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CLINICAL CHARACTERISTICS AND OUTCOME OF POSTERIOR CYSTOID MACULAR DEGENERATION IN CHORNIC CENTRAL SEROUS CHORIORETINOPATHY

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ABSTRACT

The authors are commenting on the study entitled**:** "Clinical characteristics and outcome of posterior cystoid macular degeneration in chronic central serous chorioretinopathy" published by Mohabati *et al*. in Retina Journal 2020;40(9):1742-1750, which evaluated clinical characteristics and visual outcome in 62 chronic central serous chorioretinopathy patients (83 eyes) with posterior cystoid retinal degeneration. After performing the multivariate analysis, older age at initial central serous chorioretinopathy diagnosis remained a statistically significant predictor for posterior cystoid retianal degeneration resolution. However, the validation, extrapolation, and generalizability of these findings can be made only by regression analyses including all the missing data adduced to above by us in addition to the baseline characteristics already assessed in this study, serving to identify the potential prognosticators influencing the final visual acuity and resolution of the subretinal fluid and posterior cystoid retinal degeneration

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INTRODUCTION

We read with great interest the article by Mohabati *et al.* (2020) entitled "Clinical characteristics and outcome of posterior cystoid macular degeneration in chronic central serous chorioretinopathy" published in Retina Journal 2020; 40(9):1742-1750. The study evaluated clinical characteristics and visual outcome in 62 chronic central serous chorioretinopathy (cCSC) patients (83 eyes) with posterior cystoid retinal degeneration (PCRD). 81% of the eyes received treatment and PCRD had resolved completely in 31 eyes (37%) at the final visit. Best-corrected visual acuity did not improve even after complete PCRD resolution. The authors found that PCRD is a relatively common finding in cCSC, which is often accompanied by active subretinal fluid leakage. We would like to address several challenges that have arisen from this study, which can be specifically summarized below.

The study included patients with evidence of persistent subretinal fluid for at least 3 months and retinal pigment epithelium (RPE) window defects all of them having cCSC-associated PCRD at some point during follow-up.

Although one of the exclusion criteria was the occult choroidal neovascularization, the study encompassed 30 (36%) PCRD eyes associated with pigment epithelial detachments displaying a hyperreflective irregular flat type on optical coherence tomography what showed the presence of the type 1 choroidal neovascularization and thus certifying the neovascular cCSC. Therefore, the study should have been carried out separately for the 2 types of cCSCs (neovascular and non-neovascular cCSCs) because they have completely different prognoses. It is assumed that the proportion of the neovascular cCSCs would have been higher in this study if the optical coherence tomography angiography, which allows detection of choroidal neovascularization secondary to cCSC not visible with other imaging techniques (neovascular cCSC) and which seems to be helpful to show an abnormal blood flow corresponding to choroidal neovascularization complicating the cCSC, had been used (Călugăru *et al*. 2018).

The CSC resides within the pachychoroid disease spectrum (Cheung *et al.* 2019). However, the characteristic abnormalities of the pachychoroid disease phenotype (primary choroidopathy) and the RPE dysfunction, which are primarily

involved in the CSC and have a contribution in the CSC pathogenesis, have not been fully documented with the multimodal imaging in subjects of this study.

As pertains to the pachychoroid phenotype, there were no data on the assessment of the following alterations at enrollment and at the completion of the study: the increase in subfoveal choroidal thickness (focal or diffuse) usually correlated with choroidal vessel hyperpermeability, which can result from focal or diffuse dilation of the large choroidal vessels; the increased permeability of choroidal vasculature with extravascular leakage, one of the hallmark of cCSC imaging; the distribution of the pachyvessels in the Haller's layer (in a diffuse or patchy manner) localized within the areas of choroidal vascular hyperpermeability; the focal or diffuse attenuation of the inner choroid (thinning/absence of the choriocapillaris and intermediate caliber vessels within 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 angiography.

