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Metabolic Augmentation of mREMS: Improved 30-day Mortality Prediction through Lactate and Base Excess Integration in Emergency Patients

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Abstract

Aim: To evaluate whether adding base excess (BE), alone or in combination with lactate, to the modified Rapid Emergency Medicine score (mREMS) improves 30-day mortality prediction in emergency department (ED) red-zone patients.

Materials and Methods: This observational cohort study included eligible ED red-zone patients presenting between May 2018 and December 2019 (n=1798). Vital signs, Glasgow Coma scale (GCS) scores, and blood gas parameters were collected. Because the relationship between BE and 30-day mortality was non-linear, BE was categorized using a data-driven, mortality-associated classification approach. The resulting categories were weighted using effect sizes from multivariable logistic regression and converted into an ordinal variable (0-3 points), which was integrated into the mREMS framework. Three models were developed: mREMS-Be, mREMS-L, and mREMS-LBe. Model discrimination was evaluated using the area under the curve (AUC) and comparisons were performed using the DeLong test.

Results: Thirty-day mortality was associated with older age, lower systolic blood pressure, lower SpO_2, and lower GCS ($p<0.05$). BE was not predictive when modeled as a continuous variable; however, it demonstrated an independent association with mortality when modeled as an ordinal variable (p=0.022). The AUC of the baseline mREMS was 0.753; it increased to 0.767 with the addition of BE and to 0.782 when BE was combined with lactate. Integration of BE into mREMS resulted in a significant improvement in model discrimination in both the overall and the non-traumatic red-zone patient groups.

Conclusion: Incorporating BE into mREMS provides complementary prognostic value by improving 30-day mortality discrimination among high-acuity ED patients.

Keywords: mREMS, lactate, base excess, mortality prediction, emergency department, risk stratification

Introduction

Early recognition of clinical deterioration in emergency departments (EDs) is critically important for ensuring patient safety and for directing advanced therapeutic resources efficiently (1). Accordingly, Early Warning scores (EWS), which rely on physiological parameters, are widely used as complementary tools to support the rapid and systematic identification of high-risk patients (2). However, it is well established that systems based solely on vital signs may be limited, particularly during the early phases of metabolic derangement when clinical manifestations have not yet emerged (3). For this reason, efforts to integrate metabolic biomarkers into physiologic scoring systems have gained increasing importance with the aim of improving diagnostic accuracy (4).

In this context, blood gas lactate is a powerful metabolic biomarker reflecting tissue hypoperfusion and anaerobic metabolism, and it has been shown to be closely associated with mortality in sepsis, trauma, and various critical illnesses (4,5). Base excess (BE) in

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blood gas analysis, on the other hand, reflects the severity of metabolic acidosis and serves as a comprehensive indicator of metabolic stress, particularly in patients with comorbidities or active compensatory mechanisms (6-8).

Among EWS models, the modified Rapid Emergency Medicine score (mREMS) incorporates practical parameters such as age, vital signs and neurological status and is frequently used to predict mortality in the ED (9). Although the current literature does not definitively establish mREMS as the optimal score for ED populations, its component variables and predictive potential make it a useful model (9,10). While it was initially validated in trauma patients, efforts have been made to broaden its use to general ED cohorts (9-11). Recently, Donoso Calero et al. (12) demonstrated that integrating lactate into mREMS improved its predictive performance in a general ED patient population. Considering the prognostic value of BE, its representation of a metabolic dimension distinct from that of lactate, and the theoretical advantage of combining these biomarkers to achieve a more comprehensive risk assessment, BE emerges as an additional biomarker with potential for integration into EWS models such as mREMS (13-15).

This study evaluates BE-augmented versions of mREMS in a large cohort of red-zone patients from a tertiary ED using a three-level triage system (red-yellow-green) (16). Building on established evidence for lactate integration, the primary objective is to assess the incremental prognostic value of BE, alone and in combination with lactate, for 30-day mortality. Secondary objectives include comparison with the original mREMS and between BE-augmented models. The findings are expected to support earlier identification of high-risk ED patients, more effective use of limited healthcare resources, and inform the development of metabolically augmented mREMS variants that may be suitable for future integration into digital and automated clinical decision-support systems.

Materials and Methods

Study Design and Data Source

This observational cohort study evaluated the 30-day mortality risk among adult ED patients classified as red-zone cases. The study population consisted of eligible red-zone patients who presented to a tertiary training and research hospital in Turkey between 10 May 2018 and 12 December 2019 and for whom all predefined inclusion criteria were met and prospectively recorded study parameters were available. A previous publication from our group utilized only a subset of this dataset comprising 868 patients who met ESI 1-2 criteria; (17), however, the present study includes all eligible red-zone patients who fulfilled the predefined inclusion criteria and had complete data available for analysis (n=1798), and differs from the earlier work in terms of sampling frame, analytical strategy, and study objectives. Accordingly, the current study represents a retrospective secondary analysis of prospectively recorded data.

The study complied with the Declaration of Helsinki. Written informed consent had been obtained during the initial prospective data collection. For the current retrospective analysis of the entire cohort, additional approval was obtained from the Scientific Research Ethics Committee of the University of Health Sciences Türkiye, Trabzon Faculty of Medicine (decision no: 2024/11, date: 05.11.2024), and renewed consent was not required. All data were anonymized prior to analysis and handled in accordance with institutional data confidentiality policies.

