



Scleral Thickness in Steroid-Induced Central Serous Chorioretinopathy

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Purpose: To evaluate and compare the scleral thickness of patients with idiopathic central serous chorioretinopathy (iCSC) and steroid-induced central serous chorioretinopathy (sCSC) using anterior-segment OCT.

Design: Retrospective, comparative study.

Participants: One hundred ten eyes of 110 patients with central serous chorioretinopathy.

Methods: We classified the patients into iCSC and sCSC groups and compared age, sex, spherical equivalent, axial length, subfoveal choroidal thickness (SCT), and scleral thickness. We measured scleral thickness 6 mm posterior to the scleral spur in 4 directions.

Main Outcome Measure: Scleral thickness in sCSC eyes.

Results: We enrolled 96 and 14 eyes in the iCSC and sCSC groups, respectively. The sCSC group included a greater proportion of women than the iCSC group (42.9% and 13.5%, respectively; $P = 0.020$). We observed no between-group differences in age, spherical equivalent, axial length, or SCT. Univariate analysis revealed that the sCSC group had a significantly thinner sclera at the superior ($423.4 \mu\text{m}$ vs. $346.6 \mu\text{m}$; $P < 0.001$), temporal ($440.1 \mu\text{m}$ vs. $399.4 \mu\text{m}$; $P = 0.020$), inferior ($450.1 \mu\text{m}$ vs. $395.3 \mu\text{m}$; $P = 0.001$), and nasal ($436.6 \mu\text{m}$ vs. $391.9 \mu\text{m}$; $P = 0.002$) points than the iCSC group. Multivariate analyses revealed that female sex (odds ratio, 4.322; 95% confidence interval, 1.025–18.224; $P = 0.046$) and mean scleral thickness (odds ratio, 0.972; 95% confidence interval, 0.955–0.990; $P = 0.002$) were significantly associated with sCSC.

Conclusions: The scleral thickness of eyes in the sCSC group was significantly thinner than that in the iCSC group. This suggests that the sclera has less involvement in the pathogenesis of sCSC than in that of iCSC. *Ophthalmology Science* 2022;2:100124 © 2022 by the American Academy of Ophthalmology. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

Central serous chorioretinopathy (CSC) is a pachychoroid spectrum disease characterized by serous retinal detachment, pigment epithelial detachment with choroidal vascular abnormalities, or both.^{1–8} Central serous chorioretinopathy most frequently affects men 30 to 60 years of age.^{9–11} When eyes with CSC demonstrate serous retinal detachment, pigment epithelial detachment of the central macula, or both, blurred or distorted vision occurs. Furthermore, the conditions have been shown to be exacerbated by stress and corticosteroid use.^{11–19}

To date, many reports have indicated that corticosteroid administration is a risk factor for CSC developing, a finding that has been confirmed via several case-control studies.^{12–17} Carvalho-Recchia et al¹⁴ conducted the first prospective case-control study that demonstrated the association between acute CSC and corticosteroid use. However, the reason why corticosteroid administration is a risk factor for CSC remains unclear.

Many studies have shown that the choroid in the macular region of eyes with CSC is thicker than that of healthy eyes.^{5–7} Therefore, choroidal thickening is considered to be an important clinical feature of CSC. Several studies have compared the choroidal thickness of eyes with steroid-induced CSC (sCSC) with that of eyes with idiopathic CSC (iCSC).^{20–22} However, no widely accepted consensus

regarding choroidal thickness in sCSC has been reached. For example, Honda et al²⁰ and Izumi et al²¹ reported that the choroidal thickness of patients with sCSC is thinner or similar to that of patients with iCSC, whereas Araki et al²² reported that the choroidal thickness in patients with sCSC is greater than that of patients with iCSC. Although some basic studies have suggested effects of corticosteroids on choroidal and retinal tissues,^{23–26} the precise mechanism of sCSC remains elusive.

Recent imaging studies revealed that choroidal abnormalities in CSC include choroidal vasodilation,^{1–4} choroidal vascular hyperpermeability,^{1–4} and choroidal thickening.^{5–7} Recent improvements in examination equipment have enabled researchers to obtain increasingly detailed information regarding choroidal status from the posterior pole to the vortex vein ampullas. Pang et al,²⁷ using ultra-widefield indocyanine green angiography (ICGA), reported that 83.3% of eyes with acute or chronic CSC showed dilated choroidal vessels near the vortex vein ampullas. Hiroe and Kishi²⁸ demonstrated the dilation of asymmetric vortex veins in eyes with all types of CSC using en face OCT. Taken together, these studies suggest the presence of choroidal congestion in eyes with iCSC. Therefore, choroidal circulatory disturbances are thought to be involved in the pathogenesis of CSC.

