• Clinical Research •

Reduced choroidal vascular index and choroid structural changes extended beyond subfoveal area in chronic central serous chorioretinopathy eyes with macular neovascularization

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Abstract

● **AIM:** To investigate the choroidal vascular index (CVI) and the choroidal structural changes beyond the subfoveal area (analyzed across a 20 mm×24 mm scanning area) in eyes with chronic central serous chorioretinopathy (cCSC) eyes with macular neovascularization (MNV) using ultra-widefield swept-source optical coherence tomography angiography (UWF SS-OCTA).

● **METHODS:** This retrospective comparative study included 46 cCSC with MNV eyes (With MNV group), 52 cCSC without MNV eyes (Without MNV group), and 40 age-matched healthy controls. UWF SS-OCTA imaging with a 20 mm×24 mm protocol was used to quantify CVI across 9 subfields (superotemporal, superior, superonasal, temporal, central, nasal, inferotemporal, inferior, and inferonasal). The CVI was compared among the groups.

● **RESULTS:** With MNV group demonstrated significantly older mean age than Without MNV group (56.2±6.1 *vs* 47.5±8.6y, $P<0.001$). The CVI was significantly lower in the With MNV group than in the Without MNV group, except in the superotemporal, superior, and temporal regions (all $P<0.05$). Notably, despite MNV-associated CVI reductions, the With MNV group maintained a higher CVI than the control group in all 5 subfields (superior, temporal, central, inferior, and inferonasal; all $P<0.05$). In the central region, the CVI (%) in With MNV, Without MNV, and control groups were 35.63±3.33, 37.37±2.07, and 32.67±5.00 ($P<0.05$), respectively.

● **CONCLUSION:** CVI decreases, and choroidal structural changes extend beyond the subfoveal area in cCSC with MNV eyes, providing with an imaging evidence for the important role of choroidal ischemia in the pathogenesis of MNV in cCSC.

● **KEYWORDS:** central serous chorioretinopathy; macular neovascularization; choroidal vascular index; choroidal thickness; optical coherence tomography angiography
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INTRODUCTION

Central serous chorioretinopathy (CSC) is characterized by idiopathic serous neuroepithelial detachment with defects in the outer blood-retina barrier of retinal pigment epithelium (RPE), which occurs secondary to choroidal abnormalities and dysfunction[1]. Emerging evidence has established a pivotal role for pachychoroid phenotypes in CSC pathophysiology[1-2]. First conceptualized by Warrow *et al*[3] in 2013, pachychoroid is defined as a subfoveal choroidal thickness (SFCT) exceeding 300 μm. The current understanding extends beyond anatomical thickening to include pathological hallmarks, such as mid-to-large choroidal venous dilation and choroidal capillary attenuation, which can be accompanied by progressive RPE dysfunction and neovascularization[4-5].

Macular neovascularization (MNV), a vision-threatening complication of chronic CSC (cCSC), exhibits a heightened prevalence in patients with a disease duration exceeding

six months, correlating positively with disease severity[1,6-7]. Notably, advancements in optical coherence tomography angiography (OCTA) have revealed an MNV incidence rate of 39.2% in cCSC cohorts[8], suggesting a greater prevalence than previously documented[9].

Pathophysiologically, patients with cCSCs demonstrate significantly increased choroidal thickness (ChT) compared to healthy controls, while cCSC with MNV eyes exhibit distinct structural alterations[7-8]. Multicenter studies indicated reduced ChT in cCSC with MNV eyes relative to cCSC without MNV eyes, implying potential choroidal remodeling during neovascularization[8,10]. However, the utility of ChT as a dynamic biomarker is limited by confounding physiological factors such as age, axial length (AL), and blood pressure[11-12]. In contrast, the choroidal vascular index (CVI), quantified as the ratio of choroidal vascular lumen volume to the total choroidal volume, has emerged as a robust biomarker of pachychoroid spectrum diseases. CVI's superior stability, independence from age, systolic blood pressure, AL, or intraocular pressure[13-15], and capacity to quantify vascular density, render it particularly valuable for CSC research[16-17].

