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-myo cardial 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 rate of hospitalization was 10.7%, the mortality rate was 0.9%. The rate of hospitalization was 10.7%, the mortality rate was 0.9%. Pathological findings were detected in 13 (40.6%) of 32 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

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**AKUT KARBON MONOKSIT ZEHIRLENMESI OLAN HASTALARININ KLINIK VE MANYETIK REZONANSM ORTAMINDA BULGULARI**

**ÖZET**

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 görüntülemesi (MRG)’nde tespit edilen serebral lezyonları tanımlamaya amaçlanmıştır.

Gereç ve Yöntem: 327 hasta yaş, cinsiyet, CO zehirlenme nedenleri, klinik belirtileri, nörolojik bulgular, GKS’leri, karboksijemoglobin (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.

Bulgar: Çalıştımızda hastaların yaş ortancası 31,5 yıl (IQR=19.5 yıl) olup, hastaların %72,2’si kadın idi. Hastaların %34 (%10,4)’ünde nörolojik bulgular saptandı. Nörolojik bulgusu olan hastaların dispne siklığı anlamlı olarak yüksekti (p<0.05). Çalıştımızda nörolojik bulgusu olan hastaların COHb düzeyi ve laktat düzeyleri ile anlamlı olarak yüksek, pH düzeyi anlamlı olarak düşük saptandı (p<0.05). Nörolojik bulgusu olan hastaların dispne siklığı anlamlı olarak yüksek (p<0.05). Nörolojik bulgusu olan hastaların dispne siklığı, nörolojik bulgusu olan hastaların mortalite oranları anlamaktaydı (p<0.05). Nörolojik bulgusu olan hastaların dispne siklığı, nörolojik bulgusu olan hastaların mortalite oranları anlamaktaydı (p<0.05). Nörolojik bulgusu olan hastaların dispne siklığı, nörolojik bulgusu olan hastaların mortalite oranları anlamaktaydı (p<0.05). Nörolojik bulgusu olan hastaların dispne siklığı, nörolojik bulgusu olan hastaların mortalite oranları anlamaktaydı (p<0.05). Nörolojik bulgusu olan hastaların dispne siklığı, nörolojik bulgusu olan hastaların mortalite oranları anlamaktaydı (p<0.05).

Sonuç: Akut karbonmonoksit zehirlenmelerinin akut beyin hasarı olabileceğini, klinik bulguların ve MRG’de tespit edilen serebral lezyonlarının klinik ve manyetik rezonans görüntülemesinde daha fazla dikkat edilmesi önerildi.

Anahat Kelimeler: Karboksijemoglobin, 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% neuropyschological 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, sagittal 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® (SPSS) 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 gender (p>0.05). While the relationship was found between the cause of poisoning and symptoms/dysfunction (p<0.01). The relationship between 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* |
| Gender, n (%)                               |                                 |                                 |              |        |
| Male                                        | 14 (41.2)                       | 77 (26.3)                       | 91 (27.8)    | 0.067* |
| 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  |
| Natural gas                                 | 11 (32.4)                       | 87 (29.7)                       | 98 (30)      | 0.749  |
| 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* |
| Dizziness                                   | 24 (70.6)                       | 241 (82.3)                      | 265 (81)     | 0.101  |
| Headache                                    | 14 (41.2)                       | 133 (45.4)                      | 147 (45)     | 0.640* |
| Weakness                                    | 7 (20.6)                        | 87 (29.7)                       | 94 (28.7)    | 0.267* |
| Dyspnea                                     | 7 (20.6)                        | 27 (9.2)                        | 34 (10.4)    | 0.040* |
| 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*|
| Laboratory findings                         |                                 |                                 |              |        |
| COHb (%), median (IQR)                      | 32.5 (8)                        | 26 (4.5)                        | 26 (5)       | <0.001*|
| pH, median (IQR)                            | 7.34 (0.2)                      | 7.33 (0.05)                     | 7.34 (0.03)  | 0.011* |
| Lactate (mmol/L), median (IQR)              | 1.6 (0.4)                       | 2.25 (1.2)                      | 1.6 (0.5)    | 0.004* |
| CK (UL/L), median (IQR)                     | 331 (114.3)                     | 289 (129)                       | 300 (124)    | 0.407* |
| CK-MB (UL/L), median (IQR)                  | 26 (8)                          | 25 (10)                         | 26 (9)       | 0.244* |
| Troponin-I (ng/L), median (IQR)             | 5 (2)                           | 5 (4)                           | 5 (4)        | 0.098* |
| 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* |

a: Mann-Whitney U test; b: Pearson’s Chi-Square test; c: Fisher’s exact test; GCS: Glasgow Coma Score, COHb: carboxyhemoglobin, CK: creatine kinase, CK-MB: creatine kinase- myocardial band