Recently, we reported that scleral thickness was greater in eyes with iCSC than in healthy control eyes based on anterior-segment (AS) OCT findings.²⁹ Findings suggested that scleral thickening may play an important role in choroidal circulatory disturbances in the pathogenesis of CSC.²⁹ However, in that study, we excluded patients with a history of systemic corticosteroid use and systemic conditions associated with CSC to avoid assessing patients with differing disease origins. In the current study, to better understand the nature of sCSC, we evaluated and compared the scleral thickness of patients with iCSC and sCSC using AS OCT. Moreover, we investigated clinical factors specifically related to sCSC.

Methods

This retrospective, comparative study was approved by the institutional review board of the University of the Ryukyus (approval no.: 1503) and was conducted in accordance with the tenets of the Declaration of Helsinki. We obtained informed consent from all study participants after a detailed explanation of the study protocol was provided. We retrospectively reviewed the medical records of 110 patients with a diagnosis of CSC, which was initially observed at the University of the Ryukyus Hospital between October of 2018 and October of 2020. Central serous chorioretinopathy diagnoses were based on clinical features that were assessed using multimodal imaging, as follows: (1) OCT detected subretinal fluid with or without pigment epithelial detachment, (2) fluorescein angiography detected focal or diffuse leakage through the retinal pigment epithelium level into the subretinal space, and (3) ICGA identified features indicative of choroidal circulatory disturbances such as filling delay during the early phase and choroidal vessel dilation and choroidal vascular hyperpermeability during the middle or late phase. When both eyes had CSC, we included only the right eye in the analysis.

We classified eyes into 2 groups, as follows: iCSC and sCSC. Use of corticosteroids at the onset of CSC symptoms indicated sCSC. The route of corticosteroid administration included oral, inhalation, topical, intranasal, and dermal application. We excluded patients in either group with a history of corticosteroid use, but no use at disease onset, from the study. Further, we excluded the patients in whom ICGA or OCT angiography detected choroidal neovascularization. The other exclusion criteria were as follows: (1) subretinal fluid resulting from other macular diseases, (2) uveitis, (3) glaucoma, (4) scleritis, (5) intraocular surgery performed within 6 months of CSC onset, or (6) a history of photodynamic therapy.

At the initial visit, all patients underwent comprehensive ophthalmologic examinations, including decimal best-corrected visual acuity testing with Landolt C charts (SC-1000; TOMÉY), automated refractometry or keratometry (ARK-1a; Nidek), axial length measurement (IOL Master 700; Carl Zeiss Meditec), slit-lamp biomicroscopy with or without a contact lens, indirect ophthalmoscopy, color fundus photography (TRC-50DX; Topcon), swept-source OCT (DRI-OCT Triton; Topcon), fundus autofluorescence photography, fluorescein angiography and ICGA (Spectralis HRA+OCT; Heidelberg Engineering), OCT angiography (DRI-OCT Triton between October 2018 and March 2019; Zeiss Plex Elite 9000 between April 2019 and October 2020 [Carl Zeiss Meditec]), and AS OCT (CASIA2; TOMÉY). We calculated the spherical equivalent as the principal spherical power plus the half cylinder power.

We measured subfoveal choroidal thickness (SCT) manually using a caliper function built in to swept-source OCT. We defined

SCT as the distance from the hyperreflective line corresponding to Bruch's membrane to the inner surface of the sclera at the foveal center. We measured scleral thickness via AS OCT using the method previously described (Fig 1).²⁹ Briefly, we identified the rectus muscles (superior, lateral, inferior, and medial rectus muscles) as low reflective bands by gazing in the direction opposite to each muscle. Using the raster scan mode of AS OCT, we obtained 16 B-scan images along each rectus muscle within a 16-mm diameter area. These images were 4-mm wide and included the middle of each rectus muscle. After capturing the images, we selected those that clearly crossed the middle of each rectus muscle. We then identified both anterior and posterior borders of the sclera. We defined the anterior scleral border as the line between the low reflective bands of the rectus muscles and the highly reflective area that corresponded to the sclera. We defined the posterior scleral border as the line between the highly reflective area that corresponded to the sclera and the choroid. We obtained scleral thickness measurements vertically at a point that was 6 mm posterior to the scleral spur, which was just beneath the low reflective bands of the rectus muscles. At this point, we measured scleral thickness as the distance between anterior and posterior scleral borders. We repeated the measurement 4 times in the following 4 different points: superior, temporal, inferior, and nasal.

