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ORIGINAL ARTICLE

Initial Lactate vs. Lactate Clearance in Predicting Hyperbaric Oxygen Therapy Requirement in Carbon Monoxide Poisoning

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SUMMARY

Background: **This study aimed to determine the relationship between baseline blood lactate levels and lactate clearance (C_L) and the need for hyperbaric oxygen therapy (HBOT) in patients with carbon monoxide (CO) poisoning.**
Methods: **This retrospective study included patients admitted to the emergency department for CO poisoning between January 1, 2017, and March 1, 2024. Patients were classified into normobaric oxygen therapy (NBOT) and HBOT groups based on the treatment received. Initial and peak lactate levels (measured 2 - 6 hours post-admission) were recorded in mmol/L from venous blood gas analysis. C_L was calculated using the formula: C_L = [(initial lactate - final lactate)/initial lactate] x 100.**
Results: **One hundred and sixty-nine patients were included in the study, out of which 78.7% (n = 133) did not receive HBOT, while 21.3% (n = 36) were treated with HBOT. Median lactate levels were significantly higher in the HBOT group (3 [1.2 - 10] mmol/L) compared to the NBOT group (2.1 [0.5 - 14.6] mmol/L) (p < 0.001). Median C_L values were 35.29% (-50 - 89.06) for the NBOT group, and 35.28% (-31.58 - 87.95) for the HBOT group, and no significant association was found between C_L and the treatment modality (p = 0.596). Receiver operating characteristic analysis identified lactate > 2.8 mmol/L as predictive of HBOT need, with 63.89% sensitivity and 72.93% specificity (area under the curve: 0.705, 95% confidence interval: 0.631 - 0.773, p < 0.0001).**
Conclusions: **In CO poisoning patients, initial lactate levels at admission may better indicate the need for HBOT than C_L.**
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KEYWORDS

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INTRODUCTION

Carbon monoxide (CO) is a colorless, odorless, tasteless, and non-irritating gas produced due to the incomplete combustion of carbon-based fuels [1]. CO poisoning continues to be a significant public health issue worldwide. It is among the most common causes of poisoning-related mortality and morbidity [2]. The diagno-

sis of CO poisoning is established through laboratory and clinical evaluation; a recent history of CO exposure, the presence of symptoms consistent with CO poisoning, and elevated levels of carboxyhemoglobin (COHb) in the blood are required [3]. The clinical symptoms and severity of acute CO poisoning are not always correlated with COHb concentrations at the time of presentation [2]. To facilitate the elimination of CO from the blood, either the oxygen concentration in the inhaled air must be increased or the atmospheric pressure must be elevated. Normobaric oxygen therapy (NBOT) and hyperbaric oxygen therapy (HBOT) are used for this purpose. If the patient is asymptomatic in cases of CO poisoning, 100% oxygen should be administered via a non-rebreather mask as soon as possible until the COHb levels return to normal. HBOT is indicated for patients with loss of consciousness, ischemic cardiac changes, neurological deficits, severe metabolic acidosis, pregnancy, or markedly elevated COHb [4].

Elevated blood lactate levels are a common laboratory finding in critically ill patients. Increased blood lactate concentration has been used in critically ill patients to predict the severity of the disease, morbidity, and mortality, to determine specific treatments, and to monitor the adequacy and timing of medical interventions [5]. It has been demonstrated that lactate is closely associated with morbidity and mortality in critically ill patients [6]. Recent studies have suggested that lactate clearance (C_L) may be a more reliable indicator than absolute lactate levels for risk stratification in critically ill patients [7]. In patients with cardiogenic pulmonary edema, C_L is an important parameter in predicting clinical outcomes [8]. In patients presenting to the emergency department with exacerbation of chronic obstructive pulmonary disease (COPD), monitoring lactate levels may be beneficial in clinical decision-making [9]. In intensive care patients, a low C_L can guide clinicians in clinical monitoring and treatment evaluation [10]. There are limited studies in the literature that have examined the relationship between CO poisoning and C_L.

This study aimed to investigate the relationship between blood lactate levels and C_L and the need for HBOT in patients with CO poisoning admitted to the emergency department.

MATERIALS AND METHODS

This single-center retrospective study was conducted at a tertiary hospital following approval by the local ethics committee (protocol no: SUKAEK-2023-19/3, date: October 18, 2023).

