Glaucoma and Myopia

Zeynep Kayaarası Öztürker[1]

ABSTRACT

Diagnosing glaucoma accurately in patients with high myopia is difficult because of the clinical features that resemble glaucomatous optic neuropathy. Patients may establish visual field defects that imitate glaucoma, and conventional imaging technology is less informative in disease identification and monitoring due to the absence of myopic normative data. These facts highlight the critical importance of determining the underlying pathology associated with myopia and primary open-angle glaucoma in future studies. This review discusses the intricate relationship between myopia and glaucoma and the challenges of current testing protocols and treatment strategies.

Keywords: Primary open-angle glaucoma, myopia, visual field, optical coherence tomography.

INTRODUCTION

Myopia has become more prevalent globally as a result of the significant lifestyle changes and indoor work among younger population. It is growing at an exponential rate, with 50% of the world's population expected to be impacted by 2050.[1] Although the data shows that myopia is more prevalent in younger age groups, projections for near future indicate an increase in older populations, in whom primary open-angle glaucoma (POAG) is most common. With the exception of the Ocular Hypertension Treatment Study and the Early Manifest Glaucoma Trial,[2,3] which did not find any significant association between the presence of myopia and the risk of developing or worsening glaucoma, population-based studies have demonstrated a robust correlation between myopia and POAG, suggesting that those with low to moderate myopia are twice as likely to develop POAG, and the risk is tripled for those with severe myopia.[4,5] It is suggested that axial myopia increases the risk of glaucoma, and each 1.0 D increase in myopia increases the risk of glaucoma by approximately 20%, with a more prominent increase in higher myopia.[6]

Primary open-angle glaucoma is one of the most common causes of blindness worldwide, with an estimated 30 million new cases in 2040.[7] Due to comparable pathways of optic nerve degeneration, myopia provides a substantial problem in diagnosing, monitoring, and controlling glaucoma in such cases, and convincing evidence is required to implement a novel clinical practice.

Pathophysiological Mechanisms and Clinical Findings

As high myopia and glaucoma share similar functional and anatomical alterations, distinguishing myopic optic neuropathy from glaucomatous optic neuropathy (GON) can be difficult, leading to misdiagnosis. Glaucoma is diagnosed by optic disc appearance and visual field loss, such as nasal step, central and arcuate visual field defects, which can also be found in myopic degeneration. High myopia also commonly features enlarged optic disc, large cup, peripapillary atrophy, and thinner retinal nerve fiber layer (RNFL) that may appear as a wedge during clinical examination. These similar features can mimic GON and result in fault diagnosis of glaucoma and unnecessary treatment.

Although the exact causes of the predisposition of myopic subjects to the development or aggravation of glaucoma remain unclear, the thinner sclera and the scleral tension due to longer axial length have been suggested to contribute to an increased susceptibility to glaucomatous damage.[8,9] A previous study found that optic nerve damage was more severe in highly myopic eyes with large optic discs at a given

1- VKV American Hospital American Medical Center, İstanbul, Türkiye

Received: 26.05.2023
Accepted: 26.05.2023
J Glau-Cat 2023; 18: 55-61
DOİ: 10.37844/glau.cat.2023.18.8

Correspondence Address:
Zeynep Kayaarası Öztürker
VKV American Hospital American Medical Center
Bağdat Caddesi, No:130, Feneryolu 34724 Kadıköy, Istanbul, Türkiye
Phone: +90 532 393 6432
E-mail: zeynepkayaa@yahoo.com

intraocular pressure (IOP).[10] Histomorphometric studies revealed that the lamina cribrosa is elongated and thinned when the optic disc is enlarged due to axial elongation.[11] Swept-source optical coherence tomography (OCT) studies demonstrated a posterior deformation in Bruch's membrane in correlation with functional glaucomatous damage, and confocal scanning ophthalmoscopy also provided evidence of compression of the ganglion cell axons in the optic discs in correlation with increasing myopia.[12,13] Peripapillary structural changes may also contribute to glaucomatous damage susceptibility, since β-zone peripapillary atrophy is associated with an increased risk of glaucoma progression and is more severe in patients with myopia-related glaucoma.[14-16] Additional research suggest that peripapillary delta zone is associated with axial elongation in myopic eyes with a longer peripapillary scleral flange and larger optic discs,[17,18] and the opening of Bruch's membrane in the optic nerve head is shifted, resulting in the formation of a gamma zone in the temporal disc (Figure 1).[19] Studies suggest that myopic discs may also have large shallow cupping, be tilted, and lack a clearly defined rim, making quantification difficult. Research indicates that in tilted discs, the trajectory of retinal ganglion cell axons may be disrupted, which can impede axonal transport and potentially contribute to glaucoma.[20]

