Research Article

Human fatty acid binding protein as an early predictive biomarker for carbon monoxide induced cardiotoxicity

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Abstract
Background: Carbon monoxide (CO) poisoning has deleterious cardiac effects which necessitate proper diagnoses and assessment using ECG and cardiac biomarkers; CKMB and troponinI. Human fatty acid-binding protein (H-FABP) recently used as a new cardiac marker as it is identifiable in serum by myocardial cell destruction. **Aim of the work**: To assess the role of H-FABP as an early predictor biomarker for CO induced cardiotoxicity. **Patients and Methods:** A prospective observational study was conducted on 25 adult (18-65years old) with diagnosis of acute CO poisoning of both sex, admitted to Poison Control Center of Ain shams University Hospitals **(PCCASUH)** from November 2015 to April 2016. Patients were divided into; moderately intoxicated cases7 patients (**Group 1**) and severely intoxicated patients18 patients **(Group 2)**. **Cardiac assessment** was done through ECG recording and analysis for (CKMB, CtnI and HFABP) serum level for all patients. This was done on (ER) (hour 0), after 6 hours and after 24 hours of admission. **Results:** The study recorded; chest pain 40%, syncope 60% and shock 28% in all patients which were more in **group2** than that in **group 1** and that was significant. **Group2** developed more significant ischemic ECG changes than **group 1**. The mean **CK-MB and troponin I level** showed increase with high level at 6th hour than 0hour and then decreased at 24th hour in both groups and that was statistically significant. **Group 2** had significant higher mean serum **H-FABP level** compared to **group 1** at 6th and 24th hour. Which tend to decrease continuously at 0hour, 6th hour and 24th hour in both groups this decrease with time was significant. In-addition, it showed decreasing sensitivity with time (0, 6 and 24hours) in both groups and that was significant. As well as it showed increasing specificity with time (0, 6 and 24hours) in both groups and that also was significant. **Conclusion and recommendations:** Proper diagnosis and assessment of CO induced cardiac injury using ECG, cardiac enzymes, and even echocardiography is necessary. Measurement of H-FABP is useful sensitive indicator identifying early cardiac injury in CO poisoning with recommendation to be used as bedside test for acute CO poisoning patients presented with chest pain as it is more sensitive than troponinI.
Key words: Carbon monoxide, cardiotoxicity, cardiac biomarkers, ECG, H-FABP

Introduction:
Acute carbon monoxide (CO) poisoning is considered an important clinical emergency due to it is severe cardiovascular effects with large proportion of deaths (Finegold et al., 2012). In Cairo, PCCASUH received 411 cases of acute CO poisoning in 2007, 25 patients were sever and necessitate ICU admission and 5 were died. Also in 2008 623 cases of acute CO poisoning were received; 22 patients were sever and necessitate ICU admission and 8 patients died as reported by (Tawfik and Elhelaly, 2015).

Carbon monoxide poisoning has a variety of cardiac effects including arrhythmias, coronary spasm, ventricular dysfunction and myocardial infarction (Dileo et al., 2011). Consequently, it is important to diagnose cardiac involvement rapidly in these patients (Shochat, 2016).
Increased cardiac biomarkers as: (Creatinine kinas myocardial brand (CKMB), troponinI and T and myoglobin) and ischemic (ECG) changes have been reported with severe CO poisoning (Erenler et al., 2013).

Human fatty acid-binding protein (H-FABP) is a cytosolic protein abundantly present in myocardial tissue with small molecular weight. This protein has been recently used as a new marker since it is identifiable in serum by myocardial cell destruction. In contrast to myoglobin, HFABP is more specific than

myoglobin and there is more fatty acid binding proteins in the heart compared to skeletal muscle and it can be identified earlier than CKMB and troponins in acute coronary syndromes (Ibrahim, 2014).

Aim of the Work

This study aimed to assess the role of HFABP as an early predictor biomarker for CO induced cardiotoxicity among patient admitted to the PCCASUH.

Ethical consideration:

All collected data were anonymous and confidentiality issues were preserved and valid informed consent was taken, an ethical approval from ethical committee at faculty of medicine, Ain shams university as well as an approval from director of PCCASUH were obtained.

Patient and Method

A prospective observational study was conducted on 25 adult (18-65years old) with diagnosis of acute CO poisoning of both sex, admitted PCCASUH from November 2015 to April 2016. With exclusion of smoker, anemic, pregnant patients, those with preexisting history of ischemic heart, kidney and liver diseases as well as chronic CO poisoning.

