Biometric Factors Associated With Acute Primary Angle Closure: Comparison of the Affected and Fellow Eye

Eray Atalay,[1] Monisha E. Nongpiur,[1,2] Mani Baskaran,[1,2] Sourabh Sharma,[1] Shamira A. Perera,[3] and Tin Aung[1,3,4]

[1]Singapore Eye Research Institute, Singapore
[2]Duke-NUS Graduate Medical School, Singapore
[3]Singapore National Eye Centre, Singapore
[4]Yong Loo Lin School of Medicine, National University of Singapore, Singapore

Correspondence: Tin Aung, Singapore National Eye Center, 11 Third Hospital Avenue, Singapore 168751; aung.tin@singhealth.com.sg.

Submitted: May 26, 2016
Accepted: August 25, 2016

Citation: Atalay E, Nongpiur ME, Baskaran M, Sharma S, Perera SA, Aung T. Biometric factors associated with acute primary angle closure: comparison of the affected and fellow eye. *Invest Ophthalmol Vis Sci*. 2016;57:5320–5325. DOI:10.1167/iovs.16-20006

Purpose. To compare ocular biometric and anterior segment parameters between the affected and fellow eye in subjects with acute primary angle closure (APAC).

Methods. We evaluated 76 subjects with unilateral APAC who had undergone bilateral laser peripheral iridotomy before enrollment. Imaging was done using anterior segment optical coherence tomography and a customized software was used to measure the following: angle opening distance (AOD750); trabecular–iris space area (TISA750); iris thickness (IT750); iris curvature (ICURV); iris area (IAREA); anterior chamber depth; area and volume (ACD; ACA and ACV); anterior chamber width (ACW); anterior vault (ACD+LV); lens vault (LV); and pupil diameter (PD). We used A-scan ultrasonography to measure axial length (AL) and lens thickness (LT). Mean differences in ocular biometric and anterior segment parameters were assessed using linear mixed model adjusting for PD.

Results. A total of 53 subjects (36 females, 67.9%) with a mean age of 62.7 ± 8.1 years were analyzed after excluding 17 unanalyzable images in at least one eye. Affected eyes had shallower ACD, smaller ACA, ACV, anterior vault, TISA750, AOD750, and ICURV (all $P < 0.05$). Axial length, ACW, LV, LT, IAREA, and IT750 did not differ between the eyes. In the affected eyes, IT750 was significantly associated AOD750 ($P < 0.05$); whereas in the fellow eyes, IT750 and AL was predictive of AOD750 (all $P < 0.05$).

Conclusions. Eyes with previous APAC had smaller anterior segment dimensions when compared with their fellow eyes. Iris thickness was the strongest predictor of angle width in both affected and fellow eyes.

Keywords: acute primary angle closure, AS-OCT, biometry

Although the majority of primary angle closure glaucoma cases follow an asymptomatic course, approximately 20% of patients present with acute primary angle closure (APAC) that is associated with a significant risk of vision loss if treatment is not instituted promptly.[1–5] Acute primary angle closure can occur at any time point within the primary angle closure disease spectrum and it has much higher incidence in East Asia, such as in Singapore, where the highest incidence has been reported.[6] After an APAC episode, progression to chronic angle closure glaucoma occurs in nearly 50% of eyes and one-fifth develop blindness in the following 5 years.[7]

The relative or absolute dimensions of anterior segment structures are known to determine the likelihood of developing angle closure.[8] Using imaging modalities, studies have reported several anatomic risk factors associated with angle closure which include shallower anterior chamber depth (ACD); smaller anterior chamber area (ACA); anterior chamber volume (ACV); increased lens vault (LV); and greater iris curvature (ICURV).[9–12] In an already predisposed eye, the increased pressure gradient between the anterior and posterior chamber (pupillary block) causes anterior bowing of the peripheral iris with resultant blockage of trabecular meshwork.[13,14] In eyes with APAC, it is not exactly known how these series of events cause extensive trabecular occlusion with sudden IOP elevation. However, not all fellow eyes of patients with APAC develop an acute attack; if left untreated, the risk of experiencing an acute attack in the fellow eye is 40% to 80% over 5 to 10 years.[15,16] Although primary angle closure disease is essentially a bilateral condition, roughly only 10% to 15% of cases present with bilateral APAC.[6,17–20] Factors that predispose the affected eye to APAC are currently contested and published literature, comparing anatomical characteristics of the affected and the fellow eye of the same subjects, is limited. In a study that compared biometric parameters of the eye with APAC and the fellow eye, affected eyes were shown to have shallower ACD and more anteriorly positioned lens compared with the uninvolved eye.[21] Other studies conducted using anterior segment optical coherence tomography (AS-OCT) are inconclusive, with one study showing no difference between the affected and the fellow eye and others showing LV or ACD as the main parameter associated with APAC.[22–24]

