Original Article

Role of Glycogen Phosphorylase in Prediction of Cardiotoxicity Associated with Acute Carbon Monoxide Poisoning

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ABSTRACT

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Introduction: Carbon monoxide (CO) poisoning is the leading cause of death from intoxication. The heart is extremely susceptible to CO-induced hypoxia due to its high oxygen demand. Cardiovascular involvement in CO poisoning could be clinically occult and undiagnosed due to lack of overt symptoms or specific electrocardiogram (ECG) changes. There is an emerging need for early and specific markers for myocardial hypoxia. One potential candidate is the glycogen phosphorylase BB isoenzyme (GPBB). Aim of the study: The study was planned to assess the role of glycogen phosphorylase in early prediction of myocardial toxicity due to CO poisoning. Methods: This prospective study included 2 groups; Control group and Patient group. Control group included 15 non-smokers healthy adults aged between 20 and 35 years old selected from patients' companions presented to Zagazig University Hospitals. Patient group included 30 adults aged 20-35 years old with history of CO exposure within 6 hrs. They were sub- divided into mild & moderate CO poisoning groups according to COHB% & clinical manifestations. The study was performed during the period between January 2015 to April 2016**.** All the subjects enrolled in the study were subjected to clinical examination, ECG, echocardiogram (Echo), cardiac troponin 1 (cTn-1) and GPBB assessment. Results: There was a significant increase in heart rate between patient and control groups. Also, a significant increase in GPBB was found between patient and control groups and this was associated with a significant decrease of ejection fraction evaluated by Echo in moderate CO poisoning group when compared with control group. No significant difference was found as regard ECG findings between patient and control groups. **Conclusion:** GPBB can be used as an early predictor of CO-induced myocardial ischemia which in turn may modify management plan to avoid later sequelae affecting ventricular function.

Key words: Carbon monoxide poisoning, Cardiotoxicity, GPBB, Echo

I. INTRODUCTION

Carbon monoxide (CO) is colorless, tasteless and odorless gas produced from incomplete combustion of carbon fuels, such as firewood, charcoal, gasoline, natural gas, inadequately ventilated heaters, car exhausts or from chemicals such as methylene chloride paint stripper (**Piantadosi, 2004**). Carbon monoxide poisoning is considered one of the most important toxicological global causes of

morbidity and mortality **(Chiew & Buckley, 2014).**

The signs and symptoms of CO poisoning are diverse, ranging from headache, lethargy, dizziness, nausea and confusion to cardiac and neurological disturbances. Cardiovascular complications of CO poisoning include myocardial ischemia, left ventricular dysfunction, or arrhythmias (**Chandrasekar et al., 2013)**. CO can affect the heart through several mechanisms of action; cellular hypoxia due to impaired oxygen delivery, reversible inhibition of mitochondrial respiration and oxidative stress **(Rastelli et al., 2009**).

Several clinical studies have revealed that even mild co poisoning has unfavorable acute outcome on left and right ventricular function in adults **(Ozgur et al., 2013).** A normal carboxyhemoglobin (COHb) level for a non-smoker is <2%. Slightly elevated levels of COHb (2-6%) have been shown to decrease exercise tolerance and worsen myocardial ischemia in patients with known coronary artery disease **(kalay et al., 2007).**

Unfortunately, routine cardiac biomarkers such as creatine kinase (CK), creatine kinase-MB (CK-MB), and myoglobin have low diagnostic significance. **(Teksam et al., 2010).** Also, The sensitivity of cardiac markers is low within the first 6 hours of hypoxia **(Suleyman et al., 2019). Melissa and Allen (2010)** stated that Cardiac troponin I is specific to the myocardium, but the rise in serum level may be delayed for 3–4 hours after the occurrence of cardiac symptoms in patients with acute myocardial ischemia.

Human Glycogen phosphorylase (GP) is a key enzyme of glycogenolysis. Its release depends on the degradation of glycogen. It is considered as the most promising marker among the recently proposed markers for early diagnosis of myocardial ischemia **(Nigam, 2007)**. Its isoenzyme BB (GPBB) in particular has a distinct sensitivity to myocardial oxygen deficiency in cardiomyocytes **(Sebnam et al., 2011).**

According to the recent statistics of the American Heart Association, an early diagnosis (i.e., within 3 to 6 hours from onset of the symptoms) and an efficient risk stratification are crucial for management of patients with suspected myocardial damage (**Roger et al., 2012).**

The present study aimed to investigate the role of Glycogen phosphorylase BB (GPBB) as an early biomarker of cardiac injury in acute CO poisoning and to compare the diagnostic performance of GPBB to routine cardiac marker cTn-1, ECG changes, Echo findings and clinical manifestations.

II. SUBJECTS& METHODS

This is a prospective, cross-sectional and controlled study. The study protocol was approved by the Ethical Committee for Research (Institutional Review Board), Faculty of Medicine, Zagazig University. Written consents were obtained from all participants.

