Biometric Characteristics of Eyes With Central Serous Chorioretinopathy

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Purpose. To investigate the biometric characteristics of eyes with idiopathic central serous chorioretinopathy (CSC).

Methods. Medical records of 52 consecutive patients with unilateral CSC were reviewed. Central serous chorioretinopathy was diagnosed using spectral-domain optical coherence tomography (SD-OCT) and fluorescein angiography. Data collected for comparison with fellow eyes were refractive error, biometric measurements using partial coherence interferometry, and SD-OCT parameters.

Results. Mean time from subjective symptom onset to initial visit was 8.3 ± 12.29 weeks. Mean axial length (AL) was shorter in CSC eyes than in fellow eyes by 0.24 ± 0.379 mm ($P < 0.001$), and mean anterior chamber depth (ACD) was shallower in CSC eyes than in fellow eyes by 0.03 ± 0.088 mm ($P = 0.021$). Central serous chorioretinopathy eyes also had thicker subfoveal choroidal thickness (CT) than fellow eyes by 34.0 ± 45.93 μm ($P < 0.001$). Differences in spherical equivalents between CSC and fellow eyes correlated with AL differences ($r = -0.690$, $P < 0.001$) and CT differences ($r = 0.473$, $P = 0.001$). On multiple linear regression analysis, the differences in ACD between CSC and fellow eyes were significantly correlated with AL differences ($P = 0.032$) and symptom duration ($P = 0.019$).

Conclusions. Biometric characteristics such as AL and ACD were different between eyes with CSC and fellow eyes. Variations in biometry, which correlated with CT differences, might be related to differences in refractive errors between eyes.

Keywords: central serous chorioretinopathy, anterior chamber depth, axial length, choroidal thickness, optical biometry

Central serous chorioretinopathy (CSC) is characterized by serous detachment of the neurosensory retina and retinal pigment epithelium (RPE) at the posterior pole. Central serous chorioretinopathy typically affects young to middle-aged adults.[1] Many patients with CSC first notice a minor blurring of vision followed by various degrees of visual symptoms. In approximately 90% of cases, CSC spontaneously resolves within a few months, and most patients recover their baseline visual acuity.[2,3] However, some patients experience persistent CSC without resolution for greater than 3 months, recurrence of CSC, or deterioration of vision.[4–7] The visual symptoms of a patient with CSC are caused by morphological changes in the retina.[3,8–11]

Recent advances in imaging technology have expanded our knowledge of ocular diseases including CSC.[12,13] Studies using optical coherence tomography (OCT) demonstrate that subfoveal choroidal thickness (CT) increases in CSC eyes compared with normal eyes.[14–17] The choroidal thickening might influence hypermetropization or transient hyperopic shifting in eyes with CSC, even though anteriorly, displacement of the detached neurosensory retina is the main cause of hyperopic change.[3,18,19]

The choroid is the posterior portion of the uvea and extends from the margins of the optic nerve to the pars plana, where it continues anteriorly to become the ciliary body. In addition to supplying oxygen and nutrients to the outer retina, the choroid is important for draining aqueous humor from the anterior chamber via the uveoscleral pathway.[20] Venous return from both the ciliary body and choroid primarily occurs through the vortex veins.[21] Therefore, in eyes with CSC, changes in the posterior segment might be associated with variation of the anterior segment, which could subsequently affect biometric measurements. However, detailed biometric characteristics related to choroidal thickening and the effects of choroidal thickening on optical characteristics have not been investigated in CSC eyes.

Partial coherence interferometry (PCI) offers biometric measurements with high precision and resolution.[22] Partial coherence interferometry is a noninvasive technique that might be able to measure axial length (AL), even in eyes with macular edema.[23] We hypothesized that optical and biometric characteristics in CSC eyes with subretinal fluid would be different from those in nonsymptomatic fellow eyes and that these differences would correlate with differences in OCT measurements such as retinal thickness or CT. Therefore, we used PCI and spectral-domain OCT (SD-OCT) to compare the biometric characteristics of idiopathic CSC eyes with subretinal fluid with those of nonsymptomatic fellow eyes.

