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Original Article

Long-term outcomes after acute primary angle closure of Caucasian chronic angle closure glaucoma patients

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Abstract

Importance: There is a lack of information about long-term results of chronic angle closure glaucoma following an acute primary angle closure attack in Caucasian patients.

Purpose: The aim of the study was to report morphological and functional long-term data of chronic angle closure eyes following a monolateral primary angle closure attack and to provide a comparison with their fellow eyes.

Design: Observational retrospective case series.

Participants: Fifty-seven consecutive patients (114 eyes) underwent long-term follow-up analysis.

Methods: Patients underwent ophthalmic assessment more than 5 years since the angle closure attack.

Main Outcome Measures: Intraocular pressure, best-corrected visual acuity, angle assessment, vertical C/D ratio and standard automated perimetry were the main outcome measures. Comparisons were made between angle closure attack eyes and fellow eyes and between phakic and pseudophakic eyes.

Results: Mean follow-up time was 5.86 ± 1.19 years. A significant greater damage in the angle closure eyes compared with fellow eyes in both structural (mean C/D 0.61 ± 0.16; $P < 0.001$) and functional (mean deviation: −7.98 ± 6.46 vs. −4.83 ± 4.95 dB; $P < 0.001$) terms was present. Mean IOP was 13.44 ± 2.78 and 13.89 ± 2.60 mmHg in angle closure and fellow eyes ($P = 0.11$). Thirty of 57 (53%) fellow eyes developed chronic angle closure (mean deviation: −7.74 ± 5.21 dB) even if prophylactic iridotomy was promptly performed.

Conclusion and Relevance: Our study prompts ophthalmologists to closely follow patients after an APAC attack to prevent potential glaucoma damage in both APAC and fellow eye.

Key words: APAC, glaucoma, long-term follow-up, outcome, PACG.

Introduction

Primary angle closure glaucoma (PACG) is one of the leading causes of blindness worldwide, with a pooled prevalence ranging from 0.46% in India to 1.19% in Japan.[1] Although PACG is more diffuse and studied in the Asian population, its incidence is substantial even among Caucasians accounting for 0.4% of people older than 40 years.[2] Moreover, PACG incidence in Caucasians seems to be underestimated as underlined by Ng *et al.* who found an incidence of 14.8 per 100 000 in a Scottish Caucasian cohort.[3] As PACG prevalence is correlated with age, it will be more diffuse in the western countries as the population ages. It has been estimated that the worldwide population with PACG will rise by a third of the total glaucomatous in 2020,[4] with 34 million people affected in 2040.[5] Furthermore, the prevalence of glaucoma-related bi-

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lateral versus monolateral blindness is significantly higher in PACG than in primary open angle glaucoma.[6]

Acute primary angle closure (APAC) is a highly symptomatic disease characterized by a sudden intraocular pressure (IOP) elevation often associated with severe ocular pain and systemic symptoms.[7] Persistent high IOP values following an APAC can lead to irreversible glaucomatous optic neuropathy and subsequent vision loss. It has been estimated that up to 50% of eyes after an APAC episode develop PACG.[8]

Being mainly a bilateral condition, attention is commonly given to preserve the fellow eye from potential risks, especially after an APAC episode. It has been demonstrated that fellow eyes have a significant risk of developing APAC: 2.5% of fellow eyes in an Asian population were diagnosed with PACG at the time of the acute attack, and an additional 6.5% developed glaucoma in the next 6 years.[9]

Although extensive research has been conducted in the last few years on the long-term morbidity and progression to PACG in Asian individuals following APAC, limited data are available in the Caucasian population. The aim of this cross-sectional observational case series was to provide a morphological and functional assessment of PACG in Caucasian patients more than 5 years after the occurrence of a monolateral APAC episode.

Methods

The study was approved by the local ethics committee, and the authors declare no financial or proprietary interests in the subject of this research. All study procedures adhered to the Declaration of Helsinki (as revised in Brazil 2013) for research involving humans, and written informed consent was obtained from the study subjects.