Given the largely unilateral nature of APAC, we hypothesize that there may be some inherent differences in biometric properties of the affected and unaffected fellow eyes that persist after laser peripheral iridotomy (LPI). These dissimilarities might help explain the pathogenesis of APAC. Therefore,

we compared the ocular biometric and anterior segment parameters of the affected and unaffected eye in patients with previous APAC after LPI.

Materials and Methods

Approval for this cross-sectional study was granted by the institutional review board of the hospital and was conducted in adherence to the tenets of the Declaration of Helsinki. Written informed consent was obtained from all the subjects.

Subjects previously diagnosed with unilateral APAC were prospectively recruited from glaucoma clinics of the Singapore National Eye Center. All subjects had undergone bilateral LPI prior to enrollment. Patients in whom LPI was performed more than 18 months before recruitment were excluded. Subjects who had undergone cataract surgery and/or argon laser peripheral iridoplasty in any eye were not included. Each subject underwent a standardized ophthalmic examination which included assessment of visual acuity; slit-lamp examination; stereoscopic evaluation of the optic disc using a 78-diopter lens (Volk Optical, Inc., Mentor, OH, USA); and intraocular pressure (IOP) measurement with Goldmann applanation tonometry (Haag-Streit, Koniz, Switzerland), automated refraction, and keratometry (Canon RK, 5 Auto Ref-Keratometer; Canon, Inc., Ltd., Tochigiken, Japan). A-scan ultrasonography (model US-800; Nidek Co, Ltd., Tokyo, Japan.) was used to measure axial length (AL) and lens thickness (LT). Relative lens position (RLP) was calculated using the following formula: ([ACD + 0.5 LT]/AL). Spherical equivalent was defined as sphere plus half cylinder.

Gonioscopy was performed at the time of recruitment by an experienced examiner in the dark using a 4-mirror lens (Sussman; Ocular Instruments, Inc., Bellevue, WA, USA) at ×16 magnification. The presence and extent of peripheral anterior synechiae (PAS, defined as abnormal adhesions of the iris to the angle that were at least half a clock hour in width and were present to the level of the anterior trabecular meshwork or higher) was determined.

Definitions

Acute primary angle closure was defined by the presence of any two of the following symptoms: ocular or periocular pain, nausea and/or vomiting, an antecedent history of intermittent blurring of vision with haloes, presenting IOP of greater than 21 mm Hg; and presence of at least three of the following signs: conjunctival injection, corneal epithelial edema, mid-dilated pupil, and shallow anterior chamber.

Primary angle closure suspect (PACS) was diagnosed in patients with angle closure (defined as eyes in which at least 180° of the posterior pigmented trabecular meshwork was not visible on gonioscopy in the primary position of gaze without indentation) with IOP ≤21 mm Hg, healthy optic disc, and without PAS.

Primary angle closure (PAC) was diagnosed in eyes with angle closure, healthy optic discs, and visual fields, but with elevated IOP (defined as an IOP >21 mm Hg), and/or PAS.

Primary angle closure glaucoma (PACG) was diagnosed on the basis of angle closure associated with glaucomatous optic neuropathy and compatible visual field loss on static automated perimetry.

