CORRELATION BETWEEN CORONARY ARTERY DISEASE AND GALECTIN 3 SERUM LEVEL: ANGIOGRAPHIC BASED STUDY

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ABSTRACT:

Background: Galectin 3 (Gal-3) has great impact on initiation and progression of atherosclerosis as a cardiovascular inflammatory marker. The role of Gal-3 in coronary artery disease (CAD) remains a subject of debate. CAD and atherosclerosis are an inflammatory process.

Aim of the work: to evaluate Gal-3 concentrations among patients with CAD presented with acute coronary syndrome (ACS) and chronic coronary syndrome (CCS).

Patients and Methods: 252 individuals were divided into control group with normal coronary angiography and patient group with obstructive CAD which was further subdivided according to the clinical presentations into ACS and CCS subgroups. All groups were matched for age and sex.

Results: significantly higher Gal-3 values were found in CAD group, ACS subgroup, and CCS subgroup versus control group. Gal-3 values were significantly higher in ACS subgroup compared with CCS subgroup. Gal-3 concentrations demonstrated a non-significant association with age and EF. Gal-3 levels showed significant positive relationship with Synergy Between Percutaneous Coronary Intervention with Taxus and Cardiac Surgery (SYNTAX) score in CAD group, CCS subgroup, and ACS subgroup. Gal-3 concentrations demonstrated significant positive relationship with the number of stenotic coronary arteries and segments in CAD group and CCS subgroup but not in ACS subgroup. Gal-3 was found to significantly predict coronary artery disease.

Conclusions: Gal-3 concentrations correlated with CAD's presence and severity. We recommend the integration of Gal-3 in the diagnostic, prognostic, and predictive modules of CAD.

Keywords: Galectin 3, coronary artery disease, acute coronary syndrome, stable angina.

INTRODUCTION:

Coronary artery disease is the leading worldwide cause of mortality and morbidity with a significant socioeconomic burden [1]. Multiple clinical patterns were included in the spectrum of chronic coronary syndrome (CCS) as stable angina, left ventricular dysfunction, stabilized symptoms after ACS or coronary revascularization, vasospastic angina, microcirculatory dysfunction, and asymptomatic subjects diagnosed on screening [2]. ACS included distinct clinical entities as ST-segment elevation myocardial infarction (STEMI), non-ST-segment elevation myocardial infarction (NSTEMI), as well as unstable angina [3]. Local immune

and inflammatory process had an essential role in the development of atherosclerotic CAD, in its progression [4] and also in the pathogenesis of atherothrombosis and plaque destabilization in ACS[5].

Galectin-3 is a 29-35 kDa β-galactoside-binding lectin produced in macrophages with great pathogenic role in atherosclerosis by promoting inflammatory response, oxidative stress, endothelial dysfunction, lipid endo-cytosis, and vascular smooth muscle cells migration [6]. In heart failure (HF) cases, elevated blood and myocardial Gal-3 levels reflected myocardial inflammation, remodeling, and adverse clinical outcomes [7]. The American Heart Association recommended Gal-3 for risk stratification and prognostic-cation of HF [8]. Gal-3 was extensively studied in HF populations, whereas its association with CAD severity and atherosclerotic plaque burden is still a subject of debate [9].

AIM OF THE WORK:

Our aim was to evaluate serum Gal-3 values in cases with CAD presenting with ACS and CCS, assess the relationship between Gal-3 and with CAD severity, and investigate its role as a predictor of CAD.

PATIENTS AND METHODS:

This study enrolled 252 patients recruited from the catheterization laboratory at Mansoura Specialized Medical Hospital starting October 2021 to April 2022. They were divided into patients` group with obstructive CAD and control group with normal coronary angiography (CA). Patients with CAD were further subdivided according to the clinical presentation into ACS and CCS subgroups. Obstructive CAD was defined as ≥ 70 % stenosis in the epicardial coronaries or ≥ 50 % stenosis in the left main coronary artery and normal CA was defined as normal coronary filling with no lumen narrowing or irregularities [10]. ACS and CCS were

diagnosed according to the guidelines of the European Society of Cardiology [2, 3]. CAD severity was evaluated by SYNTAX 1 score calculated online at http://www. syntaxscore. com for all coronary lesions more than 50% stenosis in vessels 2 mm or more in diameter. We excluded patients admitted for primary percutaneous coronary interventions, HF, atrial fibrillation, significant valvular disease, renal or hepatic impairment, connective tissue disease, malignancies, and subjects rejecting the informed consent.

