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Correlation of Admission Level of Galectin-3 and Cardiac Remodeling after Primary Percutaneous Coronary Intervention

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ABSTRACT

Background: Galectin-3, a biomarker associated with cardiac remodeling, has been linked to poor outcomes in patients with acute STEMI. Understanding its predictive capacity after primary percutaneous coronary intervention [PPCI] is critical for improving patient management.

The aim of the work: This study investigates the correlation between admission levels of Galectin-3 and the occurrence of cardiac remodeling in patients diagnosed with acute STEMI following PPCI.

Patients and Methods: A total of 90 consecutive patients presenting with chest pain and diagnosed with acute STEMI were included. Patients were divided into two groups based on remodeling status—Group I [non-remodelers, n=58] and Group II [remodelers, n=32]—defined by an increase in left ventricular end-diastolic volume [LVEDV] of more than 20% from baseline echocardiography. Cardiovascular metrics, including LVEDV, left ventricular end-systolic volume [LVESV], ejection fraction [EF%], and serum levels of Galectin-3, troponin, and CKMB were assessed at baseline and after three months.

Results: Significant differences were observed between groups in LVEDV, LVESV, and EF% after three months [p=0.001]. Galectin-3 levels were significantly higher in remodelers [mean 16.59] compared to non-remodelers [mean 12.62, p<0.001]. Troponin and CKMB levels also differed significantly between groups [p<0.001]. TIMI flow showed significant differences at both baseline and post-PPCI [p=0.014 and p= 0.003, respectively]. Pearson's correlation coefficient between Galectin-3 levels and cardiac remodeling was 0.84, indicating a strong positive relationship, with an AUC of 0.997 for predictive capacity.

Conclusion: Admission levels of Galectin-3 are strongly correlated with cardiac remodeling post-PPCI in STEMI patients. This biomarker could serve as a valuable predictor for assessing risk and managing treatment strategies in this population.

Keywords: Galectin-3; Cardiac Remodeling; Primary PCI; Left Ventricle; Myocardial Infarction.

INTRODUCTION

Galectin-3 is a β-galactoside–binding lectin, which received recent research interest. It is upregulated in different conditions in murine models. These conditions included hypertensive heart disease, cardiomyopathies, myocarditis, and left ventricle [LV] hypertrophy, aortic stenosis and depressed function of the LF [1-3].

In addition, galectin-3 is increased in humans with acute decompensated and chronic heart failure. In chronic heart failure [CHF], the values of galectin-3 is correlated with the New York Heart Association [NYHA] higher grades. In addition, it predicts the poor outcome [4, 5].

Furthermore, galectin-3 is correlated with echocardiographic parameters, mainly diastolic function and performance of right ventricle [RV] in patients with dyspnea who had or had not acute decompensated heart failure. However, data regarding the relation of galectin-3 to the detailed cardiac structural data are deficient [6].

Moreover, studies addressing the relation or potential role of galectin-3 to LV remodeling after acute myocardial infarction are no sufficient [6, 7]. Thus, the current work was designed to address the relation between galectin-3 plasma levels and LV cardiac remodeling in patients submitted to primary PCI after acute MI. In addition, parameters of cardiac functional and structural data were correlated with plasma galectin-3.

PATIENTS AND METHODS

This prospective study was conducted in Cardiology Department, Al-Azhar University Hospitals from June 2022 to June 2023. We included 90 consecutive patients admitted to the coronary care unit with chest pain and diagnosed as acute STEMI based on electrocardiographic to study the effects of Galectin 3 on LV remodeling after AMI.

Patients were retrogradely divided into two groups based on the occurrence of remodeling, defined as a left ventricular end-diastolic volume [LVEDV] increase of more than 20% from baseline echocardiography [8]. This resulted in Group I, consisting of non-remodelers [n=58], and Group II, comprising remodelers [n=32].

Inclusion and exclusion criteria: we included patients with acute STEMI, which was defined according to **Thygesen *et al.*** [9]. Additionally, patients had to be successfully revascularized following primary percutaneous coronary intervention [PPCI]. On the other side, we excluded patients with history of previous MI, patients presenting with symptoms of STEMI for more than 24 hours, renal failure and liver cell failure, congestive heart failure, planned coronary artery bypass surgery, and any contraindications to PPCI, such as dye allergy.

Ethical considerations: A written consent was obtained from every patient after explanation of the procedure, medical research and ethics committee approved the study.