The study was conducted at the Eye Hospital of Turin, the biggest in Piedmont (North-Western Italy). In its emergency department, more than 30 000 visits occur every year, and it represents the reference for about one-third of the 4.5 million people living in Piedmont. Subjects in this study had been previously visited at the Emergency Department of the Eye Hospital and were treated according to the Hospital Protocol, which advocates medical management to promptly reduce the IOP followed by bilateral YAG laser peripheral iridotomies (LPIs). Lens extraction/glaucoma surgery according to the clinical appearance of the angle is performed if LPI and medical treatment are not sufficient to obtain an adequate IOP control.

Consecutive Caucasian patients with chronic angle closure glaucoma (CACG), who had suffered a previous monolateral episode of APAC at least 5 years before, were included. Patients were aged between 50 and 80 years and were recruited either from our Glaucoma Service or called back based on the list of patients with APAC who had previously attended the Emergency Department. Follow-up visits were performed between September 2015 and September 2016. The patients who had not been recruited from our Glaucoma Service first underwent a telephonic interview to ascertain if they were suffering from glaucoma after the APAC episode and then called back for a follow-up visit.

A previous APAC episode was defined using the following criteria:

1. presenting IOP of more than 28 mmHg on Goldmann applanation tonometry;
2. presence of at least two of the following symptoms: ocular or periocular pain, nausea, vomiting and an antecedent history of intermittent blurred vision; and
3. presence of at least three of the following signs: conjunctival injection, corneal oedema, mid-dilated non-reactive pupil and shallow anterior chamber.

Chronic angle closure glaucoma was diagnosed based on the presence of a closed angle on indentation gonioscopy (grade 0–1 Shaffer grade, ≥180 deg), abnormal visual field according to the Hodapp–Parrish–Anderson[10] criteria and glaucomatous optic neuropathy. All visual fields underwent a second classification according to Glaucoma Staging System 2.[11]

Exclusion criteria were bilateral APAC attack, secondary angle closure, previous intraocular surgery, reported history of pre-existing glaucoma at the time of APAC and incomplete clinical data.

Demographic characteristics, and medical and ophthalmic history were gathered by a trained interviewer using a pre-defined questionnaire. Ophthalmology records regarding the APAC attack and management were reviewed, and medical charts were further analysed. Type and time of interventions after APAC were recorded.

A complete ophthalmic examination was prospectively performed for each patient and included logMAR best-corrected visual acuity, slit-lamp biomicroscopy, gonioscopy, Goldmann applanation tonometry and fundus assessment. Gonioscopy was performed to obtain the mean gonioscopic angle width (calculated by adding the Shaffer grade in each of four quadrants and dividing by four) and to assess for the presence of peripheral anterior synechiae. Fundus assessment was performed through a well-dilated pupil and a 78 D indirect lens. Optic disc analysis included the evaluation of the vertical cup:disc ratio and the neuroretinal rim. All

examinations were performed by an expert ophthalmologist (T. R.).

Patients' assessment also included ultrasound pachymetry to measure the central corneal thickness, corneal endothelial cell count with specular microscopy (CellChek Specular Microscope, Konan Medical, Nishinomiya, Hyogo, Japan), axial length (AL) and anterior chamber depth (ACD) measured with IOL Master 500 Optical Biometry (Carl Zeiss Meditec, Jena, Germany) and automated visual field test with the Humphrey Field Analyzer (Carl Zeiss Meditec).

Visual field analysis

Standard automated perimetry was performed with the Swedish Interactive Threshold Algorithm Standard strategy, programme 24-2 of the Humphrey Field Analyzer (Carl Zeiss Meditec). The criteria used for reliability were fixation losses ≤20%, false positives, and false negatives ≤33%.

Corneal endothelial cell count with specular microscopy

Specular microscopy images were obtained with the CellChek Specular Microscope (Konan Medical). Image analysis was performed using the semiautomated centre technique. Only high-quality images were included, and a manual count of the cells was performed. Over 100 contiguous endothelial cells were marked for the analysis.

Biometric factors with optical biometry

Biometry based on partial coherence interferometry was performed using the IOL Master 500 (Carl Zeiss Meditec). The AL was measured from the tear film to the retinal pigment epithelium using a modified Michelson interferometer that creates a pair of coaxial 780-nm infrared light beams with a coherence length of about 130 nm. The ACD was defined as the distance from the anterior corneal surface to the anterior lens surface using lateral slit illumination at approximately 30° to the optical axis. Five separate measurements were averaged for AL and ACD measurements.

