



# The Correlation of Carbon Monoxide Level, Lactate, Creatine Kinase Myocardial Band, Troponin T, Magnetic Resonance Imaging and Clinical Results in Acute Carbon Monoxide Poisoning

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## Abstract

**Objective:** In this study, it was aimed to investigate the correlation between arrival-carboxyl hemoglobin (COHb) levels, lactate, creatine kinase-myocardial band (CK-MB), troponin T and radiological findings in patients who were admitted to emergency service with a pre-diagnosis of carbon monoxide (CO) poisoning between the years 2014-2016.

**Methods:** The patients who were admitted to emergency service with a pre-diagnosis of CO poisoning between the years 2014-2016 were screened retrospectively. Demographic data, arrival-COHb levels, lactate, CK-MB, troponin T, and if available, computed tomography or magnetic resonance (MR) imaging of the patients, their clinical treatment, and results were compared.

**Results:** While COHb values of the patients at emergency department arrival were  $14.536 \pm 13.047\%$ , they were found as  $4.536 \pm 3.698\%$  at discharge from the hospital. A correlation was not observed statistically between arrival-COHb, lactate, troponin T, and CK-MB. Arrival-lactate level was found as  $>2.1$  mmol/L in 47 patients (36.90%). Cranial MR findings that suggest CO poisoning were determined in four cases who underwent hyperbaric oxygen treatment; millimetric signal enhancements being concordant with chronic microangiopathic ischemic gliotic foci in white matter were determined in the level of the periventricular field, bilateral corona radiata and centrum semiovale in MRI.

**Conclusion:** There is no correlation between arrival-COHb levels of the patients and lactate, CK-MB and troponin T in CO poisoning. Radiological evaluation should be used for excluding the reasons for a neurological disorder, not for diagnostic reasons.

**Keywords:** Carbon monoxide poisoning, lactate, troponin, creatine kinase

## INTRODUCTION

Carbon monoxide (CO) is a gas that is produced through incomplete combustion of fuels that contain “carbon” and it is regarded as highly “toxic”. CO poisoning is the most frequently encountered poisoning in Turkey (1).

In case of breathing, CO causes tissue hypoxia by two important effects: First, the binding capacity of CO to hemoglobin is 200-300 times more than that of oxygen, and second, the

part dissolved in plasma causes decrease in 2,3-diphosphoglyceride production thus leading to left shift in oxyhemoglobin dissociation curve, and the oxygen that is bound to hemoglobin cannot be released. Because of the first effect, oxygen content of blood decreases, and because of “carboxyhemoglobin (COHb)” produced by the second effect, oxygen offer to tissues decreases. Therefore hypoxia develops in tissues, and then anaerobic glycolysis increases, thus leading to lactic acidosis;



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the formation of free oxygen radicals, hypotension, and loss of consciousness are followed by lipid peroxidation and apoptosis (2). Multi-variance analyses suggested that arrival-lactate level, old age, leucocyte, and mental condition are independent factors related to critical complications and need of intensive medical treatment in order to determine the patient prognosis after CO poisoning (3). Nevertheless, the medical records on CO poisoning cases in our country are considered to be inadequate. Also, the number of patients applied to health institutions because of CO poisoning and how many of them were diagnosed with CO poisoning are unknown; moreover, the diagnoses are considered to be recorded with different codes in ICD-10 coding system (4).

In this study, it was aimed to investigate the correlation between arrival-CO<sub>Hb</sub> levels and lactate, creatine kinase-myocardial band (CK-MB), troponin T, and radiological findings of the patients who were admitted to emergency service with a pre-diagnosis of CO poisoning between the years 2014 and 2016.

## METHODS

### Subjects and Study Protocol

Following the approval of the Ethics Committee of Ministry of Health Okmeydanı Training and Research Hospital (approval date: 14/06/2016, decision no: 495), the patients admitted to emergency service with a pre-diagnosis of CO poisoning between the years 2014 and 2016 were screened retrospectively.

### Measurements and Calculations

Demographic data, arrival-CO<sub>Hb</sub> levels, lactate, CK, troponin T, and, if available, brain computed tomography (CT) scans or magnetic resonance imaging (MRI) of the patients, their clinical treatment and results were compared.

