ORIGINAL ARTICLE

CONTENTS

Clinical and ophthalmological patterns of myopia onset and progression in ukrainian patients of different age groups

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ABSTRACT

Aim: To investigate the clinical and ophthalmological patterns associated with the onset and rapid progression of myopia in Ukrainian patients across different age groups.

Materials and Methods: We analyzed data from 66 patients (132 eyes) with myopia, aged 6 to 17 years. In addition to a comprehensive ophthalmological examination, patients underwent OCT and OCT angiography (OCT-A), endothelial microscopy, assessment of corneal rigidity and scleral stiffness using the Sergienko–Shargorodska method, and a questionnaire survey.

Results: Myopia onset occurred most frequently at 9-11 years of age (50% of cases) in the Ukrainian pediatric cohort. Gender did not influence disease onset or the rate of progression. Key clinical factors associated with rapid myopia progression (>1 D/year) included physical inactivity, vitamin deficiency, and parental smoking. Increased corneal rigidity (KER > 4.1) was detected as early as 6-8 years of age (67% of patients), preceding the first clinical peak of disease progression. At 9-11 years of age, rapid progression was associated with corneal endothelial decompensation, grade II–IV trabecular dysgenesis (91%), significant choroidal thinning (<190 µm; 91%), and anterior scleral thinning (TAST3 <500 µm; 79%), all of which may contribute to accelerated axial elongation. OCT-A confirmed ischemia in the peritemporal quadrant, whereas macular perfusion remained stable in most patients (88%).

Conclusions: The development of myopia follows a staged pattern, progressing from refractive genesis (corneal and endothelial changes) in younger children to axial genesis (alterations of the scleral capsule) in adolescents aged 15-17 years. The preservation of macular perfusion despite peripheral ischemia suggests the presence of compensatory mechanisms during childhood.

KEYWORDS: myopia, optometry, rigidity, cornea, endothelium, perfusion

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INTRODUCTION

Modern ophthalmology considers myopia not merely a refractive error but a global public health challenge with significant medical and social implications. According to recent meta-analyses and projections by the International Myopia Institute, by 2050 the global prevalence of myopia may reach 50% of the world's population, with nearly 10% affected by high myopia [1–3]. Ukraine demonstrates a similar trend toward an earlier onset of the condition and a rising proportion of rapidly progressive myopia, which is associated with increasing near-work demands and lifestyle changes [4].

A key concept in understanding the etiology and pathogenesis of myopia is the biomechanical theory of its development [5]. This concept is based on the assumption that the primary cause of ocular elongation results from alterations in the elastic properties of the fibrous tunic of the eye. The Ukrainian ophthalmological school, represented by the work of M.M. Serhienko and Yu.M. Kondratenko [5–8], has made an invaluable contribution to the development and implementation of this concept, inspiring a new generation of researchers [5] and stimulating further investigations in this field.

According to these researchers [6], the progression of myopia is closely associated with alterations in the aqueous outflow system. In particular, trabecular dysgenesis and pigmentation have been shown to impair aqueous humor hydrodynamics, creating conditions for mechanical scleral stretching under the influence of intraocular pressure, even when intraocular pressure remains within the normal range [7].

The biomechanical properties of the fibrous tunic, including corneal and scleral rigidity, serve as critical markers of ocular stability. Several studies [5, 7, 9] have demonstrated that the rigidity index is an individual characteristic that determines the eye's resistance to deformation. A reduced rigidity coefficient is considered an unfavorable prognostic factor preceding axial elongation [5].

These changes are likely driven by extracellular matrix remodeling within the sclera, where local hypoxia activates the HIF-1α and HIF-2α signaling pathways, promoting increased expression of matrix metalloproteinase-2 (MMP-2) and degradation of type I collagen [10]. However, this hypothesis requires further investigation.

Another important component of myopia pathogenesis involves the condition of the choroid and retinal microvascular perfusion. Advanced imaging modalities, such as optical coherence tomography (OCT) and OCT angiography (OCT-A), have demonstrated that choroidal thinning is a dynamic process that often precedes axial elongation [9, 11]. The choroid functions not only as a trophic layer but also as a biomechanical buffer; therefore, its thinning may reduce its ability to regulate signaling molecules involved in scleral remodelling [12–14].

