• Clinical Research •

Association between choroidal thickness and primary open angle glaucoma in highly myopic eyes

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Abstract

● **AIM:** To investigate the relationship between choroidal thickness (CT) and primary open angle glaucoma (POAG) in highly myopic eyes across different categories of myopic atrophic maculopathy (MAM).

● **METHODS:** This observational analytical case-control study enrolled highly myopic patients. All participants underwent comprehensive ophthalmic examinations. CT was measured in peripapillary and macular regions using optical coherence tomography (OCT). Each eye was classified for POAG status and MAM category. Stepwise logistic regression was performed to identify factors independently associated with POAG.

● **RESULTS:** Among 248 highly myopic subjects, 37 (18 males, mean age 68.25±7.16y) had POAG (25 bilateral, 12 unilateral) and 211 (97 males, mean age 67.09±7.63y) were non-glaucomatous. Age and sex did not differ significantly between groups (both $P>0.05$). Seventy-eight patients had unilateral high myopia and 170 bilateral, yielding 418 highly myopic eyes, of which 58 (13.88%) had POAG. POAG prevalence across MAM categories (no lesion to complete macular atrophy) was 7.14%, 16.28%, 14.07%, 17.86%, and 17.14%, respectively ($P=0.44$). In the diffuse chorioretinal atrophy subgroup, POAG eyes showed significantly thicker CT in central, inner nasal, outer superior, inner superior, and inner inferior macular regions, and thinner central corneal thickness (all $P<0.05$). Stepwise logistic regression showed that only parafoveal inferior CT was independently associated with POAG in this subgroup (OR=1.017; 95%CI: 1.005-1.028; $P<0.01$). No significant CT-POAG association was found in other MAM categories.

● **CONCLUSION:** Increased macular CT is independently associated with POAG in highly myopic eyes with diffuse chorioretinal atrophy. This implies distinct pathogenic mechanisms underlying POAG development across different MAM categories in high myopia.

● **KEYWORDS:** glaucoma; primary open angle glaucoma; choroidal thickness; high myopia; myopic atrophic maculopathy

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INTRODUCTION

Primary open angle glaucoma (POAG) is a chronic, progressive optic neuropathy characterized by loss of the optic nerve rim and retinal nerve fiber layer (RNFL), accompanied by corresponding visual field defects. Myopia,

particularly high myopia, has been recognized as a risk factor for POAG for decades. However, its precise role in POAG pathogenesis remains incompletely understood[1]. One proposed mechanism involves optic nerve head hypoperfusion, which may increase susceptibility to glaucomatous damage in myopic eyes[2-3]. Given the shared vascular supply of the optic nerve head and choroid *via* the short posterior ciliary arteries[4], choroidal atrophy in high myopia may contribute to POAG development.

The relationship between choroidal thickness (CT), a surrogate marker for choroidal vasculature status, and glaucoma has yielded inconsistent results across studies[5-14]. Some reports describe CT as thicker[5-8], thinner[9], or unchanged[10-14] in glaucomatous eyes, including both POAG and angle-closure glaucoma. For instance, Komma *et al*[5] observed thicker peripapillary CT but unchanged posterior CT in POAG patients compared to controls, while Li *et al*[6] reported thicker macular CT in primary angle-closure glaucoma. Spraul *et al*[7] noted doubled posterior CT in end-stage POAG, contrasting with Gao *et al*[8] who found increased anterior but unchanged posterior CT in POAG. Conversely, Wang *et al*[9] documented significantly reduced CT in normal-tension glaucoma. These discrepancies likely stem from variations in study populations, given the numerous confounding factors influencing CT[15]. Despite high myopia being a significant factor for both choroidal thinning and POAG development, the relationship between CT and POAG specifically in highly myopic eyes remains unexplored.

Axial elongation in high myopia leads to diverse chorioretinal atrophy patterns, with the degree and extent of choroidal atrophy varying considerably across different stages. Based on the widely used international photographic classification and grading system for myopic maculopathy (META-PM)[16], myopic atrophic maculopathy (MAM) comprises five categories, which are not always proportional to axial length (AL). Given the distinct clinical features of each MAM category, different POAG pathogenic mechanisms may exist. Furthermore, the relationship between MAM severity and POAG prevalence has not been previously investigated.

