·Clinical Research ·

Axial length in unilateral idiopathic central serous chorioretinopathy

Hoseok Moon, Dae Yeong Lee, Dong Heun Nam

Department of Ophthalmology, Gachon University Gil Hospital, Incheon 405-760, Korea

Correspondence to: Dong Heun Nam. Department of Ophthalmology, Gachon University Gil Hospital 1198, Kuwol-dong, Namdong-ku, Incheon 405-760, Korea. eyedawns@gilhospital.com

Received: 2014-12-08 Accepted: 2015-03-17

Abstract

• AIM: To evaluate the axial length (AXL) in unilateral idiopathic central serous chorioretinopathy (CSC).

• METHODS: This retrospective case-control study was comprised of a consecutive case series of 35 patients with acute unilateral idiopathic CSC, and age- and sexmatched 50 control eyes. AXL of both eyes of unilateral CSC patients and the control eyes were investigated. AXL was measured by ultrasonic biometry, and the adjusted AXL was calculated for CSC eyes as measured AXL plus differences of foveal thickness between CSC and normal fellow eyes in millimeters. The main outcome measures were comparison of AXL between CSC, fellow and control eyes.

• RESULTS: The mean age of 35 CSC patients was 45.5y, and 31 males were included. The adjusted AXL of CSC eyes was 23.52 mm, and the AXL of fellow eyes was 23.46 mm, and of control eyes 23.94 mm. The AXL of both CSC and fellow eyes were significantly shorter than control eyes (CSC νs control, P = 0.044; fellow νs control, P = 0.026). There was no statistically significant difference in AXL between CSC and fellow eyes.

• CONCLUSION: In unilateral idiopathic CSC, the AXL of CSC and fellow eyes are shorter than that of control eyes. Short AXL may be related with choroidal circulation abnormality in CSC.

• **KEYWORDS:** axial length; central serous chorioretinopathy;

pathophysiology; choroidal circulation **DOI:10.18240/ijo.2016.05.14**

Moon H, Lee DY, Nam DH. Axial length in unilateral idiopathic central serous chorioretinopathy. *Int J Ophthalmol* 2016;9(5):717-720

INTRODUCTION

 $T \begin{array}{l} \mbox{he pathophysiology underlying central serous} \\ \mbox{chorioretinopathy (CSC) remains unclear, but current} \\ \mbox{understanding focuses on abnormal choroidal circulation.} \\ \mbox{Indocyanine green angiography (ICGA) showed a delayed} \end{array}$

filling of the choroidal arteries and choriocapillaris, choroidal venous dilatation, and increased permeability of the choriocapillaries ^[1-7]. Choroidal vascular hyperpermeability is thought to be a primary pathology, possibly as a result of stasis, ischemia, or inflammation of choroid^[8].

Thickened choroid may be an evidence supporting abnormal choroidal circulation in CSC. Recent enhanced depth imaging spectral-domain optical coherence tomography (EDI-OCT) studies showed thickening of choroid in CSC^[9-11]. Choroidal hyperpermeability through fluid accumulation and the choroidal vascular dilatation is thought to be a cause of the choroidal thickening, but the exact underlying cause of choroidal thickening is not determined. Furthermore, some reports found that the choroidal thickness did not reached normal levels in resolved CSC^[12-13]. As thickened choroid is seen in the eye with short axial length (AXL)^[14], short AXL could play a partial role for choroidal thickening in CSC.

However, some research suggests that the AXL of an eye with CSC may not be short. A recent study of resolved and treated CSC showed a significant decrease in the choroidal thickness after photodynamic therapy or spontaneous resolution, reaching normal levels in some cases ^[12]. Another consideration is refractive errors in CSC. CSC is known to be typically seen in mild hyperopic and emmetropic eyes^[15]. Recent two Korean studies also reported a refractive error of -0.50 to -0.60 diopters in CSC ^[11,16]. These findings suggest that the AXL of an eye with CSC is uncertain and hard to predict.

To the best of our knowledge, there has been no report about AXL in idiopathic CSC. Herein, we investigated the AXL in acute unilateral idiopathic CSC.

SUBJECTS AND METHODS

This retrospective case-control study followed the tenets of the Declaration of Helsinki, and approved by Institutional Review Board of Gachon University Gil Hospital. All patients enrolled in the study were made to sign an informed consent.

