RESEARCH ARTICLE

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Clinical and Magnetic Resonance Imaging Findings of Patients with Acute Carbon Monoxide Poisoning

ABSTRACT

Objective: We aim to evaluate the demographic and clinical characteristics of patients with acute carbon monoxide (CO) poisoning, who had a Glasgow Coma Score (GCS) below 15, and who had cerebral lesions detected in magnetic resonance imaging (MRI).

Methods: The age, gender, causes of CO intoxication, clinical signs, neurological findings, GCS, blood carboxyhemoglobin level (COHb), serum pH, lactate, creatine kinase (CK), creatinine kinase-myocardial band MB (CK-MB), troponin-I level, brain MRI (T1-weighted, T2-weighted, FLAIR and diffusion-weighted imaging), treatment, and mortality status of 327 patients were evaluated retrospectively.

Results: The median age of patients was 31.5 years (IQR=19.5 years), 72.2% of the patients were women. Neurological findings were detected in 34 (10.4%) of the patients. The frequency of dyspnea was significantly higher in patients with neurological findings (p<0.05). The COHb and lactate levels of patients with neurological findings were found to be significantly high, the pH level was significantly lower (p<0.05). There was no significant relationship between the presence of neurological findings and CK, CK-MB, and troponin-I levels (p>0.05). Patients with neurological findings were found to have a significantly longer follow-up period, more frequently received hyperbaric oxygen therapy (p<0.05). The rate of hospitalization was 10.7%, the mortality rate was 0.9%. Hospitalization and mortality rates were significantly high in patients with neurological findings (p <0.05). Pathological findings were detected in 13 (40.6%) of 32 of patients (except for 2 patients who did not respond to the resuscitation) who had an MRI.

Conclusions: It was determined that acute CO poisoning may lead to acute brain damage, 40.6% would be detected in brain MRIs taken in patients during the acute phase.

Keywords: Carboxyhemoglobin, Emergency Department, Magnetic Resonance Imaging, Mortality

Akut Karbonmonoksit Zehirlenmesi olan Hastalarının Klinik ve Manyetik Rezonans Görüntüleme Bulguları

ÖZET

Amaç: Bu çalışmada akut karbonmonoksit (CO) zehirlenmesi tanısı konulan hastaların demografik ve klinik özellikleri ile Glasgow Koma Skoru (GKS) 15'in altında olan hastaların manyetik rezonans görüntüleme (MRG)'de tespit edilen serebral lezyonları tanımlamayı amaçladık.

Gereç ve Yöntem: 327 hasta yaş, cinsiyet, CO zehirlenme nedenleri, klinik belirtileri, nörolojik bulguları, GKS'ları, karboksihemoglobin (COHb), serum pH, laktat, kreatin kinaz (CK), kreatini kinaz, miyokardiyal band (CK-MB), troponin-I düzeyleri ile beyin MRG (T1 ağırlıklı, T2 ağırlıklı, FLAIR, difüzyon ağırlıklı görüntü) bulguları ve mortalite durumları açısından retrospektif olarak değerlendirildi.

Bulgular: Çalışmamızda hastaların yaş ortancası 31,5 yıl (IQR=19.5 yıl) olup, hastaların %72,2'si kadındı. Hastaların 34 (%10,4)'ünde nörolojik bulgular saptandı Nörolojik bulgusu olan hastaların dispne sıklığı anlamlı olarak yüksekti (p<0,05). Çalışmamızda nörolojik bulgusu olan hastaların COHb düzeyi ve laktat düzeyi anlamlı olarak yüksek, pH düzeyi anlamlı olarak düşük saptandı (p<0,05). Nörolojik bulgu varlığının CK, CK-MB ve troponin-I düzeyi arasında anlamlı bir ilişki saptanmadı (p>0,05). Nörolojik bulgusu olan hastaların, takip süresi anlamlı olarak uzun olduğu, daha sıklıkla hiperbarik oksijen tedavisi aldığı saptandı (p<0,05). Çalışmamızda olguların yatış oranı % 10,7, mortalite oranı % 0,9 olarak saptandı. Nörolojik bulgusu olan hastalarda yatış ve mortalite oranları anlamlı olarak yüksek saptandı (p<0,05). MRG çekilen 32 olgunun (resistasyona yanıt vermeyen 2 hasta haricinde) 13'ünde (%40,6) patolojik bulgulara rastlandı.

