North Clin Istanb 2019;6(2):141–145
doi: 10.14744/nci.2018.88709

Original Article EMERGENCY MEDICINE

Analysis of the effects of COHb, lactate, and troponin levels on the clinical process and outcome in patients who were admitted to the emergency service due to carbon monoxide poisoning

Mehmet Necdet Yildiz,[1] Serkan Emre Eroglu,[2] Can Ozen,[2] Handan Aydin Yildiz,[3] Bilge Kagan Sektioglu,[4] Celil Alkan[2]

[1]Department of Emergency Medicine, T.E.V. Sultanbeyli State Hospital, Istanbul, Turkey

[2]Department of Emergency Medicine, University of Health Sciences, Umraniye Training and Research Hospital, Istanbul, Turkey

[3]Department of Psychiatry, University of Health Sciences, Erenkoy Mental Health and Neurological Diseases Training and Research Hospital, Istanbul, Turkey

[4]Department of Emergency Medicine, Sehit Prof. Dr. Ilhan Varank Sancaktepe Training and Research Hospital, Istanbul, Turkey

ABSTRACT

OBJECTIVE: The aim of the present study was to determine the demographic, medical, and treatment characteristics of patients followed up with the diagnosis of carbon monoxide (CO) poisoning in emergency care and also to determine the relationship of these patients' clinical process and outcome between carboxyhemoglobin (COHb), lactate, and troponin levels.

METHODS: The present study was conducted retrospectively between 01/01/2013 and 01/01/2016 by examining 450 patients who were referred to the emergency service for CO poisoning. The ages; sexualities; manners of application; clinical findings; levels of blood COHb, lactate, and troponin; applied oxygen treatment method; and outcome of patients were evaluated. Data analysis was done by Shapiro–Wilk, Student's t, Mann–Whitney U, and chi-square tests.

RESULTS: A total of 450 patients were included in the study. The median age of the patients was 35 (interquartile range (IQR) 26.75–45.00) years. In the study where data are not homogeneously distributed, the median levels of COHb, lactate, and troponin were 11.80% (IQR 3–23), 1.60 (IQR 1.10–2.5) mmol/l, and 0.00 (IQR 0.000–0.003) ng/ml, respectively. The levels of lactate were detected to be statistically high in patients who had syncope and who received hyperbaric oxygen treatment ($p<0.05$). In addition, the levels of lactate and troponin were significantly higher in patients who were hospitalized ($p<0.05$).

CONCLUSION: The levels of COHb, lactate, and troponin can provide an insight to the clinician about hospitalization and the type of treatment.

Keywords: CO poisoning; emergency service; lactate; troponin.

Cite this article as: *Yildiz MN, Eroglu SE, Ozen C, Aydin Yildiz H, Kagan Sektioglu B, Alkan C. Analysis of the effects of COHb, lactate, and troponin levels on the clinical process and outcome in patients who were admitted to the emergency service due to carbon monoxide poisoning. North Clin Istanb 2019;6(2):141–145.*

Carbon monoxide (CO) is a colorless, odorless, tasteless, and non-irritating gas and is formed by inadequate burning of carbon-based fuels [1, 2]. Motor vehicle exhaust gases, smokes caused by fires, gas powered engines, forest fires, and methylene chloride containing dyes are the most common sources of CO [3].

Received: July 15, 2017 *Accepted:* July 02, 2018 *Online:* May 29, 2019

Correspondence: Dr. Serkan Emre EROGLU. Saglik Bilimleri Universitesi, Umraniye Egitim ve Arastirma Hastanesi, Acil Tip Klinigi, 34776 Umraniye, Istanbul, Turkey.
Tel: +90 532 301 08 66 e-mail: drseroglu@gmail.com

CO poisonings constitute 8%–34% of all cases of poisonings [4]. However, symptoms may vary within a wide spectrum from mild headache to coma [5].

In the present study, patients with CO poisoning were admitted to the emergency department. The aim of the present study was to determine the demographic, medical, and treatment characteristics of patients and to investigate the relationship between the clinical process and outcome of patients and blood carboxyhemoglobin (COHb), lactate, and troponin levels.

MATERIALS AND METHODS

The study, which was based on dissertation, was conducted retrospectively in the emergency room of the same hospital. The study was approved by the ethics committee of Umraniye Training and Research Hospital. Patients who were aged >18 years and diagnosed with CO poisoning between 01/01/2013 and 01/01/2016 were included in the study. Patients who did not comply with the inclusion criteria or had inadequate hospital records were excluded from the study.