This study aimed to investigate the relationship between CT and POAG in highly myopic eyes across different MAM categories. We hypothesized that choroidal atrophy in highly myopic eyes may induce the development of POAG. We aimed to provide insights into the pathogenesis of POAG in highly myopic eyes.

PARTICIPANTS AND METHODS

Ethical Approval This study was conducted in accordance with the tenets of the Declaration of Helsinki and was approved by the research ethics committee of Shanghai General Hospital, affiliated to Shanghai Jiao Tong University School of Medicine (approval number: 2015KY156 and 2021KY079). All participants were informed about the procedures and gave written informed consent. Clinical trial registration number: NCT03446300.

Setting and Participants This study is a sub-study of the Shanghai High Myopia Study for Adults, a population-based, observational analytical cross-sectional study (clinical trial registration number: NCT03446300). This study included highly myopic individuals aged 50 years and older and was conducted in rural and urban regions of Shanghai, China. The inclusion criteria of the participants were 1) age ≥50y, 2) at least one eye with high axial myopia, which was defined as AL ≥26 mm. The exclusion criteria were 1) history of intraocular or refractive surgery other than cataract or glaucoma surgery, 2) significant media opacity precluding adequate fundus imaging for META-PM grading, 3) history of severe systemic diseases, 4) inability to cooperate sufficiently during examinations, resulting in unreliable visual field testing unsuitable for glaucoma diagnosis. Our research team comprised five ophthalmologists, three optometrists, and 15 auxiliary staff.

Systemic and Ophthalmologic Examinations Detailed procedures were previously described[17]. Briefly, participants' age, gender, disease history, weight, and height were collected using a questionnaire. Patients with pre-existing glaucoma provided medical records, and their glaucoma diagnosis was confirmed by documented optic nerve injury progression during follow-up. All participants underwent comprehensive ophthalmic examination. The anterior segment and fundus were observed using a slit-lamp microscope (SL130; Zeiss, Germany) and a 90 D non-contact lens (90 D, Ocular, Bellevue, WA, USA). The best-corrected visual acuity was measured using the international standard logMAR visual acuity chart. Intraocular pressure (IOP) was measured using a non-contact tonometer (Full Auto Tonometer TX-F; Topcon, Japan). AL, anterior chamber depth, lens thickness, and corneal thickness were measured by optical low-coherence reflectometry (Lenstar; Haag-Streit AG, Koeniz, Switzerland). Data on anterior chamber depth and lens thickness from aphakic or pseudophakic eyes were excluded from analysis. The fundus images centered on the optic disc and macula were obtained using a retinal camera (WX-3D, Kowa Optimed, Tokyo, Japan). The disc area and vertical cup to disc ratio were calculated from these photographs using ImageJ version 1.60 software (National Institutes of Health, MD, USA; http://rsb.info.nih.gov/ij/index.html) by two independents, blinded, well-trained observers. Averaged data were used in the final analysis. Participants without prior glaucoma diagnosis but exhibiting suspected glaucomatous optic neuropathy underwent further assessment, including Goldmann applanation tonometry, gonioscopy, and standard automated perimetry

