International Journal Dental and Medical Sciences Research
Volume 7, Issue 6, Nov – Dec 2025 pp 204-213 www.ijdmsrjournal.com ISSN: 2582-6018

Progression Pattern in Untreated Myopic Normal-Tension Glaucoma: Influence of Intraocular Pressure on RNFL Loss

Author: Dr. Punyaslok Mohanty
Co-Author: Dr. Prasant Kumar Nanda

Date of Submission: 05-12-2025 Date of Acceptance: 15-12-2025

ABSTRACT:

Précis

Although myopia is often considered a favourable prognostic factor in normal-tension glaucoma (NTG), a subset of untreated myopic NTG eyes showed significant progression within 2 years, even under low intraocular pressure (IOP).

Objective

To evaluate the natural course and risk factors for progressive retinal nerve fibre layer (RNFL) thinning in previously stable, treatment-naïve myopic NTG eyes.

Methods

A total of 111 myopic NTG eyes were included, all untreated for ≥1 year and without prior progression. RNFL thickness and visual fields were assessed every 6–12 months for >2 years. Eyes showing progressive structural or functional change formed the P(+) group; the remainder formed the P(–) group.

Results

Progression occurred in 25 eyes (22.5%). A family history of glaucoma, higher mean IOP, and higher maximum IOP were significantly associated with progression and with faster RNFL loss. No eye with a mean IOP <11 mmHg showed progression. A significant breakpoint at 14.2 mmHg ($P = 0.042$) was identified: above this level, RNFL thinning accelerated markedly ($R^2 = 0.252$, $P = 0.034$).

Conclusion

Untreated myopic NTG eyes—particularly those with a family history of glaucoma or higher IOP—face an increased risk of early progression. Early IOP-lowering treatment should be considered in high-risk patients even when clinical stability is initially observed.

I. INTRODUCTION

Optic nerve damage in normal-tension glaucoma (NTG) usually progresses more slowly than in primary open-angle glaucoma, where intraocular pressure (IOP) is high. In the Early Manifest Glaucoma Trial, 56% of early-stage NTG patients showed progression within six years without any IOP-lowering therapy. This suggests that nearly half of the untreated patients experienced little to no significant decline during that time.

Myopia has been considered a clinical feature that might differentiate NTG eyes with slower progression rates. Several studies indicate that myopia does not necessarily speed up glaucomatous damage. One study even found that some myopic patients with open-angle glaucoma showed no measurable worsening for up to seven years without IOP-lowering therapy. High myopia has also been viewed as a potential protective factor against glaucoma progression.

Despite this, NTG can still progress, sometimes quickly, and can eventually lead to blindness in some patients, including those with myopic eyes. Research shows that about 40–45% of myopic NTG eyes progress within a 3–8-year period, even when IOP-lowering treatment is given. A recent study involving untreated myopic NTG patients found visual field progression in 10.3% of cases after three years and 24.8% after five years.

Currently, lowering IOP is the only proven way to slow optic nerve damage in NTG. As a result, treatment usually starts right after diagnosis. However, this approach may not benefit individuals whose disease is less influenced by mechanical pressure factors. Given the often slow nature of NTG, some patients with mild, non-progressive cases might receive more treatment than needed, leading to unnecessary side effects, financial strain, and medication burden. As the belief grows that myopia may be linked to slower glaucoma progression, certain myopic NTG patients might also be undertreated. Yet, it is still unclear whether IOP-lowering therapy is necessary for all myopic NTG cases or if some patients could safely delay treatment.

International Journal Dental and Medical Sciences Research
Volume 7, Issue 6, Nov – Dec 2025 pp 204-213 www.ijdmsrjournal.com ISSN: 2582-6018

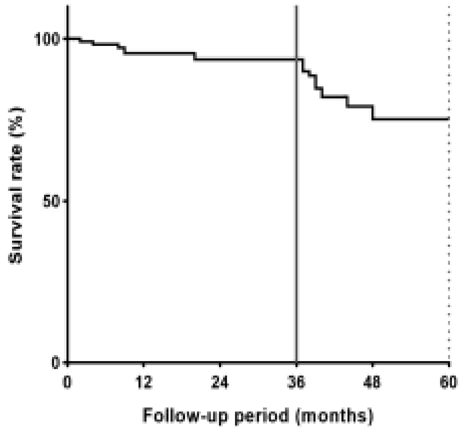

FIGURE1.Kaplan-Meiersurvivalcurveforglaucomaprogression. The overall survival rate was 93.7% at 3 years (solid line) and 75.2% at 5 years (dotted line). Patients who could not be followed up were processed as censored cases.

In this study, we looked back at Indian patients with myopic NTG who were not taking IOP-lowering medication for various reasons. Our goal was to find out how often disease progression occurred in this untreated group and to identify traits linked to a higher risk of decline. We specifically examined the role of IOP and the impact of IOP-lowering therapy in myopic NTG, aiming to better understand the IOP levels needed to prevent glaucomatous progression.