Demographic characteristics such as age, sex, spherical equivalent, axial length, SCT, and scleral thickness of patients in the iCSC and sCSC groups were compared. We evaluated the sex ratio using the Fisher exact test. The other characteristics were analyzed using the Wilcoxon rank-sum test. Additionally, a multivariate analysis was performed to determine whether a correlation existed between sCSC incidence and age, sex, and mean scleral thickness in 4 directions. Statistical analyses were performed using Statistical Analysis System software version 9.4 (SAS, Inc). All values are expressed as the mean \pm standard deviation. *P* values of < 0.05 were considered statistically significant.

Results

In this study, we enrolled 96 eyes of 96 patients with iCSC and 14 eyes of 14 patients with sCSC. Table 1 describes the detailed information of 14 patients with sCSC, including administration routes of corticosteroids, background systemic conditions, and duration and approximate total dose of corticosteroid use based on the retrospective review of medical records.

Table 2 summarizes the demographic and clinical characteristics of the patients of both groups. No significant differences regarding age, spherical equivalent, or axial length were observed. The female preponderance in the sCSC group was significant (13.5% and 42.9% in the iCSC and sCSC groups, respectively; $P = 0.015$). The mean SCT of patients in each group did not differ ($391.5 \pm 96.5 \mu\text{m}$ and $450.9 \pm 139.4 \mu\text{m}$ in patients of the iCSC and sCSC groups, respectively; $P = 0.088$). Figure 2 summarizes the scleral thickness values at each point. Compared with the iCSC group, the sCSC group showed a significantly thinner scleral thickness at the superior ($423.4 \pm 58.9 \mu\text{m}$ vs. $346.6 \pm 68.7 \mu\text{m}$, $P < 0.001$), temporal ($440.1 \pm 51.3 \mu\text{m}$ vs. $399.4 \pm 48.3 \mu\text{m}$; $P = 0.018$), inferior ($450.1 \pm 51.5 \mu\text{m}$ vs. $395.3 \pm 55.5 \mu\text{m}$;

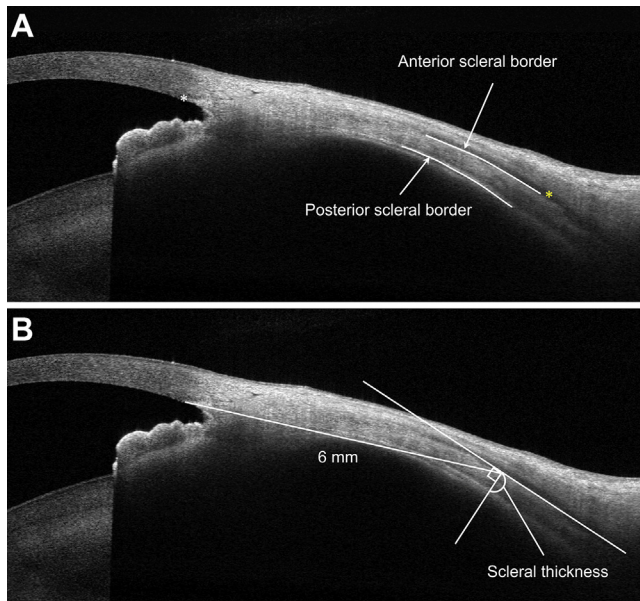


Figure 1. Scleral thickness measured using anterior-segment (AS) OCT. **A**, Scleral spur (white asterisk) and targeted rectus muscle (yellow asterisk) are shown. Rectus muscles produced low reflective bands on AS OCT. White tangential lines represent the anterior and posterior borders of the sclera. **B**, Scleral thickness was measured vertically at a point 6 mm posterior to the scleral spur, just beneath the low reflective band corresponding to the rectus muscle.

$P = 0.002$), and nasal ($436.6 \pm 54.1 \mu\text{m}$ vs. $391.9 \pm 37.3 \mu\text{m}$; $P = 0.001$) points.

Table 3 summarizes the results of the multivariate analysis of factors that contribute to the incidence of sCSC. Female sex (odds ratio, 4.322; 95% confidence

interval, 1.025–18.224; $P = 0.046$) and mean scleral thickness in 4 directions (odds ratio, 0.972; 95% confidence interval, 0.955–0.990; $P = 0.002$) were significantly associated with sCSC incidence. Representative patients from the iCSC and sCSC groups are shown in **Figure 3** and **4**, respectively.

