Karbonmonoksit Zehirlenmelerinde Klinik Semptomların Biyokimyasal Parametrelerle Değerlendirilmesi

Evaluation of Clinical Symptoms in Carbon Monoxide Poisoning with Biochemical Parameters

Handan Çiftçi[1], Ömer Çanacık[1], İlksen Dönmez[2], Turgut Dolanbay[1], Hüseyin Fatih Gül[3]

[1]Kafkas Üniversitesi Tıp Fakültesi, Acil Tıp Ana Bilim Dalı, Kars, Türkiye

[2]İstanbul Beyoğlu Göz Eğitim ve Araştırma Hastanesi, Anesteziyoloji ve Reanimasyon Anabilim Dalı, İstanbul, Türkiye

[3]Kafkas Üniversitesi Tıp Fakültesi, Tıbbi Biyokimya Anabilim Dalı, Kars, Türkiye

ÖZ

GİRİŞ ve AMAÇ: *Bu çalışmanın amacı karbonmonoksit (CO) toksisitesinde, karboksihemoglobin (COHb) düzeyi ile klinik semptomlar ve biyokimyasal parametreler (hemogram, laktat, iyonize kalsiyum ve üre) arasındaki ilişkiyi incelemektedir.*

YÖNTEM ve GEREÇLER: *Retrospektif desende yürütülen araştırmanın örneklemini 2014-2019 yılları arasında hastanemiz acil servisine CO zehirlenmesi ile başvuran 117 hasta oluşturdu. Araştırma verileri Veri Toplama Formu aracılığı ile toplandı. Analizlerde tanımlayıcı istatistikler (yüzde, ortalama, ortanca), Man Whitney U, One Way Anova F, Kruskal Wallis H testleri ve Spearman Korelasyon Analizi kullanıldı.*

BULGULAR: *Karbon monoksit zehirlenmesi tanısı alan 55'i kadın (%47) toplam 117 hasta çalışmaya alındı. Hastaların %80.3'ünün ısınma kaynağı kömür sobasıydı. Bildirilen birincil semptomlar %65.8'inde bulantı-kusma, %51.3'ünde baş ağrısı, %47'sinde baş dönmesi semptomları vardı. Ortalama COHb düzeyi %16.85 ±9.54, CO gazına maruz kaldıkları süre 5.39±4.01 saatti. COHb düzeyi ile laktat (r= 0.441, p=0.00), hemoglobin (r=0.188, p=0.042) ve üre düzeyleri (r=0.204, p= 0.027) arasında zayıf korelasyon tespit edildi. COHb düzeyi ≥%25 olan hastalarda ortalama hemoglobin düzeyi 16.06 mg/dl, üre düzeyi 40 mg/dl idi (p<0.05).*

TARTIŞMA ve SONUÇ: *Karboksihemoglobin ile laktat, üre, nötrofil, bazofil ve monosit parametreleri arasında ilişki vardı. Ayrıca akut CO zehirlenmelerinde hem COHb seviyelerinin hem de biyokimyasal parametrelerin semptomlarla doğrudan ilişkili olmadığı gözlenildi. Karboksihemoglobin seviyesi, acil servise nakil süresinin uzunluğuna ve CO gazına maruz kalma süresine bağlı olarak değişmiştir.*

Anahtar Kelimeler: *karboksihemoglobin, karbon monoksit zehirlenmesi, biyokimyasal parametreler*

ABSTRACT

INTRODUCTION: *The aim of this study is to examine the relationship between carboxyhemoglobin (COHb) level in carbon monoxide (CO) toxicity and clinical symptoms and, biochemical parameters (hemogram, lactate, ionized calcium and urea).*

METHODS: *The sample of this retrospective study consisted of 117 patients who visited the emergency department of our clinic between 2014 and 2019. The study data were collected with the Data Collection Form. Descriptive statistics (percentage, mean, and median), Mann-Whitney U, One Way ANOVA F, Kruskal-Wallis H, and Spearman Correlation Analysis were used in analyses.*

