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The inflammatory cytokine profiles and ocular biometric characteristics of primary angle-closure glaucoma

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Abstract

Objective: To investigate the pathogenesis of primary angle-closure disease (PACG) by measuring the anatomical structures of the anterior and posterior segments of the eye and inflammatory markers in the peripheral blood.

Methods: This case–control study enrolled patients diagnosed with acute PACG (APACG) and chronic PACG (CPACG). It also enrolled control subjects without PACG. The anterior and posterior anatomical features were measured in all study participants. The levels of interleukin (IL)-6, tumour necrosis factor-α and the neutrophil-to-lymphocyte ratio (NLR) in the peripheral blood were measured.

Results: This study analysed a total of 99 eyes: 34 eyes from 34 patients with APACG, 28 eyes from 28 patients with CPACG and 37 eyes from 37 control patients with senile cataract. The axis length, corneal diameter, anterior chamber depth and anterior chamber volume were significantly smaller in the APACG and CPACG groups compared with the controls. The level of IL-6 in the peripheral blood of patients with PACG was significantly lower than that of the controls. The NLR in the peripheral blood of patients with PACG was significantly greater than that of the controls.

Conclusions: Changes in the ocular anatomy and some inflammatory markers might be involved in the pathogenesis of PACG.

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Introduction

Primary angle-closure glaucoma (PACG) disease is a common eye disease that causes severe visual impairment and it has a high incidence in Asian populations.[1] The population that is affected by PACG worldwide is estimated to be more than 20 million by 2020.[2] It is characterized by elevated intraocular pressure (IOP), eye pain and headache. Primary angle closure diseases (PACD) consist of primary angle closure suspects (PACS), primary angle closure (PAC) and PACG.[3] In contrast, chronic primary angle-closure glaucoma (CPACG) patients rarely experience the dramatic symptoms.[4] Anatomical structural differences must exist between acute primary angle-closure glaucoma (APACG) and CPACG eyes. The main pathogenesis of PACG is that the peripheral iris obstructs the trabecular meshwork, which leads to the obstruction of the aqueous humor outflow and the IOP increase. Some international scholars have proposed a classification system for angle closure, including posterior lens factors and lens factors, plateau iris and pupillary block.[5] Chinese scholars believe that pupillary block is the main cause of angle closure.[6,7] Numerous studies have further confirmed the unique anatomy of PACG: short axis length, small cornea, shallow anterior chamber, narrow angle, thick lens thickness or anterior lens position.[8,9] According to previous studies,[10,11] the theory of choroidal dilatation plays an important role in PACG. Research has demonstrated that the subfoveal choroidal thickness (SFCT) of all PACD subtypes (PACS, PAC and PACG) is greater than that of normal eyes.[11]

A previous study investigated the pathogenesis of PACG from the biological morphology of the eye and explored the unique anatomical structure of PACG, but it mainly focused on the biological structure of the anterior segment and the theory of choroidal expansion.[12] It is difficult to make an exact decision about the onset of PACG. This is because a considerable number of healthy people also have the anatomical characteristics of short axial length, shallow anterior chamber and small cornea, but their IOP is normal throughout their lives.[12] It is clear that the unique anatomical structure of PACG leads to increased IOP, but what is the reason for its further development? Some scholars have proposed the theory of the inflammatory pathogenesis of PACG.[13] Studies have explored the changes of cytokines in the aqueous humor in various eye diseases, including uveitis and branch retinal vein occlusion.[14,15] A previous study found that the concentrations of several inflammation-related cytokines in the aqueous humor of APACG were increased significantly and that the inflammation was relieved when the IOP decreased.[16] The concentrations of cytokines in the aqueous humor of patients with APACG, including interleukin (IL)-6, IL-8 and monocyte chemotactic protein (MCP), were significantly increased compared with the unaffected eye of the same patient, suggesting an inflammatory response may be involved in the occurrence and development of PAC.[17] Some Studies

have compared the biological parameters of the anterior segment, choroid and inflammatory factors in the aqueous humor of PACG and normal eyes.[18,19] Yet, few comparative studies exist on the ocular biological parameters and inflammatory factors in the blood between APACG, CPACG and healthy people.[20,21]

This current study compared and analysed the anatomical structures of the eyeball, the systemic inflammatory index neutrophil/lymphocyte ratio (NLR) and the peripheral blood levels of IL-6 and tumour necrosis factor (TNF)-α in three groups of participants: APACG, CPACG and control subjects. It aimed to investigate the pathogenesis of PACG and provide a theoretical basis for the future treatment of PACG.