AS-OCT Imaging and Analysis

All subjects underwent AS-OCT imaging (Visante; Carl Zeiss Meditec, Jena, Germany) by an operator who was masked to the results of the clinical ophthalmic examination. Imaging was performed under a standardized lighting condition (0 lux, Light Meter, FC-840021; Sper Scientific, Scottsdale, AZ, USA). None of the patients were on topical miotics at the time of imaging. Scans were centered on the pupil and taken along the horizontal axis (nasal-temporal angles at 0 to 180° with a scan length of 16 mm) using the standard anterior segment single-scan protocol. The best quality images were obtained by adjusting the saturation and noise, and by optimizing the polarization for each scan during the examination. Care was taken to minimize motion artefacts, or image artefacts due to the eyelids. The examiner chose the best image, with no motion or image artifacts for further analysis.

One cross-sectional horizontal AS-OCT scan of the nasal and temporal angle was evaluated for each subject. These images were processed using customized software (Zhongshan Angle Assessment Program [ZAAP]; Zhongshan Ophthalmic Centre, Guangzhou, China)[25] by a single observer, masked to clinical data. The only observer input was to determine the location of the scleral spurs. The algorithm then automatically calculated the anterior segment parameters. The following parameters were measured: angle opening distance at 750 μm (AOD750) from the scleral spur; trabecular–iris space area at 750 μm from the scleral spur (TISA750); iris thickness at 750 μm from the scleral spur (IT750); ICURV; iris area (IAREA); ACD; anterior chamber width (ACW); anterior chamber area (ACA); ACV; and LV. Anterior vault represented the sum of ACD and LV.[26] Pupil diameter (PD) was defined as the shortest distance between the pupil edges of the iris cross-sections and measured using the custom software calipers tool (Zhongshan Ophthalmic Centre).

Statistical Analysis

Both eyes of a single patient were included in the analysis. Ocular biometric and anterior segment parameters measured by AS-OCT were compared between the affected and the nonaffected fellow eye. Linear mixed effects model was used to adjust for the nonindependence of the right and left eye. The intereye correlation was accounted for by fitting the data of both eyes as a repeated measurement in the model. Differences in mean values of parametric data between the affected and fellow eyes were analyzed after adjusting for PD. Data was expressed as mean ± SD. Univariable logistic regression was performed to determine biometric and anterior segment parameters that were associated with occurrence of APAC. Those parameters reaching $P < 0.05$, after adjusting for age sex and pupil diameter were included in the multivariable logistic regression model. Stepwise multiple linear regression (significance level to enter $P < 0.05$) was used to identify variables that predicted AOD750 (as a surrogate of angle width)[27] in the affected and fellow eyes. Variables were entered into the explanatory model based on the greatest improvement in R^2 and those variables with variance inflation factor >5 were removed so as to diminish the effect of multicollinearity. We did not include TISA750 in the stepwise multiple linear regression model due to its close association with AOD750.[28] Statistical analysis was performed using statistical software (SPSS Statistics, version 21.0; IBM Corp., Armonk, NY, USA); and statistical significance was assumed at $P < 0.05$ levels.

Results

A total of 70 patients with APAC were enrolled, of whom 17 were excluded due to poor-quality AS-OCT images, software delineation error, or indeterminate scleral spurs in at least one eye, leaving 53 (75.7%) patients with complete data for the

Table 1. Demographic Information of the Subjects

Characteristics	Total, $n = 53$
Age, y	62.7 ± 8.1
Sex, M/F	17/36
Ethnicity	
Chinese	46
Malay	4
Indian	2
Other	1
Eyes with APAC, R/L	32/21
Diagnoses of eyes with APAC	
PAC	39 (73.6%)
PACG	14 (26.4%)
Diagnoses of fellow eyes	
PACS	45 (84.9%)
PAC	5 (9.4%)
PACG	3 (5.7%)

analysis. The mean age of the included patients at presentation was 62.7 ± 8.1 years; 36 (67.9%) were female subjects and 46 (86.8%) were Chinese. The excluded patients did not differ from the included patients in terms of age, sex, and ethnicity. Table 1 lists the distribution of diagnoses between the affected and fellow eye.

