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Baseline Macular Traction Modifies the Association Between Axial Length Shortening and ATN Progression After Posterior Scleral Contraction Running title: Traction Status and Posterior Scleral Contraction

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Abstract

Background

To investigate the association between intraoperative axial length (AL) shortening and atrophy-traction-neovascularization (ATN) progression after posterior scleral contraction (PSC), and to evaluate whether baseline macular traction status modifies this relationship.

Methods

This longitudinal retrospective cohort study included 115 eyes from 77 patients with pathologic myopia (PM) who received PSC between March 2013 and December 2024. Thirty-eight eyes (33.0%) had no macular traction (T = 0) and 77 eyes (67.0%) presented with baseline traction (T ≥ 1), with a median follow-up of 25 months. AL shortening was defined as the difference between baseline AL and AL measured within 4 hours after surgery, and was stratified into three groups: ≤ 1.5 mm (Group 1), 1.6–2.5 mm (Group 2), and > 2.5 mm (Group 3). Generalized estimating equations were applied to test the interaction between AL shortening and baseline macular traction status. The primary outcome was progression of any ATN component from baseline to the last follow-up.

Results

ATN progression occurred in 30 eyes (26.1%). AL shortening's association with ATN progression differed by baseline traction (interaction P = 0.014). For T = 0 eyes, greater AL shortening reduced ATN progression odds (OR, 0.46 per mm; 95% CI, 0.23–0.93; P = 0.030). For T ≥ 1 eyes, the association trended oppositely without significance (OR, 1.46 per mm; 95% CI, 0.82–2.60; P = 0.195). T = 0 eyes had falling progression rates across Groups 1–3 (42.9%, 23.8%, 0%; trend P = 0.095), while rates rose in traction-positive eyes (13.3%, 23.5%, 32.1%; trend P = 0.169). Exploratory analyses of T ≥ 1 severe PM eyes showed progression rising from 18.2% (Group 1) to 40.0% (Group 3). T-score improved with larger shortening, and N-score rose in Group 3, mainly in traction-positive eyes.

Conclusions

Baseline traction modifies the association between intraoperative AL shortening and post-PSC ATN progression. These findings support traction-guided AL shortening, requiring larger prospective validation.

Trial registration:

Not applicable.

Background

Pathologic myopia (PM) is a leading cause of irreversible vision loss worldwide, affecting up to 3% of the global population.[1] PM is defined as high myopia with diffuse choroidal atrophy or more severe lesions, or posterior staphylomas. The ATN classification offers a comprehensive framework for describing the structural and vascular abnormalities associated with PM.[2–4] It consists of three components: atrophy (A), traction (T), and neovascularization (N), each representing a distinct pathological pathway contributing to visual loss.[5] Clinically, PM eyes with A3 and/or T3 grades exhibit significantly poorer best-corrected visual acuity (BCVA) and longer axial lengths (AL). This observation has led to a clear definition: PM with an A grade of ≥ 3, a T grade of ≥ 3, or an N grade of ≥ 2 is classified as severe PM.[2] Eyes with A ≥ 3 or T ≥ 3 share key structural features, including severe choroidal atrophy, Bruch membrane–retinal pigment epithelium complex (BM-RPE) damage, outer retinal disruption, and, in high T grades, retinoschisis or foveal detachment. These features offer a clear explanation for the visual impairment observed in eyes with high-grade PM.[3]

Myopic traction maculopathy (MTM) is a major vision-threatening complication of PM.[6] Without timely treatment, it often progresses to severe conditions and permanent visual loss.[7] At T3 or higher, it damages the photoreceptor layer and BM-RPE complex, driving irreversible structural degeneration and visual impairment.[5] Early surgical intervention before severe traction development may facilitate outer retinal structural recovery.[8] It also supports long-term preservation of vision. Thus, the baseline T grade may modify treatment response.

Posterior scleral contraction (PSC) surgery, a modified posterior scleral reinforcement technique, aims to reinforce the weakened sclera while exerting an inward force on the ocular wall to counteract posterior expansion.[9–11] Multiple studies have confirmed that PSC can control AL progression and facilitate retinal reattachment.[12–14] It also mitigates ATN classification deterioration.[10] However, previous studies on macular buckling surgery have suggested that excessive contraction may induce adverse effects, such as damage to the BM-RPE complex.[15–17] Conflicting reports exist on whether greater AL shortening consistently translates into superior anatomical and functional results.[13,15,18] Moreover, the dose-response relationship between AL shortening and long-term structural and functional stability has yet to be fully elucidated.

We hypothesized that the relationship between long-term ATN outcomes and the magnitude of AL shortening is non-uniform and may be modified by baseline ATN status, particularly baseline macular traction. The present study aimed to evaluate this association and to explore clinically relevant ranges of intraoperative AL shortening for PM patients with and without baseline tractional changes.

Methods

Study Design and Participants. This single-center, longitudinal, retrospective cohort study was performed at Tianjin Medical University Eye Hospital and included patients treated between March 2013 and December 2024. All clinical data were retrospectively retrieved from the hospital electronic medical record system. The study protocol complied with the principles of the Declaration of Helsinki and was approved by the Institutional Review Board (2026KY-47). All patients provided written informed consent before undergoing the surgical procedure.

Ocular examinations

All patients underwent comprehensive ocular examinations, including slit-lamp microscopy, fundus examination after mydriasis, intraocular pressure (IOP) measurement (CT-80 non-contact tonometer, Topcon Corporation, Tokyo, Japan), manifest refraction to determine the BCVA, wide-angle fundus photography (Optos, Daytona, Scotland), macular-centered 9-mm range OCT in star mode (RTVue, Optovue, USA, or VG200S, Version 1.42.6, Intalight, Henan, China), and B-mode ultrasound examination (UD-8000, 142 Tomey, Japan; or Cine Scan, Quantel Medical, France). Ocular biometric parameters were measured using the IOL-Master (Model 500; Zeiss, Oberkochen, Germany), including AL, central corneal thickness (CCT), anterior chamber depth (AD), lens thickness (LT), and corneal flat and steep curvatures (K1 and K2).

PM was defined according to the ATN classification system.[4] Patients were diagnosed with PM upon meeting both of the following criteria: (1) AL of ≥ 26.5 mm accompanied by posterior staphyloma; and (2) the presence of at least one ATN-related finding: diffuse choroidal atrophy or advanced choroidal atrophy (≥ A2), macular retinoschisis (≥ T1), lacquer cracks, active choroidal neovascularization, or macular scars (≥ N1). In the current study, active choroidal neovascularization was excluded from the surgical criteria.