Sonuç: Akut karbonmonoksit zehirlenmelerinin akut beyin hasarına yol açabileceği, klinik bulgu veren bu hastalarda akut dönemde çekilen MRG'sinde %40,6 oranında bulgu vereceği saptandı.

Anahtar Kelimeler: Karboksihemoglobin, Acil Servis, Manyetik Rezonans Görüntüleme, Mortalite

INTRODUCTION

Carbon monoxide (CO) poisoning is one of the most important public health problems in Turkey that can result in death if neglected. CO is a colorless, odorless, tasteless, and non-irritant gas that occurs as a result of carbon-containing fuels not being burned until the end product (1,2). CO poisoning is a clinical condition that usually develops due to the incomplete combustion of fuels used for heating such as stoves and natural gas. Also, exposure to fire and exhaust fumes, smoking, or hookah smoking leads to CO poisoning (1).

The tissues are exposed to hypoxia as the oxygen rate in the tissues of the patient exposed to CO gas decreases. The central nervous system and cardiovascular system are mostly affected by this condition.

There are publications that report that CO poisoning accounts for 34% of all poisonings (1). It was reported that deaths due to CO poisoning are in the first rank among all poisonings, and the mortality rate related to this is between 1-4.3% (3,4). Cardiac disorders (arrhythmia, left ventricular dysfunction) and up to 40% neuropsychological disorders may occur in approximately one-third of moderate and severe poisoning patients (4,5).

Symptoms of CO poisoning patients who are referred to the Emergency Department (ED) range from a mild headache to coma (6). Although the frequency of acute brain injury (ABI) due to CO poisoning has been shown to be around 37% in a study (7), little is known about the lesions developing in the acute period and the clinics that these lesions will cause (8). The diagnosis of ABI can be difficult, especially because it causes changes in the symptoms and findings of patients who use hypnotic drugs and alcohol (9).

Since correct treatment can reduce morbidity in CO poisoning, it is important to diagnose ABI in these patients (9). Magnetic resonance imaging (MRI) is a sensitive imaging method that identifies cerebral lesions in the acute period of CO poisoning (2,9).

In our study, we aimed to examine the demographic and clinical features of patients diagnosed with acute CO intoxication in the ED, and the cerebral lesions detected on MRI in patients with Glasgow Coma Score (GCS) below 15.

MATERIAL AND METHODS

Patients diagnosed with CO intoxication in the ED between December 1, 2015, and December 1, 2018, were evaluated retrospectively after obtaining approval from the hospital ethics board. 479 patients were diagnosed with acute CO intoxication in the ED, and 327 patients who met the criteria were included in the study. Age, gender, causes of CO exposure, symptoms during admission, duration of treatment, hospitalization rate, mortality rate, and GCS were determined. Blood carboxyhemoglobin level (COHb), pH,

serum lactate level, creatine kinase (CK), creatine kinase-myocardial band MB (CK-MB), and troponin-I levels, as well as MRI findings and prognoses, examined. Patients over 18 years of age diagnosed with acute CO intoxication after admission to the ED were included in the study. Patients with a lack of information in the patient files, and patients with a history of migraine, multiple sclerosis, Alzheimer's disease, dementia, hemorrhagic or ischemic stroke in the past, traumatic brain damage, and sequelae neurological findings were excluded from the study.

Syncope, loss of consciousness, altered mental status, decrease in GCS into 14 or below, isolated unable to speech or disorder, hearing impairment and vision defect or acute diplopia, and also motor deficits on physical examination were accepted as neurological impairment.