Despite numerous international studies, including MOSAIC and LAMP [15, 16], the relationship between anterior segment biomechanical alterations and posterior pole vascular biomarkers in the Ukrainian population remains insufficiently investigated. The temporal sequence of involvement of different ocular structures during myopia development across various age groups requires further clarification to identify optimal windows for preventive interventions. Therefore, an integrative analysis of clinical factors, biomechanical parameters, and modern OCT-based biomarkers remains an important research priority in contemporary ophthalmology and optometry [11, 17].

AIM

To investigate the clinical and ophthalmological patterns, including biomechanical and OCT-based structural and microvascular markers, associated with the onset and rapid progression of myopia in Ukrainian patients of different age groups.

MATERIALS AND METHODS

An analytical cohort study was conducted, including data from a comprehensive ophthalmological examination of 66 patients (132 eyes) with myopic refractive error. The study population comprised 35 females (53%) and 31 males (47%). Patients were stratified into four age groups according to the age at myopia onset and disease progression. Group I included 9 patients (18 eyes; 14%) aged 6-8 years. Group II comprised 33 patients (66 eyes; 50%) aged 9-11 years. Group III consisted of 12 patients (24 eyes; 18%) aged 12-14 years, and Group IV included 12 patients (24 eyes; 18%) aged 15-17 years.

The inclusion criteria were as follows: a clinically confirmed diagnosis of myopia (for the study group); written informed consent from the patient or the patient's legal representative to participate in the study and undergo follow-up; and the patient's ability to cooperate adequately during the study procedures. The exclusion criteria included refusal to undergo specific stages of the diagnostic examination or failure to comply with the scheduled follow-up visits.

The enrolled patients were followed for five years. Myopia progression was classified as slow when the spherical equivalent changed by <1 diopter (D) per year and as rapid when it changed by >1 D/year [5, 6].

The comprehensive ophthalmological assessment included:

• medical history – parental questionnaire covering lifestyle, nutrition, habits, and chronic conditions;

• gonioscopy – detailed visualization of the anterior chamber angle and degree of trabecular pigmentation (Ocular, USA);

• corneal topography – to measure corneal thickness and anterior chamber depth (Schwind Sirius+, Germany);

• optical coherence tomography (OCT) (REVO NX Version 10.0.0 Device SN: 2570793/06) and OCT angiography (OCT-A) (Cirrus HD-OCT 5000, Zeiss) – to assess choroidal thickness (CChT, SFCT) and vessel density (DVD) in different sectors;

• endothelial microscopy – to evaluate endothelial cell density (ECD) and hexagonality (Heidelberg Engineering Inc., Germany).

Measurements of corneal rigidity (K_{ER}) and scleral rigidity (K_{ES}) were performed using the Sergienko–Shargorodska method [5]. This in vivo technique for assessing corneal and scleral biomechanical parameters is based on determining the resistance of the fibrous ocular capsule to a controlled and evenly distributed load. The obtained data allow calculation of rigidity coefficients that reflect the elastic and biomechanical state of the ocular tunics [5, 6].

The statistical data analysis was performed using IBM SPSS Statistics v. 27.0 (Armonk, NY: IBM Corp., USA), EZR v. 1.70 and MedStat v. 5.2. Categorical variables were presented as counts and percentages. Comparisons between two groups were performed using the Fisher's exact test. For comparisons among four groups, the χ^2 test was applied, followed by *post hoc* pairwise comparisons using the Marascuilo–Liakh–Gurianov procedure when appropriate. To identify independent predictors associated with the outcome, stepwise logistic regression analysis was performed. The results of the logistic regression analysis were presented as odds ratios (ORs) with 95 % confidence intervals (CIs).

Table 1. Association between the onset of myopia and patients` sex and progression rate in the Ukrainian population

Indicator	Onset of myopia development				p
	6-8 years n = 9	9-11 years n = 33	12-14 years n = 12	15-17 years n = 12	
Males, N (%)	5 (56)	17 (52)	3 (25)	6 (50)	0.406
Females, N (%)	4 (44)	16 (48)	9 (75)	6 (50)	
Myopia progression rate >1 D/year, N (%)	7 (78)	18 (55)	8 (67)	8 (67)	0.582

Source: compiled by the authors of this study

Table 2. The signs of corneal endothelial decompensation in patients with different myopia age onset

