The Course of the Changes in Anterior Chamber Parameters After Laser Peripheral Iridotomy: Follow-up for 6 Months With a Scheimpflug-Placido Disc Topographer

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Purpose: To evaluate the course of the changes in anterior chamber (AC) parameters using a Scheimpflug-Placido disc topographer before and through 6 months after laser peripheral iridotomy (LPI).

Materials and Methods: A total of 109 eyes of 56 consecutive patients classified as primary angle closure suspect (PACS), primary angle closure (PAC), and primary angle closure glaucoma (PACG) were included in this prospective, interventional, observational case series. Anterior chamber volume (ACV), central anterior chamber depth (CACD), and anterior chamber angle (ACA) were measured by Scheimpflug photography preoperatively and at 1, 3, and 6 months after LPI. With respect to the parameters above, alterations in the measurements were assessed to determine whether the effects induced by LPI on AC morphology persisted with time.

Results: At 1 month after LPI, mean ACV, CACD, and ACA increased significantly in all groups ($P < 0.05$). However, in eyes with PACG, significant reductions at 3 months in ACA [0.00 (−1.00 to 0.00) degrees, median (Md) [quartile 1 (Q1) to quartile 3 (Q3)], $P = 0.032$] and at 6 months in ACV [−1.00 (−2.25 to 1.00) μL, $P = 0.043$) and CACD (−0.01 (−0.02 to 0.00) mm, $P = 0.006$) were shown. On analysis of all eyes together, a weak correlation was found between axial length and the change in CACD ($r = 0.266$, $P = 0.007$). Eyes with ACA ≤ 25 degrees widened by 6.6 ± 2.8 degrees versus 4.9 ± 2.4 degrees in eyes with ACA > 25 degrees at 1 month after the procedure ($P = 0.002$).

Conclusions: Scheimpflug-Placido disc topographer detected significant changes in the AC parameters after LPI in all groups. However, compared with PACS and PAC, the PACG showed significant alterations in the AC parameters through 6 months.

Key Words: Scheimpflug-Placido disc topographer, anterior chamber parameters, laser peripheral iridotomy, angle closure glaucoma

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Glaucoma is a leading cause of irreversible blindness.[1] It is estimated that by the year 2020 about 80 million people worldwide will suffer from glaucoma, and nearly quarter of these will be the angle closure glaucoma (ACG).[2] The prevalence of ACG is as low as 0.04% in people of European descent; rates up to 3% have been reported from Asian countries among people older than 40 years.[3,4] In recent years, ACG or narrow angles have been classified using terms that also describe the stages of the disease. These include primary angle closure suspect (PACS), primary angle closure (PAC), and primary angle closure glaucoma (PACG).[4] However the current treatment algorithm, which should interrupt the natural course and prevent significant morbidity, is still far from the optimum.[5]

Laser peripheral iridotomy (LPI) is currently the first step intervention for both acute and chronic forms of ACG. It eliminates the pressure gradient between anterior and posterior chambers and resolves pupillary block. In addition, LPI flattens iris convexity, increases peripheral anterior chamber angle (ACA), and thus reduces appositional angle closure.[6] Reduction in appositional angle closure is believed to be associated with cessation of peripheral anterior synechiae formation (PAS), which in turn enables better control of intraocular pressure (IOP).[7] However, despite patent iridotomies, the long-term efficacy is variable. A study from Asia reported that 58% of the eyes that underwent LPI after acute angle closure (AAC) required additional measures including trabeculectomy, which was 94% in the eyes with PACG according to another study.[8,9] It is noteworthy that regarding this substantial discrepancy, AAC in the former referred to symptomatic acute IOP elevations rather than implying any stages of the disease as PACG in the latter. Even among the eyes with PACS, which is considered as the earliest stage of disease, antiglaucoma medication was required in 17% of the cases during the 10-year follow-up.[10] Therefore, we still need a better understanding of the course of narrow angles and the role of LPI.[4]

Several studies have aimed to evaluate the changes produced in the anterior chamber (AC) after LPI. The changes were documented with the use of different imaging techniques such as gonioscopy, ultrasound biometry, anterior segment optical coherence tomography (AS-OCT), and Scheimpflug photography.[5,11–23] Gonioscopy is still an invaluable clinical tool in the diagnosis of ACG.[5] However, its variable reproducibility, potential for inducing angle distortion through contact application, and the need for a skilled examiner limit its use as an objective quantitative measure.[24] Although ultrasound biometry is a more precise and a high-definition technique particularly for studying angle recess, it is cumbersome and necessitates immersion.[25] Despite its noncontact nature and high intra-interobserver reproducibility, AS-OCT is a compromise between motion artifact and definition of scleral spur, which is essential for quantifying ACA.[24,26]

During the last years, new devices based on the Scheimpflug principle have been used more commonly in corneal refractive surgery. Depending on the brand, 1 or 2 rotating Scheimpflug cameras (RSC) provide fast and