Study Population

Patients aged ≥18 years who were classified as red-zone cases in the ED, who had complete vital signs recorded at initial presentation, who had available arterial or venous blood gas analyses were included. Completeness was defined as the availability of all variables required to calculate mREMS at initial presentation. Exclusion criteria were: presentation with cardiac arrest, pregnancy, missing mandatory parameters for mREMS calculation, and duplicate visits. The patient inclusion and exclusion steps, with corresponding numbers, are shown in Figure 1.

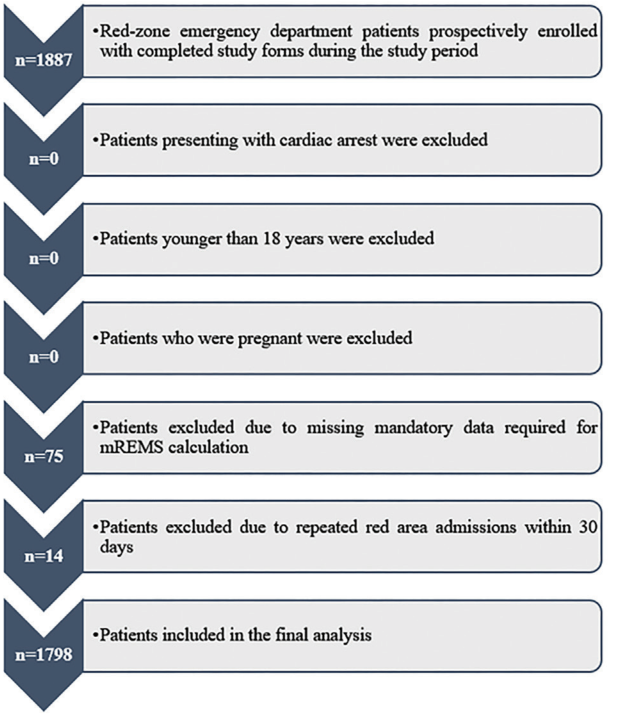


Figure 1. Flow Chart
mREMS: Modified Rapid Emergency Medicine score

Vital parameters were measured by triage nurses, and Glasgow Coma scale (GCS) was assessed by an emergency physician. Blood gas analyses were performed using a Radiometer ABL90 FLEX device integrated within the ED. Thirty-day mortality was verified through the national Death Notification System. Etiological classification (infectious or traumatic) was independently assessed by three emergency medicine specialists, and final categorization was established based on agreement between at least two specialists.

BE Classification and Scoring Method

Given that the association between BE and mortality has been shown in the literature and in logistic regression analyses of the present dataset to be non-linear, the inclusion of BE in the model as a continuous variable was considered inappropriate (8,18,19). Therefore, a data-driven approach was adopted to examine how the relationship between BE distribution and mortality risk varied across BE ranges.

Accordingly, BE values in the overall cohort were stratified into quintiles based on their distribution, and 30-day mortality rates were calculated for each quintile. This exploratory assessment indicated a non-linear risk distribution, with higher mortality observed at both markedly negative and markedly positive BE values. Adjacent quintiles exhibiting similar mortality rates were subsequently merged, resulting in four BE categories representing progressively increasing levels of risk.

In weighting the derived BE categories, reliance on crude mortality rates alone was avoided, and an approach accounting for the effects of potential confounders was employed. To this end, BE categories were entered into a multivariable logistic regression model together with established clinical variables, including age, vital signs, and GCS score. The adjusted odds coefficients obtained from this model were used to guide the assignment of ordinal risk points to the BE categories. Based on the observed risk gradient, BE categories were scored from 0 to 3 as follows:

- BE 2.2 to 4.6 mmol/L: 0 point
- BE ≥0.1 to <2.2 mmol/L or >4.6 mmol/L: 1 point
- BE ≥-3.2 to <0.1 mmol/L: 2 points
- BE <-3.2 mmol/L: 3 points

This approach ensured that BE classification and weighting were grounded in the risk structure observed within the study cohort and adjusted for relevant covariates, rather than relying on externally predefined thresholds. However, acknowledging that the prognostic signal of BE may be sensitive to the modeling approach, the maximum BE score was capped at 3 points to avoid disproportionate weighting that could obscure the prognostic contribution of lactate within the composite score.

Following score assignment, BE was treated as an ordinal variable and re-entered into multivariable logistic regression analysis. In this analysis, ordinal BE demonstrated a statistically significant association with 30-day mortality. This finding suggests that the derived BE categories exhibit internal consistency with respect to risk direction. Consequently, BE was integrated into mREMS and mREMS-based models as a complementary metabolic parameter reflecting a non-linear risk pattern.

Statistical Analysis

All primary statistical analyses were conducted using JAMOVI (v. 2.6.44). Distributional characteristics of continuous variables were assessed using the Shapiro-Wilk test in conjunction with histogram and Q-Q plot inspection. Normally distributed variables were summarized as mean ± standard deviation, whereas non-normally distributed variables were summarized as median (interquartile range). Categorical variables were summarized as counts and percentages. Between-group comparisons were performed using the Mann-Whitney U test for continuous variables and χ^2 test for categorical variables.

