



Evaluation of Patients Presenting to the Pediatric Emergency Department with Carbon Monoxide Poisoning

Çocuk Acile Karbonmonoksit Zehirlenmesi ile Başvuran Hastaların Değerlendirilmesi


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ABSTRACT

Aim: Carbon monoxide poisoning is one of the significant causes of intoxication and presentation to pediatric emergency departments, especially in winter. The primary aim of this study was to evaluate the demographic characteristics, and clinical and laboratory findings of pediatric patients who presented to the pediatric emergency department with carbon monoxide poisoning, and the secondary aim was to examine the laboratory values of the patients with impaired consciousness. **Material and Methods:** The demographic and clinical characteristics and laboratory values of 162 patients presented to the pediatric emergency department due to carbon monoxide poisoning between 2017 and 2020 were retrospectively analyzed. The laboratory parameters of the patients with and without symptoms of impaired consciousness were compared.

Results: The mean age of the patients was 8.94±5.33 years, and the gender distribution was homogenous. The highest frequency of presentation was during winter. Nausea, vomiting, and headache were the most common symptoms. Fifteen of the patients had impaired consciousness. While the laboratory values of the patients were generally within normal ranges, patients with high carboxyhemoglobin, lactate, and troponin values, and low pH were encountered. The carboxyhemoglobin and lactate levels of patients with impaired consciousness were found to be significantly higher than the patients without impaired consciousness (p<0.001 and p=0.019, respectively).

Conclusion: Elevated carboxyhemoglobin and lactate levels were associated with impaired consciousness. Although carboxyhemoglobin levels are important for diagnosis and clinical follow-up, they should not be used as the only marker. High lactate and troponin levels, and low pH should also be taken into account.

Keywords: Carbon monoxide; children; impaired consciousness; poisoning.

ÖZ

Amaç: Karbonmonoksit zehirlenmeleri özellikle kış mevsiminde zehirlenme ile çocuk acil servisine başvuruların önemli nedenlerinden biridir. Bu çalışmanın birincil amacı çocuk acil servisine karbonmonoksit zehirlenmesi ile başvuran çocuk hastaların demografik özellikleri ile klinik ve laboratuvar bulgularını değerlendirmek, ikincil amacı ise karbonmonoksit zehirlenmesine bağlı bilinç değişikliği gelişen hastaların laboratuvar değerlerini incelemektir.

Gereç ve Yöntemler: 2017 ve 2020 yılları arasında çocuk acil servisine karbonmonoksit zehirlenmesi nedeniyle başvurmuş olan 162 hastanın demografik ve klinik özellikleri ile laboratuvar değerleri geriye dönük olarak incelendi. Bilinç değişikliği semptomu olan ve olmayan hastaların laboratuvar parametreleri karşılaştırıldı.

Bulgular: Hastaların ortalama yaşı 8,94±5,33 yıl idi ve cinsiyet dağılımları homojendi. En yüksek başvuru sıklığı kış mevsimindeydi. Bulantı, kusma ve baş ağrısı en sık görülen semptomlardı. Hastaların on beşinde bilinç değişikliği vardı. Hastaların laboratuvar değerleri genel olarak normal aralıklarda iken karboksihemoglobin, laktat ve troponin değerleri yüksek olan ve pH değeri düşük olan hastalar da olduğu tespit edildi. Bilinç değişikliği olan hastaların karboksihemoglobin ve laktat düzeyi, bilinç değişikliği olmayan hastalara göre anlamlı şekilde daha yüksek bulundu (sırasıyla p<0,001 ve p=0,019).

Sonuç: Yüksek karboksihemoglobin ve laktat seviyesi bilinç değişikliği ile ilişkili bulunmuştur. Karboksihemoglobin düzeyi tanı ve klinik takip için önemli olmakla birlikte tek başına bir belirteç olarak kullanılmamalıdır. Yüksek laktat ve troponin değerleri ve düşük pH değeri de dikkate alınmalıdır.