Severe PM was defined as eyes meeting either of the following criteria: (1) A grade or T grade ≥ 3; (2) N grade ≥ 2.[2]

ATN Grading Criteria

Two experienced ophthalmologists (WYX and LYF) independently graded PM severity according to the ATN classification system. Disagreements between graders were resolved by consensus (CYH). Grade A (atrophy): 0–4, based on the severity of myopic atrophy, ranging from tessellated fundus and diffuse atrophy to patchy atrophy, with A4 indicating macular atrophy that involves the fovea; Grade T (traction): 0–5, based on the presence and severity of MTM, retinoschisis, macular hole, and retinal detachment; Grade N (neovascularization): 0–2, based on the presence of active neovascularization or macular scars.

To be eligible for the study, participants must have satisfied the above-noted PM diagnostic criteria, along with additional requirements: availability of preoperative and postoperative ATN grading and AL measurements; clear wide-field fundus photographs and macular OCT images obtained preoperatively

and at final follow-up; and a minimum follow-up period of 12 months. If both eyes met the inclusion criteria, both were enrolled in the study.

Exclusion criteria: (1) Ocular trauma, history of intraocular surgery (excluding uncomplicated cataract surgery), prior scleral surgery, or external scleral buckle/cerclage; (2) Active choroidal neovascularization; (3) Ocular fundus diseases unrelated to PM (e.g., retinal vascular diseases, glaucoma); (4) Autoimmune diseases (e.g., systemic lupus erythematosus, collagen diseases); (5) Inability to calculate intraoperative AL shortening or perform ATN grading due to missing data.

PSC Surgical Procedure.

All surgeries were performed under general anesthesia by one experienced surgeon. The PSC procedure has been described in previous studies.[9–11,13] A cross-linked donor human dural graft was placed over the posterior pole and sutured to the host sclera.

A 270° conjunctival peritomy was created. Traction sutures were placed under the lateral and inferior rectus muscles to rotate the globe superonasally. The inferior oblique muscle was identified to expose the posterior surgical field. A spindle-shaped graft was inserted between the inferior oblique muscle and the optic nerve, with its widest portion aligned to the macular region. The graft's upper and lower ends were fixed 2–3 mm posterior to the insertion sites of the superior rectus and medial rectus muscles, respectively. Anterior chamber paracentesis was performed using a 1 mL syringe to release an appropriate volume of aqueous humor. This procedure compensated for scleral contraction-induced ocular volume reduction and prevented intraoperative IOP elevation.

AL shortening was calculated as the AL difference between pre- and post-PSC (within 4 hours) using the same biometric device. The surgical target for AL shortening was determined by surgeons based on preoperative biometric parameters, the severity of tractional lesions and patient age. For patients without traction, AL shortening was controlled within the range of 0.5–2.0 mm, and for those with traction, AL shortening was appropriately increased according to the height of retinoschisis.

Postoperative Follow-up

Patients were followed according to the institutional postoperative protocol. Follow-up examinations included BCVA assessment, slit-lamp examination, IOP measurement, wide-field fundus photography, macular OCT, B-mode ultrasound examination, and ocular biometry when available. The minimum follow-up duration required for inclusion was 12 months. The final follow-up was defined as the last postoperative visit with gradable wide-field fundus photographs and macular OCT images. ATN grading at the final follow-up was compared with baseline grading to determine ATN progression.

Outcome Definition

The primary exposure was the magnitude of intraoperative AL shortening, defined as the difference between baseline AL and AL measured within 4 hours after PSC surgery.

The primary outcome was ATN progression from baseline to the final follow-up. ATN progression was defined as an increase of at least 1 grade in any ATN component, including the A, T, or N score.

Secondary outcomes included changes in individual A, T, and N scores from baseline to the final follow-up. Improvement in a component score was analyzed descriptively but was not counted as ATN progression.

Statistical Analysis.

All statistical analyses were performed using SPSS 27 (IBM Corp, Armonk, NY, USA) and Python 3.12. Statistical significance was set at a two-sided α of 0.05. Continuous variables were assessed for distribution using the Shapiro-Wilk test and are presented as mean ± standard deviation or median (interquartile range [IQR]), as appropriate. Group comparisons were performed using the independent samples t-test or the Mann-Whitney U test. Categorical variables are presented as counts (percentages) and were compared using the chi-square or Fisher's exact test, as appropriate.

Generalized estimating equations.

The primary analysis examined whether baseline traction status modified the association of intraoperative AL shortening with ATN progression. Generalized estimating equations (GEE) with a binomial family, logit link, exchangeable working correlation structure, and robust sandwich standard errors were used. Patient identity was used as the clustering variable to account for correlation between fellow eyes. The primary model included AL shortening, baseline traction status (T = 0 vs. T ≥ 1), and their interaction term. Because of the limited number of progression events, the multivariable model was kept parsimonious. Covariates were selected a priori based on clinical relevance and model stability, and the final model adjusted for baseline AL, baseline BCVA, and IOP. A main-effects-only model without the interaction term was fitted for comparison. The conditional effect of AL shortening in the T ≥ 1 subgroup was estimated using the delta method: OR = exp (βAL + βinteraction). Given only 11 events in the T = 0 subgroup, independent multivariable GEE models within each T stratum were not performed.

Dose-Response Categorization

For descriptive dose-response analyses, AL shortening was categorized as ≤ 1.5 mm (Group 1), 1.6–2.5 mm (Group 2), and > 2.5 mm (Group 3). These categories reflected clinically used ranges of intraoperative shortening in PSC surgery. Cochran-Armitage trend tests were used as exploratory analyses to assess ordered trends in ATN progression across AL shortening categories.

Nonlinearity Assessment.

Nonlinearity was explored by adding a quadratic term for AL shortening and its interaction with traction status to the full-sample GEE model. A joint Wald test was used to assess the quadratic terms.

Exploratory Subgroup Analyses.

Post hoc exploratory subgroup analyses were performed after stratifying eyes by severe PM status. Within the T ≥ 1 subgroup, ATN progression rates were compared across AL shortening categories according to severe PM status. Given the limited number of non-severe PM eyes in the T ≥ 1 subgroup (n = 19, three events), further subgroup analysis was restricted to severe PM eyes within T ≥ 1 (n = 58, 16 events).

Missing Data and Multiple Testing

Core variables, including ATN score, AL shortening, BCVA, baseline AL, age, sex, IOP, and follow-up duration, had no missing values; therefore, no imputation was performed. Descriptive statistics for ocular biometric variables with missing data (AD 10.4%, LT 13.0%, K1/K2/Astigmatism 9.6%) were computed for available cases only. No formal adjustment for multiple comparisons was applied; subgroup, nonlinearity, and trend analyses were considered exploratory.

Results

A total of 150 patients (227 eyes) undergoing PSC were initially retrieved from the electronic medical record system between March 2013 and December 2024. After applying the exclusion criteria, 115 eyes from 77 patients with PM were included in the analysis (Fig. 1). Among them, there were 22 males (28.6%) and 55 females (71.4%). The median age was 58 years (IQR, 49–64; range, 18–73 years), with a median follow-up duration of 25.00 months (IQR, 14.50–55.00; range, 12–119 months). No significant differences were observed in the baseline AL, BCVA, IOP, CCT, age, or sex between the excluded eyes and the included eyes. Detailed data are shown in Additional File 1.

