Clinical Ophthalmology

Dovepress
Taylor & Francis Group

Open Access Full Text Article

ORIGINAL RESEARCH

Choriocapillaris Flow Deficits Impact Macular Neovascularization Risk in Long-Term Follow-Up for Central Serous Chorioretinopathy

Naoya Kusunoki, Hisashi Fukuyama, Yuki Yamamoto, Fumi Gomi

Department of Ophthalmology, Hyogo Medical University, Nishinomiya, Hyogo, Japan

Correspondence: Hisashi Fukuyama, Department of Ophthalmology, Hyogo Medical University, 1-1 Mukogawa-cho, Nishinomiya, Hyogo, 663-8501, Japan, Tel +81 798 45 6462, Fax +81 798 45 6464, Email fukuyama1985@gmail.com

Purpose: To evaluate the influence of choriocapillaris flow deficit on macular neovascularization (MNV) development during extended follow-up for central serous chorioretinopathy (CSC).

Design: Retrospective study.

Participants: Thirty-six eyes from 36 patients with CSC, each followed for more than 3 years.

Methods: All patients underwent comprehensive ophthalmic examinations, including optical coherence tomography (OCT) and OCT angiography (OCTA), at baseline, 3 months from baseline, and the final follow-up. Choriocapillaris flow deficit percentage (CCFD%) was calculated using OCTA images obtained 3 months from baseline. Patients were categorized into two groups based on MNV presence or absence at the final follow-up.

Outcome Measures: Association between CCFD% and MNV development.

Results: Of the 36 patients, 11 (30.6%) developed MNV during the follow-up period (mean duration: 48.3 ± 14.5 months). The CCFD% at 3 months from baseline was significantly higher in the MNV group than in the non-MNV group (36.6 ± 1.9% vs 35.5 ± 2.3%, p = 0.027). In nine of the 11 eyes (81.8%) with MNV, neovascular lesions spatially corresponded to areas of focal geographic choriocapillaris loss identified at 3 months.

Conclusion: CCFD% measured by OCTA may serve as a prognostic biomarker for identifying CSC patients with elevated risk of MNV development. The spatial concordance between regions of choriocapillaris flow deficit and subsequent MNV formation supports the hypothesis that choriocapillaris ischemia substantially contributes to the pathogenesis of MNV in CSC.

Keywords: central serous chorioretinopathy, optical coherence tomography angiography, choriocapillaris, flow deficit, macular neovascularization

Introduction

Central serous chorioretinopathy (CSC) is a chorioretinal disease characterized by serous retinal detachment and leakage from the retinal pigment epithelium (RPE), commonly affecting middle-aged individuals.[1,2] CSC typically resolves spontaneously within a few months and carries a favorable visual prognosis. However, some cases progress to a chronic form involving persistent or recurrent subretinal fluid, which increases the risk of vision loss during long-term follow-up.[3,4] Additionally, long-term follow-up studies have shown that CSC is frequently complicated by the development of macular neovascularization (MNV).[5–7] Previous reports have estimated that MNV develops in approximately 2–18% of eyes with chronic or long-standing CSC,[8] highlighting the clinical importance of identifying reliable predictors for MNV development in this population.

In recent decades, a pachychoroid disease spectrum has been proposed, characterized by dilated choroidal vessels in Haller's layer—referred to as pachyvessels—accompanied by thinning of the overlying inner choroid and choroidal vascular hyperpermeability observed on indocyanine green angiography (ICGA). CSC is considered a representative entity within this spectrum.[9–11] According to the pachychoroid disease hypothesis, choriocapillaris alterations precede structural damage to the choriocapillaris and RPE, potentially leading to the development of MNV with disease

https://doi.org/10.2147/OPTH.S577922
Received: 13 November 2025
Accepted: 16 January 2026
Published: 27 January 2026

progression.[11] Vascular endothelial growth factor, which is upregulated under ischemic conditions, may be overexpressed in response to local hypoxia, thereby contributing to MNV formation.[12]

In eyes with age-related macular degeneration (AMD), some studies have detected an association between choriocapillaris dropout and MNV,[13,14] with dropouts frequently observed in the region surrounding neovascular lesions. Similarly, we hypothesize that sustained choriocapillaris flow deficits in CSC contribute to MNV progression. Previous studies have identified choroidal vascular abnormalities and choriocapillaris flow deficits in eyes with CSC.[15,16] However, the relationship between choriocapillaris dropout and MNV development remains unclear. This study aimed to evaluate the impact of choriocapillaris flow deficits on MNV progression in eyes with CSC during long-term follow-up.