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noncontact imaging of the anterior segment with 12 to 50 frames in 2 seconds. Then, morphometric variables such as the ACA, anterior chamber volume (ACV), and central anterior chamber depth (CACD) can be derived repeatedly using the software within the devices.[27,28]

In this study, we aimed to evaluate the AC parameters of eyes with narrow angles before and during the 6 months that followed LPI. The Sirius (Costruzione Strumenti Oftalmici, Florence, Italy) used in our study is a new device that combines an RSC with a Placido disc. Thus, it is capable of merging the data from Scheimpflug with those from Placido and may thus overcome the limitations of both.[29]

METHODS

This study was designed as a prospective, nonrandomized, interventional, and observational case series. After approval of the institutional ethics committee, 109 eyes of 56 consecutive patients who presented to the Eye Clinics of Bakirkoy Dr S. Konuk Training and Research Hospital between April 2011 and November 2012 were included into the study. Before enrollment, a signed informed consent and agreement for full compliance to follow-up visits were requested from all participants. The study was conducted in accordance with the Declaration of Helsinki.

Eyes that were found to be narrow on slit lamp examination by Van Herick testing and confirmed to have narrow angles by gonioscopy underwent comprehensive ophthalmic examination before recruitment. The baseline evaluation included medical and ophthalmic history, refraction, best corrected visual acuity (BCVA), slit lamp biomicroscopy, IOP (by Goldmann applanation tonometry), axial length (AxL) measurement, gonioscopic grading, fundoscopy, and the topographic imaging.

Gonioscopy was carried out by the same physician for uniformity reasons (Y.A.), using a 3-mirror lens (Design-OG3M-10; Ocular, Bellevue, WA) under dimly lit conditions and avoiding compression and pupillary constriction. To confirm appositional or synechial contact of the iris, indentation was performed afterward. AxL was obtained by A-scan USG (Cinescan, Quantel, France) as the average of 10 consecutive scans. Refraction was recorded as spherical equivalent (SE). If an eye was under antiglaucomatous medication at baseline or any time later, the number of topical antiglaucomatous medications (No.AGM) in use was noted. For example, if an eye was receiving only a prostaglandin analog, No.AGM was 1. In addition to that, if there was also a topical β-blocker, No.AGM was 2. The No.AGM for a fixed combination drop was also 2. Therefore, in the case of an additional single topical medication to a fixed combination, No.AGM was 3. Furthermore, raised IOP was defined as IOP $\geq$ 22 mm Hg without any antiglaucomatous medications.

Glaucoma was diagnosed on the basis of glaucomatous disc damage—a vertical cup disc ratio $\geq$ 0.7 or an asymmetry of vertical cup disc ratio $\geq$ 0.2 between 2 eyes, diffuse and focal neural rim thinning, and at least 2 reliable visual field tests (VF) [Humphrey Field Analyzer, Swedish Interactive Threshold Algorithm (SITA) 24.2 test; Carl Zeiss Meditec, Dublin, CA], which denote fixation losses $<$20% along with false positives and negatives $<$30%. Scotomas of 3 contiguous points at the level of 5% on the pattern deviation plot were sought on successive VFs. In cases of unreliable VFs, we went on with spectral domain OCT (SD-OCT) to seek for at least 1 sector of peripapillary retinal nerve fiber layer thinning (RNFL) at the level of 1% or 2 contiguous sectors of peripapillary RNFL thinning at the level of 5% on the TSNIT plot conforming to disc changes at least on 2 occasions (RNFL 3.45 protocol, RTVue-100 OCT; Optuvue Inc., Fremont, CA).

Eyes were classified according to the International Society for Geographical and Epidemiological Ophthalmology guidelines.[30] Eyes with angles in which there was iridotrabecular contact in 3 or more quadrants without PAS and without glaucoma were defined as PACS. Eyes that had evidence of either a synechiae and/or raised IOP in addition to the findings similar to those for PACS were defined as PAC. Finally, eyes that met the criteria for PAC and in addition had glaucoma were defined as PACG. PACS, PAC, and PACG groups included 48, 25, and 30 eyes, respectively.

Patients with a history of previous intraocular operations, use of miotics, ocular trauma, uveitis, and iris neovascularization were excluded. Eyes with signs of iris and/or corneal and/or lens abnormalities that precluded imaging or visualization of the cup and optic disc were excluded.

Before LPI, 1 drop of 2% pilocarpine hydrochloride was applied 3 times to all eyes, with a gap of 5 minutes between each drop. LPI was performed using a double aspheric iridectomy lens (Volk Optical Inc., Mentor, OH) in the upper nasal or upper temporal quadrant with a power of 1 to 6 mJ and 5 to 15 shots depending on iris thickness and pigmentation (Nd:Yag LightMed SYC900, Taipei, Taiwan). Immediately after LPI, a drop of brimonidine tartrate 0.15% was applied to the eyes without any antiglaucomatous medication (eyes in PACS) for preventing IOP spikes. Prednisolone acetate 1% was used in all eyes 4 times a day for 1 week. During the week after LPI, all patients were evaluated for possible complications including significant IOP elevation, hyphema, inflammation, and patency of iridotomy, which could have affected the outcomes and required exclusion. The primary objective of the study was to assess and document the course of anatomic and functional changes induced by LPI in the medium term. Therefore, the patients were scheduled for follow-up visits at 1, 3, and 6 months, and the status of refraction, BCVA, patency of iridotomy, IOP, No.AGM, and Scheimpflug-derived parameters were reevaluated and recorded. Antiglaucoma medications were adjusted to keep the IOP between 15 and 21 mm Hg. Particular attention to the patency of peripheral iridotomy was paid during follow-up; its closure required exclusion from the study.

