SERUM ACUTE PHASE PROTEINS AS NOVEL MARKERS OF MYOCARDIAL INJURY IN ACUTE CARBON MONOXIDE POISONED PATIENTS

BY

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ABSTRACT

Acute carbon monoxide (CO) poisoning is considered one of the most common types of poisoning. CO stimulates inflammatory processes that may activate acute phase response. Additionally, myocardial injury is considered a serious complication of this type of poisoning. Hence, the aim of this study was to assess some acute phase proteins in acute CO poisoned patients and their possible role as novel markers of carbon monoxide induced myocardial injury. Fifty patients with acute carbon monoxide poisoning admitted to Poison Control Unit, Tanta University from the first of May 2013 to the end of April 2015 were studied. Each patient was subjected to full history taking, clinical examination and the following investigations; arterial blood gases, serum troponin I (TnI), acute phase proteins (high sensitive serum C- reactive protein (hs-CRP), total leucocytic count (TLC), serum albumin) and carboxyhemoglobin (COHb) concentration were measured. Apparently healthy fifty individuals matched for age and sex of the studied cases represented the control group. The mean age of the studied group was 34.7 ± 10.3y with nearly equal sex distribution (24 female: 26 male). Palpitation was the most common symptom (84%), hypotension and tachycardia were recorded in 76% and 70% respectively. ECG was normal in 30%, sinus tachycardia was observed in 60%, while ischemic changes were detected in 24% of the studied patients. In addition, serum CRP and TLC were higher while serum albumin was lower in the studied patients than in control subjects. Moreover, acute phase proteins were affected more significantly and correlated well with myocardial injury. So, the determination of CRP level, TLC or serum albumin on admission could be readily available and effective tools in evaluating acute CO poisoning and detecting the presence of myocardial injury.

Keywords: *Carbon monoxide poisoning, serum acute phase proteins, myocardial injury, troponin I.*

INTRODUCTlON

Carbon monoxide (CO) is a colorless, odorless, tasteless and non-irritant gas (Stephen et al., 2012). CO poisoning is one of the most common types of poisoning, and it is the most frequent cause of death from poisoning worldwide (Iqbal et al., 2012). CO is a product of combustion of organic matter with insufficient oxygen supply, and is often produced in domestic or industrial settings by various materials. Sources of poisoning include but not limited to indoor burning of charcoal, heaters, gasoline-powered generators and vehicles exhaust (Davutoglu, 2009).

Carbon monoxide is one of known chemical asphyxiants which cause tissue hypoxia with prominent neurologic and cardiovascular injury (Jang and Park, 2010). Tissue hypoxia in CO poisoning is attributed to greater affinity of hemoglobin for CO, 200 to 250 times, than its affinity for oxygen. This results in competitive inhibition of oxygen release due to a shift in the oxygen-hemoglobin dissociation curve to the left with subsequent reduced oxygen delivery to the tissues (Lee et al., 2011).

Direct toxic effects of CO at cellular level have been established. Carbon monoxide breaks the chain of the mitochondrial cytochrome oxidase and prevents the formation of adenosine triphosphate (Cooper and Brown, 2008). In addition, it causes free oxygen radicals formation and lipid peroxidation in brain and other tissues including the heart muscle with subsequent functional and morphological alterations (Cevik et al., 2010). Carbon monoxide also binds to myoglobin, aggravating the hypoxia in the cardiac muscle (Turedi et al., 2011).

It has been suggested that CO exposure stimulates inflammatory processes that may lead to stimulation of acute phase response. The most important component of this response comprises the acute phase proteins; which are a heterogeneous group of plasma proteins. The most commonly used one in clinical practice is C- reactive protein (Kilicarslan et al., 2013).

Cardiovascular disorders are accompanied by the elevation of several positive acute phase reactants such as C–reactive protein, fibrinogen, and white blood cell count. They are also accompanied by the reduction of negative acute phase reactants such as albumin, transferrin, and transthyretin (Ahmed et al., 2012).

The aim of this study was to assess some acute phase proteins in acute CO poisoned patients and their possible role as novel markers of CO induced myocardial injury.

PATIENTS AND METHODS

This analytical cross sectional study included fifty patients with acute CO poisoning admitted to Poison Control unit, Tanta University, Egypt in the period from the first of May 2013 to the end of April 2015. Patients aged 18 years or more with a verified history of recent exposure to CO and a carboxyhemoglobin (COHb) more than 5% on admission were consecutively included in this study. Exclusion criteria were history of coronary artery disease or other known heart disease, diabetes, hypertension, liver or kidney diseases, hematological disease or cancer, acquired immunodeficiency syndrome (AIDS), acute illness or infection, chronic inflammatory conditions or chronic infections. Smokers as well were excluded.