Methods

Study Design and Participants

This retrospective study of patients with CSC was conducted at the Department of Ophthalmology, Hyogo Medical University. The Institutional Review Board of Hyogo Medical University approved the study, which adhered to the principles of the Declaration of Helsinki. The need for individual informed consent was waived by the committee due to the retrospective observational study design.

We reviewed the medical records of all patients diagnosed with CSC at Hyogo Medical University who underwent optical coherence tomography angiography (OCTA) with a follow-up interval of more than 3 years from the baseline examination, between January 2014 and August 2024. The following inclusion criteria were applied: (1) diagnosis of CSC based on the presence of submacular fluid on OCT and at least one area of active angiographic leakage on fluorescein angiography; (2) availability of OCT and OCTA images at baseline, 3 months, and final follow-up; (3) absence of MNV on ICGA and OCTA at baseline and 3 months; (4) follow-up duration exceeding 3 years; and (5) absence of high myopia, glaucoma, or optic disc pits causing retinoschisis. Eyes were excluded if the OCTA image quality was inadequate (quality score < 40) or if images were compromised by motion artifacts, poor contrast, defocus, or misalignment of the scan center.

Data Collection

All patients underwent comprehensive ophthalmologic examinations (visual acuity measurement, fundus examination, OCT, OCTA) at baseline, 3 months from baseline, and the final follow-up. Images were obtained after pupil dilation. OCT images were captured by Heidelberg Spectralis OCT (Heidelberg Engineering, Heidelberg, Germany), and OCTA images were acquired using a swept-source OCT device (DRI OCT Triton; Topcon, Tokyo, Japan). Fluorescein angiography and ICGA were performed at baseline using the Spectralis HRA 2 (Heidelberg Engineering). Demographic and clinical data (age, sex, spherical equivalent, treatment regimen) were collected. For anatomical assessments, horizontal sectional OCT images were used to measure central foveal thickness (distance from internal limiting membrane to RPE line) and central choroidal thickness (from Bruch's membrane to the choroidoscleral interface). Two independent graders (N.K. and H.F.) evaluated each eye for the presence or absence of MNV using en face and B-scan OCTA images at baseline, 3 months, and final follow-up.

Choriocapillaris Flow Deficit Image Analysis

OCTA images covering a 4.5×4.5 mm area centered on the macula were used. Choriocapillaris images were generated with Triton ImageNet 6 software (version 5.01G, Topcon) using a slab extending from Bruch's membrane to 20.8 μm beneath the membrane. Automated segmentation was reviewed and manually corrected if necessary. Images were anonymized.

ImageJ software (version 2.0.0-rc; https://imagej.net/Fiji; United States National Institutes of Health, Bethesda, MD, USA) was used for quantitative analysis, in accordance with previous reports.[17,18] Each image was resized to 1024×1024 pixels (4.39 μm/pixel). Choriocapillaris images were binarized using the Phansalkar method (window radius = 15 px). Inverted binarized images were analyzed to calculate the percentage of nonvascular (flow deficit) areas. The choriocapillaris flow deficit percentage (CCFD%) was defined as the nonperfused area relative to the total imaged area (Figure 1).