Grouping: Patients were divided into 2 groups according to the clinical grading of carbon monoxide poisoning (GoldFrank, 2014); Group1: (moderately intoxicated cases) who were 7 patients 28% of all patients. Group2: (severely intoxicated patients) who were 18 patients 72% of all patients. For all patients included sociodemographic and clinical intoxication data were collected as; exposure, delay time and previous treatment received. Clinical assessment was done including vital data recording, cardiac, pulmonary, hepatic, renal and neurological examination. Cardiac assessment through ECG recording and analysis for serum level of CKMB, CtnI and HFABP for all patients were done on (hour 0), 6 hours and 24 hours of admission.

Result

The mean age of group 1 was 29 ±10.72 (range 18-45) year old and the mean age of group2 was 31±9.80 (range 27-59) and that was insignificant between both Figure (1). 5 (92%) patients of Group1 were male and 2 (8%) were female. Six (96%) of them were nonsmoker and one (4%) was occasionally smoker. Six (96%) had no past history of chronic disease but one (4%) of them had hypertension. 13 patients (80%) of Group 2 were male and 5(20%) were female. Sixteen (92%) of them were nonsmoker and 2(8%) of them occasionally smoker. Seventeen (96%) had no past history of chronic disease but one (4%) of had hypertension. These were non-significant between both groups (Fig. 1&2).

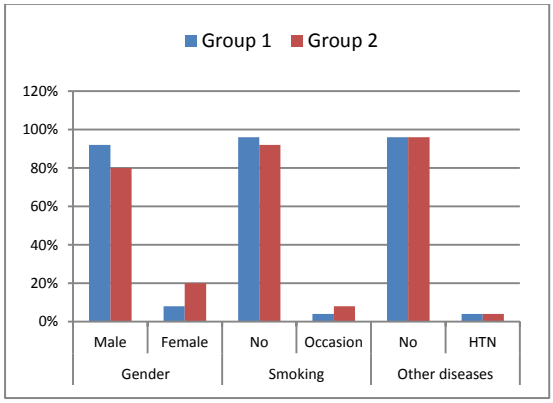

Fig (1): Number and percentage of gender, smoking habits and presence of preexisting ds

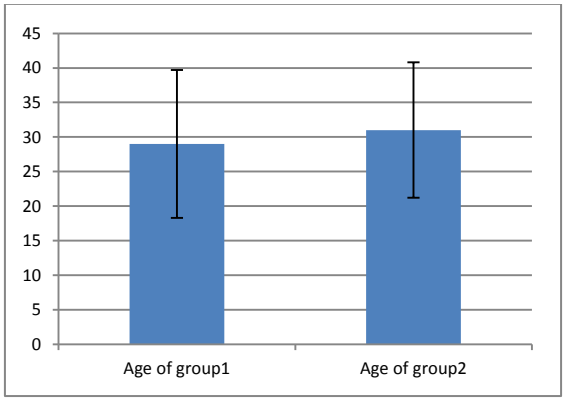

Fig (2): Mean and standard deviation of age

All patients of both groups were exposed to CO accidentally by inhalation rout. The presence of gas heater improperly installed in a poorly ventilated indoor situation was the primary cause of poisoning for the majority (85.8%) of group1 and (94.5%) of group 2 and that was nonsignificant. History of previous management; intravenous saline and normobaric O_2 received

by 4 (58%) of group1 and 10 (55%) of group 2 with one of group2 had CT brain and received mannitol before admission and that was nonsignificant. Group 2 presented after more prolonged time of exposure in relation to group 1 and this was statistically significant (Fig 3).

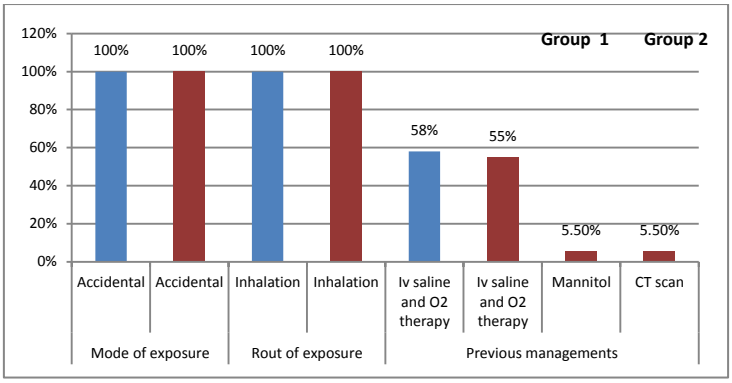

Figure (3): Intoxications data (mode of exposure, rout of exposure and previous managements)

