• Clinical Research •

Uveal effusion following acute primary angle-closure: a retrospective case series

Jian-Gang Yang[1], Jian-Jun Li[2], Hua Tian[1], Yan-Hong Li[1], Yu-Jing Gong[1], An-Le Su[1], Na He[3]

[1]Xi'an Eye Hospital, Xi'an First Hospital, Xi'an 710002, Shaanxi Province, China
[2]Department of Ophthalmology, Xian XD Group Hospital, Xi'an 710077, Shaanxi Province, China
[3]Department of Ophthalmology, the Second Affiliated Hospital of Xi'an Jiaotong University, Xi'an 710004, Shaanxi Province, China
Co-first authors: Jian-Gang Yang and Jian-Jun Li
Correspondence to: Jian-Gang Yang. Xi'an Eye Hospital, Xi'an First Hospital, Xi'an 710002, Shaanxi Province, China. jgyang0077@gmail.com; Na He. Department of Ophthalmology, the Second Affiliated Hospital of Xi'an Jiaotong University, Xi'an 710004, Shaanxi Province, China. 1312046776@qq.com
Received: 2016-03-16 Accepted: 2017-01-12

Abstract

• AIM: To evaluate the morphological changes in anterior segment in Chinese patients with uveal effusion (UE) after the attack of acute primary angle-closure (APAC) using ultrasound biomicroscopy (UBM), and to assess the clinical course and prognosis of the disease.

• METHODS: In a retrospective case series, 26 eyes in 26 consecutive patients diagnosed with UE after the treatment of intraocular pressure (IOP)-lowering medication for the attack of APAC were enrolled. The unaffected fellow eyes served as controls. The morphological changes were observed by ultrasonography, slit lamp microscopy and gonioscopy. UBM was used to assess the degree and extent of effusion based on the analysis of parameters associated with UE.

• RESULTS: The mean IOP was 9.2 (SD 2.1) mm Hg at the diagnosis of UE after IOP-lowering medication, while 14.1 (SD, 2.6) mm Hg in the fellow eyes (*P*=0.000). The anterior chamber depth (ACD) (*P*=0.000), angle opening distance at 500 μm (AOD500) (*P*<0.01) and anterior chamber angle (ACA) (*P*<0.05) were decreased significantly, while ciliary body thickness (CBT) (*P*<0.05) increased significantly in UE eyes. UE grade analysis showed 7 eyes in grade 1, 9 eyes in grade 2, and 10 eyes in grade 3. Quadrant scores were performed of 4 eyes in 1 quadrant, 3 eyes in 3 quadrants, and 19 eyes in 4 quadrants. There was the positive correlation between grade and quadrant score (*R*=0.644, *P*=0.000). The effusion on all eyes were recovered after medication, which mean IOP was 13.9 (SD, 2.8) mm Hg.

• CONCLUSION: UE is a frequent complication in Chinese patients after the attack of APAC, partially associated with hypotony. The severity of UE is correlation with height of effusion, extent of detachment, and shallower ACD.

• KEYWORDS: uveal effusion; acute primary angle-closure; intraocular pressure; ultrasound biomicroscopy

DOI:10.18240/ijo.2017.03.13

Yang JG, Li JJ, Tian H, Li YH, Gong YJ, Su AL, He N. Uveal effusion following acute primary angle-closure: a retrospective case series. *Int J Ophthalmol* 2017;10(3):406-412

INTRODUCTION

Acute primary angle-closure (APAC) is a common ophthalmic emergency in Asian population, with typical symptoms and clinical signs, such as severe ocular pain or discomfort, blurring of vision, and sudden and excessive increases in intraocular pressure (IOP). The shallow anterior chamber, forward convexity of iris and anteriorly inserted iris are factors that make the eye more prone to an acute attack of angle closure[1-2]. It is pivotal that the prompt treatment relieve the symptoms, decrease IOP, and avoid the permanent visual damage. The visual acuity recovers with IOP going to normal level. Otherwise it can lead to permanent eye damage or even blindness in a short time[3].

Conventionally, APAC treatment involves lowering IOP with topical and systemic medication, and laser peripheral iridotomy, iridoplasty or trabeculectomy is usually performed to eliminate pupillary block and to create a new exit route for aqueous humor until the sufficient corneal clarity[4]. Recently, uveal effusion (UE, ciliochoroidal effusion or ciliochoroidal detachment), an abnormal accumulation of fluid into the ciliochoroidal space, has been reported after APAC[5-6]. It is one complication associated with an attack of APAC as the IOP decreased quickly, partially coexisted with ocular hypotony[7-8]. However, little is known regarding pathological mechanism of this disorder. The potential mechanisms of UE associated with APAC are as follows, congestion of choroidal vessels due to initially marked ocular hypertension, exudation from uveal vessels due to inflammation, subsequently ocular hypotony, the adverse effects of acetazolamide and pilocarpine, and choroidal expansions[5,9-10], resulting in the reduction