All patients were clinically evaluated by full history taking, detailed general and local cardiac examination, electrocardiography and echocardiography. In addition, standard laboratory work-up [complete blood count, blood sugar, lipid profile, renal function tests, prothrombin time, partial thromboplastin time and INR] and measurement of cardiac enzymes were performed in addition to measurement of galectin-3. Then all patients were treated by PPCI.

Galectin-3 measurement was performed by an enzyme-linked immunosorbent assay kits developed by BG Medicine, Inc, Waltham, MA. It quantitatively assesses galectin-3 concentrations on plasma samples. Blood samples were collected into EDTA and aprotinin containing tubes. Then centrifuged at 2000 g for 15 minutes and plasma was collected and stored at -80 ºC until analysis. The limit of detection was 1.13 ng/mL.

Electrocardiography was recorded immediately after admission and on 6 hours intervals after admission and each 3 hours after PPCI to check dynamic changes. Papers speed of 25 mm/s with amplification of 10 mm/mv was used to complete ECG and ST-segment elevation was recorded 40 ms after the J-point and the total number of ST-segment deviations was recorded before and 60 minutes after intervention. The ST-segment resolution was graded into complete [more than 70%], partial [30 to 70%] or absent [lower than 30%].

The transthoracic Echocardiography was done at rest using available ultrasound system [HP SONOS, GE Vivid E9 and Philips envisor] and a 1.6 – 4 MHz phased array transducer, while the patient was in the left lateral decubitus position with left arm elevated to the level above the shoulder height. The left ventricle end-diastolic volume [LVEDV], LV end systolic volume [LVESV],

LV stroke volume [SV] and LV ejection fraction [LVEF] were recorded in the apical-4 chamber and 2-chamber views. Ejection fraction was calculated by the modified biplane method of Simpson. Echocardiography was performed before and at the first day and at the end of the third month after intervention. Cardiac modeling was calculated from the LV displacement and regional deformation [thickening or shortening]. However, wall motion of each cardiac segment was calculated and the LV wall motion score index as an average of all segments scores was computed. The following scoring system was used: [1] normal or hyper-kinetic, [2] hypokinetic [reduced thickening], [3] akinetic [absent or negligible thickening, e.g., scar], and [4] dyskinetic [systolic thinning or stretching, e.g., aneurysm]. An aneurysm is a morphologic entity that demonstrates focal dilatation and thinning [remodeling] with either akinetic or dyskinetic systolic deformation. WMSI Calculated by dividing the sum of the wall motion scores of each segment by 17.

PPCI [mechanical reperfusion] was performed as early as possible via percutaneous femoral or radial artery puncture. The coronary angiography was used to recognize the site of recent occlusion. Then, a metal wire was introduced and advanced past the thrombus, where a balloon catheter [with or without stent] was positioned at the occlusion site and inflated to restore antegrade flow. The TIMI flow was grades as the following: TIMI 0 flow is absence of antegrade flow; TIMI 1 flow is faint antegrade coronary flow with incomplete filling of the distal coronary bed; TIMI 2 flow is reduced flow but with complete filling of the distal territory; TIMI 3 flow is normal brisk flow.

The technical success was said to have occurred if at the end of the procedure there was no significant obstructive disease [<50% residual angiographic stenosis] within 10 mm either side of the treated lesion or there was brisk flow [TIMI III] in all major branches [>1.5 mm in diameter] involved in the treated lesion. In addition, any periprocedural complications were recorded. These included distal embolization of the thrombus, slow flow or no flow of the main vessel, new thrombus formation, transient vessel closure, coronary perforation, ventricular fibrillations, complete heart block or bradycardia required temporary pacing, neurological complications due to ischemia, allergic reactions, cardiac tamponade need draining, false aneurysm intracardiac bleeding, stent thrombus, infection, retroperitoneal bleeding, acute renal failure, pulmonary edema, death on the table and catheter tip dissection.

Statistical analysis: All statistical analyses were performed by the statistical package for social science [SPSS] for windows, version 18 [IBM®SPSS® Inc., Chicago, USA]. Continuous variables are presented as mean ± standard deviation [SD], and categorical variables as frequencies and percentages. Comparison of categorical and continuous variables was done using chi-square and independent sample "t" test, respectively. To assess the strength and direction of relationships between variables, we utilized Pearson correlation coefficients. The diagnostic performance was evaluated by receiver operating characteristic [ROC] curve analysis, calculating the area under the curve [AUC] to determine sensitivity and specificity. A significance level of $p < 0.05$ was accepted for all tests.