RESULTS: *The study included 117 patients diagnosed with CO poisoning, of which 55 were women (47%). Of the patients, 80.3% used a coal stove as their heating source. The primary reported symptoms were nausea-vomiting (65.8%), headache (51.3%), and dizziness (47%). The mean COHb level was 16.85±9.54% and the mean CO exposure time was 5.39±4.01 hours. The COHb level was weak correlated with lactate (r=0.441, p=0.00), hemoglobin (Hb) (r=0.188, p=0.042), and urea (r=0.204, p=0.027) levels. Patients with a COHb level ≥25% had a mean Hb level of 16.06 mg/dL and a mean urea level of 40 mg/dL (p<0.05).*

DISCUSSION AND CONCLUSION: *There was a relationship between COHb and lactate, urea, neutrophil, basophil, and monocyte parameters. In addition, it was observed that both COHb levels and biochemical parameters were not directly related to symptoms in acute CO poisoning. The COHb level varied depending on the length of time in transport to the emergency room and the exposure time to the CO gas.*

Keywords: *carboxyhemoglobin, carbon monoxide poisoning, biochemical parameters*

İletişim / Correspondence:

Dr. Öğr.Üyesi Handan Çiftçi

Kafkas Üniversitesi Tıp Fakültesi, Acil Tıp Ana Bilim Dalı, Kars, TürkiyeE-mail:mmanici@kuh.ku.edu.tr

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INTRODUCTION

Carbon monoxide (CO) is released from natural or anthropogenic sources, especially through the incomplete combustion of fossil fuels and biomass (1). The CO molecular weight is 28 g/mol. It is a colorless, tasteless, and odorless gas and its concentration is usually expressed in parts per million (ppm) (10,000 ppm=1% concentration). It is produced at a rate of 0.4 mL/h in a healthy individual (2).

Carbon monoxide is gaseous at normal room temperature and is inhaled. It diffuses through the lung alveolar membrane almost as easily as oxygen (3). CO first dissolves in the blood. Due to its high affinity for hemeproteins containing iron, it rapidly binds to hemoglobin in erythrocytes and forms carboxyhemoglobin (COHb) (4). The affinity of hemoglobin (Hb) for CO is 230 to 270 times higher than for oxygen (3). For this reason, CO competes with oxygen to bind to Hb and reduces its oxygen carrying capacity (3,4). COHb formation in the blood is affected by many factors such as inspired CO concentration, CO exposure time, pulmonary ventilation, and exercise. It also has a high affinity for other hemeproteins such as myoglobin and cytochrome-c oxidase (3).

The main factor in acute CO poisoning is tissue hypoxia. With the formation of COHb, the oxygen carrying capacity decreases and causes insufficient oxygenation. When CO binds to an Hb subunit, the other binding sites have an increased affinity for the oxygen molecule. Therefore, CO shifts the oxygen-dissociation curve to the left, prevents oxygen separation in the low oxygen zone, and reinforces tissue hypoxia (3-5). Exposure to CO gas as low as 10 ppm increases the blood COHb level by 2%. The World Health Organization reports that levels higher than 6 ppm per minute are potentially toxic. Carboxyhemoglobin levels of 2% and over in non-smokers and 10% and over in smokers are accepted as abnormal and symptoms may occur (4).

Frequent symptoms due to CO poisoning are headaches, dizziness, weakness, nausea, vomiting, mental state changes, chest pain, shortness of breath, and loss of consciousness (6,7). Tissue hypoxia and inflammation in CO poisoning causes relative anemia and leads to anaerobic glycolysis. Therefore, together with the COHb value, patients should be evaluated for laboratory values such as blood Hb, leukocytes, lactate, and urea, the duration of the patients' trip to the emergency room, and the oxygen treatment they received during this time (4,7,8).

In this study, it was aimed to reveal the relationship between clinical symptoms and laboratory parameters (COHb, hemogram, venous blood gas, urea) of patients who visited the emergency room of our university hospital with complaints of CO poisoning.