Recent evidence suggests that elevated CVI values in patients with CSC compared to healthy controls are attributable to choroidal vascular dilation and hyperpermeability[18]. Nevertheless, the choroidal vascular architecture in cCSC with MNV eyes remains underexplored, particularly the correlation between CVI and MNV. Traditional imaging modalities constrained by limited scan areas predominantly focus on macular ChT measurements[13]. Breakthroughs in ultra-widefield swept-source OCTA (UWF SS-OCTA) enable comprehensive visualization and volumetric quantification of the full-thickness choroidal vasculature, facilitating the three-dimensional reconstruction of choroidal vascular networks[19-20]. This study used UWF SS-OCTA to quantitatively compare CVI parameters between cCSC with and without MNV eyes. It will be helpful to clarify the pathological characteristics of choroidal vessels in cCSC with MNV eyes, explore the potential causes of MNV, and provide a new direction for the prevention and treatment of cCSC with MNV eyes in the future.

PARTICIPANTS AND METHORDS

Ethical Approval This cross-sectional study was conducted in accordance with the Declaration of Helsinki and approved by the Ethical Review Committee of the Central Theater General Hospital (Ethics No.[2024]015-01). The requirement for informed consent was waived due to the retrospective nature of this study.

Enrollment of Participants Between April 2023 and February 2024, we consecutively enrolled 98 patients diagnosed with cCSC. Of these, 18 exhibited bilateral involvement. For patients with bilateral lesions, we chose the right eye to avoid a within-subject bias. A normal group of age-matched right eyes with a normal fundus was also included.

CSC was diagnosed based on the presence of serous detachment of the neurosensory retina involving the macula, as demonstrated by optical coherence tomography (OCT), and leakage at the RPE level on fundus fluorescein angiography (FFA). cCSC is defined as a condition in which subretinal fluid persists for more than 3-4mo and may lead to permanent RPE atrophy and/or photoreceptor damage. Only cCSC eyes with AL from 22-26 mm and without anti-vascular endothelial growth factor or photodynamic therapies were included in this study.

Participants were excluded if they met the following criteria: 1) presence of drusen in the macular area; 2) glaucoma, diabetic retinopathy, exudative age-related macular degeneration (AMD), central retinal vein occlusion, retinal detachment, or any other eye diseases that may affect the vision of the study eye; 3) previous retinal laser photocoagulation or vitrectomy; 4) history of cataract surgery within the past 6mo; 5) image quality of OCT or OCTA<7; 6) images with serious artifacts preventing accurate analyzed.

Methods All patients underwent a full ophthalmic examination, including measurement of best-corrected visual acuity (BCVA) converted to the logarithm of the minimum angle of resolution (logMAR), intraocular pressure, slit-lamp examination, indirect ophthalmoscopy, fundus photography, UWF SS-OCT/SS-OCTA, FFA, and indocyanine green angiography (ICGA) examination. Baseline demographic data (sex, age, and history of hypertension) and ophthalmic examination results were collected.

The patients were divided into cCSC with MNV (With MNV group) and cCSC without MNV groups (Without MNV group). MNV can be detected using a combination of OCT, OCTA, FFA, and ICGA, although conclusive detection can be challenging[1]. Two types of MNV were identified in our study: type 1 [also known as polypoidal choroidal vasculopathy (PCV), as a special subtype] in the sub-RPE space, and type 2 crossing the RPE in the subretinal space.

The participants underwent imaging using a 400 kHz UWF SS-OCTA instrument (BM400K, Towardpi Medical Technology Co., Ltd., Beijing, China). The imaging protocol involved a raster scan, which included 1536 (horizontal) A-scans and 1280 (vertical) B-scans. This scan covered an area of 24 mm×20 mm and a depth of 6 mm, with the fovea as the central point of focus. The built-in software automatically identified both the Bruch's membrane and the choroid-sclera interface, which were manually verified if necessary.

