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Response to the Letter Regarding "Relationship Between the Severity of Coronary Artery Disease and Renal Function"

Dear Editor,

We thank Engin et al[1] for their careful evaluation of our article and their constructive remarks. We are pleased to clarify the points they have raised.

With respect to the interpretation of the correlation coefficient between estimated glomerular filtration rate (eGFR) and Gensini score ($\rho = -.352$), we agree that, according to commonly accepted statistical conventions, this magnitude is best described as a moderate inverse correlation. In our original wording, the term "strong" was used in a relative sense, as eGFR demonstrated the highest correlation with Gensini score among all measured clinical and laboratory variables in our dataset.[2] To avoid ambiguity, we agree that revising the terminology to reflect the statistically appropriate classification is preferable. Importantly, beyond the unadjusted correlation, renal function remained the strongest independent determinant of coronary atherosclerotic burden in multivariable regression analyses, supporting the clinical relevance of this association.

Regarding the characterization of diabetes mellitus (DM), our study was retrospective and included patients undergoing elective coronary angiography.[2] During the study period, HbA1c measurement prior to angiography was not routinely available for all patients; therefore, standardized longitudinal indicators of glycemic burden could not be incorporated into the analysis. For this reason, DM was modeled as a categorical variable based on established diagnostic criteria. In addition, contemporary cardiovascular risk prediction systems, including Predicting Risk of cardiovascular disease EVENTs equations (PREVENT)[3] and the European Society of Cardiology (ESC), Systematic COronary Risk Evaluation (SCORE2),[4] also use diabetes categorically within their validated risk frameworks. As our study aimed to provide anatomical insight within the context of cardiovascular-kidney-metabolic risk stratification, this approach was considered methodologically consistent. We agree that continuous measures of glycemic control may offer further mechanistic detail; however, even with DM included as a binary covariate, the association between reduced eGFR and higher Gensini score persisted after multivariable adjustment, indicating that the renal-coronary relationship observed is not solely attributable to diabetes status.

Finally, we fully concur that atherosclerosis is a systemic disease. As explicitly discussed in the Limitations section of our manuscript, the cross-sectional design precludes definitive causal inference, and we addressed the possibility of reverse causality, including systemic atherosclerosis leading to renal artery stenosis (ischemic nephropathy) and subsequent reduction in eGFR. Thus, the potential influence of extra-coronary atherosclerotic burden on renal function was directly acknowledged. Our study was intentionally focused on the anatomical coronary substrate across the renal function spectrum rather than on total systemic atherosclerotic burden.[2] We agree that future prospective investigations incorporating comprehensive vascular imaging may further clarify the bidirectional relationship between systemic atherosclerosis and renal impairment.

We appreciate the authors' thoughtful comments, which contribute to a more precise interpretation of our findings and help frame important directions for future research.

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