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The relationship between blood lactate, carboxy-hemoglobin and clinical status in CO poisoning

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Abstract. – AIM: We aimed to determine the relationship between blood lactate, carboxy-hemoglobin (COHb) levels and the severity of clinical findings in patients with CO poisoning.

PATIENTS AND METHODS: Patients over 18 years old and of both gender who were admitted to Emergency Department with the diagnosis of CO poisoning between 10.02.2008 and 17.03.2011 were enrolled in this study. Detailed physical examination of each patient was performed, patients and their relatives were informed about the study and written consents were noted. The levels of consciousness, physical examination findings, electrocardiographic findings, Glasgow Coma Scale (GCS) scores, laboratory results (lactate, COHb, CK-MB, Troponin-I levels) and applied treatments [normobaric oxygen therapy (NBOT), hyperbaric oxygen therapy (HBOT)] were recorded to standart data entry form for each patient. "SPSS for Windows version 18" package program was used for statistical analysis of the data.

RESULTS: Total 201 patients were included in this study. Thirty five patients (17.4%) received HBOT and lactate; COHb, CKMB, Troponin-I levels of this group were higher than the other patients. Lactate and COHb levels were statistically significantly higher in patients with GCS < 15 than the ones with GCS = 15 ($p < 0.01$). The patients whose both Troponin-I and CK-MB levels increased have higher lactate levels ($p = 0.038$), but COHb levels of these patients did not change ($p = 0.495$).

CONCLUSIONS: According to our study, blood lactate and COHb levels were both correlated with the changes of consciousness in CO poisoning. Blood lactate levels together with COHb in defining indications for HBO treatment might be suggested.

Key Words:
Carbon monoxide, Poisoning, Carboxyhemoglobin, Hyperbaric oxygen therapy, Lactate

Introduction

CO is a colorless, odorless, tasteless, non-irritant gas and it is easily absorbed in the lungs. CO is released as a result of incomplete burned fuel, can cause acute poisoning resulting in death. In the survivors neurological and psychiatric sequelae can be seen. Especially in regions where the climate is colder, the use of stoves, water heaters and barbecues without funnels in small spaces with inadequate ventilation causes CO poisoning and these patients frequently admitted to Emergency Services[1,2].

Most of the CO gas taken via inhalation is excreted unchanged from the lungs, 10 to 15% of it bind to myoglobin and cytochrome proteins and less than 1% dissolve in the plasma. Hemoglobin binding affinity for CO is 200-250 times greater than its affinity for oxygen. Therefore, CO quickly and easily binds with hemoglobin and displaces oxygen and as a result carboxyhemoglobin (COHb) occurs. COHb reduces the O_2 carrying capacity of blood and causes hypoxia, ischemia and tissue necrosis. At the same time CO makes it difficult to give O_2 to tissues by making structural changes in hemoglobin (carbon monoxide entry into the cell causes an inhibitory effect on cellular heme proteins, such as myoglobin), it also binds to reduced cytochrome a3 and by inactivating, it impairs cellular respiration[2-4].

Reduction in oxygen delivery causes lactate formation particularly in tissues such as brain, intestine, erythrocytes and skeletal muscle where high rate of glycolysis occurs[5]. The increase in

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lactate levels is an indicator of compromised oxygen delivery and correlated with the short term prognosis in critically ill patients[6].

In this study, we aimed to determine the relationship between blood lactate, carboxy-hemoglobin (COHb) levels and the severity of clinical findings in patients with CO poisoning.

Patients and Methods

We planned this prospective, randomized study and started after approval of Ethics Committee. Patients over 18 years old and of both gender who were admitted to emergency department with the diagnosis of CO poisoning between 10.02.2008 and 17.03.2011 were enrolled in this study. The diagnosis of CO poisoning was made due to the presence of water heater or stove poisoning in patients' history and determination of the amount of peripheral blood COHb to be > 5 g/dl. Detailed physical examination of each patient was performed, patients and their relatives were informed about the study and written consents were noted. The levels of consciousness, physical examination findings, electrocardiographic findings, Glaskow Coma Scale (GCS) scores, laboratory results (lactate, carboxy-hemoglobin (COHb), CK-MB, troponin-I levels) and applied treatments (normobaric oxygen therapy (NBOT), hyperbaric oxygen therapy (HBOT)) were recorded to standart data entry form for each patient. CK-MB levels higher than 3.75 ng/ml and Troponin I levels higher than 50 ng/ml were considered to be high. Syncope, coma, convulsion, cardiac ischemia or ventricular rhythm disturbances, COHb levels over 25%, COHb level 20% and over in pregnancy were considered as indications for HBOT and the other patients received only NBOT therapy.