Patients and methods

Study design and population

This case–control study enrolled consecutive patients diagnosed with APACG and CPACG at the Aier Eye Hospital of Shaoyang, Shaoyang, Hunan Province, China between May 2021 and May 2022. Patients with senile cataract were enrolled as the control group from the same hospital over the same time period. All study participants were aged 55–80 years. The inclusion criteria for the APACG group were as follows: (i) at least two of the following symptoms: eye or periocular pain, nausea, vomiting or both, and intermittent blurred vision and rainbow vision in the past; (ii) intraocular pressure $\geq$22 mmHg and at least three or more of the following signs: conjunctival hyperaemia, corneal epithelial oedema, moderate pupil dilation, sluggish light reflex, small anterior chamber; (iii) the presence of angle blockage confirmed by gonioscopy; (iv) after 1–10 days of an acute attack but the corneal oedema was relieved and the interstitial clarity after IOP treatment. The inclusion criteria for the CPACG group were as follows: (i) no symptoms of acute exacerbation; (ii) no previous signs of acute exacerbation; (iii) peripheral anterior synechiae (PAS) at least one quadrant by gonioscopy; (iv) IOP $>$ 21 mmHg; (v) with normal optic disc and visual field/glaucoma optic disc and visual field defect. The inclusion criteria for the control group were as follows: (i) cup-to-disk ratio $<$0.5 and the difference in the cup-to-disk ratio between the two eyes $<$0.2; (ii) normal anterior chamber depth, open angle of the entire circumference under gonioscopy and IOP $<$ 21 mmHg; (iii) spherical diopter of $-$3.00 to $+$3.00 diopters; (iv) normal visual field; (v) no family history of glaucoma.

The exclusion criteria for all participants were as follows: (i) diabetes mellitus, systemic lupus erythematosus and other systemic immune diseases; (ii) secondary glaucoma such as trauma, uveitis, surgery, or a history of any type of laser treatment; (iii) any iris or corneal abnormalities; (iv) retinal disease or neuro-ophthalmic disease; (v) uncooperative examination; (vi) myopia or hyperopia (greater than $+$3 or $-$3 diopters); (vii) clinically relevant opacity of the optical medium and a cataract or unstable fixation leading to poor image quality.

This current study was approved by the Ethics Committee of the Aier Eye Hospital of Shaoyang, Shaoyang, Hunan Province, China (no. SYER2020102801). All participants provided written informed consent for participation in the study and publication of their images. All patient details were de-identified. The reporting of this current study conforms to the STROBE guidelines.[22]

Ophthalmic examinations

All of the eyes included in the study underwent a complete ophthalmic evaluation including visual acuity measurement, slit lamp biomicroscopy, gonioscopy, IOP measurement (KAT – Keeler Applanation

Tonometer – 'Take-Away-T'; Keeler, Windsor, UK), fundus optic nerve examination and visual field (VF). Participants of control group in the study had a best-corrected visual acuity of 0.3, an intraocular pressure <21 mmHg and a refractive error in the range of −3 to +3 diopters. The biometric parameters of the eyes were measured using an IOLMaster® 500 optical biometer (Zeiss, Gottingen, Germany). The following parameters were measured: eye axis length (AL) and lens thickness (LT). Repeated measurements were undertaken with a Pentacam (WaveLight® Topolyzer® Vario Diagnostic Device; Alcon, Geneva, Switzerland) in dark room conditions by a single professional ophthalmologist. The software then automatically calculated various corneal and anterior chamber parameters as follows: corneal thickness (CT), corneal volume (CV), corneal diameter (CD), mean corneal curvature (MCC/K), anterior chamber depth (ACD) and anterior chamber volume (ACV). Sweep coherent optical tomography (SS-OCT; CIRRUS HD-OCT; Zeiss) was used to obtain images of the macular region. Five-layer thickness maps of 6 × 6 mm were automatically segmented by the manufacturer's software. SFCT and central macular thickness (CMT) measurements were performed using built-in software.

