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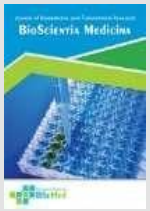

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Axial Length as the Primary Determinant of Refractive Severity in High Myopia: A Swept-Source OCT Analysis

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A B S T R A C T

Background: The escalating prevalence of high myopia represents a critical global health crisis, particularly in Southeast Asia. This pathological refractive status is driven by complex structural changes, primarily axial elongation, which predisposes the eye to sight-threatening complications. While the dominance of axial length in myopia is established, the compensatory role of the anterior segment—specifically whether the cornea or anterior chamber undergoes adaptive morphological changes to counteract elongation—remains a subject of debate. This study utilized high-resolution Swept-Source Optical Coherence Tomography (SS-OCT) to precisely quantify the relationship between Spherical Equivalent (SE) and ocular biometric parameters, isolating the specific contributions of posterior segment elongation versus anterior segment adaptation. **Methods:** This observational analytic study with a cross-sectional design was conducted at the Ophthalmology Clinic of Dr. M. Djamil General Hospital, Padang, Indonesia. The study recruited patients aged 18–40 years diagnosed with high myopia (SE defined as -6.00 Diopters or worse). Strict exclusion criteria were applied to eliminate confounding anterior segment pathologies. Ocular biometry was performed using the IOLMaster 700, which employs Swept-Source OCT technology for full-eye length tomography. We analyzed Axial Length (AL), Mean Keratometry (K), Central Corneal Thickness (CCT), and Anterior Chamber Depth (ACD). Statistical analysis utilized Pearson and Spearman correlation tests and linear regression modelling. **Results:** A total of 32 eyes from 32 high myopia patients were analyzed. The mean SE was -8.14 plus or minus 2.09 D, and the mean AL was 26.93 plus or minus 1.92 mm. We found a robust, statistically significant negative correlation between SE and AL (r = -0.86; p less than 0.001), confirming AL as the primary determinant of refractive severity. Notably, a moderate negative correlation was observed between AL and K (r = -0.41; p = 0.02), indicating a paradoxical corneal flattening in longer eyes. No significant correlations were found between SE and CCT or ACD. **Conclusion:** Axial elongation is the predominant structural mechanism driving high myopia in this cohort. The study identified a distinct compensatory response where the cornea flattens as the eye elongates; however, this emmetropization mechanism is insufficient to neutralize the profound refractive shift caused by posterior segment expansion. These findings suggest that high myopia is a disease of focal posterior scleral remodelling rather than global ocular expansion.

1. Introduction

The global ophthalmic landscape is currently undergoing a seismic shift, characterized by a myopia epidemic of unprecedented proportions.[1] What was once historically dismissed as a benign refractive error—easily corrected with spectacles or contact lenses—has now emerged as a primary driver of visual impairment and irreversible blindness on a global scale. This transition from a common optical inconvenience to a significant public health crisis is underscored by alarming epidemiological projections.[2] Data suggests that by the year 2050, half

of the world's population will be myopic, with a staggering 1 billion individuals progressing into the category of high myopia. This distinction is not merely academic; it represents a critical clinical threshold. While low to moderate myopia remains largely an optical issue, high myopia (typically defined as a Spherical Equivalent of -6.00 D or worse) is a pathological condition defined by profound structural alterations to the globe.[3]

High myopia is characterized by an excessive increase in axial length, which leads to the mechanical stretching of the eye's posterior segment. This elongation is not a benign expansion but a degenerative process that dramatically escalates the risk of sight-threatening complications.[4] As the globe stretches, the retina, choroid, and sclera are subjected to chronic mechanical stress, predisposing the eye to a suite of blinding pathologies. These include: (1) Myopic Maculopathy: A leading cause of vision loss where the central retina undergoes atrophic changes; (2) Posterior Staphyloma: An out-pouching of the posterior wall of the eye, indicating localized scleral weakness; (3) Rhegmatogenous Retinal Detachment: The thinning of the peripheral retina increases the risk of tears and subsequent detachment; and (4) Glaucoma: Highly myopic eyes often exhibit structural changes in the optic nerve head that make them more susceptible to glaucomatous damage.