Statistical Analysis

"SPSS for Windows version 18" package program (SPSS Inc., Chicago, IL, USA) was used for statistical analysis of the data. Student's *t* test was used in comparisons of normally distributed variables of quantitative data between two groups as well as descriptive statistical methods (mean, standard deviation, frequency). Spearman's rho correlation test was used for assessing the relationships between variables. According to the results, *p*-value < 0.05 was considered statistically significant.

Results

A sum of 305755 patients admitted to our Emergency Department (ED) during the study period and 210 of these patients (0.0006%) were diagnosed with CO poisoning. 201 patients who have written consents were included in the study. Of these patients 100 (49.5%) were female and 101 (50.5%) were male. The mean age was 36 ± 17 years. On admission to the Emergency Department 15.4% of patients (n=31) had no complaints, 22.9% of them (n=46) had nausea and vomiting, 20.9% of patients (n=42) had headache and 27.9% of patients (n=56) had headache and vomiting. Syncope (11 patients, 5.5%), mental confusion (9 patients, 4.5%), chest pain (3 patients, 1.5%), dyspnea (2 patients, 1%) and palpitation (1 patient, 0.5%) occurred in patients. ECG changes were observed in 15.2% of the patients (n=32). The most detected ECG changes were sinus tachycardia (25 patients, 12.4%) and others were ST segment depression and T inversion (6 patients, 3%), right bundle branch block and right axis deviation (1 patient, 0.5%) respectively. GCS score was 15 in 10 patients (5%).

Accordingly, 17.4% of the patients (n=35) had received HBOT and 82.6% (166 patients) had received only NBOT. There was no statistically significant difference between the two groups in terms of CO_2, O_2, HCO_3 levels in the first blood gas analysis performed in the ED on admission. But COHb, lactate, CK-MB and Troponin I levels were statistically higher in patients receiving HBOT (Table I).

Ten patients with GCS < 15 were sent to HBOT. According to the first laboratory results of this group, COHb and lactate levels were significantly higher than those of GCS=15 (Table II).

Table I. The comparison of first laboratory results of patients receiving HBOT and NBOT.

	HBOT (mean ± SD)	NBOT (mean ± SD)	*p* value
CO_2	31 ± 6	31 ± 5	0.843
HCO-	22 ± 4	23 ± 2	0.321
O_2	79 ± 16	74 ± 19	0.322
COHb	27 ± 7	18 ± 7	0.001
Lactate	35 ± 28	17 ± 8	0.001
CK-MB	9 ± 18	2 ± 2	0.044
Troponin-I	0.7 ± 1	0.06 ± 1	0.047

SD = Standard Deviation; NBOT = Normobaric oxygen therapy; HBOT = Hyperbaric oxygen therapy.

Table II. The Comparison of COHb and lactate levels of patients according to GCS.

	COHb (mean ± SD)	Lactate (mean ± SD)
GCS < 15	31.44 ± 7.9	50.2 ± 30
GCS = 15	19.52 ± 5.2	18.77± 13
p value	< 0.01	< 0.01

CK-MB and Troponin-I levels were increased in 10 patients and they were sent HBOT. In these patients lactate levels were higher than the other patients, whereas COHb levels were not different (Table III).

There was a positive correlation between the lactate levels and COHb, CK-MB, Troponin-I levels. There was a negative correlation between lactate, COHb levels and GCS. There was no correlation between COHb and CK-MB, Troponin-I levels (Table IV).

Thirteen patients (6.2%) were treated in the intensive care unit and 197 patients (93.8%) were followed up in the Emergency Observation Unit. All patients had been discharged. No mortality was seen due to CO poisoning.

