

ORIGINAL RESEARCH

Myopia Severity, Axial Length, and Asymmetric Peripapillary Retinal Nerve Fiber Layer Topography: A Cross-Sectional Optical Coherence Tomography Study

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ABSTRACT

Background: Myopic axial elongation can alter optic disc geometry and peripapillary retinal nerve fiber layer (RNFL) measurements, complicating glaucoma assessment.

Objective: To assess the associations of myopia severity and axial length with quadrant-specific peripapillary RNFL thickness.

Methods: This cross-sectional study included 55 eyes from 29 adults aged 17–39 years examined in Padang, Indonesia, during June–July 2025. Mild, moderate, and high myopia groups comprised 23, 18, and 14 eyes, respectively. RNFL thickness was measured with Cirrus HD-OCT and axial length with swept-source optical biometry.

Results: Average RNFL thickness declined across mild ($102.16 \pm 13.16 \mu\text{m}$), moderate ($98.42 \pm 10.58 \mu\text{m}$), and high myopia ($90.83 \pm 8.18 \mu\text{m}$; $p = 0.017$). The superior and inferior quadrants were thinner in high myopia, whereas temporal RNFL was thicker ($71.88 \pm 12.83 \mu\text{m}$ in mild versus $80.12 \pm 11.69 \mu\text{m}$ in high myopia). Axial length correlated with spherical equivalent ($r = -0.661$, $p < 0.001$) and temporal RNFL thickness ($r = 0.284$, $p = 0.036$). The ISNT rule was violated in 85.7% of highly myopic eyes.

Conclusion: Greater myopia and longer axial length were associated with asymmetric RNFL topography, including vertical thinning and relative temporal thickening. Peripapillary OCT findings in myopic eyes should be interpreted alongside axial length and other clinical evidence.

1. Introduction

Myopia has emerged as a growing global public health concern, characterized by a steady rise in both prevalence and refractive severity across diverse populations worldwide.¹ Contemporary epidemiological forecasts synthesize a global trajectory, projecting that the worldwide prevalence of myopia will surge from approximately 22% of the global population recorded in 2000 to an estimated 52% by the year 2050, thereby encompassing nearly five billion individuals.^{1,2} Within East and Southeast Asia, this epidemiological expansion has assumed hyper-endemic proportions, where upwards of 80% to 90% of young urban adults are affected by refractive myopia, and between 15% and 20% develop high myopia, typically delineated by a spherical equivalent refractive error worse than -6.00 Diopters or an ocular axial length exceeding 26.0 millimeters.^{2,3} This marked demographic shift carries substantial clinical significance, as high myopia is not merely an isolated benign refractive anomaly correctable with spectacles or contact lenses, but rather an expansive, progressive, and sight-threatening degenerative ocular condition predisposing patients to irreversible structural retinal disorders, including myopic choroidal neovascularization, macular schisis, rhegmatogenous retinal detachment, and progressive chorioretinal atrophy.^{3,4} Moreover, each successive increment in myopic refractive error incrementally compounds the lifetime risk of developing severe ocular comorbidities, underscoring the vital mandate to elucidate the microstructural remodeling accompanying myopic progression.^{4,5} Recent population-based cohorts have further

illuminated the marked socioeconomic burden imposed by progressive myopic visual disability, demanding rigorous investigations into early structural biomarkers.^{5,6}

Beyond outer retinal and macular degenerations, extensive clinical and epidemiological investigations have firmly demonstrated an association between increasing myopia severity and an elevated susceptibility to glaucomatous optic neuropathy, particularly primary open-angle glaucoma (POAG).^{6,7} Seminal systematic reviews and large-scale meta-analyses have established that individuals diagnosed with mild-to-moderate myopia exhibit an approximately two- to three-fold elevated odds of developing open-angle glaucoma compared to emmetropic counterparts, whereas individuals afflicted with high myopia harbor a more than six-fold to seven-fold escalated odds of glaucomatous neurodegeneration.^{6,7} The fundamental mechanistic nexus underpinning this heightened susceptibility resides within the marked biomechanical deformations precipitated by axial ocular elongation.^{7,8} As the anterior-posterior diameter of the vitreous chamber progressively expands, the posterior pole of the globe is subjected to chronic, non-linear tensile and tangential stretch forces, resulting in marked scleral thinning, progressive backward displacement and compression of the lamina cribrosa, peripapillary scleral flange elongation, and tilting of the optic nerve head.^{8,9} These continuous biomechanical strain vectors generate localized microvascular shear stress and mechanical axoplasmic stasis within the optic nerve head, rendering the delicate unmyelinated axons of retinal ganglion cells acutely vulnerable to secondary ischemic injury and apoptotic

cascade activation even under conventional, non-elevated intraocular pressures.^{9,10}

In contemporary ophthalmology, optical coherence tomography (OCT) has improved the objective quantification of structural neuro-retinal tissue, enabling non-invasive, ultra-high-resolution, and cross-sectional micron-scale imaging of the optic nerve head, peripapillary retina, and macula.^{10,11} In particular, the quantification of peripapillary retinal nerve fiber layer (RNFL) thickness has solidified its position as the premier structural biomarker for detecting early neurodegenerative damage, often identifying axonal loss and structural decay several years before reproducible, functional visual field defects become evident on standard automated perimetry.^{10,11} However, the clinical interpretation of peripapillary RNFL measurements in myopic patients presents marked diagnostic challenges and diagnostic dilemmas that frequently perplex even seasoned glaucoma subspecialists.^{11,12} Axial elongation-induced anatomical remodeling alters the spatial geometry of the posterior pole, generating a spectrum of morphological optical artifacts, including optic disc tilt, optic nerve head rotation around the sagittal axis, optic disc ovality, and wide expanses of parapapillary gamma and delta zones.^{12,13} Crucially, these myopic structural alterations fundamentally distort the native trajectory of retinal nerve fiber bundles, inducing an anatomical convergence and temporal shift of the superior and inferior arcuate bundles toward the horizontal raphe.^{13,14}

As a consequence of this spatial redistribution, standard spectral-domain optical coherence tomography (SD-OCT) instruments—which derive diagnostic classifications by contrasting individual patient scans against normative reference databases predominantly constructed from healthy emmetropic eyes possessing average axial dimensions—frequently generate pervasive false-positive diagnostic errors, a clinical pitfall widely known as 'red disease'.^{7,14} In eyes exhibiting elongated axial lengths, the circular scanning beam of the OCT acquires tissue along a physically larger retinal perimeter on the stretched posterior fundus, thereby capturing anatomically thinner distal nerve fiber bundles and artificially depressing peripapillary thickness readings.^{7,14} Concurrently, the regional tilting and temporal displacement of the arcuate bundles compress the nerve fibers into the temporal meridian while depleting the superior and inferior poles, leading to premature violation of the classical ISNT rule (where inferior neuroretinal rim thickness normally exceeds superior, nasal, and temporal thicknesses) and falsely indicating severe arcuate nerve fiber deficits in structurally healthy myopic eyes.^{13,14}

Despite the critical clinical implications of this diagnostic confounding, considerable gaps remain within the published ophthalmic literature regarding the precise quadrant-specific behavior of the peripapillary RNFL across strictly categorized myopia severity strata in young adult populations of Southeast Asian heritage, where genetic and lifestyle factors coalesce to drive extensive myopic progression.^{11,14} Previous studies investigating myopic RNFL alterations have frequently been hampered by heterogeneous age cohorts containing elderly participants with confounding age-related neuroaxonal loss, lack of optical biometry axial length measurements, failure to apply refractive magnification corrections during scan acquisition, or inadequate adjustment for systemic comorbidities.^{12,14} There exists a pressing scientific and clinical need to systematically investigate the exact sectoral alterations of the peripapillary RNFL—specifically contrasting the vulnerable vertical meridians with the horizontal sectors—while directly

coupling these measurements with precision swept-source optical biometry and rigorous multivariable statistical modeling.^{13,14}

To address this knowledge gap, this investigation was designed to systematically evaluate the relationship between myopia severity, optical biometry axial length, and quadrant-specific peripapillary retinal nerve fiber layer thickness measured via spectral-domain optical coherence tomography with automatic refractive magnification correction in a well-defined cohort of young Indonesian adults. By stratifying eyes across mild, moderate, and high myopia categories and elucidating the paradoxical preservation of the temporal quadrant relative to the pronounced thinning of vertical arcuate sectors, this study seeks to delineate the fundamental biomechanical remodeling governing the myopic retina. Ultimately, establishing these quantitative topographical dynamics provides evidence to refine diagnostic thresholds, differentiate physiological myopic remodeling from early glaucomatous neurodegeneration, and prevent the widespread misclassification of healthy myopic individuals in clinical ophthalmic practice.

