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Evaluation of the Relationship between Nitric Oxide Levels and Troponin, CK, CK-Mb, and CoHB Levels in Patients Presenting to the Emergency Department with Carbon Monoxide Poisoning

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Abstract

Aim: Ischemic alterations may occur due to hypoxia following carbon monoxide (CO) poisoning. As a result, the heart, brain, and other organs experience a loss of functionality. This study aimed to evaluate the relationship between the levels of nitric oxide (NO), which may be responsible for cardiac damage, and those of troponin, creatine kinase muscle-brain (CK-MB), and carboxyhemoglobin (COHb). These are markers of cardiac damage in patients who presented to the emergency department after being exposed to CO gas.

Materials and Methods: This prospective study included 103 individuals divided into three groups: troponin-negative patients with CO poisoning, troponin-positive patients with CO poisoning, and healthy controls. The NO levels of the groups were statistically compared. The correlation between NO levels and troponin, creatine kinase (CK), CK-MB, and COHb levels was also examined.

Results: The NO value was the lowest in the troponin-positive group and the highest in the control group, with significant differences between the three groups ($p<0.001$). According to the correlation analysis, the NO level was significantly and negatively correlated with the CK and COHb levels, but not the CK-MB level ($p<0.001$). This study revealed lower NO levels in patients with CO poisoning than in healthy individuals.

Conclusions: NO levels have an inverse relationship with troponin and CK values when comparing groups categorized by troponin levels. Therefore, it is postulated that cardiac damage can be prevented by administering inhaler NO therapy together with CO therapy to patients with CO poisoning.

Keywords: Carbon monoxide poisoning, nitric oxide, troponin, carboxyhemoglobin, cardiac damage

Introduction

Carbon monoxide (CO) poisoning is one of the most frequently reported toxicological causes of death. Poisoning occurs due to the incomplete combustion of compounds that contain carbon in their structure. After inhalation, CO passes into the blood through the lungs. When CO combines with hemoglobin, carboxyhemoglobin (COHb) is formed. The concentration of oxygen within the human body decreases as it is replaced by CO, leading to insufficient oxygen transport to tissues. As a result, the heart, brain, and other organs experience a loss of functionality (1).

Due to tissue hypoxia from CO poisoning, the release of electrons from the electron chain in mitochondrial cytochromes stops. The process of oxidative phosphorylation is compromised, leading to the occurrence of cellular hypoxia. CO also binds strongly to intracellular pigments, such as myoglobin. The toxic effects of CO on myocardial myoglobin result in a decrease in cardiac muscle contraction, thereby leading to a reduction in cardiac output. Hypoxia creates ischemic alterations in tissues, which subsequently lead to an increase in troponin, creatine kinase (CK), and creatine kinase muscle-brain (CK-MB) levels (2-3).

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Nitric oxide (NO) plays an important role in regulating heart contraction, heart rate, and vascular tone. Cardiovascular diseases, such as hypertension, heart failure, ischemic heart disease, coronary artery disease, and arrhythmias, are associated with an impaired NO response (4-5). NO is of primary importance in the relaxation of coronary arteries. Furthermore, the flow-induced relaxation response in the coronary microcirculation is mediated by NO (6-7).

This study aimed to evaluate the relationship between NO levels, a potential contributor to cardiac damage, and those of troponin in patients who developed cardiac damage due to exposure to CO gas and sought medical attention in the emergency department. At the same time, an attempt was made to quantitatively reveal the relationship between CK and CK-MB values and NO values, as indicators of cardiac damage. While assessing these parameters, the correlation between the COHb values and NO values of patients with CO poisoning was also evaluated. Thus, it was investigated whether new treatment principles could be developed based on NO levels prior to the occurrence of cardiac damage in patients with CO poisoning.

Materials and Methods

Study Design and Setting

This study was conducted prospectively from September 1, 2020, to April 30, 2022, at the emergency department of a tertiary hospital (Erzurum, Health Practice and Research Hospital, Atatürk University). The study was initiated following the approval of the Ethics Committee of Atatürk Medical Faculty for Clinical Research (decision number: 53, date: 05.11.2020). Patients were accepted into the study after obtaining written informed consent from all volunteers or their relatives. Written informed consent was obtained from conscious patients, while in unconscious patients, consent was obtained in writing from their first-degree relatives. The study protocol was implemented by following the tenets of the Declaration of Helsinki.