Apparently healthy fifty individuals, 25 males and 25 females, matched for age and sex were the control group.

A written informed consent was taken from patients who were conscious, the relatives of unconscious patients, and control subjects. Confidentiality of the results of investigation was respected by giving a code number for everyone.

On admission, each patient was subjected to full history taking. It included; age, sex, habits (such as smoking), circumstances of toxicity and duration of exposure, time elapsed between exposure and admission to the hospital, and underlying diseases. Complete clinical examination of patients was done including vital data (heart rate, respiratory rate, blood pressure, and temperature), level of consciousness (Glasgow Coma Scale), cardiovascular system, respiratory system, and abdominal examination. Twelve leads ECG was done to all cases on admission. Electrocardiographic analysis included the heart rate, rhythm, ST/T abnormalities, conduction defects and measurement of PR and QT intervals. The QT interval was corrected (QTc) according to the formula of Bazett (Bazett, 1920). QTc was considered prolonged when it was longer than 0.440 second (Crotti et al., 2008). Electrocardiographic records were conducted on a Cardimax ECG machine (FUKUDA DENSHI, FCP-7101, Japan), at 10 mm/mV and 25 mm/s paper speed.

After detailed clinical examination, blood samples were withdrawn from patients before giving them any medications under complete aseptic techniques with single use syringes to perform the following investigations; arterial blood gases (ABG), serum troponin I (TnI), high sensitive serum C reactive protein concentration (hs- CRP), total leucocytic count (TLC), and serum albumin. Carboxyhemoglobin measurements were performed

using pulse CO-oximeter (Masimo SET rainbow, California, USA). Carboxyhemoglobin levels of 0-5% were accepted as normal values in nonsmoker patients.

Troponin I was measured by a fluorescence immunoassay using ichroma ^{TM} Tn-I ELISA provided by Boditech Med Inc. Test levels more than 0.3 ng/ml were accepted as indicating myocardial damage.

According to reference ranges, TLC of more than 11.000/mm^3 and a serum CRP concentration of 6mg/L or more were considered as elevated. In addition, serum albumin less than 3.5g/L was considered low.

Patients received standard emergency and supportive measures including hundred percentages oxygen, and when indicated they were referred to receive hyperbaric oxygen sessions. Because of this, most of the patients had short stay in the Poisoning Control Unit so; the studied acute phase proteins could not be measured on serial times. It is to be noted that, patients who had positive cardiac troponin levels were referred for cardiology consultation for further serial biomonitoring, investigations and suitable interventions.

Statistical Analysis :

Continuous variables were expressed as means and standard deviations and categorical variables as numbers with percentages in brackets. For comparisons between the different groups, Student's t test and Chi-square test were used for quantitative and categorical variables respectively. Associations were assessed using Pearson's correlation coefficient. All analyses were performed using SPSS version 20 for Windows (SPSS Inc., Chicago, Illinois, USA). P values of 0.05 or lower were considered to be statistically significant.

RESULTS

According to inclusion criteria, fifty patients were recruited. Their age ranged from 19 to 55 years with a mean of 34.7 ± 10.3 years, with nearly equal sex distribution (24 female: 26 male). Exposure to carbon monoxide was accidental in all patients with a mean duration of 2.96 ± 0.95 h. The mean delay time (time between exposure and hospitalization) was 3.30 ± 1.25 h.

Clinical and ECG characteristics of the studied patients are shown in table (1). Examination of vital data revealed that hypotension and tachycardia were observed in 76% and 70% of the studied patients respectively. As regards the clinical complaint of the patients, palpitation was the most common symptom (84%), while only 36% of the studied patients complained of

chest pain. Electrocardiography was normal in 30 % of the studied patients, whereas the rest of the patients showed different types of arrhythmias and/or ischemic changes. Most of the studied patients presented with sinus tachycardia (60%), while only 2% presented with atrial fibrillation. As regards ischemic changes, depressed ST segment was observed in 14% of the patients, while inverted T wave was present in 10% of all the studied cases (Figures 1, 2 and 3).

Table (1) : Clinical and ECG characteristics of the studied patients (n=50).

