• Brief Report •

Clinical and multimodal imaging characteristics of acute central serous chorioretinopathy with fibrinous exudation

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Abstract

● **AIM:** To analyze the clinical and multimodal imaging characteristics of acute central serous chorioretinopathy (CSC) with or without fibrinous exudation.

● **METHODS:** Retrospective case-control study. Patients diagnosed with acute CSC with fibrinous exudation (FE group) and without fibrinous exudation (non-FE group) were consecutively included. The clinical data and multimodal images including color fundus photography, fundus autofluorescence, spectral-domain optical coherence tomography (OCT), fundus fluorescein angiography, and indocyanine green angiography at presentation were recorded. Treatment method, follow-up outcomes including best-corrected visual acuity (BCVA) and OCT characteristics were also documented.

● **RESULTS:** The FE group (n=8, 8 eyes, 6 males) had a mean age of 47.50±7.27y, and the median symptom duration was 26.50d. The non-FE group (n=20, 20 eyes, 16 males) had a mean age of 40.40±4.36y, and the median symptom duration was 7.00d. Compared to the non-FE group, the FE group exhibited significantly older age (P=0.004), longer self-reported symptom duration (P=0.02), and poorer baseline and follow-up BCVA (P=0.011, P=0.003). After more than one month follow-up, visual improvement was statistically significant in the non-FE group (P=0.017), but not in the FE group (P=0.157). Multimodal imaging results found higher prevalence of pigment epithelial detachment (PED; P=0.029) and larger subfoveal choroidal thickness (P<0.001) in the FE group, while there was no significant difference in central macular thickness between the groups. The leakage start time was earlier in the FE group (14.50±2.33s) than in the non-FE group (22.67±1.24s, P<0.001). The expanding dot sign was the most common leakage pattern of fibrinous CSC. Late leakage area usually expanded to more than 1/2 disc diameter (DD) in the FE group, while it was less than 1/2 DD in the non-FE group.

● **CONCLUSION:** Fibrinous exudation in acute CSC is a multimodal imaging biomarker indicative of severe choroidal vasculopathy and retinal pigment epithelium barrier failure. Recognizing this entity and understanding its potential mechanisms are crucial for managing patient prognosis and may guide future targeted interventions.

● **KEYWORDS:** central serous chorioretinopathy; fibrinous exudation; multimodal imaging
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INTRODUCTION

Central serous chorioretinopathy (CSC) is a type of choroidal retinopathy characterized by serous neurosensory detachment frequently involving the region of the macula with or without focal retinal pigment epithelial detachment (PED). It most commonly affects young and middle-aged men[1]. Typical symptoms include blurring of vision, relatively central scotoma, metamorphopsia, moderate dyschromatopsia, hypermetropia, micropia and reduced contrast sensitivity[2-3]. Although the exact pathogenesis of the disease is not fully understood, choroidal vascular hyperpermeability and dysfunction of retinal pigment epithelium (RPE) are the predominant pathophysiological bases[4].

Subretinal fibrinous exudation is uncommon in CSC, and has previously been reported mainly in cases secondary to pregnancy or long-term steroid usage[5-6]. The presence

of fibrin, a large molecule, possibly suggests a significant disruption to the normal physiology of the choriocapillaris and the RPE layer[7]. Previous studies have found that fibrinous exudation may exacerbate photoreceptor damage by exerting mechanical compression or triggering inflammatory responses. It is also associated with adverse outcomes such as disease chronicity, choroidal neovascularization or subretinal fibrosis[8]. Multimodal imaging techniques, such as color fundus photography (CFP), spectral-domain optical coherence tomography (SD-OCT), fundus autofluorescence (FAF), fundus fluorescein angiography (FFA), and indocyanine green angiography (ICGA) can reveal the characteristics of CSC lesions more accurately, presenting leakage points and choroidal vessel changes. The imaging features of fibrinous exudation in CSC are distinctive, presenting as large, yellow-white lesions on CFP and as subretinal hyperreflective material (SRHM) on OCT[9]. The existing literature has primarily focused on the overall characteristics of CSC, but a comprehensive analysis of the clinical and imaging features of CSC with fibrinous exudation is lacking. In this study, we retrospectively compared clinical and multimodal imaging characteristics of acute CSC with or without subretinal fibrin exudates, and investigated the underlying potential pathological mechanisms.