Based on the data obtained from the scanned patient files and hospital information management system, data related to age; sex; presentation; time of admission; state of consciousness; presence of syncope; blood COHb, troponin, and lactate levels; oxygen treatment method applied to the patients; and patient outcomes were recorded. Data were analyzed using the Statistical Package for Windows, version 23 (SPSS Inc., Chicago, IL, USA) program. In this analysis, while the distributions of data were evaluated by the Shapiro–Wilk test, mean, standard deviation, median, minimum, and maximum values were used for defining continuous variables. Number of patients and percentiles were used in the representation of categorical variables.

Student's t-test was used for comparison of two independent and normally distributed continuous variables, whereas Mann–Whitney U test was used for comparison of two variables that did not conform to normal distribution. Chi-square and Fisher's exact tests were used to examine the relationship between categorical variables. According to the treatment modality, receiver operating characteristic (ROC) analysis was used to evaluate lactate, troponin, and CO levels. Multivariate regression analysis was conducted to analyze the factors affecting the hospitalization of patients. A p value <0.05 was accepted as statistically significant.

Table 1. Information of the study patients related to admission and their clinical process

Variables	n	%
Gender		
Male	184	40.9
Female	266	59.1
Types of admission		
Ambulatory	290	64.4
In ambulance	160	35.6
Time of presentation		
07^{01}–19^{00}	195	43.3
19^{01}–07^{00}	255	56.7
Admission season		
Summer	55	12.2
Fall	81	18
Winter	202	44.9
Spring	112	24.9
Glasgow Coma Score		
15	428	95.1
14	17	3.9
13	2	0.4
12	1	0.2
3	2	0.4
History of syncope		
Yes	64	14.2
No	386	85.8
Type of oxygen therapy		
NBO*	413	91.8
NBO+HBO**	37	8.2
Outcome		
Discharge	440	97.8
Hospitalization	10	2.2

*Normobaric oxygen; **Hyperbaric oxygen.

RESULTS

A total of 450 patients who had CO poisoning during our study period were detected. It was observed that the data of these patients were not suitable for normal distribution. The median age of the patients was 35 (interquartile range (IQR) 26.75–45.00) years. Female patients constituted 59.1% (n=266) of the study population. Most (64.4%, n=290) of the patients were admitted to the outpatient clinic, whereas 56.67% (n=255) of them consulted the emergency department between 19:01 and 06:59 h and often during the winter months (n=202, 44.89%). Sixty-four (14.2%) patients had syn-

Table 2. Comparison between serum COHb, lactate, and troponin levels and other data related to the patient and clinical process

Analysis	Comparator data		No. of patient s tested	Median	p
Serum carboxyhemoglobin level (%)	Type of presentation	Ambulatory	160	12.65 (IQR 3.20–23.00)	0.679
		In ambulance	290	11.10 (IQR 2.95–23.00)	
	Syncope	Yes	64	16.05 (IQR 5.12–26.82)	0.630
		No	386	10.60 (IQR 3.0–22.3)	
	Treatment	NBO	413	10.80 (IQR 2.50–22.40)	0.006
		HBO	37	19.20 (IQR 10.00–22.50)	
	Outcome	Discharge	440	11.20 (IQR 3.00–22.87)	0.560
		Hospitalisation	10	22.85 (IQR 14.72–27.85)	
Serum lactate level (mmol/l)	Type of presentation	Ambulatory	160	1.60 (IQR 1.10–2.57)	0.605
		In ambulance	290	1.60 (IQR 1.10–2.50)	
	Syncope	Yes	64	1.85 (IQR 1.10–2.87)	0.181
		No	386	1.60 IQR 1.10–2.50)	
	Treatment	NBO	413	1.50 (IQR 1.10–2.50)	0.012
		HBO	37	2.20 (IQR 1.55–3.75)	
	Outcome	Discharge	440	1.60 (IQR 1.10–1.50)	0.022
		Hospitalisation	10	2.50 (IQR 2.17–4.32)	
Serum troponin level (ng/ml)	Type of presentation	Ambulatory	126	0.000 (IQR 0.000–0.010)	<0.010
		In ambulance	206	0.000 (IQR 0.000–0.010)	
	Syncope	Yes	53	0.001 (IQR 0.000–0.020)	0.010
		No	260	0.000 (IQR 0.000–0.002)	
	Treatment	NBO	302	0.000 (IQR 0.000–0.002)	<0.001
		HBO	30	0.001 (IQR 0.000–0.040)	
	Outcome	Discharge	322	0.000 (IQR 0.000–0.002)	<0.001
		Hospitalisation	10	0.009 (IQR 0.000–2.422)	

COHb: Carboxyhemoglobin; IQR: Interquartile range; NBO: Normobaric oxygen; HBO: Hyperbaric oxygen.

cope, and the Glasgow Coma Scale scores of 22 (22.9%) patients were <15 (Table 1).