To understand the development of high myopia, one must first examine the process of emmetropization. In a healthy, emmetropic eye, an active, vision-dependent mechanism regulates ocular growth to ensure that the eye's axial length perfectly matches its total optical power (derived from the cornea and crystalline lens). This homeostatic control ensures that incoming light is focused precisely on the retina. In cases of high myopia, this delicate homeostatic balance fails, leading to runaway axial elongation. The exact biological triggers for this failure are multifactorial, involving a complex interplay between genetic susceptibility and modern environmental pressures.[5] Key environmental drivers identified in recent literature include: (1) Reduced Outdoor Light Exposure: Lack of natural light is thought to disrupt the eye's growth signals; (2) Intense Near-Work Demands: Prolonged activities like reading or digital device use create a hyperopic defocus on the retina. These environmental inputs are believed to alter the levels of retinal neurotransmitters, specifically dopamine. Dopamine acts as a critical regulator of ocular growth; its reduction initiates a signaling cascade that reaches the sclera. This cascade triggers the release of matrix metalloproteinases (MMPs), which degrade the scleral extracellular matrix. The result is a biomechanically weakened sclera that stretches under the influence of normal intraocular pressure, leading to the characteristic elongation of the globe.[6]

A central question in modern myopia research is whether the eye attempts a rescue mechanism to counteract this pathological elongation.[7] Theoretical models of emmetropization suggest that as the eye lengthens, the anterior segment—specifically the cornea and the crystalline lens—should lose optical power by flattening. This loss of power would, in theory, move the focal point backward toward the retreating retina in an attempt to maintain clear vision. While this compensatory flattening is well-documented during the rapid growth phase of infancy, its persistence into adulthood, especially in the context of extreme axial lengths, remains a subject of intense debate. Researchers are divided on whether the cornea continues to adapt or if there is a physiological limit to this emmetropization rescue. Historically, investigating this relationship was difficult because older diagnostic tools, such as ultrasound or partial coherence interferometry (PCI), often produced inconsistent results. These technologies frequently struggled with poor signal-to-noise ratios in eyes with extreme axial lengths or dense cataracts, leading to measurement errors.

The introduction of Swept-Source Optical Coherence Tomography (SS-OCT) has revolutionized the field of ocular biometry.[8] Advanced devices like the IOLMaster 700 utilize a 1050 nm wavelength tunable

laser source. This longer wavelength offers several advantages over previous 800 nm spectral-domain systems: (1) Enhanced Penetration: It can penetrate dense cataracts and media opacities more effectively; (2) Visualization of the Foveal Pit: SS-OCT allows for a fixation check, where the clinician can visually confirm the measurement is taken along the visual axis (corneal vertex to fovea) rather than the anatomical axis; (3) Precision in Long Eyes: This technology provides highly repeatable and precise measurements of the entire ocular geometry, which is critical for patients with extreme axial lengths where alignment is often difficult due to posterior staphyloma. This leap in technology has provided a new opportunity to re-evaluate biometric correlations with a level of precision previously unattainable. It allows researchers to dissect the specific contributions of posterior segment elongation versus anterior segment adaptation with high confidence.[9]

Despite the alarming prevalence of myopia across Southeast Asia, detailed biometric profiling of Indonesian populations has remained notably sparse in international literature. Most normative datasets currently used in clinical practice are derived from East Asian (Chinese, Japanese) or Caucasian cohorts.[10] However, these datasets may not accurately reflect the biometric characteristics of the Malay-Indonesian ethnic group. Genetic variations in ocular dimensions mean that what is considered normative for a Caucasian eye may not apply to an Indonesian eye. There is a pressing need for high-resolution biometric data that specifically focuses on the high myopia phenotype within this population. Previous studies often aggregated mild, moderate, and high myopia into a single analysis, which can mask the unique structural changes that occur at the extreme end of the refractive spectrum. By strictly isolating the high myopia phenotype (SE -6.00 D or worse), researchers can investigate end-stage refractive adaptation and determine if anterior segment compensation hits a physiological limit as the eye reaches extreme lengths.

The shift in myopia research highlights that the condition is no longer just a matter of blurry vision. It is a structural disease of the globe. Current evidence points toward axial elongation as the primary driver of refractive severity, representing a disease of focal posterior scleral remodeling rather than a simple global expansion of the eye. While a compensatory corneal flattening mechanism has been identified, it appears insufficient to neutralize the profound refractive shift caused by the rapid expansion of the posterior segment. The stability of parameters like anterior chamber depth (ACD) in these eyes further supports the retro-equatorial elongation model, suggesting that the pathological stretching is localized behind the eye's equator. Understanding these biometric nuances through advanced technology like SS-OCT is essential for shifting clinical management from mere refractive correction to active interventions that target the underlying axial elongation.

This study introduces a distinct novelty by utilizing advanced SS-OCT technology to characterize a specific, homogeneous cohort of Indonesian young adults with high myopia. Unlike previous studies that often aggregated mild, moderate, and high myopia into a single analysis, this research strictly isolates the high myopia phenotype (SE -6.00 D or worse). This isolation allows for the specific investigation of end-stage refractive adaptation, determining whether anterior segment compensation hits a physiological limit in eyes with extreme elongation. Furthermore, this is one of the first studies to provide high-resolution biometric normative data for high myopes in the West Sumatra region. The primary aim of this study was to scientifically evaluate the dominance of Axial Length as the determinant of refractive severity and to investigate the presence and extent of compensatory corneal mechanisms in high myopia. Specifically, we aimed to correlate spherical equivalent with axial length, mean keratometry, central corneal thickness, and anterior chamber depth to construct a comprehensive biometric model of the highly myopic Indonesian eye.

