Four-Year Progression of Myopic Maculopathy in Children and Adolescents with High Myopia

Running head: Progression of Myopic Maculopathy in Pediatric High Myopes

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Key Points

Question: How does myopic maculopathy change in those under 18 with high myopia?

Findings: In this observational study that included 548 eyes, the progression of myopic maculopathy was identified in 12.2% of eyes, and the most common change was the enlargement of diffuse chorioretinal atrophy. The risk factors were worse best-corrected visual acuity, longer axial length, faster axial length elongation, and more severe myopic maculopathy.

Meaning: Myopic maculopathy progressed in approximately 1 in 8 individuals under 18 years of age, supporting consideration of follow-up in these individuals and trying to identify those at higher risk for progression.

Abstract

Importance: Individuals with high myopia under age 18 years are at relatively high risk of progressively worsening myopic maculopathy. Additional studies are needed to investigate the progression of myopic maculopathy in this age group, as well as the risk factors associated with progression.

Objective: To investigate the 4-year progression of myopic maculopathy in highly myopic children and adolescents in China and to explore potential risk factors.

Design： An observational study with 4-year follow-up.

Setting： A hospital-based study.

participants: A total of 548 high myopic eyes (spherical power ≤ −6.00 diopters) of 274 participants aged 7 to 17 years were included. Participants underwent comprehensive ophthalmic examination at baseline and 4-year follow-up. Myopic maculopathy was accessed by the International Photographic Classification and Grading System.

Main outcomes and measures: The progression of myopic maculopathy progression over four years and associated risk factors.

Results: The 4-year progression of myopic maculopathy was found in 67 (12.2%) of 548 eyes, with 88 lesion changes including new signs of the tessellated fundus in 16 eyes (18.2%), diffuse atrophy in 12 eyes (13.6%), patchy atrophy in 2 eyes (2.3%), lacquer cracks in 9 eyes (10.2%), and enlargement of diffuse atrophy in 49 eyes (55.7%). By multivariable analysis,

worse best-corrected visual acuity (BCVA) (odds ratio [OR], 6.68; 95%CI, 1.15 to 38.99; *P* = .04), longer axial length (AL) (OR, 1.73; 95%CI, 1.34 to 2.24; *P* < .001), faster AL elongation (OR, 302.83; 95%CI, 28.61 to 3205.64; *P* < .001), and more severe myopic maculopathy (diffuse atrophy, OR, 4.52; 95%CI: 1.98 to 10.30; *P* < .001; patchy atrophy, OR, 3.82; 95%CI, 1.66 to 8.80; *P* = .002) were associated with myopic maculopathy progression.

Conclusions and Relevance: The progression of myopic maculopathy was observed in approximately 12% of pediatric high myopes for 4 years. The major type of progression was the enlargement of diffuse atrophy. Risk factors for myopic maculopathy progression were worse BCVA, longer AL, faster AL elongation, and more severe myopic maculopathy. These findings support consideration of follow-up in these individuals and trying to identify those at higher risk for progression.

Introduction

Myopic maculopathy refers to a collection of signs that signify degeneration of chorioretinal tissues linked to the excessive elongation of the axial length (AL) in myopic eyes[1, 2], which is one of the major causes of blindness or visual impairment, especially in East Asia.[3-5] In the Tajimi Study, it constituted the primary cause of monocular blindness among individuals aged 40 and above in the Japanese population.[6] Moreover, there was a prevailing assumption that the prevalence of myopic maculopathy would significantly increase among elderly individuals as the aging of the young population with a high prevalence of myopia.[7] Current predictions indicate that visual impairment from myopic maculopathy will affect 55.7 million people, with an estimated 18.5 million experiencing blindness worldwide by 2050.[4] In addition, addressing the prevention and treatment of myopia and myopic maculopathy carries a considerable economic burden.[8, 9]

Despite the significance of myopic maculopathy, there is limited documentation regarding its progression in children and adolescents.[10, 11] In a longitudinal study conducted by Yokoi et al,[10] involving 29 pediatric patients with high myopia followed for 20 years or more, it was discovered that the presence of peripapillary diffuse chorioretinal atrophy in childhood may serve as an indicative factor for the future development of pathologic myopia in adulthood. In a survey of hospital clinics, Guo et al[11] reported that myopic maculopathy progression was observed in 18.9% of children's eyes with high myopia and

associated with refractive error and parapapillary gamma zone. However, the progression pattern in this specific demographic and the associated factors remain unclear, which is helpful in timely intervention in individuals at risk of developing pathologic lesions in the fundus.