Characteristic parameters	**n**	**Percentage**
Vital signs:		
Blood pressure (mmHg):		
Hypotension	38	76
Normal	12	24
Pulse (beat/min):		
Tachycardia	35	70
Normal	18	36
Respiratory rate (cycle/min):		
Tachypnea	21	42
Normal	29	58
Gastrointestinal manifestations:		
Vomiting	30	60
Colic	20	40
Headache	25	50
Impaired consciousness	20	40
Dyspnea	22	44
Palpitation	42	84
Chest pain	18	36
ECG changes:		
Normal	15	30
Arrhythmias		
Sinus tachycardia	30	60
Atrial fibrillation	1	2
Prolonged QTc	2	4
Premature ventricular contractions	4	8
Ischemic changes		
Depressed ST segment	7	14
Inverted T wave	5	10

n: number, QTc: corrected QT interval, ECG: Electrocardiogram.

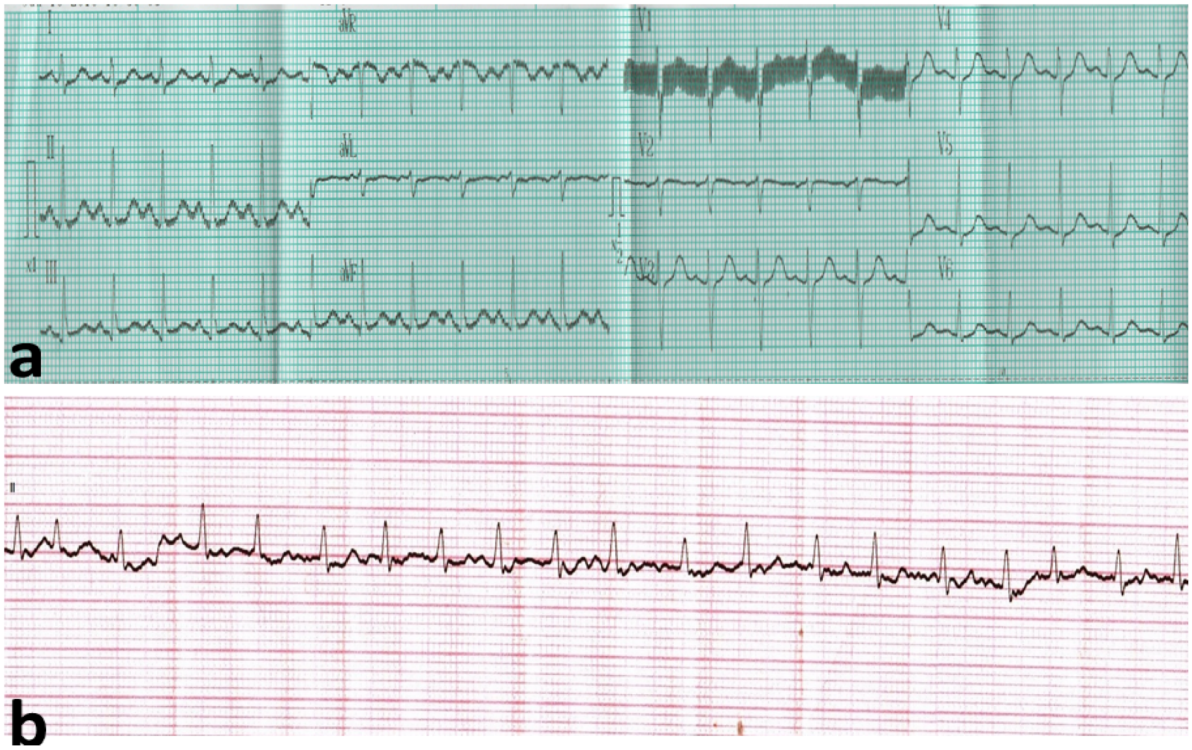

Fig. (1) : Examples of CO- induced ECG changes: a: Sinus tachycardia, b: Atrial fibrillation.

