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ORIGINAL ARTICLE

Identification value of a multi-indicator model for lactic acidosis in acute carbon monoxide poisoning: a retrospective cross-sectional study

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ABSTRACT

Objective: To investigate the association between clinical indicators at admission and lactic acidosis in patients with acute carbon monoxide (CO) poisoning, and to evaluate the diagnostic performance of a combined multi-indicator model. **Methods:** This single-center retrospective cross-sectional study included 124 adult patients with acute CO poisoning admitted between December 2021 and December 2024. Patients were classified into a lactic acidosis group (blood lactate ≥4.0 mmol/L, *n*=43) and a non-lactic acidosis group (blood lactate <4.0 mmol/L, *n*=81). Variable selection was performed using least absolute shrinkage and selection operator (LASSO) logistic regression with 10-fold cross-validation, followed by multivariable logistic regression to identify factors independently associated with lactic acidosis. A combined prediction model was developed and compared with a baseline model using receiver operating characteristic (ROC) curve analysis, the DeLong test, net reclassification improvement (NRI), integrated discrimination improvement (IDI), and decision curve analysis (DCA). **Results:** Lactic acidosis was observed in 34.7% of patients. LASSO regression identified eight candidate variables, of which respiratory rate (OR=1.25, 95% CI: 1.10–1.41; *P*<0.001), white blood cell count (OR=1.34, 95% CI: 1.18–1.53; *P*<0.001), and carboxyhemoglobin level (OR=1.20, 95% CI: 1.11–1.29; *P*<0.001) were independently associated with lactic acidosis. The combined model demonstrated excellent discriminatory performance, with an AUC of 0.939 (95% CI: 0.894–0.984), significantly outperforming the baseline model (AUC=0.802; *P*=0.0005). The categorical NRI was 0.5165 (*P*<0.001), the continuous NRI was 1.1892 (*P*<0.001), and the IDI was 0.3229 (*P*<0.001). Decision curve analysis showed a consistent positive net clinical benefit across threshold probabilities ranging from 0.1 to 0.9. **Conclusion:** Respiratory rate, white blood cell count, and carboxyhemoglobin level are independently associated with lactic acidosis in patients with acute CO poisoning. The combined model demonstrated excellent diagnostic performance and clinical utility, supporting its potential use for early risk stratification in emergency settings.

Keywords: Acute carbon monoxide poisoning, Lactic acidosis, Independent associated factors, Multi-parameter screening, Diagnostic value.

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1. INTRODUCTION

Acute carbon monoxide (CO) poisoning is the most common fatal toxic disease worldwide and a frequent critical emergency in emergency medicine. Severe cases may lead to multiple organ dysfunction, coma, or even death. Accurate assessment of disease severity on admission is essential for optimizing diagnosis and treatment [1-3]. CO has more than 200-fold higher affinity for hemoglobin than oxygen. Carboxyhemoglobin (COHb) not only drastically reduces blood oxygen-carrying capacity but also penetrates

cell membranes, inhibits key enzymes in the mitochondrial respiratory chain, blocks aerobic metabolism, and causes systemic tissue hypoxia [4,5].

Lactic acidosis is a core metabolic marker of tissue hypoperfusion and hypoxic injury in critically ill patients and is widely used for severity evaluation and risk stratification [6,7]. It is also the most specific metabolic complication of tissue hypoxia following CO poisoning, serving as a key objective indicator for evaluating hypoxia severity and poisoning grading [8,9]. Previous studies confirm that CO-poisoned patients with elevated lactate levels have higher requirements for hyperbaric oxygen therapy, higher rates of short-term adverse events, and greater risk of long-term delayed neuropsychiatric syndrome. CO poisoning-related lactic acidosis can also trigger life-threatening acute events [10]. Identifying routine admission indicators associated with lactic acidosis is therefore clinically important for optimizing emergency management.

Current studies on acute CO poisoning with lactic acidosis mostly focus on the association between lactate and prognosis, with large variations in reported incidence. Systematic analyses of the correlation between immediately available routine admission indicators and lactic acidosis in emergency patients remain limited [11,12]. Clinically, COHb reflects CO exposure burden but does not fully correspond to tissue hypoxia or metabolic disturbance; COHb alone cannot reliably identify lactic acidosis [3,13]. Respiratory rate and peripheral white blood cell count are readily obtainable in initial emergency screening. Clarifying their independent association with lactic acidosis and establishing a combined identification model may improve recognition of this metabolic disorder and streamline early emergency evaluation.