Anahtar kelimeler: Karbonmonoksit; çocuk; bilinç değişikliği; zehirlenme.

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INTRODUCTION

Carbon monoxide (CO) is a toxic gas called the "silent killer". This non-irritant gas is formed as a result of the incomplete combustion of carbon-containing materials (1,2). CO poisoning is an important public health problem in Turkey and across the world, especially in the winter months. Smoke from fires, inadequate maintenance of heating systems, and vehicle exhaust gases are the most common causes of CO poisoning. The most common reason encountered in Turkey is the inadequacy of waste gas removal in heating and hot water sources (3).

CO causes toxicity through hypoxic and inflammatory processes and can affect many systems. CO poisoning may present with non-specific symptoms, such as headache, nausea, vomiting, abdominal pain, respiratory distress, weakness, and chest pain, and it is a significant cause of morbidity, coma, and death in severe cases (4). In a study evaluating 20 years of data from Turkey, it was found that 1.42% of forensic deaths were caused by CO poisoning (5). In the United States, the Centers for Disease Control and Prevention reported that approximately 21,000 patients were admitted to the emergency department annually due to non-fire carbon monoxide poisoning, and 450 patients died (6).

The diagnosis of CO poisoning is made based on the medical history, clinical examination, and carboxyhemoglobin (COHb) levels of patients. However, the clinical symptoms and severity of acute CO poisoning are not always related to COHb concentrations at presentation (7-9). In such cases, markers including increased lactate and troponin levels as indicators of tissue hypoxia and the presence of metabolic acidosis may be useful in determining the severity of poisoning.

The primary aim of this study was to evaluate the demographic characteristics, clinical findings, and laboratory findings of pediatric patients who presented to the pediatric emergency department with carbon monoxide poisoning, and the secondary aim was to examine the laboratory values of the patients with impaired consciousness.

MATERIAL AND METHODS

In this study, patients aged one month to 18 years, who were brought to the pediatric emergency department of the University of Health Sciences Gülhane Training and Research Hospital between 1 January 2017 and 31 December 2020 due to CO poisoning, were retrospectively evaluated. The diagnosis of CO poisoning was made according to a history of exposure to CO, clinical findings, and high COHb levels. A COHb level above 5% was considered to be high (4). Patients with a history of chronic diseases (pulmonary, cardiovascular, and neurological diseases) were excluded from the study. The patients' demographic characteristics (age and sex), signs and symptoms, laboratory values (COHb, blood pH, lactate, troponin I, leukocyte, and platelet counts), treatments, and follow-up data were evaluated. A lactate level of >2 mmol/l was considered significant (10,11). There are insufficient data on reference ranges for cardiac Troponin I levels indicating cardiac injury in children (12). In this study, a significant elevation was accepted based on the reference upper Troponin I level of our hospital laboratory. Normobaric oxygen (NBO) therapy was given

to all patients. If patients had acute cardiac or neurological findings, metabolic acidosis, or high COHb values ($>25\%$), hyperbaric oxygen (HBO) therapy was given (13). We compared the laboratory results of the patients with or without impaired consciousness at the time of admission to the hospital. Impaired consciousness was assessed using the Glasgow coma scale (GCS).

To collect data, approval was obtained from the Clinical Research Ethics Committee of the University of Health Sciences, Gülhane Training and Research Hospital with the decision dated 17 June 2021 and numbered 2021/226, and official permission was received from the Medical Specialization Education Board. The study was carried out according to the principles of the Declaration of Helsinki.

Statistical Analysis

The data obtained were analyzed using the Statistical Package for the Social Sciences (SPSS) version 23.0. The data obtained by measurements were expressed as mean \pm standard deviation, median, interquartile range, and minimum-maximum values, and the grouped data are expressed as frequencies (percentages). The normality of the distribution of the data was checked by the Kolmogorov-Smirnov test, and the assumption of normality was violated for all quantitative variables. The Mann-Whitney U test was used to compare the quantitative data for the variables that did not show normal distribution between the two groups. The categorical data were analyzed using the chi-square test and Fisher's exact test. The level of statistical significance was accepted as $p<0.05$.