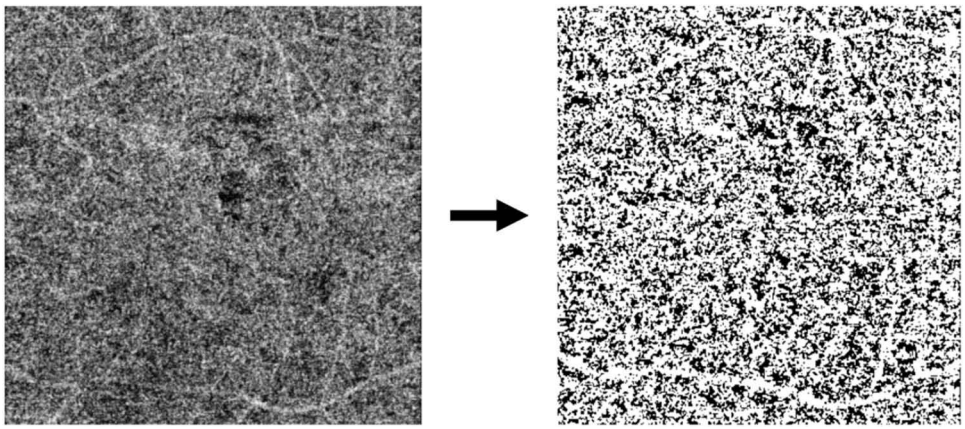

Figure 1 Method for calculating the choriocapillaris flow deficit percentage (CCFD%). An en face optical coherence tomography angiography (OCTA) image of the choriocapillaris was generated from a slab extending from Bruch's membrane (BM) to 20.8 μm below the membrane. The image of the choriocapillaris slab was binarized using the Phansalkar method. CCFD% was calculated as the proportion of the nonperfused (dark) area relative to the total imaged area.

Statistical Analysis

All statistical analyses were performed with JMP® Pro version 17.2.0 (SAS Institute, Cary, NC, USA). Best-corrected visual acuity (BCVA) was converted to logMAR units for statistical analysis. Continuous variables were expressed as mean ± standard deviation. Comparisons between the MNV and non-MNV groups were made using the Mann–Whitney *U*-test or Fisher's exact test, as appropriate. A p-value < 0.05 was considered statistically significant.

Results

We initially identified 48 eyes; 12 were excluded due to early MNV development within 3 months. Ultimately, 36 eyes from 36 patients were included. The cohort comprised 24 men and 12 women; mean age was 51.4 ± 10.6 years at baseline. Nineteen eyes were right, 17 left. Baseline mean logMAR BCVA was 0.09 ± 0.22; mean follow-up duration was 48.3 ± 14.5 months. Eleven eyes (30.6%) developed MNV during follow-up (Figure 2). Among the 27 eyes treated initially with photodynamic therapy, there was no significant difference in MNV incidence (p = 0.53). Regarding CSC characteristics, 16 eyes were classified as first-episode cases, whereas 20 eyes had a history of recurrent CSC. 16 eyes showed unifocal involvement and 20 eyes showed multifocal involvement. The duration of subretinal fluid prior to baseline was available for 32 of the 36 eyes, with a mean duration of 7.6 ± 7.3 months, while the duration was unknown in 4 eyes.

Table 1 presents a comparison of clinical characteristics between the MNV and non-MNV groups. The MNV group had a significantly higher proportion of females (p = 0.003), older age (p = 0.002), and more hyperopic spherical equivalent (p = 0.041). Table 2 compares OCT/OCTA parameters between the two groups. CCFD% at 3 months from baseline was significantly higher in the MNV group (36.6 ± 1.9%) than in the non-MNV group (35.5 ± 2.3%, p = 0.027). Baseline central foveal thickness and central choroidal thickness did not differ significantly.

At the final follow-up (48.3 ± 14.5 months), the mean logMAR BCVA was –0.005 ± 0.19 (Snellen equivalent 20/20) in the non-MNV group and 0.24 ± 0.36 (Snellen equivalent 20/35) in the MNV group (p = 0.01). Subretinal fluid recurred at least once during the follow-up period in 29 eyes (80.6%). In the MNV group, recurrence occurred in 10/11 eyes (91%); in the non-MNV group, 19/25 (76%). Of 11 MNV eyes, 8 required anti–vascular endothelial growth factor therapy; 3 were subclinical. In 9/11 eyes (81.8%), MNV lesions spatially corresponded to focal geographic choriocapillaris defects observed at 3 months from baseline (Figure 3).

Discussion

In this study, we evaluated choriocapillaris flow deficits in CSC eyes after treatment. Our findings show that CCFD% at 3 months from baseline was significantly higher in eyes that developed MNV by final follow-up than in those that did not. Moreover, in 81.8% of MNV eyes, the neovascular lesions corresponded spatially to focal choriocapillaris defects