Sample Size and Patients

The sample size of the study was calculated by using G*Power 3.1 analysis. Accepting the effect size as 0.5, the type 1 error rate as 0.05, and the power as 0.80, it was determined that a total of 90 participants (at least 30 in each group) were required. Three patient groups were formed: troponin-negative, troponin-positive, and healthy controls. A total of 30 troponin-positive and 43 troponin-negative patients presenting with CO poisoning were included in the study. Since there is no universal standard for a normal NO level, a healthy adult control group was also created, and the normal NO value was accepted as the average of the values measured in the control group. As a result, the sample consisted of 103 individuals.

Patients aged more than 18 years who were diagnosed with CO poisoning and presented to the emergency department within the first eight hours of CO exposure were included in this study. The diagnosis of CO poisoning was made based on the patients' anamnesis (medical history) and blood COHb levels above 5% (10% in smokers). The inclusion criteria of the study were as follows: being over 18 years of age, voluntary participation in the study, having a COHb level above 5 (or greater than 1 if supported by anamnesis and clinical findings), and not having a chronic disease other than hypertension. Patients excluded from the study were those aged under 18 years, those who did not volunteer to participate in the study, those with chronic diseases other than hypertension, those with other drug intoxication accompanying CO poisoning at the time of admission, pregnant women, cases in which more than eight hours had passed after CO exposure, and patients who refused treatment and follow-up. Of the total of 204 patients who presented to our emergency department with CO poisoning, 101 patients were excluded from the study because eight hours had elapsed since CO exposure, 100% oxygen therapy had been started at an external center, or due to pregnancy.

Evaluation of Patients and Data Collection

The circulation and airway scores and the Glasgow Coma Scale scores (8) of the patients were examined at the time of presentation to the emergency department, with CO poisoning.

According to this evaluation, the patients were referred to the critical care room, yellow area, or green area. Three groups were formed: the first group consisted of patients with CO poisoning who had negative troponin values (troponin-negative); the second group consisted of those with CO poisoning who had high troponin values (troponin-positive); and the third group consisted of healthy individuals without CO poisoning or chronic disease. The normal range of NO was determined based on the values measured in the control group.

The patients with CO poisoning were connected to monitors that evaluated blood pressure, respiratory rate, fever, saturation, and heart rhythm, and were placed in a security perimeter. The respiratory rate, blood pressure, pulse rate, oxygen saturation, and body temperature of the patients who were monitored in the emergency department were recorded. During this period, systemic and neurological examinations were performed. 12-lead electrocardiograms (ECGs) were taken, and the data were recorded. Levels of complete blood count, aspartate aminotransferase, alanine transaminase, arterial blood gas,

troponin, CK, CK-MB, and NO were determined as soon as possible following patient presentation. Lactate, COHb, and the presence of acidosis and alkalosis were evaluated in blood gas. The form previously prepared for patients diagnosed with CO poisoning was completed for all patients diagnosed with this condition. This form included the patient's age, gender, presentation date, emergency department protocol number, vital signs, oxygen saturation, complaints, type of CO poisoning, time since CO exposure, treatment initiation time, the presence of syncope, medical history, smoking and medication history, and arterial blood gas results. During this period, normobaric treatment (100% oxygen) was started, and hyperbaric treatment was added for those with appropriate indications. Hyperbaric treatment was planned for patients with a COHb level >25; syncope; and cardiac involvement (echocardiography findings and elevated troponin). The patients' NO levels were measured from the blood taken before hyperbaric treatment. No correlation between NO levels and hyperbaric oxygen treatment was studied. The patients were transferred to the emergency intensive care unit for follow-up.

Echocardiography was performed on the patients within the first two hours of presentation and at the 24th hour, and their ejection fraction (EF) values were measured. Cardiac changes, if any, were noted. During this process, a control ECG was taken, and any ECG changes were recorded. Echocardiography was performed by cardiologists.

Blood Collection and NO Measurement

Blood samples taken for the measurements of troponin, CK, and CK-MB levels were evaluated at the biochemistry laboratory using a Beckman Coulter AU5800 device. The data were taken from the hospital's automation system and recorded into a form. The blood samples taken for analysis of NO were transferred to 5 mL biochemistry tubes and centrifuged at 6.000 rpm for ten minutes with a Rixos 32 device. The serum obtained after centrifugation was transferred to a separate tube and stored at -80 degrees. Then, the serum was assayed collectively. A commercial enzyme-linked immunosorbent assay measurement kit (Human NO ELISA Kit, Catalog No: 201-12-1511, SunRed Biotechnology Company, China) was used to measure the NO level in the serum sample. This kit was produced for the detection of human NO levels in serum, plasma, urine, cell culture supernatant, and tissue homogenate and has a measurement range of 4-600 µmol/L, a sensitivity of 2.052 µmol/L, an intra-run precision coefficient of variation value of <10% and an inter-run precision coefficient of variation value of <12%.

