

REVIEW ARTICLE



Pathologic myopia

Kyoko Ohno-Matsui ¹✉

© The Author(s), under exclusive licence to The Royal College of Ophthalmologists 2025

Pathologic myopia (PM) represents a predominant cause of irreversible visual loss worldwide, characterised by progressive axial elongation and profound structural alterations in the posterior segment, notably posterior staphyloma. Posterior staphyloma is a cardinal feature of PM and must be differentiated from physiological myopia, as it indicates localised scleral thinning and ectasia directly impacting the macula and optic nerve. Vision-threatening complications such as myopic traction maculopathy (MTM), frequently driven by vitreoretinal traction on retinal vasculature, and macular atrophy secondary to myopic macular neovascularisation (MNV) are prevalent in PM, often culminating in severe visual impairment. The sclera serves a critical biomechanical role in maintaining ocular structural integrity; hence, targeted interventions aimed at reinforcing the posterior sclera may decelerate disease progression. Although three-dimensional magnetic resonance imaging (3D MRI) has been extensively utilised to visualise the entire shape of the eye, recent advancements such as ultra-widefield optical coherence tomography (UWF-OCT) permit more detailed, practical assessment of posterior segment alterations. Furthermore, polarisation-sensitive OCT (PS-OCT) offers non-invasive in vivo evaluation of scleral collagen fibre orientation and birefringence, deepening our understanding of staphyloma pathogenesis. Early detection of structural changes within the sclera and vitreoretinal interface is paramount for timely intervention to prevent irreversible neuroretinal damage.

Eye (2025) 39:3218–3228; <https://doi.org/10.1038/s41433-025-04072-x>

INTRODUCTION

The prevalence of myopia and high myopia has escalated markedly over recent decades, with projections estimating that nearly half of the global population will be affected by myopia by 2050. Holden et al. [1], forecast that 49.8% of individuals worldwide will be myopic, with 9.8% exhibiting high myopia (defined as spherical equivalent ≤ -5.00 dioptres). This epidemiological trend poses significant public health challenges due to the increased risk of sight-threatening complications associated with high myopia.

Pathologic myopia (PM), characterised by excessive axial elongation accompanied by progressive structural changes of the posterior segment, is a major cause of irreversible visual impairment, particularly prevalent in East Asian populations. Population-based studies consistently identify PM as a major contributor to visual impairment and legal blindness in these regions [2–6].

DEFINITION OF PM

The terminology ‘pathologic myopia (PM)’ is frequently conflated with ‘high myopia’ or ‘severe myopia,’ yet these entities are distinct. High or severe myopia refers strictly to refractive error magnitude, defined by a high degree of myopic refractive power. In contrast, PM is delineated by characteristic structural abnormalities. The International Myopia Institute (IMI) defines PM as “excessive axial elongation associated with myopia that results in structural alterations in the posterior segment (including posterior staphyloma, myopic maculopathy, and high myopia-

associated optic neuropathy) that can cause loss of best-corrected visual acuity” [7]. Ohno-Matsui et al. further define PM as eyes presenting with myopic maculopathy equal to or more severe than diffuse choroidal atrophy or those exhibiting posterior staphyloma [8–10]. Notably, staphyloma can be observed in eyes without markedly elongated axial length [11, 12]. Therefore, PM may be more accurately characterised by the presence of posterior staphyloma and subsequent choroidal atrophy.

CAUSES OF SEVERE VISUAL IMPAIRMENT IN PM

Not all individuals with PM develop severe visual impairment. Profound vision loss typically arises in association with specific complications: myopic maculopathy (notably myopic macular neovascularisation [MNV] and its long-term sequela macular atrophy), myopic traction maculopathy (MTM), and glaucoma or optic nerve damage.

MYOPIC MACULOPATHY

The META-PM study group classifies myopic maculopathy into five categories (Fig. 1): Category 0 (C0), no maculopathy; C1, tessellated fundus; C2, diffuse atrophy; C3, patchy atrophy; and C4, macular atrophy [13]. Two additional lesions, lacquer cracks and myopic MNV, are designated ‘plus lesions’ independent of background maculopathy. C0 and C1 are not unique to PM.

Diffuse atrophy (C2) presents as a yellowish fundus lesion characterised by pronounced choroidal thinning on OCT (Fig. 2) [14–16], with preservation of the outer retina and retinal pigment

¹Department of Ophthalmology and Visual Science, Institute of Science Tokyo, Tokyo, Japan. ✉email: k.ohno.oph@tmd.ac.jp

Received: 30 June 2025 Revised: 11 September 2025 Accepted: 26 September 2025

Published online: 13 October 2025

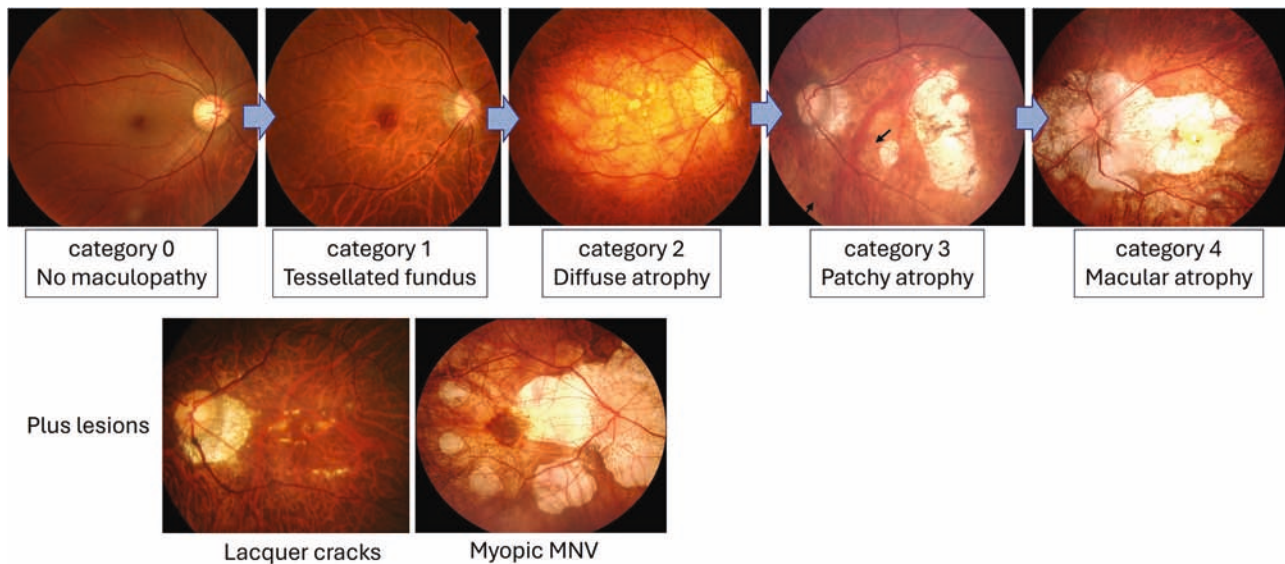


Fig. 1 According to the META-PM Study Group classification, myopic maculopathy is categorised into five grades: **Category 0, no maculopathy**; **Category 1, tessellated fundus**; **Category 2, diffuse choroidal atrophy**; **Category 3, patchy choroidal atrophy**; and **Category 4, macular atrophy**. Additional lesions such as lacquer cracks and myopic choroidal neovascularisation (myopic CNV), which may develop independently of the progression through these categories, are referred to as “plus” lesions. Categories 0 and 1 are not considered specific to pathologic myopia.

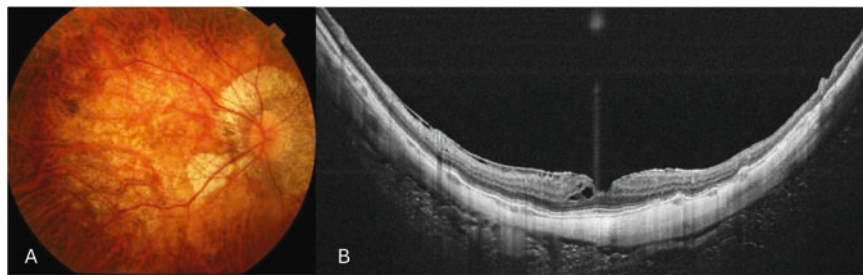


Fig. 2 **Marked choroidal thinning in Category 2 (diffuse atrophy).** The right fundus image (A) shows yellowish diffuse atrophy involving the macular region. In the corresponding OCT image (B), the choroid is almost entirely absent, with only a few large choroidal vessels remaining. Despite the severe choroidal thinning, the outer retinal layers remain relatively well preserved.

