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Comparison of Pentraxin-3 (PTX-3), and Galectin-3 (GAL-3) with troponin I as biomarkers for early diagnosis of acute coronary syndrome

Waleed H. Osman[a], Ahmed ShawkyElserafy[b],Shadia A. Fathy[a], Marwa G.A. Hegazy[a], Riham Abdel-Hamid Haroun[a*]

[a] Biochemistry department, Faculty of Science, Ain Shams University
[b]Cardiology department, Faculty of Medicine, Ain Shams University
*Corresponding author: rihamharoun8@gmail.com

Abstract

Background:Acute coronary syndrome (ACS) continues to be a leading cause of morbidity and mortality worldwide. Limited data on galectin-3 and pentraxin-3 are found as biomarkers in the ACS population. The objective of this study was to determine the variables that are most affected by high concentrations of pentraxin-3 (PTX-3) and galectin-3 (GAL-3), and the influence they have on outcomes of all-cause mortality in patients with ACS.

Patients & Methods:One hundred and fifty patients with ST elevation myocardial infarction (STEMI) were included with 30 healthy subjects served as controls. Blood samples were drawn beforeprimary percutaneous coronary intervention (PCI) and after 12 and 48 hours. Critical changes in Troponine I as well as in PTX-3, GAL-3, lipid profile, CRP, RBS and cardiac enzymes markers were examined.

Results:Highly significant time dependent increase in serum PTX-3 and Gal-3 was observed among acute STEMI patients when compared to controls. Troponin I, PXT-3, and GAL-3 showed high sensitivity and specificity in the prediction of Acute STEMI. Sensitivities of Troponin I, PXT-3, and GAL-3 were 96%, 90% and 94% respectively, while specificities were 85%, 85% and 85% respectively. Moreover, significant increase in Troponin I, LDH, AST, CK-total, CK-MB, CRP, RBS and lipid profile were observed among the acute STEMI patients as compared to controls.

Conclusion:We can conclude that both PTX-3 and GAL-3 could be excellent biomarkers of CVDACS. PTX-3 and GAL-3 plasma levels rise rapidly in acute myocardial infarction, reflecting the extent of tissue damage and predicting the risk of mortality

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Keywords: Acute coronary syndrome (ACS); Acute myocardial infarction (AMI); pentraxin-3 (PTX-3); galectin-3 (GAL-3)

1. Introduction

About 17 million people die every year from cardiovascular diseases (CVD),as reported to the World Health Organization (WHO), with important implications in terms of social costs and quality of life ***(Ristagno et al., 2019)***.

Acute coronary syndrome (ACS); which involves ST-segment elevated myocardial infarction (STEMI), non-ST-segment elevated myocardial infarction (NSTEMI), and unstable angina pectoris (UAP); is among the leading causes of mortality around the world ***(2)***and usually resulted from atherosclerotic plaque rupture or sometimes from superficial plaque erosion with subsequent thrombus formation which may cause myocardial necrosis and elevated serum levels of myocardial enzymes ***(Mashayekhi et al., 2018)***.

Successful and early myocardial reperfusion after an acute myocardial infarction (AMI) is the most effective strategy for salvaging the myocardium and improving clinical outcomes ***(4).*** Primary percutaneous coronary intervention (pPCI) with stent implantation and complete revascularization of the affected coronary vessel is the preferred method of reperfusion for patients with AMI***(Al-Salama andHashmi, 2018).*** Primary percutaneous coronary intervention (pPCI) with stent implantation and complete revascularization of the affected coronary vessel is the preferred method of reperfusion for patients with AMI***(Mehta et al., 2019).***

Biomarkers are substances derived from organs, which can be measured and evaluated as indicators of normal biological processes, pathogenic process, or pharmacological response to a therapeutic intervention ***(Ahmad and Sharma, 2012 & Sato and Fujiwara, 2012)***. Several molecules have been reportedas valuablemarkers in improving the diagnosis and prognostic classification of patients with AMI***(Val Martin et al., 2015).***