A retrospective chart review of consecutive patients diagnosed with acute unilateral idiopathic CSC between August 2009 and August 2012 at Gachon University Gil Hospital was performed. CSC was diagnosed if eyes presented with subretinal fluid in the macula associated with one or a few leaks from the retinal pigment epithelium seen in fluorescein angiography. Acute CSC was defined as the duration of symptom within 3mo. Unilateral CSC was defined as unilateral manifestation of CSC at presentation, with the normal contralateral unaffected eye showing no

Axial length in central serous chorioretinopathy

able 1 Baseline characteristics of CSC and control groups							
Characteristics	CSC group	Control group	Р				
No. of eyes	35	50	NA				
Mean age (a, range)	45.5±6.5 (34-55)	46.6±4.3 (35-55)	0.376 ^a				
Gender (M:F)	31:4	44:6	1.000 ^b				
Laterality (R:L)	15:20	25:25	0.659 ^b				
History of hypertension, <i>n</i> (%)	4 (11)	6 (12)	1.000 ^b				
History of diabetes mellitus, <i>n</i> (%)	2 (6)	4 (8)	1.000^{b}				

CSC: Central serous chorioretinopathy; NA: Not applicable. ^at-test; ^bFisher's exact test.

changes associated with CSC. Exclusion criteria were: 1) patients with systemic steroid use, pregnancy, Cushing's syndrome, end stage renal disease, collagen vascular disease, obstructive sleep apnea, Helicobacter pylori infection, or organ transplantation; 2) patients older than 55 years old; 3) spherical equivalent outside of the range from +6.0 to -6.0 diopters; 4) AXL outside of the range from 20.0 to 26.0 mm; 5) patients with previous ocular surgery damaged the cornea or sclera, for example, laser refractive surgery, cataract surgery, or vitrectomy; 6) patients with any vitreoretinal disease, for example, age-related macular degeneration, diabetic retinopathy, or retinal vein occlusion. Psychosomatic factors such as type A personality or emotional stress, and behavioral factors such as smoking or alcohol use were not considered in this study.

Medical record information, including age, sex, laterality of eyes, history of hypertension and diabetes mellitus, and ocular biometrics such as AXL, spherical equivalent, foveal thickness, radius of corneal curvature, and keratometry reading, was obtained. Ocular biometric examinations were performed at the time of manifestation of CSC at presentation. Both eyes of CSC patients, CSC eyes and fellow eyes, were examined.

AXL was measured using a 10-MHz A/B mode ultrasonography device (Cine Scan, Quantel Medical, Clermont-Ferrand, France) by one optometric specialist with an applanation technique that measured AXL from the corneal vertex to the vitreoretinal interface. A minimum of 10 AXL recordings were made for each eye and the mean calculated. Because the end point of AXL is vitreoretinal surface in A-scan ultrasound method, the AXL of a CSC eve, which presents subfoveal fluid and anterior shifting of vitreoretinal surface, can potentially be measured shorter than that without subfoveal fluid. So, we converted measured AXL to "adjusted" AXL for CSC eyes. The adjusted AXL was calculated using the following formula: adjusted AXL= (measured AXL in millimeters)+ (differences of forveal thickness between CSC and normal fellow eyes in millimeters).

Objective refraction without cycloplegia was measured by an autokeratorefractometer (NIDEK ARK-510A, Nidek Co., Ltd., Gamagori, Japan). Subjective refraction was finally determined by one trained optometrist, with objective refraction values as the starting point. The spherical equivalent refraction (SER) was calculated with the spherical dioptric power plus half the cylindrical dioptric power from the subjective refraction values measured in diopters (D). Foveal thickness was measured using the fast macular scan of Stratus OCT3 version 4.0 software (Carl Zeiss Meditec, Dublin, California, USA). Radius of corneal curvature and keratometry reading were determined as the mean of three consecutive measures using an autokeratorefractometer.

The control group consisted of randomly selected patients undergoing cataract surgery without any signs associated with CSC, and matched for age and gender. Exclusion criteria listed above was applied equally to the control group. Additional exclusion criteria of control group were patients with traumatic cataract, toxic cataract, or cataract related to known systemic or genetic diseases.

The data were analyzed using \prime -test, paired \prime -test and Fisher's exact test. Comparisons of ocular biometrics between CSC, fellow, and control eyes were performed. The statistical analyses were performed with SPSS software version 12.0 (SPSS Inc., Chicago, IL, USA), and P<0.05 was considered statistically significant.