To identify independent predictors of 30-day mortality, two multivariable binomial logistic regression models were constructed. In the first model, BE was included as a categorical variable (BE_{0-3}) to estimate category-specific adjusted odds ratios (ORs) relative to the lowest-risk reference group. In the second model, BE was incorporated as an ordinal variable (0-3 points), reflecting a progressively increasing risk of mortality based on adjusted effect sizes observed in the categorical model. This sequential modeling strategy enabled the evaluation of both category-specific effects and monotonic risk behavior for BE, while minimizing assumptions about linearity.

For multivariable models, regression coefficients (β), OR, 95% confidence intervals (CIs), and p values were reported. ORs and corresponding 95% CIs were derived directly from regression coefficients [$OR = \exp(\beta)$, $95\% CI = \exp(\beta \pm 1.96 \times \text{standard error})$]. Model fit was assessed using the Akaike information criterion and McFadden's R^2.

Model discrimination was assessed using ROC curves and area under the curve (AUC) values. Optimal cut-off points for each score were determined using the Youden index to provide an objective balance between sensitivity and specificity. Differences in AUC among the four evaluated scores (mREMS, mREMSBe, mREMS-L, and mREMS-LBe) were analyzed using pairwise DeLong tests. A p value<0.05 was considered statistically significant for all analyses.

To address potential overfitting associated with data-driven BE categorization, internal validation of all mREMS-based models was performed using bootstrap resampling with 5.000 iterations. Calibration performance, particularly for BE-containing models, was further evaluated using decile-based calibration plots in the overall study population.

Results

A total of 1,798 red-zone ED patients were included in the study (Figure 1). The most frequent diagnoses in the cohort were pneumonia (23.0%), cerebrovascular disease (14.5%), and multiple trauma (13.7%) (Table 1). The overall population was predominantly elderly, and non-survivors were significantly older than survivors (76 vs. 67 years, $p<0.001$; Table 2). Although the sex distribution was comparable between groups, respiratory rate and heart rate were higher in the mortality group, whereas systolic blood pressure (SBP), mean arterial pressure (MAP), and SpO_2 were lower (all $p<0.001$). GCS was also significantly lower among non-survivors (Table 2).

Evaluation of blood gas parameters revealed pronounced metabolic impairment in patients who died. BE values were more negative, and although median lactate levels were identical between survivors and non-survivors, non-survivors demonstrated a wider interquartile range and higher upper lactate values (both $p<0.001$; Table 2).

Distribution of mREMS and its metabolically enhanced variants showed that all scores were significantly higher among non-survivors (all $p<0.001$; Table 2). Greater score separation between survivors and non-survivors was observed for mREMS-L and mREMS-LBe compared with the baseline mREMS.

In multivariable logistic regression analysis, age, SBP, SpO_2, and GCS were identified as independent predictors of 30-day mortality [all $p<0.05$; Table 3 (a-b)]. BE when entered as a continuous variable was not independently associated with 30-day mortality and therefore did not demonstrate a significant linear effect. However, when modeled as an ordinal variable, BE showed an independent association with mortality (OR per category increase: 1.200; $p=0.022$; Table 3b). Lactate remained a strong independent predictor across all models ($p<0.001$).

In the ROC analysis, the baseline mREMS model demonstrated discriminative performance, yielding an AUC of 0.753. Augmentation of mREMS with BE resulted in a modest improvement in discrimination (AUC=0.767), while integration

Table 1. Most frequent clinical diagnoses according to ICD classification

ICD	Count	% of total
Pneumonia	414	23.0%
Cerebrovascular disease	261	14.5%
Multiple trauma	237	13.7%
Acute abdomen	167	9.3%
Gastrointestinal bleeding	66	3.7%
Acute renal failure	57	3.2%
Complicated urinary tract infection	49	2.7%
Non-STEMI	48	2.7%
Sepsis	44	2.4%
Intoxication	40	2.2%
Pulmonary edema	33	1.8%
COPD with acute exacerbation	31	1.7%
STEMI	29	1.6%
Dyspnea	29	1.6%
Spontaneous pneumothorax	27	1.5%
Acute arterial occlusion	25	1.4%
Acute pancreatitis	25	1.4%
Arrhythmia	22	1.2%
Pulmonary embolism	20	1.1%
Other (<1%)	174	9.3%

ICD: International Statistical Classification of Diseases and Related Health Problems, Non-STEMI: Non-ST-segment elevation myocardial infarction, STEMI: ST-segment elevation myocardial infarction, COPD: Chronic obstructive pulmonary disease

