



RESEARCH ARTICLE

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## OPTICAL COHERENCE TOMOGRAPHY ANGIOGRAPHY FINDINGS IN CHRONIC CENTRAL SEROUS CHORIORETINOPATHY AFTER PHOTODYNAMIC THERAPY

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### ABSTRACT

The authors are commenting on the study entitled “Optical coherence tomography findings in chronic central serous chorioretinopathy after photodynamic therapy” published by Tang *et al.* (2019), in *Ophthalmic Surg Lasers Imaging Retina* 2019; 50(1):25-32. The study concluded that optical coherence tomography angiography may provide new insights into the pathogenesis of chronic central serous chorioretinopathy, namely in identifying a correlation between structures of the deep choroid and changes in the retinal pigment epithelium and neurosensory retina. However, the validation, extrapolation, and generalizability of these findings to real-world clinical practice can be made only after analysis of all the missing baseline potential predictive factors mentioned by us in addition to the baseline characteristics already assessed in this study, serving to identify the key drivers correlated with pathogenesis of the chronic central serous chorioretinopathy.

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### INTRODUCTION

We read with great interest the prospective study by Tang *et al.* (2019) which reported the acute and long-term choroidal findings on optical coherence tomography angiography (OCTA) in 21 patients with chronic central serous chorioretinopathy (CCSC) undergoing half-fluence photodynamic therapy (PDT). Among 21 eyes, all had improved vision following PDT during a mean of 5.2 months of follow-up. Changes in the choriocapillaris were noted in 28.6% eyes following PDT and 95.2% eyes experienced resolution of subretinal fluid (SRF) within 3 months of receiving PDT. We would like to address several challenges with the study of Tang *et al.* (2019) which can be specifically summarized below.

The CCSC resides within the pachychoroid disease spectrum (Cheung *et al.* 2019). However, the characteristic abnormalities of the pachychoroid disease phenotype and the retinal pigment epithelial band – Bruch’s membrane complex, which are primarily involved in the CCSC and have a pivotal

contribution in the CCSC pathogenesis, have not been fully documented with the multimodal imaging at baseline compared to the end of the study.

Regarding the pachychoroid phenotype, there were no data on the assessment of the following relevant alterations: the increased permeability of choroidal vasculature with extravascular leakage, one of the hallmarks of the CCSC imaging; the increase in choroidal thickness (focal or diffuse) usually correlated with choroidal vascular hyperpermeability, which can result from focal or diffuse dilation of the large choroidal vessels; the distribution of the pachyvessels in the Haller’s layer (in a diffuse or patchy manner) localized within the areas of increased choroidal vascular permeability on the indocyanine green angiography (ICGA); the focal or diffuse attenuation of the inner choroid layer (thinning/absence of the choriocapillaris and intermediate caliber vessels within Sattler’s layer in areas overlying abnormally dilated Haller’s layer vessels); and the focal choroidal excavations.

Choroidal neovascular membranes (CNVM) were present in 4 of 21 eyes (19%), with evidence of distinct tangled vascular networks in all eyes prior to PDT. It is noteworthy that the neovascular network assessed by OCTA in the outer retina

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ascertained existence of the neovascular CCSC but was unable to specify the type of CNVM, namely, type 1 CNVM, located under the retinal pigment epithelium (RPE) or type 2 CNVM located in the subretinal space, above RPE. Of note, the perfusion indices (density of blood vessels and flow index) were not calculated for the choriocapillaris zone on the OCTA.

With respect to the qualitative status of the RPE, which has been compromised by choroidal abnormalities in patients with CCSC, there are no data relating to the structural optical coherence tomography patterns of some changes of the retinal pigment epithelial band – Bruch's membrane complex including pigment migration within the neurosensory retina, RPE porosity, microrips or blowouts in the RPE, focal RPE atrophy, RPE hypertrophy, existence or not of the pachydrusen that correspond to punctate hyperfluorescent spots on ICGA, diffuse or scattered leakage through the RPE, and diffuse ooze within or adjacent to the decompensated RPE (Călugăru *et al.* 2018, 2019). Likewise, there were no data regarding the existence or otherwise of the 2 optical coherence tomography patterns of the pigment epithelial detachment (PED), namely, the hyperreflective irregular flat PED (undulating RPE detachment) with a double layer sign suggesting the neovascular CCSC and the serous PED with internal hyporeflectivity certifying the non-neovascular CCSC because OCTA revealed no abnormal vessel configuration in the outer retina. The distinction should be made because neovascular CCSC is associated with a worse outcome in terms of distance and reading visual acuity (worse initial and final values) compared to non-neovascular CCSC. In addition, nothing was stated regarding classification of the CCSC patients in two groups, namely, the focal CCSC which has a maximum of 1 hot spot of leakage detected by fluorescein angiography and the diffuse CCSC which has either > 1 hot spot of leakage or a larger area of hyperfluorescent leakage (extensive RPE disruptions with widespread RPE decompensations) not directly linked to 1 point in origin.

There were no data at presentation and at the end of the study referring to the following alterations of the overlying neurosensory retina which may suffer progressive and irreversible damages in cases of the CCSC because of the persistence of the SRF caused by the pronounced dysfunctional RPE outer blood-retinal barrier with severe widespread RPE decompensation: the thinning of the outer nuclear layer, the disruption of the ellipsoid zone, the elongation of the photoreceptor outer segments, the interdigitation zone loss, the hyperreflective deposits frequently accumulated in the subretinal space below the detached neurosensory retina, and the external limiting membrane band defects allowing fluid to enter the retina and causing intraretinal fluid in some cases, sometimes referred to as "cystoid macular degeneration" (Călugăru *et al.* 2018a). Moreover, the perfusion indices for the outer retinal zone (photoreceptor) were not calculated on the OCTA. Of note, although the outer retina does not have vessels, the perfusion indices can be still determined (Călugăru *et al.*, 2018, 2018a).

There were no data referring to the baseline serum potassium levels, the renal function, the level of endogenous and exogenous corticosteroids, the type personality of the patients, and the testing of patients with regard to the *Helicobacter*

*pylori* infection (Călugăru *et al.* 2019a). Altogether, the authors of this study documented that OCTA may provide new insights into the pathogenesis of CCSC, namely in identifying a correlation between structures of the deep choroid with areas of aneurysm corresponding to areas of focal leakage on the corresponding fluorescein angiography and changes in the RPE and neurosensory retina. However, the validation, extrapolation, and generalizability of these findings to real-world clinical practice can be made only after analysis of all the missing baseline potential predictive factors mentioned by us in addition to the baseline characteristics already assessed in this study, serving to identify the key drivers correlated with pathogenesis of the CCSC.

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### Conflict of interest

All authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers' bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent-licensing arrangements), or non-financial interest (such as personal or professional relationships, affiliations, knowledge or beliefs) in the subject matter or materials discussed in this manuscript.

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