Discussion

In the current study, we evaluated and compared the scleral thickness of patients with iCSC and sCSC. Furthermore, the study evaluated the effects of other clinical characteristics. Univariate analyses identified significant between-group differences in the female ratio and scleral thickness in all 4 directions. Patients with sCSC accounted for 12.7% (14/110 patients) of the study population, and the female ratio (42.9%) of the group was significantly higher than that of the iCSC group (13.5%; $P = 0.015$). Further, multivariate analysis also revealed a significant correlation between sCSC incidence and increased female ratio and decreased mean scleral thickness in 4 directions. These results suggest that scleral thickness likely contributes less significantly to the pathogenesis of sCSC than to that of iCSC.

Many risk factors for induction of CSC have been reported. For example, male sex,^{10,18} middle age,^{10,18} smoking,¹⁶ and short axial length^{29,30} are the representative risk factors. A recent systematic review and meta-analysis identified other risk factors, including systemic conditions and drugs.^{11,15,18,19} Corticosteroid use showed high odds ratios and was noted to be a definite trigger for CSC.^{12–17} In the current study population, the proportion of corticosteroid users was 12.7%. This proportion was within the previously reported range of 3.3% to

Table 1. Detailed Information for Each Patient with Steroid-Induced Central Serous Chorioretinopathy

| Patient No. | Sex | Administration Route | Background Condition | Onset of Background Condition (mo/yr) | Duration of Corticosteroid Use | Approximate Total Dose of Corticosteroids |
|-------------|-----|----------------------|---|---------------------------------------|--|---|
| 1 | F | Oral | SLE | Unknown/1994 | Approximately 25 yrs | 73 000 mg |
| 2 | F | Oral | SLE | Unknown/1980 | Approximately 30 yrs | 83 500 mg |
| 3 | F | Oral | SLE, a history of organ transplantation | Unknown/1988 | Unknown because of lack of medical records | Unknown |
| 4 | F | Oral | SLE | October/2011 | 7 yrs and 3 mos | 10 535 mg |
| 5 | M | Oral | SLE | Unknown/2002 | Approximately 17 yrs | 28 700 mg |
| 6 | M | Oral | Myasthenia gravis | March/2013 | 7 yrs and 6 mos | 27 375 mg |
| 7 | M | Oral | Pemphigus vulgaris | Unknown/2014 | Approximately 7 yrs | 29 010 mg |
| 8 | F | Oral | IgA nephritis, a history of organ transplantation | April/2014 | 5 yrs and 8 mos | 15 000 mg |
| 9 | M | Oral, inhalation | IgG4-related disease, asthma | August/2018 | 6 mos | 3300 mg |
| 10 | M | Oral | Subacute thyroiditis | December/2019 | 7 days | 240 mg |
| 11 | M | Oral | Nephrotic syndrome | January/2020 | 6 mos | 8490 mg |
| 12 | F | Inhalation | Asthma | Unknown | Unknown | Unknown |
| 13 | M | Ointment | Hemorrhoids | Unknown | Unknown | Unknown |
| 14 | M | Ointment | Atopic dermatitis | Unknown | Unknown | Unknown |

F = female; Ig = immunoglobulin; M = male; SLE = systemic lupus erythematosus.

Table 2. Demographic and Clinical Characteristics of Patients with Idiopathic and Steroid-Induced Central Serous Chorioretinopathy

| Characteristics | Idiopathic Central Serous Chorioretinopathy (n = 96) | Steroid-Induced Central Serous Chorioretinopathy (n = 14) | P Value |
|---------------------------------|--|---|---------------------|
| Female sex | 13 (13.5) | 6 (42.9) | 0.015* |
| Age (yrs) | 52.6 ± 11.8 | 47.1 ± 11.5 | 0.089 [†] |
| Spherical equivalent (diopters) | -0.55 ± 2.02 | -0.57 ± 1.74 | 0.809 [†] |
| Axial length (mm) | 23.55 ± 0.97 | 23.50 ± 0.99 | 0.578 [†] |
| SCT (μm) | 391.5 ± 96.5 | 450.9 ± 139.4 | 0.088 [†] |
| Scleral thickness (μm) | | | |
| Superior | 423.4 ± 58.9 | 346.6 ± 68.7 | <0.001 [†] |
| Temporal | 440.1 ± 51.3 | 399.4 ± 48.3 | 0.018 [†] |
| Inferior | 450.1 ± 51.5 | 395.3 ± 55.5 | 0.002 [†] |
| Nasal | 436.6 ± 54.1 | 391.9 ± 37.3 | 0.001 [†] |

SCT = subfoveal choroidal thickness.