PARTICIPANTS AND METHODS

Ethical Approval This retrospective case-control study was approved by the Ethics Committee of Tianjin Medical University Eye Hospital (No.2025KY-29) and adhered to the tenets of the Declaration of Helsinki. Due to the retrospective nature of the study and the high rate of lost to follow-up, the research ethics board did not require consent from each individual patient. This study was retrospectively registered with the Chinese Clinical Trial Registry. Trial registration number: ChiCTR2500103950. Date of registration: June 9, 2025.

Patient Eligibility and Data Collection We conducted a retrospective chart review of all patients diagnosed with acute CSC with or without fibrinous exudation at Tianjin Medical University Eye Hospital from October 2020 to October 2023. The clinical diagnosis of acute CSC was based on the presence of typical clinical symptoms (visual loss associated with dimming, metamorphopsia or central scotoma) lasting less than 3mo and imaging characteristics including localized serous neurosensory detachment associated with focal leaks of RPE. Fibrinous exudation was defined as yellowish subretinal deposits observed on CFP or hyperreflective material deposits seen on OCT[10]. Patients diagnosed with choroidal neovascularization, other macular diseases including age-related macular degeneration, polypoidal choroidal vasculopathy, diabetic retinopathy, and chronic CSC were excluded.

Data collected included gender, age, symptom duration, baseline best-corrected visual acuity (BCVA), and clinical features at presentation. BCVA was converted to a logarithm of the minimum angle of resolution (logMAR). All patients underwent slit-lamp examination, CFP (CR2Canon, Japan), SD-OCT (Optovue RTVueXR AVANTI, USA), FAF, FFA and ICGA (Heidelberg HRA, Germany) at baseline. OCT images were obtained in linear scanning mode. The following parameters were quantitatively measured and analyzed: the presence of PEDs, central macular thickness (CMT), defined as the distance between the internal limiting membrane and the Bruch's membrane, and subfoveal choroidal thickness (SFCT), defined as the distance from the RPE to the choroid-sclera interface in the macular fovea. Leakage starts time and leakage patterns including localized leakage (ink-blot/smoke-stack) or diffuse RPE changes were recorded. Patients' treatment methods, along with follow-up BCVA and OCT outcomes obtained at visits exceeding 1mo, were also documented.

Statistical Analysis Statistical analyses were performed using Statistical Package for Social Sciences V.27.0 (SPSS V.27.0, USA). The normality of continuous data was assessed using the Shapiro-Wilk test. Continuous variables were expressed as means±standard deviations and compared using two independent samples *t*-test if they were normally distributed, otherwise expressed as median and 25th to 75th quartiles and compared using the Mann-Whitney *U* test. Nonparametric Wilcoxon signed-ranks test was used to compare BCVA changes within groups. Categorical data were presented as numbers (percentages) and compared using Fisher's exact test. Statistical significance was indicated by $P<0.05$.

RESULTS

Patient Clinical Characteristics The study included 28 eyes of 28 patients (22 males and 6 females). The mean age was 42.43±6.14y (33-59y) and the mean symptom duration was 17.73±15.47d (2-60d). Totally 8 eyes (8 patients) with fibrinous exudation were included as the FE group, and 20 eyes (20 patients) without fibrinous exudation were selected as the non-FE group. Demographic and clinical characteristics of two groups are summarized in Table 1. None of the patients were pregnant or were using steroids. There was no statistically significant difference in the gender distribution, and males constituted the majority in both groups. Compared with the non-FE group, the FE group was significantly older (P=0.004), had longer self-reported symptom duration (P=0.02), and worse baseline BCVA (P=0.011).

