups: group 1 (G1) ≤ 1 year, group 2 (G2) >1 to 10 years, and group 3 (G3) > 10 years of usage. Tonometers were checked at baseline for calibration errors and defined as 'faulty' when calibration error exceeded 2 mmHg at any testing level. Faulty GATs were repaired in-house. Subsequent calibration error checks were conducted once per month for six months.

In total, 76 slit-lamp mounted GATs were included. The number of GATs in groups 1 to 3 was 19, 36, and 21, respectively. Seven (9.2 per cent) tonometers were faulty at the baseline. None in G1, five in G2, and 16 in G3 demonstrated unacceptable calibration errors over the study course ($p < 0.01$).

The survival function of G1 tonometers was 1.0 throughout, whereas those (95% confidence interval) of the G2 and G3 tonometers were 0.97 (0.81-0.99) and 0.76 (0.51-0.89) at one month, and 0.86 (0.69-0.93) and 0.23 (0.08-0.43) at six months, respectively.

The researchers concluded that the probability of calibration error increased with increasing age of the tonometer. The frequency of use of the tonometer was not associated with the development of calibration error.

J Glaucoma 2016 Nov; 25: 11: 908-913; doi: 10.1097/IJG.0000000000000545

Jardiance approved by US FDA for adults with type 2 diabetes

To reduce the risk of cardiovascular death in adult patients with type 2 diabetes mellitus and cardiovascular disease, the US Food and Drug Administration has approved a new indication for Jardiance (empagliflozin). The announcement was made in December 2016.

According to the Centers for Disease Control and Prevention, death from cardiovascular disease is 70 per cent higher in adults with diabetes compared to those without diabetes, and patients with diabetes have a decreased life expectancy driven in large part by premature cardiovascular death.

In Australia, Jardiance is available on the Pharmaceutical Benefits Scheme as an adjunct to diet and exercise to improve glycaemic control in adults with type 2 diabetes mellitus. It has not been approved in Australia for cardiovascular disease.

The FDA conducted a post-market clinical trial of more than 7,000 patients with type 2 diabetes and cardiovascular disease. In the trial, Jardiance was shown to reduce the risk of cardiovascular death compared to a placebo when added to standard of care therapies for diabetes and atherosclerotic cardiovascular disease.

Jardiance is not intended for patients with type 1 diabetes mellitus or for the treatment of diabetic ketoacidosis. Jardiance is contraindicated in patients with a history of serious hypersensitivity reactions to Jardiance, severe renal impairment, end-stage renal disease or dialysis.

www.FDA.gov

SWAP vs OCT in the early detection of glaucoma

Researchers assessed the role and diagnostic efficacy of optical coherence tomography (OCT) and short wave automated perimetry (SWAP) to distinguish between normal subjects, glaucoma suspects and diagnosed glaucomatous eye.

In this randomised controlled, consecutive, prospective study, researchers divided 70 subjects (140 eyes) into three groups: group A: 10 healthy volunteers (20 eyes); group B: 30 glaucoma suspects (60 eyes); and group C: 30 subjects (60 eyes) with already diagnosed glaucomatous eyes.

Average retinal nerve fibre layer thickness was 75 ±9 µm in the glaucoma group; 99 ±15.5 µm in the control group; and 94 ±12 µm in glaucoma suspects. The early parameter affected was the inferior quadrant. Researchers detected significant correlation between visual field parameters and retinal nerve fibre layer thickness in glaucoma and glaucoma suspect groups.

The researchers noted that retinal nerve fibre layer defect can be difficult to identify during clinical examination, and early detection of glaucoma is still controversial, whether by OCT, SWAP or frequency-doubling technology perimetry.

The researchers concluded that while OCT parameters tended to be more sensitive than SWAP parameters, retinal nerve fibre layer thickness measured by OCT and SWAP indices were good discrimination tools between glaucomatous, glaucoma suspect and normal eyes.

Clin Ophthalmol 2016; 10: 1819-1824

Higher IOP associated with faster loss of RNFL

Higher intraocular pressure (IOP) was associated with faster rates of progressive retinal nerve fibre layer loss over three years, according to a cohort study published in *Ophthalmology*.

Researchers recruited 339 patients to two glaucoma studies at three different academic centres, and followed them for an average of 3.9 years. The mean age of patients at baseline was 65.8 years, and 53.4 per cent were women. Of these patients, 308 (56.3 per cent) had a diagnosis of glaucoma and 239 (43.7 per cent) were considered glaucoma suspects.

