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Original Article

Association of ocular biometrics with clinical severity in acute angle-closure glaucoma with cataract: a retrospective study

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Abstract: Objective: To determine the correlation between axial length (AL), anterior chamber depth (ACD), and lens thickness (LT) with clinical severity in patients with acute angle-closure glaucoma (AACG) complicated with cataracts. Methods: This retrospective study analyzed the clinical data of 247 consecutive patients (247 eyes) diagnosed with AACG complicated with cataracts. Ocular biomechanical parameters (AL, ACD, LT) were measured using IOL Master. Recorded clinical characteristics included peak intraocular pressure (IOP), best corrected visual acuity (BCVA, LogMAR), angle closure extent (quadrant number), and corneal edema grade. Statistical analysis was performed using Pearson correlation analysis and multiple linear regression. Results: The mean AL, ACD, and LT were 22.11±0.24 mm, 1.90±0.07 mm, and 5.08±0.17 mm, respectively. Pearson correlation analysis showed that ACD was strongly negatively correlated with peak IOP, BCVA (LogMAR), angle closure extent, and corneal edema grade (all $P<0.001$); LT was significantly positively correlated with all the above clinical indicators (all $P<0.001$); while AL showed a weaker negative correlation with the above indicators (all $P<0.01$). Multivariate regression identified ACD ($\beta=-3.042$, $P<0.001$) and LT ($\beta=0.732$, $P<0.001$) as independent predictors of peak IOP. Peak IOP, ACD, LT, and female gender were all independent influencing factors for BCVA (all $P<0.05$). Conclusion: In patients with AACG and cataract, shallow anterior chamber and thickened lens are important independent anatomical determinants of more severe clinical presentation. These findings support the use of ACD and LT as key biomarkers for risk assessment and individualized treatment planning.

Keywords: Acute angle-closure glaucoma, cataract, axial length, anterior chamber depth, lens thickness; clinical characteristics

Introduction

Glaucoma is one of the leading causes of irreversible vision loss worldwide. Its pathological feature is progressive optic neuropathy, manifested as damage to the retinal nerve fiber layer and optic disc, accompanied by typical visual field defects such as arcuate scotoma and nasal stepladder. This disease seriously threatens visual health and quality of life [1, 2]. In 2020, approximately 3.61 million cases of blindness and 4.14 million cases of moderate to severe visual impairment worldwide were attributed to glaucoma, an increase of over 60% compared to 1990 [3]. It is projected that by 2050, the number of cases of low vision or blindness caused by glaucoma will increase to 11.8 million [4]. Clinically, glaucoma is primarily categorized into open-angle glaucoma (OAG) and angle-closure glaucoma (ACG). Although OAG is more prevalent in Western Caucasian populations, ACG has a significantly higher prevalence in East Asia (especially China), with more acute and severe clinical manifestations and a greater risk of blindness, posing a serious public health challenge in ophthalmology [5, 6]. The core pathological change in ACG is the mechanical closure of the iridocorneal angle, obstructing the outflow of aqueous humor through the trabecular meshwork, leading to pathologically elevated intraocular pressure [7].

Among the various clinical subtypes of ACG, acute angle-closure glaucoma (AACG) is the most critical and considered one of the most

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urgent ophthalmic emergencies requiring immediate treatment [8]. The classic pathogenesis of AACG is "pupil block": when the pupil is moderately dilated, the contact area between the iris and lens increases, increasing the resistance to aqueous humor flowing from the posterior chamber into the anterior chamber through the pupil. This results in a relatively increased pressure in the posterior chamber, pushing the peripheral iris forward and bulging, eventually adhering to the trabecular meshwork, causing sudden closure of the angle and obstruction of aqueous humor outflow. During an attack, intraocular pressure can surge to over 50 mmHg within hours [9]. Clinically, AACG patients often experience severe symptoms including intense eye pain, radiating headache on the same side, nausea, and vomiting, accompanied by typical ocular manifestations such as sudden vision loss and halos around objects [10]. The rapid increase in intraocular pressure during an attack can quickly trigger corneal endothelial decompensation, leading to corneal epithelial edema and blurred vision. More seriously, the high pressure can cause mechanical compression and ischemic damage to the optic disc lamina cribriformis, and hinder axoplasmic transport. If intraocular pressure is not effectively reduced in time, a large number of retinal ganglion cells will die within 48 to 72 hours, causing permanent and irreversible vision loss [11, 12].

