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LETTER

Assessing the Role of VISTA in Vascular Cognitive Impairment: Addressing Limitations and Exploring Future Research Directions [Letter]

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Dear editor

We have read with great interest the recent publication by Liu discussing the association between VISTA expression and vascular cognitive impairment (VCI) in older Chinese adults.[1] This study is timely and provides valuable insights into the role of immune regulatory mechanisms in the pathophysiology of VCI. However, we would like to highlight several areas that warrant further exploration and propose directions for future research to strengthen the robustness of these findings.

First, these findings underscore the role of VISTA as an immunoregulatory molecule with decreased expression correlating with higher levels of pro-inflammatory cytokines, such as IL-6, and cognitive impairment severity. This aligns with existing literature highlighting the pivotal role of inflammation in the pathophysiology of VCI.[2] By demonstrating that intermediate monocytes exhibit reduced VISTA expression and are independently associated with VCI, the study adds a valuable dimension to our understanding of immune contributions to vascular dementia.

Despite its strengths, the study's cross-sectional design limits causal inferences. While the authors hypothesize that reduced VISTA expression promotes a shift toward pro-inflammatory monocyte subsets, longitudinal studies are essential to establish whether this is a cause or consequence of VCI. Furthermore, the sample size of 54 participants may not fully capture the heterogeneity of VCI pathology, especially across diverse ethnic backgrounds.[3] Expanding studies to larger cohorts and diverse populations could improve the generalizability of these findings.

Another intriguing aspect deserving attention is the observed interplay between oxidative stress markers, such as uric acid levels, and VISTA expression. The dual role of uric acid as an antioxidant and a potential contributor to inflammation merits deeper investigation.[4] Future research might focus on delineating the molecular pathways linking VISTA, oxidative stress, and neuroinflammation to better understand their collective impact on cognitive decline.

The study's implication of VISTA as a potential biomarker for VCI is noteworthy. The reported high sensitivity and specificity of VISTA expression in PBMCs and intermediate monocytes in predicting VCI offer promising avenues for early diagnosis. However, validation through multicenter studies and the integration of other biomarkers, such as neuroimaging and genetic profiling,[5] could enhance diagnostic accuracy and clinical utility.

In conclusion, the study marks a significant step toward elucidating the immunological underpinnings of VCI. Future investigations should prioritize longitudinal studies, larger and more diverse cohorts, and mechanistic insights into VISTA's role in inflammation and cognitive decline. Additionally, exploring therapeutic strategies targeting VISTA pathways could hold transformative potential for managing VCI. By building upon these findings, we can advance toward effective interventions that mitigate the burden of vascular dementia.

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Disclosure

The authors report no conflicts of interest in this communication.

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