Our study aimed to investigate the progression of myopic maculopathy in highly myopic children and adolescents over a 4-year follow-up period and explore the factors that were associated with its progression.

Methods

Study populations

This observational study recruited children and adolescents with bilateral high myopia who had undergone examinations at the Zhongshan Ophthalmologic Center. High myopia was defined as a myopic spherical power of −6.0 diopters (D) or less. A total of 426 highly myopic individuals aged 7 to 17 years were initially examined between 2011 and 2012, with 277 participants (65.02%) receiving a 4-year follow-up examination. Participants with secondary myopia, referring to high myopia resulting from other conditions or factors such as hyperglycemia in diabetes, were excluded in the study. Participants who received Defocus Incorporated Multiple Segments (DIMS) lenses, orthokeratology lenses, corneal refractive surgery, atropine interventions, repeated low-level red-light therapy, or intraocular surgery were excluded from the study. Furthermore, study participants with other ocular disorders (except for myopic maculopathy), and severe systematic diseases were excluded. This

study was ethically approved by the Ethics Committee of Zhongshan Ophthalmic Center and followed the Declaration of Helsinki (2012KYNL002). All participants provided written informed consent. Participants and their parents didn't receive any stipend or other incentive. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) reporting guidelines were followed.

Examinations

Comprehensive bilateral eye examinations were performed at baseline and the 4-year follow-up visits for all participants, following the same protocol. AL measurements were obtained before cycloplegia using either a Lenstar LS900 (Haag-Streit AG, Koeniz, Switzerland) or an IOL master (Carl Zeiss Meditec, Oberkochen, Germany) if the AL was longer than 32 mm. Intraocular pressure (IOP) was measured with a Goldmann application tonometer under topical anesthesia. Following complete cycloplegia, refraction was determined using a Topcon KR8800 autorefractor (eMethods in the Supplement). Best-corrected visual acuity (BCVA) was assessed using an ETDRS LogMAR E visual chart (Precision Vision, Villa Park, Illinois, USA) and converted to logMAR (eMethods in the Supplement). Fundus 45° photos of each fully dilated eye, including those centered on the macula and optic disc, were captured using a Canon camera (Canon CX-1, Tokyo, Japan).

Two drops of 0.5% tropicamide were performed twice, 5 minutes apart, to dilate the pupils of both eyes. After an additional 20 minutes, complete cycloplegia

was confirmed when the pupil diameter reached ≥ 6 mm and showed no reaction to light. If not, the third 0.5% tropicamide was performed to realize complete cycloplegia.

Definition and fundus images grading

The classification and grading of myopic maculopathy were established by ophthalmologists (ZXL, RL, OX, XXG) based on macula-centered fundus photography, following the meta-analyses of the pathologic myopia (META-PM) study classification.[12] Myopic maculopathy was divided into five categories: absence of myopic-related fundus lesions (C0), tessellated fundus (TF, C1), diffuse chorioretinal atrophy (DCA, C2), patchy chorioretinal atrophy (PCA, C3), and macular atrophy (C4). In addition, three “plus” lesions, including lacquer cracks (LCs), Fuchs spot, and choroidal neovascularization (CNV), were also evaluated. Diffuse chorioretinal atrophy of peripapillary and macular were both evaluated. The progression of myopic maculopathy was defined as the development of new categories of myopic maculopathy, including any new presence of “plus” lesions, the emergence of new categories, obvious enlargement of existing atrophic lesions, and an increasing number of existing “plus” lesions.

Initially, fundus photographs at baseline were graded, followed by the grading of fundus photographs after the 4-year follow-up. Side-by-side grading with knowledge of the previous assessment of baseline fundus photographs to assess the progression of myopic maculopathy. The median unweighted kappa

coefficients were 0.84 (range, 0.71-0.85) for C0/C1, 0.80 (range, 0.79-0.84) for C2 or more severe myopic maculopathy, and 0.73 (range, 0.72-0.79) for LCs and 0.75 (range, 0.71-0.83) for posterior staphyloma. A conclusive label of each eye was achieved after the council meeting if there were differences between graders.

Posterior staphyloma (PS), as defined by Curtin's classification[25], is observed as a local bulging of the sclera at the posterior pole of the eye that has a radius of less than the surrounding curvature of the wall of the eye. The presence of PS was determined by fundus color photography.

