Uveal Effusion in Primary Angle-Closure Glaucoma

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Purpose: To determine the prevalence of uveal effusion in acute and chronic primary angle-closure glaucoma (PACG) or primary angle closure (PAC) and to compare it with the prevalence in eyes with open-angle glaucoma (OAG) or ocular hypertension.

Design: Prospective consecutive case series.

Participants: Five hundred one eyes of 351 consecutive patients with PAC and 156 eyes of 116 randomly selected primary OAG or ocular hypertension patients. The PAC group included 40 eyes of 35 patients with acute PACG and 30 unaffected fellow eyes, 39 eyes and 35 fellow eyes with a history of acute PACG, and 357 eyes with chronic PAC.

Methods: Ultrasound biomicroscopic examination was performed to diagnose uveal effusion and to measure anterior chamber depth (ACD).

Main Outcome Measures: Presence of uveal effusion and ACD.

Results: Uveal effusion was demonstrated in 23 eyes (58%) with acute PACG and 7 fellow eys (23%) ($\chi^2 = 8.17$, $P = 0.0043$). Among eyes with chronic PAC, uveal effusion was present in 71 (20%), a higher prevalence than was found in open-angle patients (2 eyes [1.3%]) ($\chi^2 = 19.3$, $P<0.001$). In the chronic PAC group, the ACD of phakic eyes with uveal effusion (1.92±0.42 mm) was significantly shallower than that of phakic eyes without effusion (2.06±0.32 mm) ($P = 0.019$).

Conclusions: Uveal effusion diagnosed by ultrasound biomicroscopy is a special feature in PAC, and is prevalent in acute PACG. Uveal effusion in phakic eyes with PAC is associated with shallowing of ACD. *Ophthalmology 2005;112:413–419 © 2005 by the American Academy of Ophthalmology.*

Short axial length, shallow anterior chamber depth (ACD), and a thick and relatively anteriorly positioned lens are common anatomical features of eyes with primary angle-closure glaucoma (PACG).[1,2] Lowe described the anatomical basis of 118 eyes with PACG, noting that the average axial length of eyes with PACG is 22.01 mm (range, 18.5–26.0), more than 1 mm shorter than that of normal eyes (average, 23.10). In nanophthalmos, angle-closure glaucoma (ACG) occurs commonly. Singh et al reported that, of 32 eyes with nanophthalmos, 18 had ACG, and 4 additional eyes had closed angles without glaucoma.[3] In this report, axial length ranged between 14.5 mm and 20.5 mm (average, 17.00). Although nanophthalmos is a rare condition, and typical anatomical features of nanophthalmic eyes differ from those of eyes with PACG, these two conditions share certain anatomical features, including a small eye, shallow anterior chamber, relatively thick lens, and hyperopia.

Uveal effusion (ciliochoroidal, suprachoroidal, or choroidal effusion or choroidal or ciliochoroidal detachment), an abnormal accumulation of fluid into the suprachoroidal space, is one diagnostic feature of nanophthalmos.[4,5] Bullous serous detachment of the retina sometimes accompanies uveal effusion, and idiopathic uveal effusion and serous retinal detachment (RD) are termed *(idiopathic) uveal effusion syndrome.*[6] Gass and Jallow[6] described that idiopathic uveal effusion syndrome is differentiated from postoperative uveal effusion and from secondary uveal effusion caused by Vogt–Koyanagi–Harada syndrome, tumor, systemic diseases, or posterior scleritis,[7–9] but that uveal effusion in nanophthalmos is included in the definition of uveal effusion syndrome.[6] Uyama et al reported 16 cases of uveal effusion syndrome.[10] Six of their patients (2 male and 4 female) had nanophthalmos, with axial lengths ranging from 15.0 mm to 17.0 mm (average, 16.0).[10] Uveal effusion with nanophthalmic eyes occurs not only in unoperated eyes (uveal effusion syndrome), but also in operated eyes when intraocular pressure (IOP) is temporally lowered by any ocular surgical procedure.[11–14] Postoperative uveal effusion or uveal effusion syndrome in eyes with nanophthalmos may be caused by occult uveal effusion that is present preoperatively.

In eyes with PACG, uveal effusion may occur after surgical procedures, such as trabeculectomy[15] or laser iridotomy.[16,17] Choroidal effusion after acute PACG also has been reported.[18–20] Recently developed ultrasonic diagnos-

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Table 1. Characteristics of Subjects

	PAC	Open Angle	*P* (Student's Unpaired *t* Test)
n (eyes)	501	156	
Age (yrs) (mean ± SD)	67±8.7	60±14	<0.001
Gender (eyes) (male:female)	178:328	91:71	<0.001*
Axial length (mm) (mean ± SD)	22.5±0.82 (n = 331)	24.2±1.56 (n = 32)	<0.001
Anterior chamber depth (mm) (mean ± SD)	1.99±0.36	2.78±0.33	<0.001

PAC = primary angle closure; SD = standard deviation.
*Chi-square test.

tic technology (i.e., the ultrasound biomicroscope) allows diagnosis of uveal effusion when ophthalmoscopic observation cannot be performed because the pupil size is small or the cornea is edematous. Morever, ultrasound biomicroscopy (UBM) can depict a uveal effusion that is too small to be detected by fundus examination.[21]

In this study, we used UBM to investigate the prevalence of uveal effusion in eyes with acute PACG and chronic primary angle closure (PAC) eyes.