All eyes were imaged with SD OCT, with glaucoma progression defined as a result of likely progression on the SAP.

The researchers found the largest associations between IOP and retinal nerve fibre layer change rates in measurements from the temporal superior and temporal inferior sectors, and the smallest association for measurements from the nasal sector. For progressing eyes, OCT found that each 1 mmHg increase in IOP correlated with additional losses of 0.35 and 0.31 µm/year for the temporal superior and temporal inferior sectors of the eyes, respectively.

The authors concluded that their findings support the use of retinal nerve fibre layer thickness measurements obtained by SD-OCT in evaluating the efficacy of IOP-lowering therapies to slow the rate of disease progression, and suggested that these measurements may lead to a better understanding of the relationship between IOP and neural losses in glaucoma.

Ophthalmology 2016; 123: 10: 2058-2065

Caution urged with the use of bimatoprost after cataract surgery

The open-access journal *Ophthalmological Medicine* has published a case report on bimatoprost-induced serous macular detachment and choroidal folds following uneventful cataract surgery.

A 66-year-old male using topical bimatoprost in both eyes for open-angle glaucoma underwent uneventful cataract surgery in the right eye. Postoperatively, he was restarted on topical bimatoprost and antibiotic-steroids combination drops.

A week after surgery, he presented with conjunctival hyperaemia, serous macular detachment and choroidal folds at the posterior pole. Fundus fluorescein angiography showed perifoveal leaks in early stage with pooling of dye in late stage. Discontinuation of bimatoprost led to resolution of serous detachment and choroidal folds within three weeks with improvement in visual acuity.

The authors suggested that occurrence of serous macular detachment and choroidal folds in this case could be related to the proinflammatory property of bimatoprost. They added that bimatoprost should be used with caution in the immediate postoperative period after cataract surgery.

Case Rep Ophthalmol Med 2016; doi: 10.1155/2016/7260603

Povidone-iodine for bacterial keratitis in the developing world

Povidone-iodine 1.25% should be considered for the treatment of bacterial keratitis when antibiotic treatment is not practical, according to a study published in the *American Journal of Ophthalmology*.

The randomised, controlled, investigator-masked clinical trial compared povidone-iodine 1.25% ophthalmic solution with topical antibiotics for bacterial keratitis in areas where use of effective topical antibiotics may not be an option.

A total of 172 adults with bacterial keratitis were randomised to be treated with either povidone-iodine (40 patients in the Philippines, 38 in India) or the control antibiotic (49 patients in the Philippines, 45 in India).

Thirty patients out of 40 treated with povidone-iodine and 39 out of 49 treated with neomycin-polymyxin B-gramicidin in the Philippines achieved cure. In India, 12 individuals out of 38 treated with povidone-iodine and 10 individuals from 45 treated with ciprofloxacin achieved cure.

The researchers concluded that there is no significant difference between the effect of topical povidone-iodine 1.25% and topical antibiotics commonly available in the developing world for treatment of bacterial keratitis. Povidone-iodine 1.25%, which is widely available and inexpensive, can be considered for treatment of bacterial keratitis when antibiotic treatment is not practical.

The researchers added that the results are applicable to children as well, who are disproportionately affected by infectious keratitis in the developing world.

Am J Ophthalmol 2016; doi:10.1016/j.ajo.2016.10.004

Endoscopic cyclophotocoagulation laser for glaucoma

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THE FOCUS of current glaucoma therapy is the stabilisation of intraocular pressure (IOP). This is achieved by promoting the balance of production and outflow of aqueous humour using ophthalmic medication or surgical procedures. Surgical intervention is reserved until ophthalmic medication is no longer ideal. This occurs when the maximum medical therapy fails to reduce IOP to sufficient levels or if it is in the patient's best interest to discontinue or reduce eye-drop therapy. The patient may have intolerable adverse reactions or compliance issues, or may be unable to afford the medications, as is the case in some countries.

Most commonly-used glaucoma surgical procedures, such as trabeculectomy, aim to increase the outflow of aqueous by altering the trabecular meshwork structure. The production of aqueous can also be addressed surgically. Endoscopic cyclophotocoagulation (ECP) laser targets the ciliary processes of the ciliary body where aqueous is produced. This laser procedure decreases aqueous production by causing atrophy to the ciliary processes.

