Letter to the Editor

The value of choroidal thickness in diabetic macular oedema is contradictory

António Campos1,2,3,4, Elisa J. Campos5,6,7, João Martins1,2,6,7, and Rufino Silva1,5,8

1Coimbra Institute for Clinical and Biomedical Research (iCBR), University of Coimbra, Coimbra, Portugal; 2University of Coimbra Center for Innovative Biomedicine and Biotechnology (CIBB), Coimbra, Portugal; 3Department of Ophthalmology, Centro Hospitalar Leiria EPE, Leiria, Portugal; 4ciTechCare, Center for Innovative Care and Health Technology, Polytechnic Institute of Leiria, Leiria, Portugal; 5Association for Innovation and Biomedical Research on Light and Image (AIBILI), Coimbra, Portugal; 6Coimbra Institute for Biomedical Imaging and Translational Research (CIBIT), University of Coimbra, Coimbra, Portugal; 7Instituto de Ciências Nucleares Aplicadas à Saúde (ICNAS), University of Coimbra, Coimbra, Portugal; 8Department of Ophthalmology, Centro Hospitalar e Universitário de Coimbra (CHUC), Coimbra, Portugal

doi: 10.1111/aos.14529

Editor,

The choroidal thickness (CT) or the subfoveal choroidal thickness (SFCT) have been indicated as prognostic factors for the treatment of diabetic macular oedema (DMO), including in recent works (Mathis et al. 2020; Endo et al. 2020). Authors usually engage the decrease in CT and the decrease in central retinal thickness (CRT) under the action of anti-vascular endothelial growth factor (VEGF) agents, in a cause–effect relationship. Therefore, it is common to see the conclusion that the CT is a marker of DMO outcome. One has to consider that the VEGF action through its receptors 1 and 2 may be independent in the retina and in the choroid and acting in different cell types, including pericytes (Fig. 1). Müller cells (MC) and the fenestrated pores of the choriocapillaris (Darwich et al. 2018). The association of VEGF-dependent thinning of the retina and thinning of the choroid may be appealing, but they may be just two independent actions of the anti-VEGF agents and not necessarily being correlated one another. Drying of the retina is mainly dependent on MC at the retinal level, and of the presence of pores in the fenestrated endothelium of the choriocapillaris at the retinal pigment epithelium (RPE) level. However, while anti-VEGF agents thin the choroid, they reduce the number of pores at the choriocapillaris in a transient way, as demonstrated by electron microscopy studies (Shimomura et al. 2009).

In a prospective work, we found that the anti-VEGF thinning of the choroid has no prognostic value for the treatment of DMO, when we stratify patients by outcome. Choroidal thickness (CT) decrease was just an indicator of anti-VEGF side effect on the choroid and the time curves of CRT and SFCT did not overlap (Campos et al. 2018).

In the report of Endo et al., if CT increase in DMO was to be dependent on the level of VEGF, as stated by the authors, it is hard to explain why eyes of patients with proliferative diabetic retinopathy had lower CT, despite having higher CRT, than patients with non-proliferative diabetic retinopathy. On the other hand, VEGF levels depend on systemic treatment of diabetes, but the authors did not find that systemic treatment of diabetes significantly changed CT (Endo et al. 2020).

In the work of Mathis et al. (2020), there was a relation of CT increase with the anti-VEGFs’ subsiding effect on the choroid, but it was not clear that monitoring CT would be more useful to detect recurrence than monitoring CRT. It is possible that an anti-VEGFs’ waning effect on the choroid does not imply a need for another treatment if the CRT remains stable, that is, changes
in CT do not necessarily forecast a DMO recurrence and a need for an additional injection (Mathis et al. 2020). Furthermore, it is to be expected that the choroid thickens as the anti-VEGF effect subsides, because CT decreases as a side effect of anti-VEGF administration (Campos et al. 2018). It would be interesting if the authors would have mentioned the time elapsed from the latest injection before inclusion. Of course, the lack of data on the baseline CT before starting the treatment was also a shortcoming, since unilateral DMO implies that fellow eyes may not be alike.

In conclusion, CT as a surrogate of choroidal inflammation in diabetes, of choroidal flux or of DMO outcome is still under dispute. The value of CT thickening as an indicator of recurrence as the anti-VEGF effect wanes needs further comparison with treated patients that have no recurrence after the anti-VEGF effect subsided. It is debatable whether CT has any advantage over CRT as an indicator of DMO recurrence.

References