Baseline Characteristics

Of 115 eyes (77 patients), 38 eyes (33.0%) had no preoperative traction (T = 0), and 77 (67.0%) had traction (T ≥ 1). The T ≥ 1 group was older and had worse baseline BCVA and a higher baseline ATN total score than the T = 0 group. Among the ocular biometric parameters, corneal astigmatism was significantly higher in T ≥ 1 (median 1.22 vs. 0.82 D, $P = 0.002$), while CCT, AD, LT, K1, and K2 did not differ significantly. Baseline AL, IOP, sex, and eye laterality showed no significant differences. See Table 1.

Table 1
Baseline Characteristics by Preoperative Traction Status

Characteristic	Overall (n = 115)	T = 0 (n = 38)	T ≥ 1 (n = 77)	P Value
Age, years	58.00 (49.00–64.00)	54.00 (34.00–61.75)	60.00 (51.00–65.00)	0.013
Female sex, n (%)	87 (75.65)	25 (65.79)	62 (80.52)	0.134
Right eye, n (%)	54 (46.96)	17 (44.74)	37 (48.05)	0.891
Baseline AL, mm	30.54 ± 1.76	30.38 ± 1.68	30.61 ± 1.81	0.512
Long-term AL change (baseline − final follow-up), mm	1.42 ± 0.98	0.97 ± 0.85	1.65 ± 0.96	< 0.001
Baseline BCVA, logMAR	0.72 (0.30–1.30)	0.52 (0.22–1.00)	0.82 (0.40–1.30)	0.039
BCVA change (final − baseline), logMAR	-0.08 (-0.37–0.05)	-0.05 (-0.30–0.03)	-0.10 (-0.43–0.07)	0.768
Baseline IOP, mmHg	14.96 ± 2.78	14.36 ± 2.47	15.25 ± 2.89	0.104
CCT, μm	531.00 (510.00–558.00)	533.00 (502.25–551.75)	529.00 (514.00–561.00)	0.325
AD, mm	2.93 (2.71–3.23)	2.99 (2.69–3.35)	2.91 (2.71–3.16)	0.686
LT, mm	4.44 (4.03–4.71)	4.23 (3.88–4.69)	4.46 (4.17–4.72)	0.402
K1, D	43.64 (42.31–44.64)	43.86 (41.89–44.75)	43.56 (42.44–44.50)	0.962
K2, D	44.86 (43.55–45.83)	44.67 (42.71–45.77)	45.02 (43.62–46.09)	0.230
Astigmatism (K2 − K1), D	1.11 (0.67–1.62)	0.82 (0.51–1.15)	1.22 (0.82–1.75)	0.002
AL shortening, mm	2.01 (1.50–2.61)	1.89 (1.08–2.11)	2.18 (1.67–2.86)	0.002
Follow-up, months	25.00 (14.50–55.00)	28.00 (15.00–49.00)	24.00 (14.00–56.00)	0.795

Data are presented as mean ± SD, median (IQR), or n (%), as appropriate. Between-group comparisons were performed using the independent-samples t test, Mann-Whitney U test, chi-square test, or Fisher exact test, as appropriate. Negative BCVA change indicates visual improvement. AL = axial length; BCVA = best-corrected visual acuity; IOP=intraocular pressure; CCT = central corneal thickness; AD=anterior chamber depth; LT=lens thickness; K1/K2 = corneal flat and steep curvatures; IQR = interquartile range;

SD = standard deviation. Missing data in the overall cohort were 10.4% for AD, 13.0% for LT, 9.6% for K1/K2/astigmatism, and 0% for CCT.

Baseline ATN Component Scores and Severe PM Proportion

The T ≥ 1 group had a higher proportion of severe PM (75.3% vs. 47.4%, *P* = 0.004). ATN progression rates were similar: 29.0% in T = 0 vs. 24.7% in T ≥ 1 (*P* = 0.656). No significant intergroup differences were observed in the A scores, N scores, or the distribution of A and N grades. A ≥ 3 prevalence was 47.4% and 57.2% in the T = 0 and T ≥ 1 groups, respectively; T ≥ 3 prevalence was 40.3% in the T ≥ 1 group. Detailed data are shown in Additional File 2.

AL Shortening Distribution

The distribution of intraoperative AL shortening differed by baseline traction status (Fig. 2). In the overall cohort, AL shortening ranged from 0 to 5.20 mm, with a median of 2.01 mm (IQR, 1.50–2.61 mm). Eyes with baseline traction (T ≥ 1) had greater AL shortening than eyes without baseline traction (T = 0) (median, 2.18 vs. 1.89 mm; *P* = 0.002). In the T ≥ 1 group, 15 eyes (19.5%), 34 eyes (44.2%), and 28 eyes (36.4%) were classified into Groups 1, 2, and 3, respectively. In the T = 0 group, the corresponding numbers were 14 eyes (36.8%), 21 eyes (55.3%), and 3 eyes (7.9%). Most eyes without baseline traction were classified into Groups 1 and 2, whereas eyes with baseline traction were distributed across all three groups.

Interaction between AL Shortening and Traction Status

In univariate GEE models, baseline AL (OR, 1.46 per mm; 95% CI, 1.10–1.95; *P* = 0.010), baseline BCVA (OR, 1.10 per 0.1 logMAR; 95% CI, 1.02–1.19; *P* = 0.018), and baseline IOP (OR, 0.87 per mmHg; 95% CI, 0.76–1.01; *P* = 0.068) showed associations with ATN progression. Baseline traction status and AL shortening were not significantly associated with ATN progression when modeled as main effects alone. Given the prespecified interaction hypothesis and the limited number of progression events, the multivariable GEE model was kept parsimonious and adjusted for baseline AL, baseline BCVA, and IOP. Detailed data are shown in Additional File 3.

In the multivariable GEE model incorporating the AL shortening × baseline traction status interaction (Table 2 and Fig. 3), the interaction was statistically significant (OR, 3.18 per 1-mm AL shortening in eyes with T ≥ 1 vs. T = 0; 95% CI, 1.27–7.98; *P* = 0.014). Among eyes without baseline traction (T = 0), greater AL shortening was associated with lower odds of ATN progression (OR, 0.46 per mm; 95% CI, 0.23–0.93; *P* = 0.030). Among eyes with baseline traction (T ≥ 1), the estimated association was in the opposite direction but was not statistically significant (OR, 1.46 per mm; 95% CI, 0.82–2.60; *P* = 0.195). Baseline AL (OR, 1.42 per mm; 95% CI, 1.04–1.95; *P* = 0.030) and baseline BCVA (OR, 1.12 per 0.1 logMAR; 95% CI, 1.01–1.23; *P* = 0.025) remained independently associated with ATN progression. Baseline IOP showed a marginal inverse association (OR, 0.84 per mmHg; 95% CI, 0.70–1.00; *P* = 0.055).

