Electrocardiographic Findings and Serum Troponin I in Carbon Monoxide Poisoned Patients

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Abstract- Carbon monoxide (CO) poisoning, though with different sources, is one of the most deadly emergencies in all countries. CO can threaten men's life by several paths especially cardiac complications, which can mimic other cardiac problems such as myocardial infarction. The objective of this study was to determine ECG findings and serum troponin I levels in CO poisoned patients. In this analytical cross-sectional study, 63 CO poisoning patients were consecutively included from hospital's emergency departments. CO content was measured by a CO-oximeter and an electrocardiography was taken first thing on admission. Arterial blood gas (ABG), troponin I and other data was collected afterwards. Data were divided by age groups (adults and children) and gender. CO content was significantly higher only in subjects with normal T wave compared to patients with inverted T wave in their initial ECG (P=0.016). No other significant difference was noticed. None of the ABG findings correlated significantly with CO content. Also no significant correlation was found with CO content after stratification by gender and age groups, but pH in children (r=-0.484, P=0.026). CO content was significantly higher in adults (P=0.023), but other ABG data were not significantly different. Only 3 patients had elevated troponin I. Receiver operating characteristic (ROC) analysis showed no significant cutoff points in CO content for ECG changes. No significant specific change in electrocardiograms (ECG) could contribute carboxyhemoglobin content in carbon monoxide poisoned patients. In addition, no specific difference was found between adults and pediatric subjects' ECGs. All other findings seemed to be accidental.

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Introduction

Carbon monoxide (CO) is an odorless, non-irritable and poisonous gas, present at a concentration of 0.001 in the atmosphere (1). In all individuals, small amounts of carbon monoxide are present and important for several physiologic functions such as neurotransmission (2). Exogenous exposure to relatively mild to moderate amounts of carbon monoxide can induce either a protective or an adaptive state, but exposure to higher levels may result in toxic effects. (3) Carbon monoxide poisoning is a common condition; reported in more than 15,000 emergency department visits and 500 deaths in the United States each year (4). Sources of exogenous CO in patients with CO poisoning include fumes from automobile exhausts, poorly functioning heaters and burning charcoal as well as inhaled industrial smoke (5). CO poisoning has severe clinical effects including a high rate of death from toxic poisoning which must be considered as an important health problem in many countries (6,7). This gas causes hypoxia by forming carboxyhemoglobin and shifting the oxyhemoglobin dissociation curve to the left (1,3). Carbon monoxide's affinity for hemoglobin is 200-250 times more than oxygen, resulting in the formation of carboxyhemoglobin even with inhalation of relatively low amounts of CO (8,9). The symptoms of CO poisoning are non-specific. The major deleterious effect of CO exposure is decreased oxygen (O_2) supply to body tissue (3,10,11).

In recent years, cardiac disorders have been reported in mild to severe CO poisoned patients. These disorders

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are indicated by the presence of elevated cardiac biomarkers, e.g., troponin I, B-type natriuretic peptide, creatinine phosphokinase (CPK), and creatinine kinase-myocardial band (CK-MB) and by electrocardiographic (ECG) abnormalities e.g., ST segment and T wave changes, sinus tachycardia, premature atrial and ventricular contractions, decreased left ventricular ejection fraction and right ventricular dysfunction (12-18). Reports of the cardiovascular consequences of CO poisoning are limited to isolated cases and case-control studies of ECG changes, myocardial dysfunction, and myocardial infarction; thus, severity and duration of cardiac abnormalities are not well known. None of the previous studies has investigated all cardiac monitoring factors at the same time. Furthermore, most of them have not taken into consideration a victim's history of heart disease. (16-22). According to these points and the high number of cases of CO poisoning in Iran and all over the world (6,23-24), we conducted a study on ECG and biochemical changes in patients with acute CO poisoning.

