

Oliguria, renal function, hemodynamics, and acid-base abnormalities with 0-48-hour urine-fluid trajectories for mortality risk stratification in acute myocardial infarction: a retrospective MIMIC- IV study

Yao Zheng

yaozheng199@outlook.com

Jiaxiang County People's Hospital

Research Article

Keywords: acute myocardial infarction, cardiovascular critical care, fluid balance, in-hospital mortality, machine learning, MIMIC-IV, oliguria, prediction model

Posted Date: October 6th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-10210976/v1>

License: © ⓘ This work is licensed under a Creative Commons Attribution 4.0 International License.

[Read Full License](#)

Additional Declarations: No competing interests reported.

Abstract

Background: Risk stratification for intensive care unit patients with acute myocardial infarction requires early bedside variables that reflect circulatory stress, renal response, treatment intensity, and metabolic injury. This study evaluated an oliguria, renal function, hemodynamics, and acid-base abnormalities phenotype with early urine-output and fluid-balance trajectories for in-hospital mortality risk stratification.

Methods: This retrospective cohort study analyzed 2097 adult acute myocardial infarction-coded intensive care unit stays from MIMIC-IV. Vital signs and laboratory predictors were defined from 6 hours before to 24 hours after intensive care unit admission. Input-output variables were summarized across 0-6, 6-12, 12-24, and 24-48 hours after admission. The primary outcome was in-hospital mortality. LightGBM models were evaluated with stratified 5-fold internal cross-validation. Robustness analyses included strict 48-hour landmarking, treatment-trajectory adjustment, net fluid-balance threshold testing, continuous renal replacement therapy exclusion, no-furosemide subgroup analysis, calibration assessment, and decision-curve analysis.

Results: The cohort included 2097 intensive care unit stays and 285 in-hospital deaths (13.6%). Persistent/intermittent oliguria had the highest mortality among urine-output trajectory classes. The static phenotype model achieved an area under the receiver operating characteristic curve of 0.877, area under the precision-recall curve of 0.531, and Brier score of 0.094. Adding urine-fluid trajectories improved performance to 0.885, 0.552, and 0.089, respectively. Treatment-adjusted associations remained significant for persistent oliguria, oliguria burden, persistent positive fluid balance, and the high-risk urine-fluid phenotype.

Conclusions: The oliguria, renal function, hemodynamics, and acid-base abnormalities phenotype combined with 0-48-hour urine-output and fluid-balance trajectories provided internally validated, interpretable mortality risk stratification in acute myocardial infarction-coded intensive care unit patients. External validation is required before clinical implementation.

Background

AMI patients admitted to the ICU require rapid assessment of organ response during the first days of care. Contemporary ACS and coronary revascularization guidance emphasize early risk assessment for treatment planning, and ICU patients with AMI often have high circulatory and metabolic burden, especially when cardiogenic shock or advanced organ support is present [1, 10–12].

Urine output and fluid balance are bedside signals that update continuously. Oliguria reflects renal perfusion, venous congestion, kidney reserve, and treatment response. Acute kidney injury and cardiorenal literature links oliguria, creatinine change, and kidney-replacement therapy with adverse short-term outcomes [6–8, 14]. Fluid accumulation captures resuscitation exposure and impaired fluid

removal and should be interpreted within the broader context of critical care fluid management [9, 15–17].

The ORHA phenotype combines four domains: Oliguria, Renal function, Hemodynamics, and Acid-base abnormalities. These domains map to a coherent AMI ICU pathway: impaired circulation, reduced renal output, renal dysfunction, and metabolic injury. We added 0–48 h urine output and fluid balance trajectories to this day-1 phenotype and tested their incremental value for in-hospital mortality risk stratification.

Methods

Study design and reporting

This study was designed as a retrospective observational prognostic modeling and association analysis using deidentified intensive care data. Reporting was aligned with current recommendations for observational studies and clinical prediction models, including transparent specification of the cohort, predictor windows, outcome definition, validation approach, calibration assessment, and sensitivity analyses.

Oliguria, renal function, hemodynamics, and acid-base abnormalities phenotype

The oliguria, renal function, hemodynamics, and acid-base abnormalities (ORHA) phenotype was defined as a domain-based early intensive care unit feature framework. The oliguria domain included day-1 urine output normalized to body weight and binary oliguria using the < 0.5 mL/kg/h threshold. The renal-function domain included creatinine, blood urea nitrogen, renal comorbidity, acute kidney injury-related variables when available, and renal replacement therapy context. The hemodynamic domain included heart rate, systolic and diastolic blood pressure, mean arterial pressure, and vasopressor exposure. The acid-base domain included lactate, pH, bicarbonate, base excess, anion gap, and oxygenation-related measures. ORHA was not assigned a manual point score; instead, it served as a clinically interpretable predictor-domain structure for model development and selected association analyses relevant to cardiovascular critical care.

Data source and acute myocardial infarction cohort definition

Use of generative language tools

A large language model was used during manuscript preparation only for language editing, journal-format adaptation, and preparation of submission checklists. The tool was not used for cohort

extraction, statistical analysis, model development, or interpretation of the clinical results. The author checked and approved the final manuscript and takes full responsibility for its content.

This retrospective cohort study used MIMIC-IV, a deidentified electronic health record database from Beth Israel Deaconess Medical Center [2, 3]. Adult intensive care unit stays with acute myocardial infarction were identified from diagnosis records in the platform export. The stay_id was used as the analysis unit, and the first qualifying intensive care unit stay per hospitalization was retained. Patients without a valid intensive care unit stay identifier or outcome status were not eligible for analysis.