2. Methods

This research was designed as an observational analytic study utilizing a cross-sectional approach. The study was conducted at the Ophthalmology Outpatient Clinic of Dr. M. Djamil General Hospital, Padang, Indonesia, a tertiary referral center serving a diverse population in West Sumatra. The data collection period was confined to March 2025 through April 2025. The study protocol was reviewed and approved by the institutional ethics committee, ensuring adherence to the ethical principles outlined in the Declaration of Helsinki.

The recruitment process utilized a consecutive sampling strategy at the Ophthalmology Outpatient Clinic of Dr. M. Djamil General Hospital in Padang, Indonesia. This approach ensured that every eligible patient presenting during the designated study period was invited to participate, thereby reducing selection bias. A critical decision in the study design was the focus on young adults aged 18 to 40 years. This age bracket is clinically significant for refractive research for two primary reasons: (1) Plateau of Juvenile Myopia: By age 18, most cases of juvenile-onset myopia have stabilized, as the rapid ocular growth associated with puberty has typically reached a plateau; (2) Absence of Senile Degeneration: By capping the age at 40, the researchers successfully bypassed the confounding effects of age-related changes such as presbyopia or cataract formation. Cataracts, in particular, can impede the accuracy of optical biometry by scattering light and artificially altering refractive error through lenticular changes. The inclusion criteria were strictly limited to individuals with a diagnosis of High Myopia, defined by a Spherical Equivalent (SE) of -6.00 Diopters (D) or worse. This stringent threshold was established to ensure that the cohort represented the specific pathological phenotype of high myopia rather than mild refractive errors, which may not involve significant structural remodeling.

To ensure that the observed biometric data reflected pure myopic pathophysiology rather than secondary disease states, a robust set of exclusion criteria was enforced. Any condition that could artificially alter the dimensions or the optical power of the eye was eliminated: (1) Refractive History: Patients with a history of any intraocular or corneal refractive surgery, such as LASIK, PRK, or cataract extraction, were excluded. These procedures permanently alter corneal curvature or internal optics, rendering original biometric correlations invalid; (2) Ocular Trauma and Inflammation: A history of significant ocular trauma or active ocular inflammation (uveitis) and infection was grounds for exclusion to avoid data skewed by scarring or inflammatory structural changes; (3) Anterior Segment Pathologies: Conditions like keratoconus, corneal dystrophies, or corneal scarring were excluded. Keratoconus, for instance, causes a pathological steepening of the cornea that would confound the study's investigation into the natural compensatory corneal flattening seen in axial myopia; (4) Posterior and Systemic Factors: The study excluded posterior segment pathologies unrelated to myopia, such as diabetic retinopathy or vascular occlusions. Furthermore, systemic syndromic conditions known to affect connective tissue, such as Marfan syndrome or Stickler syndrome, were excluded because they can cause ocular elongation through mechanisms distinct from common myopic progression.

Each subject underwent a meticulous, standardized ophthalmic evaluation to ensure the highest degree of diagnostic accuracy. Distance visual acuity was first assessed monocularly using a Snellen chart at a distance of 6 meters. The refractive error was then determined through a rigorous two-step verification method: (1) Objective Refraction: Performed using streak retinoscopy to assess the total refractive error without patient input, serving as a vital anatomical baseline; (2) Subjective Refraction: Refined using a phoropter to achieve the Best Corrected Visual Acuity (BCVA), allowing the clinician to account for the patient's visual perception and refine the final prescription. The statistical cornerstone for these measurements was the Spherical Equivalent (SE), calculated using the

standard formula: SE equals Sphere power plus half of the Cylinder power.

The study's primary technological asset was the IOLMaster 700, which represents the gold standard in optical biometry by utilizing Swept-Source Optical Coherence Tomography (SS-OCT). The device operates using a tunable laser source centered at a 1050 nm wavelength. This longer wavelength is superior to the 800 nm range used in standard spectral-domain OCT systems because it penetrates ocular tissues more effectively and minimizes light scatter. This is particularly critical in eyes with extreme axial lengths, where signal quality historically suffered. In high myopia, the presence of a posterior staphyloma (a localized bulging of the posterior sclera) can make measurements along the anatomical axis inaccurate. The IOLMaster 700 addresses this through a fixation check mechanism. By visualizing the foveal dimple on the B-scan, the device ensures that the Axial Length (AL) is measured strictly along the visual axis—from the corneal vertex directly to the fovea—rather than the anatomical axis, eliminating alignment errors common in pathological eyes.