The Sirius was used to take 4 successive measurements of AC parameters (ACV, CACD, ACA) at baseline (T0) and at 1 (T1), 3 (T3), 6 months (T6) after LPI. Each session was conducted by the same physician (F.U.Y.). After instillation of 1 drop of artificial tears, at least 3 sets of Scheimpflug frames were captured and the one that met "good" acquisition quality (displayed as green check mark) with the best quality indices (ie, centration %, coverage %) was selected. It is noteworthy that a single set of frames consisted of 25 slit frames captured 7 or 8 degrees apart around the central axis by a 360-degree RSC. Built-in software then processed the selected set and produced ACV and CACD (mean values) from all 25 frames. In contrast, the software was able to produce mean ACA only from 7 of 25 frames, which were penetrable to angle structures on the nasal and temporal periphery.

All data were analyzed using SPSS version 20.0 (SPSS Inc., Chicago, IL). Demographic features such as age, sex,

TABLE 1. Demographic Features of the Eyes

	Mean ± SD/n (%)			
	PACS (n = 48)	PAC (n = 25)	PACG (n = 30)	*P*
Age (y)	60.94 ± 8.37*	53.76 ± 5.70	60.50 ± 6.83	***< 0.001***†
Sex				
Female	33 (68.8)	17 (68.0)	18 (60.0)	0.709‡
Male	15 (31.3)	8 (32.0)	12 (40.0)	
Eye				
Right	25 (52.1)	12 (48.0)	15 (50.0)	0.945‡
Left	23 (47.9)	13 (52.0)	15 (50.0)	
SE (diopter)	1.53 ± 1.19	1.12 ± 2.57	0.93 ± 1.26	0.269†
AxL (mm)	22.14 ± 0.68	21.97 ± 0.67	22.24 ± 0.71	0.355†
BCVA (LogMar)				
0	45 (93.8)	24 (96.0)	30 (100.0)	> 0.05§
0.1	3 (6.3)	1 (4.00)	0 (0.0)	

*Not homogenous with PAC.
†Analysis of variance/Tukey test.
‡χ^2 test.
§Kruskal-Wallis test.
AxL indicates axial length; BCVA, best corrected visual acuity at baseline; PAC, primary angle closure; PACG, primary angle closure glaucoma; PACS, primary angle closure suspects; SE, spherical equivalent.

selected eyes (right or left), SE, AxL, and BCVA were presented as mean ± SD or frequency and percentage values. Mean ± SD values of repeated measurements were displayed on a plot as a function of time. The Tukey test along with analysis of variance was used to correct for multiple comparisons among groups as long as the data were distributed normally (determined by the Kolmogorov-Smirnov test). Adjusted *P* values derived from the Tukey test were shown on the plotted graphs when a finding met significance level. The changes in repeated measurements on scheduled visits were displayed as median (Md) [quartile 1 (Q1) to quartile 3(Q3)] whenever they did not conform to normal distribution (ie, ΔACVs, ΔCACDs, etc.). The changes in repeated measurements (intragroup comparison) were analyzed by the paired *t* test. Bonferroni correction along with the Kruskal-Wallis test was applied for multiple comparisons among the groups in the case of non-normally distributed data (ie, ΔACVs, ΔCACDs, etc.). Independent *t* test and Mann-Whitney *U* test were the choice when only 2 sets of variables were compared (ie, baseline ACA vs. amount of changes in ACA). The χ^2 test was referred for multiple comparisons regarding qualitative variables such as sex and selected eye. The relationships between the variables were studied with Pearson or Spearman correlation. Appropriate *P* values of significance were displayed on the graphs or the tables of concern.

RESULTS

Six of the 109 eyes initially included in the study were later excluded: 2 for undergoing cataract surgery, 1 for precluding imaging, and the remainder for noncompliance to scheduled visits. Therefore 103 eyes, with 68 eyes of 34 women and 35 eyes of 19 men, met the eligibility criteria for the study. All subjects were whites. The mean age was 59.07 ± 7.89 years (range, 43 to 81 y). The mean SE was 1.26 ± 1.65 D (range, −5.00 to + 7.00). AxL ranged between 20.52 and 23.76, with a mean of 22.13 ± 0.69 mm.