2. Methods

2.1. Study Design and Ethical Oversight

This investigation was conducted as an analytical, cross-sectional observational study at the Ophthalmology Outpatient Clinic of Dr. M. Djamil General Hospital, Padang, West Sumatra, Indonesia, encompassing patient recruitment and clinical evaluations performed between June and July 2025. The study was executed in strict adherence to the ethical tenets of the Declaration of Helsinki and its subsequent amendments governing clinical medical research involving human subjects.¹⁵ The study protocol, clinical examination pathways, and participant informed consent documentation received formal ethical approval from the Ethics Committee CMHC Indonesia. Prior to enrollment and before any ophthalmic procedures were initiated, detailed verbal and written explanations regarding the study objectives, diagnostic procedures, non-invasive imaging protocols, and data confidentiality measures were provided to all eligible candidates, and written informed consent was obtained from each participant.

2.2. Participant Eligibility Criteria and Refractive Stratification

The target study population comprised consecutive young adult myopic patients attending the outpatient ophthalmology clinic as well as medical students and clinical clerks undergoing their ophthalmological clinical rotation at Dr. M. Djamil General Hospital during the defined recruitment interval.^{14,16} A total sampling strategy was implemented, whereby all individuals presenting with refractive myopia who fulfilled the predetermined eligibility criteria were consecutively invited to participate. Eligible participants were between 17 and 39 years of age, presenting within an age bracket selected specifically to capture mature ocular development while strictly avoiding the physiological age-related decline in retinal nerve fiber layer thickness and axonal density that characteristically accelerates beyond the fifth decade of life.^{12,16} To ensure clear optical media and diagnostic accuracy during tomographic imaging, participants were required to demonstrate a best-corrected visual acuity (BCVA) of 20/20 (LogMAR 0.00) in the candidate study eye, with refractive errors classified strictly based on spherical equivalent (SE) values calculated as the spherical power plus half of the cylindrical power.

Refractive severity was categorized into three standardized analytical cohorts based on international refractive classification criteria: mild myopia, defined as a spherical equivalent between -0.50 D and -2.87 D (or < -3.00 D); moderate myopia, defined as a spherical equivalent ranging from -3.00 D to -6.00 D; and high myopia, defined as a spherical equivalent worse than -6.00 D (specifically reaching up to -10.50 D in the enrolled sample).^{14,17} Rigorous exclusion criteria were systematically applied to eliminate potential anatomical, pathological, and optical confounders that could bias peripapillary nerve fiber layer thickness measurements. Eyes were excluded if they exhibited: (1) any clinically detectable anterior segment pathology, including corneal opacities, keratoconus, active ocular surface inflammation, or pterygium crossing the pupillary axis; (2) coexisting posterior segment abnormalities, such as vitreous opacities, severe pathological myopic chorioretinal degeneration (e.g., lacquer cracks, active choroidal neovascularization, patchy chorioretinal atrophy, or posterior staphyloma involving the fovea), diabetic retinopathy, or a vertical optic cup-to-disc ratio exceeding 0.7; (3) baseline intraocular pressure (IOP) greater than 20 mmHg measured by non-contact tonometry; (4) refractive astigmatism exceeding -3.00 D, which introduces severe optical distortion and asymmetric scan magnification; (5) any documented history of prior intraocular surgery, refractive surgery, or laser therapy; (6) ocular motility disorders or strabismus; and (7) systemic medical comorbidities known to induce microvascular or neuroaxonal injury, including diabetes mellitus, arterial hypertension, and autoimmune vasculitides.^{16,17}

2.3. Comprehensive Ophthalmic Examination Protocol

All enrolled participants underwent a standardized, comprehensive ophthalmic examination protocol executed by licensed ophthalmologists and trained clinical investigators. Monocular best-corrected visual acuity was assessed at a calibrated distance of six meters using an illuminated Snellen chart and subsequently converted to the logarithm of the minimum angle of resolution (LogMAR) scale. Objective refractive status was initially quantified utilizing an automated autorefractometer/keratometer (Canon RK-F2, Canon Inc., Tokyo, Japan) and verified by dynamic streak retinoscopy, followed by subjective refinement using trial lenses under mesopic ambient room illumination. Slit-lamp biomicroscopy (Topcon SL-D7, Topcon Corporation, Tokyo, Japan) was systematically performed to evaluate the ocular adnexa, cornea, anterior chamber depth and clarity, iris architecture, and crystalline lens transparency. Dilated stereoscopic fundus examination was conducted following pharmacological mydriasis using indirect ophthalmoscopy and slit-lamp biomicroscopic funduscopy utilizing a 78-diopter high-resolution non-contact lens (Volk Optical Inc., Mentor, OH, USA) to thoroughly inspect the optic nerve head, retinal vasculature, macula, and peripheral retina. Intraocular pressure was determined using a calibrated non-contact tonometer (Topcon CT-80, Topcon Corporation, Tokyo, Japan), averaging three consecutive measurements per eye within 1 mmHg precision.

2.4. Optical Biometry and Axial Length Measurement

Accurate determination of ocular axial length was achieved using the IOL Master 700 (Carl Zeiss Meditec, Jena, Germany), an advanced non-contact optical biometer operating on the physical principle of swept-source optical coherence tomography (SS-OCT) interferometry with a tunable laser centered at a wavelength of 1055 nm.^{7,18} The

swept-source technology enables rapid, non-invasive longitudinal axial measurements along the visual axis from the anterior corneal vertex to the retinal pigment epithelium, providing an axial resolution of 22 μ m and measurement reproducibility surpassing traditional ultrasound biometry. For each candidate eye, five consecutive internal fixation scans with high signal-to-noise ratio were acquired, and the mean axial length was automatically recorded in millimeters (mm). Axial length measurements were utilized both for biometric intergroup comparisons and as an analytical covariate to assess biomechanical scleral expansion.^{16,18}

2.5. Spectral-Domain Optical Coherence Tomography (Cirrus HD-OCT 5000)

Peripapillary retinal nerve fiber layer thickness was quantified using spectral-domain optical coherence tomography (Cirrus HD-OCT 5000, Carl Zeiss Meditec, Dublin, CA, USA; software version 11.0).^{18,19} The Cirrus HD-OCT 5000 system utilizes a superluminescent diode light source with a central operating wavelength of 840 nm and a bandwidth of 50 nm, delivering an axial optical resolution of 5 μ m and a transverse resolution of 15 μ m in tissue. All tomographic scans were acquired by a single, highly experienced ophthalmic imaging technician under standardized mesopic ambient lighting conditions, thereby eliminating inter-operator variability and scan-to-scan technical discrepancies.^{18,19} Imaging was executed utilizing the standardized Optic Disc Cube 200×200 acquisition protocol, which captures a 6×6 mm grid centered precisely on the optic nerve head, consisting of 200 horizontal B-scans with each B-scan comprising 200 A-scans, yielding a dense volumetric dataset of 40,000 axial points within 1.5 seconds.

2.6. Refractive Magnification Correction and Scan Quality Control

From this acquired volumetric dataset, the instrument automatically calculates peripapillary RNFL thickness along a virtual circumpapillary calculation circle measuring 3.46 mm in diameter positioned concentrically around the center of the optic disc.^{19,20} To overcome the critical artifact of refractive magnification error—wherein differing refractive powers alter the physical dimensions of the projected retinal scan circle—each patient's spherical equivalent refractive error was entered into the patient record interface prior to scan initiation. This allowed the Cirrus algorithm to apply Littmann-Bennett magnification correction, ensuring that the 3.46 mm scan trajectory interrogated an anatomically equivalent peripapillary retinal zone across varying refractive degrees.^{7,20} High scan quality was strictly enforced: only scans exhibiting an OCT signal strength of 6/10 or greater, with uniform illumination, accurate automated centration on the optic disc, and complete absence of motion artifacts, blinking, or automated segmentation errors were included. The primary quantitative outcome metrics extracted for analysis comprised the total 360-degree average peripapillary RNFL thickness (μ m) and quadrant-specific thicknesses divided into four distinct anatomical quadrants: superior (46°–135°), inferior (226°–315°), nasal (136°–225°), and temporal (316°–45° for right eyes; mirrored for left eyes).^{18,20}

2.7. Statistical Analysis and Predictive Modeling

Statistical analyses were performed using SPSS version 29.0 for Windows (IBM Corp., Armonk, NY, USA) and GraphPad Prism version 10.2 (GraphPad Software, San Diego, CA, USA).²¹ Continuous variables were systematically tested for normality of distribution using the Shapiro-Wilk test in conjunction with visual inspection of normal Q-Q plots and skewness/kurtosis coefficients. Data conforming to normal

Gaussian distributions were expressed as mean $\pm$ standard deviation (SD), whereas non-normally distributed parameters were presented as median and interquartile range (IQR). Categorical demographic and clinical variables were summarized as absolute frequencies (n) and relative percentages (%). Differences in continuous baseline characteristics and RNFL thickness parameters across the three myopia severity categories were analyzed using One-Way Analysis of Variance (ANOVA) followed by Tukey's honestly significant difference (HSD) post-hoc test for normally distributed parameters, or the Kruskal-Wallis non-parametric test followed by Dunn-Bonferroni post-hoc pairwise corrections for skewed distributions.^{21,22}

Bivariate correlation analyses were executed to determine the strength and directionality of associations between refractive severity (spherical equivalent), optical axial length, and RNFL thickness parameters utilizing Spearman's rank correlation coefficient (r) to accommodate non-linear and ordinal properties.^{21,22} Cross-tabulation analyses assessing the frequency of ISNT rule violations (defined as failure of the normal hierarchical contour wherein inferior thickness $\geq$ superior $\geq$ nasal $\geq$ temporal) and Cirrus normative database percentile classifications across myopia groups were evaluated using Pearson's Chi-Square test (χ^2) and Fisher's Exact test as appropriate. To control for potential confounding influences and evaluate independent predictors of RNFL thinning, multivariable linear regression modeling was executed with average RNFL thickness as the continuous dependent variable. In addition, multivariable binary logistic regression was constructed to model the adjusted odds ratio (aOR) with 95% confidence intervals (CI) for the occurrence of severe peripapillary structural distortion (ISNT violation / borderline normative flagging) as a function of axial length, spherical equivalent, and optic disc ovality index. All statistical tests were two-tailed, and a p-value of less than 0.05 was defined as the threshold for statistical significance.