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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*  |
| Gender                                       |               |                |         |
| Male                                         | 5 (38,5)      | 9 (47,4)       | 0,618*  |
| 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 (75,7)      | 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*  |
| pH, median (IQR)                             | 7.32 (0.14)   | 7.33 (0.05)    | 0,495*  |
| Lactate (mmol/L), median (IQR)               | 1.4 (1.6)     | 2.3 (1.1)      | 0,623*  |
| CK (U/L), median (IQR)                       | 341 (91.5)    | 321 (207)      | 0,791*  |
| CK-MB (U/L), median (IQR)                    | 25 (8,5)      | 27 (8)         | 0,910*  |
| Troponin-I (ng/ml), median                   | 5 (1.5)       | 6 (3)          | 0,520*  |
| GCS, median (IQR)                            | 13 (2.5)      | 15 (3)         | 0,013*  |
| 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 non-specific 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 non-specific 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.

Yıldız et al. stated that 91.8% of the patients received NBO₂, and 8.2% received HBO₂ + NBO₂ in their study (1). Thom et al. reported that HBO₂ reduced the delayed neurological sequel from 23% to zero in their study (24). Ducassé et al. reported that patients who received HBO₂ had less electroencephalography and brain flow abnormalities (25). Moon and DeLong reported in their study that although neurological sequelae develop in patients given HBO₂, it develops less compared to patients given only NBO₂ (26). In our study, it was found that patients with neurological findings and lesions detected on MRI received HBO₂ 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 HBO₂.

Yıldız 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 HBO₂ 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.

REFERENCES

1. Yıldız MN, Eroğlu SE, Ozen C, Yıldız HA, Sektioglu BK, 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;3(2):141–5. doi: 10.14744/nci.2018.88709.
2. Lo C-P, Chen S-Y, Lee K-W, Chen W-L, Chen C-Y, Hsueh C-J, et al. Brain injury after acute carbon monoxide poisoning: early and late complications. AJR Am J Roentgenol. 2007;189(4):W205-11. doi: 10.2214/AJR.07.2425.
3. Mukhopadhyay S, Hirsch A, Etienne S, Melnikova N, Wu J, Sircar K, Orr M. Surveillance of carbon monoxide-related incidents: Implications for prevention of related illnesses and injuries, 2005-2014. Am J Emerg Med. 2018;36(10):1837-44. doi: 10.1016/j.ajem.2018.02.011.

4. Rose JJ, Wang L, Xu Q, McTierman CF, Shiva S, Tejero J, et al. Carbon monoxide poisoning: pathogenesis, management, and future directions of therapy. Am J Respir Crit Care Med. 2017;195(5):596-606. doi: 10.1164/rccm.201606-1275CI.

5. Kim H, Choi S, Park E, Yoon E, Min Y, Lampotang S. Serum markers and development of delayed neuropsychological sequelae after acute carbon monoxide poisoning: anion gap, lactate, osmolarity, S100B protein, and interleukin-6. Clin Exp Emerg Med. 2018;5(3):185-191. doi: 10.15441/ceeem.17.217.

6. 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(2):171-7. doi: 10.1016/j.pedneo.2016.03.007.

7. Kim YJ, Sohn CH, Seo DW, Oh BJ, Lim KS, Kim WY. Clinical predictors of acute brain injury in carbon monoxide poisoning patients with altered mental status at admission to emergency department. Acad Emerg Med. 2019;26(1):60-7. doi: 10.1111/acem.13510.

8. Jeon S-B, Sohn CH, Seo D-W, Oh BJ, Lim KS, Kang D-W, et al. Acute brain lesions on magnetic resonance imaging and delayed neurological sequelae in carbon monoxide poisoning. JAMA Neurol. 2018;75(4):436-43. doi: 10.1001/jamaneurol.2017.4618.

9. Bleeker ML. Carbon monoxide intoxication. Chapter 12 In: Handb Clin Neurol. 131: Elsevier; 2015;131: 191-203. doi: 10.1016/B978-0-444-62627-1.00024-X.

