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THE INCIDENCE OF NEOVASCULARIZATION IN CENTRAL SEROUS CHORIORETINOPATHY BY OPTICAL COHERENCE TOMOGRAPHY ANGIOGRAPHY

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Purpose: To evaluate the incidence of neovascularization (NV) secondary to central serous chorioretinopathy (CSC)—a condition belonging to the spectrum of pachychoroid disorders by means of optical coherence tomography angiography.

Methods: One hundred and seventy five eyes with CSC were evaluated in this retrospective observational study. The eyes with acute or chronic CSC with no NV were included in Group 1, and those with NV were evaluated in Group 2. Only eyes that had undergone structural optical coherence tomography and optical coherence tomography angiography were included. Age, best-corrected visual acuity, and subfoveal choroidal thickness were evaluated in all eyes. In Group 2, the type and morphology of NV and the occurrence of exudation were considered.

Results: Of a total of 175 eyes with CSC, 86 had the acute form and 89 the chronic. Approximately 140 belonged to Group 1 (80%) and 35 to Group 2 (20%). Approximately 39.2% of all patient with chronic CSC developed NV. Mean age in Groups 1 and 2 was 53.3 years (±10.9) and 66.6 years (±10.2), respectively. Mean best-corrected visual acuity in Groups 1 and 2 was 45.7 (±11.7) and 30.9 (±17.9) early treatment diabetic retinopathy study letters, respectively. Mean CCT in Group 1 and 2 was 417.5 μm (±123) and 344.2 μm (±165.9), respectively. In Group 2, all patients had Type 1 NV (100%); 29 eyes (83%) had filamentous feature, and 6 eyes (17%) had irregular shape. Silent nonexudative NV was observed in 7 eyes (20%), all belonging to Group 2.

Conclusion: The use of optical coherence tomography angiography in everyday clinical practice allows for the accurate analysis of the chorioretinal vascular setting, with the identification of new vessels that could remain misdiagnosed.

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Central serous chorioretinopathy (CSC) is a chorioretinal disorder characterized by the occurrence of a serous neurosensory retinal detachment, that can involve all the retinal region, and in particular the posterior pole, is sometimes accompanied by detachments of the retinal pigment epithelium (RPE), and is likely associated with choroidal hyperpermeability.[1]

Central serous chorioretinopathy is often observed in patients with Type A personality[2] and may be related to a wide range of risk factors, including hypertension, helicobacter pylori infection, use of steroids, sleeping disturbances, autoimmune diseases, and the use of psychopharmacologic medication.[3]

Although CSC is usually regarded as a self-limiting condition (acute CSC), there are cases where it can persist for 6 months or more (chronic CSC), causing the changes in the retina, RPE, and choroid to become irreversible.[4]

Central serous chorioretinopathy has recently been included in the pachychoroid spectrum disorders, which also comprises conditions such as pachychoroid pigment epitheliopathy, pachychoroid neovasculopathy, and polypoidal choroidal vasculopathy.[5] All these disorders are characterized by increased focal or diffuse choroidal thickness, sometimes associated with dilated vessels, called pachyvessels.[6]

In addition to the presence of hyperpermeability and dilated vasculature, Kitaya et al[7] detected localized ischemic areas and a decreased choroidal blood flow, as visible on both fluorescein angiography (FA) and indocyanine green angiography.

The use of structural optical coherence tomography (OCT) by the en face scans allowed the observation of a difference in the frequency of expression of the patterns of Haller vessel arrangements between healthy eyes and those with CSC.[8]

The development of neovascularization (NV) can be regarded as an important complication of CSC, it being responsible for a reduced visual acuity during a long-term follow-up.[9]

A retrospective analysis conducted by Peiretti et al[10] revealed the presence of NV—identified as polypoidal choroidal vasculopathy on indocyanine green angiography—in 17.6% of patients affected by chronic CSC. Similarly to the result of the abovementioned study, the development of Type 1 NV was observed in 15.6% of the cases of CSC included in the analysis performed by Shiragami et al[11] and more frequently in eyes affected by chronic forms of chorioretinopathy. More recently, systemic hypertension, "double-layer sign," and RPE changes have been identified as independent risk factors for the occurrence of NV secondary to CSC in a retrospective case–control analysis conducted by Lee et al.[12]