### Statistical Analysis

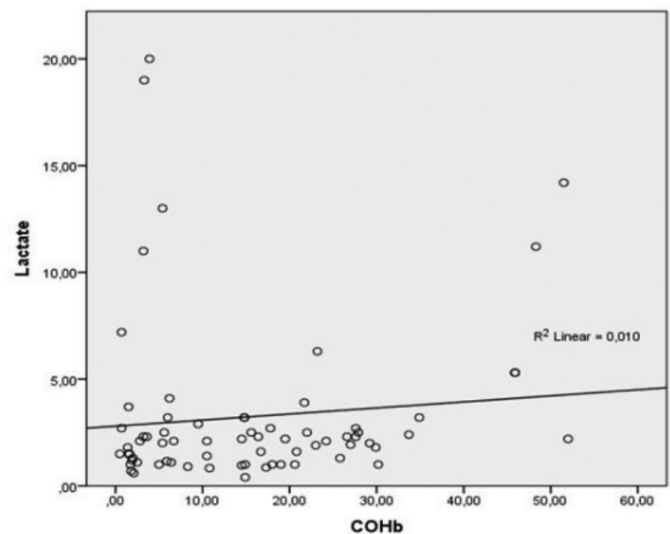
Statistical analysis of the data was performed by SPSS 21.0 (SPSS, Chicago, IL). The correlation between arrival-CO<sub>Hb</sub> and lactate, troponin T, CK-MB was analyzed by the Pearson correlation test. P values of <0.05 were considered significant.

## RESULTS

While CO<sub>Hb</sub> values of the patients at emergency department arrival were  $14.536 \pm 13.047\%$ , they were found as  $4.536 \pm 3.698\%$  at discharge from the hospital (Table 1). A correlation was not observed statistically between arrival-CO<sub>Hb</sub>, lactate, troponin T and CK-MB (Table 2, Figure 1,  $p > 0.05$ ). Arrival-lactate level was found as  $>2.1$  mmol/L in 47 patients (36.90%). Cranial MRI findings that suggest CO poisoning were determined in four cases who underwent hyperbaric oxygen (HBO) treatment; millimetric signal enhancements being concordant with chronic microangiopathic ischemic gliotic foci in white matter were determined in the level of the periventricular field, bilateral corona radiata and centrum semiovale in MRI (Figure 2).

## DISCUSSION

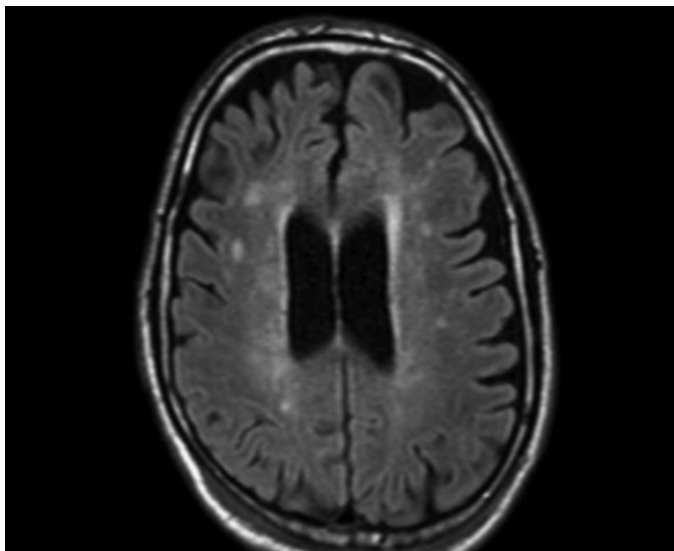
The first symptoms of CO intoxication are nonspecific; history is the most valuable finding. Physical examination has been used to a limited extent in diagnosis. Blood CO<sub>Hb</sub> levels should be measured as soon as possible; nevertheless, CO<sub>Hb</sub> levels frequently may not be useful in diagnosis and treatment (half-life is 4-6 hours at room air) (5). The relationship between the severity of poisoning and blood CO level is not drastic, but it can be used as an indicator to follow-up on the treatment. On