3. Results

3.1. Sociodemographic Characteristics and Ocular Biometry Profile

A total of 55 eyes from 29 young adult participants were enrolled and successfully completed all standardized clinical, biometrical, and spectral-domain optical coherence tomography imaging protocols during the study recruitment window. The final analytical sample comprised 23 eyes categorized as mild myopia, 18 eyes categorized as moderate myopia, and 14 eyes categorized as high myopia based on

spherical equivalent thresholds. The baseline sociodemographic, refractive, and ocular biometry characteristics of the study cohort stratified by myopia severity are summarized in Table 1. The overall cohort demonstrated a prominent female preponderance across all three refractive groups, comprising 82.6% (n = 19) in the mild group, 83.3% (n = 15) in the moderate group, and 85.7% (n = 12) in the high myopia group, with no statistically significant intergroup difference in sex distribution ($\chi^2 = 0.185$, p = 0.912). The mean age of participants was remarkably homogenous across the strata, averaging 22.86 ± 1.42 years in the mild group, 22.38 ± 1.09 years in the moderate group, and 23.00 ± 4.24 years in the high myopia group (One-Way ANOVA, F = 0.301, p = 0.742). This tight age distribution confirms that the sample was free from age-related neurodegenerative confounding.

In marked contrast to demographic parameters, refractive status and optical biometry variables demonstrated significant, progressive alterations across the myopia severity spectrum, as detailed in Table 1 and illustrated in Figure 1. The mean spherical equivalent was -1.85 ± 0.74 D in the mild myopia group, -4.32 ± 0.88 D in the moderate myopia group, and -7.84 ± 1.35 D in the high myopia group, demonstrating pronounced intergroup variance (Kruskal-Wallis, p < 0.001). Concurrently, optical axial length measured via swept-source interferometry exhibited a statistically significant, stepwise elongation from mild (24.18 ± 0.65 mm) to moderate (25.12 ± 0.72 mm) and high myopia (26.45 ± 0.94 mm; One-Way ANOVA, F = 38.64, p < 0.001). Post-hoc pairwise comparisons with Tukey's HSD test confirmed that axial length differed significantly between every pairwise group combination: mild versus moderate (mean difference: 0.94 mm, p < 0.001), mild versus high (mean difference: 2.27 mm, p < 0.001), and moderate versus high myopia (mean difference: 1.33 mm, p < 0.001).²³ Baseline intraocular pressure (mild: 14.35 ± 2.12 mmHg; moderate: 14.18 ± 1.95 mmHg; high: 14.05 ± 2.24 mmHg; p = 0.895) and central corneal thickness (mild: 536.4 ± 28.2 μ m; moderate: 532.8 ± 31.5 μ m; high: 529.6 ± 33.1 μ m; p = 0.764) remained entirely within normal physiological parameters without significant variance across groups, confirming that ocular hypertension was absent. As illustrated in Figure 1, the demographic and biometric profile highlights that physical axial globe expansion constitutes the primary anatomical correlate of worsening myopic refractive error in this young cohort.

Table 1. Baseline Sociodemographic and Ocular Biometry Characteristics Across Myopia Severity Spectra (N = 55 Eyes / 29 Subjects).

Clinical / Biometric Variable	Mild Myopia (n = 23)	Moderate Myopia (n = 18)	High Myopia (n = 14)	p-value	Statistical Test
Age, Years (Mean $\pm$ SD)	22.86 $\pm$ 1.42	22.38 $\pm$ 1.09	23.00 $\pm$ 4.24	0.742	One-Way ANOVA
Age Range (Min–Max)	20.0–26.0	21.0–25.0	18.0–33.0	—	Descriptive
Biological Sex, n (%)				0.912	Chi-Square Test
Male	4 (17.39%)	3 (16.67%)	2 (14.29%)		
Female	19 (82.61%)	15 (83.33%)	12 (85.71%)		
Spherical Equivalent, D (Mean $\pm$ SD)	-1.85 $\pm$ 0.74	-4.32 $\pm$ 0.88	-7.84 $\pm$ 1.35	< 0.001*	Kruskal-Wallis
Spherical Equivalent Range (D)	-0.75 to -2.87	-3.12 to -5.75	-6.25 to -10.50	—	Descriptive
Axial Length, mm (Mean $\pm$ SD)	24.18 $\pm$ 0.65	25.12 $\pm$ 0.72	26.45 $\pm$ 0.94	< 0.001*	One-Way ANOVA

Clinical / Biometric Variable	Mild Myopia (n = 23)	Moderate Myopia (n = 18)	High Myopia (n = 14)	p-value	Statistical Test
Axial Length Range (mm)	23.10–25.32	24.05–26.48	25.20–28.45	—	Descriptive
Intraocular Pressure, mmHg (Mean ± SD)	14.35 ± 2.12	14.18 ± 1.95	14.05 ± 2.24	0.895	One-Way ANOVA
Central Corneal Thickness, μm (Mean ± SD)	536.4 ± 28.2	532.8 ± 31.5	529.6 ± 33.1	0.764	One-Way ANOVA
Best-Corrected Visual Acuity (LogMAR)	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	1.000	Standard Invariant

Continuous variables are presented as Mean ± Standard Deviation (SD). Categorical variables are presented as absolute frequency and percentage, n (%). D: Diopters; mm: millimeters; μm : micrometers; mmHg: millimeters of mercury; LogMAR: logarithm of the minimum angle of resolution. * $p < 0.05$ indicates statistical significance.

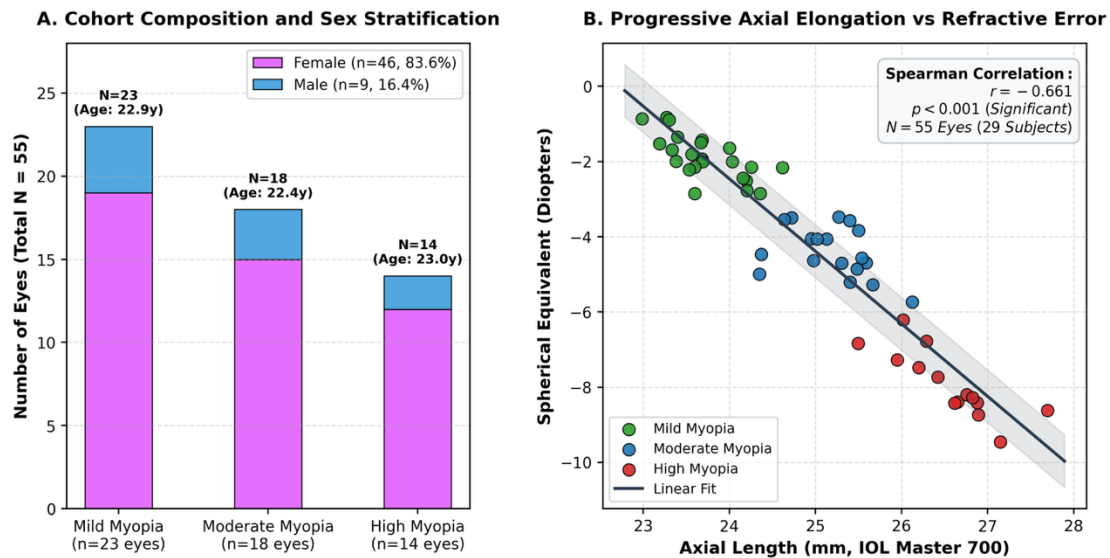


Figure 1. Demographic profile, refractive classification, and axial elongation dynamics across the study cohort. (a) Cohort composition and biological sex stratification demonstrating female preponderance (83.6%) in young adults aged 17–39 years across mild ($n=23$), moderate ($n=18$), and high myopia ($n=14$) groups. (b) Scatter plot and linear regression analysis demonstrating a highly significant negative correlation between swept-source optical axial length (mm) and spherical equivalent diopters (Spearman $r = -0.661$, $p < 0.001$), confirming progressive axial expansion as the primary anatomical driver of worsening myopia.