The comparison of biometric and anterior segment parameters between the affected and fellow eye is shown in Table 2. Fellow eyes were more hyperopic and had steeper corneal curvatures compared with the affected eyes ($P = 0.002$ and $P = 0.05$, respectively). Although the central corneal thickness was higher in the affected eyes, the mean difference was only 6.6 μm ($P = 0.03$, 95% confidence interval [CI]: −12.62, −0.56). After adjusting for pupil diameter, affected eyes had shallower ACD, smaller ACA, ACV, anterior vault, TISA750, AOD750, and ICURV (all $P < 0.05$). Axial length, ACW, LV, LT, RLP, central corneal thickness, IT750, and IAREA did not differ between the eyes. Pupil diameter was larger in the affected eye when compared with the fellow eye ($P < 0.05$). Table 3 summarizes the age, sex, and PD adjusted results of the univariable and multivariable logistic regression analyses of the association between the biometric/anterior segment parameters and occurrence of APAC. In the multivariable logistic regression, a 0.1-mm decrease in TISA750 (odds ratio [OR]: 24.97, 95% CI: 3.85–161.83), ICURV (OR: 2.63, 95% CI: 1.26–5.52), and ACD (OR: 1.52, 95% CI: 1.20–1.94), was significantly associated with APAC (all $P < 0.05$).

Table 4 presents the results of the stepwise multiple linear regression analyses for factors associated with AOD750. In the affected eyes; IT750 was the only significant variable ($P < 0.05$), explaining 43% of the variability in AOD750. The model in fellow eyes consisted of two parameters (IT750 and AL) which overall explained 43% of the variability in AOD750. By itself, IT750 explained 24% of the variation in AOD750 in the fellow eyes.

Discussion

In this cross-sectional study of subjects with APAC, the mean ACD was on average 11.7% smaller in affected eyes when compared with the fellow eyes. It is likely that narrower angles and smaller anterior chamber parameters exacerbate angle crowding in such eyes. Despite the difference in ACD, the axial length and parameters related to lens (lens thickness, RLP) were not different between the affected and fellow eyes. In the final multivariable logistic regression analysis, a smaller ACD, TISA750 and ICURV were the parameters that could differentiate the affected and fellow eyes. Although the direction of association with ICURV (OR: 2.63/0.1-mm decrease) is paradoxical, it is in agreement with previous reports.[22,23,29]

The established factors that predispose to primary angle closure disease include shorter axial length, shallower anterior chamber, smaller corneal curvature, greater LV, greater iris curvature, area, and thickness.[11] In two recent AS-OCT studies, eyes with APAC had the smallest anterior segment dimensions

Table 2. Comparison of Clinical Features, A-Scan Biometry, and AS-OCT Parameters Between Eyes With APAC and Fellow Eyes

Parameters	Eyes With APAC	Eyes w/o APAC	Adjusted Mean Difference*	95% CI of the Difference	*P* Value*
IOP at presentation, mm Hg	52.57 ± 9.49	17.39 ± 8.52	35.18	−38.83, −31.54	<0.001
Spherical equivalent	0.65 ± 2.20	1.28 ± 1.78	0.63	0.25, 1.02	0.002
Average keratometry	7.65 ± 0.24	7.63 ± 0.24	0.02	0.00, 0.045	0.05
Axial length, mm	22.44 ± 0.75	22.46 ± 0.84	0.02	−0.09, 0.13	0.70
Central corneal thickness, μm	576.7 ± 42.1	570.1 ± 37.2	6.60	−12.62, −0.56	0.03
Lens thickness, mm	4.51 ± 0.92	4.65 ± 0.86	0.14	−0.09, 0.37	0.22
Relative lens position	0.22 ± 0.02	0.22 ± 0.02	0.001	−0.005, 0.007	0.78
Mean Shaffer	1.12 ± 0.82	1.64 ± 0.90	0.52	0.29, 0.76	<0.001
Mean total PAS	3.47 ± 3.87	0.93 ± 2.44	2.55	−3.69, −1.41	<0.001
ACD, mm	1.82 ± 0.31	2.06 ± 0.24	0.24	0.16, 0.31	<0.001
Anterior vault, mm	2.83 ± 0.39	3.05 ± 0.16	0.22	0.12, 0.33	<0.001
ACW, mm	11.43 ± 0.38	11.44 ± 0.35	0.0002	−0.07, 0.07	0.99
ACA, mm^2	13.42 ± 2.22	14.93 ± 2.10	1.51	0.92, 2.10	<0.001
ACV, mm^3	85.30 ± 15.25	95.01 ± 16.08	9.71	5.75, 13.66	<0.001
AOD750, mm	0.125 ± 0.076	0.164 ± 0.077	0.039	0.017, 0.061	0.001
TISA750, mm^2	0.067 ± 0.036	0.100 ± 0.037	0.033	0.023, 0.044	<0.001
IT750, mm	0.501 ± 0.099	0.507 ± 0.103	0.006	−0.022, 0.034	0.66
IAREA, mm^2	1.501 ± 0.255	1.523 ± 0.248	0.022	−0.004, 0.049	0.09
ICURV, mm	0.145 ± 0.073	0.193 ± 0.109	0.048	0.011, 0.085	0.01
Lens vault, mm	1.01 ± 0.44	1.00 ± 0.22	0.01	−0.13, 0.10	0.81
PD, mm	4.39 ± 0.85	3.95 ± 0.72	0.44	−0.64, −0.24	<0.001