II. METHODS

Study Design

We conducted a retrospective, single-centred review of treatment-naïve, previously stable patients with myopic normal-tension glaucoma (NTG) seen at a tertiary health centre. Eligible patients had not used intraocular pressure (IOP)–lowering therapy for ≥1 year. The Institutional Review Board approved the protocol, and informed consent was waived because only anonymized data were analysed.

Examinations

All subjects underwent complete ophthalmic evaluation, including best-corrected visual acuity, Goldmann applanation tonometry, refraction, slit-lamp biomicroscopy, gonioscopy, stereoscopic disc photography, red-free fundus photography, keratometry, central corneal thickness, axial length (IOLMaster), spectral-domain OCT of the circumpapillary retinal nerve fibre layer (RNFL), and standard automated perimetry (Humphrey 24-2 SITA).

Eligibility

NTG was characterized by glaucomatous optic nerve damage, with or without visual field (VF) loss, open angles, and the highest measured IOP less than 21 mmHg during office hours. VF defects were confirmed with two reliable tests. Myopia was defined as an axial length of 24.0 mm or greater.

Inclusion criteria: age 18 years or older, best-corrected visual acuity of 20/40 or better, axial length between 24.0 and 29.0 mm, and no IOP-lowering therapy for at least one year after the NTG diagnosis.

Exclusion criteria: VF mean deviation (MD) worse than -12 dB, previous ocular trauma, intraocular surgery other than uncomplicated cataract extraction, retinal or neurologic diseases affecting the VF, or systemic medications that change IOP. If both eyes met the criteria, one was chosen at random.

Reasons for not receiving treatment included patient preference, medication intolerance, or clinician evaluation of low progression risk. This included stable findings for over one year, early VF damage, pretreatment IOP below 15 mmHg, or no family history of glaucoma.

Patients were monitored every 3 to 6 months for IOP measurement. OCT RNFL and 24-2 SAP tests were repeated every 6 to 12 months. At least five OCT scans were necessary to calculate RNFL thinning rates.

TABLE 1. Baseline Characteristics of the Study Subjects

	Total(n=111)	P(+)group(n=25)	P(-)group(n=86)	P
Demographic characteristics				
Age(y)	53.6±12.7	48.5±13.1	55.1±12.3	0.030
Sex(F),n(%)	47(42.3)	11(44.0)	36(41.9)	0.849
Family history of glaucoma,n(%)	19(17.1)	7(28.0)	12(14.0)	0.101
Systemic disease,n(%)	40(36.0)	7(28.0)	33(38.4)	0.342
Diabetes mellitus,n(%)	14(12.6)	3(12.0)	11(12.8)	0.917
Systemic hypertension,	32(28.8)	5(20.0)	27(31.4)	0.268

n(%)				
Heartdisease, n(%)	12(10.8)	1(4.0)	11(12.8)	0.213
Untreated follow-up duration(mo)	48.7±26.0	38.0±22.6	51.8±26.2	0.062
Clinical characteristics				
AXL(mm)	26.51±1.47	26.72±1.22	26.46±1.53	0.390
CCT(μm)	528.7±55.4	523.5±60.5	529.9±54.4	0.736
Mean IOP(mmHg)	12.4± 1.7	13.3± 1.4	12.1± 1.7	0.006
Maximum IOP(mmHg)	13.9± 2.1	15.1± 1.8	13.5± 2.1	0.002
IOP fluctuation (mmHg)	1.2±1.1	1.3±0.6	1.2±1.2	0.308
VFMDatbaseline(dB)	−2.27±3.01	−2.56±2.66	−2.18±3.14	0.351
Global RNFLT at baseline(μm)	78.8±12.2	82.1± 8.7	77.8±12.9	0.095
Rate of global RNFL thinning(μm/y)	−0.7±1.1	−1.7±1.5	−0.4±0.7	<0.001

Data are presented as mean ± SD or n(%).
Pvalues in bold face indicate statistical significance.
AXL indicates axial length; CCT, central corneal thickness; IOP, intraocular pressure; MD, mean deviation; RNFL, retinal nerve fiber layer; RNFLT, retinal nerve fiber layer thickness; VF, visual field.