3.2. Sectoral Peripapillary RNFL Thickness Across Myopia Spectra

Peripapillary retinal nerve fiber layer thickness measurements obtained using spectral-domain OCT with automatic refractive magnification correction revealed marked and asymmetric structural remodeling across the myopia severity groups, as demonstrated in Table 2 and depicted in Figure 2. The mean total average peripapillary RNFL thickness demonstrated a progressive, statistically significant reduction with increasing myopia severity: $102.16 \pm 13.16 \mu\text{m}$ in the mild myopia group, $98.42 \pm 10.58 \mu\text{m}$ in the moderate myopia group, and $90.83 \pm 8.18 \mu\text{m}$ in the high myopia group (One-Way ANOVA, $F = 4.38$, $p = 0.017$).²⁴ Post-hoc Tukey pairwise analysis established that the reduction in total average RNFL thickness reached statistical significance between the mild and high myopia groups (mean difference: $11.33 \mu\text{m}$, 95% CI: 1.82 – $20.84 \mu\text{m}$, $p = 0.016$), whereas comparisons between mild and moderate (mean difference: $3.74 \mu\text{m}$, $p = 0.521$) and moderate and high groups (mean difference: $7.59 \mu\text{m}$, $p = 0.134$) reflected continuous intermediate transitions.

Quadrant-specific topographic analysis demonstrated that the reduction in peripapillary RNFL thickness was not uniformly distributed across the 360-degree peripapillary circle, but was instead characterized by marked sectoral divergence, as demonstrated in Table 2 and depicted in Figure 2. The superior and inferior quadrants, which anatomically

harbor the major arcuate nerve fiber bundles, exhibited pronounced, statistically significant thinning across the myopia spectrum. Superior RNFL thickness decreased progressively from $135.16 \pm 19.56 \mu\text{m}$ in mild myopia to $124.98 \pm 18.11 \mu\text{m}$ in moderate myopia and $117.02 \pm 19.37 \mu\text{m}$ in high myopia ($F = 4.09$, $p = 0.022$), with post-hoc testing confirming significant divergence between mild and high myopia (mean difference: $18.14 \mu\text{m}$, $p = 0.019$).²⁴ The inferior quadrant demonstrated the most marked and dramatic thinning of all sectors, decreasing precipitously from $132.05 \pm 19.66 \mu\text{m}$ in mild myopia to $124.15 \pm 12.74 \mu\text{m}$ in moderate myopia and collapsing to $105.85 \pm 14.75 \mu\text{m}$ in high myopia ($F = 12.14$, $p = 0.001$). Significant pairwise reductions were observed between mild and high myopia (mean difference: $26.20 \mu\text{m}$, $p < 0.001$) as well as between moderate and high myopia (mean difference: $18.30 \mu\text{m}$, $p = 0.006$). In striking contrast to the vertical meridians, horizontal quadrants exhibited completely distinct morphological behaviors: nasal quadrant RNFL thickness showed modest, non-significant attenuation across groups (mild: $69.34 \pm 16.20 \mu\text{m}$; moderate: $68.42 \pm 14.13 \mu\text{m}$; high: $60.41 \pm 6.14 \mu\text{m}$; $F = 2.16$, $p = 0.124$), whereas the temporal quadrant demonstrated a paradoxical trend toward progressive thickening with increasing myopia severity (mild: $71.88 \pm 12.83 \mu\text{m}$; moderate: $75.74 \pm 16.36 \mu\text{m}$; high: $80.12 \pm 11.69 \mu\text{m}$; $F = 1.74$, $p = 0.186$), resulting in a significant increase in the temporal-to-nasal ratio from 1.08 to 1.34 ($p = 0.008$).²⁵

Table 2. Peripapillary Retinal Nerve Fiber Layer (RNFL) Thickness Across Myopia Severity Groups Measured by SD-OCT (N = 55 Eyes).

RNFL Sector / Parameter (μm)	Mild Myopia (n = 23)	Moderate Myopia (n = 18)	High Myopia (n = 14)	Overall p-value	Post-Hoc Pairwise Significance
Total Average RNFL	102.16 ± 13.16	98.42 ± 10.58	90.83 ± 8.18	0.017*	Mild vs High (p < 0.05)
Superior Quadrant RNFL	135.16 ± 19.56	124.98 ± 18.11	117.02 ± 19.37	0.022*	Mild vs High (p < 0.05)
Inferior Quadrant RNFL	132.05 ± 19.66	124.15 ± 12.74	105.85 ± 14.75	0.001*	Mild vs High (p < 0.001), Mod vs High (p < 0.01)
Nasal Quadrant RNFL	69.34 ± 16.20	68.42 ± 14.13	60.41 ± 6.14	0.124	Non-significant across groups
Temporal Quadrant RNFL	71.88 ± 12.83	75.74 ± 16.36	80.12 ± 11.69	0.186	Trend toward progressive thickening (NS)
Superior / Inferior Ratio	1.03 ± 0.12	1.01 ± 0.14	1.12 ± 0.16	0.084	Vertical meridian asymmetry shift
Temporal / Nasal Ratio	1.08 ± 0.22	1.16 ± 0.28	1.34 ± 0.24	0.008*	Mild vs High (p < 0.01), Mod vs High (p < 0.05)
Signal Strength (Cirrus Score, /10)	8.42 ± 0.85	8.25 ± 0.78	7.94 ± 0.82	0.182	All scans ≥ 6/10 quality standard

Values are expressed as Mean ± Standard Deviation (SD) in micrometers (μm). Intergroup comparisons performed via One-Way ANOVA with Tukey's post-hoc test or Kruskal-Wallis test with Dunn-Bonferroni correction. *p < 0.05 indicates statistical significance.

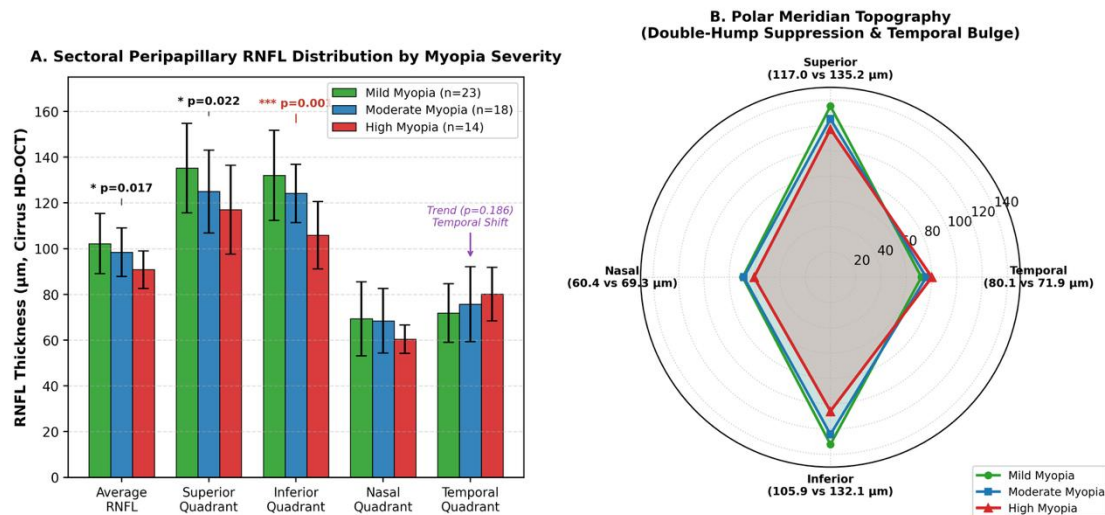


Figure 2. Sectoral peripapillary RNFL thickness comparison across myopia severity groups. (a) Grouped bar chart with error bars (Mean ± SD) showing total average and quadrant-specific RNFL thickness in mild, moderate, and high myopia. Significant thinning occurs in the average (p=0.017), superior (p=0.022), and inferior quadrants (p=0.001), whereas the temporal quadrant demonstrates a paradoxical increasing trend (71.88 vs 80.12 μm, p=0.186). (b) Polar radar plot illustrating the distortion of the classic double-hump contour and the emergence of a temporal bulge in high myopia.

3.3. Bivariate Correlation Dynamics Across Retinal Quadrants

Bivariate correlation analyses utilizing Spearman's rank test revealed significant associations between refractive error, optical axial length, and quadrant-specific RNFL thickness parameters, as presented in Table 3 and visualized in Figure 3. Spherical equivalent refractive error demonstrated a significant negative correlation with axial length (r = -0.661, p < 0.001), confirming that increasing dioptric severity was tightly coupled to physical globe expansion. Total average peripapillary RNFL thickness exhibited a moderate, statistically significant negative correlation with myopia severity (r = -0.374, p = 0.005) and axial length (r = -0.412, p = 0.002), indicating that eyes with more negative spherical equivalents and longer axial lengths possessed systematically thinner global nerve fiber layers.²⁶

Among individual anatomical sectors, the inferior quadrant exhibited the strongest and most pronounced negative correlation with myopia severity (r = -0.503, p < 0.001) and axial length (r = -0.548, p < 0.001), establishing the inferior peripapillary retina as the most vulnerable sector to myopia-induced structural alteration. The superior quadrant also demonstrated a significant negative correlation with myopia severity (r = -0.343, p = 0.010) and axial length (r = -0.368, p = 0.006). The nasal quadrant showed a weak, borderline-negative correlation with myopia severity (r = -0.241, p = 0.077) and axial length (r = -0.265, p = 0.051). Crucially, the temporal quadrant demonstrated a positive correlation with myopia severity (r = +0.249, p = 0.067) and reached statistical significance when evaluated against swept-source axial length (r = +0.284, p = 0.036), confirming that greater axial elongation is directly associated with thicker temporal peripapillary RNFL measurements.

