Myopia and glaucoma: diagnostic and therapeutic challenges

Robert T. Chang and Kuldev Singh

Purpose of review
There is strong epidemiologic evidence linking myopia with glaucomatous disease, but a myopic optic nerve can pose significant challenges with regard to making the correct diagnosis of glaucoma. This review provides an overview of these diagnostic and therapeutic challenges with a particular focus on how the growing prevalence of myopia among specific populations may impact such therapy.

Recent findings
For a given individual, the link between myopia and glaucoma remains murky in many circumstances, largely because of the fact that it is difficult to separate out myopia-related structural and functional abnormalities from 'true' glaucomatous changes. Using optical coherence tomography (OCT) imaging, myopia has been found to be associated with temporal displacement and thinning of the superior and inferior nerve fiber layer bundles. In particular, sequential generations of 'Asian' ethnicities have been noted to demonstrate increasing rates of high myopia at earlier ages, sometimes with associated visual field defects at normal intraocular pressures. As is the case with any progressive condition, it is often not possible to distinguish glaucomatous from nonglaucomatous disease based on a single examination, and thus follow-up with OCT or perimetry from an established baseline is useful.

Summary
Although myopia is a known risk factor for glaucoma, it may also result in structural and functional defects that cannot be distinguished from those caused by glaucoma based solely on cross-sectional information. Longitudinal observation may be necessary to distinguish among the multiple effects of myopia on the optic nerve and the natural history of glaucoma, which may vary substantially amongst those who are affected.

Keywords
glaucoma, myopia, spectral domain optical coherence tomography

INTRODUCTION

Diagnosing glaucoma in the setting of optic nerves characteristic of moderate or high myopia poses unique challenges. As glaucoma diagnosis relies upon determining progressive optic nerve damage and corresponding visual field deterioration, myopia can be a confounding factor. Tilted discs, peripapillary atrophy (PPA), and staphylomas may make it difficult to distinguish glaucomatous optic neuropathy from myopia-related optic nerve and retinal abnormalities. Currently, clinicians often use various optic nerve imaging modalities to measure progressive thinning of the retinal nerve fiber layer (RNFL) with corresponding visual field defects that can occur as a consequence of higher than optimal intraocular pressure for an individual patient. Thus, it is imperative to understand the effects of high myopia on spectral domain optical coherence tomography (SD-OCT) as well as perimetry, and to weigh the risk/benefit ratio of intraocular pressure (IOP)-lowering therapy based upon the likelihood of observed abnormalities being related to myopia versus glaucoma, or both conditions.

DIAGNOSING GLAUCOMA

Unfortunately, there is as yet no single discriminatory test to confirm a diagnosis of primary open angle glaucoma (POAG). The disease has a final common pathway of ganglion cell loss that is more rapid than would be expected with aging, with the potential for severe vision loss and, in end-stage

Department of Ophthalmology, Stanford University School of Medicine, Palo Alto, California, USA

Correspondence to Robert Chang, MD, Byers Eye Institute at Stanford, 2452 Watson Ct, Palo Alto, CA 94303, USA. Tel: +1 650 724 3734; e-mail: viroptic@gmail.com

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KEY POINTS

- Myopic optic nerve changes may mimic glaucomatous findings with temporal displacement and thinning of the superior and inferior nerve fiber layer bundles.
- Several 'Asian' ethnicities have been noted to demonstrate increasing rates of high myopia at earlier ages, sometimes with associated visual field defects in the setting of normal intraocular pressure.
- As is the case with any progressive condition, it is often not possible to distinguish glaucomatous from nonglaucomatous disease based on a single examination.
- Longitudinal follow-up in the setting of myopia with glaucomatous findings may be necessary to confirm the diagnosis.

cases, blindness. Currently, clinicians subjectively interpret the appearance of the optic nerve, measure and correct for factors that impact intraocular pressure, and decide if the structural and functional nerve damage assessed on optical coherence tomography (OCT) or stereo disc photos and perimetry appears to suggest glaucomatous disease either cross-sectionally or longitudinally. Treatment is guided by the patient's likelihood of experiencing vision loss in their projected lifetime with consideration of the efficacy of various treatment modalities balanced by the potential for side effects with such therapies. This complex treatment decision-making process necessitates longitudinal observation following appropriate baseline testing for each individual, as well as a solid understanding of the inter-patient variability of the natural history of open-angle glaucoma.

