

ICU day-1 arterial bicarbonate and 30-day mortality in adults with acute pancreatitis discharge diagnoses

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Article

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Abstract

Arterial bicarbonate is routinely available, but its association with short-term mortality in acute pancreatitis is uncertain. We retrospectively studied adults with an acute pancreatitis discharge diagnosis and an intensive care unit (ICU) stay in MIMIC-IV version 3.1. The exposure was the first arterial blood gas bicarbonate measured from 6 h before to 24 h after ICU admission. The primary outcome was 30-day mortality. Among 1,280 eligible patients, 1,263 had a bicarbonate measurement and 201 died within 30 days. After adjustment for age, sex, weight, Charlson comorbidity score, and ICU day-1 Sequential Organ Failure Assessment, each 5 mmol/L lower bicarbonate was associated with higher mortality (odds ratio 1.19, 95% confidence interval 1.02–1.40; $P = 0.032$). Mortality was 22.5% with bicarbonate < 22 mmol/L and 9.5% with bicarbonate ≥ 22 mmol/L; the adjusted odds ratio was 1.73 (1.20–2.49; $P = 0.003$). The categorical association remained in an enzyme-supported cohort. Lower bicarbonate was associated with higher mortality, but attenuation after severity adjustment and limited incremental model performance suggest that it mainly reflects concurrent critical illness rather than an acute pancreatitis-specific prognostic signal.

Introduction

Acute pancreatitis is an increasingly common gastrointestinal emergency with a clinical course ranging from self-limited inflammation to persistent organ failure, necrosis, and death [1–6]. Contemporary international and national guidance identifies organ dysfunction as the principal determinant of severe disease and emphasizes early supportive and nutritional care, while recent intensive care unit (ICU) cohort data continue to show substantial early and in-hospital mortality among patients requiring critical care [1–4, 7, 8].

Arterial bicarbonate is readily available from blood gas analysis and contributes to the bedside assessment of acid-base status in critically ill patients [9]. Low concentrations may accompany tissue hypoperfusion and lactate accumulation, renal dysfunction, bicarbonate loss, or mixed acid-base disorders; these abnormalities may also be influenced by early fluid resuscitation and evolving organ failure [9–12]. Earlier studies associated lower arterial or peripheral venous bicarbonate with acute pancreatitis severity and mortality, although the available cohorts were relatively small and the incremental value beyond established severity assessment remained uncertain [13–15].

Recent database studies have examined several readily available physiologic or laboratory markers in ICU patients with acute pancreatitis, including anion gap, lactate-based indices, serum lactate, creatinine-to-albumin ratio, hemoglobin-to-red cell distribution width ratio, endothelial activation indices, and dynamic heart rate measures [16–23]. Other studies have evaluated composite scores and multivariable prediction tools [21, 24]. These findings demonstrate continued interest in early risk stratification, but they also highlight the need for clearly defined exposure windows, parsimonious adjustment for concurrent severity, and cautious distinction between statistical association and clinically meaningful incremental prediction.

We evaluated the association between the first arterial bicarbonate concentration measured during ICU day 1 and 30-day mortality among adults with an acute pancreatitis discharge diagnosis in MIMIC-IV version 3.1 [25–27]. We examined bicarbonate continuously and categorically, assessed the shape of the association, and evaluated its consistency in prespecified sensitivity analyses.

Results

Cohort selection

Of 94,458 ICU records screened, 1,686 carried an acute pancreatitis discharge diagnosis and all were aged > 18 years. Restriction to the first hospital admission and then the first ICU stay yielded 1,544 and 1,280 patients, respectively. After excluding 17 patients without an arterial blood gas bicarbonate measurement in the ICU day-1 window, the analysis cohort included 1,263 patients; 621 had bicarbonate < 22 mmol/L and 642 had bicarbonate $\geq$ 22 mmol/L (Fig. 1).

Baseline characteristics

Patients with bicarbonate < 22 mmol/L had higher day-1 Sequential Organ Failure Assessment (SOFA), Simplified Acute Physiology Score II (SAPS II), and Oxford Acute Severity of Illness Score (OASIS) values, together with more pronounced renal and metabolic abnormalities. The largest between-group differences involved arterial bicarbonate, anion gap, SAPS II, day-1 SOFA, creatinine, and blood urea nitrogen (Table 1).

Table 1
Baseline characteristics according to ICU day-1 arterial bicarbonate.