Materials and Methods

Subjects

In this analytical cross-sectional study, sixty-three diagnosed CO poisoned subjects were consecutively included. Diagnosis of CO poisoning was made according to carboxyhemoglobin levels of more than 5% and the subject's present medical condition as described by themselves, their next of kin or the emergency service personnel from the ambulance. All patients were recruited to Loghman-e-Hakim and Rasoul-e-Akram Hospital Emergency Departments (ED), Tehran, Iran, by state ambulances between November 2009 and April 2010. The hospitals were known as poisoning emergency referral hospitals in western and southern parts of Tehran. Patients with prior known cardiac diseases were excluded from the study. This was taken from their medical record or past medical history given at the time of admission. Each patient or their next of kin was informed about the study and all of them consented to give access to their medical information for the purpose of this research. This investigation was approved by the ethical committee of Iran University of Medical Sciences and all authors agreed on adherence to the Helsinki announcement.

Data collection and measurements

Patients' demographic data and previous medical history were collected from their next of kin, registered documents, or hospital records. All data were collected and moved recorded on to a pre-prepared checklist. On admission, the patient's blood samples were taken; blood gas analysis samples were collected from the radial artery in a heparinized syringe and troponin I levels were measured in blood samples collected from antecubital veins in anticoagulant added tubes. Carboxyhemoglobin measurements were performed with Rapidlab 865 (Bayer) oximeter. Troponin I levels were measured with a one step immunoassay sandwich method using monoclonal antibodies (Biomérieux Vidas, Indianapolis, Indiana, USA). Test levels higher than 0.1 μg/ml were accepted as positive. Blood pressure was measured at the same time by a mercury-based manometer (Richter, Germany)

An Electrocardiography was done immediately after admission to the ED. For the purpose of our study, patients were divided into 2 groups; children (age less than 15 years) and adults (age more than 15 years), and their ECG results were reviewed by either a cardiologist or a pediatric cardiologist. ECG indices like rate, axis, rhythm, ST segment position, Invert T wave and pathologic Q wave formation, QT and PR interval and QRS duration and any significant R wave in avR lead were evaluated and recorded. Changes were recorded as positive if they were present in at least 2 adjacent leads.

Data analysis

All categorical data were expressed as absolute counts and percentages and Chi-square tests were performed to compare patient characteristics from different groups. Ordinal and continuous data were expressed as mean (SD), and were compared using the Student's t-test for normally distributed variables and the Mann-Whitney U-test if either of these conditions were not met. Receiver operating characteristics (ROC) analysis was used for the detection of a cutoff point for CO levels to predict cardiac damage due to poisoning. *P* values less than 0.05 were considered significant.

Results

Baseline evaluation

Sixty-three individuals were evaluated; consisted of 32 females and 31 males. The mean age was 23.48±15.70 years. Mean CO content was 22.17±8.84%. Met-hemoglobin levels were 1.67±0.73%. Mean pH was 7.35±0.10.

Troponin I was elevated only in 3 (4.8%) patients. Sinus tachycardia was seen in 35 (55.6%) cases. There was only one case of right axis deviation recorded from

our subjects. On ECG evaluation, 4 (6.4%) patients had ST segment elevation and 13 (20.6%) had ST segment depression, all of them in inferior (II, III and avF) leads. Only 1 patient had inverted T wave in classic (I, II, III) leads and 7 (11.2%) in chest leads (5 in V1-V4 and 2 in V1-V6). Wide QRS complex was seen in 15 (23.8%) subjects. No pathological Q wave was detected.

Tables 1 and 2 demonstrate the baseline characteristics based on gender for quantitative and qualitative data, respectively. As seen, there were no significant differences between male and female subjects in these factors.

Adults and children's baseline characteristics and blood content comparison

CO content was significantly higher in adults (*P*=0.023), but other ABG data were not significantly different. Other results are shown in Table 3. Table 4 contains information of patients with ECG changes according to the specified age groups. Sinus tachycardia was proportionately significantly high (*P*=0.004). In addition, the percentage of pediatric patients with significant R wave in avR lead was significantly higher, but other ECG findings did not show any significant difference.