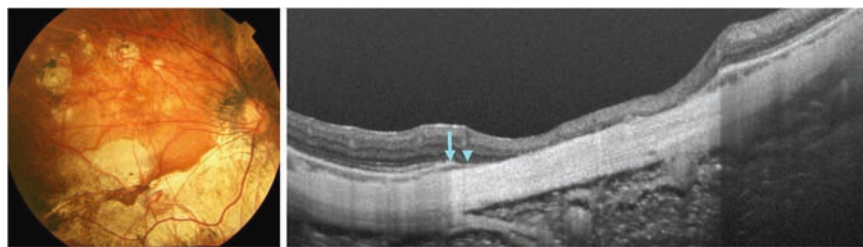


Fig. 3 **Category 3 (patchy atrophy).** Patchy atrophy appears as a well-demarcated, whitish lesion located outside the foveal area. It typically develops within regions of diffuse choroidal atrophy. The OCT image reveals a focal absence of the retinal pigment epithelium (RPE) and Bruch's membrane (BM) in the affected area. The arrow indicates the edge of the RPE, and the arrowhead marks the termination of the BM.

epithelium (RPE), often allowing maintenance of functional vision (≥ 0.5).

Patchy atrophy (C3) is a well-demarcated whitish lesion defined by defects in RPE and Bruch's membrane (BM) (Fig. 3) [17]. In C3 areas, the outer retina and choriocapillaris are absent, exposing the inner retina directly over sclera, with only major choroidal vessels remaining. This lesion corresponds to absolute scotoma but typically spares the fovea even with enlargement, preserving central vision (Fig. 4).

Macular atrophy (C4) is characterised by a well-demarcated, fovea-centred atrophic lesion (Fig. 5) with RPE and BM defects distinct from

geographic atrophy due to age-related macular degeneration [16, 18]. The foveal involvement results primarily from expansion of BM defects adjacent to myopic MNV; thus, this entity is more accurately termed 'MNV-related macular atrophy.' MNV-related macular atrophy causes central scotoma and progressive visual decline over time.

Wang et al. [19] developed machine learning models predicting best-corrected visual acuity (BCVA) at 3 and 5 years in high myopia patients, identifying baseline BCVA, prior myopic MNV, age, and category 4 macular atrophy as the most significant predictors. The prominence of MNV-related variables underscores the critical role of MNV in visual prognosis in PM.



Fig. 4 Progression of patchy atrophy over ten years, sparing the fovea. At baseline, multiple areas of patchy atrophy are observed surrounding the macula. After ten years, these lesions have enlarged and coalesced; however, the fovea remains unaffected. This suggests that patchy atrophy typically progresses without involving the central macula.

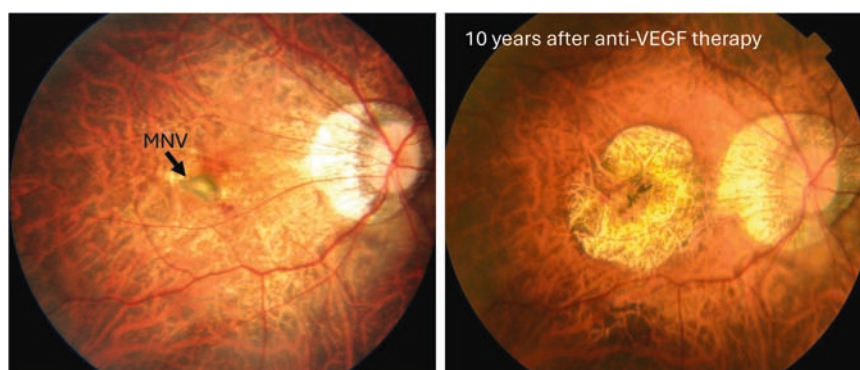


Fig. 5 Macular atrophy as a late complication of myopic macular neovascularisation (MNV). At baseline, a mildly pigmented MNV lesion is observed. Ten years after anti-VEGF therapy, the MNV has regressed with residual pigmentation; however, extensive macular atrophy has developed. Similar to patchy atrophy, MNV-related macular atrophy represents an enlargement of Bruch's membrane (BM) defects surrounding the scarred MNV lesion.

MYOPIC TRACTION MACULOPATHY (MTM)

Myopic traction maculopathy (MTM) encompasses a spectrum of macular pathologies in PM eyes caused by complex tractional forces at the posterior retina. These include incomplete posterior vitreous detachment, epiretinal membranes, internal limiting membrane (ILM) rigidity, and deformation associated with posterior staphyloma, leading to progressive structural changes. Typical manifestations include macular retinoschisis (MRS), foveal detachment, lamellar or full-thickness macular holes, and sometimes macular hole retinal detachment (Fig. 6). OCT is indispensable for detecting and monitoring MTM progression [20–22].

The Panozzo classification provides an OCT-based clinical staging system for MTM, stratifying severity by retinoschisis extent, presence of foveal detachment, macular holes, and retinal detachment [23].

MRS, particularly outer MRS, is a critical lesion often initiating extrafoveally near the optic nerve or along major retinal vessels, subsequently progressing toward the fovea [24].

Multiple causative factors include ILM stiffness, axial elongation, and posterior staphyloma as tensile forces, with vitreous traction playing a key role. At the inaugural Myopia Society meeting in Paris, global experts reached consensus favouring the term 'myopic traction maculopathy' over 'myopic tensile maculopathy' to reflect the dominant tractional mechanism, alongside simplified nomenclature based on lesion severity.

Vitreous alterations observable on OCT include multiple posterior vitreous detachments and vitreous traction at retinal vessels (Fig. 7). Takahashi et al. [25] reported a significant association between paravascular vitreous adhesions and MRS

(63% vs. 44%, $P = 0.04$), with paravascular lesions such as paravascular retinal cysts and paravascular retinoschisis more frequent in MRS eyes. Multivariate analysis confirmed paravascular adhesions as a significant risk factor for MRS.

While early MTM may be stable and asymptomatic, surgical intervention—primarily pars plana vitrectomy with modified ILM peeling—is indicated in visually significant cases, especially with foveal detachment or full-thickness macular holes [26–28]. Macular buckling is occasionally performed. Although the foveal sclera tends to protrude anteriorly by macular buckling, surrounding sclera bows more posteriorly (like pressing inflated balloon), having a risk of exacerbating mechanical damage on the optic nerve. Thus, a long-term influence on optic nerve damage should be examined.

GLAUCOMA OR ON DAMAGE

The prevalence and diagnosis of optic nerve (ON) damage in PM remain challenging due to marked optic disc morphological alterations such as extreme tilting and deformation, complicating morphological assessment [29]. Coexisting peripapillary atrophy and myopic maculopathy further hinder OCT measurements of retinal nerve fibre layer and ganglion cell complex, as well as interpretation of Humphrey visual fields. Distinguishing glaucomatous ON damage from myopic changes is also difficult [30]. Consequently, ON damage is often overlooked until advanced stages. Visual symptoms may be confounded by concurrent macular pathologies (Fig. 8), contributing to diagnostic delays. Although not restricted to PM, Nagaoka et al. [29] found glaucoma in 28% of 336 highly myopic eyes studied.

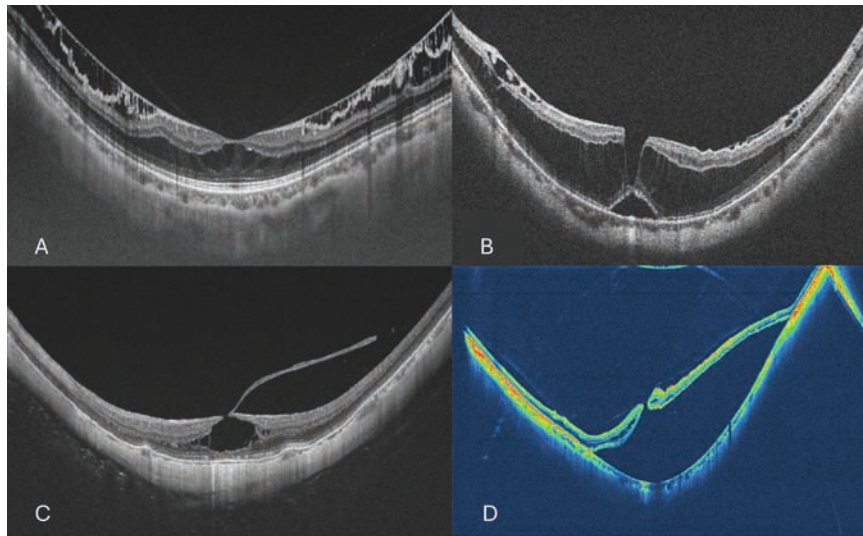


Fig. 6 Spectrum of lesions in myopic traction maculopathy. **A** Myopic macular retinoschisis (MRS), showing both inner and outer retinoschisis. **B** MRS with inner lamellar hole and foveal detachment. **C** Inner lamellar macular hole. **D** Full-thickness macular hole with associated retinal detachment.