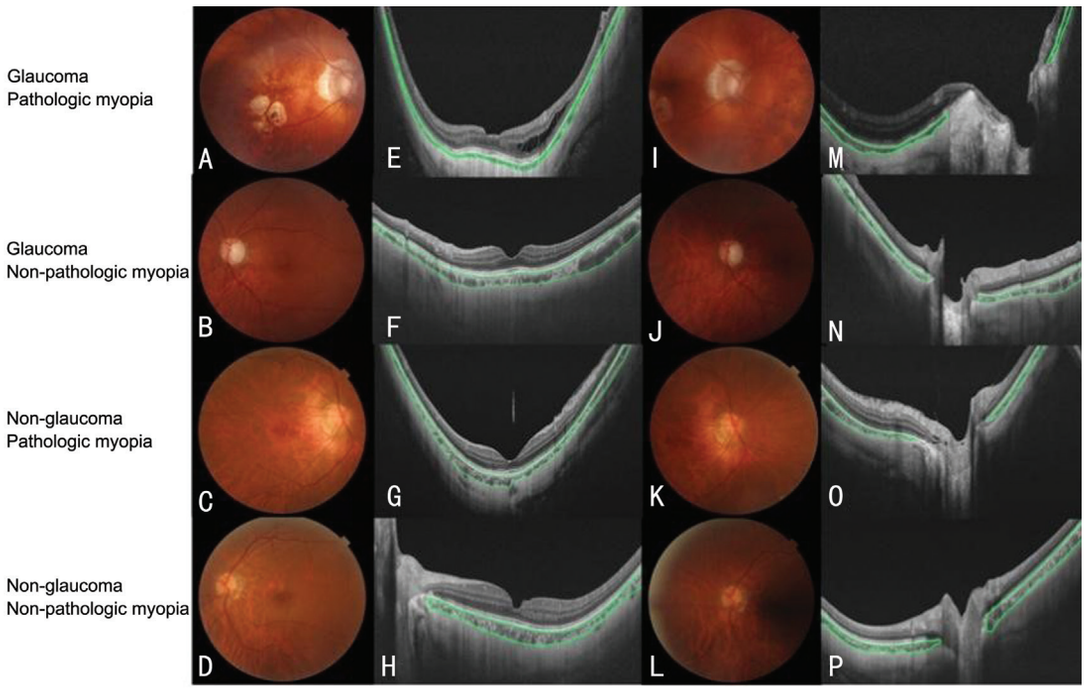

Figure 1 Examples of fundus photos of highly myopic eyes and their corresponding swept-source optical coherence tomography images A-D: Fundus photos centered in fovea; E-H: Swept-source optical coherence tomography images centered in fovea; I-L: Fundus photos centered in optic disc; M-P: Swept-source optical coherence tomography images centered in optic disc.

using the Swedish Interactive Threshold Algorithm standard 24-2 (Humphrey Visual Field Analyzer, Carl Zeiss Meditec, Inc., Dublin, CA, USA). Reliable visual fields required false-positive and false-negative error rates <15%, and fixation loss <20%.

The thickness of peripapillary and macular retina and choroid were measured by swept-source optical coherence tomography (OCT; model DRI OCT-1 Atlantis; Topcon, Japan) at a resolution of 1024 A scan/6 mm. A radial scan with 12 lines separated by 30° was used to capture images. AL was input before image acquisition for magnification correction. To minimize the diurnal fluctuation of CT, all OCT scans were performed in the morning. OCT images with poor quality was retaken to ensure the measurement of CT. Retinal and choroidal layers were automatically delineated using built-in software, with manual adjustments applied when automated segmentation was inaccurate (Figure 1). For CT evaluation, topographic maps laid over an ETDRS grid were constructed automatically, dividing the image into three concentric circles (diameter of 1, 3, and 6 mm, respectively), and the inner and outer circles were subdivided into four subfields: superior, inferior, temporal, and nasal. Average CT within a 6 mm-diameter circle at the center of the fovea and on the edge of the disc was calculated automatically using built-in software.

Diagnosis of POAG and Classification of Atrophic Myopic Maculopathy Participants without prior glaucoma diagnosis but exhibiting suspected glaucomatous optic neuropathy underwent comprehensive clinical evaluation. POAG diagnosis adhered to American Academy of Ophthalmology guidelines[18]. Stereoscopic disc photography, red-free RNFL photography, OCT measurements of circumpapillary RNFL and macular ganglion cell-inner plexiform layer (GCIPL), and visual field results were independently assessed by two masked glaucoma specialists, and discrepancies were resolved by a third specialist. The diagnosis of glaucoma was defined as structural changes of glaucomatous optic neuropathy (including generalized enlargement or vertical elongation of the optic disc cup, localized notching or narrowing of the optic disc rim, and violation of the ISNT rule), circumpapillary RNFL defects (judged by red-free RNFL photography and circumpapillary RNFL OCT measurements), macular GCIPL thinning, and corresponding glaucomatous visual field defects (pattern standard deviation beyond the 95% normal limits). Distinguishing glaucomatous optic neuropathy from highly myopic optic neuropathy is challenging due to overlapping structural and functional abnormalities in highly myopic and glaucomatous eyes[19]. Special efforts were taken to distinguish the glaucomatous optic neuropathy from those caused by myopia only. The principles we applied were similar with that concluded in a recently published review[20].

All study eyes were classified into five MAM categories (A0-A4) according to the META-PM system[16]: A0, no myopic retinal lesions; A1, tessellated fundus only, which means well-defined choroidal vessels that can be observed clearly around the fovea as well as around the arcade vessels; A2, diffuse chorioretinal atrophy, which means yellowish white

appearance of posterior pole; A3, patchy chorioretinal atrophy, which means well-defined, grayish white lesions in the macular area or around the optic disc; and A4, complete macular atrophy, which means well-defined, grayish white or whitish, round chorioretinal atrophic lesion in the foveal region. Eyes graded A2 or higher were defined as pathologic myopia (PM). Classification was performed by two independent, trained graders; disagreements were adjudicated by a retinal specialist.

