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Role of Electrocardiogram, Cardiac Biomarkers and Echocardiogram in Diagnosing Acute Carbon Monoxide Induced Myocardial Injury

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Abstract

Background: Carbon monoxide (CO) poisoning is a common accidental cause of toxicological morbidity and mortality worldwide.

Aim of this study: the aim of this study was to evaluate the cardiovascular manifestations of acute CO poisoning and the value of ECG changes, cardiac biomarkers (troponin I and CK-MB) determination and echocardiography in diagnosing cardiac damage in acute carbon monoxide poisoning cases. Additionally, evaluate the impact of delayed time of hospital presentation and duration of exposure on these parameters.

Subjects & Methods: This is a cross-sectional study that was conducted on admitted patients with acute carbon monoxide poisoning to Toxicology Unit, Emergency Hospital, Tanta University in the period from the first of March 2014 to the end of September 2015. All patients were subjected to history taking and clinical examination. Additionally, ECG, echocardiogram, cardiac biomarkers (creatinephosphokinase CK-MB, and troponin I), and carboxyhemoglobin (COHb) levels were performed for all patients.

Result: the age of the studied patients ranged from 15-55 years, the delay time from 1.5-10 hours and the duration of exposure to CO ranged from 0.5 to 5 hours. The most common cardiovascular manifestations were tachycardia; occurred in 50.9% of the patients then dyspnea (24.6%), chest pain (12.3%) and lastly hypotension (10.5%). The ECG findings showed that 48.2% of the studied patients had sinus tachycardia, 5.4% had T-wave inversion or ST depression and combination of T-wave inversion and ST depression occurred in 3.6% of the patients. All patients who had ECG suggestive of cardiac ischemia had abnormal CPK-MB, troponin I and cardiac wall motion abnormalities in echocardiography. Furthermore, significant negative correlation between delay time and duration of exposure with Glasgow coma scale was evident as well as significant positive correlation between both ECG & echocardiographic changes and delayed presentation.

Conclusion: the present study revealed that sinus tachycardia was the most common cardiac manifestation of acute CO poisoning. ECG finding that suggested myocardial ischemia occurred in only 14.4% of the patients. Therefore, a baseline ECG should be routinely performed but, it is not necessary to routinely measure troponin I and do echocardiography. But, it can be restricted to only cases with abnormal ECG findings.

Keywords Carbon monoxide; Poisoning; ECG; Troponin I; CK-MB; Echocardiogram.

Introduction

Carbon monoxide (CO) was considered to be the cause of more than one-half of the fatal poisonings reported in many countries. Fatal cases also are grossly under-reported or misdiagnosed by medical professionals. Therefore, the precise number of individuals who have suffered from CO intoxication is not known (Raub et al., 2000). An Egyptian study that was done in the Poison Control Center (PCC), Ain Shams University Hospitals in Cairo, showed that CO poisoning represented the 6th most frequent toxic exposure admitted to the PCC in 2004 (Gamalludin et al., 2005).

CO is a highly toxic gas, and its contribution to the hazardous effects of increasing air pollution is a major public health concern. Exposure to CO arises from incomplete combustion of hydrocarbons. Sources include motor exhaust fumes, gas appliances, tobacco smoke, fires, stoves, and portable heaters (Centers for Disease Control, 2004).

CO intoxication often presents a significant challenge. There is a wide spectrum of clinical features of CO poisoning; dizziness, nausea, weakness, headaches up to lethargy and confusion. Pallor of the skin is common, while classic cherry red skin is rare. Ophthalmic, noncardiogenic pulmonary edema, neurologic and/or neuropsychiatric complications were also reported (Henry et al., 2006; Lane et al., 2008).

Diagnosis of acute myocardial infarction relies upon the clinical history, interpretation of the electrocardiogram, and measurement of serum levels of cardiac enzymes. Biochemical markers of myocardial injury, such as cardiac troponin I was used instead of or along with the standard marker, the MB isoenzyme of creatine kinase (Jaffe et al., 2000).

The organs most affected by CO intoxication are those most dependent upon oxygen for their function. Most notable of these are the brain and heart (Gunn and Carter, 2005). Carbon monoxide cardiotoxicity may be clinically occult and often remains undiagnosed because of the lack of overt symptoms and specific ischemic changes in the electrocardiogram (Gandini et al., 2001; Gunn and Carter, 2005). So, Carbon monooxide cardiotoxicity commonly misdiagnosed and CO induced myocardial damage is not completely understood as a clinical entity in terms of both pathophysiology and clinical features.