Table 2
Multivariable GEE Model for ATN Progression (N = 115 eyes, 77 patients)

Variable	OR	95% CI	P Value
T ≥ 1 (vs. T = 0)	0.94	0.32–2.80	0.913
AL shortening (per mm, at T = 0)	0.46	0.23–0.93	0.030
AL shortening × T ≥ 1 interaction	3.18	1.27–7.98	0.014
Baseline AL (per mm)	1.42	1.04–1.95	0.030
Baseline BCVA (per 0.1 logMAR)	1.12	1.01–1.23	0.025
Baseline IOP (per mmHg)	0.84	0.70–1.00	0.055

The model used generalized estimating equations with a binomial distribution, logit link, exchangeable working correlation structure, robust sandwich standard errors, and patient identity as the clustering variable. The final model adjusted for baseline AL, baseline BCVA, and IOP. T = 0 indicates eyes without baseline macular traction, and T ≥ 1 indicates eyes with baseline macular traction according to the ATN classification. In the interaction model, the OR for T ≥ 1 versus T = 0 represents the conditional association when AL shortening is 0 mm. The conditional OR for AL shortening in eyes with T ≥ 1 was calculated as exp (βAL shortening + βinteraction). GEE = generalized estimating equations; ATN = atrophy–traction–neovascularization; AL = axial length; BCVA = best-corrected visual acuity; IOP = intraocular pressure; OR = odds ratio; T = traction; CI = confidence interval.

Nonlinearity Assessment.

No clear nonlinear association was detected in the continuous AL shortening model. Neither the quadratic term for AL shortening nor its interaction with traction status was statistically significant, and the joint Wald test for both quadratic terms was non-significant ($\chi^2 = 2.10$; $P = 0.350$).

Supplemental GEE Analysis of Main-Effects-Only Model

A main-effects-only GEE model without the interaction term was fitted for comparison. In this model, AL shortening was not associated with ATN progression (OR, 0.97 per mm; 95% CI, 0.60–1.58; $P = 0.916$). This finding differed from the traction-stratified estimates in the interaction model. Given the limited number of events in the T = 0 subgroup (11 events among 38 eyes), independent multivariable GEE models within each traction stratum were not performed. Detailed results are provided in Additional File 4.

Dose-Response Analysis by AL Shortening Categories

In the T = 0 group, ATN progression rates declined across increasing AL shortening categories: 42.9% (6/14) in Group 1, 23.8% (5/21) in Group 2, and 0.0% (0/3) in Group 3 (Cochran-Armitage Z = − 1.67; P =

0.095). Because only three eyes in the T = 0 group were in Group 3, this finding should be interpreted cautiously.

In the T ≥ 1 group, ATN progression rates increased across AL shortening categories: 13.3% (2/15) in Group 1, 23.5% (8/34) in Group 2, and 32.1% (9/28) in Group 3 (Z = 1.38; *P* = 0.169). Although the trend tests did not reach statistical significance, the direction of these category-based results was consistent with the interaction model. Representative cases from selected AL shortening categories are shown in Fig. 4.

Exploratory Analysis by Severe PM Status

Among eyes with T ≥ 1, 58 of 77 eyes (75.3%) had severe PM. In eyes with severe PM, ATN progression rates increased with greater AL shortening: 18.2%, 22.2%, and 40.0% in Groups 1, 2, and 3, respectively (Z = 1.45; *P* = 0.148). Among non-severe PM eyes with T ≥ 1, only three progression events occurred, with an overall progression rate of 15.8% (3/19), precluding reliable category-based analysis. See Table 3.

Table 3
ATN Progression Rates by AL Shortening Category, Traction Status, and Severe PM

AL shortening category	**T = 0 (N = 38, 11 events)**	**T ≥ 1 overall (N = 77, 19 events)**	**T ≥ 1, Non-severe PM (N = 19, 3 events)**	**T ≥ 1, Severe PM (N = 58, 16 events)**	**overall (N = 115, 30 events)**
Group 1	6/14 (42.9%, 21.4–67.4)	2/15 (13.3%, 3.7–37.9)	0/4 (0.0%, 0.0–49.0)	2/11 (18.2%, 5.1–47.7)	8/29 (27.6%, 14.7–45.7)
Group 2	5/21 (23.8%, 10.6–45.1)	8/34 (23.5%, 12.4–40.0)	2/7 (28.6%, 8.2–64.1)	6/27 (22.2%, 10.6–40.8)	13/55 (23.6%, 14.4–36.3)
Group 3	0/3 (0.0%, 0.0–56.1)	9/28 (32.1%, 17.9–50.7)	1/8 (12.5%, 2.2–47.1)	8/20 (40.0%, 21.9–61.3)	9/31 (29.0%, 16.1–46.6)
Total	11/38 (28.9%, 17.0–44.8)	19/77 (24.7%, 16.4–35.4)	3/19 (15.8%, 5.5–37.6)	16/58 (27.6%, 17.8–40.2)	30/115 (26.1%, 18.9–34.8)

Data are shown as n/N (%) with Wilson 95% confidence intervals. Events indicate eyes with ATN progression, defined as progression in any A, T, or N component from baseline to final follow-up. Group 1 indicates intraoperative AL shortening ≤ 1.5 mm; Group 2, 1.6–2.5 mm; and Group 3, > 2.5 mm. T = 0 indicates eyes without baseline macular traction, and T ≥ 1 indicates eyes with baseline macular traction according to the ATN classification. Severe PM was defined as meeting any of the following ATN thresholds: A ≥ 3, T ≥ 3, or N ≥ 2. AL = axial length; ATN = atrophy–traction–neovascularization; PM = pathologic myopia.

An additional multivariable GEE model including both traction status and severe PM showed that the AL shortening × traction status interaction remained statistically significant (OR, 2.88; 95% CI, 1.17–7.09; *P*

= 0.022). The AL shortening × severe PM interaction was not statistically significant (OR, 2.10; 95% CI, 0.82–5.38; *P* = 0.123). The limited number of events in this subgroup precluded a reliable stratified statistical analysis.

ATN Component Changes from Baseline to Final Follow-up

Changes in ATN component scores from baseline to final follow-up are shown in Fig. 5. In the overall cohort, total ATN score change decreased with greater AL shortening, mainly driven by improvement in T-score. A-score change remained relatively stable. N-score change was close to neutral in Groups 1 and 2 but increased in Group 3.

In eyes without baseline traction (T = 0), total ATN score change decreased across AL shortening groups, although the Group 3 estimate should be interpreted cautiously because only three eyes were included. T-score change remained relatively stable. In eyes with baseline traction (T ≥ 1), T-score improved across all AL shortening groups, whereas N-score change increased from Group 1 to Group 3.