Statistical Analysis Statistical analyses were performed using SAS (version 9.3, SAS Institute, NC, USA). For systemic factor analysis, only right-eye data were included when both eyes were eligible. Otherwise, both eyes were included. Data distribution was assessed using the Kolmogorov-Smirnov test. Normally distributed continuous variables were compared using *t*-tests; non-normally distributed variables used the Kruskal-Wallis *H* test. Categorical variables were compared using Chi-square tests. Stepwise logistic regression analysis was used to assess the factors independently associated with POAG, covariables were adjusted for, and odds ratio (OR) and 95% confidence interval (CI) were calculated. Data were shown as means±standard deviations for continuous data and as counts (proportions) for categorical data. Statistical significance was defined as $P<0.05$ (two-tailed). False discovery rate (FDR) was applied for multiple comparisons.

RESULTS

A total of 248 highly myopic participants were included in the study, of whom 37 (14.92%) had POAG, including 25 patients with bilateral glaucoma and 12 with unilateral glaucoma. Among the 12 unilateral glaucoma patients, neither optic nerve atrophy nor retinal ganglion cell loss was observed in the unaffected eye at the time of the survey. No significant differences were found in age, gender, body mass index (BMI), or hypertension rates between participants with and without glaucoma (Table 1).

Among the 248 highly myopic participants, 78 had unilateral high myopia and 170 had bilateral high myopia. Thus, a total of 418 highly myopic eyes was included, of which 58 had POAG (13.88%) and 307 were phakic (73.44%). Among the 58 glaucomatous eyes, 42 had been diagnosed before the survey at hospitals. Compared to non-glaucomatous eyes, POAG eyes exhibited significantly higher IOP, a greater vertical cup to disc ratio, larger disc area, thinner circumpapillary RNFL, thinner macular GCIPL, and thinner corneas. No significant differences were found in the rate of PM, anterior chamber depth, lens thickness, AL, peripapillary CT, or macular CT between the groups (Table 2).

All study eyes were divided into five groups according to their MAM degrees (Table 3), and group A2 was the most prevalent in both POAG (48.28%) and control eyes (47.50%). The prevalence of POAG was 7.14%, 16.28%, 14.07%, 17.86%, and 17.14% in the A0, A1, A2, A3, and A4 group, respectively; however, the inter-group differences were not statistically significant ($P=0.44$). Central macular CT, average macular CT, average peripapillary CT, AL, corneal thickness, and IOP were compared between eyes with and without POAG in each MAM degree group (Table 3). The central macular CT was significantly higher ($P<0.01$, $Q=0.01$), and corneal thickness was significantly lower ($P<0.01$, $Q<0.01$) in POAG eyes of the A2 group. In the A4 group eyes, IOP was significantly higher in POAG eyes ($P<0.01$, $Q<0.01$).

Table 1 Demographic and systemic characteristics compared between high myopic participants with and without open angle glaucoma

Parameters	Glaucoma	Non-glaucoma	*P*
Total	37	211	
Age (y)	68.25±7.16	67.09±7.63	0.39
Male, *n* (%)	18 (48.65)	97 (45.97)	0.76
Body mass index (kg/m^2)	23.47±2.44	23.79±3.05	0.55
High blood pressure, *n* (%)	20 (54.05)	97 (45.97)	0.36

Perifoveal and parafoveal CT were assessed in A2 group eyes (Figure 2). CT in the inner nasal ($P=0.01$, $Q=0.04$), outer superior ($P=0.02$, $Q=0.03$), inner superior ($P<0.01$, $Q=0.02$), and inner inferior ($P<0.01$, $Q<0.01$) regions were significantly higher in POAG eyes compared to non-glaucomatous eyes. A stepwise logistic regression analysis was conducted to evaluate the independent factors associated with glaucoma in A2 group eyes. The CT in the inner nasal, outer superior, inner superior, inner inferior, and central macular regions, as well as corneal thickness, were included in the analysis as potential associated factors and covariables. The result indicated that in A2 group eyes, only parafoveal inferior CT was independently associated with POAG (OR=1.017; 95%CI=1.005-1.028, $P<0.01$).