In the FE group, 1 eye underwent conventional laser treatment, 1 eye was lost to follow-up, and the remaining 6 were observed. In the non-FE group, 11 eyes underwent laser treatment (8 conventional and 3 micropulse), 3 eyes

Table 1 Comparison of demographic and clinical characteristics between two groups

Characteristics	FE group (*n*=8)	Non-FE group (*n*=20)	*P*
Male gender, *n* (%)	6 (75)	16 (80)	1.00[c]
Age (mean±SD, y)	47.50±7.27	40.40±4.36	0.004[a]
Symptom duration (d)	26.50 (16.75, 30.00)	7.00 (5.00, 15.00)	0.02[b]
Baseline BCVA	0.301 (0.097, 0.398)	0.097 (0.000, 0.191)	0.011[b]
Follow-up BCVA	0.222 (0.097, 0.373)	0.000 (0.000, 0.097)	0.003[b]

[a]Two independent samples *t*-test; [b]Mann-Whitney *U* test; [c]Fisher's exact test. FE: Fibrinous exudation; SD: Standard deviation; BCVA: Best-corrected visual acuity.

Table 2 Summary of multimodal imaging characteristics and follow-up data of two groups

Characteristics	FE group (*n*=8)	Non-FE group (*n*=20)	*P*
Yellowish exudation/hyperreflective material, *n* (%)	8 (100)	0	<0.001[b]
PED, *n* (%)	8 (100)	11 (55)	0.029[b]
CMT (mean±SD, μm)	535.00±144.15	434.94±112.83	0.067[a]
SFCT (mean±SD, μm)	402.00±20.50	346.10±10.97	<0.001[a]
Leakage starts (mean±SD, s)	14.50±2.33	22.67±1.24	<0.001[a]
Leakage pattern			
Localized (ink-blot/smoke-stack)	8 (7/1)	16 (11/5)	0.295[b]
Diffused	0	4	
Follow-up OCT features			
SRF	5/7	7/17	0.371[b]
PED	6/7	8/17	0.172[b]
Hyperreflective material	5/7	4/17	0.061[b]

[a]Two independent samples *t*-test; [b]Fisher's exact test. FE: Fibrinous exudation; PED: Pigment epithelial detachment; SD: Standard deviation; CMT: Central macular thickness; SFCT: Subfoveal choroidal thickness; OCT: Optical coherence tomography; SRF: Subretinal fluid.

were lost to follow-up, while the remaining 6 were observed. Vision improvement was seen in both groups, but there was only a statistically significant difference in the non-FE group (*P*=0.017), not in the FE group (*P*=0.157). The last follow-up visual acuity differed significantly between groups, with the non-FE group demonstrating superior outcomes compared to the FE group (*P*=0.003).

Multimodal Imaging Characteristics Multimodal imaging characteristics and follow-up data of the two groups are summarized in Table 2. All patients showed focal serous neuroretina detachment outlines of varying sizes on CFP, and patients in the FE group exhibited diffuse yellowish exudation lesions. SD-OCT revealed significant intergroup differences. All eyes in the FE group (8/8, 100%) presented with PED, in contrast to only 55% (11/20) in the non-FE group (*P*=0.029). Furthermore, SRHM, indicative of fibrinous exudation, was observed exclusively in the FE group (8/8, 100%) and corresponded to fundus-visible fibrin area. The SFCT was significantly greater in the FE group than the non-FE group (*P*<0.001), while there was no difference in CMT between groups. In the FE group, fibrin exhibited hypofluorescence in FAF and was surrounded by hyperfluorescent serous retinal detachment. In contrast, only hyperfluorescent detachment outlines were observed in the non-FE group. FFA showed that hyperfluorescent leakage commenced earlier in the FE group (14.50±2.33s) than in the non-FE group (22.67±1.24s; *P*<0.001). All eyes had a localized leakage (7 ink-blot and 1 smoke-stack) in the FE group, while 16 eyes had a localized leakage (11 ink-blot and 5 smoke-stack) and the remaining 4 eyes had diffuse RPE changes in the non-FE group. In the late phase, the leakage area in the FE group usually expanded to more than 1/2 disc diameter (DD), whereas it was less than 1/2 DD in the non-FE group. In ICGA, both groups observed dilated choroidal vessels in the early phase. Geographic areas of hyperfluorescence with blurred margins, interpreted as choroidal vascular hyperpermeability, could be observed in the mid-phase. In the late phase, hyperfluorescence areas persisted, showed peripheral extension, or washed out.