Patients aged > 18 years who presented to the emergency department with CO poisoning between January 1, 2017, and March 1, 2024, and had initial and follow-up venous blood gas (VBG) results (2 - 6 hours post-admission) were included.

Patients with incomplete or unavailable data were excluded from the study. Additionally, individuals with conditions potentially causing tissue hypoxia, such as sepsis, renal failure, COPD, diabetic ketoacidosis, cardiogenic shock, recent epileptic seizure, use of medications known to elevate lactate (e.g., metformin), or major trauma, were excluded.

Demographic characteristics, comorbidities, laboratory parameters, initial blood lactate levels at presentation, and the highest lactate levels observed within 2 - 6 hours were recorded from the hospital's electronic medical records and patient files.

Patients were stratified as NBOT and HBOT according to treatment modality. Those meeting indications for HBOT at the presentation initially received NBOT until HBOT could be administered. Initial lactate levels at presentation to the emergency department and the peak lactate levels within 2 - 6 hours were recorded in mmol/L. C_L was calculated using the formula: [(initial lactate - final lactate)/initial lactate] x 100 [11].

Data were initially entered into Microsoft Excel and then analyzed using IBM SPSS (version 25) and MedCalc (version 20; MedCalc Software, Ostend, Belgium). The normality of the variables was assessed via the Kolmogorov-Smirnov test. Since the numerical data did not follow a normal distribution, they were presented as median (minimum - maximum); categorical variables were expressed as count (n) and percentage (%). For comparisons between two independent groups, the Mann-Whitney U test was used, while the Wilcoxon test was applied for paired (dependent) group comparisons. Categorical variables were compared using the chi-squared test or Fisher's exact test. A receiver operating characteristic (ROC) analysis was used to determine optimal cutoff values for laboratory parameters (lactate, COHb) that are predictive of HBOT indication. Area under the curve (AUC), sensitivity, specificity, and 95% confidence intervals were calculated. A p-value < 0.05 was considered statistically significant in all analyses.

RESULTS

A total of 358 patient records with CO poisoning were screened; after applying the exclusion criteria, 169 patients were included in the study. The study flowchart is shown in Figure 1.

The mean age of the patients was 44.81 ± 18.77 years; 99 (58.6%) were female and 70 (41.4%) were male. The mean age was 45.17 ± 18.90 years for males and 44.55 ± 18.77 years for females, respectively, with no significant difference between genders ($p = 0.834$).

The most common presenting symptoms were headache in 36 patients (21.3%) and nausea in 28 patients (16.6%). Patients' symptoms, state of consciousness, comorbidities, and laboratory parameters are summarized in Table 1.

VBG parameters, including pH, COHb, lactate, bicarbonate (HCO_3^-), and base excess, were compared between baseline and follow-up measurements. Accord-

Table 1. Patients' symptoms, state of consciousness, comorbidities, and laboratory parameters.

Symptoms *	
Headache	36 (21.3)
Nausea	28 (16.6)
Fatigue	25 (14.8)
Dyspnea	23 (13.6)
Dizziness	20 (11.8)
Syncope	18 (10.7)
Altered level of consciousness	10 (5.9)
Chest pain	9 (5.3)
Consciousness *	
Alert	159 (94.1)
Confusion	6 (3.6)
Lethargy	0 (0)
Stupor	2 (1.2)
Coma	2 (1.2)
Comorbidity *	
HT	34 (20.1)
CAD	16 (9.5)
CVD	12 (7.1)
DM	11 (6.5)
CHF	3 (1.8)
CABG	2 (1.2)
Laboratory parameters **	
Glucose (mg/dL)	119 (81 - 432)
Creatinine (mg/dL)	0.7 (0.43 - 1.2)
Urea (mg/dL)	32 (11 - 70)
Sodium (mmol/L)	137 (131 - 143)
Potassium (mmol/L)	4.1(3.1 - 5.7)
AST (U/L)	25 (11 - 97)
WBC (10^9/L)	9.9 (4.54 - 23.34)
NEU (10^9/L)	6.28 (2.14 - 21.08)
HB (g/dL)	13.6 (6.1 - 19)
LYM (10^9/L)	2.2 (0.2 - 12.7)
Troponin (ng/mL)	0.0 (0.0 - 3.54)

*** n (%), ** median (minimum - maximum), HT hypertension, CAD coronary artery disease, CVD cerebrovascular disease, DM diabetes mellitus, CHF congestive heart failure, CABG coronary artery bypass graft, AST aspartate aminotransferase, ALT alanine amino-transferase, WBC white blood count, NEU neutrophil, HB hemoglobin, LYM lymphocyte.**

Table 2. Comparison of baseline and control blood gas results.