RESULTS

During the period covered in the study, 162 patients were diagnosed with CO poisoning in the pediatric emergency department. The mean age of the patients was 8.94 ± 5.33 years (1 month-17 year), and 81 (50%) were female. Seventeen patients were foreign nationals. The times of the day with the highest frequency of admissions were between 00:01 and 06:00 in the winter months (December and January). Among the patients, 53.1% ($n=86$) were brought to the emergency department by the 112 ambulance service. More than half ($n=107$) of the patients had no complaints and were brought to the hospital with the suspicion of poisoning because they had a family history of CO poisoning. Percentages for symptoms are given over the total number of patients. The most common symptoms during admission were nausea (43.6%), vomiting (32.7%), and headache (32.7%). Twenty-five patients had multiple symptoms. Fifteen (27.3%) cases had a history of impaired consciousness (sleepiness in nine patients, syncope in four, and convulsions in two). In the physical examinations, three patients had a GCS score of 14 and had only a tendency to sleep. Other systemic examinations were normal in all patients. The demographic and clinical characteristics of the patients were given in Table 1. The electrocardiograms of all patients were within normal limits.

COHb levels at the time of admission were 20% or higher in 48 (29.6%) of the patients, lactate levels in blood gas tests were high in 76 (46.9%), and troponin I levels were high in 12 (7.4%). The laboratory values of the patients are presented in Table 2.

Table 1. Demographic and clinical characteristics of patients

Age (years), mean±SD (min-max)	8.94±5.33 (1-17)
Gender, n (%)	
Female	81 (50.0)
Male	81 (50.0)
Admission time, n (%)	
00:01-06:00	46 (28.4)
06:01-12:00	36 (22.2)
12:01-18:00	38 (23.5)
18:01-00:00	42 (25.9)
Means of transport to the hospital, n (%)	
112 ambulance service	86 (53.1)
By own means	76 (46.9)
Admission season, n (%)	
Winter	94 (58.0)
Spring	44 (27.2)
Summer	8 (4.9)
Fall	16 (9.9)
Has symptoms, n (%)	
Yes	55 (34.0)
No	107 (66.0)
Signs and symptoms at admission[#], n (%)	
Nausea	24 (43.6)
Vomiting	18 (32.7)
Headache	18 (32.7)
Impaired consciousness	15 (27.3)
Dizziness	14 (25.4)
Cough	2 (3.6)
Fatigue	2 (3.6)
Shortness of breath	1 (1.8)

SD: standard deviation, min: minimum, max: maximum, #: twenty-five patients had multiple symptoms, percentages for symptoms were given over the total number of patients

Table 2. Laboratory parameters of patients

	Mean±SD	Median (IQR) [min-max]
COHb (%)	13.08±10.23	13.6 (19.1) [0.2-38.9]
pH	7.38±0.05	7.40 (0.05) [7.08-7.50]
Lactate (mmol/L)	2.42±1.42	2.0 (1.4) [0.8-11.5]
Troponin-I (pg/mL)	11.36±22.96	4.5 (5.8) [0.9-150]
WBC (x10 ³ /mm ³)	10.01±3.23	9.5 (4.3) [2.2-21.2]
PLT (x10 ³ /mm ³)	314.48±8.24	304 (108) [181-753]
Hemoglobin (g/L)	13.12±1.46	13.1 (1.7) [9.0-17.6]
Urea (mg/dL)	25.95±7.18	25.5 (10.0) [8-45]
Creatinine (mg/dL)	0.60±0.18	0.56 (0.23) [0.33-1.50]
AST (IU/L)	29.58±13.61	28 (15) [4-114]
ALT (IU/L)	17.83±14.12	14 (8) [4-108]

SD: standard deviation, IQR: interquartile range, min: minimum, max: maximum, COHb: carboxyhemoglobin, WBC: white blood cell, PLT: platelet, AST: aspartate aminotransferase, ALT: alanine aminotransferase