In correlation analysis, none of the ABG findings correlated significantly with CO content. Except pH in children (r=-0.484, *P*=0.026), no significant correlation was found with CO content after stratification by gender and age.

We calculated the mean age of each ECG findings and compared them. This value was significantly higher only in patients with ST segment depression compared with patients with normal ST segment (*P*=0.001). Other specific ECG changes did not show any significant differences in mean age.

Table 1. Baseline characteristics of subjects, divided by gender.

	All Subjects	Male (n=31)	Female (n=32)	*P* value*
Age (years)	23.48 ± 15.70	24.48 ± 16.899	22.5 ± 14.66	0.620
CO content (%)	22.17 ± 8.84	23.00 ± 9.02	21.37 ± 8.72	0.471
Methemoglobin Content (%)	1.67 ± 0.73	1.71 ± 0.75	1.63 ± 0.73	0.673
pH	7.35 ± 0.091	7.357 ± 0.098	7.353 ± 0.085	0.888
PCO_2 (%)	36.73 ± 7.92	37.38 ± 9.28	36.1 ± 6.43	0.528
BE	-4.69 ± 14.77	-6.47 ± 20.46	-3.37 ± 5.16	0.410
HCO_3 (mM)	21.22 ± 4.93	21.75 ± 4.85	20.7 ± 5.04	0.403
PO_2 (%)	64.17 ± 40.12	61.26 ± 34.94	67 ± 44.95	0.575
Systolic Blood pressure (mmHg)	105.17 ± 24.16	105.96 ± 15.35	104.4 ± 13.64	0.671
Diastolic Blood Pressure (mmHg)	69.81 ± 9.68	70.32 ± 9.93	69.31 ± 9.57	0.683
Pulse (beat/min)	106.57 ± 24.16	106.58 ± 24.64	106.56 ± 24.09	0.998

* Student's t-test

Table 2. Electrocardiographic changes in subjects, divided by gender.

	All subjects	Male (n=31)	Female (n=32)	*P* value*
Sinus Tachycardia	35 (55.6%)	17 (54.4%)	18 (56.2%)	0.91
ST elevation	3 (4.7%)	1 (3.2%)	2 (6.2%)	0.589
ST depression	13 (20.6%)	8 (25.6%)	5 (15.5%)	0.318
Inverted T wave	8 (15.8%)	4 (13.1%)	4 (12.4%)	0.962
QT interval Prolongation	12 (19%)	5 (16.1%)	7 (21.7%)	0.561
PR interval Prolongation	9 (14.3%)	5 (16.1%)	4 (12.4%)	0.611
P wave widening	9 (14.3%)	6 (19.3%)	3 (9.3%)	0.254
QRS widening	15 (23.8%)	8 (25.6%)	7 (21.7%)	0.714
Significant R wave in avR lead	17 (27%)	10 (32.2%)	7 (21.7%)	0.353

* Chi-square test

Table 3. Quantitative parameter in children and adults. As seen above, blood pressure and pulse rate are significantly different. Significant *P* values are presented in bold font.

	CO[1]	Met[2]	pH	PCO_2[3]	HCO_3[3]	PO_2[3]	SBP[4]	DBP[4]	Pulse rate
Children (n=21)	18.6 ± 7.8	1.74 ± 0.74	7.366 ± 0.086	35.25 ± 6.9	20.22 ± 4.3	71.66 ± 39.42	98.62 ± 11.62	66 ± 8.6	122.38 ± 18.3
Adult (n=42)	23.95 ± 8.8	1.64 ± 0.74	7.354 ± 0.094	37.48 ± 8.3	21.72 ± 5.1	60.43 ± 40.40	108.45 ± 14.4	71.71 ± 9.7	98.67 ± 22.90
P value*	0.23	0.593	0.511	0.293	0.258	0.299	**0.01**	0.026	> **0.001**

1: CO content (percent), 2: Methemoglobin content (percent), 3: percent, 4: Systolic and diastolic blood pressure (mmHg), 5: Pulse rate per minute.