https://doi.org/10.2147/OPTH.S577922

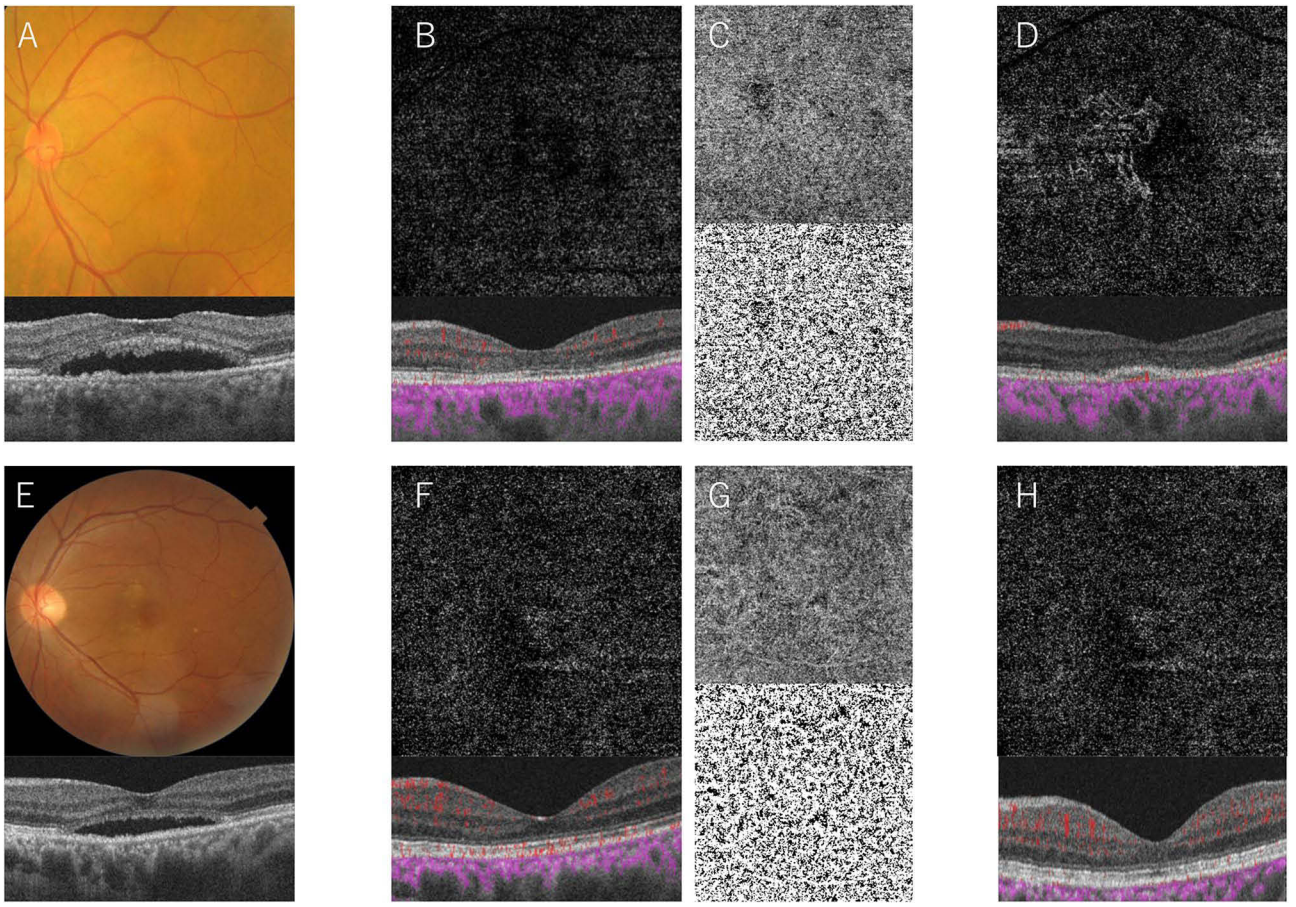

Figure 2 Representative cases of patients with and without macular neovascularization (MNV) development. A 65-year-old woman presented with subretinal fluid in the macular region and underwent photodynamic therapy (**A**). At 3 months post-treatment, en face OCTA and corresponding B-scan OCTA images showed no evidence of MNV (**B**), while the choriocapillaris slab revealed flow deficits with a CCFD% of 38.2% (**C**). At the final follow-up (72 months), en face and B-scan OCTA demonstrated the presence of MNV (**D**). A 49-year-old man presented with subretinal fluid in the macula and received photodynamic therapy (**E**). At 3 months, OCTA images showed no MNV (**F**), with a CCFD% of 34.3% (**G**). At 62 months, MNV remained absent (**H**).

identified earlier. These results support the hypothesis that sustained hypoxia in the choriocapillaris contributes to MNV development in CSC.