Studies suggets that the enlargement of the optic disc due to axial elongation is a significant factor in the increased susceptibility to myopic glaucoma. Additionally, the enlarged peripapillary delta zone that accompanies this elongation is associated with a higher prevalence of glaucoma.[21]

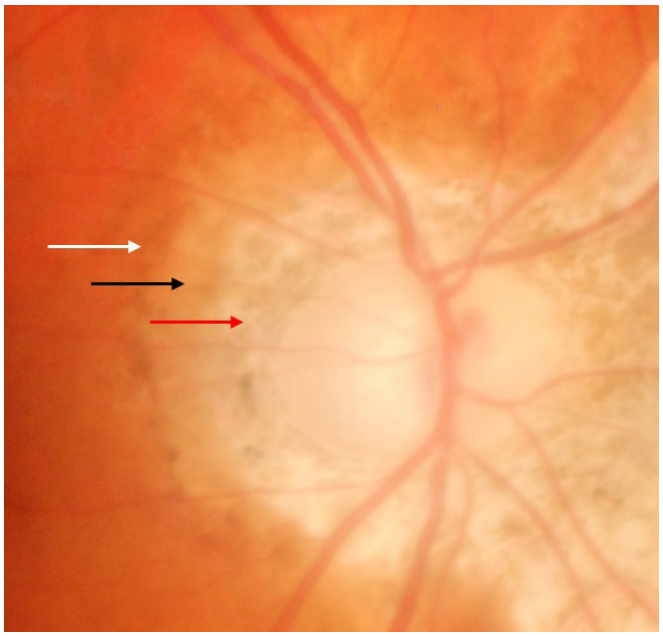

Figure 1: *The photograph shows the optic nerve of a glaucoma patient with a myopic fundus and large optic disc. White arrows indicate the peripapillary gamma zone in the temporal disc, black arrows indicate the delta zone, and red arrows indicate the β-zone peripapillary atrophy.*

Barriers to Diagnosis

Visual Field

As glaucoma and myopia can coexist, it is often difficult to diagnose glaucoma in high myopia, especially when the IOP is within the normal range. When visual field defects were classified in the High Myopic Registry Study, 16% of juvenile high myopic patients had visual field defects that resembled classical glaucomatous defects.[22] In a series of young myopic cases previously diagnosed with suspected glaucoma, no change was observed in the visual field and optic disc cupping when followed for seven years.[23] These findings emphasize that it is important to distinguish between myopic patients and glaucoma patients, as visual field defects can potentially lead to misdiagnosis. Therefore, longitudinal monitoring of high-risk myopic patients is crucial for management as progression over time is the most significant factor in detecting early glaucomatous changes.

Eyes with myopic tilted optic discs tend to have visual field defects more frequently than non-myopic eyes. The Blue Mountains Eye Study found that out of 62 eyes with a tilted disc, 19.4% had visual field defects commonly found in the superotemporal quadrant of the visual field.[24] Peripapillary intrachoroidal cavitation, known as peripapillary detachment in pathologic myopia, is also associated with visual field defects. It was found that eyes with peripapillary intrachoroidal cavitation were three times more likely to have glaucoma-like visual field defects than those without.[25]

A recent study developed a visual field classification system for myopic eyes without glaucoma and pathological changes, allowing for accurate and reliable categorization of visual field defects in myopic patients.[26] Glaucoma-like (paracentral defects, nasal step, paracentral arcuate and arcuate), myopia-like (enlarged blind spot, vertical step, partial peripheral rim, and non-specific), and combined defects were described, and glaucoma-like defects were associated with longer axial length in 10% of eyes.[26]

The results suggest that interpreting visual field defects in myopic eyes is still challenging in clinical practice. Myopic eyes may have visual field defects, which may be associated with abnormalities in the myopic retina or optic disc. In some cases, they may appear similar to glaucoma-like disc cupping but remain stable for several years.