Subjects and Methods

We performed a prospective, observational, high-resolution echographic study on 501 eyes of 351 consecutive patients with PAC and 156 eyes of 116 randomly selected patients with primary open-angle glaucoma (POAG) or ocular hypertension. All subjects were Japanese. A total of 35 patients were diagnosed with acute PACG (40 affected eyes and 30 fellow eyes) and underwent immediate medical therapy. In 431 eyes of 316 patients with chronic PAC, 37 patients had a history of acute PACG (39 affected eyes and 35 fellow eyes).

The criterion for acute PACG was presence of all the following symptoms and physical observations: ocular and/or periocular pain or irritation, nausea and/or vomiting, blurred vision, IOP ≥ 40 mmHg, conjunctival hyperemia, corneal epithelial edema, middilated pupil, and bilateral shallow anterior chamber with an occludable angle. Criteria for PAC were a bilateral shallow anterior chamber and synechial or appositional angle closure diagnosed by both indentation gonioscopy and UBM. Patients with secondary angle closure associated with uveitis, ocular surgery, laser trabeculoplasty, ocular trauma, or scleritis were excluded from the clinical diagnosis of PAC.

At initial examination, all patients underwent ophthalmic evaluation, which included slit-lamp examination, gonioscopy, and applanation tonometry. Ultrasound biomicroscopic examinations were performed by a single skilled physician (HS). Axial length measured by A-mode ultrasonography was obtained in 294 eyes of patients with PAC and 32 eyes of patients with open-angle glaucoma or ocular hypertension. Informed consent was obtained from all subjects. Variables that were recorded include age; gender; history of ocular surgery or laser treatment; existence of ocular hypotony (IOP < 10 mmHg); use of antiglaucoma medications, including pilocarpine eyedrops and intravenous and/or oral acetazolamide; ACD; and existence and degree of uveal effusion.

On the first visit, patients with acute PACG were given immediate medical treatment, which included application of 2% pilocarpine eyedrops, oral and/or intravenous injection of acetazolamide, and administration of an intravenous hyperosmotic agent to release an acute attack. Two percent pilocarpine eyedrops were applied every 15 minutes for 2 to 4 hours to affected acute PACG eyes and continued every 6 hours until surgical treatment had been performed. Unaffected fellow eyes were also given 2% pilocarpine

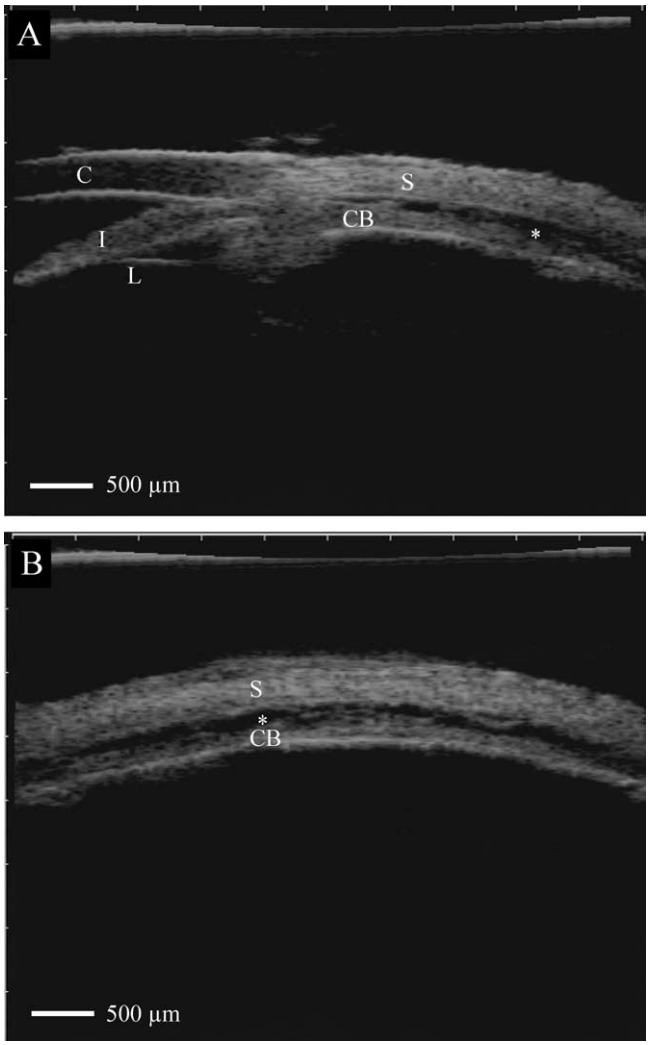

Figure 1. Radial (**A**) and transverse (**B**) sections of ultrasound biomicroscopic findings of a 44-year-old female's eye with acute primary-angle glaucoma. Intraocular pressure was 44 mmHg at initial examination, and was successfully reduced to 12 mmHg on the next day. Grade 3 uveal effusion (*) was evident as a hypoechographic area between the sclera (S) and the pars plana of the ciliary body (CB) on the next day. Note that the angle was still closed. C = cornea; I = iris; L = lens.