The aim of this study was to evaluate the cardiovascular manifestations of CO poisoning and the value of ECG changes, cardiac biomarkers (troponin I and CK-MB) determination and echocardiography in diagnosing cardiac damage in acute carbon monoxide poisoning cases. Additionally, evaluate the impact of delay time and duration of exposure on these parameters.

Subjects and methods

This is a cross-sectional study that was conducted on admitted patients with acute carbon monoxide poisoning to Toxicology Unit, Emergency hospital, Tanta University in the period from the first of March 2014 to the end of September 2015.Pregnant patients, patients with medical disease such as diabetes, hypertension, anemia, smoking, dyslipidemia, history of coronary artery disease and myocardial infarction and patients with mixed poisoning or prehospital treatment were excluded from the study. An informed consent was obtained from every patient or his guardian before the start of the study. Privacy of patients and confidentiality of data and the results of investigations were maintained by using coding number.

At admission, all patients were subjected to the following:

I- History:

-Personal history (age, sex) and toxicological history: it included manner of poisoning, delay time and the duration of CO exposure.

II- Clinical examination: it includedpupil size, vital signs, level consciousness, and systems examination (central nervous, respiratory, cardiovascular, gastrointestinal and urinary systems examination).

III- Investigations:

1- **Carboxyhemoglobin (CO-Hb) level:** it was done by Co-oximeter.

2- **Laboratory investigations:** laboratory investigations were carried on blood. Two samples of blood were drawn from each subject under the study. The non heparinized blood sample (4ml) was used for measurement of serum creatine phosphokinase (CK-MB) and troponin Ilevels using the commercially available enzyme immunoassay kits according to the manufacture instructions. The heparinized blood sample (4ml) was used for evaluation of arterial blood gases (ABG).

3- **Electrocardiogram (ECG):** 12 lead ECG was done for every patient.

4-Echocardiogram: it was done to detect the presence or absence of wall motion and was reviewed independently from clinical data by a cardiologist.

Treatment protocol: All patients with CO toxicity are usually treated with high-flow oxygen by face mask in addition to symptomatic treatment as indicated. Hyperbaric oxygen treatment is indicated in the following conditions; COHb level above 25 percent, evidence of ongoing end-organ ischemia (eg, profound metabolic acidosis, myocardial ischemia), loss of consciousness, or in pregnant women with a COHb >20 percent or evidence of fetal distress.

Statistical analysis

Statistical presentation and analysis of the present study was conducted, using SPSS Version.16. Chi-square test was used for comparison between groups as regards qualitative data. The strength of the linear association between two variables was quantified by Spearman correlation coefficient test.

RESULTS

The total number of the studied patients was 57, their age ranged from 15-55 years and 35.1% of the patients were males whereas, 64.9% were females. The delay time ranged from 1.5-10 hours andthe duration of exposure from 0.5 to 5 hours. All cases were accidentally exposed to Co poisoning. In addition, no cases of deaths were recorded during the period of the study and all cases had normal temperature and pupil size.

Table (1) revealed that most of the patients complained from vomiting (87.7%), history of loss of consciousness (45.6%), and headache (22.8%). The most common cardiovascular manifestation was tachycardia; occurred in 50.9% of the patients then dyspnea that occurred in about one fourth (24.6%) of patients and lastly chest pain (12.3%) and hypotension (10.5%). The ECG findings showed that 48.2% of the studied patients had sinus tachycardia (**figure 1).** Additionally, ECG findings that indicated myocardial ischemia occurred in 14.4% [in the form of T-wave inversion (5.4%), ST depression (5.4%) and combination of T-wave inversion and ST depression (3.6%)] as shown in **figure 2**.

All patients who had ECG suggestive of cardiac ischemia had abnormal serum levels of CPK-MB and troponin I as well as wall motion abnormalities in echocardiography **(Figure 3).** Additionally, most patients who had tachycardia had abnormal serum level of CPK-MB and normal troponin I level. However, most patients who had normal ECG had normal serum levels of CPK-MB and troponin I as well as echocardiogram (**Table 2)**. **Table (3)** illustrated significant negative correlation between delay time and duration of exposure with Glasgow coma scale. While, significant positive correlation was noticed between both ECG and echocardiographic changes and delayed presentation.