Postoperative Complications

Postoperative conjunctival congestion was common within the first 3 days and resolved spontaneously within 7 days. Transient IOP elevation occurred in 10 eyes and normalized within 7 days after IOP-lowering treatment. Inferotemporal subretinal hemorrhage was observed in five eyes and resolved spontaneously within 14 days. Postoperative metamorphopsia or diplopia was observed in 75 eyes (65.2%) and resolved spontaneously within 6 months. No major adverse events, including scleral graft rejection or intraocular infection, occurred during follow-up, and no patient required graft removal.

Discussion

This study demonstrated a significant interaction between intraoperative AL shortening and baseline macular traction status. In eyes without baseline traction, greater AL shortening was associated with lower odds of ATN progression. In eyes with baseline traction, the estimated association was reversed but did not reach statistical significance. Category-based analyses showed a similar directional pattern, and component-score changes suggested a trade-off between T-score improvement and N-score progression at higher shortening levels. These findings support individualized AL shortening according to baseline traction status, while the proposed thresholds should be interpreted as preliminary.

The inverse association observed in eyes without baseline traction may reflect the structural characteristics of less traction-affected PM. In these eyes, the BM-RPE complex and outer retinal architecture may be relatively preserved compared with eyes with established MTM.[4] Adequate AL shortening may stabilize the posterior staphyloma, reduce chronic choroidal stretching, and slow chorioretinal atrophy progression.[12,19] A relatively preserved BM-RPE complex may retain some elasticity, enabling it to better tolerate stress changes induced by scleral contraction.[20,21] In the present study, 73.7% of T = 0 eyes had A2 or A3 grade atrophy, and 52.6% had A2 grade atrophy (diffuse choroidal atrophy without extensive BM–RPE damage). The 1.6–2.5 mm range of AL shortening was

associated with a relatively low progression rate, whereas the apparent absence of progression in the > 2.5 mm group should be interpreted cautiously because only three eyes were included in this category.

Eyes with baseline traction differed clinically and structurally from eyes without traction. The T ≥ 1 group had poorer baseline BCVA, higher baseline ATN total scores, and a higher proportion of severe PM. In these eyes, PSC-related shortening may have competing effects. Greater AL shortening may improve tractional anatomy, as reflected by T-score improvement.[22] However, excessive posterior wall indentation may also increase mechanical stress on vulnerable outer retinal, choroidal, and BM-RPE structures.[23,24] In the present study, N-score change increased at higher shortening levels, particularly in eyes with baseline traction. This pattern suggests that larger AL shortening may improve traction-related changes while potentially being accompanied by greater N-component progression.[25]

The exploratory severe PM analysis further supports the need for risk stratification among eyes with baseline traction. In T ≥ 1 eyes with severe PM, ATN progression increased from Group 1 to Group 3. However, the trend test did not reach statistical significance, and the AL shortening × severe PM interaction was not significant. Therefore, severe PM should not be interpreted as a confirmed independent effect modifier in this study. Rather, severe PM may identify a higher-risk subgroup in which aggressive shortening should be approached with caution. This interpretation is biologically plausible because severe PM is associated with advanced atrophic, tractional, and neovascular changes, including BM-RPE complex damage, outer retinal disruption, and choroidal impairment.[2,26] The BM is essential for retinal homeostasis as it regulates biomolecule diffusion and sustains the structural integrity of the RPE layer.[20,24,27] Severe injury to the BM weakens macular mechanical stability, making the tissue more vulnerable to mechanical stress and less tolerant of excessive ocular deformation.[28]

These findings are consistent with the biomechanical principles shared by PSC and macular buckling procedures. Both approaches apply an inward force to the posterior ocular wall and can reduce tractional separation in myopic traction maculopathy.[15,17] However, excessive deformation of the posterior pole may impose additional stress on fragile chorioretinal tissues. Prior macular buckling studies have reported structural complications related to excessive indentation, supporting the possibility that overly aggressive shortening may have adverse consequences in susceptible eyes.[15–17] In the current study, the increase in N-score change in Group 3 supports caution when AL shortening exceeds 2.5 mm, especially in eyes with baseline traction and severe PM.

In addition to the AL shortening interaction, baseline AL and BCVA were independently associated with postoperative ATN progression. Longer AL and poorer BCVA likely reflect more advanced PM and greater cumulative choroidal impairment.[29] These readily available clinical parameters may help identify eyes at higher risk of postoperative progression and may inform individualized surgical planning. Taken together, the present findings suggest that AL shortening should not be treated as a uniform surgical dose. Instead, the target range should be considered in the context of baseline traction status, severe PM features, and overall structural vulnerability.

Limitations

This study has several limitations. First, its retrospective, single-center design may have introduced selection bias and residual confounding. Intraoperative AL shortening was determined by the surgeon rather than randomly assigned; therefore, confounding by indication cannot be excluded. Second, the study lacked a nonsurgical or alternative surgical control group, limiting separation of postoperative ATN changes from the natural course of PM. Third, subgroup analyses were limited by small sample sizes and few events, particularly in eyes without baseline traction and eyes with extreme AL shortening. This reduced statistical power and precluded independent multivariable GEE models within each traction stratum. Fourth, ATN progression was defined as progression in any A, T, or N component. Although clinically practical, this composite definition combines components with different biological mechanisms. Component-score changes were evaluated descriptively. Finally, vascular imaging was not systematically available, and the prolonged enrollment period may have introduced temporal confounding. Larger prospective studies with standardized imaging protocols and prespecified AL shortening targets are needed.

Conclusion

This study demonstrated that baseline macular traction status modified the association between intraoperative AL shortening and ATN progression after PSC surgery. Greater AL shortening was associated with lower odds of ATN progression in eyes without baseline traction, whereas eyes with baseline traction showed an opposite but nonsignificant trend, particularly among those with severe PM. Component-score analyses suggested a trade-off between T-score improvement and N-score progression at higher shortening levels. These findings support individualized AL shortening based on baseline traction status, but the proposed thresholds remain preliminary and require validation in larger prospective studies.

Abbreviations

AL
axial length
ATN
atrophy–traction–neovascularization
PM
pathologic myopia
PSC
posterior scleral contraction
BCVA
best-corrected visual acuity
BM-RPE
Bruch’s membrane–retinal pigment epithelium complex

MTM
myopic traction maculopathy
IOP
intraocular pressure
CCT
central corneal thickness
AD
anterior chamber depth
LT
lens thickness
K1
flat keratometry
K2
steep keratometry
CI
confidence interval
GEE
generalized estimating equations
IQR
interquartile range
OR
odds ratio.