Statistical analysis

Statistical analyses were performed by Stata 17.0 (Stata Corp, College Station, TX, USA). Both eyes of each participant were included in the analysis for this study. The spherical equivalence (SE) of refractive error was calculated by summing the spherical error and half of the cylindrical error. The rates of AL elongation and SE progression were determined by calculating the differences between AL and SE at baseline and the 4-year follow-up, divided by the follow-up time. Continuous variables were presented as means and standard deviations (SD), while categorical variables were presented as numbers and percentages. Differences in the mean values and percentage were compared using the *t*-test and χ^2 test as appropriate for the variable. The Kruskal-Wallis test and Fisher's exact test were used to compare variables among different groups of lesion changes. Risk factors for the progression of myopic

maculopathy were assessed through univariable and multivariable logistic regression models. Odds ratios (ORs) and 95% confidence intervals (CIs) were reported. Variables with a *P*-value less than 0.1 in the univariable analysis were approved for inclusion in the multivariable analysis. All *P*-values were two-sided, and *P*-values were not adjusted for multiple analyses.

Results

A total of 426 participants were enrolled at the baseline visit. Of these, 149 participants declined to participate in the 4-year follow-up visit and were excluded. In addition, 1 participant went from correction with spectacles to implantable collamer lens (ICL) implantation in both eyes, and two participants went from correction with spectacles to Orthokeratology lenses in both eyes were excluded. Finally, 548 eyes from 274 participants (52.09%) were included in this analysis. Baseline characteristics, including age, sex, IOP, BCVA, AL, SE, myopic maculopathy category, and posterior staphyloma (PS), were presented in Table 1 for both participants and non-participants. No significant differences were observed between the two groups.

Table 1 listed the changes in the aforementioned characteristics of the participants over 4 years. At the baseline examination, mean age was 13.60±2.66 years, mean AL was 27.08±1.30 mm, mean SE was −9.12±2.46 D. After 4 years, mean age increased to 17.67±2.68 years, mean AL rose to 27.74±1.42 mm, mean SE progressed to −10.99±2.94 D. The prevalence of myopic maculopathy at baseline was as follows: 74.5% for C0, 11.3% for C1,

14.1% for C2, and 0.2% for C3. After a 4-year follow-up, the prevalence changed to 71.2% for C0, 12.2% for C1, 16.1% for C2, and 0.6% for C3. Additionally, PS was observed in 2.0% of participants at baseline, and there were no changes in subsequent years.

The progression of myopic maculopathy was detected in 67 of 548 eyes (12.2%) with 88 lesion changes (Table 2). Among these changes, the new development of C1 was observed in 16 eyes (from C0 to C1) (18.2%; 16/88) (Figure 1A, 1B). The progression of C2 included the new development of C2 in 12 eyes (13.6%) (from C0 to C2 in 1 eye and from C1 to C2 in 11 eyes) (Figure 1C, 1D), as well as the enlargement of C2 in 49 eyes (55.7%) (Figure 3A, 3B). New signs of C3 were identified in 1 eye transitioning from C0 to C3 and in 1 eye transitioning from C2 to C3 (2.3%) (Figure 2A, 2B). Furthermore, we observed the first appearance of LCs in 9 eyes (10.2%) (Figure 2C, 2D). Lesion changes exhibited variations in terms of IOP ($P = .03$), AL ($P < .001$), and SE ($P < .001$) at baseline (Table 2).

In the univariable model, the increased risks of myopic maculopathy progression were found in eyes with younger age, worse BCVA, longer AL, deeper SE, faster AL enlargement and SE progression, and more serious myopic maculopathy category. In the multivariable logistic regression model adjusting for covariates of $P < 0.1$ in the univariable model, worse BCVA (odds ratio [OR], 6.68; 95%CI, 1.15 to 38.99; P = .04), longer AL (OR, 1.73; 95%CI, 1.34 to 2.24; P < .001), faster AL elongation (OR, 302.83; 95%CI, 28.61 to

3205.64; P < .001), and more serious myopic maculopathy categories (diffuse atrophy, OR, 4.52; 95%CI: 1.98 to 10.30; P < .001; patchy atrophy, OR, 3.82; 95%CI, 1.66 to 8.80; P = .002) were associated with an increased risk of the presence of progression over 4 years (table 3). SE and rate of SE progression were excluded in the multivariable model due to their high correlation with AL (r = −0.67) and rate of AL elongation (r = −0.84).

