

Early bicarbonate normalization and discharge alive from the ICU in adults with diabetic ketoacidosis and high-anion-gap acidosis: a retrospective MIMIC-IV landmark cohort study

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Research Article

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Abstract

Background: Published DKA risk studies have largely relied on admission biomarkers, organ-failure scores, or multivariable prediction models. These approaches describe baseline severity but do not show whether metabolic acidosis is improving after treatment begins. We examined whether bicarbonate normalization during hours 6–12 after intensive care unit (ICU) admission was associated with discharge alive from the ICU by 72 hours.

Methods: This exploratory retrospective landmark study used MIMIC-IV. Adults with DKA were identified using prespecified ICD-9-CM and ICD-10-CM diagnosis codes. The primary cohort required baseline bicarbonate <18 mmol/L, anion gap >12 mmol/L, ICU stay of at least 12 hours, and a bicarbonate measurement during hours 6–12. The exposure was bicarbonate ≥ 18 mmol/L during hours 6–12. The primary outcome was failure to achieve discharge alive from the ICU by 72 hours, defined as ICU stay ≥ 72 hours or ICU death. Multivariable logistic regression adjusted for baseline clinical and metabolic variables. Selection associated with repeat measurement was examined with inverse-probability-of-observation weighting.

Results: Among 1,243 ICU stays involving adults with DKA, 630 met the active high-anion-gap acidosis phenotype and 569 entered the landmark cohort. Bicarbonate normalized in 261 patients (45.9%). The primary outcome occurred in 52 of 261 patients with normalization (19.9%) and 89 of 308 without normalization (28.9%). In the complete-case model ($n=558$; 136 events), normalization was associated with lower adjusted odds of the primary outcome (odds ratio [OR], 0.46; 95% confidence interval [CI], 0.28–0.78; $P=0.004$). The observation-weighted estimate was similar (OR, 0.47; 95% CI, 0.28–0.78). The estimate weakened in the glucose ≥ 200 mg/dL subgroup and when the last rather than the first bicarbonate value in the initial window was used for adjustment.

Conclusions: Bicarbonate normalization during hours 6–12 was associated with a greater likelihood of discharge alive from the ICU by 72 hours. The result was sensitive to the baseline time definition and should be considered hypothesis-generating. Exact laboratory timestamps, detailed treatment data, and external validation are required before clinical use.

Background

Diabetic ketoacidosis (DKA) remains a common metabolic emergency and a substantial source of emergency, hospital, and ICU resource use [1, 3, 4]. Contemporary consensus and practice guidance assess resolution using ketone suppression together with recovery of pH or serum bicarbonate [1, 2]. Yet patients admitted to the ICU differ substantially in pretreatment exposure, precipitating illness, renal function, and response to therapy. An admission value can describe severity at one point in time, but it cannot show whether acidosis is resolving.

Recent studies have approached DKA prognosis through prediction models for prolonged ICU stay and mortality [5, 6], analyses of acid-base status and organ-failure scores [7, 8], and single laboratory

markers or composite ratios such as the blood urea nitrogen-to-albumin ratio, lactate-to-albumin ratio, and serum osmolality [9–12]. A separate validated cohort also showed that acidosis-related variables were important in identifying adults requiring ICU admission [13]. These studies provide useful risk stratification, but their predictors are usually drawn from admission or first-day data. A separate clinical question remains unanswered: does a routinely repeated acid-base measurement after treatment begin to identify patients with a different subsequent ICU course?

We addressed this question with a 12-hour landmark design. The analysis was restricted to adults with DKA and active high-anion-gap acidosis at baseline. The exposure was a simple follow-up state—bicarbonate ≥ 18 mmol/L during hours 6–12—rather than a data-derived biomarker ratio or a multivariable prediction score. The landmark separated exposure assessment from the subsequent ICU outcome and made the target population explicit.