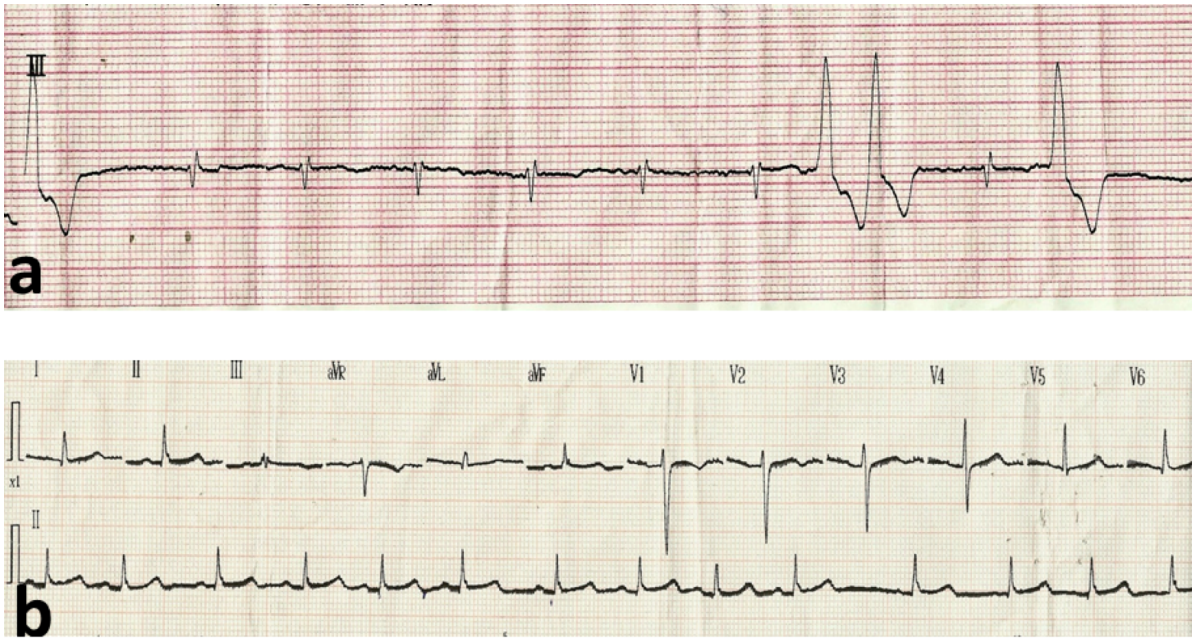

Fig. (2) : Examples of CO- induced ECG changes: a: Premature ventricular contractions, b: prolonged QTc (corrected QT) interval.

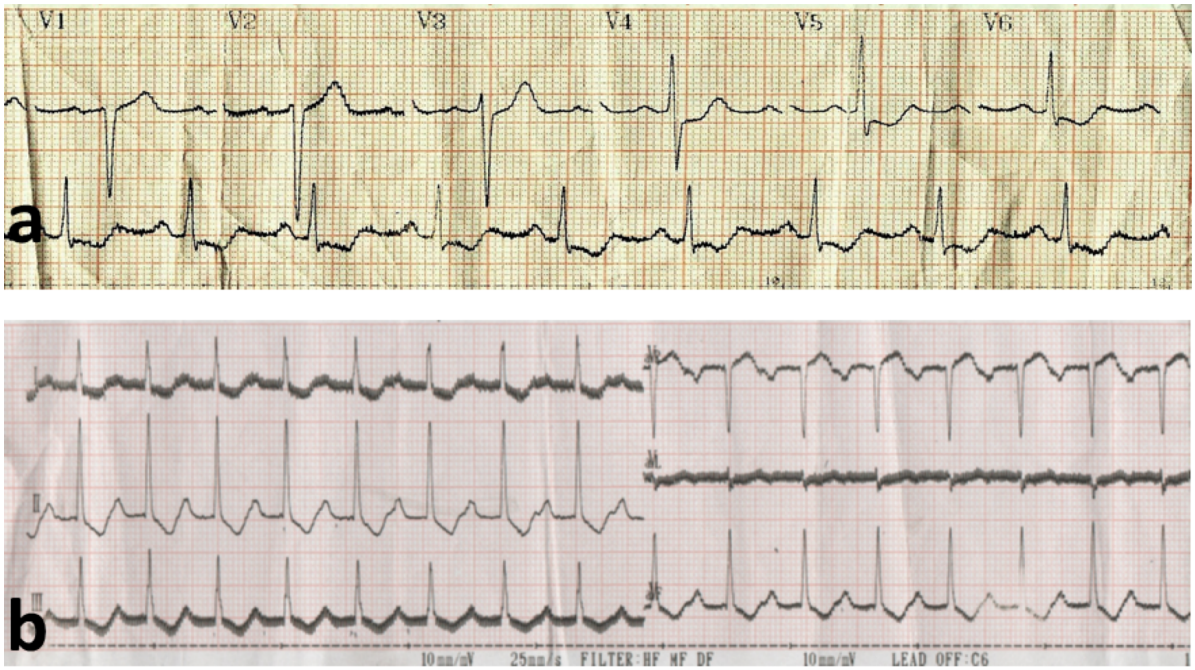

Fig. (1) : Examples of CO- induced ECG changes: a: Depressed ST segment, b: T wave inversion.