Table 2. Baseline characteristics of the study population according to 30-day mortality				
Variable	**Overall cohort (n=1798)**	**Survivors (n=1560)**	**Non-survivors (n=238)**	**p value**
Demographics				
Age, years	73±20	67±20	76±18	**<0.001**
Male sex, n (%)	965 (53.6)	845 (54.2)	120 (50.4)	0.313
Vital signs				
Temperature, °C	37 (36-37)	37 (36-37)	37 (36-37)	0.441
Heart rate, bpm	86 (75-100)	86 (75-100)	90 (75-110)	**0.022**
Respiratory rate/min	18 (15-22)	18 (15-21)	20 (15-28)	**<0.001**
SpO_2, %	95 (90-98)	95 (92-98)	92 (85-96)	**<0.001**
SBP, mmHg	130 (110-150)	130 (110-150)	110 (90-134)	**<0.001**
MAP, mmHg	93 (80-106)	93 (83-106)	80 (70-96)	**<0.001**
Neurological				
GCS	15 (15-15)	15 (15-15)	14 (9-15)	**<0.001**
Blood gas parameters				
Base excess, mmol/L	1 (-2-4)	1 (-2-4)	-1 (-6-3)	**<0.001**
Lactate, mmol/L	2 (1-2)	2 (1-2)	2 (1-4)	**<0.001**
Clinical scores				
mREMS	4 (3-7)	4 (2-6)	7 (5-11)	**<0.001**
mREMS-Be	5 (3-7)	5 (3-7)	8 (5-12)	**<0.001**
mREMS-L	6 (4-9)	6 (4-8)	10 (7-15)	**<0.001**
mREMS-LBe	7 (5-9)	6 (4-9)	11 (7-15)	**<0.001**
Clinical course				
Intubation on arrival, n (%)	36 (2.0)	16 (1.0)	20 (8.4)	**<0.001**
Hospitalization, n (%)	1672 (93.0)	1421 (91.2)	235 (98.7)	**<0.001**
ICU admission, n (%)	489 (27.2)	344 (22.1)	145 (60.9)	**<0.001**
Etiology				
Infective, n (%)	757 (42.1)	651 (41.7)	106 (44.5)	0.455
Traumatic, n (%)	247 (13.7)	227 (14.6)	20 (8.4)	**0.014**

SBP: Systolic blood pressure, MAP: Mean arterial pressure, GCS: Glasgow Coma scale, mREMS: Modified Rapid Emergency Medicine score, mREMS-Be: Modified REMS with base excess, mREMS-L: Modified REMS with lactate, mREMS-LBe: Modified REMS with lactate and base excess, ICU: Intensive care unit

Table 3a. Multivariable binomial logistic regression analysis for 30-day mortality with categorical base excess							
Predictor	**Estimate**	**SE**	**Z**	**p**	**OR**	**95% CI**	
						Lower	**Upper**
Intercept	3.22766	1.21392	2.659	0.008	25.220	2.336	272.299
Age	0.02583	0.00518	4.987	<0.001	1.026	1.016	1.037
HR	-0.00505	0.00370	-1.365	0.172	0.995	0.988	1.002
RR	0.01402	0.01132	1.238	0.216	1.014	0.992	1.037
SpO_2	-0.03781	0.00974	-3.881	<0.001	0.963	0.945	0.981
SBP	-0.01297	0.00261	-4.973	<0.001	0.987	0.982	0.992
GCS	-0.17668	0.02276	-7.763	<0.001	0.838	0.801	0.876
BE-categoric							
BE_1-BE_0	0.09246	0.24780	0.373	0.709	1.097	0.675	1.783
BE_2-BE_0	0.42258	0.26623	1.587	0.112	1.526	0.906	2.571
BE_3-BE_0	0.47844	0.26375	1.814	0.070	1.614	0.962	2.706
Lactate	0.15577	0.03662	4.254	<0.001	1.169	1.088	1.256

BE: Base excess, SE: Standard error, OR: Odds ratio, CI: Confidence interval, SBP: Systolic blood pressure, HR: Heart rate, RR: Respiratory rate, GCS: Glasgow Coma scale

Table 3b. Multivariable binomial logistic regression analysis for 30-day mortality with ordinal base excess							
Predictor	**Estimate**	**SE**	**Z**	**p**	**OR**	**95% CI**	
						Lower	**Upper**
Intercept	3.17117	1.19578	2.65	0.008	23.835	2.288	248.356
Age	0.02576	0.00518	4.98	<0.001	1.026	1.016	1.037
HR	-0.00509	0.00370	-1.38	0.169	0.995	0.988	1.002
RR	0.01388	0.01132	1.23	0.220	1.014	0.992	1.037
SpO_2	-0.03760	0.00970	-3.88	<0.001	0.963	0.945	0.982
SBP	-0.01296	0.00261	-4.97	<0.001	0.987	0.982	0.992
GCS	-0.17584	0.02272	-7.74	<0.001	0.839	0.802	0.877
BE-ordinal	0.18193	0.07921	2.30	0.022	1.200	1.027	1.401
Lactate	0.15526	0.03627	4.28	<0.001	1.168	1.088	1.254

Two multivariable binomial logistic regression models evaluating the association between base excess (BE) and 30-day mortality are presented. The primary outcome was 30-day all-cause mortality, coded as a binary variable (1= death, 0= survival).

In the first model, BE was included as a categorical variable, derived using data-driven mortality-based thresholds, to estimate category-specific odds ratios. In the categorical model, BE was grouped into four mortality-BE_{0}-$_{3}$ derived from a data-driven threshold analysis. BE_0 represents the reference category associated with the lowest observed mortality risk. BE_1, BE_2, and BE_3 represent progressively higher-risk BE strata based on increasing 30-day mortality rates. These adjusted odds ratios were subsequently used to assign weighted risk scores to each BE category.