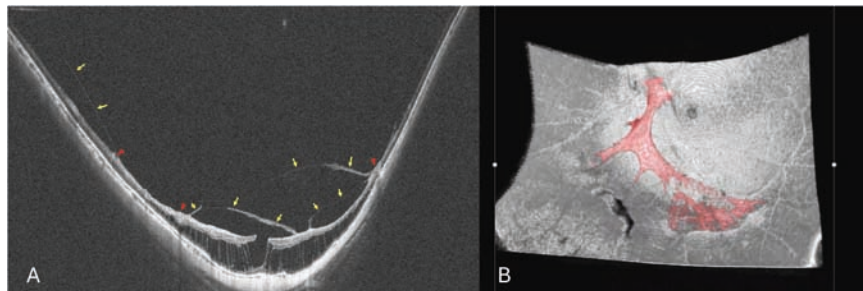


Fig. 7 Vitreous adhesion along retinal vessels. **A** Optical coherence tomography (OCT) image from a patient with macular retinoschisis shows posterior vitreous thickening (arrows), with firm vitreous cortex adhesion to the inner retinal surface at multiple points. Closer examination reveals that the borders of multiple partial posterior vitreous detachments (PVDs) correspond to the locations of retinal vessels (arrowheads). **B** A three-dimensional reconstruction highlights vitreous adhesions at several sites along the retinal vessels, revealing a starfish-shaped configuration of partial PVDs.

Papillary and peripapillary lesions inducing visual field deficits in PM can be categorised as PM-specific and non-specific. PM-specific lesions include conus pits [31], retinal thinning over scleral ridges temporal to the optic nerve [32, 33], and retinal defects along peripapillary intrachoroidal cavitation (ICC) (Fig. 9) [33–35]. Non-specific lesions include lamina cribrosa defects, which tend to be larger and deeper in PM eyes (Fig. 10) [31, 33]. However, Xie et al. [33] demonstrated that visual field loss correlates more strongly with overlying retinal thickness rather than lamina cribrosa defect size.

Given the diagnostic challenges, clinical practice benefits from Goldmann perimetry when optic disc morphologies, such as large or small disc, severely tilted disc, ICC, deformed optic disc, asymmetrical optic disc (Fig. 11). Because inner isopter is affected by large PPA and maculopathy lesions, the outermost isopter is a good marker for roughly diagnosing the existing ON damage.

POSTERIOR STAPHYLOMA; UNDERLYING CAUSES OF PM-RELATED COMPLICATIONS

Posterior staphyloma is a localised outpouching of the posterior segment of the eye and represents a hallmark deformity in eyes with PM. It serves as a reliable diagnostic indicator for PM. However, confusion exists in differentiating true staphylomas from generalised posterior scleral bowing observed in eyes with

axial elongation. In axially elongated eyes, a generalised bowing of the posterior sclera is confused with staphyloma. In contrast, staphyloma is an outpouching of a limited area of the posterior fundus. Thus, detecting its edges is important to diagnose staphylomas.

Spaide [36] defined a staphyloma as “an outpouching of the eye wall that has a radius of curvature less than that of the surrounding eye wall.” Clinically, however, peripapillary and nasal staphylomas often lack abrupt curvature transitions and instead manifest as nasal distortion of the posterior segment of the eye, leading to the inclusion of “nasally distorted” morphologies within staphyloma classification (Fig. 12) [37]. Additionally, staphylomas can also develop in eyes without long axial length (D in Fig. 12) [38].

Historically, detection of staphylomas depended on subjective methods such as indirect ophthalmoscopy, which limited recognition to pronounced staphylomas with pigmentary changes at their margins, often missing early or mild staphyloma edges.

Three-dimensional magnetic resonance imaging (3D MRI) has emerged as a powerful objective tool to evaluate morphology of the eye [37, 39, 40]. Unlike traditional imaging modalities, 3D MRI enables comprehensive visualisation of the entire globe, including posterior eye morphology, without geometric distortion (Fig. 13). It allows for the accurate assessment of

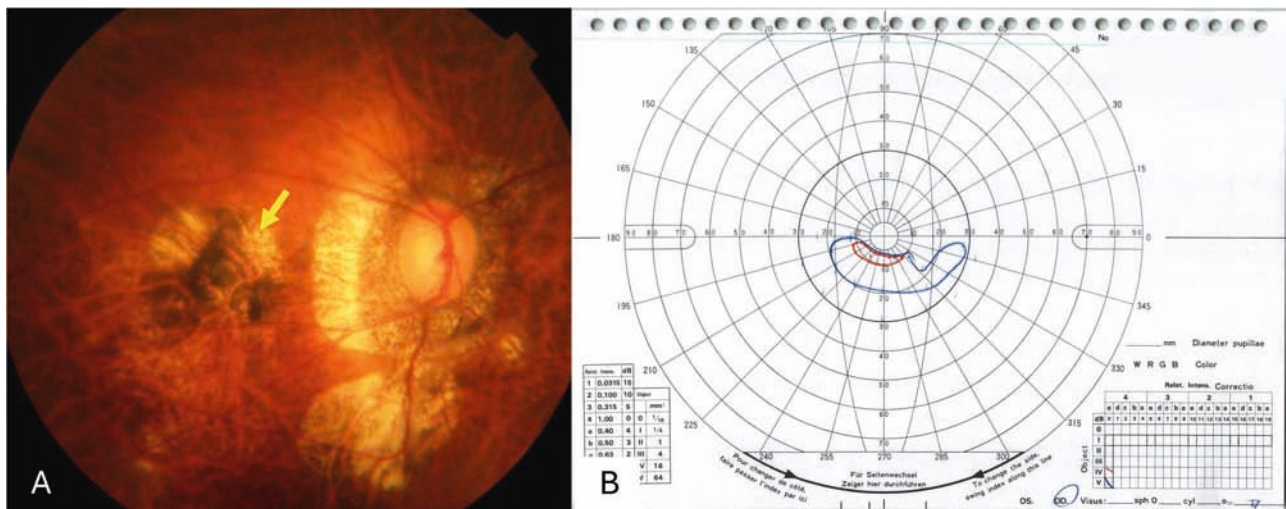


Fig. 8 Underdiagnosis of optic nerve damage until advanced stages. **A** Fundus photograph of the right eye of a 60-year-old woman with an intraocular pressure (IOP) of 11 mmHg and a visual acuity of 0.02. Despite repeated complaints of vision loss, she was consistently diagnosed with macular atrophy (arrow) as the sole cause of her impairment. **B** Goldmann perimetry reveals severe visual field loss, with only a small remaining area in the inferior field.

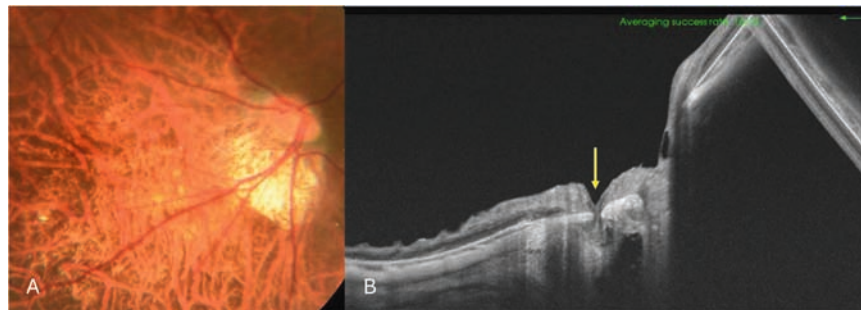


Fig. 9 Conus pit and herniation of the retinal nerve fibre layer into the pit. **A** Fundus photograph of the right eye showing an inferotemporally tilted optic disc. The conus pit is not clearly visible. **B** Optical coherence tomography (OCT) image revealing the pit as a discontinuity in the peripapillary sclera (arrow). The overlying neural retina is herniated into the pit.