In reference to the qualitative status of the RPE, which has been compromised by choroidal abnormalities in patients with CSC, the study presented thoroughly the RPE window defects and the presence of the diffuse atrophic RPE alterations directly underneath PCRD which were observed in 83% of the cases. However, nothing was stated referring to the optical coherence tomography patterns of the pigment epithelial detachment associated with PCRD at final visit (e.g., serous pigment epithelial detachment with internal hyporeflectivity, hyperreflective irregular flat pigment epithelial detachment with a double layer sign or no pigment epithelial detachment association) and the diffuse ooze within or adjacent to the decompensated RPE.

There were no data referring to the multimodal imaging of the overlying photoreceptor cell layer which may suffer progressive and irreversible damages in cases of the cCSC because of the persistence of the subretinal fluid caused by pronounced dysfunctional RPE outer blood-retinal barrier with severe widespread RPE decompensation. Specifically, these alterations include: thinning of the outer nuclear layer, external limiting membrane band defects, discontinuity of the junction between inner and outer segments, 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 hyperreflective deposits frequently accumulated in the subretinal space below the detached neurosensory retina. Moreover, the perfusion indices (density of blood vessels and flow index) for the outer retina zone (photoreceptor) were not calculated on optical coherence tomography 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 satisfactory. That is, 82% of the eyes had complete resolution of the subretinal fluid, the PCRD, which is more therapy-resistant than subretinal fluid in cCSC, was completely resolved in 37% of the eyes, and the best-corrected visual acuity improved significantly in patients with PCRD located between the optic disc and the foveal region without peripapillary or foveal involvement (papillomacular region). Taking these outcomes into account we believe that the wording of PCRD cannot be applied to all examined and treated PCRD eyes in this study. The term "degeneration" implies an irreversible retinal cell loss (cell disorganization and cell death by atrophy) with loss of natural pumping function of the RPE cells and the Muller cells, followed by the intraretinal cystoid cavity formation with no possibility of reversibility of the condition after treatment, even after PCRD resolution. This term was suitable for 63% of the PCRD eyes in this study, with reduced (21%), increased (6%), and unchanged (36%) PCRD after photodynamic therapy. For all the other eyes with intraretinal fluid and subretinal fluid (37%) that have responded to treatment with complete resolution of the PCRD at the final visit, the term posterior cystoid retinal edema seems to be more appropriate being associated with vascular hyperpermeablity and active leakage and emphasizing the possibility of reversion of the condition after treatment, once the edema has resolved. Therefore, that is why the differentiation between the PCRD and the posterior cystoid retinal edema should have been done.

The 2 components of the pathogenetic mechanism in PCRD manifestation were carefully presented in this study, namely, a homeostatic fluid imbalance component, leading to intraretinal and subretinal fluid, which seems more likely to respond to photodynamic therapy, and a degenerative component leading to tissue loss, which is less likely to respond to treatment. However, nothing was stated about the third component of this mechanism, namely, the external limiting membrane band defects allowing fluid to enter the retina, which is probably the most important trigger driver of the condition, without which the fluid could not have entered the retina, and thus causing subretinal fluid in some cases, sometimes referred to as "cystoid macular degeneration".

Altogether, the authors concluded that the treatment may be beneficial to stop subretinal fluid leakage component but is less likely to result in a complete PCRD resolution and/or a best-corrected visual acuity improvement. After performing the multivariate analysis, older age at initial cCSC diagnosis remained a statistically significant predictor for PCRD resolution. However, the validation, extrapolation, and generalizability of these findings can be made only by regression analyses including all the missing data referred to above by us in addition to the baseline characteristics already assessed in this study, serving to identify the potential prognosticators influencing the final visual acuity and resolution of the subretinal fluid and PCRD.

REFERENCES

Mohabati D, Hoyng CB, Yzer S, et al. Clinical characteristics and outcome of posterior cystoid macular degeneration in chronic central serous chorioretinopathy. Retina Journal 2020:40(9):1742-1750.

Călugăru D, Călugăru M. Clinical features of central serous chorioretinopathy with type 1 choroidal neovascularization. Am J Ophthalmol 2018;195(November);245-246.

Cheung CMG, Lee WKI, Koizumi H, et al. Pachychoroid disease. Eye 2019;33(1):14-33.

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