Correspondence

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

DEAR EDITOR:

WE READ WITH GREAT INTEREST THE RECENTLY PUBLISHED article entitled "Foveal Crack Sign: An Optical Coherence Tomography Sign Preceding Macular Hole After Vitrectomy for Rhegmatogenous Retinal Detachment."[1] In this study, Ishibashi and associates assessed the relatioship between thisparticular structural optical coherence tomography (OCT) alteration, namely, a vertical hyperreflective intraretinal foveal line, and the onset and progression of macular holes (MHs).

This alteration was very similar to what our group previously described as hyperreflective foveal spots (HFSs).[2] In particular, we found an association between this structural OCT alteration and different vitreomacular alterations, including MH, eyes without vitreomacular interface abnormalities (VMIAs), with VMIAs, or with vitreous adhesion. Furthermore, we found a significant impairment of the retinal vascular network, compared to healthy eyes, thus suggesting an involvement of the intraretinal vascular plexa also.[2]

More recently, Amoroso and associates reported the presence of this vertical hyperreflective alteration, which they named intraretinal hyperreflective lines (IHLs), in extremely heterogeneous retinal disorders, thus expanding this finding also beyond vitreomacular-related disorders.[3]

In addition, another recent article by Scharf and associates further confirm the link between this biomarker, which they named hyperreflective stress lines (HSLs), and MH.[4]

Although conducted by independent research groups, all these studies agreed in interpreting HFS/IHL/HSL alteration as the result of a primary alteration of the Müller cells, with eventual secondary effects caused by traction forces coming from the altered vitreoretinal interface, thus leading to the development of MH.

Indeed, Müller cells are the most represented cytotype of the vertebrate retina,[5] with a maximal concentration localized in the foveal region. Considering the peculiar organization of foveal Müller cells, with a Müller cells–photoreceptors ratio of 1:1,[6] it is perfectly assumable that a primary alteration of this specific cytotype might cause the onset of IHL/HFS/HSL alteration.

However, the spectrum of presentation of IHL/HFS/HSL may involve diseases characterized by totally different pathogeneses,[3] thus suggesting a more complex etiopathogenetic mechanism for the development of this lesion. Moreover, the effective localization of IHL/HFS/HSL may vary from full foveal thickness to limited containment in the outer retinal layers, leading to the hypothesis of varying involvement of other retinal cytotypes, including retinal pigmented epithelium cells and other glialcells.[3]

All these considerations strongly suggest that further larger studies are warranted to effectively characterize the pathogenesis of IHL/HFS/HSL and its possible role in each specific macular disease. Furthermore, intriguing perspectives should include the assessment of the relationship among the onset of IHL/HFS/HSL, the progression of each macular disease, and the involvement of the retinal vascular network.

In conclusion, we would like to praise Ishibashi and associates[1] for their excellent study, which strongly reinforced what previously described regarding this peculiar structural OCT alteration.

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