Ultrasound pachymetry

Central corneal thickness was measured using ultrasound pachymetry (SW 1000 A-Scan Pachymeter, Tianjin Suowei Electronic Technology, Tianjin, China) after the instillation of one drop of topical anaesthesia (0.4% oxybuprocaine hydrochloride); the examiner manually placed the probe pachymeter as perpendicularly as possible to the centre of the cornea while the patient was asked to gaze a distant fixation target straight ahead. The mean of five measurements was then calculated.

Statistical analysis

Continuous variables were checked to meet the normality conditions of Shapiro–Wilk test. Nonparametric tests were used: the Wilcoxon signed rank test for comparisons of two correlated samples involving matched pairs and the Mann–Whitney test for comparisons of two independent distributions.

The binary variables were arranged in cross-correlation tables and studied using the chi-squared test with the Yates correction.

The relationship between presenting features (presenting IOP and duration of acute attack) and structural and functional findings was examined using linear regression analysis and the Spearman's rank correlation coefficient.

Statistical analysis was performed using a statistical software (SPSS Statistics, version 19.0, Chicago, IL, USA). Statistical significance was set at $P < 0.05$.

Definition

In the paper, the eye that previously suffered APAC will be defined as the affected eye, and the contralateral eye as the fellow eye, irrespective of the presence of PACG.

Results

Baseline data

Seventy consecutive patients with previous APAC were enrolled. Eight patients were excluded owing to unavailability of data from the APAC episode, whereas five patients called from our Emergency Department lists were excluded owing to the inclusion criteria for PACG not being met (three absence of significant disk damage and two inconsistent visual field defects). Fifty-seven CACG patients were analysed in terms of structural and functional damage at a mean of 5.86 ± 1.19 years (range 5–9 years) after APAC.

Demographic data are summarized in Table 1.

The most common symptom of presentation was ocular pain (76% of subjects) followed by blurred vision (71%), ocular hyperemia (21%), nausea (20%) and vomiting (14%).

All the patients were treated medically to resolve the attack. Whenever possible, LPI was performed within 24 h from presentation in the Emergency Department (22 eyes). In 32 eyes, it was deferred but performed within 48 h. In three eyes (5%), the IOP was insufficiently controlled by medical therapy, and LPI was impossible to perform owing

Table 1. Patients characteristics and management at the time of APAC

	Study subjects (n = 57)	
	Mean ± SD	Range
Age (years)	68.84 ± 9.60	50–80
Gender, F/M, *n* (%)	42/15 (74%/26%)	
Presenting IOP (mmHg)	53.19 ± 9.15	30–70
Duration of acute attack (hours)	22.07 ± 18.42	2–72
Medications used for acute attack (*n*/patient)	3.77 ± 0.85	2–6
Topical medications (*n*/patient)	2.70 ± 0.53	2–4
Systemic medications (*n*/patient)	1.07 ± 0.75	0–2
Phakic patients at the time of APAC, *n* (%)	57 (100%)	
LPI in the APAC eye, *n* (%)	54 (95%)	
LPI within 24 h from the presentation in the emergency department	22 (39%)	
LPI within 48 h from the presentation in the emergency department	32 (56%)	
Phaco-emulsification to resolve APAC	3 (5%)	
LPI in fellow eye, *n* (%)	57 (100%)	

SD, standard deviation; F, female; M, male; IOP, intraocular pressure; APAC, acute primary angle closure; LPI, laser peripheral iridotomy.

to corneal haziness, and phaco-emulsification with epithelial removal was performed. LPI was performed in the fellow eye of all patients as a prophylactic intervention. No serious complications were reported to have occurred after laser iridotomy in any eye.

Follow-up data

Data from affected and fellow eyes at last follow-up are reported in Table 2.

Mean IOP at the time of follow-up was respectively 13.44 ± 2.78 and 13.89 ± 2.60 mmHg in the affected and fellow eyes. All the eyes presented an IOP < 21 mmHg, whereas 86% and 72% of affected eyes and 89% and 77% of fellow eyes had an IOP < 18 and <16 mmHg, respectively. Data about IOP and medication status are provided in Table 3. All iridotomies were patent at the follow-up and, no APAC recurrencies were reported in either the affected or fellow eyes.