Table 3. Characteristics of patients with and without impaired consciousness due to carbon monoxide poisoning

	With IC (n=15)		Without IC (n=147)		p
	Mean±SD	Median (IQR) [min-max]	Mean±SD	Median (IQR) [min-max]	
Age (years)	11.73±5.44	14 (10) [2-17]	8.66±5.25	9 (9) [1-17]	0.027
COHb (%)	22.76±8.91	23.8 (8.9) [3.0-38.9]	12.10±9.86	12.0 (18.5) [0.2-36.3]	<0.001
pH	7.34±0.10	7.38 (0.10) [7.08-7.44]	7.39±0.40	7.40 (0.06) [7.27-7.50]	0.130
Lactate (mmol/L)	3.60±2.73	2.5 (2.3) [1.7-11.5]	2.30±1.17	2.0 (1.3) [0.8-6.8]	0.019
Troponin-I (pg/mL)	20.85±43.79	3.3 (6.8) [1.8-150]	10.22±19.13	4.6 (5.8) [0.9-141]	0.796
WBC (x10 ³ /mm ³)	9.93±2.98	7.8 (3.6) [5.9-18.0]	10.02±3.27	9.5 (4.4) [2.2-21.2]	0.944
PLT (x10 ³ /mm ³)	275.40±6.37	229 (79) [186-381]	319.13±8.37	307 (110) [181-753]	0.047
Length of hospital stay (hours)	12.13±5.40	12 (4) [6-24]	5.93±4.10	6 (2) [2-34]	<0.001

IC: impaired consciousness, SD: standard deviation, IQR: interquartile range, min: minimum, max: maximum, COHb: Carboxyhemoglobin, WBC: white blood cell, PLT: platelet

The demographic characteristics and laboratory findings of the patients with and without impaired consciousness were given in Table 3. The mean ages of the patients with and without impaired consciousness were 11.73±5.44 and 8.66±5.25 years, respectively, and a statistically significant difference was found between the two groups in terms of age (p=0.027). The COHb and lactate levels of the patients with impaired consciousness were significantly higher than those of the patients without impaired consciousness (p<0.001 and p=0.019, respectively). Although troponin I levels were higher in the patients with impaired consciousness, this difference was not statistically significant (p=0.796). There was also no significant difference between the groups with and without impaired consciousness in terms of their leukocyte counts (p=0.944), while platelet counts were significantly higher in the group without impaired consciousness (p=0.047).

All patients were followed up in the pediatric emergency department with 100% oxygen therapy. Additionally, HBO therapy was applied to 30 (18.5%) patients. The mean hospital stay duration of the patients was 6.5±4.6 hours. The patients with impaired consciousness were followed up statistically significantly longer in the hospital compared to the group without impaired consciousness (p<0.001). All patients were discharged from the pediatric emergency department with recovery.

DISCUSSION

Carbon monoxide poisoning is one of the most common causes of pediatric emergencies presenting with intoxication, in both Turkey and the world. The causes of CO poisoning vary according to geographical regions, as well as social and cultural factors (14).

CO poisoning often develops due to acute accidents in childhood. It has been stated that CO poisoning is generally caused by fire and exhaust fumes in developed countries. In Turkey, it has been reported that the sources of poisoning are coal stoves and water heaters working with gas cylinders or natural gas (3,15,16). In this study, 55.6% of the patients presented to the hospital with CO poisoning due to smoke inhalation, and 44.4% presented due to cylinder gas and natural gas exposure. In the literature, it has been shown that CO poisoning is frequently seen in the winter months (9,17,18). In the present study, we determined that the patients were brought to the emergency department most frequently in the winter season and at night (00:01-06:00).

Poisoning develops with the release of CO formed by the incomplete combustion of carbon-containing compounds. Clinical findings in CO poisoning may be non-specific; however, these cases can cause symptoms through damage to many organs that need more oxygen, especially the brain and the heart. The most common symptoms have been reported as nausea, vomiting, and headache (15,19,20).

