

Quantitative Optical Coherence Tomography Angiography of Retinal and Choriocapillaris Microvasculature in Central Serous Chorioretinopathy

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Abstract

Objective: We aimed to quantitatively evaluate choriocapillaris (CC) patterns in central serous chorioretinopathy (CSC) using optical coherence tomography angiography (OCTA).

Methods: Macular OCTA images (6 × 6 mm) were acquired using the RTVue-XR Avanti with the AngioVue imaging system (Luneau Technology, Pont-de-l'Arche, France). Quantitative analysis of the superficial and deep retinal capillary plexuses and CC was performed using en face projection images. Evaluated parameters included vessel density, fractal dimension, and the area and density of CC flow deficits.

Results: Forty-seven patients diagnosed with CSC (56 affected eyes and 38 fellow eyes) were included. Abnormal OCTA features were observed in 49 (87.5%) affected eyes and 14 (36.8%) fellow eyes ($p < 0.001$). Dark areas were seen in 28 (50.0%) affected eyes and eight (21.1%) fellow eyes, respectively ($p = 0.002$), and dark spots were noted in 20 (35.7%) affected eyes and four (10.5%) fellow eyes, respectively ($p = 0.002$); abnormal vessels were observed in 10 (17.9%) affected eyes and two (5.3%) fellow eyes, respectively ($p = 0.045$). Affected eyes showed significantly lower CC flow deficit (mean difference -1.94 , $p = 0.019$) and CC flow area (mean difference -0.24 , $p < 0.001$) compared to fellow eyes. No significant differences ($p > 0.05$) were observed in outer retinal flow area, subfoveal choroidal thickness, or choroidal vascularity index.

Conclusion: OCTA showed that eyes with CSC frequently exhibit dark areas and dark spots, along with significantly lower CC flow deficit ($P=0.019$) and reduced CC flow area ($P < 0.001$) compared with fellow eyes. These findings suggest that OCTA provides valuable insights into CC alterations and the pathogenesis of CSC.

Categories: Radiology, Ophthalmology, Healthcare Technology

Keywords: angiography, central serous chorioretinopathy, choriocapillaris, indocyanine green angiography, optical coherence tomography

Introduction

Central serous chorioretinopathy (CSC) is a common macular disorder characterized by serous neurosensory retinal detachment, often in association with pigment epithelial detachment [1]. First described by von Graefe in 1866 as recurrent central syphilitic retinitis [2], CSC has since been renamed multiple times, including capillarospastic central retinitis, central angiospastic retinopathy, and central serous retinopathy [1]. It is the fourth most common retinal disorder after age-related macular degeneration, diabetic retinopathy, and retinal vein occlusion [3]. CSC shows male predominance, with an incidence of 9.9 per 100,000 in men versus 1.7 per 100,000 in women, and typically affects individuals aged 25–50 years [4,5].

Recognized risk factors include corticosteroid use [6], pregnancy, type A personality [7], psychiatric disorders, and *Helicobacter pylori* infection [8]. Other reported associations include hypertension, gastroesophageal reflux disease, obstructive sleep apnea, alcohol consumption, and sympathomimetic drug use [9]. Although the pathophysiology remains incompletely understood, evidence suggests that choroidal vascular hyperpermeability, ischemia, and inflammation lead to disruption of the retinal pigment epithelium (RPE) barrier, causing fluid leakage into the subretinal space [10]. RPE dysfunction, with impaired fluid transport, may further contribute to subretinal fluid accumulation. Corticosteroids may exacerbate this process by increasing choroidal vessel sensitivity to catecholamines and upregulating adrenergic receptor expression. Experimental studies have demonstrated choroidal vasodilation and leakage following glucocorticoid or aldosterone administration, suggesting a role for mineralocorticoid receptors in the choroid [11–14].

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Clinically, CSC presents with blurred vision, metamorphopsia, micropsia, reduced brightness and color perception, and decreased visual acuity. Although it typically affects one eye, bilateral involvement occurs in approximately 4% of cases [6,15-17]. Optical coherence tomography (OCT) supports the diagnosis by demonstrating subretinal and/or sub-RPE fluid with choroidal thickening. In chronic or recurrent cases, fluorescein angiography is used to identify characteristic leakage patterns (ink blot, smoke stack, or diffuse) [12,18-20], whereas indocyanine green angiography demonstrates choroidal hyperpermeability [21,22].