At the final follow-up OCT examination, 5 eyes showed residual subretinal fluid (SRF), 6 eyes still exhibited PED changes, and 5 eyes retained subretinal fibrin exudation in the FE group. There was 1 eye lost to follow-up, and thus no follow-up data were available. In the non-FE group, 7 eyes showed residual SRF, 8 eyes exhibited PED, and 3 eyes were lost to follow-up. Furthermore, 4 eyes presented SRHM during the follow-up and 1 eye exhibited a small RPE break at the final visit in the non-FE group. Examples of acute CSC with or without fibrinous exudation were shown in Figures 1 and 2.

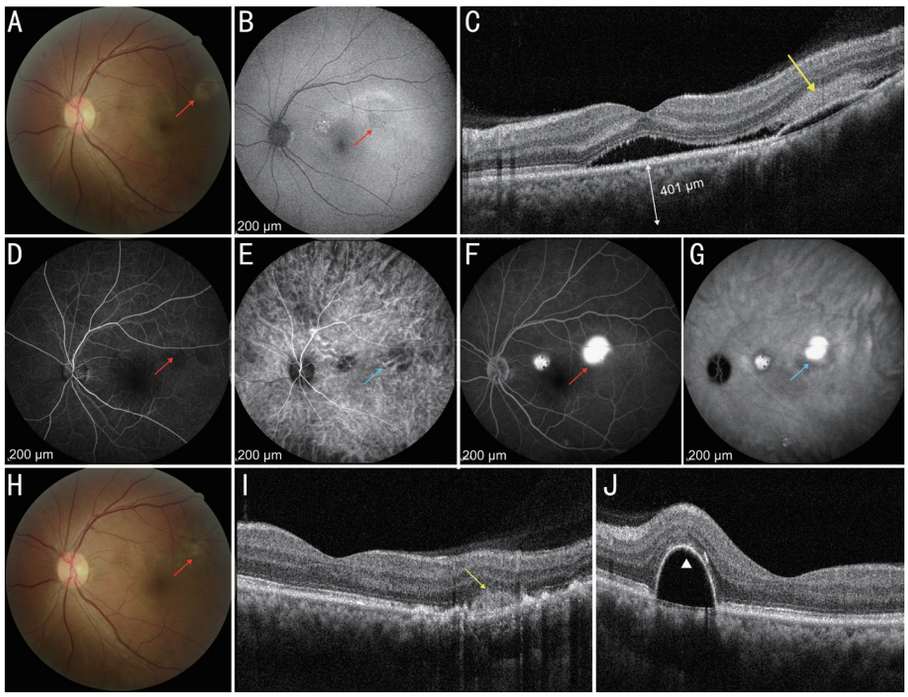

Figure 1 Multimodal imaging characteristics of acute CSC with fibrinous exudates A: CFP shows the outline of neuroretinal detachment in the macula, with yellow-white lesions in the temporal aspect of the macula (red arrow); B: FAF shows hypofluorescence in the yellow-white lesions surrounded by hyperfluorescence (red arrow); C: OCT shows increased subfoveal choroidal thickness, presence of SRF with serous PED, and subretinal hyperreflective fibrin (yellow arrow). The SFCT was measured as 401 μm; D, F: FFA shows pinpoint hyperfluorescent leakage spot in the arterial phase (13s) and leakage area expanding to more than 1 DD in the late phase (15min; red arrows); E, G: ICGA shows choroidal vasodilation at the corresponding lesion without obvious hyperfluorescence in the early phase (13s), and hyperfluorescence of virtually the same size and morphology as seen on FFA in the late phase (15min; blue arrow); H, I, J: After one month of observation, CFP showed persistent yellowish lesions (red arrow). OCT showed SRF had been absorbed, but PED (triangle) and hyperreflective materials were still present (yellow arrow). CSC: Central serous chorioretinopathy; CFP: Color fundus photography; FAF: Fundus autofluorescence; OCT: Optical coherence tomography; SRF: Residual subretinal fluid; PED: Pigment epithelial detachment; SFCT: Subfoveal choroidal thickness; FFA: Fundus fluorescein angiography; DD: Disc diameter; ICGA: Indocyanine green angiography.

