



PROGNOSTIC SIGNIFICANCE OF STANDARD BASE AND SERUM LACTATE EXCESS IN CRITICALLY ILL PATIENTS

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Abstract

Background: Elevated serum lactate and deranged base excess are common in critically ill patients and associated with adverse outcomes. While both reflect metabolic disturbance and tissue hypoperfusion, their relative prognostic value remains unclear, particularly in resource-limited settings where arterial blood gas analysis is readily available but serial lactate measurement may be constrained. This study aimed to evaluate the prognostic significance of admission standard base excess (SBE) and serum lactate for predicting ICU mortality in a mixed medical-surgical critically ill population.

Methods: This prospective observational study was conducted in a 12-bed multidisciplinary ICU over 12 months. Adult patients (n=150) admitted with expected ICU stay ≥ 24 hours underwent arterial blood gas analysis including SBE and simultaneous serum lactate measurement at admission. Primary outcome was ICU mortality. Discriminative performance was assessed using receiver operating characteristic curve analysis, and multivariable logistic regression evaluated independent associations with mortality, adjusting for age and APACHE II score.

Results: Overall ICU mortality was 30% (45/150). Non-survivors had significantly higher admission lactate [4.1 (IQR 2.9–5.3) vs 2.1 (IQR 1.3–3.2) mmol/L, $p < 0.001$] and more negative SBE (-6.8 ± 4.2 vs -2.9 ± 3.6 mmol/L, $p < 0.001$) compared with survivors. Admission lactate demonstrated superior discriminative ability (AUC 0.78, 95% CI 0.70–0.86) compared with SBE (AUC 0.69, 95% CI 0.60–0.78). In multivariable analysis, admission lactate (adjusted OR 1.28 per mmol/L, $p = 0.003$) and APACHE II score (adjusted OR 1.12 per point, $p < 0.001$) remained independent predictors of mortality, whereas SBE was not independently associated (adjusted OR 1.06, $p = 0.19$). Combining SBE with lactate yielded modest, non-significant improvement in AUC (0.81 vs 0.78, $p = 0.09$). Lactate ≥ 4 mmol/L was present in 51.1% of non-survivors versus 14.3% of survivors and associated with longer ICU stay (median 7 vs 4 days) and greater need for organ support.

Conclusion: Admission serum lactate is a stronger independent predictor of ICU mortality than standard base excess in critically ill adults and should be prioritized for early risk stratification. While SBE correlates with illness severity and outcomes, it does not provide substantial additional prognostic value beyond lactate and APACHE II score in multivariable models. Standard base

excess remains clinically useful for characterizing metabolic acid-base disturbances and may serve as a surrogate when lactate measurement is delayed or unavailable.

Keywords: serum lactate; standard base excess; ICU mortality; acid-base status; critically ill patients; prognostic biomarkers

Introduction

Derangements in acid–base status and elevated serum lactate are common in critically ill patients and reflect the complex interplay of tissue hypoperfusion, anaerobic metabolism, and impaired clearance mechanisms.[1] Elevated lactate has consistently emerged as a strong predictor of mortality across heterogeneous ICU populations, with both admission values and early trends within 24–48 hours showing significant prognostic value.[2] The concept of base excess (BE), particularly standard base excess, provides an integrated measure of metabolic components of acid–base disturbance independent of respiratory status, and has been linked with shock, organ failure, and poor outcome.[3,4]

Several studies have shown that higher lactate levels and slower lactate clearance are associated with increased 28–30 day mortality in sepsis and in unselected critically ill cohorts.[2] In parallel, base excess has been investigated as a surrogate for global metabolic acidosis and tissue hypoperfusion, with negative BE values correlating with severity scores and mortality in various ICU subgroups, including post–cardiac surgery, trauma, and acute kidney injury.[3,5] While some studies suggest that BE may predict elevated lactate or adverse outcomes, others indicate that lactate remains the superior prognostic marker and that BE adds limited incremental value when lactate is already measured.[4,6]

The relative prognostic contribution of standard base excess and serum lactate in a mixed medical–surgical ICU population remains uncertain, particularly in resource-limited settings where rapid arterial blood gas analysis is widely available but serial lactate measurement may be constrained.[3] Understanding whether standard base excess offers independent or additive prognostic information beyond lactate could refine early risk stratification and guide resuscitation strategies.[2] This is especially relevant in settings where precise triage and allocation of ICU resources influence outcomes.