Type 1 MNV has been reported in CSC during long-term follow-up.[5] CSC has recently been classified as part of the pachychoroid disease spectrum, in which chronic hypoxic conditions drive neovascular changes, potentially progressing to pachychoroid neovasculopathy or polypoidal choroidal vasculopathy over time.[19] One proposed mechanism involves choriocapillaris occlusion secondary to prolonged vortex vein congestion or compression by dilated Haller's vessels, resulting in inner choroidal thinning and ischemia that ultimately promotes MNV formation.[20] Our study supports this pathophysiological mechanism by demonstrating significantly enhanced choriocapillaris flow deficit at the early post-treatment stage in eyes that subsequently developed MNV.

Some previous studies have identified factors associated with the development of MNV secondary to CSC. Zhou et al reported that older age, a higher incidence of chronic CSC and recurrence, and foveal leakage points on fluorescein angiography constituted risk factors for MNV.[21] Other studies have also shown that pachychoroid neovascularization is more likely to occur in older patients with a prolonged history of CSC.[5,10,21,22] Consistent with these findings, our study demonstrated a significantly higher percentage of older patients in the group with MNV at final follow-up ($p = 0.002$). Although not statistically significant, bilateral involvement—with CSC in the fellow eye—was more common in the MNV group than in the non-MNV group. Choroidal structure varies across populations and may be influenced by genetic background.[23] Clinical characteristics such as older age and a higher incidence of bilateral CSC may reflect an underlying

Table 1 Baseline Clinical Characteristics of Patients with Central Serous Chorioretinopathy

Variable	Overall	MNV Present (n=11)	MNV Absent (n=25)	P-value
Number of patients	36	11	25	
Eye laterality (right/left)	19/17	3/8	16/9	0.070
Age (years), mean±SD	51.4±10.6	60.7±11.8	47.4±7.1	0.003
Sex (male)	24 (66.7%)	3 (27.3%)	21 (84.0%)	0.002
Smoker (current or former)	18 (50%)	3 (27.3%)	15 (60.0%)	0.15
Phakic eyes	33 (91.7%)	9 (81.8%)	24 (96.0%)	0.22
Spherical equivalent (D), mean ± SD	−0.58±2.10	0.35±1.75	−0.98±2.15	0.041
Baseline BCVA (logMAR), mean±SD	0.09±0.22	0.23±0.30	0.03±0.15	0.056
History of CSC	20 (55.6%)	5 (45.5%)	15 (60.0%)	0.42
History of CSC in fellow eye	21 (58.3%)	9 (81.8%)	12 (48.0%)	0.077
Multiple leakage on FA	35	5 (50%) (n=10)	15 (60%)	0.59
CVH on ICGA	30 (83.3%)	8 (72.7%)	22 (88.0%)	0.34
PDT	27 (75.0%)	7 (63.6%)	20 (80.0%)	0.41
Follow-up duration (months), mean ± SD	48.3±14.5	43.8±12.7	50.2±15.0	0.24

Abbreviations: MNV, macular neovascularization; BCVA, best-corrected visual acuity; logMAR, logarithm of the minimum angle of resolution; CSC, central serous chorioretinopathy; CVH, choroidal vascular hyperpermeability; ICGA, indocyanine green angiography; PDT, photodynamic therapy; SD, standard deviation; D, diopters; FA, fluorescein angiography.

Table 2 OCT and OCTA Characteristics

Characteristic	Overall (n=36)	MNV Present at Final Follow-Up (n=11)	MNV Absent at Final Follow-Up (n=25)	P-value
Baseline CFT (μm), mean±SD	358.3±153.7	343.5±95.3	364.8±174.6	0.93
Baseline CCT (μm), mean±SD	350.3±80.6	305.9±102.1	369.8±62.0	0.050
Baseline CFT in fellow eye (μm), mean±SD	200.7±45.9 (n=33)	194.5±34.4 (n=10)	203.4±50.6 (n=23)	0.68
Baseline CCT in fellow eye (μm), mean±SD	291.9±51.0 (n=33)	273.7±39.6 (n=10)	299.8±54.1 (n=23)	0.26
Month 3 CFT (μm), mean±SD	234.2±79.3	232.5±87.8	235.0±77.2	0.76
Month 3 CCT (μm), mean±SD	333.4±74.4	301.1±91.0	347.6±62.7	0.18
Month 3 CCFD%, mean±SD	35.8±2.2	36.6±1.9	35.5±2.3	0.027

Abbreviations: MNV, macular neovascularization; CFT, central foveal thickness; CCT, central choroidal thickness; CCFD%, choriocapillaris flow deficit percentage.

predisposition; thus, further investigation is warranted to clarify risk factors for MNV development, including potential genetic influences.