Declarations

Ethics approval and consent to participate

This study protocol complied with the principles of the Declaration of Helsinki and was approved by the Tianjin Medical University Eye Hospital Institutional Review Board (2026KY-47). All patients provided written informed consent before undergoing the surgical procedure.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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Author Contribution

WRH and CYH were responsible for research design, data analysis and interpretation, and manuscript preparation. WangYX was responsible for data acquisition and research execution, data analysis and interpretation, and manuscript preparation. XAQ contributed to research design and manuscript preparation. LYF, ZHY, WD, XR, CMD, ZXY, CX, WeiYX and LXT were responsible for data acquisition and research execution. HYN, PEE and WX contributed to manuscript preparation. No authors obtained funding. All authors read and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

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Not applicable.

Availability of data and materials

All data generated or analysed during this study are included in this published article and its supplementary information files.

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Figures

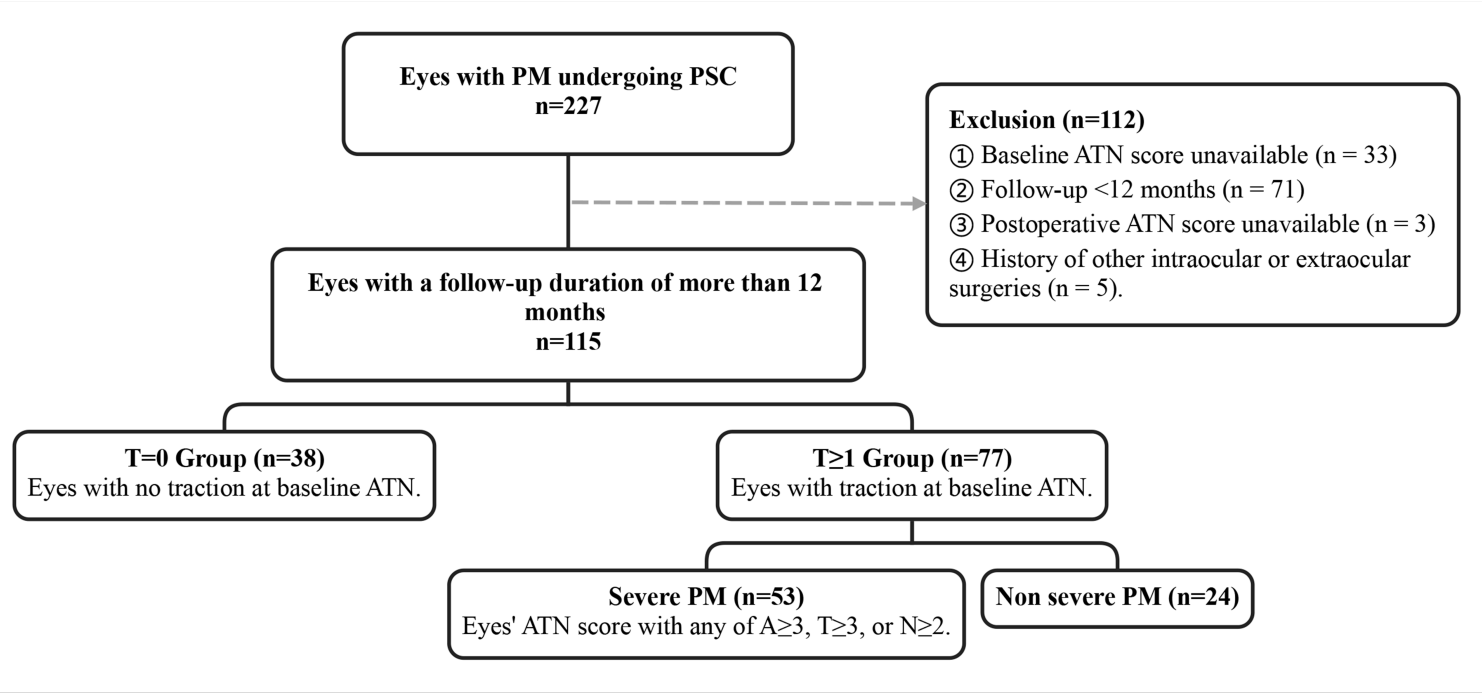

Figure 1

Flow diagram of study inclusion and exclusion. Eyes with pathologic myopia (PM) undergoing posterior scleral contraction (PSC) between March 2013 and December 2024 were screened. Eyes were excluded for unavailable baseline ATN score, follow-up duration <12 months, unavailable postoperative ATN score, or a history of other intraocular or extraocular surgery. Included eyes were stratified by baseline macular traction status. Severe PM was defined as meeting any of the following ATN thresholds: A≥3, T≥3, or N≥2. AL = axial length; ATN = atrophy–traction–neovascularization; PM = pathologic myopia; PSC = posterior scleral contraction.