Against this background, this retrospective cross-sectional study analyzed clinical data of adult emergency patients with acute CO poisoning to identify admission baseline indicators independently associated with lactic acidosis and to evaluate the identification value of a multi-indicator combination for lactic acidosis. The aim is to provide a reference for emergency risk stratification and support early clinical decision-making.

2. OBJECTS AND METHODS

Study Population

This was a single-center retrospective cross-sectional study that consecutively enrolled adult patients with acute carbon monoxide (CO) poisoning admitted to the emergency department from December 2021 to December 2024.Inclusion criteria: age ≥ 18 years; confirmed acute CO poisoning with definite exposure history, clinical manifestations, arterial blood gas analysis, and carboxyhemoglobin (COHb) test; complete baseline data; no pre-hospital interventions (hyperbaric oxygen, fluid infusion, or acidosis correction) before admission. Exclusion criteria: other causes of lactic acidosis (severe trauma, septic shock, diabetic ketoacidosis); advanced malignancy or end-stage organ diseases; missing key data; cardiopulmonary resuscitation on arrival. The study was approved by the Ethics Committee of our hospital (No. Bo Yi Lun Shen 2025 No. 263). All data were anonymized, and informed consent was waived due to the retrospective observational design.

Data Collection

Clinical data from the first assessment upon admission, prior to any targeted in-hospital intervention, were collected via the hospital's electronic medical record and laboratory information systems. The collected information included demographic characteristics, admission vital signs, past medical history, laboratory test results, and the Glasgow Coma Scale (GCS) score.

Grouping Criteria

According to conventional diagnostic criteria for lactic acidosis [6,14], patients were divided into two groups based on their initial arterial blood lactate level upon admission: the lactic acidosis group (blood lactate ≥ 4.0 mmol/L) and the non-acidosis group (blood lactate < 4.0 mmol/L).

Statistical Methods

Statistical analyses were performed using R 4.4.2. The sample size met the 10 events per variable (EPV) rule for logistic regression. Normally distributed continuous data were presented as mean ± standard deviation and compared using an independent-samples t-test. Non-normally distributed data were presented as median (interquartile range) and compared using the Mann–Whitney U-test. Categorical data were presented as number (percentage) and compared using χ^2 or Fisher's exact test for baseline description only. Candidate variables were screened by LASSO binary logistic regression with 10-fold cross-validation (lambda.1se as the optimal parameter). After excluding multicollinearity (variance inflation factor < 5), multivariate binary logistic regression was performed to determine factors independently associated with lactic acidosis, reported as odds ratio (OR) with 95% confidence interval (CI).A combined indicator model was developed and compared with a baseline model using receiver operating characteristic (ROC) analysis,

DeLong test, net reclassification improvement (NRI), integrated discrimination improvement (IDI), and decision curve analysis (DCA). A two-sided P < 0.05 was considered statistically significant.

3. RESULTS

Comparison of Baseline Characteristics

A total of 124 patients with acute carbon monoxide poisoning were enrolled in this study, including 43 patients in the lactic acidosis group (incidence rate: 34.7%) and 81 patients in the non-lactic acidosis group (65.3%). A comparison of baseline characteristics revealed that the lactic acidosis group had significantly higher respiratory rate, heart rate, white blood cell count, creatinine, alanine aminotransferase (ALT), and carboxyhemoglobin (COHb) levels, as well as significantly lower Glasgow Coma Scale (GCS) scores and prevalence of history of hypertension, compared with the non-lactic acidosis group (all P < 0.05).There were no statistically significant differences in other baseline indicators, including age, sex, body mass index (BMI), body temperature, systolic blood pressure, urea, total bilirubin, and history of diabetes mellitus between the two groups (all P > 0.05).Detailed core baseline data are presented in Table 1.Notably, a history of hypertension was significantly more prevalent in the non-lactic acidosis group (35.80% vs. 13.95%, P=0.010). This seemingly paradoxical finding may be attributed to several factors: (1) hypertensive patients may have heightened health awareness, leading to earlier medical attention and shorter CO exposure durations; (2) antihypertensive medications, particularly calcium channel blockers and ACE inhibitors, may confer partial mitochondrial protection against hypoxic injury; (3) survival bias, as hypertensive patients with severe CO poisoning may have experienced higher pre-hospital mortality, resulting in a selected survivor population in the non-acidosis group. Importantly, this association lost significance in multivariate analysis (P>0.05), indicating it was confounded by other variables such as age and CO exposure severity.