RESULTS

The age of non-remodelers ranged from 36 to 71 years, with an average of 53.5, while remodelers were older, aged 42 to 71 years with a mean of 60.94, showing a significant difference [p=0.001]. The Body Mass Index [BMI] in Non-remodelers averaged 28.9 [range 25.4 to 33.4] compared to 31.39 [range 23.8 to 34.5] in Remodelers, also significant [p=0.001]. Sex distribution showed no significant difference [p= 0.5]. Notably, risk factors such as smoking, diabetes mellitus [DM], dyslipidemia, and family history were significantly different [p=0.001], except for hypertension [p=0.15]. Heart rates and blood pressures were higher in the Remodelers group, with significant differences in heart rate [mean 85.56 vs. 72.7], systolic blood pressure [SBP], and diastolic blood pressure [DBP] [all p=0.001]. Additionally, there were significant differences regarding infarction location [p= 0.001] [Table 1].

After three months, there were significant differences in left ventricular end-diastolic volume [LVEDV] and left ventricular end-systolic volume [LVESV] between the studied groups [p=0.001]. Both groups also showed significant differences in ejection fraction [EF%] at baseline and after three months [p=0.001]. However, the baseline wall motion score showed no significant difference [p=0.6], and follow-up scores had no significant difference either [p=0.4]. The scores were 1.91 for Remodelers and 2.01 for Non-remodelers at baseline, and 2.68 vs. 1.9 after three months, respectively [Table 2].

In the study, Galectin-3 levels in the Non-remodelers group ranged from 11 to 16, with a

mean of 12.62, while in the Remodelers group, the range was 12 to 18, with a mean of 16.59, showing a statistically significant difference [p< 0.001]. Troponin levels also differed significantly, with Non-remodelers averaging 3.42 [range 2.6 to 4.3] and Remodelers averaging 4.21 [range 3.1 to 5] [p<0.001]. For CKMB, Non-remodelers had a mean of 48.50 [range 37 to 59] compared to 56.61 [range 38 to 66] in Remodelers, again showing a significant difference [p<0.001] [Table 3].

Regarding TIMI flow, there was a significant difference between the two studied groups both at baseline and post-PPCI [p = 0.014 and p = 0.003, respectively] [Table 4].

Pearson's correlation coefficient [r] between the admission level of Galectin-3 and cardiac remodeling after primary PCI was 0.84, indicating a strong positive relationship [Table 5].

Regarding the baseline Galectin-3 level, the AUC was 0.997, the cutoff value was 14.5, the sensitivity was 93.8%, and the specificity was 100% [Table 6, Figure 1].

Table [1]: Demographic and clinical data of the study groups

		non-Remodeling	Remodeling	P value
Age [years]	Mean ± SD	53.50 ± 6.84	60.94 ± 7.08	**0.001***
Body mass index [Kg/m^2]	Mean ± SD	28.90 ± 2.12	31.39 ± 2.89	**0.001***
Sex, n [%]	Male	32 [44.8%]	20 [37.5%]	0.5
	Female	26 [55.2%]	12 [62.5%]	
Risk factors	Smoking	44 [75.9%]	9 [81.3%]	**0.001***
	Hypertension	39 [67.2%]	26 [81.3%]	0.15
	DM	14 [24.1%]	26 [81.3%]	**0.001***
	Dyslipidemia	12 [20.7%]	23 [71.9%]	**0.001***
	FH	14 [24.1%]	24 [75%]	**0.001***
Heart rate [Bpm]	Mean ± SD	72.71 ± 10.49	85.56 ± 12.38	**0.001***
Systolic blood pressure [mmHg]	Mean ± SD	126.60 ± 20.15	149.06 ± 21.61	**0.001***
Diastolic blood pressure [mmHg]	Mean ± SD	77.31 ± 9.73	89.25 ± 12.39	**0.001***
Infarction location	Anterior wall	21 [36.2%]	29 [90.6%]	**0.001***
	Non anterior wall	37 [63.8%]	3 [9.4%]	

Table [2]: Comparison of ECHO data between studied groups

		Non-Remodeling		Remodeling		P value
		Mean	**SD**	**Mean**	**SD**	
LVEDV [mL]	Baseline	102.29	8.32	103.16	10.49	0.66
	3 months follow up	108.02	14.44	135.59	13.20	**0.001***
LVESV [mL]	Baseline	45.28	13.08	64.59	9.70	**0.001***
	3 months follow up	46.64	11.33	80.28	11.79	**0.001***
EF [%]	Baseline	56.38	8.94	37.22	8.94	**0.001***
	3 months follow up	57.14	7.39	39.00	7.32	**0.001***
WMSI	Baseline	2.01	4.64	1.59	0.26	0.6
	3 months follow up	1.91	3.65	2.68	5.54	0.4