Table (2) shows basic laboratory characteristics of the studied patients. Mean carboxyhemoglobin level was elevated above the normal detected value (25.74±12.9 %). Metabolic acidosis was diagnosed according to ABG findings (pH =7.31 ± 0.05, HCO_3= 17.37 ± 2.0 mmol/l, $PaCO_2$ = 32.62 ± 4.9 mmHg). Myocardial injury was diagnosed by elevation of serum TnI (0.71 ±. 0.61 ng/ml). As regards acute phase reactants studied in this work, it was found that serum hs- CRP, and total leucocytic count were elevated (17.85 ± 11.2 mg/L and 15632 ± 3886 x 10^9/L respectivelly), while serum albumin was decreased (3.36 ± 0.48 g/L).

Comparison between study and control groups is demonstrated in table (3). There were significant high levels of COHb and TnI level in the study group compared to the control group. As regards the acute

Table (2): Basic laboratory characteristics of the studied patients (n=-50).

Laboratory characteristics	Mean ± SD
Blood pH	7.31 ± 0.05
Serum HCO_3 (mmol/l)	17.37 ± 2.0
P_aCO_2(mmHg)	32.62 ± 4.9
COHb level (%)	25.74 ± 12.9
Serum TnI level (ng/ml)	0.71 ± 0.61
Serum hs- CRP (mg/L)	17.85 ± 11.2
Total leucocytic count (x10^9/L)	15632 ± 3886
Serum albumin (g/L)	3.36 ± 0.48

n: number, SD: standard deviation, COHb: Carboxyhemoglobin, TnI: troponin I, hs-CRP: high sensitive serum C reactive protein.

phase reactants, serum CRP and TLC were significantly higher, while serum albumin was significantly lower in the studied cases as compared with the control subjects. ECG changes were observed in 70% of the studied patients while they were observed in 12% of the control group only in the form of sinus tachycardia.

According to the level of serum cardiac TnI, the studied patients were divided into myocardial injury group (20%) and non-myocardial group (80%). Myocardial injury was diagnosed by elevation of TnI level. It was found that COHb level, CRP concentration and total leucocytic count were significantly higher while serum albumin was significantly lower in myocardial injury group compared to non-myocardial injury one (Table 4).

Carboxyhemoglobin level showed significant positive correlation with TnI, CRP and total leucocytic count, whereas it showed significant negative correlation with serum albumin level. Moreover, serum TnI had significant positive correlation with serum CRP and total leucocytic count while it showed significant negative correlation with serum albumin (Table 5).

Table (3): Comparison between study and control groups as regards demographic data, vital signs, ECG findings, COHb level, and the studied acute phase proteins (n =100)

		Study group (n=50)	Control group (n=50)	P value
Age	mean ± SD	34.7 ± 10.3	33.7± 10.4	0.08
Sex	(m/f)	26/24	25/25	0.06
Heart rate (beats/min)	mean ± SD	94.1 ± 18.5	77.3 ± 12.4	< 0.001*
SBP (mm Hg)	mean ± SD	90.3 ± 10.8	115.8 ± 12.3	0.05*
DBP(mm Hg)	mean ± SD	55.7 ± 10.2	70.4 ± 9.8	0.03*
Respiratory rate (cycle/ min)	mean ± SD	28.5± 10.9	16.3 ± 4.4	0.001*
ECG				
Normal Abnormal	n (%) n (%)	15 (30%) 35 (70%)	44 (88 %) 6 (12 %)	< 0.001*
COHb level (%)	mean ± SD	22.74 ± 12.9	1± 0.19	< 0.001*
Serum TnI level (ng/ml)	mean ± SD	0.71 ± .61	0.15 ± 0.05	< 0.001*
Serum hs CRP (mg/L)	mean ± SD	17.85 ± 11.2	3.5 ± 1.11	< 0.001*
Total leucocytic count ($x10^9$/L)	mean ± SD	15632 ± 3886	7535.8 ± 1358.96	< 0.001*
Serum albumin (g/L)	mean ± SD	3.36 ± .48	4.05 ±.64	0.001*

n: number, f: female, m: male, SD: standard deviation, SBP: systolic blood pressure, DBP: diastolic blood pressure, ECG: Electrocardiogram, COHb: carboxyhemoglobin, TnI: troponin I, hs-CRP: high sensitive serum C reactive protein, *: significant at p <0.05.

Table (4) : Comparison of myocardial and non-myocardial injury groups as regards COHb level and the studied acute phase proteins (studied group n=50).