Table 1. Comparison of Baseline Characteristics Between the Two Groups.

Indicator	Total Population (n=124)	Non-Lactic Acidosis Group (n=81)	Lactic Acidosis Group (n=43)	Statistic	P value
Age [M (Q_1, Q_3), years]	56.00 (46.75, 70.00)	56.00 (50.00, 67.00)	57.00 (42.50, 74.00)	Z=-0.26	0.797
Male [n (%)]	65 (52.42)	46 (56.79)	19 (44.19)	χ^2=1.79	0.181
Respiratory Rate [M (Q_1, Q_3), /min]	18.00 (16.00, 20.00)	18.00 (15.00, 20.00)	20.00 (17.00, 22.50)	Z=-3.09	0.002
Heart Rate [M (Q_1, Q_3), /min]	87.50 (78.00, 100.50)	82.00 (76.00, 96.00)	99.00 (87.00, 113.50)	Z=-4.00	<.001
White Blood Cell Count [M (Q_1, Q_3), $\times10^9$/L]	11.29 (8.85, 15.91)	10.04 (8.09, 13.50)	16.43 (11.48, 22.55)	Z=-4.58	<.001
Creatinine [M (Q_1, Q_3), μmol/L]	59.50 (47.98, 76.33)	55.50 (46.80, 67.90)	70.80 (57.20, 101.15)	Z=-3.30	<.001
ALT [M (Q_1, Q_3), U/L]	25.50 (18.00, 38.25)	24.00 (18.00, 30.00)	37.00 (20.00, 48.00)	Z=-2.55	0.011
COHb [M (Q_1, Q_3), %]	25.50 (14.02, 33.75)	19.40 (12.80, 30.00)	33.10 (24.90, 42.85)	Z=-4.80	<.001
History of Hypertension (Yes) [n (%)]	35 (28.23)	29 (35.80)	6 (13.95)	χ^2=6.62	0.010
History of Diabetes (Yes) [n (%)]	12 (9.68)	8 (9.88)	4 (9.30)	χ^2=0.00	1.000
GCS Score [M (Q_1, Q_3), points]	12.00 (8.00, 15.00)	13.00 (10.00, 15.00)	8.00 (6.50, 12.00)	Z=-4.81	<.001

Note: M, median; Q_1, Q_3, quartiles; Z, Mann-Whitney U test statistic; χ^2, chi-square test statistic; ALT, alanine aminotransferase; COHb, carboxyhemoglobin; GCS, Glasgow Coma Scale.

Variable Selection and Results of Multivariate Regression Analysis

All baseline candidate variables were entered into LASSO binary logistic regression. With 10-fold cross-validation, the optimal regularization parameter λ was determined as 0.0495 by the lambda.1se criterion, and 8 core variables with non-zero coefficients were screened: respiratory rate, heart rate, white blood cell count, lymphocyte count, albumin, GCS score, COHb, and hypertension history. The coefficient path and binomial deviance curve are displayed in Figure 1 and Figure 2.

After excluding multicollinearity, these 8 variables were included in multivariate binary logistic regression. After adjustment for confounders, respiratory rate, white blood cell count, and COHb on admission were independently and positively associated with lactic acidosis in acute CO poisoning patients (P < 0.05). Heart rate, lymphocyte count, albumin, GCS score, and hypertension showed no significant association (P > 0.05) and were excluded from the final model. Details are shown in Table 2.