in hydrostatic pressure gradient and consequent outflow. Ultrasound biomicroscopy (UBM), a high-resolution imaging technique, can be performed to observe ocular structure in the anterior segment. UBM has been detected classical signs of the anterior segment, consisted of pre-existing narrow angles or closed angles, shallow anterior chamber, relatively anteriorly positioned lens, and more important, supraciliary effusion[11-14]. Drug-induced UE associated with primary angle closure glaucoma (PACG)/APAC has been reported in previous literatures[15-16], involving medications such as acetazolamide[17], pilocarpine[18] and timolol[19]. There is a correlation between UE and hypotony with administration of both oral acetazolamide and topical pilocarpine after cataract surgery[15,20]. The ciliary body edema and detachment, relaxation of zonules along with anterior rotation of the ciliary body, and forward shift of the iris-lens diaphragm are reported to contribute to drug-associated UE after the attack of APAC. These changes promote shallow anterior chamber, leading to an increase of supraciliochoroidal fluids[15-16].

In this study we retrospectively investigate 26 consecutive Chinese patients presented with the UE after the attack of unilateral APAC. The aims were to determine the clinical findings, identify the time course of UE in APAC eyes, focus on the morphometric features of anterior segment using UBM, and compare the anterior segment structures between the UE in APAC eyes and the fellow eyes.

SUBJECTS AND METHODS

Ethical approval of the study was obtained from the Ethics Committee of the Xi'an Eye Hospital, China. The work was performed in accordance with the principles of the Declaration of Helsinki.

Study Design In this retrospective study, we reviewed the records of 26 consecutive Chinese patients presented with the UE after the attack of unilateral APAC between September 2011 and October 2016 in our Hospital. The fellow eyes served as controls.

Inclusion Criteria At initial presentation, the diagnosis of attack of APAC with the following symptoms and physical observations: ocular pain with/without periocular pain or irritation, nausea with/without vomiting, blurred vision, IOP ≥30 mm Hg, conjunctival hyperemia, corneal epithelial edema, mid-dilated pupil, and the shallow anterior chamber with an occludable angle[21]. All fellow eyes were observed as a pigmented trabecular meshwork in the eye not visible for at least 180° on static gonioscopy, with an IOP lower than 21 mm Hg, and without peripheral anterior synechiae or glaucomatous neuropathy. Acute attacks were treated according to a standard protocol[19]. In brief, acetazolamide 500 mg by mouth, then 250 mg 6 hourly; pilocarpine drops 2% to the affected eye frequently; timolol eyedrops 0.5% 12 hourly; with/without brimonidine tartrate eyedrops 0.15% 12 hourly; hyperosmotic agent intravenously. The nonattack eyes were also given pilocarpine eyedrops 2%.

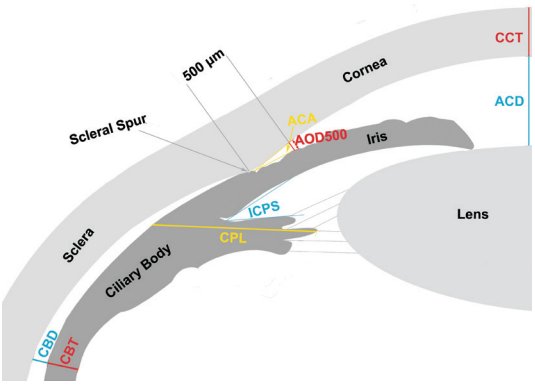

Figure 1 Schematic diagram showing the measurement of anterior segment parameters by UBM.

Following initial medication a further examination was performed, which included assessment of best corrected visual acuity (BCVA), tonometer, gonioscopy, slit lamp microscopy, A/B-scan ultrasonoscopy, perimetry and fundus examination. UBM were diagnose the UE by a single skilled ophthalmologist. Patients with unilateral UE in APAC detected by UBM were enrolled in this study. All eyes with UE accepted to stop medication to lower the IOP, and were treated with fluorometholone drops 0.1% with/without diclofenac Sodium drops 0.1%. The criteria for cure were defined as the reattachment of ciliochoroidal detachment by UBM.

Exclusion Criteria Patients with secondary angle closure associated with uveitis, ocular surgery, laser trabeculoplasty, neovascular, ocular trauma or scleritis were excluded from the clinical diagnosis of APAC associated with UE.