IL-6, TNF-α and NLR measurements

On the second day of hospitalization at the Aier Eye Hospital of Shaoyang, participants provided three tubes (3 ml) of cubital venous blood in the morning after an overnight fast. The blood samples were centrifuged using a TD4A desk centrifuge (Changsha Yingtai Instrument, Changsha, China) at 3000 *g* for 5 min at 4°C. Serum was aliquoted into Eppendorf tubes and stored at –80°C until use. Serum samples were diluted 1:5 and aliquoted into microplate wells and stored at 2–8°C for a maximum of 7 days.

An IL-6 enzyme-linked immunosorbent assay (ELISA) kit (Changsha Weimi Biotechnology, Changsha, China) was used to measure serum IL-6 levels. The minimum detectable concentration was 2 pg/ml. A TNF-α ELISA kit (Changsha Weimi Biotechnology) was used to measure serum TNF-α levels. The minimum detectable concentration was 2 pg/ml for TNF-α. Intra- and interassay coefficients of variation for both ELISAs were <8% and <10%, respectively.

White blood cell, neutrophil and lymphocyte counts were measured via a complete blood cell count using a blood cell analyser (Mindray BC-5380 Auto Haematology Analyser; Mindray, Shenzhen, China).. NLR was calculated manually.

Statistical analyses

All statistical analyses were performed using IBM SPSS Statistics for Windows, Version 22.0 (IBM Corp., Armonk, NY, USA). Clinical data and measurements were tabulated for all participants and diagnostic groups. The Shapiro–Wilk test was used to evaluate the normal distribution of dat. Normally distributed data are expressed as mean ± SD. The differences in parameters such as AL, LT, CT, CV, CD, MCC/K, ACD, ACV, SFCT, CMT, IL-6 and TNF-α among the three groups were compared using a multiple comparison analysis method. Independent samples *t*-test was used to compare two groups. When data were not normally distributed, the median (interquartile range) is presented and nonparametric tests (Kruskal–Wallis) were used for comparison. The correlation between choroidal thickness and inflammatory factors IL-6 and TNF-α was analysed using Pearson correlation

coefficient test. A P-value <0.05 was considered statistically significant.

Results

This case–control study analysed a total of 99 eyes: 34 eyes from 34 patients with APACG, 28 eyes from 28 patients with CPACG and 37 eyes from 37 control patients with senile cataract. The mean ± SD age of the study cohort was 68.78 ± 8.15 years (range, 55–80 years). There were 38 males and 61 females.

Comparison of the anterior and posterior segment parameters between APACG, CPACG and control eyes are presented in Table 1. There were significant differences among the three groups in terms of AL, CD, MCC/K, ACD, ACV, LT and SFCT ($P < 0.05$ for all comparisons). There were no statistically significant differences in CMT, CT and CV among the three groups. The AL, CD, ACD and ACV were significantly smaller in the APACG and CPACG groups compared with control eyes ($P < 0.001$, $P = 0.002$, $P < 0.001$ and $P < 0.001$, respectively). The LT was significantly greater in the APACG and CPACG groups than in the controls ($P < 0.001$). The MCC/K was significantly greater in the APACG group than in the controls ($P = 0.044$). The SFCT was significantly thicker in the APACG group than in the controls ($P = 0.012$).