In an effort to standardize the approach to diagnosing glaucoma, various consortiums have created guidelines, such as the American Academy of Ophthalmology (AAO) Preferred Practice Pattern Guidelines http://one.aao.org/asset.axd?id=a860f57a-0e6a-4c4f-b0f7-1a42e05073ff [Accessed on 5 October 2012]), the World Glaucoma Association (WGA) consensus statements [1] and the National Institute for Health and Excellence (NICE) document in the United Kingdom (http://pathways.nice.org.uk/pathways/glaucoma/glaucoma-diagnosis [Accessed on 5 October 2012]). For every existing structural and functional test for glaucoma, the overlap between normals and eyes damaged by glaucoma is large, and traditionally, there has been reliance upon glaucoma expert assessment of stereo optic disc photographs as the gold standard. However, this standard has been eroding due to the subjective nature of photo interpretation and poor agreement, even among leading glaucoma experts [2]. Additionally, digitization of disc photos has reduced the usage of older slide stereo pairs while the dramatic increase in adoption of SD-OCT has led some clinicians to replace the utilization of fundus photography with OCT in their practices.

EPIDEMIOLOGY

Multiple large, cross-sectional, population-based studies have linked high myopia with POAG. A few such notable studies include the Blue Mountain Eye Study [3], the Beijing Eye Study [4], the Los Angeles Latino Eye Study (LALES) [5], and the Singapore Malay Eye Study [6]. The Blue Mountain Eye Study screened 3654 Australians in an urban setting and found that glaucoma was twice as common in phakic eyes with low myopia of approximately 1D [odds ratio (OR), 2.1; confidence interval (CI), 1.2–3.8] and with a stronger association being noted for moderate-to-high myopia (OR, 3.3; CI, 1.7–6.4). In an effort to account for under or over classification of glaucoma due to myopic discs, particularly in elderly phakic patients for whom the refractive shift was a result of cataract, the LALES and Singapore studies measured axial length and found that axial myopia was associated with glaucoma with ORs ranging from 2 to 3. The Beijing Eye Study also assessed myopia divided into four categories: high myopia (<−8D), marked myopia (−6 to −8 D), moderate myopia (−3 to −6 D), and low myopia (−0.5 to −3 D). The investigators found that myopia greater than −6 D was more likely to be associated with glaucoma than moderate or low myopia. A recent meta-analysis of myopia as a risk factor for POAG, which combined data from 11 population-based studies conducted between 1994 and 2010 involving 48 161 individuals, found a pooled OR of 1.92 (95% CI, 1.54–2.38) and concluded that progressively higher myopia increases the likelihood of glaucoma [7▪▪]. One important caveat is that prevalence studies are cross-sectional in design and thus may incorrectly classify individuals as glaucomatous when in fact the defects may be due to other causes [8]. Although high myopia is clearly associated with glaucomatous disease, there remains no effective intervention for myopia, at this time, to favorably impact this important risk factor.

STRUCTURAL CHALLENGES WITH THE MYOPIC DISC

The two most commonly used contemporary modalities to image the optic nerve are subjectively interpreted stereo disc photos and objectively measured, automated SD-OCT RNFL, ganglion cell

(GCC or GCA), and optic nerve head (ONH) parameters. An important advantage of disc photos is that the image technology does not change over time, and thus a captured picture serves as a direct comparison indefinitely. While the photo storage format may have changed with the transition from film to digital, the picture is the same and not postprocessed. Thus, clinicians have a reliable reference to ascertain enlarged cupping, new notching or new RNFL defects, bayoneting of the vessels, increasing PPA, and other features characteristic of glaucoma. In particular, RNFL defects identified on disc photographs, especially atypical ones involving the papillomacular bundle, can be found in highly myopic eyes and may indicate early glaucomatous damage compared with nonhighly myopic eyes [9]. One software released to aid clinicians in detecting change over time is called MatchedFlicker (EyeIC, Pennsylvania, USA) which has been shown to help determine optic nerve change by auto-aligning and alternating two disc images back and forth quickly to highlight areas of possible progression [10].