As regard vital data, Group 1 showed that mean heart rate was (105.48±6.59/min), mean systolic BP was (108±24.11mmhg) mean diastolic BP was (67.60±24.88mmhg), mean temperature was (36.9±.23C˚) and mean respiratory rate was (24.83±4.09/min). As regard GCS the mean value was (10.1±1.2) with range from 9 to13. Group 2 showed that mean heart rate was (120.48±14.59/min), mean systolic BP was (85±20.11mmhg), mean diastolic BP was (55.60±15.88 mmhg), mean temperature was (37.1±.21C˚) and mean respiratory rate was (35.83±10.09/min). As regard GCS the mean value was (7.92±2.2) with range from 3 to 9. Tachycardia, hypotension, tachypnea and decreased GCS were more prominent in group 2 in comparison to group1 and these were significant. Unlike the change in temperature between both groups was not significant (Figure 3)

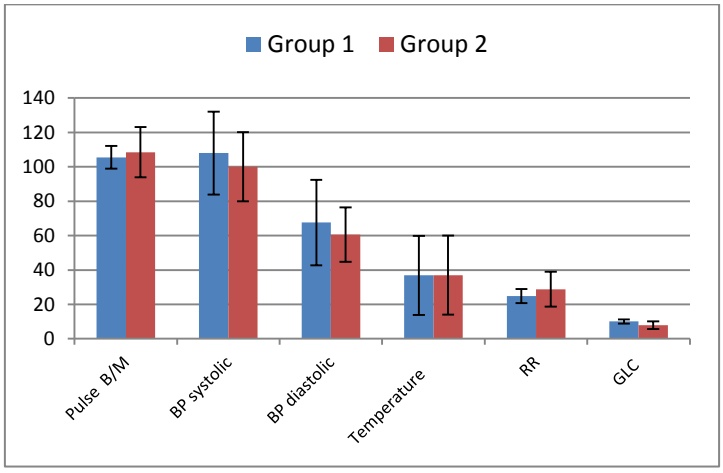

Figure (3): Mean and standard deviation of recorded vital signs and Glasgow coma scale (GCS)

Regarding clinical manifestations Nausea and vomiting were common presentations. All patients of group 1 were disoriented and drowsy, 3 (48%) were agitated and none developed lateralization. Four of them (58%) developed tachypnea with mild respiratory distress. Three of patients developed chest pain, 4 (58%) came with history of syncope and none had shocked. Seven patients of group 2 (38.5%) were confused, 11 (71.5%) were comatose, half of them developed agitation and only one patient (5.5%) showed lateralization signs.

Moreover, 13 (71.5%) of them developed tachypnea with 11 (61.5%) had cyanosis and severe respiratory distress. Ten (55%) patients developed chest pain, 13(71%) with history of syncope and 7 (38.5%) were shocked. The common cardiac manifestation were chest pain 40%, syncope 60% and shock 28% in all patients. Neurological, cardiac and respiratory morbidities were more recorded in group 2 than that in group 1 and this was significant.

Table (2): Chi-square test comparison of clinical manifestations between both groups admitted to PCCASUH, from November 2015 to April 2016.

Manifestations	Groups	N	%	P
Nausea and vomiting	Group1	7	100%	>0.05
	Group2	18	100%	
Conscious level	Group1	7(disoriented)	100%	<0.05
	Group2	7(confused)	38.5%	
		11coma	61.5%	
Agitation	Group1	3	48.0%	
	Group2	9	50.0%	
Lateralization signs	Group1	0	00.0%	
	Group2	1	5.5%	
Tachypnea	Group1	4	58.0%	<0.05
	Group2	13	71.5%	
Respiratory distress RD	Group1	4	58.0%	
	Group2	11(cyanosed)	61.5%	
Chest pain	Group1	3	48.0%	<0.05
	Group2	11	61.5%	
Syncope	Group1	4	58.0%	
	Group2	13	71.5%	
Shock	Group1	0	00.0%	
	Group2	7	38.5%	

ECG was done on admission and 6 hours later for **Group1,** and revealed that 3 (48%) of patients showed normal finding, 4 (52%) of patients had sinus tachycardia to be subsided at 24 hours of admission with all patients were normal. Group 2, ECG was done on admission and 6 hours later revealed that 4 (22%) of them had normal finding, 10 (55%) had sinus tachycardia, one (5.5%) had occasional attacks of bradycardia and 3 (16.5%) had ischemic changes in form of ST segment depression and T wave inversion. At 24 hours of admission 16 (89%) of patients showed normal ECG finding and 2 (11%) of still had sinus tachycardia. There were no significant changes in ECG finding at different time between both groups. However, the patients with severe toxicity developed significant ischemic ECG changes than moderately intoxicated patient (Figure 3&4).