Characteristic	Overall (n = 1263)	Bicarbonate $\geq$ 22 (n = 642)	Bicarbonate < 22 (n = 621)	SMD
Age, years	59.6 (46.7–72.6)	59.1 (47.4–72.3)	60.2 (46.1–73.6)	0.02
Male sex	726 (57.5%)	392 (61.1%)	334 (53.8%)	-0.15
Weight, kg	81.5 (70.0–98.2)	81.5 (71.0–99.4)	81.3 (68.7–96.4)	-0.07
Charlson comorbidity score	4.0 (2.0–6.0)	3.0 (2.0–6.0)	4.0 (2.0–6.0)	0.2
ICU day-1 SOFA score	5.0 (2.0–8.0)	4.0 (2.0–6.0)	6.0 (3.0–10.0)	0.62
SAPS II score	35.0 (25.0–46.0)	30.0 (22.0–40.0)	40.0 (29.0–55.0)	0.68
OASIS score	32.0 (26.0–39.0)	30.0 (24.0–36.0)	34.0 (28.0–42.0)	0.46
Creatinine, mg/dL	1.1 (0.7–1.8)	0.8 (0.6–1.3)	1.4 (0.9–2.7)	0.57
Blood urea nitrogen, mg/dL	19.0 (12.0–35.0)	16.0 (10.0–25.0)	26.0 (15.0–46.0)	0.55
Anion gap, mmol/L	15.0 (13.0–19.0)	13.0 (11.0–15.0)	18.0 (15.0–22.0)	1.08
Chloride, mmol/L	103.0 (99.0–108.0)	102.0 (98.0–107.0)	104.0 (100.0–109.0)	0.25
Glucose, mg/dL	125.0 (102.0–168.0)	120.0 (100.0–152.0)	134.0 (106.0–189.0)	0.38
Total calcium, mg/dL	8.1 (7.4–8.6)	8.3 (7.7–8.7)	7.9 (7.1–8.5)	-0.43
White blood cell count, K/uL	12.4 (8.5–17.8)	12.1 (8.5–17.1)	12.7 (8.5–18.5)	0.07
Platelet count, K/uL	187.0 (128.0–266.8)	200.0 (142.0–283.5)	172.0 (115.5–249.5)	-0.2
Arterial bicarbonate, mmol/L	22.0 (18.0–25.0)	25.0 (23.0–27.0)	18.0 (15.0–20.0)	-2.44
Early invasive ventilation	475 (37.6%)	209 (32.6%)	266 (42.8%)	0.21
Early vasopressor use	397 (31.4%)	149 (23.2%)	248 (39.9%)	0.37
Values are median (interquartile range) or n (%). Positive standardized mean differences indicate higher values or prevalence in the bicarbonate < 22 mmol/L group. OASIS, Oxford Acute Severity of Illness Score; SAPS II, Simplified Acute Physiology Score II; SMD, standardized mean difference; SOFA, Sequential Organ Failure Assessment.				

Characteristic	Overall (n = 1263)	Bicarbonate $\geq$ 22 (n = 642)	Bicarbonate < 22 (n = 621)	SMD
Acute pancreatitis as principal diagnosis	430 (34.0%)	245 (38.2%)	185 (29.8%)	-0.18
Values are median (interquartile range) or n (%). Positive standardized mean differences indicate higher values or prevalence in the bicarbonate < 22 mmol/L group. OASIS, Oxford Acute Severity of Illness Score; SAPS II, Simplified Acute Physiology Score II; SMD, standardized mean difference; SOFA, Sequential Organ Failure Assessment.				

Mortality outcomes

Thirty-day mortality from ICU admission occurred in 201 patients. Mortality was 22.5% among patients with bicarbonate < 22 mmol/L and 9.5% among those with bicarbonate $\geq$ 22 mmol/L. The low-bicarbonate group also had higher unadjusted 90-day, in-hospital, and ICU mortality (Table 2).

Table 2
Unadjusted mortality outcomes according to ICU day-1 arterial bicarbonate.

Outcome	Bicarbonate $\geq$ 22	Bicarbonate < 22	P value
ICU-based 30-day mortality	61/642 (9.5%)	140/621 (22.5%)	< 0.001
ICU-based 90-day mortality	97/642 (15.1%)	178/621 (28.7%)	< 0.001
In-hospital mortality	58/642 (9.0%)	138/621 (22.2%)	< 0.001
ICU mortality	29/642 (4.5%)	91/621 (14.7%)	< 0.001
P values were obtained using Pearson's chi-square test. ICU, intensive care unit.			

Primary and secondary association analyses

In the severity-adjusted model, each 5 mmol/L lower arterial bicarbonate was associated with higher odds of 30-day mortality (odds ratio [OR] 1.19, 95% confidence interval [CI] 1.02–1.40; $P = 0.032$). In the secondary categorical analysis, bicarbonate < 22 mmol/L was associated with higher mortality in the unadjusted model (OR 2.77, 95% CI 2.00–3.83) and after adjustment for day-1 SOFA (OR 1.73, 95% CI 1.20–2.49; $P = 0.003$). The adjusted associations with 90-day, in-hospital, and ICU mortality were in the same direction (Table 3 and Fig. 2).