Glaucoma can be confirmed by observing reproducible visual field defects and their progression, which correspond to structural changes in the optic disc, RNFL, or ganglion cell layer.

Intraocular pressure

The average IOP of myopic subjects appears to be higher than that of normal subjects. Although statistically significant, this difference, which varies between 0.5 mmHg and 1.25 mmHg depending on the series, is probably not clinically significant and does not on its own explain the clear difference in the prevalence of glaucoma between the healthy subjects and myopic subjects.[27] While some studies reported that up to 17% of high myopes have an average IOP greater than or equal to 21 mmHg,[28] other research indicate lower average IOP levels in highly myopic glaucomatous patients for the same level of glaucomatous loss.[10] The Blue Mountains Eye Study demonstrated in multivariate analysis that the risk of developing glaucoma for a myopic subject was not only higher than that of a non-myopic adult of the same age, but also that it was independent of other risk factors for glaucoma and IOP in particular.[29]

IOP is the only parameter that can be modified in glaucoma and glaucoma suspect patients. However, initiating treatment for myopic glaucoma suspects requires careful consideration due to their unique anatomical structure. The elongation of a myopic eye can result in greater vulnerability of the optic nerve to injury, as the posterior pole is subjected to more stretching than in a nonmyopic eye when IOP increases. Elevated IOP can lead to stress and strain in the lamina cribrosa and peripapillary sclera. This can result in various changes at the structural, cellular, or molecular level in the connective tissue. The stress and strain caused by IOP may have negative effects on blood flow in the laminar region, resulting in reduced delivery of oxygen and nutrients to the axons of the retinal ganglion cells.[30] The results of two population-based studies, Tajimi Study and Kumejima Study, indicate a significant positive correlation between higher myopia and higher IOP. These findings are consistent with the outcomes of the Beijing Eye Study.[31-33] Research also provides that there is a correlation between elevated IOP and the onset of glaucoma in myopic eyes with an axial length of less than 27.5 mm, and the presence of a large optic disc and advanced age further contribute to the development of glaucoma in individuals with a longer axial length.[34]

High myopic eyes may also be more sensitive to IOP fluctuations, and the baseline IOP and diurnal IOP fluctuation differ between myopic and non-myopic eyes. Myopic eyes have greater daytime IOPs and higher nighttime supine IOP elevations than those without myopia.[35] In myopic glaucoma, a greater 24-hour IOP range and nocturnal IOP rise was reported compared to controls, as well as greater IOP variability following exercise.[36] The reduction of ocular blood flow in myopia is also suggested to contribute to the development of glaucoma and increase susceptibility to the impact of IOP on the optic nerve head.[37]

The susceptibility of the myopic optic nerve to fluctuations in IOP can be attributed to various factors. These include the weakness of the fibroglial matrix that supports the nerve fibers at the optic disc level, structural alterations of the cribriform plate that becomes thinner, and the disappearance of the peripapillary choriocapillaris in highly myopic individuals can lead to disturbances in microcirculation at this level.[10,38]

Due to these distinctions, clinicians face difficulties in interpreting and managing the IOP of patients with myopic glaucoma or suspected glaucoma. Establishing a target IOP can pose a greater challenge, particularly when the patients exhibit normal IOP levels. Given the high risk of hypotony maculopathy following filtration surgery in myopes], targeting for a lower IOP in the absence of progression should be approached with caution.

Structural Imaging

Diagnostic technologies with improved sensitivity and specificity are still struggling to accurately assess the optic nerve in myopic eyes with glaucoma due to aberrant optic nerve morphology. The factors that contribute to the difficulty of detecting GON include a decrease in spatial and color contrast between the neuroretinal rim and the optic cup, peripapillary retinoschisis that can lead to incorrect segmentation of the retinal nerve fiber layer, a large gamma zone that makes it difficult to use the opening of Bruch's membrane as a reference point for measuring the neuroretinal rim, and a large gamma zone that reduces the value of the parapapillary beta zone as an indicator for GON.