It is noteworthy that the peak incidence of AACG typically is in middle-aged and elderly people over 50 years old, which coincides with the high incidence of cataracts [13]. Therefore, AACG and cataracts often coexist clinically, with the two being mutually causal in pathophysiology, forming a vicious cycle. Cataracts are essentially lens opacities, but their progression involves more than just decreased transparency, which are accompanied by complex biological and morphological changes [14]. With aging, the lens not only continues to grow, increasing in size and thickness, but also shifts forward due to physiological zonular laxity [15]. This age-related thickening and forward shift of the lens, known as "lens-related factor", compresses the posterior chamber space, worsening pupillary block and pushing the entire iris root forward, further crowding the already anatomically vulnerable narrow angle and making it more prone to closure [16]. Consequently, in elderly patients, the natural progression of cataract itself is a key intrinsic factor inducing and aggravating AACG. The comorbidity of cataracts and glaucoma not only increases the complexity of clinical diagnosis but also places higher demands on treatment options.

The onset of AACG is not accidental, whose fundamental cause lies in the specific anatomical vulnerability of the anterior segment of the patient, namely, "anterior segment crowding" [17]. A series of ocular biometric parameters collectively determine the spatial structure of the anterior segment. Among them, axial length, anterior chamber depth, and lens thickness are recognized as three key quantitative indicators for assessing anterior segment crowding and predicting the risk of ACG. It is generally accepted that short axial length, shallow anterior chamber depth, and large lens thickness are independent risk factors for predicting the occurrence and progression of primary ACG and its early stages [18-20]. Modern ophthalmic imaging techniques and high-precision optical or ultrasonic biometric equipment can perform precise and repeatable quantitative analysis of these minute intraocular anatomical structures at the millimeter or even micrometer level, providing solid technical support for clinical risk screening and assessment.

Current research mostly uses these ocular anatomical parameters as static indicators for predicting acute angle closure. However, in-depth research on how these parameters quantitatively affect the clinical manifestations during acute attacks, especially in patients with cataracts, remains relatively lacking. Therefore, this study employed a retrospective translational medicine research methodology to collect biometric and clinical data from patients with AACG combined with cataract. The correlation between axial length (AL), anterior chamber depth (ACD), lens thickness (LT), and clinical severity indicators was analyzed to elucidate the intrinsic mechanisms by which anatomical structures influence clinical manifestations, providing a basis for more precise risk stratification and treatment decisions in clinical practice. The innovation of this study lies in its systematic quantification of the association between static anatomical parameters and dynamic clinical severity indicators in the acute phase, providing clinicians with evidence-based thresholds for identifying high-risk patients and

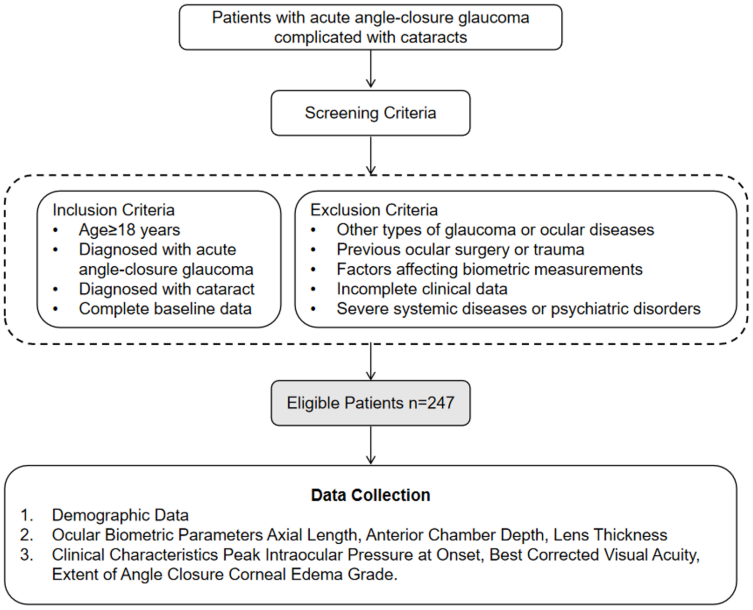

Figure 1. Patient screening process.

guiding the development of individualized treatment plans.