Table 1: Distribution of clinical data among acute CO intoxicated patients (total number: 57 cases)

	Number	**Percentage%**
Headache		
Yes	13	22.8
No	44	77.2
Vomiting		
Yes	50	87.7
No	7	12.3
Dyspnea		
Yes	14	24.6
No	43	75.4
Chest pain		
Yes	7	12.3
No	50	87.7
Loss of consciousness		
Yes	26	45.6
No	31	54.4
Respiratory rate		
Normal	36	66.7
Tachypnea	19	33.3
Pulse		
Normal	28	49.1
Tachycardia	29	50.9
Blood pressure		
Normal	51	89.5
Hypotension	6	10.5
ECG		
Sinus tachycardia	27	48.2
T wave inversion	3	5.4
ST Depression	3	5.4
T wave inversion and ST Depression	2	3.6

Table 2: Chi-square association between ECG changes and serum levels of CPK-MB and troponin I and echocardiographic findings

		ECG changes					
		Normal (21)	**Sinus tachycardia** (28)	**ST depression** (3)	**T wave inversion** (3)	**ST depression & T inversion** (2)	**Total** (57)
Troponin I	Normal	16	26	0	0	0	42
	Abnormal	5	2	3	3	2	15
X^2	27.78						
P value	0.000*						
CPK-MB	Normal	18	3	1	0	0	22
	Abnormal	3	25	2	3	2	35
X^2	32.03						
P value	0.000*						
Echo-cardiographic changes	Absent	18	25	0	0	0	43
	Present	3	3	3	3	2	14
X^2	28.67						
P value	0.000*						

*X^2 chi-square test,*P value significant if <0.05.*

Table 3: Spearman correlation between delay time of hospital presentation and duration of exposure to CO and Glasgow coma scale, ABG, ECG changes, carboxy hemoglobin, creatine phosphokinase (CK-MB) and troponin I levels

	Minimum-maximum	**Delay time**		**Duration of exposure**	
		r	**P**	**r**	**P**
Glasgow coma scale	**9-15**	-0.54	0.00*	-0.39	**0.003***
pH	7.22-7.42	0.10	0.34	0.2	0.14
Pco2	32.4-58.7	-.104	0.44	-.16	0.23
Hco3	17.3-29	.03	0.48	0.176	0.19
Carboxy hemoglobin	7-45	0.14	0.29	0.14	0.3
Creatine phosphokinase (CK-MB)	17-110	-0.22	0.10	-0.14	0.31
Troponine I	**0**.12-4	0.03	0.28	0.15	0.26
ECGchanges		0.38	0.004*	0.48	0.72
Echo-cardiographic changes		0.29	0.03*	0.233	0.08