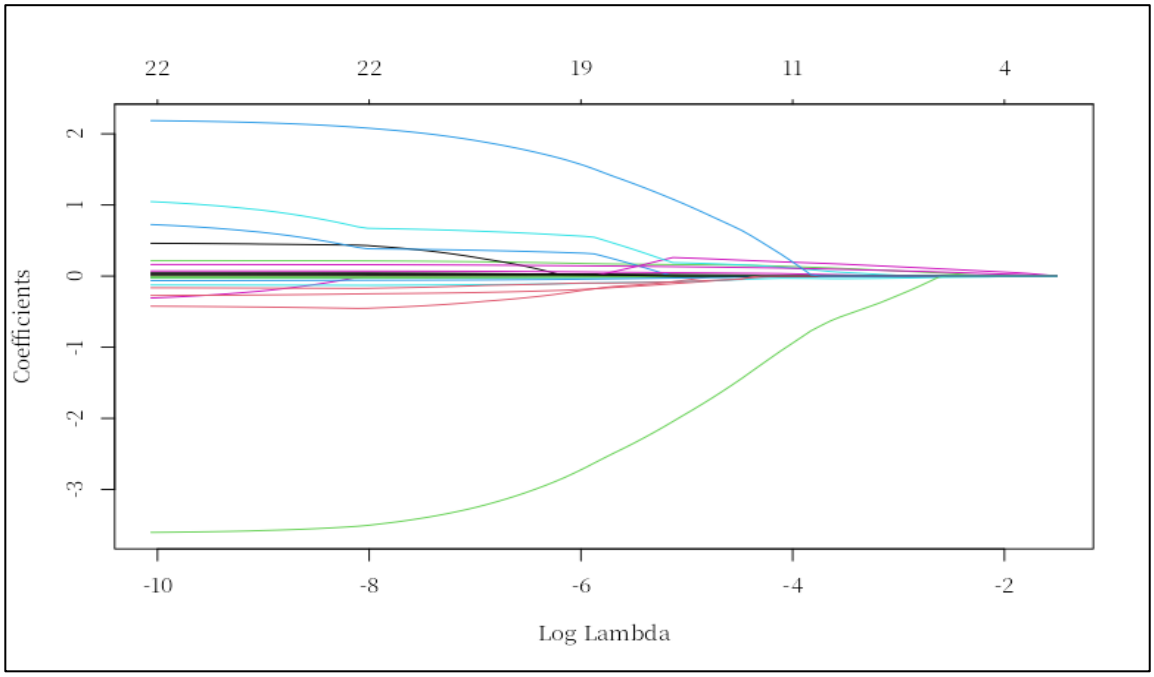

Figure 1. Trajectory of variable coefficients in the LASSO regression as a function of Log(λ).

Note: The horizontal axis shows Log(λ), and the vertical axis shows regression coefficients of variables. Each curve represents the coefficient path of one variable. The number at the top indicates the number of variables with non-zero coefficients retained at the corresponding λ value.