UBM Examination UBM examination was carried out with the UBM Model 840 (Zeiss-Humphrey Inc, San Leandro, CA, USA) with 50 MHz transducer. Each eye was examined in radial sections, and observed the anterior segment at 3, 6, 9 and 12 o'clock positions, including centered on the pupil, the angular region, the ciliary process and pars plana, and peripheral choroid. In the present study, we observed the morphologic changes in the anterior segment, and measured some parameters on UBM images (Table 1; Figure 1)[21]. The degree of UE was classified as previously reported[6]: grade 0 (none), grade 1 (slitlike; supraciliochoroidal space less than half the ciliary body thickness), grade 2 (bandlike; supraciliochoroidal space greater than half the ciliary body thickness), and grade 3 (obvious; supraciliochoroidal space greater than the ciliary body thickness) (Figure 2). Informed consent was obtained from all subjects. Variables that were recorded included age, gender, history of APAC, existence of ocular hypotony (IOP ≤10 mm Hg), use of antiglaucoma medications, including eyedrops, intravenous solutions and oral tablets, and existence and degree of UE.

Statistical Analysis All analyses were performed using SPSS Statistics version 21. Differences in mean values of parametric

Table 1 Anterior segment parameters measured by UBM and their definitions

Parameters	Definition
CCT	The axial thickness from the corneal epithelium to the endothelium
ACD	The axial distance from the corneal endothelium to the anterior lens surface
AOD500	The distance between the posterior corneal surface and the anterior iris surface on a line perpendicular to the trabecular meshwork 500 μm from the scleral spur
ACA	The apex of the iris sulcus and the arms passing through the point 500 μm from the scleral spur and the point perpendicularly opposite on the iris
ICPA	The angle between the posterior iris surface and the ciliary process
CPL	The length of the axis of ciliary process through the apex of ciliary process linked with zonule
CBT	The ciliary body thickness measured at 3 mm from scleral spur
CBD	The distance between the detached ciliary body and sclera measured at 3 mm from scleral spur

CCT: Central corneal thickness; ACD: Anterior chamber depth; AOD500: Angle opening distance at 500 μm; ACA: Anterior chamber angle; ICPA: Iris-ciliary process angle; CPL: Ciliary process length; CBT: Ciliary body thickness; CBD: Ciliary body detachment.

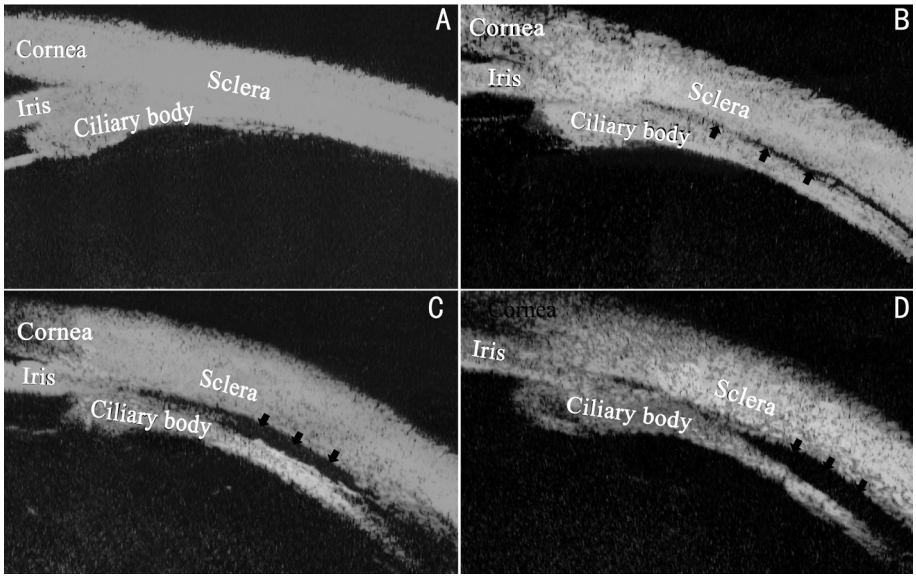

Figure 2 UBM images of each degree of UE after the attack of APAC A: Grade 0. The effusion is observed as a hypoechographic line (B) between the ciliary body and sclera (black arrows), suggesting the presence of serous detachment (grade 1). Panels C and D demonstrate more obvious hypoechographic spaces (black arrows), defines as grades 2 and 3, respectively.

data between bilateral eyes or between baseline and follow-ups were examined using an independent samples Student's *t*-test. We used Pearson's correlation coefficient between two quantitative variables, and Spearman's rank correlation coefficient for non-parametric test. A *P*-value of less than 0.05 was considered statistically significant.

RESULTS

Twenty-six consecutive patients with APAC coexisted with UE were recruited. The mean IOP was 49.5 mm Hg (95%CI: 46.7, 52.3) at initial presentation. The most common symptoms were blurring of vision (96.1%), followed by pain (92.3%), vomiting (46.1%) and halos (34.6%). The mean IOP was 15.2 mm Hg (95%CI: 14.0, 16.4) in fellow eyes. There was significantly different of the IOPs between bilateral eyes (*P*=0.000) (Figure 3). Demographic and clinical characteristics of the patients are summarized in Table 2.