Cardiac enzymes have long been used as frontline diagnostic tools in the detection of myocardial injury caused by myocardial ischaemia. However, the most commonly cardiac enzymes biomarkers to be used; including creatine kinase (CK) and its myocardial fraction CK myocardial band (MB), aspartate aminotransferase (AST), and lactate dehydrogenase (LDH), are limited in their ability to detect myocardial injury by short diagnostic windows, limited sensitivities, and lack of specificity because of their presence in skeletal muscle ***(Karki et al., 2015&Pareek et al., 2017)***. Therefore, WHO recommended the panel of CK, AST and LDH for the diagnosis of AMI.However, the assessment of the cardiac biomarkers was revolutionized in after the development of immunoassays ***(Mythil and Malathi, 2015)***.With the introduction of new more sensitive and cardiac-specific biochemical markers of myocardial injury, such as cardiac troponin I (cTnI), smaller myocardial injuries could be recognized. cTnI currently considered to be the gold standard biomarker test for AMI. Moreover, cTnI measurements by a new

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generation of high-sensitivity cTnI assays could be helpful for long-term risk stratification of different patient groups, including patients with MI or coronary vascular diseases ***(Agnello et al., 2017&Katus et al., 2017)***.

Pentraxin-3 (PTX-3) is an inflammatory biomarker from the C-reactive protein (CRP) family which has been shown to be expressed at the site of atherosclerotic plaques and cardiac myocytes(**George etal., 2015).**Particularly, the expression of PTX-3 is increased in human atherosclerotic plaques **(Chu et al., 2019).** Galectin-3 (GAL-3) has emerged as a promising, novel risk marker in patients with cardiovascular disease **(Singsaas et al., 2016)**. Gal-3 is a 29 to 35 kDa protein, member of a β-galactoside binding lectin family expressed in cardiac cells which has emerged as a potential regulator of physiological and pathological processes including inflammation and fibrosis ***(Ibarrola et al., 2019&Tsai et al., 2012)*.**

A number of studies have investigated the role of PTX-3 and GAL-3 in AMI. However, there is a dearth of data in the ACS population, giving us the impetus to perform this study. We thus sought to determine the variables that are most affected by high concentrations of PTX-3 and GAL-3, and the influence they have on outcomes of all-cause mortality in ACS patients.

2. Patients and Methods

2.1.Study design and participants:

The study protocol was approved by the ethics committee of Faculty of Medicine, Ain Shams Universityand written informed consent was given by all patients. A total of 150 patients with acute STEMI treated by pPCI were enrolled in the study between April 2018 and June 2019. All were successfully revascularized achieving normal coronary blood flow during the PCI procedure.Inclusion criteria were as follows: age ≥18 years, ischaemic symptoms since <12 hours, eligible for pPCI, ECG with ST-segment elevation. Thirty healthy subjects without current or past medical disorders were included and served as controls. All study subjects were investigated for age, gender, body mass index (BMI), smoking history, the presence or absence of hypertension, diabetes mellitus, and dyslipidemia.Laboratory tests were conducted at admission, includinga blood levels of random blood sugar, CRP, triglyceride, high-density lipoprotein cholesterol, and LDL cholesterol levels in all study subjects.

2.2.*Blood sampling and biochemical analysis*:

When a diagnosis of acute STEMI was established and a decision to perform pPCI was made, blood samples were obtained from peripheral vein before starting intervention (zero time), and 12 hand 48 h(2nd day) after intervention. Blood samples for troponin I (TnI), MB fraction of creatine kinase (CK-MB), creatine kinase total (CK-Total), Lactate dehydrogenase (LDH), aspartate aminotransferase (AST), PTX-3 and GALl-3 determination were allowed tocoagulate at room temperature for 30 min, and thencentrifuged at 4000 rpm for 10 min to obtain serumsamples. Serum PTX-3 was

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measured using Human Pentraxin-related protein PTX3, ELISA Kit (cat# E0277h) (EIAab, Inc., China) according to the protocol of manufacturer. Serum GAL-3 was determined using Human Galactin-3, Gal-3 ELISA Kit (cat# E0497h) (EIAab, Inc., China) according to the protocol of manufacturer.