The CVI can be automatically measured and computed using built-in software for 9 subfields (superotemporal, temporal,

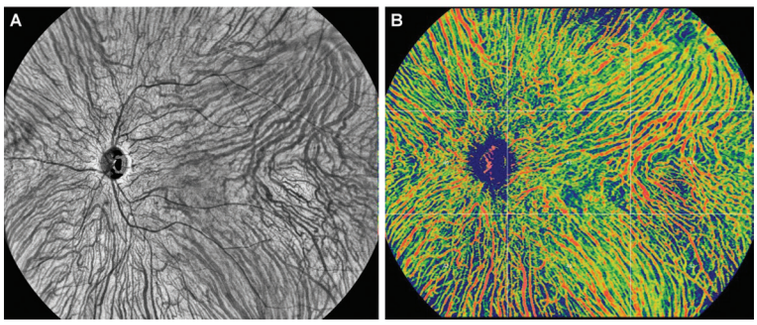

Figure 1 Ultra-widefield swept-source optical coherence tomography images A: An enface image; B: The choroidal vascularity index values in 9 subfields (superotemporal, temporal, inferotemporal, superior, central, inferior, superonasal, nasal, inferonasal).

Table 1 Clinical data in eyes of cCSC with/without MNV and control groups mean±SD

Characteristics	With MNV group (*n*=46)	Without MNV group (*n*=52)	Control group (*n*=40)	*P*	*P*1	*P*2	*P*3
Male/female	40/6	41/11	32/8	0.247	-	-	-
Age, y	56.2±6.1	47.5±8.6	54.0±9.0	<0.001	<0.001[a]	0.164[a]	<0.001[a]
Hypertension, *n* (%)	13 (28.3)	15 (28.8)	12 (30.0)	0.644	-	-	-
Duration, mo	39.8±9.2	15.1±4.5	-	<0.001	<0.001[a]	-	-
AL, mm	23.22±1.32	23.48±1.54	23.24±1.24	0.759	-	-	-
BCVA, logMAR	0.38±0.18	0.29±0.07	0.10±0.08	<0.001	<0.001[a]	<0.001[a]	<0.001[a]
SFCT, μm	298.22±79.42	362.45±83.02	255.82±81.13	<0.001[a]	<0.001	0.054	<0.001
Pachychoroid, *n* (%)	46 (100)	52 (100)	12 (30.0)	<0.001	>0.05[a]	<0.05[a]	<0.05[a]
MNV type (1/2)	44/2	-	-	-	-	-	-

cCSC: Chronic central serous chorioretinopathy; MNV: Macular neovascularization; SD: Standard deviation; AL: Axial length; BCVA: Best-corrected visual acuity; logMAR: Logarithm of the minimum angle of resolution; SFCT: Subfovel choroidal thickness. *P*: Chi-square or analysis of variance test. *P*1: With MNV group *vs* Without MNV group; *P*2: With MNV group *vs* control group; *P*3: Without MNV group *vs* control group. [a]Post hoc pairwise comparisons were performed using Bonferroni's method.

inferotemporal, superior, central, inferior, superonasal, nasal, and inferonasal; Figure 1). It is worth noting that the principle of CVI measurement is based on cross-sectional scanning images; however, the instrument itself has an automatic quantification function for the CVI. To better illustrate the CVI results in different regions, we used en-face scanning images for display, which is one of the advantages of using UWF SS-OCTA. All image assessments were performed by two experienced independent retinal specialists. Disagreements regarding image interpretation were resolved by open adjudication.