TABLE 2.Comparison of RNFLT and VFMD at Baseline and Final Follow-Ups in Myopic NTG Eyes With and Without Glaucoma Progression

	P(+)group(n=25)	P(-)group(n=86)	P*
RNFLT			
Global RNFLT at baseline(μm)	82.1± 8.7	77.8±12.9	0.095
Global RNFLT at final follow-up(μm)	78.2± 8.8	76.2±13.3	0.456
P†	<0.001	<0.001	—
Amont of change in the RNFLT between follow-ups(μm)	−3.9±2.6	−1.6±3.0	<0.001
VFMD			
VFMD at baseline(dB)	−2.56±2.66	−2.18±3.14	0.351
VFMD at final follow-up(dB)	−2.16±3.231	−2.48±3.05	0.351
P†	0.338	0.109	—
Amount of change in the VFMD between follow-ups(dB)	0.40±1.86	−0.30±1.48	0.239

Data are presented as mean ± SD.
*Mann-Whitney Utest.
†Paired T test.
Pvalues in bold face indicate statistical significance.
MD indicates mean deviation; NTG, normal tension glaucoma; RNFLT, retinal nerve fiber layer thickness; VF, visual field.

Definition of Progression
Progression was determined by structural OCT RNFL thinning or functional VF deterioration.

OCT progression required RNFL thinning greater than test-retest variability (global >5 μm, superior or inferior >8 μm) on two consecutive scans. Two masked graders assessed progression, and any disagreements were resolved by a third reviewer.

VF progression required three identical points on the pattern deviation plot to show significant worsening in three consecutive tests compared to the baseline.

Eyes that showed progression were classified as P(+) and were recommended to start IOP-lowering treatment. The date of confirmed progression was used as the survival endpoint.

International Journal Dental and Medical Sciences Research
Volume 7, Issue 6, Nov – Dec 2025 pp 204-213 www.ijdmsrjournal.com ISSN: 2582-6018

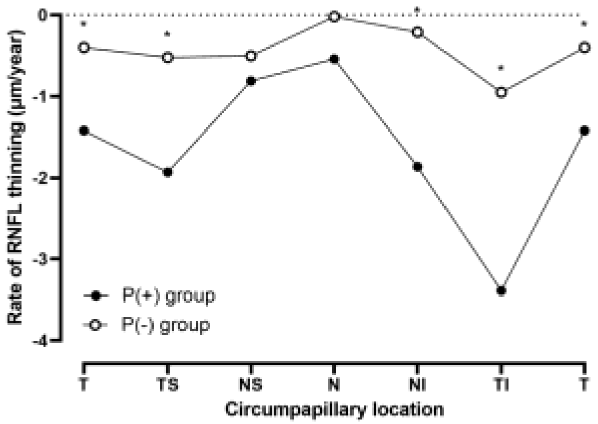

FIGURE 2. Rates of sectoral RNFL thinning in the progression[P (+)] and no-progression [P(–)] groups. "*" indicates significant differences.Nindicatesnasal;NI,nasal-inferior;NS,nasal- superior; RNFL, retinal nerve fiber layer; T, temporal; TI, temporal- inferior; TS, temporal-superior.

Statistical Analysis

Interobserver agreement for OCT progression was assessed using the kappa statistic. Kaplan–Meier curves were constructed for time to progression. Continuous variables were compared with independent t tests or Mann–Whitney U tests, and categorical variables with χ^2 tests. Cox proportional hazards models were used to identify predictors of progression. Linear regression estimated RNFL thinning rates and factors associated with thinning. Variables with P <0.10 in univariable analyses were included in multivariable models. The Davies test (R version 4.3.1) was used to detect structural breakpoints. Other analyses were performed with SPSS 27.0. Statistical significance was set at P <0.05.

TABLE 3.Factors Associated With Initiation of Medical Treatment Due to Glaucoma Progression in Treatment-Naïve Myopic NTG

	Uni variable analysis		Multi variable analysis1		Multi variable analysis 2	
	HR(95%CI)	P	HR(95%CI)	P	HR(95%CI)	P
Age(per1yolder)	0.970(0.941,1.000)	0.054	0.985(0.956,1.015)	0.331	0.987(0.957,1.019)	0.424
Sex(F)	1.162(0.527,2.563)	0.709	—	—	—	—
Family history of glaucoma	2.614(1.077,6.348)	0.034	3.308(1.341,8.162)	0.009	2.593(1.070,6.283)	0.035
Systemic disease	0.630(0.263,1.509)	0.300	—	—	—	—
Diabetes mellitus	0.948(0.283,3.179)	0.932	—	—	—	—
Systemic hypertension	0.645(0.241,1.729)	0.383	—	—	—	—
Heart disease	0.258(0.035,1.912)	0.185	—	—	—	—
AXL(per1mmlonger)	1.100(0.805,1.503)	0.549	—	—	—	—
CCT (per1µm thicker)	0.998(0.991,1.006)	0.622	—	—	—	—
Mean IOP(per1mmHg higher)	1.419(1.083,1.860)	0.011	1.495(1.131,1.976)	0.005	—	—
Maximum IOP (per1 mm Hg higher)	1.221(1.009,1.479)	0.040	—	—	1.207(1.009,1.443)	0.039
IOP fluctuation (per 1mm Hg larger)	0.864(0.545,1.372)	0.536	—	—	—	—
Global RNFLT at baseline (per	1.019(0.983,1.057)	0.30	—	—	—	—

International Journal Dental and Medical Sciences Research
Volume 7, Issue 6, Nov – Dec 2025 pp 204-213 www.ijdmsrjournal.com ISSN: 2582-6018

1μm thicker)						
VFMD at baseline (per 1dB higher)	0.984(0.870,1.113)	0.797	—	—	—	—

Variables with Pvalue<0.1 in the univariable analysis were included in the multi variable analysis.
Pvalues in bold face indicate statistical significance.