Unfortunately, the use of dye-based angiography for the correct visualization of the NV in the eyes affected by CSC can be misleading because features including intraretinal or subretinal fluid, cystoid macular degeneration, retinal atrophy, and diffuse hyperfluorescence can be observed in the eyes affected by CSC either with or without secondary NV.[13]

Since the introduction of OCT angiography (OCTA), this limitation has been overcome, at least partially. Optical coherence tomography angiography has provided a new, noninvasive, dyeless technique, capable of visualizing retinal circulation directly, in a multilayered, tridimensional way.[14–16]

The sensitivity and specificity of this imaging method (combined with the information supplied by structural OCT scans) in detecting NV secondary to CSC has been emphasized, since OCTA has been effective in defining the morphologic aspect of the NV and in clearly distinguishing the NV from the surrounding tissue.[13]

Moreover, this imaging tool has introduced the possibility of describing NV in the eyes with pachychoroid pigment epitheliopathy, a disorder falling within the pachychoroid disease spectrum, in which OCTA—compared with FA or indocyanine green angiography—proved to be successful in detecting NV earlier.[17]

In addition, OCTA performed on eyes affected by chronic CSC was able to disclose a higher incidence of NV at the RPE detachment level than other imaging tools.[18,19]

The purpose of this study is to retrospectively assess the incidence of NV secondary to CSC using OCTA.

Methods

This single-centered retrospective study was performed at the private eye center "Centro Italiano Macula" in Rome, Italy.

The study was conducted in accordance to the tenets of the Declaration of Helsinki.

Subjects with CSC were diagnosed with chronic or acute CSC on the basis of binocular clinical examinations and OCT imaging. About acute CSC, we made diagnosis in the case of acute neuroepithelium detachment associated to pachychoroid. Chronic CSC eyes showed structural OCT details peculiar to the diagnosis. Chronic CSC was defined as the presence of visual symptoms for at least 6 months, with documented clinical features of CSC, including changes in macular subretinal fluid and RPE documented on OCT imaging.

Neovascular maculopathy (e.g., pathological myopia, idiopathic CNV, and angioid streaks) were excluded from the analyses. Polypoidal lesions polypoidal choroidal vasculopathy masquerading as CSC with signs of a branching vascular choroidal network or polypoidal lesions were carefully assessed and excluded.

We excluded pseudophakic eyes and assessed the refractive error (spherical equivalent). Eyes with more than ±3 diopters (D) spherical equivalent were excluded from enrolment.

Exclusion criteria also comprised poor quality images. In case of bilateral involvement, both eyes

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The data sets generated and/or analyzed during the current study are available from the corresponding author upon reasonable request.

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Table 1. Clinical Characteristics of Patients With Central Serous Chorioretinopathy (CSC), With and Without Neovascularization

	Group 1 (w/NV)	Group 2 (w/o NV)	*P**
n (eyes)	140	35	—
CSC type			
Acute	86 (61.4%)	0	<0.001†
Chronic	54 (38.6%)	35 (100%)	0.75
Sex			
Male	104 (74.3%)	22 (62.8%)	<0.001†
Female	36 (25.7%)	13 (37.2%)	0.11
Age (years)	53.3 (±10.9)	66.6 (±10.2)	<0.001†
SCT (μm)	417.5 (±123)	344.2 (±165.9)	<0.001†
BCVA	45.7 (±11.7)	30.9 (±17.9)	0.63
Refractive error (spherical equivalent)	0.48 ± 2	−0.09 ± 2.2	0.73
NV type in structural OCT	—	Type 1 = 35 (100%)	<0.001†
NV morphology in OCTA	—	Filamentous = 29 (83%)	<0.001†
		Irregular = 6 (17%)	0.001†
Exudation	0	28 (80%)	<0.001†
No exudation	140	7 (20%)	<0.001†

*Calculated using the Chi-square test.
†$P < 0.05$.

were considered. We exclude any patient who did not have OCT and/or OCTA to begin with.

The eyes included in the study were further divided into two groups. Eyes with acute or chronic CSC with no NV evidence were included in Group 1, and those with NV were considered in Group 2.

Both structural OCT and OCTA images were evaluated for the analysis using the Optovue device (RTVue XR Avanti with AngioVue, Optovue Inc, Fremont, CA). With regard to OCTA, segmentation boundaries were automatically provided by the visualization software. Only good quality images were retained for further investigations.