OCT angiography (OCTA) is a noninvasive imaging technique that enables visualization of the retinal and choroidal microvasculature without the need for dye injection [23-28]. Recent studies have described distinctive choriocapillaris (CC) alterations in CSC, including “dark areas,” “dark spots,” and abnormal vessels [28,29], as well as flow signal patterns such as concentric high/low-flow zones and mosaic configurations. Notably, up to 61% of unaffected fellow eyes have also been reported to exhibit CC abnormalities. Quantitative OCTA analysis has demonstrated reduced vessel density in retinal and choroidal layers, increased subfoveal choroidal thickness, and enlargement of the foveal avascular zone [30].

Based on these observations, this study aimed to characterize qualitative and quantitative CC microvascular alterations associated with CSC using OCTA and to compare these findings between CSC-affected eyes and clinically unaffected fellow eyes.

Materials And Methods

This was a cross-sectional multicenter study conducted at the Retina and General Ophthalmology clinics of Mettapracharak (Wat Rai Khing) Hospital, Nakhon Pathom, Thailand, and the Department of Ophthalmology, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand, between August 2019 and February 2020. Written informed consent was obtained from all eligible participants prior to enrollment.

Study population

Patients with either acute or chronic CSC were enrolled. Chronic cases were defined by symptom persistence for over four months. Affected and clinically unaffected fellow eyes were included for analysis. A total of 47 participants (56 affected eyes and 38 unaffected fellow eyes) were recruited using a convenience sampling approach. In view of the limited existing literature on OCTA in CSC, the sample size was determined with reference to previous studies that reported comparable sample sizes. For example, Chan et al. [24] included 20 participants (22 eyes), and Costanzo et al. [28] studied 32 participants (33 eyes).

Eligibility Criteria

The inclusion and exclusion criteria for participant selection are summarized in Table 1. Eligible participants were adults (≥18 years) with a clinical diagnosis of CSC confirmed by multimodal imaging who were able to provide written informed consent. Participants were excluded if they had coexisting retinal diseases, a recent history of intraocular surgery (except uncomplicated cataract surgery), or media opacities that precluded high-quality imaging.

Inclusion Criteria	Exclusion Criteria
Aged ≥18 years	Coexisting retinal diseases (e.g., diabetic retinopathy or age-related macular degeneration)
Clinically diagnosed with central serous chorioretinopathy (CSC) using multimodal imaging	History of intraocular surgery (other than uncomplicated cataract surgery) within the past 6 months
Able to provide written informed consent	Media opacities that precluded high-quality imaging

TABLE 1: Inclusion and exclusion criteria for study participants

Ophthalmic examination

All participants underwent a comprehensive ophthalmic examination, including measurement of best-corrected visual acuity using the Early Treatment Diabetic Retinopathy Study chart [31], slit-lamp biomicroscopy, and a dilated fundus examination.

Imaging protocol

Spectral-domain OCT, enhanced depth imaging OCT, and fundus autofluorescence were performed using the Spectralis HRA+OCT system (Heidelberg Engineering GmbH, Heidelberg, Germany). OCTA was performed

using the RTVue-XR Avanti with AngioVue (Visionix, Pont-de-l'Arche, France). For each eye, a 6×6 mm scan centered on the fovea was obtained, with dual acquisition to ensure repeatability. Three en face slab segmentations were analyzed, including the outer retina, a custom slab extending from the RPE to $30\text{ }\mu\text{m}$ below the RPE, and the CC. The outer retina and CC were segmented automatically, while the custom slab was segmented manually. The CC slab corresponded to a depth of 34–44 μm below the RPE-Bruch's membrane complex to minimize projection artifacts.

Image analysis

Quantitative OCTA analysis was performed using ReVue software (Visionix; formerly Optovue Inc.). CC images were further processed using ImageJ software (National Institutes of Health, Bethesda, Maryland, United States) and binarized using the Phansalkar local thresholding method (radius: 15 pixels) to evaluate flow deficits. The following parameters were measured: vessel density and fractal dimension in the superficial and deep capillary plexuses, flow deficit area and density in the CC, and flow area in the outer retina. Morphologic abnormalities in the CC were qualitatively classified as dark areas, dark spots, or abnormal vessels, based on previously described criteria [28,29].

