Volume 1, Issue 1

THE MYOPIA ISSUE

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We are tremendously proud of the content in this issue and the forthcoming issues in 2022. We are also incredibly grateful to all the authors who have contributed to this journal. Of course, we also want to thank all the advertising partners for their support in helping us launch this exciting peer-to-peer initiative.

As the journal continues to grow, we welcome new ideas, new topics and new submissions which can be sent directly to info@catalytichealth.com. The journal's aim is to provide practical and pragmatic real-world content that helps to inform disease management for Canadian clinicians.

We do sincerely hope you enjoy this first issue, and we look forward to your readership and your ideas for future articles as we grow and expand the reach of this publication!

Best wishes,

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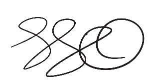

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The evolving role of OCT in pathologic myopia

Carl Shen, MD Mark Seamone, MD Mark Greve, MD

Introduction

The global burden of myopia represents a significant public health concern that is expected to continue to increase in the near future. It is estimated that 50% of the world's population will be affected by myopia by 2050[1], with a disproportionately high prevalence in Asia. High myopia, where the spherical equivalent refractive error is equal to or higher than 6.00 diopters, is expected to increase in prevalence from 2.7% to 10% during this period[1]. The severity of myopia is of paramount concern to clinicians as higher levels are associated with pathologic myopia (PM) and increased risk of vision loss. Pathologic myopia, as recently defined by the International Myopia Institute, is an excessive axial elongation associated with myopia that leads to structural changes in the posterior segment of the eye that can lead to loss of best-corrected visual acuity[2]. These structural changes and their complications include posterior staphyloma, myopic choroidal neovascularization, myopic maculopathy, myopic traction maculopathy, dome shaped maculopathy, optic disc changes and glaucoma associated with myopia, and retinal detachments.

The advent of optical coherence tomography (OCT) has facilitated the characterization, diagnosis, and management of several of these complications associated with PM and will be the focus of this article. Imaging the highly myopic eye represents a crucial step in the identification of these complications and poses its own unique challenges. Researchers have demonstrated the advantage of 3D cube scans in the detection of pathology compared to 1- and 5-line rasters[3]. Using vertical scanning patterns aligning where the radius of curvature is larger relative to the horizontal plane of the myopic eye can minimize associated artifacts. Wide scans, facilitated by emerging technologies such as swept- source OCT and ultra wide-field OCT, are useful in cases of PM where the pathology can initiate peripherally[4].

Myopic Choroidal Neovascularization

It is estimated that 5-11% of patients with PM will develop myopic choroidal neovascularization (MCNV)[5]. Effective treatment, primarily with anti-VEGF agents, can alter the natural history of MCNV that would otherwise result in significant vision loss.

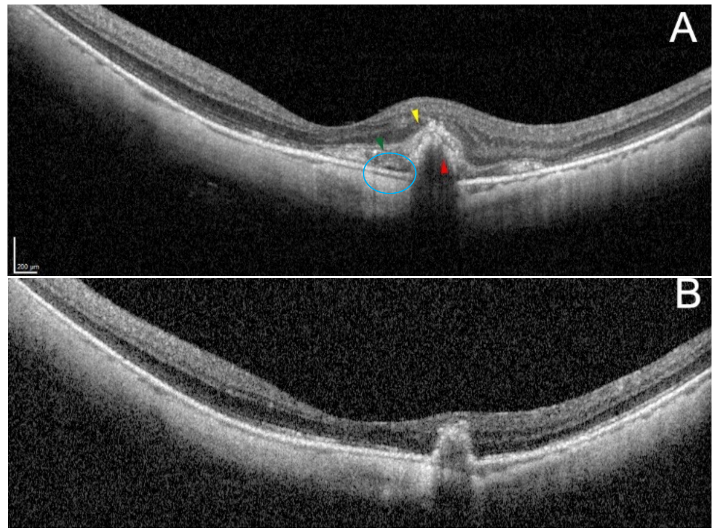

Figure 1. A) Pigment epithelial detachment (red) with overlying disruption of the ellipsoid and external limiting membrane (yellow arrow), subretinal hyper-reflective material (green arrow), and small amount of subretinal fluid (blue circle).
B) Follow up OCT after 1 year of "treat and extend" anti-VEGF. Note the consolidation of the borders of the PED, and absence of subretinal fluid and sub-retinal hyperreflective material.

Diagnosis: MCNV often appears as a type 2 CNV on OCT with a hyperreflective mound above the retinal pigment epithelium (RPE) **(Figure 1)**. Additional OCT features of MCNV include a loss of the ellipsoid layer and Bruch's membranes, absence of the external limiting membrane, and retinal thickening[6]. The degree of intra- and subretinal fluid by OCT is less in MCNV compared to other etiologies and thus may be a less sensitive marker for its detection if used in isolation[7]. At the same time, features such as the absence of subretinal fluid, may aid in the differentiation of MCNV from neovascularization caused by age-related macular degeneration, and inflammatory causes[8]. The agreement between OCT and reference standard fluorescein angiography (FA) in the detection of MCNV has been reported to be about 70-90%[9,10]. Optical coherence tomography-angiography (OCT-A) may be more sensitive, being able to detect over 90% of MCNVs[11], and may also be useful in differentiating MCNV from hemorrhage related to lacquer cracks[12]. However, to date, FA remains the gold standard for diagnosis of MCNV.

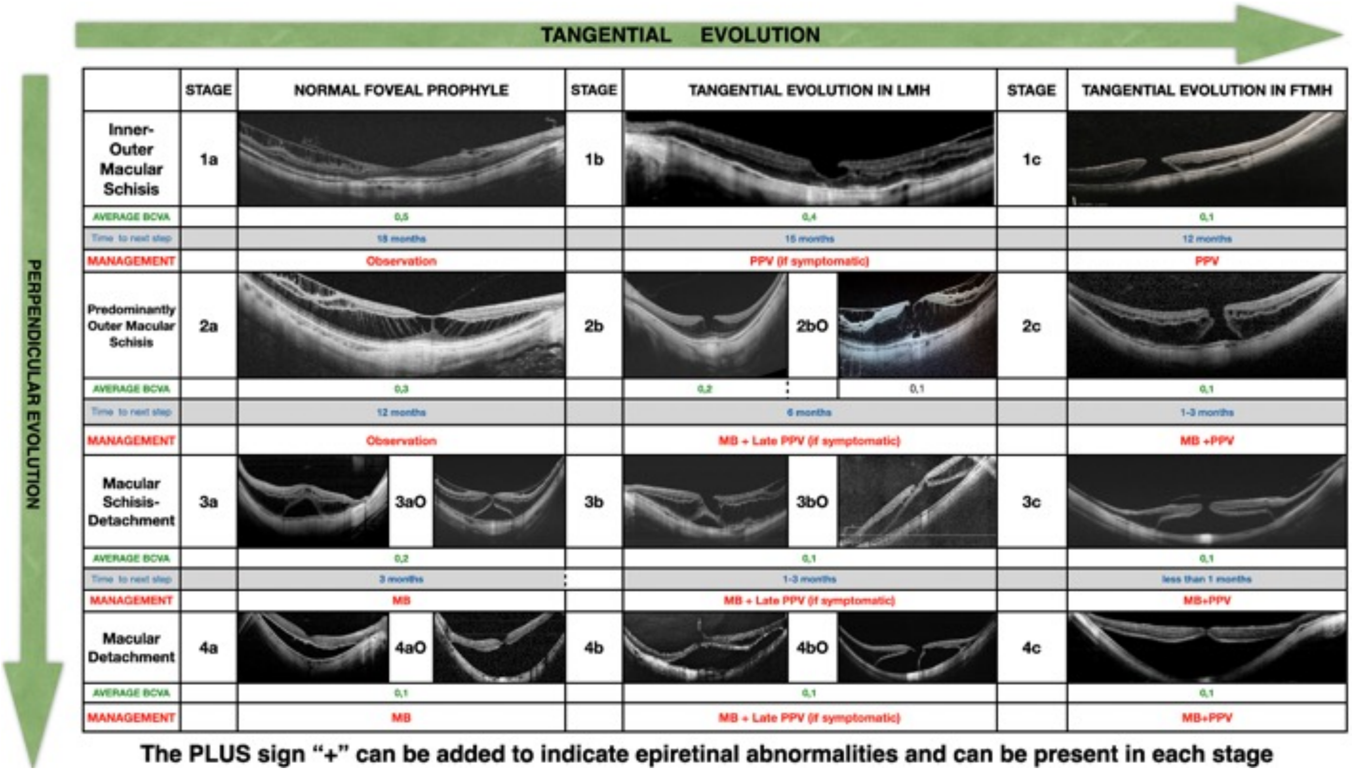

Figure 2. Myopic Tractional Maculopathy Staging System based on tangential and perpendicular evolution of changes in MTM; Adpated from Parolini, B et al., 2021"

Disease Monitoring: OCT provides a rapid, non-invasive means of monitoring disease activity and response to treatment. A characteristic "fuzzy area" of the hyperreflective lesion often corresponds with active FA leakage[13]. Subretinal hyperreflective exudation is similarly predictive for MCNV activity[14]. Several studies have sought to identify OCT characteristics capable of prognosticating development of MCNV, response to treatment, and recurrence. Eyes with MCNV have been found to have reduced choroidal thickness compared to fellow eyes[15]. Thinner choroids have also been associated with recurrence of MCNV at the 1-year follow up and with a lower resolution rate after single injections of anti-VEGF therapy[16].