The peripapillary RNFL bundles may be displaced towards the temporal region, leading to thicker temporal quadrants and relatively thinner nasal quadrants, and this can be misleading as the standard normative database of the OCT does not represent patients with moderate to high myopia.[39,40] The presence of anatomical variations and peripapillary alterations can pose a challenge in accurately segmenting the optic disc, which can result in a higher probability of errors or artifacts in the measurement of RNFL.[41] It is essential to carefully examine the circumpapillary image to assess the scan quality and RNFL

segmentation. Utilizing the inner circle scan report can assist in accurately interpreting RNFL scans, ultimately contributing to the reliability of the outcomes.[42]

Recent advancements in swept-source OCT (SS-OCT) and OCT angiography (OCTA) have led to an improvement in the detection of glaucoma-like optic neuropathy associated with myopia, suggesting that these technologies have the potential to enhance the diagnosis and treatment. SS-OCT has the capability to visualize the complete layer of the choroid and sclera, and a correlation between the severity of visual defects and the angle of scleral bending has been reported in high myopia.[43] The OCTA technology has proven to be effective in detecting early glaucomatous nerve damage by identifying focal defects in peripapillary retinal perfusion area, and choroidal microvascular drop-out around the optic nerve.[44] The use of SS-OCT and OCTA technology to analyze glaucoma characteristics in myopic eyes, along with the artificial intelligence to analyze a large database of fundus photographs would contribute to create algorithms to detect glaucoma in populations with high myopia rates.[45]

Ganglion cell complex (GCC) parameters have been shown to be superior to RNFL thickness in detecting myopic glaucoma, as GCC is not significantly associated with refractive error and better match with visual field defects than RNFL.[46] A recent longitudinal study found that macular ganglion-inner plexiform layer thinning, as opposed to RNFL thinning, has a significantly higher risk of developing visual field progression in highly myopic eyes (Figure 2).[47] Currently, there is a debate on which OCT parameters are best to use for glaucoma suspect patients with high myopia. Nevertheless, previous studies suggest that monitoring the GCC and visual field longitudinally could provide significant benefits for these patients (Figure 3). It is important to acknowledge that macular disease, which has been associated with high myopia, could potentially hinder ganglion cell analyses and subsequently

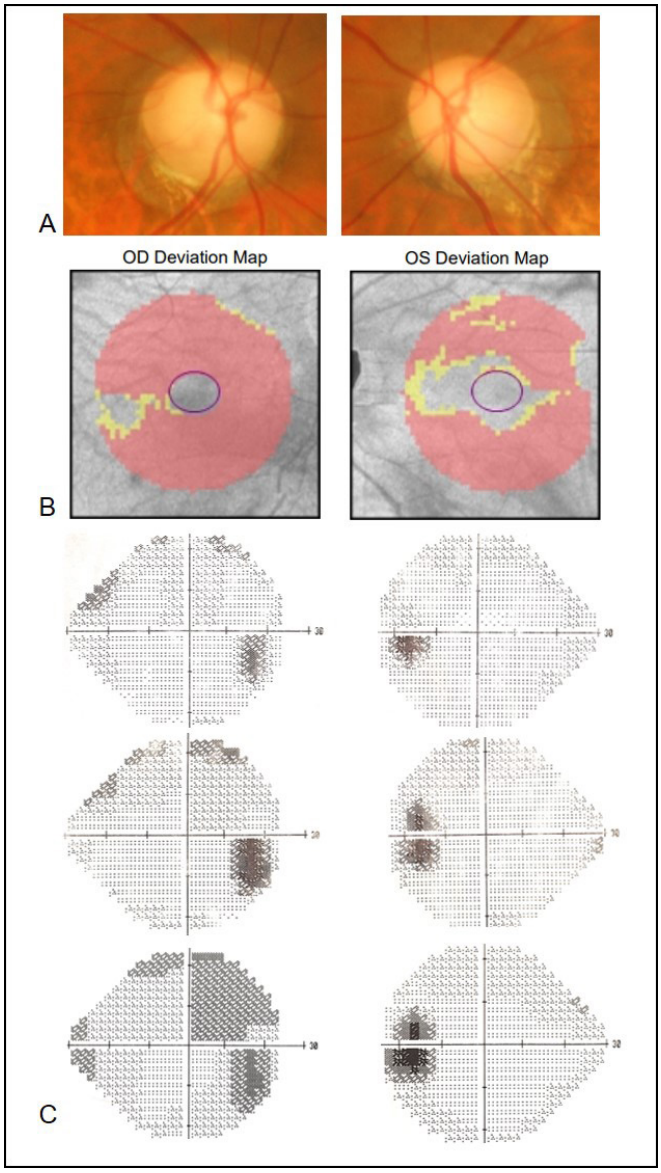

Figure 2: *The fundus photograph of a myopic glaucoma patient reveals a shallow cup and β-zone peripapillary atrophy (A). Ganglion cell analysis from Cirrus-OCT (Carl-Zeiss Meditec, Dublin, CA, USA) reveals bilateral, widespread ganglion cell loss (B). Despite normal intraocular pressure, there is an expansion of visual field defects on follow-up (C).*