Table 3
Association between arterial bicarbonate < 22 mmol/L and mortality outcomes.

Outcome	Model	n	Events	OR (95% CI)	P value
ICU-based 30-day mortality	Model 1	1263	201	2.77 (2.00–3.83)	< 0.001
ICU-based 30-day mortality	Model 2	1262	201	2.68 (1.91–3.78)	< 0.001
ICU-based 30-day mortality	Model 3	1260	201	1.73 (1.20–2.49)	0.003
ICU-based 90-day mortality	Model 1	1263	275	2.26 (1.71–2.98)	< 0.001
ICU-based 90-day mortality	Model 2	1262	275	2.15 (1.59–2.89)	< 0.001
ICU-based 90-day mortality	Model 3	1260	275	1.43 (1.04–1.97)	0.029
In-hospital mortality	Model 1	1263	196	2.88 (2.07–4.00)	< 0.001
In-hospital mortality	Model 2	1262	196	2.75 (1.96–3.87)	< 0.001
In-hospital mortality	Model 3	1260	196	1.66 (1.14–2.40)	0.008
ICU mortality	Model 1	1263	120	3.63 (2.35–5.60)	< 0.001
ICU mortality	Model 2	1262	120	3.57 (2.29–5.55)	< 0.001
ICU mortality	Model 3	1260	120	1.79 (1.10–2.90)	0.019
Model 1 was unadjusted. Model 2 adjusted for age, sex, weight, and Charlson comorbidity score. Model 3 additionally adjusted for ICU day-1 SOFA. CI, confidence interval; ICU, intensive care unit; OR, odds ratio; SOFA, Sequential Organ Failure Assessment.					

Table 4
Continuous, quartile, and sensitivity analyses for 30-day mortality.

Analysis	n	Events	Effect (95% CI)	P value
Per 5 mmol/L lower arterial bicarbonate	1260	201	1.19 (1.02–1.40)	0.032
Quartile: Q3 vs Q4 (23–25 mmol/L)	1260	201	0.89 (0.47–1.68)	0.73
Quartile: Q2 vs Q4 (19–22 mmol/L)	1260	201	1.27 (0.73–2.23)	0.398
Quartile: Q1 vs Q4 (3–18 mmol/L)	1260	201	1.68 (0.96–2.92)	0.068
Ordinal trend per lower quartile	1260	201	1.24 (1.04–1.47)	0.016
Deaths within the first ICU day excluded	1232	173	1.56 (1.07–2.27)	0.020
Acute pancreatitis as principal discharge diagnosis	428	41	0.92 (0.41–2.07)	0.841
Fixed-threshold enzyme-supported phenotype	489	77	2.16 (1.13–4.15)	0.020
Alternative laboratory-adjusted model	1240	200	2.47 (1.69–3.59)	< 0.001
Modified Poisson model (adjusted risk ratio)	1260	201	1.49 (1.13–1.97)	0.004
Multiple imputation (20 datasets)	1263	201	1.73 (1.20–2.49)	0.003
Unless otherwise stated, effects are adjusted odds ratios from Model 3. Quartile 4 (26–39 mmol/L) was the reference group. Sensitivity-analysis rows from exclusion of first-ICU-day deaths through multiple imputation evaluate bicarbonate < 22 versus ≥ 22 mmol/L unless another exposure scale is stated. The fixed-threshold enzyme-supported phenotype required maximum blood amylase ≥ 300 U/L or lipase ≥ 180 U/L between hospital admission and 24 h after ICU admission. The modified Poisson estimate is an adjusted risk ratio. The alternative laboratory-adjusted model included age, sex, weight, Charlson score, creatinine, blood urea nitrogen, glucose, total calcium, white blood cell count, and platelet count.				

Dose-response and sensitivity analyses

The restricted cubic spline indicated an overall association between arterial bicarbonate and 30-day mortality ($P = 0.041$) without evidence of nonlinearity ($P = 0.164$; Fig. 3). The ordinal quartile trend was statistically significant ($P = 0.016$), although individual quartile estimates were imprecise and were not strictly monotonic.

Associations were directionally consistent across alternative ICU day-1 bicarbonate summaries (Supplementary Table S2). Exploratory subgroup estimates are presented in Supplementary Table S4 and Supplementary Fig. S1.

