THE ANATOLIAN JOURNAL OF CARDIOLOGY

Official journal of the TURKISH SOCIETY OF CARDIOLOGY

Comments on "Evaluation of Whole Blood Viscosity to Predict Stent Restenosis"

LETTER TO THE EDITOR

To the Editor,

I read with great interest the recent article by Çalapkulu et al,[1] which investigated the relationship between whole blood viscosity (WBV) and in-stent restenosis (ISR) in patients with chronic coronary syndrome and prior coronary stent implantation. The authors are to be commended for being the first to demonstrate that both high-shear rate and low-shear rate WBV, calculated via the De Simone formula, are independent predictors of ISR, with excellent discriminative performance in receiver operating characteristic analysis. I believe this important work contributes to expanding the range of simple, inexpensive biomarkers that may assist in pre-procedural risk stratification. Nevertheless, I would like to offer several observations that may help refine the interpretation of the findings.

Firstly, although the authors applied multivariate Cox regression to account for confounding variables, the retrospective, single-center design inherently limits the internal validity of the results. Selection bias may also be present, as the clinical or angiographic factors influencing the decision for initial stent type, diameter, and length—each found to be independent predictors—were not fully detailed. Moreover, procedural details such as balloon inflation time, pre-dilatation, post-dilatation, and use of adjunctive techniques (e.g., high-pressure non-compliant balloons) were not reported, despite their known association with ISR risk.[2,3]

Secondly, WBV was not directly measured with a viscometer but estimated from hematocrit and plasma protein levels using a validated formula. While this approach is practical and has been used in previous cardiovascular cohorts,[4] its accuracy can be influenced by acute-phase responses, hydration status, or laboratory measurement variability. The lack of repeated WBV assessments or evaluation of other hemorheological indices—such as plasma viscosity, erythrocyte deformability, or aggregation—may have limited the mechanistic insights into the pathophysiology of ISR.

Finally, the study population was limited to patients undergoing coronary angiography for chronic coronary syndrome, which may restrict external validity to other clinical settings such as acute coronary syndromes, where inflammatory and rheological profiles differ substantially.[5] Additionally, although the authors highlight the predictive value of WBV, the clinical utility of integrating this parameter into decision-making remains to be determined. Whether a high WBV should prompt intensified secondary prevention, closer surveillance, or even influence stent selection warrants investigation in prospective multicenter studies.

In conclusion, Çalapkulu et al[1] have provided valuable preliminary evidence supporting WBV as a potential predictor of ISR. Future large-scale, prospective trials with standardized procedural protocols, direct hemorheological measurements, and diverse clinical populations are essential to validate these findings and to define how WBV can be integrated into personalized PCI follow-up strategies.

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Cite this article as: Süygün H. Comments on "evaluation of whole blood viscosity to predict stent restenosis". *Anatol J Cardiol.* 2025;XX(X):1-2.

DOI:10.14744/AnatolJCardiol.2025.5731

Declaration of Interests: The author have no conflicts of interest to declare.

Funding: The author declare that this study received no financial support.

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