The mean IOP was 9.2 mm Hg at the diagnosis of APAC secondary to UE, which hypotony existed in 18 eyes (69.2%), meanwhile the mean IOP was 14.1 mm Hg in fellow eyes

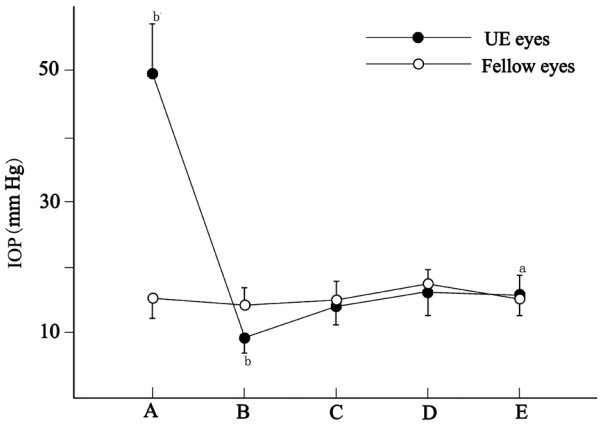

Figure 3 IOP curves of bilateral eyes during the course of unilateral UE associated with APAC A: IOPs at the attack of APAC. The difference was significant between bilateral eyes (*P*=0.000); B: IOPs at initial presentation of UE after APAC (*P*=0.000); C: IOPs at the disappearence of UE; D: IOPs before surgery; E: IOPs at one month after surgery (*P*=0.036). Data were given as mean±SD. [a]*P*<0.05; [b]*P*<0.01.

(*P*=0.000) (Table 3; Figure 3). Subsequently, UBM was per-

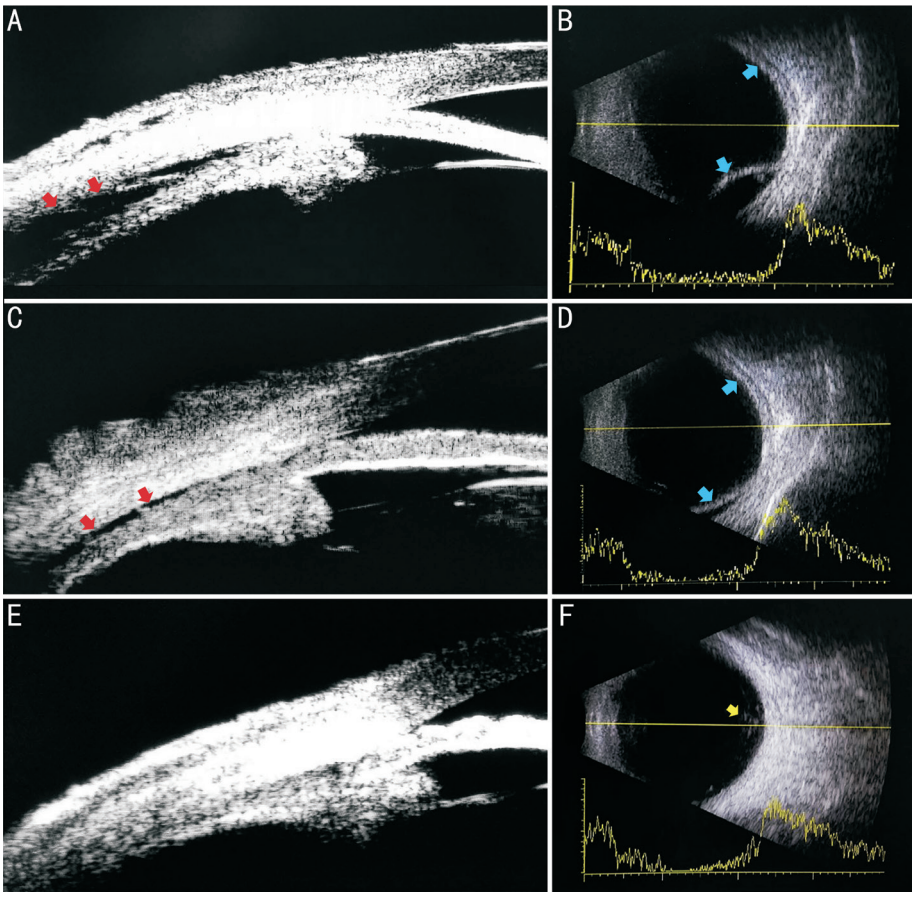

Figure 4 The time course of recovery in a case with UE after the attack of APAC monitored by UBM and B-scan ultrasonography
A 51-year-old female patient presented with IOP of 8.2 mm Hg had a supraciliary effusion of grade 3 in left eye (A, red arrows) on UBM image, and the choroidal efffusion (B, blue arrows) on B-scan image after the attack of APAC. Seven days later, both the supraciliary effusion (C, red arrows) and choroidal effusion (D, blue arrows) were alleviated. Twelve days later, UE disappeared on UBM and B-scan images (E, F), however, it remained the marked imflammatory opacities in posterior vitreous (F, yellow arrow) on B-scan image.