In the present study, the most common signs and symptoms were nausea, vomiting, and headache. CO poisoning should be considered in the differential diagnosis of patients presenting to the emergency department with unexplained nausea, vomiting, and headache, especially in the winter months.

Severe CO poisoning can cause neurological symptoms such as seizures, syncope, or impaired consciousness, as well as cardiac symptoms such as myocardial ischemia and ventricular arrhythmias, and metabolic findings related to lactic acidosis (21).

In this study, myocardial ischemia and arrhythmias did not develop in any of the patients during follow-up. Impaired consciousness (sleepiness, syncope, and convulsions) was observed in fifteen patients.

Hemogram parameters can also be affected in cases of CO poisoning. Ertekin et al. (22) determined that leukocyte and platelet values increased in patients with CO poisoning in comparison to healthy controls. In another study, platelet and leukocyte counts did not significantly differ between CO poisoning cases of mild and moderate severity (23). In the study conducted by Coşkun et al. (24), patients were divided into 2 groups as those with severe poisoning and those without severe poisoning, and leukocyte values were found to be high in the severe poisoning cases. In two studies, leukocyte values were found to be high in CO poisoning cases that required HBO treatment (25,26). In the study carried out by Bağcı et al. (27), who divided patients presenting with CO poisoning into two groups as mild-moderate and severe, no difference was found between the two groups in terms of platelet counts. In the present study, there were patients with leukocytosis and thrombocytosis. The platelet counts were significantly higher in the group without impaired consciousness. There was no statistically significant difference between the groups in terms of their leukocyte counts. These results may be due to the limited number of patients included in the study.

Previous studies have shown that patients with severe system involvement have higher COHb levels (7,9,28). When COHb levels rise above 20%, the heart and brain are severely affected (29). In a study conducted in Turkey, it was found that a COHb level of >20% measured at the time of admission in pediatric CO poisoning cases can be used to predict target organ damage (30). Similarly, in this study, the mean COHb level was found to be above 20% in the patients with impaired consciousness at presentation, which was significantly higher than in the patients without impaired consciousness.

In CO poisoning, lactate levels increase due to tissue hypoxia and cytotoxicity, and metabolic acidosis is observed (31,32). However, the relationship between a high lactate level and the severity of clinical findings is debated. Many studies have shown that

morbidity and mortality rates are higher in patients with lactate levels of >2 mmol/L (10,11).

In the literature, lactate levels have been found to be significantly higher in patients with neurological symptoms related to CO poisoning (33,34). In this study, the lactate levels of the patients with impaired consciousness at the time of their presentation were found to be significantly higher than those without impaired consciousness, but no significant difference was found between the groups in relation to pH levels. In patients presenting with CO poisoning, there is a need to closely monitor serum lactate levels.

In cases of CO poisoning, the risk of developing toxicity is high in tissues that are more sensitive to hypoxia, such as the central nervous system and the cardiovascular system. Therefore, in patients admitted to the hospital with suspected CO poisoning, troponin I levels should be measured as an important variable in demonstrating myocardial injury. Studies have shown that troponin I levels increase in CO poisoning cases (35,36). In the present study, the mean troponin level of the entire patient group (11.36 ± 22.96 pg/mL) was within normal limits, and troponin I elevation was detected in only 12 patients. Troponin I levels were found to be higher in the patients with impaired consciousness, but there was no statistically significant difference compared to the patients without impaired consciousness. We consider that in pediatric CO poisoning cases, cardiac enzyme levels should be monitored closely during both admission and follow-up.

HBO therapy is applied in the presence of cardiac and neurological involvement, end-organ damage, metabolic acidosis, worsening or no regression of symptoms despite four-hour NBO therapy, and a COHb level above 25% (13). In this study, 30 patients were treated with HBO therapy in accordance with these criteria. All patients were followed up in the emergency department and were discharged without any problem.

This study had certain limitations. The small number of patients was one of these limitations. Moreover, the duration of the CO exposure of the patients and the time of arrival at the hospital could not be evaluated. Additionally, the data reflected the experience of a single center, and the study had a retrospective design. There is a need for prospectively designed, larger multicenter case series on CO poisoning.