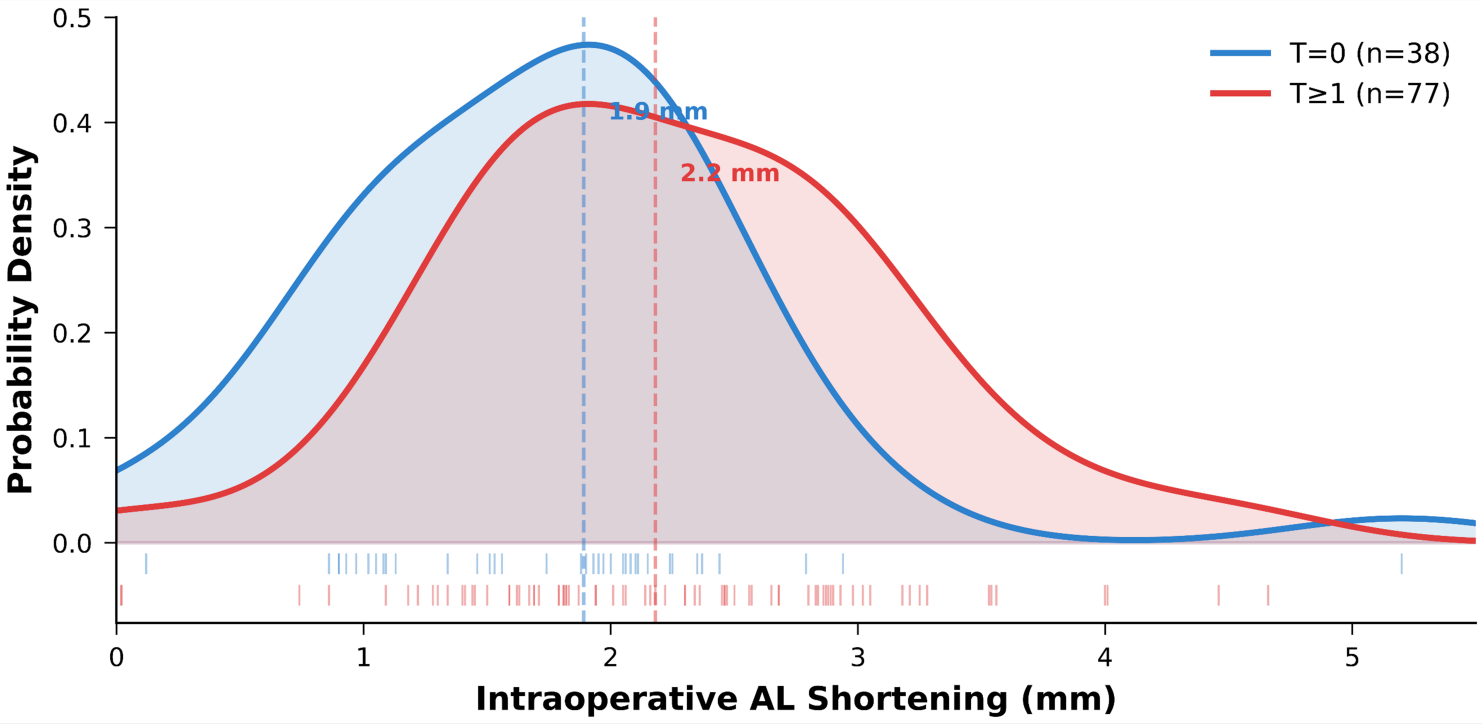

Figure 2

Distribution of intraoperative axial length shortening according to baseline macular traction status. Kernel density curves show the distribution of intraoperative AL shortening in eyes without baseline macular traction (T=0) and eyes with baseline macular traction (T≥1). Rug marks indicate individual eyes. Dashed vertical lines indicate median AL shortening values in each group. AL = axial length.

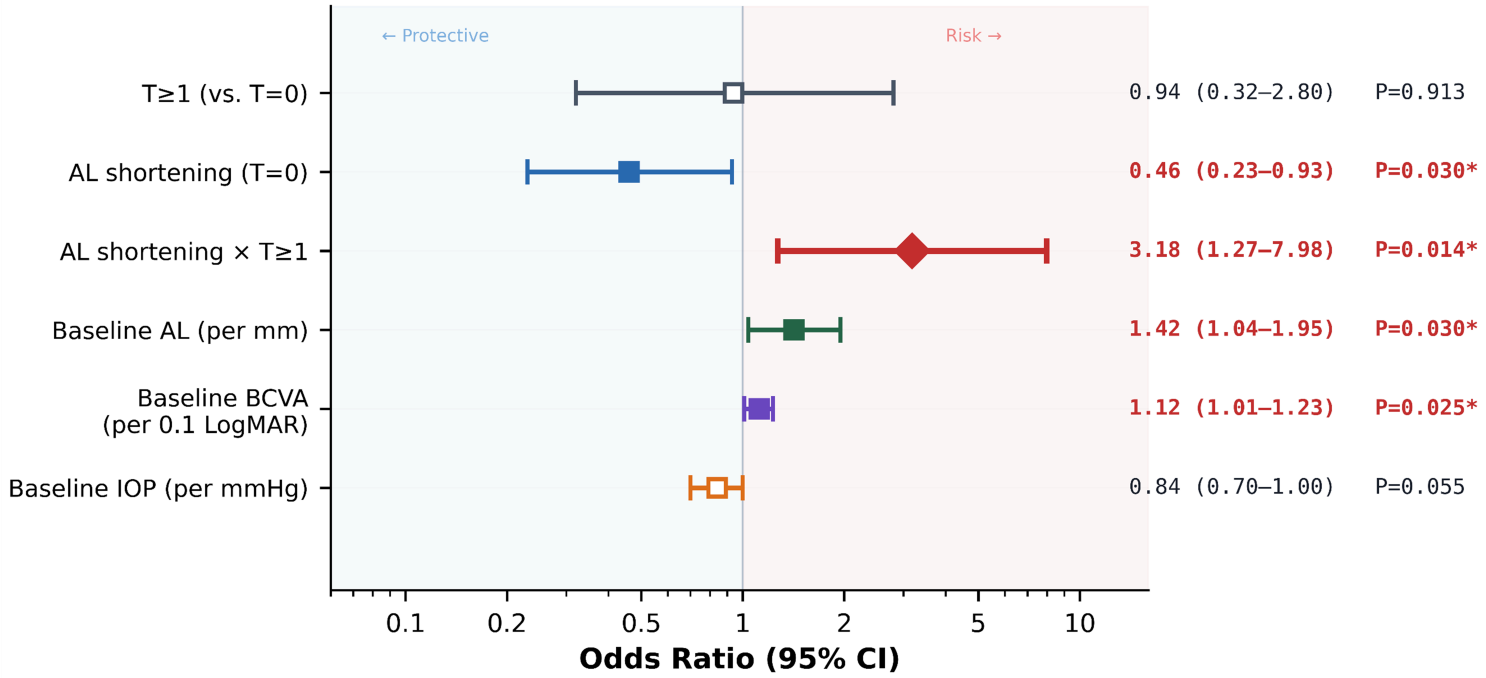

Figure 3

Multivariable generalized estimating equation model for ATN progression. Odds ratios and 95% confidence intervals are shown for the interaction model. This model tested effect modification by baseline traction status. The vertical reference line indicates an odds ratio of 1. Values to the left indicate lower odds of ATN progression, and values to the right indicate higher odds. *P<0.05. CI = confidence interval; AL = axial length; ATN = atrophy–traction–neovascularization; BCVA = best-corrected visual acuity; logMAR = logarithm of the minimum angle of resolution; IOP = intraocular pressure.