Two different 1.5 Tesla Magnetic Resonance Imaging scanners; (Magnetom®, Aera, Siemens-Erlangen, Germany and Philips Achieva®, Philips Medical Systems, Eindhoven, The Netherlands) with a standard head coil were used for performing the magnetic resonance imaging of patients. The non-contrast conventional brain MRI, protocol constituted the following sequences, axial T1-weighted, axial T2-weighted, axial FLAIR, coronal T2-weighted, sagittal FLAIR, and diffusion-weighted imaging. The patients' images were evaluated on MRI via Extreme Picture Archiving and Communications System (PACS, Ankara, Turkey).

Statistical Analysis: All statistical analyses were performed using the Statistical Package for Social Sciences® (SSPS) software (SPSS for Windows, Version 24, SPSS Inc., USA). The distribution of the data was analyzed using the Kolmogorov-Smirnov test. Quantitative (parametric) variables were presented as median and interquartile range (IQR) deviation, and qualitative (nonparametric) variables were expressed as observed numbers and percentages. Pearson's Chi-Square test was used to test if differences between dichotomous groups were significant. Fisher's exact test was used when a table had a cell with an expected frequency of less than 5. Upon determining that quantitative data is non-parametric, a Mann-Whitney U test was used to analyze the data with categorical variables. All analyses were performed within a 95% confidence interval and a p value <0.05 was considered statistically significant.

RESULTS

Of the 34 (10.4%) accepted patients with neurological findings, 82.4% had syncope, and loss of consciousness developed in 29.4%, visual disturbance in 26.5%, impaired speech in 26.5%, hemiparesis in 2%, and cardiopulmonary arrest in 2.9% and GCS of the patients were below 15 in 55.9%.

It was determined that out of the three patients who developed cardiopulmonary arrest, one that was brought as cardiopulmonary arrest responded to resuscitation and an MRI was taken during this process. MRI could not be performed on the other two patients of cardiopulmonary arrest, as they did not respond to resuscitation.

The median age of patients in our study was 31.5 years (IQR=19.5 years), and 72.2% of the patients were women. No relationship was found between the presence of neurological findings and age and gender (p>0.05). It was determined that poisoning developed most frequently from the stove (64.5%). No relationship was found between the cause of poisoning and neurological findings (p>0.05). The most frequently recorded symptoms were dizziness (81%; n = 265) and nausea (66.7%; n = 218). While the frequency of dyspnea development was significantly higher in patients with neurological findings (p<0.05), there was no relationship found between neurological findings and nausea, dizziness, headache, weakness, and chest pain with the presence of neurological findings (p>0.05). In our study, the COHb and lactate levels of patients with neurological findings were found to be significantly high, and the pH level was significantly lower (p<0.05).

There was no significant relationship between the presence of neurological findings and CK, CK-MB, and troponin-I levels (p>0.05). The follow-up period of patients with neurological findings was significantly longer (p<0.05). It was found that 44 (13.5%) of the patients received additional hyperbaric oxygen therapy (HBO2).

The frequency of HBO2 treatment was significantly higher in patients with neurological findings (p<0.05). In our study, the rate of hospitalization was 10.7%, and the mortality rate was 0.9%. Hospitalization and mortality rates were significantly high in patients with neurological findings (p<0.05). The relationship of the presence of neurological findings with clinical, demographic, and laboratory features is presented in Table 1.

Table 1. The relationship between clinical and demographic features and the presence of neurological findings