We acknowledge several limitations in this study. First, the retrospective design and small sample size may limit generalizability of our findings. In particular, the small number of eyes that developed MNV may have reduced the statistical power of more complex analyses. Due to the limited sample size and potential model overfitting, we therefore focused on univariate analyses in this study. Additionally, MNV may have developed in patients without MNV at the final follow-up if they had been observed for a longer duration. Previous studies have shown that secondary MNV prevalence in resolved CSC ranges from 5.8% to 24.0%.[1,5,22,24] In contrast, the prevalence of MNV in our cohort was 30.6%, which is substantially higher than previously reported rates. Second, OCTA device and data quality might affect the result. Segmentation errors and image artifacts remain a concern in OCTA quantification. In addition, from a clinical perspective, the identification of a potential threshold of choriocapillaris flow deficits may be useful for risk stratification of MNV development in CSC. However, quantitative CCFD measurements can vary depending on the OCTA device, image acquisition protocols, and binarization or post-processing methods; therefore, we considered that generalizing a specific cutoff value should be approached with caution at this stage. Further validation using standardized imaging

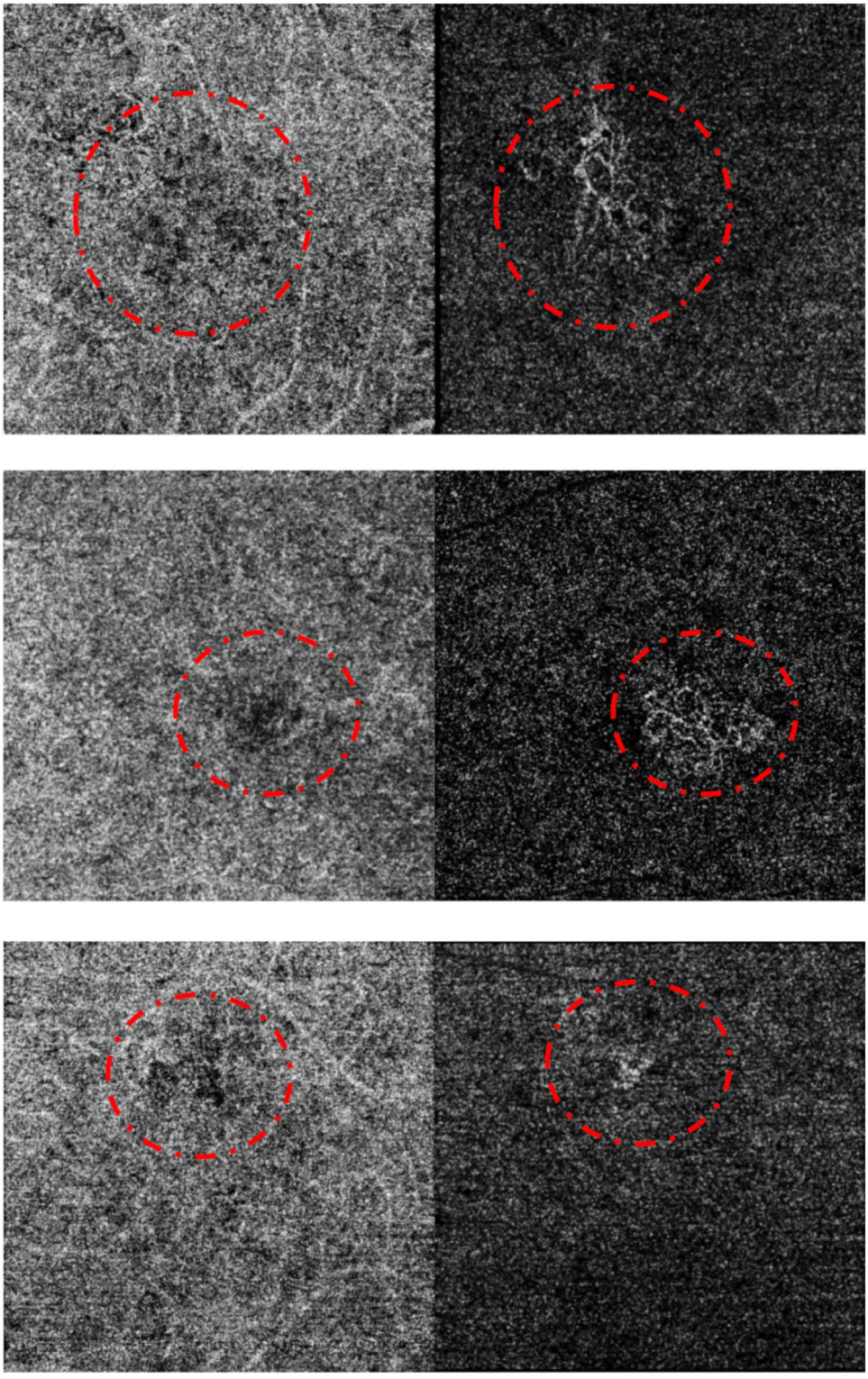

Figure 3 Spatial correspondence between early choriocapillaris flow deficits and future macular neovascularization (MNV). Choriocapillaris images at 3 months post-treatment (left panels) and outer retinal slab OCTA images at the final visit showing MNV (right panels) are shown. Red circles denote areas of reduced choriocapillaris flow that spatially correspond to later MNV development.

protocols and larger prospective cohorts is required before CCFD-based thresholds can be applied in routine clinical practice. Despite these limitations, the strong spatial correlation and statistical significance indicate that CCFD evaluation merits further study.

Conclusions

In conclusion, greater choriocapillaris flow deficits at early post-treatment may predict higher risk of MNV in CSC. OCTA-derived CCFD% is a promising metric to guide risk stratification and monitoring in this patient population.

Data Sharing Statement

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Ethics Approval and Informed Consent

This study was approved by the Institutional Review Board of Hyogo Medical University Hospital (approval number: 2426) and was conducted in accordance with the tenets of the Declaration of Helsinki. The requirement for individual informed consent was waived by the committee because of the retrospective observational study design. All patient data were anonymized prior to analysis, and confidentiality of patient information was strictly maintained throughout the study.