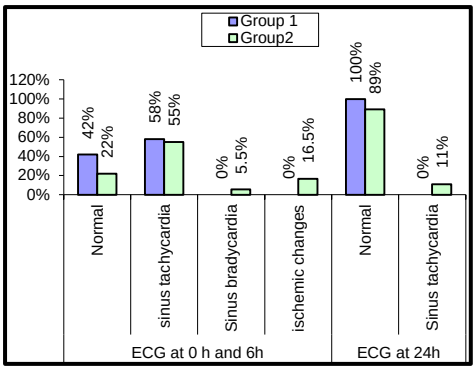

Figure (3): Number and percentage of (ECG) finding

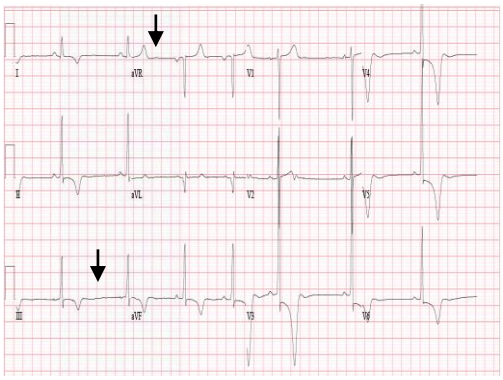

Figure (4): ECG finding in the group2 patients showing ST segment depression

Group 1, on admission (0h) the study revealed that 1 (14.20%) of the 7 patients had elevated serum CK-MB levels (25.01± 30.02U/L), 1(14.20%) had elevated serum troponin I levels (0.02 ±0.02 ng/mL) and 5(71.60%) had elevated serum H-FABP levels (0.45 ±0.12 ng/mL). At 6th hours of admission we found that 2(28.40%) had elevated serum CK-MB levels (27.90 ±35.27U/L), 2(28.40%) had elevated serum troponin I levels (0.02 ± 0.02ng/mL) and 4(58%) had elevated serum H-FABP levels (0.14 ± 0.11ng/mL). At 24th hours of admission 1(14.20%) of the 7 patients had elevated serum CK-MB levels (27.20 ± 34U/L), 1 (14.20%) had elevated serum troponin I levels (0.02± 0.02ng/mL) and 2(28.40%) had elevated serum H-FABP levels (0.10±0.009 ng/mL). Group 2, on admission (0h) the study found that 7 (39%) of the 18 patients had elevated serum CK-MB levels (39.07± 30.22U/L), 6(33%) had elevated serum troponin I levels (0.02 ±0.02 ng/mL) and 13(72%) had elevated serum H-FABP levels (0.47 ±0.88 ng/mL). At 6th hours of admission we found that 9(50%) had elevated serum CK-MB levels (49.24 ±36.27U/L), 8(44%) had elevated serum troponin I levels (0.03 ± 0.04ng/mL) and 12(66.5%) had elevated serum H-FABP levels (0.32 ± 0.68ng/mL). At 24th hours of admission 6(33.5%) of the 18 patients had elevated serum CK-MB levels (49.12 ± 35U/L), 8 (44%) had elevated serum troponin I levels (0.02± 0.02ng/mL) and 9(50%) had elevated serum H-FABP levels (0.21±0.54 ng/mL).

The mean CK-MB levels increased with time showing high level at 6th hour than 0hour and then decreased at 24th hour in both groups and the changes in serum CKMB levels between two groups were statistically significant. The mean serum troponin I levels increased with time showing high level at 6th hour than 0hour and then decreased at 24th hour in both groups and the changes in serum troponin I levels were statistically significant in group 2. There was no statistically significant difference between the groups in mean serum H-FABP levels at 0 hour. However, the patients of group2 had significant higher mean serum H-FABP levels compared to the patients of group1 at 6th hour and 24th hour

Human fatty acid binding protein (HFABP) levels tend to decrease continuously at 0hour, 6 hour and 24 hour in both groups and the decrease with time was significant. At 6th hour of admission the study revealed that 2 (8%) only of the 25 patients had higher serum H-FABP levels than at 0 hour, and 9(36%) of them had higher serum troponin I levels than at 0 hour in all patients of both groups (table 3 & fig. 6).

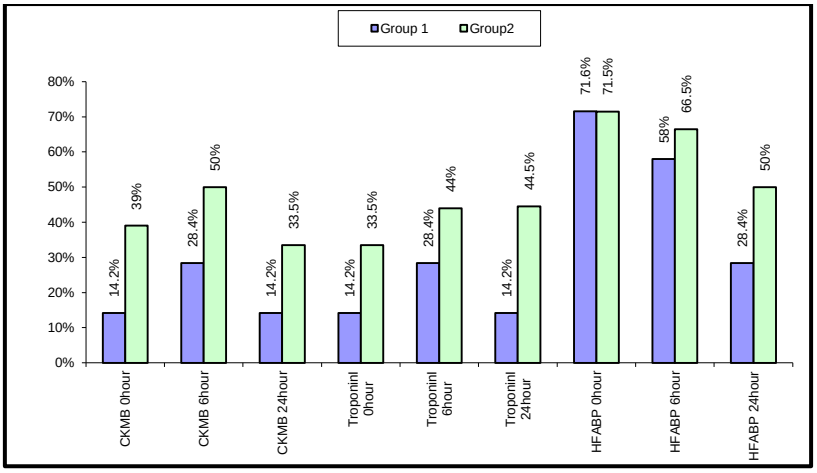

Figure (6): Number and percentage of CKMB, troponin I and HFAB at 0 hour, 6 hour and 24 hour