Clinical, demographic and laboratory findings	Neurological symptoms (+) (n=34)	Neurological symptoms (-) (n=293)	Total (n=327)	P value
Age (year), (IQR)	31.5 (19.5)	33 (20)	31.5 (19.5)	0,681[α]
Gender, n (%)				
Male	14 (41,2)	77 (26,3)	91 (27,8)	0,067[β]
Female	20 (58,8)	216 (73,7)	236 (72,2)	
Causes of poisoning, n (%)				
Stove	22 (64,7)	189 (64,5)	211 (64,5)	0,982[β]
Natural gas	11 (32,4)	87 (29,7)	98 (30)	0,749[β]
Hookah	0	10 (3,4)	10 (3,1)	0,507*
Automobile exhaust	1 (2,9)	7 (2,4)	8 (2,4)	0,589*
Symptoms / Findings, n (%)				
Nausea	18 (52,9)	200 (68,3)	218 (66,7)	0,073[β]
Dizziness	24 (70,6)	241 (82,3)	265 (81)	0,101[β]
Headache	14 (41,2)	133 (45,4)	147 (45)	0,640[β]
Weakness	7 (20,6)	87 (29,7)	94 (28,7)	0,267[β]
Dyspnea	7 (20,6)	27 (9,2)	34 (10,4)	0,040[β]
Syncope	28 (82,4)	0	28 (8,6)	<0,001*
Loss of consciousness	10 (29,4)	0	10 (3,1)	<0,001*
Chest pain	2 (5,9)	6 (2)	8 (2,4)	0,798*
Visual disturbance	9 (26,5)	0	9 (2,8)	<0,001*
Impaired speech	9 (26,5)	0	9 (2,8)	<0,001*
Cardiopulmonary arrest	3 (8,8)	0	3 (0,9)	0,001*
Hemiparesis	1 (2,9)	0	1 (0,3)	0,104*
GCS, median (IQR)	14 (2)	15 (0)	15 (0)	<0,001[α]
Laboratory findings				
COHb (%), median (IQR)	32,5 (8)	26 (4,5)	26 (5)	<0,001[α]
pH, median (IQR)	7.34 (0,2)	7.33 (0,05)	7,34 (0,03)	0,011[α]
Lactate (mmol/L), median (IQR)	1.6 (0.4)	2,25 (1.2)	1,6 (0,5)	0,004[α]
CK (U\L), median (IQR)	331 (114,3)	289 (129)	300 (124)	0,407[α]
CK-MB (U\L), median (IQR)	26 (8)	25 (10)	26 (9)	0,244[α]
Troponin-I (ng/L), median (IQR)	5 (2)	5 (4)	5 (4)	0,098[α]
Follow-up period (hour), median (IQR)	72 (24)	6,8 (2,8)	7 (2,8)	0,001*
Treatment				
Normobaric oxygen	3 (2.9)	280 (95.6)	283 (86.5)	<0,001*
Hyperbaric oxygen+normobaric oxygen	31 (97,1)	13 (4,4)	44 (13,5)	
Hospitalization (day), n (%)	31 (91.2)	4 (1.4)	35 (10.7)	<0.001*
Mortality, n (%)	3 (8,8)	0	3 (0,9)	0,001*

α: Mann-Whitney U test, β: Pearson's Chi-Square test; *: Fisher's exact test, GCS: Glasgow Coma Score, COHb: carboxyhemoglobin, CK: creatine kinase, CK-MB: creatinine kinase- myocardial band

Pathological findings were observed in 13 (40.6%) of 32 patients with MRI. The incidence of MRI findings in the study group was 4% (n = 13/327). Symmetric hyperintensities in the globus pallidus in 7 (53.8%) patients and asymmetric hyperintensities in the caudate nucleus, putamen, and thalamus in 4 (30.7%) patients in the T2-weighted and FLAIR images. 1 (%7.6) patient restricted diffusion in the caudate nucleus, putamen, and 1 (%7.6) patient restricted diffusion in the globus pallidus in the diffusion-weighted imaging (Figure 1 a,b). In our study, no relation was found between the presence of pathological findings on the MRI and age, gender, cause of poisoning, and symptoms/findings (p>0.05). Patients with pathology in their MRI were found to have significantly low GCS and a longer follow-up time (p<0.05). All patients received HBO$_2$ + NBO$_2$ (normobaric oxygen) therapy and all patients with neurological findings were hospitalized. Pathological findings were seen in the MRI of the patient who responded to cardiopulmonary resuscitation. The relationship between clinical, demographic, and laboratory characteristics of the patients and the presence of pathological findings on MRI are shown in Table 2.
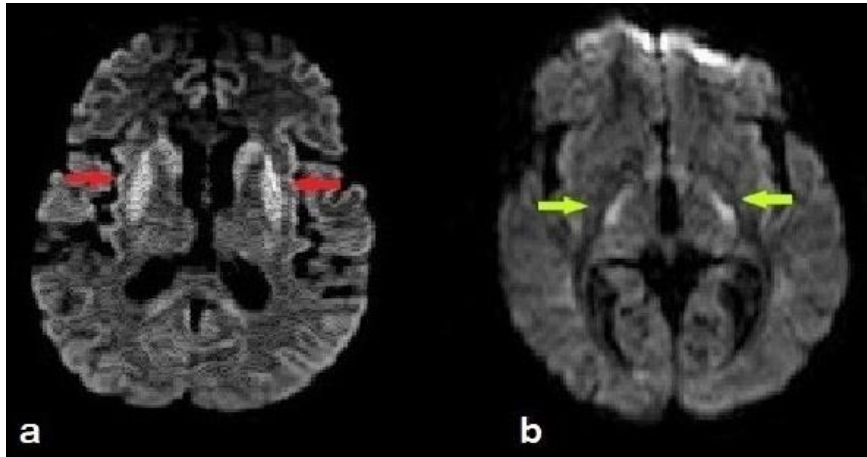