We examined whether early bicarbonate normalization was associated with discharge alive from the ICU by 72 hours after accounting for initial acid-base severity, renal function, comorbidity, and vital signs. The analysis was exploratory and not preregistered. The objective was to estimate a clinically interpretable association, test its robustness to alternative definitions, and define the boundary of the evidence.

Methods

Data source and study design

This retrospective landmark cohort study used the Medical Information Mart for Intensive Care IV (MIMIC-IV), a deidentified electronic health record database from Beth Israel Deaconess Medical Center, a tertiary academic hospital in Boston, Massachusetts, USA [14]. Data were extracted using a structured online query platform. All study windows were anchored to ICU admission. Reporting followed STROBE and RECORD; completed checklists are provided in Additional file 2 [15, 16].

Study population

Adults with DKA were identified using prespecified ICD-9-CM and ICD-10-CM diagnosis codes. For patients with more than one eligible hospitalization or ICU stay, only the first hospitalization and first ICU stay were retained. Encounters with a diagnosis code for hyperosmolar hyperglycemic state were excluded.

The primary phenotype required the first bicarbonate value in the – 6 to + 6-hour baseline window to be < 18 mmol/L and the first anion gap to be > 12 mmol/L. This operational definition identifies active high-anion-gap metabolic acidosis but does not replace direct biochemical confirmation with beta-hydroxybutyrate.

The 12-hour landmark cohort required an ICU stay of at least 12 hours and a bicarbonate measurement during hours 6–12. The analysis therefore applies to patients who survived, remained in the ICU, and underwent repeat laboratory testing through the exposure window.

Exposure

The primary exposure was bicarbonate normalization, defined as the last serum bicarbonate value recorded during hours 6–12 being ≥ 18 mmol/L. A secondary analysis modeled the 6–12-hour bicarbonate value continuously per 5-mmol/L increment while adjusting for the earlier value. A model containing both a change score and the baseline value is algebraically equivalent to a model containing the later and baseline values; the analysis therefore does not claim that the change score is an independent marker.

Outcome

The primary outcome was failure to achieve discharge alive from the ICU by 72 hours, defined as ICU length of stay ≥ 72 hours or ICU death. Patients discharged alive before 72 hours were classified as having achieved discharge alive from the ICU and therefore did not experience the primary outcome. Sensitivity analyses used 48-hour and 96-hour thresholds and substituted in-hospital death for ICU death.

Covariates

The adjustment set included age, sex, body weight, diagnosis-derived renal disease and heart failure indicators, baseline bicarbonate, anion gap, glucose, creatinine, potassium, mean arterial pressure, heart rate, and respiratory rate. Laboratory and vital-sign variables were the first values in the baseline window. Diagnosis-derived comorbidity indicators were calculated from discharge coding and do not prove that the conditions were present before admission. Post-baseline treatment variables were not included in the primary adjustment set because they could lie on the pathway between early illness severity and the outcome.

Statistical analysis

Continuous variables are reported as median and interquartile range (IQR), and categorical variables as number and percentage. Baseline differences are summarized with absolute standardized mean differences (SMDs); values > 0.10 indicate potentially meaningful imbalance. No a priori power calculation was performed because the study included all eligible records available in the database.

Logistic regression with heteroscedasticity-robust standard errors estimated ORs and 95% CIs. The primary analysis used complete cases because 11 of 569 patients lacked at least one model covariate.

No imputation was performed.

The landmark design can select patients according to ICU duration and repeat testing. We therefore compared included and excluded patients with active high-anion-gap acidosis. A logistic model for landmark inclusion generated stabilized inverse-probability-of-observation weights, which were truncated at the 1st and 99th percentiles and applied in a weighted outcome model.

Sensitivity analyses required baseline glucose ≥ 200 mg/dL; excluded diagnosis-derived renal disease, septic shock, or sodium bicarbonate treatment; used alternative 48-hour and 96-hour outcome thresholds; used in-hospital death in the composite; and replaced the first baseline bicarbonate with the last value in the initial window. These analyses were exploratory, and no multiplicity adjustment was applied. Two-sided $P < 0.05$ was considered statistically significant.