	Baseline blood gas result	Control blood gas result	p
	Median (min - max)	Median (min - max)	
Ph	7.37 (7.03 - 7.60)	7.37 (7.15 - 7.58)	0.304
COHb (%)	16.7 (0.5 - 47.9)	2.9 (0.2 - 14)	< 0.001
Lactate (mmol/L)	2.20 (0.5 - 14.6)	1.4 (0.4 - 9.40)	< 0.001
HCO_3^- (mmol/L)	25.4 (10.4 - 34.5)	25.8 (14.5 - 33.1)	0.002
BE (mmol/L)	0.4 (-19.3 - 8.80)	0.8 (-14.2 - 6.6)	0.009

COHb carboxyhemoglobin, HCO_3^- bicarbonate, BE base excess.

Table 3. Comparison of patients according to the treatment modality.

Parameters	HBOT (-)	HBOT (+)	p
Age **	41 (18 - 92)	51 (20 - 89)	0.067
Gender *			
Male	56 (80)	14 (20)	0.875
Female	77 (77.8)	22 (22.2)	
Consciousness *			
Alert	129 (81.1)	30 (18.9)	0.002
Confusion	1 (16.7)	5 (83.3)	
Lethargy	0	0	
Stupor	2 (100)	0 (0)	
Coma	1 (50)	1 (50)	
Laboratory parameters **			
Glucose (mg/dL)	112 (81 - 432)	144.7 (93.3 - 321)	< 0.001
Creatinine (mg/dL)	0.7 (0.5 - 1.20)	0.73 (0.43 - 1.20)	0.523
Urea (mg/dL)	31.2 (11 - 60.4)	34 (16 - 70)	0.048
Na (mmol/L)	138 (131 - 143)	137 (133 - 142)	0.153
K (mmol/L)	4.06 (3.1 - 5.7)	4.2 (3.2 - 5.56)	0.394
AST (U/L)	24 (12 - 71)	28.45 (11.9 - 97)	0.063
ALT (U/L)	18 (7 - 76)	18 (6.4 - 76)	0.579
Trop (ng/mL)	0 (0 - 2.23)	0.108 (0 - 3.54)	< 0.001
WBC (10^9/L)	9.1 (4.54 - 23.34)	11.9 (6.20 - 21.64)	< 0.001
Neutrophils (10^9/L)	5.4 (2.14 - 21.08)	8.9 (3.5 - 19.70)	< 0.001
Lymphocytes (10^9/L)	2.2 (0.6 - 6.20)	1.57 (0.2 - 12.70)	0.029
Platelets (10^9/L)	249 (43 - 438)	293 (123 - 520)	0.019
pH	7.37 (7.14 - 7.60)	7.36 (7.03 - 7.44)	0.02
COHb (%)	14.3 (0.5 - 39.1)	22.5 (1.3 - 47.9)	0.001
Lactate (mmol/L)	2.1 (0.5 - 14.6)	3 (1.2 - 10)	< 0.001
BE (mmol/L)	0.7 (-17 - 8.80)	-0.65 (-19.3 - 7.20)	0.011
HCO_3^- (mmol/L)	25.6 (10.4 - 34.5)	24.2 (10.5 - 29.9)	0.104
C_L (%)	35.29 (-50 - 89.06)	35.28 (-31.58 - 87.95)	0.596

*** n (%), ** median (minimum - maximum), HBOT (-) not receiving hyperbaric oxygen therapy, HBOT (+) hyperbaric oxygen therapy recipeents, COHb carboxyhemoglobin, HCO_3^- bicarbonate, BE base excess, AST aspartate aminotransferase, ALT alanine amino-transferase, WBC white blood count, C_L lactate clearance.**

Table 4. ROC analysis of patients receiving hyperbaric oxygen therapy.

	AUC (95% CI)	Cutoff value	Sensitivity	Specificity	p
Lactate (mmol/L)	0.705 (0.631 - 0.773)	> 2.8	63.89	72.93	< 0.0001
COHb (%)	0.680 (0.604 - 0.750)	> 18	66.67	60.15	< 0.001

ingly, COHb and lactate levels were statistically significantly lower during follow-up than at baseline ($p < 0.001$ for both). Other parameters are presented in Table 2.