Statistical analysis

Continuous variables were presented as mean $\pm$ standard deviation (SD), whereas categorical variables were presented as frequency and percentage. Comparisons between affected and fellow eyes were performed using the paired t-test. Comparisons between independent groups were performed using the independent samples t-test or the Mann-Whitney U test, as appropriate. A two-sided p-value of < 0.05 was considered statistically significant. Statistical analyses were performed using IBM SPSS Statistics for Windows, Version 31.0 (IBM Corp., Armonk, New York, United States).

Results

Demographic and clinical characteristics

A total of 47 patients with CSC were included, comprising 56 affected eyes and 38 unaffected fellow eyes. The mean age was 51.26 ± 13.31 years (range: 31–77 years), and 37 patients (78.7%) were male. Current and former smoking were reported in nine (19.1%) and six (12.8%) participants, respectively. Among systemic comorbidities, hypertension in 15 (31.9%), diabetes mellitus in nine (19.1%), dyslipidemia in eight (17.0%), and obstructive sleep apnea in five (10.6%). A history of corticosteroid use was noted in six patients (12.8%). The median disease duration was six months (interquartile range: 3–16 months). Among chronic CSC cases (>4 months), previous treatments included focal laser photocoagulation in eight cases (17%), photodynamic therapy in eight (17%), and intravitreal injection therapy in six (12.7%) (Table 2).

Characteristics		Values
Age (years)	Mean ± SD	51.26 ± 13.31
	Range	31–77
Sex, n (%)	Female	10 (21.3)
	Male	37 (78.7)
Smoking, n (%)	Non-smoker	32 (68.1)
	Current smoker	9 (19.1)
	Ex-smoker	6 (12.8)
Systemic comorbidities, n (%)	Hypertension	15 (31.9)
	Diabetes mellitus	9 (19.1)
	Dyslipidemia	8 (17)
	Chronic kidney disease	1 (2.1)
	Coronary artery disease	1 (2.1)
	Obstructive sleep apnea	5 (10.6)
	Others	10 (21.3)
Disease duration (months), median (IQR)		6 (3-16)
Previous treatment in chronic CSC (symptoms > 4 months) (n=21)*, n (%)	Focal laser	8 (17.0)
	Photodynamic therapy	8 (17.0)
	Intravitreal therapy	6 (12.7)

TABLE 2: Demographic and clinical characteristics of the patients (N = 47)
*One patient received more than one treatment modality; therefore, the treatment categories are not mutually exclusive.

Representative qualitative OCTA findings of the CC are shown in Figure 1. The en face OCTA image demonstrates a representative dark area, defined as an area of apparent flow reduction within the CC (Figure 1a), which corresponds to the area of serous retinal detachment identified on the cross-sectional OCT B-scan with angio overlay (Figure 1b).