DISCUSSION

In our study, patients with fibrinous CSC were older, reported a longer symptom duration, and exhibited poorer baseline and follow-up visual acuity outcomes compared to patients without fibrin exudates. Multimodal imaging systems revealed distinct characteristics of fibrinous CSC, including a higher incidence of PEDs, larger SFCT values, earlier leakage start time, and a potentially larger late leakage area. These findings suggest that fibrinous CSC may be associated with more severe choroidal abnormalities and RPE dysfunction. Interpretation of these findings should therefore be considered alongside relevant research findings.

The pathogenesis of CSC is mainly attributed to choroidal congestion, vascular hyperpermeability, and RPE dysfunction. Many pathophysiological mechanisms, including vortex vein congestion, inflammation, mineralocorticoid pathway activation, and systemic and genetic factors, can lead to choroidal hyperpermeability[11-12]. This hyperpermeability increases tissue hydrostatic pressure, which can lead to PED and subsequent RPE defects. These defects, in turn, result in focal leakage and accumulation of SRF. Fibrinous CSC is an atypical form and its exact pathophysiology remains unclear. It is thought that disturbance of the posterior blood-retinal barrier, Bruch's membrane, or the RPE may be the primary site of damage[7]. Normally, proteins do not leak from the choriocapillaris, but increased capillary permeability can lead to the extravasation of large molecules. These proteinaceous exudates then gain access to the subretinal space through blowout areas of PEDs and RPE defects[13]. In pathology, fibrin leakage is an important marker of severe vessel wall damage and increased vascular permeability.

Genetic predisposition, endogenous or exogenous corticosteroids, androgens, pregnancy, type A personality, psychosocial stress, sleep disturbance, and phosphodiesterase-5 inhibitors (*e.g.*, sildenafil) are acknowledged risk factors for CSC[1,4,14-16]. CSC is more prevalent among working-age males, and its development may be associated with testosterone, a vasoactive hormone with vasodilatory effects[17-18]. Aging leads to morphological alterations in the basal plasma membrane, loss of the regular hexagonal shape of RPE cells, increased