RESULTS

The CSC group with unilateral idiopathic CSC consisted of 35 patients (31 men) with a mean age of 45.5 years old (range 34-55y), and both eyes were phakic in all patients without cataract. The control group undergoing cataract surgery included 50 patients (44 men) with a mean age of 46.6 years old (range 35-55y). No statistically significant difference of the baseline characteristics, including age, gender, laterality of eyes, and history of hypertension or diabetes mellitus, was noted between study and control group (Table 1).

The adjusted AXL of CSC eyes was 23.52 mm, and the AXL of fellow eyes was 23.46 mm and of control eyes 23.94 mm. The AXL of both CSC and fellow eyes were significantly shorter than that of control eyes (CSC νs control, P=0.044, and fellow νs control, P=0.026), and no difference in AXL between CSC and fellow eyes was detected (Table 2, Figure 1). Spherical equivalent, corneal radius and keratometry did not differ among the three groups (Table 2).

DISCUSSION

The most common form of CSC is idiopathic, although many conditions, such as systemic steroid or symphatomimetics use, pregnancy, Cushing's syndrome, collagen vascular Int J Ophthalmol, Vol. 9, No. 5, May 18, 2016 www. ijo. cn Tel:8629-82245172 8629-82210956 Email: jopress @163.com

Biometrics	CSC eyes	Fellow eyes	Control eyes	Р		
				CSC vs control ^b	Fellow vs control ^b	CSC vs fellow ^c
Spherical equivalent (D)	-0.09 ± 0.82	-0.24 ± 0.77	-0.48 ± 1.40	0.142	0.371	0.412
Foveal thickness (µm)	376±121	196±25	201±26	< 0.001	0.381	< 0.0001
Axial length (mm)	$23.52{\pm}0.84^a$	23.46±0.84	23.94±1.05	0.044	0.026	0.757
Corneal radius (mm)	7.77±0.22	7.77±0.21	7.71±0.27	0.341	0.357	0.974
Keratometry (D)	43.51±1.31	43.50±1.29	43.80±1.56	0.371	0.365	0.995

his matrice between CSC follow and control aver

CSC: Central serous chorioretinopathy. ^aAdjusted axial length for CSC eyes; ^bt-test; ^cpaired t-test.

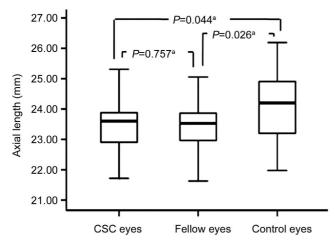


Figure 1 Comparison of AXL between CSC, fellow and control eyes The *t*-test revealed statistically significant differences in AXL between CSC and control eyes and between fellow and control eyes, but not between CSC and fellow eyes.ªt-test.

diseases, obstructive sleep apnoea, antibiotic use, alcohol use, allergic respiratory disease, and untreated hypertension, were determined as risk factors of CSC^[17]. The subject of this study was idiopathic CSC, and the AXL was shorter in CSC eyes than control eyes. This suggests that shorter AXL could be an underlying condition of idiopathic CSC.

We suppose two possible mechanisms that short AXL affects the abnormal choroidal circulation and development of CSC. The first is increased resistance of choroidal venous outflow. The choroidal venous drainage pathway from choroidal vessels to superior and inferior ophthalmic veins consists of the vortex veins which penetrate the sclera. In the eye with short AXL, the sclera is thick and rigid, and the scleral flexibility may decrease. The resistance of the vortex veins passing the rigid sclera can increase, resulting in decreased choroidal venous outflow and abnormal choroidal circulation. The second is decreased trans-scleral outflow. Because the sclera of the eye with short AXL is thick and the scleral tissues are compact, the scleral thickening may decrease the transscleral fluid outflow associated with the stasis of choroidal circulation. Increased resistance of choroidal venous outflow and decreased transscleral outflow can result in increased choroidal vascular pressure, and increased stress on vessel wall may induce the choroidal vascular hyperpermeability. Furthermore, decreased choroidal outflow can result in decreased perfusion pressure

of the choroid, and resultant decrease in choroidal blood flow may induce ischemia and inflammation of choriocapillaries and RPE disruption, and partially affect poor retinal cooling, making the RPE vulnerable to oxidative stress

Bilateral involvement of CSC has been reported to occur in up to 40% of cases [7]. Choroidal abnormalities of the contralateral unaffected eye in unilateral CSC have been noted. Choroidal vascular hyperpermeability in the unaffected fellow eye of unilateral CSC has been reported in ICGA studies [3,5-7,10,18-19]. Recent EDI-OCT studies noted increased choroidal thickness in the unaffected fellow eye of unilateral CSC [9-10]. In the current study, the AXL of the fellow eyes was shorter than the control eyes. Short AXL as an underlying condition of the unaffected fellow eye is a new finding, supporting choroidal abnormalities in the clinically unaffected fellow eye in unilateral CSC.