The available platform export did not provide a complete, auditable table of acute myocardial infarction subtype, including STEMI/NSTEMI status, type 1/type 2 myocardial infarction status, culprit-vessel information, revascularization timing, or mechanical circulatory support timing for all stays. Therefore, the analysis is framed as a prognostic study of acute myocardial infarction-coded intensive care unit stays rather than a subtype-specific acute coronary syndrome study. These missing clinical details were treated as key limitations and external-validation priorities.

Predictor windows

Vital signs and laboratory predictors used the ICU day-1 window from 6 h before ICU admission to 24 h after ICU admission. Input-output variables used four post-ICU-admission windows: 0–6 h, 6–12 h, 12–24 h, and 24–48 h. This window structure keeps the physiologic phenotype within a standard early ICU assessment period and evaluates dynamic bedside trajectories after ICU admission.

Phenotype and trajectory variables

After defining ORHA as a four-domain feature framework, trajectory variables were constructed to capture dynamic urine output and fluid balance. Implausible physiologic values were cleaned before modeling. Window-specific urine output was calculated as urine volume divided by cleaned body weight and window duration. Oliguria was defined as urine output < 0.5 mL/kg/h, consistent with KDIGO urine-output criteria [8]. Net fluid balance was calculated as total input minus total output. Persistent oliguria was defined as oliguria in at least two of four trajectory windows. A high-risk urine-fluid phenotype combined persistent oliguria, persistent positive fluid balance, or cumulative net fluid balance > 50 mL/kg.

Because CRRT and diuretics can alter urine output and fluid balance interpretation, treatment-trajectory variables were incorporated and additional CRRT-exclusion and no-furosemide analyses were performed.

Outcomes

The primary outcome was in-hospital mortality. Secondary outcomes included ICU mortality and post-discharge mortality endpoints available in the source data. The outcome represents all-cause mortality;

cause-specific cardiovascular death was not available.

Missing data and preprocessing

Missing values were handled within each cross-validation training fold through median imputation for continuous predictors and most-frequent imputation for categorical predictors. Missingness indicators were added for continuous variables during model preprocessing. No information from validation folds was used to estimate imputation values. Missing trajectory categories were retained descriptively because missingness may reflect early death, transfer, recording intensity, or organ-support complexity.

Sensitivity analyses

The strict 48 h landmark cohort included patients who were alive and hospitalized at 48 h after ICU admission. This analysis evaluated mortality after the trajectory assessment window. Treatment-trajectory adjustment included vasopressor exposure, furosemide use, CRRT, and invasive ventilation across the four 0–48 h windows. Additional analyses tested cumulative net fluid balance thresholds of 30, 40, 50, and 60 mL/kg, excluded patients exposed to CRRT during 0–48 h, and repeated key associations among patients without 0–48 h furosemide exposure.

Modeling and reporting

LightGBM models were evaluated with stratified 5-fold internal cross-validation [21]. The main hyperparameters were `n_estimators = 120`, `learning_rate = 0.04`, `num_leaves = 31`, `min_child_samples = 20`, `subsample = 0.85`, `colsample_bytree = 0.85`, `objective=binary`, `class_weight=balanced`, and `random_state = 42`. Performance metrics included the area under the receiver operating characteristic curve, area under the precision-recall curve, and Brier score. The area under the precision-recall curve was prespecified as important because in-hospital death occurred in 13.6% of the cohort. Bootstrap resampling was used to estimate uncertainty for model performance. Calibration was assessed with decile plots, calibration intercept, calibration slope, observed/expected ratio, and Brier score. Decision-curve analysis evaluated net benefit across clinically relevant risk thresholds.

Results

The cohort included 2097 ICU stays. In-hospital death occurred in 285 patients (13.6%). Baseline clinical characteristics are summarized by outcome in Supplementary Table S14.

Table 1
Cohort overview

Metric	Value
Total ICU stays	2097.00
In-hospital deaths	285.00
In-hospital mortality, %	13.60
ICU deaths	211.00
28-day deaths	317.00
90-day deaths	422.00
365-day deaths	563.00
Median urine output, mL/kg/h	0.895
Oliguria prevalence, %	20.70
Median net fluid balance, mL	1277.70
Positive fluid balance prevalence, %	69.00

Persistent/intermittent oliguria identified the highest-risk urine-output trajectory class. This group had lower median 0–48 h urine output and higher median net fluid balance than patients with adequate urine output.

Table 2
Outcomes by 0–48 h urine output trajectory class

Trajectory class	N	Deaths	Mortality, %	Median urine 0-48h, mL/kg/h	Median net balance 0-48h, mL/kg
Persistent/intermittent oliguria	264	82	31.10	0.333	54.51
Missing	49	14	28.60	NA	38.91
Oliguria recovery	247	34	13.80	0.851	35.45
Late/worsening oliguria	551	73	13.20	0.620	23.91
Single-window oliguria	200	17	8.500	0.882	14.41
Adequate urine output	786	65	8.300	1.202	7.610

Urine-fluid trajectories improved prediction beyond the static ORHA phenotype. The same direction was observed in the strict 48 h landmark cohort. Bootstrap intervals, fold-level results, calibration metrics, and decision-curve net benefit are provided in the supplement.