II.1. SUBJECTS

The studied groups were classified into control group and patient group as follow:

Control group: Included 15 age and sex matched non-smoker healthy adults aged between 20 and 35 years old selected from patients' companions presented to Zagazig University Hospitals during the period between January 2015 and April 2016. They had no history of any medical diseases or toxic exposure and were subjected to clinical examination, cTn-1, GPBB assessment, ECG and Echo.

Patient group: Included 30 CO poisoned patients selected from Poison Control Center, Zagazig University Hospitals during the period between January 2015 to April 2016. Diagnosis of CO poisoning was made based

on history, COHB levels of more than 10 % with the following inclusion and exclusion criteria:

- Inclusion criteria:

• History of CO exposure

•Within 6 hours of exposure

•The selected patients were those diagnosed as mild CO poisoning (15 patients) and moderate CO poisoning (15 patients).

Severity of co poisoning was determined according to **Craig &Kent (2010)** as follow**:**

-Mild CO poisoning: COHB level of over 10% with minor clinical signs and symptoms including headache, nausea and vomiting.

-Moderate CO poisoning:

COHB level of over 10%, but under 20-25% associated with generalized feeling of weakness, dizziness, unsteadiness and problems with concentration and thinking

-Severe CO poisoning: COHb level of over 20-25%, loss of consciousness, and confusion or signs of cardiac ischemia.

- Exclusion criteria:

• Patients with any history of heart diseases, such as congenital heart diseases, rheumatic or valvular diseases, arrhythmias, pericarditis, coronary arterial disease, anemia, pulmonary diseases, diabetes, obesity, hepatic or renal diseases were excluded.

• Smokers (Heavy smokers may have COHb% up to 15 %).

• Severe cases of CO poisoning were also excluded from the study as they were already presented with neurological and/or cardiac manifestations and usually associated with abnormalities in ECG and in routine cardiac biomarkers. Also patients with severe CO poisoning are urgent cases in which hyperbaric oxygen will be recommended without delay or waiting for performing diagnostic or prognostic assessment included in the study and this in turn would affect the accuracy of the study.

II.2. METHODS

On admission before giving any treatment, the included patients were subjected to the following:

Clinical evaluation:

- Biochemical assessment: ABG, COHb%, cTn-1 and GP-BB.
- ECG
- ECHO

II.2.1. Clinical evaluation: Included assessment of heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), respiratory rate (RR) and temperature (Temp.).

II.2.2. Biochemical assessment: Carried out in Zagazig University Hospitals Laboratories except for GP-BB which was measured at Biochemical Department, Faculty of Medicine, Zagazig University.

Six ml blood samples were drawn from participants and divided into three portions; 2 ml on heparin containing tube used for COHB%, 2 ml was collected in plain tube for serum separation to measure c-Tn-I. The remaining 2ml was collected in Ethylene diamine tetra acetic acid (EDTA) treated tube and was centrifuged at 1,300 xg for 10 min at 4°C for plasma separation which was kept frozen at -80°C till assessment of GPBB. The methods used included the following:

- Carboxyhemoglobin % (COHB%: COHB% was analyzed by blood gas analyzer, Bayer 855.

- Cardiac troponin-I (cTn-I) level: was measured by VIDAS Troponin I Ultra (TNU, BioMerieux Inc., France) according **to Apple et al. (2008).** The cut off level for cTn-I was 0.01 ug/L.
- Glycogen phosphorylase BB(GPBB) level: was assessed using Enzyme-linked Immunosorbent Assay (ELISA) kit (Diagenics SE, Essen, Germany), according to the manufacturer's protocols **(Lee et al., 2012).** The cut off level was 10 ng/ml.

II.2.3. Electrocardiogram (ECG):

Twelve-lead ECG was recorded with a paper speed of 25 mm/s for all patients included in the study on admission.

II.2.4.Echocardiographic examination:

Echocardiographic examination was performed for all patients on admission. Each subject was examined using an Acuson Sequoia C256 Echocardiography System (Acuson, Mountain View, CA, USA) equipped with 3V2c and 5V2c broadband transducers with second harmonic capability. Echocardiographic images were recorded on videotape. M-mode images were used to measure left ventricular ejection fraction (LVEF%), left ventricular internal dimension in diastole (LVIDd) & left ventricular internal dimension in systole (LVIDs). The echocardiography was performed by an investigator who was blinded for clinical data and the analysis of the echocardiographic recordings.

II.3. Statstical analysis:

Data were processed using the Statistical Package for Social Science version 16 (SPSS Inc., Chicago, IL). Quantitative data were summarized as mean ± standard deviation (X ±SD). Qualitative data were summarized as percentage. Test of significance for qualitative data was Chi-square test. Test of significance for quantitative data was done using ANOVA test for comparison between groups. Least significant difference (LSD) for multiple comparison **(Norusis, 1997)** Spearman correlation test was used to evaluate the association between two variables. The significance level was considered at p value < 0.05.