Table 2 Demographic features at baseline of patients with the attack of APAC

Characteristics	Subjects of study
Patients/eyes (*n*)	26/52
Mean age (a, $\bar{x} \pm s$)	61.7±9.3
Gender (M/F)	5/21
Laterality (APAC), OD/OS	12/14
Duration of experienced attack, d (SD)	5.1 (2.5)
No. of glaucoma medications (SD)	5.2 (1.2)
Pilocarpine (eyes)	26
Acetazolamide (eyes)	26
Timilol (eyes)	26
Brimonidine (eyes)	20
Mannitol (eyes)	21
Fluorometholone (eyes)	15

SD: Standard deviation; APAC: Acute primary angle-closure.

formed to document the morphological structures of anterior segment. The fellow eyes had shallower anterior chamber, narrow angle, and the anterior eminence of peripheral iris. In comparison of the fellow eyes, the features on UE eyes were as follows (Figures 4A, 5A), the shallower anterior chamber [supported by parameter anterior chamber depth (ACD)], the extremely narrow angle, even the appositional angle closure [supported by angle opening distance at 500 μm (AOD500), anterior chamber angle (ACA)], and anterior shift of iris-lens diaphragm [supported by iris-ciliary process angle (ICPA)], which was confirmed as the iris root angulated forward and the closed entrance of the angle using gonioscopy. The ciliary body rotated forward about its attachment to the scleral spur with the extensive edema at ciliary process and pars plana [supported by ciliary process length (CPL) and ciliary body thickness (CBT)]. However, the configuration of ciliary body remained identified (Figures 4, 5). The corneal edema remained observed on partial UE eyes [supported by central corneal thickness (CCT)], confirmed in 10 eyes (47.6%) by slit lamp microscopy.

Of all 26 patients with UE, grade 1 was detected in 7 eyes (26.9%), grade 2 in 9 eyes (34.6%), and grade 3 in 10 eyes (38.5%) [supported by CBT and ciliary body detachment (CBD)]. In addition, the effusion was observed between 1 quadrant and 4 quadrants. Four eyes (15.4%) showed effusion in 1 quadrant, 3 eyes (11.5%) in 3 quadrants, and 19 eyes (73.1%) in 4 quadrants. Ten eyes of grade 3 effusion were all detected in 4 quadrants. A positive correlation is relationship

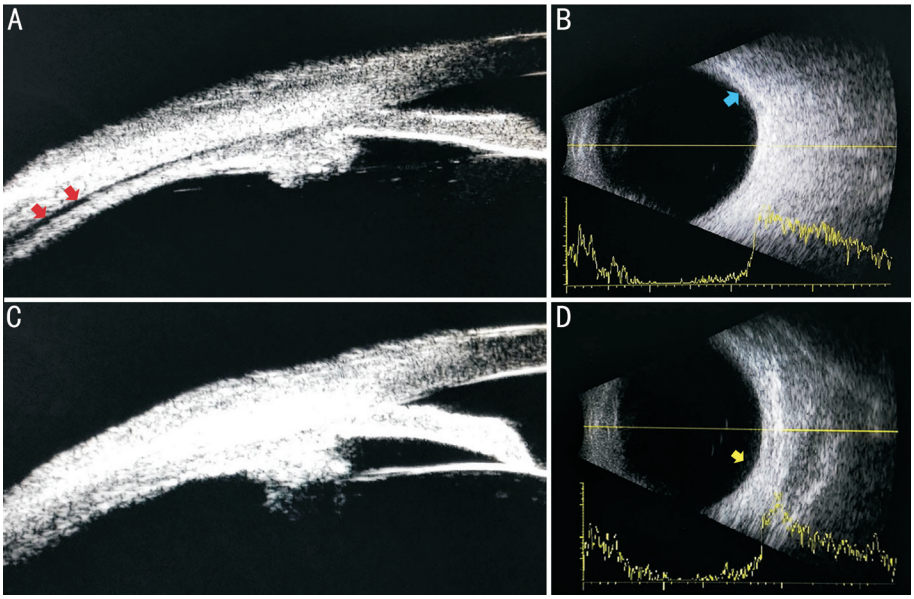

Figure 5 The time course of recovery in another case with UE after the attack of APAC IOP was 9.4 mm Hg in a female patient of 60y. Supraciliary effusion was grade 1 in left eye (A, red arrows) on UBM image, and the choroidal efffusion (B, blue arrow) was crevice-like by B-scan after APAC. Four days later, UE disappeared on UBM and B-scan images (C and D), however, there was the imflammatory opacities in posterior vitreous (D, yellow arrow).