* Student's t-test

Table 4. Number of subjects with specific electrocardiographic findings divided by age groups is shown. Significant P values are presented in bold font.

	Sinus Tachycardia	ST segment elevation	ST segment depression	Inverted T wave	QT interval prolongation	PR interval prolongation	P wave prolongation	Widened QRS	Significant R wave in avR lead
Children (n=21)	17 (79.9%)	0	3 (14.2%)	5 (23.5%)	5 (23.5%)	3 (14.2%)	4 (18.8%)	4 (18.8%)	9 (42.6%)
Adults (n=42)	18 (42.8%)	3 (7.1%)	10 (23.8%)	3 (7.1%)	7 (16.6%)	6 (14.2%)	6 (11.9%)	11 (26.1%)	8 (19.4%)
P value*	**0.004**	**0.001**	0.367	0.07	0.502	1	0.453	0.53	**0.045**

* Chi-square test

CO content and Electrocardiography analysis

The average CO content in patients with or without each ECG changes is demonstrated in Table 5. CO content was significantly higher only in subjects with normal T wave compared to patients with inverted T wave in their initial ECG (*P*=0.016). No other significant difference was noticed.

For further analysis, we divided our subjects into 2 other groups; one with and another without any ECG changes; the groups consisted of 45 and 18 subjects, respectively. All parameters were calculated and compared between these groups. No significant difference was found between the number of individuals with ECG changes within gender (*P*=0.30) and age groups (*P*=0.76). We combined the number of patients with ST segment depression and elevation, and formed a group that demonstrated the patients with ST segment changes. Patients with ST segment changes had significantly higher ages compared to others, *P*=0.028.

In ROC analysis, CO content, PO_2 and met-hemoglobin percentages in blood were evaluated for positive ECG changes. No suitable cutoff point was found (Figure 1). The best cutoff point for CO content was 23.5% (42% sensitivity, 78% specificity; AUC=0.532 and *P*=0.692), for PO2 was 55.5 (55% sensitivity, 78% specificity; AUC= 0.609 and *P*=0.178) and for met-hemoglobin content was 1.00 (60% sensitivity, 18% specificity; AUC= 0.428 and *P*= 0.378).

Table 5. CO content (%) in each group is calculated in each ECG findings, divided by having or not having the specific change.

	Sinus tachycardia	ST segment elevation	ST segment depression	Inverted T wave	QT interval prolongation	PR interval prolongation	P wave prolongation	QRS widening	Significant R wave in avR lead
Yes	23.29 ± 9.3	21.66 ± 8.65	21.75 ± 9.01	23.18 ± 8.7	21.75 ± 9.01	21.5 ± 8.94	21.76 ± 8.88	21.88 ± 8.65	22.43 ± 8.37
No	22.00 ± 8.58	29.75 ± 9.28	24.01 ± 8.16	15.25 ± 6.4	24.00 ± 8.16	26.22 ± 7.37	24.67 ± 8.55	23.13 ± 9.66	21.47 ± 10.37
P value*	0.863	0.076	0.431	0.016	0.431	0.131	0.365	0.634	0.704

* Student's t-test or Mann-Whitney U-test

Table 6. Arterial blood gas findings in subjects divided by weather having Specific ECG changes or not.