Statistical Analysis

The IBM SPSS v. 20.0 (SPSS Inc., Chicago, IL, United States) program was used to analyze the data obtained. Continuous variables were expressed as mean (standard deviation) or median (interquartile range) values. Categorical data were presented as numbers (percentages). A normality analysis was performed. In the comparison between two independent groups, the independent-samples t-test was used when the normal distribution condition was met, and the Mann-Whitney U test was used otherwise. When comparing more than two independent groups and continuous variables, the ANOVA test was used for normally distributed data and the Kruskal-Wallis test for non-normally distributed data. Following ANOVA, post-hoc methods were employed using the Tukey test in cases where the variances were homogeneous and using the Tamhane T2 test in those where the variances were not homogeneous. Following Kruskal-Wallis analysis, an appropriate post-hoc test was used. However, the description of using one-way ANOVA (k-samples) may not accurately fit the context since ANOVA is not typically used as a post-hoc for Kruskal-Wallis. A p-value of <0.05 was considered significant in all statistical analyses.

Results

The study included a total of 103 patients. In the troponin-negative group, 47.7% (n=20) of the patients were female and 52.3% (n=23) were male. In the troponin-positive group, 45.2% (n=14) of the patients were female and 54.8% (n=16) were male. In the control group, 53.3% (n=16) of the patients were female and 46.7% (n=14) were male. The mean age of the entire cohort at the time of presentation was 33.44±13.82 years, and the median age was 27 (minimum: 18 - maximum: 79) years (Table 1).

Table 1 presents the patients' complaints, the presence of chest pain and syncope, the causes of CO poisoning, any ECG changes, the treatment plan, and the EF status, according to the groups.

Vital signs, pH, lactate, myoglobin values, and the EF status of the patients according to the groups are detailed in Table 2.

The comparison of the study parameters among the three groups revealed significant differences: in the COHb value between the troponin-positive group and the control group, between the troponin-negative group and the control group ($p<0.001$); the CK value between the troponin-positive group and the control group, between the troponin-negative group and the control group ($p<0.001$); the CK-MB value between the troponin-positive and troponin-negative groups ($p=0.003$); and the troponin value between the troponin-positive and troponin-negative groups, as well as between the troponin-negative group and the control group ($p<0.001$). Furthermore, the NO value significantly differed between the three groups ($p< 0.001$). The NO value was the lowest in the troponin-positive group and the highest in the control group (Table 3).

According to the correlation analysis, the NO level was significantly and negatively correlated with the CK and COHb levels ($p < 0.001$), but not with the CK-MB level ($p=0.265$) Furthermore, as the troponin value increased, the NO value decreased. Therefore, it can be stated that as cardiac involvement increases, the NO value decreases (Table 4).

Table 1. Demographic and clinical characteristics of the groups

		Troponin-negative (n=43, 100%)	Troponin-positive (n=30, 100%)	Control (n=30, 100%)
Age (year/mean ± SD)		32.81±11.02	39.17±18.66	28.60±9.46
Gender	Female	20 (46.5%)	14 (46.7%)	16 (53.3%)
	Male	23 (53.5%)	16 (53.3%)	14 (46.7%)
Presentation complaint	Headache	22 (51.2%)	7 (23.3%)	0 (0%)
	Dizziness	11 (25.6%)	9 (30.3%)	0 (0%)
	Fainting	1 (2.3%)	4 (13.3%)	0 (0%)
	Nausea and vomiting	8 (18.6%)	2 (6.7%)	0 (0%)
	Altered state of consciousness	1 (2.3%)	8 (26.7%)	0 (0%)
	No complaint	0 (0%)	0 (0%)	30 (100%)
Cause of CO poisoning	Stove fume	24 (55.8%)	15 (50%)	0 (0%)
	Natural gas	7 (16.3%)	7 (23.3%)	0 (0%)
	Tandoor	8 (18.6%)	1 (3.3%)	0 (0%)
	Exhaust smoke	4 (9.3%)	1 (3.3%)	0 (0%)
	Fire smoke	0 (0%)	6 (20.1%)	0 (0%)
	No known exposure	0 (0%)	0 (0%)	30 (100%)
ECG changes	Present	0 (0%)	1 (3.3%)	0 (0%)
	Not present	43 (100%)	29 (96.7%)	30 (100%)
Chest pain	Present	0 (0%)	3 (10%)	0 (0%)
	Not present	43 (100%)	27 (90%)	30 (100%)
Syncope	Present	6 (14%)	9 (30%)	0 (0%)
	Not present	37 (86%)	21 (70%)	30 (100%)
Treatment plan	Normobaric	37 (86%)	0 (0%)	0 (0%)
	Hyperbaric	6 (14%)	30 (100%)	0 (0%)
	No treatment	0 (0%)	0 (0%)	30 (100%)

