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RESEARCH ARTICLE

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LONG-TERM VISUAL OUTCOMES AND CAUSES OF VISION LOSS IN CHRONIC CENTRAL SEROUS CHORIORETINOPATHY

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ABSTRACT

The authors are commenting on the study entitled “Long-term visual outcomes and causes of vision loss in chronic central serous chorioretinopathy”, published by Mrejen *et al.* in *Ophthalmology* 2019;126(4):576-588, which evaluated the long-term visual outcomes and causes of vision loss in chronic central serous chorioretinopathy. The authors of this study concluded that the age at presentation and outer retinal changes on multimodal imaging were associated with long-term best-corrected visual acuity changes and may be predictors of long-term visual outcomes. However, the validation, extrapolation, and generalizability of these outcomes can be made only after inclusion in the stepwise multivariate logistic regression analysis of all the missing data mentioned by us in addition to the baseline characteristics already assessed in this study, serving the identification of the significant putative biomarkers of long-term visual outcomes.

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INTRODUCTION

We enjoy reading the study by Mrejen *et al.* (2019) which evaluated the long-term visual outcomes and causes of vision loss in chronic central serous chorioretinopathy (CSC). The authors concluded that later age of disease onset was associated with significantly greater best-corrected visual acuity (BCVA) change at the 10-year follow-up. We would like to discuss several issues that have arisen from this study, which can be specifically summarized below.

The work relied on a retrospective study design with rather low percentages of eyes/patients with chronic CSC examined at the 1, 5, and 10-year follow-up visits (60.3%/61.65%, 67.28%/69.17%, and 58.98%/57.89, respectively), which might cause an inadvertent bias. The 7 years difference between the onset of the disease and the first visit was a significant determinant of the change in visual acuity that strengthens this bias. In addition, 47.9 % of eyes did not receive any treatment and the remaining of 52.1% of eyes received a variety of treatments (photodynamic therapy, thermal laser or anti-vascular endothelial growth factor [VEGF] therapy or a combination of thereof). Likewise, there was a history of corticosteroid use which is an important risk factor

for the development of CSC, either orally, parentally or via inhalation in 33 patients (24.8%). Taken together, these issues make interpretation of the results challenging.

The CSC resides within the pachychoroid disease spectrum (Cheung *et al.* 2019). However, the following characteristic abnormalities of the pachychoroid disease phenotype, which is primarily involved in the CSC and has a contribution in the CSC pathogenesis, have not been documented with the multimodal imaging technique at presentation in subjects of this study: the subfoveal choroidal thickness and the choroidal vascular hyperpermeability highlighted on indocyanine green angiography; the increase in choroidal thickness (focal or diffuse); the distribution of the pachyvessels in the Haller's layer (in a diffuse or patchy manner); the focal or diffuse attenuation of the inner choroid (thinning/absence of the choriocapillaris and intermediate caliber vessels within the Sattler's layer in areas overlying abnormally dilated Haller's layer vessels); and the foveal choroidal excavations. Of note, the perfusion indices (density of blood vessels and flow index) were not calculated for the choriocapillaris zone on the optical coherence tomography (OCT) angiography. In addition, the OCT angiography should have been used for detection of the choroidal neovascularization (CNV) secondary to chronic CSC not visible with the conventional imaging techniques (dye

angiography) and which appears to be helpful to show an abnormal blood flow corresponding to CNV complicating the chronic CSC. Conceivably, the percentage of eyes with the type 1 CNV, which nourishes the outer retina and retinal pigment epithelium (RPE), would have been higher than that found by the authors in this study (23.9%), if the OCT angiography had been used (Călugăru *et al.* 2018).