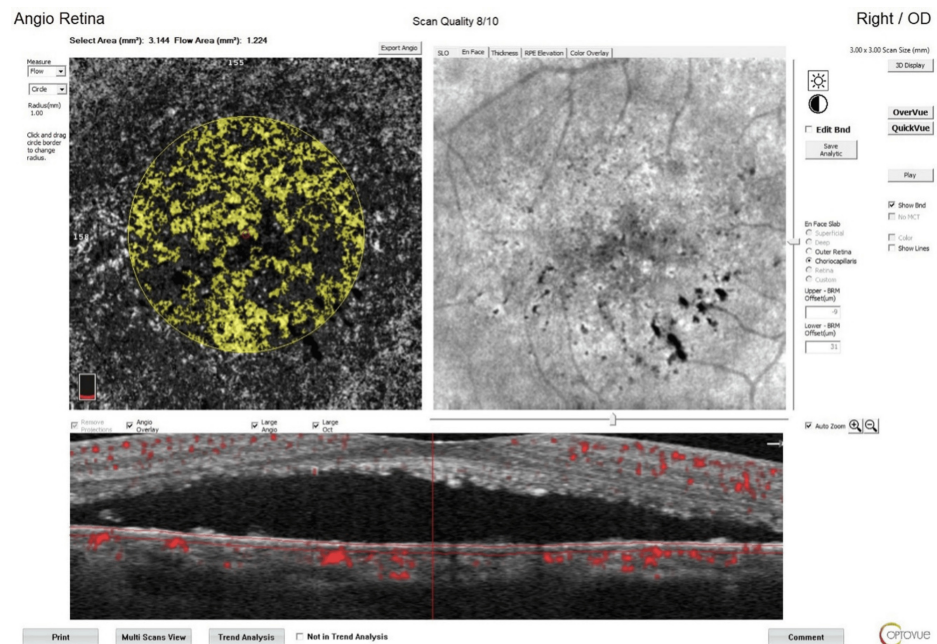


FIGURE 1: Quantification of flow void area in the CC using en face OCTA and corresponding cross-sectional OCT B-scan with angio overlay

(a) OCTA at the choriocapillaris: the yellow circle delineates an area of apparent blood flow reduction, called dark area in this study; (b) Cross-sectional OCT B-scan with angiographic overlay. The red vertical lines indicate the limits of the serous retinal detachment (SRD) that corresponds to the OCTA.

OCTA: optical coherence tomography angiography; OCT: optical coherence tomography; CC: choriocapillaris

Quantitative OCTA and choroidal parameters

OCTA abnormalities were detected in 49/56 affected eyes (87.5%) compared with 14/38 fellow eyes (36.8%) ($p < 0.001$). Among the qualitative CC abnormalities, dark areas were the most common finding, occurring in 28/56 affected eyes (50.0%) compared with 8/38 fellow eyes (21.1%) ($p = 0.002$). Dark spots were observed in 20/56 affected eyes (35.7%) and 4/38 fellow eyes (10.5%), respectively ($p = 0.002$), whereas abnormal vessels were detected in 10/56 affected eyes (17.9%) and 2/38 fellow eyes (5.3%), respectively ($p = 0.045$).

Quantitative OCTA analysis demonstrated that CSC eyes had a significantly lower CC flow deficit than fellow eyes (47.60 ± 3.45 vs. 49.54 ± 4.24 ; mean difference, -1.94 ; 95% confidence interval (CI), -3.56 to -0.31 ; $p = 0.019$) and significantly smaller CC flow area (1.77 ± 0.29 vs. 2.00 ± 0.16 ; mean difference, -0.24 ; 95% CI, -0.34 to -0.14 ; $p < 0.001$) compared with fellow eyes.

No significant difference was observed in outer retinal flow area (0.45 ± 0.35 vs. 0.44 ± 0.31 ; $p = 0.968$). Central subfield thickness (CST) was significantly greater in affected eyes than in fellow eyes ($289.39 \pm 102.06 \mu\text{m}$ vs. $253.87 \pm 30.03 \mu\text{m}$; $p = 0.037$). No statistically significant differences were seen between the groups in subfoveal choroidal thickness, choroidal vascularity index, total choroidal area, or luminal area (all $p > 0.05$) (Table 3).

Variables	Diseased eyes (n = 56)	Non-diseased eyes (n = 38)	Absolute difference (95% CI)	p-value
Abnormal OCTA features	49 (87.5)	14 (36.8)	50.66 (33.04, 68.27)‡	<0.001
Dark areas	28 (50.0)	8 (21.1)	28.95 (10.52, 47.37)‡	0.002
Dark spots	20 (35.7)	4 (10.5)	25.19 (9.29, 41.08)‡	0.002
Abnormal vessels	10 (17.9)	2 (5.3)	12.59 (0.30, 24.88)‡	0.045
CC flow deficit	47.60 ± 3.45	49.54 ± 4.24	-1.94 (-3.56, -0.31)†	0.019
CC flow area	1.77 ± 0.29	2.00 ± 0.16	-0.24 (-0.34, -0.14)†	<0.001
Outer retinal flow area	0.45 ± 0.35	0.44 ± 0.31	0.00 (-0.13, 0.14)†	0.968
Central subfield thickness	289.39 ± 102.06	253.87 ± 30.03	35.52 (2.09, 68.96)†	0.037
Subfoveal choroidal thickness	368.25 ± 105.91	351.29 ± 109.21	16.96 (-27.22, 61.14)†	0.452
Choroidal vascular index	64.62 ± 4.24	65.09 ± 4.50	-0.47 (-2.26, 1.32)†	0.607
Total choroidal area	1.31 ± 0.56	1.36 ± 0.54	-0.05 (-0.28, 0.17)†	0.653
Luminal area	0.83 ± 0.31	0.87 ± 0.31	-0.03 (-0.16, 0.09)†	0.598

TABLE 3: Comparison of CC patterns between affected and fellow eyes in CSC.