The subfoveal choroidal thickness (SCT) was also measured on cross-sectional OCT B-scans. Two independent masked graders individually assessed all choroidal thickness measurement in the fovea region, from the rear edge of the RPE to the choroid-sclera junction. The agreement between the two observers was determined through Cohen's kappa statistic,[20] which revealed an agreement of 91%.

The presence/absence of CNV was determined using clinical evaluations, structural OCT and OCTA imaging. Neovascularization in Group 2 was defined as a hyperreflective, flat, irregular RPE elevation on OCT, and as a well-defined flow signal on OCTA.

According to morphology aspect for NV associated to pachychoroid neovasculopathy previously described by Azar et al, [17] we classified all observed NVs in filamentous aspect and irregular. We referred to filamentous morphology only for the forms that were evidently filamentous; all other, clearly nonfilamentous shape, have been defined as irregular.

Data Analysis

Data are presented as mean values ± SD, where applicable. Best-corrected visual acuity (BCVA) measurements are expresses in early treatment diabetic retinopathy study letters.

Categorical subjects and the ocular characteristics potentially associated with NV (CSC type [chronic or acute], sex, age, and SCT) were examined using the Chi-square test. A multiple logistic regression analysis was also performed, to determine whether CSC type, sex, age, BCVA, and SCT affected the presence/absence of NV (Group 1/Group 2). All statistical analyses were performed with GraphPad Prism 6.0 (GraphPad software, Inc, San Diego, CA). A *P* value of <0.05 was considered statistically significant.

Results

Overall, 175 eyes with CSC pachychoroid disease were evaluated in this retrospective study. Of a total of

Table 2. Correlation Between SCT and Age and Visual Acuity in Eyes With Central Serous Chorioretinopathy (CSC) in Group 1 and Group 2

	P (r^2)	
	Group 1 (w/o NV)	Group 2 (w/NV)
Age and SCT	0.002 (0.06)†	0.03 (0.12)†
BCVA and SCT	0.0003 (0.09)†	0.47 (0.01)

Calculated using the Pearson correlation coefficient test.
†$P < 0.05$

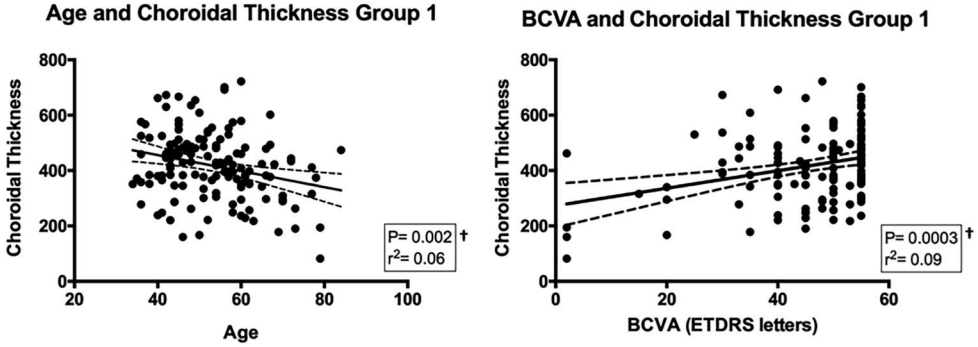

Fig. 1. Linear regression between choroidal thickness and age and between choroidal thickness and best-corrected visual acuity in Group 1. Both correlations were significant ($P < 0.001$).

175 eyes with CSC, 86 had the acute form and 89 the chronic. Approximately 140 belonged to Group 1 (80%) and 35 to Group 2 (20%). Approximately 39.2% of all patient with chronic CSC developed NV. Mean age in Groups 1 and 2 was 53.3 years (±10.9) and 66.6 years (±10.2), respectively. Mean BCVA in Groups 1 and 2 was 45.7 (±11.7) and 30.9 (±17.9) early treatment diabetic retinopathy study letters, respectively. Refractive error (spherical equivalent) in Groups 1 and 2 was 0.48 ± 2 and −0.09 ± 2.2, respectively. Mean CCT in Groups 1 and 2 was 417.5 μm (±123) and 344.2 μm (±165.9), respectively. These differences between eyes with and without CNV were almost statistically significant ($P < 0.001$). By contrast, female sex, BCVA, and the refractive error were not significantly different between the two groups ($P = 0.11$, $P = 0.63$, $P = 0.73$, respectively) (Table 1). We observed the absence of association to NV in acute CSC.

In addition, mean SCT was significantly correlated to age in both groups; the same was valid for BCVA in Group 1, unlike Group 2 (Table 2).