The mean time after APAC to phaco-emulsification was 35.24 ± 12.07 months in the affected eyes and 45.65 ± 11.19 months in the fellow eyes. This difference was statistically significant (P = 0.012). None of the eyes required additional glaucoma surgery.

Tables 4, 5 and 6 represented respectively the distribution of vertical cup:disc ratio and of visual field damage in affected and fellow eyes.

Table 2. Characteristics of the enrolled eyes at last examination

	APAC eye	Fellow eye	*P*-value
Vertical C/D ratio	0.68 ± 0.15 (0.4–0.9)	0.61 ± 0.16 (0.3–0.9)	<0.0001†
MD (dB)	−7.98 ± 6.46 (−0.9–26.8)	−4.83 ± 4.95 (1.24–23.6)	<0.0001†
PSD (dB)	5.17 ± 3.38 (1.15–14.33)	3.43 ± 2.24 (0.23–11.9)	<0.0001†
BCVA, logMAR	0.37 ± 0.49 (0–1.7)	0.17 ± 0.20 (0–1)	<0.0001†
ECD (cells/mm^2)	1923.93 ± 497.08 (673–3300)	2066.02 ± 429.70 (1028–2994)	0.002†
CCT (μm)	551.98 ± 39.09 (436–653)	551.98 ± 40.75 (450–669)	0.79†
ACD (mm)	2.58 ± 0.70 (1.28–4.57)	2.58 ± 0.70 (1.42–4.58)	0.65†
AL (mm)	21.68 ± 0.99 (18.88–24.21)	21.86 ± 1.17 (18.65–25.76)	0.013†
Gonioscopy, grade	1.44 ± 0.76 (0–3)	1.60 ± 0.78 (0–4)	0.029†
Gonioscopy PAS, eyes (%)	23/57 (40.35%)	10/57 (17.54%)	0.013‡
Lens status			
Phakic	32 (56%)	39 (68%)	
Pseudophakic	25 (44%)	18 (32%)	0.24‡

All parameters are expressed as mean ± standard deviation (range). BCVA, best-corrected visual acuity; ACD, anterior chamber depth; AL, axial length; APAC, acute primary angle closure; MD, mean deviation; PAS, peripheral anterior synechiae; PSD, pattern standard deviation; CCT, central corneal thickness; ECD, endothelial cell density.

†Wilcoxon test.

‡Chi-square test.

Table 3. Long-term IOP and medication status in affected and fellow eyes

	APAC eye	Fellow eye	*P*-value
IOP (mmHg)	13.44 ± 2.78 (9–19)	13.89 ± 2.60 (10–20)	0.38†
Medication-free eyes (%)	19/57 (33%)	31/57 (54%)	0.04‡
Medications (all eyes), *n*/eye	1.19 ± 1.19	0.73 ± 1.04	<0.001§
Medications in treated eyes, *n*/eye	1.79 ± 1.02 (1–5)	1.62 ± 0.98 (1–5)	0.45§

APAC, acute primary angle closure; IOP, intraocular pressure.
†Mann–Whitney test.
‡Chi-squared test.
§Wilcoxon test.

Table 4. Distribution (%) of vertical cup:disc ratio in affected and fellow eyes

	Vertical cup:disc ratio				*P*†
	<0.5	0.5–0.7	0.7–0.9	>0.9	
APAC eyes	4 (7%)	25 (44%)	25 (44%)	3 (5%)	0.035
Fellow eyes	14 (25%)	26 (46%)	16 (28%)	1 (2%)	

APAC, acute primary angle closure.
†Chi-squared test.

Table 5. Distribution (%) of visual field damage, according to the Hodapp–Parrish–Anderson criteria in affected and fellow eyes

	Visual field damage (Hodapp–Parrish–Anderson)				*P*†
	No damage	<6 dB	6–12 dB	>12 dB	
APAC eyes	3 (5%)	28 (49%)	13 (23%)	13 (23%)	0.02
Fellow eyes	10 (18%)	35 (61%)	7 (12%)	5 (9%)	

†Chi-squared test.

Visual acuity (VA) at follow-up was 0.37 ± 0.48 logMAR in the affected eyes and 0.17 ± 0.20 logMAR in the fellow eyes. The distribution of VA in the affected eyes is presented in Figure 1.