Sample size:

It was calculated by Power Analysis and Sample Size (PASS) software v 15.0.5 for windows (2017) based on data reported by Kusaka et al. [11] with the difference of Gal-3 concentrations between CAD cases and normal controls as the primary outcome. The null hypothesis was the absence of statically significant difference between both groups as regards Gal-3 values. According to Kusaka et al. [11], Gal-3 level was 6.13±1.4 in CAD group and 5.38±1.14 in Control group. We adopted group allocation ratio of 4:1 (CAD: Control). A sample size of 170 patients in CAD group and 43 patients in control group was required for achieving 95% power (1-β or the probability of rejecting null hypothesis when it is false) using two-sided two-sample unequal-variance t-test with a significance level (α or the probability of rejecting null hypothesis when it is true) of 5%. The sample size was rounded up to 202 CAD patients and 50 controls to ensure sufficient power of our research.

Each patient was subjected to thorough clinical examination, electrocardiogram, and detailed transthoracic echocardiography based on the guidelines of the American Society of Echocardiography [12] using GE Vivid E9 XDclear Dimensions ultrasound system (GE Healthcare, USA).

Laboratory analysis:

A blood sample was withdrawn from each patient before undergoing coronary

angiography for measuring serum Galectin-3 level in all categories of patients. Sera were obtained by centrifuging samples at 2000-3000 RPM over 20 min, then sera underwent separation and were kept at -80° C. Enzyme-Linked Immunosorbent Assay (ELISA) kit was utilized for accurate quantitative detection of Human Galectin-3 (LGALS3) by sandwich technique (Human Galectin-3 ELISA, Bioassay Technology Laboratory kit, Catalogue No. E1951Hu). ELISA plate was pre-coated with Human LGALS3 antibody. LGALS3 in the sample was added to bind to antibodies coated on wells. After that, biotinylated Human LGALS3 Antibody was added to bind to LGALS3 in the sample. Streptavidin-HRP was then added to bind to the Biotinylated LGALS3 antibody. Following incubation, unbound Streptavidin-HRP was washed away. Then, substrate solution was added, and color developed in proportion to Human LGALS3 level. The reaction was stopped by adding an acidic solution and absorbance was detected at 450 nm. The concentrations of Gal-3 in sera were estimated by following the manufacturer´s guidelines and underwent calculation from a standard curve. The Gal-3 concentrations were expressed as pg./mL and the linear analytical measurement range of the Human Gal-3 ELISA was 5 - 2000 pg./mL Serum galectin 3 level may be affected by use of anti-inflammatory drugs, however none of our patients received such medications.

Statistical Analysis:

Data were analyzed by statistical package of social science (SPSS, IBM, Inc, Chicago; US) v 26 for windows. Quantitative data underwent testing for normality by Kolmogorov-Smirnov test and described as means ± SDs. Categorical data were described as percents and frequencies. One-way ANOVA with LSD post hoc analysis and Kruskal Wallis with Dunn's post hoc analysis was utilized to compare between the groups for parametric and non-parametric continuous data respectively. Chi square test or Fisher's exact test was utilized to compare two of categorical data. Bivariate Correlations were evaluated utilizing Pearson's or Spearman's correlation coefficient. A binary logistic regression model was utilized for the determination of effects of Gal-3 level on the risk of developing CAD (R^2). Probability ($P<0.05$) was statistically significant.

Ethical considerations:

The study obtained its approval from the Institutional Research Board of Faculty of Medicine, Mansoura University (proposal code number R.21.11.1504) on 28/11/2021. Each subject gave informed consent, and he was granted confidentiality and privacy.

RESULTS:

This study included 252 patients admitted for elective CA (84 females, 168 males; mean age 58.90±7.06 years). Patients were randomized into control group of 50 patients with normal CA (17 females, 33 males; mean age 57.90±7.83 years) and patients` group including 202 patients with CAD (67 females, 135 males; mean age 59.15±6.85 years). Patients` group was further subdivided according to the clinical presentations into 2 subgroups; 90 patients presented with ACS (31 females, 59 males; mean age 59.40±6.49 years) and 112 patients presented with CCS (36 females, 76 males; mean age 58.96±7.14 years). All groups were age and sex matched Table (1). The risk profile was comparable among all groups except diabetes mellitus and smoking which were significantly lower in controls than CAD group, ACS subgroup, and CCS subgroup but no significant difference between ACS and CCS subgroups Table (1). Angina class more than 2 was significantly higher in ACS subgroup than control patients, but no significant difference among other groups Table (1). Abnormal electrocardiogram was significantly lower while ejection fraction (EF) was significantly higher in control group than CAD group,