The study tracked four key biometric parameters to construct a comprehensive ocular model: (1) Axial Length (AL): Measured from the anterior corneal surface to the retinal pigment epithelium (RPE); (2) Keratometry (K): Measured as reflection from 18 points in hexagonal patterns across three zones (1.5 mm, 2.5 mm, and 3.5 mm). The study utilized the Mean K reading for analysis; (3) Central Corneal Thickness (CCT): Pachymetry measured at the exact optical center; (4) Anterior Chamber Depth (ACD): Measured from the corneal epithelium to the anterior capsule of the crystalline lens.

Data management and analysis were performed using SPSS Statistics version 30.0. To maintain mathematical integrity, the distribution of all continuous variables (Age, SE, AL, K, CCT, and ACD) was first scrutinized using the Shapiro-Wilk test to determine normality. The strength and direction of the relationships between these variables were quantified using: (1) Pearson's Correlation Coefficient (r): For normally distributed variables; (2) Spearman's Rank Correlation (r): For non-normally distributed data; (3) Linear Regression Modeling: Specifically used to quantify the impact of Axial Length on Spherical Equivalent and Keratometry, allowing the researchers to determine how much of the variance in refractive error could be explained by axial elongation. A p-value of less than 0.05 was established as the threshold for statistical significance, ensuring that the findings represented genuine clinical trends rather than random chance.

3. Results

Table 1 provides a comprehensive overview of the demographic characteristics of the study cohort, which consisted of 32 eyes from 32 high myopia patients. The analysis revealed a mean age of 25.97 plus or minus 6.96 years. This focus on young adults was a deliberate methodological choice to examine ocular biometry during a phase of relative refractive stability. By restricting the age range to 18–40 years, the researchers effectively minimized the confounding effects of age-related ocular changes, such as presbyopia or early cataract formation, which can artificially skew refractive and biometric measurements.

The gender distribution within the cohort showed a significant female predominance, with females comprising 68.8% (n = 11) of the participants, while males represented 31.3% (n = 5). While this gender skew is notable, the cohort's homogeneity in terms of age and refractive status (SE of -6.00 D or worse) allows for a precise investigation into the structural mechanisms of high myopia in the Indonesian population. This demographic profile serves as the foundational baseline for the study's primary objective: determining how axial elongation serves as the dominant factor in refractive severity and investigating potential compensatory changes in the anterior segment.

Table 1. Demographic Profile

Summary of characteristics for the high myopia study cohort in West Sumatra (N = 32 eyes from 32 patients).

CHARACTERISTIC	MEAN / FREQUENCY (N)	SD / PERCENTAGE (%)
CONTINUOUS METRICS		
Age (Years)	**25.97**	± 6.96
GENDER DISTRIBUTION		
Male	5	31.3%
Female	**11**	**68.8%**

ℹ The cohort represents a specific demographic of young adults, predominantly female, within the 18–40 year age range designed to minimize age-related ocular confounding.

Table 2 provides a detailed descriptive analysis of the ocular biometric parameters that define the structural profile of the high myopia cohort. The mean Spherical Equivalent (SE) was recorded at -8.14 ± 2.09 D, with a range extending to -12.50 D, confirming the severe refractive status of the participants. This high degree of myopia is fundamentally driven by significant globe elongation, as evidenced by a mean Axial Length (AL) of 26.93 ± 1.92 mm. Notably, the maximum recorded AL reached 31.03 mm, a value that significantly exceeds the dimensions of a typical emmetropic eye and highlights the pathological expansion of the posterior segment in these individuals.

Regarding the anterior segment, the mean Keratometry (K) was 44.27 ± 1.75 D, representing the corneal refractive power. The data also revealed a mean Central Corneal Thickness (CCT) of 513.59 ± 32.64 µm and an Anterior Chamber Depth (ACD) of 3.67 ± 0.30 mm. The stability of these anterior metrics, particularly the ACD, despite extreme variations in axial length, suggests that the structural remodeling in high myopia is localized to the retro-equatorial region rather than representing a global, uniform expansion of the entire eye. These descriptive statistics serve as the biometric foundation for identifying axial elongation as the primary determinant of refractive severity in the Indonesian population.

Table 3 delineates the primary correlation matrix between the Spherical Equivalent (SE) and various ocular biometric parameters, isolating the structural determinants of high myopia. The most profound finding is the robust, statistically significant negative correlation between SE and Axial Length ($r = -0.86$; $p < 0.001$). This strong association provides biometric confirmation that axial elongation is the predominant structural mechanism driving refractive severity in this Indonesian cohort. Clinically, this indicates that as the posterior segment expands, the refractive error becomes progressively more negative, effectively shifting the focal point in front of the retreating retina.