Figures 1 and 2 show the linear regression between SCT and age and between SCT and BCVA in both groups. The correlation between SCT and age was significant ($P < 0.001$) in both groups; by contrast, the correlation between SCT and BCVA was significant only in Group 1 and not significant in Group 2 ($P = 0.47$).

In Group 2, all NVs were Type 1 (100%), with 29 eyes (83%) having a filamentous feature (Figure 3) and 6 eyes (17%) having an irregular shape (Figure 4). Group 2 included also silent nonexudative NV, which was observed in 7 eyes (20%) (Figure 5).

A multiple logistic regression analysis, conducted to evaluate factors related to the occurrence of NV, revealed that all the eyes with chronic CSC were more likely to generate NV than the eyes with acute CSC (100% of eyes suffered from chronic CSC). In addition, in Group 2, factors such as older age and SCT ($P \leq 0.03$) were more likely to generate NV than younger age and greater choroidal thickness. No other parameter was associated with the occurrence of NV (Table 3).

Discussion

The introduction of OCTA in clinical practice allowed to observe several morphological features, mainly in choriocapillary vascular and NV-related disorders. One of the most exciting topic was that related to new vessels secondary to CSC. As part of the spectrum of pachychoroid disorders, CSC was associated to a greater choroidal thickness and, sometimes, to a vessel diameter enlargement, both of which has become the target for a correct diagnosis and a therapeutic monitoring.[21]

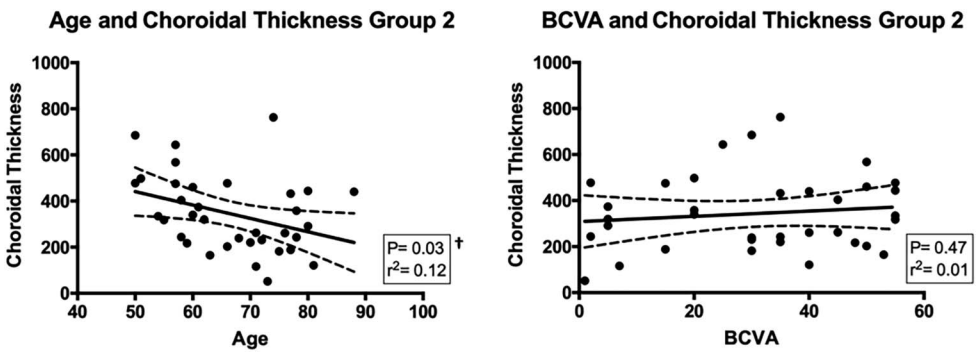

Fig. 2. Linear regression between SCT and age and between SCT and BCVA in Group 2. Correlation between SCT and age was significant ($P < 0.001$), unlike correlation between SCT and BCVA ($P = 0.47$).

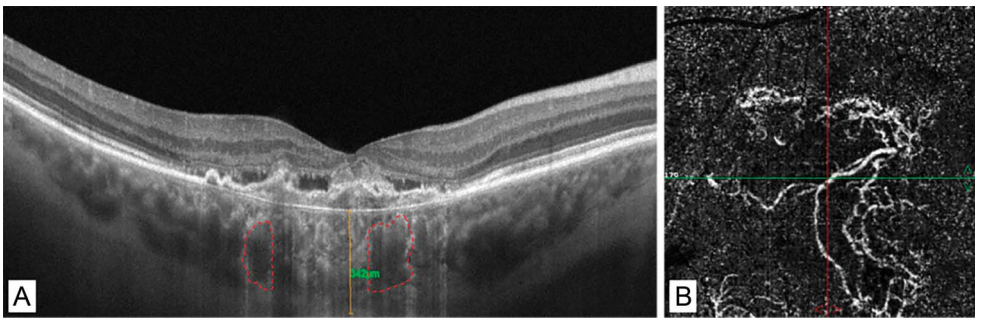

Fig. 3. Structural OCT (**A**) shows the SCT (CCT) with pachychoroid report (342 μm) and the pachyvessel feature (red outline). Type 1 new vessels can be observed below the RPE. The exudation is defined as the occurrence of subretinal fluid. The OCT angiography (**B**) highlights the filamentous aspect of the new vessels.