Comparison of the anterior and posterior segment parameters between 62 PACG and 37 control eyes are presented in Table 2. There were significant differences between the two groups in terms of AL, CD, MCC/K, ACD, ACV, SFCT and LT ($P < 0.05$ for all comparisons). The AL, CD, ACD and ACV were significantly smaller in the PACG group than in the controls ($P < 0.001$ for all comparisons). The MCC/K, LT and SFCT were significantly greater in the PACG groups than in the normal controls ($P = 0.039$, $P < 0.001$, $P = 0.002$, respectively).

Table 1. Comparison of the biometric parameters of the eyes in patients with acute primary angle-closure glaucoma (APACG), chronic primary angle-closure glaucoma (CPACG) and control subjects.

Biometric parameter	Control $n = 37$	APACG $n = 34$	CPACG $n = 28$	F/H	Statistical analyses
CD	11.32 ± 0.40	10.99 ± 0.34[a]	11.05 ± 0.49[b]	6.721	$P = 0.002$
CT	537.27 ± 33.58	536.71 ± 36.88	529.21 ± 39.35	0.461	*NS*
SFCT	151.79 ± 29.64	176.96 ± 42.98[a]	167.68 ± 26.81	6.674	$P = 0.012$
LT	4.32 ± 0.50	4.93 ± 0.30[a]	4.80 ± 0.24[b]	25.826	$P < 0.001$
MCC/K	44.27 ± 1.42	45.50 ± 1.87[a]	44.76 ± 2.80	3.228	$P = 0.044$
CMT	213.00 (202.00–225.00)	212.00 (196.00–220.25)	215.50 (201.00–230.25)	1.701	NS
ACD	2.46 (2.26–2.70)	1.64 (1.49–1.91)[a]	1.93 (1.71–2.10)[b]	51.930	$P < 0.001$
ACV	114.00 (93.50–137.50)	58.50 (52.50–69.25)[a]	74.00 (62.25–90.50)[b]	40.730	$P < 0.001$
CV	58.00 (56.20–60.30)	59.90 (53.55–61.95)	59.45 (55.78–63.75)	0.717	NS
AL	23.41 (22.89–23.99)	22.30 (22.10–22.59)[a]	22.63 (22.19–22.94)[b]	35.140	$P < 0.001$

Data presented as mean ± SD or median (interquartile range).
[a]$P < 0.05$ control group versus APACG group; [b]$P < 0.05$ control group versus CPACG group; [c]$P < 0.05$ APACG group versus CPACG group; multiple comparison analysis method; NS, no significant between-group difference ($P \geq 0.05$).
CD, corneal diameter; CT, corneal thickness; SFCT, subfoveal choroidal thickness; LT, lens thickness; MCC/K, mean corneal curvature; CMT, central macular thickness; ACD, anterior chamber depth; ACV, anterior chamber volume; CV, corneal volume; AL, axial length.

Table 2. Comparison of the biometric parameters of the eyes and inflammatory markers in patients with primary angle-closure glaucoma (PACG) and control subjects.

Parameter	Control eyes		PACG eyes		Z/F	Statistical analyses
	Eyes, *n*		Eyes, *n*			
ACD	37	2.46 (2.26–2.70)	62	1.70 (1.49–1.95)	–6.930	$P < 0.001$*
ACV	37	114.00 (93.50–137.50)	62	58.50 (51.00–68.25)	–6.084	$P < 0.001$*
AL	37	23.41 (22.89–22.99)	62	22.32 (22.21–22.66)	–5.602	$P < 0.001$*
CMT	37	213.00 (202.00–225.00)	62	209.5 (194.00–220.50)	–0.659	NS*
CV	37	58.40 ± 4.31	62	58.62 ± 6.08	4.186	NS
MCC/K	37	44.27 ± 1.42	62	45.16 ± 2.34	8.098	$P = 0.039$
LT	37	4.32 ± 0.50	62	4.87 ± 0.28	11.812	$P < 0.001$
CD	37	11.32 ± 0.40	62	11.02 ± 0.41	0.032	$P < 0.001$
CT	37	537.27 ± 33.58	62	533.32 ± 37.89	0.050	NS
SFCT	37	151.11 ± 29.44	62	173.11 ± 34.45	1.001	$P = 0.002$
Interleukin-6, pg/ml	32	2.16 (2.00–6.81)	31	1.50 (1.50–1.71)	–5.280	$P < 0.001$*
Neutrophil-to-lymphocyte ratio	32	1.56 (1.17–2.89)	31	2.23 (1.80–3.00)	2.331	$P = 0.020$*
Tumour necrosis factor-α, pg/ml	32	5.63 (3.31–11.04)	31	5.63 (3.31–11.04)	–0.098	NS*