	Myocardial injury group (n= 10) Mean ± SD	**Non myocardial injury group (n=40) Mean ± SD**	**P value**
COHb level (%)	32.04 ±10.27	14.81± 9.19	< 0.001*
Serum hs- CRP (mg/L)	25.45 ±8.42	11.37 ± 9.05	0.001*
Total leucocytic count (x 10^9 / L)	18278.26 ±2698.4	1277.77 ± 3291.63	0.02*
Serum albumin (g/L)	3.01 ±.28	3.65 ±.42	0.001*

n: number, SD: standard deviation, COHb: carboxyhemoglobin, hs-CRP: high sensitive serum C reactive protein. *: significant at p <0.05.

Table (5) : Pearson's correlation between serum troponin I, COHb level and the studied acute phase proteins (studied group n=50).

	COHb level		**Serum TnI level**	
	r	**p value**	**r**	**p value**
Serum hs- CRP (mg/L)	0.926	< .001*	0.722	< .001*
Total leucocytic count (x 10^9 / L)	0.914	< .001*	0.960	< .001*
Serum albumin (gm/L)	- 0.8250	< .001*	-0.649	< .001*
Serum TnI level (ng/ml)	0.762	< .001*	Not applicable	

n: number, r: correlation coefficient, hs-CRP: high sensitive serum C reactive protein, TnI: troponin I, *: significant at p <0.05.

DISCUSSION

The current study demonstrates that serum CRP and total leucocytic count were higher, while serum albumin was lower in patients with acute CO poisoning than in control subjects. Moreover, these acute phase proteins changes were more significantly detected and correlated well with myocardial injury.

The study participants showed nearly equal sex distribution (24 female: 26 male). In contrast, other studies showed that males were more represented in CO poisoning (Homer et al., 2005; Henry et al., 2006; Ismail et al., 2013). The mean age of the studied patients was 34.7 ± 10.3 y. In this regard, Ismail et al. (2013) reported a mean age of 26.5 ± 8.3 y, but Satran et al. (2005) and Grieb et al. (2010) reported a higher mean age of their studied patients (47.2 and 42 years respectively).

In the present study, the heart rate, blood pressure and respiratory rate were affected and this was proven in previous studies (Aslan et al., 2006; Cevik et al., 2010). Carbon monoxide intoxication resulted in cardiovascular manifestations e.g. palpitation (84%), dyspnea (44%), chest pain (36%) and ECG abnormalities (70%), with the palpitation was the most common complaint. Palpitation is due to tachycardia which is usually considered as a compensatory response to systemic hypoxemia and decreased cardiac systolic function. Chest discomfort or pain can result from myocardial ischemia or necrosis. Shortness of breath and low blood pressure can be symptoms of cardiac dysfunction (Jang and Park, 2010).

The current work demonstrated arrhythmic and/ or ischemic changes in ECG with the following percentages: sinus tachycardia (60%), atrial fibrillation (2%), prolonged QTc interval (4%), premature ventricular contractions (8%), depressed ST segment (14%) and inverted T wave (10%). Satran et al. (2005) reported ECG abnormalities in 84 % of the studied patients with a nearly similar incidence of ischemic changes (26%). Moreover, Hajsadeghi et al. (2011) documented ECG abnormalities in CO poisoned patients with sinus tachycardia represented 55.6% , whereas ST segment depression and T wave inversion were 20.6% and 12 % respectively. The illustrated prolonged QTc interval in this study is supported by Sener et al. (2014) who described that all wave durations including QTc interval were significantly prolonged in ECG in adult patients with acute CO poisoning.

ECG changes in cases of acute CO poisoning can be attributed to the primary toxic effect of CO on the heart or due to CO-induced depression of respiratory and central nervous systems causing cardiac affection (Ismail et al., 2013). Dallas et al. (2012) identified the molecular basis for the proarrhythmic effects of acute CO exposure and attributed them to the striking increase in the late Na current arising from nitric oxide formation. Additionally, Akilli et al. (2013). reported that myocardial repolarization heterogeneity might play an important role in the formation of arrhythmias.

Cardiac troponins are markers of myocardial injury, not just myocardial infarction (Hickman et al., 2010). The current study showed that cardiac troponin I was significantly higher in CO poisoned patients compared to control group with significant positive correlation between it and COHb level. Similarly, Satran et al. (2005), Kao et al. (2009) and Akilli et al. (2013) documented myocardial injury accompanied by release of troponin in acute CO poisoning cases. The incidence of myocardial injury in this study was 20%. In contrast, Satran et al. (2005) and Kao et

al. (2009) reported higher incidence (37% and 59.2%) respectively. This difference is explained by the characters of their study population, where they included only moderate to severe cases of CO poisoning. Likewise, Akilli et al. (2013) reported lower incidence of myocardial injury (only 14 patients) in their study that included 94 patients with CO poisoning.