* Linear Mixed Model: adjusted for intereye correlation, pupillary diameter.

Investigative Ophthalmology & Visual Science

Table 3. Relationship of Biometric and AS-OCT Parameters With Presence of APAC

Predictor Variable	Univariable Logistic Regression			Multivariable Logistic Regression*		
	OR	95% CI	*P* Value	OR	95% CI	*P* Value
Spherical equivalent, per D increase	0.86	0.70, 1.06	0.17			
Average keratometry, per 0.1 mm decrease	0.92	0.77, 1.10	0.35			
Axial length, per mm increase	0.98	0.93, 1.04	0.46			
Lens thickness, per mm increase	0.98	0.94, 1.03	0.47			
ACD, per 0.1 mm decrease	1.43	1.18, 1.72	**<0.001**	1.52	1.20, 1.94	**0.001**
Anterior vault, per 0.1 mm decrease	1.46	1.18, 1.80	**<0.001**	1.25	0.97, 1.61	0.08
ACW, per 0.1 mm decrease	1.06	0.94, 1.19	0.33			
Lens vault, per 0.1 mm increase	0.99	0.88, 1.12	0.86			
AOD750, per 0.1 mm decrease†	1.92	1.06, 3.46	**0.03**			
TISA750, per 0.1 mm^2 decrease	12.47	3.35, 46.35	**<0.001**	24.97	3.85, 161.83	**0.001**
IT750, per 0.1 mm increase	0.77	0.49, 1.22	0.27			
IAREA, per 0.1 mm^2 increase	1.06	0.89, 1.26	0.52			
ICURV, per 0.1 mm decrease	1.85	1.05, 3.27	**0.03**	2.63	1.26, 5.52	**0.01**

Univariable and multivariable analyses were adjusted for age, sex, and pupillary diameter; bold values are $P < 0.05$.

* Including variables with $P < 0.05$ in univariable analysis.

† Not included in the multivariable analysis due to multicollinearity with TISA750.

and the greatest LV when compared with PACS, PAC, and PACG.[24,30] Moghimi et al.[24] showed that anterior segment features of fellow eyes mimicked those of affected eyes with no significant differences between the two groups. In our patients, fellow eyes had greater anterior segment dimensions (ACD, ACA, ACV, and anterior vault) and wider anterior chamber angles (TISA750 and AOD750) when compared to the affected eyes. These findings are consistent with previous studies that have reported shallower ACD,[21–23,29,31,32] smaller ACA,[22,23,29] and narrower angle widths[22,29,32] (TISA and AOD) in eyes with APAC. We also found that eyes with APAC had flatter corneas when compared with the fellow eyes. This observation corroborates early biometric reports by Lowe,[33] Alsbirk,[34] and a more recent work by Friedman et al.[5] It is likely that the composite effect of shallower anterior chamber and flatter cornea might be leading to crowding of the angle and subsequent appositional closure.[5]