Methods

This study was approved by the Institutional Review Board of the Korea University Medical Center in Seoul, South Korea. All research was conducted in accordance with the tenets of the Declaration of Helsinki.

Patients and Data Collection

We retrospectively reviewed the medical records of patients with idiopathic CSC who were examined at the Korea University Medical Center between March 2010 and October 2013. Idiopathic CSC was diagnosed as the presence of serous detachment of the neurosensory retina involving the macula, confirmed by SD-OCT and leakage at the level of the RPE on fluorescein angiography (FA). We included patients with unilateral involvement at the time of examination, who underwent both biometry with PCI and choroidal imaging with SD-OCT. Exclusion criteria were (1) no evidence of serous retinal detachment on SD-OCT; (2) steroid-induced CSC; (3) other retinal disease such as age-related macular degeneration, polypoidal choroidal vasculopathy, idiopathic choroidal neovascularization, diabetic retinopathy, retinal vascular occlusion, intraocular inflammation, or posterior segmental tumor; or (4) a history of intraocular treatments such as intravitreal injection of antivascular endothelial growth factor within the previous 3 months, or photodynamic therapy, filtering surgery, or pars plana vitrectomy.

In addition to FA and SD-OCT, patients underwent best-corrected visual acuity (BCVA) measurements using a Snellen chart, automated refraction, biometry with PCI, applanation tonometry, biomicroscopy examination, indirect ophthalmoscopy, and fundus photography. Additional data recorded for each patient included sex, age, laterality, and duration of subjective symptoms (such as relative central scotoma, metamorphopsia, micropsia, dyschromatopsia, and blurring of vision) prior to the initial visit. We compared measurements of BCVA, refractive error, intraocular pressure, keratometric (K) value, AL, anterior chamber depth (ACD), central subfield retinal thickness (CRT), and subfoveal CT in CSC eyes with those in fellow eyes. Additional comparisons were made after patients were classified into two groups according to symptom duration. One group experienced onset of subjective symptoms within 3 months of the initial visit, and the other group experienced symptoms sooner than 3 months before the initial visit. Patients with recurrent CSC were included in the former group, when their most recent symptoms developed within 3 months of the initial visit.

FA and SD-OCT

FA was performed with a fundus camera (model FF 450 Plus; Carl Zeiss Meditec AG, Jena, Germany). Using FA images, we classified leakage patterns as expansile dot, smokestack, or diffuse pattern.[24] The SD-OCT unit (three-dimensional [3D] OCT-1000 Mark II model; version 3.20 software; Topcon Corp., Tokyo, Japan) we used had a wavelength of 840 nm, a horizontal resolution of $\leq$20 μm, and an axial resolution of up to 5 μm. Its imaging speed was 27,000 axial scans per second. Patients underwent SD-OCT evaluation using 3D scanning protocols with 128 B-scans (512 A-scans per B-scan) of a 6-mm $\times$ 6-mm area and line scanning protocols with an average of 50 B-scans (1024 A-scans per B-scan) with a 6-mm length. Central subfield retinal thickness was assessed at the macular center (1-mm diameter) using topographic mapping. Choroidal mode with horizontal 6-mm line scans was used to obtain choroidal images. Subfoveal CT was measured perpendicularly from the outer surface of the hyper-reflective line corresponding to the RPE to the inner surface of the sclera at the foveal center.