AXL indicates axial length; CCT, central corneal thickness; HR, hazard ratio; IOP, intraocular pressure; MD, mean deviation; NTG, normal tension glaucoma; RNFLT, retinal nerve fiber layer thickness; VF, visual field.

TABLE 4. Factors Associated With the Rate of Global RNFL Thinning in Treatment-Naïve Myopic NTG

	Univariable Analysis		Multi variable analysis 1		Multivariable analysis 2	
	β(95%CI)	P	β(95%CI)	P	β(95%CI)	P
Age (per1yolder)	0.019(0.003,0.034)	0.054	0.013(−0.002,0.029)	0.090	0.013(-0.002,0.029)	0.098
Sex (F)	−0.234(−0.647,0.180)	0.709	—	—	—	—
Family history of glaucoma	−0.624(−1.157,−0.092)	0.034	−0.605(−1.123,−0.086)	0.023	−0.574(−1.100,−0.048)	0.033
Systemic disease	0.226(−0.200,0.651)	0.300	—	—	—	—
Diabetes mellitus	0.268(−0.349,0.884)	0.932	—	—	—	—
Systemic hypertension	0.356(−0.093,0.804)	0.383	—	—	—	—
Heart disease	−0.226(−0.886,0.434)	0.185	—	—	—	—
AXL (per 1mm longer)	0.071(−0.081,0.224)	0.549	—	—	—	—
CCT(per 1μm thicker)	0.000(−0.004,0.004)	0.622	—	—	—	—
Mean IOP(per 1mmHg higher)	−0.168(−0.285,−0.052)	0.011	−0.170(−0.284,−0.056)	0.004	—	—
Maximum IOP(per 1mmHg higher)	−0.110(−0.205,−0.016)	0.040	—	—	−0.105(−0.198,−0.013)	0.026
IOP fluctuation(per 1mmHg larger)	0.030(−0.162,0.222)	0.536	—	—	—	—
Global RNFLT at baseline (per1μm thicker)	−0.009(−0.026,0.008)	0.307	—	—	—	—
VFMD at baseline (per 1dB higher)	−0.012(−0.083,0.060)	0.797	—	—	—	—

Variables with Pvalue<0.1 in the univariable analysis were included in the multivariable analysis.
Pvalues in bold face indicate statistical significance.
AXL indicates axiallength; CCT, central corneal thickness; IOP, intraocular pressure; MD, mean deviation; NTG, normal tension glaucoma; RNFL, retinalnerve fiber layer; RNFLT, retinal nerve fiber layer thickness; VF, visual field.

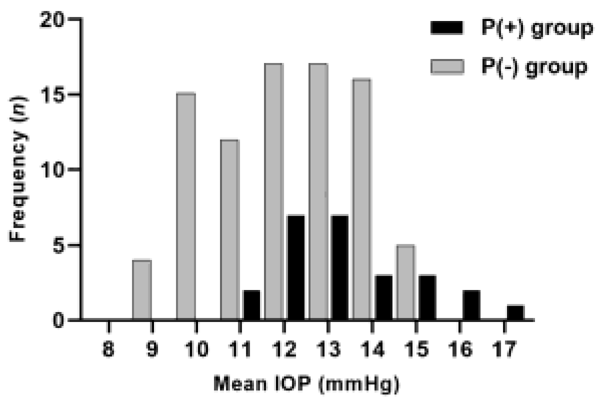

FIGURE 3. Distributions of the mean IOP in the progression [P(+)] and no-progression [P(–)] groups. None with a mean IOP<11 mmHg were in the P(+)group.IOP indicates intraocular pressure.

III. RESULTS

Study Population

A total of 111 myopic NTG eyes were included. Over a mean follow-up of 48.7 ± 26.0 months, 25 eyes (22.5%) demonstrated progression on OCT (n = 24) or visual field (VF) testing (n = 4) and were classified as P(+). Interobserver agreement for OCT progression was excellent (κ = 0.921). Kaplan–Meier analysis showed cumulative survival rates of 93.7% at 3 years and 75.2% at 5 years, with a mean survival time of 38.0 ± 22.6 months.