In stark contrast, no significant correlations were identified between the degree of myopia and anterior segment parameters, including Mean Keratometry ($r = 0.15$; $p = 0.42$), Central Corneal Thickness ($r = 0.06$; $p = 0.77$), or Anterior Chamber Depth ($r = 0.05$; $p = 0.79$). This statistical dissociation is critical as it

suggests that while the cornea and lens contribute to the eye's total refractive power, they do not dictate the severity of the myopic state in eyes that have already progressed to high myopia. These findings reinforce the pathophysiological model that high myopia is fundamentally a disease of focal posterior scleral remodeling. Consequently, the data support the necessity of monitoring axial length as the primary anatomical marker of disease burden rather than relying solely on refractive measurements.

Table 2. Ocular Biometric Parameters

Descriptive statistics summarizing the refractive and structural profile of high myopes.

OCULAR PARAMETER	MEAN (± SD)	MINIMUM	MAXIMUM
Refractive Status **Spherical Equivalent (SE) [D]**	**-8.14 ± 2.09**	-12.50	-6.00
Global Elongation **Axial Length (AL) [mm]**	**26.93 ± 1.92**	24.42	31.03
Corneal Power **Mean Keratometry (K) [D]**	**44.27 ± 1.75**	40.75	49.25
Pachymetry **Central Corneal Thickness [μm]**	**513.59 ± 32.64**	457	559
Anterior Chamber **Anterior Chamber Depth (ACD) [mm]**	**3.67 ± 0.30**	3.16	4.32

Key Observation: The study cohort exhibits significant axial elongation (up to 31.03 mm). While the mean SE is -8.14 D, the extreme range confirms the pathological nature of high myopia in this Indonesian population.

Table 3. Primary Correlation Matrix

Statistical association between Spherical Equivalent (SE) and Ocular Biometry.

BIOMETRIC PARAMETER	CORRELATION (R)	P-VALUE	INTERPRETATION
Axial Length (AL)	**-0.86**	**< 0.001**	STRONG NEGATIVE
Mean Keratometry (K)	0.15	0.42	NO CORRELATION
Central Corneal Thickness (CCT)	0.06	0.77	NO CORRELATION
Anterior Chamber Depth (ACD)	0.05	0.79	NO CORRELATION

Statistical Insight: Axial Length serves as the sole biometric variable with a statistically significant correlation to refractive error severity ($p < 0.001$). Anterior segment parameters (K, CCT, ACD) show no direct statistical impact on the degree of myopia in this cohort.

A secondary analysis was performed to see how the elongation of the eye (AL) affected the shape of the anterior segment (Table 4). Notably, a moderate negative correlation was found between Axial Length and Mean Keratometry, suggesting that longer eyes tended to have flatter corneas. There was no correlation between AL and the depth of the anterior chamber or corneal thickness.

Table 4. Secondary Correlation Matrix

Analysis of Axial Length (AL) relative to Anterior Segment dimensions and compensatory adaptation.

BIOMETRIC PARAMETER	CORRELATION COEFFICIENT (R)	P-VALUE	INTERPRETATION
Mean Keratometry (K)	**-0.41**	**0.02**	MODERATE NEGATIVE
Anterior Chamber Depth (ACD)	0.18	0.33	NO CORRELATION
Central Corneal Thickness (CCT)	0.11	0.56	NO CORRELATION

Structural Evidence: The data confirms a statistically significant moderate negative correlation (**r = -0.41**) between Axial Length and Mean Keratometry. This indicates a compensatory flattening of the cornea as the globe elongates, though this mechanism is ultimately insufficient to neutralize high myopic refractive shifts.

Linear regression modelling was performed to quantify the relationship (Table 5). The model for AL and SE showed a high coefficient of determination (R-squared = 0.643), meaning AL explains 64.3% of the variance in refractive error.

Table 5. Linear Regression Model Summaries

Quantifying the predictive impact of Axial Length on refractive and corneal parameters.

DEPENDENT VARIABLE (Y)	INDEPENDENT VARIABLE (X)	REGRESSION EQUATION	R-SQUARED VALUE	P-VALUE
Spherical Equivalent (SE)	Axial Length (AL)	`Y = 15.3 - 0.87(X)`	**0.643**	< 0.001
Mean Keratometry (K)	Axial Length (AL)	`Y = 54.37 - 0.37(X)`	**0.170**	0.02

Predictive Analysis: Axial Length (AL) is a powerful predictor of refractive error, explaining **64.3%** of the variance in Spherical Equivalent. Furthermore, for every 1 mm increase in axial length, the cornea flattens by approximately **0.37 D**, confirming a statistically significant, albeit partial, compensatory response.