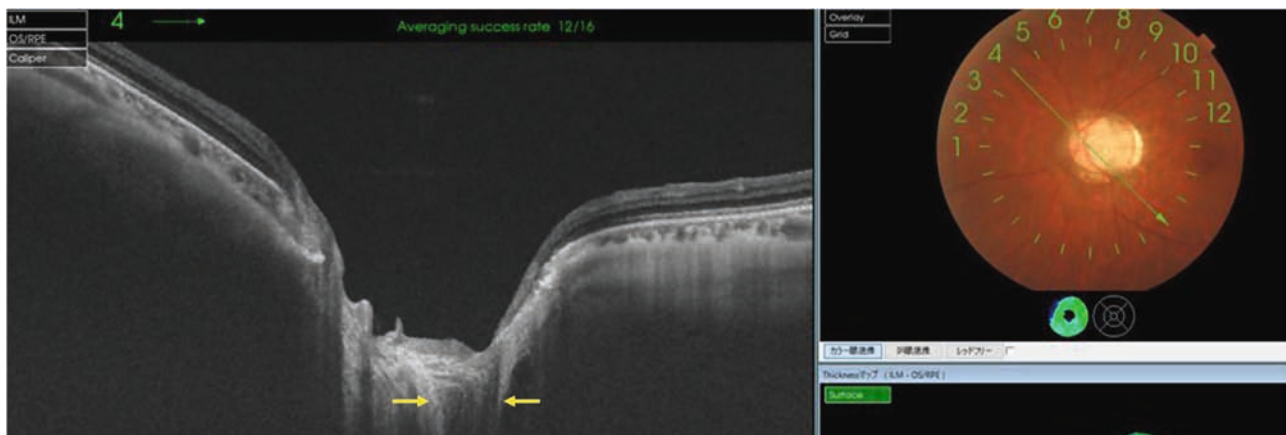


Fig. 10 Large lamina cribrosa (LC) defect in eyes with pathologic myopia. The OCT image demonstrates a prominent LC defect (between arrows) along the temporal border of the optic nerve, accompanied by laminar projections. The overlying retina appears markedly thinned.

asymmetry of the eye, posterior staphyloma, and the spatial relationships between ocular structures and the surrounding tissues. This non-invasive technique provides critical insights into the anatomical changes associated with PM, facilitating research on the relationship between eye shape and fundus complications due to PM. Takahashi et al. [41] used 3D MRI and

showed that the volume of eyes with and without staphyloma did not significantly differ, suggesting that local outward protrusion of the posterior eye wall is not directly caused by global expansion of eyes.

Ultra-widefield optical coherence tomography (UWF-OCT) offers significant advantages over 3D MRI for evaluating the

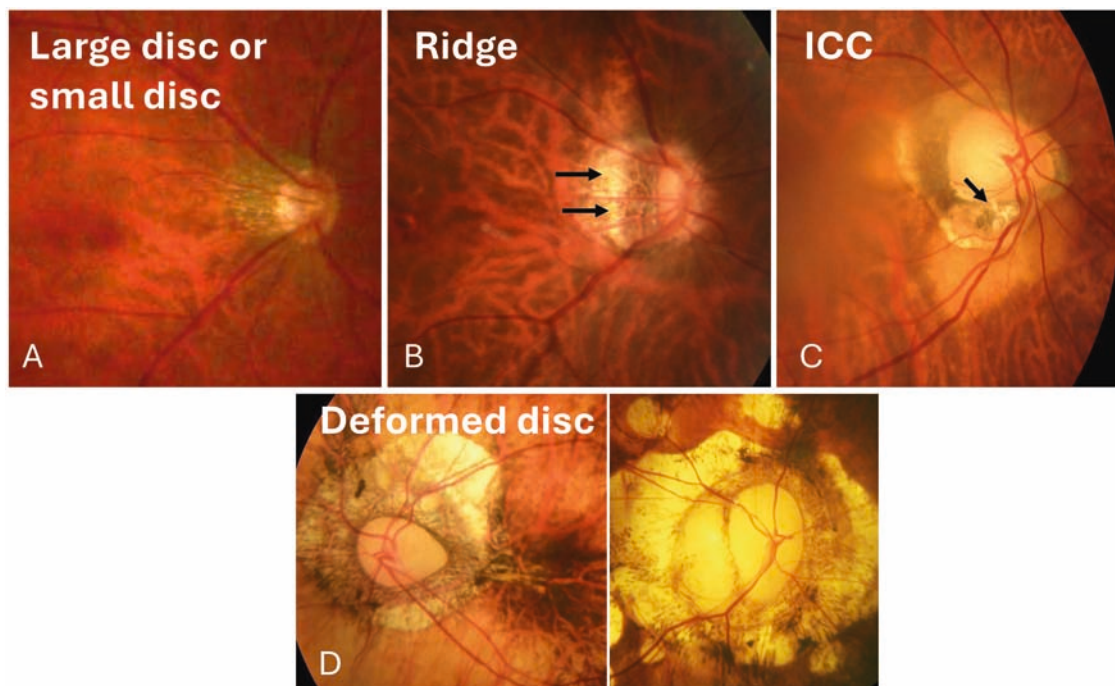


Fig. 11 Optic disc features associated with suspected visual field defects. **A** Large or small optic disc. **B** Scleral ridge temporal to the optic nerve (arrows). **C** Peripapillary intrachoroidal cavitation (ICC), with full-thickness retinal defects occurring along the ICC margin (arrow). **D** Deformed or asymmetrical optic disc. Lamina cribrosa defects frequently develop in asymmetrically stretched regions of the optic nerve head.

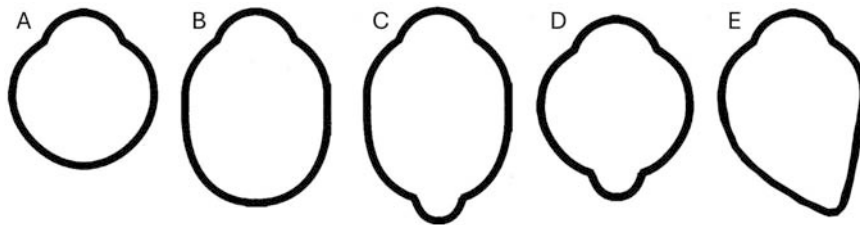


Fig. 12 Schematic illustration of various eye shapes. **A** Emmetropic eye. **B** Axially elongated eye with simple myopia without posterior staphyloma. **C** Axially elongated eye with posterior staphyloma. **D** Eye with posterior staphyloma without significant axial elongation. **E** Eye with nasal or peripapillary staphyloma.

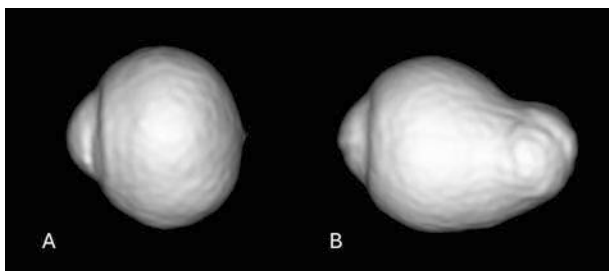


Fig. 13 Three-dimensional magnetic resonance imaging (3D MRI) of the eye. 3D MRI allows visualisation of the entire eye shape from any angle. Emmetropic eyes (**A**) appear nearly spherical, whereas eyes with pathologic myopia (**B**) show severe deformation beyond simple elongation. The patterns of deformity vary among patients.

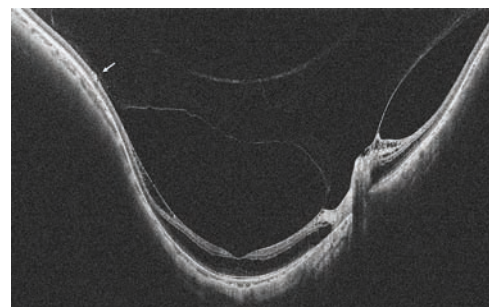


Fig. 14 Ultra-widefield OCT imaging of posterior staphyloma. In this widefield view, the edge of the staphyloma (arrow) is clearly identified as an inward protrusion of the sclera. The image illustrates the spatial relationship among the staphyloma, vitreous adhesion, and myopic traction maculopathy.

shape of the posterior segment of the eye. While 3D MRI provides the visualisation of the entire eye, it has relatively low spatial resolution and requires lengthy acquisition times. In contrast, UWF-OCT delivers high-resolution cross-sectional images of the retina and choroid with much greater detail and speed. It enables precise visualisation of posterior staphylomas, scleral curvature, and fundus complications (like

MTM) over a wide field (Fig. 14) [25, 42], often exceeding 100 degrees. Furthermore, UWF-OCT is more accessible in clinical settings, non-invasive, and does not require exposure to magnetic fields, making it a more practical tool for routine assessment of posterior eye morphology, especially in patients with high or pathologic myopia. Shinohara et al. [42, 43]