**Figure 1.** Correlation between arrival-CO<sub>Hb</sub> and lactate  
CO<sub>Hb</sub>: Carboxyl hemoglobin

Table 1. Carboxyl hemoglobin, lactate, troponin T, creatine kinase-myocardial band levels of the cases on admission to the emergency department					
	Mean ± SD	n=132			
Arrival CO <sub>Hb</sub> (%)	Discharge CO <sub>Hb</sub> (%)	Lactate (mmol/L)	Troponin T	CK-MB	Length of hospital stay
14.536±13.047	4.536±3.698	3.172±3.670	0.086±0.478	3.226±6.849	1.410±1.668
CO <sub>Hb</sub> : Carboxyl hemoglobin, CK-MB: Creatine kinase-myocardial band, SD: Standard deviation					

the other hand, if a long time passed after CO exposure or if supportive oxygen treatment was administered, blood CO level can erroneously be detected as low. Detection of high-level CO in the blood is important for diagnosis, but low CO level in blood does not detract from diagnosis (6). In the early period or weeks after the exposition, various symptoms occur in organs because of hypoxia. In addition to major symptoms affecting the neuropsychiatric and cardiovascular systems, other systems and organs are also affected (7-9). Especially when the children with acute CO poisoning were compared with those who were healthy, the heart seems to be the most critical organ and subclinical systolic and diastolic left ventricle dysfunction, and ventricular repolarization failure were observed (10). Similar findings ranging from simple arrhythmia to myocardial infarction are also seen in the poisoning of adults (11). Thus, a correlation is expected between blood COHb and cardiac troponin T. In their



**Figure 2.** Cranial MRI; millimetric signal enhancements being concordant with chronic microangiopathic ischemic gliotic foci in white matter were determined in the level of the periventricular field, bilateral corona radiata and centrum semiovale in MRI  
MRI: Magnetic resonance imaging

retrospective study in 141 patients, Huysal et al. (1) found a weak correlation for this, although the COHb level greater than 25% was described as severe acute CO intoxication and, they found no correlation with CK-MB. Also, in our study, a correlation was not found between arrival-COHb ( $14.536 \pm 13.047$ ) and troponin T and CK-MB. In another study on correlation from the point of arrival-blood lactate, COHb, and electrocardiography (ECG) findings (COHb values on average  $21.5 \pm 13.9\%$ ), normobaric oxygen treatment was applied to 67.6% of the patients while HBO treatment was administered to 32.4% of the patients. Blood lactate levels of HBO treated patients were found greater when admitted to emergency service (2.3 mmol/L vs 1.0 mmol/L,  $p < 0.001$ ), and a positive correlation was determined between lactate level and clinical findings of poisoning and COHb values. Nevertheless, this study was criticized on the basis of its results, such that HBO treatment is a therapy method rather than a measure of clinical outcome, and more than one factor is required for the decision regarding HBO therapy, and it does not indicate more severe poisoning (12). Also, in our study, although arrival-lactate values of the patients were above 3, no correlation was found between arrival-COHb and arrival-lactate values. Arrival-COHb values above 30%, low lactate, troponin T, and Glasgow Coma scale (GCS) show that more than one factor was considered in the decision-making process for four patients whose HBO treatments were planned. In another study, HBO treatment was planned for 37 of 57 patients who admitted to the emergency department for CO poisoning (COHb levels  $10.1 \pm 5.7\%$  (range=3-25%) (13). HBO treatment was administered to four patients in our study. The first patient had a GCS score of 4 in the neurologic examination, was hypotensive, had metabolic acidosis in arterial blood gas, COHb level was 30%, and lactate was 2.2 mmol/L, ECG showed dysrhythmia, ST-T variances and had pulmonary edema. The second patient had a COHb level of 52%, lactate was 2.2 mmol/L. The third patient had a COHb level of 51% and lactate was 14.2 mmol/L. The fourth patient

**Table 2. Correlation of arrival- carboxyl hemoglobin, lactate, troponin T, creatine kinase-myocardial band**

	Arrival COHb	Lactate (mmol/L)	Troponin T	CK-MB	
Arrival COHb	1	0.098	0.059	-0.003	Pearson correlation
		0.421	0.662	0.983	Sig. (2-tailed)
Lactate	0.098		-0.082	-0.017	Pearson correlation
	0.421		0.556	0.906	Sig. (2-tailed)
Troponin T	0.059	-0.082		0.138	Pearson correlation
	0.662	0.556		0.269	Sig. (2-tailed)
CK-MB	-0.003	-0.017			Pearson correlation
	0.983	0.906			Sig. (2-tailed)

$p < 0.05$ , COHb: Carboxyl hemoglobin, CK-MB: Creatine kinase-myocardial band, Sig: Strikethrough

had a COHb level of 45.9% and lactate was 5.3 mmol/L. The normal level of COHb is 0.5-3%, and it is 3-7% in neonatal and 4-12% in smokers. Poisoning begins when the concentration is 15%. The toxic level is 20-50% and the lethal level is above 50-60%. COHb level was found 50% in people staying several hours in an environment with CO, and it was found as 25% in a person staying in such an environment for 34 minutes (14). Mild poisoning can have subtle symptoms such as headache, fatigue, weakness, gasping, nausea, and vertigo (COHb=15-30%). When the level of COHb exceeds 20%, the heart and brain are affected severely, and if it is above 30-70%, it leads to dizziness, vomiting, loss of muscular coordination, unconsciousness and death (15).