Data for the second and third columns are given as n (%) and mean ± SD.

†The absolute difference represents the mean difference estimated using a generalized linear model with a Gaussian distribution.

‡The absolute difference represents the difference in proportions estimated using a risk difference regression model (generalized linear model with an identity link and binomial distribution).

CSC: central serous chorioretinopathy; CC: choriocapillaris

Discussion

This study evaluated qualitative and quantitative changes in the CC in eyes with CSC using OCTA, with fellow eyes serving as controls. Affected eyes demonstrated a significantly higher frequency of OCTA abnormalities, predominantly dark areas and dark spots, together with significantly lower CC flow deficit and CC flow area. The CST was significantly increased in affected eyes; however, no statistically significant inter-eye differences were observed in subfoveal choroidal thickness, choroidal vascularity index, total choroidal area, or luminal area.

The high prevalence of OCTA abnormalities in affected eyes (n=49, 87.5%) and their detection in fellow eyes (n=14, 36.8) are consistent with previous reports demonstrating subclinical CC alterations in the contralateral eye [28]. These findings support the concept of CSC as a choroidal disorder with asymmetric clinical expression, as proposed by Maruko et al. [16].

Qualitatively, the higher frequency of dark areas and dark spots in affected eyes is consistent with the morphological descriptions reported by Costanzo et al. [28], who associated dark areas with hypoperfusion in regions of serous retinal detachment and dark spots with pigment epithelial detachment. The presence of these abnormalities in fellow eyes, as also reported by Shinjima et al. [29] and Sodhi et al. [30], may indicate early or subclinical disease. Abnormal vessels were more frequently observed in CSC-affected eyes than in fellow eyes, and the difference was statistically significant (17.9% vs. 5.3%; risk difference, 12.59 percentage points; 95% CI, 0.30-24.88; p = 0.045).

Quantitatively, the finding of lower CC flow deficit in affected eyes compared with fellow eyes contrasts with those of several previous studies, which reported increased CC flow voids in CSC [25,26,29]. Because our cohort included both acute and chronic CSC, this discrepancy may reflect partial restoration of CC perfusion during the chronic stage or following treatment [23,24]. In addition, differences in OCTA device algorithms, segmentation boundaries, and image binarization methods have been shown to influence CC flow deficit measurements [25].

The reduced CC flow area in affected eyes supports the hypothesis that CC hypoperfusion contributes to the pathogenesis of CSC. This finding is consistent with previous reports by Spaide et al. [22] and Shinjima et

al. [29] using indocyanine green angiography and OCTA. The absence of statistically significant differences in subfoveal choroidal thickness, choroidal vascularity index, total choroidal area, or luminal area between affected and fellow eyes suggests that macroscopic choroidal parameters may not fully reflect microvascular alterations. This interpretation is supported by the findings of Imamura et al. [12] and Ma et al. [23].

The significantly greater CST observed in affected eyes is consistent with previous reports describing neurosensory retinal detachment and subretinal fluid accumulation in active CSC [4,15]. CST therefore remains a useful biomarker of disease activity and correlates with visual function impairment

Strengths and limitations

The strengths of our study include the use of qualitative and quantitative OCTA parameters, standardized imaging protocols, and a paired-eye comparison design, which reduces intersubject variability. However, certain limitations need to be acknowledged. The cross-sectional design precluded temporal analysis, and variability in image quality may have influenced the findings; in addition, functional assessments such as microperimetry were not performed.

Future directions

Future longitudinal studies should investigate whether CC flow parameters can predict recurrence or treatment response, particularly following PDT [23,25] or mineralocorticoid receptor antagonist therapy [11,14]. Integrating OCTA with other noninvasive imaging modalities could further clarify the relationship between choroidal perfusion and retinal structural changes in CSC

Conclusions

Eyes with CSC demonstrate characteristic OCTA abnormalities and quantitative CC alterations, with evidence of subclinical involvement in fellow eyes. These findings highlight the potential value of OCTA in detecting microvascular changes associated with CSC and contribute to a better understanding of the underlying disease pathophysiology.