In subgroup analysis, PACS, PAC, and PACG groups included 48, 25, and 30 eyes, respectively. The mean ages of the patients were 60.94 ± 8.37 years in PACS, 53.76 ± 5.7 years in PAC, and 60.5 ± 8.37 years in PACG. The mean age of PAC patients was significantly lower than the other 2 groups ($P < 0.001$; statistical significance if $P < 0.05$, analysis of variance-Tukey). Comparison of other demographic parameters including female to male ratio, right to left ratio, SE, and AxL and BCVA at baseline did not reveal any statistical significance between the groups (Table 1).

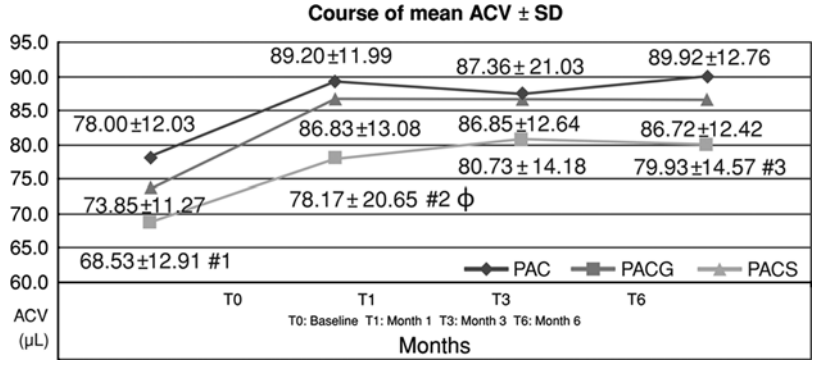

FIGURE 1. #1, #2, #3: not homogenous with time-matched ACV of PAC, respectively ($P = 0.015$, 0.018, and 0.016; statistical significance if $P < 0.05$/analysis of variance-Tukey. Not homogenous with time-matched ACV of PACS). ACV indicates anterior chamber volume; PAC, primary angle closure; PACS, primary angle closure suspect; PACG, primary angle closure glaucoma.

TABLE 2. Changes in Anterior Chamber Volume

ΔACV (μL)	Median (Q1-Q3) PACS (n = 48)	PAC (n = 25)	PACG (n = 30)	*P*
Δ[T1 − T0]	13.50 (9.00-17.00)	12.00 (7.50-15.00)	12.00 (8.75-15.00)	0.340*
P	***< 0.001***	***< 0.001***	***0.007***	
Δ[T3 − T1]	0.00 (−2.00 to 1.25)	1.00 (−1.00 to 3.50)	0.00 (−2.00 to 1.25)	0.251*
P	0.951	0.624	0.386	
Δ[T6 − T3]	0.00 (−1.00 to 2.00)	0.00 (−2.00 to 1.50)	−1.00 (−2.25 to 1.00)	0.172*
P	0.563	0.485	***0.043***	

Paired *t* test for △ACVs, statistical significance if $P < 0.05$.
*Kruskal-Wallis test, statistical significance if $P < 0.017$ (Bonferroni correction) for *P* on the rightmost column.

Figure 1 shows the mean ACV in PACS, PAC, and PACG at baseline (T0) and at 1 (T1), 3 (T3), and 6 months (T6) after LPI. In addition, the mean changes in ACV from previous measurements, with Δ[T1 − T0] showing the change from baseline to 1 month, Δ[T3 − T1] the change from 1 month to 3 months, and Δ[T6 − T3] the change from 3 months to 6 months, are demonstrated (Table 1). The increase in ACV from baseline to 1 month after LPI (Δ[T1 − T0]) was statistically significant ($P < 0.01$, paired *t* test) in all groups. Subsequently, ACV remained stable at 3 and 6 months for PACS and PAC (Δ[T3 − T1] and Δ[T6 − T3] were not statistically significant, $P > 0.05$, paired *t* test). However in PACG, a slight decrease (Δ[T6 − T3]) with borderline significance was found at 6 months ($P = 0.043$, paired *t* test). *P* values marked in Figure 1 and those on the right column in Table 2 indicate statistical homogeneity of the measurements or the changes among the groups with appropriate statistical test (statistical significance if $P < 0.05$ for paired *t* test).

Figure 2 shows the mean CACD in PACS, PAC, and PACG at baseline (T0) and at 1 (T1), 3 (T3), and 6 months (T6) after LPI. The mean changes in CACD, with Δ[T1 − T0] showing the change from baseline to 1 month, Δ[T3 − T1] showing the change from 1 month to 3 months, and Δ[T6 − T3] showing the change from 3 months to 6 months, are shown in Table 3. PACS, PAC, and PACG revealed significant increases in CACD at 1 month (Δ[T1 − T0]) after LPI ($P = 0.047$, $P < 0.001$, $P = 0.003$, respectively/paired *t* test). In PACS and PAC, the increase in CACD was preserved at 3 and 6 months. However, in PACG, the CACD varied significantly from the previous level at 6 months (Δ[T6 − T3]) $P = 0.006$/paired *t* test). *P* values marked in Figure 2 and those on the right column in Table 3 indicate statistical homogeneity of the measurements or the changes among the groups with appropriate statistical test (statistical significance if $P < 0.05$ for paired *t* test).