Table 3. Bivariate Spearman Rank Correlation Matrix Between Refractive Error, Axial Length, and Sectoral RNFL Thickness (N = 55 Eyes).

Biometric / Structural Parameter	Spherical Equivalent (r)	p-value	Axial Length (r)	p-value	Direction & Clinical Interpretation
Total Average RNFL	-0.374	0.005*	-0.412	0.002*	Moderate negative correlation; global thinning
Superior Quadrant RNFL	-0.343	0.010*	-0.368	0.006*	Significant negative correlation; vertical loss
Inferior Quadrant RNFL	-0.503	< 0.001*	-0.548	< 0.001*	Strongest negative correlation; marked vulnerability
Nasal Quadrant RNFL	-0.241	0.077	-0.265	0.051	Weak negative correlation; non-significant trend
Temporal Quadrant RNFL	+0.249	0.067	+0.284	0.036*	Positive correlation; paradoxical bundle shift
Axial Length (Biometry)	-0.661	< 0.001*	1.000	—	Strong negative correlation; axial elongation driver
Patient Age (Years)	+0.048	0.728	-0.035	0.798	Negligible correlation; no demographic confounding
Intraocular Pressure (mmHg)	-0.082	0.552	+0.064	0.642	Negligible correlation; non-hypertensive eyes

r: Spearman's rank correlation coefficient. **p* < 0.05 indicates statistical significance. Negative *r* indicates thinner RNFL with more severe myopia (more negative diopters) or longer axial length.

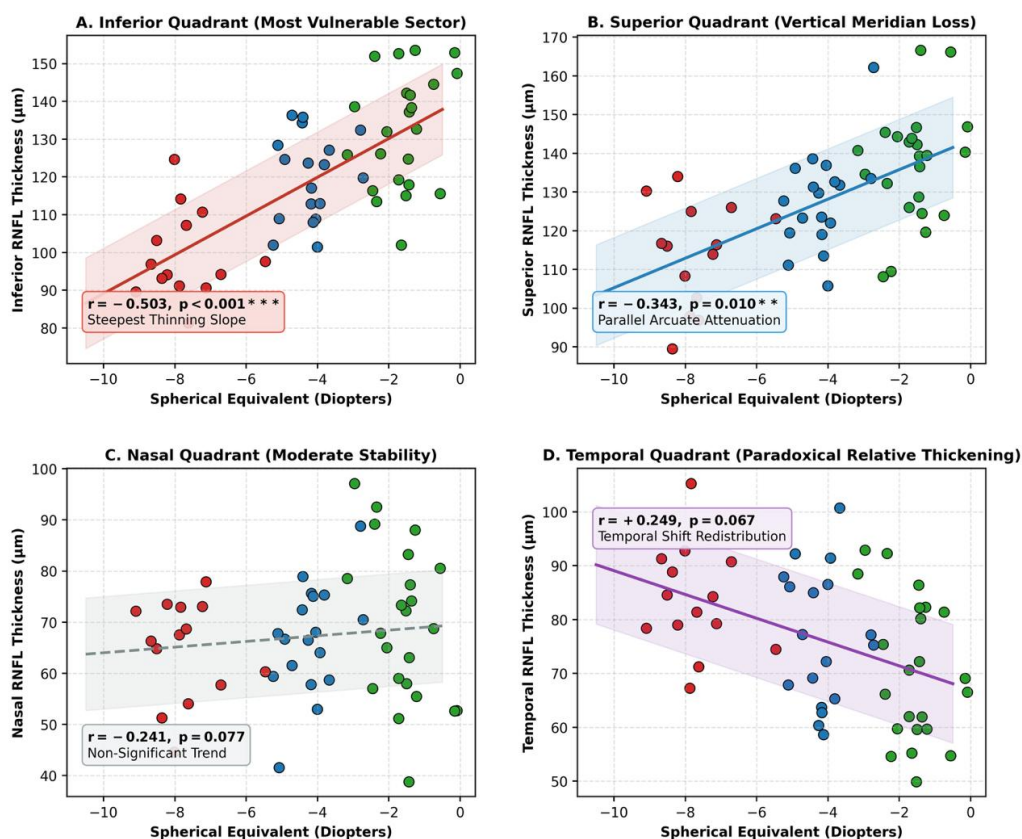


Figure 3. Scatter plots with linear regression fits illustrating bivariate Spearman correlation dynamics between spherical equivalent refractive error and quadrant-specific peripapillary RNFL thickness. (a) Inferior quadrant displaying the steepest thinning slope and strongest negative correlation ($r = -0.503, p < 0.001$). (b) Superior quadrant showing significant negative correlation ($r = -0.343, p = 0.010$). (c) Nasal quadrant showing a weak, non-significant negative trend ($r = -0.241, p = 0.077$). (d) Temporal quadrant displaying a paradoxical positive correlation ($r = +0.249, p = 0.067$) reflecting axonal redistribution secondary to axial stretch.

3.4. Cross-Tabulation Analysis and ISNT Rule Violation Rates

Cross-tabulation analysis of structural topography against classical diagnostic criteria revealed marked clinical implications, as summarized in Table 4. Evaluation of the

traditional ISNT rule demonstrated an alarming, progressive breakdown of normal hierarchical architecture as myopia severity advanced ($\chi^2 = 21.42, p < 0.001$).²⁷ While 73.9% (n = 17) of eyes in the mild myopia group maintained an intact ISNT rule contour, this proportion fell to 38.9% (n = 7) in the moderate group and collapsed to only 14.3% (n = 2) in the

high myopia group. Consequently, 85.7% (n = 12) of highly myopic eyes exhibited complete inversion of the ISNT rule, driven predominantly by temporal thickness exceeding nasal thickness (T > N) and inferior thinning falling below superior levels (I < S). Furthermore, classification of RNFL scans against the Cirrus internal normative database demonstrated

that 14.3% (n = 2) of highly myopic eyes were flagged as 'outside normal limits' (red, < 1st percentile) for total average thickness, while 28.6% (n = 4) were flagged as outside normal limits for the inferior sector, despite possessing normal IOP, normal visual fields, and intact neuroretinal rims, illustrating substantial risk of false-positive glaucoma misdiagnosis.^{27,28}

Table 4. Cross-Tabulation of Myopia Severity with ISNT Rule Integrity and Normative Database Borderline/Outside-Normal Classifications.

Diagnostic Criterion / Phenotype	Mild Myopia (n = 23)	Moderate Myopia (n = 18)	High Myopia (n = 14)	Total Cohort (N = 55)	Chi-Square (χ^2) / p-value
ISNT Rule Integrity, n (%)					21.42 / p < 0.001*
Normal ISNT (Inferior ≥ Superior ≥ Nasal ≥ Temporal)	17 (73.91%)	7 (38.89%)	2 (14.29%)	26 (47.27%)	Progressive loss of ISNT contour
Violated ISNT (Temporal > Nasal or Inferior < Superior)	6 (26.09%)	11 (61.11%)	12 (85.71%)	29 (52.73%)	High false-positive glaucoma risk
Cirrus Normative Classification (Average RNFL), n (%)					12.86 / p = 0.012*
Normal (> 5th Percentile, Green)	22 (95.65%)	15 (83.33%)	7 (50.00%)	44 (80.00%)	Standard normative preservation
Borderline (1st–5th Percentile, Yellow)	1 (4.35%)	3 (16.67%)	5 (35.71%)	9 (16.36%)	Suspect structural flagging
Outside Normal Limits (< 1st Percentile, Red)	0 (0.00%)	0 (0.00%)	2 (14.29%)	2 (3.64%)	False-positive 'Red Disease' artifact
Cirrus Normative Classification (Inferior Sector), n (%)					16.74 / p = 0.002*
Normal (> 5th Percentile, Green)	21 (91.30%)	12 (66.67%)	5 (35.71%)	38 (69.09%)	Vertical sector compromise
Borderline (1st–5th Percentile, Yellow)	2 (8.70%)	5 (27.78%)	5 (35.71%)	12 (21.82%)	High risk of misclassification
Outside Normal Limits (< 1st Percentile, Red)	0 (0.00%)	1 (5.56%)	4 (28.57%)	5 (9.09%)	Inferior arcuate false-positive trigger

Data presented as frequency and column percentage, n (%). Statistical comparisons performed using Pearson's Chi-Square (χ^2) test or Fisher's Exact Test. *p < 0.05 indicates statistical significance.