ACS subgroup, and CCS subgroup but no significant difference between ACS and CCS subgroups Table (1). Wall motion abnormalities and Gal-3 values were significantly lower among control patients compared with CAD group, ACS subgroup, and CCS subgroup and was significantly higher in ACS subgroup than CCS subgroup Table (1). Analysis of CA showed no significant difference between ACS and CCS subgroups as regard coronary arteries affection and SYNTAX score Table (2). In CAD group and CCS subgroup, Gal-3 concentrations demonstrated significant positive relationship with the number of stenotic vessels and segments and SYNTAX score Table(3) and Diagram (1a & 1b), whereas in ACS subgroup, Gal-3 concentrations demonstrated significant positive association with SYNTAX score Table (3) and Diagram (1c) but not with the number of stenotic vessels and segments Table (3). In CAD group, ACS subgroup, and CCS subgroup, Gal-3 demonstrated no significant correlation with age, body mass index (BMI), creatinine clearance, angina class, and EF Table (3). The current study revealed that Gal-3 was found to significantly predict CAD using a univariate logistic regression model (R^2 = 22.1%, P value < 0.001) Table (4). ROC analysis revealed that the ideal cut-off level of Gal-3 for prediction of CAD was 251.48 pg./mL (P value < 0.001, 74.2 % accuracy, 88 % specificity, 70.8 % sensitivity, 96 % negative predictive value, 42.7 % positive predictive value, 0.842 area under the curve, and 0.588 Youden's index) Diagram (2).

Table 1: Clinical, ECG, echocardiographic, and laboratory characteristics of all patients:

	All patients (no=252)	CAD group (no=202)			Control group (no=50)	P1 value	P2 value	P3 value	P4 value
		All CAD group (no=202)	ACS subgroup (no=90)	CCS subgroup (no=112)					
Age (years)	58.90±7.06	59.15±6.85	59.40±6.49	58.96±7.14	57.90±7.83	0.262	1	0.690	1
Male gender	168(66.7%)	135(66.8%)	59(65.6%)	76(67.9%)	33(66.0%)	0.911	>0.05	>0.05	>0.05
body mass index (kg/m2)	30.15±5.21	29.97±4.65	30.09±4.32	29.88±4.91	30.86±7.04	0.279	1	1	0.802
CrCl (mL/min)	92.03±31.35	93.22±30.37	92.31±24.67	93.96±34.43	87.27±34.91	0.231	1	1	0.638
HCV	71(28.2%)	56(27.7%)	26(28.9%)	30(26.8%)	15(30.0%)	0.749	>0.05	>0.05	>0.05
Smoking	131(52.0%)	114(56.4%)	50(55.6%)	64(57.1%)	17(34.0%)	0.004*	>0.05	<0.05*	<0.05*
Diabetes	103(409%)	92(45.5%)	42(46.7%)	50(44.6%)	11(22.0%)	0.002*	>0.05	<0.05*	<0.05*
Hypertension	128(50.8%)	105(52.0%)	48(53.3%)	57(50.9%)	23(46.0%)	0.449	>0.05	>0.05	>0.05
Dyslipidemia	117(46.4%)	93(46.0%)	44(48.9%)	49(43.8%)	24(48.0%)	0.803	>0.05	>0.05	>0.05
FH of CAD	58(23.0%)	45(22.3%)	21(23.3%)	24(21.4%)	13(26.0%)	0.576	>0.05	>0.05	>0.05
Angina class >2	161(63.9%)	135(66.8%)	66(73.3%)	69(61.6%)	26(52.0%)	0.051	>0.05	<0.05*	>0.05
Abnormal ECG	196(77.8%)	171(84.7%)	74(82.2%)	97(86.6%)	25(50.0%)	<0.001*	>0.05	<0.05*	<0.05*
Ejection fraction	52.76±6.65	51.10±5.62	50.86±5.13	51.29±6.01	59.46±6.30	<0.001*	1	<0.001*	<0.001*
WMA	120(47.6%)	108(53.5%)	57(63.3%)	51(45.5%)	12(24.0%)	<0.001*	<0.05*	<0.05*	<0.05*
Gal-3 level	536.29±380.99	637.59±426.32	701.1±457.49	556.5±394.21	194.22±170.08	<0.001*	0.016*	<0.001*	<0.001*

CrCl=creatinine clearance, ECG= electrocardiogram, HCV=hepatitis C seropositivity, WMA=wall motion abnormalities, P1 value: CAD group versus control group, P2 value: ACS subgroup versus CCS subgroup, P3 value: ACS subgroup versus control group, P4 value: CCS subgroup versus control group, *=significant.