The combined qualitative and quantitative assessment of the CC using OCTA may provide clinically useful information for disease evaluation and monitoring. Further prospective longitudinal studies with larger sample sizes are warranted to validate these findings and to determine the prognostic value of OCTA-derived CC parameters in predicting disease progression and treatment response.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. Ethics Committee of Mettaphracharak (Wat Rai Khing) Hospital issued approval COA005-2562. The study adhered to the tenets of the Declaration of Helsinki. **Animal subjects:** All authors have confirmed that this study did not involve animal subjects or tissue. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an

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References

1. Abouammoh MA: Advances in the treatment of central serous chorioretinopathy . Saudi J Ophthalmol. 2015, 29:278-86. [10.1016/j.sjopt.2015.01.007](#)
2. Graefe AV: About central recurrent retinitis (Article in German) . Graefes Arch Clin Exp Ophthalmol. 1866, 12:211-5.
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, 91:1554-72. [10.1016/S0161-6420\(84\)34117-3](#)
4. Wang M, Munch IC, Hasler PW, Prunte C, Larsen M: Central serous chorioretinopathy. Acta Ophthalmol. 2008, 86:126-45. [10.1111/j.1600-0420.2007.00889.x](#)
5. Gackle HC, Lang GE, Freissler KA, Lang GK: Central serous chorioretinopathy. Clinical, fluorescein angiography and demographic aspects (Article in German). Ophthalmologie. 1998, 95:529-33. [10.1007/s003470050310](#)
6. Wakakura M, Song E, Ishikawa S: Corticosteroid-induced central serous chorioretinopathy. Jpn J Ophthalmol. 1997, 3:180-5. [10.1016/S0021-5155\(97\)00027-0](#)
7. Yannuzzi LA: Type-A behavior and central serous chorioretinopathy . Retina. 1987, 7:111-31. [10.1097/00006982-198700720-00009](#)
8. Haimovici R, Koh S, Gagnon DR, Lehrfeld T, Wellik S: Risk factors for central serous chorioretinopathy: a case-control study. Ophthalmology. 2004, 111:244-9. [10.1016/j.ophtha.2003.09.024](#)
9. Brodie FL, Charlson ES, Aleman TS, et al.: Obstructive sleep apnea and central serous chorioretinopathy. Retina. 2015, 35:238-43. [10.1097/IAE.0000000000000326](#)
10. Mudvari SS, Goff MJ, Fu AD, McDonald HR, Johnson RN, Ai E, Jumper JM: The natural history of pigment epithelial detachment associated with central serous chorioretinopathy. Retina. 2007, 27:1168-73. [10.1097/IAE.0b013e318156db8a](#)
11. Daruich A, Matet A, Dirani A, et al.: Central serous chorioretinopathy: recent findings and new physiopathology hypothesis. Prog Retin Eye Res. 2015, 48:82-118. [10.1016/j.preteyeres.2015.05.003](#)
12. Imamura Y, Fujiwara T, Margolis R, Spaide RF: Enhanced depth imaging optical coherence tomography of the choroid in central serous chorioretinopathy. Retina. 2009, 29:1469-73. [10.1097/IAE.0b013e3181be0a83](#)
13. Jampol LM, Weinreb R, Yannuzzi L: Involvement of corticosteroids and catecholamines in the pathogenesis of central serous chorioretinopathy: a rationale for new treatment strategies. Ophthalmology. 2002, 109:1765-6. [10.1016/S0161-6420\(02\)01303-9](#)
14. Zhao M, Célérier I, Bousquet E, et al.: Mineralocorticoid receptor is involved in rat and human ocular chorioretinopathy. J Clin Invest. 2012, 122:2672-9. [10.1172/JCI61427](#)
15. Maruko I, Iida T, Ojima A, Sekiryu T: Subretinal dot-like precipitates and yellow material in central serous chorioretinopathy. Retina. 2011, 31:759-65. [10.1097/IAE.0b013e3181fbce8e](#)
16. Maruko I, Iida T, Sugano Y, Ojima A, Sekiryu T: Subfoveal choroidal thickness in fellow eyes of patients with central serous chorioretinopathy. Retina. 2011, 31:1603-8. [10.1097/IAE.0b013e31820f4b39](#)
17. Shukla D, Aiello LP, Kolluru C, Baddela S, Jager RD, Kim R: Relation of optical coherence tomography and unusual angiographic leakage patterns in central serous chorioretinopathy. Eye (Lond). 2008, 22:592-6. [10.1038/sj.eye.6702818](#)
18. Bujarborua D, Nagpal PN, Deka M: Smokestack leak in central serous chorioretinopathy . Graefes Arch Clin Exp Ophthalmol. 2010, 248:339-51. [10.1007/s00417-009-1212-5](#)
19. Friberg TR, Campagna J: Central serous chorioretinopathy: an analysis of the clinical morphology using image-processing techniques. Graefes Arch Clin Exp Ophthalmol. 1989, 227:201-5. [10.1007/BF02172748](#)
20. Gómez-Ulla F, Seoane I, Labella F, Torreiro J, Ruiz C: An image analyzer study of central serous chorioretinopathy. Optom Vis Sci. 1993, 70:118-22. [10.1097/00006324-199302000-00006](#)
21. Katsimpris J, Donati G, Kapetanios A, Pharakakis N, Pournaras CJ: Value of indocyanine green angiography in detection of central serous chorioretinopathy (Article in French). Klin Monbl Augenheilkd. 2001, 218:335-7. [10.1055/s-2001-15893](#)
22. Spaide RF, Hall L, Haas A, et al.: Indocyanine green videoangiography of older patients with central serous chorioretinopathy. Retina. 1996, 16:203-13. [10.1097/00006982-199616030-00004](#)
23. Ma DJ, Park UC, Kim ET, Yu HG: Choroidal vascularity changes in idiopathic central serous chorioretinopathy after half-fluence photodynamic therapy. PLoS One. 2018, 13:e0202930. [10.1371/journal.pone.0202930](#)
24. Chan SY, Pan CT, Wang Q, Shi XH, Jonas JB, Wei WB: Optical coherent tomographic angiographic pattern of the deep choroidal layer and choriocapillaris after photodynamic therapy for central serous chorioretinopathy. Graefes Arch Clin Exp Ophthalmol. 2019, 257:1365-72. [10.1007/s00417-019-04318-x](#)
25. Cakir B, Reich M, Lang S, et al.: OCT angiography of the choriocapillaris in central serous chorioretinopathy: a quantitative subgroup analysis. Ophthalmol Ther. 2019, 8:75-86. [10.1007/s40123-018-0159-1](#)
26. Nicolò M, Rosa R, Musetti D, Musolino M, Saccheggiani M, Traverso CE: Choroidal vascular flow area in central serous chorioretinopathy using swept-source optical coherence tomography angiography. Invest Ophthalmol Vis Sci. 2017, 58:2002-10. [10.1167/iov.17-21417](#)
27. Hu J, Qu J, Piao Z, Yao Y, Sun G, Li M, Zhao M: Optical coherence tomography angiography compared with