Although the clinical use of structural OCT and enface scans permitted the observation of some helpful details for the diagnosis of CSC,[22] the use of OCTA allowed for an easier identification of NV in CSC. According to Quaranta-El Maftouhi et al,[23] OCTA allows to detection NV in CSC that are not visible with other imaging techniques. The chorioretinal hyperfluorescence on FA and ICG angiography does not allows the clear evidence of neovascular networks. The dye leakage provides the masking of the effective NV visualization. Further sometimes the clear leakage, pooling, and staining effect are not so obvious, especially in the advanced phases of chronic CSC.

The development of NV is one of the major causes of reduced vision seen during the long-term follow-up of patients with CSC.

The aim of this study was to provide an overview of NV incidence through the use of OCTA in acute and chronic CSC eyes. As described in Table 1, in our study, we never observed NV associated to acute CSC. This aspect may be justified by the absence of dysfunction and abnormalities of the RPE peculiar of chronic disease.

Neovascularization was observed only in chronic CSC mainly in case of advanced alteration of RPE as flat irregular PEDs (FIPEDs), recently described in patients with chronic CSC.[19] The detection of NV in CSC patients with FA and ICG can be more challenging because of the diffuse abnormalities of the RPE during CSC. Previous studies reported that the incidence of NV secondary to CSC ranges from 2 to 15.6%.[9,11] In our cases, incidence was 20%, and we also found that CNV was significantly associated with chronic CSC. The difference, and the greater incidence, could be due to the use of OCTA, which can better show the presence of NV.

All our results described Type 1 NV secondary to CSC. This observation is in contrast with the outcomes of Lee et al,[12] who reported a higher incidence of Type 2 NV, occurring in 76.7% of cases. In addition, we observed that NV secondary to CSC was related to age, with an incidence mainly observed in older patient (mean age in Group 2 was 66.6 ± 10.2 years). A possible explanation of this result could be a long-standing CSC insult to RPE. As described, chronic CSC may predispose to the development of choroidal NV.[10]

Although speculatively, and without any previous scientific evidence, the greater choroidal thickening in Group 1 (415.3 ± 126.5 μm) versus Group 2 (344.2 μm ± 165.9) identified in our observation could suggest that a considerable choroidal thickening could prevent the development of NV. It is not yet known whether CSC occurs as a choroidal thickening or undergoes thickening over time or during its evolution. Recently, Baek et al[24] described a large choroidal vessel layer and pachyvessel pattern in CSC, suggesting a common pathophysiology involving choroidal changes in these eyes.

Optical coherence tomography angiography allowed in those eyes a silent, nonexudative diagnosis of NV (20%) that probably would have remained misdiagnosed.

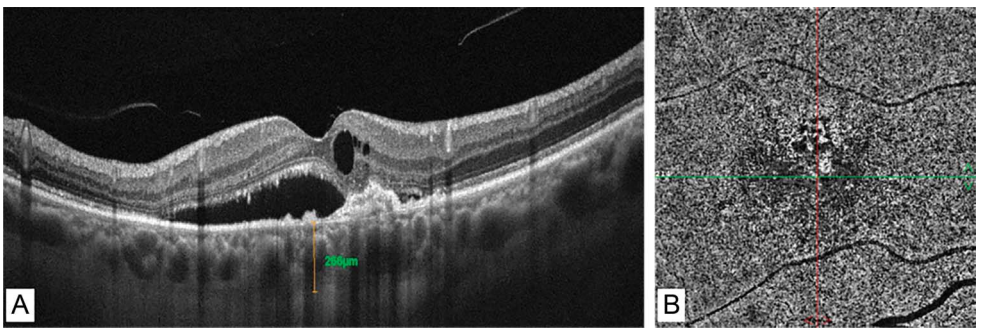

Fig. 4. Structural OCT (**A**) shows the SCT measurement (266 μm). Focal hyperreflective accumulation under RPE elevation is related to Type 1 new vessels subretinal fluid detachment. The OCT angiography (**B**) highlights the irregular shape of the new vessels.

