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MYOPIC PRESENTATION OF CENTRAL SEROUS CHORIORETINOPATHY

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Purpose: To increase insight into the myopic presentation of central serous chorioretinopathy (CSC) by comparing a large group of myopic patients with CSC with reference groups with only one of the diagnoses.

Methods: Myopic patients with CSC (spherical equivalent ≤ -3D, n = 46), emmetropic patients with CSC (spherical equivalent -0.5 to 0.5 D, n = 83), and myopic, non-CSC patients (n = 50) were included in this multicenter cross-sectional study. Disease characteristics and imaging parameters, such as subfoveal choroidal thickness and indocyanine green angiography patterns, were compared between cases and reference groups.

Results: In myopic patients with CSC, median subfoveal choroidal thickness (286 μm [IQR 226–372 μm]) was significantly thicker than subfoveal choroidal thickness in myopic, non-CSC patients (200 μm [IQR 152–228 μm], $P < 0.001$) but thinner than emmetropic patients with CSC (452 μm [IQR 342–538 μm], $P < 0.001$). They also had pachyvessels in 70% of the eyes comparable with emmetropic CSC (76%, $P = 0.70$). Choroidal hyperpermeability was frequently present on indocyanine green angiography in both myopic and emmetropic CSC eyes. Need for treatment, treatment success, and recurrence rate were not significantly different between CSC groups.

Conclusion: Myopic CSC presents with similar imaging and clinical characteristics as emmetropic CSC, apart from their thinner choroids. Keeping in mind the structural changes of myopia, other imaging characteristics could aid the diagnostic process.

RETINA 41:2472–2478, 2021

Central serous chorioretinopathy (CSC) is a chorioretinal disease characterized by a serous retinal detachment with or without a retinal pigment epithelial (RPE) detachment and presumably correlated to changes in the choroidal vasculature.[1–3] Previous studies in patients with CSC identified choroidal vascular hyperpermeability and an increased choroidal thickness due to enlargement of vessels in Haller's layer.[4–6] This notion that CSC is part of the pachychoroid spectrum was fortified by studies showing that subfoveal choroidal thickness (SFCT) decreased with resolution of CSC and increased again with recurrences.[7,8] Myopia with its elongated axial length (AL) is known to be inversely associated with choroidal thickness, every 1 mm increase in AL decreases the choroid by 32 μm.[9] Previous studies identified myopia as a protective factor for CSC and hyperopia as a risk factor.[10]A diagnosis of CSC indeed seems to be rare in myopic persons, only 10 cases of CSC with myopia could be identified in the literature.[11–13] Subfoveal choroidal thickness in these myopic CSC cases was thicker than expected based on their refractive error and age but within the normal range for emmetropic eyes.[6] This suggests that identification of a pachychoroid disease in a myopic patient can be easily overlooked. The current study aims to increase insight into the clinical and imaging features of myopic CSC by comparing a large group of myopic patients with CSC with reference groups with only one of the diagnoses.

Materials and Methods

Study Population

This study had a multicenter cross-sectional design. Data were collected from various clinical sites and from a population-based study. The case group consisted of n = 42 myopic patients with a history of CSC. Of these, n = 36 were ascertained from the Rotterdam Eye Hospital (Rotterdam, the Netherlands), n = 3 from Leiden

University Medical Centre (Leiden, the Netherlands), and n = 3 from Vitreous Retina Macula Consultants of New York (New York, NY). The first reference group consisted of n = 76 emmetropic patients with CSC collected at the Rotterdam Eye Hospital. The second reference group consisted of n = 50 myopic, non-CSC controls from the Rotterdam Study, a prospective population-based cohort study (RS I-III; registered at trialregister.nl [NTR6831]) in the Netherlands.[14] The myopic reference group matched with the case group on age and AL. This study was approved by the Medical Ethics Committee of the Erasmus Medical Centre, Rotterdam (MEC-2018-109), and was registered at www.toetsingsonline.nl (NL65899.078.18). Written informed consent was obtained from all participants.