Data are presented as no. (%) or mean ± standard deviation, unless otherwise indicated.

*Fisher exact test.

[†]Wilcoxon rank-sum test.

52.0%.^{9,10,13,14,16,17,22} Wakakura et al¹³ and Bouzas et al¹⁵ previously reported that sCSC tended to affect a greater proportion of women than iCSC. Moreover, Quillen et al¹² analyzed women with CSC exclusively to demonstrate that a relatively high number of women used corticosteroids. In the current study, a greater proportion of women was included in the sCSC group than the iCSC group, a finding that is in agreement with previous reports.^{12,13,15} Taken together, these findings indicate that women tend to demonstrate CSC in association with

corticosteroid use at a rate that is greater than that of men. Because steroid hormones such as corticosteroids are sex hormones, sex differences in sex hormone regulation may play important roles in the development of CSC.

As mentioned above, choroidal thickening was easily evaluated by swept-source OCT, and choroidal abnormalities are known to play important roles in the pathogenesis of CSC. Several comparative studies have investigated SCT in idiopathic and steroid-induced CSC.^{20–22} Honda et al²⁰ reported that SCT values of patients with sCSC were less

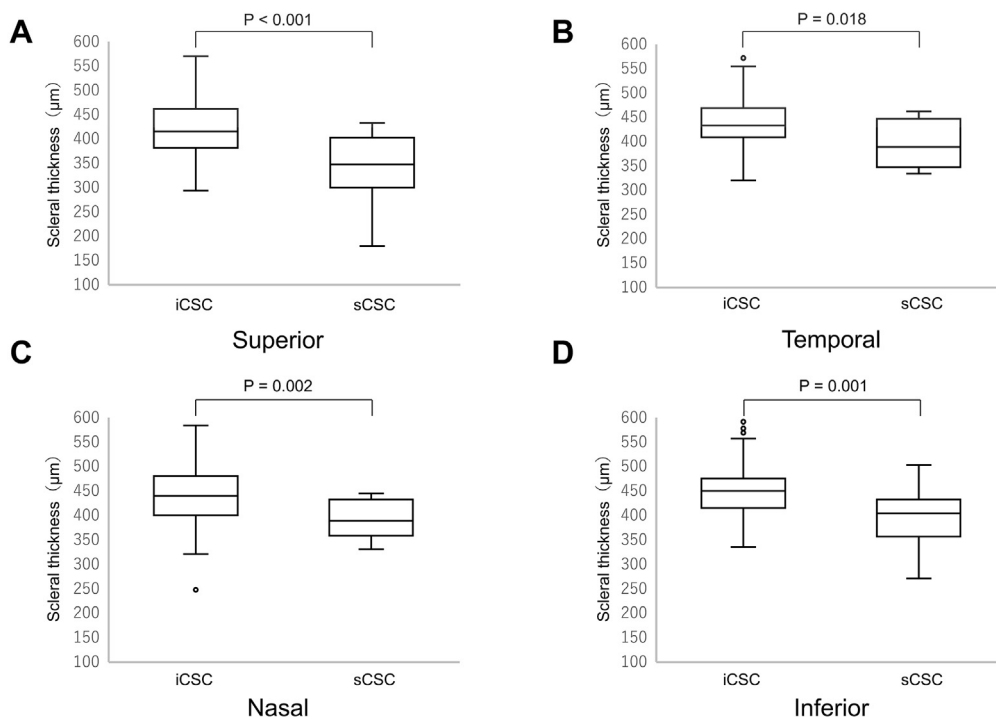


Figure 2. Box-and-whisker plots depicting scleral thicknesses in 4 directions in idiopathic central serous chorioretinopathy (iCSC) and steroid-induced CSC (sCSC): (A) superior, (B) temporal, (C) nasal, and (D) inferior. Scleral thickness at each point in the sCSC group was significantly thinner than that in the iCSC group.

Table 3. Multivariate Analysis of Factors Associated with Steroid-Induced Central Serous Chorioretinopathy

| | Odds Ratio | 95% Confidence Interval | P Value |
|--|------------|-------------------------|---------|
| Female sex (vs. male) | 4.322 | 1.025–18.224 | 0.046 |
| Age (yrs) | 0.954 | 0.897–1.015 | 0.139 |
| Mean scleral thickness (μm) | 0.972 | 0.955–0.990 | 0.002 |