Table 2: Angiographic features of CAD group:

	All CAD group (no = 202)	ACS subgroup (no = 90)	CCS subgroup (no = 112)	P value
LM	26(12.9%)	13(14.4%)	13(11.6%)	0.550
LAD	169(83.7%)	75(83.3%)	94(83.9%)	0.909
LCX	128(63.4%)	58(64.4%)	70(62.5%)	0.776
RCA	52(25.7%)	25(27.8%)	27(24.1%)	0.553
Number of stenotic coronary segments	3.39±1.21	3.46±1.23	3.34±1.21	0.500
Number of stenotic coronary arteries	1.85±0.65	1.88±0.65	1.83±0.65	0.609
SYNTAX score	26.30±7.36	26.64±7.39	26.03±7.36	0.558

LAD = significant left anterior descending artery disease, LCX = significant left circumflex disease, LM = significant left main disease, RCA = significant right coronary artery disease

Table 3: Relationship between galectin-3 concentrations and different parameters in CAD group:

	All CAD group (no = 202)		ACS subgroup (no = 90)		CCS subgroup (no = 112)	
	R	P	R	P	r	P
Age (years)	0.007	0.920	-0.032	0.762	0.031	0.748
body mass index (kg/m2)	-0.020	0.781	-0.071	0.508	0.014	0.883
CrCl (mL/min)	-0.071	0.321	-0.126	0.235	-0.029	0.762
Angina class > 2	-0.073	0.301	-0.163	0.124	-0.049	0.605
Ejection fraction	0.093	0.188	0.042	0.694	0.150	0.114
Number of stenotic coronary segments	0.338	< 0.001*	0.154	0.146	0.502	< 0.001*
Number of stenotic coronary arteries	0.391	< 0.001*	0.149	0.160	0.603	< 0.001*
SYNTAX score	0.367	< 0.001*	0.390	< 0.001*	0.344	< 0.001*

* = significant.

Table 4: Regression analysis for the value of galectin-3 concentrations in the prediction of CAD versus control groups:

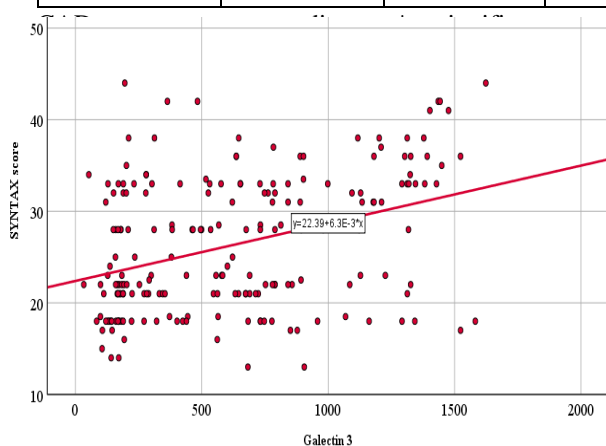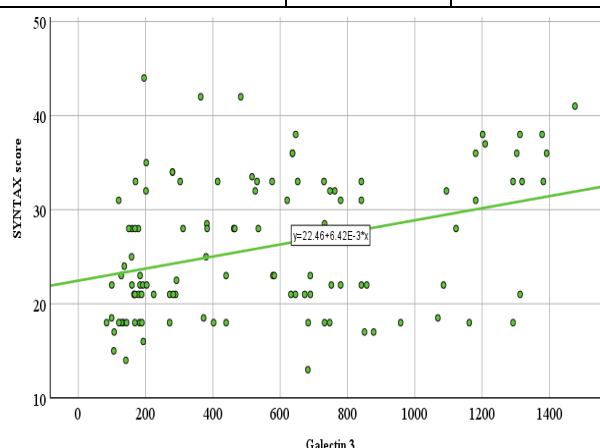|	R^2	Exb (B)	95 % confidence interval	constant	P value
Galectin-3	22.1%	1.006	1.004 - 1.008	-0.544	< 0.001*