MATERIAL AND METHODS

The study was conducted as a retrospective cross-sectional study with ethics committee approval dated 26.06.2019 and numbered 80576354-050-99/173 received from Kafkas University Faculty of Medicine Dean's Local Ethics Committee.

Study Pattern

The study was conducted by retrospectively evaluating the data of patients who visited the emergency room of Kafkas University Health Research and Training Hospital between January 1, 2014 and May 31, 2019 and who were diagnosed with CO poisoning.

Study Population and Sample

Patients who were found to be exposed to CO gas, had a detectable COHb level in the blood, and whose data file were complete, over 18 years of age were included in the study. Patients with incomplete data file, under the age of 18, smoking, pregnant, patients with systemic comorbidities (such as the presence of lung, heart disease or cancer) were excluded from the study. Thus, 143 patient files were examined, 26 patients with missing data were excluded, and 117 patients were included in the study.

Data Collection Formand Groups

A data collection form was created for the evaluation of the cases poisoned by CO gas. With this form, the patients' gender, age, place of poisoning, exposure time to CO gas, type of fuel that caused the poisoning, COHb level, complaints during admission to the emergency room, venous blood gas taken from the patients (lactate, COHb, normalized ionized calcium [NICA]), hemogram, and urea levels were recorded. When the level of COHb is between 2.5% and 10%, in brain functions may be deteriorate or it may be asymptomatic. With the level of CO-Hb is 10% and above neurological symptoms such as nausea, headache and dizziness

are observed. When the COHb level is 25% and above and exposure to acute co gas, syncope and confusion may occur (4,9,10). Hyperbaric oxygen therapy may be required especially in pregnant women and those with cancer or systemic diseases (cardiovascular, neurological diseases) (11). Therefore the COHb levels were grouped as (1) 2.5-9.9%, (2) 10-24.9%, and (3) 25% and over.

Analyses of Laboratory Parameters

The complete blood count analysis was performed using a flow cytometric method with an ABX-Pentra DX 120 analyzer (Horiba LTD, Japan) device. Serum urea analysis was performed using the enzymatic method with a Cobas C501 analyzer (Roche Diagnostic, Germany), (All kits used in analyzes were compatible with the brands of the devices). The collection tubes (Vacuette® -Greiner Bio-One GmbH, Germany) were used for serum analysis, with K2EDTA (BD Vacutainer®, BD Diagnostic, U.K.) for complete count analysis. In blood gas analysis of venous blood samples of patients taken into dried Lithium Heparin tubes (BD Vacutainer®, BD Diagnostic, U.K.); Blood lactate and NICA levels were measured by ion-selective electrode (ISE) method, and COHb levels were measured by potentiometric methodwith Radiometer ABL90 FLEX analyzer (Radiometer, Kopenhagen, Denmark) device that Co-oximeter unit. COHb level is similar for arterial and venous blood in blood gas analyzers and Co-oximetry is an accepted standard method of measurement for the evaluation of COHb (2).

Reference assay ranges of the analytical methods used are; White blood count (WBC): 3.7-10.4 ×109 L–1, Neutrophil (%): 39.9- 75.4, Lymphocyte (%): 16.1-48.7, Monocyte (%): 3.8-11.1, Basophil (%): 0-2.5, Eosinophil (%): 0.8-7.3, Hb:10.8-15.1 g/dL, Urea: 10-50 mg/dL, Lactate: 0.5-1.6 mmol/L, NICA: 0.5-2.5 mmol/L, COHb (%):0.5-2.5 (Depending on the level of carbon monoxide exposure).