DISCUSSION

To our knowledge, this is the first study investigating the relationship between CT and POAG specifically across different MAM categories in high myopia. Our key finding is a positive association between thicker parafoveal inferior CT and POAG risk in eyes with diffuse chorioretinal atrophy (A2), suggesting that choroidal thickening may be a biomarker for glaucoma in this subgroup. However, no such association was observed in other MAM categories.

The finding of thicker macular choroid in POAG eyes within the A2 group is consistent with several former studies conducted in patients without high myopia[7,21-22]. Spraul *et al*[7] analyzed 20 eye bank eyes with end stage POAG and compared them with 20 age-matched control eye bank eyes. Their results indicated that although the density of capillaries, veins and arteries all decreased, posterior CT was twice as thick in POAG eyes compared to controls. Cennamo *et al*[21] investigated 21 healthy eyes and 16 eyes with POAG

Table 2 Comparison of ocular features between high myopic eyes with and without open angle glaucoma

Parameters	Glaucoma	Non-glaucoma	*P*
Number of eyes	58	360	
IOP (mm Hg)	14.57±4.08	12.97±2.96	<0.01
PM eyes, *n* (%)	39 (67.24)	223 (61.94)	0.44
Corneal thickness (μm)	526.72±33.48	538.20±35.10	0.02
Anterior chamber depth (mm)	3.60±0.65	3.54±0.63	0.49
Lens thickness (mm)	4.54±0.48	4.54±0.32	0.93
Axial length (mm)	27.64±1.49	27.86±1.68	0.33
Vertical cup to disc ratio	0.77±0.18	0.57±0.32	0.02
Disc area (mm^2)	3.49±0.82	2.96±0.73	<0.01
Macular choroidal thickness (μm)			
Center	95±62	89±58	0.54
Inner temporal	99±59	100±61	0.93
Inner superior	99±59	94±61	0.68
Inner nasal	80±49	88±51	0.35
Inner inferior	92±55	99±63	0.47
Outer temporal	108±61	107±55	0.93
Outer superior	110±60	104±61	0.58
Outer nasal	73±40	68±35	0.43
Outer inferior	92±52	95±50	0.71
Average	96±48	93±50	0.79
Peripapillary choroidal thickness (μm)			
Inner temporal	54±33	60±44	0.28
Inner superior	58±27	65±36	0.27
Inner nasal	74±31	75±50	0.79
Inner inferior	63±42	68±40	0.53
Outer temporal	60±29	67±44	0.18
Outer superior	79±32	86±38	0.22
Outer nasal	99±46	91±47	0.31
Outer inferior	69±39	71±35	0.69
Average	73±27	77±35	0.56
Macular ganglion cell-inner plexiform layer thickness (μm)			
Center	45±18	50±18	0.08
Inner temporal	67±25	76±23	0.01
Inner superior	70±27	76±21	0.10
Inner nasal	72±26	79±22	0.02
Inner inferior	71±25	78±21	0.03
Outer temporal	55±19	62±20	0.01
Outer superior	54±21	60±16	0.05
Outer nasal	57±20	60± 18	0.26
Outer inferior	47±16	51± 16	0.11
Average	57±18	62±15	0.02
Circumpapillary retinal nerve fiber layer thickness (μm)			
Inner temporal	73±37	110±49	<0.01
Inner superior	70±34	98±37	<0.01
Inner nasal	113±50	134±54	<0.01
Inner inferior	89±35	100±43	0.06
Outer temporal	109±50	135±50	<0.01
Outer superior	70±21	74±21	0.13
Outer nasal	77±36	89±33	<0.01
Outer inferior	47±30	49±24	0.52
Average	73±27	86±30	<0.01

IOP: Intraocular pressure; PM: Pathologic myopia.