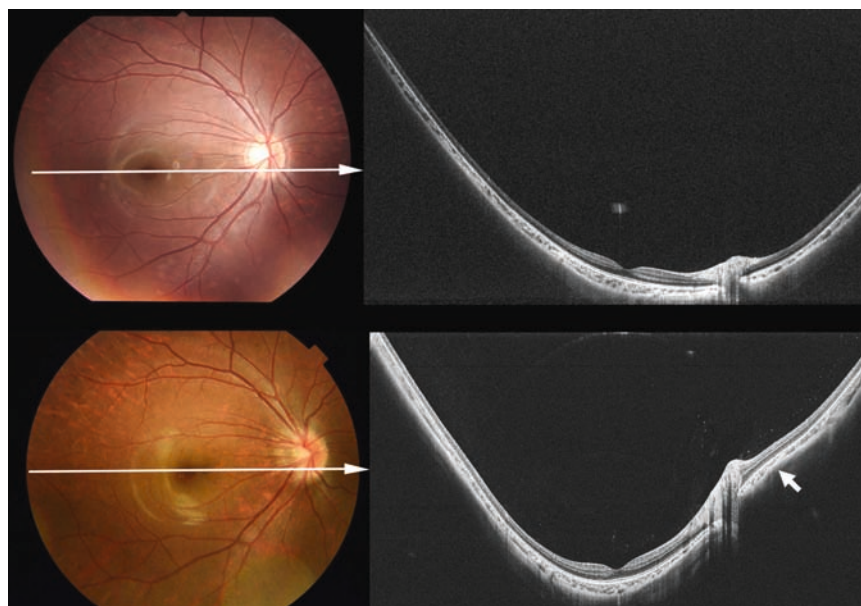


Fig. 15 Development of staphyloma in children. Top row: Fundus photograph of the right eye of an 8-year-old girl with an axial length of 26 mm. At baseline, the inner scleral curvature is inclined toward the optic disc, and no staphyloma edge is observed. Bottom row: Three years later, the axial length has increased by 1 mm. An inward scleral protrusion, suggestive of a staphyloma edge, has formed nasal to the optic disc (arrow). The macular sclera bows more posteriorly, resulting in a symmetrical curvature around the fovea.

showed that the presence and types of staphylomas judged by UWF-OCT was comparable to 3D MRI. Thus, UWF-OCT can be used to detect staphylomas as efficiently as 3D MRI.

Importantly, UWF-OCT can be used in paediatric populations. Chen et al. [44] longitudinally monitored 47 eyes of 24 highly myopic children and adolescents for 2–4 years using UWF-OCT. They found that 14% of eyes developed new posterior staphylomas, predominantly located near the optic disc, with earlier onset in children younger than 10 years (Fig. 15). This underscores that staphyloma formation may begin early in life, and UWF-OCT provides a valuable modality for early detection and monitoring in paediatric high myopia.

Although the precise mechanisms underlying staphyloma formation remain unclear, McBrien et al. [45] hypothesised that scleral collagen fibre orientation plays a crucial role in determining staphyloma location and development.

Scleral collagen fibre architecture is considered fundamental to scleral biomechanics and the pathogenesis of staphylomas. However, observing the direction of scleral fibres *in vivo* in human eyes has been challenging, and most previous investigations have relied on histological analyses. Recently, polarisation-sensitive OCT (PS-OCT) has emerged as a promising tool that enables the visualisation of scleral collagen fibre orientation *in vivo*, providing new insights into the biomechanical properties of the sclera related to staphyloma formation.

Polarisation-sensitive OCT (PS-OCT) is an advanced imaging modality that extends conventional OCT by incorporating polarisation contrast in addition to standard intensity-based contrast [46, 47]. Although the resolution of PS-OCT is generally not sufficient to visualise individual collagen lamellae within the sclera [48], it has enabled the assessment of polarisation characteristics on the scale of tens of micrometres in both animal and human tissues [49–53]. Importantly, the sclera exhibits birefringence—a property of fibrous tissues with regularly arranged nanostructures where the refractive index varies with polarisation state. PS-OCT not only quantifies the magnitude of birefringence, which corresponds to fibre density, but also visualises the optic axis, indicating the directional alignment of collagen fibres.

In highly myopic eyes, retinal and choroidal thinning facilitates imaging of the entire scleral thickness with PS-OCT. Ohno-Matsui et al. [54] described distinct collagen fibre orientation patterns in non-PM highly myopic eyes: inner scleral fibres radiate from the optic nerve head peripherally, whereas outer scleral fibres run vertically and obliquely (Fig. 16). This differentiation permits separate analysis of inner and outer scleral layers. In PM eyes, thinning predominantly affects the inner sclera, while the outer sclera remains relatively preserved.

Prior to staphyloma edge analysis, Ohno-Matsui et al. [54] analysed dome-shaped macula (DSM), which is anterior protrusion of the macular sclera [55–59]. DSM exhibited an aggregation and thickening of low-birefringence fibres in the inner sclera at the dome site, while outer scleral fibres appeared compressed and thinner (Fig. 17). This suggests DSM formation is primarily driven by inner scleral fibre reorganisation.

Wu et al. [60] further demonstrated with PS-OCT that inner scleral horizontal fibres originally spanning the fovea become displaced and aggregate along staphyloma edges. Upper and lower to the staphyloma edges, opposite directions of oblique fibres were seen to be dragged toward the staphyloma edge (Fig. 18). These findings indicate that both DSM and staphyloma edges arise from localised aggregation of inner scleral fibres, while the outer sclera plays a lesser role in these anterior protrusions. Thus, the inner and outer scleral layers may serve distinct physiological and pathological functions, with the inner sclera chiefly responsible for scleral curvature irregularities.

FUTURE THERAPIES FOR STRENGTHENING SCLERA

Given that posterior staphylomas underlie many PM-related fundus complications, early therapeutic strategies aimed at reinforcing the sclera prior to irreversible damage are highly desirable.

Several approaches have targeted the posterior sclera, including scleral collagen cross-linking (SXL) and posterior scleral reinforcement (PSR).

SXL aims to biomechanically strengthen the sclera by increasing collagen cross-links, thus potentially halting or slowing myopic

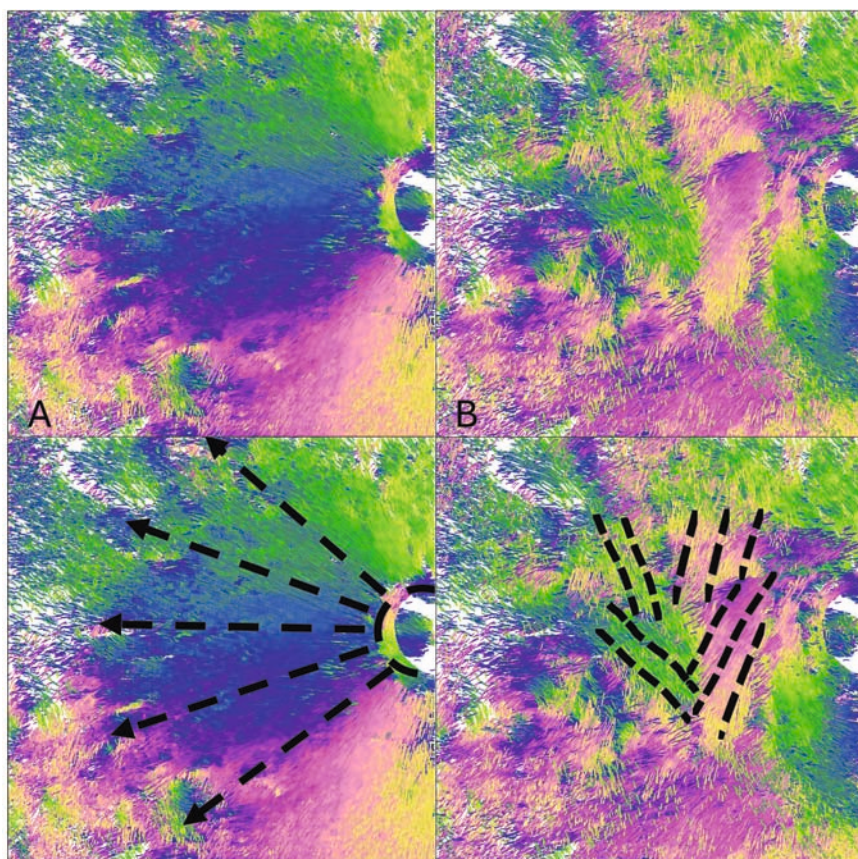


Fig. 16 Streamline images from polarisation-sensitive OCT of a highly myopic eye. The orientation of scleral collagen fibres is colour-coded as follows: blue indicates horizontal, yellow vertical, and pink/green oblique fibres. **A** View from inside the eye showing fibres of the inner sclera radiating outward from the optic nerve toward the periphery. **B** View from outside the eye illustrating outer scleral fibres with predominantly vertical and oblique orientations. The two images in the bottom row illustrate the orientation of scleral fibres.

progression [61]. Traditional photochemical SXL employs ultraviolet-A (UV-A) light combined with riboflavin but its invasiveness and the risk of cytotoxicity should be further elucidated. Consequently, alternative protocols using UV-B light, rose bengal activated by green light (RGX), or methylene blue with red light have been explored, each varying in penetration depth, efficacy, and safety profiles. Chemical (non-light) cross-linking methods avoid irradiation but face challenges in effective delivery and localisation. Novel modalities such as low-level laser therapy (LLLT) and near-infrared (NIR) light combined with bacteriochlorophyll derivatives offer minimally invasive options. Although animal studies show encouraging results, further research is required to establish safe, effective protocols for human application.