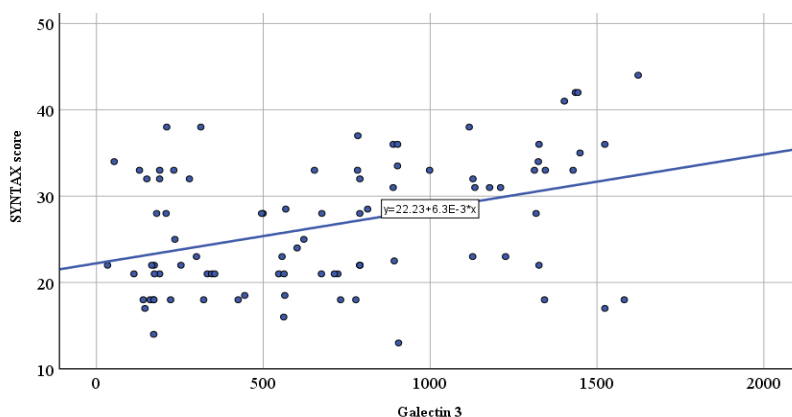Diagram 1a: CAD group

Diagram 1b: CCS subgroup

Diagram 1c: ACS subgroup

Diagram 1: Correlation between galectin-3 levels and SYNTAX score in CAD group, CCS subgroup and ACS subgroup.
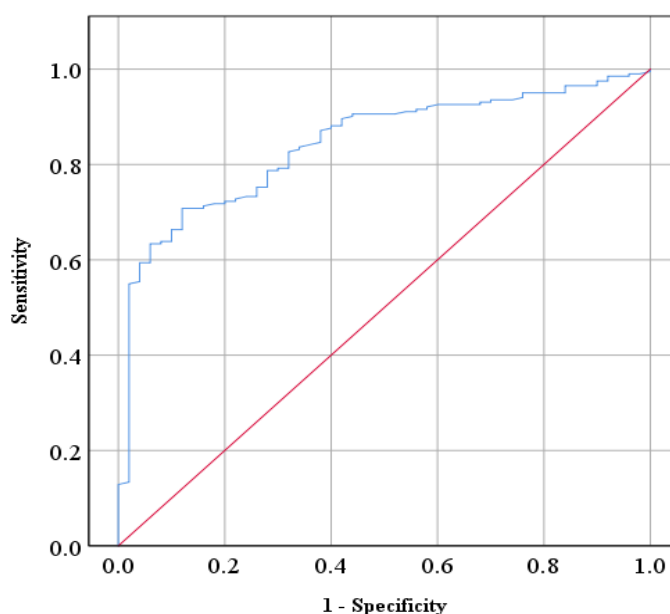

Diagram 2: Receiver operating characteristic (ROC) curve for diagnostic profile of galectin-3 levels in differentiating coronary artery disease and control groups.

DISCUSSION:

Inflammation and local innate immune response are considered cornerstone pathogenic mechanisms of atherosclerotic CAD [13]. *Ibrahim et al.* [14] concluded that Gal-3 expression level was a CAD risk factors in diabetic patients. Our aim was to assess serum Gal-3 levels among CAD cases and its role in the evaluation of CAD severity and prediction. To achieve these aims, 252 patients were divided into 50 patients with normal CA as control group, 90 patients with CAD presented by ACS, and 112 patients with CAD presented by CCS, all groups were age and sex matched.

We found significantly greater Gal-3 values in CAD patients, ACS subgroup, and CCS subgroup compared to control group with normal CA. Also, Gal-3 values were significantly greater in ACS subgroup versus

the CCS subgroup. The current study results may propose that Gal-3 as a major inflammatory marker, can have a substantial role in the pathogenetic process of plaque disturbance and ACS. Compared to patients with normal CA, earlier studies reported significantly high Gal-3 concentrations among CAD cases [11,15-22], CCS[17,23&24], ACS[25], NSTEMI[16,17&26], STEMI[17,27&28], and premature myocardial infarction[29]. On the other hand, compared to patients with CCS, earlier studies demonstrated that Gal-3 values were significantly greater among those with ACS [16,24,30&31] and STEMI[17]. *Lisowska et al.,*[20] found that Gal-3 concentrations were not significantly different between STEMI and NSTEMI cases.