To sum up, in the present study, the patients with impaired consciousness due to CO poisoning had lower serum pH and higher COHb, lactate, and troponin I levels, and these patients were followed up in the emergency department for a longer period. Despite this, the COHb levels of three of our patients with neurological involvement were below 20%.

CONCLUSION

Although high COHb levels are important for the diagnosis of CO poisoning, low values do not exclude the possibility of this condition. COHb levels alone should not be used as a marker for treatment and follow-up. Lactate, troponin I, and pH levels should also be evaluated in pediatric patients presenting to the hospital with CO poisoning.

Ethics Committee Approval: The study was approved by the Ethics Committee of the University of Health Sciences, Gülhane Training and Research Hospital (17.06.2021, 226).

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REFERENCES

- Byard RW. Carbon monoxide - the silent killer. *Forensic Sci Med Pathol.* 2019;15(1):1-2.
- Ernst A, Zibrak JD. Carbon monoxide poisoning. *N Engl J Med.* 1998;339(22):1603-8.
- Uysalol M, Paslı Uysalol E, Kayaoğlu S, Varol Saraçoğlu G. A retrospective analysis of pediatric patients admitted to the pediatric emergency service for carbon monoxide intoxication. *Balkan Med J.* 2011;28(3):237-43.
- Tomaszewski C. Carbon monoxide. In: Nelson LS, Howland MA, Lewin NA, Smith SW, Goldfrank LR, Hoffman RS, editors. *Goldfrank's toxicologic emergencies.* 11th ed. New York: McGraw-Hill; 2019.
- Yetiş Y, Karbeyaz K, Güneş A. 20-year analysis of deaths due to carbon monoxide poisoning in Eskisehir. *ADYÜ Sağlık Bil Derg.* 2017;3(1):396-406. Turkish.
- cdc.gov [Internet]. Centers for Disease Control and Prevention. Carbon monoxide poisoning. [Updated: 2018 September 16; Cited: 2019 March 28]. Available from: <https://www.cdc.gov/co/>.
- Raub JA, Mathieu-Nolf M, Hampson NB, Thom SR. Carbon monoxide poisoning--a public health perspective. *Toxicology.* 2000;145(1):1-14.
- Shimazu T. Pathophysiology, myths and mysteries of acute carbon monoxide poisoning. *Chudoku Kenkyu.* 2006;19(1):23-33. Japanese.
- Hampson NB, Hauff NM. Carboxyhemoglobin levels in carbon monoxide poisoning: do they correlate with the clinical picture? *Am J Emerg Med.* 2008;26(6):665-9.
- Jones AE, Shapiro NI, Trzeciak S, Arnold RC, Claremont HA, Kline JA, et al. Lactate clearance vs central venous oxygen saturation as goals of early sepsis therapy: A randomized clinical trial. *JAMA.* 2010;303(8):739-46.
- Aydın A, Kaçmaz O, Öterkuş M, Miniksar ÖH. The relationship between MPV, RDW, lactate, sodium and albumin levels and mortality in intensive care patients. *Dicle Med J.* 2022;49(1):168-75. Turkish.
- Clerico A, Aimo A, Cantinotti M. High-sensitivity cardiac troponins in pediatric population. *Clin Chem Lab Med.* 2021;60(1):18-32.
- Weaver LK, Hopkins RO, Chan KJ, Churchill S, Elliott CG, Clemmer TP, et al. Hyperbaric oxygen for acute carbon monoxide poisoning. *N Engl J Med.* 2002;347(14):1057-67.
- Baum CR. What's new in pediatric carbon monoxide poisoning? *Clin Ped Emerg Med.* 2008;9(1):43-6.