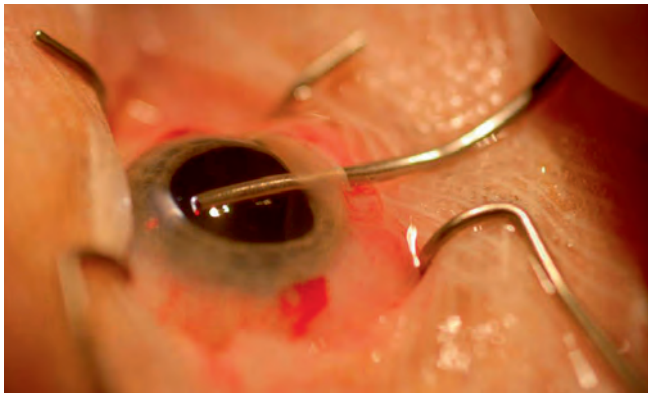
ECP can be used for most types of glaucoma from mild to severe. ECP has been indicated for the treatment of primary open-angle glaucoma (POAG), normal-tensive glaucoma and secondary glaucomas including pigmentary and pseudoexfoliative glaucoma. It can also be used when angle remodelling surgeries such as selective laser trabeculoplasty (SLT) are contraindicated. These include chronic closed-angle glaucoma, angle recession, plateau iris, neovascular glaucoma, iridocorneal syndrome and difficult to treat congenital glaucoma.[1,2] Although there are no absolute contraindications, ECP should not be used in uveitic glaucoma and when the eye pressures exceed 40 mmHg.[3]

Procedure

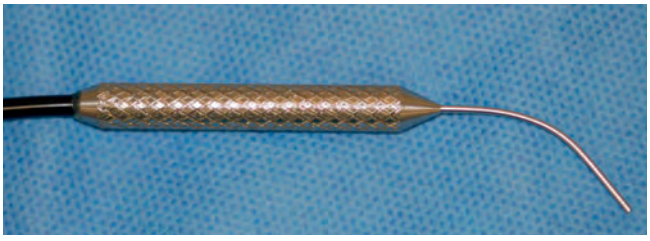
ECP utilises a hand-held endoscopic probe that is inserted intraocularly and provides the surgeon with the precision required to selectively target the ciliary body processes, thereby limiting subsequent laser damage to the nearby structures. The instrument approach can be either an anterior approach through the pupil or posteriorly by entering the eye through the pars plana. The pars plana approach is best for aphakic eyes or eyes with a posterior synechiae and uses an incision into the sclera which is placed 3.5 mm posterior to the limbus.[2-4] The anterior approach uses a limbal incision. This approach is more commonly used and is suitable for phakic, posterior chamber pseudophakic or aphakic eyes.[3]

The incision used in phaco-emulsification cataract extraction is easily adapted to the ECP probe, allowing the two procedures to be performed concurrently. Though ECP can be performed independently, combining these two procedures reduces any additional risk to the patient associated with an extra procedure. When combined, the ECP procedure is completed after the cataract has been removed and the intraocular lens (IOL) is in position.

Following IOL placement, viscoelastic is injected underneath the iris to expand the area surrounding the ciliary body. The endoscopic probe is inserted through the limbal incision and directed beneath the iris (Figure 1). Within the 20-gauge endoscopic probe are four components: a light source, 810 nm diode laser, helium-neon aiming laser and video camera (Figure 2).[5] The video feed is directed to a separate screen that the surgeon uses to visualise the procedure.



▲ Figure 1. ECP probe placed intraocularly through limbal incision



▲ Figure 2. Endoscopic probe used in ECP

A single incision site allows 300 degrees of treatment and a second incision must be placed to treat a full 360 degrees.[2] While treatment of 180 degrees is necessary to achieve significant reduction in IOP, treatment should be applied to 360 degrees to reach target IOPs in many adults.[4] In paediatric patients, the response is more unpredictable and treatment is applied to 180 to 270 degrees in the initial procedure.[4] If the response is insufficient, the surgery can be repeated to ablate the remaining ciliary processes.

During the procedure, each individual ciliary process is visualised while the photocoagulation laser is applied. The foot-pedal-controlled diode laser emits pulsed, continuous energy that is absorbed by the pigment, causing necrosis of the ciliary body epithelium and stroma (Figure 3).[2,6] As each ciliary process is treated, it shrinks and becomes whitened (Figure 4).