Table [3]: Laboratory results among the study groups

	Non-remodeling		Remodeling		P value
	Mean	**SD**	**Mean**	**SD**	
Galectin-3	12.62	0.81	16.59	1.10	**0.001**
Troponin	3.42	0.40	4.21	0.52	**0.001**
Creatine kinase-MB	48.50	5.19	56.61	6.20	**0.001**

Table [4]: Comparison of TIMI flow between studied groups

TIMI flow		Non-Remodeling		Remodeling		P value
		No.	**%**	**No.**	**%**	
Initial	0	43	74.1	13	40.6	**0.014***
	1	5	8.6	4	12.5	
	2	6	10.3	8	25	
	3	4	6.9	7	21.9	
Post PPCI	1	0	0	1	3.1	**0.003***
	2	0	0	5	15.6	
	3	58	100	26	81.3	

Table [5]: Correlation between Baseline Galectin-3 and LV remodeling after PCI

	r	P value
Baseline Galectin-3	0.84	**0.001***

Table [6]: Sensitivity and specificity of Galectin-3 in prediction of LV remodeling

	Diagnostic parameters			
	AUC	**Cutoff value**	**Sensitivity**	**Specificity**
Galectin-3	0.997	14.5	93.80%	100%

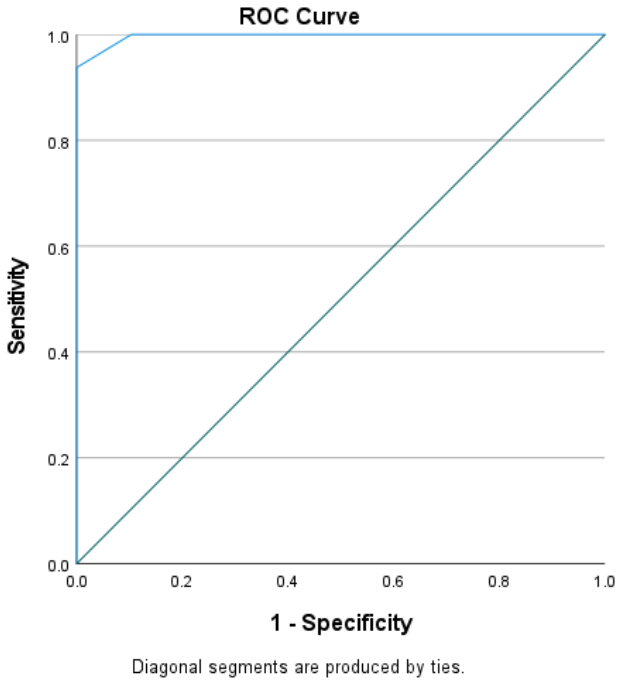

Figure [1]: Receiver operating characteristic curve for Baseline Galectin-3 level

DISCUSSION

The remodeling of the LV describes a set of changes in the structure and function of LV after acute MI. it is associated with a progressive increase in LVESV and LVEDV. These changes can lead to the deterioration of the LV systolic function measured by left ventricle EF [LVEF] with further complications of the cardiovascular system [10, 11].

Age and BMI showed statistically significant differences between patients with and without cardiac remodeling, while gender differences were not significant. In line with the current study, **Bendary *et al.*** [12] analyzed the role of Galectin-3 in predicting LV remodeling after anterior STEMI in 100 patients, finding that 13 [13%] experienced remodeling. Their results showed no significant differences in sex or age between those with and without remodeling [p>0.05]. Similarly, **Andrejic *et al.*** [13] studied Galectin-3 levels in 57 patients, reporting 22 [38.6%] with remodeling and also finding no significant differences in sex or age [p>0.05]. Conversely, **Di Tano *et al.*** [14] investigated 99 patients, identifying 26 [23.9%] with remodeling, where a significant difference in gender was observed [more females, p<0.05]. This discrepancy may be attributed to variations in mean age and gender distribution.

Our study found that left ventricular end-diastolic volume [LVEDV] was significantly higher in the remodeling group after three months, with no baseline differences. **Bendary *et al.*** [12] also noted significant increases at six months [p< 0.001]. In contrast, **Andrejic *et al.*** [13] reported higher baseline LVEDV in the remodeling group [p=0.001], but our study showed no significant differences at one and six months, possibly due to sample size variations.