The cardiotoxic effect of CO and specifically myocardial injury might be caused by its ischemic changes in the heart and / or its direct toxic effect on the myocardium (Lippi et al., 2012). Ischemic injury is caused by carboxyhemoglobin that shifts the oxygen-hemoglobin dissociation curve toward the left and leads to impairment of tissue oxygen delivery and makes cellular hypoxia (Weaver, 2009). Moreover, Gawlikowski et al. (2013) recently reported increased thrombin generation and impaired fibrinolysis in patients with acute CO poisoning, which might enhance ischemic complications. The direct toxic effect of CO on the myocardium was attributed to several different mechanisms including cytochrome C oxidase inhibition, an increase in free radical formation, and cellular apoptosis due to nitric oxide (Hajsadeghi et al., 2011; Ismail et al., 2013).

According to Aslan et al. (2006), some patients exposed to CO were found to have no cardiovascular symptoms or signs with silent myocardial injury and so they concluded that cardiotoxicity may be overlooked. Gandini et al. (2001) stated other barriers to identify CO-induced myocardial damage e.g. CO poisoning may not be correctly diagnosed, the pathophysiological and clinical characteristics of CO induced myocardial injury are not well understood, commonly used markers of myocardial damage have a low diagnostic value when skeletal muscle damage is also present, and changes in the ECG are often nonspecific.

Indeed early detection of myocardial injury with subsequent early intervention is very important and might improve the patient outcome. This is supported by Henry et al. (2006) and Kao et al. (2009) who reported the prognostic value of myocardial injury for both short-term and long-term mortality in patients with moderate to severe CO poisoning. So, this study had focused on early detection of myocardial injury in CO poisoned patients by using readily available, valid and relatively cheap biomarkers.

In this study, serum CRP–as one of the most common acute phase proteins-was significantly higher in CO intoxicated patients compared to control group. Moreover, the increased level of CRP was significantly higher in patients with myocardial injury with significant positive correlation with COHb and cardiac troponin I. Considering that all inflammatory processes

or damaging factors affecting human organism increase the levels of pro-inflammatory proteins in the serum (Sawiniec et al., 2004), CO exposure might stimulate CRP production from the liver. The inflammatory response in acute CO intoxicated patients was documented by Thom et al. (2010). This may either result from its hypoxic or non-hypoxic mechanisms of action (Satran et al., 2005; Cevik et al., 2010). In accordance with this finding, Sawiniec et al. (2004) evaluated the level of the CRP as a diagnostic and prognostic marker in acute poisonings. They reported CRP elevation in CO intoxicated patients. Likewise, Grieb et al. (2010) revealed an elevation of CRP in CO intoxicated patients and it was correlated with the severity and outcome of their patients.

The significant rise of CRP in patients with myocardial injury in this study is supported by Suleiman et al. (2006) who reported that CRP as an inflammatory biomarker rises acutely after tissue injury and necrosis including myocardial infarction where, intense cytokine production and inflammatory cell infiltration occur in the area of ischemia and necrosis. Li and Fang (2004) also demonstrated that CRP was not only an inflammatory marker of cardiovascular diseases, but it may have direct proinflammatory effects and plays a pivotal role in their pathogenesis. The role of CRP as a strong independent predictor for cardiovascular risk and events was demonstrated by many authors (Clearfield, 2005; Mora et al., 2009).

The study of total leucocytic count as another acute phase reactant in this study revealed significant rise in CO intoxicated patients in comparison with control group. This rise was higher in myocardial injury group with significant positive correlation with COHb and cardiac troponin I. in accordance with this, Grieb et al. (2010) revealed an elevation in the number of leucocytes in CO poisoned patients with significant correlation with the severity of intoxication. Furthermore, Thom et al. (2006) stated that acute CO poisoning causes intravascular neutrophil activation with platelet–neutrophil interactions inducing a cascade of effects resulting in oxidative injury. Leukocytosis results from inflammatory response of neurohormonal system. White blood cells are major mediators of inflammation; the infiltration of them into necrotic tissue in response to ischemia has a major role in secreting mediators which contribute to oxidative and proteolytic injury (Salehi et al., 2013).