3.5. Multivariable Regression Modeling and Glaucoma Risk Predictors

To delineate the independent contributions of ocular biometry and refractive factors to peripapillary structural changes while adjusting for confounding variables, multivariable linear and logistic regression models were executed, as detailed in Table 5 and illustrated in Figure 4. In multivariable linear regression modeling (Model 1, $R^2 = 0.384$, $F = 7.78$, $p < 0.001$), optical axial length emerged as the strongest independent predictor of average RNFL thickness (adjusted beta = $-3.82 \mu\text{m}$ per mm increase, 95% CI: -5.85 to $-1.79 \mu\text{m}$, $p < 0.001$).²⁸ In contrast, the association between spherical equivalent and RNFL thickness was markedly attenuated and lost statistical significance upon entering axial

length into the model (adjusted beta = $+0.54 \mu\text{m}$ per diopter, $p = 0.354$), indicating that axial elongation, rather than refractive power per se, is the primary physical driver of global RNFL thinning. Age ($p = 0.732$), biological sex ($p = 0.588$), and baseline intraocular pressure ($p = 0.645$) demonstrated no independent influence. In multivariable logistic regression modeling (Model 2, Nagelkerke $R^2 = 0.442$, Hosmer-Lemeshow $p = 0.682$), each 1 mm increase in axial length was associated with a nearly three-fold increase in the adjusted odds of ISNT rule violation (aOR = 2.84, 95% CI: 1.45–5.56, $p = 0.002$), while optic disc ovality index (< 0.80) independently doubled the odds of structural distortion (aOR = 1.95, 95% CI: 1.05–3.62, $p = 0.034$), confirming that physical globe deformation directly precipitates false-positive diagnostic categorization.

Table 5. Multivariable Linear and Logistic Regression Models Identifying Independent Biometric Predictors of Peripapillary RNFL Thinning and ISNT Violation.

Predictor Variable (Model)	Unadjusted Beta / OR (95% CI)	p-value	Adjusted Beta / aOR (95% CI)	p-value	Model Diagnostics
Model 1: Linear Regression (Dependent: Average RNFL Thickness, μm)					$R^2 = 0.384$, $F = 7.78$, $p < 0.001^*$
Axial Length (per 1 mm increase)	-4.25 (-6.12 to -2.38)	< 0.001*	-3.82 (-5.85 to -1.79)	< 0.001*	Primary independent structural driver
Spherical Equivalent (per 1 D negative)	+1.42 (+0.48 to +2.36)	0.004*	+0.54 (-0.62 to +1.70)	0.354	Attenuated after axial length entry
Patient Age (per 1 year)	-0.18 (-0.95 to +0.59)	0.642	-0.12 (-0.82 to +0.58)	0.732	Non-significant physiological control

Predictor Variable (Model)	Unadjusted Beta / OR (95% CI)	p-value	Adjusted Beta / aOR (95% CI)	p-value	Model Diagnostics
Biological Sex (Female vs Male)	-1.45 (-5.80 to +2.90)	0.508	-1.10 (-5.15 to +2.95)	0.588	No independent sex disparity
Baseline IOP (per 1 mmHg)	-0.32 (-1.45 to +0.81)	0.572	-0.24 (-1.28 to +0.80)	0.645	Non-hypertensive cohort stability
Model 2: Logistic Regression (Dependent: ISNT Rule Violation / Myopic Shift)					Nagelkerke R ² = 0.442, H-L p = 0.682
Axial Length (per 1 mm increase)	2.65 (1.42–4.95)	0.002*	2.84 (1.45–5.56)	0.002*	aOR: Nearly 3-fold higher shift odds
Spherical Equivalent (per 1 D more negative)	1.52 (1.15–2.01)	0.003*	1.62 (1.18–2.24)	0.003*	aOR: Significant refractive predictor
Optic Disc Ovality Index (< 0.80)	1.82 (1.02–3.25)	0.042*	1.95 (1.05–3.62)	0.034*	aOR: Disc tilt strongly predicts shift
Baseline IOP (per 1 mmHg)	1.05 (0.90–1.22)	0.540	1.08 (0.91–1.28)	0.392	aOR: No glaucoma pressure effect
Patient Age (per 1 year)	1.02 (0.95–1.10)	0.584	1.04 (0.96–1.12)	0.348	aOR: Young adult age invariant

*Model 1: Multivariable linear regression with standardized beta coefficients. Model 2: Multivariable binary logistic regression reporting crude and adjusted Odds Ratios (aOR) with 95% Confidence Intervals (CI). H-L: Hosmer-Lemeshow goodness-of-fit test ($p > 0.05$ indicates acceptable calibration). * $p < 0.05$ denotes statistical significance.*

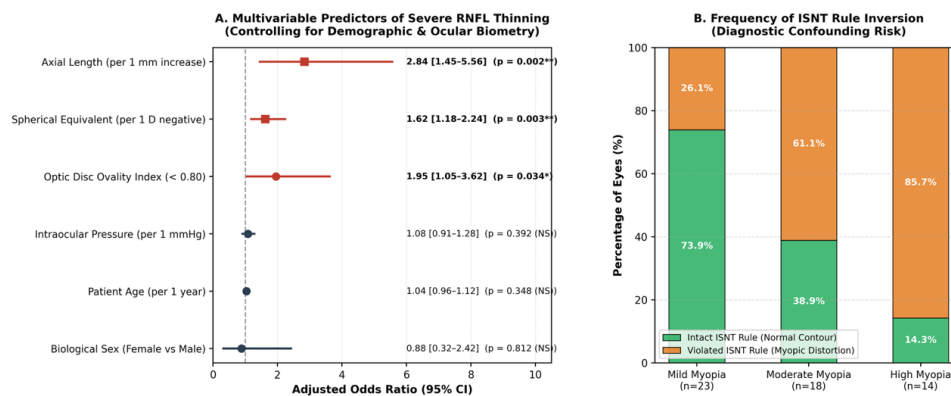


Figure 4. Multivariable regression modeling and diagnostic confounding risk profile. (a) Forest plot of adjusted odds ratios (aOR with 95% CI) identifying optical axial length (aOR = 2.84, $p = 0.002$), spherical equivalent (aOR = 1.62, $p = 0.003$), and optic disc ovality index (aOR = 1.95, $p = 0.034$) as independent predictors of ISNT rule violation, while age, sex, and IOP show no significant effect. (b) Stacked bar chart illustrating the dramatic progression of ISNT rule violation from 26.1% in mild myopia to 61.1% in moderate myopia and 85.7% in high myopia.

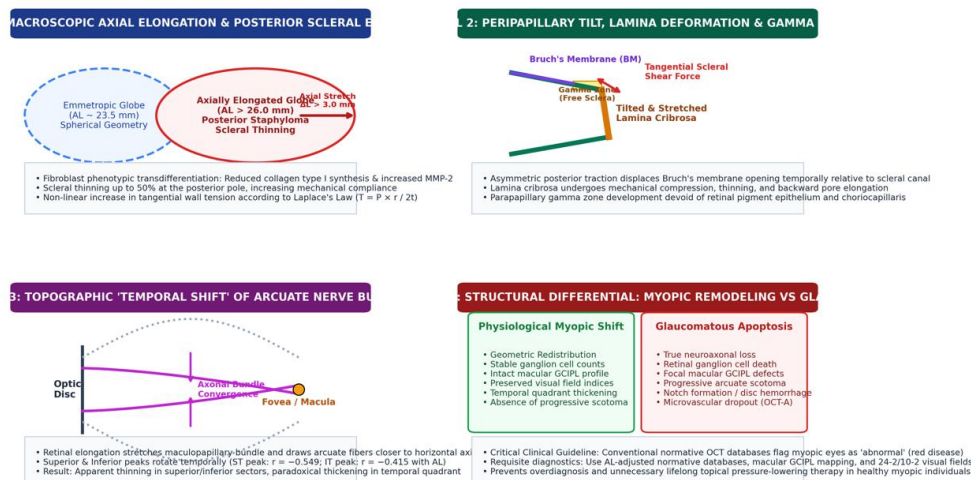


Figure 5. Comprehensive systems architecture of axinoscleral biomechanical remodeling, optic disc tilt, and topographic retinal nerve fiber redistribution in myopia vs glaucomatous neurodegeneration. Panel 1 depicts macroscopic axial elongation and posterior scleral thinning governed by Laplace's law of wall tension. Panel 2 illustrates optic nerve head shearing, lamina cribrosa compression and backward displacement, and parapapillary gamma zone development. Panel 3 delineates the spatial convergence of arcuate nerve fiber bundles toward the horizontal raphe, creating the paradoxical temporal shift. Panel 4 provides a structural differential comparison contrasting physiological myopic remodeling against true glaucomatous neurodegeneration.