Our study demonstrated that left ventricular end-systolic volume [LVESV] was significantly

higher in the remodeling group compared to non-remodelers at both baseline and three months postoperatively [p=0.001]. **Bendary *et al.*** [12] similarly observed elevated LVESV in remodelers at baseline and six months [p<0.001]. While **Andrejic *et al.*** [13] reported higher baseline left ventricular end-diastolic volume [LVEDV] in remodelers [p=0.002], they found no significant differences at one and six months. **Di Tano *et al.*** [14] noted increased LVEDV in remodeling patients at six months versus baseline, while it decreased in non-remodelers [p<0.05].

The average baseline wall motion score was 1.59 for the Remodelers group and 2.01 for the non-Remodelers group, with no significant difference [p=0.6]. After three months, scores were 2.68 for remodelers and 1.9 for non-remodelers, also not significant [p=0.4]. This contrasts with **Mousa *et al.*** [15], which found significant differences and identified WMSI [>1.5] as a predictor of left ventricular remodeling post-PCI.

The current study found that Galectin-3 levels in the Non-remodelers group ranged from 11 to 16, with a mean of 12.62, while in the Remodelers group, levels ranged from 12 to 18, with a mean of 16.59, showing a statistically significant difference [p<0.001]. Similarly, **Di Tano *et al.*** [14] reported that higher baseline Galectin-3 levels were linked to an increased risk of left ventricular remodeling [LVR], with multivariable analysis identifying Gal-3 as an independent predictor [OR 1.22, 95% CI 1.06 to 1.42 per 1 ng/mL change]. **Bendary *et al.*** [12] also observed significantly higher baseline Galectin-3 in remodelers [p<0.001], but no significant difference was noted at six months post-surgery. Additionally, **Andrejic *et al.*** [13] found that Gal-3 levels on day 1 were higher in the LVR group [10.34 ng/ml ± 3.81 vs. 8.22 ng/ml ± 2.34, p= 0.01], with multivariable analysis indicating that Gal-3 at day 30 independently predicted LVR.

Infarction markers Troponin and CKMB were significantly higher in the remodelers group, which also had more anterior wall infarctions than the non-remodelers. Supporting this, **Hendriks *et al.*** [16] indicated that peak CKMB levels correlate with infarction size and ventricular remodeling. **Silveira *et al.*** [17] suggested peak CKMB during acute STEMI predicts remodeling outcomes, while **Kubo *et al.*** [18] linked elevated cTnT to LV remodeling progression, indicating its predictive value for end-stage conditions. **Wang *et al.*** [19] found higher remodeling incidence in patients with anterior wall infarctions, with peak troponin I levels independently associated with early LV remodeling [odds ratio: 1.035].

The study found a strong positive relationship between admission levels of Galectin-3 and cardiac remodeling after primary PCI, with a Pearson's correlation coefficient of 0.84.

Several studies showed a significant association between Galectin-3 and cardiac remodeling after primary PCI, although Pearson's correlation was not used [13, 14]. **Andrejic *et al.*** [13] reported positive correlations of Galectin-3 levels from the aortic root, coronary sinus, and femoral vein. Notably, Galectin-3 concentration in the cubital vein on day 30 was identified as an independent risk factor for left ventricular remodeling [LVR] six months post-AMI, indicating a 1.5-fold increased risk. A meta-analysis by **Tian *et al.*** [20] found a significant negative correlation between Galectin-3 and left ventricular ejection fraction [LVEF], with higher levels linked to increased all-cause mortality in AMI patients.

This study's ROC curve analysis revealed that admission levels of Galectin-3 effectively predict cardiac remodeling after primary PCI, with a baseline AUC of 0.997, a cutoff of 14.5 ng/ml, sensitivity of 93.8%, and specificity of 100%. **Di Tano *et al.*** [14] found a cutoff of ≥ 13.5 ng/ml with sensitivity of 76.9% and specificity of 65.2%. **Bendary *et al.*** [12] reported an AUC of 0.897 with a cutoff of >19.3 ng/ml, sensitivity of 92.3%, and specificity of 87.2%. **Andrejic *et al.*** [13] noted lower values for Galectin-3 levels at a cutoff of 9.42 ng/ml.

Limitations of the current study include a small sample size, a single-center design, and short follow-up, highlighting the need for further research with larger samples and longer follow-up to confirm these results.

Conclusion: This study found that Gal-3 serum levels after PPCI are a reliable biomarker for left ventricular remodeling in patients with anterior STEMI. The positive correlation raises the question of using Gal-3 in screening for those at higher risk of post-STEMI heart failure.

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