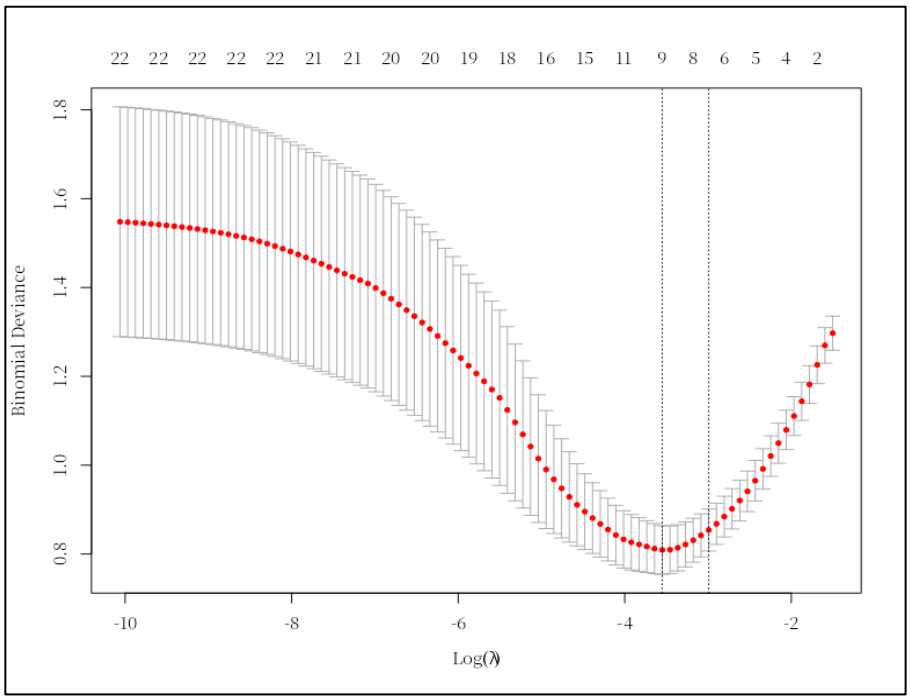

Figure 2. Curve of binomial deviance in the LASSO regression as a function of Log(λ).

Note: The horizontal axis represents the Log(λ) value, and the vertical axis represents the binomial deviance. The vertical dashed lines correspond to the positions of lambda.min and lambda.1se, respectively. In this study, variables were selected based on the lambda 1se criterion.

Table 2. Results of Multivariate Binary Logistic Regression Analysis.

Variable	Regression Coefficient	Standard Error	Wald Z	P value	OR (95% CI)
Intercept	-13.30	2.58	-5.16	<0.001	0.00 (0.00 ~ 0.00)
Respiratory Rate (/min)	0.22	0.06	3.46	<0.001	1.25 (1.10 ~ 1.41)
White Blood Cell Count (×10^9/L)	0.30	0.07	4.44	<0.001	1.34 (1.18 ~ 1.53)
COHb (%)	0.18	0.04	4.66	<0.001	1.20 (1.11 ~ 1.29)

Note: OR, odds ratio; 95% CI, 95% confidence interval; COHb, carboxyhemoglobin.

Model Performance Comparison

The final combined model (Model 2) is expressed by the following logistic regression equation: logit(P) = -13.30 + 0.22 × (Respiratory Rate, /min) + 0.30 × (White Blood Cell Count, ×10^9/L) + 0.18 × (COHb, %). For clinical application, the linear predictor (LP) score can be computed as LP = 0.22 × RR + 0.30 × WBC + 0.18 × COHb. Based on multivariate regression results, a combined multi-indicator model (Model 2) was established using respiratory rate, white blood cell count, and COHb. A baseline model (Model 1) including age, sex, comorbidities, and COHb was used for comparison. Model 2 achieved an AUC of 0.939 (95% CI: 0.894–0.984), significantly higher than Model 1 (AUC = 0.802, 95% CI: 0.721–0.884; DeLong test: P = 0.0005). Model 2 also showed better accuracy, sensitivity, specificity, and a lower Brier score, reflecting stronger discriminative and calibration performance (Table 3; Figure 3).Reclassification analyses demonstrated that Model 2 achieved a categorical NRI of 0.5165 (95% CI: 0.2932–0.7398, P < 0.001), continuous NRI of 1.1892 (95% CI: 0.8877–1.4907, P < 0.001), and an IDI of 0.3229 (95% CI: 0.2306–0.4152, P < 0.001), indicating significantly improved classification performance.