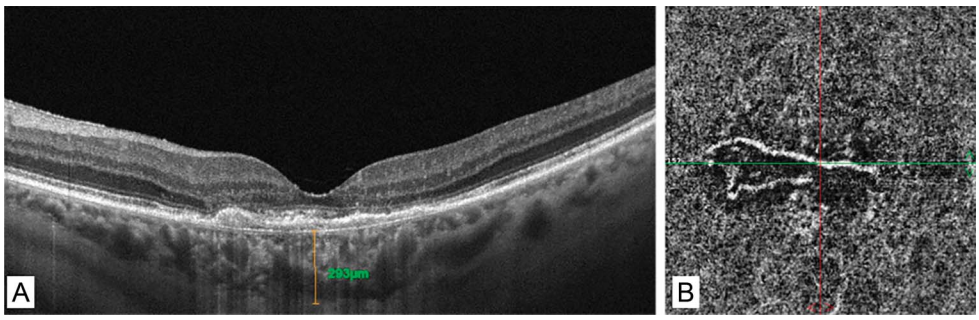

Fig. 5. Structural OCT (**A**) shows the SCT assessment (293 μm). Type 1 new vessels can be observed below the RPE, with no signs of exudation. The OCT angiography (**B**) highlights the filamentous aspect of the new vessels.

A possible future prospect will be to evaluate the risk of exudation of nonexudative NV in CSC eyes over time. As recently reported by Freund et al on the development of NV in wet AMD, this nonexudative NV carries a high risk of development of exudation within the first year after detection.[25]

Our study has some limitations as not performing FA and or ICG for CSC at first observation. According to Fujimoto et al,[26] we believe that OCT findings may offer information to understand acute CSC occurrence. Furthermore, all chronic CSC eyes had structural OCT evidences showing details helpful for the diagnosis as previously described as pachychoroid[6] or RPE dysfunction and abnormalities.[19] Other limitations were that the analysis was limited by the unavailability of established regulatory data. We did not include an healthy population as controls because of the differences in age between the study groups. Also, baseline demographics between the groups differed in terms of age and sex distribution. Furthermore, there are evidences that CCT and choroidal volume are influenced by ethnicity, age, sex, axial length, and age can also have an impact on choroidal vascular density.[27–30] Compared with AMD, mean age was younger in Group 2 and much younger in Group 1. Male dominance in both groups (1 and 2) might partly contribute to the occurrence of NV and the greater choroidal thickness observed in this study. In addition, all our subjects were white, and we did not measure the axial length of the eyes because we had excluded pseudophakic eyes and assessed the refractive error. Our results reported a thinner choroid in eyes with NV associated to chronic CSC. This evidence can be related to older age in patients with NV who physiologically expect a thinner choroid.[27] Another possible explanation could be a choroidal blood flow dysregulation in patients with NV secondary to CSC as recently described by Lupidi et al.[31] Further studies are necessary to better understand this phenomenon.

According to Mrejen et al,[32] our study agreed with their three points results: 1) later age of disease onset is significantly associated with change in BCVA in chronic CSC; 2) type 1 NV is the most frequent subtype of NV associated with chronic CSC; and 3) CNVs were associated with poor VA in chronic CSC.

In conclusion, our results showed that younger female subjects with greater choroidal thickness and BCVA preservation experienced have a lower occurrence of NV. The incidence of NV in CSC pachychoroid disorder was 20%. The occurrence of NV in our results is a little higher than that previously reported; this could be due to OCTA being able to better show the onset of NV. The morphological aspect of all NVs was mainly filamentous towards the irregular shape. In our series, we have not seen medusa, sea-fan, or tangled shapes as previously described for exudative AMD.[33,34]

We believe our findings will be useful and will lead to more sophisticated diagnoses and treatments for the CSC disorder for a personalized medicine.

Table 3. Multiple Logistic Regression Model of Variables Associated With Central Serous Chorioretinopathy, With and Without Choroidal Neovascularization

	Group 1 (w/o NV)		Group 2 (w/NV)	
Independent Variable	*P*	95% CI	*P*	95% CI
CSC type (acute: 0, chronic: 1)	**0.002***	52.21 to 70.85	—	—
Sex (male: 0, female: 1)	**0.005***	−0.02 to 7.83	0.62	−8.89 to 5.45
Age	<**0.01***	52.21 to 70.85	<**0.01***	63.77 to 83.95
BCVA (ETDRS letters)	0.20	−0.28 to 0.05	0.65	−0.15 to 0.24
Subfoveal choroidal thickness (μm)	**0.03***	−0.03 to −0.01	**0.03***	−0.04 to 0.01

*P <0.05.
ETDRS, early treatment diabetic retinopathy study; CI, confidence interval.

Key words: central serous chorioretinopathy, choroidal thickness, neovascularization, pachychoroid spectrum disorders, pachyvessels, optical coherence tomography angiography, personalized medicine.

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