Use of generative artificial intelligence

OpenAI ChatGPT was used during manuscript preparation to assist with language editing. It was not used to generate or alter study data or to make autonomous decisions about cohort definitions, statistical models, or interpretation. The author verified all definitions, analyses, numerical results, citations, and interpretations and takes full responsibility for the manuscript.

Results

Cohort construction and selection

Among 1,243 adult ICU stays involving patients with DKA, 630 met the active high-anion-gap acidosis phenotype. Sixty-one were excluded because the ICU stay was < 12 hours and/or no bicarbonate measurement was available during hours 6–12, leaving 569 patients in the landmark cohort. Eleven additional patients had incomplete adjustment variables; the primary model therefore included 558 patients and 136 events (Fig. 1).

Patients excluded from the landmark cohort were older and had higher frequencies of diagnosis-derived renal disease, sepsis, ICU death, and in-hospital death than included patients (Additional file 1: Table S1). The comparison shows that repeat measurement and continued ICU stay were related to patient characteristics, so an observation-weighted analysis was added rather than assuming that landmark inclusion was random.

Bicarbonate status and baseline profile

Bicarbonate normalized during hours 6–12 in 261 patients (45.9%); 308 patients (54.1%) remained below 18 mmol/L. The normalization group had a different baseline profile, including higher initial bicarbonate and glucose and differences in age, renal disease, and treatment indicators. Several absolute

standardized mean differences exceeded 0.10 (Table 1), indicating that the crude event-rate comparison required adjustment.

Table 1
Baseline characteristics by bicarbonate status at 6–12 hours

Characteristic	HCO ₃ < 18 mmol/L (n = 308)	HCO ₃ ≥ 18 mmol/L (n = 261)	Absolute SMD
Age, years	46.0 [27.0, 61.3]	51.8 [37.3, 64.5]	0.253
Male sex	142 (46.1%)	153 (58.6%)	0.251
Weight, kg	73.6 [62.3, 85.0]	73.6 [63.2, 87.5]	0.011
Diagnosis-derived renal disease	47 (15.3%)	67 (25.7%)	0.258
Diagnosis-derived heart failure	36 (11.7%)	47 (18.0%)	0.178
Sepsis diagnosis	106 (34.4%)	86 (33.0%)	0.031
Septic shock diagnosis	41 (13.3%)	19 (7.3%)	0.198
Baseline bicarbonate, mmol/L	10.0 [7.8, 13.0]	14.0 [12.0, 16.0]	0.858
Baseline anion gap, mmol/L	22.0 [18.0, 27.0]	23.0 [19.0, 28.0]	0.194
Baseline glucose, mg/dL	270.0 [194.5, 357.0]	325.0 [234.0, 508.0]	0.357
Baseline creatinine, mg/dL	1.1 [0.8, 2.0]	1.3 [0.9, 2.0]	0.047
Baseline potassium, mmol/L	4.3 [3.8, 4.7]	4.4 [4.0, 5.0]	0.225
Mean arterial pressure, mmHg	83.0 [74.0, 95.0]	85.5 [74.8, 97.0]	0.140
Heart rate, beats/min	105.0 [92.0, 118.5]	100.0 [90.0, 111.0]	0.255
Respiratory rate, breaths/min	21.0 [17.0, 26.0]	20.0 [16.0, 24.0]	0.210
Sodium bicarbonate treatment	46 (14.9%)	18 (6.9%)	0.258
Vasopressor treatment	56 (18.2%)	27 (10.3%)	0.224
Antibiotic treatment	121 (39.3%)	86 (33.0%)	0.132
<i>Values are median [IQR] or n (%). SMD, standardized mean difference.</i>			

Primary association

Failure to achieve discharge alive from the ICU by 72 hours occurred in 52 of 261 patients with bicarbonate normalization (19.9%) and 89 of 308 without normalization (28.9%), an absolute difference of 9.0 percentage points. The unadjusted OR was 0.61 (95% CI, 0.41–0.91; P = 0.014). After adjustment, normalization remained associated with lower odds of the outcome (OR, 0.46; 95% CI, 0.28–0.78; P = 0.004) (Table 2).