We have found novel associations between angle width (AOD750) and other AS-OCT parameters in subjects with APAC after LPI. The single predictor of angle width in the affected eyes was IT750; whereas in the fellow eyes, AL and IT750 were predictive of AOD750. These results are in contrast to a large population-based cross-sectional study which has found the angle width to be largely dependent on ACA, ACV and LV.[28] Considering that anterior chamber dimensions are very small in our patients, it is reasonable to assume that there exists a threshold level (floor effect) in related parameters (ACA and ACV) below which the association with AOD750 is diminished. In our study, IT750 as a single predictor explained 43% and 24% of the variability in AOD750, in affected and fellow eyes, respectively. This contrasts with the findings of the aforementioned population-based study in which IT750 only explained 4% to 6% of the variability in AOD750.[28] The consistent and strong association between IT750 and AOD750 in affected and fellow eyes collectively suggests that the angle width becomes more dependent on iris thickness with smaller ocular dimensions. It is also possible that the APAC eyes and fellow eyes may have the extreme iris morphology (as described by Quigley[35]) predisposing them to such events.

Table 4. Stepwise Multiple Linear Regression Analysis on AOD750 in Affected and Fellow Eyes

Variables in Model, *n*	Variable	β Regression Coefficient (95% CI)	Partial R Square	Model R Square
Affected eye				
1	IT750, μm	−0.653 (−0.644, −0.314)*	0.43	0.43
Fellow eye				
1	IT750, μm	−0.486 (−0.780, −0.211)*	0.24	0.24
2	AL, mm	0.433 (0.185, 0.673)*	0.19	0.43

* Regression coefficient is significant at $P \leq 0.001$.

Lens vault, an AS-OCT parameter that is a measurement of the extent of lens located anterior to the plane of the anterior chamber angles, is independently associated with angle closure.[12] In our study, where AS-OCT imaging was done post-LPI, neither LV nor lens thickness was different between the two eyes. This contrasts with the findings of previous reports which have demonstrated a greater LV in the affected APAC eyes at the time of the attack before any laser intervention.[22,23,29] The timing of AS-OCT imaging may underlie the discrepancy in LV findings. Based on the differential effect of LPI in the affected and fellow eye, Quigley et al.[36] postulated that a greater increase in ACD 4 months after LPI in the affected eyes might be due to the forward movement of the lens at the time of the attack. Choroidal expansion is the inciting event in this model which causes the anterior lens movement. As a corollary to this hypothesis, Moghimi et al.[37] in a longitudinal study have noted a decrease in LV and a shift in lens position after the resolution of the attack. They reasoned that these changes may not be due to the LPI but to a cessation of ciliary body and choroidal inflammation.[37] Zonular laxity might be responsible, in part, for forward movement of the lens and the increase in LV in acute angle closure.[36] Considering the post-LPI nature and the relatively long interval between the acute attack and imaging in our patients, we speculate that reversal of choroidal expansion and inflammation might have already occurred, thereby explaining the similarity in LV in our sample.

One of the limitations of our study was the cross-sectional design and the sample population consisting mostly of Chinese subjects. Secondly, although expected to be representative of the global anterior segment morphology, only a single cross-section image was used. Furthermore, post-LPI evaluation of the anterior segment might not reflect the exact status of the eye at the time of the attack. Although the analyses were adjusted for PD, the effect of a statistically significant difference between the affected and fellow eyes on the results is

unknown. While the RLP was not different between the affected and fellow eyes, we were unable to rule out the presence of zonular laxity and lens subluxation. The aforementioned methodological limitations preclude drawing causal inferences and establishing temporal relationships. However, due to the visually destructive nature of APAC, it is ethically inappropriate to withhold treatment with an aim to assess such temporal relationships in a longitudinal study. The strengths of our study include a relatively large sample size compared with similar previous studies. We also used an appropriate statistical model to account for the nonindependence of fellow eyes of the same patient which has not been considered in many of the previous similar studies.[21,23,24,29,31,32]

In conclusion, we observed that affected eyes with APAC have smaller anterior segment dimensions when compared to their fellow eyes. Although smaller TISA750, ICURV, and ACD were significantly associated with the occurrence of APAC, the only plausible causative factor among these would be a decreased ACD, as the other factors are likely to be altered by the acute attack and/or the LPI. We have also identified a novel association between iris thickness and angle width in patients with APAC which explains approximately one third of the variability in AOD750.