Data presented as mean ± SD or median (interquartile range).
*Kruskal–Wallis; independent samples *t*-test was used for the other comparisons; NS, no significant between-group difference ($P \geq 0.05$).
ACD, anterior chamber depth; ACV, anterior chamber volume; AL, axial length; CMT, central macular thickness; CV, corneal volume; MCC/K, mean corneal curvature; LT, lens thickness; CD, corneal diameter; CT, corneal thickness; SFCT, subfoveal choroidal thickness.

Comparison of IL-6 and TNF-α between 31 patients with PACG and 32 control subjects are presented in Table 2. The IL-6 level in the peripheral blood of patients with PACG was significantly lower than that in the control group ($P < 0.001$). There was no significant difference in TNF-α levels in the peripheral blood between the two groups. The NLR in the peripheral blood of patients with PACG was significantly higher than that in the control group ($P = 0.020$).

Pearson correlation coefficient analysis of inflammatory markers and choroidal thickness demonstrated that the IL-6 level was inversely correlated with SFCT in patients with APACG ($r = -0.577$; $P = 0.024$) (Table 3). The IL-6 level was inversely correlated with NLR in patients with CPACG ($r = -0.603$; $P = 0.006$). TNF-α levels and NLR were not correlated with SFCT in patients with either APACG or CPACG.

Discussion

Studies have confirmed that PACG eyes often have unique anatomical features: eye axial length, small cornea, shallow anterior chamber, thicker lens and anterior lens position.[23,24] A previous study found that the position of the ciliary process and the curvature and thickness of the iris were also closely related to the narrow angle.[25] The two main types of pathological mechanism that are involved in the development of PACG are pupillary and non-pupillary block. Pupillary block is caused by increased resistance to aqueous flow due to the proximity of the lens to the posterior

Table 3. Pearson correlation coefficient analysis between subfoveal choroidal thickness (SFCT), neutrophil-to-lymphocyte ratio (NLR), interleukin (IL)-6 and tumour necrosis factor-α (TNF-α) in patients with acute primary angle-closure glaucoma (APACG) and chronic primary angle-closure glaucoma (CPACG).

	APACG $n=34$				CPACG $n=28$			
	SFCT		NLR		SFCT		NLR	
IL-6	$r=-0.577$	$P=0.024$	$r=-0.171$	$P=0.542$	$r=0.151$	$P=0.537$	$r=-0.603$	$P=0.006$
TNF-α	$r=0.202$	$P=0.470$	$r=-0.419$	$P=0.120$	$r=0.009$	$P=0.972$	$r=0.419$	$P=0.074$
NLR	$r=0.072$	$P=0.749$			$r=0.051$	$P=0.827$		

APACG, acute primary angle-closure glaucoma; CPACG, chronic primary angle-closure glaucoma; IL-6, White interleukin-6; NLR, neutrophil to lymphocyte ratio; TNF, Tumor necrosis factor-α; SFCT, subfoveal choroidal thickness.