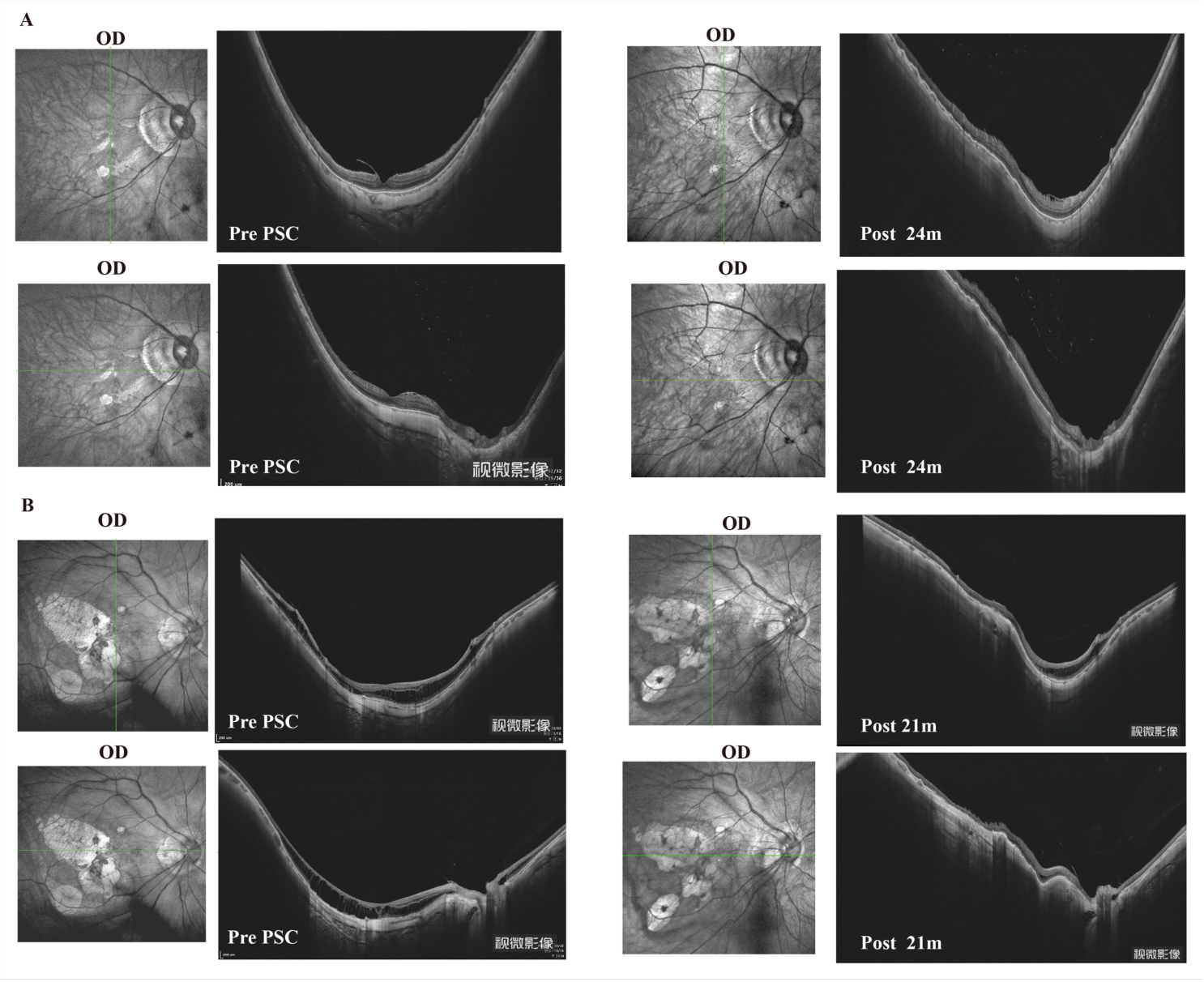

Figure 4

Representative scanning laser ophthalmoscopy and optical coherence tomography images at baseline and final examination. A, Representative eye without baseline macular traction (T=0), followed for 24 months. Baseline AL was 30.76 mm, intraoperative AL shortening was 2.37 mm, and final AL was 29.53 mm. The ATN grade remained A2T0N1 from baseline to final examination. B, Representative eye with baseline macular traction (T≥1), followed for 21 months. Baseline AL was 33.12 mm, intraoperative AL shortening was 1.69 mm, and final AL was 31.70 mm. The ATN grade changed from A3T3N0 at baseline to A3T2N0 at final examination. AL = axial length; ATN = atrophy–traction–neovascularization; OCT = optical coherence tomography; SLO = scanning laser ophthalmoscopy.

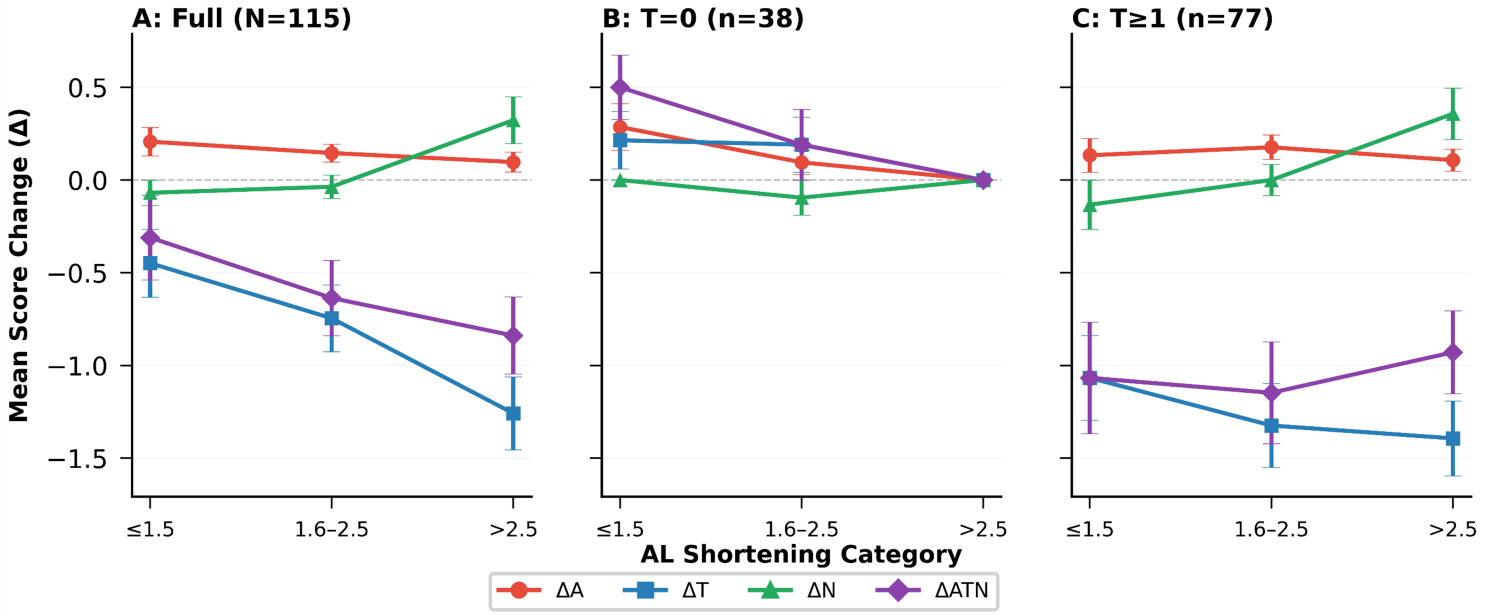

Figure 5

Changes in ATN component scores from baseline to final follow-up according to intraoperative axial length shortening category. Score change was calculated as final score minus baseline score. Negative values indicate improvement, whereas positive values indicate progression. ΔA, ΔT, ΔN, and ΔATN represent mean score changes in the A, T, N, and total ATN scores, respectively. Panel A shows the full cohort, panel B shows eyes without baseline macular traction (T=0), and panel C shows eyes with baseline macular traction (T≥1). AL shortening categories were defined as ≤1.5 mm, 1.6–2.5 mm, and >2.5 mm. Error bars indicate 95% confidence intervals. A = atrophy; T = traction; N = neovascularization; ATN = atrophy–traction–neovascularization; AL = axial length.

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