LETTER TO THE EDITOR

Inflammation and Hemorrhagic Stroke Outcomes: *Other Players in the Nexus*

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Dear Editor,

In the recent most issue of the *Indian Journal of Critical Care Medicine*, Babu et al. highlighted the on-admission neutrophil–lymphocyte ratio (NLR, a pro-inflammatory marker) as an important predictor of 90-day functional outcome in patients with hemorrhagic stroke.[1] The authors' pragmatic research approach is heralded by the simultaneous inclusion of a clinical-radiological prognostication in the form of intracranial hemorrhage (ICH) score in their prospective observational study.[1] Nonetheless, inflammatory predisposition to poor functional outcomes in stroke is essentially multifaceted wherein additional insights into the topic can prove to be beneficial for the readers of the *Journal*.

Appropriate to the context, the relevance of apprising the inflammatory links of hypoalbuminemia (not accounted for in the Babu et al. study) cannot be overemphasized, particularly amid a prevalent poor nutritional status in our patient subset.[1–5] Moreover, literature exists to suggest poor functional outcomes in background of hypoalbuminemia in patients with an underlying spontaneous subarachnoid hemorrhage (SAH) and intracerebral hemorrhage.[2,3] It would be worthwhile to mention the Kapoor et al. study that refers to albumin as much more than a mere nutritional status marker in a setting of SAH.[2] Staging a prospective evaluation of 273 patients, the research group concluded that a lower on-admission albumin $\leq$3.5 g/dL entails an elevated 3-month risk of new neurological deficits, infarcts, unfavorable Glasgow outcome scale (GOS), and mortality.[2] Interestingly, a negative association of the albumin levels with a hemorrhagic transformation after thrombolysis has additionally been described in ischemic stroke patients.[4]

Talking from a generalized perspective also, recent literature features the incremental prognostic role of composite inflammatory indices such as albumin/NLR score, (or the ANS as coined by Kao et al.)implying an inflammatory interplay of hypoalbuminemia and leukocytic alterations.[5] Needless to say, an elucidation of the potential inflammatory nexus only assumes an enhanced importance in research endeavors like the one by Babu et al., which are aligned with the covetable aim of favorably modulating the functional outcomes in stroke patients.[1,5]

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