Diagnostic Criteria

The exclusion criteria were photodynamic therapy (PDT) 1 year before enhanced depth imaging optical coherence tomography (EDI-OCT) measurement because PDT significantly decreases the choroidal thickness in the first year after treatment[7,15] and poor visibility of the choroidal–scleral junction on EDI-OCT. The inclusion criteria were moderate myopia (spherical equivalent (SE) ≤ -3 diopters (D)) and AL ≥ 24.0 mm or emmetropia SE between -0.5 D and 0.5 D. High myopia was defined as a SE ≤ -6 D or more. Central serous chorioretinopathy was defined as subretinal fluid (SRF) with or without pigment epithelium detachment on OCT, presence of leakage on fluorescein angiography (FA), and/or choroidal hyperpermeability on indocyanine green angiography (ICGA). The eyes with other possible causes of fluid accumulation, such as dome-shaped macula, were excluded with the use of multimodal imaging. Subretinal fluid either resolved spontaneously or after half-time PDT when SRF did not resolve after 6 months. Central serous chorioretinopathy was considered acute when symptoms or SRF resolved within 6 months or chronic when symptoms lasted for at least 6 months or extensive RPE abnormalities covering an area equivalent to at least two times the optic disk diameter were present.[16] Recurrence was defined as a new episode of CSC at least 3 months after proven complete resolution of SRF.

Study Outcomes and Data Collection

The primary study outcome was SFCT. Secondary outcomes were pachyvessels on EDI-OCT, choroidal hyperpermeability and intervortex vein anastomoses on ICGA, best-corrected visual acuity and disease characteristics (type of CSC and need for treatment), recurrence of CSC, and secondary macular neovascularization. Cofactors of the outcomes were age at the onset, SE, and AL. Spherical equivalent was measured by autorefraction; AL was measured using ocular biometry (IOLMaster 700; Zeiss, Germany or LENSTAR LS 900; Haag–streit, Switserland). Subfoveal choroidal thickness was measured using EDI-OCT (SPECTRALIS; Heidelberg, Germany) or OCT (3D OCT-1000 and 2000; Topcon, Japan) manually by M.R. and S.Y. and defined as the vertical distance between the hyperreflective outer border of the RPE and the hyperreflective line of the choroidal–scleral junction at the center of the fovea.[17] The suprachoroidal space, visible in 50% of patients with CSC as a second hyperreflective line beneath Haller's layer, was not included in the total SFCT.[18] Pachyvessels were defined as dilated outer choroidal vessels in an increased Haller's layer proportion, which correlated with areas of maximal choroidal thickness.[19] Dilation was considered present if the lumen of the vessels took up more than two-thirds of the total choroidal thickness. Leakage points and atrophic RPE alterations on FA and choroidal congestion and hyperpermeability on ICGA were assessed by a senior retinal specialist (S.Y.). Early ICGA was assessed for the presence of anastomotic connections between vortex vein systems. An intervortex venous anastomosis was considered to be present if 2 or more anastomotic vessels connected adjacent quadrants of vortex veins.[20] FA and ICGA were only performed in patients with CSC who potentially needed PDT or to exclude alternative diagnosis. Chorioretinal changes on color fundus and OCT were graded by the Eye-NED reading centre

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Supported by Rotterdamse Stichting Blindenbelangen (RSB), Rotterdam, the Netherlands. B20170076. The sponsor had no role in the design or conduct of this research.

None of the authors has any financial/conflicting interests to disclose.

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(Erasmus Medical Centre, Rotterdam, the Netherlands) according to a standardized protocol.

Statistical Analysis

All data were analyzed using version 25.0 of SPSS (IBM SPSS, Chicago, IL). Subfoveal choroidal thickness and SE were presented as median with interquartile range; age and AL as mean ± standard deviation. Medians between groups were compared with the Mann–Whitney test, means of continuous variables with an independent Student *t*-test, and differences in proportions of categorical variables with the chi-square test or Fisher exact test. *P* value less than 0.05 was considered statistically significant.