III.RESULTS

In this study 65% of CO- poisoned cases resulted from burning of charcoal, wood, kerosene, or natural gas for heating and cooking while 35% was due to inadequately ventilated bathroom heater. Demographic criteria showed non-significant difference between CO- poisoned patient and control groups regarding age and sex distribution (Table 1)

Table (1): demographic data between control & patient groups

	Control group N=15	CO-poisoned group N=30	P value	Significance
Age(mean±SD)	25.3±4.1	26.3±3.9	0.63*	NS
Sex ▪Male ▪Female	 9(60%) 6(40%	 16(53.3%) 14(46.7%)	1.00**	NS

*T test **Chi-square test NS: Non-significant (p >0.05)

III.1.Clinical evaluation:

There was a statistically significant difference in the heart rate between CO-

poisoned patients and control group (P<0.05). However, no significant difference in the mean values of heart rate between mild and moderate co poisoning was detected (P>0.05). On the other hand, non-significant difference regarding systolic blood pressure (SBP), diastolic blood pressure (DBP), respiratory rate &temperature among the studied groups were found (Table 2)

Table (2): Statistical comparison among control, mild and moderate co -poisoned patients as regard mean values of vital signs using ANOVA and LSD.

Vital signs	Control N= 15	CO poisoned group		ANOVA test	
		Mild N= 15	Moderate N= 15	F	P
HR	100.51 ± 9.76	110.64 ± 8.68[a]	116.54±14.42[b,c]	9.58	0.001*
SBP	110.78 ± 12.56	111.65±10.56	11٠.11±9.35	0.10	0.91
DBP	68.52 ± 7.34	68.73 ± 8.51	67.59±9.54	0.08	0.93
Respiratory rate	18.61± 1.34	20.31 ± 2.76	20.93±4.12	8.11	0.09
Temperature	37.04±0.01	37.01±0.12	37.02±0.02	0.70	0.50

HR: heart rate; SBP: systolic blood pressure; DBP: diastolic blood pressure; RR: respiratory rate Temp: temperature; min.: minute; F: ANOVA; N: Number of patients in each group=15. NB: All values are expressed as mean± SD; *: Significant (p<0.05); LSD for repeated measure ANOVA expressed as letters (a (p<0.05) vs control group, b(p<0.05) vs control group & c (p>0.05) vs mild co- poisoned group

III.2. Biochemical assessment:

Non-significant differences among the studied groups regarding ABG parameters & cTn-1 levels were found (P>0.05). On the other hand, a significant increase in the mean values of COHB% and GPBB in CO-poisoned groups when compared with the control group was detected (p<0.05) with a significant increase in moderate CO poisoned patients when compared with mild group (p<0.05) (Table 3).

Table (3): Statistical comparison among control, mild and moderate co poisoned patients as regard mean values of Biochemical Parameters (ABG, COHB%, cTn-I, GPBB) at admission using ANOVA &LSD.

Biochemical	Control group	CO poisoned group	ANOVA test

Parameters		N=15				
					F	P
			Mild N=15	Moderate N=15		
ABG	PH	7.36±0.٠٩	7.35 ± 0.١3	7.35±0.٢١	0.04	0.96
	PO2	90.51 ± 6.32	88.96± 4.56	96.76±5.72	0.44	0.66
	PCO2	35.87 ± 5.79	35.98± 6.11	36.65±6.47	0.07	0.93
	HCO3	23.77 ± 3.85	22.67± 6.12	22.11± 6.65	0.33	0.72
COHB %		5.45±1.97	12.43±2.71[a]	15.73±4.11[b,c]	44.09	0.000*
C troponin-I (c-Tn-I)(µg/L)		0.037±0.014	0.042±0.023	0.051±0.041	0.94	0.39
GPBB(ng/ml)		12.53±2.43	27.83±6.03[d]	32.94±7.56[e,f]	51.05	0.000*

ABG: Arterial blood gases; COHB%: Carboxyhemoglobin %; GPBB: Glycogen phosphorylase BB F: ANOVA; N: Number of patients in each group=15. P>0.05: non- significant; *: Significant (p<0.05); LSD for repeated measure ANOVA expressed as letters (a (p<0.001) vs control group, b (p<0.001) vs control group, c (p<0.05) vs mild co poisoned group, d ((p<0.001) vs control group, e (p<0.001) vs control group & f (p<0.05) vs mild co poisoned group).

III.3. ECG findings:

Electrocardiogram **(**ECG) in the studied groups showed Sinus tachycardia with significant difference between control & CO–poisoned patients. Sinus tachycardia with ST-T wave changes was detected in 6.6% of CO-poisoned patients with no significant difference between mild CO-group and moderate CO-group (Table 4) (Fig. 1).

Table (4): Statistical comparison among control, mild and moderate co poisoned patients as regard ECG changes using Chi square & Fisher Exact probability tests.