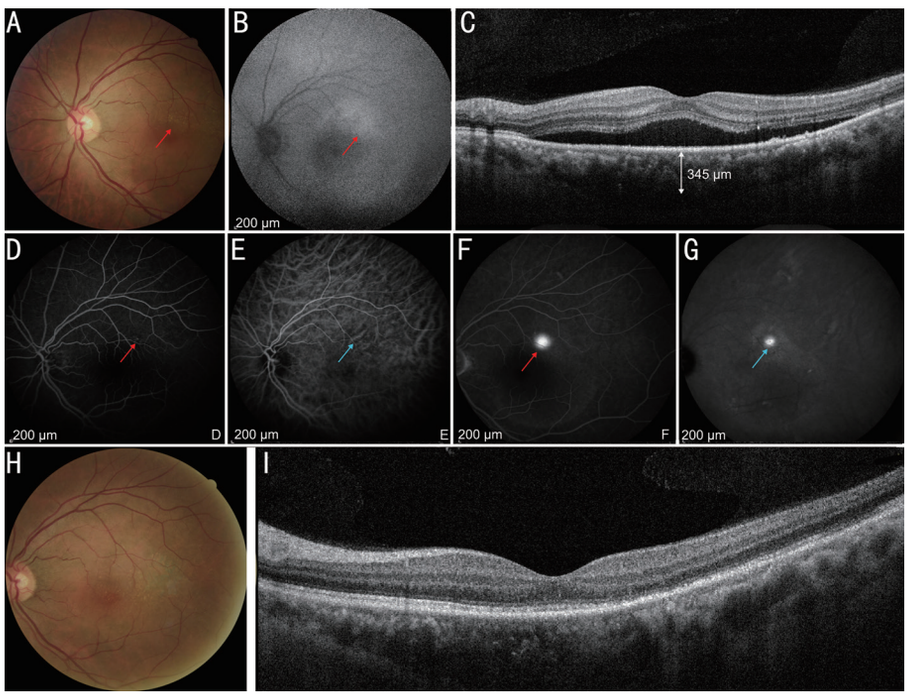

Figure 2 Multimodal imaging characteristics of acute CSC without fibrinous exudates A: CFP shows the outline of neuroretinal detachment in the macula (red arrow) without yellowish-white exudation; B: FAF shows hyperfluorescence in the area of neuroretinal detachment (red arrow); C: OCT shows increased subfoveal choroidal thickness and SRF in the macula. The SFCT was measured as 345 μm; D, F: FFA shows pinpoint hyperfluorescent leakage spot in the venous phase (23s) and leakage area expanding to 1/4 DD in the late phase (16min; red arrow); E, G: ICGA shows choroidal vasodilation at the lesion without obvious hyperfluorescence in the early phase (23s) and hyperfluorescent leakage significantly smaller than that seen on FFA in the late phase (16min; blue arrow); H, I: After one month of observation, SRF was completely absorbed. CSC: Central serous chorioretinopathy; CFP: Color fundus photography; FAF: Fundus autofluorescence; OCT: Optical coherence tomography; SRF: Residual subretinal fluid; SFCT: Subfoveal choroidal thickness; FFA: Fundus fluorescein angiography; DD: Disc diameter; ICGA: Indocyanine green angiography.

basal infoldings, and disruption of tight junctions[19-20]. Yu *et al*[21] found that age is an independent risk factor for persistent or recurrent CSC, as RPE in elderly eyes is more vulnerable to increased hydrostatic pressure and choroidal hyperpermeability. In our study, patients in the FE group were significantly older than those in the non-FE group, potentially suggesting that elderly individuals are more prone to develop fibrin exudation during the course of CSC. However, the direct relationship between advanced age and fibrin exudation is unclear. In addition, the clinical significance of symptom duration in relation to fibrin exudation remains controversial. Yu *et al*[22] found that the symptom duration was shorter in eyes with subretinal exudation (18.85±21.25d) than in those without (30.33±26.41d), supporting that subretinal exudates may be an early indicator of acute CSC. Rajesh *et al*[9] suggested that the duration of the disease is unrelated to fibrin exudation, as fibrin formation can be observed as early as 1wk after the onset of symptoms or persist for years. Cong *et al*[23] reported that SRHM is more common in eyes with longer disease duration, while their study cohort included both acute and chronic CSC patients. These disparate findings across studies might be explained by a dynamic model of fibrin formation and resolution. We hypothesize that fibrin can appear early in severe cases with massive leakage, and it can also accumulate and persist over time in cases with ongoing permeability. The timing of patient presentation relative to this dynamic process likely influences the observed relationship. In our study, although both groups presented within the acute phase, we observed longer self-reported symptom durations in the FE group (median 26.5d) than the non-FE group (median 7d). Furthermore, during follow-up reviews, 4 patients in the non-FE group developed fibrinous exudation, with the earliest case occurring 7d and the latest 45d after the initial diagnosis. These observations support the dynamic nature of this process; the formation of visible fibrinous exudates evolves over time, concurrent with increasing vascular permeability and RPE defects, rather than indicating a specific disease stage. Further, larger, prospective studies are required to clarify the relationship between symptom duration and fibrinous exudation.