*r Spearman correlation,*P value significant if <0.05*

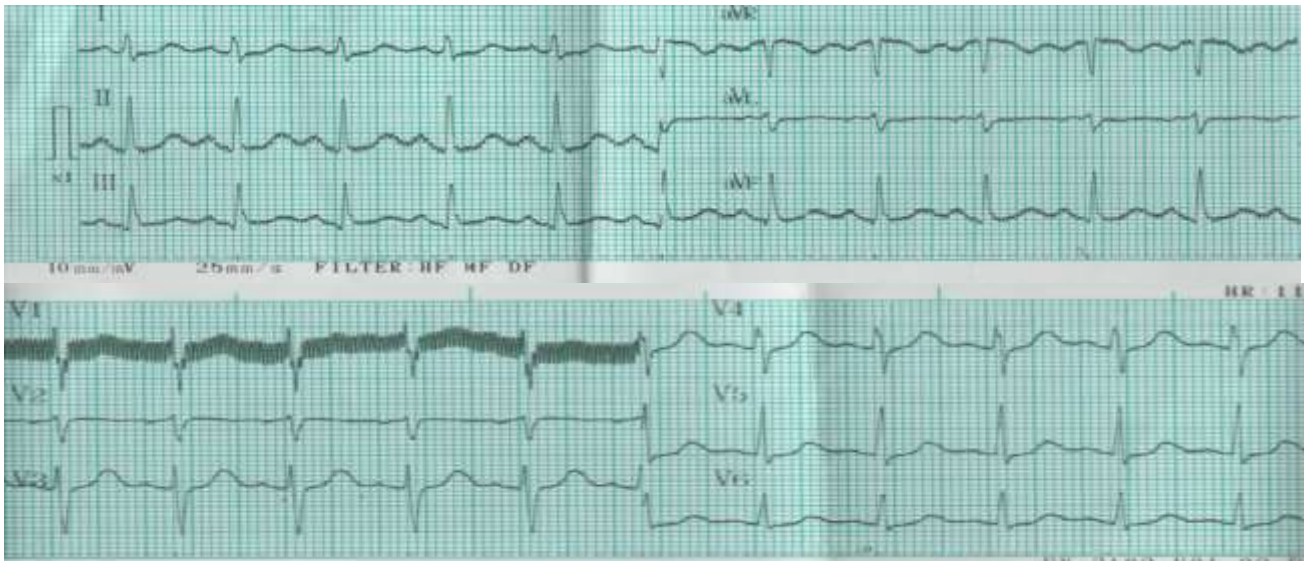

Figure 1: An ECG of a patient intoxicated by carbon monoxide showing sinus tachycardia (rat= 120 beat/ minute).

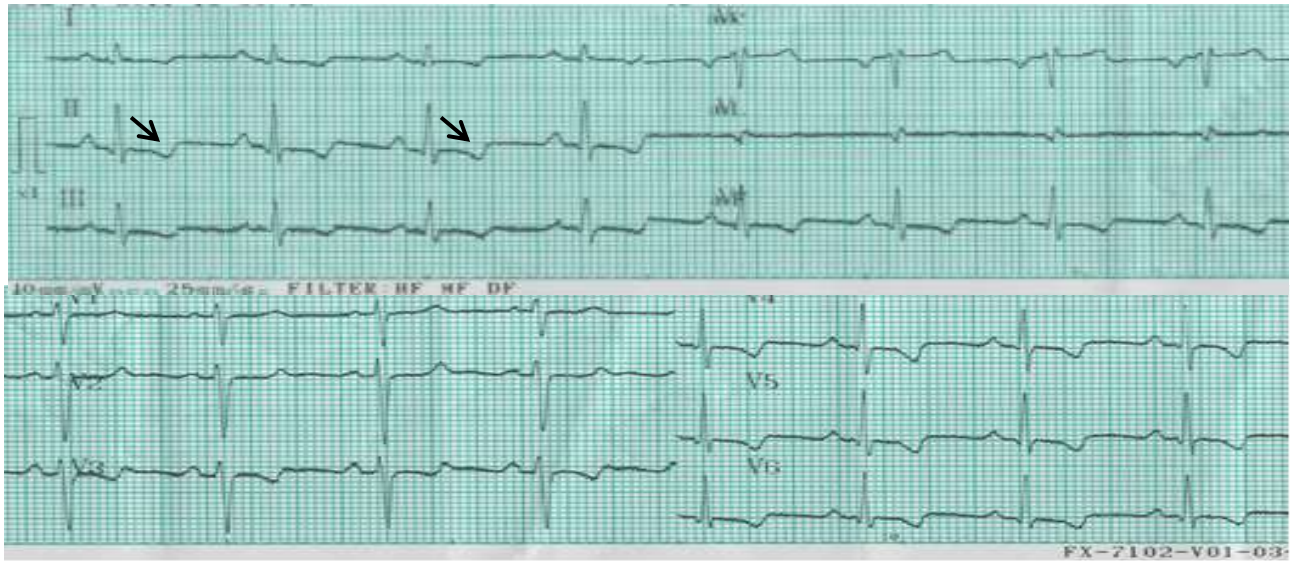

Figure 2: An ECG of a patient intoxicatedby carbon monoxide showing sinus rhythm (rate= 70 beat/ minute), scoping ST segment & T wave inversion.

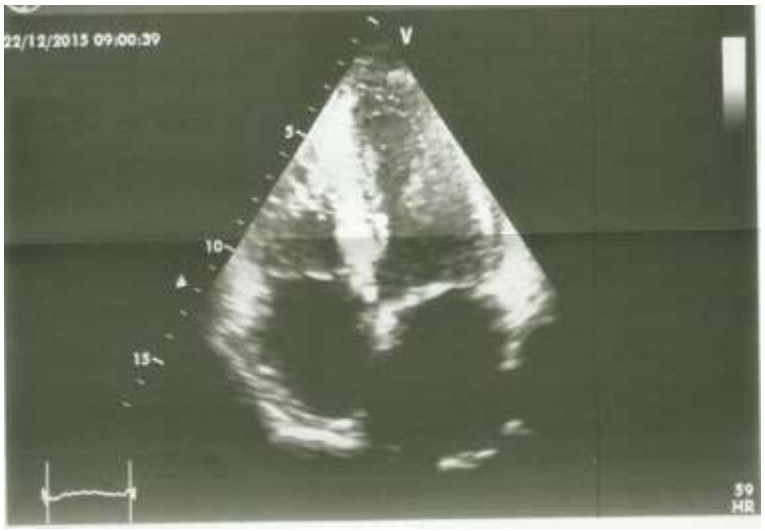

Figure 2: Echocardiographic image of apical four chambers view showing normal wall motion.