Myopic Traction Maculopathy

Myopic traction maculopathy (MTM) refers to a collection of conditions including vitreomacular traction, epiretinal membrane, lamellar hole, macular hole, myopic foveoschisis/macular schisis that are unified by their underlying etiology of tractional forces acting on the retina in highly myopic eyes[2]. MTM may affect up to 30% of patients with PM[17]. OCT findings in MTM have recently been organized into a staging system[18] called the MTM Staging System (MSS) **(Figure 2).** The MSS organizes the evolution of changes from forces perpendicular to the retina (stage 1-4) with forces tangential to the retina (a-c), to arrive at a number and letter staging (e.g 2B). Stage 1 represents inner-outer macular schisis, stage 2 represents predominantly outer macular schisis, stage 3 represents macular schisis detachment, and stage 4 represents macular detachment. Stage *a* represents a normal foveal profile, stage *b* represents lamellar hole changes, and stage *c* represents macular hole changes. Epiretinal abnormalities are designated with a "+" and can occur at any stage.

Myopic Macular Schisis: Myopic Macular Schisis (MMS), or foveoschisis, is evident on OCT as retinoschisis in multiple retinal layers, bridged vertically by presumed Müller cells. Separation of the internal limiting membrane (ILM) from the remaining retinal layers can occur. In early stages (stage 1-2 MSS), the ellipsoid zone is usually well-preserved and visual function is minimally affected. A large natural history study of MTM[19] found that over 36.2 ± 6.2 months of follow up, 11.6% (n= 24/207) of eyes experienced progression of MTM **(Figure 3)**, with higher rates of progression (42.9%) in eyes with more extensive MMS.

Myopic Macular Hole: Macular holes are reported to occur in approximately 8.4% of eyes with PM[20]. Data has shown that there is a differentiation between two types of macular holes in myopic eyes. The first is a "flat type", characterized by cystic changes at the edge of the hole and similar success rates of surgical closure similar to macular holes in non-myopic eyes[21]. The second is a "schisis type" characterized by accompanying retinoschisis changes which are evident at the borders of the hole. These latter holes have a lower success rate of closure and are at high risk of progression to macular hole retinal detachments.

Treatment: Intervention at the MMS stage is generally guided by progressively worsening visual symptoms, metamorphopsia, and reduction in visual acuity. Treatment consists largely of surgical intervention across the spectrum of MTM and comprises vitrectomy and/or macular buckling. In a prospective, non-randomized study of 62 consecutive eyes, rates of visual improvement with surgery were shown to be greater when foveal detachment or disruption was evident[22]. Several variations of vitrectomy have been proposed, vitrectomy alone, with membrane peeling, with ILM peeling, ILM flaps, subretinal expansion, retinal incisions, and grafting procedures of amniotic membrane or retinal tissue. There has been renewed interest in macular buckling for the management of MTM with the hypothesis that anatomical and functional outcomes may be superior to vitrectomy alone[23,24] **(Figure 4).** Several modifications to the standard macular buckling technique have been proposed to facilitate the surgery including modified buckle shapes[24] and internal chandelier placement[25]. Intraoperative OCT has been used to augment surgery in MTM eyes through the detection of residual epiretinal membrane (ERM), ILM, and cortical vitreous and has revealed undetected holes post peeling[26]. Clinicians have proposed a treatment algorithm based on their MSS using permutations of vitrectomy and macular buckling depending on the stage of disease, with vitrectomy better suited to address tangential traction and macular buckling better suited to address perpendicular traction[27]. Such studies underscore the utility of OCT in cases of MTM in potentially identifying optimal treatment strategies corresponding to pathophysiologic principles.

Myopic Maculopathy

Myopic maculopathy (MM), or myopic macular degeneration, are terms often used interchangeably to describe several of the degenerative features in PM that contribute loss of best corrected visual acuity in myopia. The most widely used classification system is the meta-analysis of pathologic myopia (META-PM)[28] which divides PM into five distinct categories as follows: category ‘0’: no myopic retinal lesions, category ‘1’: tessalated fundus, category ‘2’: diffuse chorioretinal atrophy, category ‘3’: patchy chorioretinal atrophy, and category ‘4’: macular atrophy. Additional features of “plus lesions” can be assigned for lacquer cracks, choroidal neovascularization, and Fuchs’ spots. MM tends to progress. Over a mean follow up of 18 years in a retrospective observational case series of 810 eyes, the authors reported progression of MM in 58.6% of all eyes, and 74.3% in eyes with pre-existing PM[29]. Notably, META-PM is based on fundus photographs

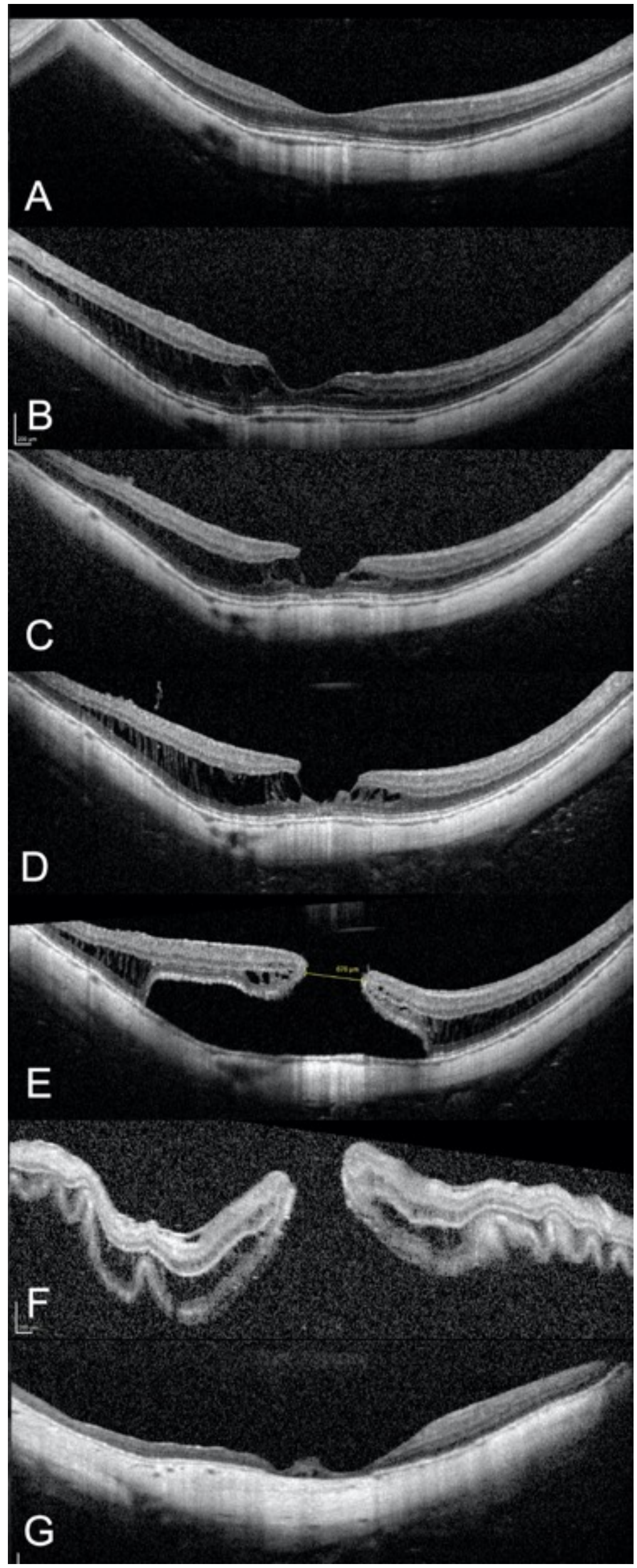

Figure 3. Progression of MTM from baseline (A) to predominantly outer retinal schisis/stage 2a at 3 years (B) followed by progressive outer retinal schisis and increasing lamellar changes/stage 2b at 4 years (C) and 5 years (D). 9 months after (D) the appearance of a macular schisis detachment/stage 3c was noted (E) with extension of the retinal detachment to outside the macula after another month (F). The patient was treatment with vitrectomy, membrane and ILM peel, and silicone oil with continued anatomical success and 20/200 vision at 2 years follow up but significant retinal atrophy (G)

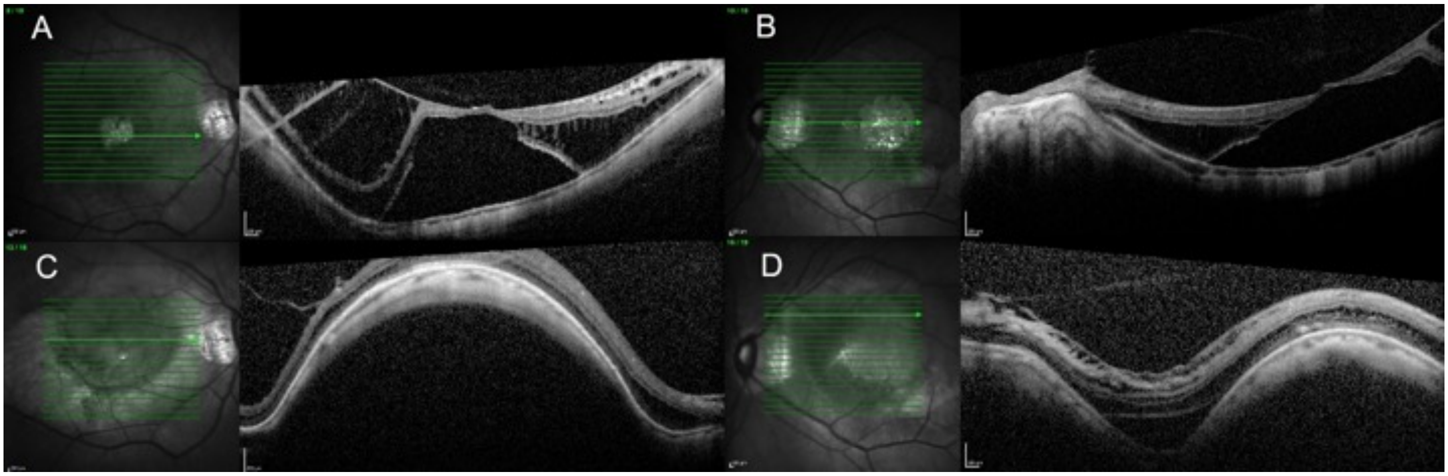

Figure 4. Pre-operative OCT of stage 4b MTM in the right (A) and left eye (B) of a -12 myope with symptomatic visual decline to 20/100 and 20/70 respectively. Post-operative OCT after successful macular buckling surgery in the right (C) and left eye (D) with reduction in retinoschisis and resolution of macular detachment. Visual acuity improved to 20/30 OU with a concurrent reduction of 5 diopters of myopia.

and does not incorporate OCT findings. Researchers have recently attempted to supplement the META-PM classification with OCT features and demonstrated that choroidal thinning is associated with the progression of myopic maculopathy. They established cut-off values of choroidal thickness of <56.5 μm located 3000 μm nasally from the fovea to define peripapillary choroidal thinning, and <62 μm subfoveally to define macular choroidal thinning, two terms proposed as the OCT equivalent to the subtypes of category 2 diffuse chorioretinal atrophy in META-PM. Unfortunately, to date, no established treatment for MM exists.