With SD-OCT, postprocessed images utilizing custom analysis algorithms result in color printouts with normative database cutoffs indicated by green, yellow, and red to denote normal, borderline, and outside normal limits, respectively. These cutoffs are different for the various competing SD-OCT machines, thus making direct comparisons impossible, which is in stark contrast to fundus photography. In hindsight, such a color scheme was an overly simplistic attempt to encompass the wide range of population variability that exists, even independent of imaging artifacts. It might have been preferable to label the normative database as a 'reference range' that incorporated built-in covariates that are now known to include age and disc size, and to a lesser extent, axial length. The arbitrary cutoffs currently set at 5% and 1%, then, would not automatically be associated with disease but merely act as statistical guidelines to indicate when an SD-OCT parameter was merely outside the range likely to be found in a healthy person without any optic nerve disease. Instead, the current assessments subject the clinician to 'red disease,' in which one is tempted to assume that a red result is abnormal and suspicious for glaucoma, or even 'green disease' in which a clinician may miss an early abnormality because he or she is only viewing a green color indicative of 'normal' in someone who is in fact abnormal [11▪].

When SD-OCT was introduced approximately 5 years ago, there was an expectation that additional data with more robust algorithms would resolve some of the problems encountered when scanning those with high myopia. Although image acquisition has improved with centration being less of a problem given present day automated RNFL circle alignment, there remain multiple artifacts with SD-OCT. A recent study of neuroretinal rim parameters with Cirrus High Definition (HD)-OCT in 255 highly myopic eyes demonstrated that at least 17.6% had a measurement error as a result of large areas of peripapillary atrophy, severely tilted discs, steep retinal slopes, or vitreous opacities [12]. As was the case with Stratus (Zeiss, Dublin, California), SD-OCT algorithms tend to measure thinner RNFL and macular thickness in myopia [13–17]. However, one group was able to demonstrate that this can be a result of ocular magnification and may be mathematically adjusted for based on axial length and camera magnification [14]. Another study of false-positive RNFL color results found that longer axial length and smaller disc area were significantly associated with an increased incidence of false-positives when other factors were controlled (ORs, 2.422 and 0.165, respectively) [18]. Some of this variation may be explained by the fact that high myopia also causes lateral shifts in the contour of the RNFL thickness profile, which can make those with normal RNFL thickness fall outside the 'normal' range as determined by data derived from a normal, nonmyopic population. This artifactual shifting of the RNFL bundle peaks was reported by Hwang *et al.* [19,20], who analyzed 255 eyes of myopes from 0 to −11D using Cirrus HD-OCT and found that horizontal or temporally tilted nerves played a significant role in RNFL thinning as well as inducing more temporally positioned superior and inferior peaks.

A recent article by Leung *et al.* [21▪▪] elucidates this RNFL peak variation in normal myopic eyes using the Cirrus HD-OCT. The normative database for Cirrus only includes 284 individuals of low or no myopia, and therefore the reference range does not account for increased axial length or high myopia. With increasing axial length, there is a decrease in the angle bounded by the superotemporal and inferotemporal RNFL bundles. This RNFL distribution angle is the primary factor in the RNFL deviation map being coded 'red' independent of refractive error, axial length, age, average RNFL thickness, and disc area. The authors hypothesize that the variation of RNFL distribution bundles may be related to the shape of the globe with asymmetrical anteroposterior elongation and/or posterior staphyloma that is characteristically seen in those with myopia. This draws the bundles closer to the macula as compared to the anatomy in those with relatively more spherical emmetropic eyes [21▪▪]. Therefore, it is best to use symmetry in the RNFL deviation map to determine if there is a specific localized loss rather

than an abnormally labeled area due to a small RNFL distribution angle.

DIAGNOSTIC PERFORMANCE STUDIES OF SPECTRAL DOMAIN OPTICAL COHERENCE TOMOGRAPHY IN MYOPIA

Oftentimes, the optic nerves of high myopes are difficult to interpret in terms of classic glaucomatous cupping, and therefore the first SD-OCT test, as previously mentioned, acts as an initial baseline for future comparison. SD-OCT diagnostic performance studies typically employ a cross-sectional case–control study design of normal eyes versus those of individuals with glaucoma confirmed by abnormal perimetric testing. Thus, the sensitivity and specificity results do not accurately reflect the positive attributes of OCT in a glaucoma suspect population. However, with preperimetric disease, there is no definitive biochemical or other marker with which to compare OCT and thus the only definitive way to diagnose POAG is longitudinal follow-up, which requires lengthy studies that are difficult and expensive.