Methods

Study design and patient selection

This was a single-center, retrospective observational study conducted in the Department of Ophthalmology, at the Ninth People's Hospital of Zibo City, China. We retrospectively reviewed the hospital information system and ophthalmology examination database of the hospital, screening for hospitalized patients diagnosed with both AACG and cataracts who were admitted between January 2022 and December 2025. The diagnostic criteria for AACG were: acute onset, accompanied by symptoms such as eye pain, headache, and nausea; conjunctival hyperemia, corneal edema, shallow anterior chamber, and moderate pupillary dilation; intraocular pressure (IOP) >21 mmHg; and gonioscopy confirming angle closure. Cataracts were diagnosed by slit-lamp examination revealing lens opacity.

Inclusion criteria were: (1) Age ≥ 18 years; (2) Confirmed diagnosis of AACG with cataract; (3) Completion of baseline ocular biometry and clinical feature assessment within 24 hours of admission; and (4) Complete medical records.

Exclusion criteria were: (1) Other types of glaucoma (e.g., OAG, secondary glaucoma); (2) Other serious ocular diseases (e.g., retinal detachment, uveitis); (3) History of intraocular surgery or ocular trauma; (4) Cases where biometry could not be accurately completed (e.g., severe corneal opacity); and (5) Incomplete clinical data. The patient screening process is detailed in **Figure 1**.

Data collection and extraction

Treatment options, including medication selection, timing of surgical intervention, and surgical procedures (such as phacoemulsification cataract extraction combined with intraocular lens implantation, and, if necessary, goniosynostosis or trabeculectomy), were determined by the attending ophthalmologist based on the patient's ocular condition, overall health, and clinical judgment. Before any intervention, the ophthalmologist has thoroughly explained all treatment options, including potential benefits, risks, and limitations, to the patient and their legal guardian. The study adheres to the principles of the Declaration of Helsinki and hospital ethical guidelines.

The study used standardized data collection forms to extract data from patient medical records. Data extraction was performed by two independent researchers; any disagreements were resolved through consensus or consultation with a third senior researcher. Extracted data included demographic information: age, sex, and affected eye.

Ocular bioparameters: AL, ACD, and LT were measured at admission using an IOL Master 700 (Carl Zeiss Meditec, Germany). Measurements were taken three times per eye, and the average value was used for analysis.

Clinical characteristics: Peak IOP was measured using a Topcon non-contact tonometer (mmHg). Best corrected visual acuity (BCVA) was measured using a standard visual acuity chart and converted to log minimum resolution angle (LogMAR) for analysis. Angle closure extent was assessed by gonioscopy and recorded as the number of quadrants with closure (0-4).

Table 1. Basic characteristics of the study population (N=247)

Characteristic	Value
Age (years), mean ± SD	67.87±8.68
Gender, n (%)	
Male	81 (32.79%)
Female	166 (67.21%)
Affected eye, n (%)	
Right	126 (51.01%)
Left	121 (48.99%)

Corneal edema severity was graded by slit-lamp examination, ranging from grade 0 (no edema) to grade 3 (severe edema, inability to visualize intraocular structures).

Outcome measures

All examinations were completed within 24 hours of admission, at baseline, and before any treatment intervention.

(1) Primary outcome measures: Ocular biometrics: All patients underwent ocular biometrics measurements upon admission using an IOL Master 700 (Carl Zeiss Meditec, Germany), performed by an experienced ophthalmologist. Measurements were taken three times per eye, and the average value was used as the final result. Specific parameters included: AL: Distance from the corneal apex to the retinal pigment epithelium; ACD: Distance from the corneal endothelium to the anterior surface of the lens; LensT: Distance from the anterior surface to the posterior surface of the lens. Peak IOP: Measured at admission using a Topcon non-contact tonometer.

(2) Secondary outcome measures: BCVA: Measured at admission using an international standard visual acuity chart and converted to LogMAR values for statistical analysis. Higher LogMAR values indicate poorer visual acuity. Corneal edema severity: Graded based on slit-lamp examination results upon admission. The grading criteria are as follows: Grade 0: No corneal edema; Grade 1: Mild edema, slight decrease in corneal transparency, corneal texture clearly visible; Grade 2: Moderate edema, significant decrease in corneal transparency, blurred corneal texture; Grade 3: Severe edema, cornea appears milky white and cloudy, intraocular structures cannot be observed. Higher grades indicate more severe edema. Angle closure extent: Assessed by gonioscopy upon admission, the number of quadrants with angle closure was recorded, ranging from 0 to 4 quadrants. Higher values indicate a wider angle closure extent.