Table 3
Main and landmark model performance

Cohort	Model	AUROC	AUPRC	Brier score
Full cohort	Static ORHA	0.877	0.531	0.094
Full cohort	Static ORHA + urine-fluid trajectories	0.885	0.552	0.089
Full cohort	Static ORHA + trajectories + scores	0.898	0.580	0.083
Strict 48 h landmark	Static ORHA	0.864	0.465	0.088
Strict 48 h landmark	Static ORHA + urine-fluid trajectories	0.874	0.486	0.086
Strict 48 h landmark	Static ORHA + trajectories + scores	0.892	0.532	0.078

Treatment-trajectory adjustment reduced effect estimates and retained statistically significant associations for the main trajectory exposures.

Table 4
Treatment-trajectory adjusted associations

Cohort	Exposure	Adjusted OR (95% CI)	P value
Full cohort	Persistent oliguria in ≥ 2 windows	2.899 (2.167–3.878)	< 0.001
Full cohort	Each additional oliguria window	1.463 (1.314–1.63)	< 0.001
Full cohort	Positive balance in ≥ 2 windows	2.174 (1.509–3.133)	< 0.001
Full cohort	Net fluid balance per 10 mL/kg	1.053 (1.029–1.078)	< 0.001
Full cohort	High-risk urine/fluid trajectory phenotype	1.922 (1.336–2.767)	< 0.001
Strict 48 h landmark	Persistent oliguria in ≥ 2 windows	2.464 (1.793–3.387)	< 0.001
Strict 48 h landmark	Each additional oliguria window	1.376 (1.223–1.548)	< 0.001
Strict 48 h landmark	Positive balance in ≥ 2 windows	2.348 (1.563–3.529)	< 0.001
Strict 48 h landmark	Net fluid balance per 10 mL/kg	1.054 (1.028–1.08)	< 0.001
Strict 48 h landmark	High-risk urine/fluid trajectory phenotype	2.016 (1.349–3.014)	< 0.001

Discussion

This study identified a clinically interpretable early cardiovascular critical care pattern in acute myocardial infarction-coded intensive care unit stays: persistent or recurrent oliguria across the first 48 hours was associated with higher in-hospital mortality. The ORHA framework organized this signal with renal function, hemodynamics, and acid-base injury, while fluid-balance trajectories provided additional information about resuscitation exposure, impaired fluid removal, and overall treatment intensity.

The trajectory findings were consistent across descriptive, association, and prediction analyses. Patients with persistent/intermittent oliguria had the highest mortality and the greatest cumulative fluid burden. Adding urine-fluid trajectories to the static phenotype produced modest but consistent improvements in discrimination and overall prediction error. Because the absolute performance gain was small, the main value of the trajectory extension should be interpreted as enhanced cardiovascular bedside interpretability and incremental prognostic information rather than as evidence of a stand-alone deployable clinical tool.

The landmark analysis improves temporal credibility. Patients who died during the first 48 h were removed from the strict landmark cohort. Persistent/intermittent oliguria remained the highest-risk trajectory class, and trajectory variables retained incremental model value. The full cohort results should be interpreted as overall early-trajectory associations, while the landmark results provide the time-consistent prognostic analysis.

Treatment-trajectory adjustment addresses confounding by clinical intensity. Vasopressors, diuretics, CRRT, and ventilation influence urine output and fluid balance. The main associations remained significant after these adjustments, and additional CRRT-exclusion and no-furosemide analyses supported the direction of the findings. The attenuated effect sizes indicate that treatment burden explains part of the risk signal, while the urine-fluid trajectory retains prognostic information within the observed treatment context.

Positive fluid balance should be interpreted as a prognostic marker, not as proof of causal harm [9, 15–17]. In AMI ICU care, positive balance may reflect shock resuscitation, low perfusion pressure, renal dysfunction, delayed fluid removal, mechanical ventilation, CRRT timing, and documentation patterns. The study evaluates risk stratification, not fluid-treatment effects.

Clinical use should be considered only after external validation and workflow testing. At this stage, the ORHA trajectory framework is best viewed as a prognostic research model that may help clinicians recognize persistent low-output and fluid-accumulation patterns requiring reassessment of perfusion, congestion, renal reserve, and organ-support needs. It should not be used as an automated decision-support system or treatment trigger without additional validation.

This study has limitations. MIMIC-IV comes from a single health system, and external validation is required before clinical use. Residual confounding may remain, especially for fluids, vasopressors, diuretics, continuous renal replacement therapy, and mechanical ventilation. The study used diagnosis-record based acute myocardial infarction identification from the platform export. Reliable STEMI/NSTEMI classification, type 2 myocardial infarction status, revascularization timing, mechanical circulatory support timing, and cause-specific cardiovascular death were not available. Vital signs and laboratory predictors were defined in the intensive care unit day-1 window, whereas the dynamic analysis focused on input-output and treatment variables. Future studies should validate the framework in independent cardiovascular cohorts, assess recalibration, and evaluate whether the trajectories improve clinical workflow beyond established severity scores.

Conclusions

The ORHA phenotype combined with 0-48-hour urine-output and fluid-balance trajectories provided internally validated, interpretable mortality risk stratification in acute myocardial infarction-coded intensive care unit patients. Persistent/intermittent oliguria and sustained fluid-balance burden remained informative after landmark and treatment-focused sensitivity analyses. External validation is required before clinical implementation.