	CO[1]	Met[2]	pH	PCO_2[3]	BE[4]	HCO_3[3]	PO_2[3]
With ECG findings (n=45)	21.28 ± 8.4	1.82 ± .68	7.362 ± 0.098	37.81 ± 6.75	-3.2 ± 7.6	22.2 ± 4.68	53.87 ± 40.34
Without ECG findings (n=18)	22.53 ± 9.00	1.61 ± 0.75	7.352 ± 0.089	36.30 ± 8.37	-5.5 ± 16.8	20.8 ± 5.02	68.3 ± 39.79
P value*	0.615	0.325	0.688	0.497	0.569	0.293	0.2

1: CO content (percent), 2: Methemoglobin content (percent), 3: percent, 4: Base excess.

* Mann-Whitney U-test

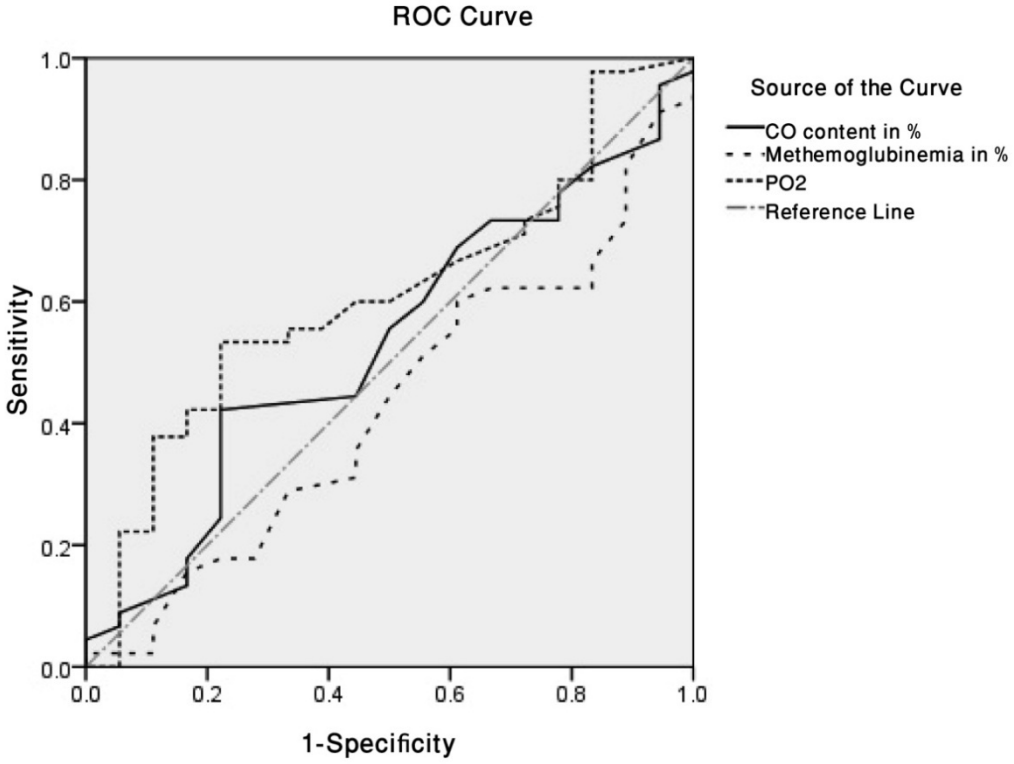

Figure 1. ROC curve conducted for CO content, Met-hemoglobin levels and atrial O2 pressure to find a cut-off point to find at least ECG change in CO poisoned patients. No significant value could be extracted.

Discussion

This study evaluated detailed changes of ECG in patients with CO poisoning. No specific changes in ECG were found that could be specifically related to high CO or methemoglobin contents. Even after dividing our subjects into adults and children, the only parameter that differed significantly was the number of patients with sinus tachycardia (*P*=0.004). Comparing the ECG changes between adults and children, as far as we know, was done for the first time in this study.