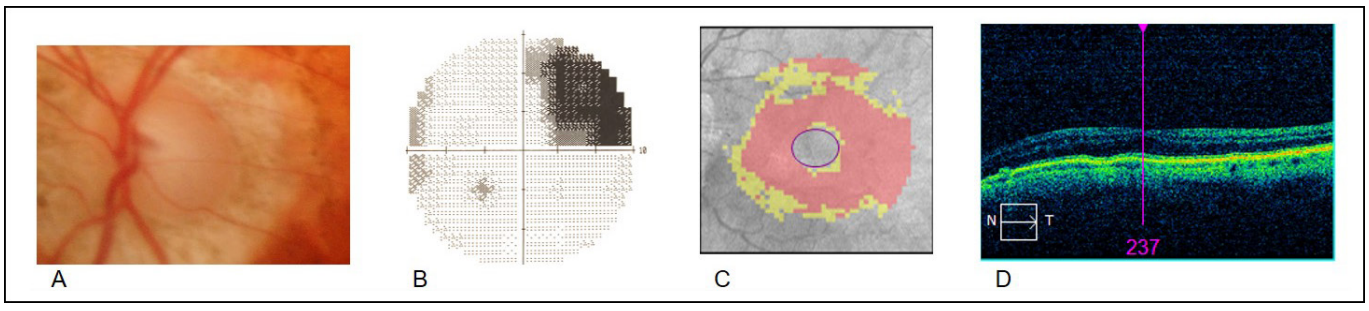

Figure 3: *Representative myopic glaucoma suspect with normal IOP levels. Optic disc photograph reveals diffuse peripapillary atrophy and large, shallow cupping (A). Humphrey visual field 10-2 shows paracentral scotoma (B). The macular GCC thickness map shows perifoveal ganglion cell loss (C), and there is no myopic macular degeneration (D). Monitoring the visual field and GCC thickness longitudinally can provide accurate diagnosis and treatment for this patient.*

reduce the reliability of the readings.[48]

The current diagnostic technologies for detecting myopic glaucoma need to be improved and new imaging modalities for both structural and functional analyses need to be developed. Additionally, a normative database for myopic patients needs to be established to enhance the objectivity of the diagnostic criteria.

Glaucoma Therapy in Myopia

The correlation between GON and IOP in individuals with high myopia remains an area of investigation, and no randomized trial has examined lowering IOP to treat glaucoma in highly myopic eyes. However, many researchers suggest that lowering IOP is advisable for highly myopic patients with glaucoma. The treatment approach for reducing IOP in myopic glaucoma is comparable to that of POAG in non-myopic eyes. This includes topical medications that decrease the production of aqueous humor or increase its outflow, laser procedures that target the trabecular meshwork to enhance aqueous humor outflow and surgical interventions that either increase outflow or decrease production.

Parasympathomimetics and cholinergic compounds in miotic eye drops rarely might cause retinal detachment, among other possible side effects. As patients with high myopia are at increased risk for retinal detachment, fundus examination with special attention to the peripheral retina, should be undertaken before miotics are prescribed. Myopia induction is another major problem, and patients should be warned that there is a risk of worsening their nearsightedness when using miotics.

Performing fistulating procedures like trabeculectomy may increase the risk of postoperative complications such as hypotony, choroidal detachment and expulsive hemorrhage. The choroidal thinning and the oblique course of the short posterior ciliary arteries and vortex veins through the sclera are potential reasons for suprachoroidal hemorrhage in highly myopic eyes.