Similar estimates were observed after exclusion of deaths during the first ICU day and after multiple imputation. The modified Poisson model yielded an adjusted risk ratio of 1.49 (95% CI 1.13–1.97). The laboratory-adjusted estimate was larger than the SOFA-adjusted estimate, indicating that the magnitude depended on how concurrent illness severity was modeled. Pancreatic amylase or lipase was measured in 807 patients, and 491 met the fixed-threshold enzyme-supported definition. Among 490 with an arterial bicarbonate result, mortality was 22.5% in the low-bicarbonate group and 7.3% in the reference group. In 489 patients with complete Model 3 covariates, bicarbonate < 22 mmol/L remained associated with 30-day mortality (OR 2.16, 95% CI 1.13–4.15; $P = 0.020$), while the continuous estimate per 5 mmol/L decrease was less precise (OR 1.22, 95% CI 0.92–1.63; $P = 0.170$). The ICU-proximate and principal-diagnosis-plus-enzyme estimates were in the same direction but had wide confidence intervals (Supplementary Table S7). In the principal-diagnosis subgroup without requiring enzyme support, the adjusted estimate remained close to the null and imprecise (OR 0.92, 95% CI 0.41–2.07); the interaction by diagnostic position was not statistically significant ($P = 0.124$).

Discussion

Lower arterial bicarbonate during ICU day 1 was associated with higher 30-day mortality in the overall cohort of ICU patients identified using acute pancreatitis discharge codes. The categorical association was also observed in a fixed-threshold enzyme-supported sensitivity cohort, although continuous and stricter subgroup estimates were imprecise. Arterial bicarbonate should be viewed as a readily available marker of overall critical illness; its acute pancreatitis-specific prognostic value remains uncertain.

The findings are consistent with earlier studies linking lower arterial or peripheral venous bicarbonate to acute pancreatitis severity and mortality [13–15]. They also complement recent MIMIC-based reports describing associations between mortality and anion gap, lactate-to-albumin ratio, serum lactate, creatinine-to-albumin ratio, hemoglobin-to-red cell distribution width ratio, and the endothelial activation and stress index [16–22]. The present study differs by evaluating the first arterial bicarbonate value within a prespecified – 6-hour to + 24-hour ICU window and by using a severity score restricted to the first ICU day. The reduction in effect size after SOFA adjustment indicates that arterial bicarbonate and concurrent organ dysfunction capture overlapping prognostic information.

Arterial bicarbonate may reflect several components of critical illness, including impaired perfusion, lactate accumulation, renal acid-base dysfunction, bicarbonate loss, and treatment-related shifts in chloride and extracellular volume [9–12]. The low-bicarbonate group had higher anion gap, creatinine, blood urea nitrogen, and SOFA scores, supporting this interpretation. Persistent organ failure is strongly linked to mortality in severe and necrotizing acute pancreatitis [7, 28], making it plausible that

bicarbonate partly functions as an integrated marker of systemic illness rather than a disease-specific mediator.

The diagnostic sensitivity analyses refine, but do not eliminate, concerns about disease specificity. The association was not demonstrated in the subgroup defined only by a principal acute pancreatitis discharge diagnosis. In contrast, the categorical estimate remained elevated in the fixed-threshold enzyme-supported phenotype and was directionally similar in the subgroup requiring both a principal diagnosis and enzyme support. The latter estimate was based on only 20 deaths and was imprecise. The continuous estimate within the enzyme-supported cohort was imprecise. These findings support the plausibility that acute pancreatitis was clinically present in at least part of the cohort, but they do not establish a disease-specific bicarbonate threshold or resolve residual diagnostic misclassification.

Arterial bicarbonate is inexpensive and rapidly available, but it should be interpreted together with perfusion, renal function, fluid balance, and concurrent organ failure. The exploratory model-performance analysis showed negligible improvement in discrimination beyond age, comorbidity burden, and day-1 SOFA (Supplementary Table S5). Observational findings should not be used to infer benefit from bicarbonate administration: studies in acute pancreatitis with hyperlactatemia and randomized evidence in severe metabolic acidemia do not support routine correction based on bicarbonate concentration alone [29, 30]. Accordingly, the present data support bicarbonate as a nonspecific marker of concurrent illness severity rather than a stand-alone acute pancreatitis-specific predictor or treatment target.

Strengths include a relatively large ICU cohort, a prespecified early arterial blood gas window, ICU-based mortality endpoints, use of a time-restricted day-1 SOFA score, parsimonious adjustment models, and several sensitivity analyses.

The study has limitations. It was retrospective and single-center, and residual confounding remains possible. Pancreatic enzymes were not measured in all patients, and the phenotype could not confirm abdominal pain or characteristic imaging. It should not be interpreted as a fully adjudicated revised Atlanta diagnosis. The first arterial bicarbonate value was obtained according to clinical need rather than a study protocol. Secondary and subgroup analyses were exploratory and were not adjusted for multiplicity.