Abbreviations

ACS

acute coronary syndrome

AMI

acute myocardial infarction

AUPRC

area under the precision-recall curve

AUROC

area under the receiver operating characteristic curve

BUN

blood urea nitrogen

CRRT

continuous renal replacement therapy

ICU

intensive care unit

MIMIC-IV

Medical Information Mart for Intensive Care IV

NSTEMI

non-ST-elevation myocardial infarction

ORHA

oliguria,renal function,hemodynamics,and acid-base abnormalities

PCI

percutaneous coronary intervention

SAPS II

Simplified Acute Physiology Score II

SOFA

Sequential Organ Failure Assessment

STEMI

ST-elevation myocardial infarction.

Declarations

Ethics approval and consent to participate

This study used deidentified MIMIC-IV data. MIMIC-IV was approved for research use by the Institutional Review Boards of the Massachusetts Institute of Technology and Beth Israel Deaconess Medical Center; the requirement for individual informed consent was waived because the database contains deidentified health records. Access to MIMIC-IV requires completion of the required data-use training and credentialing process. The current secondary analysis used only deidentified data and complied with the PhysioNet data-use agreement.

Clinical trial number

Not applicable. This study was a retrospective analysis based on the MIMIC-IV database and did not involve prospective patient enrollment or clinical intervention.

Consent for publication

Not applicable. The manuscript contains no individual person's identifiable data, images, or videos.

Availability of data and materials

The MIMIC-IV dataset analyzed during the current study is available to credentialed users through PhysioNet (MIMIC-IV version 3.1; <https://doi.org/10.13026/kpb9-mt58>). The minimal summary results supporting the conclusions are included in this article and its Additional file 1. The derived analytic tables, variable definitions, and analysis code can be made available by the corresponding author on reasonable request and subject to the MIMIC-IV data-use agreement.

Competing interests

The author declares that he has no competing interests.

Funding

No specific funding was received for this study.

Authors' contributions

YZ conceived the study, performed data curation and analysis, interpreted the results, drafted the manuscript, revised the manuscript critically for important intellectual content, and approved the final version for submission.

Acknowledgements

The author thanks the developers and maintainers of the MIMIC-IV database and PhysioNet. A large language model was used only for language editing, formatting adaptation, and preparation of

submission checklists; it was not used to generate or analyze study data. The author reviewed and approved all final content and takes full responsibility for the manuscript.

Authors' information

Not applicable.

Generative AI and large language model disclosure: A large language model was used only for language editing, formatting adaptation, and checklist preparation. It was not used to generate or analyze the study data. The author reviewed and approved all manuscript content and remains fully accountable for the work.

References

1. Byrne RA, Rossello X, Coughlan JJ, Barbato E, Berry C, Chieffo A, et al. 2023 ESC Guidelines for the management of acute coronary syndromes. *Eur Heart J*. 2023;44:3720–826. 10.1093/eurheartj/ehad191.
2. Johnson AEW, Bulgarelli L, Shen L, Gayles A, Shammout A, Horng S, Pollard TJ, et al. MIMIC-IV, a freely accessible electronic health record dataset. *Sci Data*. 2023;10:1. 10.1038/s41597-022-01899-x.
3. Johnson A, Bulgarelli L, Pollard T, Gow B, Moody B, Horng S, Celi LA, Mark R. MIMIC-IV (version 3.1). PhysioNet. 2024. <https://doi.org/10.13026/kpb9-mt58>
4. Collins GS, Moons KGM, Dhiman P, Riley RD, Beam AL, Van Calster B, et al. TRIPOD + AI statement: updated guidance for reporting clinical prediction models that use regression or machine learning methods. *BMJ*. 2024;385:e078378.
5. Vasey B, Nagendran M, Campbell B, Clifton DA, Collins GS, Denaxas S, et al. Reporting guideline for the early-stage clinical evaluation of decision support systems driven by artificial intelligence: DECIDE-AI. *BMJ*. 2022;377:e070904. 10.1136/bmj-2022-070904.
6. Kellum JA, Romagnani P, Ashuntantang G, Ronco C, Zarbock A, Anders HJ. Acute kidney injury. *Nat Reviews Disease Primers*. 2021;7:52. 10.1038/s41572-021-00284-z.
7. Ostermann M, Zarbock A, Goldstein S, Kashani K, Macedo E, Murugan R et al. Recommendations on acute kidney injury biomarkers from the Acute Disease Quality Initiative consensus conference. *JAMA Network Open*. 2020;3:e2019209. 10.1001/jamanetworkopen.2020.19209
8. Kidney Disease: Improving Global Outcomes Acute Kidney Injury Work Group. KDIGO Clinical Practice Guideline for Acute Kidney Injury. *Kidney Int Supplements*. 2012;2:1–138.
9. Evans L, Rhodes A, Alhazzani W, Antonelli M, Coopersmith CM, French C, et al. Surviving Sepsis Campaign: international guidelines for management of sepsis and septic shock 2021. *Intensive Care Med*. 2021;47:1181–247. 10.1007/s00134-021-06506-y.
10. Samsky MD, Morrow DA, Proudfoot AG, Hochman JS, Thiele H. Cardiogenic shock after acute myocardial infarction: a review. *JAMA*. 2021;326:1840–50. 10.1001/jama.2021.18323.