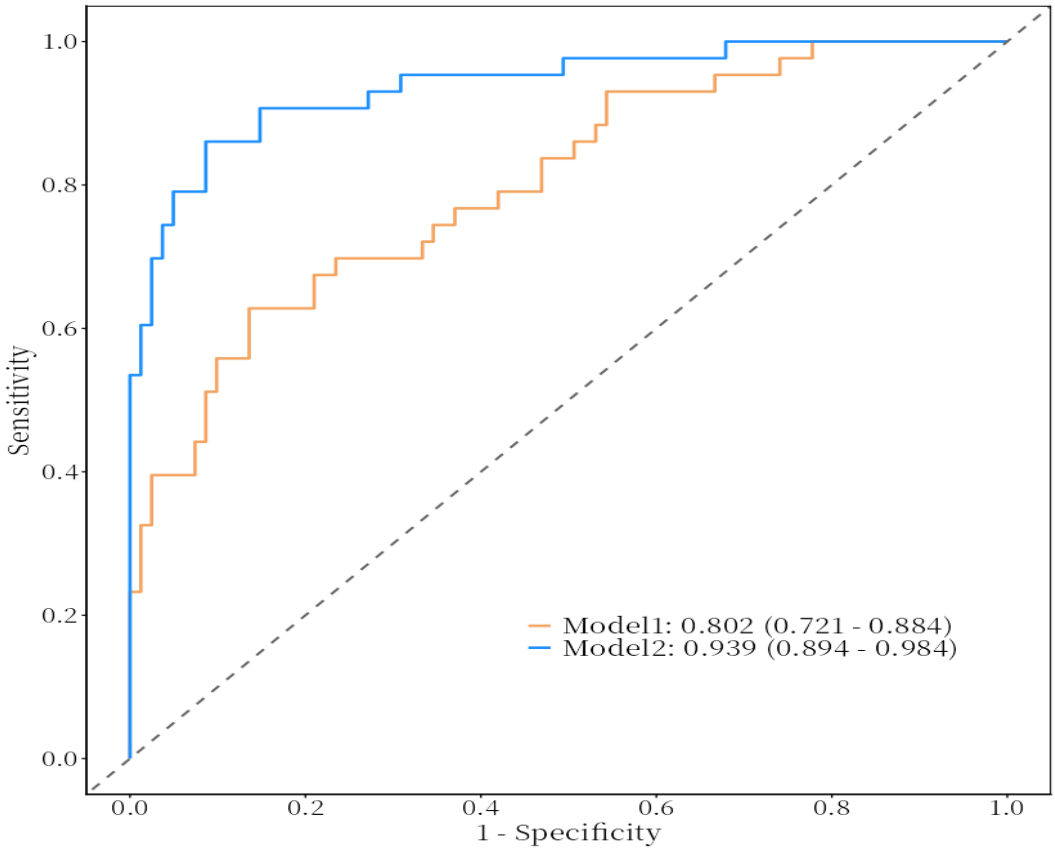

Figure 3. ROC Curves of the Two Models for Identifying Lactic Acidosis in Patients with Acute CO Poisoning.

Note: The orange curve represents the baseline model (Model 1), the blue curve represents the multi-indicator combined model (Model 2), and the grey dashed line is the reference line.

Table 3. Performance Comparison of the Two Models for Identifying Lactic Acidosis in Acute CO Poisoning Patients.

Model	AUC (95% CI)	Accuracy (95% CI)	Sensitivity (95% CI)	Specificity (95% CI)	Positive Predictive Value (95% CI)	Negative Predictive Value (95% CI)	F1 Score	Brier Score	Optimal Cut-off
Model 1	0.802 (0.721–0.884)	0.78 (0.70–0.85)	0.63 (0.48–0.77)	0.86 (0.79–0.94)	0.71 (0.57–0.85)	0.81 (0.73–0.90)	0.67	0.16	0.4698
Model 2	0.939 (0.894–0.984)	0.90 (0.83–0.94)	0.86 (0.76–0.96)	0.91 (0.85–0.97)	0.84 (0.73–0.95)	0.93 (0.87–0.98)	0.85	0.09	0.4079

Evaluation of Clinical Net Benefit

Decision curve analysis (DCA) results demonstrated that across the clinically reasonable threshold probability range of 0.1 to 0.9, the net benefit of the multi-indicator combined model (Model 2) was consistently higher than that of the baseline model (Model 1). Furthermore, Model 2's net benefit remained above the two reference lines representing the "treat-all" and "treat-none" strategies throughout this range, with no interval of negative net benefit. In contrast, Model 1 maintained a positive net benefit only within a narrower threshold range of 0.2 to 0.7, and the magnitude of its net benefit was significantly lower than that of Model 2. Details are shown in Figure 4.