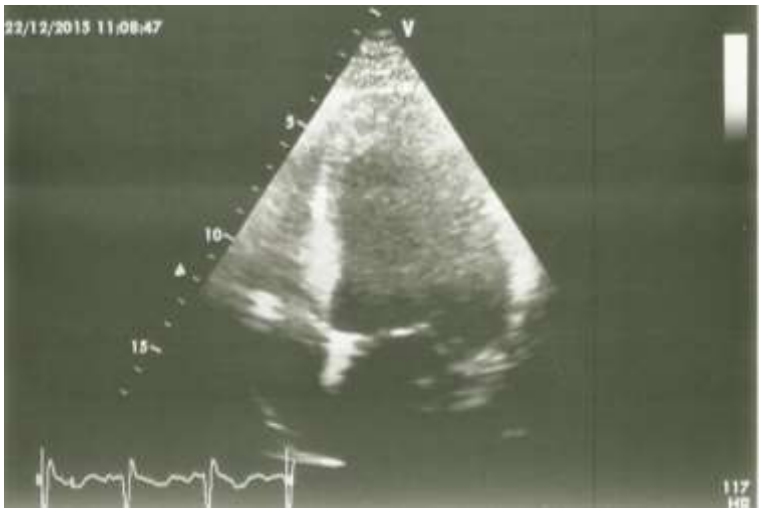

Figure 3: Echocardiographic image of apical four chambers view of a carbon monoxide poisoning patient showing mid and basal septal wall motion abnormalities.

Discussion

Cardiovascular disease is a global health problem causing nearly 1 in 3 deaths every year. Many of these deaths are due to coronary heart disease. Myocardial infarction (MI) is a major manifestation of coronary heart disease. In 1959, the World Health Organization defined MI as a combination of 2 of 3 characteristics: typical symptoms, enzyme rise, and a typical electrocardiographic (Patil, 2011).

In this study, the age of the studied patients ranged from 15-55 years with female predominance. All cases were accidentally exposed to acute Co poisoning with no recorded deaths. This is disagreeing with other studies in which the mean age was 47.2 years with male sex and accidental mode predominance (Satran et al., 2005; Henry et al., 2006).

Most of the patients in this study were complaining of vomiting (87.7%), history of loss of consciousness (45.6%), and headache (22.8%).These finding are in accordance with Harper and Croft-Baker (2004); Kao and Nañagas (2004) who revealed that the clinical findings of carbon monoxide poisoning are highly variable and largely nonspecific as the patients often present with constitutional symptoms as headache, malaise, nausea, dizziness, and may be misdiagnosed with acute viral syndromes.

The most common cardiovascular manifestation in the current study was tachycardia (occurred in about half of the patients (50.9%)) then dyspnea (occurred in about one fourth (24.6%) of patients and lastly chest pain (12.3%) and hypotension (10.5%). Similar results were obtained in a study done by Ismail et al. (2013). However, Hampson and Zmaeff (2001) stated that bradycardia was found among 10 cases out of 18 cases (55.5%) in a study to examine the outcome of group of patients with extreme CO poisoning in the United States. Tachycardia could be attributed to the compensatory response to systemic hypoxemia induced by CO and decreased blood pressure detected in the present study. Chest pain and low blood pressure may be symptoms of myocardial ischemia or decreased cardiac output caused by cellular hypoxia. Kourembanas (2002) reported that, CO

induced smooth muscle relaxation and vasodilatation of blood vessels as well as inhibition of platelet aggregation. This could be due to activation of guanyl cyclase enzyme and generation of cyclic GMP. Additionally, hypotension may result from myocardial injury secondary to hypoxia, ischemia, a direct myocardial depressant activity from myoglobin binding, peripheral vasodilation, or a combination of the aforementioned and may persist even after neurologic and metabolic symptoms have resolved as reported by Yanir et al. (2002).