Results

In total, 46 eyes with CSC from 42 myopic cases, 83 eyes with CSC from 76 emmetropic patients, and 50 eyes without CSC from 50 myopic patients were included in the analysis. The emmetropic patients with CSC (age 45 ± 9 years) were significantly younger at the OCT-EDI measurement than the myopic case group with CSC (age 53 ± 10 years, $P < 0.001$) and the myopic reference group without CSC (age 54 ± 4 years, $P < 0.001$). The proportion of men was slightly lower in the case group (69%) than reference groups, 79% in emmetropic CSC eyes and 72% in myopic non-CSC eyes, respectively (Table 1). The mean SE and AL in the myopic case and myopic, non-CSC patients corresponded closely: SE −5.1 D (−6.8 to −4.3 D) versus SE −5.4 D (−5.9 to −4.6 D, $P = 0.53$) and AL 26.0 ± 1.1 mm versus 25.9 ± 0.4 mm ($P = 0.54$). High myopic eyes represented 33% of the case group (n = 15) and 24% of the myopic control group (n = 12, $P = 0.35$).

Subfoveal Choroidal Thickness and Other Imaging Characteristics

Myopic eyes with CSC had a median SFCT of 286 μm (226–372 μm), which was significantly thinner than emmetropic eyes with CSC (452 μm [342–538 μm], $P < 0.001$) but thicker than myopic, non-CSC eyes (200 μm [152–228 μm], $P < 0.001$). Pachyvessels were detected in 32 eyes of the myopic case group and 63 eyes of the emmetropic CSC reference group (70 and 76%, respectively, $P = 0.70$). Fluorescein angiography showed similar leakage patterns in the myopic case group and emmetropic CSC reference group with focal leakage being the most common observation in 23/34 myopic eyes and 28/42 emmetropic eyes ($P = 0.97$). Indocyanine green angiography was performed in 16 myopic eyes (34%) and 11 emmetropic eyes (13%) with CSC and showed typical choroidal hyperpermeability and multifocal choroidal congestion in all cases. Intervortex vein anastomoses were identified in 10 myopic case eyes (63%) and 8 emmetropic CSC eyes (72%, Figure 1).

Clinical Characteristics of Central Serous Chorioretinopathy

Clinical characteristics of CSC were compared in the subanalyses between the case group (myopic eyes with CSC) and reference group (emmetropic eyes with CSC). The mean age at the disease onset corresponded closely as follows: 48 ± 9 years in the case group and 43 ± 9 years in the reference group ($P = 0.02$). The best-corrected visual acuity significantly improved in both groups after spontaneous resolution and treatment (Table 2). Figure 2 shows a representative case of a high myopic patient with CSC with an increased SFCT and pachyvessels as identified on EDI-OCT.

Subretinal fluid was persistent in 25 myopic eyes (54%) and 47 emmetropic eyes (67%) with acute or

Table 1. Subfoveal Choroidal Thickness and Cofactors in the Case Group (Myopic and CSC) and Reference Groups (Emmetropic and CSC and Myopic, Non-CSC)

Variables	Case Group	Reference Group 1		Reference Group 2	
	Myopic CSC	Emmetropic CSC	*P**	Myopic, Non-CSC	*P**
Number of eyes	46	83	—	50	—
SFCT, μm (median, IQR)	286 (226–372)	452 (342–538)	**<0.001**	200 (152–228)	<0.001
Cofactors					
Age† (mean ± SD)	53 ± 10	45 ± 9	**<0.001**	54 ± 4	0.54
Gender, male (%)	29 (69)	60 (79)	0.23	36 (72)	0.76
SE, D (median, IQR)	−5.1 (−6.8 to −4.3)	0.0 (−0.4–0.5)	**<0.001**	−5.4 (−5.9 to −4.6)	0.53
AL, mm (mean ± SD)	26.0 ± 1.1	NA	NA	25.9 ± 0.4	0.68

**P* values are calculated for each reference group (compared with the case group).
†Age was calculated for the moment of OCT measurement.
NA, not applicable; OCT, optical coherence tomography.