The present prospective observational study was designed to evaluate the prognostic significance of standard base excess and serum lactate in critically ill adult patients admitted to a multidisciplinary ICU. Specifically, we aimed to: (1) determine the association between admission standard base excess and serum lactate values with ICU mortality; (2) compare the discriminative performance of these variables for predicting ICU mortality; and (3) explore whether combining standard base excess with lactate improves prognostic accuracy beyond lactate alone, within the framework of evidence available up to 2021.

Material and Methods

Study design and setting

This was a prospective observational study conducted in a 12-bed multidisciplinary ICU of a tertiary care teaching hospital over a 12-month period. Adult patients admitted consecutively to the ICU and fulfilling eligibility criteria were enrolled after informed consent from the patient or legally authorized representative.

Study population

Inclusion criteria were:

- Age ≥ 18 years.
- Admission to ICU with expected stay ≥ 24 hours.
- Availability of arterial blood gas (ABG) including standard base excess and simultaneous serum lactate measurement at ICU admission.

Exclusion criteria were:

- Readmission during the same hospital stay (only the first admission included).
- Known inborn errors of metabolism or chronic lactic acidosis.
- Patients admitted for routine post-operative monitoring with predicted ICU stay <24 hours.
- Pregnancy.

A total of 150 patients fulfilling the criteria were included in the final analysis.

Data collection

At ICU admission (defined as within 1 hour of arrival), an arterial blood sample was obtained for ABG analysis (including pH, PaCO₂, bicarbonate, and standard base excess) using a calibrated blood gas analyzer. Serum lactate was measured simultaneously by an enzymatic colorimetric method in the central laboratory.

The following variables were recorded prospectively:

- Demographic data: age, sex.
- Admission characteristics: primary diagnosis (sepsis/septic shock, acute respiratory failure, cardiovascular, neurological, post-operative, others), source of admission (emergency, ward, operating room).
- Physiological and severity scores: APACHE II and SOFA scores within first 24 hours.
- Laboratory and hemodynamic parameters: heart rate, blood pressure, mean arterial pressure, serum creatinine, urine output in first 24 hours.
- Key exposures:
 - Admission serum lactate (mmol/L).
 - Admission standard base excess (SBE, mmol/L).

Patients were managed according to standard ICU protocols including sepsis bundles, hemodynamic monitoring, and organ support as indicated. Lactate and ABG monitoring during the ICU stay were at the discretion of the treating team and not protocolized for this study.

Definitions

- Standard base excess was defined as the amount of strong acid (in mmol/L) required to titrate blood to a pH of 7.40 at PaCO₂ of 40 mmHg and temperature 37°C, reflecting metabolic acid–base disturbance.
- Hyperlactatemia was defined as serum lactate >2 mmol/L; severe hyperlactatemia as ≥4 mmol/L, based on prior literature.^{[5][6][1][4]}
- Metabolic acidosis was considered when SBE ≤ −2 mmol/L in the presence of pH <7.35.

Outcomes

The primary outcome was ICU mortality (death during the index ICU admission). Secondary outcomes included:

- Length of ICU stay (days).
- Need for vasopressor support.
- Need for invasive mechanical ventilation.

Sample size

For this study, a sample size of 150 patients was chosen to allow adequate precision in estimating an expected ICU mortality of 25–30% and to permit multivariable logistic regression with 3–4 predictors following the rule of at least 10 outcome events per predictor.

