

Enhancing the Predictive Value of SII and NLR in LAA Stroke: Addressing Unexplored Limitations and Future Directions [Letter]

E Zhao¹, Zhengting Duan², Jingmei Li¹

¹Department of Rehabilitation, Guangyuan Central Hospital, Guangyuan, People's Republic of China; ²Department of Rehabilitation, The First People's Hospital of Guangyuan, Guangyuan, People's Republic of China

Correspondence: Jingmei Li, Department of Rehabilitation, Guangyuan Central Hospital, Guangyuan, People's Republic of China, Email 13981261881@163.com

Dear editor

We have read with great interest the article by Liu et al, titled "Systemic Immune-Inflammation Index (SII) and Neutrophil-to-Lymphocyte Ratio (NLR): A Strong Predictor of Disease Severity in Large-Artery Atherosclerosis (LAA) Stroke Patients".¹ This study provides valuable clinical insights into the predictive value of SII and NLR for assessing the severity of LAA stroke. However, upon reflection, we would like to highlight several limitations that were not addressed in the study and propose suggestions for improvement to enhance future research in this area.

First, the study does not account for lifestyle factors, such as smoking, alcohol consumption, dietary habits, and physical activity, which can significantly influence systemic inflammatory responses. These lifestyle variables may confound the association between SII, NLR, and stroke severity. For example, smoking is known to elevate inflammation levels,² while regular physical activity may reduce it.³ The absence of adjustment for these factors could lead to potential biases. Future studies should collect detailed information on lifestyle factors and control for their potential effects in statistical analyses to improve the reliability of the findings.

Second, while the authors adjusted for certain clinical factors such as age and comorbidities, they did not adequately address the impact of medication use on the results. Drugs like anti-inflammatory agents (eg, aspirin), statins,⁴ antiplatelet therapies, or corticosteroids can substantially influence inflammation levels or platelet counts, thereby affecting SII and NLR values. Failure to account for patients' medication histories could result in biased interpretations. Future research should systematically collect data on medication use and incorporate it as a covariate in regression models or stratify analyses based on medication exposure to improve the accuracy and clinical relevance of the conclusions.

Third, the study does not explore the differences in inflammatory responses across sex and age groups. Hormonal differences between males and females may influence inflammation levels, while older patients, due to immunosenescence, typically exhibit higher baseline inflammation. Thus, the predictive value of SII and NLR may vary across these subgroups. Additionally, baseline conditions such as diabetes and hypertension may further modulate inflammatory states, complicating the interpretation of these biomarkers. Future research should include stratified or subgroup analyses to evaluate the applicability of SII and NLR in diverse patient populations, thereby supporting more personalized diagnostic and therapeutic strategies.

In conclusion, while the study lays a solid foundation for the clinical application of SII and NLR, addressing the aforementioned limitations would further enhance the robustness and translational value of future research. We highly appreciate the authors' contribution and look forward to further advancements in this important field.

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