PSR surgery, developed initially to mechanically stabilise axial elongation, has demonstrated promising outcomes in paediatric populations. A recent meta-analysis of eight studies comprising 357 children revealed that PSR significantly slowed an increase of axial length and the progression of myopic refractive error, with minimal adverse events [62]. These findings suggest that PSR may be a valuable intervention for controlling myopia progression from an early age, although further high-quality, global studies are needed to assess its broader applicability.

Cell-based regenerative therapies represent an innovative avenue. Shinohara et al. [63] investigated transplantation of human fibroblasts (hFBs) onto the sclera in a rat model of form-deprivation myopia. In this model, form deprivation induced axial elongation, scleral thinning, and smaller collagen fibrils. Transplantation of hFBs promoted synthesis of mature type I collagen fibrils within the sclera, integrating into the tissue and significantly reducing axial elongation and myopic progression. These

findings highlight the potential of cell-mediated scleral reinforcement as a therapeutic strategy in humans.

MYOPIA PREVENTION THERAPIES AND ITS IMPACT ON PATHOLOGIC MYOPIA

In recent years, numerous interventions have been developed to prevent or slow the progression of myopia in children. These include low-dose atropine eye drops [64, 65], orthokeratology [66], spectacles with Defocus Incorporated Multiple Segments (DIMS) [67], Highly Aspherical Lenslet (HAL) lenses [68, 69], Diffusion Optics Technology (DOT) [70], soft multifocal contact lenses [71], and low-level red-light therapy (RLRL) [72, 73]. In addition, new treatment modalities continue to emerge and are anticipated in the near future.

Myopia is a well-established risk factor for sight-threatening conditions such as glaucoma, cataract, and retinal detachment [74, 75]. Axial elongation in myopic children typically occurs in the equatorial region, which may differ from the posterior pole deformation observed in pathologic myopia, such as posterior staphyloma. It remains unclear whether controlling myopia in childhood can effectively reduce the incidence of pathologic myopia in adulthood, or whether pathologic myopia is predominantly genetically determined and distinct from common axial myopia. Long-term studies are needed to clarify this relationship.

CONCLUSIONS

Posterior staphyloma is a defining feature of PM and a critical determinant of associated fundus complications. It must be

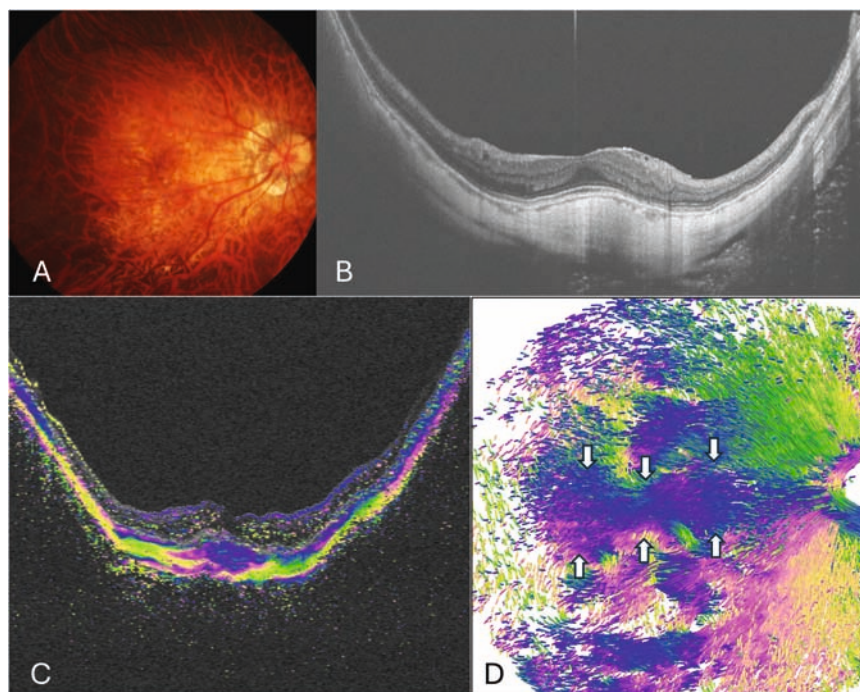


Fig. 17 Aggregation of inner scleral fibres at the dome-shaped macula (DSM). **A** Fundus photograph of the right eye showing diffuse choroidal atrophy. **B** OCT image demonstrating inward scleral protrusion characteristic of DSM. **C** Vertical scan along the optic axis reveals aggregation of inner scleral horizontal fibres within the dome, whereas surrounding regions predominantly contain outer scleral vertical fibres. In the dome area, the yellow-coloured outer scleral fibres appear compressed rather than thickened. **D** Streamline image viewed from inside the eye highlights intense aggregation of horizontal fibres across the macula (between arrows).

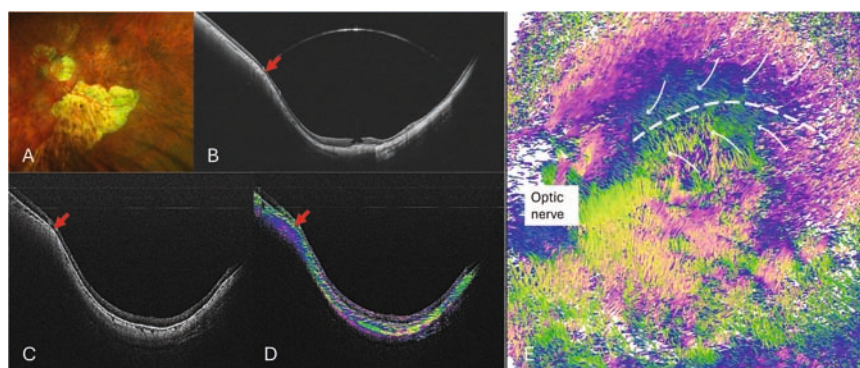


Fig. 18 Aggregation of inner scleral fibres at the edge of staphyloma. **A** Fundus photograph of the left eye showing patchy atrophy inferior to the macula. **B, C** Vertical OCT scans revealing the upper edge of the staphyloma (arrow). **D** Polarisation-sensitive OCT (PS-OCT) B-scan illustrating aggregation of inner scleral horizontal fibres (blue) at the upper staphyloma edge. **E** Streamline image showing displacement of the normally macula-distributed horizontal blue fibres toward the upper edge of the staphyloma. Above and below this edge, oblique fibres oriented in opposite directions appear to be pulled toward the edge.

distinguished from physiological myopia since staphylomas cause localised posterior scleral protrusion leading to progressive damage of the macular retina and optic nerve. Such structural alterations underlie major vision-threatening complications, including myopic neovascularisation (MNV), MNV-related macular atrophy, myopic traction maculopathy (MTM), and optic nerve injury, which collectively account for severe visual impairment in PM.

Given the pivotal role of the sclera in maintaining ocular structural integrity and protecting intraocular neural tissues, early detection and therapeutic intervention targeting the sclera prior to irreversible neuroretinal damage hold promise as effective strategies. Nonetheless, larger clinical studies with extended follow-up are required to confirm the long-term safety and efficacy of emerging scleral-targeted treatments in human PM.

REFERENCES

- Holden BA, Fricke TR, Wilson DA, Jong M, Naidoo KS, Sankaridurg P, et al. Global prevalence of myopia and high myopia and temporal trends from 2000 through 2050. *Ophthalmology*. 2016;123:1036–42.
- Xu L, Wang Y, Li Y, Cui T, Li J, Jonas JB. Causes of blindness and visual impairment in urban and rural areas in Beijing: the Beijing Eye Study. *Ophthalmology*. 2006;113:1134 e1131–1111.
- Iwase A, Araie M, Tomidokoro A, Yamamoto T, Shimizu H, Kitazawa Y. Prevalence and causes of low vision and blindness in a Japanese adult population: the Tajimi Study. *Ophthalmology*. 2006;113:1354–62.
- Foster PJ, Jiang Y. Epidemiology of myopia. *Eye (Lond, Engl)*. 2014;28:202–8.
- Morgan IG, Ohno-Matsui K, Saw SM. Myopia. *Lancet*. 2012;379:1739–48.
- Wong YL, Saw SM. Epidemiology of pathologic myopia in Asia and worldwide. *Asia-Pac J Ophthalmol (Philos, Pa)*. 2016;5:394–402.
- Flitcroft DI, He M, Jonas JB, Jong M, Naidoo K, Ohno-Matsui K, et al. IMI - Defining and Classifying Myopia: A Proposed Set of Standards for Clinical and Epidemiologic Studies. *Investig Ophthalmol Vis Sci*. 2019;60:M20–m30.