Conclusion

OCT has emerged as a foundational technology in the management of PM. The relative hypopigmentation of myopic fundi and microstructural changes associated with pathology means that biomicroscopy alone is insufficient in the assessment of PM eyes. Advances in OCT imaging continue to enhance our understanding of PM and it is imperative for the eyecare specialist to leverage these advantages in the care of our patients.

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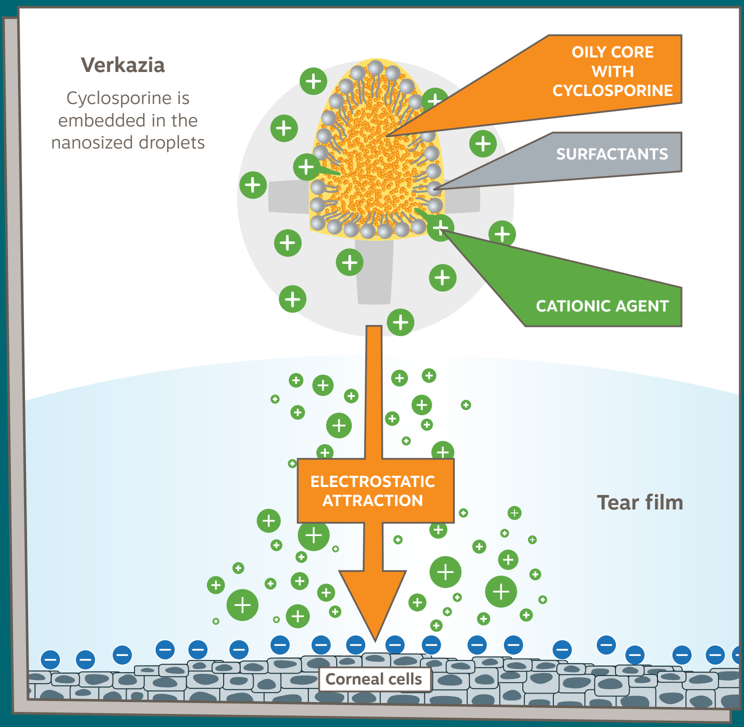

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Methods to treat myopia progression in pediatric patients

Michael J. Wan, MD, FRCSC

Background

Myopia is an enormous, and growing, public health issue across the globe. The prevalence of myopia has doubled in just the past 50 years and it is estimated that approximately half of the world's population (4.8 billion people) will be affected by 2050.[1,2] The increase has been especially pronounced in individuals of East Asian descent, where 80-90% of young adults are now myopic.[3] Myopia is now the most common cause of visual impairment and the second most common cause of blindness worldwide.[4]

While often considered a "correctable" cause of vision loss, people with myopia have an increased lifetime risk of complications, such as macular degeneration and retinal detachment, which can cause long-term visual impairment or even blindness.[5] Although all levels of myopia are associated with an increased risk of complications, the risk is substantially greater in people with high myopia (defined by the World Health Organization as a refractive error of ≤-5 diopters).[6] In addition to a large burden of visual impairment, myopia also has a significant global economic cost, estimated to be $250 billion per year in lost productivity, which is almost certain to rise.[7]

With these factors in mind, preventing the progression of myopia is a global public health priority. The purpose of this article is to review the currently available methods to treat myopia progression in children.

Treatment

Atropine

The use of topical atropine to treat myopia progression is supported by an impressive number of high-quality studies. The first randomized, placebo-controlled, double masked clinical trial (RCT), Atropine in the Treatment of Childhood Myopia Study (ATOM1), was published in 2006.[8] ATOM1 results demonstrated that daily 1% topical atropine reduced the progression of myopia in children aged 6-12 years old (both refractive error and axial length) compared to placebo over 2 years of treatment. However, eyes treated with 1% atropine suffered significant visual side effects related to cycloplegia and mydriasis, and there was a significant rebound effect after the atropine was stopped.[9]

The follow-up RCT (aptly named ATOM2) compared 3 lower doses of atropine – 0.5%, 0.1%, and 0.01% - over 2 years.[10] The main outcome measure of myopic progression was comparable between the different doses, but the 0.01% atropine dose was the best tolerated. In phase 2 of the ATOM2 trial, treatment was stopped for 12 months, and a rebound effect was again noted. The severity of the rebound was directly related to the concentration of atropine, such that the 0.01% atropine had the most sustained effect on minimizing myopic progression.[11] In phase 3 of the study, all children with myopic progression were treated with 0.01% atropine for 2 additional years and a convincing reduction in myopic progression was achieved.[12]

Since the publication of the ATOM trials, there have been additional studies on the optimal use of topical atropine. The Low-Concentration Atropine for Myopia Progression (LAMP) study compared even lower doses of atropine – 0.05%, 0.025%, and 0.01% - and found that 0.05% atropine was approximately twice as effective as 0.01% at reducing myopic progression over 2 years with no additional side effects.[13,14] However, longer-term results and the risk of rebound have yet to be published from the LAMP study. There are also ongoing clinical trials in North America and Europe aimed at addressing generalizability concerns in light of both the ATOM and LAMP studies having been conducted exclusively in children of East Asian descent. While the optimal treatment regimen for atropine may change with time, the cumulative evidence strongly supports the use of daily atropine (0.01% or 0.05%) to reduce progression in myopic children.

Orthokeratology (Ortho-k)

In addition to low-dose atropine, there are several non-pharmacologic treatments which have strong evidence of reducing myopic progression in children. Orthokeratology (Ortho-k) is one of the most well studied of these. Ortho-k involves the use of specially designed, rigid contact lenses which are worn overnight to flatten the central cornea. The cornea remains flattened for a period of time when the lenses are removed, allowing myopic children to achieve acceptable visual acuity without correction during the day. Ortho-k can correct refractive errors of approximately -5 diopters, but may induce optical aberrations and daytime vision can fluctuate.[15] Several systematic reviews and meta-analyses have concluded that ortho-k does reduce axial length elongation compared to control subjects.[16-19] However, the long-term effect and potential for myopic rebound with ortho-k have yet to be elucidated. The risk of complications is low when ortho-k lenses are used properly, but there are several reports of severe complications such as infectious keratitis.[20] Therefore, ortho-k is an effective management option for myopic control, and the only modality that improves daytime vision without correction, but it does require nightly application of specially designed contact lenses and has a small risk of serious adverse effects.

Peripheral myopic defocus lenses (contact lenses and glasses)

Another non-pharmacologic intervention for myopic progression is the use of peripheral defocus lenses. The proof-of-concept for the use of peripheral defocus lenses came from animal studies demonstrating that artificially focusing light in front of the retina (i.e. myopic defocus) could inhibit growth of the eye.[21] To harness this effect, contact lenses were designed with a central zone to correct distance refractive error combined with peripheral zones of additional plus power to create myopic defocus.[22,23] These soft contact lenses have been shown to be effective in reducing myopic progression in high-quality RCTs.[24] Furthermore, follow-up studies have reported no evidence of myopic rebound after discontinuation.[25] In addition, vision-related quality-of-life measures have been shown to be similar (and may even be superior) in children wearing the contact lenses compared to those wearing spectacles.[26] However, like all contact lenses, peripheral defocus lenses require proper care and have a small risk of keratitis.

For children who cannot manage the daily use and care of contact lenses, peripheral defocus spectacles will soon be an option. Recent RCTs have shown that peripheral defocus spectacles also effectively slow myopic progression for at least 3 years compared to single vision glasses.[23,27] Peripheral defocus spectacles present an attractive option as a non-pharmacologic intervention with no risk of microbial keratitis. However, the advanced design means that these spectacles are likely to be significantly more costly than single vision glasses.

Environmental Factors

The dramatic increase in myopia in just a single generation strongly suggests that environmental factors play a significant role. A large prospective study from the Netherlands found that the risk of childhood myopia was almost equally related to genetic and environmental factors.[28] Another epidemiological study compared age- and ethnicity-matched children (to minimize the effect of genetics) in Singapore and Sydney and found that the prevalence of myopia was almost 10 times higher in Singaporean children.[29] This has led to a concerted effort to identify which environmental factors are most influential and, as such, potential targets for behavioral modification. To date, outdoor activity time has been found to be the most powerful environmental factor contributing to childhood myopia.[29,30] A recent meta-analysis looking at five studies with over 3,000 children aged 6 to 12 years concluded that, in children who spent more time spent outdoors, there were fewer de novo cases of myopia and less myopic progression.[31] Some studies have also identified near work as a risk factor for myopia,[30] but others have not.[29] Therefore, the evidence to date indicates that environmental factors do play a role in the development of myopia and that more outdoor time is strongly associated with decreased risk.

Comparison of Treatments

Having several effective treatments for myopia progression is critical in ensuring optimal patient outcomes.