Statistical Analyses

Statistical analyzes were performed using the Statistical Package for the Social Sciences (SPSS) 20.0 package program. Descriptive statistical methods (mean, median, percentage) were implemented to evaluated the data. The normal distribution of the data set was evaluated considering the ± 3 kurtosis and skewness values (12). 'One Way Anova F' was used for analysis of more than two categorical variables demonstration normal distribution (COHb level and Hg, neutrophil, lymphocyte, urea, NICA, exposure time to CO gas). "Man Whitney U" test was used for analysis two categorical variables that do not demonstration a normal distribution (COHb level with sourch of CO, and the duration of CO gas exposure with the place of origin of the patients). 'Kruskal Wallis H' test was used for analysis of more than two categorical variables that did not demonstration a normal distribution (COHb and WBC, monicide, eosinophil, basophil, lactate). The correlation between continuous variables was evaluated by Spearman correlation test. A p value of <0.05 was considered statistically significant.

RESULTS

A total of 117 patients, aged 18-82 years (median: 31), of which 55 (47%) were men and 62 (53%) were women, were included in the study. The patients' mean COHb level was 16.85±9.54% (min: 2.5%, max: 45%) and the mean exposure time to CO gas was 5.39±4.01 hours (min:10 minutes, max:15 hours). The COHb levels of the patients, the place from which they came to the emergency room, the place where the poisoning occurred, the type of fuel that caused the poisoning, and the symptoms during admission to the emergency room are given in table 1.

Carbon monoxide poisoning was mostly seen in the winter (52.1%), then the spring (33.3%), and lastly the fall (14.5%).

A statistically significant difference was found between the exposure time of the patients to CO gas and the place they traveled from to the emergency room (province-district center and rural area) (p=0.024). In addition, there was a statistically significant difference between the CO source and COHb levels of the patients (p=0.00) (Table 2).

The patients' mean WBC values were 9.67 ±3.82 ×109 L–1, the mean neutrophil (%) values were 67.77±12.68, the mean lymphocyte (%) values were 23.67 ±12.68, the mean monocyte (%) values were 5.29±3.08, the mean basophil (%) values were 0.40±0.35, the mean urea values were 33.67 ±9.82 mg/dL, the mean lactate values were 1.89±1.65 mmol/L, the mean Hb values were 15.02 ±1.90 g/dL, and the mean NICA were 0.95 ±.019 mmol/L. Examination of the COHb values obtained from the venous blood gases of the patients during the first admission to the emergency room showed that there

was a statistically significant difference regarding COHb levels and Hb (p=0.007), neutrophil (%) (p=0.028), and urea values (p=0.01). Bonferroni and Tamhane's tests were used in Post Hoc paired comparisons to find the group that created the difference. According to the Bonferroni result, the mean Hb value of those with a COHb value of ≥25% was higher than of those with a COHb value of 10-24.9%. Those with a COHb value of ≥25% had a higher urea value than those with a COHb value of 10-24.9% and those with a value of 2.5-9.9% (Table 3).

According to Tamhane's result, those with a COHb value of 10-24.9% had a higher mean neutrophil (%) value than those with a COHb value of 2.5-9.9% (Table 3). However, there was no statistically significant difference between the patients' COHb values and lymphocyte (%) (p=0.502), NICA (p=0.246), and exposure time to CO gas (h) (p=0.073) (Table 3).

A statistically significant difference between the patients' COHb levels and monocyte (%) (p=0.001), basophil (%) (p= 0.03), and lactate levels was determined (p=0.00). The corrected Bonferroni test determined that the difference was distributed among the groups. According to this result, the mean monocyte (%) value was higher in the COHb group with a value of 2.5-9.9% than the COHb group with a value of 10-24.9%, the mean basophil (%) value was higher in the COHb group with a value of 25% ≥ than the group with a COHb value of 10-24.9%, and the mean lactate value of the group with a COHb value of 25% ≥ was higher than of those patients in the other groups. However, no statistically significant difference was found between the groups in terms of COHb values and WBC (p=0.644) or eosinophil (%) (p=0.057) (Table 4).

A significant positive relationship was found between COHb and lactate levels (r=0.441, p=0.00). In addition, a significant very weak relationship was determined between COHb and Hb (r=0.188, p=0.042) and urea levels (r=0.204, p=0.027). Similarly, very weak relationships were found between lactate and leukocyte values (r=0.227, p=0.014), urea (r=0.185, p=0.045), NICA values (r=0.217, p=0.019); very weak relationships were found Hb and urea values (r=0.318, p=0.00).