Statistical analysis

SPSS 26.0 software was used for statistical analysis. Normally distributed continuous data were expressed as mean ± standard deviation (±SD), and categorical data were presented as number of cases and percentages (n, %). Pearson correlation analysis was used to assess the linear correlation between ocular biometric parameters and clinical indicators. To identify independent risk factors, this study constructed two multiple linear regression models using a stepwise selection method (inclusion criterion α=0.05, exclusion criterion α=0.10). The first model used peak IOP as the dependent variable, with age, sex, AL, ACD, and LT as independent variables. The second model used BCVA (LogMAR) as the dependent variable, with age, sex, peak IOP, AL, ACD, and LT as independent variables. The variance inflation factor (VIF) was calculated to test for multicollinearity, and a two-sided *p*-value <0.05 was considered statistically significant.

Results

Basic characteristics

This study included 247 patients (247 eyes) with acute angle-closure glaucoma complicated by cataracts. All patients had unilateral disease and complete clinical data. Among them, 81 were male (32.79%) and 166 were female (67.21%), with a significantly higher proportion of female patients. The mean age of the patients was 67.87±8.68 years. Regarding the distribution of affected eyes, 126 cases (51.01%) involved the right eye and 121 cases (48.99%) involved the left eye; there was no significant difference in the proportion of patients affected by both eyes (see **Table 1** for details).

Ocular biometric parameters and clinical characteristics

The AL was 22.11±0.24 mm, the ACD was 1.90±0.07 mm, and the LT was 5.08±0.17 mm;

Table 2. Measurement results of ocular biological parameters of patients and clinical characteristics of the patients

Ocular biometric parameters	Measured value (mm)	Range
Axial length	22.11±0.24	21.62-22.48
Anterior chamber depth	1.90±0.07	1.76-2.04
Lens thickness	5.08±0.17	4.80-5.46
Clinical characteristics	Measured values/grading	Range
Peak intraocular pressure at onset	56.36±4.49	48.5-66.1
Best corrected visual acuity	1.61±0.35	1.0-2.5
Extent of angle closure	3.46±0.34	1.0-4.0
Corneal edema grade	2.38±0.38	0-3

Table 3. Results of the correlation analysis between ocular biometric parameters and clinical characteristic indices

Indicators	Axial length		Anterior chamber depth		Lens thickness	
	r (95% CI)	*P*	r (95% CI)	*P*	r (95% CI)	*P*
Age	-0.199 (-0.316 to -0.076)	0.002	-0.384 (-0.485 to -0.272)	<0.001	0.307 (0.190 to 0.416)	<0.001
Peak intraocular pressure at onset	-0.168 (-0.286 to -0.044)	0.008	-0.648 (-0.715 to -0.569)	<0.001	0.535 (0.440 to 0.618)	<0.001
Best corrected visual acuity	-0.173 (-0.291 to -0.049)	<0.001	-0.690 (-0.750 to -0.619)	<0.001	0.623 (0.540 to 0.694)	<0.001
Extent of angle closure	-0.200 (-0.317 to 0.077)	0.002	-0.703 (-0.761 to -0.634)	<0.001	0.608 (0.523 to 0.682)	<0.001
Corneal edema grade	-0.182 (-0.300 to -0.058)	0.004	-0.697 (-0.756 to 0.626)	<0.001	0.621 (0.538 to 0.692)	<0.001

the peak IOP during an attack was 56.36±4.49 mmHg; the BCVA (converted to LogMAR) was 1.61±0.35; the angle closure extent was 3.46±0.34 quadrants; and the corneal edema grade was 2.38±0.38. See **Table 2**.

Pearson correlation analysis of ocular biometric parameters and clinical characteristic indicators

Pearson correlation analysis showed that AL, ACD, and LT were significantly correlated with the clinical indicators. See **Table 3**.

- AL was significantly negatively correlated with age, peak IOP during an attack, angle closure extent, corneal edema, and best corrected visual acuity (LogMAR) (all *P*<0.01).