In the second model, BE was incorporated as an ordinal variable (0-3 points) reflecting increasing mortality risk based on the magnitude of odds ratios observed in the categorical model. For the ordinal BE variable, the reported odds ratio represents the change in the odds of 30-day mortality associated with a one-category increase in BE score. Mortality was coded as 1 (event) and 0 (non-event). Estimates (β), SEs, Wald Z statistics, ORs, and 95% CIs for ORs are reported. The model was estimated with n=1.798

OR: Odds ratio, CI: Confidence interval, SE: Standard error, HR: Heart rate (beats per minute), RR: Respiratory rate (breaths per minute), SBP: Systolic blood pressure (mmHg), GCS: Glasgow Coma scale, BE: (mmol/L), Lactate (mmol/L)

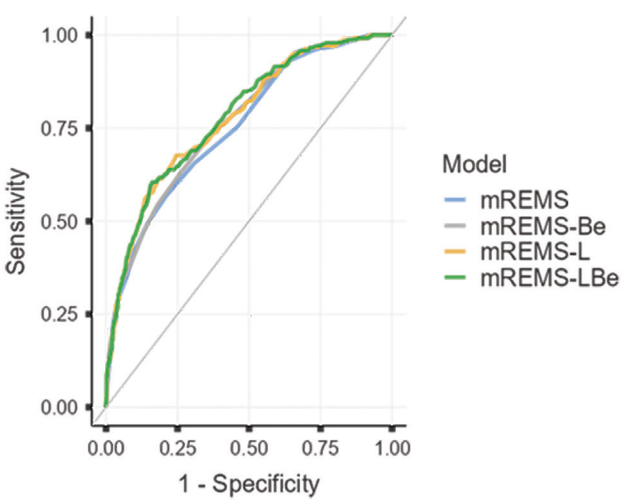


Figure 2a. Combined ROC Curves of mREMS and mREMS-based models in the overall cohort

mREMS: modified Rapid Emergency Medicine score, mREMS-Be: Modified REMS with base excess, mREMS-L: Modified REMS with lactate, mREMS-LBe: Modified REMS with lactate and base excess

of lactate led to a further increase in AUC to 0.778. The combined mREMS-LBe achieved an AUC of 0.782. Although mREMS-LBe yielded the numerically highest AUC, its performance was not statistically superior to that of mREMS-L (Table 4). Pairwise DeLong tests demonstrated that mREMS-Be, mREMS-L, and mREMS-LBe all performed significantly better than mREMS (p=0.006, 0.002, and 0.002, respectively). While mREMS-LBe also outperformed mREMS-Be (p=0.014), it did not show a statistically significant advantage over mREMS-L (p=0.228). ROC curves for the overall cohort are shown in Figure 2a.

Internal validation using bootstrap resampling demonstrated minimal optimism across all models. Optimism-corrected AUCs were 0.7663 for mREMS-Be and 0.7816 for mREMS-LBe, closely approximating apparent performance. Model accuracy assessed by the Brier score was 0.0976 for mREMS-Be and 0.0966 for mREMS-LBe. Decile-based calibration analyses demonstrated good agreement between predicted and observed 30-day mortality across risk strata in the overall cohort (Figure 3a-b; Table 5). In subgroup analyses, all models demonstrated high discriminative ability in trauma patients (AUCs: 0.873 for mREMS, 0.865 for mREMS-Be, 0.846 for mREMS-L, and 0.837 for mREMS-LBe), however, pairwise DeLong test comparisons revealed no statistically significant differences between the models (all p>0.05; Table 4). In the non-traumatic subgroup, integration of both BE and lactate yielded higher AUC values compared with baseline mREMS (p<0.05 for all relevant DeLong comparisons). Although mREMS-LBe showed the numerically highest AUC in this subgroup as well, it did not show a significant advantage over mREMS-L. ROC curves for the traumatic and non-traumatic subgroups are presented in Figures 2b and 2c, respectively.

Overall, metabolically augmented mREMS models demonstrated improved discrimination in predicting 30-day mortality compared with the baseline score.

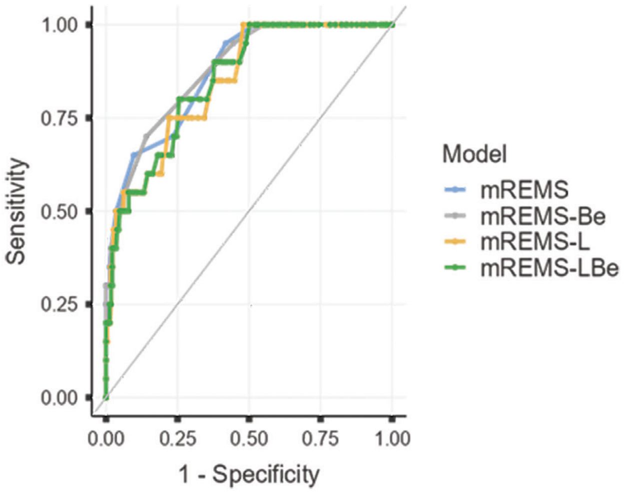


Figure 2b. Combined ROC curves of mREMS and mREMS-based models in the traumatic subgroup

mREMS: modified Rapid Emergency Medicine score, mREMS-Be: Modified REMS with base excess, mREMS-L: Modified REMS with lactate, mREMS-LBe: Modified REMS with lactate and base excess