Table 3 Demographics and ocular biometric parameters of in APAC affected eyes and fellow eyes

Parameters	Affected eyes			Fellow eyes			*P*
	Mean	SD	95%CI	Mean	SD	95%CI	
IOP (mm Hg)	9.2	2.1	8.4-10.0	14.1	2.6	13.0-15.1	0.000
BCVA	20/100		20/125-20/80	20/40		20/50-20/32	
AL (mm)	22.3	0.6	22.1-22.5	22.4	0.6	22.2-22.6	0.778
CCT (μm)	596.2	86.2	566.9-635.1	536.5	36.2	532.1-550.4	0.004
ACD (μm)	1648.1	277.5	1525.4-1748.8	1918.8	153.0	1861.9-1975.4	0.000
AOD500 (μm)	51.6	91.9	26.9-77.7	99.6	87.4	73.6-122.4	0.001
ACA (μm)	5.5	9.4	3.0-8.1	8.5	7.8	6.4-10.5	0.044
CPL (μm)	1195.2	240.7	1134.2-1260.6	1052.3	291.8	971.5-1133.2	0.000
ICPA (μm)	39.8	23.5	33.9-46.8	48.7	21.1	43.0-54.4	0.067
CBT (μm)	322.7	81.1	299.4-345.0	284.6	78.6	264.4-305.4	0.026
CBD (μm)	270.6	169.4	224.6-317.1				

between grades and quadrants (R=0.644, P=0.000). The coexistence with choroidal detachment was observed in 5 eyes (19.2%) with B-scan ultrasound (Figures 4B, 4D, 5B). In addition, a negative correlation existed between ACD and UE (ACD and CBD, R=-0.408, P=0.003; ACD and grade, R=-0.540, P=0.000). However, there was no relationship between ACD and quadrants (R=-0.162, P=0.250).

The median BCVAs were 20/100 and 20/40 in the affected eyes and the fellow eyes, respectively. The mean axial length (AL) was detected with A-scan ultrasonoscopy, which there was no significant difference between bilateral eyes. In comparison of the fellow eyes, CCT in the affected eyes was significantly thicker, suggesting that the corneas remained edema. ACD, AOD500 and ACA were significantly decreased, showing the shallower anterior chamber, and narrower even closed angle. CPL and CBT were increased, indicating that the edema of cilary body. Whereas, ICPA were not different, indicating the anterior rotation of ciliary process both the bilateral eyes.

The eyes with UE were treated with fluorometholone 0.1% and diclofenac sodium 0.1%, while the antiglaucoma medications were stopped. The effusion in all eyes disappeared after medication, accompanied by a significant deepening of the anterior chamber (P<0.05) (Table 4), which indicated as a reduction of the anterior rotation of ciliary process. The mean duration for UE recovery was 5.7d (SD: 3.6, 95%CI: 4.4, 7.1). The prognosis analysis showed the recovery duration was negative correlation with IOP at the presentation of UE (R=-0.457, P=0.019) and quadrants (R=-0.390, P=0.049), but no relationship with grade (R=0.334, P=0.095). The mean IOP increased to 13.9 mm Hg, which was significantly different compared with that at the first UE presentation (P<0.01) (Figure 3). However, the noted

Table 4 Demographics and biometric parameters after UE recovery

Parameters	Affected eyes			Fellow eyes			*P*
	Mean	SD	95%CI	Mean	SD	95%CI	
IOP (mm Hg)	16.0	3. 7	14.6-17.6	17.4	2.0	16.6-18.3	0.000
BCVA	20/50		20/80-20/40	20/40		20/50-20/32	
CCT (μm)	573.7	34.2	561.3-587.0	555.8	34.6	542.1-570.1	0.039
ACD (μm)	1812.3	132.2	1763.5-1860.4	1904.2	162.3	1842.7-1968.4	0.017
LPI (eyes)	14			26			
Trabeclectomy (eyes)	7			0			
Phacotrbeclectomy (eyes)	5			0			

The median BCVAs were 20/50 and 20/40 in the affected eyes and the fellow eyes, respectively. LPI: Laser peripheral irodotomy.

inflammatory opacities developed in the posterior vitreous body during the UE recovery (Figures 4F, 5D). APAC eyes with UE were performed laser iridotomy, trabeculectomy or phacotrabeculectomy, when IOP was increased and maintained at a relatively constant level. The fellow eyes were undergone laser iridotomy. The mean IOP was 15.2 mm Hg (SD: 2.5, 95%CI: 14.2, 16.2) in the eyes of APAC coexisted with UE at 1mo after surgery (Figure 3).

