

Assessing the Role of Serum Prealbumin in Prognostic Studies of Stroke: Reflections on Existing Research Methods [Letter]

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Dear editor

We are writing to discuss the study entitled “Serum Prealbumin Levels and Risks of Adverse Clinical Outcomes After Ischemic Stroke¹”, published by Shi et al. This research examines the association between serum prealbumin levels and poor prognosis in ischemic stroke patients, proposing that serum prealbumin could serve as a valuable prognostic biomarker. The study highlights the importance of maintaining good nutrition in managing stroke outcomes. While the methodology is rigorous and the insights are valuable, certain aspects related to variable consideration and data categorization require further exploration.

First, although the authors addressed different stroke subtypes, they did not account for the potential impact of infarct size and location on patient outcomes. Infarcts covering larger areas often result in more severe brain damage, increasing the complexity and challenge of recovery. This can lead to significant neurological deficits and poorer functional outcomes.² Moreover, the specific location of the infarct crucially influences the neurological functions affected.³ For example, infarcts in the anterior circulation region typically result in hemiparesis and speech disorders, whereas those in the posterior circulation region can impair visual and balance functions.⁴

In addition, in making model adjustments, the authors simplified the four baseline lipid measurements from continuous variables to dichotomous variables (normal versus abnormal lipids), adjusting only for abnormal lipids. This may have oversimplified the complex interactions between lipid molecules and ischemic stroke prognosis, potentially resulting in the loss of important information on continuous variables. In the baseline table (Table 1), there were significant correlations ($P < 0.0001$) between different ranges of serum prealbumin levels and the four lipid parameters, suggesting that detailed lipid data can significantly impact outcomes. Therefore, I recommend maintaining the original continuity of lipid data when performing modeling adjustments and subgroup analyses to improve the reliability of study results.

Finally, it is puzzling that the study did not include a history of chronic conditions such as diabetes, hypertension, and dyslipidemia in the subgroup analysis. These comorbidities are considered high-risk factors for poor prognosis in stroke patients and may interact with prealbumin levels.⁵ We are particularly interested in the correlation between prealbumin levels and poor stroke prognosis among these patient subgroups. We believe that including this analysis could demonstrate how targeted nutritional interventions benefit specific subgroups of patients.

The authors conducted a rigorous analysis and laid a solid foundation for future research. However, acknowledging and potentially addressing these limitations could strengthen the study's implications and its application in clinical settings. We recommend that future studies incorporate these variables in their analyses to provide a clearer picture of the interactions between nutrition, lipid management, and stroke recovery.

Thank you for considering these comments. I appreciate the opportunity to contribute to the discussion on this important topic and look forward to further developments in the area of stroke research.

Disclosure

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this communication.

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