Statistical Analysis All statistical analyses were performed using SPSS software (version 25.0; IBM, Chicago, USA). Continuous variables were compared using analysis of variance for multiple group comparisons and Bonferroni's method for post-hoc pairwise comparisons when the parameters followed a normal distribution. For parameters that did not follow a normal distribution, the Kruskal-Wallis test was used for multiple group comparisons and post hoc pairwise comparisons. Categorical variables were compared using the Chi-squared test. For the correlation of the CVI with the SFCT and With MNV group, Spearman correlation analysis was used. *P*-values of <0.05 were considered statistically significant.

RESULTS

General Characteristics A total of 98 eyes with cCSC from 98 patients were included in this study. Among them, 46 eyes of 46 patients were in the With MNV group (Figure 2), and 52 eyes of 52 patients were in Without MNV group (Figure 3). The incidence of MNV in cCSC eyes was 43.10%. Forty eyes of 40 age-matched healthy subjects were included in the control group. The demographic and clinical characteristics of the patients were summarized in Table 1.

With MNV group were significantly older aged (56.2±6.1 *vs* 47.5±8.6, *P*<0.001), with worse visual acuity (0.38±0.18 logMAR *vs* 0.29±0.07 logMAR, *P*<0.001), longer duration (months) of diseases (39.8±9.2 *vs* 15.1±4.5, *P*<0.001) and thinner SFCT (298.22±79.42 *vs* 362.45±83.02 μm, *P*<0.001) than that of the Without MNV group. There were no statistical differences between the groups regarding the history of hypertension and AL. All eyes with cCSC and 12 (30.0%) eyes in the control group had a pachychoroid. In the With MNV group, 46 (95.7%) eyes had type 1 MNV, and 2 (4.3%) eyes had type 2 MNV. No PCV was observed in type 1 MNV in the With MNV group. No differences in detection indices were found between the two types of MNV; thus, they were assigned to the With MNV group.