Table 1. The type of fuel causing the poisoning, the place of poisoning, carboxyhemoglobin (COHb) levels, and the complaints during admission to the emergency room

Variable	n	%
Gender		
Female	62	53
Male	55	47
Came from		
Province and district center	64	54.7
Village (rural area)	53	45.3
Type of fuel		
Coal	94	80.3
Other (Biomass, fire, natural gas)	23	19.7
Place of poisoning		
Home	104	88.9
Workplace	13	11.1
Carboxyhemoglobin level (COHb) (%)		
2.5-9.9	32	27.4
10-24.9	60	51.3
25 and over	25	21.4
Complaints during admission		
Nausea-vomiting	77	65.8
Headache	60	51.3
Dizziness	55	47.0
CVS (chest pain, dyspnea)	9	7.7
Clouding of consciousness	7	6.0
Syncope	3	2.6
Stomachache	2	1.7

Table 2. Duration of CO gas exposure according to the place of origin of the patients and COHb level according to the source of CO

Exposure time (h) to carbon monoxide (CO) gas by place of origin						
Place of origin	N	Maximum	Minimum	Median	U	P
Province-district center	64	15:00	0:10	3:31	1285	0.024
Village	53	14:30	0:30	6:20		
Total	117	15:00	0:10	4:19		
Carboxyhemoglobin level (COHb) (%) relative to CO source						
Type of fuel	N	Maximum	Minimum	Median	U	P
Coal	94	45.00	2.50	18.3000	492.500	0.00
Other	23	39.30	2.50	7.3000		
Total	117	45.00	2.50	16.10		
Mann-Whitney U Test, p<0.05						

Table 3. Relationship between exposure times and hemoglobin, neutrophil (%), lymphocyte (%), urea, and NICA according to COHb level (%)

	COHb (%)	N	Mean	SD	F	*P*	***Post hoc***
Hb (g/dL)	2.5-9.9 (1)	32 (27.35%)	14.8972	1.89145	5.246	0.007*	Bonferroni 3>2
	10-24.9 (2)	60 (%51.28)	14.6557	1.99468			
	≥ 25 (3)	25 (%21.36)	16.0604	1.29332			
	Total	117 (100%)	15.0219	1.90576			
Neutrophil (%)	2.5-9.9 (1)	32 (27.35%)	63.8919	10.95135	(Welch) 3.841	0.028*	Tamhane 2>1
	10-24.9 (2)	60 (51.28%)	70.5837	11.75851			
	≥ 25 (3)	25 (21.36%)	65.9948	15.49779			
	Total	117 (100%)	67.7729	12.68817			
Lymphocyte (%)	2.5-9.9 (1)	32 (27.35%)	25.1222	10.81993	0.692	0.502	
	10-24.9 (2)	60 (51.28%)	22.4925	10.23852			
	≥ 25 (3)	25 (21.36%)	24.6744	13.94285			
	Total	117 (100%)	23.6779	11.24016			
Urea (mg/dL)	2.5-9.9 (1)	32 (27.35%)	33.0313	8.84812	7.67	0.01*	Bonferroni 3>1 3>2
	10-24.9 (2)	60 (51.28%)	31.3833	9.22063			
	≥ 25 (3)	25 (21.36%)	40.0000	10.06231			
	Total	117 (100%)	33.6752	9.82680			
NICA (mmol/L)	2.5-9.9 (1)	32 (27.35%)	.9094	.22861	1.422	0.246	
	10-24.9 (2)	60 (51.28%)	.9580	.18193			
	≥ 25 (3)	25 (21.36%)	.9956	.17652			
	Total	117 (100%)	.9527	.19539			
Exposure time (h)	2.5-9.9 (1)	32 (27.35%)	4:47	3:48	2.678	0.073	
	10-24.9 (2)	60 (51.28%)	6:29	4:02			
	≥ 25 (3)	25 (21.36%)	4:47	3:58			
	Total	117 (100%)	5:39	4:01			
One Way ANOVA F (p<0.05), Hb: Hemoglobin, NICA: Normalized Ionized Calcium, COHb: Carboxyhemoglobin							