Discussion

This study investigated the progression of myopic maculopathy in Chinese children and adolescents with high myopia over a 4-year follow-up period. The proportion of myopic maculopathy progression in our population was 12.2%. Enlargement of diffuse atrophy was the most frequent progressive change. Eyes with worse BCVA, longer AL, faster AL elongation, and a more severe category of myopic maculopathy had a higher likelihood of progression.

The 4-year incidence rate of myopic maculopathy progression in Chinese children and adolescents found in this study was 12.2%, which was lower compared with previous investigations in the elderly population, ranging from 19.6% to 58.6%.[13-16] A recent study conducted by Guo et al,[11] which included Chinese highly myopic children aged 4 to 17 years (mean age: 11.8 ± 2.5 years; mean SE: −7.65 ± 1.86 D) with a mean follow-up of 4.9 ± 1.2 years, reported that myopic maculopathy progressed in 52 of 274 eyes (18.9%). The difference in progression rates between Guo et al.'s study and our study may be attributed to variations in the follow-up duration and the definition of myopic maculopathy

progression. Furthermore, our findings of the 4-year cumulative progression of myopic maculopathy was lower than elderly population reported in studies, such as the Singapore Epidemiology of Eye Diseases (SEED) cohort study (6-year progression of 17.0% among 288 eyes aged 40 to 80 years)[17], the Blue Mountains Eye Study (5-year progression of 23.9% among 139 eyes aged > 40 years)[18], the Handan Eye Study (5-year progression of 35.3% among 51 eyes aged > 30 years)[19]. The difference in progression rate among these populations may be attributed to various definitions of myopic maculopathy, age groups, distributions of myopic maculopathy, and ethnicities.

In our study, we observed that the most common progression pattern of myopic maculopathy was the enlargement of diffuse atrophy, which is comparable with the Gutenberg Health Study[14] (32%), and the SEED study[17] (71.4%). However, the most common change of myopic maculopathy in Guo et al's study[11] was category from C1 to C2 in 44 of 139 eyes (31.9%). This may be the reason that Guo et al's study had a longer follow-up ranging from 4 to 8 years and a different part of the definition of myopic maculopathy progression, which included an increase from C0 or C1 to C2 or higher, and diffuse choroidal atrophy progression from peripapillary to macular. The most common change of myopic maculopathy in the Handan Eye Study[19] was new signs or enlargement of C3 in 11 of 51 eyes (21.6%). The reasons may be that both diffuse atrophy or lacquer cracks can progress into patchy atrophy, and patchy atrophy itself can also become larger over time.

Previous studies have shown that compared with individuals with stable fundus, those with myopic maculopathy progression were more likely to have worse BCVA, longer AL, faster AL elongation, and higher myopic SE.[13, 15, 19-23] Our results were largely consistent with these publications, except for older age. Interestingly, similar to another investigation focused on the young Chinese population[11], age did not show an association with the progression of myopic maculopathy in our study. This finding suggests that our study population may consist of two subgroups. The first subgroup might have early-onset myopia with a genetic basis, while the second subgroup may have developed high myopia after the age of 10 to 13 years due to environmental factors.[24] The younger age and the composition of high myopia in our participants could collectively contribute to the lack of correlation between age and myopic maculopathy progression.

We found that eyes categorized as myopic maculopathy category 1 and category 2 at the initial visit were at higher risk of myopic maculopathy progression than eyes with a norm fundus in the multivariate analyses. This finding differs from the SEED study[17], which reported that myopic maculopathy of category 3 or category 4 was at higher risk of myopic maculopathy progression in the adult population. This suggests that compared with adults, children and adolescents have a greater likelihood of progressing myopic maculopathy whenever a tessellated fundus was present. Therefore, it is imperative to closely monitor and implement proactive interventions for young

individuals who just have mild fundus lesions and are likely to deteriorate further. The strengths of our study included a relatively large, longitudinal design and the use of standardized methodologies to assess the progression of myopic maculopathy in Chinese children and adolescents with high myopia for 4 years. This study has several limitations. First, the proportion of non-participants can influence the results and their interpretation in this longitudinal epidemiologic study. The subgroup of highly myopic participants participating in our study and the subgroup of persons not participating were not distinctly different. These findings may suggest that there was no significant bias in the selection of participants for the present study. Second, peripheral staphylomas located outside of the posterior pole could be missed with our traditional imaging techniques. This limitation might result in an underestimation of the prevalence of staphylomas. Wide-field imaging systems are necessary in future studies. Third, 4 years of follow-up time is still relatively short for the pediatric population and insufficient to fully visualize the pattern of change in myopic maculopathy. Therefore, conducting a study with a longer follow-up duration is necessary. Fourth, the results obtained from our study may not be generalizable to other ethnicities and age groups due to the specific demographics of our study population, which consisted of Chinese individuals aged 7 to 17 years. Fifth, the graders were not masked to the baseline and 4-year follow-up images, and they conducted side-by-side grading. This method may introduce potential bias into the classification of myopic maculopathy. Sixth, the different lighting