Lower arterial bicarbonate during ICU day 1 was associated with higher 30-day mortality in the overall cohort of ICU patients identified using acute pancreatitis discharge codes. The association was attenuated after adjustment for concurrent illness severity and was not demonstrated in the smaller principal-diagnosis subgroup. Arterial bicarbonate should be viewed as a readily available marker of overall critical illness; its acute pancreatitis-specific prognostic value remains uncertain.

Methods

Data source and study design

This retrospective cohort study used de-identified data from MIMIC-IV version 3.1, an electronic health record database from Beth Israel Deaconess Medical Center that contains admissions from 2008 through 2022 [25–27]. Study variables were extracted directly from the MIMIC-IV database. Reporting followed the STROBE recommendations [31]. The MIMIC-IV project was approved 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 de-identified data. All methods were performed in accordance with relevant guidelines and regulations.

Study population

The source population comprised 94,458 ICU records available for screening. Acute pancreatitis was identified using ICD-9 discharge diagnosis code 577.0 or ICD-10 code K85 and its subcodes, yielding 1,686 records. Restriction to patients aged > 18 years left 1,686 records. We then retained the first hospital admission per patient ($n = 1,544$) and the first ICU stay within the retained admission ($n = 1,280$). Patients without an arterial blood gas bicarbonate measurement during the predefined ICU day-1 window were excluded. To reduce concern about diagnostic misclassification, an enzyme-supported sensitivity phenotype required an acute pancreatitis discharge code together with a maximum blood amylase concentration ≥ 300 U/L or maximum blood lipase concentration ≥ 180 U/L from hospital admission through 24 hours after ICU admission. This supportive phenotype was not considered equivalent to confirmation of all revised Atlanta diagnostic criteria, and absence of enzyme testing was not interpreted as a negative test.

Exposure assessment

The primary exposure was the first bicarbonate value reported from arterial blood gas analysis from 6 hours before to 24 hours after ICU admission. When several measurements were available, the earliest value was selected. Bicarbonate was modeled continuously per 5 mmol/L decrease. A prespecified secondary categorical analysis, selected for clinical interpretability, compared concentrations < 22 with ≥ 22 mmol/L. Minimum, average, median, last, and maximum values within the same window were evaluated in sensitivity analyses.

Outcomes

The primary outcome was all-cause mortality within 30 days of ICU admission. Secondary mortality outcomes were 90-day mortality from ICU admission, in-hospital mortality, and ICU mortality. Death dates were obtained from MIMIC-IV mortality records. Renal outcomes, including acute kidney injury, renal replacement therapy, and continuous renal replacement therapy during the index hospitalization, were exploratory and are reported in Supplementary Tables S3A and S3B.

Covariates

Covariates were selected before analysis to limit redundant adjustment. The demographic and comorbidity model included age, sex, body weight, and Charlson comorbidity score. The severity-adjusted model additionally included the maximum SOFA score during the first 24 hours after ICU

admission. An alternative model replaced SOFA with the first ICU day-1 values of creatinine, blood urea nitrogen, glucose, total calcium, white blood cell count, and platelet count. Early invasive ventilation and vasopressor use were described but were not included in the principal model because they may reflect concurrent treatment and illness severity. Operational definitions and extraction windows are provided in Supplementary Table S6.

Statistical analysis

Continuous variables are presented as medians with interquartile ranges because normal distributions were not assumed; categorical variables are presented as counts and percentages. Standardized mean differences describe between-group imbalance. Unadjusted categorical outcome comparisons used Pearson's chi-square test. Logistic regression estimated odds ratios (ORs) and 95% confidence intervals (CIs). Model 1 was unadjusted. Model 2 adjusted for age, sex, weight, and Charlson score. Model 3 additionally adjusted for ICU day-1 SOFA and was used to assess whether the association persisted after accounting for concurrent illness severity; it was not intended to estimate a causal effect independent of potential mediators. All tests were two-sided with an alpha level of 0.05. Exact P values are reported where available; values below 0.001 are shown as $P < 0.001$. Variable-level missingness is reported in Supplementary Table S1.

A restricted cubic spline with four degrees of freedom evaluated the continuous association, using 22 mmol/L as the reference. Likelihood-ratio tests assessed the overall and nonlinear spline terms. Sensitivity analyses excluded patients who died during the first ICU day, restricted the cohort to patients with acute pancreatitis as the principal discharge diagnosis, used the fixed-threshold enzyme-supported phenotype, used an alternative laboratory-adjusted model, estimated adjusted risk ratios with modified Poisson regression and robust variance [32], and used 20 iteratively imputed datasets for missing covariates following established chained-equation principles [33]. Within the enzyme-supported cohort, bicarbonate was evaluated both categorically and continuously using Model 3. An ICU-proximate analysis required the first qualifying pancreatic enzyme measurement to occur from 6 hours before to 24 hours after ICU admission. Because the subgroup combining a principal acute pancreatitis diagnosis and enzyme support contained only 20 deaths, it was adjusted parsimoniously for age, sex, and day-1 SOFA. Exposure and outcome values were not imputed. Secondary, subgroup, and exploratory analyses were not adjusted for multiplicity and should be interpreted as hypothesis-generating. Analyses were conducted with Python 3.13.5, pandas 2.2.3, and statsmodels 0.14.6.