In the current study, 45.6% of the patients experienced loss of consciousness and Glasgow Coma Scale Score ranged from 9-15 on arrival at the emergency toxicology center and significant negative correlation was found between the delay time and duration of exposure with Glasgow Coma Scale Score. This result is consistent with the result of a study done by Henry et al. (2006) who found that 81% of the patients experienced transient or persistent loss of consciousness and 79% had an abnormal Glasgow Coma Scale Score (<15) on arrival at the medical center. Additionally, Satran et al. (2005) revealed that loss of consciousness occurred in 81% and Glasgow Coma Scale Score was abnormal (≤14 range 3 to 15) in 46% of the patients upon arrival at Hennepin County Medical Center (HCMC). Raub et al.(2000)found that the severity of clinical condition is related to the duration of CO exposure and the prevailing clinical disposition of the patient.

Significant positive correlation was noticed between both ECG & echocardiographic changes and delayed presentation to the hospital in the present study. While, Salih et al. (2013) found that, the most important factors for myocardial injury or dysfunction in patients with CO poisoning are carboxyhemoglobin level and duration of CO exposure.

In the present study carboxyhemoglobin levels did not correlate with either the duration or time pass since exposure. This may be attributed to the fact that carboxyhemoglobin level depend on multiple factors; magnitude and duration of exposure, degree of alveolar ventilation, blood volume, and metabolic activity (Hampson &Hauff, 2008) .

The ECG findings of CO-poisoned patients in this study showed that 48.2% of the studied patients had sinus tachycardia. Additionally, ECG finding that indicate myocardial ischemia occurred in 14.4% [in the form of T-wave inversion ((5.4%), ST depression (5.4%) and combination of T-wave inversion and ST depression (3.6%)]. This is in partial agreement with Satran et al. (2005) who found that sinus tachycardia was present in 41% of patients and diagnostic ischemic changes were present in 30% of patients (26% with ST inversion- or T-wave changes and 4% with ST-segment elevation). Additionally, Khater (2006) revealed that 16.7% of acute CO intoxicated patient in PCC, Ain Shams University Hospitals had ECG changes in the form of ST-segment abnormalities, inverted T wave, premature ventricular contractions, right bundle branch block and QT interval prolongation. Furthermore, in another study, patient who had normal coronary artery developed ST segment elevation in D2, D3 and AVF after CO intoxication (Gonullu et al., 2011). On the other hand, Sorodoc et al. (2004); Chamberland et al. (2004) showed another ECG changes in co intoxicated patients; a non Q-wave anteroseptal acute myocardial infarction and right bundle branch block respectively. Moreover, Salih et al. (2013) showed myocardial ischemia associated with arrhythmias such as 1st degree AV block in a case report intoxicated by CO. An animal study reported that CO exposure at different intensities caused increase in PR and QT intervals in a dose-dependent manner (Rezaee et al., 2012).

The ECG changes in cases of acute CO poisoning can be attributed to the direct toxic effect of CO on the heart or due to CO-induced depression of both respiratory and central nervous systems causing cardiac affection (Blumenthal, 2001). Additionally, Yanir et al. (2002) reported that binding of CO to myocardial myoglobin and mitochondrial cytochrome chain enzymes might explain cardiac failure. The good response to inotropic agents and the findings of repeated echocardiographic studies support the probable diagnosis of myocardial stunning. The stunned myocytes, although unable to contribute to efficient ventricular contractility, still preserve metabolic viability suggesting that myocardial dysfunction can occur without ECG changes or elevation of enzyme levels typical of ischemia. These findings were supported with those of Aslan et al. (2005), they stated that it is not necessary to routinely measure CK, CK-MB and troponin, and perform myocardial perfusion SPECT in acute CO poisoning cases without any ECG abnormality, ischemic cardiac symptoms. In addition, Teksam et al. (2010) revealed that myocardial damage may exist with normal ECG and elevated cardiac biomarkers.

Cardiac injury occurs when there is disruption of normal cardiac myocyte membrane integrity. This results in loss of intracellular biologically active cytosolic and structural proteins constituents as biomarkers (such as troponin, creatine kinase, myoglobin, heart-type fatty acid binding protein, and lactate dehydrogenase) into the extracellular space including blood. When a sufficient number of myocytes have died (myocyte necrosis) or lost function, acute clinical disease is apparent. Ischemia, with or without infarction, consequent to an imbalance between the supply and demand of oxygen and nutrients is the most common cause of cardiac injury. Other causes include trauma, toxins, and viral infection (Jaffe et al., 2000).

Carbon monoxide poisoning can exacerbate angina and cause cardiac injury even in persons with normal coronary arteries (Satran et al., 2005). Therefore, the evaluation of cardiac markers can be of considerable diagnostic value in the presence of chest discomfort or ischemic electrocardiographic changes. Troponin I and troponin T have been successfully used in the diagnosis of CO-induced cardiotoxicity (Henry et al., 2006).