Acknowledgments

Supported by the Singapore Ministry of Health's National Medical Research Council, under its Singapore Translational Research Investigator Award (NMRC/STAR/0023/2014) and Translational and Clinical Research Flagship Programme-Tier 2 (NMRC/TCR/008-SERI/2013), Singapore, Republic of Singapore.

Disclosure: **E. Atalay**, None; **M.E. Nongpiur**, None; **M. Baskaran**, None; **S. Sharma**, None; **S.A. Perera**, None; **T. Aung**, None

References

1. Aung T, Ang LP, Chan SP, Chew PT. Acute primary angle-closure: long-term intraocular pressure outcome in Asian eyes. *Am J Ophthalmol*. 2001;131:7–12.
2. Dandona L, Dandona R, Mandal P, et al. Angle-closure glaucoma in an urban population in southern India. The Andhra Pradesh eye disease study. *Ophthalmology*. 2000;107:1710–1716.
3. Foster PJ, Baasanhu J, Alsbirk PH, Munkhbayar D, Uranchimeg D, Johnson GJ. Glaucoma in Mongolia. A population-based survey in Hovsgol Province Northern Mongolia. *Arch Ophthalmol*. 1996;114:1235–1241.
4. Foster PJ, Oen FT, Machin D, et al. The prevalence of glaucoma in Chinese residents of Singapore: a cross-sectional population survey of the Tanjong Pagar district. *Arch Ophthalmol*. 2000;118:1105–1111.
5. Friedman DS, Gazzard G, Foster P, et al. Ultrasonographic biomicroscopy, Scheimpflug photography, and novel provocative tests in contralateral eyes of Chinese patients initially seen with acute angle closure. *Arch Ophthalmol*. 2003;121:633–642.
6. Seah SK, Foster PJ, Chew PT, et al. Incidence of acute primary angle-closure glaucoma in Singapore. An island-wide survey. *Arch Ophthalmol*. 1997;115:1436–1440.
7. Aung T, Friedman DS, Chew PT, et al. Long-term outcomes in Asians after acute primary angle closure. *Ophthalmology*. 2004;111:1464–1469.
8. Ritch R, Liebmann JM. Role of ultrasound biomicroscopy in the differentiation of block glaucomas. *Curr Opin Ophthalmol*. 1998;9:39–45.
9. Nongpiur ME, Sakata LM, Friedman DS, et al. Novel association of smaller anterior chamber width with angle closure in Singaporeans. *Ophthalmology*. 2010;117:1967–1973.
10. Wang BS, Narayanaswamy A, Amerasinghe N, et al. Increased iris thickness and association with primary angle closure glaucoma. *Br J Ophthalmol*. 2011;95:46–50.
11. Nongpiur ME, Ku JY, Aung T. Angle closure glaucoma: a mechanistic review. *Curr Opin Ophthalmol*. 2011;22:96–101.
12. Nongpiur ME, He M, Amerasinghe N, et al. Lens vault, thickness, and position in Chinese subjects with angle closure. *Ophthalmology*. 2011;118:474–479.
13. Gazzard G, Friedman DS, Devereux JG, Chew P, Seah SK. A prospective ultrasound biomicroscopy evaluation of changes in anterior segment morphology after laser iridotomy in Asian eyes. *Ophthalmology*. 2003;110:630–638.
14. Nolan WP, Foster PJ, Devereux JG, Uranchimeg D, Johnson GJ, Baasanhu J. YAG laser iridotomy treatment for primary angle closure in east Asian eyes. *Br J Ophthalmol*. 2000;84:1255–1259.
15. Edwards RS. Behaviour of the fellow eye in acute angle-closure glaucoma. *Br J Ophthalmol*. 1982;66:576–579.
16. Lowe RF. The natural history and principles of treatment of primary angle-closure glaucoma. *Am J Ophthalmol*. 1966;61:642–651.
17. Bain WE. The fellow eye in acute closed-angle glaucoma. *Br J Ophthalmol*. 1957;41:193–199.
18. Lowe RF. Acute angle-closure glaucoma. The second eye: an analysis of 200 cases. *Br J Ophthalmol*. 1962;46:641–650.
19. Hillman JS. Acute closed-angle glaucoma: an investigation into the effect of delay in treatment. *Br J Ophthalmol*. 1979;63:817–821.
20. Ang LP, Aung T, Chew PT. Acute primary angle closure in an Asian population: long-term outcome of the fellow eye after prophylactic laser peripheral iridotomy. *Ophthalmology*. 2000;107:2092–2096.
21. Lim MC, Lim LS, Gazzard G, et al. Lens opacity, thickness, and position in subjects with acute primary angle closure. *J Glaucoma*. 2006;15:260–263.
22. Sng CC, Aquino MC, Liao J, et al. Pretreatment anterior segment imaging during acute primary angle closure: insights into angle closure mechanisms in the acute phase. *Ophthalmology*. 2014;121:119–125.
23. Lee JR, Sung KR, Han S. Comparison of anterior segment parameters between the acute primary angle closure eye and the fellow eye. *Invest Ophthalmol Vis Sci*. 2014;55:3646–3650.
24. Moghimi S, Vahedian Z, Fakhraie G, et al. Ocular biometry in the subtypes of angle closure: an anterior segment optical coherence tomography study. *Am J Ophthalmol*. 2013;155:664–673, 673.e1.
25. Console JW, Sakata LM, Aung T, Friedman DS, He M. Quantitative analysis of anterior segment optical coherence tomography images: the Zhongshan Angle Assessment Program. *Br J Ophthalmol*. 2008;92:1612–1616.
26. Kim YK, Yoo BW, Kim HC, Aung T, Park KH. Relative lens vault in subjects with angle closure. *BMC Ophthalmol*. 2014;14:93.
27. Narayanaswamy A, Sakata LM, He MG, et al. Diagnostic performance of anterior chamber angle measurements for detecting eyes with narrow angles: an anterior segment OCT study. *Arch Ophthalmol*. 2010;128:1321–1327.
28. Foo LL, Nongpiur ME, Allen JC, et al. Determinants of angle width in Chinese Singaporeans. *Ophthalmology*. 2012;119:278–282.
29. Moghimi S, Zandvakil N, Vahedian Z, et al. Acute angle closure: qualitative and quantitative evaluation of the anterior segment using anterior segment optical coherence tomography. *Clin Exp Ophthalmol*. 2014;42:615–622.
30. Guzman CP, Gong T, Nongpiur ME, et al. Anterior segment optical coherence tomography parameters in subtypes of