Refractive Error and Optical Biometric Data

Refractive error was measured with an automated refractor (RK-F1 model; Canon, Inc., Tochigi, Japan). The spherical equivalent was defined as the sum of the sphere and one-half of the cylinder power and was used to calculate the refractive error in diopters (D). Refractive astigmatism was obtained from cylinder power. Keratometric, AL, and ACD values were measured using PCI (IOLMaster, version 5.0; Carl Zeiss Meditec). Horizontal K value was defined as the K value of the axis from 0° to 45° and 136° to 180°, and vertical K value was defined as K value of the axis from 46° to 135°. Corneal astigmatism was calculated as the difference between flat and steep K values, and the axis of the flat K was recorded. The axis differences between refractive astigmatism and corneal astigmatism were (1) if $|$ refractive astigmatic axis $-$ corneal astigmatic axis $| \leq 90$, axis difference $= |$ refractive astigmatic axis $-$ corneal astigmatic axis $|$; and 2) if $|$ refractive astigmatic axis $-$ corneal astigmatic axis $| > 90$, axis difference $= 180 - |$ refractive astigmatic axis $-$ corneal astigmatic axis $|$. Anterior chamber depth was not analyzed in eyes with a history of cataract surgery or peripheral iridotomy or in cases with measurements taken after pharmacologic mydriasis.

Statistical Analysis

All data were analyzed using SPSS version 20.0 software (SPSS, Inc., Chicago, IL). Results are expressed as means $\pm$ SD. The Kolmogorov-Smirnov test was used to verify distribution normality for continuous variables. To determine statistically significant differences between groups, we used *t*-tests or paired *t*-tests for normally distributed continuous variables. Wilcoxon signed-rank test or Mann-Whitney *U* test was used for non-normally distributed continuous variables, and Fisher's exact test was used for categorical variables. Linear correlations were analyzed with Pearson's correlation coefficient (r) for normally distributed continuous variables. Multiple linear regression analysis was used to evaluate correlations among differences between ACD and related variables. P values less than 0.05 were considered statistically significant.

Results

Patient Characteristics

The study included 52 patients with a mean age of 46.5 $\pm$ 7.51 years with 38 (73.1%) men and 14 (26.9%) women. Of the affected eyes, 29 (55.8%) were on the right side and 23 (44.2%) were on the left side. The mean time from onset of subjective symptoms to the initial visit was 8.3 $\pm$ 12.29 weeks. The most common angiographic leakage pattern among the 52 eyes with CSC was the expansile dot pattern in 37 eyes (71.2%), with 13 eyes (25.0%) with diffuse pattern and two eyes (3.8%) with smokestack pattern. Pigment epithelial detachments (PEDs) were observed in 19 cases (36.5%). Age was not significantly correlated with differences in ACD ($P = 0.833$), AL ($P = 0.899$), or spherical equivalent between the two eyes ($P = 0.237$).

Biometric Measurements of CSC Eyes Versus Fellow Eyes

Axial length was shorter in CSC eyes than in fellow eyes by 0.24 $\pm$ 0.379 mm ($P < 0.001$) (Table 1). In addition, ACD was shallower in CSC eyes than in fellow eyes by 0.03 $\pm$ 0.088 mm ($P = 0.021$). CSC eyes also had thicker CRTs and CTs than fellow eyes ($P < 0.001$ and $P < 0.001$, respectively).

The difference in ACD between eyes correlated with AL differences ($r = 0.297$, $P = 0.047$) (Fig. 1) but not with CRT differences ($P = 0.158$), CT differences ($P = 0.271$), or patient ages ($P = 0.833$). The mean differences between ACD in CSC eyes and those in fellow eyes were not significantly different between groups of patients categorized as younger or older

Table 1. Measurements of Eyes With Central Serous Chorioretinopathy and Fellow Eyes

Measurement	No. of Patients	CSC Eyes	Fellow Eyes	*P* Value‡
BCVA, logMAR	52	0.16 ± 0.168	0.01 ± 0.029	<0.001
Snellen equivalent*		20/29	20/20	
IOP, mm Hg	51	15.6 ± 2.94	15.5 ± 3.07	0.728
Spherical equivalent†, D	47	−1.40 ± 2.560	−1.30 ± 2.767	0.443
Refractive astigmatism, D	47	−0.62 ± 0.655	−0.79 ± 1.136	0.163
Mean K value, D	52	44.10 ± 1.270	44.01 ± 1.235	0.069
Horizontal K value, D	52	43.76 ± 1.293	43.65 ± 1.226	0.088
Vertical K value, D	52	44.44 ± 1.477	44.38 ± 1.429	0.342
Corneal astigmatism, D	52	−0.88 ± 0.797	−1.01 ± 0.775	0.126
Axis difference between refractive and corneal astigmatism	32	32.2° ± 27.98°	31.3° ± 30.53°	0.797
AL, mm	52	23.76 ± 1.343	24.00 ± 1.349	<0.001
ACD, mm	45	3.47 ± 0.365	3.50 ± 0.348	0.021
CRT, μm	52	413.1 ± 129.14	231.2 ± 26.43	<0.001
Subfoveal CT, μm	52	298.4 ± 58.67	264.4 ± 52.10	<0.001