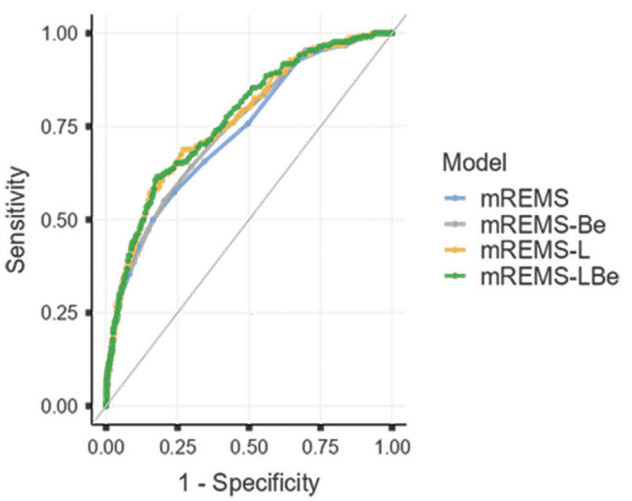
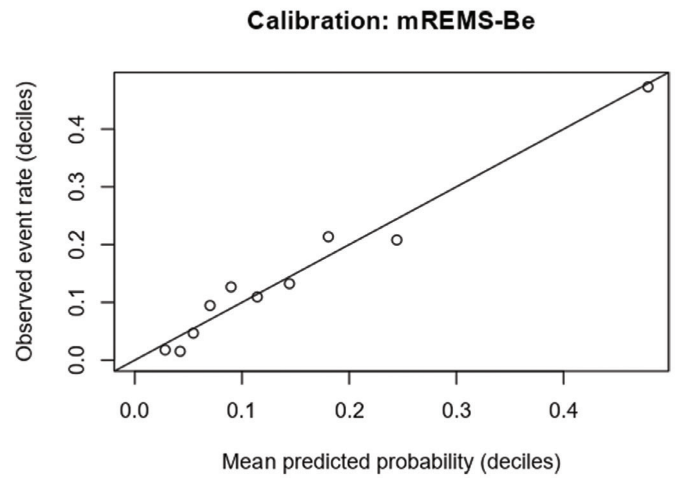


Figure 2c. Combined ROC curves of mREMS and mREMS-based models in the non-traumatic subgroup

mREMS: modified Rapid Emergency Medicine score, mREMS-Be: Modified REMS with base excess, mREMS-L: Modified REMS with lactate, mREMS-LBe: Modified REMS with lactate and base excess



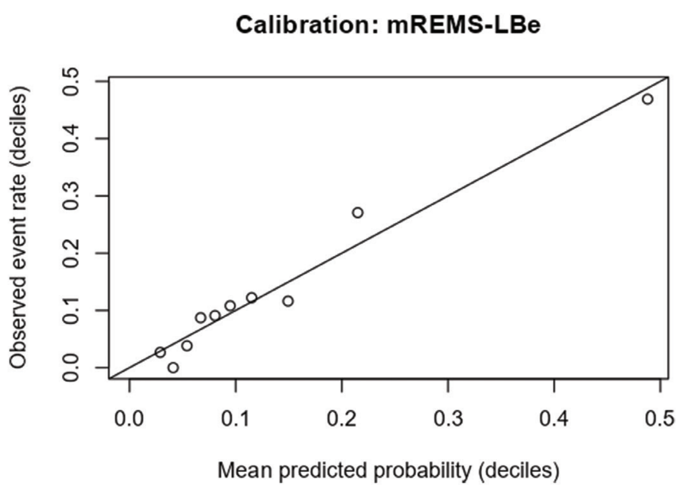


Figure 3 (a-b). Decile-based calibration plots of the mREMS-Be and mREMS-LBe models in the overall cohort

Decile-based calibration plots for the mREMS-Be and mREMS-LBe models in the overall cohort following bootstrap internal validation (B=5.000). Predicted 30-day mortality risks are plotted against observed event rates within deciles of predicted risk. The dashed diagonal line represents ideal calibration.

mREMS: modified Rapid Emergency Medicine score, mREMS-Be: Modified REMS with base excess, mREMS-L: Modified REMS with lactate, mREMS-LBe: Modified REMS with lactate and base excess

Table 4. ROC analysis with DeLong test comparisons of mREMS and mREMS-based models in the overall cohort and clinical subgroups