TREATMENT	PROS	CONS
Topical atropine (0.01% or 0.05%)	Strong evidence Minimal side effects No risk of infectious keratitis	Needs to be compounded Does not replace refractive correction
Ortho-k	Able to see well uncorrected during the day Parents can perform all aspects of lens care	Requires specially designed lenses Small risk of infectious keratitis Cannot correct high myopia Daytime vision can fluctuate
Peripheral defocus lenses	Able to correct high myopia Option of contact lenses or glasses	Small risk of infectious keratitis with contact lenses Higher cost than single vision glasses or contact lenses
Environmental factor	Very cost-effective May augment other treatments	Uncertainty about the effect of some factors (e.g. near work)

Table 1. Pros and cons of currently available methods to treat myopia progression in children; courtesy of Michael Wan, MD

Unfortunately, the optimal treatment for a given patient is often uncertain as there are few studies directly comparing various interventions.[32] A meta-analysis[33] and a Cochrane Review [34] synthesized the available evidence in order to indirectly compare treatments and both concluded that the most effective intervention for myopia control in pediatric patients was pharmacologic (i.e. atropine) followed by specially designed contact lenses (i.e. ortho-k and peripheral defocus lenses).

The "best" treatment also depends on individual patient preferences. Parents and children may prefer the strong scientific evidence supporting the use of atropine, the correction-free vision provided by ortho-k, the soft contact lenses of peripheral defocus lenses, or the convenience of peripheral defocus spectacles. Combining treatments, such as topical atropine and ortho-k, may provide synergistic effects but these combined treatment approaches have yet to be studied. Finally, it is always a good idea to encourage children to spend time outdoors, an intervention with important benefits and minimal cost (**Table 1**).

Who to Treat and How to Monitor?

Existing atropine studies on the management of myopia in pediatric patients have been restricted to children who are already myopic and at least 4 years old. In contact lens studies, the age cut-off has been 8 years old and inclusion criteria have similarly included only those subjects with pre-existing myopia.[34] However, these age cut-offs are chosen specifically for clinical trials and may not reflect clinical practice. It is reasonable (and possibly beneficial) to start treatment in younger children if myopia develops early. It may also be useful to treat high-risk children prior to the development of myopia (the ongoing ATOM3 trial is treating high-risk children with atropine to see if myopia prevention is possible).[35]

Once treatment has been initiated, regular monitoring of refractive error and axial length is crucial. It is also important to monitor for any complications, such as photophobia with atropine use or keratitis from contact lens use. Most existing clinical trials have a treatment duration and/or evaluation timepoint at 2 years, but the optimal treatment duration is unknown. It is reasonable to treat for longer periods of time (many centers continue treatment into late adolescence) or restart treatment if myopic rebound is detected.[36]

Summary

Myopia in children is a significant and increasing worldwide public health issue. There are several evidence-based methods available to treat myopia progression. Topical atropine has the strongest evidence of efficacy; ortho-k provides correction-free vision during the day; and peripheral defocus lenses provide the option of soft contact lenses or spectacles. The optimal method in any given situation must consider the likelihood of success based on the evidence available to the clinician as well as the preferences of the patient. Initiating treatment early and monitoring closely for effect and tolerance can help to minimize the economic burden and vision-threatening complications of myopia in our pediatric patient population.

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Spot the differences: challenges in detecting glaucoma in the myopic patient

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Clinical Case Conundrum:
A 53-year-old male was referred for glaucoma consultation regarding paracentral visual field defects in the right eye. Prior to phacoemulsification and intraocular ocular lens (IOL) implantation his refraction was -10.50 D and -11.00 D in the right and left eyes, respectively. Axial length was 28 mm in both eyes (OU). He was medically healthy. Visual acuities were 20/25 OU. Intraocular pressures (IOPs) were 16 mmHg OU. Central corneal thicknesses were 652 µm OU. Anterior segment, gonioscopy, and fundus findings are indicated in **Figure 1.** Optical coherence tomography (OCT) analysis of the retinal nerve fibre layer (RNFL) is shown in **Figure 2**. This patient's visual fields and structure-function correlations are highlighted in **Figure 3.**

Asymptomatic myopic patients often present with normal intraocular pressures, anomalous nerves, and visual field defects that are challenging to interpret in the context of one another. This is further complicated by myopia being a known risk factor for glaucomatous progression. Decisions regarding diagnosis and management in this patient population remain challenging.

Epidemiology:
Myopia is a growing epidemic. By 2050, it is predicted that 5 billion people (50% of the global population), will have myopia. Of these, 1 billion are projected to incur vision-threatening complications of high myopia.[1] High, or pathologic, myopia is defined as a spherical equivalent > -6.00 D or an axial length > 26.5 mm.[2] Patients with high myopia are at higher risk for several conditions believed to arise from excessive axial stretching of the globe, including retinal atrophy and degenerations predisposing to detachments. Myopic patients can lose central vision from a host of macular conditions including choroidal neovascularization, chorioretinal atrophy, peripapillary crescents, Bruch's membrane dehiscences, and a thin macular choroid, thereby limiting visual potential.[3,4]

Several landmark trials have identified myopia as an important risk factor for developing primary open angle glaucoma (POAG).[5] Glaucoma represents a group of disorders culminating in optic neuropathy with stereotypic optic nerve changes, loss of retinal ganglion cells, and corresponding visual field defects.[6] High myopia may increase the risk of POAG by as much as six-fold[7] and make patients more susceptible to incurring POAG at an earlier age.[8] POAG often presents despite "normal" pressures, and several studies have found plausible associations between myopia and normal-tension glaucoma.[5] In high myopes, it has been hypothesized that axial stretching or torsional forces at the lamina cribrosa may induce glaucoma through axonal strain.[9,10] Notwithstanding their frequent coexistence, glaucoma may be challenging to diagnose in high myopia. Due to departures from normative data for eyes with myopia and glaucoma, management of the coexisting conditions remains a common clinical conundrum.

Optic Nerve Analysis:
Glaucomatous optic nerve changes are often difficult to detect in myopic nerves for several reasons. With increasing axial length, the position of the optic disc can be displaced nasally in relation to the fovea, resulting in a rotation or 'tilt' of the disc with an associated shift in the Bruchs' Membrane opening and affording only an oblique view of the optic nerve head (ONH).[11,12] The ONH may be more pallid, reducing contrast between the pink neuroretinal rim and the optic cup. Distinguishing zones of peripapillary atrophy (PPA), with the increase in the beta zone having been linked to glaucoma, may be difficult. The lamina cribrosa can stretch and become thin. Further, choroidal thinning and subsequent retinal pigment epithelium (RPE) atrophy can make it difficult to detect peripapillary RNFL changes.[12] However, in myopes, RNFL defects may be an early sign of glaucoma, especially if they involve the papillomacular bundles.[13]

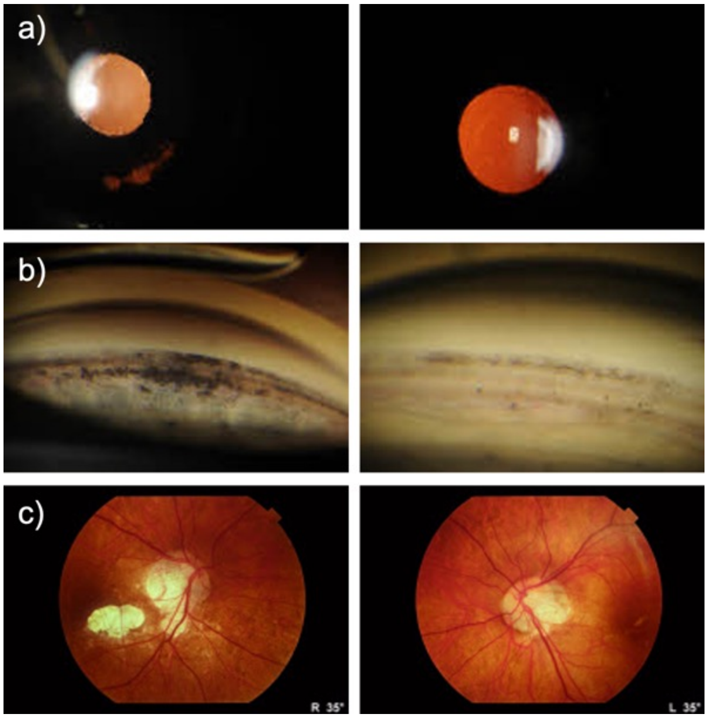

Figure 1: Clinical photograph compilation of salient exam findings including a) transillumination defects OD, b) pigment deposition OD on gonioscopy, c) fundus photographs of the optic nerve heads and peripapillary retina; courtesy of Cindy Hutnik et al.

Interpreting OCT measurements of the RNFL can also be nuanced in myopic patients. The normative database for the Cirrus® (Zeiss, Dublin, California) HD-OCT contains data from only 284 individuals, with limited representation from high myopes and hyperopes.[14] The lack of a myopic reference often biases clinicians towards "red disease" in which the OCT suggests abnormal thinning when the patient in fact has a healthy RNFL (a false positive).[15,16] Indeed, the disc-margin definition algorithm on HD-OCT can be influenced by myopic changes such as tilt, magnification or media artefact and scan circle misalignment from peripapillary atrophy. The superotemporal and inferotemporal RNFL bundles can be temporally displaced. Symmetry, correlation of other clinical parameters, and serial observations are essential, in the evaluation of a myopic patient who is considered at risk for glaucoma.[14,17]

Visual Fields:

Visual fields may also be difficult to interpret in myopic patients. Significant PPA can cause visual field defects. Macular pathology, including choroidal neovascularization or atrophy, can cause paracentral scotomas mimicking glaucomatous field loss, especially the paracentral changes seen in normal-tension glaucoma (NTG).