Figure 3 shows the mean ACA in groups before (T0) and at 1 (T1), 3 (T3), and 6 months (T6) consecutively after LPI. The mean changes in ACA, with Δ[T1 − T0] showing the change from baseline to 1 month, Δ[T3 − T1] showing the change from 1 month to 3 months, Δ[T6 − T3] showing the change from 3 months to 6 months, are shown in Table 4. Similarly the ACA increased significantly at 1 month (Δ[T1 − T0], $P < 0.001$/paired *t* test), and remained stable at 3 and 6 months except for PACG. The mean ACA in PACG decreased when compared with the previous level at 3 months (Δ[T3 − T1]), which was statistically significant ($P = 0.032$, paired *t* test). *P* values marked in Figure 3 and those on the right column in Table 4 indicate statistical homogeneity of the measurements or the changes among the groups with appropriate statistical test (statistical significance if $P < 0.05$ for paired *t* test).

Figure 4 shows the mean IOP in PAC, PACG, and PACS before (T0) and at 1 (T1), 3 (T3), and 6 months (T6) after LPI. Changes from the previous measurements as described above, Δ[T1 − T0], Δ[T3 − T1], and Δ[T6 − T3], are shown in Table 5. The mean IOP decreased significantly in PACS, PAC, and PACG at 1 month ($P < 0.001$, $P = 0.010$, $P < 0.001$, respectively/paired *t* test) and coursed steadily thereafter (Δ[T3 − T1]) and Δ[T6 − T3] were not statistically significant, $P > 0.05$/paired *t* test) except for PACG. At 3 months in PACG, the mean IOP showed a significant change from the previous level at 1 month (Δ[T3 − T1], $P = 0.001$, paired *t* test) in addition to borderline change at 6 months (Δ[T6 − T3], $P = 0.063$,

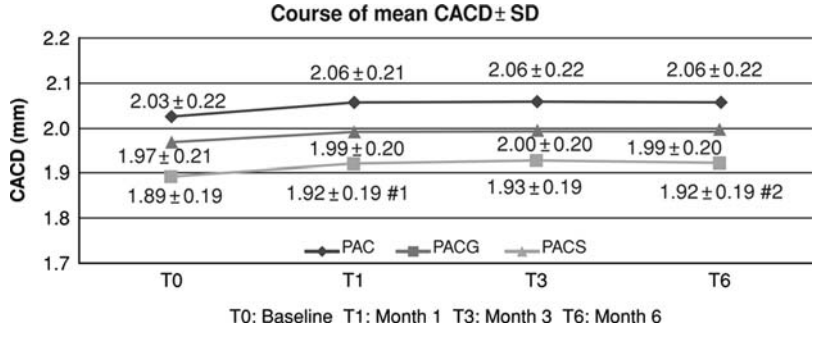

FIGURE 2. #1, #2: not homogenous with time-matched CACD of PAC, respectively ($P = 0.043$ and 0.043; statistical significance if $P < 0.05$/analysis of variance-Tukey). CACD indicates central anterior chamber depth; PAC, primary angle closure; PACS, primary angle closure suspect; PACG, primary angle closure glaucoma.

TABLE 3. Changes in Central Anterior Chamber Depth

	Median (Q1-Q3)			
ΔCACD (mm)	**PACS (n = 48)**	**PAC (n = 25)**	**PACG (n = 30)**	***P***
Δ[T1 − T0]	0.02 (0.01-0.04)	0.02 (0.01-0.05)	0.03 (0.01-0.04)	0.747*
P	***0.047***	***< 0.001***	***0.003***	
Δ[T3 − T1]	0.00 (−0.02 to 0.02)	0.00 (−0.02 to 0.02)	0.01 (−0.01 to 0.02)	0.648*
P	0.303	0.780	0.080	
Δ[T6 − T3]	0.00 (−0.01 to 0.02)	0.00 (−0.01 to 0.01)	−0.01 (−0.02 to 0.00)†	***0.015****
P	0.132	0.485	***0.006***	

Paired *t* test for ΔCACDs, statistical significance if $P < 0.05$.
*Kruskal-Wallis test, statistical significance if $P < 0.017$ (Bonferroni correction) for *P* on the rightmost column.
†Not homogenous with PAC and PACS.

paired *t* test). *P* values on the rightmost column on Table 5 indicate statistical homogeneity of the measurements or the changes among the groups with appropriate statistical test (statistical significance if $P < 0.05$ for paired *t* test).

Table 6 shows the Md (Q1 to Q3) of No.AGM prescribed to keep the IOP between 15 and 21 mm Hg. By definition, PACS included eyes without antiglaucomatous medication at baseline (T0), which required no medication thereafter. In PAC and PACG, the Md (Q1 to Q3) of No.AGM decreased significantly at 1 month (△[T1 − T0], $P = 0.020$, paired *t* test, statistical significance if $P < 0.05$) and remained stable afterward.