	Model	AUC	95% CI	Cut-off	Sensitivity	Specificity	Model comparisons with DeLong test			
							vs	mREMS-Be	mREMS-L	mREMS-LBe
Overall cohort	mREMS	0.753	0.720-0.786	**≥7.0**	56.7%	79.0%	mREMS	**0.006**[*]	**0.002**[*]	**0.002**[*]
	mREMS-Be	0.767	0.735-0.799	**≥7.0**	63.4%	73.5%	mREMS-Be	-	0.124[*]	**0.014**[*]
	mREMS-L	0.778	0.746-0.810	**≥9.0**	61.3%	81.9%	mREMS-L	-	-	0.228[*]
	mREMS-LBe	0.782	0.750-0.813	**≥9.9**	60.5%	83.9%	mREMS-LBe	-	-	-
Traumatic subgroup	mREMS	0.873	0.800-0.945	**≥6.0**	65.0%	90.3%	mREMS	0.683[*]	0.083[*]	0.233[*]
	mREMS-Be	0.865	0.790-0.940	**≥6.0**	70.0%	85.9%	mREMS-Be	-	0.140[*]	0.054[*]
	mREMS-L	0.846	0.765-0.928	**≥6.8**	75.0%	78.0%	mREMS-L	-	-	0.777[*]
	mREMS-LBe	0.837	0.752-0.923	**≥6.8**	80.0%	74.4%	mREMS-LBe	-	-	-
Non-traumatic subgroup	mREMS	0.733	0.697-0.770	**≥8.0**	50.0%	83.6%	mREMS	**0.007**[*]	**<0.001**[*]	**<0.001**[*]
	mREMS-Be	0.750	0.715-0.785	**≥8.0**	55.0%	79.7%	mREMS-Be	-	**0.028**[*]	**0.002**[*]
	mREMS-L	0.766	0.731-0.800	**≥9.0**	62.4%	79.8%	mREMS-L	-	-	0.236[*]
	mREMS-LBe	0.772	0.738-0.806	**≥9.9**	61.5%	82.2%	mREMS-LBe	-	-	-

[*]DeLong p values represent pairwise comparisons of ROC AUCs between the model listed in each row and the corresponding model listed in the column header. P values are reported in the intersecting cells. Optimal cut-off values were determined using the Youden index

AUC: Area under the curve, mREMS: Modified Rapid Emergency Medicine Score, mREMS-Be: Modified REMS with base excess, mREMS-L: Modified REMS with lactate, mREMS-LBe: Modified REMS with lactate and base excess

Table 5. Apparent and optimism-corrected performance metrics of mREMS and BE-augmented mREMS models

	Model	N	Events	AUC (apparent)	AUC (optimism-corrected)	Calibration intercept (corrected)	Calibration slope (corrected)	Brier (apparent)	Brier (corrected)
Overall cohort	mREMS	1798	238	0.7530	0.7530	0.0102	1.004	0.0989	0.0993
	mREMS-Be	1798	238	0.7669	0.7671	0.0102	1.003	0.0976	0.0980
	mREMS-L	1798	238	0.7779	0.7783	0.0088	1.004	0.0969	0.0971
	mREMS-LBe	1798	238	0.7819	0.7819	0.0052	1.002	0.0966	0.0969
Traumatic subgroup	mREMS	247	20	0.8728	0.8731	-0.0120	0.9804	0.0510	0.0529
	mREMS-Be	247	20	0.8649	0.8648	0.0042	0.9871	0.0522	0.0541
	mREMS-L	247	20	0.8461	0.8459	0.0115	0.9895	0.0579	0.0604
	mREMS-LBe	247	20	0.8371	0.8377	0.0119	0.9956	0.0576	0.0595
Non-traumatic subgroup	mREMS	1551	218	0.7335	0.7340	0.0123	1.006	0.1063	0.1066
	mREMS-Be	1551	218	0.7499	0.7501	0.0081	1.004	0.1047	0.1051
	mREMS-L	1551	218	0.7658	0.7655	0.0066	1.001	0.1030	0.1036
	mREMS-LBe	1551	218	0.7716	0.7715	0.0042	1.002	0.1027	0.1030

The Apparent AUC and Brier score represent model performance in the original sample. Optimism-corrected values were obtained using bootstrap internal validation with 5.000 resamples. Calibration intercept and slope values closer to 0 and 1, respectively, indicate better calibration.

AUC: Area under the curve, mREMS, modified Rapid Emergency Medicine score, mREMS-Be, mREMS augmented with base excess, mREMS-L, mREMS augmented with lactate, mREMS-LBe, mREMS augmented with lactate and base excess, N, number of patients, Events, number of deaths within 30 days; CI: Confidence interval, mREMS-Be: Modified REMS with base excess, mREMS-L: Modified REMS with lactate, mREMS-LBe: Modified REMS with lactate and base excess, BE: Base excess

Discussion

This study demonstrates that integrating mREMS-Be significantly improves 30-day mortality prediction among critically ill ED patients. The mREMS model enriched with lactate and BE provides superior discrimination compared with mREMS-Be and demonstrates predictive performance comparable to that of the previously validated mREMS-L model in critically ill ED patients not limited to trauma.

Integration of mREMS-L is not a novel concept. A recent multicenter study by Donoso Calero et al. (12) in a mixed ED population showed that mREMS-L significantly improved the performance of the original mREMS. Our results were consistent with those reported in this study. These findings suggest that mREMS, which has demonstrated strong prognostic capability primarily in trauma populations, may also be applicable to non-trauma ED populations when combined with lactate (9,10,12).