Table values are means ± SD. LogMAR, logarithm of the minimum angle of resolution.
* Snellen equivalent was calculated from the mean of logMAR BCVA.
† Spherical equivalent = sphere + cylinder/2.
‡ *P* values were calculated using paired *t*-tests.

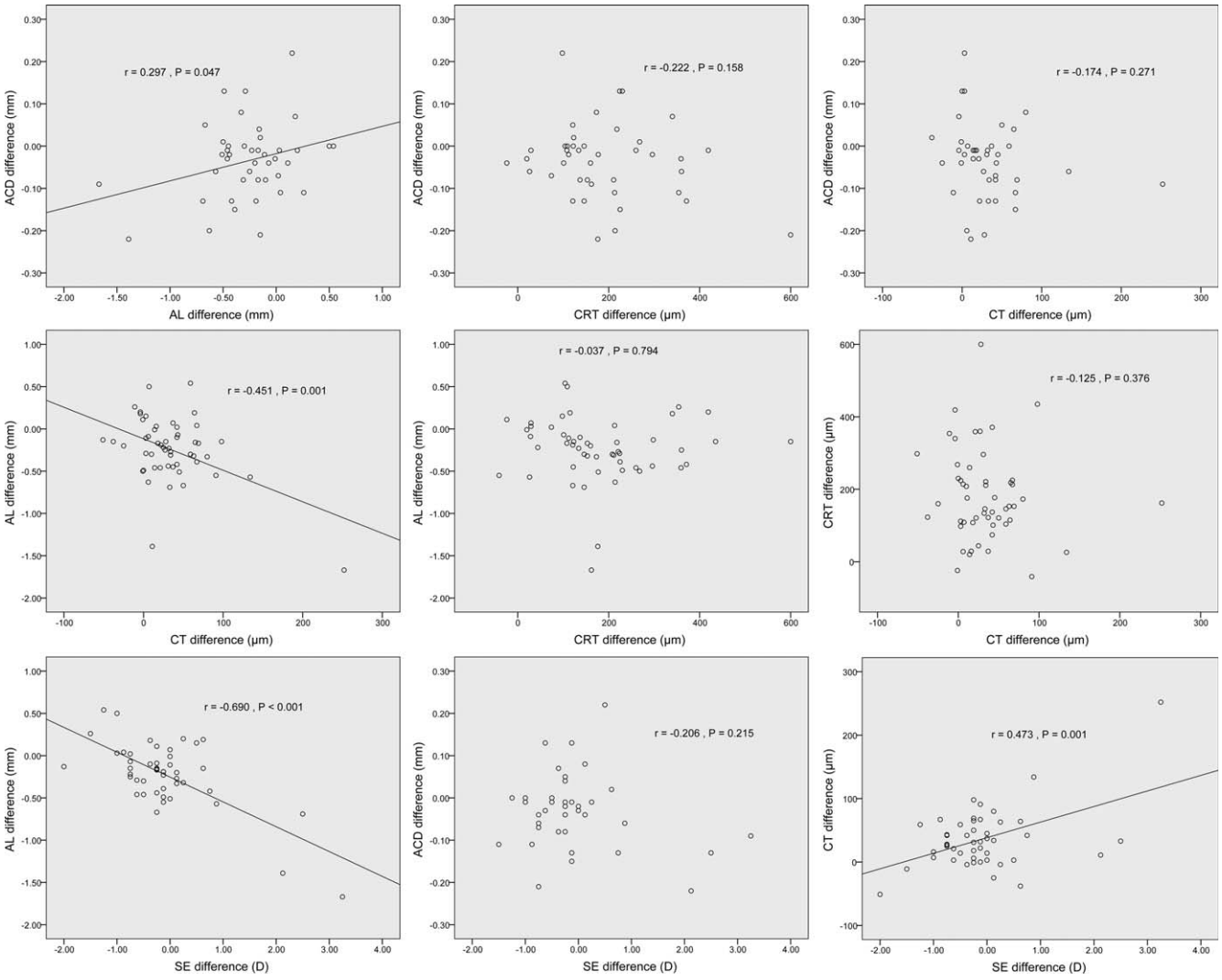

Figure 1. Scatterplots of differences in ACD, AL, CRT, subfoveal CT, and spherical equivalent between eyes with central serous chorioretinopathy and fellow eyes. Correlation coefficients (*r*) and *P* values are shown.

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than 45 years of age ($P = 0.809$). Axial length differences correlated with CT differences ($P = 0.001$) but not with CRT differences ($P = 0.794$). Variations in AL were not significantly different between groups of patients with and those without PED ($P = 0.126$).

Biometric Measurements and Refractive Error

Best-corrected visual acuity was worse in CSC eyes than in fellow eyes ($P < 0.001$). The mean spherical equivalent of CSC eyes was −1.40 ± 2.560 D. Mean refractive astigmatism was −0.62 ± 0.655 D. Table 1 summarizes comparisons between refractive errors in CSC eyes and those in fellow eyes. Corneal astigmatism correlated with refractive astigmatism in CSC eyes ($r = 0.549$, $P < 0.001$) and fellow eyes ($r = 0.667$, $P < 0.001$) (Fig. 2). Axis differences between refractive astigmatism and corneal astigmatism were not different between CSC and fellow eyes ($P = 0.797$). Differences between spherical equivalents in CSC eyes and those in fellow eyes correlated with AL differences ($r = -0.690$, $P < 0.001$) and CT differences ($r = 0.473$, $P = 0.001$) but not with ACD ($P = 0.215$) or CRT difference ($P = 0.348$) (Fig. 2). Differences in mean K values between eyes did not correlate with differences in spherical equivalents ($P = 0.363$), AL ($P = 0.347$), ACD ($P = 0.474$), CRT ($P = 0.303$), or CT ($P = 0.819$).