In analysis of 103 eyes together, there was a weak correlation between the AxL and the change in CACD 1 month (△[T1 − T0]) after LPI ($r = 0.266$, $P = 0.007$; statistical significance if $P < 0.05$, Pearson/Spearman) (Fig. 5). Moreover, when all eyes were classified according to the Scheimpflug topographer–based ACA scores at baseline (T0), eyes with ACA ≤ 25 degrees showed a 6.6 ± 2.8-degree (mean ± SD) increase in ACA at 1 month, whereas eyes with ACA > 25 degrees had a 4.9 ± 2.4-degree (mean ± SD) increase ($P = 0.002$, independent *t* test/Mann-Whitney *U* test, statistical significance if $P < 0.05$). Corresponding Md (Q1 to Q3) values of increase for eyes with ACA ≤ 25 degrees and eyes with ACA > 25 degrees were 7.00 (4.00 to 8.00) and 5.00 (3.00 to 6.25) degrees, respectively.

DISCUSSION

The introduction of Scheimpflug imaging devices into clinical practice led to several studies that focused on changes in the AC parameters of eyes with narrow angles that underwent LPI. The changes in ACA, CACD, peripheral AC depth, and central corneal thickness have been documented previously.[5,20–23] The device used in these studies is almost always the Pentacam (Oculus; Optikgeräte GmbH, Wetzlar, Germany), which has been shown to be accurate and reproducible in quantifying AC parameters.[27] In our study we used the Sirius device, which is basically similar albeit slightly different, and as far as we know it has not been used previously in the evaluation of AC parameters after LPI.

Studies on reproducibility and consistency of the measurements taken by the Sirius have been reported previously. A 0.49% coefficient of variation with intraclass correlation coefficient > 0.99 for CACD, and 1.62% coefficient of variation with intraclass correlation coefficient > 0.99 for ACV have been documented in healthy eyes using the Sirius. These figures are comparable with those of Pentacam reported by Savini et al.[28] Bedei and colleagues reported substantially repeatable measurements of ACV, ACA, and CACD for both Pentacam and Sirius, which were not statistically equivalent or interchangeable. They attributed these discrepancies to the different reference systems or algorithms that the devices used for calibration

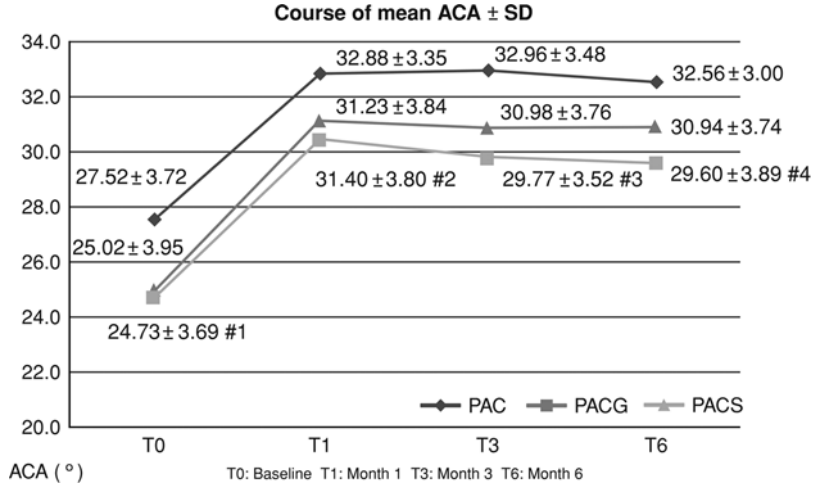

FIGURE 3. #1, #2, #3, #4: not homogenous with time-matched ACA of PAC, respectively ($P = 0.014$, 0.048, 0.006, and 0.013; statistical significance if $P < 0.05$/analysis of variance-Tukey). ACA indicates anterior chamber angle; PAC, primary angle closure; PACS, primary angle closure suspect; PACG, primary angle closure glaucoma.

TABLE 4. Changes in Anterior Chamber Angle

	Median (Q1-Q3)			
△ACA (deg.)	PACS (n = 48)	PAC (n = 25)	PACG (n = 30)	*P*
Δ[T1 − T0]	6.00 (4.00-8.00)	5.00 (3.50-6.50)	5.50 (4.00-7.00)	0.475*
P	***< 0.001***	***< 0.001***	***< 0.001***	
Δ[T3 − T1]	0.00 (−1 00 to 0.00)	0.00 (−1.00 to 1.00)	0.00 (−1.00 to 0.00)	0.501*
P	0.234	0.784	***0.032***	
Δ[T6 − T3]	0.00 (−1.00 to 1.00)	0.00 (−1.00 to 0.00)	0.00 (−1.00 to 1.00)	0.493*
P	1.000	0.144	0.455	

Paired *t* test for △ACAs, statistical significance if $P < 0.05$.
*Kruskal-Wallis test, statistical significance if $P < 0.017$ (Bonferroni correction) for *P* on the rightmost column.

and calculations.[31] Thus, when comparing the outcomes of the similar studies conducted with different commercial brands or even with different imaging modalities, it may be better to analyze proportional changes in measurements rather than absolute changes.