11. Thiele H, Zeymer U, Akin I, Behnes M, Rassaf T, Mahabadi AA, et al. Extracorporeal life support in infarct-related cardiogenic shock. *N Engl J Med*. 2023;389:1286–97. 10.1056/NEJMoa2307227.
12. Lawton JS, Tamis-Holland JE, Bangalore S, Bates ER, Beckie TM, Bischoff JM, et al. 2021 ACC/AHA/SCAI guideline for coronary artery revascularization. *J Am Coll Cardiol*. 2022;79:e21–129. 10.1016/j.jacc.2021.09.006.
13. Goldberger AL, Amaral LAN, Glass L, Hausdorff JM, Ivanov PC, Mark RG, et al. PhysioBank, PhysioToolkit, and PhysioNet: components of a new research resource for complex physiologic signals. *Circulation*. 2000;101:e215–20. 10.1161/01.CIR.101.23.e215.
14. Ronco C, Bellasi A, Di Lullo L. Cardiorenal syndrome: an overview. *Adv Chronic Kidney Dis*. 2018;25:382–90. 10.1053/j.ackd.2018.08.004.
15. Malbrain MLNG, Van Regenmortel N, Saugel B, De Tavernier B, Van Gaal PJ, Joannes-Boyau O, et al. Principles of fluid management and stewardship in septic shock: it is time to consider the four D's and the four phases of fluid therapy. *Ann Intensiv Care*. 2018;8:66. 10.1186/s13613-018-0402-x.
16. Zampieri FG, Machado FR, Biondi RS, Freitas FGR, Veiga VC, Figueiredo RC, et al. Effect of slower versus faster intravenous fluid bolus rates on mortality in critically ill patients: the BaSICS randomized clinical trial. *JAMA*. 2021;326:830–8. 10.1001/jama.2021.11444.
17. Semler MW, Self WH, Wanderer JP, Ehrenfeld JM, Wang L, Byrne DW, et al. Balanced crystalloids versus saline in critically ill adults. *N Engl J Med*. 2018;378:829–39. 10.1056/NEJMoa1711584.
18. Van Calster B, McLernon DJ, van Smeden M, Wynants L, Steyerberg EW. Calibration: the Achilles heel of predictive analytics. *BMC Med*. 2019;17:230. 10.1186/s12916-019-1466-7.
19. Vickers AJ, van Calster B, Steyerberg EW. A simple, step-by-step guide to interpreting decision curve analysis. *Diagn Prognostic Res*. 2019;3:18. 10.1186/s41512-019-0064-7.
20. Wolff RF, Moons KGM, Riley RD, Whiting PF, Westwood M, Collins GS, et al. PROBAST: a tool to assess the risk of bias and applicability of prediction model studies. *Ann Intern Med*. 2019;170:51–8. 10.7326/M18-1376.
21. Ke G, Meng Q, Finley T, Wang T, Chen W, Ma W, et al. LightGBM: a highly efficient gradient boosting decision tree. *Adv Neural Inf Process Syst*. 2017;30:3146–54.
22. Hyland SL, Faltys M, Hüser M, Lyu X, Gumbsch T, Esteban C, et al. Early prediction of circulatory failure in the intensive care unit using machine learning. *Nat Med*. 2020;26:364–73. 10.1038/s41591-020-0789-4.
23. Shillan D, Sterne JAC, Champneys A, Gibbison B. Use of machine learning to analyse routinely collected intensive care unit data: a systematic review. *Crit Care*. 2019;23:284. 10.1186/s13054-019-2564-9.

Figures

ICU day-1 ORHA phenotype linked with dynamic urine-fluid trajectories

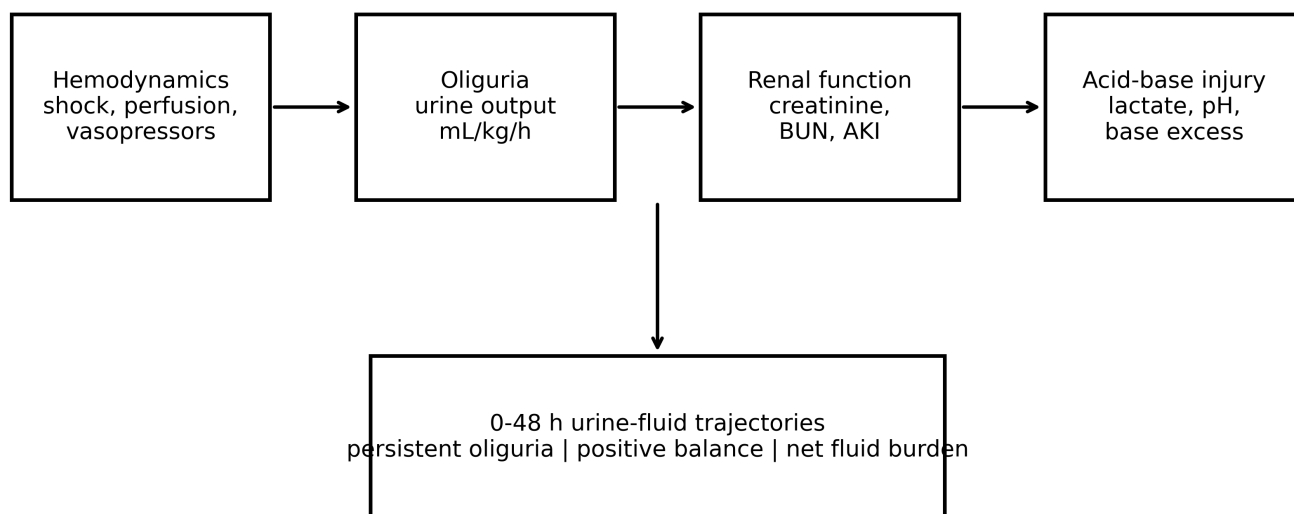


Figure 1

ORHA conceptual framework and dynamic urine-fluid trajectory domain.