- ACD was significantly negatively correlated with all clinical characteristics (all P<0.001), making it the biometric parameter with the strongest correlation to clinical characteristics.

- LT was significantly positively correlated with peak intraocular pressure during an attack, angle closure extent, corneal edema, and best corrected visual acuity (LogMAR) (all P<0.001), and also positively correlated with age (P< 0.001).

Results of linear regression analysis

Linear regression model with peak IOP during attacks as the dependent variable: Age, gender, AL, ACD, and LT were used as independent variables for univariate and multivariate linear regression analysis. The results are shown in **Table 4**.

Univariate analysis revealed that AL, ACD, and LT, were all significantly correlated with peak intraocular pressure (all P<0.05): AL was negatively correlated (β=-0.313, P=0.008), ACD was significantly negatively correlated (β=-3.971, P<0.001), and LT was positively correlated (β=1.418, *P*<0.001).

After variable screening, the multivariate stepwise regression analysis retained ACD and LT as independent predictors in the final model. ACD showed the strongest predictive effect (β=-3.042, P<0.001), while LT remained statistically significant (β=0.732, P<0.001). However, after controlling for other variables, AL, age, and gender did not show independent statistical significance (all P>0.05). The variance inflation factor for all independent variables was <10, indicating that the model did not have significant multicollinearity.

Table 4. Results of multiple linear regression analysis for factors influencing peak intraocular pressure during attack

Univariate	β	S.E	95% CI	t	*P*
Age	0.163	0.031	0.101 to 0.224	5.190	<0.001
Gender/female	-0.152	0.609	-1.352 to 1.048	-0.250	0.803
Aaxial length	-0.313	0.117	-0.544 to -0.081	-0.266	0.008
Anterior chamber depth	-3.971	0.298	-4.558 to -3.384	-1.332	<0.001
Lens thickness	1.418	0.143	1.136 to 1.699	0.991	<0.001
Multivariate	β	S.E	95% CI	t	*P*
Age	0.019	0.026	-0.033 to 0.071	0.714	0.476
Gender/female	0.118	0.443	-0.754 to 0.990	0.267	0.790
Aaxial length	-0.241	0.899	-2.012 to 1.530	-0.268	0.789
Anterior chamber depth	-3.042	0.340	-3.712 to -2.373	-8.948	<0.001
Lens thickness	0.732	0.145	0.447 to 0.102	5.058	<0.001

Table 5. Results of multiple linear regression analysis for factors influencing best-corrected visual acuity

Univariate	β	S.E	95% CI	t	*P*
Age	0.014	0.002	0.009 to 0.019	5.867	<0.001
Gender/female	0.028	0.048	-0.066 to 0.122	0.585	0.559
Peak intraocular pressure at onset	0.068	0.003	0.063 to 0.073	26.480	<0.001
Axial length	-0.254	0.092	-0.435 to -0.072	-2.747	0.006
Anterior chamber depth	-3.329	0.223	-3.768 to -2.890	-14.928	<0.001
Lens thickness	1.299	0.104	1.093 to 1.504	12.455	<0.001
Multivariate	β	S.E	95% CI	t	*P*
Age	0.001	0.001	-0.002 to 0.004	0.789	0.431
Gender/female	0.044	0.022	0.001 to 0.087	2.031	0.043
Peak intraocular pressure at onset	0.050	0.003	0.044 to 0.057	15.925	<0.001
Axial length	0.018	0.044	-0.069 to 0.105	0.412	0.681
Anterior chamber depth	-0.854	0.193	-1.234 to -0.475	-4.435	<0.001
Lens thickness	0.396	0.075	0.249 to 0.543	5.297	<0.001

Linear regression model with "BCVA (LogMAR value)" as the dependent variable: Univariate and multivariate linear regression analyses were conducted with age, gender, peak IOP, AL, ACD, and LT as independent variables. The results are shown in **Table 5**.

Univariate analysis indicated that peak IOP, AL, ACD, and LT during an attack were all significantly correlated with BCVA (LogMAR) (all *P*< 0.01): peak IOP was positively correlated (β= 0.068, P<0.001), AL was negatively correlated (β=-0.254, P=0.006), ACD was strongly negatively correlated (β=-3.329, *P*<0.001), and LT was positively correlated (β=1.299, *P*<0.001).