Table 4. Blood gas and hemogram values according to the COHb level (%)

	COHb level (%)	n (%)	Median	Minimum	Maximum	X^2	*P*	*Post hoc* (Bonferroni)
WBC ($\times 10^9$ L^{-1})	2.5-9.9 (1)	32 (27.35%)	9.1000	5.15	34.60	0.879	0.644	
	10-24.9 (2)	60 (51.28%)	8.9000	4.80	14.70			
	≥ 25 (3)	25 (21.36%)	9.3000	5.50	16.70			
	Total	117 (100%)	9.1000	4.80	34.60			
Monocyte (%)	2.5-9.9 (1)	32 (27.35%)	6.4000	.00	10.20	13.240	0.001*	1>2
	10-24.9 (2)	60 (51.28%)	4.2500	.00	10.90			
	≥ 25 (3)	25 (21.36%)	5.9000	1.62	28.98			
	Total	117 (100%)	4.8000	.00	28.98			
Basophil (%)	2.5-9.9 (1)	32 (27.35%)	.3500	.00	1.20	7.046	0.030*	3>2
	10-24.9 (2)	60 (51.28%)	.2050	.00	1.00			
	≥ 25 (3)	25 (21.36%)	.5000	.10	1.40			
	Total	117 (100%)	.3000	.00	1.40			
Eosinophil (%)	2.5-9.9 (1)	32 (27.35%)	2.7000	.00	23.10	5.713	0.057	
	10-24.9 (2)	60 (51.28%)	1.5000	.00	6.56			
	≥ 25 (3)	25 (21.36%)	2.1000	.80	7.54			
	Total	117 (100%)	1.9000	.00	23.10			
Lactate (mmol/L)	2.5-9.9 (1)	32 (27.35%)	1.0500	.50	14.10	22.947	0.000*	3>2 2>1 3>1
	10-24.9 (2)	60 (51.28%)	1.5500	.40	3.70			
	≥ 25 (3)	25 (21.36%)	2.2000	.90	8.40			
	Total	117 (100%)	1.5000	.40	14.10			

Kruskal-Wallis H Test (p<0.05), WBC: White Blood Cell, COHb: Carboxyhemoglobin

DISCUSSION

Most CO poisoning occurs by accident and can be prevented (13). While CO poisoning in Turkey occurs often due to heating devices such as coal ovens and water heaters, it also occurs due to suicides, exhaust gases, and fires in other countries (14-16). In the current study,CO poisoning mostly (80.3%) occurred due to coal stoves, then gas-fired boilers, biomass burning, and lastly fires.

Previous studies on CO poisoning show that emergency room admissions are higher in winter and on windy days (14-16). In the current study, in accordance with the literature, admissions to the emergency room due to poisoning were higher during the winter months. The use of coal stoves as a means of heating in winter, especially in rural areas, causes a lower and longer exposure to CO gas (15,16). In the current study, patients coming from districts or villages took longer to come to the emergency service and were exposed longer to CO gas.

COHb measurement is necessary to diagnose acute CO poisoning. However, although it is thought that there is a relationship between blood COHb level and clinical symptoms related to CO poisoning, it is insufficient alone to determine clinical severity. The severity of clinical symptoms is related to exposure time as well as to CO concentration or blood COHb level. Although short-term exposure to high levels of CO increases COHb levels, it may not cause any symptoms whereas prolonged exposure to low levels of CO gas may cause more severe clinical symptoms. In addition, as COHb is reduced by moving away from the environment with CO gas and with oxygen therapy, it may not reflect the severity of the exposure (7). Hampton et al. reported a mean COHb level of 22.3±11.0%. However, they showed that despite the significantly higher COHb level measurements in patients with unconsciousness, it was not clinically significant (6). Ku et al. reported a mean COHb level of 21.9±17.6% and a mean exposure time to CO gas of 6±10 hours. However, they showed that these values do not have a descriptive feature in determining the severity of mortality and poisoning (8). This situation may be attributed to the toxic effects of CO at the cellular level by binding to mitochondrial proteins such as cytochrome-c, disrupting ATP production, and producing reactive oxygen species. As the COHb level decreases, cellular functions may return but neuronal necrosis or apoptosis may still occur,