- indocyanine green angiography in central serous chorioretinopathy. *Sci Rep.* 2019, 9:6149. [10.1038/s41598-019-42623-x](https://doi.org/10.1038/s41598-019-42623-x)
28. Costanzo E, Cohen SY, Miere A, et al.: Optical coherence tomography angiography in central serous chorioretinopathy. *J Ophthalmol.* 2015, 2015:134783. [10.1155/2015/134783](https://doi.org/10.1155/2015/134783)
 29. Shinojima A, Kawamura A, Mori R, Fujita K, Yuzawa M: Findings of optical coherence tomographic angiography at the choriocapillaris level in central serous chorioretinopathy. *Ophthalmologica.* 2016, 236:108-13. [10.1159/000448436](https://doi.org/10.1159/000448436)
 30. Sodhi PK, Rao KC, R AT, et al.: Optical coherence tomography angiography parameters in Indian patients with central serous chorioretinopathy. *Cureus.* 2023, 15:e46467. [10.7759/cureus.46467](https://doi.org/10.7759/cureus.46467)
 31. Ferris FL III, Kassoff A, Bresnick GH, Bailey I: New visual acuity charts for clinical research. *Am J Ophthalmol.* 1982, 94:91-6. [10.1016/0002-9394\(82\)90197-0](https://doi.org/10.1016/0002-9394(82)90197-0)