The first treatment option in CO poisoning is 100% O<sub>2</sub> (normobaric oxygen) treatment at least for six hours until reaching the normal values of COHb. The aim is to decrease COHb. Continuous oxygen treatment should be carried on until the patient becomes asymptomatic, or the level of COHb is below 10%. It should be decreased to 2% in those with cardiovascular or pulmonary symptoms (16).

HBO modulates the inflammatory processes that cannot be maintained with normobaric oxygen, enhances mitochondrial function, and temporarily inhibits lipid peroxidation. It also repairs leukocyte adhesion to damaged microvasculature and promotes myelin formation in the brain. Therefore, HBO treatment is recommended in acute symptomatic CO poisoning and, in the same way, in poisoning due to CO inhalation for patients with unconsciousness and those with the permanent neurological deficit; nevertheless, it remains somehow unclear (17,18). Brain CT or MRI should be used for excluding the reasons for a neurological disorder, not for diagnostic reasons. Hypoxic brain damage is primarily observed in the cerebral cortex, cerebral white matter, and basal ganglions, especially in Globus pallidus (19). In our study, cranial MRI findings that suggest CO poisoning were determined in four cases; millimetric signal enhancements being concordant with chronic microangiopathic ischemic gliotic foci in white matter were determined in the level of the periventricular field, bilateral corona radiata, and centrum semiovale.

A standard HBO treatment protocol for CO poisoning includes 100% oxygen for 90 minutes. Cases with moderate symptoms can benefit from the initial treatment. However, for the patients whose symptoms do not regress, second or third treatment sessions could be planned. Temporary unconsciousness, coma or seizure, ischemic ECG changes, focal neurological deficit, pregnant women with COHb levels >15%, COHb levels exceeding 40% together with headache and nausea are the indications

for HBO treatment. The only absolute contraindication for HBO treatment is untreated pneumothorax (20). In a study involving 68 centers from 23 countries, temporary or prolonged unconsciousness was accepted as an indication for HBO treatment in 95% of the centers; also, positive neurologic findings, acute cardiac ischemia and, pregnancy were of priority for HBO treatment (21). Our priorities were formed by these data in our four cases; for our first case, the symptoms regressed after the first session, and therefore, the treatment program was terminated, nevertheless for our second case, there was no regression even after three sessions. In their ten years of study, Chan et al. (22) administered HBO treatment for 24 of 93 patients, and none of these patients were found to have neurological sequelae in the follow-up. Neurological sequelae were found in seven patients who were not treated with HBO. The degree of the patient's consciousness on admission to the hospital, GCS score, increased troponin levels, creatine kinase, and intubation requirement were defined as prognostic factors for the development of neurological sequelae (21,23,24).

## CONCLUSION

There was no correlation between arrival-COHb levels of the patients and lactate, CK-MB and troponin T in CO poisoning. Radiological evaluation should be used for excluding the reasons for a neurological disorder, not for diagnostic reasons. The first treatment option is 100% oxygen treatment for at least six hours. HBO treatment plan to reduce the half-life of COHb should be prepared considering prognostic factors; national protocols are required for the issue.

### Ethics

**Ethics Committee Approval:** Following the approval of the Ethics Committee of Ministry of Health Okmeydanı Training and Research Hospital (approval date: 14/06/2016, decision no: 495), the patients admitted to emergency service with a pre-diagnosis of CO poisoning between the years 2014 and 2016 were screened retrospectively.

**Informed Consent:** Retrospective analysis.

**Peer-review:** Externally peer-reviewed.

### Authorship Contributions

Surgical and Medical Practices: N.T., B.A., N.A., S.K., S.D.Ö., E.D., H.F., Y.İ., T.M., Concept: N.T., B.A., Design: N.T., B.A., Data Collection or Processing: N.T., B.A., N.A., İ.G., E.D., H.F., Y.İ., Analysis or Interpretation: N.T., B.A., İ.G., Literature Search: N.T., B.A., S.K., T.M., İ.G., Writing: N.T., B.A., T.M.

**Conflict of Interest:** The authors declare no conflict of interest.

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