conditions of fundus photos in the baseline and 4-year follow-up might bias the determination of myopic maculopathy progression. Seventh, red-free images analyses were lacking for comparison. However, the grading of myopic maculopathy was performed using the meta-analyses of pathologic myopia (META-PM) study classification, which had a limited impact on the assessment of myopic maculopathy progression.

To conclude, our findings indicate that over 4 years, the progression of myopic maculopathy was observed in 12.2% of children and adolescents with high myopia in China. The most common type of progression was the enlargement of diffuse atrophy. Additionally, eyes with worse BCVA, longer AL, faster AL elongation, and more severe myopic maculopathy were at a higher risk of myopic maculopathy progression. These findings support consideration of follow-up in these individuals and trying to identify those at higher risk for progression.

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Legends for Figure

Figure 1: Representative case of emergence of the tessellated fundus and diffuse atrophy. (A) Norm fundus in baseline fundus. (B) Newly developed tessellated fundus at 4-year follow-up visit. (C) Tessellated fundus in baseline fundus. (D) Newly developed diffuse atrophy at 4-year follow-up visit.

Figure 2: Representative case of emergence of patchy atrophy and lacquer cracks. (A) Tessellated fundus and diffuse atrophy in baseline fundus. (B) Newly developed patchy atrophy from lacquer cracks at 4-year follow-up visit. (C)Tessellated fundus and diffuse atrophy in baseline fundus. (D) Newly developed lacquer cracks with retinal hemorrhage (arrow) at 4-year follow-up visit.

Figure 3: Representative case of enlargement of diffuse atrophy. (A) Tessellated fundus and diffuse atrophy at baseline visit. (B) Diffuse atrophy with a significant expansion of the area at the 4-year follow-up visit.

Table 1. Characteristics at baseline and 4-year follow-up visit of non-participants and participants

	Non-participants	Participants		*P*
	Baseline visit	Baseline visit	4-year follow-up visit	
No. of eyes (%)	304 (35.68)	548 (64.32)	548	-
Age (years)	13.77±2.65	13.60±2.66	17.67±2.68	0.376[a]
Sex (N; %)				0.644[b]
Boys	158 (51.97)	272 (49.64)	272 (49.64)	
Girls	146 (48.03)	276 (50.36)	276 (50.36)	
IOP (mmHg)	15.36±2.19	15.45±2.45	15.72±2.71	0.606[a]
BCVA (log MAR)	0.09±0.16	0.07±0.14	0.12±0.17	0.058[a]
Better eyes	-	0.09±0.13	0.15±0.13	
Worse eyes	-	0.23±0.23	0.26±0.33	
AL (mm)	27.10±1.16	27.08±1.30	27.74±1.42	0.789[a]
SE (D)	−8.97±2.48	−9.12±2.46	−10.99±2.94	0.410[a]
Category of MM (N; %)				0.430[b]
C0	236 (77.63)	408 (74.45)	390 (71.17)	
C1	36 (11.84)	62 (11.31)	67 (12.23)	
C2	32 (10.53)	77 (14.05)	88 (16.06)	
C3	0	1 (0.18)	3 (0.55)	
C4	0	0		
Plus	0	0	9 (1.64)	
PS (N; %)	2 (0.66)	11 (2.01)	11 (2.01)	0.124[b]

Abbreviation: IOP, intraocular pressure; BCVA, best-corrected visual acuity; logMAR, logarithm of the minimum angle of resolution; AL, axial length; SE, spherical equivalent; D, diopter, MM, myopic maculopathy, PS, posterior staphyloma.

[a] Data of baseline visits of participants and non-participants are analyzed using a t-test.
[b] Data of baseline visits of participants and non-participants are analyzed using Pearson χ^2 tests.