Table 2
Primary and boundary analyses

Analysis	N	Events	OR	95% CI	P value
Unadjusted primary analysis	569	141	0.61	0.41–0.91	0.014
Adjusted primary analysis	558	136	0.46	0.28–0.78	0.004
Continuous HCO ₃ at 6–12 h, per 5 mmol/L	558	136	0.46	0.32–0.68	< 0.001
Observation-weighted analysis	558	136	0.47	0.28–0.78	0.004
Baseline glucose $\geq$ 200 mg/dL	435	108	0.60	0.34–1.05	0.075
Last initial-window HCO ₃ adjustment	558	136	0.70	0.40–1.24	0.217

The continuous analysis led to the same clinical interpretation: each 5-mmol/L higher bicarbonate value during hours 6–12, conditional on the earlier bicarbonate and other covariates, was associated with lower odds of the outcome (OR, 0.46; 95% CI, 0.32–0.68; $P < 0.001$). This result supports the relevance of the follow-up value; it does not establish a separate effect of a change score.

Robustness and boundaries

The observation-weighted estimate was nearly identical to the complete-case estimate (OR, 0.47; 95% CI, 0.28–0.78; $P = 0.004$), suggesting that measured determinants of landmark inclusion did not account for the main association. The direction was also preserved after excluding renal disease, septic shock, or sodium bicarbonate treatment and when the alive-discharge threshold was changed to 48 or 96 hours (Fig. 2; Additional file 1: Table S2).

The evidence was not uniform across all definitions. In patients with baseline glucose $\geq$ 200 mg/dL, the estimate was weaker and crossed the null (OR, 0.60; 95% CI, 0.34–1.05; $P = 0.075$). When the last rather than the first bicarbonate in the initial window was used for adjustment, the association was also attenuated (OR, 0.70; 95% CI, 0.40–1.24; $P = 0.217$). These analyses identify the temporal anchor as the main source of uncertainty.

Discussion

Principal findings

Bicarbonate normalization during hours 6–12 was associated with a higher probability of discharge alive from the ICU by 72 hours. The absolute event-rate difference was 9.0 percentage points, and the adjusted and observation-weighted estimates were similar. The association weakened under a stricter glucose criterion and an alternative baseline time anchor. The finding is therefore clinically interpretable but remains exploratory.

Comparison with previous DKA studies

Across recent DKA studies, strengths have included multicenter sampling, explicit training and validation cohorts, model calibration, nonlinear exposure assessment, and clear presentation of clinically important outcomes [5–13]. Most analyses have focused on mortality, acute kidney injury, ICU admission, or prolonged ICU stay using admission biomarkers, laboratory ratios, serum osmolality, organ-failure scores, or multivariable prediction models. Our study addresses a narrower and complementary question. It evaluates a routinely repeated acid-base measurement and preserves temporal ordering through a landmark design. The aim is not to outperform multivariable prediction models but to estimate whether early follow-up bicarbonate status is associated with the subsequent ICU course after the earlier acid-base profile is known.

Clinical interpretation

Bicarbonate normalization may reflect suppression of ketogenesis, restoration of perfusion, renal clearance of organic anions, adequate insulin delivery, and control of the precipitating illness. It is also affected by fluid composition, renal dysfunction, sodium bicarbonate treatment, and the timing of laboratory measurement. Recent randomized and synthesized evidence has shown that fluid composition can alter chloride and acid-base trajectories during DKA treatment [17, 18]. The follow-up bicarbonate value should therefore be interpreted as a summary of the observed metabolic and treatment trajectory, not as a causal treatment target.