10. Pepe G, Castelli M, Nazerian P, Vanni S, Del Panta M, Gambassi F, et al. Delayed neuropsychological sequelae after carbon monoxide poisoning: predictive risk factors in the Emergency Department. A retrospective study. Scand J Trauma Resusc Emerg Med. 2011;19:16. doi: 10.1186/1757-7241-19-16.

11. Thom SR, Taber RL, Mendiguren, II, Clark JM, Hardy KR, Fisher AB. Delayed neuropsychologic sequelae after carbon monoxide poisoning: prevention by treatment with hyperbaric oxygen. Ann Emerg Med. 1995;25(4):474-80. doi: 10.1016/s0196-0644(95)70261-x.

12. Kim Y, Cha Y, Kim M, Kim H, Lee Y, Youk H, et al. The usefulness of diffusion-weighted magnetic resonance imaging performed in the acute phase as an early predictor of delayed neuropsychiatric sequelae in acute carbon monoxide poisoning. Hum Exp Toxicol. 2018;37(6):587-95. doi: 10.1177/09603271177722821.

13. O'donnell P, Buxton P, Pitkin A, Jarvis LJ. The magnetic resonance imaging appearances of the brain in acute carbon monoxide poisoning. Clin Radiol. 2000;55(4):273-80. doi: 10.1053/crad.1999.0369.

14. Kaya H, Coskun A, Beton O, Kurt R, Yildirim M, 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(13):2891-8.

15. Stearns D, Sircar K. National unintentional carbon monoxide poisoning estimates using hospitalization and emergency department data. Am. J. Emerg. 2019;37(3):421-6. doi: 10.1016/j.ajem.2018.06.002.

16. Hassan, OA, Abdelaleem, SA, Hamdy, L. A prospective comparative study between three chemical markers for predicting delayed neurological sequelae in patients with acute carbon monoxide poisoning of poison control center in Minia University Hospital. Ain-Shams J Forensic Med Clin Toxicol 2018; 31: 23–32. doi: 10.21608/AJFM.2018.15874.

17. Genç S, Aygün D. Karbonmonoksit Zehirlenmesinde Karboksihemoglobin Düzeni, Zehirlenmenin Şiddet ve Mini Mental Durum Testi Skalası Arasındaki İlişki. Turk J Emerg Med. 2013;13(1): 25-32. doi: 10.5505/1304.7361.2013.36002.

18. Sohn CH, Huh JW, Seo DW, Oh BJ, Lim KS, Kim WY. Aspiration pneumonia in carbon monoxide poisoning patients with loss of consciousness: prevalence, outcomes, and risk factors. Am J Med. 2017;130(12):1465. e21-. e26. doi: 10.1016/j.amjmed.2017.06.038.

19. Sokal JA, Krallkowska E. The relationship between exposure duration, carboxyhemoglobin, blood glucose, pyruvate and lactate and the severity of intoxication in 39 cases of acute carbon monoxide poisoning in man. Archives of toxicology. 1985;57(3):196-9. doi: 10.1007/bf00290887.

20. Keles A, Demircan A, Kurtoglu G. Carbon monoxide poisoning: how many patients do we miss?. Eur J Emerg Med. 2008;15(3):154-7. doi: 10.1097/MEJ.0b013e3282ef5d19.

21. Besli GE, Ergüven M, Karadogan M, Yılmaz Ö. Carbon Monoxide Poisoning in Children. Eurasian J Emerg Med 2010;9:26–30. doi: 10.4170/JAEM.2009.19480.

22. 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. doi: 10.1007/s00134-003-1866-0.

23. Chu K, Jung K-H, Kim H-J, Jeong S-W, Kang D-W, Roh J-K. Diffusion-weighted MRI and 99mTc-HMPAO SPECT in delayed relapsing type of carbon monoxide poisoning: evidence of delayed cytotoxic edema. Eur Neurol. 2004;51(2):98-103. doi: 10.1159/000076536.
24. Thom SR, Taber RL, Mendiguren II, Clark JM, Hardy KR, Fisher AB. Delayed neuropsychologic sequelae after carbon monoxide poisoning: prevention by treatment with hyperbaric oxygen. Ann Emerg Med. 1995;25(4):474-80. doi: 10.1016/s0196-0644(95)70261-x.

25. Ducassé JL, Celsis P, Marc-Vergnes JP. Non-comatose patients with acute carbon monoxide poisoning: hyperbaric or normobaric oxygenation?. Undersea Hyperb Med. 1995, 22(1):9-15.

26. Moon RE, DeLong E. Hyperbaric oxygen for carbon monoxide poisoning. Med J Aust. 1999 Mar 1;170(5):197-9.