Biometry and Symptom Duration

Of all study patients, 44 (84.6%) experienced symptoms for 3 or less months. The mean age of this patient subset was 45.9 ± 7.34 years, and the mean time from onset of subjective symptoms to initial visit was 4.1 ± 3.63 weeks. Pigment epithelial detachments were observed in 16 CSC eyes (36.4%). Spherical equivalent refractive errors and mean K values were not different between CSC eyes and fellow eyes; however, AL was shorter in CSC eyes ($P < 0.001$) (Table 2). Anterior chamber depth was shallower in CSC eyes than in fellow eyes ($P = 0.001$), and the differences between ACD in CSC and those in fellow eyes correlated with AL differences ($r = 0.372$, $P = 0.026$; also see Supplementary Fig. S1).

Eight patients (15.4%) experienced symptom duration of more than 3 months. The mean age of this patient subset was 49.8 ± 8.08 years, and the mean time from symptom onset to initial visit was 31.0 ± 17.91 weeks. PEDs were observed in three eyes (37.5%). Axial length was shorter in CSC eyes than in fellow eyes ($P = 0.012$) (Table 2); however, the differences in ACD were not significant ($P = 0.159$).

Multiple Linear Regression Analysis

Multiple linear regression analysis of age, sex, symptom duration, and differences in AL, CRT, and CT between CSC eyes and fellow eyes showed that differences in ACD (mm) positively correlated with AL differences (mm; $R^2 = 0.113$, $P = 0.032$) and symptom duration (weeks; $R^2 = 0.133$, $P = 0.019$). Parameter estimates of all other tested variables showed no statistical significance. A stepwise method was used, and multiple R^2 values for the model were 0.210.