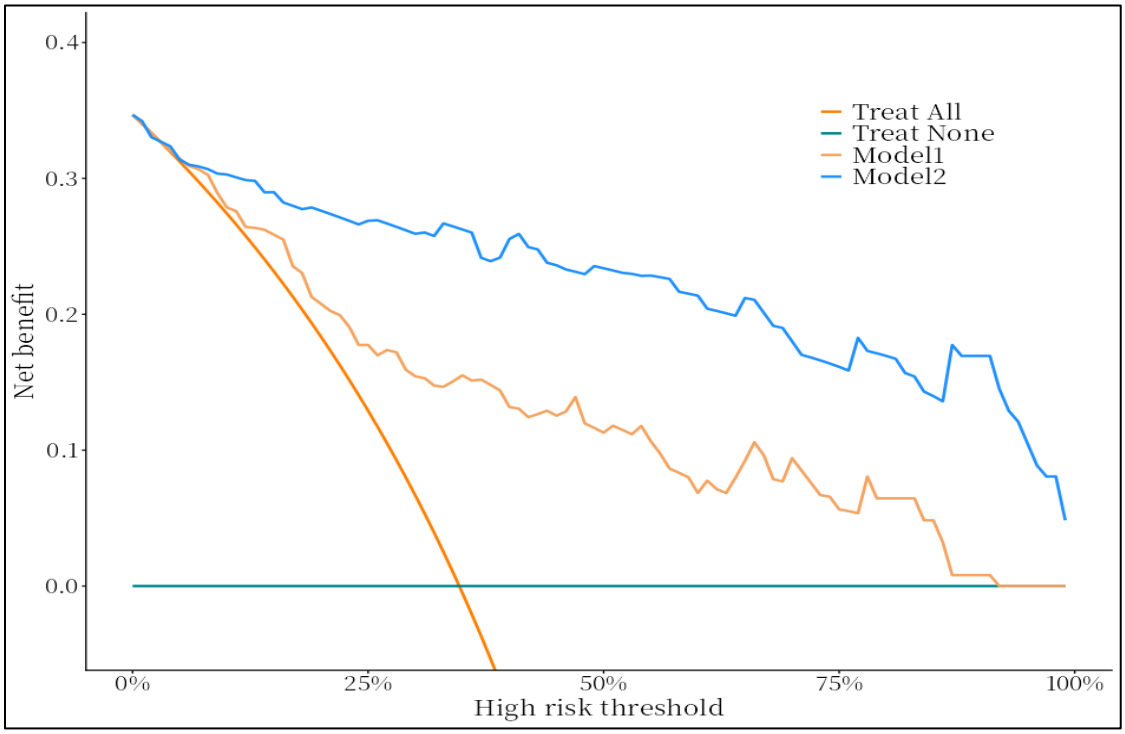

Figure 4. Decision Curves of the Two Models for Identifying Lactic Acidosis in Patients with Acute CO Poisoning.

Note: The horizontal axis represents the high-risk threshold probability, and the vertical axis represents the net benefit. The solid orange line represents the "treat-all" strategy, the cyan solid line represents the "treat-none" strategy, the light orange solid line represents the baseline model (Model 1), and the blue solid line represents the multi-indicator combined model (Model 2).

4. DISCUSSION

This study enrolled 124 adult patients with acute CO poisoning in the emergency department. The incidence of lactic acidosis on admission was 34.7%, similar to international reports. Doğan et al. (2015) and Icme et al. (2014) reported rates of 31.2% [13] and 36.8% [15] for blood lactate ≥4.0 mmol/L, respectively. Discrepancies across studies are mainly attributed to differences in inclusion criteria, poisoning severity, and pre-hospital interventions. Patients with pre-hospital oxygen or fluid therapy were excluded, and all samples were collected before intervention, allowing objective reflection of the initial metabolic state. Regarding the lactate threshold selection, we acknowledge that 2.0 mmol/L is widely used as a sensitive indicator for early tissue perfusion deficits. However, using

this lower threshold would increase detection rate but decrease specificity, potentially diluting the model's discriminative performance by including patients with clinically insignificant mild hyperlactatemia. The 4.0 mmol/L threshold is more appropriate for our study aim of identifying high-risk patients requiring urgent intervention. Previous studies confirmed that lactic acidosis is common in CO poisoning and closely related to disease severity and neurological injury [16,17]. Elevated lactate is associated with adverse outcomes in critically ill patients, with etiological heterogeneity, especially in sepsis and trauma [11,18]. Severe hyperlactatemia predicts higher ICU admission and mortality [12], highlighting the clinical value of identifying associated indicators.

LASSO regression was used for variable selection to avoid bias from univariate screening. After adjustment for confounders, increased respiratory rate, elevated white blood cell count, and higher COHb were independently and positively associated with lactic acidosis in acute CO poisoning. Respiratory rate is readily available at the bedside; white blood cell count and COHb are routine emergency tests. The combined three-index model performed significantly better than COHb alone for risk stratification.