Table (3): ANOVA test comparison of some cardiac markers (CKMB, TroponinI and HFABP) at 0hour, 6hour and 24hour between both groups admitted to PCCASUH, from November 2015 to April 2016.

Cardiac markers	Groups	Mean	SD	P value
CKMB 0hour (U\L)	Group1	25.01	±30.02	<0.05
	Group2	39.07	±30.22	
CKMB 6hour (U\L)	Group1	27.90	±35.27	<0.05
	Group2	49.24	±36.27	
CKMB 24hour (U\L)	Group1	27.20	±34	<0.05
	Group2	49.12	±35	
TroponinI 0hour (ng\ml)	Group1	0.02	±0.02	>0.05
	Group2	0.02	±0.02	
TroponinI 6hour (ng\ml)	Group1	0.02	±0.02	<0.05
	Group2	0.03	±0.04	
TroponinI 24hour (ng\ml)	Group1	0.02	±0.02	>0.05
	Group2	0.02	±0.02	
HFABP 0hour (ng\ml)	Group1	0.45	±0.12	>0.05
	Group2	0.47	±0.88	
HFABP 6hour (ng\ml)	Group1	0.14	±0.11	<0.05
	Group2	0.32	±0.68	
HFABP 24hour (ng\ml)	Group1	0.10	±0.09	<0.05
	Group2	0.21	±0.54	
HFABP level decreased with time and p value was <0.001				

SD=standard deviation CKMB= creatinine kinas myocardial brand HFABP=human fatty acid binding protein

At time of admission (0h) HFABP showed 100% sensitivity in both groups and 38.9% specificity in group 1 and 46.62% in group 2 in relation to troponin I. The change in specificity between two groups at time of admission was insignificant (table 4 & Fig. 7). At 6 hours of admission HFABP showed 66.5% sensitivity in group1 and 88.8% in the group2, and showed 50% specificity in group1 and 55% in group2, in relation to troponin I. These changes in

sensitivity and specificity between two groups at 6 hours of admission was insignificant (table 5& Figure 8). At 24 hours of admission HFABP showed 50% sensitivity in group1 and 60% in group2, and showed 60% specificity in group1 and 62.5% in group2 in relation to troponin I (table 6 & Fig. 9). These changes in sensitivity and specificity between both groups at 24hours of admission was insignificant. HFABP showed decreasing sensitivity with time (0, 6 and 24hours) in both groups and the decrease was significant. It also showed increasing specificity with time (0, 6 and 24hours) in both groups and that was significant.

Table (4): Sensitivity and specificity of HFABP relative to Troponin I at 0 hour obtained for both groups admitted to PCCASUH, from November 2015 to April 2016.

HFABP at 0hour		Troponin I at 0hour				HFABP at 0hour	
		Negative		Positive		Sensitivity	Specific
		N	%	N	%		
Group1	Negative	2	38.9%	0	0.0%	100.0%	38.9%
	Positive	4	61.1%	1	100.0%		
Group2	Negative	7	46.62%	0	0.0%	100.0%	46.62 %
	Positive	8	53.29%	3	100.0%		
P value						>0.05	>0.05

N=number HFABP=human fatty acid binding protein

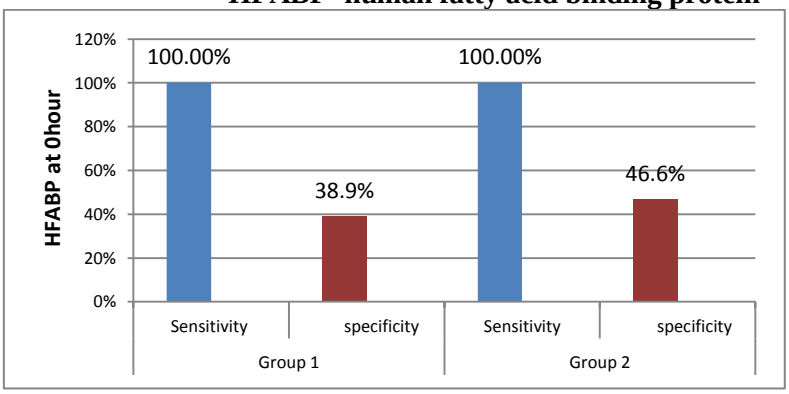

Figure (7): Sensitivity and specificity of HFABP relative to Troponin I at 0 hour obtained.