Discussion

We compared biometric characteristics of CSC eyes with those of fellow eyes. Refractive error and corneal power were not different between eyes; however, mean AL was shorter in CSC eyes than in fellow eyes. Differences in AL between eyes were significantly correlated with differences in subfoveal CT. This finding suggests that changes in AL in CSC eyes are closely related to subfoveal CT. Whereas ultrasonographic biometry uses reflection from the internal limiting membrane to measure AL, PCI uses signals from the RPE, calculating AL after adjusting for the normal retinal thickness of healthy eyes.[25] Partial coherence interferometry might detect anteriorly shifted RPE layers because of increased subfoveal CT in CSC eyes.[14–17] Kim et al.[17] and Jirarattanasopa et al.[15] reported greater subfoveal CT in eyes with unilateral CSC than in fellow eyes by approximately 67 μm and 126 μm, respectively. Thus, subfoveal choroidal thickening might push the RPE layer inward, and anterior shifting of the RPE might affect AL measurements in CSC eyes. Chakraborty et al.[26] demonstrated that diurnal variation in AL was negatively associated with CT. Increases in the distance between photoreceptors caused by subretinal fluid is suggested to be the mechanism causing micropsia in eyes with CSC.[8–11] Associated metamorphopsia might occur if photoreceptor separation is irregular.[8] Hypermetropization is also explained by anterior shifting of the retina, caused by subretinal fluid in CSC.[3] Transient hyperopic shifting is restored, it has been suggested, after resolution of

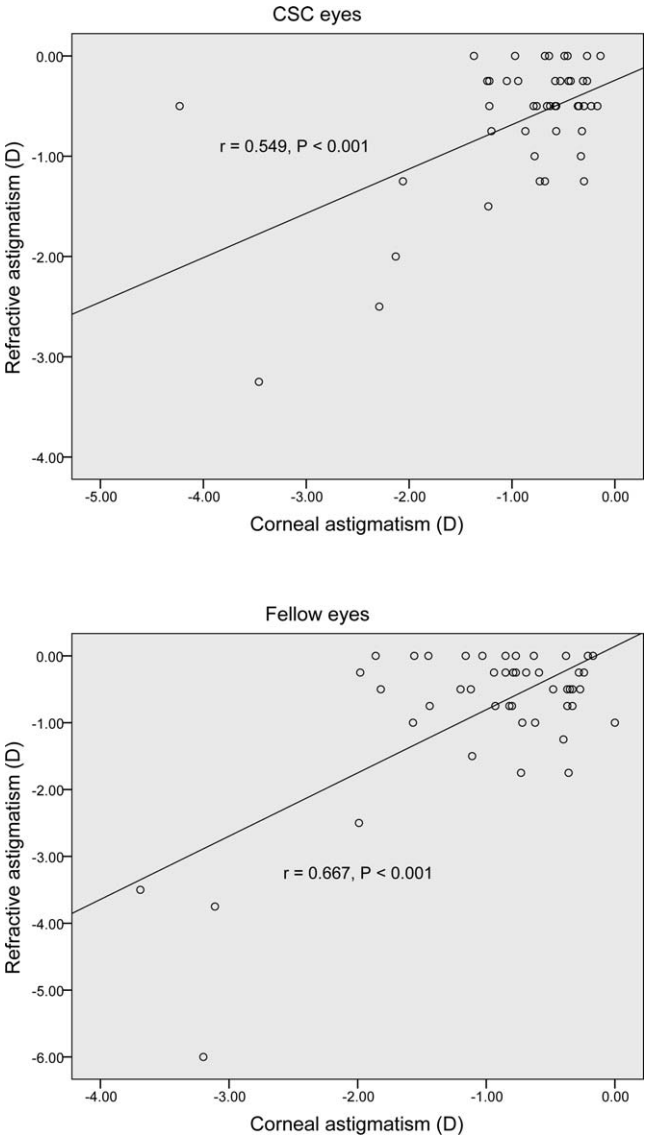

Figure 2. Scatterplots of corneal astigmatism and refractive astigmatism in eyes with CSC and fellow eyes. Correlation coefficients (r) and P values are shown.