A thin sclera can challenge in creating a sufficient partial-thickness scleral flap and suturing during trabeculectomy. In myopic eyes, the risk of hypotony can be reduced by implementing specific measures such as meticulous conjunctival closure to prevent wound leak, using a larger scleral flap, tightening scleral flap sutures to prevent over-filtration, and minimizing exposure to antifibrotic agents. To ensure successful surgery, it is also essential to tightly control blood pressure, reduce preoperative IOP to the maximum extent possible, gradually reduce IOP during the surgery, and maintain the anterior chamber throughout the procedure. If a glaucoma drainage tube is considered, there is a risk that the tube will be positioned further in the anterior chamber than intended, increasing the risk of corneal decompensation. Therefore, a longer intrascleral tube path or placement of the tube in the ciliary sulcus will help reduce the risk of corneal damage.

Minimally invasive and nonpenetrating glaucoma surgery may be safer as they gradually reduce IOP with less fluctuation during the procedure. However, the IOP outcome may be less effective than conventional filtering surgery.[49,50]

CONCLUSION

The presence of myopia can make it more difficult to diagnose and treat glaucoma. However, understanding the structural features of the myopic optic disc and retina and accurately interpreting ocular imaging and visual field test results can help overcome challenges. Myopic eyes may have glaucoma-like symptoms, but the condition may never progress; therefore, patients may benefit from conservative treatment and closer monitoring. If the intraocular pressure is normal, this may prevent unnecessary treatment until the glaucoma is diagnosed and continues progressing. The patients should be monitored and treated in accordance with the severity of the structural and functional damage, the location of visual field defects in relation to the fixation point, disc hemorrhage, central corneal thickness, pseudoexfoliation syndrome, pigment dispersion syndrome, and family history.

There is no evidence for an increased risk of glaucoma for low myopia (<-3D), and monitoring can be equivalent to that of a non-myopic person in the absence of a family history. The evidence shows that moderate myopia raises the risk of glaucoma, and the risk is greatest for high myopia (> 6 diopters), when the IOP is high in particular. High myopia increases optic nerve head susceptibility and complicates glaucoma. Therefore, regular monitoring is essential to ensure the best outcome.

The prevalence of myopia is on the rise, which calls for glaucoma diagnostic and treatment protocols that are explicitly tailored to myopia. This is essential to address the anticipated increase in the complexity of glaucoma cases. Myopic optic neuropathy, which is distinct from glaucoma, requires additional investigation to be conducted.