In an effort to assess the ability to cross-sectionally diagnose glaucoma with imaging in myopes, Shoji *et al.* [22] examined 82 patients with −5D or below of myopia using RTVue SD-OCT in which, 31 patients without field defects and 51 matched patients with perimetric glaucoma were compared. Of all the parameters studied – RNFL, ONH, and GCC (ganglion cell complex) – the global loss volume from GCC had the highest area under the curve (AUC) of 0.954 in differentiating normal from glaucoma patients. The authors did point out that variability of scans in high myopes could affect the reliability of the results and 9% of eyes were excluded due to low signal strength scores. The same authors also published a similar study indicating that GCC was the ideal test for diagnosing glaucoma in high myopes [23]. Kim *et al.* [24] published a similar study with RTVue SD-OCT comparing 73 normal controls and 77 perimetric glaucoma patients with a sub-analysis group of 45 high myopes ≤-6D. Once again, the GCC macula parameter was found to be ideal in discriminating normals from those with glaucoma, showing the highest AUC of 0.889. A potential criticism of both studies was that, without longitudinal data, investigators may have erroneously classified myopic individuals as being glaucomatous when some may have had minimally progressive field defects due to high myopia rather than glaucoma [25]. It is noteworthy, that macular disease, which is more common in pathologic myopia, can distort the ganglion cell readings as well.

VISUAL FIELD ABNORMALITIES IN MYOPIA

A major source of difficulty in diagnosing glaucoma in myopes is that a large percentage of myopic discs are tilted with PPA, which can lead to glaucomatous-appearing visual field defects. Researchers have previously published that optic disc ovality, as an index of tilt, is significantly correlated with greater myopia, longer axial length, and a higher mean defect on visual fields [26]. In a study of 127 eyes with high myopia compared with 40 low myopic and 46 emmetropic controls, 11% had a peripapillary 'detachment' around the optic nerve, which led to glaucoma-like field defects compared with those without peripapillary 'detachment' [27]. PPA-β has been shown to be associated with glaucoma and SD-OCT of this region revealed the lack of a Bruch's membrane within PPA to be significantly associated with myopic refractive error [28]. In a Korean normal tension glaucoma (NTG) population, optic disc tilt (45.5%) and optic disc torsion (75.9%) were found to be significantly more prevalent in myopes with NTG relative to those with NTG without myopia (3.4%, $P < 0.001$; and 33.9%, $P < 0.001$; respectively) [29]. Torsion degree was the only factor related to visual field defect location, and the myopic NTG group happened to be significantly younger with an average age of 42 years compared with 60 years for the nonmyopic NTG group. A prior work by Doshi *et al.* [30] described a series of young to middle-aged men of Chinese origin who presented with a constellation of ocular findings suggestive of glaucoma that did not significantly progress for up to 7 years. At least 40% were high myopes, and 75% and 81% were found to have tilted discs and peripapillary atrophy, respectively, features commonly associated with myopia. In a small follow-up study of similar patients, increasing axial length was not found to correlate with degree of visual field loss in asymmetric disease but it is noteworthy that this study may not have been powered to detect such a difference if one existed [31].

To further characterize this group of minimally progressive patients less than 50 years old with myopia and suspected glaucomatous optic neuropathy, a case–control study was carried out comparing 21 normals, 32 young myopic glaucoma suspects, and 43 patients with known glaucoma [32▪▪]. Approximately 70% of the young myopic glaucoma suspects were receiving IOP-lowering therapy due primarily to the existence of significant visual field defects. Clinical features of young myopic glaucoma suspects alone were not enough to differentiate these individuals from older glaucoma patients with confirmed disease. The problem with using visual fields in the setting of high myopia

is that threshold sensitivity is reduced in moderate and high myopia, regardless of the method of trying to correct for the refractive error [33]. Additionally, tilted discs can increase the likelihood of false apparent visual field deterioration or improvement with small changes in myopic correction [34]. While a retrospective review of myopia and perimetric glaucoma progression revealed worsening mean deviation with higher myopia, this may have been impacted by multiple other factors [35]. A recent retrospective review of 492 eyes with high myopia less than −8D and no glaucoma revealed newly developed significant visual field defects in 13.2% of eyes over 5 years [36▪]. An oval optic disc was more common in the eyes that developed field defects, and over 10 years, 73.8% of eyes showed progression of defects on Goldmann perimetry. Although the defects did not follow a classic glaucomatous pattern, normal tension glaucoma could not be completely ruled out. When it comes to diagnosing glaucoma in the setting of myopia, visual field testing, as is the case with optic nerve imaging, remains less than ideal.