Table 2. Measurements of Eyes With Central Serous Chorioretinopathy Versus Fellow Eyes Classified by Symptom Duration Greater Than or Less Than 3 Months

Measurement	Acute Symptoms ≤3 Months				Long-Lasting Symptoms >3 Months			
	n	CSC Eyes	Fellow Eyes	*P* Value	*n*	CSC Eyes	Fellow Eyes	*P* Value‡
BCVA, logMAR	44	0.14 ± 0.162	0.01 ± 0.022	<0.001	8	0.24 ± 0.188	0.02 ± 0.055	0.035
Snellen equivalent*		20/28	20/20			20/35	20/21	
IOP, mm Hg	43	15.6 ± 3.19	15.5 ± 3.30	0.829	8	15.4 ± 0.92	15.1 ± 1.46	0.739
Spherical equivalent†, D	40	−1.68 ± 2.630	−1.64 ± 2.825	0.803	7	0.16 ± 1.395	0.64 ± 1.294	0.027
Refractive astigmatism, D	40	−0.63 ± 0.686	−0.79 ± 1.211	0.241	7	−0.61 ± 0.476	−0.79 ± 0.603	0.059
Mean K value, D	44	43.97 ± 1.283	43.90 ± 1.285	0.147	8	44.89 ± 0.885	44.70 ± 0.536	0.327
Horizontal K value, D	44	43.60 ± 1.204	43.53 ± 1.235	0.254	8	44.69 ± 1.487	44.35 ± 0.953	0.123
Vertical K value, D	44	44.33 ± 1.536	44.26 ± 1.487	0.381	8	45.10 ± 0.860	45.04 ± 0.787	0.611
Corneal astigmatism, D	44	−0.90 ± 0.837	−1.04 ± 0.822	0.095	8	−0.83 ± 0.561	−0.80 ± 0.416	0.889
Axis difference between refractive and corneal astigmatism	26	33.3° ± 29.75°	28.3° ± 28.95°	0.119	6	27.5° ± 19.89°	44.3° ± 36.55°	0.225
AL, mm	44	23.9 ± 1.37	24.1 ± 1.39	<0.001	8	23.0 ± 0.83	23.3 ± 0.81	0.012
ACD, mm	37	3.49 ± 0.383	3.54 ± 0.361	0.001	8	3.37 ± 0.261	3.33 ± 0.227	0.159
CRT, μm	44	418.5 ± 129.78	233.6 ± 27.01	<0.001	8	383.5 ± 129.83	217.9 ± 19.24	0.025
Subfoveal CT, μm	44	308.1 ± 55.39	271.1 ± 48.55	<0.001	8	245.1 ± 48.89	227.8 ± 59.05	0.176

Results are means ± SD.

* Snellen equivalent was calculated from the mean of BCVA logMAR.

† Spherical equivalent = sphere + cylinder/2.

‡ *P* values were calculated using Wilcoxon signed-rank tests.

subretinal fluid.[27–29] However, we found that differences between spherical equivalent in CSC eyes and those in fellow eyes correlated well with AL differences or CT differences but not with CRT. This finding suggests that AL changes related to choroidal thickening might explain transient changes in refractive error.