ECG changes	Control group N=15	CO poisoned patients		P
		Mild N=15	Moderate N=15	
Normal	13(86.66%)	3(20%)[a]	1(6.66%)[a]	0.000*
Sinus tachycardia	2(13.33%)	11(73.33%)[a]	13(86.66%)[a]	0.000*
Sinus tachycardia +ST-T wave changes	……..	1(6.66%)	1(6.66%)	1**

*Chi –square test; **Fisher Exact Probability Test; P>0.05: non- significant; P<0.05: significant; Chi-square test expressed as letter [a] (p<0.05) vs control group

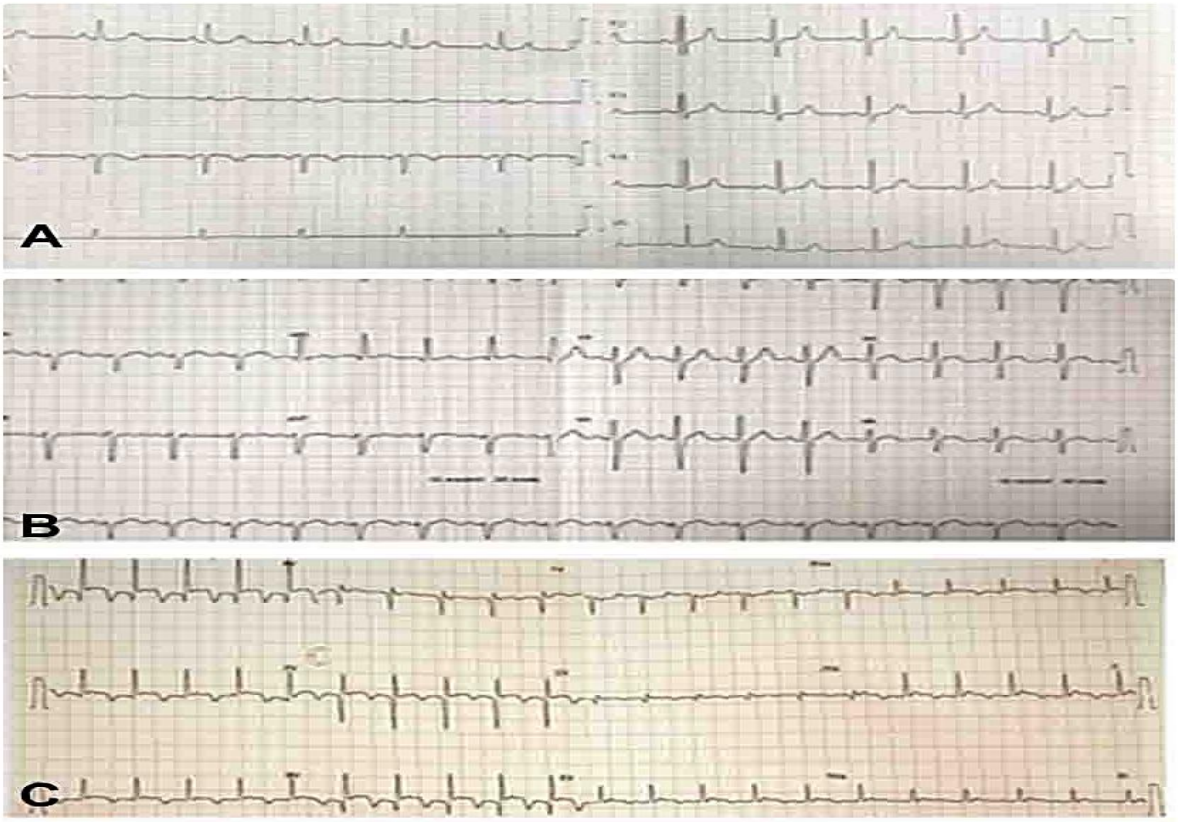

Fig. (1): ECG findings showed normal ECG (A), tachycardia (B)& tachycardia with ST wave changes (C).

III.4. Echocardiographic findings:

Regarding LVEF%; a significant decrease in moderate CO poisoned patients was detected when compared with both control & mild CO poisoned groups. On the other hand, there were no-significant differences among the studied groups regarding LVIDd and LVIDs (Table 5) (Fig. 2).

Table (5): Statistical comparison among control, mild and moderate co poisoned patients as regard mean values of LVEF%, LVEDD & LVESD using ANOVA &LSD.

ECHO findings	Control group N =15	CO poisoned group		ANOVA test	
		Mild N=15	Moderate N=15	F	P
LVEF %	67.96±4.89	65.76±5.78[a]	61.09±4.21[b,c]	6.49	0.003*
LVIDd (cm)	3.7±0.54	3.6±0.51	3.6±0.49	0.19	0.83
LVIDs(cm)	2.3±0.31	2.4±0.32	2.3±0.36	0.44	0.65

EF: ejection fraction; LVIDd: left ventricle internal dimension in diastole; LVIDs: left ventricle internal dimension in systole; P>0.05: non- significant; p<0.05: significant; LSD for repeated measure ANOVA expressed as letters (a (p>0.05) vs control group, b (p<0.05) vs control group, c (p<0.05) vs mild co poisoned group).