The causes of VA ≥ 1 logMAR were cataract in one patient, macular oedema in two eyes and severe glaucoma damage in the remaining six eyes.

No statistically significant correlation was found between presenting IOP or duration of APAC attack and the structural and functional measurements at last follow-up visit.

Fellow eye data

Of the 57 fellow eyes, 30 (53%) had glaucomatous neuropathy at the time of the last follow-up; the mean deviation (MD) was −7.74 ± 5.21 dB (range −3.14 to −23.6). Table 7 shows the comparison between fellow CACG eyes and fellow healthy eyes. Nine out of 27 non-CACG fellow eyes were on therapy owing to ocular hypertension.

In order to evaluate the influence of phaco-emulsification on the outcomes, we compared the affected eyes that underwent phaco-emulsification with the affected phakic eyes. Patients who underwent phaco-emulsification were older than the phakic patients (71.8 ± 9.27 and 66.53 ± 9.34, $P = 0.026$), had lower mean IOP values and took less anti-glaucomatous medications (IOP = 12.72 ± 2.32 with 0.68 ± 1.03 medications in the pseudophakic eyes; IOP = 14.00 ± 3.01 with 1.59 ± 1.16 in the phakic eyes, $P = 0.13$ for IOP, $P = 0.001$ for medication). Statistically significant differences emerged also in terms of ACD and angle grading (ACD = 2.82 ± 0.85, angle grading = 1.92 ± 0.77 in pseudophakic eyes and ACD = 2.39 ± 0.50, $P = 0.034$ angle grading = 1.06 ± 0.49, $P < 0.001$ in phakic eyes).

Discussion

Primary closed angle glaucoma is regarded as an uncommon disease in Caucasian, and this accounts for the lack of long-term data on the morbidity and progression of this disease.

Our study focused on the long-term morphological and functional outcomes in Caucasian who

Table 6. Distribution (%) of visual field damage, according to the GSS 2 in affected and fellow eyes

	GSS 2							*P*†
	Stage 0	Stage borderline	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	
APAC eyes	4 (7%)	9 (16%)	9 (16%)	11 (19%)	11 (19%)	7 (12%)	6 (11%)	0.04
Fellow eyes	12 (21%)	6 (11%)	18 (32%)	10 (17%)	7 (12%)	2 (4%)	2 (4%)	

APAC, acute primary angle closure; GSS 2, Glaucoma Staging System 2.
†Chi-squared test.

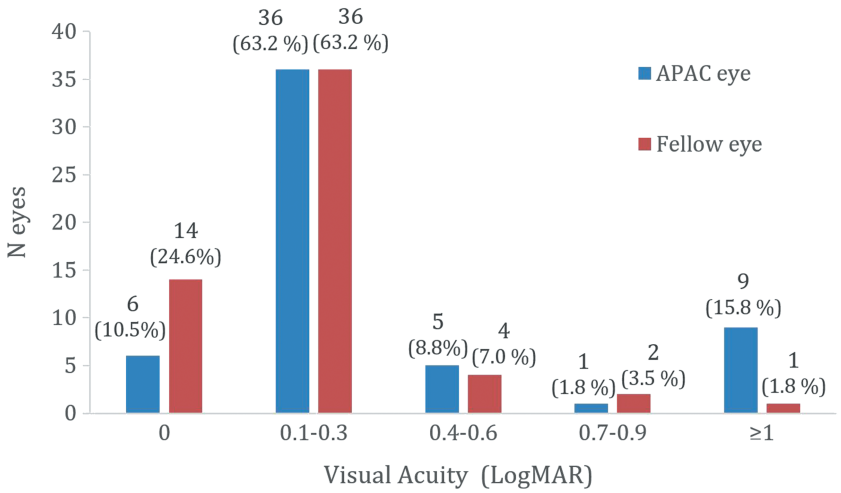

Figure 1. Best-corrected visual acuity (number of eyes and percentage) in eyes with previous acute primary angle closure and fellow eye. Chi-squared test, $P = 0.04$.

developed a CACG in one eye following an APAC attack. Clinically significant structural and functional damage of both the affected and fellow eyes was observed. In our relatively large cohort of patients, almost half of the eyes that developed the acute attack, when examined more than 5 years later, presented in the affected eye a moderate to severe visual field defect with both the Hodapp (46%) and Brusini (42%) classifications.