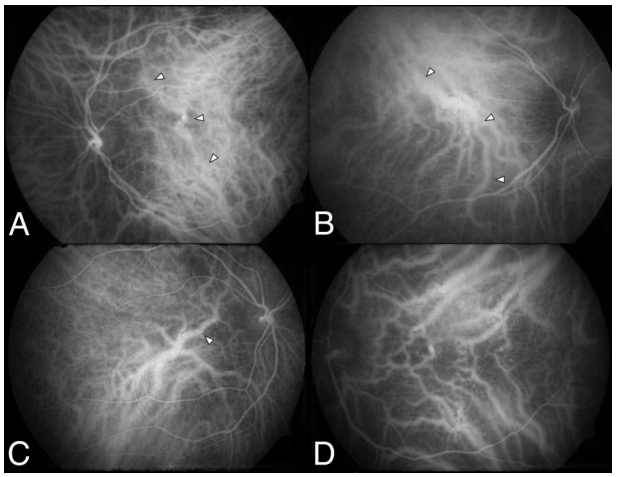

Fig. 1. A–D. Four cases of central serous chorioretinopathy. A 34-year-old man with spherical equivalent (SE) of −4.25 D (**A**), a 41-year-old woman with SE of −4.25 D (**B**), a 44-year-old man with SE of −0.25 D (**C**), and a 44-year-old man with SE of 0.75 D (**D**). Large choroidal veins with anastomotic connections uniting the venous outflow systems. These typical anastomoses (arrowheads) could be found in myopic (**A** and **B**) and emmetropic (**C** and **D**) cases.

chronic CSC. In both groups, only a few patients were treated with oral medication (two vs. three eyes), others received half-time PDT. Most of the patients in both groups were successfully treated with one session half-time PDT (Table 2). Recurrence of CSC occurred in 21 myopic case eyes (46%) with a mean of 1.5 ± 0.9 recurrences per eye. In emmetropic CSC, recurrences developed in 42 eyes (51%) with a mean of 1.1 ± 0.3 recurrences per eye. Although there was no significant difference between myopic and emmetropic eyes in developing a CSC recurrence, myopic eyes with recurrences developed these more often than emmetropic eyes ($P = 0.04$). Choroidal neovascularization during follow-up occurred in 8 myopic eyes and 4 emmetropic eyes, which was close to significance ($P = 0.03$).

Discussion

This study investigated the relatively rare myopic presentation of CSC. We showed that myopic patients with CSC had a thicker SFCT compared with myopic, non-CSC patients. Other typical pachychoroid disease characteristics, such as pachyvessels, choroidal hyperpermeability, and intervortex vein anastomoses, were equally present in myopic eyes and emmetropic eyes with CSC. These results show that the thinner choroids, as usually observed in myopia, are not excluded from developing a pachychoroid disease. Besides similar disease characteristics between myopic and emmetropic CSC eyes, we additionally showed that the presence of myopia did not influence the need for treatment, treatment success, or recurrence rate. Where the diagnosis of CSC in myopia can be challenging, standard treatment and follow-up guidelines can be followed.

Previous studies that investigated the relationship between CSC and refractive error, identified hyperopia as a risk factor for the disease.[10,21] By contrast, myopia was suggested to be a protective factor for the development of CSC because a longer AL is correlated to a thinner choroid and may therefore be less likely to develop pachychoroid disease. In this study, SFCT in the myopic case group was significantly thinner than

Table 2. Clinical Characteristics of Central Serous Chorioretinopathy in Myopic Eyes (Cases) and Emmetropic Eyes (References)