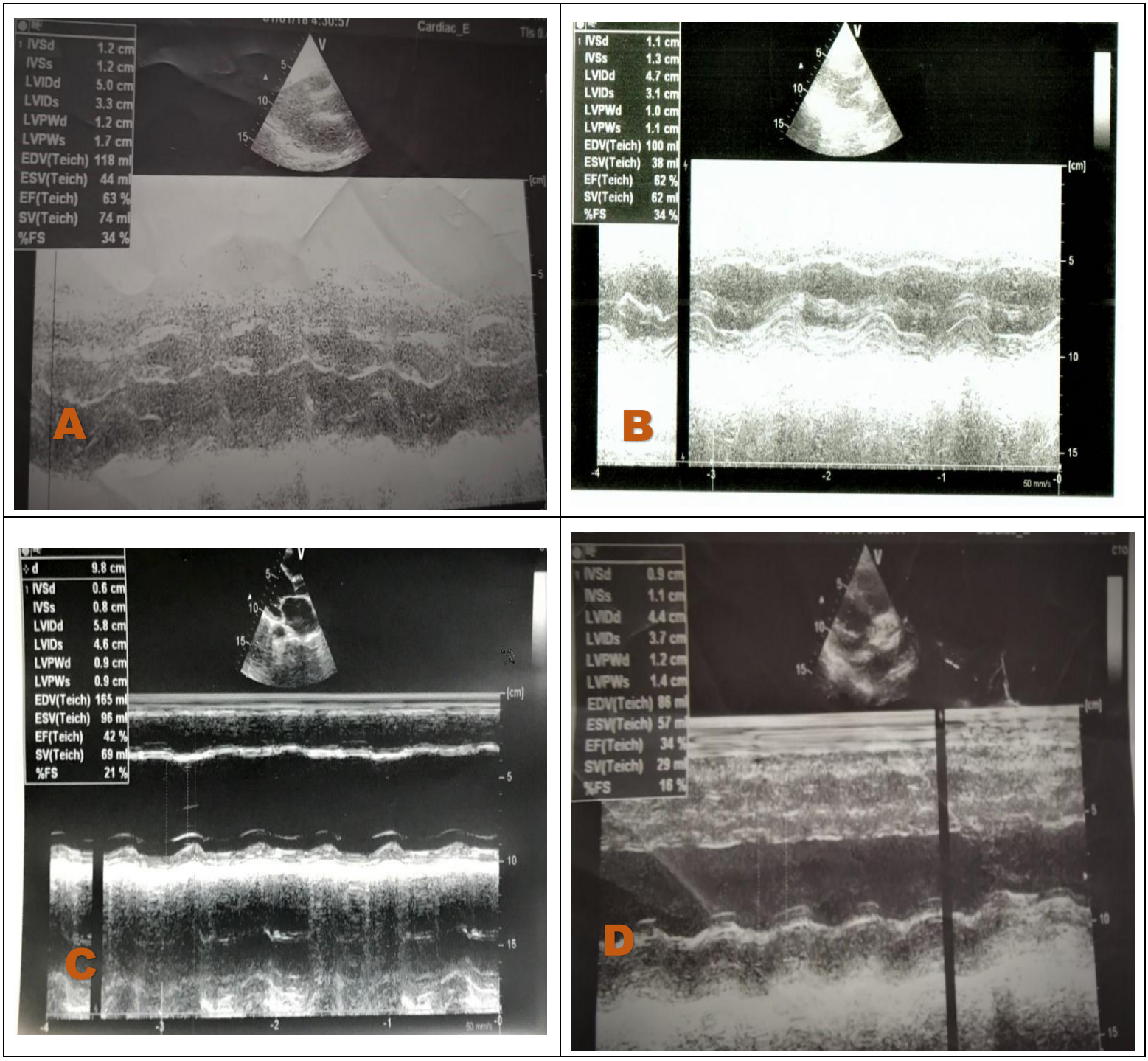

Fig.(2): Echo findings showing: A&B: Normal echo with normal EF% (63%)and (62%) respectively. C&D: Decreased EF% (42%) and 34%. EF respectively. EF%: ejection fraction, LVIDd: left ventricle internal diameter in diastole. LVIDs: left ventricle internal diameter in systole.

By studying the correlation between Glycogen Phosphorylase BB level(ng/ml) and left ventricular ejection fraction, there was a strong negative correlation meaning

that decreased ejection fraction was associated with increased GPBB level (Table 6) (Fig. 3). On the other hand, no correlation was found between GPBB (ng/ml) and COHB (%) (Table 7, Fig.4)

Table (6): Correlation between Glycogen Phosphorylase BB (GPBB) level(ng/ml) and Left ventricular Ejection Fraction (LVEF) % by Spearman correlation test.