In our study, Gal-3 showed no significant correlation with age, BMI, creatinine clearance, and angina class. There was no significant correlation between Gal-3 level and traditional risk variables of IHD as hypertension, diabetes mellitus, obesity, and smoking. Concordant to our results, *Falcone et al.,* [30] concluded that Gal-3 levels had no significant correlation with age and BMI, whereas *Bastawesy et al.,*[23], *Quisi et al.,*[26] and *Iribarren et al.,* [32] showed that Gal-3 concentrations had significant positive association with age. Characteristically, our results showed that Gal-3 levels had no significant correlation with ejection fraction (EF). Our results may be explained by the exclusion of patients presented with HF. Previous results showed contradictory results regarding the association between Gal-3 concentrations and EF. In CCS cases, some studies demonstrated that Gal-3 concentrations had significant negative correlation with EF [31-34], whereas other studies showed no significant correlate-ion[23,30&36]. In those with NSTEMI, *Quisi et al.,*[26] reported that Gal-3 concentrations had significant negative association with EF. In contrast, a non-significant association was found between Gal-3 values and EF among patients with ACS [25] and STEMI [32&36].

In CAD group, CCS subgroup, ACS subgroup, we showed significant positive association between Gal-3 levels and CAD severity measured by SYNTAX score. Also, we showed significant positive relationship between Gal-3 values and the number of stenotic coronary arteries and segments in CAD group and CCS subgroup but not in ACS subgroup. This may reflect that Gal-3 values in ACS cases may reflect acute inflammation rather than the extent of coronary involvement. Previous reports demonstrated that Gal-3 concentrations had significant positive association with CAD severity evaluated by SYNTAX score in cases with CAD [17], NSTEMI [16&26], and STEMI [28]. Similarly, previous studies showed that Gensini score as a measure of CAD severity, had significant positive association with Gal-3 values among those with CAD [16,18,23&24], NSTEMI [19], and ACS [25]. Some studies showed that Gal-3 showed significant positive association with the number of stenotic vessels [12,15,16,18,20,21,23,25&27] and the number of coronary stenotic segments [21], whereas other studies showed, Gal-3 concentrations did not differ with number of stenotic coronary arteries [30&37].

In our study, ROC analysis revealed that the ideal cut-off level of Gal-3 for the prediction of CAD was 251.48 pg/mL. *Quisi et al.,*[26] reported a cut-off level at 14.0 ng/mL for Gal-3 to predict an intermediate or high SYNTAX score with sensitivity and specificity of 75 % and 51%, respectively. *Li et al.,* [16] concluded that Gal-3 independently predicted ACS with 79% specificity and 60% sensitivity for a cut-off at 3.93 ng/ml. *George et al.,* [31] showed a cut-off level of 11.02 ng/mL for Gal-3 to predict ACS. *Soltan et al.,* [25] found that a Gal-3 cut-off value of ≥ 12.5 ng/mL was a predictor of ACS with sensitivity and specificity of 88.9% and 70%, respectively. Using a univariate logistic regression model, our study revealed that Gal-3 significantly predicted CAD. Earlier studies focused on the prognostic role of Gal-

3. *Maiolino et al.,*[38] demonstrated that Gal-3 independently predicted mortality in those having atherosclerotic CAD. *Aksan et al.,*[18] concluded that Gal-3 level may be considered a significant marker of inflammation in atherosclerotic CAD. *Li et al.,*[17] found that Gal-3 independently predicted the presence of CAD. *Kishimoto et al.,* [21] study revealed that Gal-3 was an independent factor for CAD and was correlated with its presence and its severity. On the contrary, *Goenka et al.,*[22] concluded that despite being significantly increased among CAD cases compared with those with normal CA, Gal-3 levels do not assess CAD severity.

Therefore, the clinical value of serum Gal-3 was to predict presence of obstructive CAD and its severity via syntax score in different clinical presentations of CAD.

Conclusion:

We conclude that Gal-3 concentrations are significantly greater in CAD cases than in control patients with normal CA. In those with CAD and CCS, Gal-3 concentrations correlated with the severity and extent of CAD measured by SYNTAX score and the number of stenotic vessels and segments. Patients with ACS had significant positive association between Gal-3 concentrations and CAD severity assessed by SYNTAX score but not with extent of CAD assessed by the number of stenotic coronary arteries and segments. This finding, together with the significantly greater Gal-3 concentrations among ACS cases than cases with CCS, may reflect the role of Gal-3 in the inflammatory pathogenesis of atherothrombosis and plaque destabilization in ACS. Also, Gal-3 concentrations significantly predicted CAD using a univariate logistic regression model with an ideal cut-off level of 251.48 pg./mL. We recommend integrating Gal-3 in the diagnostic, prognostic, and predictive modules of CAD. Also, we recommend follow up studies to evaluate the causal association and the prognostic role of Gal-3 in CAD patients.