Variables	Case Group Myopic CSC	Reference Group Emmetropic CSC	*P*
Number of eyes	46	87	—
Age at disease onset, years (mean ± SD)	48 ± 9	43 ± 9	0.02
Type of CSC, acute versus chronic (%)	34/12 (74/26)	50/33 (60/40)	0.12
Corticosteroids (%)	9 (17)	15 (20)	0.93
BCVA at disease onset, Snellen (logMar)	20/25 (0.12 ± 0.15)	20/32 (0.14 ± 0.23)	0.55
BCVA after resolution, Snellen (logMar)	20/20 (0.06 ± 0.12)	20/25 (0.05 ± 0.21)	0.79
P value (before vs. after active CSC)	0.008	<0.001	—
Spontaneous resolution (%)	22 (48)	36 (43)	0.63
Treatment; half-time PDT (%)	22 (48)	44 (53)	0.57
1 session (%)	20 (91)	39 (89)	—
>1 session (%)	2 (9)	5 (12)	—
Recurrence of CSC (%)	21 (46)	42 (51)	0.50
Macular neovascularization (%)	8 (17)	4 (5)	0.03

BCVA, best-corrected visual acuity; SD, standard deviation.

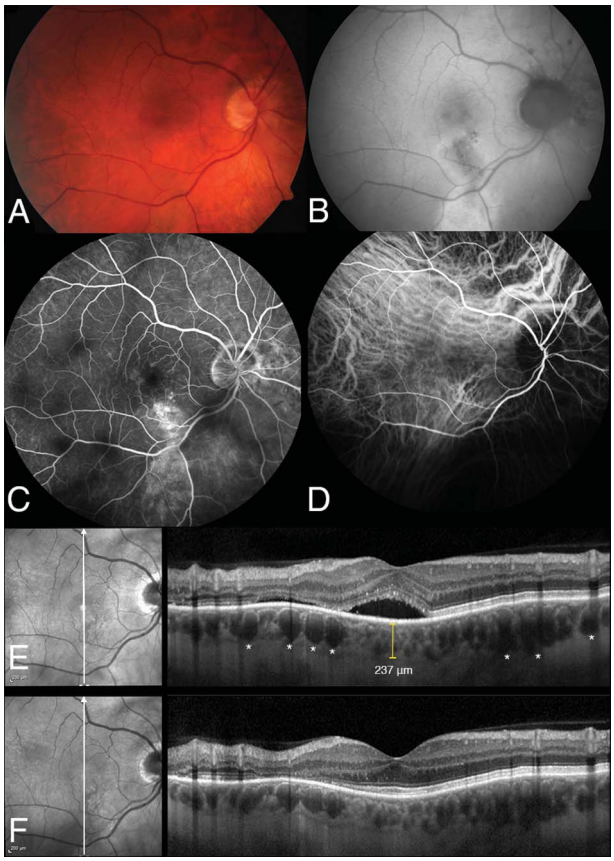

Fig. 2. A–F. Multimodal imaging of a 58-year-old man, with a spherical equivalent of −10 D and axial length of 26.69 mm, with chronic CSC. Color fundus photograph shows mild fundus tessellation and no signs of myopic macular degeneration (**A**). A descending hypofluorescent gravitational track from the macular area is shown on fundus autofluorescence (**B**). (**C**) Fluorescein angiography shows leakage in the late phase, and (**D**) indocyanine green angiography shows dilated choroidal vessels and hyperfluorescence (midphase). **E.** A vertical single B scan obtained with a EDI-OCT scan shows a neurosensory detachment with pachyvessels (white asterisks) in Haller's layer with a nearly invisible overlying choriocapillaris. The yellow line shows the position of subfoveal choroidal thickness measurement, being 237 μm. **F.** Enhanced depth imaging optical coherence tomography single B scan of the choroid 4 months after half-time photodynamic therapy with complete resolution of the neurosensory detachment.

emmetropic CSC. Subfoveal choroidal thickness in our myopic, non-CSC controls was as expected when using the formula published by Flores–Moreno et al.[22] These choroids were significantly thinner than the choroids of our myopic CSC cases with similar AL. This is in concordance with an earlier study on myopic CSC and SFCT.[11] It also strengthens the hypothesis that although choroidal thickness in myopic CSC eyes cannot match the choroids of emmetropic CSC eyes, the choroids of these myopic eyes with CSC are unquestionably enlarged. From our findings, we conclude that AL should therefore be taken into consideration when interpreting choroidal thickness in pachychoroid-related diseases.