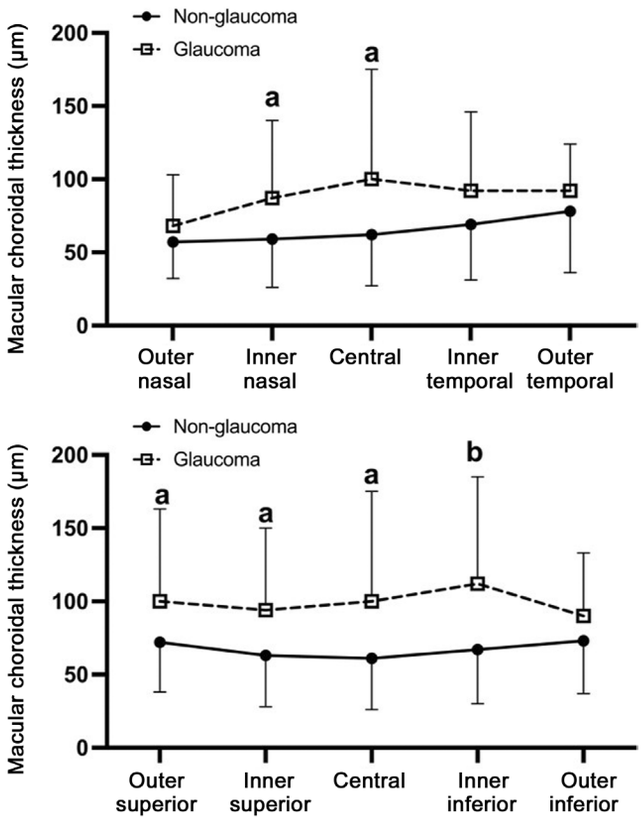

Figure 2 Comparing perifoveal and parafoveal choroidal thickness between glaucomatous and non-glaucomatous eyes with diffuse chorioretinal atrophy. [a]*Q*<0.05 (false discovery rate), [b]*Q*<0.01 (false discovery rate).

and found that CT was increased in glaucomatous eyes due to an increase in both the vertical diameter and the luminal area of the vessels. A more recent study from the same group further revealed that advanced glaucoma patients exhibited a significant increase in subfoveal CT compared to healthy controls, whereas no difference was found between preperimetric glaucoma patients and healthy controls. The finding of a thicker choroid in POAG eyes seems to contrast with our initial hypothesis linking choroidal atrophy to POAG. There are two possible reasons for this contradiction. First, the opposite change between CT and choroidal vessel density may result in this unexcepted results. Former postmortem and *in vivo* studies both indicated that because of a significantly reduced choriocapillary vessel density in POAG eyes, a compensatory dilation of the choroidal vessels results in an increased vessel diameter, and an increased perfusion pressure in the still permeable vessels results in an increased luminal area of the choroidal vessels. And the increased vessel diameter and luminal area of the choroidal vessels together results in an increased CT[7,21-22]. Second, axial elongation may cause abnormal branching of the short posterior ciliary arteries, resulting in increased blood flow to the macular choroid and reduced perfusion of the optic nerve head, which

Table 3 Ocular features of high myopic eyes of different myopic atrophy maculopathy categories

Parameters	A0	A1	A2	A3	A4	*P*
Total	70	86	199	28	35	
Glaucoma, *n* (%)	5 (7.14)	14 (16.28)	28 (14.07)	5 (17.86)	6 (17.14)	0.44
Central macular choroidal thickness (μm)						
Non-glaucoma	140±65	105±43	62±35	42±30	26±8	<0.01
Glaucoma	124±55	117±46	100±75	32±4	47±44	<0.01
P	0.60	0.36	<0.01	0.58	0.09	
Q	0.82	0.92	0.01	0.82	0.32	
Average macular choroidal thickness (μm)						
Non-glaucoma	140±52	106±35	69±31	51±28	34±8	<0.01
Glaucoma	127±51	121±35	90±43	38±11	54±43	<0.01
P	0.59	0.16	0.03	0.45	0.10	
Q	0.60	0.27	0.17	0.56	0.25	
Average peripapillary choroidal thickness (μm)						
Non-glaucoma	97±34	85±33	67±30	45±24	43±13	<0.01
Glaucoma	98±30	79±17	76±26	44±21	45±19	<0.01
P	0.95	0.54	0.31	0.95	0.80	
Q	0.96	0.96	0.96	0.96	0.96	
Axial length (mm)						
Non-glaucoma	26.67±0.88	26.94±0.79	28.07±1.53	29.84±1.58	30.03±1.63	<0.01
Glaucoma	26.25±0.34	26.93±0.77	27.68±1.34	29.39±2.15	29.07±1.61	<0.01
P	0.30	0.97	0.21	0.62	0.20	
Q	0.50	0.98	0.50	0.78	0.50	
Corneal thickness (μm)						
Non-glaucoma	540.30±28.46	540.27±27.19	537.27±36.19	536.17±50.31	535.88±44.44	0.95
Glaucoma	552.50±42.74	529.43±25.34	512.54±29.43	545.80±36.73	553.50±33.60	<0.01
P	0.42	0.17	<0.01	0.69	0.37	
Q	0.43	0.35	<0.01	0.56	0.43	
Intraocular pressure (mm Hg)						
Non-glaucoma	13.05±2.72	12.92±3.20	12.90±2.73	12.56±3.99	13.67±3.21	0.69
Glaucoma	14.80±0.84	13.42±3.44	13.57±3.63	16.30±4.82	19.92±4.61	<0.01
P	0.16	0.61	0.26	0.08	<0.01	
Q	0.21	0.49	0.26	0.16	<0.01	