Sub-foveal choroidal thickness has been shown to be the most significant predictor of visual acuity in highly myopic eyes,[3] and choroidal and outer retinal thinning has been implicated in early visual field defects in myopes.[18] High myopes can have higher mean deviations[19] and lower threshold sensitivities than emmetropes.[20] Therefore, even at a microarchitectural level, myopic macular degenerations can confound visual fields in glaucoma evaluations. It is interesting to note that choroidal thinning has also been linked to the progression of visual field damage in a cohort of NTG patients, possibly implicating choroidal thinning in NTG pathogenesis.[21]

Structural Imaging: New Frontiers and New Management Considerations:

A host of novel imaging techniques may enhance our ability to detect glaucoma in myopic patients. For instance, new sd-OCT imaging strategies may more accurately detect the neuro-retinal rim (NRR). The Bruchs' membrane opening minimal rim width (BMO-MRW) is the minimum distance from the BMO to the internal limiting membrane. Measuring the NRR using the BMO-MRW has been shown to reduce rates of false positive errors in healthy myopic eyes with tilted ONHs, yielding more accurate RNFL analyses.[11,22]

Other imaging approaches have been introduced to better differentiate between glaucomatous optic neuropathy and myopia. Ganglion cell analysis in sd-OCT relies largely on the fact that retinal ganglion cells in the peripapillary RNFL (ppRNFL) are mainly located within the macula. Given the artefactual OCT changes that can confound analysis in myopic eyes, OCT of the ganglion cell-inner plexiform layer (GC-IPL) may aid in the differential diagnosis between glaucoma and myopia, especially in myopic eyes where the optic disc structure may introduce inaccuracies in ppRNFL measurements. Compared to RNFL analyses, macular ganglion cell complex parameters may be less susceptible to artefacts from refractive error or ONH morphology.[23]

Various scoring systems for identifying glaucoma in myopic eyes have been proposed. One study involving 195 highly myopic eyes identified that the GC-IPL hemifield test has an especially high sensitivity for discriminating between myopia and glaucoma. This test measures GC-IPL thickness differences across the horizontal temporal raphe on an OCT macula. The presence of temporal hemifield asymmetry on OCT GC-IPL thickness maps ("raphe sign") can be a particularly useful parameter for detecting glaucomatous changes in high myopes.[22]

In recent years, many clinicians have advocated for the development of an OCT normative database of myopic eyes as a more meaningful reference group for clinical decision making. Several small myopic normative databases have been developed for these purposes and have shown promise in improving the specificity and sensitivity for detecting RNFL abnormalities in eyes with

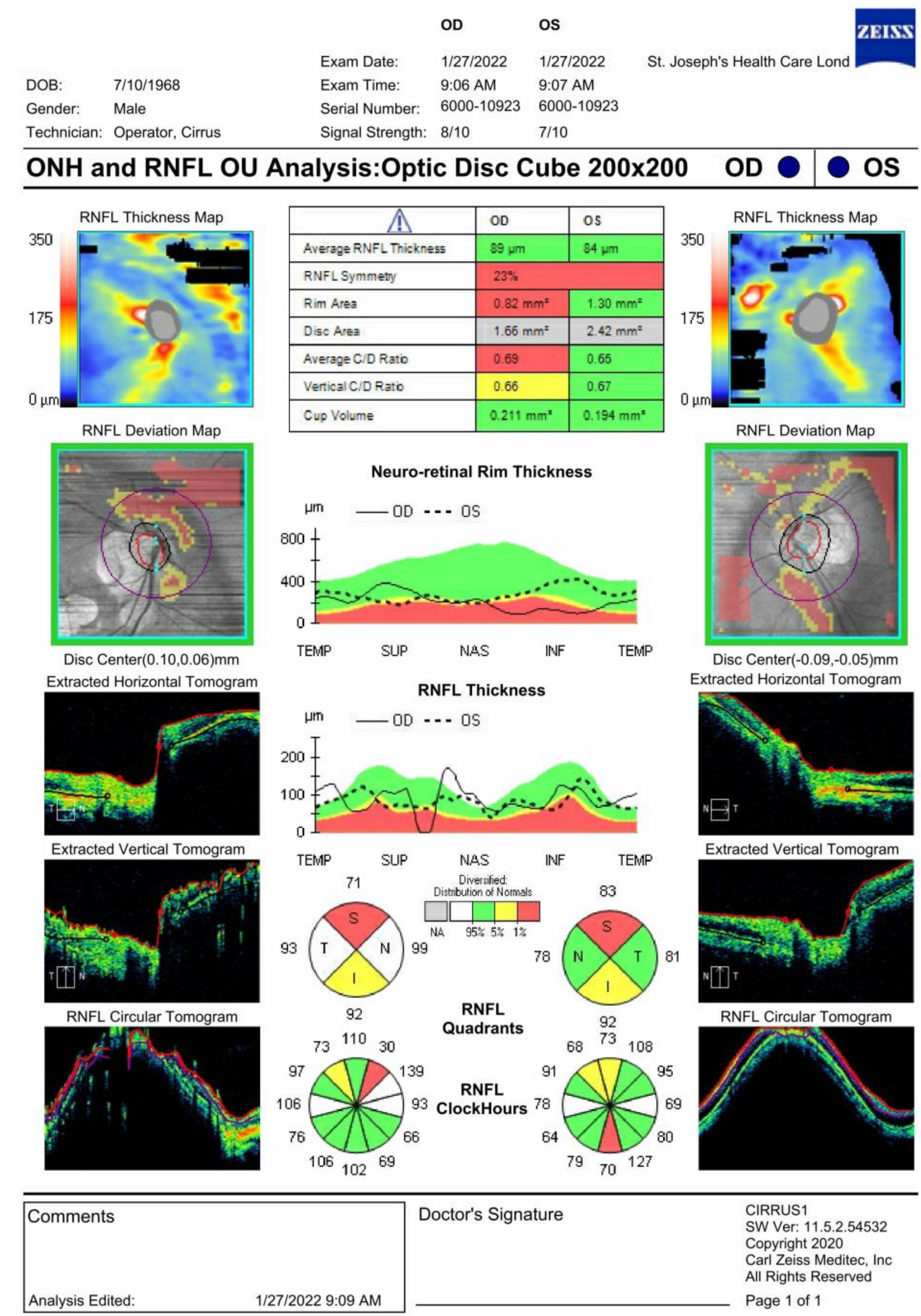

Figure 2: Zeiss Cirrus® HD-OCT analysis of the retinal nerve fibre layer queried superior thinning OU and inferior thinning OS; courtesy of Cindy Hutnik et al.

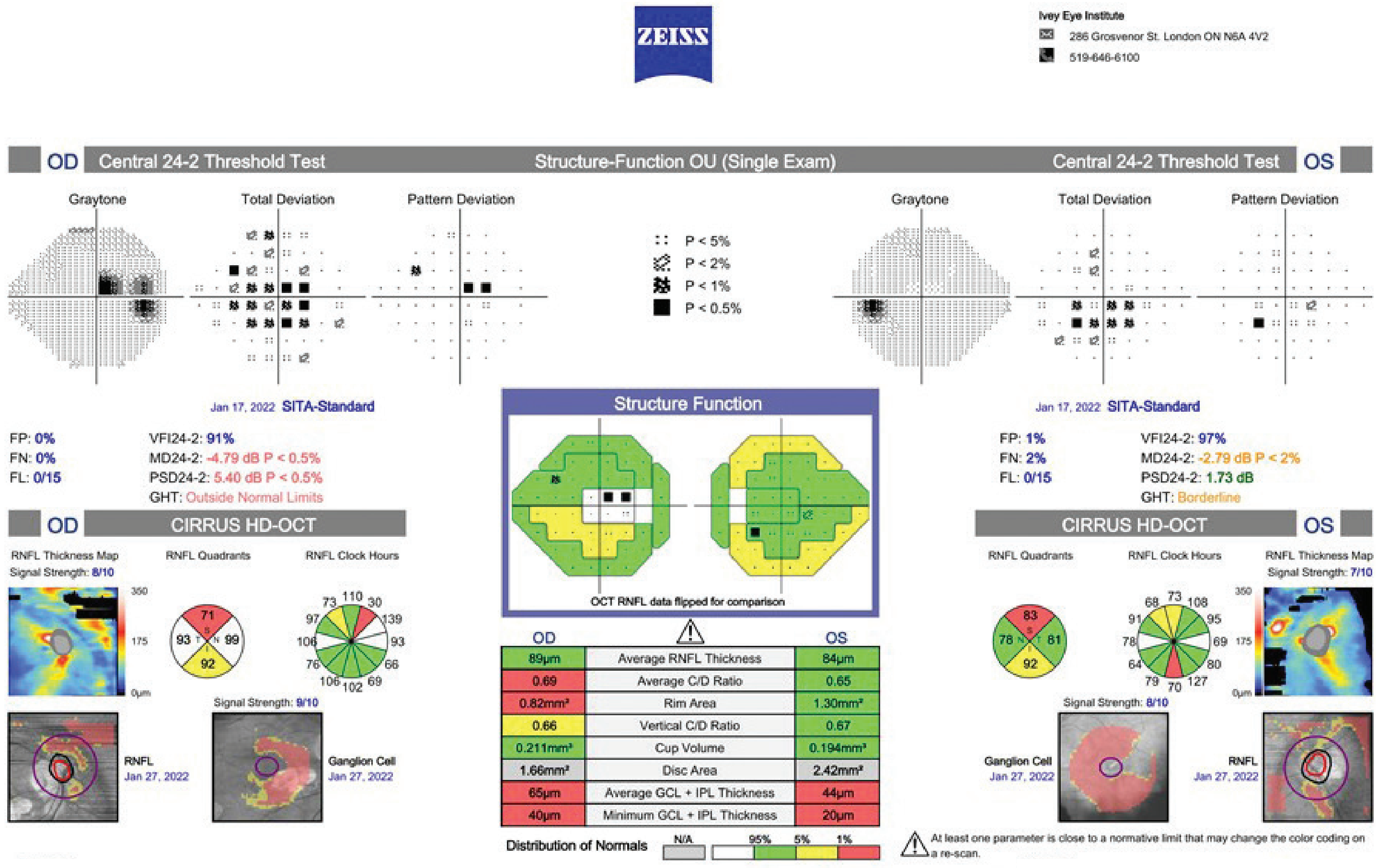

Figure 3: Structure-Function Maps (Zeiss Cirrus® HD-OCT), containing 24-2 visual field data and querying glaucoma OU; courtesy of Cindy Hutnik et al.

high myopia.[24,25] One study found that integrating a myopic normative database for RNFL thickness analyses can lead to a substantial improvement in reduction of false positive errors in myopic eyes compared to the emmetropic normative database contained in the Zeiss Cirrus® HD-OCT.[25] Myopic normative databases have proven challenging to develop for recruitment and proprietary reasons. Further, the considerable diversity of ONH configurations and refractive errors seen in myopia make it challenging to identify a 'standard normal' as a reference group.