According to the treatment received, patients were divided into NBOT (n = 133; 78.7%) and HBOT (n = 36; 21.3%) groups. Median initial lactate was significantly higher in the HBOT group (3.0 [1.2 - 10] mmol/L) com-

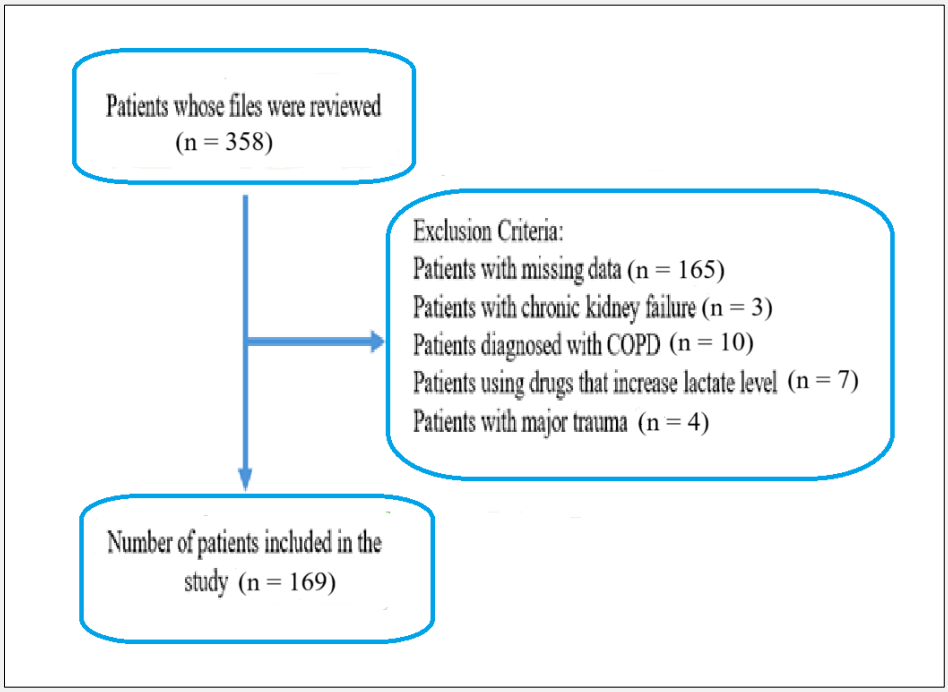

Figure 1. Study flow chart.

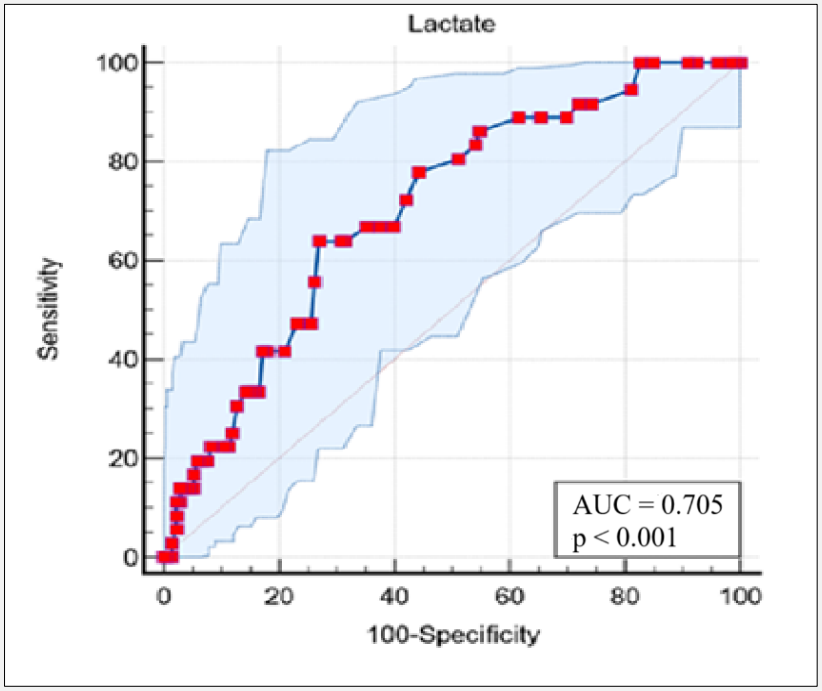

Figure 2. ROC analysis of lactate levels in patients receiving hyperbaric oxygen therapy.