Robustness and boundary of the evidence

The analysis used several complementary checks rather than relying on a single significant model. Observation weighting addressed measured differences in repeat testing; sample restrictions examined renal disease, shock, glucose level, and bicarbonate treatment; and alternative outcome thresholds tested the dependence on the 72-hour cutoff. The nonsignificant alternative-baseline analysis is especially important because it shows that the result depends on how the early measurement is anchored.

Strengths and limitations

Strengths include a clinically focused exposure, explicit temporal separation between exposure and outcome, restriction to active high-anion-gap acidosis, comparison of included and excluded patients, observation weighting, and transparent reporting of nonsignificant boundary analyses. Limitations remain. DKA was identified using diagnosis codes, consistent with several prior MIMIC and eICU studies [5, 6, 8–12], and beta-hydroxybutyrate was unavailable for cohort-wide confirmation. Exact laboratory timestamps, emergency-department fluids, and reliable insulin-infusion data were unavailable. Diagnosis-derived comorbidities may include conditions recorded after admission. The landmark cohort excludes short ICU stays and patients without repeat testing. The outcome is influenced by local ICU practice, and the single-center study lacks external validation.

Implications and next steps

Failure of bicarbonate to normalize by hours 6–12 may justify a structured reassessment of insulin delivery, perfusion, renal function, fluid composition, infection, and alternative causes of acidosis. The data do not support sodium bicarbonate administration as a strategy to improve outcomes and do not define a treatment threshold. A confirmatory study should anchor time zero to biochemical DKA confirmation, use exact laboratory timestamps and longitudinal models, capture insulin and fluid exposure, and validate the result in an independent database.

Conclusions

Among adults admitted to the ICU with DKA and active high-anion-gap acidosis, bicarbonate normalization during hours 6–12 was associated with a greater likelihood of discharge alive from the ICU by 72 hours. The result adds a follow-up treatment-response perspective to a literature dominated by static biomarkers and prediction models. Because the association was sensitive to the baseline time anchor, it should be treated as a hypothesis-generating signal pending validation with exact timestamps, detailed treatment data, and an external cohort.

Abbreviations

CI	confidence interval
DKA	diabetic ketoacidosis
HCO ₃	serum bicarbonate
ICU	intensive care unit
IQR	interquartile range
MIMIC-IV	Medical Information Mart for Intensive Care IV
OR	odds ratio
RECORD	REporting of studies Conducted using Observational Routinely-collected health Data
SMD	standardized mean difference
STROBE	Strengthening the Reporting of Observational Studies in Epidemiology.

Declarations

Ethics approval and consent to participate: The institutional review boards of the Massachusetts Institute of Technology and Beth Israel Deaconess Medical Center approved the creation and research use of MIMIC-IV and waived individual informed consent because the data are deidentified. The author completed the required human-subjects training and accessed the database under the PhysioNet data use agreement.

Consent for publication: Not applicable.

Data availability: The deidentified MIMIC-IV electronic health record database used in this retrospective landmark cohort study is available to qualified researchers through the PhysioNet platform (<https://physionet.org/content/mimiciv/>) [19]. Access requires completion of the required human subjects research training and acceptance of the PhysioNet data use agreement. In accordance with the MIMIC-IV data use agreement, the individual-level data used in this study cannot be shared or redistributed by the author. Variable definitions, aggregate results, and supporting analyses are provided in the main manuscript and Additional file 1. The statistical analysis scripts used in this study are available from the corresponding author upon reasonable written request.

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Authors' contributions: YZ conceived and designed the study, performed the analysis, interpreted the results, drafted and critically revised the manuscript, and approved the final manuscript.