Blood levels of COHb (n=450), lactate (n=448), and troponin (n=332) were analyzed in a respective number of patients. The median values of COHb, lactate, and troponin at the time of presentation were 11.80% (IQR 3–23) (range 1.3%–1.5%), 1.60 (IQR 1.10–2.50) (range 1.2–2.2) mmol/l, and 0.00 (IQR 0.000–0.003) (0–0.03) ng/ml, respectively. In our study, the relationships of these biomarkers with normobaric oxygen (NBO) or hyperbaric oxygen (HBO) treatments were also analyzed (Table 2). According to these analyses, lactate, troponin, and COHb levels of patients receiving HBO treatment were as follows: 2.20 (IQR 1.55–3.75), 0.001 (IQR 0.000–0.040), and 19.20 (IQR 10.00 (26.50), respectively, and statistically significantly higher than those receiving NBO (Mann–Whitney U, p<0.01). With the help of ROC analysis, the presence of the threshold values of these levels was investigated (Fig. 1).

Accordingly, the areas under the curve drawn for HBO therapy were 0.594 for COHb, 0.641 for lactate, and 0.646 for troponin, but a significant threshold value with high specificity and sensitivity could not be achieved (p=0.088, p=0.011, and p=0.008, respectively) (Table 3). The mean values for COHb, lactate, and troponin markers, each according to the status of admission and discharge, were examined separately. Accordingly, the mean values of patients at admission were higher than those discharged. In patients admitted for hospitalization, median lactate, troponin, and COHb values were 1.85 (IQR 1.10–2.87), 0.001 (IQR 0.000–0.020), and 16.05 (IQR 5.12–26.82), respectively (Table 2). The median levels of each parameter were statistically signif-

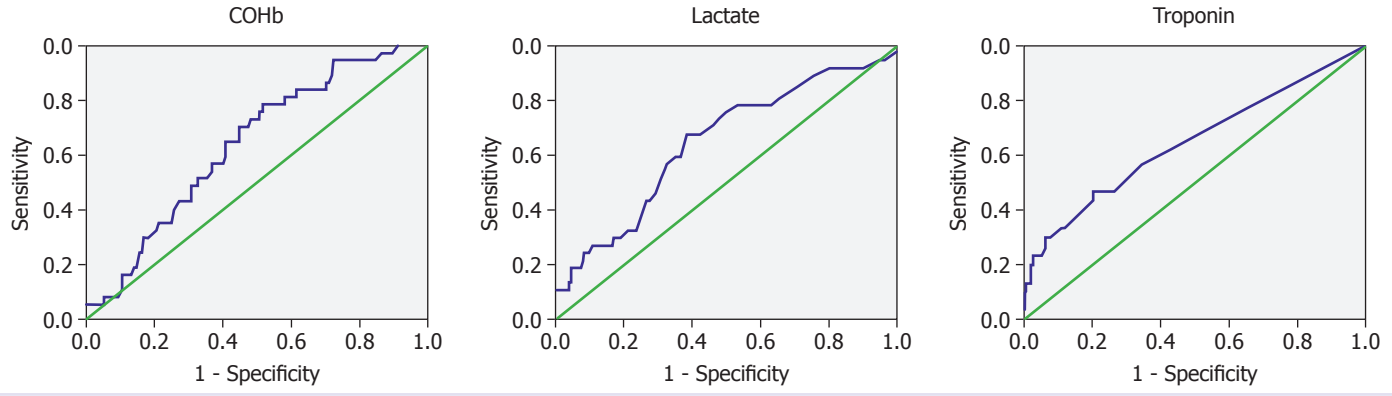

FIGURE 1. ROC analysis of COHb, lactate, and troponin values required for HBO treatment.

TABLE 3. ROC analyses performed separately for lactate, troponin, and CO levels in patients receiving HBO and NBO treatments

	AUC	95% CI	p
COHb (%)	0.594	0.540–0.741	0.088
Lactate (mmol/l)	0.641	0.495–0.694	0.011
Troponin (ng/ml)	0.646	0.530–0.762	0.008

AUC: Area under curve; CI: Confidence interval.

icantly higher than those of discharged patients (Mann–Whitney U, $p<0.01$).

DISCUSSION

CO poisoning is the most important cause of poisoning-related mortality in our country as it is worldwide [1, 3].

In some studies, it has been reported that the average age of the adult population who were admitted to the emergency service with CO poisoning varies between 27 and 41.7 years and consists mainly of female patients [6–8]. Our study has also yielded similar results with respect to age and gender of the poisoned population. The fact that most of the poisonings in our study occurred at night and female patients more frequently referred to hospitals with symptoms of poisoning may be related to greater contribution of men working late at night and women spending more time at home compared with men. Therefore, women are more frequently exposed to sources of CO, such as combi boiler and stoves that use natural gas.