Figure 1 a,b. Axial diffusion-weighted magnetic resonance image (a) shows restricted diffusion in the caudate nucleus and putamen (red arrows), (b) axial diffusion-weighted magnetic resonance image shows restricted diffusion in the globus pallidus (yellow arrows).

Table 2. Relationship between magnetic resonance imaging finding and clinical and demographic characteristics

Clinical, demographic and laboratory findings	MRI (+) (n=13)	MRI (-) (n=19)	P value
Age (year), Median (IQR)	35 (29)	31 (12)	0,209$^\alpha$
Gender			
Male	5 (38,5)	9 (47,4)	0,618$^\beta$
Female	8 (61,5)	10 (52,6)	
Causes of poisoning, n (%)			
Stove	7 (53,8)	13 (68,4)	0,473*
Natural gas	6 (46,2)	5 (26,3)	0,283*
Automobile exhaust	0	1 (2,9)	>0,999*
Symptoms / Findings, n (%)			
Nausea	7 (53,8)	11 (57,9)	0,821
Dizziness	11 (84,6)	12 (63,2)	0,249*
Headache	5 (46,2)	7 (36,8)	0,598
Weakness	3 (23,1)	4 (21,1)	>0,999*
Dyspnea	3 (23,1)	2 (10,5)	0,374*
Syncope	12 (92,3)	14 (73,7)	0,361*
Loss of consciousness	3 (23,1)	6 (31,6)	0,704*
Chest pain	2 (15,4)	0	0,157*
Visual disturbance	4 (30,8)	4 (21,1)	0,684
Impaired speech	4 (30,8)	5 (26,3)	>0,999*
Hemiparesis	1 (5,3)	0	>0,999*
Laboratory findings			
COHb (%), median (IQR)	32 (9)	33 (14)	0,495$^\alpha$
pH, median (IQR)	7.32 (0,14)	7.33 (0,05)	0,495$^\alpha$
Lactate (mmol/L), median (IQR)	1.4 (1.6)	2,3 (1.1)	0,623$^\alpha$
CK (U/L), median (IQR)	341 (91,5)	321 (207)	0,791$^\alpha$
CK-MB (U/L), median (IQR)	25 (8,5)	27 (8)	0,910$^\alpha$
Troponin-I (ng/ml), median	5 (1,5)	6 (3)	0,520$^\alpha$
GCS, median (IQR)	13 (2,5)	15 (1)	0,013$^\alpha$
Follow-up period (hour), median (IQR)	72 (48)	40,3 (39)	0,001*
Mortality, n (%)	1 (7.7)	0	0,406*

α: Mann-Whitney U test, β: Pearson's Chi-Square test; *: Fisher's exact test, MRI: magnetic resonance imaging, GCS: Glasgow Coma Score, COHb: carboxyhemoglobin, CK: creatine kinase, CK-MB: creatinine kinase-myocardial band

DISCUSSION

ABI, which develops in acute CO intoxications, can develop even within minutes in relation to the CO concentration and contact time of the medium. However, even if this damage did not appear on an MRI at the beginning, these patients were shown to experience highly delayed neuropsychiatric pathologies (2). The affinity of CO to hemoglobin is 200 times higher than that of oxygen. Therefore, the developed hypoxia causes ischemia in the brain and reduces energy production. As a result, edema develops in the cells and free oxygen radicals begin to accumulate. Loss of consciousness caused by an excessive increase in COHb leads to lipid peroxidation and apoptosis (9,10).