There are no data at presentation and at the end of the follow-up period relating to the OCT patterns of the following alterations of the retinal pigment epithelial band – Bruch membrane complex which has been compromised by choroidal abnormalities: pigment migration within the neurosensory retina, retinal pigment epithelium (RPE) porosity, microrips or blowouts in the RPE, focal RPE atrophy, RPE hypertrophy, types of pigment epithelial detachment (PED) (serous PED with internal hyporeflectivity and hyperreflective shallow irregular flat, undulating PED with double layer sign), diffuse ooze within or adjacent to the decompensated RPE, and patterns of the RPE leak of fluid into the subretinal space on dye angiography, namely, no leakage; single focal spot of leakage (ink blot/smoke stack/vague); or multifocal diffuse leakage.

There were no data at presentation referring to the multimodal imaging of the following alterations of the overlying photoreceptor cell layer, which may suffer progressive and irreversible damages in cases of chronic CSC because of the persistence of the subretinal fluid caused by pronounced dysfunctional RPE outer blood-retinal barrier: thinning of the outer nuclear layer, disruption of the ellipsoid zone, elongation of the photoreceptor outer segments, interdigitation zone loss, morphologic changes in the appearance of the outer border of the photoreceptor layer (smooth, granulated, or as scattered dots attached to external limiting membrane), and external limiting membrane band defects allowing fluid to enter the retina, sometimes referred to as “cystoid macular degeneration” (Călugăru *et al.* 2018a). Likewise, the location of these intraretinal cystoid spaces on OCT without intraretinal leakage on fluorescein angiography (ganglion cell layer or inner/outer retinal layers) was not highlighted. Moreover, the perfusion indices for the outer retinal zone (photoreceptor) were not calculated on the OCT angiography. Of note, although the outer retina does not have vessels, the perfusion indices can be still determined.

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.

The final results of this study were poor. Although 79.7 % of patients maintained at final visit driving-standard vision with BCVA of 20/40 or better in at least one eye, there was a significant mean loss of 4.5 Early Treatment Diabetic Retinopathy Study letters over a period of 10 years and 12.8 % of patients were legally blind with BCVA of 20/200 or worse in both eyes at the end of the study. Cystoid macular degeneration persisted at 21.7% of eyes, central subfoveal thickness had pathological value (436.8 μm) certifying unresolved macular edema due to insufficient macular deturgescence, and disruption of the ellipsoid zone and external limiting membrane were present in quite high percentages of eyes (41.5% and 67.3%, respectively). Of note, there is no mention of the proportion of eyes with completely resolved subretinal fluid at the end of the follow-up period.

One explanation of these unsatisfactory outcomes is that 47.9% of the eyes did not receive any treatment during a period of 18.3 years ranging between the onset of the disease and the last visit of this study whereas the remaining 52.1% of the eyes received treatment over a 11.3-year period beginning with 7 years after the onset of the disease and spanning between the first and last visit of this study. On the other hand the study included patients whose disease onset occurred at the mean age of 53.7 years and their first visit was made at the mean age of 60.7 years, an age above the middle age of the CSC patients whose peak normally lies between 40 and 50 years. Importantly, the elderly group patients are associated with a lower resolution of the PED, increased impairment of the RPE layers, foveal thinning, and worse vision outcomes compared with the young group suggesting a chronic insult to the choroidal vessels involving more severe damages to the outer retinal layers (Bae *et al.* 2019).

Altogether, the authors of this study concluded that the age at presentation and outer retinal changes on multimodal imaging were associated with long-term BCVA changes and may be predictors of long-term visual outcomes. However, the validation, extrapolation, and generalizability of these outcomes can be made only after inclusion in the stepwise multivariate logistic regression analysis of all the missing data mentioned by us in addition to the baseline characteristics already assessed in this study, serving the identification of the significant putative biomarkers of long-term visual outcome (Călugăru *et al.* 2018a).

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All authors have completed and submitted the ICMJE form for disclosure of potential conflicts of interest. No financial disclosures. Both authors (D.C and M.C) were involved in the design and conduct of the study; collection, management, analysis and interpretation of the data; and preparation, review or approval of the manuscript. The authors have full control over the primary data and they agree to allow the International Journal of Development Research to review their data if requested.

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