Serum albumin was also studied as one of the negative acute phase reactants. The present study revealed significant decrease in serum albumin in acute CO poisoned patients compared to the control group. This finding is supported by Liao et al. (2005) who reported low serum albumin concentration as a result of environ-

mental CO exposures. Barle et al. (2006) stated that the reduction in plasma albumin concentration is a characteristic feature of acute inflammatory states. A previous study has indicated that the decrease in plasma albumin concentration observed during acute inflammatory response is strongly related to an increase in albumin efflux, i.e. redistribution of albumin to the extravascular space, as well as to changes in the size of the intravascular fluid space (Smeets et al., 1994). In the present study serum albumin was significantly lower in patients with myocardial injury and was inversely associated with both troponin I and COHb. This goes in agreement with Djoussé et al. (2002) who concluded that low serum albumin appears to be a risk factor of myocardial infarction. Whether the detected low albumin has a direct causal role for myocardial injury or it is only an indicator of an underlying inflammatory condition is not yet clear.

CONCLUSION

Results support a view that Co exposure causes acute inflammatory events in humans. Furthermore, based on the results from the present study, the determination of C-reactive protein levels, total leucocytic count or serum albumin on admission could be considered inexpensive and readily available tools help in evaluating acute CO poisoning cases and detecting the presence of myocardial injury. Consequently, their usefulness is applied in day to day clinical practice with suspected impact on patient's outcome.

LIMITATIONS

Study has some limitations. (1) The small sample size may be regarded as a limitation. (2) Since we did not follow up our study groups, we cannot provide any information on mortality and morbidity. Based on the results from the present study, it is not possible to discern exactly how the acute phase response develops over time, especially not in the early phase after CO exposure, or how the degree and type of CO poisoning affect this development. These issues certainly warrant further study.

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بروتينات المرحلة الحادة فى المصل كدلالات جديدة لحدوث إصابة بعضلة القلب فى مرضى التسمم الحاد بأول أكسيد الكربون

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يعد التسمم الحاد بأول أكسيد الكربون نوعاً من أنواع التسمم الأكثر شيوعاً. يحفز أول أكسيد الكربون حدوث التهابات والتي قد تنشط استجابة المرحلة الحادة. بالإضافة إلى ذلك تعتبر إصابة عضلة القلب من المضاعفات الخطيرة لهذا النوع من التسمم. لذا كان الهدف من هذه الدراسة هو تقييم بعض بروتينات المرحلة الحادة فى مرضى التسمم الحاد بأول أكسيد الكربون والدور المحتمل لهم كدلالات جديدة لحدوث إصابة بعضلة القلب. تم دراسة خمسين من مرضى التسمم الحاد بأول أكسيد الكربون من الذين تم دخولهم وحدة علاج التسمم بجامعة طنطا في الفترة من أول مايو ٢٠١٣ إلى نهاية أبريل ٢٠١٥ . تم أخذ التاريخ المرضي والفحص الإكلينيكي وعمل الفحوصات التالية لكل مريض: غازات بالدم ومستوى التروبونين I وبروتينات المرحلة الحادة (سي آر بي شديد الحساسية) ومستوى الألبومين بالمصل ومجموع عدد كرات الدم البيضاء) وتم أيضاً قياس تركيز الكربوكسي هيموجلوبين. وتمثلت المجموعة الضابطة بخمسين من الأصحاء مطابقين لعمر وجنس الحالات محل الدراسة. كان متوسط عمر الحالات محل الدراسة ٣٤ , ٠ ± ٣ , ١٠ سنة وتكونت من ٢٤ أنثى و٢٦ ذكرا ,كان خفقان القلب أكثر الأعراض شيوعا (٨٤٪) وسجل انخفاض ضغط الدم وزيادة ضربات القلب ٧٦٪ و ٧٠٪ على التوالي. وكان رسم القلب الكهربائي طبيعياً في ٣٠٪ ولوحظ زيادة ضربات القلب المنتظمة ٦٠٪ في حين التغيرات المميزة للإسكيميا سجلت ٢٤٪ من المرضى الخاضعين للدراسة. بالإضافة إلى ذلك كان مستوى سي آر بي شديد الحساسية في المصل ومجموع كرات الدم البيضاء أعلى بينما كان مستوى الألبومين في المصل أقل في المرضى الخاضعين للدراسة مقارنة بالمجموعة الضابطة. علاوة على ذلك فقد تأثرت بروتينات المرحلة الحادة أكثر وارتبطت بقوة وبدلالة إحصائية مع وجود إصابة بعضلة القلب. لذلك فإن تحديد مستوى سي آر بي شديد الحساسية ومجموع عدد كرات الدم البيضاء والألبومين بالمصل عند دخول المريض من الممكن أن تكون أدوات متاحة وفعالة في تقييم التسمم الحاد بأول أكسيد الكربون والكشف عن وجود إصابة بعضلة القلب.