Reply to Comment on: Foveal Crack Sign: An Optical Coherence Tomography Sign Preceding Macular Hole After Vitrectomy for Rhegmatogenous Retinal Detachment

WE THANK ARRIGO AND ASSOCIATES FOR THEIR POSITIVE and insightful comments on our work[1] and agree with their opinion that a primary alteration of the Müller cell cones might cause the onset of intraretinal hyperreflective lines (IHLs)/hyperreflective foveal spots (HFSs)/hyperreflective stress lines (HSLs) and foveal crack sign (FCS) alteration. We clarified that hyperrefractive lines on optical coherence tomography were preceding secondary macular holes (MHs) after pars plana vitrectomy for rhegmatogenous retinal detachment. We named these lines the *foveal crack sign* and interpreted FCS as the dehiscence of Müller cell cones caused by parafoveal epiretinal membrane (ERM) traction.

The IHL/HFS/HSL reported in previous studies[2–4] probably represents the same and the change or damage to Müller cell cones by inflammatory, degenerative, or tractional conditions. Basically, IHL/HFS/HSL in the previous studies includes the FCS of our study. IHL/HFS/HSL could, thus, be roughly divided into 2 types in terms of the development of MH.[2–4] The first type reflects the disruption of Müller cell cones by tangential traction and cause MH. The other type reflects the stress or damage to Müller cell cones by conditions other than traction, such as after closure of MH and resolution of macular hemorrhage, which would not cause MH. FCS would be of the first type because all of them were accompanied by parafoveal ERM[1]; that is, FCS would be included in IHL/HFS/HSL. Also, the representative case (Case 5, Figure 1 in our article[1]) showed that localization of this line extended from the inner retinal layer to the outer retinal layer as time proceeds; this suggest that the localization of this line may vary depending on the progression of tractional condition.

Future studies about this line (IHL/HFS/HSL, including FCS),[2–4] caused by either tangential traction or of the other type, should be separated (based on previous studies) because the data become noise when various case types are mixed. Additionally, as Arrigo and associates pointed out, the involvement of the retinal vascular network on this hyperreflective line (IHL/HFS/HSL) is interesting, especially in cases where the line resulted from damage or stress on the Müller cell cones by conditions other than traction. Although we focused on the tractional line, and clarified that this line (FCS) would be associated with MH, there are many unknowns, such as the relationship between the localization of this line and MH development; therefore, further larger studies are required.

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[1–4]SEE THE ORIGINAL ARTICLE[1] FOR ANY DISCLOSURES OF the authors.

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Comment on: Morphologic Features of Buried Optic Disc Drusen on En Face Optical Coherence Tomography and Optical Coherence Tomography Angiography

EDITOR:

WE READ WITH INTEREST THE WORK OF KIM AND ASSOCIates[1] and their use of optical coherence tomography angiography (OCTA) to define peripapillary vascular structures in eyes with presumed optic disc drusen (ODD) and optic disc edema (ODE) in an effort to understand pathogenesis and local architecture. We leave the question of their definition of ODD to a separate letter for which we are co-authors and focus here on the reported vascular abnormalities in various etiologies of ODE including papilledema, optic neuritis, and nonarteritic anterior ischemic optic neuropathy (NAION). They report a significant qualitative decrease in retinal peripapillary capillary (RPC) densities both in eyes with ODE of all causes together and ODD, and state that this occurs without