	GPBB level(ng/ml)	
	r	P-value
LVEF (%)	-0.93	0.02*

r: Correlation coefficient. P<0.05: significant

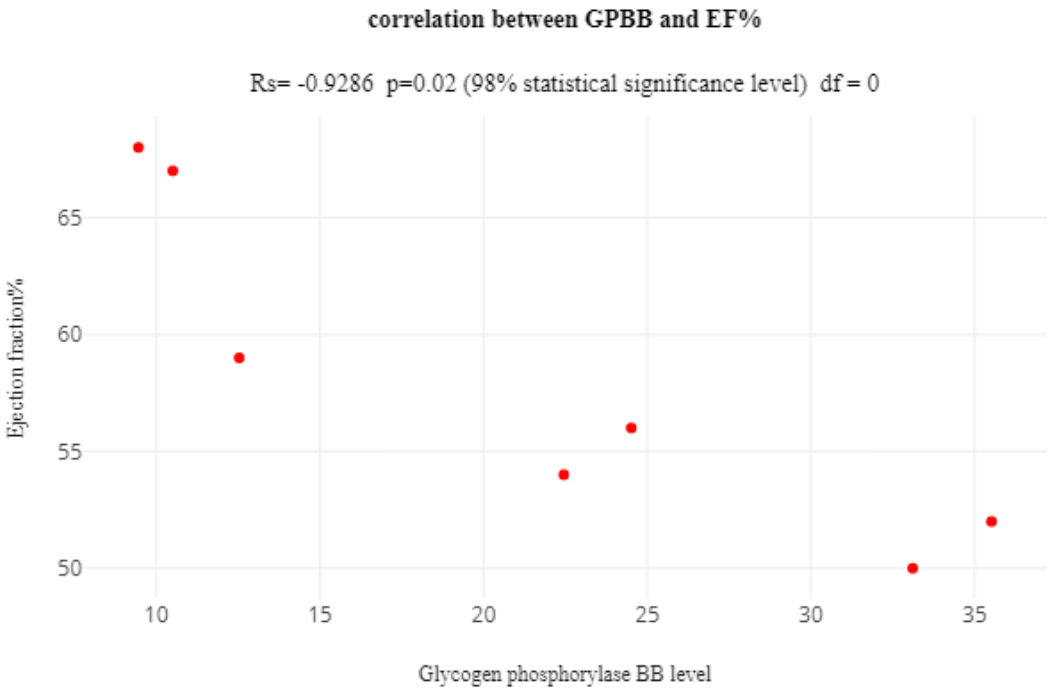

Fig.(3): Correlation between GPBB(ng/ml) and LVEF% by Spearman correlation test.

Table (7): Correlation between Glycogen Phosphorylase BB (GPBB)level(ng/ml) and Carboxy-hemoglobin (COHB) (%) by Spearman correlation test.

	GPBB level(ng/ml)	
	r	P-value
COHB (%)	0.46	0.5

r: correlation coefficient. P>0.05: non-significant.

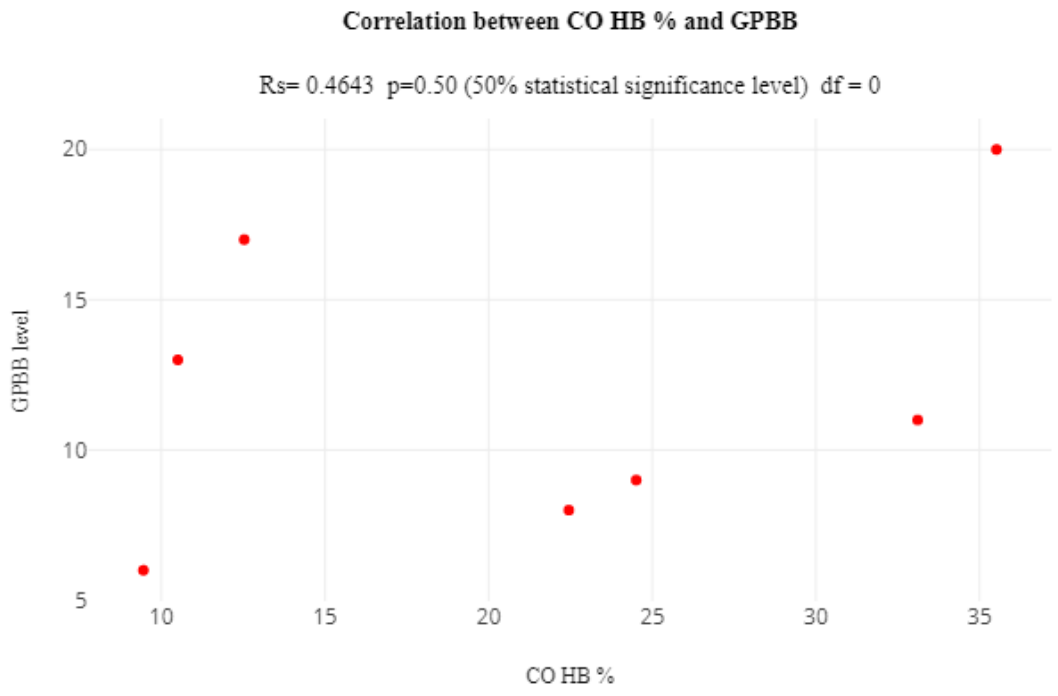

Figure (4): Correlation between GPBB level (ng/ml) and COHB %.