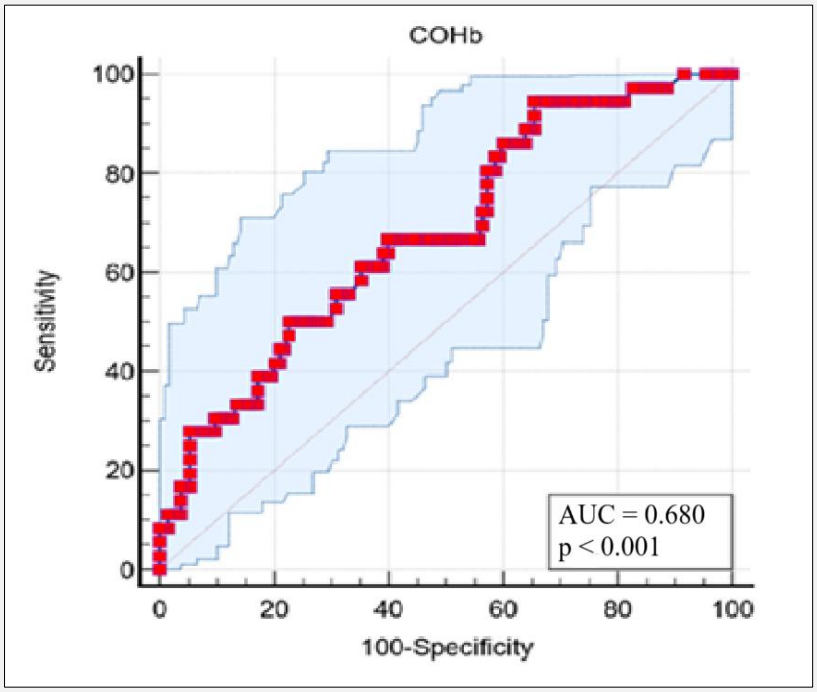

Figure 3. ROC analysis of COHb levels in patients receiving hyperbaric oxygen therapy.

pared to the NBOT group (2.1 [0.5 - 14.6] mmol/L; $p < 0.001$). Median C_L did not differ significantly between NBOT (35.29% [-50 - 89.06]) and HBOT (35.28% [-31.58 - 87.95]) groups ($p = 0.596$). Median COHb was also significantly higher in the HBOT group (22.5% [1.3 - 47.9]) compared to the NBOT group (14.3% [0.5 - 39.1]; $p = 0.001$) (Table 3). A detailed comparison of the groups in terms of other parameters is presented in Table 3.

ROC analysis was used to evaluate the predictive value of initial lactate for HBOT indication (Table 4). This analysis identified lactate > 2.8 mmol/L as predictive of HBOT need, with 63.89% sensitivity and 72.93% specificity (AUC: 0.705, 95% confidence interval (CI): 0.631 - 0.773, $p < 0.0001$) (Table 4) (Figure 2).

Similarly, a COHb threshold > 18% provided 66.67% sensitivity and 60.15% specificity for predicting HBOT need (AUC: 0.680, 95% CI: 0.604 - 0.750, $p < 0.001$) (Table 4, Figure 3).

DISCUSSION

This study was conducted to determine the relationship between blood lactate levels, C_L, and the chosen treatment modality in patients presenting to the emergency department with CO poisoning. The findings indicate that the initial blood lactate level is an effective parameter for determining the need for HBOT in patients with CO poisoning. In contrast, C_L did not show a significant difference between treatment groups, and therefore, appears not to be a useful parameter for predicting the need for HBOT.

To the best of our knowledge, this is the first study to investigate the effectiveness of C_L in predicting the need for HBOT in this patient group. Given the need for objective criteria in determining HBOT indications for CO poisoning, we believe that our findings make a valuable contribution to the literature.

The diagnosis of CO poisoning is established based on the clinical presentation, COHb level, and a history of CO exposure. It is important to note that COHb levels can be affected by factors such as smoking and environmental pollution. Furthermore, COHb levels may decrease after the patient is removed from the CO source and due to oxygen administration prior to hospital admission; therefore, a normal COHb level does not exclude CO poisoning [12]. In a study by Grieb et al., it was reported that the initial COHb level alone is insufficient to assess the severity of poisoning [13]. In another study, Dogruyol et al. identified COHb as a parameter that could influence the decision to administer HBOT in cases of CO poisoning [14]. Dogan et al. reported that patients receiving HBOT had significantly

higher COHb levels than those treated with NBOT, suggesting that COHb levels should be considered [15]. Consistent with the literature, COHb values of patients receiving HBOT were significantly higher in the present study.