Although a thick choroid is frequently observed, it is not the most important criterion for the diagnosis of pachychoroid disease.[19] Instead, choroidal vascular abnormalities, such as pachyvessels with thinning of the overlying choriocapillaris and choroidal hyperpermeability are essential for the diagnosis. Choroidal hyperpermeability results in increased hydrostatic pressure in the choroid and choriocapillaris, which causes leakage from one or more areas through a defect in the RPE outer blood–retina barrier, and ultimately leads to a serous retinal detachment in patients with CSC.[1,2] In this study, we specifically looked for pachyvessels in all CSC eyes and found them present in 71% of the myopic cases. Comparable proportions were found in emmetropic CSC controls. A recent study suggested that the typical pachyvessels as seen on OCT-EDI are anastomoses that obliterate the watershed zone in the choroidal venous draining system and can be seen on early ICGA.[20] We found these intervortex vein anastomoses to be present in most of the cases that underwent ICGA, in both myopic and emmetropic eyes with CSC. In addition, choroidal hyperpermeability was visible in all ICGAs available on myopic cases and emmetropic CSC control eyes.[23] Therefore, these results suggest that both myopic CSC and emmetropic CSC are likely to have the same underlying pathogenesis in the choroid.

Central serous chorioretinopathy is typically diagnosed in middle-aged patients with a peak incidence between 40 and 50 years.[24,25] Our myopic CSC cases (mean 48 years) were older than our emmetropic CSC cases (mean 44 years) at the disease onset. Despite the difference in age, both myopic CSC and emmetropic CSC cases were within the range of CSC first diagnosis according to the literature. Clinically, CSC is self-limiting in most cases; treatment is reserved for eyes with persistent SRF or chronic CSC. Based on the current level of evidence, PDT with reduced settings is considered the treatment of choice.[16,26] In this study, complete resolution of the SRF was acquired in 93% and 89% of the myopic, respectively, emmetropic CSC eyes. Considering a direct decreasing effect of PDT on choroidal thickness,[7] we hypothesized that myopic patients may be at risk of developing chorioretinal atrophy after PDT. Fortunately, none of the patients experienced complications or developed chorioretinal atrophy after treatment, even 7 years after therapy. The results of the current study indicate that half-time PDT is an effective and safe treatment in myopic patients with typical CSC. It should be kept in mind that other complications explaining the accumulation of SRF, such as serous maculopathy in the context of a tilted disk with inferior staphyloma and/or subretinal neovascularization, must be excluded.[16]

Recurrences of CSC appear in 15% to 50% of the eyes after a first episode depending on CSC type and previous

treatment.[16,27] In this cohort, a relatively high number of recurrences were reported in myopic (46%) and emmetropic eyes (52%). This could be explained by the patient population of the different involved centers, which are all tertiary hospitals and referral centers for CSC. Previous reports suggested that the recurrence rate of CSC was negatively correlated with the magnitude of SFCT reduction after resolution. We could not find differences in recurrences between myopic and emmetropic eyes and, therefore, favor the opinion that not the absolute volume of the choroid determines recurrence, but the relative change in SFCT from active to resolved CSC. This has been found to be independent of the refractive error and the choroidal thickness per se.

Secondary MNV is a severe complication of CSC, which occurs in 2% to 16% of CSC eyes.[25,28,29] In our study, myopic eyes developed significant more MNV's after CSC resolution compared with emmetropic eyes. An explanation may be that some myopic eyes had developed a primary myopic MNV independent or earlier CSC. The prevalence of myopic MNV has been estimated to be 10% in eyes with pathologic myopia, which occurs predominantly in high myopia with abnormalities of the posterior segment.[30] Because only a small portion of our patients with CSC was highly myopic and we did not observe other retinal lesions, we think that it is unlikely that myopic MNV independent of CSC explained the differences between myopic and emmetropic eyes.