This visual feedback is useful for the surgeon to monitor for the treatment's endpoint. If too much energy is applied to a single ciliary process, it may rupture, causing bleeding and reduced visibility.[3] Direct visualisation is also important to ensure adequate treatment. In pseudoexfoliation cases, the processes are smaller and covered with pseudoexfoliative material (Figure 5). More energy is required and the laser needs to be placed closer to the process to achieve adequate absorption and atrophy.[3]

▲ Figure 3. ECP instrument panel

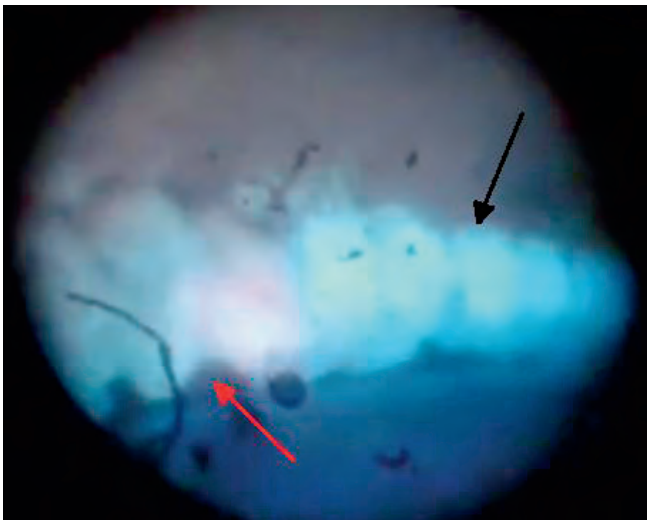
As with any procedure, there is risk of infection. Therefore, the patient should be prescribed a topical antibiotic beginning three days prior to surgery and continued for one week after surgery. To help with inflammation and prepare the eye for surgery, a topical non-steroidal anti-inflammatory is used for three days prior and one week following the procedure. A topical steroid is also used following surgery, every two hours for the first week followed by a slow taper for one month. This regimen is similar to that of post-cataract care, making the two surgeries even more compatible.

Patients should remain on their anti-glaucoma medications for up to one month following the procedure. This allows the IOP to stabilise. Anti-glaucoma medications should be stopped one at a time at one month intervals if multiple medications were used prior to surgery. The number that can be discontinued will depend on the final target IOP.

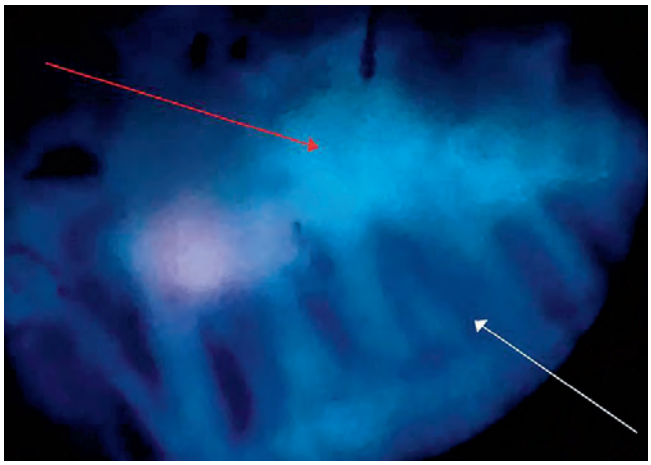
ECP versus TCP

While similar in mechanism of action, trans-scleral cyclophotocoagulation (TCP) has an entirely different approach. TCP is done extraocularly through the sclera. The biggest limitation of TCP is the lack of direct visualisation of the ciliary body, leading to increased risk of collateral

Continued page 24



▲ Figure 4. Ciliary processes viewed during ECP. The whitening and shrinking of the ablated ciliary processes is seen on the right (black arrow). The ciliary processes on the left have not been ablated (red arrow).



▲ Figure 5. Ciliary processes in an eye with pseudoexfoliation. The pseudoexfoliative material is apparent on the zonules (white arrow) and ciliary processes (red arrow).

Endoscopic cyclo-photocoagulation laser

From page 23

damage and variable treatment efficacy. A majority of the TCP misses the ciliary body processes and instead affects the pars plana, which is located further posteriorly.[2] Even when the application is more accurate, most of the ciliary processes are destroyed only partially. This difficulty arises because the only feedback of the treatment's endpoint is an audible popping sound, indicating over-treatment has occured.[2]

The efficacy of TCP is variable due to the nature of the approach. The absorption depends on the thickness of scleral tissue, angle of the probe and the pressure applied against the sclera.[2] As little as 35 per cent of the energy emitted during TCP actually reaches the pigmented epithelial cells of the ciliary body, with the remainder being absorbed by other tissues or just being reflected.[2,6]

Following TCP, patients may experience pain, inflammation, cystoid macular oedema, hypotony and a phthisical eye with permanent vision loss.[3] Due to the increased risk of these vision-threatening complications, this blind approach is often reserved for eyes with limited visual potential or severe pain associated with uncontrolled increased IOP.