especially at high COHb levels (17). In the current study, the mean COHb level according to venous blood gas analysis was 16.85±9.54% and the mean exposure time to CO gas was 5.39±4.01 hours. In addition, the most common symptoms associated with poisoning were nausea-vomiting (65.8%), headache (51.3%), and dizziness (47%). However, no relationship could be found between CO gas exposure time and COHb level and the relationship between measured COHb level and symptoms was not clinically significant.

Lactate, which is associated with tissue hypoxia and inflammation, rises due to anaerobic glycolysis and correlates with COHb. Moon et al. reported that as the COHb level increases, lactate also increases, and there is a weak correlation between them. In addition, patients with high blood lactate levels also have leukocytosis and hyperglycemia (18). Sokal et al. reported a weak relationship between blood lactate levels and COHb ($r=0.340$, $p<0.05$) (19). In the current study, there was a weak correlation between COHb and lactate ($r=0.441$, $p=0.00$) and lactate and leucocytes ($r=0.227$, $p=0.014$).

Inflammation in tissues, together with tissue hypoxia caused by CO poisoning due to the formation of COHb and relative anemia, may be another factor that causes an inability to establish a relationship between COHb level and clinical findings (7,8). Ku et al. reported the mean value of COHb was 21.9±17.6%, Hb was 13.8±2.6 g/dL, WBCwas 21.3±9.6 ×109 L–1, blood urea nitrogen was 21.9±26.3 mg/dL, and blood calcium was 8.2±1.1 mg/dL (8). In the study on the examination of cardiac markers by Özdemir et al., the mean COHb was 25.35±8.1%, Troponin I value was 0.001 ng/mL, mean Hb value was 13.4±1.6 g/dL, and mean leukocyte value was 10.5±3.6 ×109 L–1 (20). In the current study, the mean COHb level was 16.85±9.54% and the mean Hb (16.06 g/dL) and urea (40.0 mg/dL) values were higher in the group with a COHb value of ≥25%. In addition, the neutrophil value (70.58%) was higher in the group with a COHb value of 10-24.9% and the monocyte (6.4%) value was higher in the group with a COHb value of <10%. The mean WBC was similar in all three groups and within normal limits (mean 9.67±3.82 ×109 L–1) and the mean NICA value was 0.95±.019 mmol/L. We believe that the higher mean Hb value in the current study compared to previous studies is related to the geographical conditions (high altitude) of the province where the study was conducted.

STUDY LIMITATION

Some limitations should be considered when examining the results in our study. Post-emergency treatment data of the patients were not available. The small sample may limit the generalizability of the findings. The duration of exposure to CO gas and oxygen administration during transport to the emergency room may have caused changes in COHb values measured in patients.

CONCLUSION

In conclusion, CO poisoning is more common in winter months. Although coal stoves are responsible for most cases of CO poisoning, poisonings due to water heaters, exhaust gas, biomass, or natural gas can also be seen. Therefore, awareness-raising activities for heating devices, especially in rural areas where it takes longer to access emergency services, will help reduce the number of poisoning cases. Blood COHb values measured for diagnosis can be affected by the length of exposure time of patients, the time until the emergency room is reached, and oxygen applications applied during this time. High COHb values, an indicator of CO poisoning, are not always related to the severity of clinical symptoms. Because the increase in COHb also causes an increase in lactate levels, we believe that the lactate level can be used as a follow-up indicator. COHb level and biochemical parameters were not directly related to clinical symptoms in acute CO poisoning.

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