In our study, statistically significant increases in ACV, ACA, and CACD were observed in PACS, PAC, and PACG after LPI. Our findings agree with previous studies regarding the increase in ACV and ACA.[5,20–23] In contrast, it is controversial whether LPI deepens the CACD or not. Caballero et al reported a significant increase in CACD ($P = 0.004$), whereas Antoniazzi et al stated that this increase had a borderline significance by Pentacam ($P = 0.0468$).[20,22] Moreover, Gazzard et al[32] have shown significant increases in CACD with the A-scan; however they could not show this with optical pachymetry on the 55 fellow eyes of acute PACG patients. Lei et al[19] used AS-OCT and found a mean increase of 31 μm in CACD in eyes with PAC, similar to our results. Rabsilber et al[27] showed a correlation between ACV and CACD, whereas Talajic et al[21] reported a significant increase in CACD after pilocarpine administration. In addition to a significant increase in CACD, we found a correlation between AxL and CACD ($r = 0.266$, $P = 0.007$). We feel that the changes in CACD induced by LPI are so subtle that factors such as artifacts, accommodation or miosis, antiglaucomatous medications in use, device resolutions, small sampling size, and heterogeneity in biometric values of the eyes may easily influence whether the results reach statistical significance.

Shortly after LPI, we showed a significant decrease in IOP along with a reduction in No.AGM in eyes with PAC and PACG ($P = 0.010$, $P < 0.001$). In PACS, in which no antiglaucomatous medication was being used at baseline because of lack of consensus on benefits of treatment, the mean decrease in IOP was −2.00 (−3.00 to 0.00) mm Hg (△[T1 − T0], Md (Q1 to Q3), $P < 0.001$). These results were similar to those of previous studies.[20,21] However, the long-term efficacy of LPI is a major concern in eyes with narrow angles. Despite the fact that LPI widens the ACA in all ethnic groups with narrow angles, the efficacy of the procedure depends on the stage of the disease and the underlying mechanism causing angle closure.[32–34] Apart from pupil block, a higher presenting IOP, larger cup:disc ratio, greater extent of PAS, and African and Asian descent are proposed as poor predictors of IOP control after LPI.[35] Undoubtedly, the eyes with the above-mentioned poor predictors mostly fall into the group PACG according to the new classification system. Thus, eyes that have PACG that denote advanced stage are likely to require additional treatment for IOP control before PACS and PAC. Nolan et al[13] reported that new PAS is likely to develop despite patent iridotomies in eyes with advanced stage; therefore we anticipated reversal of the effects in AC parameters induced by LPI. As expected, our study showed significant changes in ACA and IOP at 3 months, and in ACV and CACD and at 6 months in PACG. In our opinion, these findings may relate to the progressive nature of PAS formation. Alternatively, given the fact that these eyes are under

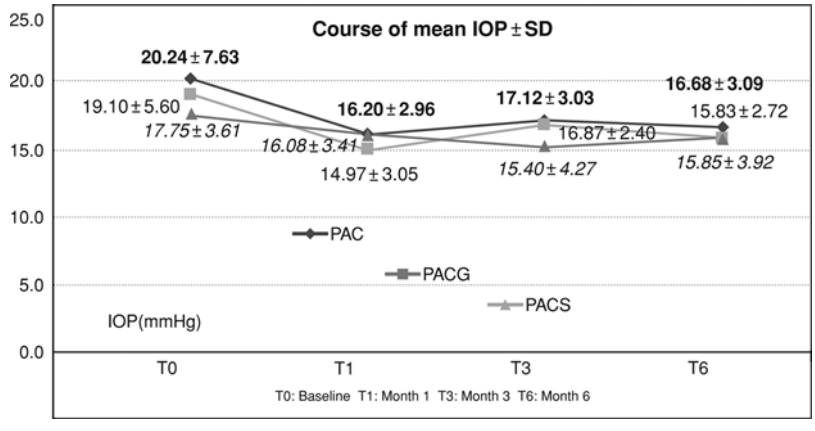

FIGURE 4. Bold: PAC values; italic: PACS values. Time-matched comparisons of measurements between groups showed statistical homogeneity ($P > 0.05$, analysis of variance-Tukey, statistical significance if $P < 0.05$). IOP indicates intraocular pressure; PAC, primary angle closure; PACS, primary angle closure suspect; PACG, primary angle closure glaucoma.