REFERENCES

1. Holden BA, Fricke TR, Wilson DA, et al. Global prevalence of myopia and high myopia and temporal trends from 2000 through 2050. Ophthalmology. 2016;123:1036-42.
2. Gordon MO, Beiser JA, Brandt JD, et al. The Ocular Hypertension Treatment Study. Baseline factors that predict the onset of primary open-angle glaucoma. Arch Ophthalmol 2002;120:714-20.
3. Leske MC, Heijl A, Hussein M, et al. Early Manifest Glaucoma Trial Factors for glaucoma progression and the effect of treatment: the Early Manifest Glaucoma Trial. Arch Ophthalmol 2003;121:48-56.
4. Mitchell P, Hourihan F, Sandbach J, et al. The Relationship between Glaucoma and Myopia: The Blue Mountains Eye Study. Ophthalmology 1999;106:2010-2015.
5. Grødum K, Heijl A, Bengtsson B. Refractive error and glaucoma. Acta Ophthalmol Scand. 2001;79:560-6.
6. Ha A, Kim CY, Shim SR, et al. Degree of Myopia and Glaucoma Risk: A Dose-Response Meta-Analysis. Am. J. Ophthalmol. 2022;236:107-19.
7. Tham YC, Li X, Wong TY, et al. Global prevalence of glaucoma and projections of glaucoma burden through 2040: a systematic review and meta-analysis. Ophthalmology. 2014;121:2081-90.
8. Saw SM, Gazzard G, Shih-Yen EC, et al. Myopia and Associated Pathological Complications. Ophthalmic Physiol. Opt. J. Br. Coll. Ophthalmic Opt. Optom. 2005;25:381-91.
9. Cahane M, Bartov E. Axial Length and Scleral Thickness Effect on Susceptibility to Glaucomatous Damage: A Theoretical Model Implementing Laplace's Law. Ophthalmic Res. 1992;24:280-4.
10. Jonas JB, Budde WM. Optic nerve damage in highly myopic eyes with chronic open-angle glaucoma. Eur J Ophthalmol. 2005;15:41-7.
11. Jonas JB, Berenshtein E, Holbach L. Lamina cribrosa thickness and spatial relationships between intraocular space and cerebrospinal fuid space in highly myopic eyes. Invest Ophthalmol Vis Sci. 2004;45:2660–5.
12. Lee S, Han SX, Young M, et al. Optic Nerve Head and Peripapillary Morphometrics in Myopic Glaucoma. Investig. Ophthalmol. Vis. Sci. 2014;55:4378-93.
13. Oh SH, Chung SK, Lee NY. Topographical Analysis of Non-Glaucomatous Myopic Optic Discs Using a Confocal Scanning Laser Ophthalmoscope (TopSS). Semin. Ophthalmol. 2015;30:397-409.
14. Jonas, J.B. Clinical Implications of Peripapillary Atrophy in Glaucoma. Curr. Opin. Ophthalmol. 2005;16:84-88.
15. Zhou D, Cao M, Duan X. Prevalence and Diagnostic Ability of β-Zone Parapapillary Atrophy in Open-Angle Glaucoma: A Systematic Review and Meta-Analysis. BMC Ophthalmol. 2022;22:72.
16. Teng CC, De Moraes CGV, Prata, et al. Beta-Zone Parapapillary Atrophy and the Velocity of Glaucoma Progression. Ophthalmology 2010;17:909-15.
17. Jonas JB, Jonas SB, Jonas RA, et al. Parapapillary Atrophy: Histological Gamma Zone and Delta Zone. PLoS ONE 2012;7:e47237.
18. Jonas JB, Ohno-Matsui K, Spaide RF, et al. Macular Bruch's Membrane Defects and Axial Length: Association with Gamma Zone and Delta Zone in Peripapillary Region. Investig. Ophthalmol. Vis. Sci. 2013;54:1295-302.
19. Jonas JB, Wang YX, Dong L, et al. Advances in Myopia Research Anatomical Findings in Highly Myopic Eyes. Eye Vis. 2020;7: 45.
20. 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;139:247-52.
21. Jonas JB, Weber P, Nagaoka N, et al. Glaucoma in high myopia and parapapillary delta zone. PLoS One. 2017;12:e0175120.
22. Ding X, Chang RT, Guo X, et al. Visual Field Defect Classification in the Zhongshan Ophthalmic Center-Brien Holden Vision Institute High Myopia Registry Study. Br. J. Ophthalmol. 2016;100:1697-702.
23. Doshi A, Kreidl KO, Lombardi L, et al. Nonprogressive Glaucomatous Cupping and Visual Field Abnormalities in Young Chinese Males. Ophthalmology 2007;114:472-9.
24. Vongphanit J, Mitchell P, Wang JJ. Population prevalence of tilted optic disks and the relationship of this sign to refractive error. Am J Ophthalmol. 2002;133:679-85.
25. Shimada N, Ohno-Matsui K, Nishimuta A, et al. Peripapillary changes detected by optical coherence tomography in eyes with high myopia. Ophthalmology. 2007;114:2070-6.
26. Lin F, Chen S, Song Y, et al. Classification of Visual Field Abnormalities in Highly Myopic Eyes without Pathologic Change. Ophthalmology 2022;129:803-12.
27. Wong TY, Klein BE, Klein R, et al. Refractive errors, intraocular pressure, and glaucoma in a white population. Ophthalmology 2003;110:211-7.
28. Loyo-Berrios NL, Blustein JN. Primary open glaucoma myopia: a narrative review. Wis Med J 2007;106:85-95.
29. Mitchell P, Hourihan F, Sandbach J, et al. The relationship between glaucoma and myopia: the Blue Mountains Eye Study. 1999;106:2010-15.
30. Crawford Downs J, Roberts MD, Sigal IA. Glaucomatous cupping of the lamina cribrosa: a review of the evidence for active progressive remodeling as a mechanism. Exp Eye Res. 2011;93:133-40.
31. Kawase K, Tomidokoro A, Araie M, et al. Tajimi Study Group; Japan Glaucoma Society. Ocular and systemic factors related to intraocular pressure in Japanese adults: the Tajimi study. Br J Ophthalmol. 2008 Sep;92:1175-9.
32. Tomoyose E, Higa A, Sakai H, et al. Intraocular pressure and related systemic and ocular biometric factors in a population-based study in Japan: the Kumejima study. Am J Ophthalmol. 2010;150:279-86.
33. Xu L, Li J, Zheng Y, Cui T, et al. Intraocular pressure in Northern China in an urban and rural population: the Beijing eye study. Am J Ophthalmol. 2005;140:913-5.