SD: Standard deviation, CO: Carbon monoxide, ECG: Electrocardiogram

Table 2. Vital signs and EF status of the patients according to the groups

		Troponin-negative (n=43)	Troponin-positive (n=30)	Control (n=30)
Vital signs	GCS score (median)	15	15	15
	SBP (mmHg)	122.84±11.395	124.67±19.161	119.70±13.35
	DBP (mmHg)	69.51±8.93	67.67±9.91	66.53±9.64
	sPO_2 (%), median	97	95	98
	Pulse/min, median	80	88	80
pH, median (IQR)		7.39 (7.33)	7.38 (7.36)	7.38 (7.34)
Lactate, median (IQR)		1.4 (0.8)	3.0 (2.6)	0.950 (0.5)
Myoglobin, median (IQR)		18.4 (14.4)	31.0 (95.1)	12.5 (7.3)
EF (%), median (IQR)		60 (5)	55 (5)	60 (0)

EF: Ejection fraction, GCS: Glasgow Coma Scale, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, sPO_2: Oxygen saturation, IQR: Interquartile range

Table 3. Evaluation of the patients' NO, CK, CK-MB, troponin, and COHb levels according to the groups					
Parameter	**Group 1**	**Group 2**	**Group 3**	**p**	**Post-hoc**
NO (µmol/L)	45 (26.0)	17.5 (31.3)	110 (95)	0.000	1-2, 1-3, 2-3
Troponin (ng/LT)	2.40 (2.4)	37.0 (1875.5)	1.35 (2.7)	0.000	2-1, 2-3
CK-MB (U/L)	24 (20)	50 (101)	31 (22)	0.000	1-2
CK (U/L)	90 (75)	146 (11)	16 (9)	0.000	3-1, 3-2
COHb (%)	10.2 (15.9)	18.1 (24.3)	0.8 (0.9)	0.000	3-1, 3-2
NO: Nitric oxide, COHb: Carboxyhemoglobin, Group 1: Troponin-negative, Group 2: Troponin-positive, Group 3: Control, CK-MB: Creatine kinase muscle-brain					

Table 4. Evaluation of the relationship between NO and CK, CK-MB, COHb, and troponin in patients with CO poisoning					
	NO	COHb	Troponin	CK	CK-MB
Spearman's rank correlation	1	0-.499	0-.535	0-.537	0-.110
P		0.000	0.000	0.000	0.265
N		103	103	103	103
NO: Nitric oxide, COHb: Carboxyhemoglobin, CK-MB: Creatine kinase muscle-brain					

Table 5. Evaluation of the relationship between NO and length of hospital stay in patients with CO poisoning		
	NO	**Length of hospital stay**
Spearman's rank correlation	1	0-0.725
P		0.000
N		103
NO: Nitric oxide, Carbon monoxide (CO)		

There was an inverse correlation between the NO level and length of hospital stay in patients with CO poisoning (p=0.001). In other words, as the NO value decreased, the length of hospital stay increased (Table 5).

Discussion

In most fatalities caused by CO gas, acute myocardial damage plays an important role in increased long-term mortality. The role of NO in the regulation of heart contraction, heart rate, and vascular tone is significant. In the current study, individuals with CO poisoning who had elevated troponin levels were observed to have a significantly lower NO level compared to both troponin-negative patients and healthy controls. Furthermore, the NO value of the troponin-negative patient group was significantly lower than that of the control group. Further investigations are warranted to explore the efficacy of inhaled NO interventions as a potential measure to prevent future cardiac events and as a supplementary approach to oxygen therapy in individuals with CO poisoning, NO is considered an important intracellular and intercellular, bioactive molecule that affects various physiological and pathophysiological functions in the body, including cardiac contractility and vasodilation regulation (9). During ischemia, NO synthesis increases with increased NO synthase activity. Cardiac damage increases with cellular cytotoxicity. However, oxygenation during reperfusion facilitates delayed NO production, especially in the periinfarct region, and the conversion of NO into peroxynitrite reduces the NO level (10).

In this study, the NO level was found to be very high in the healthy adult control group, while it was the lowest in the group exhibiting elevated troponin values. The NO level also significantly differed between the troponin-negative and troponin-positive patients with CO poisoning, with lower values being detected in the latter.