THE ROLE OF INTRAOCULAR PRESSURE

A common diagnostic dilemma in clinical practice is the presentation of a young, moderate to highly myopic patient with abnormal tilted nerves and glaucomatous-appearing cupping with associated field defects and borderline high or normal IOP. One is faced with the difficult decision of initiating treatment versus careful follow-up to determine if the disease is truly progressing. Even if there is progression, it may be difficult to determine if the progression is due primarily to myopia or glaucoma and a distinction between these entities is often impossible to resolve. Myopes with glaucoma may commonly have IOP in the normal range, and while one study using a linear mixed model showed that the extent of myopia was found to be a prognostic factor for visual field progression, another showed that myopia did not have such an effect [37,38]. There are data from a study suggesting that there is greater IOP fluctuation in high myopes with POAG on pharmacologic therapy than in nonmyopes with POAG [39]. It remains controversial, however, whether or not short-term or long-term IOP fluctuations are independent risk factors for glaucomatous progression. Typically, clinicians favor more aggressive treatment for glaucoma, all other things being equal, when: IOP is higher, there is greater optic nerve damage, and the central corneal thickness is less than average.

Setting the correct target IOP range can be difficult in myopes with glaucoma, particularly if IOP is not higher than average relative to nonglaucomatous individuals. One cautious approach that has some merit is to establish baseline structural and functional parameters and begin the process of quantifying rate of progression as best as possible with regular testing rather than immediately targeting a low IOP that might require glaucoma surgery. It is noteworthy that myopes are at particularly high risk of hypotony maculopathy following glaucoma filtration surgery [40,41]. As some myopes with field defects may not show progression that is characteristic of glaucomatous disease, initial conservative treatment with glaucoma medications rather than surgery for those in whom the pathologic processes are not well understood may be wise, particularly in circumstances wherein the visual field defects at the time of initial diagnosis do not appear to threaten central fixation. A review of the Collaborative Normal Tension Glaucoma Study (CNTGS) results reminds us that half of the patients in the untreated arm of this study did not show progression at 5 years [42].

CONCLUSION

Myopia is a risk factor for open-angle glaucoma that may complicate both the diagnosis and treatment of glaucomatous disease. Currently, there is a growing prevalence of myopia in many regions of the world, particularly in several countries on the Asian continent. Epidemiologic studies in China, for example, have revealed this to be primarily an urban rather than rural phenomenon [43]. This will only increase the number of difficult diagnostic cases and deserves attention. Further research is needed to determine better ways to confirm a diagnosis of glaucoma in myopes without long periods of follow-up and to assess appropriate algorithms for treating glaucomatous disease in those with myopia.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES AND RECOMMENDED READING

Papers of particular interest, published within the annual period of review, have been highlighted as:
- ▪ of special interest
- ▪▪ of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (p. 176).

1. Weinreib R and Greve E, editors. Glaucoma diagnosis. Consensus Statement 1. NY: Kugler; 2004.

2. Jampel HD, Friedman D, Quigley H, *et al.* Agreement among glaucoma specialists in assessing progressive disc changes from photographs in open-angle glaucoma patients. Am J Ophthalmol 2009; 147:39–44.
3. Mitchell P, Hourihan F, Sandbach J, *et al.* The relationship between glaucoma and myopia: the Blue Mountains Eye Study. Ophthalmology 1999; 106:2010–2015.
4. Xu L, Wang Y, Wang S, *et al.* High myopia and glaucoma susceptibility: the Beijing Eye Study. Ophthalmology 2007; 114:216–220.
5. Kuzin AA, Varma R, Reddy HS, *et al.* Los Angeles Latino Eye Study Group. Ocular biometry and open-angle glaucoma: the Los Angeles Latino Eye Study. Ophthalmology 2010; 117:1713–1719.
6. Perera SA, Wong TY, Tay WT, *et al.* Refractive error, axial dimensions, and primary open-angle glaucoma: the Singapore Malay Eye Study. Arch Ophthalmol 2010; 128:900–905.
7. ■■ Marcus MW, de Vries MM, Junoy Montolio FG, Jansonius NM. Myopia as a risk factor for open-angle glaucoma: a systematic review and meta-analysis. Ophthalmology 2011; 118:1989–1994.