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Figures

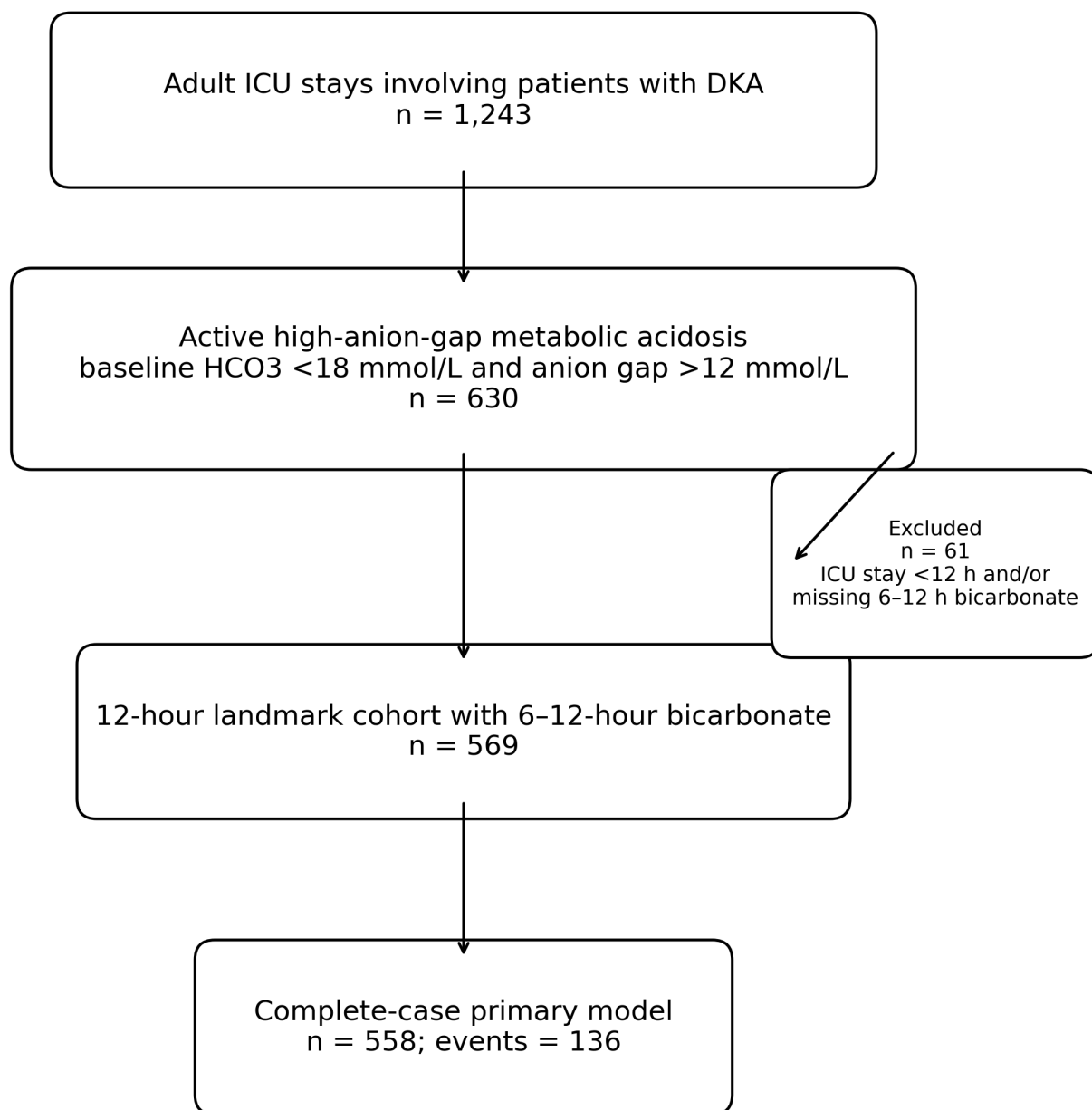


Figure 1

Cohort selection for the active high-anion-gap acidosis landmark study.