DISCUSSION

UE after the attack of APAC is a frequent disease in Asia, defined as the hypoechographic space between the uvea internally and sclera externally by UBM[22-23]. However, only a few studies have demonstrated the clinical features of the disorder. Kumar *et al*[14] demonstrated as a significant proportion of UE in Asian population that the prevalence of UE was 25.0% (3 of 12 eyes) in APAC eyes, and 15.7% (11 of 70 eyes) in PACG eyes on UBM. Recently, You *et al*[23] reported that the prevalence of UE was 29.3% in Chinese patients with APAC.

It is well known that UBM can reveal the characteristics of APAC, such as the shallow anterior chamber and the closed angle. In the present study, we investigated the morphological changes of UE in APAC by ultrasonoscopy, composed of the shallower anterior chamber, extremely narrow even closed angle, more forward angulation of peripheral the iris root, and more anterior shift of iris-lens diaphragm on the basis of fellow eyes. The scleral spur is an important anatomical landmark in the anterior segment of UE, which is the point of attachment to eye wall when anterior rotation of ciliary body, with the extensive edema at ciliary process and pars plana, and edema of anterior choroids (quantitative measurement was not performed in this study). The supraciliary effusion was observed by UBM, further 19.2% coexistence choroidal effusion by B-scan ultrasonography. UE is contributed to shallowness of the anterior chamber. In the present study, the eyes with UE had shallower anterior chambers compared with those without effusion. The severity of a shallow anterior chamber was correlation to the height of ciliary detachment (R=-0.408) or UE grade (R=-0.540), but no relationship with quadrants. The ciliary body edema or detachments causes forward rotation of the ciliary body, displacing the iris forward to close the angle and resulting in shallower anterior chamber. In addition, ciliary body edema produces relaxation of the zonules, which results in lens thickening. The thick and relatively anteriorly positioned lens may also contribute to the shallower anterior chamber[6].

An attack is associated with remarkable ocular hypertension in the eyes with APAC. Subsequently, the sudden decrease of IOP develops in the partial eyes due to ciliary body shutdown induced by the prolonged attack[24]. The ischemia of ciliary body lead to an alteration in the capillary permeability, and reduction in aqueous production by virtue of the persistent exposure of eyes to high IOP, also possibly due to the inflammation in ciliary body[25-26]. In the present study, the suppression of aqueous humor may persist for one week in most cases after the halt on aqueous suppressants, thus causing the transient hypotony.

UE recovered as a mean duration of 5.7d, and IOP recover to normal level. The vitreous body showed the marked posterior inflammation. Use of corticosteroids may be considered as necessary, and thought to stabilize the blood-aqueous, relieve uveitis gradually, regain the ciliary function to decrease the damage of choroidal circle, advance the functional recovery of ciliary epithelium to increase aqueous humor formation, thereby promoting resolution of the edema and effusions, and elevating the IOP[27]. The discontinuation of topical treatment for APAC results in the reattachment of ciliochoroidal detachment. Topical miotics should be avoided as they may promote anterior movement of the lens-iris diaphragm, and worsen the effusion[7]. By analyzing the outcomes, we considered as the prognosis factors were IOP at pesentation of UE, quadrants, as well as grade. The recovery of UE was associated with hypotony, severity of detachment and extension of effusion quadrants.

In conclusion, UE diagnosed by UBM examination is frequently observed in eyes with APAC, which is usually associated with hypotony. The severity of UE is correlation

with height of effusion, extent of detachment, as well as shallower ACD. Corticosteroids is necessary to recover the disorder. The UBM examination is a very useful technique in the diagnosis and management of UE in eyes with APAC.

ACKNOWLEDGEMENTS

Foundation: Supported by the Key Science and Technology Program of Shaanxi Province, China (No.2015SF146).

Conflicts of Interest: Yang JG, None; **Li JJ,** None; **Tian H,** None; **Li YH,** None; **Gong YJ,** None; **Su AL,** None; **He N,** None.

REFERENCES

1 Nongpiur ME, Foo VHX, Leon JM, *et al*. Evaluation of choroidal thickness, intraocular pressure, and serum osmolality after the water drinking test in eyes with primary angle closure. *Invest Ophthalmol Vis Sci* 2015;56(4):2135-2143.

2 Park HYL, Shin HY, Jung KI, Park CK. Changes in the lamina and prelamina after intraocular pressure reduction in patients with primary open-angle glaucoma and acute primary angle-closure. *Invest Ophthalmol Vis Sci* 2014;55(1):233-239.

3 Lee DA, Higginbotham EJ. Glaucoma and its treatment: a review. *Am J Health Syst Pharm* 2005;62(7):691-699.

4 Boey PY, Singhal S, Perera SA, Aung T. Conventional and emerging treatments in the management of acute primary angle closure. *Clin Ophthalmol* 2012;6:417-424.

5 Gazzard G, Friedman DS, Devereux J, Seah S. Primary acute angle closure glaucoma associated with suprachoroidal fluid in three Chinese patients [letter]. *Eye* 2001;15:358-360.