8. Ohno-Matsui K, Lai TYY, Cheung CMG, Lai CC. Updates of pathologic myopia. *Prog retinal eye Res.* 2016;52:156–87.
9. Ohno-Matsui K, Wu PC, Yamashiro K, Vutipongsatorn K, Fang Y, Cheung CMG, et al. IMI pathologic myopia. *Investig Ophthalmol Vis Sci.* 2021;62:5.
10. Ohno-Matsui K. Definition of pathologic myopia. In: Richard F Spaide KO-M, Lawrence A. Yannuzzi (ed). *Pathologic Myopia*, 2 ed. Springer Nature: Switzerland; 2021: 13–15.
11. Wang NK, Wu YM, Wang JP, Liu L, Yeung L, Chen YP, et al. Clinical characteristics of posterior staphylomas in myopic eyes with axial length shorter than 26.5 mm. *Am J Ophthalmol.* 2016;162:180–90.
12. Ohno-Matsui K, Jonas JB. Posterior staphyloma in pathologic myopia. *Prog Retinal Eye Res.* 2019;70:99–109.
13. Ohno-Matsui K, Kawasaki R, Jonas JB, Cheung CM, Saw SM, Verhoeven VJ, et al. International photographic classification and grading system for myopic maculopathy. *Am J Ophthalmol.* 2015;159:877–83.
14. Ohno-Matsui K. Myopic chorioretinal atrophy. In: Spaide RF, Ohno-Matsui K, Yannuzzi LA (eds). *Pathologic Myopia*. Springer: New York; 2014. pp 187–210.
15. Fang Y. Macular Diffuse Choroidal Atrophy. In: Ohno-Matsui K (ed). *Atlas of Pathologic Myopia*. Springer; 2020. pp 53–56.
16. Fang Y, Yokoi T, Nagaoka N, Shinohara K, Onishi Y, Ishida T, et al. Progression of myopic maculopathy during 18-year follow-up. *Ophthalmology.* 2018;125:863–77.
17. Ohno-Matsui K, Jonas JB, Spaide RF. Macular Bruch membrane holes in highly myopic patchy chorioretinal atrophy. *Am J Ophthalmol.* 2016;166:22–28.
18. Yoshida T, Ohno-Matsui K, Yasuzumi K, Kojima A, Shimada N, Futagami S, et al. Myopic choroidal neovascularization: a 10-year follow-up. *Ophthalmology.* 2003;110:1297–305.
19. Wang Y, Du R, Xie S, Chen C, Lu H, Xiong J, et al. Machine learning models for predicting long-term visual acuity in highly myopic eyes. *JAMA Ophthalmol.* 2023;141:1117–24.
20. Takano M, Kishi S. Foveal retinoschisis and retinal detachment in severely myopic eyes with posterior staphyloma. *Am J Ophthalmol.* 1999;128:472–6.
21. Ikuno Y, Sayanagi K, Oshima T, Gomi F, Kusaka S, Kamei M, et al. Optical coherence tomographic findings of macular holes and retinal detachment after vitrectomy in highly myopic eyes. *Am J Ophthalmol.* 2003;136:477–81.
22. Gaucher D, Haouchine B, Tadayoni R, Massin P, Erginay A, Benhamou N, et al. Long-term follow-up of high myopic foveoschisis: natural course and surgical outcome. *Am J Ophthalmol.* 2007;143:455–62.
23. Panozzo G, Mercanti A. Optical coherence tomography findings in myopic traction maculopathy. *Arch Ophthalmol (Chic, Ill : 1960).* 2004;122:1455–60.
24. Shimada N, Ohno-Matsui K, Baba T, Futagami S, Tokoro T, Mochizuki M. Natural course of macular retinoschisis in highly myopic eyes without macular hole or retinal detachment. *Am J Ophthalmol.* 2006;142:497–500.
25. Takahashi H, Tanaka N, Shinohara K, Uramoto K, Yokoi T, Yoshida T, et al. Importance of paravascular vitreal adhesions for development of myopic macular retinoschisis detected by ultra-widefield OCT. *Ophthalmology.* 2021;128:256–65.
26. Shimada N, Sugamoto Y, Ogawa M, Takase H, Ohno-Matsui K. Fovea-sparing internal limiting membrane peeling for myopic traction maculopathy. *Am J Ophthalmol.* 2012;154:693–701.
27. Ho TC, Chen MS, Huang JS, Shih YF, Ho H, Huang YH. Foveola nonpeeling technique in internal limiting membrane peeling of myopic foveoschisis surgery. *Retin (Philos, PA).* 2012;32:631–4.
28. Okuda T, Higashide T, Kobayashi K, Ikuno Y, Sugiyama K. Macular hole closure over residual subretinal fluid by an inverted internal limiting membrane flap technique in patients with macular hole retinal detachment in high myopia. *Retin Cases Brief Rep.* 2016;10:140–4.
29. Nagaoka N, Jonas JB, Morohoshi K, Moriyama M, Shimada N, Yoshida T, et al. Glaucomatous-type optic discs in high myopia. *PLoS one.* 2015;10:e0138825.
30. Lu Y, Ji Z, Jia J, Shi R, Liu Y, Shu Q, et al. Progress in clinical characteristics of high myopia with primary open-angle glaucoma. *Biotechnol Genet Eng Rev.* 2024;40:4923–42.
31. Ohno-Matsui K, Akiba M, Moriyama M, Shimada N, Ishibashi T, Tokoro T, et al. Acquired optic nerve and peripapillary pits in pathologic myopia. *Ophthalmology.* 2012;119:1685–92.
32. Ohno-Matsui K, Shimada N, Yasuzumi K, Hayashi K, Yoshida T, Kojima A, et al. Long-term development of significant visual field defects in highly myopic eyes. *Am J Ophthalmol.* 2011;152:256–65.
33. Xie S, Kamoi K, Igarashi-Yokoi T, Uramoto K, Takahashi H, Nakao N, et al. Structural abnormalities in the papillary and peripapillary areas and corresponding visual field defects in eyes with pathologic myopia. *Investig Ophthalmol Vis Sci.* 2022;63:13.
34. Spaide RF, Akiba M, Ohno-Matsui K. Evaluation of peripapillary intrachoroidal cavitation with swept source and enhanced depth imaging optical coherence tomography. *Retin (Philos, PA).* 2012;32:1037–44.
35. Shimada N, Ohno-Matsui K, Yoshida T, Yasuzumi K, Kojima A, Kobayashi K, et al. Characteristics of peripapillary detachment in pathologic myopia. *Arch Ophthalmol (Chic, Ill : 1960).* 2006;124:46–52.
36. Spaide RF *Staphyloma: Part 1*. Springer: New York; 2014.
37. Ohno-Matsui K. Proposed classification of posterior staphylomas based on analyses of eye shape by three-dimensional magnetic resonance imaging and wide-field fundus imaging. *Ophthalmology.* 2014;121:1798–809.
38. Wang N, Wu Y, Wang J, Liu L, Yeung L, Chen Y, et al. Clinical characteristics of posterior staphylomas in myopic eyes with axial length shorter than 26.5 millimeters. *Am J Ophthalmol.* 2016;162:180–90.
39. Moriyama M, Ohno-Matsui K, Hayashi K, Shimada N, Yoshida T, Tokoro T, et al. Topographic analyses of shape of eyes with pathologic myopia by high-resolution three-dimensional magnetic resonance imaging. *Ophthalmology.* 2011;118:1626–37.
40. Moriyama M, Ohno-Matsui K, Modegi T, Kondo J, Takahashi Y, Tomita M, et al. Quantitative analyses of high-resolution 3D MR images of highly myopic eyes to determine their shapes. *Investig Ophthalmol Vis Sci.* 2012;53:4510–8.
41. Takahashi T, Yokoi-Igarashi T, Takahashi H, Nakao N, Uramoto K, Yoshida T, et al. Evaluating the volume of eyes with pathologic myopia using 3D MRI. *Investig Ophthalmol Vis Sci.* 2025;66:13.
42. Shinohara K, Tanaka N, Jonas JB, Shimada N, Moriyama M, Yoshida T, et al. Ultra-widefield optical coherence tomography to investigate relationships between myopic macular retinoschisis and posterior staphyloma. *Ophthalmology.* 2018;125:1575–86.
43. Shinohara K, Shimada N, Moriyama M, Yoshida T, Jonas JB, Yoshimura N, et al. Posterior staphylomas in pathologic myopia imaged by widefield optical coherence tomography. *Investig Ophthalmol Vis Sci.* 2017;58:3750–8.
44. Chen C, Du R, Xie S, Lu H, Xiong J, Wang Y, et al. Longitudinal study of changes in inner scleral curvature patterns and development of posterior staphylomas in highly myopic eyes of children and adolescents. *Retin (Philos, PA).* 2023;43:454–63.
45. McBrien NA, Gentle A. Role of the sclera in the development and pathological complications of myopia. *Prog Retinal Eye Res.* 2003;22:307–38.
46. Baumann B. Polarization sensitive optical coherence tomography: a review of technology and applications. *Appl Sci.* 2017;7:474.
47. de Boer JF, Hitzengerber CK, Yasuno Y. Polarization sensitive optical coherence tomography - a review [Invited]. *Biomed Opt Expr.* 2017;8:1838–73.
48. Li Q, Karnowski K, Untracht G, Noble PB, Cense B, Villiger M, et al. Vectorial birefringence imaging by optical coherence microscopy for assessing fibrillar microstructures in the cornea and limbus. *Biomed Opt Expr.* 2020;11:1122–38.
49. Yamanari M, Ishii K, Fukuda S, Lim Y, Duan L, Makita S, et al. Optical rheology of porcine sclera by birefringence imaging. *PLoS One.* 2012;7:e44026.
50. Nagase S, Yamanari M, Tanaka R, Yasui T, Miura M, Iwasaki T, et al. Anisotropic alteration of scleral birefringence to uniaxial mechanical strain. *PLoS One.* 2013;8:e58716.
51. Sugiyama S, Hong YJ, Kasaragod D, Makita S, Uematsu S, Ikuno Y, et al. Birefringence imaging of posterior eye by multi-functional Jones matrix optical coherence tomography. *Biomed Opt Express.* 2015;6:4951–74.
52. Fialová S, Augustin M, Glösmann M, Himmel T, Rauscher S, Gröger M, et al. Polarization properties of single layers in the posterior eyes of mice and rats investigated using high resolution polarization sensitive optical coherence tomography. *Biomed Opt Expr.* 2016;7:1479–95.
53. Fialová S, Augustin M, Fischak C, Schmetterer L, Handschuh S, Glösmann M, et al. Posterior rat eye during acute intraocular pressure elevation studied using polarization sensitive optical coherence tomography. *Biomed Opt Expr.* 2017;8:298–314.
54. Ohno-Matsui K, Igarashi-Yokoi T, Azuma T, Sugisawa K, Xiong J, Takahashi T, et al. Polarization-sensitive oct imaging of scleral abnormalities in eyes with high myopia and dome-shaped macula. *JAMA Ophthalmol.* 2024;142:310–9.
55. Gaucher D, Erginay A, Lecleire-Collet A, Haouchine B, Puech M, Cohen SY, et al. Dome-shaped macula in eyes with myopic posterior staphyloma. *Am J Ophthalmol.* 2008;145:909–14.
56. Caillaux V, Gaucher D, Gualino V, Massin P, Tadayoni R, Gaudric A. Morphologic characterization of dome-shaped macula in myopic eyes with serous macular detachment. *Am J Ophthalmol.* 2013;156:958–67.
57. Ellabban AA, Tsujikawa A, Matsumoto A, Yamashiro K, Oishi A, Ooto S, et al. Three-dimensional tomographic features of dome-shaped macula by swept-source optical coherence tomography. *Am J Ophthalmol.* 2012;3:578.
58. Fang Y, Jonas JB, Yokoi T, Cao K, Shinohara K, Ohno-Matsui K. Macular Bruch's membrane defect and dome-shaped macula in high myopia. *PLoS one.* 2017;12:e0178998.
59. Ohno-Matsui K, Fang Y, Uramoto K, Shinohara K, Yokoi T, Ishida T, et al. Peridome choroidal deepening in highly myopic eyes with dome-shaped maculas. *Am J Ophthalmol.* 2017;183:134–40.