DISCUSSION

The heart is a critical organ in CO poisoning. Myocardial damage may be manifested clinically in several ways, from nonfatal arrhythmias to myocardial infarction or cardiomyopathy **(Mirvis & Goldberger, 2018).** Myocardial damage including necrosis is commonly seen at narcolepsy after fatal carbon monoxide poisoning. It has also been shown in experimental animals which were sacrificed after non-fatal poisoning **(Szponar et al., 2012)**.

Ischemia is a process that occurs when the demand for oxygen exceeds the available supply**.** Heart being a high-oxygen demand

organ is affected by co poisoning through impaired oxygen delivery, disrupting oxygen utilization and respiration at the cellular level. This explains myocardial ischemia in cases of CO poisoning **(Rastelli et al., 2009).**

Hila et al. (2015) reported a 26 years old male presented to the emergency department with headache, nausea and vomiting after CO exposure. Although there were no cardiac signs and symptoms, COHB level was 20% with high troponin level and echocardiography demonstrated global hypokinesia of the left ventricle. They concluded that, myocardial injury in patients with CO poisoning are difficult to identify, especially in asymptomatic patients. All patients considered to have CO poisoning should be evaluated with electrocardiograms, cardiac necrosis marker measurements, and an echocardiogram for myocardial injury regardless of the level of COHB or the absence of cardiac symptoms and signs.

In the current study, clinical examination revealed a significant increase in HR of Co poisoned patients. These results are in accordance with **Rose et al. (2017)** who reported that tachycardia was one of the most common presenting signs in cases of acute Co poisoning. However, **Deniz et al. (2017)** stated that while tachycardia is a common finding in CO poisoning, it cannot be considered a specific sign of CO related cardiac damage.

Early identification and confirmation of acute myocardial injury is essential for appropriate patient care and management in the emergency department **(Andrew et al., 2017)**. It is important to know the nature of the proteins immediately released into the blood after cardiac ischemia **(Aleksandra et al., 2017)**. Also, echocardiography has become a well-accepted, practical, safe and noninvasive method for diagnosing left and right ventricular systolic and diastolic function in the clinical setting **(Jae et al., 2011).**

Glycogen Phosphorylase is a glycolytic enzyme that plays an essential role in the regulation of carbohydrate metabolism. It catalyzes the first step of glycogenolysis, in which glycogen is converted to glucose 1-phosphate (**Lippi et al., 2013**). The clinical application of GPBB as a marker of ischemic myocardial injury is a very promising tool for increasing our knowledge of the severity of myocardial ischemic events. The properties of this new marker are most likely explained by its exclusive role in cardiac glycogenolysis, rapidly accelerating the supply of energy to the ischemic heart **(Ravi & Ramachandran, 2017).** In this study, GPBB was significantly increased while no significant increase in cTn-I was detected within the first 6 hours of CO poisoning.

These results are consistent with **Peetz et al., (2005)** who conducted a study on 61 patients presented with acute myocardial ischemia. They found that GPBB showed the highest sensitivity and specificity within the first 6 hours of myocardial ischemia, while established cardiac markers as cTn-1& creatine kinase isoenzyme MB increased later in the time course. Also, **Bozkurt et al., 2011** reported that GPBB released into circulation 2-4 hrs after onset of ischemia and return baseline level 1-2 days after cardiac ischemia.

These results are in parallel with that of **Neelima et al. (2018)** who stated that GPBB plays an essential role in the anaerobic energy metabolism during myocardial oxygen deficiency where it is released and escapes the cell through diffusion early into the blood.

Although GPBB was elevated in Co poisoned patients, no significant changes in ECG were detected except for sinus tachycardia. On the other hand, ECHO showed decreased LVEF % in moderated CO poisoned patient with signification negative correlation with GPBB level.

These findings are in agreement with that of **Teksam et al. (2010**), who found that myocardial injury may exist in CO poisoning despite the absence of abnormal ECG findings, including ST and T wave changes. On the other hand, they reported decreased EF % in five cases of non-fatal Co poisoning indicating left ventricular dysfunction.

Moreover, **Lee et al. (2011)** reported that some clinical studies have revealed that even mild CO poisoning has acute unfavorable effects on left and right ventricular function in adults with the sub-endocardial and papillary muscle areas of the left ventricle are most frequently involved. In addition, **Feng-You et al. (2015**) stated that myocardial dysfunction has been reported by echocardiography in several studies on myocardial injury related to CO poisoning.

This study revealed no correlation between the COHB & GPBB levels. This indicates that COHB level cannot be used as a predictor of myocardial ischemia in cases of mild or moderate Co poisoned patients and is poorly correlated with cardiac affection.