The available data on the progression to PACG in the affected eye of purely Caucasians are limited to a single report by Andreatta[12] on seven patients who developed PACG 27 ± 14 months after an acute attack.

Our data are similar to those of that study in several respects as the IOP in the affected eye at the last follow-up visit (13.44 ± 2.78 vs. 14 ± 2 mmHg), the C/D (0.68 ± 0.15 vs. 0.69 ± 0.11), the MD (−7.98 ± 6.46 vs. −8.2 ± 2.8 dB) and in some respect as the post-APAC management. In the study of Andreatta, PACG patients underwent repeated LPI and cataract extraction more frequently, and in one patient, trabeculectomy was necessary, whereas only 57% of the PACG patients were on topical medications.

The main difference is that Andreatta was able to exclude the presence of a PACG before the occurrence of the APAC attack because he had access to the optic nerve and visual field data immediately after the event.

The IOP at presentation and at the last visit in our sample is in line with previous studies in Asian or mixed populations. The mean presenting IOP in the affected eye in our series (53.2 ± 9.2 mm Hg) was similar to that found in Eastern Asians (Lee *et al.*[13,14]: 50.2 ± 12.6 and 49.2 ± 14 mmHg) and in a mixed New Zealand population (Chew *et al.*[15]: 53.3 ± 12.4 mmHg).

Considering the long-term follow-up data on the affected eyes, 43 Asian PACG patients with a previous APAC attack observed for an average of 6.3 years presented a more severe visual field defect (MD: −11 ± 10.8 dB and pattern standard deviation: 4.1 ± 2.9 dB).[8] Tham[16] reported an MD of −14.7 ± 9.4 dB and a cup:disc ratio of 0.7 ± 0.2 in 13 eyes after an unspecified time period after the attack, although this group of patients had a higher IOP and medication use (17.8 ± 7.1 mmHg and 2.2 ± 0.9 drug/patient) compared with ours (13.61 ± 2.84 mmHg and 1.24 ± 1.24 drug/patient). Chen *et al.*,[17] who reported similar IOP at the last follow-up visit, found a more advanced optic nerve damage (cup:disc ratio: 0.76 ± 0.14 and MD: −8.5 ± 6.65 dB) in 40 eyes, albeit with a shorter and more variable follow-up (3 months to 8 years).

The management of our patients developing CACG is somehow different from what is reported by previous authors.[8,13,18] The percentage of our patients undergoing phaco-emulsification during the follow-up was 39%, whereas no patients needed additional glaucoma surgery. The percentage of glaucoma surgery reported by other authors after APAC is higher in both Caucasian and Asian patients: 16% and 63% filtration surgeries were respectively performed in the series by Andreatta[12] and Alsagoff.[18] Nevertheless, published studies in White populations found LPI to be effective with a low incidence of eventual trabeculectomy ranging from 0% to 8%.[19,20]

Our data seem to confirm that the prognosis of APAC is generally worse in Asians than in Caucasians. Quek *et al.*[21] found that in Chinese with PACG, higher mean overall IOP and history of previous APAC were positively correlated with poorer visual field and visual acuity outcomes over a 10 years' period. Andreatta,[12] within his APAC Caucasians series, reported that a longer time to resolve the APAC attack and a higher duration of symptoms correlated with a poorer prognosis. We could not confirm these findings, and this may be

Table 7. Clinical outcomes of fellow eyes divided on the basis of glaucomatous damage at the last follow-up