Discussion

The rapid binding of carbon monoxide to hemoglobin causes tissue hypoxia by lowering O_2 carrying capacity of blood. Brain, heart and kidneys are the most sensitive organs to the hypoxic effects of CO exposure because of high oxygen demand. Central nervous system involvement is responsible for most symptoms of CO poisoning[4]. In the treatment of CO poisoning O_2 which competitively removes carbon monoxide from the regions where it was bounded constitutes the basis of treatment. The elimination of carbon monoxide in the blood may be accelerated by increasing the concentration of O_2 in the breathing air or by increasing the atmospheric pressure[2,4]. NBO or HBO are the modalities used for this purpose[7]. However, certain objective criteria are not defined for the implementation HBO in CO poisoning. It is often decided according to clinical findings. Loss of consciousness, neurological pathology signs, evidence of cardiac ischemia and severe acidosis are considered to be the indications of hyperbaric oxygen therapy[2]. The COHb level is important among biochemical parameters. COHb levels higher than 25%, COHb levels of 20% and higher in patients with ischemic heart disease and 15% and higher in pregnant women are considered to be indications for HBOT.

It must be noted that the CO levels which were higher before may be measured lower at the first admission of the patient to the Clinic, depending on the transportation process[8,9]. On the other hand, there are several publications suggesting an association between COHb levels and the severity of clinical symptoms (especially neurological symptoms). Keles et al[10] reported that with high-

Table III. The comparison of COHb and lactate levels in patients with and without cardiac enzyme elevation.

	Patients with elevated cardiac enzyme levels (mean±SD)	Patients with normal cardiac enzyme levels (mean±SD)	*p* value
COHb	18 ± 12	22 ± 10	0.495
Lactate	45 ± 34	19 ± 8	0.038

Table IV. The correlations between COHb and lactate levels of all patients with GCS, CK-MB, and Troponin I.

	COHb	CK-MB	Troponin I	GCS
Lactate				
Spearman's rho correlation	r = 0.331	r = 0.449	r = 0.313	r = –0.325
p value	*p* = 0.001	*p* = 0.001	*p* = 0.001	*p* = 0.001
COHb				
Spearman's rho correlation	1	r = 0.073	r = –0.053	r = –0.267
p value		*p* = 0.342	*p* = 0.491	*p* = 0.001

er COHb levels (≥ 20%) they observed an increase in the frequency of syncope. In another study[3] it is observed that in patients who didn't received O_2 therapy have higher mean COHb levels and neurological symptoms. Depending on these results, it is reported that COHb level is an important indicator of diagnosis and clinical follow-up in acute CO poisoning, but low COHb levels do not exclude the diagnosis.

According to results of our study it is observed that COHb levels were statistically significantly higher in patients with GCS < 15 than the patients with GCS = 15. We detected a negative correlation between GCS and COHb. This finding is similar with the results of the study of Aslan et al[11]. Transient myocardial dysfunction or signs of ischemia (ST and T wave changes, increased myocardial enzymes), pulmonary edema and symptoms ranging from cardiogenic shock to rhythm disorders may be seen in CO poisoning[3,12-14]. However, the studies evaluating the relationship between COHb level and cardiac involvement are controversial. Satran et al[12] suggested COHb levels among risk factors for myocardial damage. Yelken B et al[14] reported a correlation between COHb levels and QT interval, CK-MB, troponin-I levels. Moreover, Hajsadeghi et al[13] reported that COHb level was high only in the cases with T wave inversion and there was no significant difference with other ECG changes. Aslan et al[11] detected no correlation between COHb levels and CK, CK-MB levels. We have obtained similar results like this study. There was no significant correlation between COHb levels and CK-MB, Troponin I levels in our study.

Lactate levels were significantly higher in patients with GCS < 15 than the patients with GCS = 15 in our study. We also observed that the patients whose both troponin-I and CK-MB levels increased have higher lactate levels, whereas COHb levels of these patients did not change. In the study of Benaissa et al[15], the lactate levels were increased in patients who developed neurological symptoms in CO poisoning. Besli et al[3] identified a positive correlation between lactate and COHb levels and detected higher lactate levels in patients with neurological findings. Sokal et al[16] reported that lactate levels of patients with severe CO poisoning are significantly higher than mild poisoning. Inoue et al[17] reported that initial lactate levels may be associated with clinical course and outcome. They suggested that it can be used as a good marker for clinical course. Moon at al[18] suggested that the first lactate value is an independent factor for the change of mental status and determining the need for intensive medical treatment and it may be useful in predicting prognosis.

Conclusions

Blood lactate and COHb levels were both correlated with the changes of consciousness in CO poisoning. Blood lactate levels together with COHb in defining indications for HBO treatment might be suggested.

Conflict of Interest

The Authors declare that there are no conflicts of interest.

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