In animal experiments, endothelial cells have been implicated in the development of ischemic damage. Myocardial ischemia is considered to develop after endothelial damage from CO poisoning. The disruption of the NO synthase enzyme, released from the endothelium due to endothelial damage, decreases NO synthesis. NO has an inverse correlation with ischemia-induced cardiac damage and troponin elevation (11). Similarly, in the current study, it was observed that as the NO level decreased, the troponin level of the patients with CO poisoning increased.

In a study comparing serum NO levels before and after the treatment of CO poisoning, it was found that the NO level was higher after treatment (12). This can be considered an indicator that the NO level decreases in the presence of CO exposure.

It plays an important role in regulating heart contraction, beat rate, and vascular tone. Cardiovascular diseases such as

hypertension, heart failure, ischemic heart disease, coronary artery disease and arrhythmias are associated with impaired NO response (5). Myocardial damage due to CO poisoning occurs with myocardial hypoperfusion; the CO has a toxic effect on myocardial mitochondria. Consequently, various arrhythmias, including ventricular extrasystole and fibrillation, may develop rapidly after CO exposure, without tachycardia or myocardial damage, which can affect mortality (13). In the current study, consistent with the literature, the NO level of the patients with cardiac damage was found to be lower than that of the healthy controls.

Intracellular heme protein function is affected by the partial pressure of at least three gases. These are CO, O^2 and NO. Even at low COHb levels, the CO concentration is 109 times greater than the NO concentration. CO gas binds to the heme competitively with NO. When CO binds to heme, free NO remains in the environment. This free NO has been shown to directly impair myocardial contraction (14). As the CO concentration in the environment increases, NO is expelled from heme proteins and replaced with CO (15).

CK-MB, troponin, myoglobin, and brain natriuretic peptide are the most expressed cardiac proteins in the heart muscle, and they increase after myocardial damage (16-18). The most frequently used biomarkers are troponin I, cardiovascular isoenzyme CK-MB, and CK (19-20). Icme et al. (21) reported statistically significantly higher lactate, CK-MB, and troponin I levels in patients receiving hyperbaric treatment than in those receiving normobaric treatment. In another study, Toksoy (13) detected no correlation between COHb and troponin I levels. In the same study, the mean CK level was found to be 282 U/L, and the mean CK-MB level was 27.45 U/L. In the current study, there was a negative correlation among other cardiac parameters, such as troponin, CK, and CK-MB.

The COHb level is one of the main diagnostic criteria after exposure to CO gas. CO can be produced endogenously and exogenously. In a study conducted by Toksoy (13), a statistically significant difference was found between the presentation complaints of the patients and their COHb levels. In contrast, Hampson et al. (22) found no significant correlation between presentation complaints and COHb values. Karakuzu (23), evaluating retrospective data, determined that high COHb levels did not correlate with clinical findings. Yurtseven et al. (24) detected no correlation between troponin I and COHb levels in patients with CO gas exposure and high troponin levels. In the current study, there was an inverse correlation between the NO and COHb levels, i.e., as the COHb value increased, the NO value decreased.

Study Limitations

The first limitation of our study is the very short half-life of NO, and that NO levels were not examined according to the time of presentation to the hospital in patients with CO poisoning. Moreover, several patients were transported from the incident site by ambulance, and the administration of oxygen therapy through a nasal cannula in the ambulance was a variable that influenced the NO level; however, this treatment was not included in our evaluation. Another limitation of our study is that it is a single-center study and the long-term results are unknown.

Conclusion

Patients with CO poisoning exhibit lower NO levels compared to healthy individuals. The NO level decreases further patients with high troponin levels as compared to in troponin-negative cases. Therefore, it is postulated that inhaled NO therapy in conjunction with CO therapy may serve as a preventive measure against cardiac damage in individuals exposed to CO.

Ethics

Ethics Committee Approval: The study was initiated following the approval of the Ethics Committee of Atatürk Medical Faculty for Clinical Research (decision number: 53, date: 05.11.2020).

Informed Consent: Patients were accepted into the study after obtaining written informed consent from all volunteers or their relatives.

Footnotes

Authorship Contributions

Surgical and Medical Practices: B.K.Ç., A.G., Concept: A.G., Design: B.K.Ç., A.G., Data Collection or Processing: B.K.Ç., A.G., Analysis or Interpretation: A.G., Literature Search: B.K.Ç., A.G., Writing: B.K.Ç., A.G.

Conflict of Interest: The authors declare that they have no conflicts of interest.

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