Q: FDR was used for multiple comparisons; A0: No myopic retinal lesions; A1: Tessellated fundus only; A2: Diffuse chorioretinal atrophy; A3: Patchy chorioretinal atrophy; A4: Complete macular atrophy.

potentially contribute to POAG development. Although CT may not directly reflect choroidal blood supply in POAG eyes, our results establish a CT-POAG association in highly myopic eyes with diffuse chorioretinal atrophy.

Conversely, we found no relation between CT and POAG in highly myopic eyes with no myopic retinal lesions (A0), tessellated fundus only (A1), patchy chorioretinal atrophy (A3), and complete macular atrophy (A4). These results are consistent with two prior meta-analyses[23-24] and one recently published study[25] conducted in patients without high myopia. One Meta-analysis, which included 22 case-control or cross-sectional studies, found no significant difference in subfoveal CT between patients with POAG and controls[23]. Wang and Zhang[24] conducted a cross-sectional study including 76 healthy and 52 POAG subjects, and they found no significant differences in CT between the POAG and healthy eyes after adjusting for IOP, age, and AL. A subsequent Meta-analysis including 875 POAG patients and 871 controls also found no significant difference in CT between the two groups. Wang *et al*[25] reported that the subfoveal CT was not different in 69 patients with POAG and 44 healthy controls, but the proportion of luminal area to the total choroidal area in the POAG groups was significantly lower than that in the control group. The inconsistent findings between eyes of diffuse chorioretinal atrophy (A2) group and eyes of other MAM categories may stem from three potential reasons: First, the sample size of A2 group was bigger than the other groups, which is beneficial for reaching statistical significance. Second,

the CT of PM eyes have high individual variability potentially masking associations. Take Figure 1 for example, different size and pattern of tilt optic nerve head and peripapillary atrophy leads to high variability in peripapillary CT, and the same situation applies for the effect of patchy chorioretinal atrophy and complete macular atrophy on macular CT. Third, different mechanism may exist in highly myopic eyes of different MAM categories in the development of POAG. Which implies that choroid may play a significant role in glaucoma development in eyes with diffuse chorioretinal atrophy, but not in other highly myopic eyes.

This study has several limitations. First, the sample size was small for a population-based study, especially in the glaucoma group, potentially leading to bias. Second, we could not account for the potential effects of medications or prior surgeries on CT. For example, prostaglandin analogues eye drops decrease IOP by increasing the uveoscleral outflow, which may result in the increase of CT. But the drugs our participants used for IOP control was not included into analysis. Moreover, both cataract and glaucoma surgeries could result in an increased CT[26-29]. However, since the participants of our study were elderly high myopic patients with a mean age over 65, more than a quarter of them had received cataract surgeries before our survey. Thus, the influence of surgeries and medications on our results cannot be eliminated. Third, because of the small sample size of glaucomatous eyes, we used the data of both eyes in patients with bilateral glaucoma, which may lead to systematic confounding factors such as age being neglected. Fourth, despite AL-based OCT magnification correction, posterior staphyloma curvature may cause artifactual ETDRS grid misalignment and measurement error.

High myopic eyes are at high risk of developing POAG[30]. Given the global rise in myopia prevalence[31], understanding POAG pathogenesis in this group is clinically significant. This observational analytical case-control study identifies thicker macular choroid as an independent factor associated with POAG in highly myopic eyes with diffuse chorioretinal atrophy. Our result also suggested that different mechanism may exist in highly myopic eyes of different MAM categories in the development of POAG. Future studies using OCT angiography could quantify choroidal and optic nerve head blood flow to test the perfusion redistribution hypothesis. Histological analysis may clarify if choroidal thickening is driven by vascular dilation or stromal expansion.

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