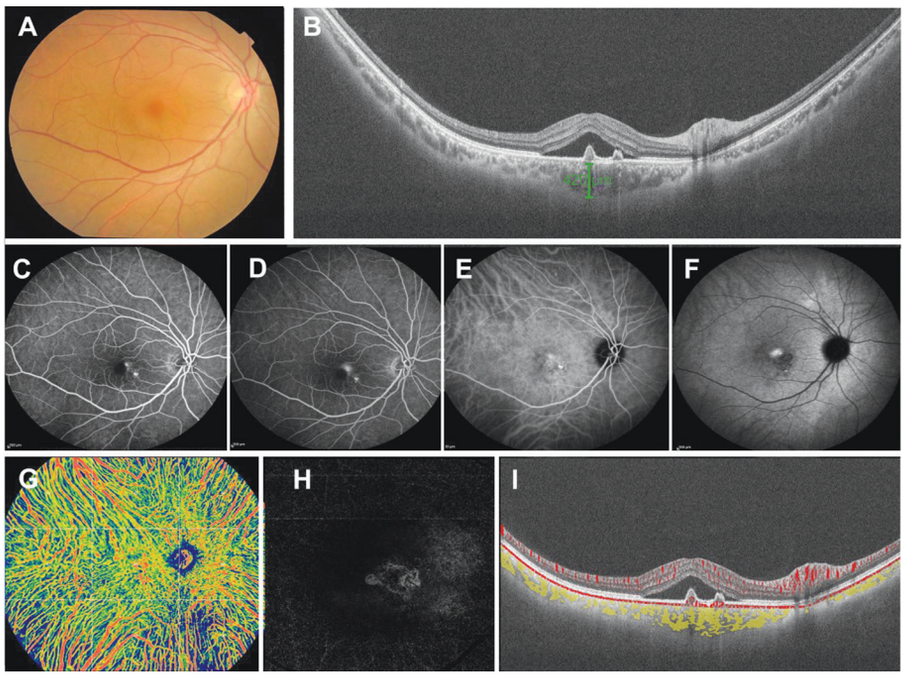

Figure 2 Representative case of cCSC with MNV A 52-year-old man was diagnosed with cCSC with MNV in the right eye. Fundus photograph shows serous retinal detachment in the central macula (A). OCT image shows subretinal neuroepithelial fluid, irregular RPE bulge, and PED formation with SFCT of 420 μm (B). FFA images in the middle (C) and late (D) stages show smoke-stack fluorescence leakage. In the middle (E) and late (F) ICGA images, neovascular-like hyperfluorescence can be observed. OCTA images show CVI of 35% in the central region (G) and choroidal neovascularization in the avascular layer of the retina (H). OCTA B-scan image shows blood flow signals in the PED (I). cCSC: Chronic central serous chorioretinopathy; MNV: Macular neovascularization; OCT: Optical coherence tomography; RPE: Retinal pigment epithelium; PED: Pigment epithelium detachment; SFCT: Subfoveal choroidal thickness; FFA: Fundus fluorescein angiography; ICGA: Indocyanine green angiography; OCTA: Optical coherence tomography angiography; CVI: Choroidal vascular index.

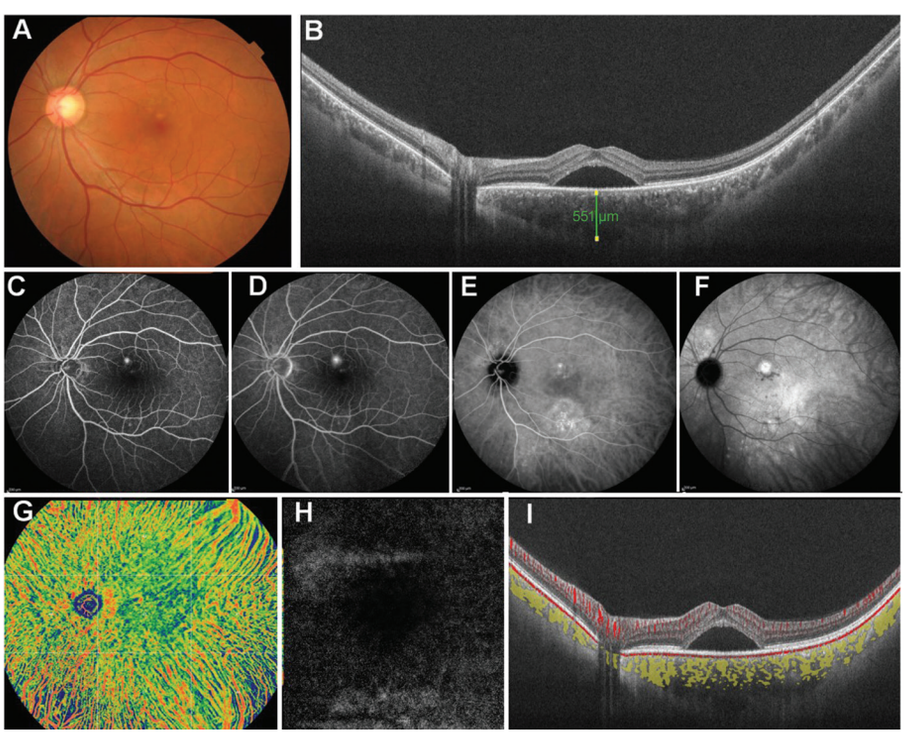

Figure 3 Representative case of cCSC without MNV A 44-year-old man was diagnosed with chronic recurrent CSC in the left eye. Fundus photograph shows serous retinal detachment in the central macula (A). OCT image shows a subretinal neuroepithelial fluid collection with SFCT of 551 μm (B). FFA images in the middle (C) and late (D) stages show smoke-stack fluorescence leakage. In the middle (E) and late (F) ICGA images, multifocal areas of choroidal hyperpermeability are seen. OCTA image shows CVI of 43% in the central region (G). No MNV can be observed on OCTA (H and I). cCSC: Chronic central serous chorioretinopathy; MNV: Macular neovascularization; OCT: Optical coherence tomography; SFCT: Subfoveal choroidal thickness; FFA: Fundus fluorescein angiography; ICGA: Indocyanine green angiography; OCTA: Optical coherence tomography angiography; CVI: Choroidal vascular index.