It is well known that CO poisoning can exacerbate angina and cause cardiac injury, even in persons with normal coronary arteries. Therefore, it can be recommended that poisoned patients undergo cardiovascular investigation (12-24). If cardiac injury is present, a cardiology consultation is recommended. But there was not a precise study in which changes may have been specifically induced by CO poisoning, because all papers concentrated on ischemic changes (12,14,16,24). The mechanism that most researchers accept is the competitive inhibition of oxygen release due to a shift in the oxygen-hemoglobin dissociation curve, causing reduced oxygen delivery, and resulting in subsequent tissue hypoxia (1-3,6-8). In the living cell, CO binds to cytochrome oxidase of the electron transport chain, resulting in asphyxiation at the cellular level (15). High concentrations of CO have been shown to induce cellular apoptosis mediated by nitric oxide (25). In preclinical models, global and relative sub-endocardial hypoperfusion was seen in CO poisoned dogs (26). But as seen in this study, there were other changes that cannot be fully explained by ischemia of heart muscles. Prolonged intervals associated with some

ischemic ECG manifestations such as ST alternation or wide QRS (as the most frequent changes in most subjects of this study), could not be contributed to hypoxic and ischemic changes alone. While this study cannot correlate any cause to these changes, ischemia is the most likely theory that can be applied (12-18).

There are not many studies on CO poisoning in relation to ECG changes. A study in Minnesota, USA, showed that ischemic ECG changes were present in 30% of patients, but only 16% of their population had a normal ECG. Cardiac biomarkers (CK-MB or troponin I) were elevated in 35% of patients (15). Compared to the results of this study, demonstrating that 28% or our subjects did not have any ECG changes and only 3 patients had elevated troponin I levels. This difference may originate from single evaluation of troponin I in our patients due to their short length of stay in the ED. Another study in Turkey demonstrated that P wave duration and QT interval duration and corrected QT (QT_c) were significantly longer in CO poisoned patients compared with normal population (17).

In the literature, reports of children with cardiac findings, including long QT, ST-T wave changes, fibrosis and cardiogenic shock, were published in a few case reports (12,16,22,27-28). Yanir *et al.* presented an 8-year-old girl with reversible cardiac failure, including mild myocardial ischemic findings, such as T wave inversion on ECG, reduction in left ventricular function, low ejection fraction, and moderate mitral regurgitation, despite the lack of increase in cardiac enzyme levels (16). Similarly, Gandini *et al.* reported a child with cardiac damage associated with a relatively low carboxy-hemoglobin level after prolonged CO exposure. This patient had prolonged QT interval and ST-T changes recorded by ECG (12). As reported in several studies, it is very difficult to identify patients with CO-induced cardiac damage due to the lack of correlation between CO levels and the symptoms of CO poisoning, for both adult and pediatric patients (12-18).

In study by Kalay *et al.,* cardiovascular manifestations in adult patients with CO contents greater than 25% were treated with Hyperbaric Oxygen Therapy; of which 35% of the study population had positive cardiac biomarkers for myocardial injury and 30% of them had diagnostic ischemic changes on ECG (14). In this study, the frequency of pediatric patients with myocardial injury was lower compared to the adult patients in this study. Furthermore, patients with ST segment changes, identified by a notable change in myocardial ischemic damage, were significantly older compared to other patients. We theorized that different inclusion criteria used in Kalay *et al.* (14) study may have had an effect on the frequency of patients with myocardial injury. As our study population included all patients with CO poisoning. However, they included more critical patients. The other possible reason could be that adult patients were more prone to cardiovascular complications.

Study limitations and recommendations

In this study, we could not evaluate the time length that patients were exposed to CO. This can influence the results based on some previous studies that have shown the possibility of higher cardiac damage due to longer exposure time. Furthermore, the patient's length of stay at hospital could not be retrieved due to fast discharge from hospital in the majority of cases. We suggest that other studies should consider a larger sample size to evaluate other biomarkers such as CK in multiple sessions and other cardiac parameters like echocardiographic indices with the consideration of more factors, for example, considering the time that patients were exposed to CO and the type and source of CO present in their environments.

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