Table (5): Sensitivity and specificity of HFABP relative to Troponin I at 6hours of admission obtained for both group admitted to PCCASUH, from November 2015 to April 2016.

HFABP at 6hour		Troponin I at 6hour				HFABP at 6hour	
		Negative		Positive		Sensitivity	Specificity
		N	%	N	%		
Group1	Negative	2	50.0%	1	33.5%	66.5%	50.0%
	Positive	2	50.0%	2	66.5%		
Group2	Negative	5	55.0%	2	22.2%	77.8%	55.0%
	Positive	4	45.0%	7	77.8%		
P value						>0.05	>0.05

N=number HFABP=human fatty acid binding protein

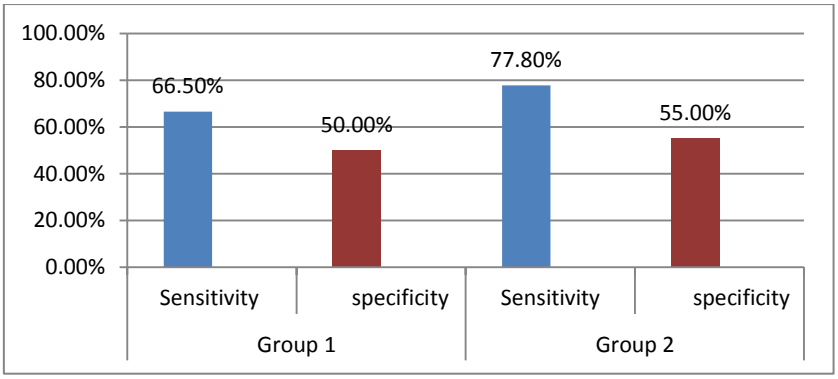

Figure (8): Sensitivity and specificity of HFABP relative to Troponin I at 6 hours of admission obtained for both group

Table (6): Sensitivity and specificity of HFABP relative to Troponin I at 24 hours of admission obtained for both groups admitted to PCCASUH, from November 2015 to April 2016.

HFABP at 24hour		Troponin I at 24hour				HFABP at 24 hour	
		Negative		Positive		Sensitivity	Specificity
		N	%	N	%		
Group 1	Negative	3	60.0%	1	50.0%	50.0%	60%
	Positive	2	40.0%	1	50.0%		
Group 2	Negative	5	62.5%	4	40.0%	60.0%	62.5%
	Positive	3	38.5%	6	60.0%		
P value						>0.05	>0.05

N=number HFABP=human fatty acid binding protein

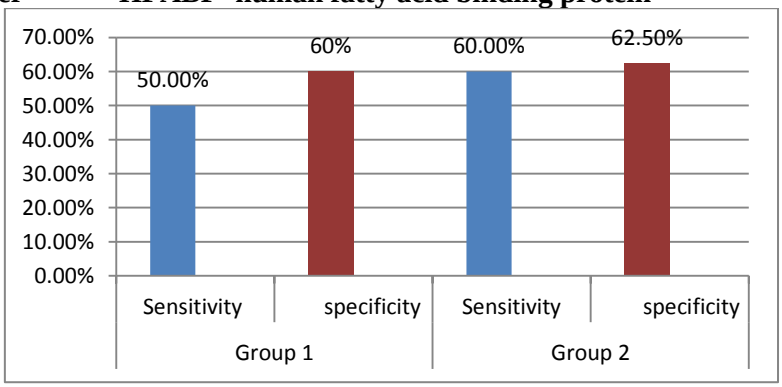

Figure (9): Sensitivity and specificity of HFABP relative to Troponin I at 24 hour obtained for both groups

Discussion

Acute carbon monoxide (CO) poisoning is considered an important clinical emergency due to it is severe cardiovascular effects with large proportion of deaths (Finegold et al., 2012). This was reported in the current observational prospective study which was carried out on 25 acute moderate and severe CO intoxicated patients admitted to PCCASUH from November 2015 to April 2016. The study showed that 7 patients were diagnosed as moderate acute CO poisoning (Group I) and 18 patients were diagnosed as severe acute CO poisoning (Group 2).

The study aimed to assess the role of H-FABP as an early predictor biomarker for carbon monoxide induced cardiotoxicity among patients included. Lippi et al., (2012) attributed myocardial injury induced by CO poisoning to tissue hypoxia as well as damage at the cellular level, with release of cardiac biomarkers. Tolles, (2016) investigated CO induced myocardial injury and which was diagnosed by ECG

findings and elevated cardiac biomarkers as CKMB, troponin-I and myoglobin which considered as early marker of myocardial infarction. Recent studies support the use of the new biochemical indicators, H-FABP as its high concentration inside myocardium that help in identifying the cardiotoxicity of CO poisoning at an early phase (Altintop et., al. 2018).