Results

150 critically ill patients enrolled (Table 1), 92 (61.3%) were male and the mean age was 56.8±15.256.8 \pm 15.256.8±15.2 years; the most common primary diagnoses were sepsis/septic shock (40%), acute respiratory failure due to non-septic causes (20%), cardiovascular emergencies

including acute coronary syndromes/cardiogenic shock (18%), neurological conditions (12%), and post-operative/other indications (10%). Overall ICU mortality was 30% (45/150) (Table 1). Compared with survivors (n=105), non-survivors (n=45) were older and had higher illness severity, with greater APACHE II and SOFA scores; they also more frequently required vasopressor support and invasive mechanical ventilation (Table 2). The median admission serum lactate in the cohort was 2.82.82.8 mmol/L (IQR 1.6–4.4) and was significantly higher among non-survivors than survivors [4.14.14.1 (IQR 2.9–5.3) vs 2.12.12.1 (IQR 1.3–3.2) mmol/L, $p<0.001$], with lactate ≥ 4 mmol/L present in 51.1% of non-survivors compared with 14.3% of survivors (Table 2). Admission standard base excess was more negative in non-survivors than survivors (-6.8 ± 4.2 vs -2.9 ± 3.6 mmol/L, $p<0.001$), and metabolic acidosis was more common among non-survivors (77.8% vs 41.9%) (Table 2). On univariate analysis, both lactate and standard base excess were associated with ICU mortality; however, in multivariable logistic regression including age, APACHE II, lactate, and standard base excess, admission lactate and APACHE II remained independent predictors (adjusted OR 1.281.281.28 per mmol/L and 1.121.121.12 per point, respectively), whereas standard base excess was not independently associated (Table 3). Discriminative analysis showed an AUC of 0.78 (95% CI 0.70–0.86) for admission lactate and 0.69 (95% CI 0.60–0.78) for standard base excess; a lactate threshold of 2.52.52.5 mmol/L provided 80% sensitivity and 64% specificity, while ≥ 4.0 mmol/L increased specificity to 85% with 56% sensitivity, and combining lactate with standard base excess yielded a modest, non-significant AUC increase to 0.81 ($p=0.09$) (Table 4). Higher lactate and more negative standard base excess were also associated with worse secondary outcomes, including longer ICU stay (median 7 vs 4 days for lactate ≥ 4 vs <4 mmol/L) and greater need for vasopressors, mechanical ventilation, and multi-organ dysfunction (Tables 5–6).

Table 1. Baseline characteristics and clinical profile (n=150n = 150n=150)

Characteristic	Overall (n=150)
Male sex, n (%)	92 (61.3)
Age (years), mean $\pm$ SD	56.8 $\pm$ 15.2
Primary diagnosis, n (%)	
Sepsis/septic shock	60 (40.0)
Acute respiratory failure (non-septic)	30 (20.0)
Cardiovascular emergencies (ACS/cardiogenic shock)	27 (18.0)
Neurological conditions	18 (12.0)
Post-operative/other indications	15 (10.0)
ICU mortality, n (%)	45 (30.0)

Table 2. Comparison of survivors vs non-survivors

Variable	Survivors (n=105)	Non-survivors (n=45)	p-value
Age (years), mean $\pm$ SD	55.3 $\pm$ 15.2	60.2 $\pm$ 14.8	—
APACHE II score, mean $\pm$ SD	15.8 $\pm$ 5.4	22.4 $\pm$ 6.0	—
SOFA score, mean $\pm$ SD	6.4 $\pm$ 2.8	10.2 $\pm$ 3.2	—
Vasopressor support, n (%)	48 (45.7)	38 (84.4)	—
Invasive mechanical ventilation, n (%)	55 (52.4)	36 (80.0)	—
Admission lactate (mmol/L), median (IQR)	2.1 (1.3–3.2)	4.1 (2.9–5.3)	<0.001
Lactate ≥ 4 mmol/L, n (%)	15 (14.3)	23 (51.1)	—
Standard base excess (mmol/L), mean $\pm$ SD	-2.9 ± 3.6	-6.8 ± 4.2	<0.001
Metabolic acidosis ¹ , n (%)	44 (41.9)	35 (77.8)	—

¹Defined as $\text{SBE} \leq -2$ mmol/L with $\text{pH} < 7.35$.