Efficacy of ECP

The main mechanism of IOP reduction in ECP is the destruction of the secretory ciliary epithelium.[6] Additional effects are from vascular damage and ischaemia of the ciliary body processes.[6] Due to vascular damage and uveitis, the IOP decreases significantly during the first week, increases over the course of the next month and finally stabilises.[6] Following ECP, ciliary epithelial remodelling may occur to a greater extent than with TCP, leading to regression of treatment and increased IOP. However, this newly-remodelled epithelial tissue's capability to secrete aqueous is unclear.[6]

A greater reduction in IOP and higher rate of success are seen in eyes with higher preoperative IOP or chronic angle-closure glaucoma.[7] Patients with a secondary glaucoma have shown a greater lowering effect on IOP than POAG. Patients with POAG had an average of 22 per cent IOP decrease while secondary glaucoma patients had an IOP decrease of 29 per cent.[2] Previous failed glaucoma surgery may also have an impact on efficacy of ECP as patients with no previous surgical intervention showed a decrease in IOP of 30 per cent, while those who had had previous surgeries had a decrease of 17 per cent.[2]

ECP is safer and comparable in IOP lowering effect when compared against other glaucoma surgeries. A randomised, prospective study found a similar decrease in IOP with a 29 per cent reduction with ECP and a 32 per cent reduction with trabeculectomy.[2] One study found no significant difference in effect of ECP compared to that of an Ahmed valve (shunt surgery) at 20 months.[6] While the effect was the same, complications associated with the Ahmed valve were more frequent than with ECP.[6]

Cataract surgery itself perpetuates a certain degree of IOP-lowering effect; however, this effect tends to diminish after two years. When paired with ECP, the decrease in IOP remains significant with an additional benefit of decreased anti-glaucoma medication dependence.[3,7] At year three, there is no significant difference in IOP between cataract extraction alone and the combined procedure; however, there is a significant decrease in the number of topical medications used.[7] In a study with multiple types of glaucoma, the mean reduction in IOP with the combined procedure was 20 per cent at one year.[7] In a Brazilian study of 368 eyes, the average IOP decreased from 23.1 mmHg to 12.1 mmHg at two years. The use of anti-glaucoma medication also was reduced from 1.4 to 0.4 ophthalmic medications.[7]

When cataract extraction and ECP are combined, the manipulation of the ciliary processes with ECP also has an effect on the position of the implanted IOL. A study of 139 eyes found a significant increase in deviation from the desired refractive endpoint when ECP was performed in conjunction with cataract surgery.[1] Eyes which were treated with ECP had an average myopic shift of 0.54 dioptres compared to an average 0.26 dioptres myopic shift outcome of eyes that had cataract surgery alone.[1] Therefore, when the procedures are combined, consideration should be made when choosing the appropriate IOL power.

Complications of ECP include endophthalmitis, hyphaema, phthisis bulbi and hypotony although the incidence of these complications is rare. Neovascular and paediatric patients are at higher risk of long-term hypotony. Hypotony in these patients can be averted with more conservative laser treatment.[2,6]

Conclusion

ECP has been shown to be effective in managing multiple types of glaucoma. It has previously been reserved for end-stage glaucoma due to concerns of potentially visually compromising complications such as hypotony and phthisis bulbi. With the increase in safety compared to TCP, ECP should be considered earlier in the disease process. It should especially be considered in glaucoma patients who are also having cataract surgery as the two procedures can be combined without any significant additional risk. ▲

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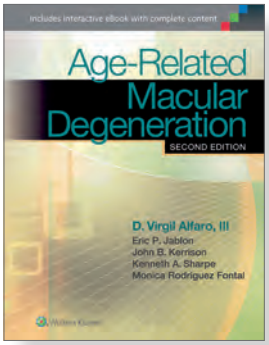

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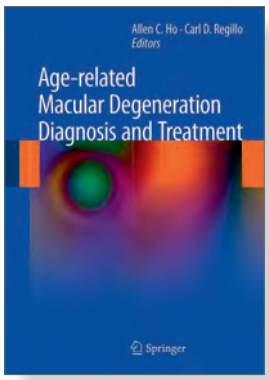

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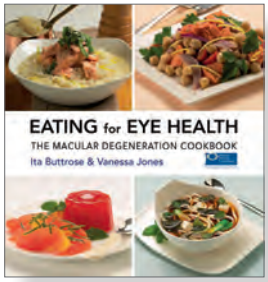

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