Artificial Intelligence:

The application of artificial intelligence (AI) approaches to identifying glaucoma in the setting of myopia remains an exciting prospect. Current research efforts in this area have largely focused on disease classification and prediction for both glaucoma and myopia. Machine learning models (a branch of AI) have been shown to identify both glaucoma and myopia from OCT printouts and may predict glaucomatous progression.[26] AI approaches have made significant inroads in detecting glaucomatous disc damage from fundus photographs and OCTs. Other algorithms may successfully predict progression of glaucoma in eyes with myopia.[27,28] Since machine learning models in glaucoma rely heavily on ONH morphology in making decisions, many of the developments in AI in this field are still limited by excluding highly myopic eyes from training datasets. AI remains an exciting frontier and may become an additional clinical tool in distinguishing between glaucoma and myopia. The incorporation of AI platforms into routinely used electronic medical records may be very helpful in deciphering the complexities posed by managing glaucoma in the setting of myopia.

Conclusion

The growing public health burden associated with high or pathologic myopia is of concern as these patients are at higher risk for other related conditions including POAG. Optic nerve analysis in high myope patients with glaucomatous optic nerve changes may be difficult to detect. In myopes, RNFL changes may be predictive of glaucoma but OCT measurements of the RNFL are hampered by the lack of a myopic reference group which may lead to false positives. Visual fields can also be difficult to interpret with many variables contributing to visual field defects and/or mimicking glaucomatous field loss. The emergence of new imaging techniques and the prospect of AI hold much promise in helping clinicians differentiate between glaucoma and myopia.

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Cataract Surgery in the Myope: What You Should Know

Joshua Teichman, MD, MPH, FRCSC

Background

Improvements in technology have led to an increased safety profile in cataract surgery. Accordingly, in recent decades there has been an increasing clinical focus on providing improved refractive outcomes.

Initially using manual keratometers and ultrasound biometry, advances in intraocular lens (IOL) formulae lead to increased precision and accuracy of IOL calculations. This manifested in the form of decreased requirement for spectacle correction, usually at a distance target.

Later, optical biometry supplanted ultrasound as a more accurate method for the measurement of axial length and anterior chamber depth; it may also be useful in the measurement of lens thickness and white-to-white limbal distance. Newer biometers have built-in topographers with accurate keratometry.

It is beneficial to have an experienced ultrasonographer to perform testing in myopes, but it should be noted that posterior staphyloma can cause issues for the most experienced technicians. Some of the newer optical biometers capture a small OCT image at the time of testing to be used to test for foveal alignment, which is especially important in staphylomatous eyes.

More accurate testing, combined with newer-generation IOL formulae, has resulted in further improvement in the accuracy and precision of IOL calculation and increased spectacle independence for patients, commonly at distance. Moreover, newer intraocular lenses including multifocal and trifocal IOLs have increased the probability of spectacle independence at both distance and near. Furthermore, extended range of vision IOLs can provide distance and intermediate vision, with less dysphotopsia than current multifocal lenses.

Despite these advances, there are patient populations in which special attention is required to achieve improved refractive outcomes. Patients with increased axial length (myopes) have suffered from systematic errors in IOL calculation. Initially, modifications to previously developed IOL formulae were developed to compensate for this. The Wang-Koch correction (including its newer-generation correction) to various formulae would be an example of this.[1]

Currently, the newest generation of IOL formulae perform better, without correction, than the previous generation of formulae, with or without correction.[2]

Axial length is important

All formulae perform relatively well for the average length

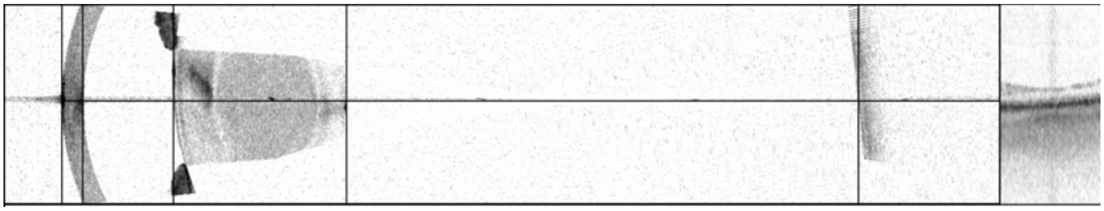

Figure 1. Image from optical biometer centered on the fovea; courtesy of Joshua Teichman, MD

eye, however as eyes become shorter or longer, IOL formulae become less reliable when plotting mean absolute error vs axial length. Most studies suggest that the Barrett Universal II Formula is the most accurate IOL formula for calculating the intraocular lens in patients with a long axial length[2],which aligns with the author's current recommendation in this patient population. Given the increased variability of IOL calculations, it is important to consider using multiple formulae and comparing the results between them; however, care should be taken not to use formulae that are known to perform poorly in longer axial lengths as this may skew the surgeon towards less accurate results. Clinicians should be aware that these formulae continue to evolve and that newer generations are constantly being released. For instance, the Hill-RBF Version 3.0 accepts eyes that would previously have been "out of bounds" in previous versions of the formula.

In many provinces in Canada the provincial health plan will cover ultrasound biometry but not optical biometry. Thus, for patients that choose not to pay for optional, non-medically necessary, although advanced testing, it is important that the surgeon be aware of modifications that may still improve results in these patients. The Wang-Koch axial length modification may be performed on ultrasound biometry derived measurements to improve the accuracy of IOL calculations. While the Barrett formula is meant for optical biometry, if the surgeon does not select an IOL and the ultrasound A-constant is manually inserted, the formula can be used, albeit not as accurately. As a final point, the Wang-Koch modification is for virgin eyes only and should not be used in those patients who have undergone prior refractive surgery.

Prior refractive surgery

This category of myopic patients—those that have undergone prior refractive surgery--- must be considered carefully. In the setting of prior surgery for myopia, there are multiple intraocular lens formulae and calculations that may be used. With or without the availability of historical data, measurements can be entered into an online calculator such as the one available at the ASCRS website and multiple formulae may be calculated simultaneously. Personal experience suggests that the Barrett True K is the most accurate formula and is also available for toric IOLs, as well as with total keratometry (TK), which uses posterior corneal measurements as opposed to predicted values. Clinicians should not use TK values in non-TK formulae, nor vice versa, as this will induce error in IOL calculations.

While these techniques are vast improvements on the previous methods of calculating IOLs in patients with previous refractive surgery, they are less accurate than IOL calculations on virgin eyes and the proportion of patients within half a diopter of plano is expected to be lower in patients with previous refractive surgery than in patients with virgin eyes .

IOL target

Another important consideration in myopic patients undergoing cataract surgery is the IOL target. While most patients undergoing cataract surgery opt for distance vision correction and expect to wear glasses for reading if they chose a monofocal lens, this is not a scenario that the average myope anticipates as they are accustomed to wearing glasses for distance tasks and removing them to read. It is important that these patients are counseled about the fact that they may lose their ability to read without glasses if they are aiming for a plano outcome. Clinicians should also be aware that multifocal IOLs require a close-to-plano outcome to minimize issues and maximize their efficacy and both myopes and those with previous refractive surgery are less likely to achieve this outcome without careful planning.

An additional option for these patients is monovision; however, a contact lens trial is recommended prior to considering the surgical approach.

The presence of a unilateral cataract in a myope is not uncommon. The discussion of the IOL target in the eye with the cataractous lens is more challenging as compared with a patient having a -6D or -9D refraction in an eye with a clear lens. These patients have the option of aiming the eye for plano and it would be practical to aim each eye for the ideal life-long target (distance or near), as opposed to compromising in the interim and leaving the patient with a sub-optimal long-term refractive outcome. However, even if targeted at near, a patient with a refraction of -9D in the contralateral eye is very unlikely to be able to function and extremely likely to experience symptoms of anisometropia. When the difference is not as extreme, the patient may be able to tolerate a near target. If symptoms of anisometropia occur, attempting a contact lens trial is the next step. If the patient is unable to tolerate a contact lens, then an earlier lens extraction can be considered. While the scope of this article is not meant to cover surgical complications in myopes, it is important to note that these surgeries carry higher risks than in those patients with normal axial lengths (e.g. retinal detachment), so patients are accepting additional risk, especially in an eye that can be corrected to a standard of acceptable vision. In a younger patient, one may consider an implantable collamer lens (ICL) with its own set of inherent risks, however these may be lower than those associated with cataract surgery. Moreover, these lenses can be removed if a cataract develops, and cataract surgery can then be performed at that time. This may be preferable to laser vision correction in high ametropia; however the individual procedural risk-benefit considerations must be carefully weighed, and the patient's preferences included as part of an approach centered on shared decision-making.

Conclusion

With optical biometry fixated at the fovea, the continued evolution of newer IOL formulae and newer IOLs, information regarding patients preferred visual outcomes, and informed discussion, clinicians can be confident that even very highly myopic patients may obtain excellent visual outcomes.