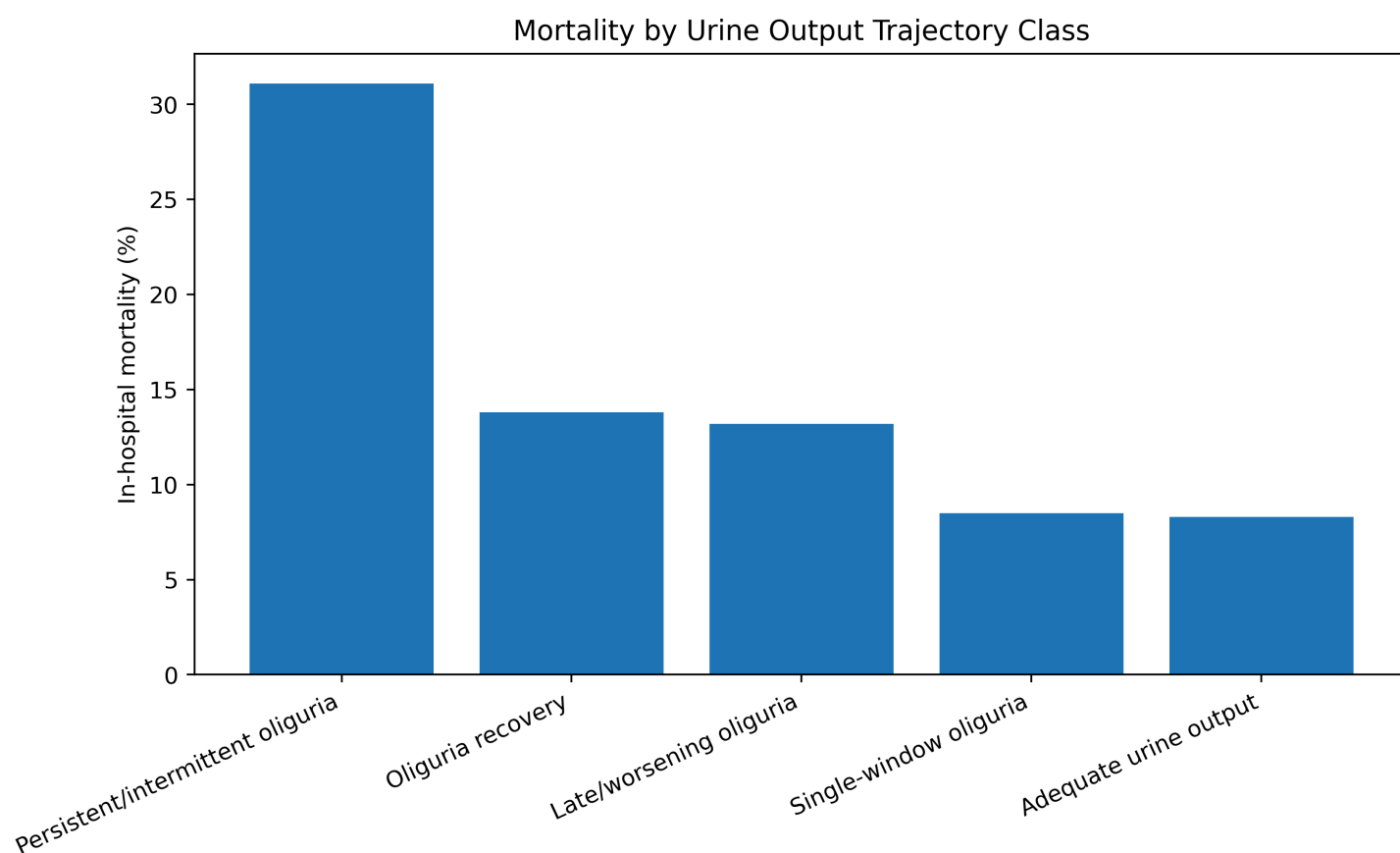


Figure 2

In-hospital mortality by 0-48 h urine output trajectory class.