Indicator	Onset of myopia development				p
	6-8 years n = 9	9-11 years n = 33	12-14 years n = 12	15-17 years n = 12	
ECD 1601–2000 cells/mm^2, N (%)	1 (11)	15 (45)	4 (33)	1 (8)	0.053
ECD 2401–2800 cells/mm^2, N (%)	0	1 (3)	4 (33)	3 (25)	0.013
Mean endothelial cell size <500.0 μm^2, N (%)	1 (11)	3 (9)	4 (33)	3 (25)	0.207
Hexagonality >50 %, N (%)	1 (11)	1 (3)	4 (33)	3 (25)	0.037

Source: compiled by the authors of this study

Table 3. The presence and degree of trabecular pigmentation in patients with different myopia age onset

Indicator	Onset of myopia development				p
	6-8 years n = 9	9-11 years n = 33	12-14 years n = 12	15-17 years n = 12	
Absence or trabecular pigmentation degree I, N (%)	5 (56)	3 (9)	9 (75)	10 (83)	p_{2-3}= 0.001 p_{2-4}<0.001
Trabecular pigmentation degree II, N (%)	2 (22)	12 (36)	0	0	p_{2-3}= 0.093 p_{2-4}= 0.094
Trabecular pigmentation degrees III-IV, N (%)	2 (22)	18 (55)	3 (25)	2 (17)	0.047

Notes: p_{2-3} – the significance of the difference between 9-11 and 12-14 years groups; p_{2-4} – the significance of the difference between 9-11 and 15-17 years groups

Source: compiled by the authors of this study

The threshold for statistical significance was set at $p < 0.05$ [17].

ETHICS

The study was conducted in accordance with the ethical principles of the Declaration of Helsinki (2000) and the requirements of the current legislation of Ukraine governing biomedical research involving human participants. All study procedures were approved by the local Ethics Committee of Bogomolets National Medical University. Written informed consent for participation in the study and the processing of personal data was obtained from all parents or legal representatives of the participants prior to enrollment.

AI STATEMENT

Artificial intelligence tools, including Microsoft Copilot, Google Gemini, and Grammarly, were used under the full supervision and control of the authors solely for language editing, stylistic improvement of the manuscript, and reference formatting. Artificial intelligence tools were not used for study design, data collection, data analysis, statistical processing, interpretation of results, generation of scientific conclusions, or decision-making related to the findings of the study. All scientific content, methodological decisions, analyses, interpretations, and conclusions were developed, verified, and approved exclusively by the authors. The authors take full responsibility for the accuracy, integrity, and final content of the manuscript.

FRAMEWORK

The work was carried out at the Department of Ophthalmology and Optometry of Postgraduate Education of the Institute of Postgraduate Education of Bogomolets National Medical University and is a fragment of the initiative-research project «Theoretical and practical

Table 4. The macular perfusion and OCT biomarkers in patients with different myopia age onset

Indicator		Onset of myopia development				p
		6-8 years n = 9	9-11 years n = 33	12-14 years n = 12	15-17 years n = 12	
Subfoveal choroid thickness <190.0 μm, N (%)		5 (56)	30 (91)	5 (42)	5 (42)	p_{2-3}= 0.035 p_{2-4}= 0.035
Macular perfusion, deep vascular density in the fovea < 18,0 %, N (%)		1 (11)	5 (15)	1 (8)	1 (8)	0.893
Deep vessel density reduction in the peritemporal quadrant, N (%)	<47.0 mm^{-1}	5 (56)	29 (88)	5 (42)	5 (42)	p_{2-3}= 0,069 p_{2-4}= 0,069
	47.1 – 48.0 mm^{-1}	2 (22)	3 (9)	2 (16)	3 (25)	0,528
	48.1 – 49.0 mm^{-1}	1 (11)	1 (3)	0	4 (33)	0,010
	>49.0 mm^{-1}	1 (11)	0	5 (42)	0	p_{2-3}= 0,018
Thinning of the anterior sclera in the temporal region (TAST3) to <500.0 μm, N (%)		5 (56)	26 (79)	5 (42)	5 (42)	0.040

Notes: p_{2-3} – the significance of difference between 9-11 and 12-14 years groups; p_{2-4} – the significance of difference between 9-11 and 15-17 years groups
Source: compiled by the authors of this study

aspects of improving clinical and experimental methods of diagnosis, treatment and prevention of diseases and injuries of the organ of vision and their complications» (state registration number 0123U104207; term: 2023-2026).