Table 2 Comparisons of choroidal vascularity index in 9 subfields among cCSC with/without MNV and control eyes %, mean±SD

Subfields	With MNV group	Without MNV group	Control group	*P*	*P*1	*P*2	*P*3
Superotemporal	34.74±3.96	36.50±4.01	33.18±4.41	0.001	0.086	0.417	0.001
Superior	35.85±3.41	37.31±3.70	32.13±5.34	<0.001[a]	0.194	0.005	<0.001
Superonasal	33.91±4.07	36.89±3.78	30.36±6.48	<0.001[a]	0.002	0.084	<0.001
Temporal	31.72±3.00	33.46±3.59	28.77±4.99	<0.001[a]	0.075	0.018	<0.001
Central	35.63±3.33	37.37±2.07	32.67±5.00	<0.001[a]	0.011	0.016	<0.001
Nasal	31.61±5.34	34.54±3.82	27.31±7.99	<0.001[a]	0.011	0.054	<0.001
Inferotemporal	30.85±4.98	34.07±2.97	28.77±5.82	<0.001[a]	0.006	0.074	<0.001
Inferior	32.67±5.53	36.33±4.30	29.21±6.45	<0.001[a]	0.004	0.047	<0.001
Inferonasal	26.24±5.83	31.89±5.50	21.26±7.39	<0.001[a]	0.000	0.028	<0.001
Average	32.43±3.02	34.78±3.06	29.33±4.94	<0.001[a]	0.003	0.028	<0.001

cCSC: Chronic central serous chorioretinopathy; MNV: Macular neovascularization; SD: Standard deviation. [a]Kruskal-Wallis test; Post hoc pairwise comparisons were performed using Kruskal-Wallis test for parameters with nonnormal distribution. *P*1: With MNV group *vs* without MNV group; *P*2: With MNV group *vs* control group; *P*3: Without MNV group *vs* control group.

Table 3 Correlation of CVI of the central region with SFCT and with MNV group

Groups	CVI	SFCT	*r*	*P*
All subjects (*n*=139)	35.47±3.97	309.11±85.37	0.657	<0.001
With MNV group (*n*=46)	35.63±3.33	302.07±78.36	0.625	<0.001
Without MNV group (*n*=52)	37.37±2.07	353.28±77.64	0.245	0.047
Control group (*n*=40)	32.67±5.00	256.26±71.34	0.839	<0.001
With MNV group (Without MNV group=0, With MNV group=1)	-	-	-0.310	<0.001

CVI: Choroidal vascularity index; SFCT: Subfoveal choroidal thickness; MNV: Macular neovascularization.

Comparison of CVI Among Groups Table 2 summarized the comparisons of the CVI in the With MNV, Without MNV, and control groups. The With MNV group showed significantly higher CVI values than the control group for all 9 subfields (all $P<0.05$). The CVI was significantly lower in the With MNV group than in the Without MNV group, except in the superotemporal, superior, and temporal regions (all $P<0.05$). However, in the superior, temporal, central, inferior, and inferior nasal regions, the CVI in With MNV group remained higher than that in the control group (all $P<0.05$). Among the 9 subfields in the 3 groups, the CVI was relatively lower in the inferonasal region.

Furthermore, the CVI (%) of the central region in With MNV, Without MNV, and control groups were 35.63±3.33, 37.37±2.07, and 32.67±5.00 ($P<0.001$; Table 2). The CVI in the Without MNV group was approximately 14.4% higher than that in the control group, and that in the With MNV group was approximately 4.7% lower than that in the Without MNV group. However, the CVI of the With MNV group was approximately 9.1% higher than that of the control group.