2.3.*Statistical analysis*

Statistical analysis was performed using the statistical package for social sciences, version 20.0 (SPSS Inc., Chicago, Illinois, USA). Quantitative data were expressed as mean± standard deviation (SD). Qualitative data were expressed as frequency and percentage.

The corresponding parameters in the patients group at baseline were compared by means of independent-samples T-test. Results after 48 h of pPCI were compared with their own baseline values (paired-sample *t*-tests). Correlations between the changes (as mean± standard deviation) in the different parameters with troponine I were carried by Pearson correlation coefficient analysis. The level of statistical significance was set at $P < 0.05$.

3. Results

3.1. Clinic-biological Characteristics

The current study included 150 patients (92 males and 58 females) with Acute STEMI treated by pPCI; with mean age 48.40±10.82 years, and mean BMI 24.24±3.07. The comparison between control group and patients group according to baseline characteristics are summarized in **table (1)**. As the study was designed to match the age, gender and BMI between cases and controls, no statistically significant difference was observed for these parameters (P >0.05).

We compared the mean level of laboratory investigations between patients group and control group regarding troponin I, LDH, CRP, CK-Total, CK-MB and AST, and the mean levels were significantly higher in patients group in all investigations (Table 2). Our results revealed that there is a significant (P<0.05) increase in the serum troponin I (0.55±0.76) as compared to controls (0.02±0.01). Results of different laboratory investigations showed that a highly significantly (P<0.001) increase in LDH, AST, CK-total, CK-MB, CRP, RBS, Cholesterol, Triglyceride, HDL, LDL, and VLDL were observed among the acute STEMI patients as compared to controls. Also, a highly significant (P<0.001) increase was found in the serum PTX-3 (5.69±1.37), and GAL-3 (13.39±1.80) when compared to controls (2.68±0.61& 7.16±0.95; respectively) (Table 3). On comparing the mean levels of PTX-3 and GAL-3 at zero time, after 12 hrs.and48 hrs. there was highly significant increase with p- value < 0.001**. (Table 4-5).**

3.2.Correlation of PTX-3 and GAL-3:

Data recorded in **table (6)** showed the correlation of PTX-3 and GAL-3 with the different measured parameters, results revealed that there is a highly significant positive correlation between PTX-3 with Troponin I, AST, CK-total, CK-MB and CRP (p value <

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0.001);while there is significant negative correlation between PTX-3 with Age, diastolic BP and RBS. While for GAL-3; it was found that it has a significant positive correlation with Troponin I, AST, CK-total, CK-MB and CRP, and significant negative correlation with RBS.

3.3.Receiver operating characteristic (ROC) curves analysis:

ROC curve analysis was used to calculate the area under the curve (AUC) of Troponin I, PXT3, and GAL3 in order to evaluate the sensitivity and specificity of these factors in the prediction of Acute STEMI. In discrimination of myocardial infarction patients; results of the current study showed that AUC for troponin Iis 0.922(cut off value **>0.03)** with sensitivity of 96% and specificity of 85%, PTX- 3is 0.892(cut off value **>3.9)**with sensitivity of 90% and specificity of 85%, and GAL-3is 0.961(cut off value **>8.5)**with sensitivity of 94% and specificity of 85%; as shown in **table (7) & fig (1)**.

4. Discussion

Acute coronary syndrome (ACS) describes the range of myocardial ischemic states that includes unstable angina (UA), non-ST elevated myocardial infarction (NSTEMI), or ST-elevated myocardial infarction (STEMI). The diagnosis and classification of ACS is based on a thorough review of clinical features, including electrocardiogram (ECG) findings and biochemical markers of myocardial necrosis (***Smith et al., 2015)***.Atherothrombotic events such as myocardial infarction and ischemic stroke are major causes of morbidity and mortality worldwide. Atherosclerotic plaque disruption and subsequent thrombus formation are considered to be critical processes involved in the onset of atherothrombotic events. However, autopsy studies have revealed that these processes do not always result in thrombotic occlusion followed by acute symptomatic events. These findings indicate that thrombus propagation is also critical to the onset of clinical events (***Asada et al., 2020***). Therefore; in this study, we hypothesized that the serum PTX-3 and GAL-3 levels might be useful for the early diagnosis of atherothrombosis and might be an easy way to diagnose the disease and determine its intensity. Also we investigated whether or not there are any associations of the PTX-3 and GAL-3 levels with the presence and intensity of CAD in a group of stable patients who were suspected of having CAD.

