Comment on: Vitreomacular Adhesion and Its Association With Age-Related Macular Degeneration in a Population-Based Setting: The Alienor Study

We read with interest the recent study of Gattoussi et al.1 investigating the association between vitreomacular adhesion (VMA) and AMD. The authors did not find any statistically significant associations of VMA with any stage of AMD. This result is in contrast to other previous studies that have found an association between VMA and exudative AMD, suggesting that VMA might be a risk factor for choroidal neovascularization (CNV) development.

As Gattoussi et al.1 appropriately pointed out, there are a number of limitations in previous studies2–5 that were overcome in their recent study. In addition to that already emphasized by the authors, the larger study population and the use of spectral-domain optical coherence tomography (SD-OCT) also should be considered. Many previous studies2–5 were performed before SD-OCT became available. The low resolution and higher number of artifacts of time-domain OCT compared with SD-OCT reduce the accuracy of evaluation of the vitreoretinal interface and may compromise the results. Moreover, they used the OCT-based International Classification System developed by the International Vitreomacular Traction Study Group for grading vitreomacular interface status. This allowed a standardized definition of the vitreomacular interface configurations, reducing the risk of misinterpretations. In our view, these aspects significantly contribute to increase the reliability of their results.

Nevertheless, as specified by the authors, the temporal relationship of VMA and AMD could not be assessed due to the cross-sectional design. In this regard, the authors consider the possibility that patients might have had AMD for many years, with VMA at the moment they develop AMD, which then progressed to complete posterior vitreous detachment (PVD). Thus, they suggest that a causative association between VMA and AMD might have been masked by occurrence of a complete PVD after the onset of AMD. Another possibility, not mentioned in the article, is that a role of VMA as a consequence of exudative AMD might have been masked, because, due to the cross-sectional design, they could not determine a reduction in the occurrence of PVD consequent to the activity of CNV. In fact, the study does not allow to differentiate the prevalence of VMA in eyes with early stages of exudative AMD compared with late stages.

In agreement with the results of Gattoussi et al.,1 in our recent study about the prevalence of VMA in AMD,6 we found no association between the state of the vitreomacular interface and the presence of AMD. Interestingly, we also found that the incidence of release of VMA at the end of the follow-up was significantly lower in exudative AMD eyes compared with nonexudative AMD. This implies the possibility that VMA might be a consequence instead of a cause of exudative AMD.

Despite the cross-sectional design of their study,1 we believe that a different subgroup classification of the study population could have allowed the authors to provide some information about the likelihood that the exudative processes owing to CNV might increase the incidence of VMA. In particular, they gathered together heterogeneous types of eyes in the “late AMD” group, including all the stages of exudative AMD, of both recent onset and advanced. We think that a partition of the eyes into “early” exudative AMD, “advanced” exudative AMD (i.e., fibrotic scars), and nonexudative AMD could have provided additional meaningful information about the cause-effect relation between VMA and CNV.

As stated by the authors, additional prospective studies are required to define precisely the role of VMA in AMD development.

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References


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