In patients with CO poisoning, it was found that there was a 10-26% finding in MRI in the long term, and this rate was reported to be 14 times higher than those that developed acutely (9,11-13). Kim et al. reported that ABI developed in 37.2% of the patients (7). O'Donnell et al. reported that in 63% of unconscious patients, they observed findings in the diffusion-weighted MRI examination (13). Jeon et al. stated that ABI developed in 26.9% of the patients and this rate was higher in unconscious patients (8). In our study, neurological findings were detected in 10.4% of the patients, and MRI findings were detected in 40.6% of these patients. The main reason for the low frequency of neurological findings in our study may be due to the evaluation of patients in the acute process. The frequency of lesions in patients with MRI is consistent with the literature.

CO poisonings are among the pathologies that can be seen in all age groups, and studies reported that it is mostly observed in the female population between the ages of 35-41 and at the rate of 57-68% (1,7,14,15). Stearns et al. reported that female patients showed lower levels of symptoms in CO intoxications depending on physiological factors between genders (15). Kim et al. could not find a relationship between ABI development and age and gender (7). In our study, the median age of patients with acute CO poisoning was 31.5 years and 72.2% were females which is consistent with the literature. No relationship was found between the neurological findings and MRI characteristics and age and gender. We think that the results found in our country are due to the fact that the female population is not sufficiently involved in business life and the national average age is young. We believe that neurological findings are related to exposure time and CO concentration in the environment rather than demographic factors such as age and gender, which is why there is no difference between the groups. In addition, the fact that suicide attempts are more frequent in women may have contributed to this process.

In our study, no relationship was found between the neurological and MRI findings of the cause of poisoning in patients. We think that individuals are exposed to a higher rate of neurological involvement due to the long-term exposure due to both sleeping and CO being odorless and that patients who smoke hookahs are less affected by CO as it leaves the environment in a shorter time. However, we believe that the difference is not significant in relation to the small number of patients who are poisoned by hookah.

The symptoms that develop when the COHb level is 15–30% are not specific. Headache, dizziness, nausea, fatigue, and a decrease in dexterity are the most common symptoms (10). Hassan et al. stated that the most common symptoms include headache, nausea/vomiting, and weakness (16). In the study conducted by Genç and Aygün, the most common symptoms reported were dizziness, nausea/vomiting, weakness, and headache (17). Consistent with the literature, the most common symptoms in our study were nausea, dizziness, and headache.

CO intoxications are clinical dose-dependent and the clinical deterioration occurs as the COHb level increases. In severe poisonings, loss of consciousness, chest pain, cardiovascular diseases, delayed neurological sequelae, coma, and death can be seen (18). It has been reported that there is no clear relationship between exposure time and ABI (19). Kim et al. reported that the COHb level of patients who developed ABI was higher than the group without ABI (7). Hassan et al. stated that COHb levels were high in patients with neurological disorders (16). In a study, it was stated that the COHb level is high in patients who developed syncope (20). In our study, while the COHb levels of patients with neurological findings were significantly high, no relationship was found between MRI findings and COHb levels. We think that neurological findings develop more frequently due to the increase in the level of hypoxia, edema, and free radicals as the COHb level increases. It is important to remember that factors such as exposure amount, COHb intensity of the environment, pre-hospital duration and amount of oxygen delivered, and the anxiety status of patients may change the presence of neurological symptoms and MRI findings.