The independent risk factors retained by the multivariate step wise regression model included: peak IOP during an attack (β=0.050, P< 0.001), ACD (β=-0.854, P<0.001), LT (β=0.396, P<0.001), and sex (female) (β=0.044, P= 0.043). AL and age did not show independent effects in the multivariate model (P>0.05). The variance inflation factor for all variables was <10, indicating a good model fit.

Discussion

AACG complicated with cataract is a major ophthalmic emergency in the middle-aged and elderly population, which can lead to sudden vision loss and even permanent blindness [21]. Its core pathophysiological mechanism is the crowding of the anterior segment anatomy, causing sudden angle closure and a sharp increase in intraocular pressure [22]. AL, ACD, and LT, as key biometric parameters reflecting the spatial structure of the anterior segment,

are not only important indicators for predicting the risk of ACG but may also directly influence the clinical severity of acute attacks [23]. The results of this study are consistent with previous research on anatomical risk factors for ACG [24, 25], and extend the relevant understanding to the clinical manifestations during acute attacks. Demographic analysis of this study showed that the proportion of female patients was significantly higher (67.21%), with a mean age of 67.87±8.68 years, which is highly consistent with the epidemiological features of ACG reported by Xiao et al. [26] and the globally recognized conclusion that ACG is highly prevalent in elderly women [3]. The decline in estrogen levels in postmenopausal women may lead to zonular laxity and anterior displacement of the lens, which in turn aggravates anterior segment crowding [27]. The high incidence of this disease in middle-aged and elderly people is closely related to age-related physiological thickening of the lens. In this study, LT was significantly positively correlated with age, further confirming this pathophysiological basis [28]. Ocular biometrics revealed that patients exhibited typical anatomical features compared to the general population, including shorter AL, shallower ACD and thicker lens. This supports the classic theory that "anterior segment crowding" is the core anatomical basis for the pathogenesis of AACG [29, 30].

Correlation analysis showed that ocular biometrics were closely related to clinical characteristics. AL was significantly negatively correlated with peak IOP during an attack, angle closure extent, the corneal edema grade, and BCVA, suggesting that patients with shorter AL had more severe acute attacks. The pathophysiological mechanism may be that a shorter AL reduces the anteroposterior diameter of the eyeball, causing compression of the anterior segment structures, shortening the distance between the iris and lens, and significantly enhancing the pupillary block effect, thereby inducing sudden angle closure and a sharp increase in intraocular pressure [31]. In addition, a short AL is often accompanied by increased scleral rigidity, and the mechanical compression and ischemic damage to the optic nerve lamina cribriformis under high pressure are more severe, thus exacerbating optic function impairment. It is worth noting that this result is consistent with the study by Wang et al. [32], which also pointed out that short axial length and shallow anterior chamber often coexist in ACG, and the two have a synergistic effect on intraocular pressure and visual prognosis. However, this study found that the correlation strength between AL and various clinical indicators was weaker than that between anterior chamber depth and lens thickness, suggesting that in AACG patients complicated with cataracts, AL, as a macroscopic anatomical parameter, may indirectly affect the condition through intermediate variables such as ACD and LT.

In this study, ACD demonstrated the most significant predictive value, showing the most significant negative correlation with all clinical characteristic indicators, becoming the anatomical parameter most closely related to the severity of the condition in this investigation. Multivariate linear regression analysis further confirmed that anterior chamber depth is an independent risk factor affecting peak IOP and best corrected visual acuity during an attack. This result is consistent with the study by Zheng et al. [33], which pointed out that shallow anterior chamber is an anatomical risk factor independent of LT and AL. This study expands its clinical significance, demonstrating that anterior chamber depth not only predicts disease risk but also quantitatively reflects the severity of acute attacks. A shallower ACD means a narrower aqueous humor outflow channel (angle), resulting in a more pronounced pupillary block effect caused by lens displacement. Once the angle is completely closed and normal aqueous humor drainage is obstructed, IOP can rise sharply within a short period. Simultaneously, in a shallow anterior chamber state, corneal endothelial cells experience relatively insufficient nutrient supply, making them more prone to functional decompensation under high-pressure stress, leading to increased corneal edema and consequently affecting vision. Therefore, anterior chamber depth can serve as a core indicator for clinically assessing the severity of AACG complicated with cataracts. Patients with extremely shallow ACD (e.g., <1.8 mm) should be classified as high-risk individuals requiring enhanced monitoring and timely intervention.