Baseline Characteristics

Table 1 summarizes baseline comparisons. Relative to P(–) eyes, the P(+) group was younger (48.5 ± 13.1 vs 55.1 ± 12.3 years, P = 0.030), had higher mean IOP (13.3 ± 1.4 vs 12.1 ± 1.7 mmHg, P = 0.006), higher peak IOP (15.1 ± 1.8 vs 13.5 ± 2.1 mmHg, P = 0.002), and faster RNFL loss (–1.7 ± 1.5 vs –0.4 ± 0.7 μm/year, P < 0.001). No significant differences were observed in axial length, central corneal thickness, IOP fluctuation, baseline global RNFLT, or baseline VF mean deviation (MD).

Longitudinal Structural and Functional Changes

Table 2 shows RNFLT and VF outcomes. Global RNFLT decreased significantly in both groups (P < 0.001), with greater thinning in the P(+) eyes (P < 0.001). VF MD remained stable in both groups (P = 0.338 and P = 0.109), with no intergroup difference at baseline or final follow-up (P = 0.239).

Sectoral analysis (Figure 2) demonstrated significantly faster RNFL thinning in the P(+) group in the temporal (P = 0.037), temporal-superior (P = 0.018), nasal-inferior (P = 0.002), and temporal-inferior (P = 0.003) regions.

Factors Associated With Progression

Risk factors for being in the P(+) group are summarized in Table 3. On univariable Cox analysis, family history of glaucoma, higher mean IOP, and higher maximum IOP were significant predictors. In multivariable models accounting for IOP interactions, family history (HR = 3.308 and 2.593), higher mean IOP (HR = 1.495), and higher maximum IOP (HR = 1.207) remained independently associated with progression.

Factors Associated With Faster RNFL Thinning

Linear regression results are shown in Table 4. Univariable analyses revealed that family history, higher mean IOP, and higher maximum IOP correlated with faster RNFL loss. In multivariable analyses, all three remained significant predictors:

- Family history: β = –0.605 and –0.574
- Mean IOP: β = –0.170
- Maximum IOP: β = –0.105

Relationship Between Mean IOP and RNFL Thinning

Figure 3 shows the distribution of mean IOP in both groups. Notably, no P(+) eyes had a mean IOP < 11 mmHg. Figure 4A demonstrates a significant linear relationship between mean IOP and the global RNFL thinning rate overall (R^2 = 0.058, P = 0.006). This relationship was absent in the P(–) group (R^2 < 0.000, P = 0.918), but strong in the P(+) group (R^2 = 0.157, P = 0.041; between-group slope difference, P = 0.048). No eye with a mean IOP < 11 mmHg showed progressive structural damage requiring initiation of IOP-lowering therapy.

Davies test identified a significant breakpoint at 14.2 mmHg (P = 0.042; Fig. 4B). Below this threshold, the slope was not significant (R^2 = 0.007, P = 0.382). Above the breakpoint, RNFL thinning increased significantly with higher mean IOP (R^2 = 0.252, P = 0.034; slope difference, P = 0.042).

Change in IOP and RNFL Thinning After Starting Treatment

To evaluate treatment effects, follow-up was extended in the P(+) group for an additional 29.8 ± 17.9 months. After initiation of IOP-lowering therapy, mean IOP, maximum IOP, and IOP fluctuation were all significantly reduced compared with pre-treatment levels (all P < 0.05; Table 5).

The global RNFL thinning rate improved from –1.7 ± 1.5 to –1.2 ± 1.2 μm/year, although this change did not reach statistical significance (P = 0.223). Despite this improvement, the rate remained significantly faster in P(+) eyes compared with P(–) eyes (–1.2 ± 1.2 vs –0.4 ± 0.7 μm/year, P < 0.001).

Figure 5 illustrates sectoral changes in the P(+) group. RNFL thinning slowed across most sectors after treatment, with a significant reduction in the temporal-superior sector (P = 0.038).

International Journal Dental and Medical Sciences Research
Volume 7, Issue 6, Nov – Dec 2025 pp 204-213 www.ijdmsrjournal.com ISSN: 2582-6018