6 Sakai H, Morine-Shinjyo S, Shinzato M, Nakamura Y, Sakai M, Sawaguchi S. Uveal effusion in primary angle-closure glaucoma. *Ophthalmology* 2005;112(3):413-419.

7 Singer JR, Pearce ZD, Westhouse SJ, Siebert KJ. Uveal effusion as a mechanism of bilateral angle-closure glaucoma induced by chlorthalidone. *J Glaucoma* 2015;24(1):84-86.

8 Mukherji S, Ramanathan S, Tarin S. Uveal effusion associated with campylobacter jejuni infection presenting as bilateral angle closure glaucoma. *J Glaucoma* 2011;20(9):587-588.

9 Fineman MS, Katz LJ, Wilson RP. Topical dorzolamide-induced hypotony and ciliochoroidal detachment in patients with previous filtration surgery. *Arch Ophthalmol* 1996;114(8):1031-1032.

10 Quigley HA, Friedman DS, Congdon NG. Possible mechanisms of primary angle closure and malignant glaucoma. *J Glaucoma* 2003;12(2):167-180.

11 Alimgil ML, Benian O. Choroidal effusion and shallowing of the anterior chamber after adjunctive therapy with latanoprost in a trabeculotomized patient with angle closure glaucoma. *Int Ophthalmol* 2001;24(3):129-131.

12 Alexander P, Ramirez-Florez S. Hypotony and choroidal detachment as a complication of travoprost after trabeculectomy surgery. *Eye* 2008;22(5):736-737.

13 Sugimoto K, Ito K, Esaki K, Miyamura M, Sasoh M, Uji Y. Supraciliochoroidal fluid at an early stage after trabeculectomy. *Jpn J Ophthalmol* 2001;105(11):766-770.

14 Kumar RS, Quek D, Lee KY, *et al*. Confirmation of the presence of uveal effusion in Asian eyes with primary angle closure glaucoma: an ultrasound biomicroscopy study. *Arch Ophthalmol* 2008;126(12):1647-1651.

15 Hanna R, Tiosano B, Dbayat N, Gaton D. Unilateral angle-closure glaucoma with ciliochoroidal effusion after the consumption of cannabis: a case report. *Case Rep Ophthalmol* 2014;5(3):439-443.

16 Ramos-Esteban JC, Goldberg S, Danias J. Drug induced acute myopia with supraciliary choroidal effusion in a patient with Wegener's granulomatosis. *Br J Ophthalmol* 2002;86(5):594-596

17 Lachkar Y, Bouassida W. Drug-induced acute angle closure glaucoma. *Curr Opin Ophthalmol* 2007;18(2):129-133.

18 Malagola R, Arrico L, Giannotti R, Pattavina L. Acetazolamide-induced cilio-choroidal effusion after cataract surgery: unusual posterior involvement. *Drug Des Devel Ther* 2013;7:33-36.

19 Vela MA, Campbell DG. Hypotony and ciliochoroidal detachment following pharmacologic aqueous suppressant therapy in previously filtered patients. *Ophthalmology* 1985;92(1):50-57.

20 Sabti K, Lindley SK, Mansour M, Discepola M. Uveal effusion after cataract surgery: an echographic study. *Ophthalmology* 2001;108(1):100-103.

21 Moghimi S, Ramezani F, He M, Coleman AL, Lin SC. Comparison of anterior segment-optical coherence tomography parameters in phacomorphic angle closure and acute angle closure eyes. *Invest Ophthalmol Vis Sci* 2015;56(13):7611-7617.

22 Kawahara S, Nagai Y, Kawakami E, Ida RY, Takeuchi M, Uyama M. Ciliochoroidal detachment following scleral buckling surgery for rhegmatogenous retinal detachment. *Jpn J Ophthalmol* 2000;44(6):692-693.

23 You YA, Zhu LR, Wen JQ, Liu YH. Ultrasound Biomicroscopic evaluation of uveal effusion in acute primary angle closure. *J Glaucoma* 2015;24(9):656-661.

24 Fricke TR, Mantzioros N, Vingrys AJ. Management of patients with narrow angles and acute angle-closure glaucoma. *Clin Exp Optom* 1998;81(6):255-266.

25 Rao A, Gupta V, Garudadri CS. Atypical ocular ischaemia in angle-closure glaucoma and anaemia. *Int Ophthalmol* 2013;33(3):295-297.

26 Amerasinghe N, Aung T. Angle-closure: risk factors, diagnosis and treatment. *Prog Brain Res* 2008;173:31-45.

27 Benson BE, Mandal K, Bunce CV, Fraser SG. Is post-trabeculectomy hypotony a risk factor for subsequent failure? A case control study. *BMC Ophthalmol* 2005,5:7.