LT, as a core indicator of "lens-related factor", demonstrated significant clinical relevance in this study. It is significantly positively correlated with peak IOP, angle closure extent, corneal

edema grade and BCVA during the attack; and in the multivariate regression model, LT is an independent predictor of peak IOP and BCVA. This result is consistent with the study by Liu et al. on the role of lens morphology in age-related cataracts and angle closure [34], highlighting the active role of the lens in the pathogenesis of AACG. With aging, the lens gradually thickens and may shift forward, which not only directly occupies the anterior segment space and makes the anterior chamber shallower, but also aggravates pupillary block by increasing the contact area between the iris and the lens, while pushing the iris root forward, further squeezing the already narrow angle, and finally inducing angle closure and acute attack. In addition, the increase in LT can also indirectly affect the magnitude of intraocular pressure increase and the degree of optic nerve damage by changing the intraocular refractive state and increasing scleral hardness. The positive correlation between lens thickness and age observed in this study also explains the high incidence of AACG and cataracts in middle-aged and elderly people. This suggests that for elderly cataract patients with significantly thickened lenses, routine gonioscopy and intraocular pressure monitoring are necessary to be alert to the occurrence of AACG [35, 36].

The linear regression analysis in this study also yielded noteworthy findings. After controlling for other variables, AL was no longer an independent risk factor for peak intraocular pressure and best-corrected visual acuity during an attack. This seems to contradict the significant association between AL and clinical indicators in the correlation analysis, but it actually reflects the complex interaction between ocular anatomical parameters. As a comprehensive anatomical indicator, the effect of axial length on anterior segment crowding may be indirectly mediated by ACD and LT, i.e., short AL is usually accompanied by a shallow anterior chamber and a thick lens; in the multivariate model, the latter two, as more direct anatomical risk factors, may mask the independent effect of axial length itself [37]. Furthermore, regression models with best-corrected visual acuity as the dependent variable revealed that peak IOP during an attack was an independent risk factor, along with gender (female). This indicates that, in addition to anatomical structure, acute damage to retinal ganglion cells and corneal endothelial cells caused by elevated intraocular pressure, as well as potential anatomical differences in women (such as a shallower anterior chamber and a thicker lens), jointly determine the degree of visual impairment in patients. This result points to a crucial direction for clinical diagnosis and treatment: treatment strategies should not only target potential anatomical susceptibility factors but also emphasize early and rapid intraocular pressure reduction to minimize irreversible damage to ocular tissues caused by high pressure.

Study limitations

This study employed a single-center retrospective design. Although the sample size was sufficient and the statistical analysis rigorous, certain limitations still exist. First, all included patients had clinically significant cataracts; therefore, the study conclusions cannot be directly extrapolated to AACG patients without cataracts. Second, the study primarily relied on the IOL Master optical biometry system for detection. While this method is highly accurate, it cannot dynamically assess the morphology of the iris-ciliary complex at the moment of angle closure. Finally, this study is a cross-sectional analysis. While it confirms a strong correlation, it cannot establish a clear causal relationship or temporal sequence, requiring further verification through prospective studies. Based on these limitations, future research could combine ultrasound biomicroscopy and anterior segment optical coherence tomography to conduct dynamic observations at multiple time points during acute attacks, remission, and postoperative periods. This would quantitatively analyze the changes in parameters such as anterior chamber depth, lens thickness, and angle opening distance, and their correlation with intraocular pressure control stability. Simultaneously, prospective cohort studies could be conducted, stratifying patients based on the aforementioned anatomical parameters to compare the long-term efficacy and safety of various surgical procedures in different anatomical subgroups, and constructing an individualized surgical approach selection model based on anatomical characteristics. In addition to geometric parameters, integrating more dimensions of imaging and functional indicators, such as lens density, iris volume, and elasticity assessment, could potentially lead to a more comprehensive risk assessment and prognostic prediction system for AACG.

Conclusion

In summary, this study, by combining static anatomical parameters with dynamic clinical manifestations, deepens our understanding of the pathological mechanisms of complication with cataracts. The results support the use of ACD and LT as key biomarkers for assessing the severity of acute attacks in these patients, laying the foundation for future personalized treatment pathways based on multi-parameter models.

Disclosure of conflict of interest

None.

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