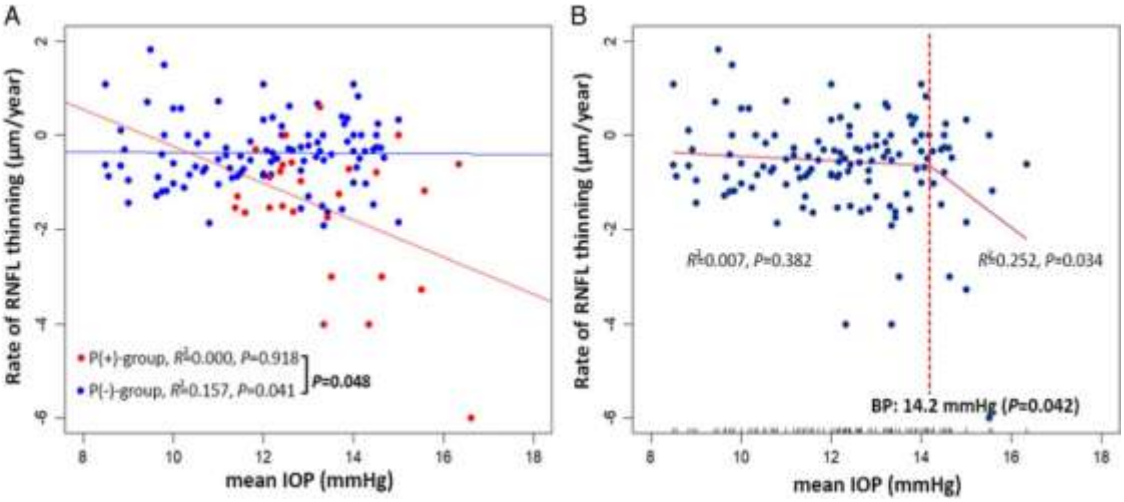

FIGURE 4. A, Relationship between the mean IOP and the rate of RNFL thinning in progression[P(+)] and no-progression [P(–)]groups. B, ABP in the relationship between the mean IOP and the rate of RNFL thinning identified by the Davies test. Rate of RNFL thinning significantly accelerated after the BP of 14.2mmHg.BP indicates break point; IOP, intraocular pressure; RNFL, retinal nerve fiber layer. Figure 4 can be viewed in color online at www.glaucomajournal.com.

TABLE 5.Changes of IOP and Rate of RNFL Thinning After Initiation of IOP-Lowering Treatment in P(+) Group (n=25)

	Before treatment	After treatment	P
Follow-up duration(mo)	38.0±22.6	19.8±17.9	0.006
Mean IOP(mmHg)	13.3± 1.4	12.1± 1.8	<0.001
Maximum IOP(mmHg)	15.1± 1.8	13.1± 2.3	0.001
IOP fluctuation(mmHg)	1.3±0.6	0.8±0.5	0.023
Rate of global RNFLT decrease(μm/y)	−1.7±1.5	−1.2±1.2	0.223

Data are presented as mean ± SD.
Pvalues in bold face indicate statistical significance.
IOP indicates intraocular pressure; RNFL, retinal nerve fiber layer; RNFLT, retinal nerve fiber layer thickness.

IV. DISCUSSION

The CNTGS described the natural course of untreated NTG, but that group included patients at different stages of the disease. This made treatment decisions tough for eyes that seemed stable at first. While myopia itself does not strongly relate to glaucoma progression in adults, myopic eyes can still worsen at low IOP levels. In this study of myopic NTG patients with at least one year of documented stability, 22.5% showed progression that needed treatment over about four years.

RNFL thinning was measurable even in the P(–) group, but the rate (–0.4 µm/y) was comparable to age-related decline. VF MD remained unchanged in both groups, likely reflecting early-stage disease where structural changes precede functional loss.

Consistent with prior work, IOP remained a key determinant of progression in myopic NTG. A significant breakpoint was identified at a mean IOP of 14.2 mmHg; above this value, RNFL loss accelerated. No eye with a mean IOP < 11 mmHg progressed, echoing previous reports that single-digit IOP is generally protective. These findings reinforce the pathogenic relevance of IOP even in myopic NTG but highlight the need to avoid excessively low IOP in highly myopic eyes due to the risk of hypotony maculopathy.

Treatment initiation in the P(+) group led to significant drops in mean, maximum, and fluctuating IOP. Even though sectoral RNFL thinning, especially in the temporosuperior region, slowed after treatment, global RNFL thinning still happened faster than in untreated stable eyes. This might be due to not having enough follow-up time, a small sample size, insufficient IOP reduction, or ongoing mechanical stress from earlier axial elongation in younger patients.

Family history came up as an extra risk factor for progression, in line with previous NTG studies. It’s unclear if this points to early detection patterns or genuine genetic risk, and more research is needed using modern tools like polygenic risk assessment.

The study has some limitations. It is retrospective, has a small sample size, and only includes early-stage patients without disc hemorrhage, which may affect how widely the results apply. We need prospective studies with larger and more diverse groups to confirm these findings.

