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LETTER TO THE EDITOR

Radial Wall Strain and μQFR as Complementary Predictors of Risk After Myocardial Infarction

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To the Editor,

The study by Hou et al. offers compelling evidence for the prognostic significance of angiography-derived radial wall strain (RWS) in patients with acute myocardial infarction (AMI) following complete revascularization (CR) [1]. The demonstration that a maximum RWS (RWSmax) > 14.5% in nonsignificant stenoses is independently predictive of major adverse cardiovascular events (MACE) supports the emerging notion that biomechanical stress on the vessel wall—beyond traditional anatomical and flow-based parameters—may indicate plaque vulnerability and future clinical risk.

A particularly notable strength lies in the study's integration of RWS with μQFR analysis. The finding that RWSmax retains predictive value regardless of whether lesions are deemed flow-limiting by μQFR (> 0.8 or ≤ 0.8) is clinically relevant, as it underscores the additive role of biomechanical indices in identifying high-risk lesions often considered physiologically benign. This may call into question the sufficiency of μQFR or FFR-based deferred strategies alone in post-AMI patients and suggests a combined functional and mechanical risk assessment framework could better stratify residual risk [2].

Nevertheless, the study invites further discussion regarding the mechanistic underpinnings of high RWS in nonsignificant lesions. Could localized strain patterns reflect underlying microstructural features not visible by angiography, such as lipid cores or thin fibrous caps? While the authors correlate RWS with OCT-derived characteristics in prior studies, a direct comparison within this specific AMI cohort using intravascular imaging would provide stronger pathophysiological validation. Additionally, the relatively high optimal cutoff for RWSmax (> 14.5%) compared to prior studies raises the question of whether acute coronary syndrome modifies plaque strain behavior or artifactually elevates RWS values. Clarifying this discrepancy through head-to-head RWS analysis in stable versus unstable coronary syndromes could refine its clinical utility.

In summary, this work broadens the paradigm of post-AMI risk stratification. Validation in prospective multicenter trials and exploration of mechanistic links between strain and plaque morphology are warranted to determine whether incorporating RWS into routine assessment can guide therapeutic decisions or justify preventive interventions for angiographically mild lesions.

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Conflicts of Interest

The authors declare no conflicts of interest.

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