These results are in parallel with those of **Zahger and Slutzky (2002)** who found that myocardial injury had occurred with 20% COHB without any cardiac symptoms. Also, **Neil et al., 2012** concluded that elevated COHB level is diagnostic for CO poisoning, but does not predict the mortality or severity of clinical signs and symptoms.

Moreover, **Kirk & Rutherfoord (2017)** reported that the use of COHB level as an indicator of CO poisoning severity or to predict treatment options is limited because COHB level is affected by removal from CO exposure sources and any oxygen treatment given before assessment of its level. Additionally, people with comorbidities that make them more sensitive to the hypoxia associated with CO can present with symptoms of poisoning at either low or within the normal range of COHB level.

CONCLUSION

- Myocardial ischemia may exist during the first 6 hours of Co exposure despite the absence of obvious clinical manifestations or ECG findings and regardless of COHb level.
- GPBB increases early within the first 6 hours after myocardial ischemia. On the other hand, cardiac enzyme (cTn-I) may not increase until 6 hours after ischemia and so, the myocardium may be affected early by hypoxia but not detected by routine cardiac markers. Measuring GPBB is considered an early indicator and suitable cardiac marker in cases of CO induced myocardial ischemia since, early detection of myocardial ischemia may modify treatment to avoid later sequalae affecting ventricular function

RECOMMENDATIONS

-GPBB is recommended to be used as a routine diagnostic tool in mild & moderate Co poisoned patients as an early marker of myocardial ischemia regardless of clinical manifestations, COHb level, routine cardiac markers or ECG findings.

-Patients with elevated GPBB are recommended for follow up of the cardiac

function by ECHO to exclude ventricular dysfunction

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دور الجليكوجين فسفوريلاز في التنبؤ بسمية القلب المرتبطة بالتسمم الحاد بأول اكسيد الكربون

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مقدمة: التسمم بأول اكسيد الكربون يعد من اشهر اسباب الوفاة الناتجة عن التسمم. ان القلب معرض بشكل كبير لنقص الاكسجسن الذى يسببه غاز اول اكسيد الكربون نتيجة لاحتياجه العالي من الاكسجين. علي الرغم من ذلك فان اصابة القلب في حالات التسمم باول اكسيد الكربون قد يكون غيرواضح اكلينيكيا بسبب عدم وجود اعراض واضحة وكذلك يصعب تشخيصه لعدم وجود تغييرات محددة في رسم القلب ولذلك فان هناك حاجة ملحة للتشخيص المبكر لتاثر عضلة القلب نتيجة لنقص الاكسجين باستخدام بعض المؤشرات المبكرة مثل الجليكوجين فسفوريلازBB . **الهدف من الدراسة**: تقييم دور الجليكوجين فسفوريلاز BB في التنبؤ المبكر لتاثرعضلة القلب نتيجة التسمم باول اكسيد الكربون. **الطريقة:** اشتملت هذه الدراسة علي مجموعتين ; مجموعة ضابطة تكونت من ١٥ شخص صحيح بالغ ما بين ٢٠ و٣٥ سنة غير مدخنين وقد تم اختيارهم من رفقاء المرضي بمشتشفيات جامعة الزقازيق ومجموعة المرضي والتي اشتملت علي ٣٠ شخص بالغ ما بين ٢٠ و ٣٥ سنة تعرضوا لاول اكسيد الكربون في خلال ٦ ساعات حيث تم تقسيمهم الي مجموعة ذات تعرض بسيط ومجموعة ذات تعرض متوسط حسب الاعراض الاكلينيكية ونسبة الهيموجلوبين المحمل باول اكسيد الكربون. جميع المشتركين في الدراسة خضعوا للفحص الاكلينيكي ، رسم القلب الكهربائي، مخطط صدى القلب ،قياس انزيم التروبونين للقلبI و مستوى الجليكوجين فسفوريلاز BB في الدم . **النتائج**: وجدت زيادة ذات دلالة احصائية في سرعة نبضات القلب بين مجموعة المرضي والمجموعة الضابطة وايضا وجدت زيادة ذات دلالة احصائية في مستوى الجليكوجين فسفوريلازBB بين مجموعة المرضى والمجموعة الضابطة وقد صحب ذلك نقص ذو دلالة احصائية في كفاءة عضلة القلب الذى تم تقييمه بواسطة مخطط صدى القلب بين المجموعة ذات التعرض المتوسط لاول اكسيد الكربون والمجموعة الضابطة بينما لم توجد تغييرات ذو دلالة احصائية في مستوى انزيم التروبونين للقلب I، او في رسم القلب الكهربائي عندما تم مقارنة محموعة المرضى بالمجموعة الضابطة. **الاستنتاج:** يمكن ان يستخدم الجليكوجين فسفوريلازBB كمؤشر مبكر لتاثر عضلة القلب نتيجة لنقص الاكسجين وهذا بدوره قد يغير من خطة العلاج لتفادى حدوث مضاعفات متأخرة من شأنها ان تؤثر علي وظيفة القلب.