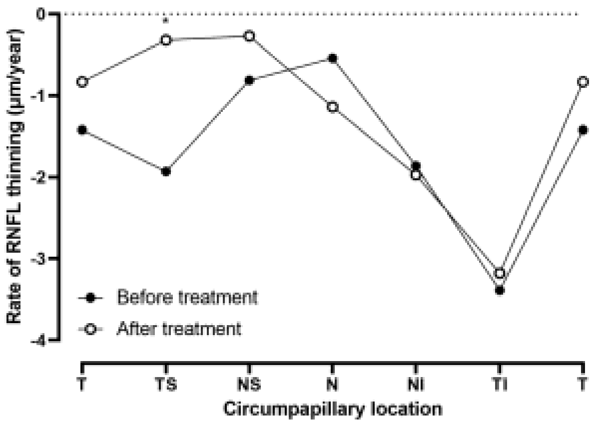

FIGURE 5.Changesin the ratesof sectoral RNFL thinning after the initiation of IOP-lowering treatment in the progression[P(+)] group. “*” indicates a significant difference. IOP indicates intraocular pressure; N, nasal; NI, nasal-inferior;NS,nasal- superior; RNFL, retinal nerve fiber layer; T, temporal; TI, temporal- inferior; TS, temporal-superior.

V. CONCLUSION

A notable proportion of myopic NTG eyes progressed despite prior stability without treatment. Maintaining a low IOP appeared to be the main factor associated with stability, while younger age and family history were associated with higher progression risk. Myopia should not be assumed protective, and treatment may need to be initiated earlier in myopic NTG eyes with higher-risk profiles, even when the disease initially appears stable.

REFERENCES

[1]. Heijl A, Bengtsson B, Hyman L, et al; Early Manifest Glaucoma Trial Group. Natural history of open-angle glaucoma. Ophthalmology. 2009;116:2271–2276.

[2]. Sohn SW, Song JS, Kee C. Influence of the extent of myopia on the progression of normal-tension glaucoma. Am J Ophthalmol. 2010;149:831–838.

[3]. Araie M, Shirato S, Yamazaki Y, et al. Risk factors for progression of normal-tension glaucoma under β-blocker monotherapy. Acta Ophthalmol. 2012;90:e337–e343.

[4]. Lee JY, Sung KR, Han S, et al. Effect of myopia on the progression of primary open-angle glaucoma. Invest Ophthalmol Vis Sci. 2015;56:1775–1781.

[5]. Doshi A, Kreidl KO, Lombardi L, et al. Nonprogressive glaucomatous cupping and visual field abnormalities in young Chinese males. Ophthalmology. 2007;114:472–479.

[6]. Collaborative Normal-Tension Glaucoma Study Group. Natural history of normal-tension glaucoma. Ophthalmology. 2001;108:247–253.

[7]. Sawada A, Rivera JA, Takagi D, et al. Progression to legal blindness in patients with normal-tension glaucoma: hospital-based study. Invest Ophthalmol Vis Sci. 2015;56:3635–3641.

[8]. Kim S, Sung KR. Factors associated with loss of visual function in medically treated advanced normal-tension glaucoma. Curr Eye Res. 2017;42:429–435.

[9]. Lee J, Ahn EJ, Kim YW, et al. Impact of myopia on the association of long-term intraocular pressure fluctuation with the rate of progression in normal-tension glaucoma. Br J Ophthalmol. 2021;105:653–660.