	CACG eye (n = 30)	Non-CACG eye (n = 27)	P-value
Age (years)	72.90 ± 7.48 (50–80)	64.33 ± 9.79 (50–80)	0.001†
IOP (mmHg)	13.43 ± 2.75 (10–20)	14.41 ± 2.36 (10–20)	0.11†
Medications, *n*/patient	1.07 ± 1.26 (0–5)	0.37 ± 0.56 (0–2)	0.023†
Medication-free, eyes (%)	13/30 (43%)	18/27 (66%)	0.13‡
Lens status			
Phakic	17 (57%)	22 (81%)	
Pseudophakic	13 (43%)	5 (19%)	0.08‡
BCVA, logMAR	0.24 ± 0.24 (0–1)	0.09 ± 0.10 (0–0.4)	0.006†
MD (dB)	−7.74 ± 5.21 (−3.14–23.60)	−1.61 ± 1.42 (1.24–4.03)	<0.001†
PSD (dB)	4.70 ± 3.06 (1.50–11.90)	2.02 ± 1.13 (0.23–6.50)	0.004†
ECD	2021.63 ± 481.54 (1028–2923)	2115.33 ± 368.38 (1365–2994)	0.59†
Vertical C/D ratio	0.68 ± 0.14 (0.4–0.9)	0.53 ± 0.14 (0.3–0.8)	<0.001†
ACD (mm)	2.78 ± 0.77 (1.43–4.58)	2.36 ± 0.54 (1.42–3.83)	0.012†
AL (mm)	21.83 ± 1.16 (18.65–24.04)	21.90 ± 1.20 (19.85–25.76)	0.707†
Gonioscopy, grade	1.70 ± 0.76 (0–3)	1.48 ± 0.80 (0–4)	0.17†
CCT (μm)	553.60 ± 48.78 (450–669)	550.19 ± 30.28 (488–615)	0.701†
Gonioscopy PAS, eyes (%)	7/30 (23.3%)	3/27 (11.1%)	0.39‡

ACD, anterior chamber depth; AL, axial length; MD, mean deviation; PAS, peripheral anterior synechiae; PSD, pattern standard deviation.

†Mann–Whitney test.

‡Chi-squared test.

due to the shorter duration of the attack in our patients. Furthermore, LPI was performed within 48 h, and this may have prevented IOP spikes. Other authors could not find any correlation between the duration of the symptoms and the long-term prognosis.[8,14]

As expected, the affected eye was more seriously compromised compared with the fellow eye. The difference between the affected eye and the fellow eye was both structural (vertical C/D) and functional (standard automated perimetry and VA).

In our series, more than 50% of the fellow eyes (30/57) had CACG, although they underwent a prophylactic LPI within 48 h after the APAC attack. Twelve of the fellow eyes (21%) had a significant visual field defect (more than 6 dB) according to the Hodapp classification and 11 (19.3%) according to Brusini's classification. A visual acuity ≥1 logMar determined by glaucoma was present in a single eye.

In Singapore, Friedman *et al.*[9] found glaucomatous optic nerve damage in seven out of 79 (8.9%) of fellow eyes, 6.3 ± 1.5 years since APAC. In two eyes, the glaucomatous optic neuropathy (GON) was pre-existing before the attack. The MD (−15.0 ± 11.9 dB in six eyes) was higher than in our series (−4.83 ± 4.95 dB). The lower prevalence in the Singapore Study can be explained by the stricter criteria of definition of the GON used by the author. Furthermore, owing to the design of our study, it was impossible to determine if CACG in some of the fellow eyes was determined by previous subacute attacks or by an increase of the IOP during the follow-up, although the latter seems less likely as they underwent an LPI, which was patent and presented an IOP within normal limits. If this is the case, our case series underlines the fact that PACG is largely underestimated and treated in Caucasian patients.

Phaco-emulsification in our study was performed at a mean of 35 months after the attack in the affected eye and 45 months in the fellow eye. The affected eyes that underwent phaco-emulsification had a significantly lower use of medications. These findings corroborate the efficacy of phaco-emulsification in the management of patients with PACG, as recently pointed out by the Eagle Clinical Trial.[22]

Our study has several limitations mainly due to its retrospective nature. No or incomplete data regarding the C/D ratio and the visual field defect at baseline were available, no longitudinal data during the follow-up were available, multiple ophthalmologists were involved both in the initial management of the APAC attack and between the attack and the last follow-up visit, and the follow-up period was variable. Nevertheless, our study has the advantage of showing a snapshot of both the affected eye and the fellow eye a reasonable time after the acute attack in a Caucasian population living in a region with prompt access to both the emergency and regular follow-up visits. It also confirms that, although the damage following an APAC attack seems less severe in Caucasians, there is a consistent risk even for this population to develop a significant visual damage both in the affected eye and in the fellow eye.

Our study prompts ophthalmologists to closely observe the patients with APAC to prevent potential long-term harm to the optic nerve. It also suggests

that the incidence of glaucoma after APAC may be underestimated in Caucasians. Our study is unique as it investigates several parameters at least 5 years after APAC in a purely Caucasian population.

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