In the current study all patients who had ECG suggestive of cardiac ischemia had abnormal CPK-MB and troponin I with significant correlation between the level of CPK-MB and troponin I with ECG. Most patients who had normal ECG had normal CPK-MB and troponin I. This is in accordance with Satran et al. (2005) who reported that 44% of patients with biomarkers drawn had cardiac biomarkers diagnostic of myocardial ischemia. Furthermore, a study was performed by Szponar et al. (2012) on a group of 44 patients treated from carbon monoxide poisoning, the level of troponin I was increased in all cases with ECG changes, but also in 13 patients with normal ECG. Another prospective cohort study of 230 consecutive adult patients treated for moderate to severe CO poisoning with hyperbaric oxygen and admitted to the Hennepin County Medical Center showed an increased in both cardiac troponin I and creatine kinase-MB levels and/or diagnostic electrocardiogram changes in 85 (37%) of 230 patients (Henry et al., 2006). On contrary, carbon monoxide cardiotoxicity may be clinically occult and often remains undiagnosed because of the lack of overt symptoms and specific ischemic changes in the electrocardiogram. Routine myocardial necrosis markers have low diagnostic efficiency, particularly in patients with concomitant skeletal muscle necrosis or multiple organ failure complicating carbon monoxide poisoning (Gandini et al., 2001; Gonullu et al., 2011).

In clinical studies, echocardiography is sensitive than electrocardiography in detecting cardiac damage and more effective for severity assessment (Adams et al., 1994). In the current study, all patients who had ECG suggestive of cardiac ischemia after CO exposure had wall motion abnormalities in echocardiography. Satran et al. (2005) analyzed 230 patients with intentional CO exposure. Of them, 53 patients underwent echocardiographic examination and 57 percent of patients showed abnormal left ventricular function. They mentioned that the finding is non-specific, and attributed it to myocardial hypoxia.

The exact mechanism of CO induced myocardial damage is unknown; however, tissue hypoxia at the cellular level is often responsible because myocardial tissues are most sensitive to the hypoxic effects of CO. The affinity of hemoglobin for CO is 200 to 250 times greater than its affinity for oxygen. This results in competitive inhibition of oxygen release due to a shift in the oxygen-hemoglobin dissociation curve, reduced oxygen delivery, and subsequent tissue hypoxia (Weaver, 1999; Prockop and Chichkova, 2007). However, clinical findings have generally excluded hypoxia as a cause of arrhythmias because carboxyhemoglobin levels do not correlate with the observed ECG changes (Carnevaliet al., 1987; Gandini et al., 2001). Myocardial stunning as a result of CO poisoning or unmasking of underlying coronary arterial disease by creating a myocardial demand/supply mismatch may be the proposed mechanisms of myocardial damage. Furthermore, blood viscosity, and platelet dysfunction have been implicated as an important pathophysiological mechanisms in patients with acute myocardial infarction with normal coronary arteries. An increasing thrombotic tendency secondary to platelet stickiness and polycythemia has been reported in patients with CO poisoning (Kimet al., 2012). Carbon monoxide, after being released from hemoglobin, could also bind to myoglobin, forming carboxymyoglobin, thereby reducing the amount of oxygen transported by myoglobin, diminishing the amount of ATP synthesized, and decreasing the ability of muscle cells to contract, this may be an additional mechanism of the cardiotoxicity (Wattel et al., 2006). Also, impaired cellular respiration provokes a stress response, including the activation of hypoxia-inducible factor 1α,15 by gene regulation resulting in neurologic and cardiac protection or injury, depending on the dose of carbon monoxide (Weaver, 2009).

Conclusion

The present study revealed that sinus tachycardia is the most common cardiac manifestation of CO poisoning and ECG finding that suggest myocardial ischemia occurred in only 14.4% of the patients. All patients who had ECG suggestive of cardiac ischemia had abnormal CPK-MB and troponin I. However, most patients who had normal ECG had normal CPK-MB, troponin I and echocardiogram. Therefore, a baseline ECG should be performed but, it is not necessary to routinely measure troponin I and do echocardiogram and it can be restricted to only cases with abnormal ECG findings.

Limitations & recommendations

This study limited by the small sample size and absence of control group. Further work should include determination of hematocrit, blood viscosity, and platelet function which implicated as an important pathophysiological mechanism in patients with acute myocardial infarction and the role of different antioxidants in cardiotoxicity induced by acute Co poisoning. Follow up of patients parameters after treatment is also recommended after 24 and then after 48 hours.