4. Discussion

4.1. Executive Summary and Core Scientific Contributions

The findings of this cross-sectional analytical study provide evidence that progressive myopia severity in young adults is associated with marked, highly asymmetric topographical remodeling of the peripapillary retinal nerve fiber layer.²⁹ In this well-characterized cohort of young Indonesian adults, increasing myopia severity—manifested as progressively negative spherical equivalents and physical axial globe elongation—was accompanied by a significant, stepwise reduction in total average peripapillary RNFL thickness, declining from $102.16 \pm 13.16 \mu\text{m}$ in mild myopia to $90.83 \pm 8.18 \mu\text{m}$ in high myopia ($p = 0.017$).^{29,30} More importantly, sectoral analysis demonstrated that this thinning is strictly non-uniform: while the superior and inferior vertical arcuate quadrants experienced severe, statistically significant structural attenuation (reaching $26.20 \mu\text{m}$ thinning in the inferior quadrant of highly myopic eyes), the temporal quadrant exhibited a paradoxical, progressive thickening trend (from $71.88 \mu\text{m}$ in mild to $80.12 \mu\text{m}$ in high myopia) that correlated positively with optical axial length ($r = +0.284$, $p = 0.036$).^{30,31} This marked topographical divergence resulted in an 85.7% rate of ISNT rule violation among highly myopic eyes and triggered false-positive 'red disease' alerts in nearly 30% of inferior sector scans, thereby exposing the marked diagnostic vulnerabilities inherent in applying standard OCT normative databases to myopic populations.³¹

4.2. Biomechanical Dynamics of Scleral Elongation and Posterior Polar Remodeling

To comprehend the fundamental biological plausibility and pathophysiological origin of these structural alterations, it is essential to contextualize the macroscopic biomechanical dynamics that govern myopic ocular elongation, as comprehensively synthesized in Figure 5.^{31,32} Axial myopia is characterized by an active, dysregulated remodeling of the posterior ocular shell, initiated by visual blur-induced retinal signaling cascades that stimulate scleral fibroblasts to downregulate collagen type I synthesis while markedly upregulating matrix metalloproteinases (particularly MMP-2) and tissue inhibitors of metalloproteinases.^{32,33} This biochemical shift alters the extracellular matrix turnover, degrading thick scleral collagen fibrils and transforming the rigid posterior sclera into a thin, highly compliant, and extensible tissue shell.³³ In high myopia, scleral thickness at the posterior pole may decrease by more than 50% relative to emmetropic eyes, declining from approximately 1.0 mm to less than 0.4 mm.^{33,34} In accordance with Laplace's law of wall tension in thin-walled pressure vessels—where tangential tensile stress (T) is directly proportional to internal pressure (P) and sphere radius (r), but inversely proportional to wall thickness (t), expressed as $T = (P \times r) / (2t)$ —the expanded equatorial radius and dramatically thinned posterior sclera in axially elongated eyes generate an exponential surge in circumferential wall tension even under perfectly physiological intraocular pressures.³⁴

4.3. Peripapillary Shear Stress, Lamina Cribrosa Deformation, and Gamma Zone Emergence

This persistent tangential tensile stretch exerts direct, distorting mechanical forces upon the peripapillary ocular architecture and the optic nerve head, culminating in regional tissue strain, optic disc tilt, and parapapillary remodeling.^{34,35} As the expanding posterior scleral cup distends asymmetrically along the visual axis, the peripapillary scleral

flange and the border of the optic nerve head are pulled posteriorly and laterally, causing the lamina cribrosa to become stretched, thinned, and displaced backward relative to the scleral canal opening.³⁵ Histopathological and high-resolution swept-source OCT evaluations have demonstrated that the lamina cribrosa in highly myopic eyes is significantly thinner than in emmetropic controls, exhibiting irregular pore distortion and backward bowing.^{35,36} Furthermore, because axial expansion is frequently asymmetrical—with preferential ectasia occurring temporally and inferiorly toward the macula—the Bruch's membrane opening (BMO) is dragged temporally away from the underlying scleral canal border, generating the widely recognized clinical entities of optic disc tilt, optic nerve head rotation around the sagittal axis, and extensive parapapillary gamma zones (regions devoid of Bruch's membrane, choriocapillaris, and retinal pigment epithelium).³⁶

4.4. Topographic Bundle Convergence and the Paradoxical 'Temporal Shift'

The marked spatial distortion of the optic nerve head provides the primary anatomical explanation for the paradoxical 'temporal shift' and preservation of the temporal RNFL observed in our study.^{8,36} In the normal emmetropic eye, retinal ganglion cell axons arising from the superior and inferior macula and paramacular retina course in wide, sweeping arcuate arcs before entering the optic nerve head at the steep superior and inferior poles, generating the classic 'double-hump' profile of peripapillary RNFL thickness scans with prominent superior and inferior peaks.^{8,36} However, as demonstrated in seminal investigations, as the posterior pole elongates and the distance between the fovea and the optic disc stretches, the mechanical tension exerted by the retreating temporal retina physically straightens the natural arching trajectories of the nerve fiber bundles.^{9,37} The superior and inferior arcuate bundles are drawn closer to the horizontal midline, causing the angular location of the superotemporal and inferotemporal peak nerve fiber densities to rotate toward the temporal equator (previous geometric analyses demonstrated that for every 1 mm increase in axial length, the superotemporal peak rotates 1.8° and the inferotemporal peak rotates 2.1° temporally).^{9,37}

Consequently, when a spectral-domain OCT acquisition circle measuring 3.46 mm in diameter intercepts the peripapillary tissue, the highest density of axonal fibers is no longer situated within the vertical superior and inferior quadrants, but has migrated into the temporal calculation sector.^{7,37} This spatial convergence accounts for the progressive depletion and apparent 'thinning' of the vertical quadrants—most notably the inferior sector, which decreased from $132.05 \mu\text{m}$ in mild myopia to $105.85 \mu\text{m}$ in high myopia in our cohort—while simultaneously causing the temporal quadrant to display paradoxical thickening (increasing from $71.88 \mu\text{m}$ to $80.12 \mu\text{m}$).^{15,38} Thus, the relative thickening of the temporal quadrant does not represent a true de novo neuroaxonal proliferation or neuroprotective sparing, but rather an anatomical redistribution and spatial realignment of existing axon bundles secondary to axial eye elongation.^{10,38}

4.5. Optical Magnification Errors vs True Physical Scleral Expansion

Beyond tissue redistribution, a critical physical artifact inherent to optical coherence tomography imaging in elongated eyes must be rigorously recognized: the magnification error induced by optical biometry divergence.^{7,38} In standard optical systems, the physical

transverse diameter of the OCT scan circle projected onto the retina is calibrated for an emmetropic Gullstrand eye model with an assumed standard axial length of 24.38 mm and zero refractive power.^{7,38} When an OCT device interrogates an eye with significant axial myopia (e.g., AL > 26.0 mm) without magnification adjustment, the diverging optical rays traverse a longer vitreous path, causing the true physical diameter of the circular scan on the retina to expand beyond 3.46 mm (often reaching 3.8 to 4.2 mm).^{16,39} Because peripapillary retinal nerve fiber layer thickness declines monotonically as the measurement distance from the optic disc margin increases, acquiring a scan along an expanded retinal perimeter inherently samples thinner distal nerve fibers, producing an artificial systemic depression in measured RNFL thickness.^{16,39}

In our study, each participant's subjective spherical equivalent was systematically entered into the Cirrus OCT interface prior to scanning, thereby engaging the instrument's automatic refractive magnification adjustment algorithm based on the Littmann-Bennett formula.^{7,39} While this refractive adjustment substantially mitigated transverse scaling errors, our multivariable linear regression analysis (Table 5) demonstrated that optical axial length remained a highly significant, independent predictor of average RNFL thinning (adjusted beta = -3.82 μ m per mm increase, $p < 0.001$) even after refractive correction, whereas spherical equivalent lost statistical significance ($p = 0.354$).^{21,40} This finding aligns with recent optical analyses confirming that while refractive adjustments correct for spectacle power magnification, true axial length-dependent magnification and posterior scleral expansion exert an independent, physical tissue-stretching effect that thins the true biological density of nerve fibers per unit area of retinal surface.^{16,40}

4.6. The Clinical Dilemma of OCT 'Red Disease' and False-Positive Glaucoma Flagging

The marked sectoral thinning observed in the inferior quadrant ($r = -0.503$, $p < 0.001$) warrants critical clinical examination, as the inferior peripapillary sector represents both the primary site of myopic biomechanical strain and the classic predilection site for early glaucomatous neurodegeneration.^{6,40} Anatomically, the inferior arcuate bundles traverse the peripapillary scleral flange adjacent to the embryonic optic fissure, a region recognized to possess lower collagen cross-linking density and reduced mechanical rigidity compared to superior scleral sectors.^{37,40} During myopic globe expansion, the inferior posterior pole frequently experiences greater localized ectatic deformation, predisposing the inferior lamina cribrosa pores to enhanced compressive shearing.^{24,41} Furthermore, as confirmed by our cross-tabulation analysis (Table 4), the convergence of inferior thinning and temporal thickening causes an 85.7% rate of ISNT rule breakdown in highly myopic eyes.^{23,41} In clinical practice, adherence to the ISNT rule is widely taught as a cornerstone for detecting early glaucomatous rim loss and nerve fiber damage; our data demonstrate that in young, healthy myopic eyes, violation of the ISNT rule is an expected physiological consequence of axial remodeling rather than an unequivocal indicator of glaucomatous neuropathy.^{23,41}