Patient visual acuity at presentation and final outcomes are associated with various factors such as persistence of SRF or PED, alternations in CMT, disruption of the foveal ellipsoid zone, damage to the external limiting membrane, and the

development of choroidal neovascularization[10,24-26]. Patients with fibrinous exudates tend to have relatively worse vision. Yu *et al*[22] found patients with subretinal exudation had lower BCVA than those without. Suwal *et al*[24] demonstrated that hyperreflective dots in the intra/subretinal layer are associated with poor visual outcome and persistence of SRF. Our results are consistent with these observations. However, patient age and disease duration are potential confounding factors, as studies have demonstrated their negative correlation with visual acuity[25].

Previous researchers suggested that an SFCT value >300 μm should be considered a pathological indicator, but no universally accepted threshold currently exists[27]. Choroidal thickening is caused by fluid accumulation resulting from vascular hyperpermeability, as well as hypertrophic or congested vascular structures[27-28]. PEDs are common in CSC and usually occur in areas with expanded Sattler and Haller choroidal structures[29]. It could represent a form of RPE decompensation in response to high choroidal hydrostatic pressure. Normally, RPE pumps fluid towards the choroid; however, under abnormal microenvironment, its pump function would be reversed, causing fluid accumulation beneath the retinal neural epithelium[30]. Our study found a significant increase in SFCT in both groups, with the degree being significantly higher in the FE group. Furthermore, all patients in the FE group had PED, compared to only 55% in the non-FE group. These findings suggest that choroidal vasodilation, vascular hyperpermeability changes, and RPE dysfunction may be more pronounced in CSC patients with fibrinous exudation. Fibrinous exudation is a result of choroidal vasodilation and increased permeability. Notably, no significant differences were observed between the two groups in CMT values, which represent the distance between the inner limiting membrane to Bruch's membrane, incorporated the height of SRF. Its association with fibrin exudation remains unclear, but this may be explained as CMT values are likely influenced by multiple complex factors.

Multimodal imaging reveals the characteristic features of fibrinous exudate. It is usually described as yellowish or yellow-white subretinal material on CFP, with or without a central dark dot. FAF typically shows ill-defined hypofluorescence areas with surrounding hyperfluorescence changes, caused by fluid exudation. Diffuse or dense hyperreflective deposits beneath the retina observed in SD-OCT represent the most typical manifestation of fibrinous exudation. Vacuole sign, described as a hyporeflective oval-shaped vacuole-like clearing area, could be observed in the area of fibrin and is often coupled with RPE defects[9,13]. It was thought to be caused by clear fluid leaking into the subretinal space and displacing the fibrin. However, vacuolation and typical RPE defects were not observed in our FE group patients; instead, fibrin frequently appeared as an adhesive bridging the neurosensory retina and the RPE. This may be because RPE defect or rip may not be visible when fibrin is minimal or in the early cases. On FFA, the early phase shows focal dye leakage corresponding to the site of subretinal exudate, while the late phase shows staining of the exudates themselves. The subretinal exudate did not block the underlying fluorescence. ICGA showed choroidal vasodilation in the early phase with mid-phase indistinct hyperfluorescence and late staining. Interestingly, we found that leakage start time appeared earlier in the FE group, supporting a link between fibrinous exudation and more severe vascular hyperpermeability. Larger areas of late hyperfluorescence leakage were frequently observed, but the statistical significance requires further investigation. Further, the expanding dot sign was the most common leakage pattern of fibrinous CSC. Recently, Pu *et al*[31] found that patients with SRHM had higher frequencies of PED and RPE defects, larger SRD dimensions, and larger focal choroidal vascular hyperpermeability areas. They suggested that alternations in choroidal vasculature and compromised RPE barrier function might play important roles in the development of SRHM. However, their study neither correlated patients' clinical characteristics with multimodal imaging features nor provided a detailed description of the imaging manifestations of fibrinous exudation.