surface of the iris.[26] This creates a pressure difference between the posterior and anterior chambers that forces the peripheral iris to bend forward.[27] Among the non-pupillary block factors are thick peripheral iris, anteriorly located peripheral iris, anteriorly circumflexed ciliary body and plateau iris. However, why do some closed-angle eyes develop acute disease and others develop chronic disease? Anatomical risk factors cannot explain some angle closures sufficiently. The use of new anterior chamber assessment techniques such as anterior segment optical coherence tomography and Pentacam have allowed the parameters of the anterior chamber to be more accurately quantified. Pentacam has proven to be a reliable and accurate measurement method of the cornea and anterior chamber.[28] The image of the anterior segment is captured by a rotating Scheimpflug camera in a fast and non-contact manner, which can provide a complete and clear 3-dimensional image of the anterior segment that can be evaluated objectively. It also quantifies anterior segment parameters (cornea and anterior chamber), such as MCC/K, CV, CD, CT, ACD and ACV.[29] A previous study showed that ACV is independently associated with the presence of narrow angle and ACV has good sensitivity and specificity in eyes with a narrow angle.[30] Research has found that measuring the ACV using a Pentacam is a better way to diagnose a narrow angle and when the patient's ACV was <110 mm, the probability of angle stenosis by gonioscopy was 9.42-times higher.[31,32] This current study found that there were significant differences in the AL, CD, ACD and ACV of the APACG and CPACG groups compared with the control subjects. The LT was significantly greater in the APACG and CPACG groups than in the controls. The MCC/K was significantly greater in the APACG group than in the controls, but there were no significant differences in CT and CV between the two groups. Consistent with the conclusions of other studies,[23,24] measuring more anterior segment parameters demonstrates that PACG has its unique anatomical characteristics and is an important factor in glaucoma onset.

In addition to the unique anatomy of PACG, there may be other physiological factors that trigger acute attacks and not all narrow-angle glaucoma will present with acute attacks throughout the patient's lifespan. The posterior choroid may play an important role in PACD according to the choroidal dilatation theory.[10] Research has demonstrated that the anterior choroid of primary open angle glaucoma (POAG)/PACD eyes is thicker than that of controls, suggesting that the anterior choroid may play an important role in the pathogenesis

of glaucoma.[33] The theory of choroidal dilatation proposes that dilation of the choroid leads to the advance of the lens-iris septum, leading to an acute attack.[34] However, further comparative research has demonstrated that the lens position during an acute attack in PACG eyes did not change.[35] Therefore, whether the choroid is the cause of acute attacks needs to be further explored. This current study found that the SFCT was significantly thicker in the APACG group compared with the control subjects. In the combined PACG group, the SFCT was significantly greater than in the control group. There was no significant difference in the CMT between the PACG and control groups in the current study. These current findings suggest that the SFCT is associated with the pathogenesis of PACG. The pathogenesis of choroidal dilatation needs to be further demonstrated in studies with larger sample sizes and more advanced methods.

Acute primary angle-closure glaucoma shows an early 'acute inflammatory' phase that has not been fully elucidated in the development of PACG.[13] Glaucoma damage is caused by a variety of factors, but the common pathway of various factors is the apoptosis of retinal ganglion cells, with cytokines being involved in the apoptotic process of retinal ganglion cells through various pathways.[36] Research is attempting to find glaucoma biomarkers in different body fluids, such as the blood, tears, aqueous humor, vitreous humor and cerebrospinal fluid.[37] IL-6 is a proinflammatory cytokine released by T cells and macrophages when stimulated by infection, trauma or other tissue damage, which in turn triggers a series of immune responses.[38] TNF-α is a pluripotent proinflammatory cytokine involved in retinal ganglion cell apoptosis in glaucoma.[39] A study found no significant difference in aqueous IL-6 and IL-8 concentrations between the PACG group and the cataract control group.[40] However, some studies found that the concentrations of IL-8, IL-6 and TNF-α in the aqueous humor of the APAG group were significantly higher than those of the cataract group.[13,41] The concentrations of IL-6, IL-8 and TNF-α in the aqueous humor of APACG were significantly higher than those of the cataract group; and CPACG patients had significantly higher levels of IL-6, IL-8 and MCP-1 in their aqueous humor than did patients with cataracts.[42] However, the level of TNF-α in the aqueous humor of CPACG was significantly different from that of non-glaucoma eyes, and the level of IL-6 was no difference between CPACG and non-glaucoma eyes.[43] These findings suggested that an inflammatory response may be involved in the occurrence and development of PAC. There are differences in the findings of the various studies, which is probably related to differences in sample size and the methods used. Most of the studies measured the levels of cytokines in the aqueous humor, but none of them measured the levels in the peripheral blood.[41–43] The levels of IL-2 and IL-6 in the peripheral blood of patients with PACG were significantly lower than the control group; and the IL-2 and IL-6 levels were significantly reduced under the condition of high IOP.[44] This current study measured the levels of IL-6 and TNF-α in the peripheral blood of the patients in the APACG, CPACG and control groups. The IL-6 level in the peripheral blood of patients with PACG was significantly lower than that in the control group. There was no significant difference in TNF-α levels between the PACG and control groups. These current findings were consistent with the results of a previous study that demonstrated significantly lower serum IL-6 levels and no significant difference in serum TNF-α levels between Posner-Schlossman syndrome patients and cataract controls.[45] This may lead to the increase of cortisol secretion *in vivo* due to a stress reaction, which then inhibits the synthesis and release of