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Glaucoma and myopia: risk factors, pathophysiology, and treatment

Jing Wang, MD

Myopia is one of five most important risk factors for the development of primary open angle glaucoma (POAG) along with older age, elevated intraocular pressure (IOP), sub-Saharan African ethnicity and positive family history of glaucoma.[1] There are a few facets to consider when we discuss myopia and glaucoma. First, axial myopia (high myopia) increases the susceptibility of the optic nerve head (ONH) to IOP-related damage, therefore POAG occurs more frequently in a physiological normal IOP range in patients with high myopia. Second, there is evidence that POAG progresses faster in high myopes when IOP is elevated due to this increased susceptibility. Third, many myopes have undergone laser corrective surgery which can lead to an underestimation in the measurement of IOP and may delay the diagnosis of POAG in this group of patients. Fourth, high axial myopia is associated with atypical optic disc appearance and myopic macular degeneration. Both can cause visual impairment and make clinical assessment of glaucoma difficult. Moderate myopia is also associated with pigmentary glaucoma which is a common sub-type of open angle glaucoma. Finally, myopic patients are at risk of retinal detachment. The surgical treatment of retinal detachment can lead to a secondary form of glaucoma and worsen pre-existing POAG.

Clinicians should be reminded that patients with childhood glaucoma develop secondary myopia as a result of high IOP on very elastic developing eyes, particularly if the IOP was uncontrolled at a young age. The focus of this article is on the relationship between primary myopia and glaucoma.

Myopia is a risk factor for glaucoma - pathophysiology

Population-based landmark studies have consistently identified myopia as an independent risk factor impacting both the prevalence and incidence of POAG across different ethnic groups (Asians, Caucasians and Hispanics).[2-4] Myopia is associated with a 2-3 fold increased risk of developing glaucomatous optic neuropathy (GON).[2] High myopia, defined as -6 or -8 diopters or an axial length of ≥ 26.5 mm, is more strongly associated with POAG than low-to-moderate myopia.[4] However, pigmentary glaucoma, a special type of open-angle glaucoma, is associated with moderate myopia instead of high myopia.[5]

The pathophysiology underlying high myopia as a risk factor for POAG is thought to be related mainly to the biomechanics of the ONH.[6] The ONH is the structure in the posterior ocular fundus that allows the exit of the retinal ganglion cell axons and the entry and exit of the retinal blood vessels through a specialized connective tissue called the lamina cribrosa (LC). The retinal ganglion cell axons at the level of the ONH form the rim of optic nerve as seen on fundus exam and the part of the LC without retinal ganglion cell axons forms the cup of the optic nerve. The LC is a porous structure continuous with the oval sclera opening which is referred to as the scleral canal; the portion of sclera surrounding the LC is referred to as the scleral flange. The biomechanical properties of the LC and that of adjacent sclera are different. The LC serves as pressure barrier between the intravitreal compartment with the pulsating IOP and the retrobulbar compartment with the cerebral space fluid pressure. Axial elongation of myopic eyes is associated with both enlargement and rotation of the ONH. This disc enlargement is achieved by the stretching and thinning of the LC as well as the enlargement of the scleral canal opening and the thinning of the scleral flange. The thinning of the LC reduces its mechanical support to the surrounding axons, making the latter more susceptible to the stress of IOP. The thinning of the LC potentially steepens the pressure gradient between the IOP and cerebrospinal fluid (CSF) pressure, presumably increasing glaucoma susceptibility of highly myopic eyes. The effect of myopia on the development of POAG is the increased susceptibility of the ONH to IOP-related damage. GON is more likely to occur in high myopia even within a normal IOP range. However, this susceptibility of the ONH is more accentuated with higher IOP. Several smaller clinical studies have suggested that myopia and IOP have synergistic effects on the risk of POAG development and progression.[7]

Myopia and intraocular pressure (IOP)

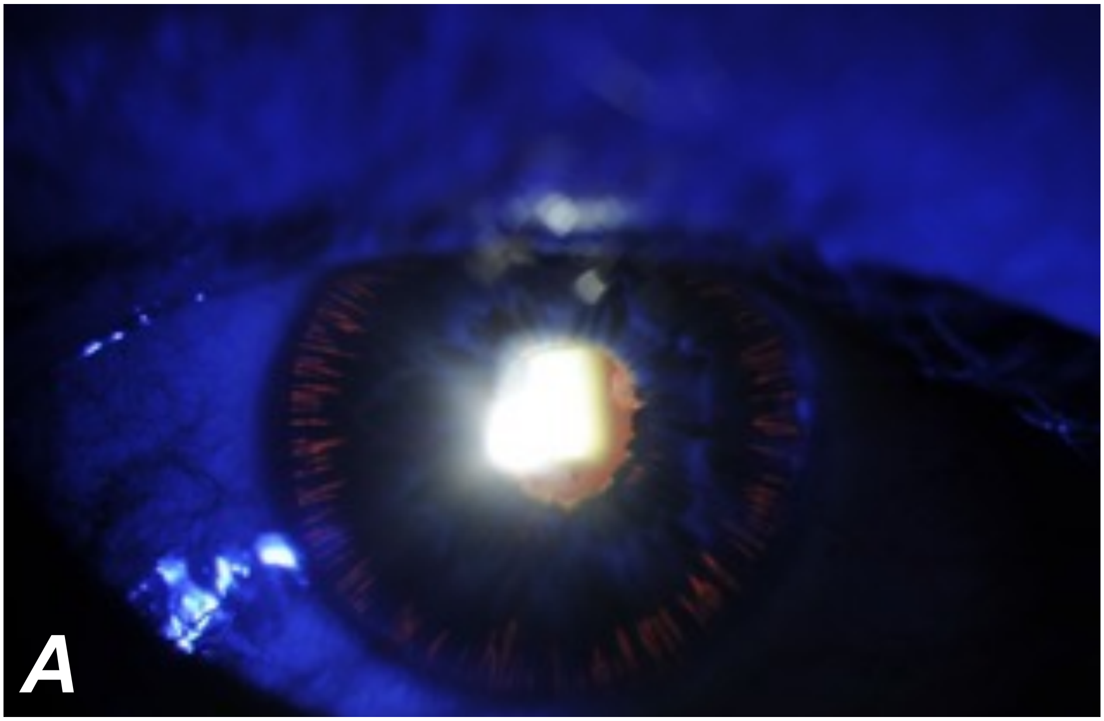
IOP is not necessarily more elevated in myopic patients compared to emmetropic or hypermetropic patients.[4] Some studies have suggested that the development of POAG in moderate myopia is associated with elevated IOP, but in patients with high myopia and POAG, IOP has been shown to be normal, suggesting an increased susceptibility of the ONH to a normal range of IOP in high myopia.[8] There are two particular scenarios that clinicians should keep in mind. First, moderate myopia is associated with pigmentary glaucoma which is a high-IOP open angle glaucoma. Second, with the popularity and ease of access to laser refractive surgery, many myopic patients have previously undergone Laser in situ keratomileusis (LASIK) to correct their myopia by thinning their corneas. Patients who have undergone LASIK are at a higher risk of developing glaucoma compared to the general population due to their myopia. By thinning the cornea, LASIK surgery renders the measurement of IOP inaccurate and mostly underestimates the true IOP. Numerous studies have tried to select the most accurate tonometer in measuring IOP for patients who have undergone laser refractive surgery.[9] While some tonometers are more accurate than others, the simplest way of monitoring IOP in post-LASIK patients is to use the widely available gold standard Goldmann Applanation Tonometer while keeping in mind that the true IOP may be underestimated. Precise and reproducible measurements of IOP are perhaps more important for longitudinal follow-up of patients and for monitoring their response to IOP-lowering therapy. It can be an exercise in futility to establish baseline IOP in post-LASIK patients with POAG as the target IOP depends ultimately on the speed of deterioration and severity of glaucomatous damage.

Myopia and pigmentary glaucoma

Pigmentary dispersion syndrome (PDS) is a well-recognized clinical entity that is associated with moderate myopia due to posterior bowing of the iris which causes the release of pigments from the posterior iris epithelium and the heavily pigmented trabecular meshwork (TM) due to the deposit of these pigments.[5] Clinical signs include posterior bowing of the iris (reverse pupillary block), trans-illumination of the iris viewed with retroillumination technique, vertical deposits of phagocytosed pigments on the posterior cornea due to the convection of aqueous humour (Krukenberg's spindle), deposits of pigments on the posterior lens zonules and posterior capsules of the lens (Scheie stripe or Zentmayer ring when it is circumferential) **(Figure 1).** On gonioscopy exam, an even, heavily-pigmented, velvety and thickened TM line is pathognomonic of PDS when other clinical signs are absent. Pigmentary glaucoma (PG) is almost always a high-IOP glaucoma with the primary pathology at the TM. This pathology includes the TM being overloaded with phagocytosed pigments leading to TM cell necrosis, the elevation of IOP and development of GON. PDS and PG occur most commonly in young, moderately myopic males. The dispersion of pigments tends to decrease with age. After active pigmentary dispersion stops, the patient's IOP can remain elevated, or it may normalize depending on the degree of TM damage during the active pigmentary dispersion phase. However, clinicians should keep in mind that further deterioration of PG can still occur at normalized IOP if the damage to the optic nerve is severe enough. Regardless of the initial high IOP associated with PG, advanced stage PG typically requires eye pressure to be maintained in the single digit-to-low-teen range in order to slow down or prevent further deterioration.