Acknowledgments

The authors would like to thank all members of the Department of Ophthalmology, Hyogo Medical University, for their support in data collection and patient care. This work was presented at the American Academy of Ophthalmology (AAO) Annual Meeting, 2024.

Author Contributions

N.K. designed the study, collected and analyzed the data, and drafted the manuscript. H.F. reviewed the results and critically revised the manuscript for important intellectual content. F.G. provided final approval of the version to be published. Y.Y. contributed to data collection. All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Funding

The sponsor or funding organization had no role in the design or conduct of this research.

Disclosure

The authors report no conflicts of interest in this work.

References

1. Spaide RF, Campeas L, Haas A, et al. Central serous chorioretinopathy in younger and older adults. *Ophthalmology*. 1996;103(12):2070–2079. doi:10.1016/s0161-6420(96)30386-2
2. Kitzmann AS, Pulido JS, Diehl NN, Hodge DO, Burke JP. The incidence of central serous chorioretinopathy in Olmsted County, Minnesota, 1980–2002. *Ophthalmology*. 2008;115(1):169–173. doi:10.1016/j.ophtha.2007.02.032
3. Laatikainen L. Diffuse chronic retinal pigment epitheliopathy and exudative retinal detachment. *Acta Ophthalmol*. 1994;72(5):533–536. doi:10.1111/j.1755-3768.1994.tb07175.x
4. Mrejen S, Balaratnasingam C, Kaden TR, et al. Long-term visual outcomes and causes of vision loss in chronic central serous chorioretinopathy. *Ophthalmology*. 2019;126(4):576–588. doi:10.1016/j.ophtha.2018.12.048
5. Fung AT, Yannuzzi LA, Freund KB. Type 1 (sub-retinal pigment epithelial) neovascularization in central serous chorioretinopathy masquerading as neovascular age-related macular degeneration. *Retina*. 2012;32(9):1829–1837. doi:10.1097/IAE.0b013e3182680a66
6. Bonini FMA, de Carlo TE, Ferrara D, et al. Association of choroidal neovascularization and central serous chorioretinopathy with optical coherence tomography angiography. *JAMA Ophthalmol*. 2015;133(8):899–906. doi:10.1001/jamaophthalmol.2015.1320
7. McClintic SM, Jia Y, Huang D, Bailey ST. Optical coherence tomographic angiography of choroidal neovascularization associated with central serous chorioretinopathy. *JAMA Ophthalmol*. 2015;133(10):1212–1214. doi:10.1001/jamaophthalmol.2015.2126
8. Kaye R, Chandra S, Sheth J, Boon CJF, Sivaprasad S, Lotery A. Central serous chorioretinopathy: an update on risk factors, pathophysiology and imaging modalities. *Prog Retin Eye Res*. 2020;79:100865. doi:10.1016/j.preteyeres.2020.100865
9. Warrow DJ, Hoang QV, Freund KB. Pachychoroid pigment epitheliopathy. *Retina*. 2013;33(8):1659–1672. doi:10.1097/IAE.0b013e3182953df4