Although studies reported that there is a relationship between the development of neurological findings, pH, and lactate levels, it has been stated that this relationship is not clinically important (21,22). Yildiz et al. reported that syncope development was unrelated to CO and lactate levels, and those who developed syncope had high troponin levels (1). Kaya et al. reported that while the CO levels of patients who are troponin positive in CO poisoning is high; they stated that CK and CK-MB levels were similar to troponin negatives (14). Hassan et al. found that patients with neurological findings had high

troponin, CK, and CK-MB levels, and low pH levels (16). Kim et al. reported that patients with neuropsychiatric disorders had high troponin levels, while lactate levels were similar to those that did not (12). In our study, the presence of neurological findings and MRI findings revealed a high lactate level and a low pH level. There was no relationship between CK, CK-MB, and troponin-I levels between the groups. This may be due to the low resistance of axons to COHb. The axon damage can be related to axons being affected before other organ systems.

In a study, it was stated that while patients with neuropsychiatric disorders in the later periods had low GCS, there was no relationship with the symptoms (12). The prominence of neurological findings has been linked to the sensitivity of neurons to CO (2). In our study, there was no relationship between neurological findings and non-specific symptoms (nausea, dizziness, headache, and chest pain); however, patients with neurological findings had lower GCS and higher dyspnea frequency. We believe that nonspecific symptoms develop even at low COHb levels since neurons are more sensitive to CO than other cells. We also believe that developing hypoxia causes many nonspecific symptoms. We believe that CO increases the central effectiveness and therefore, the frequency of dyspnea increases in patients with neurological symptoms.

In the studies, it was stated that the most common finding in MRI due to CO poisoning was in the globus pallidus, followed by the caudate nucleus, putamen, and thalamus. Frequent lesions in these areas have been linked to their susceptibility to hypoxia (2). It has been reported that lesions in globus pallidus generally develop in a short time (23). In our study, the most common lesions were found in the globus pallidus which is consistent with the literature. This may be due to the fact that some regions have higher oxygen requirements and are more sensitive to ischemia.

Yildiz et al. stated that 91.8% of the patients received NBO_2, and 8.2% received HBO_2 + NBO_2 in their study (1). Thom et al. reported that HBO_2 reduced the delayed neurological sequel from 23% to zero in their study (24). Ducassé et al. reported that patients who received HBO_2 had less electroencephalography and brain flow abnormalities (25). Moon and DeLong reported in their study that although neurological sequelae develop in patients given HBO_2, it develops less

compared to patients given only NBO_2 (26). In our study, it was found that patients with neurological findings and lesions detected on MRI received HBO_2 more frequently. This may be due to the fact that the development of neurological findings is related to high COHb level, and both high COHb level and the presence of neurological findings are criteria for HBO_2.

Yildiz et al. stated that 2.2% of the patients were hospitalized in their study (1). Chang et al. stated in their study that 15% of patients needed intensive care and 30.8% of them were treated by hospitalization (6). In our study, 10.7% of the patients were admitted and the frequency of hospitalization and follow-up were longer in patients with neurological findings and lesions detected on MRI. We believe that patients with neurological findings are hospitalized for treatment due to the requirement of longer treatment and the fact that HBO_2 treatment consists of several sessions.

In studies conducted, the mortality rates in CO intoxications have been reported to be between 1-3% (4,18). In a study, it was stated that the frequency of ABI is high in patients with mortality (14). In our study, the mortality rate was 0.8%. We believe that one of the main causes of cardiopulmonary arrest in patients is the cardiac adverse effects at high COHb levels as in ABI.

Jeon et al. reported that the diffusion-weighted MRI imaging showed 75.2% sensitivity and 90.2% specificity in showing ABI (8). In our study, the sensitivity was 76.5% and specificity was 85.3% for the 29.5% cut-off value for neurological findings, and the sensitivity was 76.9% and specificity was 88.5% for the cut-off value of 30.5 for the MRI findings. We believe that high sensitivity and specificity support that MRI is highly diagnostic.

Limitations
As our study is retrospective, we believe that some data are insufficient. Again, information on factors affecting neurological findings such as exposure amount, duration, and the COHb density of the environment; pre-hospital duration; and the amount and duration of oxygen delivered are limited.

Conclusion
It was determined that acute CO poisoning may lead to acute brain damage, and 40.6% would be detected in brain MRIs taken in patients during the acute phase.

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