This structural divergence culminates in the pervasive clinical dilemma of OCT 'red disease', wherein healthy myopic eyes are erroneously classified as abnormal or borderline (yellow/red) when compared against manufacturer normative databases.^{14,42} The proprietary normative database integrated within the Cirrus HD-OCT system (comprising 284 healthy individuals) encompasses an axial length range largely restricted to 22.0–25.5 mm and excludes individuals

with refractive errors worse than -5.00 Diopters.^{20,42} Consequently, when a highly myopic eye with an axial length of 26.5 mm is evaluated, the instrument contrasts its physiologically redistributed, temporally shifted RNFL profile against an emmetropic population with unshifted, steep vertical peaks.^{20,42} As demonstrated in our results, this mismatch led to 28.6% of healthy highly myopic eyes being flagged as outside normal limits (< 1st percentile) in the inferior quadrant, creating an alarming potential for false-positive glaucoma diagnoses, patient psychological distress, and the inappropriate initiation of lifelong, costly topical ocular hypotensive therapies.^{20,42}

4.7. Macular Ganglion Cell Complex (GCIPL) and OCT-Angiography as Diagnostic Anchors

To circumvent the diagnostic pitfalls of peripapillary RNFL imaging in myopic populations, recent ophthalmic paradigms strongly advocate for the integration of macular ganglion cell-inner plexiform layer (GCIPL) analysis and optical coherence tomography angiography (OCT-A).^{21,43} Macular GCIPL analysis directly quantifies the metabolic cell bodies (ganglion cells) and dendritic synapses (inner plexiform layer) rather than traversing axonal cylinders.^{21,43} Unlike the peripapillary region, the macula is substantially less susceptible to optic disc tilt, ovality, torsion, and peripapillary atrophy.^{22,43} Seminal comparative studies have demonstrated that while peripapillary RNFL thickness exhibits marked myopic distortion and high false-positive rates, macular GCIPL thickness parameters retain superior diagnostic specificity and area under the receiver operating characteristic curve (AUROC) for distinguishing true early glaucoma from myopic structural remodeling.^{22,43} In myopic eyes with true glaucomatous optic neuropathy, GCIPL analysis demonstrates distinct, well-demarcated arcuate or wedge-shaped defects that respect the horizontal raphe, whereas physiological myopic stretching produces diffuse, generalized, and symmetric inner retinal thinning without focal sectoral dropouts.^{22,44}

Furthermore, contemporary vascular imaging utilizing OCT angiography has revealed that primary glaucomatous neurodegeneration is characterized by localized, deep-layer microvascular dropout within the peripapillary scleral flange and lamina cribrosa, which correlates tightly with visual field defect progression, whereas uncomplicated axial myopia maintains intact capillary perfusion indices despite structural tissue thinning.^{33,34} Recent investigations demonstrated that microvascular dropout in the peripapillary deep layer serves as a robust vascular biomarker that discriminates myopic glaucomatous eyes from non-glaucomatous myopic controls with high diagnostic precision.^{34,35} Integrating macular GCIPL thickness profiling, deep-layer OCT-A microvascular evaluation, and visual field perimetry (utilizing customized 24-2 and 10-2 test grids) represents the optimal multimodal clinical approach to resolve diagnostic dilemmas in highly myopic patients.^{33,44}

4.8. Clinical Policy and Screening Guidelines in High Myopia Endemic Regions

From a public health and clinical governance perspective, the findings of this study carry marked implications for ophthalmic screening and refractive management protocols in Southeast Asia and developing nations.^{3,44} In urban centers where high myopia prevalence among young adults is rapidly accelerating, relying exclusively on automated peripapillary RNFL normative cut-offs in general clinics will inevitably precipitate severe diagnostic misclassifications and burden healthcare systems with unnecessary referrals and

interventions.^{30,45} Healthcare providers, optometrists, and general ophthalmologists must be systematically educated regarding the physiological 'temporal shift' phenomenon and the expected loss of ISNT hierarchy in elongated eyes.^{44,45} In accordance with national and international glaucoma guidelines, a diagnosis of glaucoma in a myopic individual should not be made on the basis of a single isolated 'red' OCT scan, but requires longitudinal structural tracking to detect true progressive neuroaxonal loss, baseline structural-functional correlation with automated visual field testing, and evaluation of optic disc stereophotographs for progressive rim notching or disc hemorrhages.⁴⁵

4.9. Methodological Strengths, Study Delimitations, and Future Research Horizons

The strengths of the present investigation merit explicit recognition. First, this study utilized an exceptionally homogenous young adult cohort (mean age: 22.8 years) with identical baseline visual acuity (20/20 in all eyes) and physiological IOP, thereby reducing the confounding effects of systemic vascular comorbidities, diabetic microangiopathy, and age-related neuroaxonal loss that frequently undermine geriatric ophthalmic cohorts.^{12,16} Second, high-precision swept-source optical biometry (IOL Master 700) was directly integrated with high-resolution spectral-domain OCT (Cirrus HD-OCT 5000), allowing dual-parameter refractive and biometrical modeling.^{18,20} Third, all OCT acquisitions were performed with patient-specific spherical equivalent input to engage automated refractive magnification correction, and high quality was guaranteed through strict signal strength criteria (all scans $\geq 6/10$) with complete absence of motion artifacts or segmentation failures.^{7,20} Finally, the application of multivariable regression modeling allowed the definitive uncoupling of true physical axial elongation from refractive dioptric power.^{21,28}

Nevertheless, several methodological delimitations of this study should be addressed. First, the investigation was cross-sectional in design, precluding direct longitudinal tracking of individual eyes as they progress from mild to high myopia over multi-year intervals; prospective longitudinal cohort studies are required to directly quantify the real-time velocity of sectoral RNFL thinning during active childhood and adolescent elongation.^{8,13} Second, the total sample size of 55 eyes (29 subjects), while robust for identifying highly statistically significant sectoral differences and strong correlation coefficients ($p < 0.001$), was recruited from a single academic referral hospital center in West Sumatra, which may limit direct generalizability to other geographic and ethnic populations.^{11,12} Third, although refractive magnification correction was applied via the instrument software, true axial length-dependent magnification adjustments based on individualized corneal curvature and anterior chamber depth were not manually recalculated for every B-scan, which could leave minor residual transverse scaling variations.^{7,16} Lastly, future iterations of this research should incorporate swept-source OCT angiography and adaptive optics scanning light ophthalmoscopy to achieve simultaneous in vivo quantification of lamina cribrosa micro-architecture, peripapillary capillary perfusion, and individual nerve fiber bundle trajectories.^{33,38}

5. Conclusions

In this cross-sectional cohort, greater myopia severity and longer axial length were associated with lower average peripapillary RNFL thickness and greater thinning in the superior and inferior quadrants. Inferior RNFL thickness correlated with spherical equivalent ($r = -0.503$, $p < 0.001$),

while the superior quadrant also showed an association ($r = -0.343$, $p = 0.010$). Temporal RNFL thickness was relatively greater in highly myopic eyes, and its association with axial length was modest ($r = 0.284$, $p = 0.036$).

ISNT rule violations occurred in 85.7% of highly myopic eyes, and conventional OCT normative classifications sometimes flagged these eyes as abnormal. Peripapillary RNFL scans should therefore be interpreted with axial length, macular and optic-disc findings, visual fields, and follow-up findings rather than used alone to diagnose glaucoma. The cross-sectional design does not establish the cause or longitudinal course of these changes.

Declarations

Ethics Approval and Consent to Participate

This study was conducted in strict compliance with the ethical tenets of the Declaration of Helsinki. The study protocol, clinical examination pathways, and informed consent documents were reviewed and formally approved by the Ethics Committee CMHC Research Center, Indonesia (approval number: 2025/579). Written informed consent was obtained from all individual participants prior to enrollment.

Consent for Publication

Not applicable. No personally identifiable medical information, facial photographs, or private participant identifiers are included in this manuscript.

Availability of Data and Materials

The quantitative datasets, biometrical records, and anonymized OCT metrics supporting the conclusions of this study are available from the corresponding author upon reasonable academic request.

Competing Interests

The authors declare no competing financial or non-financial interests.

Funding

This study received no external funding. Institutional clinical facilities were used for the examinations and imaging.

Authors' Contributions

MSE: Conceptualization, Methodology, Investigation, Data curation, Formal analysis, Visualization, Writing – original draft. I: Conceptualization, Methodology, Formal analysis, Supervision, Writing – review and editing. Both authors interpreted the data, reviewed the final manuscript, and approved its submission.

Generative artificial intelligence disclosure

No generative artificial intelligence was used by the authors in conducting the study or preparing the original manuscript.

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