CSC is typically a self-limiting disease with relatively good functional and structural recovery. Observation is often the first-line approach in acute CSC management, while early interventions such as focal photocoagulation, micropulse laser, photodynamic therapy or intravitreal injection of anti-vascular endothelial growth factor are all considered to accelerate the resolution of serous neuroretinal detachment and shorten the disease course, possibly reducing disease recurrences and RPE degeneration[32]. The presence of fibrin seems to have an impact on the prognosis. Research has found that patients with hyperreflective deposits have longer SRF resolution duration, thinner final CMT and poorer final BCVA[23,33], whereas Chhablani *et al*[10] reported good visual recovery in most CSC patients with SRHM after SRF absorption. We found that patients with fibrinous exudation usually maintain acceptable visual outcomes after one to three months of follow-up, though significant improvement is uncommon. This may be related to the difficulty in achieving complete absorption of SRF and fibrinous exudates. Regardless of the presence of fibrin, PED may persist even after complete resolution of SRF. However, due to the small sample and loss to follow-up in some patients, this study did not analyze the impact of different treatment regimens on the visual and structural outcomes of fibrinous exudate.

Although our study did not compare different treatment regimens, our findings have clear clinical implications. The identification of fibrinous exudation in acute CSC may serve as a key imaging biomarker for disease severity. Clinicians encountering such patients should be aware that it potentially indicates a more prolonged disease course and a less favorable visual outcome, warranting a more proactive management strategy, such as closer follow-up intervals, early intervention for persistent leaks, and counseling patients about the potential for slower recovery. Regarding therapeutic choices, given the severe choroidal vasodilation and hyperpermeability indicated by fibrinous exudation, therapies targeting the choroid, such as photodynamic therapy, could be more effective at addressing the underlying pathophysiology than conventional laser photocoagulation, although this needs to be confirmed in future prospective studies[3-4].

The study has several limitations. First, the retrospective nature of the study and its small sample size are primary constraints, which may limit the statistical power and generalizability of our findings. Furthermore, we did not assess potential risk factors such as personality traits (*e.g.*, Type A behavior) or vortex vein morphology, which have been implicated in CSC pathogenesis. Future prospective studies incorporating these factors may provide further insights. Additionally, more comprehensive quantitative morphological measurements (*e.g.*, height and volume of PED, quantitative thresholds for fibrin) were not performed. The inclusion of minute PEDs in OCT poses challenges for quantitative measurements, and morphological variations in PEDs across different scanning planes complicate standardized cross-sectional comparisons. Identification of fibrinous exudation relies on qualitative imaging criteria, without further stratification based on spatial distribution or quantitative ranges. A standardized quantitative threshold for fibrin exudation is currently lacking. In addition, enhanced-depth-imaging OCT technology has not been employed for measuring choroidal thickness, which might provide more accurate SFCT assessments. Manual interpretation of imaging results may introduce increased inter-observer variability due to image quality and subjective factors. Future large-scale, prospective studies are warranted to conduct reliable multidimensional quantitative analyses of morphological characteristics of fibrinous CSC and to evaluate the impact of different treatment regimens on its long-term prognosis. Despite these limitations, this study provides a comprehensive clinical and multimodal imaging comparison and analysis of acute CSC with or without fibrinous exudation, elucidating the potential pathophysiological mechanisms underlying fibrin exudation.

In conclusion, we found that acute CSC with fibrinous exudation was associated with older patient age, longer symptom duration, and poorer baseline and follow-up BCVA outcomes. Multimodal imaging characteristics suggest that fibrinous exudation might result from more severe choroidal vasodilation, increased vascular permeability, and RPE barrier dysfunction. Recognizing fibrinous CSC and understanding its underlying mechanisms are crucial for managing patient prognosis and may guide future research into targeted interventions.

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Data Availability Statement: The data that support the findings of this study are not openly available due to reasons of sensitivity and are available from the corresponding author upon reasonable request.

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Conflicts of Interest: Liu ZC, None; **Zhang J,** None; **Han JD,** None.

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