To evaluate AXL as an underlying condition of CSC, ultrasonic measurement of AXL after resolution of acute CSC or before development of CSC would be more informative than that in acute CSC, but it is difficult in the usual clinical settings. In this study, "adjusted" AXL was used for acute CSC eyes, because ultrasonically measured AXL might have an error in acute CSC with serous retinal detachment. However, an adjusted AXL, calculated from measured AXL, should be regarded as "presumed true" AXL which may be the AXL after resolution of acute CSC or before the development of CSC. In addition, the AXL of the clinically unaffected fellow eye could be an alternative for the AXL in acute unilateral CSC. In acute CSC with anterior shifting of vitreoretinal surface, partial coherence laser interferometry may be more precise because this device measures AXL through detecting the retinal pigment epithelial (RPE) layer. However, in acute CSC with retinal pigment epithelial detachment (PED), PED should be assessed and the AXL may need to be adjusted in partial coherence laser interferometry as in ultrasonic biometry of our study.

Our study has some considerations. First, our control group was not a normal population. The control eyes of current study were randomly selected patients undergoing cataract surgery, mean age of 46.6y, 88% males and mean axial of 23.94 mm. Because our control group was not a normal population, it is essential to compare the AXL of our control

Axial length in central serous chorioretinopathy

Fable 3 AXL of normal males aged 40-49y in east and southeast Asian population or cohort-based studies						
Ethnicity	Study	No. of eyes	Axial length (mm)	Methods		
Koreans	Healthy twin study ^[20]	133	24.14	Ultrasonic biometry		
Singaporean Malay	Singapore Malay eye study ^[21]	361	23.88	Partial coherence laser interferometry		
Singaporean Chinese	Tanjung Pagar survey ^[22]	120	23.80	Ultrasonic biometry		
Singaporean Indian	Singapore Indian eye study ^[23]	427	23.71	Partial coherence laser interferometry		

eyes with that of an age, sex and ethnicity matched normal population. Among the 50 control eyes of this study, 44 eyes were of males aged from 40 to 49y with a mean AXL of 23.98 mm. The AXL of normal males aged 40-49y in east or southeast Asian population- or cohort-based studies were 24.14 mm in one Korean study and from 23.71 to 23.88 mm in three Singaporean studies (Table 3)^[20-23]. Although the AXL of our study was similar to prior studies, further study is needed to compare the AXL of CSC eye with normal population. Second, the sample size of the subjects in current study was small. Although this study showed a statistically significant difference of P < 0.05 in AXL between study and control eyes, a sample size of 35 study eyes and 50 control eyes achieved an 53.2% statistical power of a two-sided *t*-test. Regarding this study as a reference, further large study should verify the differences in the AXL between CSC and normal eyes. Third, because the subjects of this study were Koreans, ethnic variations of AXL should be considered. Fourth, as this study showed shorter AXL in CSC eye, the relation between the AXL and choroidal thickness by EDI-OCT or choroidal circulation abnormality by ICGA would be interesting.

In conclusion, in unilateral idiopathic CSC, the AXL of CSC and fellow eyes was shorter than that of control eyes. Short AXL may be related with choroidal circulation abnormality in CSC.

ACKNOWLEDGEMENTS

Conflicts of Interest: Moon H, None; Lee DY, None; Nam DH, None.

REFERENCES

1 Scheider A, Nasemann JE, Lund OE. Fluorescein and indocyanine green angiographies of central serous choroidopathy by scanning laser ophthalmoscopy. *Am.J Ophthalmol* 1993;115(1):50-56.

2 Guyer DR, Yannuzzi LA, Slakter JS, Sorenson JA, Ho A, Orlock D. Digital indocyaninegreen videoangiography of central serous chorioretinopathy. *Arch Ophthalmol* 1994;112(8):1057-1062.

3 Piccolino FC, Borgia L. Central serous chorioretinopathy and indocyanine green angiography. *Retina* 1994;14(3):231-242.

4 Prunte C. Indocyanine green angiographic findings in central serous chorioretinopathy. *Int Ophthalmol* 1995;19(2):77-82.

5 Menchini U, Virgili G, Lanzetta P, Ferrari E. Indocyanine green angiography in central serous chorioretinopathy. ICG angiography in CSC. *Int Ophthalmol* 1997;21(2):57–69.