4. Discussion

This study presents a comprehensive biometric analysis of high myopia in an Indonesian cohort, utilizing the precision of Swept-Source OCT to dissect the structural determinants of refractive error. The findings underscore the complex pathophysiology of myopic progression, revealing a dominant role of axial elongation and a secondary, yet distinct, compensatory response of the cornea.[11]

The most definitive finding of this investigation is the robust negative correlation (r = -0.86) between Spherical Equivalent and Axial Length. This statistical relationship serves as biometric confirmation that high myopia is fundamentally a disease of posterior segment expansion. Our regression model suggests that for every millimeter of axial extension, the refractive error shifts by approximately -0.87 Diopters. This finding aligns with the hyperopic defocus theory of myopia pathogenesis. According to this theory, when the focal point of the eye falls behind the retina (hyperopic blur), it triggers a local biochemical feedback loop within the retina. Amacrine cells in the retina modulate the release of dopamine, a neurotransmitter critical for regulating ocular growth.[12] Reduced dopamine levels lead to a cascade involving the retinal pigment epithelium (RPE) and the choroid, ultimately resulting in the release of matrix metalloproteinases (MMPs) in the sclera. These enzymes degrade the scleral collagen matrix, reducing its biomechanical stiffness and allowing the eye to elongate under the influence of normal intraocular pressure.

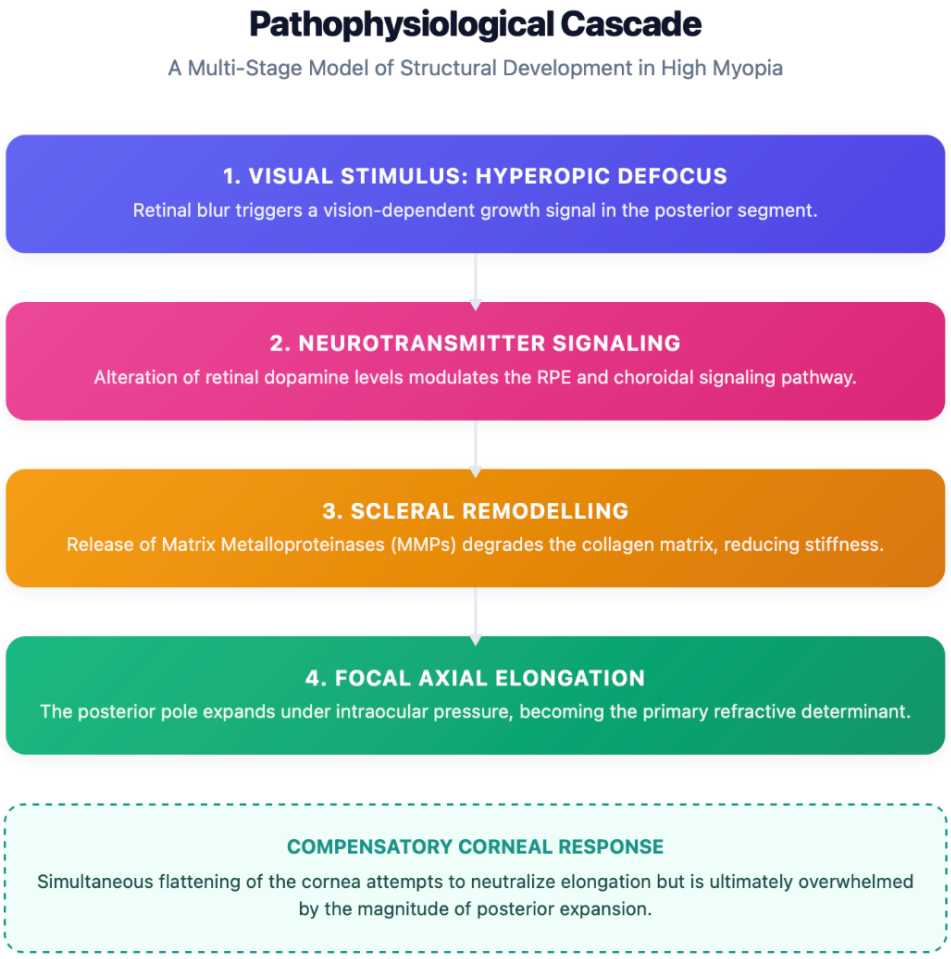

Figure 1. Pathophysiological cascade.