10. Pang CE, Freund KB. Pachychoroid neovasculopathy. *Retina*. 2015;35(1):1–9. doi:10.1097/IAE.0000000000000331
11. Cheung CMG, Lee WK, Koizumi H, Dansingani K, Lai TYY, Freund KB. Pachychoroid disease. *Eye*. 2019;33(1):14–33. doi:10.1038/s41433-018-0158-4
12. Forsythe JA, Jiang BH, Iyer NV, et al. Activation of vascular endothelial growth factor gene transcription by hypoxia-inducible factor 1. *Mol Cell Biol*. 1996;16(9):4604–4613. doi:10.1128/mcb.16.9.4604
13. Scharf JM, Corradetti G, Alagorie AR, et al. Choriocapillaris flow deficits and treatment-naïve macular neovascularization secondary to age-related macular degeneration. *Invest Ophthalmol Vis Sci*. 2020;61(11):11. doi:10.1167/iovs.61.11.11
14. Nesper PL, Ong JX, Fawzi AA. Exploring the relationship between multilayered choroidal neovascularization and choriocapillaris flow deficits in AMD. *Invest Ophthalmol Vis Sci*. 2021;62(3):12. doi:10.1167/iovs.62.3.12
15. Rochepeau C, Kodjikian L, Garcia MA, et al. Optical coherence tomography angiography quantitative assessment of choriocapillaris blood flow in central serous chorioretinopathy. *Am J Ophthalmol*. 2018;194:26–34. doi:10.1016/j.ajo.2018.07.004
16. Ruggeri ML, Passamonti M, Quarta A, et al. Choroidal and choriocapillaris OCT-A analysis in patients affected by active central serous chorioretinopathy. *Transl Vis Sci Technol*. 2024;13(12):14. doi:10.1167/tvst.13.12.14
17. Chu Z, Zhang Q, Gregori G, Rosenfeld PJ, Wang RK. Guidelines for imaging the choriocapillaris using OCT angiography. *Am J Ophthalmol*. 2021;222:92–101. doi:10.1016/j.ajo.2020.08.045
18. Cheng W, Song Y, Lin F, et al. Choriocapillaris flow deficits in normal Chinese imaged by swept-source optical coherence tomographic angiography. *Am J Ophthalmol*. 2022;235:143–153. doi:10.1016/j.ajo.2021.09.018
19. Yanagi Y. Pachychoroid disease: a new perspective on exudative maculopathy. *Jpn J Ophthalmol*. 2020;64(4):323–337. doi:10.1007/s10384-020-00740-5
20. Kishi S, Matsumoto H. A new insight into pachychoroid diseases: remodeling of choroidal vasculature. *Graefes Arch Clin Exp Ophthalmol*. 2022;260(11):3405–3417. doi:10.1007/s00417-022-05687-6
21. Zhou X, Komuku Y, Araki T, et al. Risk factors and characteristics of central serous chorioretinopathy with later development of macular neovascularisation detected on OCT angiography: a retrospective multicentre observational study. *BMJ Open Ophthalmol*. 2022;7(1):e000976. doi:10.1136/bmjophth-2022-000976
22. Borrelli E, Battista M, Gelormini F, et al. Rate of misdiagnosis and clinical usefulness of the correct diagnosis in exudative neovascular maculopathy secondary to AMD versus pachychoroid disease. *Sci Rep*. 2020;10(1):20344. doi:10.1038/s41598-020-77566-1
23. Morino K, Miyake M, Kamei T, et al. Association between central serous chorioretinopathy susceptibility genes and choroidal parameters. *Jpn J Ophthalmol*. 2022;66(6):504–510. doi:10.1007/s10384-022-00945-w
24. Hage R, Mrejen S, Krivosic V, Quentel G, Tadayoni R, Gaudric A. Flat irregular retinal pigment epithelium detachments in chronic central serous chorioretinopathy and choroidal neovascularization. *Am J Ophthalmol*. 2015;159(5):890–903.e3. doi:10.1016/j.ajo.2015.02.002