60. Wu Y, Lu H, Chen C, Xiong J, Yamanari M, Takahashi H, et al. Polarization-Sensitive Optical Coherence Tomography Imaging of Posterior Staphyloma Edges in Eyes With High Myopia. *Invest Ophthalmol Vis Sci*. 2025;66:6.
61. Yasir ZH, Sharma R, Zakir SM. Scleral collagen cross linkage in progressive myopia. *Indian J Ophthalmol*. 2024;72:174–80.
62. Chen J, Tang Y, Lin Z, Tao Z, Deng H. Effect and safety posterior scleral reinforcement on controlling myopia in children: a meta-analysis. *Int Ophthalmol*. 2024;44:8.
63. Shinohara K, Yoshida T, Liu H, Ichinose S, Ishida T, Nakahama KI, et al. Establishment of novel therapy to reduce progression of myopia in rats with experimental myopia by fibroblast transplantation on sclera. *J Tissue Eng Regenerative Med*. 2018;12:e451–e461.
64. Chia A, Chua WH, Cheung YB, Wong WL, Lingham A, Fong A, et al. Atropine for the treatment of childhood myopia: safety and efficacy of 0.5%, 0.1%, and 0.01% doses (Atropine for the Treatment of Myopia 2). *Ophthalmology*. 2012;119:347–54.
65. Yam JC, Li FF, Zhang X, Tang SM, Yip BHK, Kam KW, et al. Two-year clinical trial of the low-concentration atropine for myopia progression (LAMP) study: phase 2 report. *Ophthalmology*. 2020;127:910–9.
66. Hiraoka T, Kakita T, Okamoto F, Takahashi H, Oshika T. Long-term effect of overnight orthokeratology on axial length elongation in childhood myopia: a 5-year follow-up study. *Invest Ophthalmol Vis Sci*. 2012;53:3913–9.
67. Lam CSY, Tang WC, Tse DY, Lee RPK, Chun RKM, Hasegawa K, et al. Defocus Incorporated Multiple Segments (DIMS) spectacle lenses slow myopia progression: a 2-year randomised clinical trial. *Br J Ophthalmol*. 2020;104:363–8.
68. Bao J, Huang Y, Li X, Yang A, Zhou F, Wu J, et al. Spectacle lenses with aspherical lenslets for myopia control vs single-vision spectacle lenses: a randomized clinical trial. *JAMA Ophthalmol*. 2022;140:472–8.
69. Sankaridurg P, Weng R, Tran H, Spiegel DP, Drobe B, Ha T, et al. Spectacle lenses with highly aspherical lenslets for slowing myopia: a randomized, double-blind, cross-over clinical trial: parts of these data were presented as a poster at the annual research in vision and ophthalmology meeting, 2022. *Am J Ophthalmol*. 2023;247:18–24.
70. Neitz J, Neitz M. Diffusion optics technology (DOT): a myopia control spectacle lens based on contrast theory. *Transl Vis Sci Technol*. 2024;13:42.
71. Chamberlain P, Peixoto-de-Matos SC, Logan NS, Ngo C, Jones D, Young G. A 3-year randomized clinical trial of MiSight lenses for myopia control. *Optom Vis Sci*. 2019;96:556–67.
72. Jiang Y, Zhu Z, Tan X, Kong X, Zhong H, Zhang J, et al. Effect of repeated low-level red-light therapy for myopia control in children: a multicenter randomized controlled trial. *Ophthalmology*. 2022;129:509–19.
73. Liu G, Liu L, Rong H, Li L, Liu X, Jia Z, et al. Axial shortening effects of repeated low-level red-light therapy in children with high myopia: a multicenter randomized controlled trial. *Am J Ophthalmol*. 2025;270:203–15.
74. Tideman JW, Snabel MC, Tedja MS, van Rijn GA, Wong KT, Kuijpers RW, et al. Association of axial length with risk of uncorrectable visual impairment for europeans with myopia. *JAMA Ophthalmol*. 2016;134:1355–63.
75. Bullimore MA, Brennan NA. Myopia control: why each diopter matters. *Optom Vis Sci*. 2019;96:463–5.

ACKNOWLEDGEMENTS

The article is based on the Duke Elder lecture which was held at the 2025 RCOphth Congress.

FUNDING

This work was supported by a Mission-Driven Strategic Research Grant from Institute of Science Tokyo.

COMPETING INTERESTS

The author declares no competing interests.

ADDITIONAL INFORMATION

Correspondence and requests for materials should be addressed to Kyoko Ohno-Matsui.

Reprints and permission information is available at <http://www.nature.com/reprints>

Publisher's note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Springer Nature or its licensor (e.g. a society or other partner) holds exclusive rights to this article under a publishing agreement with the author(s) or other rightsholder(s); author self-archiving of the accepted manuscript version of this article is solely governed by the terms of such publishing agreement and applicable law.