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الملخص العربى

دور رسم القلب الكهربي والعلامات البيولوجيه للقلب و رسم صدى القلب في تشخيص إصابات عضلة القلب الناتج عن التسمم الحاد بأول أكسيد الكربون

مرفت عريبى وايناس ابراهيم المداح[١]

مقدمه البحث: يعد التسمم بغاز أول أكسيد الكربون (CO) من اكثر الاسباب العرضيه للامراض والوفيات الناتجة عن التسمم في جميع أنحاء العالم.

الهدف من هذه الدراسة: هو تقييم مظاهر التسمم الحاد بأول أكسيد الكربون علي القلب والأوعية الدمويه وقيمة التغييرات التي تحدث في رسم القلب الكهربائى و العلامات البيولوجية للقلب (الكرياتين فوسفو كينيز، والتروبونين اي) ورسم صدى القلب في تشخيص أضرار القلب في حالات التسمم الحاد بأول أكسيد الكربون. بالإضافة إلى ذلك، تقييم أثر تأخر وقت المجئ للمستشفى ومدة التعرض علي هذه المعايير.

الاشخاص وطرق البحث: هذه دراسة مستعرضة أجريت على المرضى الذين يتم إدخالهم بسبب التسمم الحاد بأول أكسيد الكربون إلى وحدة علاج التسمم ، مستشفى الطوارئ بجامعة طنطا في الفترة الزمنيه من أول مارس ٢٠١٤ إلى آخر سبتمبر ٢٠١٥. جميع المرضى تعرضوا لأخذ التاريخ المرضي مع اجراء الفحص السريري. بالإضافة إلى ذلك، تم إجراء رسم القلب الكهربي، رسم صدى القلب، العلامات البيولوجية للقلب (الكرياتين فوسفو كينيز م ب، و التروبونين أى)، ومستويات الكربوكسي هيموغلوبين لجميع المرضى.

نتائج البحث: تراوحت أعمار المرضى من ١٥-٥٥ عاما، و تأخر وقت المجئ للمستشفى من ١,٥ حتي ١٠ ساعات و مدة التعرض لاول اكسيد الكربون من ٠,٥-٥ ساعات . وكانت سرعة دقات القلب من أكثر أعراض القلب والأوعية الدموية شيوعا وقد حدثت في ٥٠,٩ ٪ من المرضى ثم يليها صعوبة في التنفس (٢٤,٦ ٪) واخيرا ألم في الصدر (١٢,٣ ٪)، و انخفاض ضغط الدم (١٠,٥ ٪). أظهرت النتائج تغيرات رسم القلب الكهربي في 48.2 ٪ من المرضى الخاضعين للدراسة وقد عانوا من سرعة فى دقات القلب، ٥,٤ ٪ من قلب موجة-T ، ٥,٤ ٪ من انخفاض ST، و حدوث كلاهما معا في ٣,٦ ٪. جميع المرضى الذين كان لديهم رسم القلب الكهربي الذي يشير الي اسكيمية عضلة القلب كان لديهم زياده غير طبيعيه ذو دلالة احصائية في الكرياتين فوسفو كينيز م ب ، التروبونين اي و تغييرات في حركةجدار القلب في رسم صدى القلب. بالإضافة إلى ذلك، كان هناك ارتباط سلبى ذو دلالة واضح بين تأخر وقت المجئ للمستشفى ومدة التعرض مع مؤشر غلاسكو للغيبوبة و ارتباط ايجابى ذو دلالة احصائية بين تغيرات رسم القلب الكهربي و رسم صدى القلب مع تأخر وقت المجئ للمستشفى.

الخلاصة: وتخلص هذه الدراسة أن سرعة فى دقات القلب هو مظهر من مظاهر القلب الأكثر شيوعا للتسمم بغاز أول أكسيد الكربون . تغيرات رسم القلب الكهربي الذى يشير الى اسكيمية عضلة القلب حدثت فقط فى ١٤,٤% من المرضى . ولذلك، ينبغي إجراء رسم القلب الكهربائى في بادىء الامر وبشكل روتينى، ولكن ليس من الضروري قياس روتيني للتروبونين ورسم صدى القلب و لكن يمكن أن يقتصر فقط على حالات بها رسم القلب الكهربي الغير طبيعية.

١ قسم الطب الشرعي و السموم الاكلينيكية، كلية الطب، جامعة طنطا.