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# Reply to Letter to the Editor: "Comments on 'Evaluation of Whole Blood Viscosity to Predict Stent Restenosis'"

LETTER TO THE EDITOR
REPLY

To the Editor,

I sincerely thank you for the interest and constructive comments[1] regarding my recent article.[2] In this study, I investigated the association between whole blood viscosity (WBV) and in-stent restenosis (ISR) in patients with chronic coronary syndrome who had undergone coronary stent implantation. I demonstrated that both high-shear rate and low-shear rate WBV were independent predictors of ISR, thereby contributing to the growing body of evidence in this field.[2]

As noted, the retrospective and single-center design inherently carries limitations in terms of internal validity. However, I believe that the relatively large sample size in my study helped reduce the potential impact of selection bias. Furthermore, while certain procedural details such as stent type, diameter, length, and adjunctive techniques were not fully available, I explicitly acknowledged this as a limitation. These factors are indeed recognized to influence ISR outcomes, as reported in previous literature.[3,4]

In my study, WBV was not directly measured using a viscometer but instead estimated from hematocrit and plasma protein levels using the validated De Simone formula. Although this approach may be influenced by biological variability, it has been widely utilized in cardiovascular research.[5] Importantly, the recent work of Nwose and Bwititi reaffirmed its clinical applicability, showing that the method provides acceptable sensitivity and specificity.[6] For this reason, I consider my approach both practical and valid for large patient populations. Nevertheless, I agree that additional hemorheological parameters, such as plasma viscosity and erythrocyte deformability, could provide deeper insights into the mechanisms of ISR.

My cohort was restricted to patients undergoing angiography for chronic coronary syndrome, which inevitably limits generalizability to acute coronary syndromes, where inflammatory and rheological conditions are different. However, I believe that my findings provide preliminary evidence supporting WBV as a potential predictor of ISR and can form the basis for larger, multicenter, prospective studies.

In conclusion, I acknowledge the limitations emphasized and appreciate the perspective that further studies are warranted. I would like to highlight once more that while my results suggest a promising role for WBV, its integration into clinical decision-making will only be clarified through prospective investigations with standardized protocols and diverse populations.

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**Yunus Çalapkulu**

*Department of Cardiology, University of Health Sciences, Ankara Mamak State Hospital, Ankara, Türkiye*

**Corresponding author:**
Yunus Çalapkulu
✉ yunuscalapkulu@gmail.com

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