Recent researches investigated the role of inflammatory mediators such as PTX-3 and GAL-3 in cardiovascular events due to their contribution in the formation and rupturing of the atheromatous plaques***(Li et al., 2019; Ristagno et al; 2019;Stanojevic et al, 2019;Nerkiz et al.,2015 &Helseth et al., 2014).***Our study showed correlation between the levels of these inflammatory biomarkers and acute myocardial infarction.

Both GAL-3 and PTX-3 increase during the inflammatory process, our results showed increasing levels of both biomarkers during 48 hours of admission and diagnosis of AMI

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to the patients group, as evident by ***Bivona et al, (2016) who*** indicates that GAL-3 level increase after the occurrence of AMI and decrease only after 5 days**.**

The analysis of the data obtained from this study revealed that the serum PTX-3 and GAL-3 levels were higher in the patients with Acute STEMI in comparison to the patients with a normal coronary vessel anatomy.

Our results agree with a study by ***Altay et al,*** (2016) which showed that PTX-3 levels increase in ACS and AMI and can be used in the diagnosis of AMI. Another study correlating the level of GAL-3 and the insult of AMI was conducted by ***Alturfan et al***, (2014) that showed significant rise of GAL-3 level in AMI patients. The results of the mentioned studies about the two biomarkers are compatible with our results.

On the other hand, this study is limited by the small sample size to further confirm the value and accuracy of the studied biomarkers in the diagnosis of AMI, also the short duration of follow-up didn't allow the proper study of the prognostic value of these markers. Another limitation is that the normal range of these biomarkers was not investigated for clinical practice.

So we recommend further studying and clinical investigation of the diagnostic and prognostic impact of these biomarker with larger and multi-centric studies which can give a more detailed results for better clinical practice and updated guidelines. Also more cost-efficiency studies to determine the convenience of the application of these laboratory investigations in clinical practice.

Conclusion:

In conclusion, PTX-3 and GAL-3 represent novel and accurate laboratory alternatives to conventional cardiac enzymes such as Troponin I with accuracy and predictive value very similar to the standard, which suggest the possibility of the implementation of these biomarker in the clinical practice as new investigations to diagnose acute myocardial infarction.

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Baseline characteristics	**Control group** ***(n=30)*** Mean±SD	**Patients Group (n=150)** Mean±SD	**p-value**

Table (1): Comparison between control group and patients group according to baseline characteristics.

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Sex Female Male	 12 (40.0%) 18 (60.0%)	 58 (38.7%) 92 (61.3%)	 0.913
Age (years)	46.80±6.69	48.40±10.82	0.192
BMI [wt/(ht)^2]	22.50±1.96	24.24±3.07	0.254
Systolic BP (mmHg)	116.50±6.90	168.69±7.95	<0.001**
Diastolic BP (mmHg)	75.00±6.88	95.28±3.39	<0.001**

*p-value>0.05 NS; *p-value <0.05 S; **p-value <0.001 HS*

Laboratory investigations	**Control group** ***(n=30)*** Mean±SD	**Patients Group (n=150)** Mean±SD	**t-test**	**p-value**
Troponin I (ng/mL)	0.02±0.01	0.55±0.76	9.542	0.003*
LDH (U/L)	91.70±11.97	566.76±121.07	30.779	<0.001**
AST (U/L)	21.75±7.00	41.80±20.09	19.177	<0.001**
CK-total (U/L)	69.25±16.56	322.59±209.65	28.929	<0.001**
CK-MB (U/L)	13.40±4.38	54.98±26.57	48.237	<0.001**
CRP (mg/dL)	3.21±1.45	11.52±10.27	12.955	<0.001**
RBS (mg/dL)	87.85±11.72	190.07±94.05	23.345	<0.001**
Cholesterol (mg/dL)	153.28±19.10	200.93±35.73	32.888	<0.001**
T.G (mg/dL)	87.70±10.21	157.48±51.63	35.876	<0.001**
HDL (mg/dL)				