than those of patients with iCSC, whereas Araki et al²² reported contradictory results. However, Izumi et al²¹ reported that no significant differences in SCT between patients with iCSC and sCSC were observed. The evaluation of SCT in patients with sCSC remains controversial, and further investigation using a larger study cohort and a strict definition of steroid use has the potential to provide a more accurate means to compare SCT values of patients with iCSC and sCSC. In this study, mean SCT values determined for the iCSC and sCSC groups did not significantly differ. Additionally, one of the authors (H.K.) reported that he observed intrachoroidal structural differences between the 2

groups.²¹ Results revealed that eyes with sCSC significantly differed from eyes with iCSC as follows: (1) reduced large choroidal vessel layer thickness versus SCT ratio and (2) reduced luminal versus entire choroidal area ratio. Accordingly, eyes with sCSC may have different intrachoroidal structures compared with those with iCSC. Steroid-induced CSC may develop in the absence of structural changes typically observed in iCSC, including pachyvessel or choroidal vasodilation,⁸ which are thought to be associated scleral thickening.²⁹

Several reports have indicated an association between the sclera and fluid accumulation in the choroid.^{29–34} Uveal effusion syndrome is a disease that causes exudative retinal and choroidal detachment resulting from scleral thickening.³¹ Brockhurst³² emphasized that the thickened sclera may have compressed vortex veins, causing drainage route resistance. Sclerotomy is reportedly effective for uveal effusion syndrome, even if the surgery does not decompress vortex vein congestion, because the procedure improves the permeability of the sclera itself.³³ Additionally, Spaide and Ryan³⁴ suggested that hydrostatic pressure within the choroid leads to the loculation of fluid in patients with CSC. They also suggested that scleral anatomic features are related to the

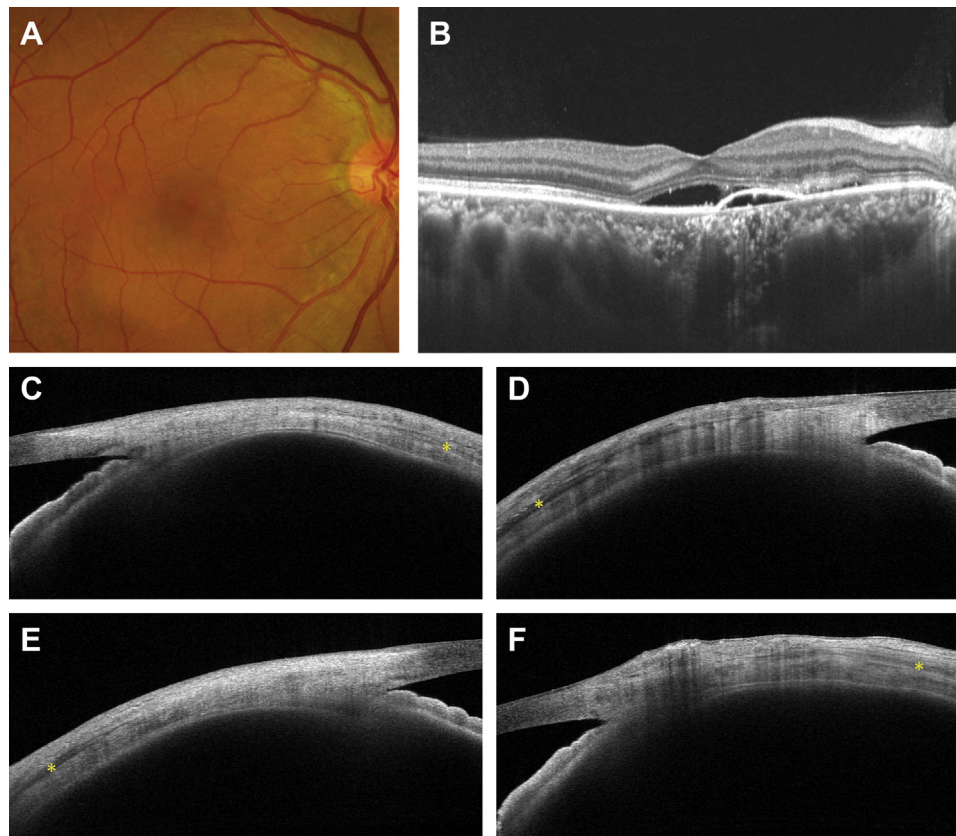


Figure 3. Images obtained from the right eye of a 47-year-old man with idiopathic central serous chorioretinopathy. Spherical equivalent was +0.125 diopters, and the axial length was 23.80 mm. **A**, Color fundus photograph revealing subretinal fluid in the macular region. **B**, Horizontal OCT image demonstrating subretinal fluid with shallow pigment epithelial detachment and a thickened choroid. The subfoveal choroidal thickness was 544 μm . **C–F**, Anterior-segment OCT images revealing cross-sectional images of the sclera in 4 directions: scleral thickness was 461 μm at the superior point (**C**), 409 μm at the temporal point (**D**), 480 μm at the inferior point (**E**), and 508 μm at the nasal point (**F**). Yellow asterisks indicate rectus muscles.