Our study had some limitations. Axial length was not measured in emmetropic eyes, whereas refractive error and AL are important factors for SFCT. In this study, AL was a tool to have a comparable myopic control group and to show that myopia was truly caused by axial elongation and not, for instance, by cataract of corneal changes. Besides AL, age is also known to be a determinant of choroidal thickness. Our emmetropic controls were significantly younger than the myopic cases and controls, and it is therefore reasonable to assume that the difference in choroidal thickness between myopic cases and emmetropic controls may be partly explained by age. The myopic CSC cases and myopic, non-CSC patients had the same age; hence, this did not determine the difference between these two groups. We encountered a problem with the timing of OCT measurement. A previous study reported a more pronounced SFCT during an active phase of CSC when SRF is present.[8] Enhanced depth imaging optical coherence tomography measurements in myopic cases were predominantly performed after SRF resolution because of logistic reasons, whereas emmetropic CSC references were ascertained at a different time point allowing EDI-OCT measurements during the active period. Given these constraints, we may have overestimated the difference in SFCT between myopic cases and emmetropic references and underestimated the difference in SFCT between myopic cases and myopic references.

In conclusion, CSC is an uncommon diagnosis in myopic eyes and may be overlooked if a neurosensory detachment or typical smokestack leakage on FA is absent. Given the rapidly increasing prevalence of myopia, clinicians will encounter more diseases on a myopic background. We demonstrated that myopic patients show similar features to emmetropic patients with CSC, apart from the choroidal thickness. A choroid that is thicker than expected based on AL should be carefully evaluated and should be considered a pathological finding in case SRF is also present. In our myopic cases, we did not only find an increased choroidal thickness but we also found other typical vascular abnormalities in pachychoroid disease, such as pachyvessels and choroidal hyperpermeability. This suggests a similar origin of disease in emmetropic and myopic patients with CSC and implies that CSC in myopia is not a different disease entity but a rare presentation of CSC.

Key words: central serous chorioretinopathy, pachychoroid, myopia, choroidal thickness, pachyvessels, indocyanine green angiography.

Acknowledgments

The authors thank Dr. G. Ledesma from the Vitreous Retina Macula Consultants of New York and Drs. Y. Coskun of the Rotterdam Eye Hospital for their efforts to review numerous patient files and T. Verzijden for his efforts in this study.

References

1. Gass JD. Pathogenesis of disciform detachment of the neuroepithelium. Am J Ophthalmol 1967;63:1–139.
2. Spaide RF, Hall L, Haas A, et al. Indocyanine green videoangiography of older patients with central serous chorioretinopathy. Retina 1996;16:203–213.
3. Yannuzzi LA, Shakin JL, Fisher YL, Altomonte MA. Peripheral retinal detachments and retinal pigment epithelial atrophic tracts secondary to central serous pigment epitheliopathy. Ophthalmology 1984;91:1554–1572.
4. Kim YT, Kang SW, Bai KH. Choroidal thickness in both eyes of patients with unilaterally active central serous chorioretinopathy. Eye (Lond) 2011;25:1635–1640.
5. Chung YR, Kim JW, Choi SY, et al. Subfoveal choroidal thickness and vascular diameter in active and resolved central serous chorioretinopathy. Retina 2018;38:102–107.
6. Chen G, Tzekov R, Li W, et al. Subfoveal choroidal thickness in central serous chorioretinopathy: a meta-analysis. PLoS One 2017;12:e0169152.
7. Kang NH, Kim YT. Change in subfoveal choroidal thickness in central serous chorioretinopathy following spontaneous res-