Limitation of our study:

The major limitation of our study is being a single center study with relatively low number of patients. Because of the descriptive, cross-sectional design, our study was unable to determine the causal relationship between Gal-3 and CAD.

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Our study did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Conflict of interest:

none.

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العلاقة بين مرض قصور الشريان التاجي ومستوى جالكتين 3 فى السيرم دراسة تعتمد على التصوير الوعائي

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المقدمة: تعتبر أمراض القلب والأوعية الدموية من الأسباب الرئيسية للوفاة. على الرغم من التقدم الكبير فيما يتعلق بتشخيص وعلاج هؤلاء المرضى ، لا يزال التقسيم الطبقي للدرجة الخطورة يمثل تحدي.

يعد الجالاكتين 3 كعلامة مخاطر واعدة ومبتكرة في المرضى الذين يعانون من امراض القلب و الاوعية الدموية وهو من عائلة اللاكتين التى لها دور كبير فى التهاب وتليف الكثير من الاعضاء ومنها القلب.

وقد اثبتت الدراسات العدية دور الجالاكتين 3 فى التنبؤ بضعف عضلة القلب فى المرضى الذين يعانون من قصور الشرايين التاجية.

كما ان هناك دور للجالاكتين 3 في تغيير مسار العلاج الدوائى في مرضى ضعف عضلة القلب. ويعتبر قصور الشرايين التاجية سبب رئيسى لضعف عضلة القلب وهناك دراسات حديثة لاثبات دور الجالاكتين 3 فى مرضى قصور الشريان التاجى و كيفية التنبؤ بحدوث وفيات علي المدى الطويل في وضى قصور الشرايين التاجية.

الهدف من الدراسة: دراسة مستوى الجالاكتين 3 فى الدم وعلاقته بشدة قصور الشرايين التاجية.

هذه دراسة مستقبلية مركزية واحدة . اشتملت الدراسة 252 مريضا من مرضى قصور الشرايين التاجية فوق الثمانية عشر سنة و المقرر لهم اجراء قسطرة علي الشرايين التاجية للقلب بقسم أمراض القلب بمستشفى الباطنة التخصصي لجامعة المنصورة، خلال الفترة من أكتوبر 2021 وحتى ابريل 2022.

تم استبعاد المرضى الذين يعانون من أي من الأمراض التالية من هذه الدراسة:

المرضى الذين يعانون من فشل فى وظائف القلب .المرضى الذين يعانون من امراض في صمامات القلب .المرضى الذين يعانون من فشل كلوى مزمن و مرضى السرطان .المرضى الذين يعانون من امراض النزف والمرضى الرافضين الخضوع لهذه الدراسة.

تم اجراء هذه الدراسة بعد اخذ موافقة لجنة الاخلاقيات و البحوث بكلية الطب جامعة المنصورة.

طرق البحث: تم عمل الاتي لكل الحالات :-

1. التاريخ المرضي كاملا للحالات مع دراسة كافة عوامل الخطورة في امراض قصور الشرايين التاجية مثل ارتفاع ضغط الدم و داء السكرى و ارتفاع نسبة الدهون بالدم.
2. فحص اكلينيكي شامل.
3. رسم قلب.
4. فحوصات بالدم واهمها سحب عينة دم وقياس نسبة انزيم الجالاكتين 3 بالدم من خلال سحب عينة من الوريد وذلك قبل اجراء القسطرة.
5. اجراء قسطرة تشخيصية للشرايين التاجية للقلب بالجراءات المتعارف عليها مع حساب درجة قصور الشرايين التاجية وفقا لمعدل سينتاكس والذى يتم حسابه عن طريق تطبيق معين علي الانترنت.

وسوف يتم تقسيم المرضى الى مجموعتين :

المجموعة الاول : مرضى ذو شرايين تاجية سليمة

المجموعة الثانية: المرضى ذو تصلب في الشرايين التاجية

واثبتت النتائج ارتفاع مستوى الجالاكتين 3 فى الدم فى مرضى قصور الشرايين التاجية عن ذوى الشرايين التاجية السليمة.