Signs and symptoms of CO poisoning may be non-specific and variable. In their study on CO poisoning in children, Besli et al. detected a correlation between increased lactate levels and neurological signs [9]. In the study by Benaissa et al., lactate levels were found to be significantly higher in patients with CO poisoning who developed neurological symptoms, but the clinical significance was reported to be controversial due to the slight increase in lactate levels [10]. In another study examining syncope development, an increased frequency of syncope was observed in patients with high COHb levels (especially ≥20%) [6]. Contrary to this observation, in our study, although the frequency of syncope was observed to be as high as 14%, it was found that the development of syncope did not correlate with COHb levels. However, our finding of higher level of troponin in these patients, in consideration of the half-life, and production of CO may be related to the time elapsed after exposure to CO gas, which affected negatively lactate and COHb and positively troponin levels.

HBO is one of the most important treatments for CO poisoning. There are several studies reporting different opinions comparing NBO and HBO therapy in the literature. In a Cochrane review, six randomized controlled trials were examined and any clear-cut evidence indicating that HBO alleviates the neurological side effects of CO poisoning [11]. However, Repplinger et al. suggested that lactate can be used for HBO therapy [12]. Although our study did not question the success of the treatment, it was observed that patients who were referred for HBO treatment had high COHb, lactate, and troponin levels, and it was important in that this observation demonstrated our physicians' choice of treatment. In fact, the detection of COHb, lactate, and troponin levels in each patient whom we think has CO poisoning may be useful with respect to treatment planning and

decision-making process for admission or discharge.

Retrospective design of our study, missing file records, failure to bring the patients who died at the event site to our hospital, and lack of any information about the final status of the patients restrict our interpretation. The fact that patients who were brought to our hospital were not included in our study despite the fact that their file records were not lost, and also after the hospitalization in the emergency room, the final status (e.g., referral to an external center and exitus) of the patients restrict our interpretation.

In conclusion, our study has reflected some demographic, medical, and therapeutic characteristics of the patients presented with CO poisoning, which are frequently encountered in both press and organization of emergency services, and reinforced the need for hospitalization and follow-up of the patients with elevated blood COHb, lactate, and troponin levels.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study has received no financial support.

Authorship Contributions: Concept – MNY, CA; Supervision – SEE; Materials – HY; Data collection and/or processing – MNY, CO Analysis and/or interpretation – BKS, CO; Writing – MNY; Critical review – MNY, SEE.

REFERENCES

1. Malaney G. Carbon monoxide. In: Tintinalli JE, editors. Emergency Medicine: A Comprehensive Study Guide. 8th ed. McGraw-Hill 2016. p. 1437–40.
2. Sykes OT, Walker E. The neurotoxicology of carbon monoxide - Historical perspective and review. Cortex 2016;74:440–8.
3. Bulut M. Kabonmonoksit zehirlenmesi. In: Kekeç Z, editor. Tüm Yönleriyle Acil Tıp. Adana; 2014. p. 715–20.
4. Denizbaşı A. Zehirlenmiş hastaya yaklaşım. In: Kekeç Z, editor. Tüm Yönleriyle Acil Tıp. Adana; 2014. p. 657–66.
5. Chang YC, Lee HY, Huang JL, Chiu CH, Chen CL, Wu CT. Risk Factors and Outcome Analysis in Children with Carbon Monoxide Poisoning. Pediatr Neonatol 2017;58:171–7.
6. Keleş A, Demircan A, Kurtoğlu G. Carbon monoxide poisoning: how many patients do we miss? Eur J Emerg Med 2008;15:154–7.
7. Sá MC, Rodrigues RP, Moura D. Carbon monoxide intoxications in Portugal. [Article in Portuguese]. Acta Med Port 2011;24:727–34.
8. Kaya H, Coskun A, Beton O, Kurt R, Yildirimli MK, Gul I. A cost effective parameter for predicting the troponin elevation in patients with carbon monoxide poisoning: red cell distribution width. Eur Rev Med Pharmacol Sci 2016;20:2891–8.
9. Besli GE, Ergüven M, Karadogan M, Yilmaz Ö. Carbon Monoxide Poisoning in Children. Eurasian J Emerg Med 2010;9:26–30.
10. Benaissa ML, Mégarbane B, Borron SW, Baud FJ. Is elevated plasma lactate a useful marker in the evaluation of pure carbon monoxide poisoning? Intensive Care Med 2003;29:1372–5.
11. Buckley NA, Juurlink DN, Isbister G, Bennett MH, Lavonas EJ. Hyperbaric oxygen for carbon monoxide poisoning. Cochrane Database Syst Rev 2011;CD002041.
12. Repplinger DJ, Slomer A, Nolan B, Su MK. Lactate and carbon monoxide poisoning: More questions regarding its prognostic role. Hum Exp Toxicol 2016;35:794.