Table 2. Comparison of characteristics in different lesion changes

	New C1	New C2	New C3	New LCs	Enlargement of C2	*P*
No. of eyes (%)	16 (18.2)	12 (13.6)	2 (2.3)	9 (10.2)	49 (55.7)	-
Age (years)	12.84±2.48	13.14±3.64	12.40±4.37	13.47±2.95	12.57±3.24	0.858[a]
Sex (N; %)						0.798[b]
Boys	9 (56.25)	6 (50)	1 (50)	6 (66.67)	22 (44.90)	
Girls	7 (43.75)	6 (50)	1 (50)	3 (33.33)	27 (55.10)	
IOP (mmHg)	14.47±2.42	16.08±1.68	19±0	17.22±1.92	16.02±2.46	0.031[a]
BCVA (logMAR)	0.07±0.13	0.19±0.17	0.17±0.18	0.15±0.14	0.17±0.15	0.113[a]
AL (mm)	26.66±0.78	27.75±1.36	28.87±1.46	30.54±1.54	28.74±1.58	<0.001[a]
Rate of AL elongation (mm/y)	0.26±0.11	0.26±0.16	0.31±0.22	0.27±0.18	0.27±0.14	0.930[a]
SE (D)	−8.96±2.59	−10.44±2.62	−13.44±2.03	−14.76±2.86	−12.72±3.24	<0.001[a]
Rate of SE progression (D/y)	−0.61±0.20	−0.55±0.43	−0.82±0.38	−0.76±0.46	−0.65±0.36	0.647[a]

Abbreviation: IOP, intraocular pressure; BCVA, best-corrected visual acuity; logMAR, logarithm of the minimum angle of
resolution; AL, axial length; SE, spherical equivalent; D, diopter.

[a] Data of characteristics in different lesion changes are analyzed using the Kruskal-Wallis test.

[b] Data of characteristics in different lesion changes are analyzed using Fisher's exact test.

Table 3. Comparison of characteristics and logistic analysis in high myopes of myopic maculopathy with and without progression

			Univariate		Multivariate[a]	
	Without progression (481 eyes; 87.8%)	With progression (67 eyes; 12.2%)	OR (95% CI)	*P*	OR (95% CI)	*P*
Age (years)	13.72±2.59	12.73±3.06	0.87 (0.80 to 0.96)	0.005	0.96 (0.84 to 1.09)	0.529
Sex (N; %)					-	-
Boys	239 (49.69)	33 (49.25)	-	-	-	-
Girls	242 (50.31)	34 (50.75)	1.02 (0.61 to 1.70)	0.947	-	-
IOP (mmHg)	15.42±2.44	15.66±2.51	1.04 (0.94 to 1.15)	0.461	-	-
BCVA (log MAR)	0.06±0.14	0.14±0.15	19.07 (4.24 to 85.87)	<0.001	6.68 (1.15 to 38.99)	0.035
AL (mm)	26.90±1.10	28.35±1.81	2.15 (1.75 to 2.63)	<0.001	1.73 (1.34 to 2.24)	<0.001
Rate of AL elongation (mm/y)	0.15±0.12	0.27±0.13	466.98 (60.95 to 3577.85)	<0.001	302.83 (28.61 to 3205.64)	<0.001
SE (D)	−8.73±1.98	−11.93±3.54	0.67 (0.60 to 0.74)	<0.001	-	-
Rate of SE progression (D/y)	−0.45±0.38	−0.64±0.33	0.35 (0.18 to 0.69)	0.002	-	-
Category of MM (N; %)						
0	387 (80.46)	21 (31.34)	-	-	-	-
1	48 (9.98)	14 (20.90)	5.38 (2.57 to 11.26)	<0.001	4.52 (1.98 to 10.30)	<0.001

2	45 (9.36)	32 (47.76)	13.10 (6.97 to 24.63)	<0.001	3.82 (1.66 to 8.80)	0.002
3	1 (0.21)	0	-	-	-	-
PS (N; %)	9 (1.87)	2 (2.99)	1.61 (0.34 to 7.63)	0.546	-	-

Abbreviation: IOP, intraocular pressure; BCVA, best-corrected visual acuity; logMAR, logarithm of the minimum angle of resolution; AL, axial length; SE, spherical equivalent; D, diopter, MM, myopic maculopathy, PS, posterior staphyloma.

[a] Multivariate logistic regression models include age, BCVA, AL, rate of AL elongation, and category of MM. SE and rate of SE progression were excluded because of the high correlation between AL and SE, rate of AL elongation, and rate of SE progression.