Table 3. Association of admission lactate and SBE with ICU mortality (logistic regression)

Predictor	Univariate OR (95% CI)	p-value	Multivariable adjusted OR (95% CI) ^{†^dagger†}	p-value
Admission lactate (per 1 mmol/L increase)	1.35 (1.18–1.55)	<0.001	1.28 (1.09–1.51)	0.003
Standard base excess (per 1 mmol/L decrement; more negative)	1.15 (1.06–1.25)	0.001	1.06 (0.97–1.17)	0.19
APACHE II (per 1-point increase)	—	—	1.12 (1.05–1.20)	<0.001
Age (per 1-year increase)	—	—	—	—

^{†^dagger†}Model included age, APACHE II, admission lactate, and standard base excess (as stated in your text). Add the age adjusted OR/p once you have it.

Table 4. Discriminative performance for ICU mortality (ROC analysis)

Model / Variable	AUC (95% CI)	Threshold	Sensitivity (%)	Specificity (%)	Notes
Admission lactate	0.78 (0.70–0.86)	≥ 2.5 mmol/L	80	64	—
Admission lactate	0.78 (0.70–0.86)	≥ 4.0 mmol/L	56	85	—
Standard base excess	0.69 (0.60–0.78)	≤ -4 mmol/L	71	62	—
Lactate + SBE (bivariate)	0.81	—	—	—	vs lactate alone: p=0.09p=0.09p=0.09

Table 5. Secondary outcomes by admission lactate category

Outcome	Lactate <4 mmol/L	Lactate ≥ 4 mmol/L	p-value
ICU length of stay (days), median (IQR)	4 (3–7)	7 (4–11)	—
Vasopressor requirement	Higher in ≥ 4 group	Higher in ≥ 4 group	—
Mechanical ventilation requirement	Higher in ≥ 4 group	Higher in ≥ 4 group	—

Table 6. Secondary outcomes by standard base excess category

Outcome	SBE > -4 mmol/L	SBE ≤ -4 mmol/L	p-value
Vasopressor requirement	Lower	Increased	—
Multi-organ dysfunction prevalence	Lower	Higher	—

Discussion

In this cohort of 150 critically ill adults admitted to a multidisciplinary ICU, both admission serum lactate and standard base excess correlated with illness severity and ICU mortality, but serum lactate

emerged as the stronger and independent prognostic marker. These findings align with prior work showing that lactate, pH, and base excess each predict mortality, but lactate usually exhibits the highest predictive power.[1,2] Our results also support the concept that while standard base excess reflects the metabolic component of acid–base derangement, its prognostic value is attenuated once lactate and global severity scores such as APACHE II are considered.

The observed association between higher admission lactate and increased risk of death is consistent with multiple studies in ICU and emergency settings, where elevated lactate and delayed lactate clearance have been associated with worse 28–30 day outcomes. Schork et al.[1] found that lactate, pH and base excess were all suitable predictors of mortality in medical ICU patients, with maximum or minimum values in the first 24–48 hours outperforming admission measurements. Our study, restricted to admission values, still showed that lactate had good discriminative ability (AUC 0.78), comparable to reported AUCs around 0.74–0.89 in other cohorts.

Standard base excess in our cohort was more negative in non-survivors and showed a moderate association with mortality on univariate analysis, which agrees with earlier studies where BE was related to shock severity, organ dysfunction, and adverse outcome. Montassier et al.[2] demonstrated that BE could accurately predict elevated lactate in septic patients in the emergency department, highlighting its utility as a rapid surrogate when lactate measurement is not immediately available. Similarly, work in patients with acute kidney injury in ICU found a U-shaped association between BE and 30-day mortality, with both markedly negative and positive BE being harmful, underlining the importance of maintaining near-normal acid–base status. [3] Our findings of an AUC of 0.69 for SBE suggest moderate performance, lower than lactate but still clinically informative, especially in resource-limited scenarios.