In non-traumatic etiologies, clinical deterioration may progress more insidiously and vital signs may lag behind early metabolic impairment; consequently, classical EWS models such as mREMS may be less sensitive in the early recognition of clinical deterioration (20-22). In this context, lactate, as a marker of metabolic stress and early hypoperfusion (4,23) may help mitigate this limitation. As another clinically informative component of blood gas analysis reflecting metabolic reserve, BE has clear theoretical relevance; however, to our knowledge, its integration into an EWS has not been widely evaluated (7,8,18,19,24). Although mREMS does not represent all EWSs, incorporating BE into mREMS allowed us to explore this question within a pragmatic and commonly used framework. Given lactate's established prognostic capacity and BE's ability to reflect metabolic reserve, the combined use of these biomarkers may offer complementary clinical information.

On the other hand, the question of whether BE or lactate better predicts clinical or metabolic deterioration in critically ill patients remains unresolved (25-28). Caputo et al. (26) reported that lactate and BE were equivalent in predicting shock among trauma patients with normal vital signs, whereas other studies have shown context-dependent differences, with lactate or BE demonstrating relative advantages in specific populations (28,29). Several investigations further suggest that combining lactate and BE may improve the prediction of clinical deterioration compared with either biomarker alone (8,23). Accordingly, lactate and BE were incorporated into the mREMS framework both standalone and in combination.

Our results show that BE, consistent with the literature, did not exhibit a linear association with mortality, but became prognostically informative when modeled as an ordinal variable (4,19,24). This pattern may reflect threshold-dependent depletion of metabolic reserve rather than a uniform linear effect. Accordingly, ordinally scored BE, both alone and combined with lactate, improved prognostic accuracy when integrated into mREMS in ED patients, not limited to trauma. These findings are also consistent with evidence from previous systematic reviews and meta-analyses demonstrating that the integration of metabolic biomarkers into physiology-based scoring systems enhances the prediction of clinical deterioration in critically ill patients, not limited to trauma (20,21,30). In the overall cohort and the non-traumatic subgroup, mREMS-LBe demonstrated superior discriminative performance compared with those of the original mREMS and mREMS-Be. The lack of a significant performance advantage over mREMS-L suggests that the observed improvement is largely attributable to the dominant prognostic contribution of lactate (18,23). In this context, BE appears to function as a complementary metabolic biomarker rather than as an independent determinant of prognostic discrimination, and integration of BE and lactate may represent an option for enhanced risk stratification in general ED populations, pending external validation.

In the trauma cohort, incorporation of BE, lactate, or both biomarkers did not yield a statistically significant improvement in the discriminative performance of mREMS. This finding may reflect limited statistical power due to the relatively small trauma sample size. Nonetheless, prior studies have demonstrated that BE performs comparably to lactate and may be superior in specific clinical contexts for predicting mortality and adverse outcomes in trauma populations (29,31,32). Given the established prognostic utility of mREMS in trauma patients, further evaluation of BE integration in larger, adequately powered trauma cohorts is warranted as part of future model refinement efforts.

In non-trauma populations, relatively high mortality rates and the limited ability of conventional vital sign–based parameters to reliably detect early physiological deterioration highlight the increasing importance of integrating metabolically sensitive biomarkers, such as lactate and BE, into physiology-based EWS (20-22,33). However, in the context of the present study, translating this physiological rationale into a statistically robust and clinically applicable modeling strategy required several methodological assumptions, including data-driven categorization of BE. Although this approach was intended to account for the non-linear association between BE and 30-day mortality, the derivation of cohort-specific thresholds may introduce optimism bias and limit model transportability to other ED populations. While bootstrap-based internal validation and calibration analyses demonstrated minimal optimism

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and acceptable internal stability, these methods do not ensure external validity. Therefore, the performance of BE-augmented mREMS models should be interpreted with caution, and multicenter external validation is warranted before broader clinical application.

Study Limitations

Limitations of this study include the relatively small trauma subgroup, which prevented adequate statistical power; the possibility that arterial-venous differences in blood gas sampling introduced additional variability in BE; and the single-center retrospective design, which may limit generalizability. Thus, validation of the integration of metabolic biomarkers into mREMS requires multicenter, prospective studies.

Conclusion

This study demonstrates that integration of BE into mREMS, whether alone or combined with lactate, is associated with improved discrimination in predicting 30-day mortality among high-acuity ED patients, with BE serving as a complementary metabolic parameter rather than supplanting the established prognostic role of lactate. Following external validation, BE-augmented mREMS models may be considered for integration into automated clinical decision-support systems in acute care settings.

Ethics

Ethics Committee Approval: The study protocol was approved by the Scientific Research Ethics Committee of the University of Health Sciences Türkiye, Trabzon Faculty of Medicine (decision no: 2024/11, date: 05.11.2024).

Informed Consent: Written informed consent had been obtained during the initial prospective data collection.

Footnotes

Authorship Contributions

Surgical and Medical Practices: G.M., Concept: G.M., Design: G.M., Data Collection or Processing: G.M., S.G., R.Ö., A.K., Analysis or Interpretation: G.M., S.G., Literature Search: G.M., R.Ö., A.K., Writing: G.M., S.G., R.Ö., A.K.

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