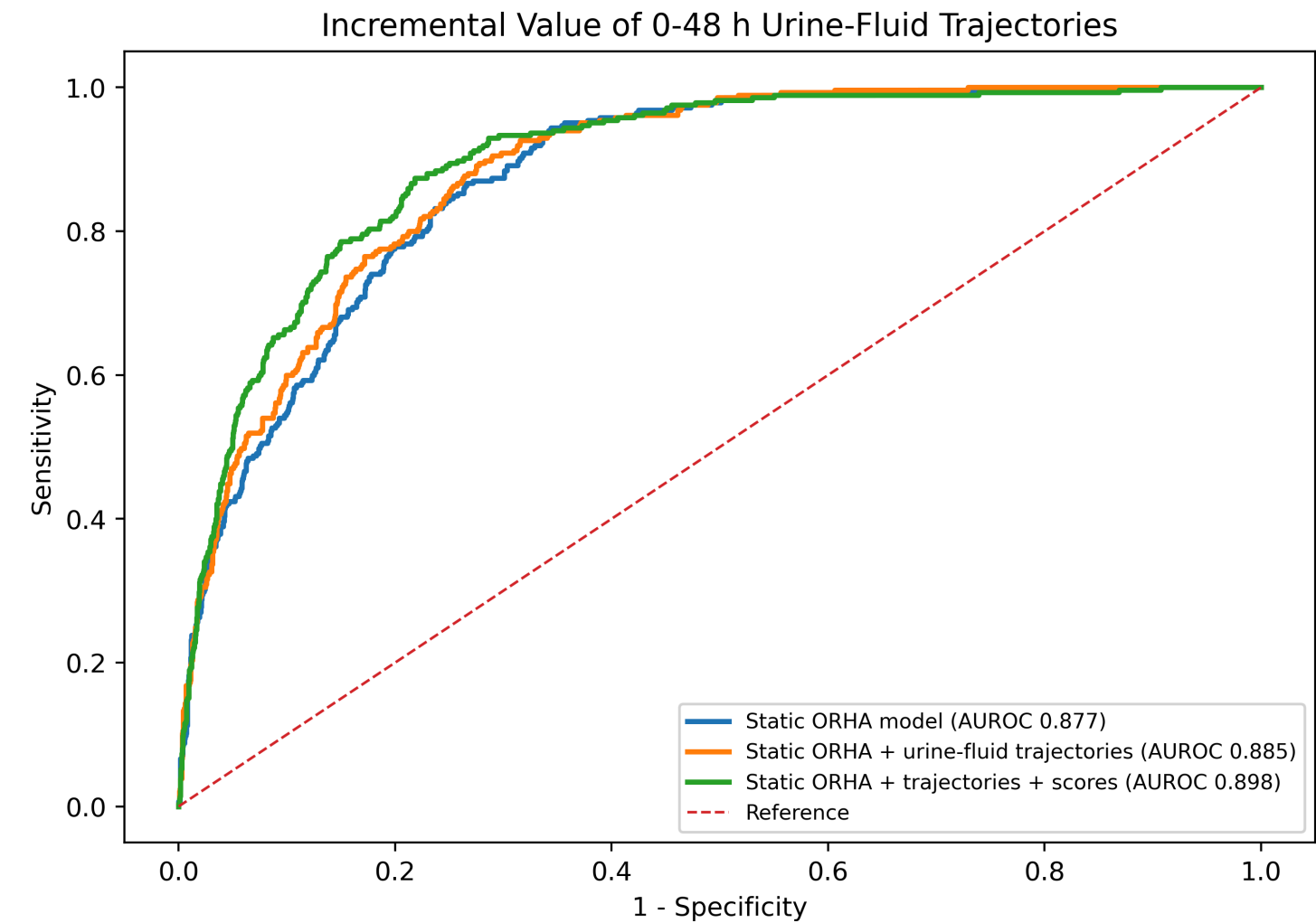


Figure 3

Incremental ROC comparison after adding urine-fluid trajectory variables.