Declarations

Competing interests

The author declares no competing interests.

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Author Contribution

Y.Z. conceived the study, specified the data extraction, curated the data, performed and verified the analyses, interpreted the findings, drafted the manuscript, prepared the figures, and approved the final version.

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Data Availability

The raw deidentified clinical data supporting this retrospective cohort analysis are sourced from the MIMIC-IV version 3.1 database (<https://doi.org/10.13026/kpb9-mt58>), hosted on PhysioNet. MIMIC-IV is restricted to credentialed researchers who complete required human subjects training and sign a formal Data Use Agreement via the PhysioNet platform. No raw patient-level data can be publicly shared by the author due to privacy and data licensing constraints. Aggregate summary statistics, supplementary tables, figures, and reproducible analytical code that support all findings presented in this manuscript are available from the corresponding author upon reasonable written request.

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Figures

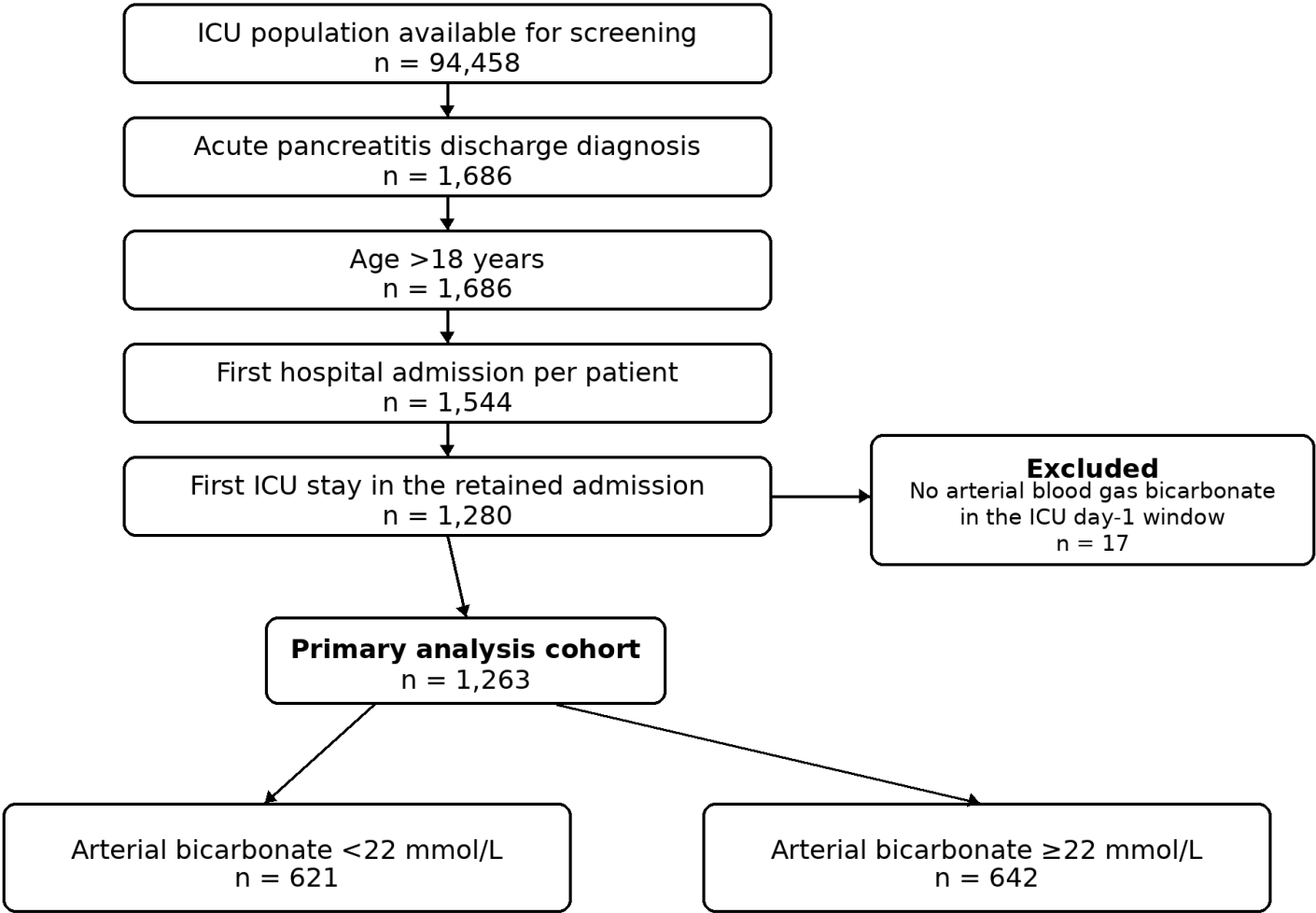


Figure 1

Study population flow diagram. The diagram shows cohort selection from the MIMIC-IV ICU population to the primary analysis cohort and the two arterial bicarbonate groups. ICU, intensive care unit.

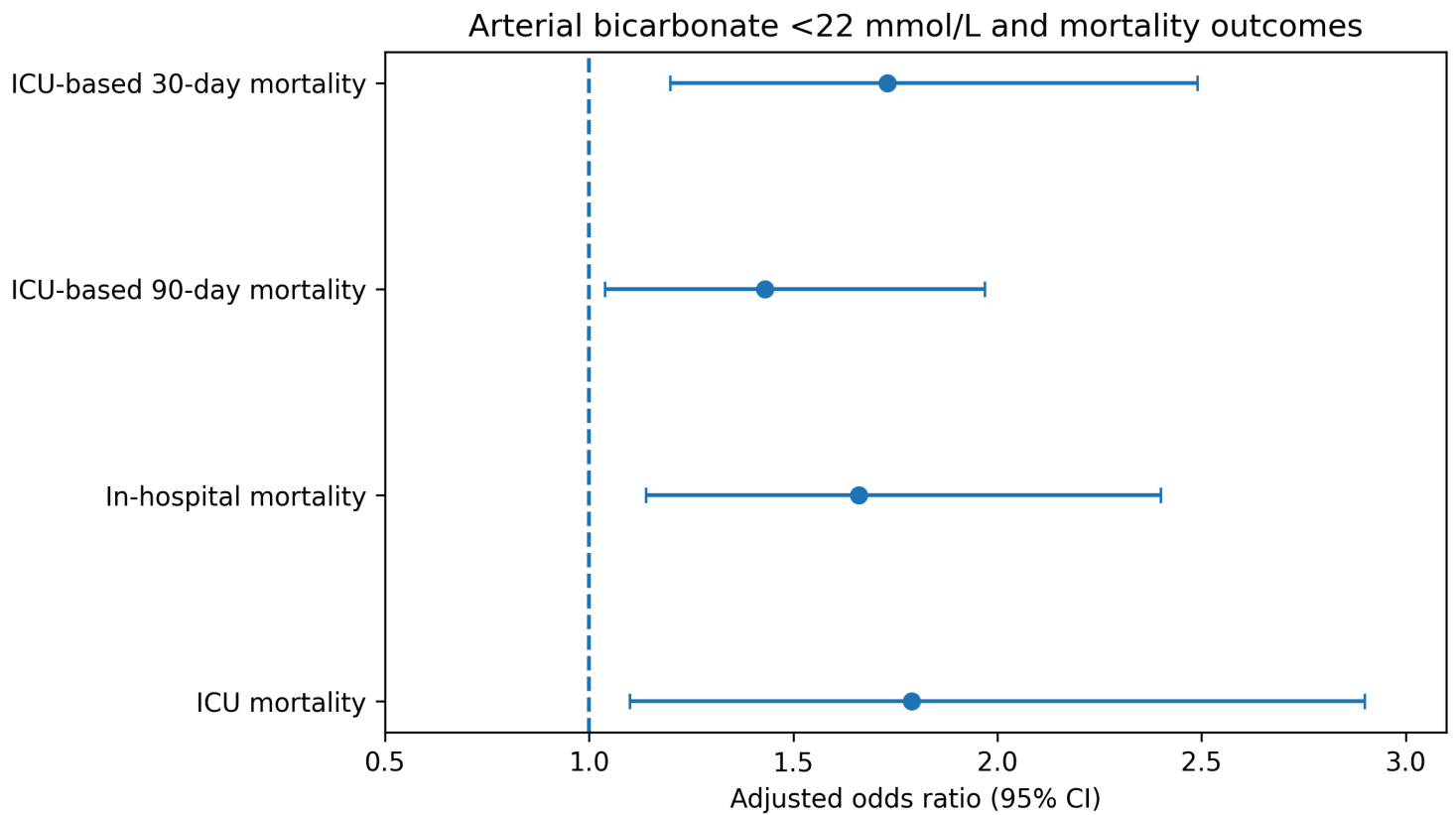


Figure 2

Severity-adjusted associations between arterial bicarbonate <22 mmol/L and mortality outcomes. Points show adjusted odds ratios and horizontal bars show 95% confidence intervals from Model 3, adjusted for age, sex, weight, Charlson comorbidity score, and ICU day-1 SOFA. The dashed vertical line indicates an odds ratio of 1. CI, confidence interval; ICU, intensive care unit; SOFA, Sequential Organ Failure Assessment.