olution and low-fluence photodynamic therapy. Eye (Lond) 2013;27:387–391.
8. Kim DY, Joe SG, Yang HS, et al. Subfovael choroidal thickness changes in treated idiopathic central serous chorioretinopathy and their association with recurrence. Retina 2015;35: 1867–1874.
9. Wei WB, Xu L, Jonas JB, et al. Subfoveal choroidal thickness: the Beijing eye study. Ophthalmology 2013;120:175–180.
10. Ersoz MG, Arf S, Hocaoglu M, et al. Patient characteristics and risk factors for central serous chorioretinopathy: an analysis of 811 patients. Br J Ophthalmol 2019;103:725–729.
11. Yzer S, Fung AT, Barbazetto I, et al. Central serous chorioretinopathy in myopic patients. Arch Ophthalmol 2012;130: 1339–1340.
12. van Dijk EHC, de Roon Hertoge KL, Boon CJF. Central serous chorioretinopathy in a myopic patient with pachychoroid. Ophthalmol Point Care 2017;1:e26–e29.
13. Peponis VG, Chalkiadakis SE, Nikas SD, et al. Bilateral central serous retinopathy following laser in situ keratomileusis for myopia. J Cataract Refract Surg 2011;37:778–780.
14. Ikram MA, Brusselle GGO, Murad SD, et al. The Rotterdam study: 2018 update on objectives, design and main results. Eur J Epidemiol 2017;32:807–850.
15. Maruko I, Iida T, Sugano Y, et al. One-year choroidal thickness results after photodynamic therapy for central serous chorioretinopathy. Retina 2011;31:1921–1927.
16. van Rijssen TJ, van Dijk EHC, Yzer S, et al. Central serous chorioretinopathy: towards an evidence-based treatment guideline. Prog Retin Eye Res 2019;73:100770.
17. Staurenghi G, Sadda S, Chakravarthy U, Spaide RF. Proposed lexicon for anatomic landmarks in normal posterior segment spectral-domain optical coherence tomography: the IN • OCT consensus. Ophthalmology 2014;121:1572–1578.
18. Spaide RF, Ryan EH. Loculation of fluid in the posterior choroid in eyes with central serous chorioretinopathy. Am J Ophthalmol 2015;160:1211–1216.
19. Cheung CMG, Lee WK, Koizumi H, et al. Pachychoroid disease. Eye (Lond) 2019;33:14–33.
20. Spaide RFM, Ledesma-Gil GMD, Gemmy Cheung CMM. Intervortex venous anastomosis in pachychoroid-related Disorders. Retina 2020. Available at: https://journals.lww.com/retinajournal/Abstract/9000/Intervortex_Venous_Anastomosis_in.95698.aspx. Accessed March 8, 2021.
21. Manayath GJ, Arora S, Parikh H, et al. Is myopia a protective factor against central serous chorioretinopathy? Int J Ophthalmol 2016;9:266–270.
22. Flores-Moreno I, Lugo F, Duker JS, Ruiz-Moreno JM. The relationship between axial length and choroidal thickness in eyes with high myopia. Am J Ophthalmol 2013;155:314–319.e1.
23. Yang L, Jonas JB, Wei W. Choroidal vessel diameter in central serous chorioretinopathy. Acta Ophthalmol 2013;91:e358–e362.
24. Castro-Correia J, Coutinho MF, Rosas V, Maia J. Long-term follow-up of central serous retinopathy in 150 patients. Doc Ophthalmol 1992;81:379–386.
25. Spaide RF, Campeas L, Haas A, et al. Central serous chorioretinopathy in younger and older adults. Ophthalmology 1996; 103:2070–2080.
26. van Dijk EHC, Fauser S, Breukink MB, et al. Half-dose photodynamic therapy versus high-density subthreshold micropulse laser treatment in patients with chronic central serous chorioretinopathy: the PLACE trial. Ophthalmology 2018; 125:1547–1555.
27. Liew G, Quin G, Gillies M, Fraser-Bell S. Central serous chorioretinopathy: a review of epidemiology and pathophysiology. Clin Exp Ophthalmol 2013;41:201–214.
28. Loo RH, Scott IU, Flynn HW, et al. Factors associated with reduced visual acuity during long-term follow-up of patients with idiopathic central serous chorioretinopathy. Retina 2002; 22:19–24.
29. Shiragami C, Takasago Y, Osaka R, et al. Clinical features of central serous chorioretinopathy with type 1 choroidal neovascularization. Am J Ophthalmol 2018;193:80–86.
30. Wong TY, Ferreira A, Hughes R, et al. Epidemiology and disease burden of pathologic myopia and myopic choroidal neovascularization: an evidence-based systematic review. Am J Ophthalmol 2014;157:9–25.e12.