TABLE 5. Changes in Intraocular Pressure

	Median (Q1-Q3)			
ΔIOP (mm Hg)	**PACS (n = 48)**	**PAC (n = 25)**	**PACG (n = 30)**	***P***
Δ[T1 − T0]	−2.00 (−3.00 to 0.00)	−1.00 (−5.50 to 0.00)	−3.00 (−7.00 to −1.00)	0.128*
P	***< 0.001***	***0.010***	***< 0.001***	
Δ[T3 − T1]	0.00 (−1.75 to 0.75)	1.00 (−0.50 to 2.00)	1.50 (0.00-3.25)†	***0.001****
P	0.122	0.085	***0.001***	
Δ[T6 − T3]	0.00 (−1.00 to 1.00)	0.00 (−2.00 to 1.50)	−0.50 (−3.00 to 2.00)	0.266*
P	0.310	0.406	0.063‡	

Paired *t* Test for ΔIOPs, statistical significance if $P < 0.05$.
*Kruskal-Wallis test, statistical significance if $P < 0.017$ (after Bonferroni correction) for *P* on the rightmost column.
†Not homogenous with PACS.
‡Borderline.

antiglaucomatous medication with the most No.AGM, variations in IOP along with sensitivity to medications may be the reason.

Our study did not show significant changes in eyes with PACS and PAC. The effects in AC parameters induced by LPI coursed steadily along 6 months of follow-up, which might be too short in displaying reversal of the effects. Moreover, with respect to efficacy of LPI, our study showed that after LPI, ACA widened by 7.00 (4.00 to 8.00) degrees in eyes with ACA ≤ 25 degrees versus 5.00 (3.00 to 6.25) degrees in eyes with ACA > 25 degrees [Md (Q1 to Q3), $P = 0.002$]. These findings may be useful in the clinical setting, because a recent paper showed that the eyes with ACA ≤ 26 degrees were at high risk of developing AAC and they required LPI.[36]

The Sirius has some disadvantages as stated before for Pentacam. Rather than direct visualization as in Pentacam, the Sirius-Scheimpflug provides angle calculation based on interpolation or extrapolation.[22] According to device user manual, only 7 of 25 scans intercepting the meridian 180 ± 20 degrees of nasal and temporal iridocorneal periphery are processed for averaging in ACA calculation, whereas the superior and inferior quadrants are not taken into consideration. Approximately 270 of 360 degrees of the entire ACA is ignored. Therefore, possible changes in ACA on superior and inferior iridocorneal periphery cannot be assessed as a limitation. In this case, ACA calculated by the Sirius can only be complementary to gonioscopy. CACD and ACV may be more reliable parameters in the assessments as stated before.[36] In our view, ACV may be the best, because volumetric calculation technically requires the highest number of raw data (variables) retrieved by the device.

Inclusion of both eyes, relatively small sample size when considering separate groups, nonrandomized design, and lack of healthy controls are the major limitations of our study. Furthermore, apart from No.AGM and IOP, the AC parameters of interest repeatedly showed statistical heterogeneity in the measurements among the groups. This seems mostly related to PAC, in which the measurements are relatively dissimilar. It is noteworthy that the mean age in PAC is significantly lower than the 2 others ($P < 0.001$). Kim et al[37] showed that ACD decreases 0.12 mm per decade, presumably because of the continuously increasing lens thickness. Thus, heterogeneity in age between the groups in our study may result in heterogeneity in the AC parameters measured.

In conclusion, our study showed significant changes in AC parameters in eyes with PACG at 6 months after LPI. A longer follow-up is likely to show more prominent changes in PACG and even in PAC, and maybe in PACS. However, this study highlights that Scheimpflug imaging along with the new simpler classification system may help document the course of narrow angled eyes with or without treatment.

TABLE 6. Changes in Number of Antiglaucomatous Medication in Use

	Median (Q1-Q3)		
No.AGM	**PACS (n = 48)**	**PAC (n = 25)**	**PACG (n = 30)**
T0	0 (0-0)	2 (1-3)	2 (1-3)
T1	0 (0-0)	2 (1-2)	2 (1-2)
T3	0 (0-0)	2 (1-2)	2 (1-2)
T6	0 (0-0)	2 (1-2)	2 (1-2)

No.AGM indicates number of antiglaucomatous medication in use; T0, baseline; T1, month 1; T3, month 3; T6, month 6.

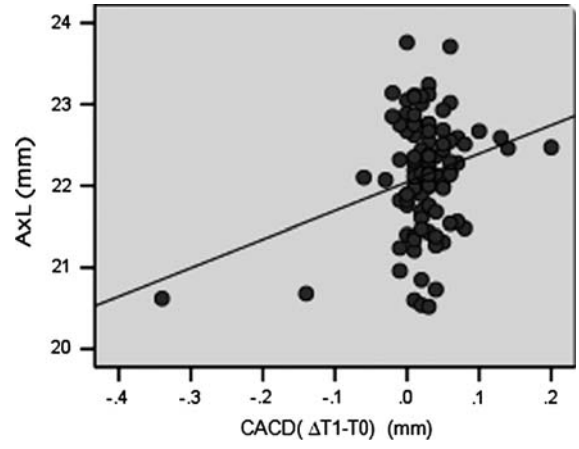

FIGURE 5. Scatterplot of correlation between AxL and change in CACD at 1 month ($r = 0.266$, $P = 0.007$; Pearson/Spearman; statistically significance if $P < 0.05$). AxL indicates axial length; CACD, central anterior chamber depth.

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