primary angle closure. *Invest Ophthalmol Vis Sci*. 2013;54: 5281–5286.

31. Lan YW, Hsieh JW, Hung PT. Ocular biometry in acute and chronic angle-closure glaucoma. *Ophthalmologica*. 2007;221: 388–394.
32. Zhang HT, Xu L, Cao WF, Wang YX, Jonas JB. Anterior segment optical coherence tomography of acute primary angle closure. *Graefes Arch Clin Exp Ophthalmol*. 2010;248:825–831.
33. Lowe RF. Aetiology of the anatomical basis for primary angle-closure glaucoma. Biometrical comparisons between normal eyes and eyes with primary angle-closure glaucoma. *Br J Ophthalmol*. 1970;54:161–169.
34. Alsbirk PH. Primary angle-closure glaucoma. Oculometry epidemiology, and genetics in a high risk population. *Acta Ophthalmol Suppl*. 1976;5–31.
35. Quigley HA. The iris is a sponge: a cause of angle closure. *Ophthalmology*. 2010;117:1–2.
36. Quigley HA, Friedman DS, Congdon NG. Possible mechanisms of primary angle-closure and malignant glaucoma. *J Glaucoma*. 2003;12:167–180.
37. Moghimi S, Chen R, Johari M, et al. Changes in anterior segment morphology after laser peripheral iridotomy in acute primary angle closure. *Am J Ophthalmol*. 2016;166:133–140.

Investigative Ophthalmology & Visual Science