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LETTER TO THE EDITOR OPEN ACCESS

Methodological Considerations in Blood Pressure Variability Assessment for Branch Atheromatous Disease

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Dear Editor,

The recent multicenter study examining the relationship between systolic blood pressure variability (BPV) and 90-day functional outcomes in branch atheromatous disease (BAD) provides valuable data on a clinically challenging stroke subtype [1]. Although the authors highlight an independent association between elevated BPV and adverse outcomes, several methodological issues require caution before accepting BPV as a robust prognostic marker in this setting.

A principal limitation is the restricted number of systolic blood pressure measurements used for calculating variability since only three values obtained between 24 and 72 h plus Day-7 readings. BPV metrics such as SD, CV, and VIM require sufficient temporal density to achieve statistical stability, and sparse sampling risks producing artificially inflated or unstable estimates, diverging from standards recommended for short-term BPV assessment [2]. Consequently, the reported tertile-based differences may partially reflect measurement sparsity rather than pathophysiological variability.

Another concern is the incomplete adjustment for hemodynamic and clinical factors known to influence early neurological deterioration (END) and functional outcomes. Variables such as pain, hydration status, sleep–wake patterns, sympathetic activation, and acute-phase medications can modify SBP fluctuations and are not captured in the regression models. Prior work in ischemic stroke populations shows that BPV is highly sensitive to treatment intensity, particularly antihypertensive titration and antithrombotic protocols, which may confound associations attributed to intrinsic disease mechanisms [3].

Interpretation of the discriminative capacity of BPV indices also warrants caution. Although the authors present area-under-the-curve (AUC) values for SD, CV, and VIM, these metrics fall in a range considered weak for clinical prediction, with limited incremental value relative to established prognostic variables. Previous analyses have emphasized that BPV adds marginal discriminatory benefit unless supported by more granular BP monitoring or complementary physiological markers [4]. As such, the study's conclusions may overstate the practical relevance of BPV for risk stratification in BAD.

The distinct anatomical and microvascular features of BAD further complicate the interpretation of BPV as a causal or near-causal factor. BAD lesions lack substantial collateral support, and progression may be driven primarily by perforator occlusion dynamics rather than systemic BP behavior. Emerging mechanistic research suggests that END in BAD is more closely related to local microthrombotic propagation and dynamic perfusion mismatch than to brief systemic BP oscillations [5]. These considerations indicate that BPV may function more as an epiphenomenon than as a modifiable determinant of outcome in BAD.

Sincerely,

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All of the authors have no conflict of interest.

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