Elevated COHb was independently associated with lactic acidosis, consistent with previous finding [13,15,19].The underlying mechanism involves reduced oxygen-carrying capacity, mitochondrial respiratory chain inhibition, blocked aerobic metabolism, and enhanced anaerobic glycolysis, leading to lactate accumulation [2,5,20]. In this study, each 1% increase in COHb raised the risk of lactic acidosis by 20% (OR=1.20, 95% CI: 1.11–1.29, P<0.001).

Increased respiratory rate was also independently associated with lactic acidosis. Tachypnea represents a compensatory response to tissue hypoxia. Severe hypoxia overcomes physiological compensation and activates the respiratory center; however, increased respiratory muscle work elevates oxygen consumption and worsens hypoxia-lactate vicious cycle. Central nervous system injury may further impair respiratory regulation [3,21], consistent with previous reports [11].The inverse association between hypertension history and lactic acidosis in univariate analysis warrants cautious interpretation. While it might suggest a protective effect, the loss of significance after multivariate adjustment indicates confounding by healthcare-seeking behavior, medication effects, or unmeasured variables related to exposure characteristics. This finding should not be interpreted as evidence for a protective role of hypertension against CO poisoning-induced metabolic derangement.

Elevated white blood cell count was independently associated with lactic acidosis, reflecting stress and inflammatory responses to hypoxic tissue injury [22]. Hypoxia and cell damage activate innate immunity, trigger inflammatory cell infiltration and oxidative stress, aggravate mitochondrial dysfunction and microcirculatory disturbance, and promote lactate production [5,22]. Previous studies also linked systemic inflammation to organ injury and metabolic disturbance in CO poisoning, supporting our findings.

This study has several limitations. First, it is a single-center retrospective design with a relatively small sample, which may cause selection bias; results require validation in multicenter prospective studies. Second, potential confounders such as exposure duration and pre-hospital transport time were not included. The time interval between cessation of CO exposure and blood sampling in the emergency department was not systematically recorded. This represents a critical measurement bias, as COHb has a half-life of approximately 4-6 hours on room air (and 40-80 minutes on 100% oxygen), while lactate levels can change rapidly with tissue perfusion and pre-hospital interventions [1,2]. Variations in transport time and pre-hospital oxygen administration (despite exclusion criteria) may have influenced the measured values. Future prospective studies should standardize sampling timepoints and record exact exposure durations to minimize this bias. Third, only admission lactic acidosis was analyzed, without follow-up of lactate clearance or long-term neurological outcomes. Additionally, the impact of different lactate thresholds (e.g., 2.0 mmol/L or 3.0 mmol/L) on model performance was not evaluated in this study. Future research should assess whether the combined model maintains its discriminative ability across varying definitions of hyperlactatemia. Fourth, the study included only adults; generalizability to children and elderly patients needs further verification. Fifth, the study inclusion period (December 2021 to December 2024) overlapped with the COVID-19 pandemic. COVID-19 infection can independently alter respiratory rate, inflammatory markers, and lactate metabolism, representing a potential unmeasured confounding factor. Due to the retrospective design, emergency medical records did not consistently document COVID-19 screening results, preventing systematic exclusion or adjustment for this variable. This limitation means that lactate levels or inflammatory parameters in some patients may have been partially attributable to COVID-19 rather than pure CO poisoning, potentially underestimating or overestimating the effect sizes of certain indicators. We recommend validating this model in independent samples outside the pandemic period and standardizing the recording of respiratory viral infection status in future prospective studies.

5. CONCLUSION

The results indicate that increased respiratory rate, elevated white blood cell count, and higher COHb on admission are independently and positively associated with lactic acidosis in acute CO poisoning patients. The multi-indicator combined model shows favorable

identification performance and clinical net benefit for lactic acidosis, which can provide evidence-based support for emergency risk stratification and lay a foundation for future prospective studies.

Conflict of Interest: The authors declare that they have no conflict of interest.

Data Availability: Data are unavailable publicly for privacy protection, and may be obtained from the corresponding author upon ethical approval.

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