6 Spaide RF, Hall L, Haas A, Campeas L, Yannuzzi LA, Fisher YL, Guyer DR, Slakter JS, Sorenson JA, Orlock DA. Indocyanine green videoangiography of older patients with central serous chorioretinopathy. *Retina* 1996;16(3):203–213.

7 Iida T, Kishi S, Hagimura N, Shimizu K. Persistent and bilateral choroidal vascular abnormalities in central serous chorioretinopathy. *Retina*

1999;19(6):508-512.

8 Yannuzzi LA. Central serous chorioretinopathy. a personal perspective. *Am J Ophthalmol* 2010;149(3):361–363.

9 Imamura Y, Fujiwara T, Margolis R, Spaide RF. Enhanced depth imaging optical coherence tomography of the choroid in central serous chorioretinopathy. *Retina* 2009;29(10):1469-1473.

10 Maruko I, Iida T, Sugano Y, Ojima A, Sekiryu T. Subfoveal choroidal thickness in fellow eyes of patients with central serous chorioretinopathy. *Retina* 2011;31(8):1603-1608.

11 Kim YT, Kang SW, Bai KH. Choroidal thickness in both eyes of patients with unilaterally active central serous chorioretinopathy. *Live (Lond)* 2011; 25(12):1635–1640.

12 Kang NH, Kim YT. Change in subfoveal choroidal thickness in central serous chorioretinopathy following spontaneous resolution and low-fluence photodynamic therapy. *Eye (Lond)* 2013;27(3):387–391.

13 Brandl C, Helbig H, Gamulescu MA. Choroidal thickness measurements during central serous chorioretinopathy treatment. *Int Ophthalmol* 2014;34 (1):7–13.

14 Li XQ, Larsen M, Munch IC. Subfoveal choroidal thickness in relation to sex and axial length in 93 Danish university students. *Invest Ophthalmol Vis Sci* 2011;52(11):8438-8441.

15 Yannuzzi LA, Gitter KA, Schatz H. Central serous chorioretinopathy. In Yannuzzi LA, Gitter KA, Schatz H, eds. *The macula: a comprehensive text and atlas Baltimore, MD.* Williams & Wilkins;1979:145–165.

16 Kim SW, Oh J, Kwon SS, Yoo J, Huh K. Comparison of choroidal thickness among patients with healthy eyes, early age-related maculopathy, neovascular age-related macular macular degeneration, central serous chorioretinopathy, and polypoidal choroidal vasculopathy than in control or age-realted maculopathy groups. *Retina* 2011;31(9):1904–1911.

17 Liew G, Quin G, Gillies M, Fraser-Bell S. Central serous chorioretinopathy: a review of epidemiology and pathophysiology. *Clin Experiment Ophthalmol* 2013;41(2):201-214.

18 Spaide RF, Campeas L, Haas A, Yannuzzi LA, Fisher YL, Guyer DR, Slakter JS, Sorenson JA, Orlock DA. Central serous chorioretinopathy in younger and older adults. *Ophthalmology* 1996;103(12):2070–2079.

19 Okushiba U, Takeda M. Study of choroidal vascular lesions in central serous chorioretinopathy using indocyanine green angiography. *Nippour Ganka Cakkai Zasshi* 1997;101(1):74–82.

20 Kim MH, Zhao D, Kim W, Lim DH, Song YM, Guallar E, Cho J, Sung J, Chung ES, Chung TY. Heritability of myopia and ocular biometrics in Koreans: the healthy twin study. *Invest Ophthalmol Vis Sci* 2013;54(5): 3644–3649.

21 Lim LS, Saw SM, Jeganathan VS, Tay WT, Aung T, Tong L, Mitchell P, Wong TY. Distribution and determinants of ocular biometric parameters in an Asian population: the Singapore Malay eye study. *Invest Ophthalmol Vis Sci* 2010;51(1):103–109.

22 Wong TY, Foster PJ, Ng TP, Tielsch JM, Johnson GJ, Seah SK. Variations in ocular biometry in an adult Chinese population in Singapore: the Tanjong Pagar Survey. *Invest Ophthalmol Vis Sci* 2001;42(1):73–80.

23 Pan CW, Wong TY, Chang L, Lin XY, Lavanya R, Zheng YF, Kok YO, Wu RY, Aung T, Saw SM. Ocular biometry in an urban Indian population: the Singapore Indian Eye Study (SINDI). *Invest Ophthalmol Vis Sci* 2011; 52(9):6636–6642.