Elevated blood lactate levels are a common laboratory finding in critically ill patients [5]. Lactate measurement is useful in monitoring patients with cardiogenic shock and serves as a strong predictor of mortality [7]. Several studies have underscored the importance of tailoring early resuscitation based on lactate levels [5]. In their study, Lucas et al. demonstrated that high initial lactate levels in critically ill patients are associated with poor prognosis [16]. Dogan et al. showed that patients receiving HBOT had higher lactate levels than those treated with NBOT and identified a lactate cutoff value of 1.85 mmol/L for predicting HBOT [15]. In a study by Kaldirim et al., patients undergoing HBOT were also found to have higher lactate levels, and the authors recommended that both COHb and blood lactate levels be considered when determining HBOT indications [17]. Uysalol et al. reported that the duration of CO exposure, COHb levels, neurological symptoms, and lactate levels are guiding parameters in determining the need for HBOT [18]. Guzel et al. suggested that lactate levels may assist clinicians in predicting the length of hospital stay [19]. Cervellin et al. reported lactate levels of 1.5 mmol/L among patients receiving HBOT compared to 1.1 mmol/L among those not receiving HBOT, and 1.8 mmol/L among patients admitted to the hospital compared to 1.2 mmol/L among those who were not. They also indicated that blood lactate levels might be useful in risk stratification and determining the need for hospital admission in patients with CO poisoning [20]. Consistent with the literature, lactate levels were significantly higher in HBOT recipients in the present study. Moreover, a lactate level > 2.8 mmol/L predicted the need for HBOT with a sensitivity of 63.89% and a specificity of 72.93%. These findings suggest that the initial blood lactate level may be a valuable parameter in determining the need for HBOT in patients with CO poisoning.

Recent research suggests that C_L may be a more reliable indicator of clinical severity and prognosis than absolute lactate levels in critically ill patients [7]. Yadigaroglu et al. reported that in COVID-19 patients, high lactate levels and low lactate kinetics are predictive of mortality [21]. In patients with shock, serial lactate measurements during follow-up have been shown to possess greater prognostic value than a single initial lactate measurement [22]. Haas et al. demonstrated that in critically ill patients with severe hyperlactatemia (> 10 mmol/L), particularly when significant C_L is not achieved within 12 hours, there is an association with high in-hospital mortality [10]. Durmus et al. indicated that monitoring lactate levels in the emergency department may be a useful tool in determining whether patients presenting with COPD exacerbations should be discharged or admitted [9]. In their study, Efgan et al. found that a C_L < 14.29% in patients with cardiogenic pulmonary edema could serve as an indicator for early initiation of treatment and predict poor prognosis [8]. Liu et al. observed that in patients with delayed encephalopathy due to CO poisoning, C_L measured after 24 hours of treatment was significantly lower in the group with a favorable prognosis. Moreover, patients with high C_L had a significantly lower prevalence of delayed encephalopathy compared to those with low C_L [23]. Wang et al. reported that blood C_L is critically important for evaluating neurological damage and prognosis in cases of acute CO poisoning [24]. Wang et al. reported a negative correlation between early C_L (at 6, 24, and 72 hours) and the incidence of delayed encephalopathy in hyperlactatemic CO poisoning [25]. There are limited studies in the literature investigating the association between delayed encephalopathy due to CO poisoning and C_L. Furthermore, the association between C_L and the chosen treatment modality has not yet been sufficiently investigated. In the present study, C_L did not show a significant difference between treatment groups, indicating that it may not be a useful parameter for predicting the need for HBOT.

Limitations

Our study has several limitations. One of the most significant limitations is that patients received oxygen therapy during the transfer from the poisoning environment and from other healthcare facilities to our hospital. Additionally, the retrospective and single-center design of the study limits the generalizability of the findings. The exclusion of patients who were discharged from the emergency department without a repeat blood gas analysis because of their mild clinical condition represents another limitation.

CONCLUSION

In patients with CO poisoning, the initial lactate level measured at presentation appears to be a more reliable and effective indicator than C_L for predicting the need for HBOT and guiding clinical decision-making.

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Declaration of Interest:
The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publica-

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