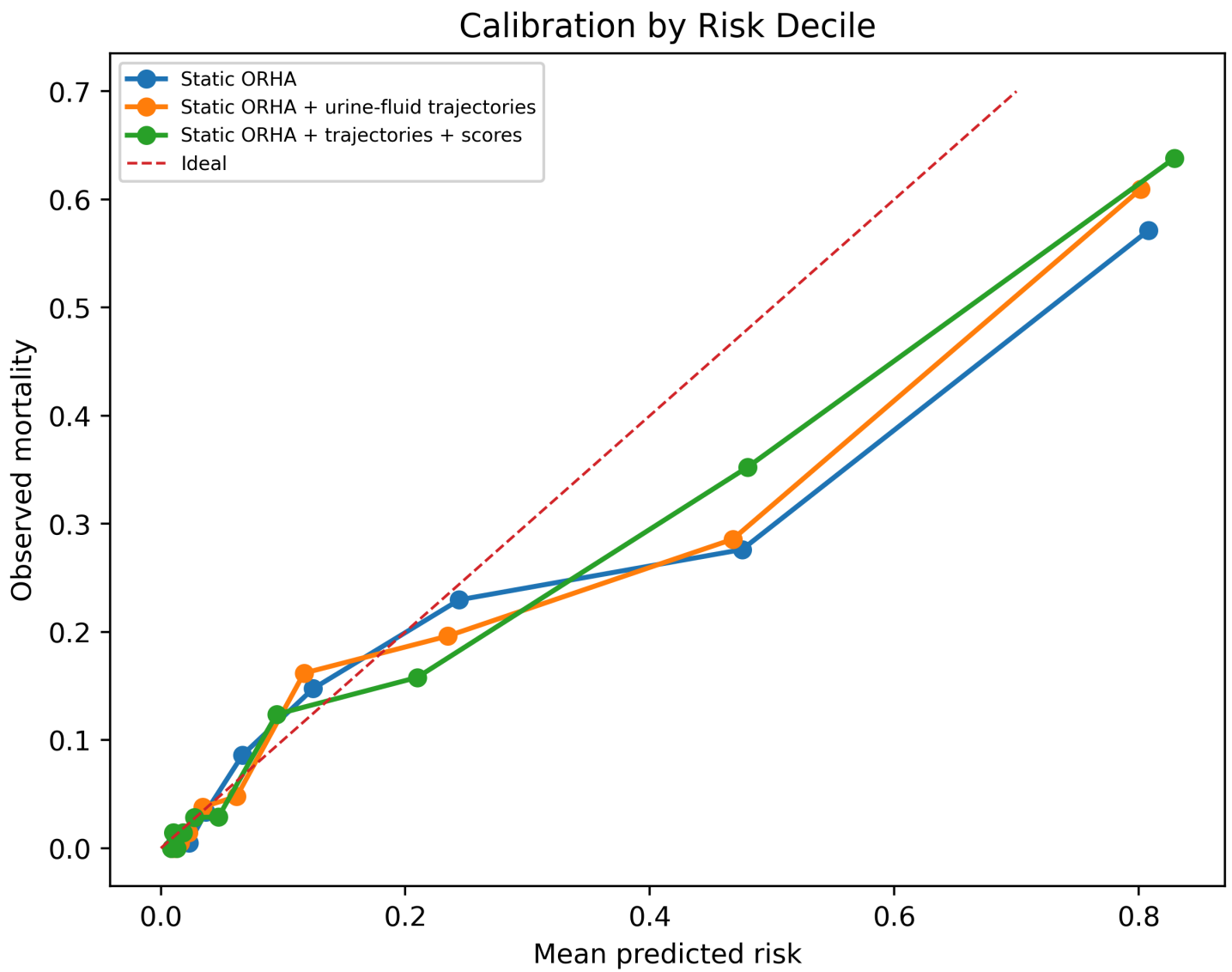


Figure 4

Calibration by risk decile in the full cohort.

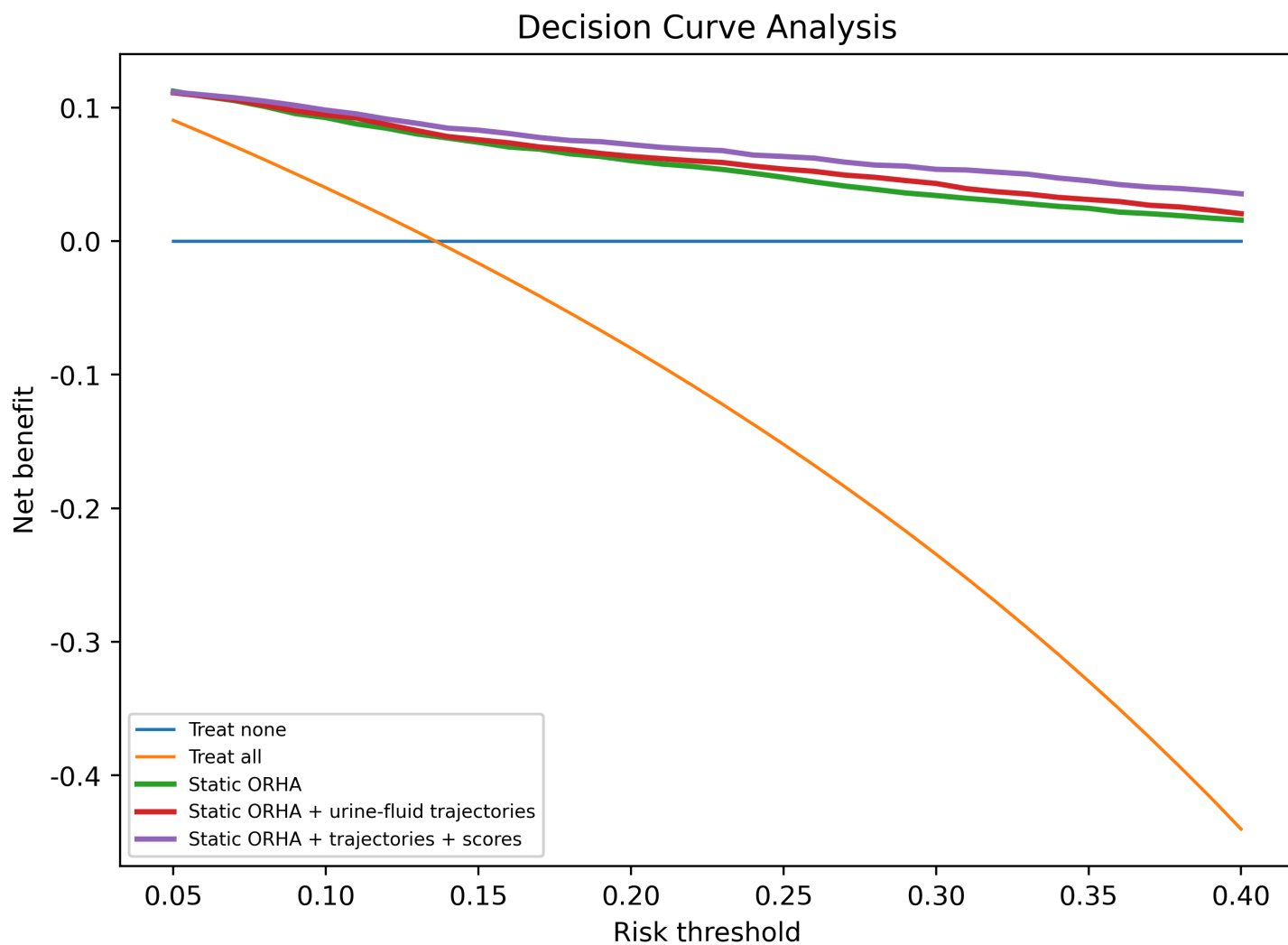


Figure 5

Decision-curve analysis in the full cohort.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [Additionalfile1ORHABMCCVDFinal.docx](#)