

Reply to the Letter to the Editor: Assessing the Predictive Value of Kolmogorov–Arnold Networks for the No–Reflow Phenomenon in ST–Segment Elevation Myocardial Infarction: A Comparative Machine Learning Study

Editöre Mektup Yanıtı: ST Segment Yükselmesi Miyokard Enfarktüsünde No–Reflow Fenomeni için Kolmogorov–Arnold Ağlarının Öngörü Değerinin Değerlendirilmesi: Karşılaştırmalı Makine Öğrenimi Çalışması

To the Editor,

We would like to thank the authors of both Letters to the Editor^{1,2} for their thoughtful and constructive comments on our article, "Assessing the Predictive Value of Kolmogorov–Arnold Networks for the No–Reflow Phenomenon in ST–Segment Elevation Myocardial Infarction: A Comparative Machine Learning Study."³ We appreciate their interest in our work and agree that transparent reporting, calibration assessment, clinical utility analysis, leakage–safe validation, and external validation are essential before any machine learning model can be considered for clinical implementation.

However, we would like to emphasize that our study was designed primarily not to develop a ready–to–use clinical decision support system but to evaluate the comparative discriminative performance, variable contribution, and computational efficiency of the Kolmogorov–Arnold Network (KAN) model in a single–center STEMI cohort. Therefore, the main outcomes of the study were structured around ROC–AUC, F1–score, precision, recall, runtime, and SHAP–based explainability.³ In this context, the study should be interpreted as a methodological and comparative machine learning investigation rather than a definitive clinical deployment study.

We agree with the authors that discrimination alone does not establish clinical readiness. Calibration–in–the–large, calibration slope, Brier score, and calibration plots would provide valuable complementary information regarding the agreement between predicted and observed probabilities.⁴ Similarly, decision–curve analysis would be useful for determining whether the model provides a net clinical benefit across clinically relevant risk thresholds.⁵ Reporting split–specific no–reflow event counts in the training and test datasets would also improve transparency, particularly in the setting of an imbalanced outcome. These analyses are important for future clinical translation; nevertheless, their absence does not invalidate the main findings of the present study, which focused on internal comparative discrimination and model interpretability.

The concern regarding potential overfitting is also important. In our study, the dataset was divided into a stratified 70% training set and a 30% independent test set, and five–fold cross–validation was restricted to the training data for hyperparameter optimization.³ SMOTE was applied only during the training process to reduce the risk of data leakage. Final model performance was obtained from the independent test set, which was not used during model training. We acknowledge, however, that a more detailed pipeline diagram would improve reproducibility and make the sequence of imputation, feature selection, oversampling, hyperparameter tuning, and testing easier to verify. Future studies should report this workflow explicitly and, where possible, use nested cross–validation or external validation to further minimize optimism and strengthen reliability.⁶

LETTER TO THE EDITOR REPLY EDİTÖRE MEKTUP YANITI

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We also agree that the intended time point of model use should be clarified. Because some of the strongest predictors included procedural variables such as stent length and total ischemic time, the current model should not be interpreted as a purely preprocedural decision-support tool. Rather, in its present form, it is more appropriately considered a periprocedural or early postprocedural risk stratification framework. Future models intended for pre-PCI decision support should be developed using only variables available before coronary intervention.

The comment regarding the definition of no-reflow is also well taken. Our study focused on angiographic no-reflow, defined using TIMI flow and Myocardial Blush Grade criteria, because these measures are immediately available in the catheterization laboratory and are widely used in interventional cardiology studies. Cardiac magnetic resonance imaging may provide a more detailed assessment of microvascular obstruction and infarct characteristics; however, it is not routinely available in the acute procedural setting and was beyond the scope of the present analysis. Future studies incorporating both angiographic and CMR-based endpoints would provide a more comprehensive evaluation of no-reflow and microvascular obstruction.^{7,8}

We further agree that SHAP analysis should not be interpreted as a causal inference method. In our study, SHAP was used to improve transparency and evaluate whether model predictions were driven by clinically plausible variables, including ejection fraction, BNP, baseline troponin, total ischemic time, and stent length.^{3,9} Therefore, SHAP-based findings should be considered explanatory and associative rather than causal. Combining machine learning with causal modeling frameworks may be a valuable direction for future research.

In summary, we agree that full calibration reporting, decision-curve analysis, split-specific event reporting, leakage-safe workflow visualization, nested or temporal validation, external multicenter validation, and prospective impact analyses would strengthen the clinical translation of KAN-based no-reflow prediction. At the same time, these additional requirements relate mainly to clinical implementation rather than to the

primary objective of our study. Our findings support the potential value of KAN as a promising research framework for modeling complex nonlinear relationships in STEMI-related no-reflow; however, as stated in our original conclusion, KAN should currently be considered a research tool rather than a ready-to-use clinical decision support system.³

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