We demonstrated that mean ACD was significantly shallower in CSC eyes than in fellow eyes, although the reasons for this difference are unclear. Choroidal hyperpermeability is considered influential in CSC pathogenesis.[7] Prünte and Flammer[30] suggested that capillary or venous congestion might result in choroidal hyperpermeability associated with CSC. Although venous return from both the ciliary body and the choroid occurs via the vortex veins,[21] choroidal venous congestion in CSC eyes might be associated with ciliary body congestion and edema. Consequently, ciliary body edema would cause a forward shift of the lens or an increase in lens thickness and a shallow anterior chamber.[31,32] We found that the differences between ACD in CSC eyes and those in fellow eyes correlated with AL differences. Capillary or venous congestion of the choroid might induce increased subfoveal CT and ciliary body edema, resulting in a short AL and shallow ACD in CSC eyes. Future studies using ultrasonographic biomicroscopy will be helpful in evaluating ciliary body thickness and understanding CSC pathogenesis.[33,34] Anterior chamber depth thinning might also be attributed to a forward shift of the lens that results from an anterior shift in the vitreous body after serous detachment of the neurosensory retina or RPE at the posterior pole.[35,36] However, the degree of ACD differences between eyes did not correlate with CRT differences in our analysis. Also, previous studies found that ACD was not affected by intravitreal injection of ranibizumab, 0.05 mL, and a decrease in ACD 5 minutes after intravitreal injection of triamcinolone acetonide, 0.1 mL, normalized within 45 minutes.[37,38] Anterior chamber depth evaluation in other retinal diseases with serous retinal detachment and without ocular inflammation, such as age-related macular degeneration and polypoidal choroidal vasculopathy, would be helpful for understanding the origin of shallow ACD in CSC eyes. Another possibility for the cause of shallow ACD in CSC eyes is that ACD thinning might result from accommodation in eyes with CSC. Although we did not find a significant difference in refractive error between eyes, using an automated refractor, hyperopic shift is a common finding that corresponds to serous foveal detachment in CSC eyes.[39] Hyperopic shifts might result in accommodation during PCI measurement. Accommodation with increased lens thickness and forward shifting of the anterior lens surface could result in a shallower anterior chamber in CSC eyes.[40–44] Notably, however, the mean ACD differences between eyes of younger patients (<45 years old) were not significantly different from those observed in older patients (≥45 years), who had a diminished ability to accommodate.

On multiple linear regression analysis, the differences in ACD between both eyes positively correlated with symptom duration. We currently do not have a clear explanation for these results. In connection with the hypotheses mentioned above, increased collateral drainage of the ciliary body into the intrascleral venous plexus and the episcleral veins or remodeling of the anteriorly shifted vitreous body might develop in patients with a longer symptom duration. In addition, reduced accommodation due to impaired vision and a shallower serous retinal detachment in eyes with longer symptom duration might contribute to the positive correlation between differences in ACD and symptom duration; however, our study was limited by a small sample size, and further investigation in a larger patient population is needed.

In addition to our retrospective study design and small number of patients, this study was cross-sectional; therefore, longitudinal changes in AL and ACD measurements with respect to disease course should be investigated in future studies. Comparing biometric measurements of nonsymptomatic fellow eyes with those of normal control eyes would also be interesting. Central serous chorioretinopathy involves both eyes in approximately 40% of cases.[1] In a previous study, Kim et al.[17] demonstrated that subfoveal CT was significantly increased in unaffected fellow eyes compared with that in normal control eyes. Although PCI uses signals from the RPE, PCI might detect signals from the detached retina rather than from the RPE.[45] However, Ueda et al.[23] reported that measurements of AL using PCI were 0.27 mm longer than measurements using ultrasonography in eyes with macular edema; this difference was positively correlated with macular

thickness. Further prospective studies are needed to compare ALs measured using PCI and ultrasonographic biometry in CSC eyes, as well as longitudinal studies of AL in eyes with resolved CSC. We did not find that difference in AL between eyes correlated with CRT differences. Additionally, PEDs were apparent in 36.5% of CSC eyes in our study. In some cases, particularly those with PED in the foveal center, PCI might detect the signal from the anterior surface of the PED. Another possible explanation is misalignment of the measurement axis due to poor fixation in CSC eyes. Impaired vision might make it difficult for patients with CSC to obtain good fixation, causing the PCI to measure an off-foveal AL.[23,46] Incorrect alignment would results in an underestimation of eye length.[47] Lastly, CSC might occur more frequently in eyes with a shorter AL. However, since our study was cross-sectional, we cannot draw definitive conclusions from our data.

In conclusion, biometric characteristics such as AL and ACD were different between eyes with CSC and fellow eyes. Variations in biometry, which correlated with CT differences, might be related to differences in refractive error between eyes. This study is the first to demonstrate a difference between ACD in CSC eyes and that in fellow eyes, particularly in early stage disease. These findings could be helpful in understanding idiopathic CSC.

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