The authors pooled data from 11 cross-sectional studies and found that low myopia less than −1D and high myopia less than −3D had a pooled OR of 1.65 (1.26–2.17) and 2.46 (1.93–3.15), respectively.

8. Chang RT. Myopia and glaucoma. Review. Int Ophthalmol Clin. 2011; 51:53–63.
9. Kimura Y, Hangai M, Morooka S, *et al.* Retinal nerve fiber layer defects in highly myopic eyes with early glaucoma. Invest Ophthalmol Vis Sci 2012; 53:6472–6478.
10. Syed ZA, Radcliffe NM, De Moraes CG, *et al.* Detection of progressive glaucomatous optic neuropathy using automated alternation flicker with stereophotography. Arch Ophthalmol 2011; 129:521–522.
11. ■ Chong GT, Lee RK. Glaucoma versus red disease: imaging and glaucoma diagnosis. Curr Opin Ophthalmol 2012; 23:79–88.

A review of why normative databases need to be approached with caution and limitations of using color interpretation of ocular imaging results.

12. Hwang YH, Kim YY, Jin S, *et al.* Errors in neuroretinal rim measurement by Cirrus high-definition optical coherence tomography in myopic eyes. Br J Ophthalmol 2012; 96:1386–1390.
13. Qiu KL, Zhang MZ, Leung CK, *et al.* Diagnostic classification of retinal nerve fiber layer measurement in myopic eyes: a comparison between time-domain and spectral-domain optical coherence tomography. Am J Ophthalmol 2011; 152:646–653.
14. Kang SH, Hong SW, Im SK, *et al.* Effect of myopia on the thickness of the retinal nerve fiber layer measured by Cirrus HD optical coherence tomography. Invest Ophthalmol Vis Sci 2010; 51:4075–4083.
15. Savini G, Barboni P, Parisi V, Carbonelli M. The influence of axial length on retinal nerve fibre layer thickness and optic-disc size measurements by spectral-domain OCT. Br J Ophthalmol 2012; 96:57–61.
16. Mohammad Salih PA. Evaluation of peripapillary retinal nerve fiber layer thickness in myopic eyes by spectral-domain optical coherence tomography. J Glaucoma 2012; 21:41–44.
17. Hwang YH, Kim YY. Macular thickness and volume of myopic eyes measured using spectral-domain optical coherence tomography. Clin Exp Optom 2012; 95:492–498.
18. Kim NR, Lim H, Kim JH, *et al.* Factors associated with false positives in retinal nerve fiber layer color codes from spectral-domain optical coherence tomography. Ophthalmology 2011; 118:1774–1781.
19. Hwang YH, Yoo C, Kim YY. Myopic optic disc tilt and the characteristics of peripapillary retinal nerve fiber layer thickness measured by spectral-domain optical coherence tomography. J Glaucoma 2012; 21:260–265.
20. Hwang YH, Yoo C, Kim YY. Characteristics of peripapillary retinal nerve fiber layer thickness in eyes with myopic optic disc tilt and rotation. J Glaucoma 2012; 21:394–400.
21. ■■ Leung CK, Yu M, Weinreb RN, *et al.* Retinal nerve fiber layer imaging with spectral-domain optical coherence tomography: interpreting the RNFL maps in healthy myopic eyes. Invest Ophthalmol Vis Sci 2012; 53:7194–7200.

The authors concluded that the RNFL angle distribution of the superotemporal and inferotemporal RNFL bundles converged temporally in normal healthy myopes as axial length increased, which could lead to falsely abnormal RNFL deviation maps.