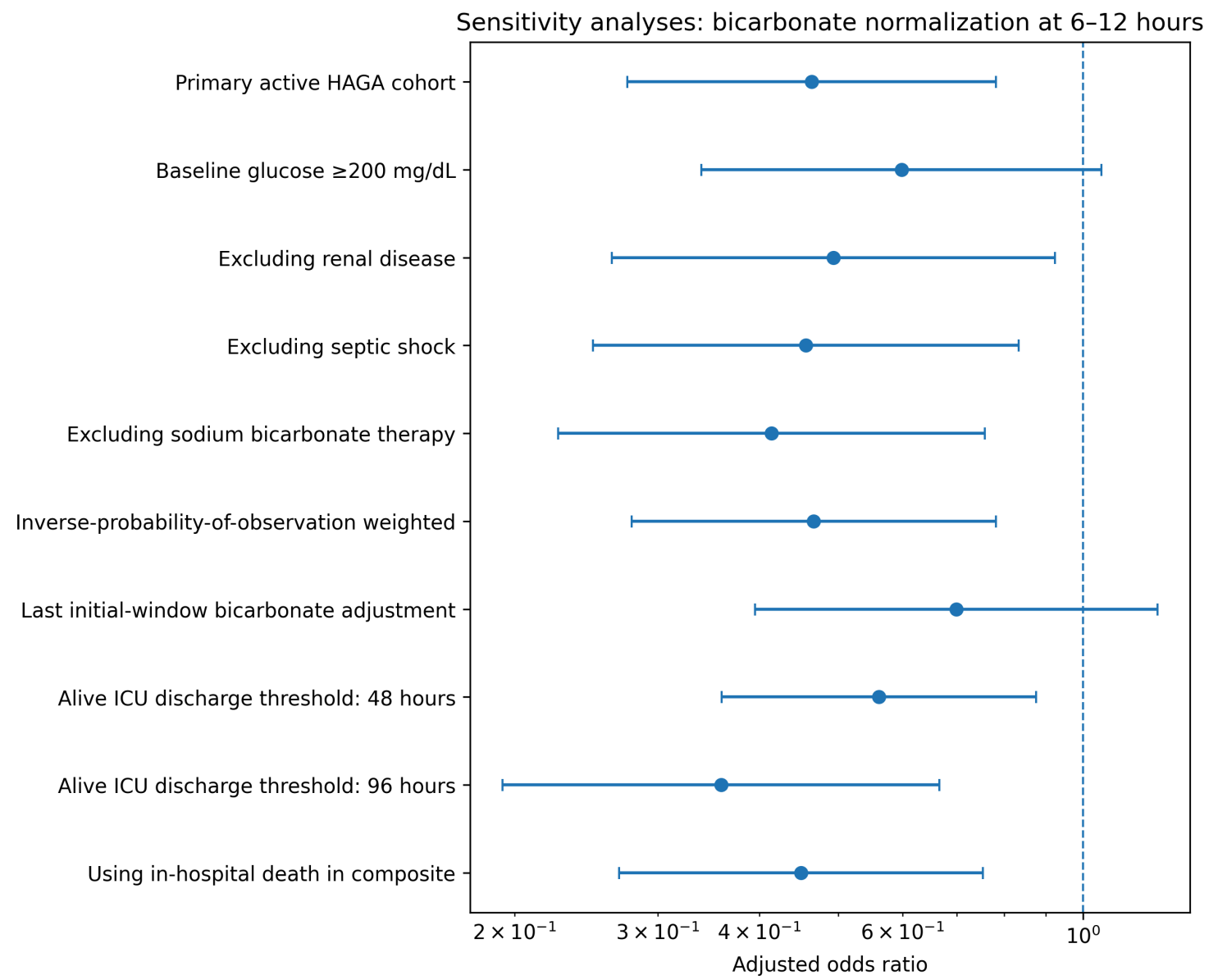


Figure 2

Sensitivity analyses of bicarbonate normalization at 6–12 hours and failure to achieve discharge alive from the ICU. ORs compare $\text{HCO}_3 \geq 18$ mmol/L with $\text{HCO}_3 < 18$ mmol/L.

Supplementary Files

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- [Additionalfile1.docx](#)
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