There was no significant difference regarding demographic data (age, gender, special habits and presence of previous diseases) between the two studied groups in the current study as well as intoxication data: mode of exposure, rout of exposure and previous management were received before ER admission this came in agreement with Pepe et al., (2011), who studied 340 CO poisoned patients, and found that no correlation between age, sex, suicide attempt, source of carbon monoxide and development of severe manifestations.

The study showed that, the presence of gas heater improperly installed in a poorly ventilated indoor situation was the primary cause of carbon monoxide poisoning in the majority of patients of **group 1** and (94.5%) of **group 2** and that was proved according to Quinn et al., (2013); Sharma et al., (2011), who stated that home accidents due to CO leakage from water heaters or stoves were the primary causes of CO intoxication in developing coun-tries but suicidal attempts using charcoal burning in confined spaces were more common in developed countries. There was significant difference between the two studied groups regarding delay time and this is usually associated with development of severe symptoms and signs. These findings were in agreement with Ku et al., (2015), who studied 43 CO poisoned patients retrospectively and reported that CO exposure duration and delay time was an important factor for the development of severe neurological and cardiac sequelae.

Regarding vital signs the study showed that tachycardia, hypotension, tachypnea and decreased GCS were more significantly prominent in severe group than moderate group however; the change in temperature between both groups was not significant. The significant decrease in systolic blood pressure in group 1 when compared with group 2 after acute CO poisoning, could be explained according to Beppu, (2014) who stated that myocardial depression and peripheral vasodilatation causing hypotension were important in the genesis of CO-induced cardiac and neurological injury**.** Moreover, Shen et al., (2015) study conducted on 148 CO poisoned patients found that hypotension was associated with poor outcome including cardiogenic shock and death. On other hand Zengin et al., (2015), who postulated tachycardia and tachypnea occurred early in CO poisoned patients as a compensatory response to cellular hypoxia and decreased cardiac systolic function but could not be used as a predictor of severity or poor outcome after acute CO poisoning.

The present study also revealed that nausea, vomiting and headache were the common clinical manifestations and occurs in all patients of both groups, and that was insignificant between both groups this was in agreement with Park et al., (2012), who found insignificant difference between severely manifested patients and moderate one as regard incidence of headache, vomiting and loss of consciousness.

Cardiological manifestations were common in patients of group 2 after acute CO poisoning with chest pain 61.5%, syncope 71.5% and shock 38.5% and that could be explained according to, Shen et al., (2015), who stated that the most susceptible organs for late sequelae after acute CO poisoning were the brain and heart because of their high metabolic demand. This finding also goes hand in hand with Ismail et al., (2013), who studied 50 patients with acute CO poisoning and found that (58%) were complaining of dyspnea, (96%) had palpitation, (50%) had chest pain, and (96%) had ECG changes. ECG was done for all cases in both groups on admission, 6 hours and 24hours post admission, the most common ECG changes were sinus tachycardia 56%, 12% of them had ischemic changes in form of ST segment depression and T wave inversion and 4% had occasional attack of bradycardia. These findings, were in accordance with Ramazan et al., (2011) who investigated ECG finding among 80 patients with acute CO poisoning on admission, 41.2% of patients had normal ECG sinus tachycardia was present in 48.8% of

patients, 5% of them had ischemic changes, 3.8% had sinus arrhythmia and 1.2% had ventricular extrasystole. They also added that ischemic ECG changes were found in severely intoxicated patients compared to other patients. Ismail et al., (2013) attributed ECG changes in acute CO poisoning to the direct toxic effect of CO on the heart or due to CO induced respiratory and central nervous systems depression. Also they reported atrial flutter, atrial fibrillation, tachycardia or ischemia especially with COHB levels of > 20-25% at time of admission.

Regarding Serum CK-MB, the current study showed that the CK-MB level increased with time showing high level at the 6th hour than 0hour and then decreased at 24th hour in both groups and the changes in serum CKMB levels were statistically significant between both groups. Açikalin et al., (2011) assumed that, CO exposed patients, high CK-MB might originate from hypoxia present in all tissues rather than cardiac injury. The use of CK-MB as a marker of cardiotoxicity was substantially limited due to lack of tissue specificity and sensitivity. This could be attributed to the fact that CO intoxication produce cardiotoxicity and frequently produce myotoxicity, which further decreases tissue specificity of CK-MB.