RESULTS

Myopia onset occurred most frequently at 9-11 years, accounting for half of all cases in the included patient sample (33/66; 50%). The remaining cases were distributed across 12-14 years (12/66; 18%), 15-17 years (12/66; 18%), and 6-8 years (9/66; 14%).

The relationship of the myopia age onset to patients` sex and progression rate is presented in Table 1.

According to the presented data (Table 1), no significant gender differences in the onset of myopia were found. In addition, gender did not affect the rate of myopia progression. Among 31 male patients, 22 (71%) cases of rapid progression; among 35 female patients, 19 (54%) such cases (p=0.207).

Environmental and behavioural risk factors for myopia development and progression have been widely studied [2, 4]. In our study, the key clinical factors associated with rapid myopia progression (>1 D/year) were insufficient physical activity (OR 27.8 [95 CI 5.7-136.8]; p<0.001), vitamin deficiency (OR 24.8 [95% CI 5.1-121.2]; p<0.001) and parental smoking (OR 4.2 [95% CI 1.3-13.3%]; p=0.015). Meanwhile, urban residence (63.4% vs. 44.0% in rapid and slow progression groups, respectively; p=0.136) and having at least one myopic parent (56.1% vs. 60.0% in rapid and slow progression groups, respectively; p=0.802) did not show a significant association with rapid myopia progression.

The relationship between myopia onset and signs of corneal endothelial decompensation is summarised in Table 2.

A decrease in endothelial cell density (ECD) to 1601-2000 cells/mm^2 was observed in 45% of patients aged 9-11 years, whereas ECD values within the normal range (2401-2800 cells/mm^2) were recorded in 33% of patients aged 12-14 years. In addition, a reduced mean endothelial cell area (<500.0 μm^2) and increased hexagonality (>50%) were observed in 33% of patients aged 12-14 years (Table 2).

In the youngest age group (6-8 years), most patients showed no changes in endothelial cell density, endothelial cell area, or hexagonality (Table 2).

Data on trabecular pigmentation according to age at myopia onset are presented in Table 3.

In group I (6-8 years), grade I trabecular dysgenesis was detected in 56% of patients, grade II - 22%, and grades III-IV were observed in 22% of cases. In Group II (ages 9-11 years), corneal endothelial decompensation and trabecular dysgenesis III-IV degrees were observed in 55 % of patients, and the II degree - in 36%. The majority of patients in group III (12-14 years) and group IV (15-17 years) had grade I trabecular dysgenesis – 75% and 83%, respectively (Table 3).

Assessment of biomechanical markers revealed that increased K_{ER} values (+)3.1 – (+)4.0 occurred in 50% of patients aged 6-8 years, and higher values (+)5.4 were observed in 44 % of cases, preceding the peak of myopia progression. It was found that in Group I (ages 6-8 years), 67% of patients already demonstrated elevated K_{ER} >(+)4.1, which preceded the clinical progression of the condition.

Analysis of macular perfusion and OCT-based biomarkers revealed a selective pattern of vascular

impairment. Clinically significant structural alterations were predominantly observed in patients aged 9-11 years, identifying this period as a critical stage at which ocular compensatory mechanisms begin to be overwhelmed by structural remodeling (Table 4).

Significant thinning of the subfoveal choroid at a distance of 3 mm from the fovea in the vertical meridian (SFCT, S3+I3) to <190.0 μm was observed in 91% of patients aged 9-11 years, indicating active remodeling of the posterior segment.

A notable finding was that macular perfusion remained stable and within the normal range (>18.0%) in most patients (88%), whereas reduced perfusion was detected in the peritemporal quadrant.

A deep vessel density of <47.0 mm^{-1} in the peritemporal quadrant was observed more frequently in patients aged 9-11 years than in those aged 12-14 and 15-17 years. Deep vessel density values of 48.1-49.0 mm^{-1} occurred more frequently in the 15-17-year age group. Values >49.0 mm^{-1} were observed in 42% of patients aged 12-14 years but were absent in the 9-11- and 15-17-year groups and were detected in only one patient aged 6-8 years.

These findings indicate selective vascular vulnerability. The pediatric eye appears to preserve perfusion of the foveal region, which is responsible for central visual acuity, through autoregulatory mechanisms, whereas the peripheral retina is affected earlier by biomechanical stretching of the ocular tunic.