- Cho CH, Chiu NC, Ho CS, Peng CC. Carbon monoxide poisoning in children. *Pediatr Neonatol.* 2008;49(4):121-5.
- Kurt F, Bektaş Ö, Kalkan G, Öncel MY, Yakut HI, Kocabaş CN. Does age affect presenting symptoms in children with carbon monoxide poisoning? *Pediatr Emerg Care.* 2013;29(8):916-21.
- Ait El Cadi M, Khabbal Y, Idrissi L. Carbon monoxide poisoning in Morocco during 1999-2007. *J Forensic Leg Med.* 2009;16(7):385-7.
- Salameh S, Amitai Y, Antopolsky M, Rott D, Stalnicowicz R. Carbon monoxide poisoning in Jerusalem: epidemiology and risk factors. *Clin Toxicol (Phila).* 2009;47(2):137-41.
- Jüttner B, Busch HJ, Callies A, Dormann H, Janisch T, Kaiser G, et al. S2k guideline diagnosis and treatment of carbon monoxide poisoning. *Ger Med Sci.* 2021;19:Doc13.
- Eichhorn L, Thudium M, Jüttner B. The diagnosis and treatment of carbon monoxide poisoning. *Dtsch Arztebl Int.* 2018;115(51-52):863-70.
- Nañagas KA, Penfound SJ, Kao LW. Carbon monoxide toxicity. *Emerg Med Clin North Am.* 2022;40(2):283-312.
- Ertekin B, Koçak S, Acar T, Öztürk E, Demir LS. Role of whole blood markers in carbon monoxide poisoning. *Cukurova Med J.* 2019;44(Suppl 1):197-201. Turkish.
- Tursun S, Alpcan A, Karahan İ. The retrospective analysis of carbon monoxide poisoning admissions to pediatric emergency room of a university hospital. *KÜ Tıp Fak Derg.* 2020;22(2):199-207. Turkish.
- Coşkun A, Eren FA, Eren ŞH, Korkmaz İ. Predicting of neuropsychosis in carbon monoxide poisoning according to the plasma troponin, COHb, RDW and MPV levels: Neuropsychoses in carbon monoxide poisoning. *Am J Emerg Med.* 2019;37(7):1254-9.
- Karaman K, Armagan HH. Predictive value of platelet lymphocyte ratio in carbon monoxide poisoning. *Ann Med Res.* 2021;28(9):1754-7.
- Atiş ŞE, Sarıkaya T, Yamaç A. The relationship of blood parameters with the severity of carbon monoxide poisoning. *J Contemp Med.* 2022;12(4):481-4.
- Bağcı Z, Arslan A, Arslan D. The value of neutrophil:lymphocyte ratio and platelet:lymphocyte ratio in predicting clinical severity in children with carbon monoxide poisoning. *Indian J Pediatr.* 2021;88(11):1121-6.
- Keleş A, Demircan A, Kurtuluş G. Carbon monoxide poisoning: how many patients do we miss? *Eur J Emerg Med.* 2008;15(3):154-7.
- Bleecker ML. Carbon monoxide poisoning. *Handb Clin Neurol* 2015;131:191-203.
- Yurtseven A, Ulaş Saz E. Carboxyhemoglobin levels should be considered in carbon monoxide poisoning. *J Curr Pediatr.* 2017;15(2):10-8. Turkish.
- Guzman JA. Carbon monoxide poisoning. *Crit Care Clin.* 2012;28(4):537-48.
- Weaver LK. Clinical practice. Carbon monoxide poisoning. *N Engl J Med.* 2009;360(12):1217-25.

33. Besli GE, Ergüven M, Karadoğan M, Yılmaz Ö. Carbon Monoxide Poisoning in Children. *Eurasian J Emerg Med.* 2010;9(1):26-30. Turkish.
34. 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(8):1372-5.
35. Cha YS, Cha KC, Kim OH, Lee KH, Hwang SO, Kim H. Features and predictors of myocardial injury in carbon monoxide poisoned patients. *Emerg Med J.* 2014;31(3):210-5.
36. Boztepe H, Yalaki Z, Bilge YD. Evaluation of neurological and cardiological findings in carbonmonoxide poisoning in children. *Turk Arch Pediatr.* 2014;49(4): 314-22.