[10]. Hsueh C-M, Yeh J-S, Ho J-D. Effect of myopia on the progression of normal-

tension glaucoma. PLoS One. 2023;18:e0287661.
[11]. Lee K, Kim BY, Seong GJ, et al. Risk factors for the structural progression of myopic glaucomatous eyes with a history of laser refractive surgery. J Clin Med. 2021;10:2408.
[12]. Han JC, Han SH, Lee EJ, et al. Clinical course and risk factors for visual field progression in normal-tension glaucoma with myopia without glaucoma medications. Am J Ophthalmol. 2020;209:77–87.
[13]. Collaborative Normal-Tension Glaucoma Study Group. The effectiveness of intraocular pressure reduction in the treatment of normal-tension glaucoma. Am J Ophthalmol. 1998;126:498–505.
[14]. Collaborative Normal-Tension Glaucoma Study Group. Comparison of glaucomatous progression between untreated patients with normal-tension glaucoma and patients with therapeutically reduced intraocular pressures. Am J Ophthalmol. 1998;126:487–497.
[15]. Drance S, Anderson DR, Schulzer M; Collaborative Normal-Tension Glaucoma Study Group. Risk factors for progression of visual field abnormalities in normal-tension glaucoma. Am J Ophthalmol. 2001;131:699–708.
[16]. Sakata R, Yoshitomi T, Iwase A, et al. Factors associated with progression of Japanese open-angle glaucoma with lower normal intraocular pressure. Ophthalmology. 2019;126:1107–1116.
[17]. Leske MC, Heijl A, Hyman L, et al. Predictors of long-term progression in the early manifest glaucoma trial. Ophthalmology. 2007;114:1965–1972.
[18]. Forsman E, Kivelä T, Vesti E. Lifetime visual disability in open-angle glaucoma and ocular hypertension. J Glaucoma. 2007;16:313–319.
[19]. Oliver JE, Hattenhauer MG, Herman D, et al. Blindness and glaucoma: a comparison of patients progressing to blindness from glaucoma with patients maintaining vision. Am J Ophthalmol. 2002;133:764–772.
[20]. Chen PP. Risk and risk factors for blindness from glaucoma. Curr OpinOphthalmol. 2004;15:107–111.
[21]. Grant WM, Burke JF Jr. Why do some people go blind from glaucoma? Ophthalmology. 1982;89:991–998.
[22]. Lee J, Kong M, Kim J, et al. Comparison of visual field progression between relatively low and high intraocular pressure groups in normal tension glaucoma patients. J Glaucoma. 2014;23:553–560.
[23]. Leske MC, Wu S-Y, Hennis A, et al; Barbados Eye Studies Group. Risk factors for incident open-angle glaucoma: the Barbados Eye Studies. Ophthalmology. 2008;115:85–93.
[24]. Shin YI, Jeong Y, Huh MG, et al. Longitudinal evaluation of advanced glaucoma: ten year follow-up cohort study. Sci Rep. 2024;14:476.
[25]. Ratanawongphaibul K, Tsikata E, Zemplenyi M, et al. Earlier detection of glaucoma progression using high-density 3-dimensional spectral-domain OCT optic nerve volume scans. Ophthalmol Glaucoma. 2021;4:604–616.
[26]. Lee EJ, Lee D, Kim MJ, et al. Different glaucoma progression rates by age groups in young myopic glaucoma patients. Sci Rep. 2024;14:2589.
[27]. Leung CK, Yu M, Weinreb RN, et al. Retinal nerve fiber layer imaging with spectral-domain optical coherence tomography: a prospective analysis of age-related loss. Ophthalmology. 2012;119:731–737.
[28]. Lee YA, Shih YF, Lin LLK, et al. Association between high myopia and progression of visual field loss in primary open-angle glaucoma. J Formos Med Assoc. 2008;107:952–957.
[29]. Chihara E, Liu X, Dong J, et al. Severe myopia as a risk factor for progressive visual field loss in primary open-angle glaucoma. Ophthalmologica. 1997;211:66–71.
[30]. Quigley HA. Reappraisal of the mechanisms of glaucomatous optic nerve damage. Eye. 1987;1:318–322.
[31]. Cahane M, Bartov E. Axial length and scleral thickness effect on susceptibility to glaucomatous damage: a theoretical model implementing Laplace’s law. Ophthalmic Res. 1992;24:280–284.
[32]. Kim YW, Lee EJ, Kim TW, et al. Microstructure of β-zone parapapillary atrophy and rate of retinal nerve fiber layer thinning in primary open-angle glaucoma. Ophthalmology. 2014;121:1341–1349.

[33]. Naito T, Fujiwara M, Miki T, et al. Effect of trabeculectomy on visual field progression in Japanese progressive normal-tension glaucoma with intraocular pressure <15 mm Hg. PLoS One. 2017;12:e0184096.

[34]. Iverson SM, Schultz SK, Shi W, et al. Effectiveness of single-digit IOP targets on decreasing global and localized visual field progression after filtration surgery in eyes with progressive normal-tension glaucoma. J Glaucoma. 2016;25:408–414.

[35]. Schultz SK, Iverson SM, Shi W, et al. Achieving single-digit intraocular pressure targets with filtration surgery in eyes with progressive normal-tension glaucoma. J Glaucoma. 2016;25:217.

[36]. Aoyama A, Ishida K, Sawada A, et al. Target intraocular pressure for stability of visual field loss progression in normal-tension glaucoma. Jpn J Ophthalmol. 2010;54:117–123.

[37]. Koenig SF, Montesano G, Fang CEH, et al. Effect of trabeculectomy on the rate of progression of visual field damage. Eye. 2023;37:2145–2150.

[38]. Azuma K, Saito H, Takao M, et al. Frequency of hypotonic maculopathy observed by spectral domain optical coherence tomography in post glaucoma filtration surgery eyes. Am J Ophthalmol Case Rep. 2020;19:100786.

[39]. Park HYL, Shin DY, Jeon SJ, et al. Predicting the development of normal-tension glaucoma and related risk factors in normal-tension glaucoma suspects. Sci Rep. 2021;11:16697.

[40]. Craig JE, Han X, Qassim A, et al. Multitrait analysis of glaucoma identifies new risk loci and enables polygenic prediction of disease susceptibility and progression. Nat Genet. 2020;52:160–166.

[41]. Siggs OM, Han X, Qassim A, et al. Association of monogenic and polygenic risk with the prevalence of open-angle glaucoma. JAMA Ophthalmol. 2021;139:1023–1028.

[42]. Marshall HN, Mullany S, Han X, et al. High polygenic risk is associated with earlier initiation and escalation of treatment in early primary open-angle glaucoma. Ophthalmology. 2023;130:830–836.