34. Jonas JB, Nagaoka N, Fang YX, et al. Intraocular Pressure and Glaucomatous Optic Neuropathy in High Myopia. Investig. Ophthalmol. Vis. Sci. 2017;58: 5897-906.

35. Liu JHK, Kripke DF, Twa MD, et al. Twenty-Four-Hour Pattern of Intraocular Pressure in Young Adults with Moderate to Severe Myopia.Investig. Ophthalmol. Vis. Sci. 2002;43:2351-5.

36. Jeong DW, Kook MS, Lee KS, et al. Circadian Pattern of Intraocular Pressure Fluctuations in Young Myopic Eyes with Open-Angle Glaucoma. Investig. Ophthalmol. Vis. Sci. 2014;55:2148-56.

37. Shimada N, Ohno-Matsui K, Harino S, et al. Reduction of retinal blood fow in high myopia. Graefes Arch Clin Exp Ophthalmol. 2004;242:284-8.

38. Fong DS, Epstein DL, Allingham RR. Glaucoma and myopia: are they related? Int Ophthalmol Clin 1990;30:215-8.

39. 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. Investig. Ophthalmol. Vis. Sci. 2010;51:4075-83.

40. Mwanza JC, Sayyad FE, Aref AA, et al. Rates of Abnormal Retinal Nerve Fiber Layer and Ganglion Cell Layer OCT Scans in Healthy Myopic Eyes: Cirrus versus RTVue. Ophthalmic Surg. Lasers Imaging Off. J. Int. Soc. Imaging Eye 2012;43:S67-S74.

41. Yılmaz H, Köylü MT, Yeşiltaş YS, et al. Alterations in the Retinal Nerve Fiber Layer Thickness Color Map in Non- Glaucomatous Eyes with Myopia. Turk J Ophthalmol 2021;51:26-31.

42. Zemborain ZZ, Jarukasetphon R, Tsamis E, et al. Optical Coherence Tomography Can Be Used to Assess Glaucomatous Optic Nerve Damage in Most Eyes with High Myopia. J. Glaucoma 2020;29:833845.

43. Meng LH, Yuan MZ, Zhao XY, et al. Wide-Field Swept Source Optical Coherence Tomography Evaluation of Posterior Segment Changes in Highly Myopic Eyes. Eur. J. Ophthalmol. 2022;32:2777-2788.

44. Na HM, Lee EJ, Lee SH, et al. Evaluation of Peripapillary Choroidal Microvasculature to Detect Glaucomatous Damage in Eyes With High Myopia. J Glaucoma. 2020;29:39-45.

45. Lim WS, Ho HY, Ho HC, et al. Use of Multimodal Dataset in AI for Detecting Glaucoma Based on Fundus Photographs Assessed with OCT: Focus Group Study on High Prevalence of Myopia. BMC Med. Imaging 2022;22:206.

46. Zhang Y, Wen W, Sun X. Comparison of Several Parameters in Two Optical Coherence Tomography Systems for Detecting Glaucomatous Defects in High Myopia. Investig. Ophthalmol. Vis. Sci. 2016;57:4910-5.

47. Shin JW, Song MK, Sung KR. Longitudinal Macular Ganglion Cell-Inner Plexiform Layer Measurements to Detect Glaucoma Progression in High Myopia. Am. J. Ophthalmol. 2021;223:9-20.

48. Wong YL, Sabanayagam C, Ding Y, et al. Prevalence, Risk Factors, and Impact of Myopic Macular Degeneration on Visual Impairment and Functioning Among Adults in Singapore. Investig. Ophthalmol. Vis. Sci. 2018;59:4603-13.

49. Ahmed IIK, Fea A, Au L, et al. COMPARE Investigators. A Prospective Randomized Trial Comparing Hydrus and iStent Microinvasive Glaucoma Surgery Implants for Standalone Treatment of Open-Angle Glaucoma: The COMPARE Study. Ophthalmology. 2020;127:52-61.

50. Hamel M, Shaarawy T, Mermoud A. Deep sclerectomy with collagen implant in patients with glaucoma and high myopia. J Cataract Refract Surg. 2001;27:1410-7.