Correlation of CVI of the Central Region with SFCT and With MNV Group Table 3 represented the correlation between the CVI of the central region and the SFCT and With MNV group. The CVI of the central region was significantly correlated with the SFCT in all subjects ($r=0.657$, $P<0.001$), With MNV ($r=0.625$, $P<0.001$), Without MNV ($r=0.245$, $P<0.001$), and control groups ($r=0.839$, $P<0.001$). Further analysis of the correlation between the CVI of the central region and the With MNV groups (Without MNV group=0, With MNV group=1) showed that the CVI in the central region was related to the With MNV group ($r=-0.310$, $P<0.001$).

DISCUSSION

This retrospective study analyzed choroidal vascular characteristics in 98 eyes with cCSC, and revealed significantly lower CVI in cCSC eyes with MNV(With MNV group) than in those without MNV (Without MNV group). Notably, structural alterations extend beyond the subfoveal region, a finding that not only corroborates but also substantially expands upon previous small-scale studies. Crucially, our quantitative CVI measurements provide compelling evidence for the pivotal role of choroidal ischemia in MNV development secondary to cCSC.

The marked reduction in CVI in the With MNV group suggests a direct association between diminished choroidal vascular components and MNV pathogenesis. Of particular significance, our study identified diffuse choroidal structural remodeling in eyes with MNV, exceeding previously reported macular-confined changes[13]. The implementation of 24 mm×20 mm widefield imaging provided novel insights into pan-choroidal alterations in cCSC with MNV, offering a more comprehensive perspective than conventional macular-focused assessments. While these findings parallel the CVI reduction observed in exudative AMD[21-22], the ischemic etiology of cCSC with

MNV remains pathologically unverified and requires further histopathological validation[13].

Our findings reinforce the "pachychoroid-driven pathogenesis" hypothesis in cCSC with MNV eyes. As a key component of the pachychoroid disease spectrum, cCSC manifests pathological hallmarks, including outer choroidal vascular dilation with concomitant capillary layer atrophy[23]. Chronic ischemia resulting from such structural derangements may trigger compensatory neovascularization[24]. The observed pan-choroidal CVI reduction not only substantiates choroidal hypoperfusion, but also implies that vascular alterations may precede MNV formation and perpetuate disease progression. This mechanism complements Bruch's membrane rupture theory, wherein prolonged RPE detachment and subretinal fluid accumulation synergistically promote MNV through mechanical barrier disruption and hypoxic microenvironmental changes[25-26].

Notably, while all cCSC groups exhibited elevated CVI compared with healthy controls, reflecting inherent choroidal venous stasis and vascular dilation[27-29] and the With MNV group demonstrated significantly lower CVI than the Without MNV group. This suggests that MNV-associated vascular remodeling exacerbates pathological changes, though it is insufficient to fully counteract primary choroidal dilatation in the central regions. Furthermore, CVI's superior clinical utility of CVI over SFCT is evident through its strong MNV correlation and reduced susceptibility to confounding factors[30-31], making it a promising biomarker for cCSC-MNV assessment.

Our study has some limitations. The single-center retrospective design and modest sample size may have limited the generalizability of the results. This cross-sectional nature precludes the determination of temporal relationships between CVI changes and MNV formation. Future multicenter longitudinal studies should investigate: 1) dynamic CVI evolution during cCSC-to-MNV progression, 2) CVI response patterns post-MNV treatment, 3) regional CVI gradient characteristics. Such investigations could establish CVI's prognostic value of the CVI in clinical practice.

In conclusion, through wide-field quantitative choroidal analysis, this study demonstratesd extensive extramacular CVI reduction in cCSC-MNV eyes, providing critical imaging evidence for the central role of choroidal ischemia in MNV pathogenesis. These findings advance our understanding of the pachychoroid disease mechanisms and highlight the potential of CVI as a novel biomarker for early MNV detection.

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