The dominance of AL in our data supports the concept that this scleral remodeling is the final common pathway for all forms of myopia, regardless of the upstream trigger (genetic or environmental).[13] The high R-squared value indicates that while lens power and corneal power play a role, the sheer magnitude of axial growth in high myopia overwhelms these optical variables. This is consistent with histopathological studies showing that in high myopia, the sclera is significantly thinned, particularly at the posterior pole, leading to the formation of staphylomas (Figure 2). Our data confirms that monitoring AL is a more direct measure of myopic pathology than refraction alone, as refraction can be influenced by accommodative spasm or lenticular sclerosis, whereas AL represents the true anatomical burden of the disease.[14]

A particularly intriguing finding in our study is the significant negative correlation (r = -0.41) between Axial Length and Mean Keratometry. This indicates that as the eye elongates, the cornea becomes flatter (lower dioptric power). This phenomenon represents a persistence of the emmetropization mechanism into adulthood. During emmetropization in infancy, the eye grows rapidly in length, and to prevent the development of myopia, the cornea flattens, and the lens thins.[15] Our data suggest that this biological coupling between posterior elongation and anterior flattening is maintained even in the pathological state of high myopia.

Physiologically, this could be explained by two competing theories. The first is the passive stretch theory, which posits that the expansion of the globe exerts a mechanical tractional force on the limbal ring, physically stretching the cornea and flattening its curvature. The second is the active regulation theory, which suggests that the same retinal signaling pathways driving scleral growth also send inhibitory signals to the anterior segment to reduce optical power in a futile attempt to maintain emmetropia. The fact that the correlation is moderate (r = -0.41) rather than strong suggests that this compensatory capacity is limited. In high myopia, the axial elongation eventually outstrips the cornea's ability to flatten. This uncoupling point is likely a key marker for the transition from physiological eye growth to pathological myopia. This finding has significant clinical implications for refractive surgery; patients with high myopia typically have flatter corneas to begin with, which limits the amount of tissue that can be safely ablated during LASIK, often necessitating intraocular lens implantation instead.[16]

Contrary to the intuitive assumption that a larger eye would have a deeper anterior chamber, our study found no significant correlation between Axial Length and anterior chamber depth (ACD). This lack of correlation is a critical pathophysiological clue. It suggests that the elongation in high myopia is not a uniform, balloon-like expansion of the entire globe.[17] Instead, it supports the model of retro-equatorial elongation. In this model, the stretching of the eye occurs almost exclusively in the posterior segment, behind the insertion of the extraocular muscles. The anterior segment, including the anterior chamber and the lens zonular complex, remains relatively dimensionally stable.

This finding distinguishes high myopia from other causes of buphthalmos (large eyes), such as congenital glaucoma, where high intraocular pressure expands the entire globe, including the cornea and anterior chamber. The preservation of normal ACD dimensions in our high myopia cohort implies that the pathological weakness is localized to the posterior sclera. This localization is likely due to the differential gene expression in the posterior sclera compared to the anterior sclera, making the posterior pole more susceptible to the matrix metalloproteinase-mediated remodeling triggered by myopic stimuli. This stability of the ACD is clinically relevant for phakic IOL implantation (ICL), as it implies that high myopes do not necessarily have roomier anterior chambers to accommodate these implants, requiring careful sizing based on standard ACD measurements rather than assuming depth based on axial length.[18]

Our study found no correlation between central corneal thickness (CCT) and spherical equivalent or

axial length. This reinforces the concept of biomechanical independence between the cornea and the sclera. Although they are continuous connective tissues, the cornea and sclera have distinct collagen fibril organizations and proteoglycan compositions.[19] The sclera in high myopia undergoes thinning and ectasia, but our data show this does not translate to corneal thinning. The cornea appears to be genetically hard-coded to maintain its thickness regardless of the refractive error. This contradicts some historical beliefs that myopic eyes have thinner, weaker corneas. The clinical takeaway is that a patient with high myopia does not inherently have a higher risk of false-low intraocular pressure readings due to thin corneas, nor are they inherently more susceptible to ectasia based on thickness alone, although their flatter curvature must be considered.[20]

5. Conclusion

This study provides compelling biometric evidence that high myopia in the Indonesian population is driven primarily by excessive axial elongation, confirming the dominance of posterior segment pathology in determining refractive severity. We identified a distinct compensatory mechanism characterized by corneal flattening in response to ocular growth; however, this emmetropization response is partial and ultimately overwhelmed by the magnitude of axial extension. Furthermore, the dissociation between axial length and anterior chamber depth supports the pathophysiological model of focal retro-equatorial elongation rather than global expansion. These findings highlight the necessity of shifting clinical myopia management from merely correcting refraction to actively targeting axial elongation through pharmacological or optical interventions to prevent the structural sequelae of this progressive disease.