Glaucoma in high myopia - a diagnostic dilemma

The diagnosis of GON in patients with high myopia can be challenging. Whereas the development of POAG in moderate myopia is often accompanied by elevated IOP, in high myopia, this may not be the case.[8] High axial myopia is associated with parapapillary changes, myopic



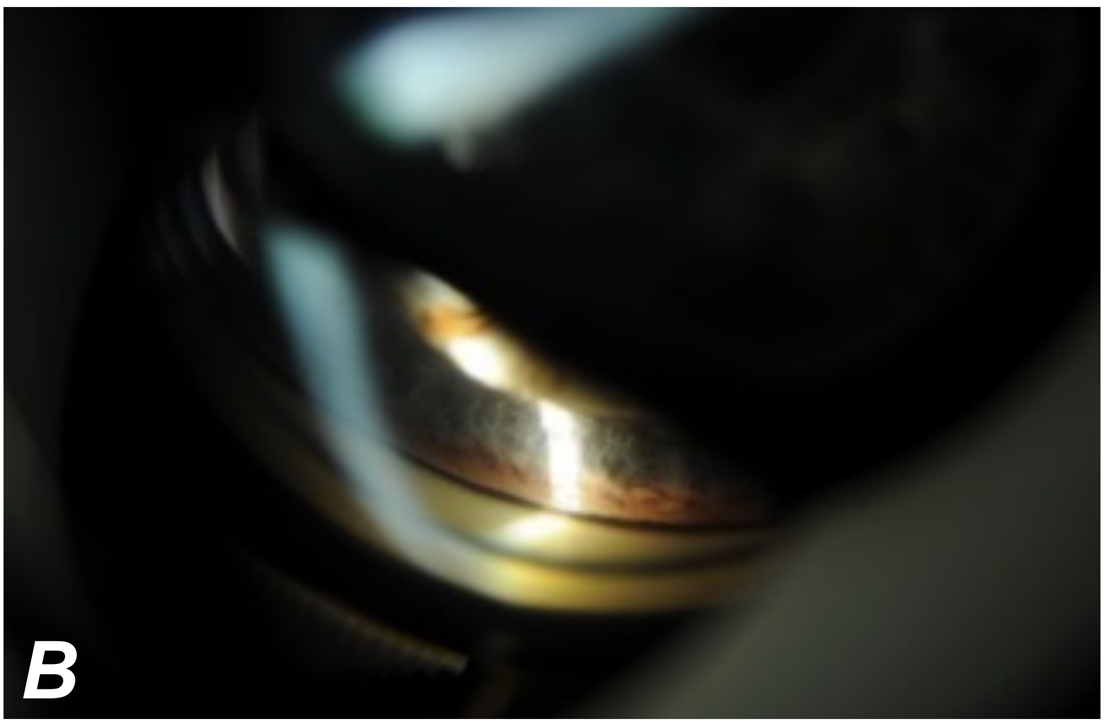


Figure 1. Pigmentary dispersion syndrome (A) Spoke like transillumination viewed with retroillumination technique at slit-lamp; B) Gonioscopic view of heavily pigmented trabecular meshwork and posterior bowing of iris; photo courtesy of Jing Wang, MD

macular degeneration and posterior staphyloma that can lead to visual field deficit similar to that of POAG. In addition, the assessment of the optic nerve in high myopic eyes is challenging for multiple reasons. First, the ONH is rotated due to axial elongation which gives rise to an oblique view on clinical exam. Secondly, both the colour and height contrast between the optic nerve rim and cup in myopic eyes are less obvious due to the enlargement of the ONH in high myopes. Thirdly, parapapillary changes such as those seen in the gamma and delta zones are frequent in high myopic eyes and they are difficult to distinguish from the glaucoma-related changes in the beta

	HISTOLOGICAL FINDINGS	CLINICAL ASSOCIATION
Alpha zone	Irregular pigmentation of retinal pigment epithelium (RPE)	Found in normal eyes
Beta zone	Absence of RPE over Bruch's membrane	Associated with glaucoma and speculated to be IOP dependent
Gamma zone	Absence of RPE and Bruch's membrane with bare sclera	Found in moderate-to-high myopic eyes
Delta zone	Absence of RPE and Bruch's membrane with elongated and thinned peripapillary scleral flange - i.e. the inner zone of Gamma zone	Found in high myopic eyes

Table 1. Four types of parapapillary zones and their clinical associations; photo courtesy of Jing Wang, MD

zone on clinical exam alone **(Table 1).** These myopia-related parapapillary zones also make assessment of the retinal nerve fiber layer (RNFL) in the peripapillary region extremely difficult.[10]

The accuracy of diagnostic imaging tools such as optical computed tomography (OCT) is reduced in highly myopic eyes.[(11)] One of the commonly encountered diagnostic errors using OCT with high myopes is the abnormal RNFL measurement of the RNFL due to the temporal convergence of RNFL bundles. This require clinicians to give careful consideration to the interpretation of the RNFL thickness map.[(12)] The inferior and superior temporal rims and RNFL bundles are thickest in a healthy optic nerve (shown as double humps on the RNFL distribution map). The thinning of these two areas are early signs of GON. The superior and inferior temporal RNFL bundles are temporally displaced in the high myopic eyes compared to the normal emmetropic eyes. This temporal displacement results in false-positive results on the deviation map (i.e. the deviation map will show thinning despite the average RNFL thickness being within the normal limit**.** Besides RNFL thickness, the macular ganglion cell inner plexiform layer (GCIPL) thickness has emerged as another structural parameter for diagnosing and monitoring glaucoma. However, this parameter also has its limitations in high myopic eyes. Long axial length eyes have been reported to have thinner maculae and may lead to false positive findings of thinning on the GCIPL thickness map.[13] Furthermore, the presence of myopic macular degeneration and patchy atrophy can cause abnormalities in the macular region, independent of established glaucoma.

An accurate diagnosis of POAG in high myopia requires a multitude of clinical signs taking into account IOP, disc assessment, and auxiliary tests such as fundus colour photo, OCT and visual field. Clinicians should bear in mind that the IOP can be normal despite the presence of advanced glaucomatous damage or the true IOP can be underestimated if patients have undergone previous LASIK surgery. The disc can be difficult to evaluate due to the above-mentioned reasons. Auxiliary tests can have false positive results and visual field deficit may not always be evident due to glaucomatous change. In cases where the diagnosis is uncertain and the IOP is below the normal range, it is not unreasonable to closely monitor patients through ongoing testing and IOP measurement without any intervention.[14] The hallmark of POAG is that it is a progressive disease. Further deterioration of the visual field with longitudinal follow-up can clarify the diagnosis and help in tailoring the appropriate treatment plan.

Glaucoma management in myopic patients

POAG is often over-diagnosed and over-treated in the setting of high myopia due to false-positive results on OCT, large disc and large cupping, the presence of parapapillary changes and visual field deficits that may not be due to glaucomatous damage.On the other hand, we also encounter many post-LASIK patients who are under-diagnosed and under-treated due to inaccurately low IOP readings. Establishing an accurate diagnosis and initiating appropriate treatment is critical for patients' well-being and quality of life. Clinicians should avoid initiating treatment solely based on OCT abnormalities, especially when the visual field is normal. In the presence of visual field deficit, clinicians should ensure that these deficits are not due to macular atrophy associated with myopic macular degeneration. If the visual field deficit is severe and progressive, clinicians should have a low threshold to initiate or augment treatment.

It is worth emphasizing that while angles are likely to be open in high myopes, gonioscopy is absolutely mandatory in the clinical evaluation of glaucoma. Lenticular myopia can lead to low, moderate or even high myopia. Causes for lenticular myopia include an evolving cataract, the forward movement of cilio-lenticular complex, and retinopathy of prematurity which can occasionally lead to lenticular myopia and anterior position of the lens and consequent angle closure. In these cases, the angle can be narrow or even closed on gonioscopy. As a result, the treatment approach would be different in the case of angle closure as the goal would include reversing the angle closure in addition to IOP lowering.

Once the diagnosis of POAG has been established, treatment is focused on the lowering of IOP regardless of baseline IOP. The target IOP depends on the severity of the glaucoma and its rate of progression. Both medical treatment with topical glaucoma medication and selective laser trabeculoplasty (SLT) should be considered as first line treatment options.[15] The maximum baseline IOP in untreated high myope and post-LASIK patients can be low (usually in the mid-teens). It is common to see post-LASIK patients with very thin corneas who have developed GON at IOP levels in the low teens. In these situations, a single-digit IOP level is likely required to arrest disease progression. In case of PG, the treatment principle is similar although SLT should be considered with caution given the higher risk of IOP spike. The use of laser iridotomy to reverse the posterior bowing of the iris has not been shown to provide clinical benefit in the treatment of PDS.[15]

When the visual field continues to deteriorate despite aggressive and comprehensive treatment strategies, glaucoma surgery is required to further reduce IOP with the hope of preventing further deterioration. The target IOP fundamentally depends on the severity of glaucoma damage to the visual field regardless of the untreated baseline IOP. In the last decade, numerous angle-based techniques involving minimally-invasive glaucoma surgery (MIGS) have emerged and broadened the surgical options that can be offered to patients. Despite the higher safety profile of MIGS and angle-based surgery, these surgical techniques rarely result in patients reaching single-digit IOP levels that are required in the management of advanced glaucoma. Filtering surgery such as trabeculectomy involving the use of antimetabolites and glaucoma drainage devices is still the most effective IOP-lowering surgery that surgeons can offer to glaucoma patients with severe damage. One particularly frequent complication of filtering surgery in myopic patients is hypotony maculopathy, which arises due to a more elastic sclera in myopic eyes. Hypotony maculopathy can unfortunately develop when the IOP is at the desired target but the macular folds are visually handicapping for the patients. The incidence of hypotony maculopathy is lower with the use of glaucoma drainage devices and newer MIGS filtering surgery, however this lower incidence is partially because the final surgical IOP is not as low as that which can be achieved by trabeculectomy. Ultimately, the visual function of the patient is still more important than the numerical IOP value.

Summary

Myopia and glaucoma are both common clinical entities. The diagnosis of GON can be challenging in high myopic eyes as the patient's IOP may not necessarily be elevated. The atypical disc and myopic macular degeneration in high myopes decreases the accuracy of auxiliary imaging tests and both can cause visual field deficits that resemble POAG. Myopic patients who have undergone previous LASIK surgery have inaccurately low IOP readings with all tonometers, which may delay the diagnosis of glaucoma in this particular sub-group of patients. Clinicians should also pay particular attention to the higher prevalence of PG in myopes and rare cases of angle closure associated with myopia. The management of POAG with coexisting myopia follows the same principles of open-angle glaucoma management - to further lower IOP and to monitor deterioration. Hypotony maculopathy is a particular complication of filtering surgery that is more frequent in myopic eyes.

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