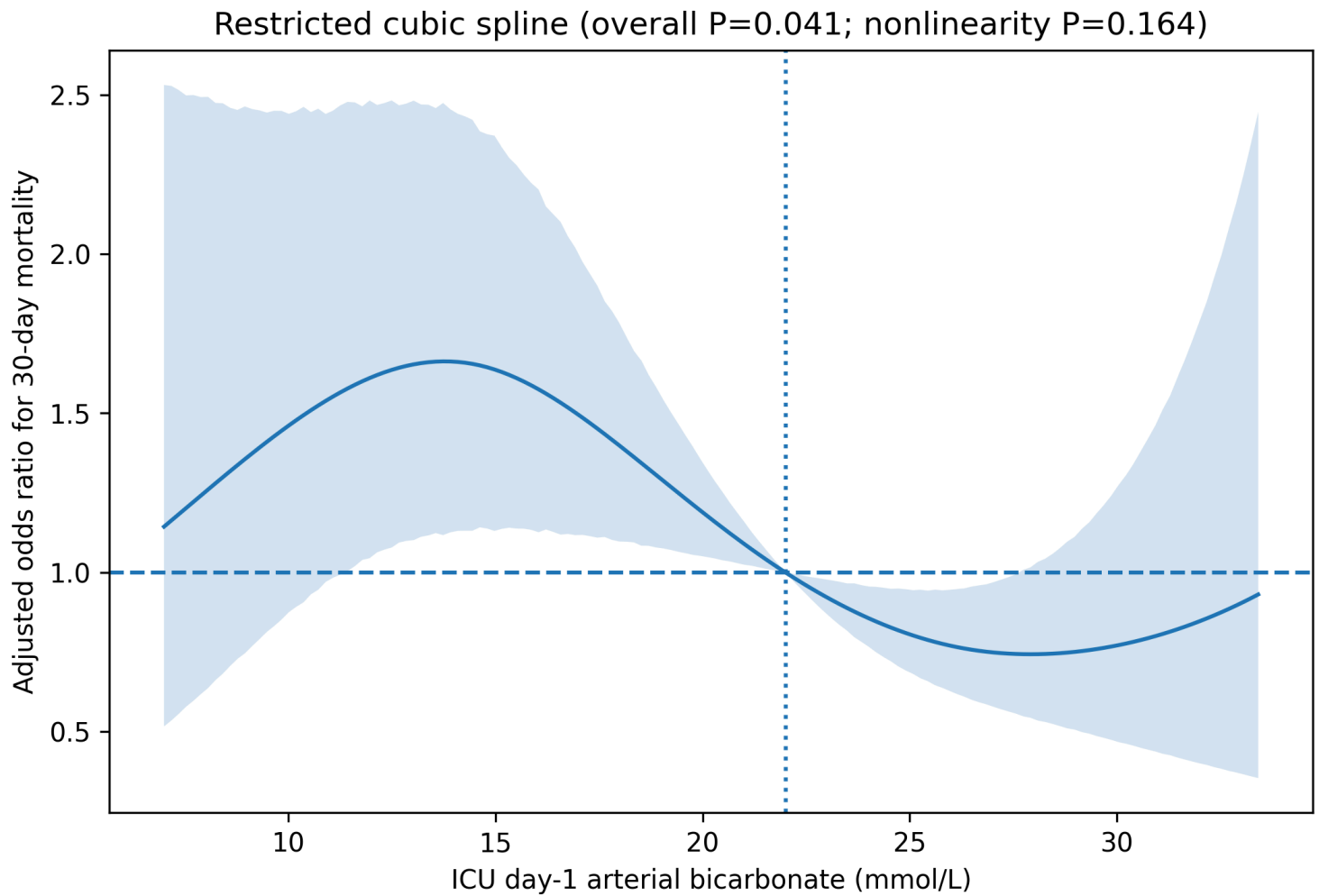


Figure 3

Restricted cubic spline for ICU day-1 arterial bicarbonate and 30-day mortality. The solid line shows adjusted odds ratios from Model 3 relative to 22 mmol/L, and the shaded area shows 95% confidence intervals. The horizontal dashed line indicates an odds ratio of 1 and the vertical dotted line indicates the reference value. The spline used four degrees of freedom (overall P = 0.041; nonlinearity P = 0.164). The plot displays the central range of observed bicarbonate values.

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