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	50.92±4.69	43.46±5.98	26.639	<0.001**
LDL (mg/dL)	84.82±21.98	125.98±34.68	25.335	<0.001**
VLDL (mg/dL)	17.54±2.04	31.49±10.33	35.877	<0.001**

Table (2): Comparison between control group and patients group according to laboratory investigations.

t-Independent Sample t-test;

**p-value <0.05 S; **p-value <0.001 HS*

Table (3): Comparison between control group and patients group according to PTX-3 and Gal-3 markers at zero time.

Markers at zero time	**Control group *(n=30)*** Mean±SD	**Patients Group (n=150)** Mean±SD	**t-test**	**p-value**
PTX-3 (ng/mL)	2.68±0.61	5.69±1.37	91.682	<0.001**
Gal-3 (ng/mL)	7.16±0.95	13.39±1.80	22.309	<0.001**

*t-Independent Sample t-test; **p-value <0.001 HS*

Table (4): Difference of PTX-3 level over several periods among patients group.

PTX-3 (ng/mL)	**Mean±SD**	**Mean Diff.**	**t-test**	**p-value**
Zero time	5.69±1.37			
After 12 hrs	9.63±2.33	3.94	-23.037	<0.001**
After 48 hrs	11.95±2.44	6.26	-31.149	<0.001**

*t-Paired Sample t-test; **p-value <0.001 HS*

Table (5): Difference of GAL-3 level over several periods among patients group.

Gal-3 (ng/mL)	**Mean±SD**	**Mean Diff.**	**t-test**	**p-value**
Zero time	13.39±1.80			
After 12 hrs	16.81±1.77	3.42	-26.326	<0.001**
After 48 hrs	21.41±2.73	8.02	-37.308	<0.001**

*t-Paired Sample t-test; **p-value <0.001 HS*

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Table (6): Correlation between PTX-3 and GAL-3 with all parameters at zero time.

Patients Group	PTX-3 (ng/mL)		Gal-3 (ng/mL)	
	r	p-value	r	p-value
Age (years)	-0.407	<0.001**	-0.123	0.291
BMI [wt/(ht)^2]	-0.083	0.478	-0.010	0.931
Systolic BP (mmHg)	-0.121	0.300	-0.130	0.267
Diastolic BP (mmHg)	-0.271	0.018*	0.141	0.227
Troponin I (ng/mL)	0.470	<0.001**	0.255	0.027*
LDH (U/L)	-0.117	0.320	0.044	0.707
AST (U/L)	0.603	<0.001**	0.329	0.004*
CK-total (U/L)	0.559	<0.001**	0.281	0.015*
CK-MB (U/L)	0.567	<0.001**	0.312	0.006*
CRP (mg/dL)	0.707	<0.001**	0.322	0.005*
RBS (mg/dL)	-0.347	0.002*	-0.390	<0.001**
Cholesterol (mg/dL)	0.043	0.714	-0.196	0.092
T.G (mg/dL)	-0.083	0.481	0.047	0.688
HDL (mg/dL)	0.191	0.101	0.011	0.928
LDL (mg/dL)	0.036	0.759	-0.218	0.060
VLDL (mg/dL)	-0.083	0.481	0.047	0.688

r-Pearson Correlation Coefficient

*p-value>0.05 NS; *p-value <0.05 S; **p-value <0.001 HS*

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Table (7): Sensitivity, specificity and cut-off value of Troponin I, PTX-3 and Gal-3 in AMI patients.

Markers	Cut-off	Sen.	Spe.	PPV	NPV	Accuracy
Troponin I (ng/mL)	**>0.03**	96%	85%	96%	85%	0.922
PTX-3 (ng/mL)	**>3.9**	90%	85%	95%	68%	0.892
Gal-3 (ng/mL)	**>8.5**	94%	85%	96%	85%	0.916

Fig. (1): Receiver-operating characteristic (ROC) curve for prediction of myocardial infarction patients using the troponin I, PTX-3 and Gal-3.