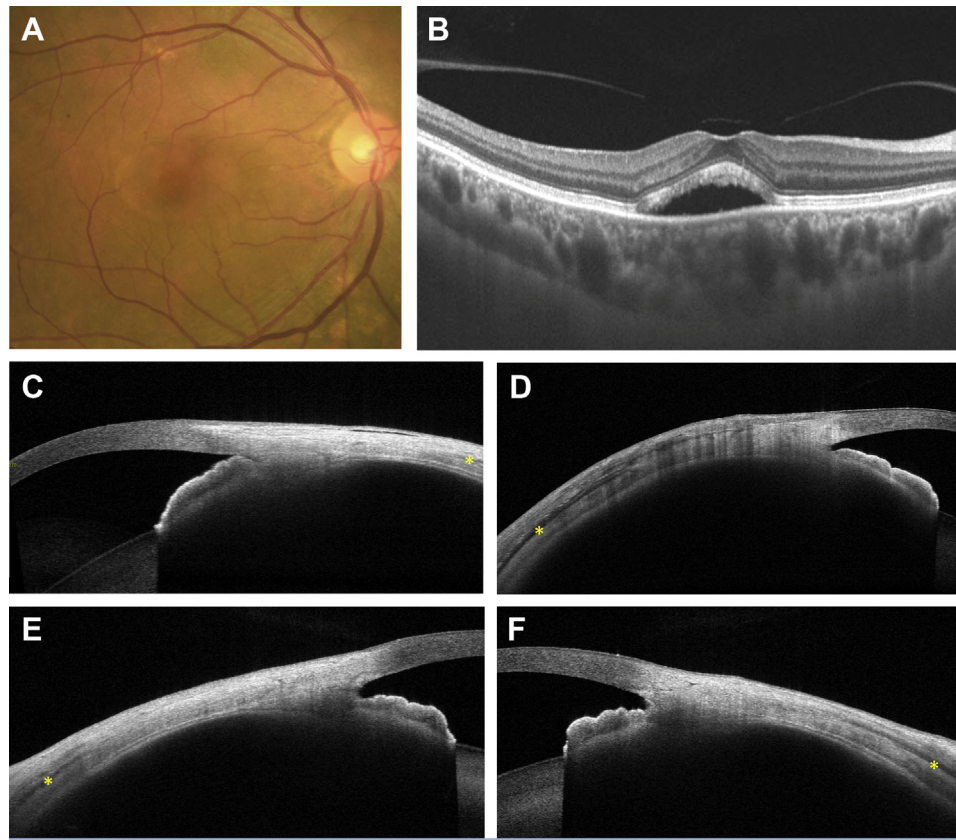


Figure 4. Images obtained from the right eye of a 59-year-old woman with steroid-induced central serous chorioretinopathy who received oral prednisolone for systemic lupus erythematosus. Spherical equivalent and axial length measured were +0.75 diopters and 23.31 mm, respectively. **A**, Color fundus photograph revealing subretinal fluid in the macular region. **B**, Horizontal AS OCT image revealing the presence of subretinal fluid and a thickened choroid. The subfoveal choroidal thickness was 437 μm . **C–F**, Anterior-segment OCT revealing cross-sectional images of the sclera in 4 directions: scleral thickness was 276 μm at the superior point (**C**), 348 μm at the temporal point (**D**), 426 μm at the inferior point (**E**), and 383 μm at the nasal point (**F**). Yellow asterisks indicate rectus muscles.

loculation of the fluid. In accordance with prior reports, we devised a method to measure scleral thickness using AS OCT and reported that eyes with iCSC harbored thicker sclera than normal control eyes.²⁹ In that article, we suggested that the presence of a thickened sclera may result in increased vortex vein outflow resistance, making choroidal congestion more likely to occur in eyes with iCSC.²⁹ In the current study, we paid particular attention to the scleral thickness of eyes with sCSC, which revealed for the first time that scleral thickness values at multiple points within eyes with sCSC were less than those of eyes with iCSC. Kuroda et al³⁵ reported AS OCT findings in patients with scleritis, which showed that scleral thickness values fluctuate. It cannot be denied that the long-term administration of corticosteroids likely affects scleral thickness, quality, or both. However, further investigation is needed to evaluate scleral characteristics and to clarify the association between scleral characteristics and the pathogenesis of CSC.