In addition, Serum troponinI analysis in the current study showed that troponin I increased with time showing high level at 6th hour than 0hour and then decreased at 24th hour in both groups and the changes in serum troponin I was statistically significant in severe intoxicated group. This finding was in concordance with Ramzan et al., (2011), who showed that the serum troponin I and CK-MB levels increased at 6th hour and then decreased at 24th hour in both groups, and the changes in serum troponin I levels were statistically significant only in severely intoxicated patients. Ismail et al., (2013) added that cardiac troponin I was significantly higher in CO poisoned patients due to hypoxic cardiotoxic effects with no significant correlation between COHb level and cardiac troponin I.

As regard human fatty acid binding protein the study found that the serum H-FABP levels tended to decrease continuously at 0 hour, 6th hour and 24th hour in both groups and this decrease was significant, there was no statistically significant difference between the studied groups in terms of serum H-FABP levels at 0 hour. However, the patients of group 2 had significant higher serum H-FABP levels compared to the patients of group 1 at 6^{th} hour and 24^{th} hour, and these finding were in agreement with study conducted by Ramzan et al., (2011) on 80 patients of acute CO poisoning and found that the serum H-FABP levels tended to decrease continuously at 0 hour, 6^{th} hour and 24^{th} hour in both groups and this decrease was significant. They also recorded that, H-FABP levels increase earlier than other cardiac biomarkers and this are higher in patients with pathological results on echocardiography (ECHO). The current study also evaluated on a case basis, only four (5%) had higher H-FABP level at the 6th hour than that of at 0 hour; the troponin I levels of (29%) were higher at 6th hour than that of at 0 hour. This obtained data supports the use of H-FABP in identifying the cardiotoxicity in CO intoxications in patients presenting to the ED. This could be explained by their kinetic as HFABP early released with minor tissue injury unlike troponin I which appears within 4-6 hours of tissue injury. This was correlated with experimental study conducted by Ibrahim, (2014) who assessed FABP in rats exposed to CO poisoning, he reported that serum FABP levels increased just after the CO exposure, however, the serum troponin I levels only increased at 6 hours after exposure.

The current study found that at time of admission (0h) H-FABP showed 100% sensitivity in both groups and 38.9% specificity in the group 1 and 46.62% in the group 2 in relation to troponin I and the change in specificity between the two groups at time of admission was insignificant. At 6 hours of admission H-FABP showed 66.5% sensitivity in group 1 and 88.8% in group 2, and showed 50% specificity in group 1 and 55% in group 2 in relation to troponin I, and also these changes were insignificant. At 24 hours of admission H-FABP showed 50% sensitivity in group 1 and 60% in group 2, and showed 60% specificity in group 1 and 62.5% in group 2 in relation to troponin I. The change in sensitivity and specificity between the two groups at 24 hours of admission was insignificant. However, H-FABP showed decreasing sensitivity with time (0, 6 and 24 hours) in both groups and the decrease

was significant and showed increasing specificity with time (0, 6 and 24 hours) in both groups and that was significant. This finding was in concordance with Gerede et al., (2015) study, who compare the qualitative measurement of H-FABP with other cardiac markers as an early diagnostic marker of non-ST-segment elevation myocardial infarction. They recorded that, H-FABP was a better diagnostic marker compared to CK-MB and troponin I (accuracy index 85%), with a high sensitivity (79%) and specificity (93%) for early diagnosis (≤six hours). They also recommended bedside H-FABP measurements contribute to correct early diagnoses, as its levels are elevated soon following MI, and measurement is easy and rapid

Moreover, clinical study conducted by Ramazan et al., (2011) found that 70% of patients presenting to ED with moderate or severe CO intoxication had elevated H-FABP levels, whereas 36% had elevated troponin I levels. H-FABP levels decreased at the 6th and the 24th hours while troponin I and CK-MB levels increased at the 6th hour and decreased at the 24th hour. These findings suggest that in CO intoxication, cardiac injury starts with exposure to CO and H-FABP can be used as an early marker of acute cardiac injury there was lack of information on the duration and the severity of CO exposure. Further studies are necessary in this regard.

Conclusions:

Identification of the degree of cardiac injury is essential in patients admitted and followed-up with the diagnosis of CO intoxication. ECG, cardiac enzymes, and even echocardiography should be performed in the ED on these patients to determine the level of injury. Measurement of H-FABP is useful sensitive indicator in identifying the early cardiac injury in patients presenting within 2 to 4 h of after the onset of chest pain as the serum and that is more sensitive than troponin I.

Recommendations

It is recommended to do H-FABP levels at admission after acute CO poisoning as a useful early senstive biomarker for predicting cardiac injury. Appropriate cardiological assessment should be done to every CO poisoned patient during hospitalization especially those with risk factors, to allow prompt recognition of sequelae. Further prospective studies are needed to evaluate the role of more specific type of HFABP for cardiac injury as heart type fatty acid binding proteins.

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