At the same time, thinning of the anterior sclera in the temporal region (TAST3) to <500.0 μm was observed in 79% of patients aged 9–11 years (Table 4). Previous studies [13, 14] have demonstrated that anterior scleral thickness is directly associated with overall ocular rigidity. Our findings suggest that, in children aged 9-11 years, reduced scleral rigidity increases the susceptibility of the ocular wall to intraocular pressure–related deformation, thereby facilitating axial elongation and the progression of axial myopia.

DISCUSSION

The findings of the present study support the concept of the stepwise involvement of ocular structures in myopia development. The earliest alterations were observed in corneal rigidity (at 6–8 years of age), followed by the appearance of peripheral retinal perfusion abnormalities and signs of corneal endothelial decompensation during the 9-11-year period. In contrast to adults, the preservation of macular perfusion in children may reflect compensatory autoregulatory mechanisms operating during the phase of active ocular growth. At the same time, thinning of the anterior sclera was also identified in patients aged 9-11 years.

Our findings are consistent with the theory proposed by Yu. Kondratenko and N. Serhienko [8], emphasise the key role of the ocular drainage system in the pathogenesis of myopia. The high prevalence of trabecular abnormalities (grades II–IV; 91%) observed during the first peak of disease progression (9-11 years of age) suggests that impaired aqueous humour outflow and subclinical elevation of intraocular pressure may represent important triggers of axial elongation in myopia [7, 8, 13, 18].

The present study further supports the concept of the sequential involvement of ocular structures during myopia development. At the earliest stage (6-8 years of age), the process appears to be predominantly refractive in nature and is characterized by alterations in the biomechanical properties of the fibrous ocular tunic, increased rigidity indices, and early corneal endothelial decompensation. The subsequent stage (9-11 years of age) is marked by the involvement of the choroid and sclera, indicating a transition toward axial myopia. This interpretation is supported by anterior scleral thinning (TAST3 <500 μm), which was observed in 79% of children in this age group and is consistent with previous reports [9, 13, 14]. Such thinning may serve as a marker of reduced biomechanical stability of the eye and increase susceptibility to progressive axial elongation.

Previous studies have shown that a 23-μm reduction in choroidal thickness is associated with an approximately 1-mm increase in axial length [9, 12, 14]. Consistent with these findings, our study demonstrated marked choroidal thinning (<190 μm), suggesting an increased risk of subsequent myopic staphyloma formation.

Comparison of our OCT angiography findings with previously published data [11–13] revealed a pattern of selective vascular vulnerability: macular perfusion remains preserved for a prolonged period, likely owing to autoregulatory mechanisms, whereas the peritemporal region appears to be the earliest site of perfusion impairment [11–13]. These findings emphasise the value of segmental OCT angiography analysis for the early identification of patients at increased risk of disease progression.

LIMITATIONS OF THE STUDY

Previous studies in the area studied are few. The study included 66 Ukrainian patients (132 eyes), which limits the generalizability of the results to larger populations. However, the sample size allowed for an in-depth qualitative analysis that identified peaks in myopia and significant clinical predictors. The duration of follow-up of the patient groups is five years and needs to be extended in the future.

PERSPECTIVES OF SUBSEQUENT SCIENTIFIC RESEARCH

The increase in the corneal rigidity coefficient, the decrease in the density of the deep vascular plexus in the peritemporal quadratus, and the thickness of the choroid 3 mm from the fovea obtained in this clinical study are the basis for a more detailed study of the problem of the influence of changes in the stiffness of the fibrous capsule of the eye and the blood supply to the retina and posterior pole of the eye on the development and progression of myopia.

CONCLUSIONS

1. Assessment of the corneal rigidity coefficient is the most informative early biomechanical marker for identifying potential risks of myopia development and progression. Its alterations can be detected 2-3 years before the clinical manifestation of disease progression.
2. The age of 9-11 years represents a critical pathogenetic stage, characterized by structural deformation of the ocular tunics and ischemia of the peripheral retina.
3. Myopia progression in Ukrainian patients follows a trajectory from refractive changes to axial elongation.
4. Rapid myopia progression is accompanied by ischemia of the peritemporal zone while macular perfusion remains preserved.
5. Insufficient physical activity, vitamin deficiency and parental smoking are significant clinical risk factors associated with the progression of myopia in the Ukrainian patients.

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CONFLICT OF INTEREST
The Authors declare no conflict of interest

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