Considering the fact that mean scleral thickness values of eyes with sCSC were thinner than those of eyes with iCSC, a mechanism other than choroidal vascular congestion likely plays an important role in the pathophysiologic

characteristics of sCSC. Previous studies provide insights into the pathophysiologic characteristics of sCSC. Saito et al³⁶ demonstrated an association between increased sympathetic activity and the development of acute CSC using laser speckle flowgraphy. The same authors also showed that increased sympathetic activity may lead to increases in vascular resistance, which causes imbalanced choroidal blood flow distributions in eyes with acute CSC.³⁷ Curiously, Valamanesh et al²⁵ showed that structural changes such as choriocapillary rarefaction result from intravitreal triamcinolone acetonide injection in mice. Systemic or local effects of steroids may affect the choroid via sympathetic activity or choroidal vasculature homeostasis directly, even if scleral factor involvement and resultant pachychoroid status in sCSC do not exert as much impact as they do in iCSC. In fact, steroids reportedly inhibit norepinephrine uptake by nonneuronal cells, thereby increasing norepinephrine concentration at the sympathetic nerve ending and leading to sympathetic nervous system dominance.³⁸ Exogenous steroid use may have an additive effect on the choroid in sCSC. However, the precise mechanism involved remains undetermined.

The current study has several limitations that mainly owe to its retrospective nature and small sample size. First, the duration of steroid use varied. Second, we could not take into consideration the dose-dependent effect of steroids on patients. Systemic steroid use may impact the changes in scleral thickness and characteristics of the sclera such as rigidity. We need to clarify this issue in future studies. Third, systemic disorders that required steroid administration, rather than steroid use, may affect scleral and choroidal status. Fourth, the use of AS OCT to measure scleral thickness has not been automated, and therefore, measurements were obtained manually. Automated measurement methods are needed to ensure more objective analyses. Finally, we measured the scleral

thickness 6 mm posterior to the scleral spur in 4 directions under the rectus muscles, and the sclera evaluated in the current study did not affect vortex vein drainage routes directly. However, previous histomorphometric studies demonstrated significant correlation in scleral thickness between anterior and posterior portions of enucleated globes.^{39,40} Accordingly, we believe that our results reflect scleral thickness in the more posterior part of the eyes.

In conclusion, we used AS OCT to show that the mean scleral thickness value of eyes with sCSC was significantly thinner than that of eyes with iCSC. This suggests that the sclera has less involvement in the pathogenesis of sCSC than in that of iCSC.

Footnotes and Disclosures

Originally received: August 8, 2021.

Final revision: January 28, 2022.

Accepted: February 1, 2022.

Available online: February 8, 2022.

Manuscript no. D-21-00140

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Disclosure(s):

All authors have completed and submitted the ICMJE disclosures form.

The author(s) have made the following disclosure(s): S.S.: Lecturer — Senju

N.T.: Lecturer — Topcon, Novartis, AbbVie, Kowa, Bayer

N.I.: Lecturer — HOYA, Senju

S.W.: Lecturer — Novartis, Senju

T.T.: Lecturer — Novartis, Alcon, Senju

Y.Y.: Lecturer — Senju, Alcon, HOYA

H.K.: Financial support — Novartis, Bayer, Alcon, HOYA, Senju, Otsuka, AMO, Kowa, Santen; Lecturer — Novartis, Bayer, Alcon, HOYA, Topcon, Senju, Nidek, AbbVie, Otsuka, Pfizer, AMO, JFC, Kowa, Bausch & Lomb, Allergan, Canon, TOMEY, Sumitomo Dainippon Pharma, Sanofi, Star Japan, Santen

Supported by Japan Society for the Promotion of Science KAKENHI, Tokyo, Japan (grant no.: JP20K18349).

HUMAN SUBJECTS: Human subjects were included in this study. The human ethics committees at University of the Ryukyus approved the study.

All research adhered to the tenets of the Declaration of Helsinki. All participants provided informed consent.

No animal subjects were included in this study.

Author Contributions:

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Analysis and interpretation: Sawaguchi, Terao, Koizumi

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Abbreviations and Acronyms:

AS = anterior-segment; CSC = central serous chorioretinopathy; ICGA = indocyanine green angiography; iCSC = idiopathic central serous chorioretinopathy; sCSC = steroid-induced central serous chorioretinopathy; SCT = subfoveal choroidal thickness.

Keywords:

Central serous chorioretinopathy, Choroidal thickness, Pachychoroid, Scleral thickness, Steroid.

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