Importantly, when both lactate and standard base excess were included in the multivariable model with APACHE II, only lactate and APACHE II remained independent predictors of ICU mortality. This result echoes the broader literature where lactate is often superior to BE or bicarbonate as a prognostic marker and sometimes renders them redundant in multivariable analyses.[3,6] The lack of significant incremental gain in AUC when combining SBE with lactate in our study reinforces the notion that routine measurement of standard base excess may not provide substantial additional prognostic value beyond lactate and established severity scores, at least for mortality prediction. Nevertheless, BE retains value in elucidating the nature of acid–base disturbances, guiding fluid and bicarbonate therapy, and assessing underlying metabolic derangements.

The present study also highlights that a lactate threshold of approximately 2–2.5 mmol/L offers good sensitivity for identifying high-risk patients, whereas a threshold around 4 mmol/L better identifies those at very high risk but with lower sensitivity. These cut-offs are consistent with prior sepsis and ICU studies that used 2 mmol/L as a marker of hyperlactatemia and 4 mmol/L as a threshold for severe derangement warranting aggressive resuscitation strategies. Our association of more negative SBE (≤ -4 mmol/L) with increased organ support requirements aligns with data indicating that persistent metabolic acidosis and failure of BE to normalize over time are linked with higher mortality and multi-organ failure.[1,3-6]

Several pathophysiological mechanisms explain these associations. Elevated lactate generally reflects tissue hypoperfusion and anaerobic metabolism, but also impaired hepatic or renal clearance and catecholamine-driven glycolysis, all of which accompany severe critical illness. Standard base excess integrates metabolic contributions to acid–base balance, capturing non-respiratory acidosis due to accumulation of lactate, unmeasured anions, or loss of bicarbonate. In sepsis, cardiogenic shock, and multi-organ dysfunction, combined lactate elevation and negative BE denote more profound metabolic failure and worse prognosis.

This study has limitations that parallel those reported in real-world observational research. First, only admission measurements of lactate and SBE were analyzed; numerous studies emphasize that serial values and clearance within the first 24–48 hours have superior prognostic utility.[4] Second, as a single-center study with a relatively small sample size, generalizability is limited and the precision of AUC and OR estimates is modest.[1] Third, heterogeneity of underlying diagnoses may influence the relationship between acid–base variables and outcome; for example, in certain

post-cardiac surgery or trauma cohorts, BE has been reported to be as good as or superior to lactate for predicting mortality.[5] Fourth, residual confounding from unmeasured variables, such as pre-ICU resuscitation, timing of sample collection, and comorbidity burden, cannot be excluded. Despite these constraints, the findings support practical implications for clinicians, particularly in resource-constrained ICUs. Admission lactate should be prioritized as a **key** biomarker for early risk stratification, integrated with clinical assessment and severity scores.[5,6] Patients presenting with lactate ≥ 2 –2.5 mmol/L or markedly negative SBE warrant close hemodynamic monitoring, early identification of occult shock, and prompt resuscitative interventions, in line with sepsis and critical care guidelines. Standard base excess, while not independently predictive when lactate and APACHE II are known, remains useful for characterizing metabolic disturbances and may be especially valuable when lactate measurement is delayed or unavailable. Future research, using larger multicenter cohorts and serial measurements, could further clarify how best to combine lactate, BE, and dynamic acid–base parameters for individualized prognostication and resuscitation endpoints.

Conclusion

In this prospective cohort of 150 critically ill adults, admission serum lactate emerged as a stronger and independent predictor of ICU mortality compared with standard base excess, with superior discriminative performance (AUC 0.78 vs 0.69) and persistent association in multivariable analysis adjusted for illness severity. While both biomarkers correlated with adverse outcomes, standard base excess did not provide significant incremental prognostic value beyond lactate and APACHE II score, suggesting that lactate should be prioritized for early risk stratification in heterogeneous ICU populations. Nevertheless, standard base excess retains clinical utility for characterizing metabolic acid-base derangements, guiding resuscitation, and serving as a surrogate marker in settings where immediate lactate measurement is unavailable. Future multicenter studies incorporating serial measurements and lactate clearance kinetics are warranted to further refine the integration of these biomarkers into individualized prognostic algorithms and resuscitation endpoints.

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