6. References

1. Micheletti E, El-Nimri N, Nishida T, Moghimi S, Rezapour J, Fazio MA, et al. Central visual field damage in glaucoma eyes with choroidal microvasculature dropout with and without high axial myopia. Br J Ophthalmol. 2024; 108(3): 372–9.
2. Kayabaşi M, Köksaldi S, Demirel N, Saatci AO. The effect of axial length on macular vascular density in eyes with high myopia. Rom J Ophthalmol. 2025; 69(1): 88–100.
3. Gaafar AAM, Mohamed El Zankalony YAM, Riad BFA, Khalil AKI. Corneal biometric features and their association with axial length in high myopia. QJM. 2025; 118(Suppl_1).
4. Wu C-W, Chen H-Y. Comparison of the diagnostic performance of two spectral-domain OCT machines in detecting glaucoma in high and non-high axial myopia eyes. Int Ophthalmol. 2025; 46(1): 20.
5. Li M, Shao Y, Liu L, Shi Y, Sun L, Li H, et al. The influence of axial length on visual quality following SMILE surgery in patients with high myopia. Photodiagnosis Photodyn Ther. 2025; 52(104497): 104497.
6. Liu T, Wang YX, Jonas JB, Hoang QV, Girard MJA, Wang X. Gaze-induced optic nerve head deformations are greater in high myopia and strains increase with axial length. Invest Ophthalmol Vis Sci. 2025; 66(9): 21.
7. Borkenstein AF, Borkenstein E-M. Intraocular lens calculation for cataract surgery in high myopia: a case report of an extreme axial eyeball length of 35.7 mm. Front Med (Lausanne). 2025; 12: 1450922.
8. Huang Y, Xu H, Yang X, Huang H, Zhi Z, Chen S. Correlation and agreement of anterior chamber biometric parameters and sulcusto-sulcus diameter in Chinese patients with high myopia and varying axial lengths. J Refract Surg. 2025; 41(8): e805–13.
9. Zhai J, Chen H, Ye X-J, Cai W-J, Hou L-J, Shen M-X, et al. Ocular biological characteristics of children with myopia and rapid axial length changes treated with

spectacles with highly aspherical lenslets. Int J Ophthalmol. 2025; 18(8): 1522–8.
10. Murata M, Miyake M, Suda K, Mori Y, Morino K, Okayama W, et al. Potential negative feedback between age and baseline axial length on axial elongation in high myopia. Ophthalmol Sci. 2026; 6(1): 100937.
11. Kong K, Liu X, Fang Z, Jiang J, Jiang J, Wang D, et al. Axial elongation in nonpathologic high myopia: Ocular structural changes and glaucoma diagnostic challenges. Asia Pac J Ophthalmol (Phila). 2024; 13(6): 100123.
12. Bartol-Puyal F de A, Isanta C, Ruiz-Moreno Ó, Abadia B, Calvo P, Pablo L. Distribution of choroidal thinning in high myopia, diabetes mellitus, and aging: a swept-source OCT study. J Ophthalmol. 2019; 2019: 3567813.
13. Yu X, Yuan J, Chen ZJ, Li K, Yao Y, Xing S, et al. Whole-exome sequencing among school-aged children with high myopia. JAMA Netw Open. 2023; 6(12): e2345821.
14. Xing S, Jiang S, Wang S, Lin P, Sun H, Peng H, et al. Association of mitochondrial DNA variation with high myopia in a Han Chinese population. Mol Genet Genomics. 2023; 298(5): 1059–71.
15. Jiang J, Song Y, Kong K, Wang P, Lin F, Gao X, et al. Optic nerve head abnormalities in nonpathologic high myopia and the relationship with visual field. Asia Pac J Ophthalmol (Phila). 2023; 12(5): 460–7.
16. Kong K, Jiang J, Wang P, Song Y, Lin F, Li F, et al. Progression patterns and risk factors of axial elongation in young adults with nonpathologic high myopia: Three-year large longitudinal cohort follow-up. Am J Ophthalmol. 2024; 267: 293–303.
17. Zhang X, Jiang J, Kong K, Li F, Chen S, Wang P, et al. Optic neuropathy in high myopia: Glaucoma or high myopia or both? Prog Retin Eye Res. 2024; 99(101246): 101246.
18. Yuan J, Qiu R, Wang Y, Chen ZJ, Sun H, Dai W, et al. Exome-wide genetic risk score (ExGRS) to predict high myopia across multi-ancestry populations. Commun Med (Lond). 2024; 4(1):280.
19. Wang P, Kong K, Jiang J, Jiang J, Xie Z, Lin F, et al. Intraocular pressure is a promising target for myopia control. J Transl Med. 2025; 23(1): 556.
20. Sharma P. Complicated case of coexisting cataract, uveitis, high myopia & glaucoma-surgical management. high myopia. 2024; I(1): 117–20.