22. Shoji T, Sato H, Ishida M, *et al.* Assessment of glaucomatous changes in subjects with high myopia using spectral domain optical coherence tomography. Invest Ophthalmol Vis Sci 2011; 52:1098–1102.
23. Shoji T, Nagaoka Y, Sato H, Chihara E. Impact of high myopia on the performance of SD-OCT parameters to detect glaucoma. Graefes Arch Clin Exp Ophthalmol 2012 Dec; 250(12):1843–1849.
24. Kim NR, Lee ES, Seong GJ, *et al.* Comparing the ganglion cell complex and retinal nerve fibre layer measurements by Fourier domain OCT to detect glaucoma in high myopia. Br J Ophthalmol 2011; 95:1115–1121.
25. Aref AA. The diagnosis of glaucoma in highly myopic individuals requires longitudinal data. Graefes Arch Clin Exp Ophthalmol 2012 [Epub ahead of print].
26. Tay E, Seah SK, Chan SP, *et al.* Optic disk ovality as an index of tilt and its relationship to myopia and perimetry. Am J Ophthalmol 2005; 139:247–252.
27. Shimada N, Ohno-Matsui K, Nishimuta A, *et al.* Peripapillary changes detected by optical coherence tomography in eyes with high myopia. Ophthalmology 2007; 114:2070–2076.
28. Hayashi K, Tomidokoro A, Lee KY, *et al.* Spectral-domain optical coherence tomography of β-zone peripapillary atrophy: influence of myopia and glaucoma. Invest Ophthalmol Vis Sci 2012; 53:1499–1505.
29. Park HY, Lee K, Park CK. Optic disc torsion direction predicts the location of glaucomatous damage in normal-tension glaucoma patients with myopia. Ophthalmology 2012; 119:1844–1851.
30. Doshi A, Kreidl KO, Lombardi L, *et al.* Nonprogressive glaucomatous cupping and visual field abnormalities in young Chinese males. Ophthalmology 2007; 114:472–479.
31. Chao DL, Shrivastava A, Kim DH, *et al.* Axial length does not correlate with degree of visual field loss in myopic Chinese individuals with glaucomatous appearing optic nerves. J Glaucoma 2010; 19:509–513.
32. ■■ Kumar RS, Baskaran M, Singh K, Aung T. Clinical characterization of young Chinese myopes with optic nerve and visual field changes resembling glaucoma. J Glaucoma 2012; 21:281–286.

The authors concluded that the clinical features of young myopic glaucoma suspects were not enough to differentiate from older glaucoma patients, and longitudinal data was still needed to see if the untreated myopes would have progressed over time.

33. Aung T, Foster PJ, Seah SK, *et al.* Automated static perimetry: the influence of myopia and its method of correction. Ophthalmology 2001; 108:290–295.
34. Vuori ML, Mäntyjärvi M. Tilted disc syndrome may mimic false visual field deterioration. Acta Ophthalmol 2008; 86:622–625.
35. Perdicchi A, Iester M, Scuderi G, *et al.* Visual field damage and progression in glaucomatous myopic eyes. Eur J Ophthalmol 2007; 17:534–537.
36. ■ Ohno-Matsui K, Shimada N, Yasuzumi K, *et al.* Long-term development of significant visual field defects in highly myopic eyes. Am J Ophthalmol 2011; 152:256–265.

High myopia is a risk factor for visual field defects and these defects can be progressive even without glaucoma.

37. Sakata R, Aihara M, Murata H, *et al.* Contributing factors for progression of visual field loss in normal-tension glaucoma patients with medical treatment. J Glaucoma 2012 [Epub ahead of print].
38. Sohn SW, Song JS, Kee C. Influence of the extent of myopia on the progression of normal-tension glaucoma. Am J Ophthalmol 2010; 149:831–838.
39. Yang Y, Li Z, Wang N, *et al.* Intraocular pressure fluctuation in patients with primary open-angle glaucoma combined with high myopia. J Glaucoma 2012 [Epub ahead of print].
40. Silva RA, Doshi A, Law SK, Singh K. Postfiltration hypotony maculopathy in young Chinese myopic women with glaucomatous appearing optic neuropathy. J Glaucoma 2010; 19:105–110.
41. Costa VP, Arcieri ES. Hypotony maculopathy. Acta Ophthalmol Scand 2007; 85:586–597.
42. Anderson DR, Normal Tension Glaucoma Study.. Collaborative normal tension glaucoma study. Curr Opin Ophthalmol 2003; 14:86–90.
43. Cheng JW, Cheng SW, Ma XY, *et al.* The prevalence of primary glaucoma in mainland china: a systematic review and meta-analysis. J Glaucoma 2011 [Epub ahead of print].