cytokines.[46] At present, there are few studies on serum IL-6 and TNF-α levels in patients with PACG and there is no evidence to confirm that serum cytokine levels are directly correlated with the cytokine levels in the eyes.[47] This area of research requires further investigation with larger sample sizes.

In tumour tissue, neutrophils promote the proliferation and metastasis of cancer cells by forming an inflammatory microenvironment; with lymphocytes being the main components of anti-tumour cellular immunity, so a high NLR indicates an enhanced inflammatory state and a weakened anti-tumour immune function.[48] Research into role of NLR in ocular diseases is ongoing. For example, a review of the laboratory and clinical data of PACG patients and controls found that NLR was independently related to PACG, suggesting that NLR might be used as a biomarker of PACG and that a low-level inflammatory response might be involved in the occurrence and development of PACG.[49] A previous study found that the NLR in patients with high IOP and POAG was higher than that in elderly cataract controls; and the standard deviation of the glaucoma visual field defect index pattern was also positively correlated with NLR.[50] This current study found that the NLR in the peripheral blood of patients with PACG was significantly higher than that of the control group, which was consistent with the conclusions of other studies.[49,50] These current findings suggest that a low-level inflammatory response is involved in the occurrence and development of PACG. Studies have demonstrated the anatomical differences between APACG and CPACG.[9,51]

To date, there have been no studies on the relationship between choroidal thickness and cytokine levels. This current study found that the IL-6 levels in the peripheral blood were inversely correlated with SFCT in APACG and inversely correlated with NLR in CPACG.

This current study had several limitations. First, although the findings reached statistical significance, only 62 PACG eyes were analysed. Future research in larger sample sizes should be undertaken. Secondly, the role of the dynamic changes in choroidal thickness in the pathogenesis of PACG requires further research.

This current study used an optical biometer, a Pentacam and SS-OCT to simultaneously measure the biometric parameters of APACG, CPACG and control eyes. It also measured the levels of IL-6, TNF-α and NLR in the peripheral blood. The current findings suggest that IL-6 and NLR are associated with the pathogenesis of PACG. The mechanism behind this association requires further study.

Author contributions

Joint direction of the study: Zhi-Neng Ning and Xian Wang; conception and design: Hong-Bo Zeng, Jun-Di Jiang, Zhi-Neng Ning and Xian Wang; administrative support: Rong Li and Jian-Guo Xie; provision of study materials and/or patients: Jian Li and Wen-Juan Zeng; data collection: Xiong-Kai Li, Bin Hu, Fei Zhou and Hai-Ying Yu; data analysis and interpretation: Hong-Bo Zeng and Jun-Di Jiang; manuscript preparation: all authors; final approval of the manuscript: all authors.

Declaration of conflicting interests

The authors declare that there are no conflicts of interest.

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Supplemental material

Supplemental material for this article is available online.

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