

Original Article

Correlation of plasma galectin-3 and plasma lipoprotein-associated phospholipase A2 with the severity and prognosis of coronary artery disease

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Abstract: Objective: To evaluate the correlation of galectin-3 (Gal-3) and lipoprotein-associated phospholipase A2 (Lp-PLA2) with the severity of coronary artery disease and major adverse cardiovascular events (MACE). Methods: 130 patients diagnosed with coronary heart disease (CHD) by coronary angiography from October 2018 to August 2019 in the Department of Cardiology, the First Affiliated Hospital of Hebei North University, were matched into the CHD group, with 68 cases in the mild stenosis (MS) group (degree of stenosis 50%~75%), and 62 cases in the severe stenosis (SS) group (degree of stenosis $\geq 75\%$). For comparison, patients with negative results of angiography during the same period (stenosis degree $< 50\%$) were assigned to the normal group. Indicators for detection included plasma Gal-3, Lp-PLA2 concentrations, Gensini scores, and MACE events in a 30-day follow-up visit. Results: Remarkably higher plasma Gal-3 and Lp-PLA2 concentrations in the CHD group were observed in comparison with the normal group. The SS group obtained a more positive result regarding plasma Gal-3 and Lp-PLA2 concentrations and Gensini scores than the MS group ($P < 0.05$). The highest concentration of plasma Gal-3 and Lp-PLA2 was detected in the multi-vessel disease (MVD) group, followed by the double-vessel disease (DVD) group, and finally the single-vessel disease (SVD) group. Pearson correlation analysis revealed a positive correlation of plasma Gal-3 and Lp-PLA2 concentrations with Gensini scores ($P < 0.05$). Results of the follow-up visit presented strong relevance between noticeably higher concentrations of the plasma Gal-3 and Lp-PLA2 and MACE events ($P < 0.05$). Increased Gal-3 and Lp-PLA2 are risk factors for the prognosis of coronary artery disease. Conclusion: Plasma Gal-3 and Lp-PLA2 concentrations in patients with CHD are strongly related to the severity of coronary artery disease and MACE events, which is valuable for assessing the risk of patients in clinical practice.

Keywords: Plasma Gal-3, Lp-PLA2, CHD, gensini scores, MACE

Introduction

Coronary atherosclerotic heart disease (CHD) is a common disease among the middle-aged and elderly population, and has been showing a gradual rise in its morbidity and also in the number of young patients in recent years [1]. The formation and rupture of the plaque, indeterminacy in its pathogenesis notwithstanding, has been proved to be crucially related to the inflammatory response [2]. Plasma Gal-3, one type of galectin, is an inflammatory factor involved in cell adhesion, activation, growth, and apoptosis, and can accelerate the formation of atherosclerosis [3, 4]. Plasma Lp-PLA2, one of the subtypes of the phospholipase superfamily, is mainly synthesized by macrophages and T lymphocytes. It is a new marker

of reactive vascular inflammation with stable properties that can reflect neurohumoral changes and participate in the occurrence and development of atherosclerosis [5, 6]. This study investigated the correlation of plasma Gal-3 and Lp-PLA2 concentrations with the severity of CHD and MACE events, aiming to provide new markers for CHD risk assessment.

Materials and methods

General information

One hundred and thirty patients diagnosed with CHD from October 2018 to August 2019 by coronary angiography in the Department of Cardiology, the First Affiliated Hospital of Hebei

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Table 1. Scores of different degree of stenosis and coefficients of different branches

	<25% (1) point	25~49% (2) points	50~74% (4) points	75~89% (8) points	90~99% (16) points	≥99% (32) points
Left main stenosis (×5)	5	10	20	40	80	160
Left anterior descending 1 (×2.5)	2.5	5	10	20	40	80
Left anterior descending 2 (×1.5)	1.5	3	6	12	24	48
Diagonal branch 1 (×1)	1	2	4	8	16	32
Diagonal branch 2 (×0.5)	0.5	1	2	4	8	16
Left circumflex branch 1 (×2.5)	2.5	5	10	20	40	80
Left circumflex branch 2 (×1)	1	2	4	8	16	32
Posterior descending branch (×1)	1	2	4	8	16	32
Posterior lateral branch (×0.5)	0.5	1	2	4	8	16
Right coronary artery (1, 2, 3 and posterior descending branch) (×1)	1	2	4	8	16	32

North University, were matched into the CHD group, with 68 cases in the MS group (mild stenosis, degree of stenosis 50%~75%) and 62 cases in the SS group (severe stenosis, degree of stenosis ≥75%).

Inclusion criteria: Patients were diagnosed with coronary angiography. Patients were between 18 and 75 years old. Patients had good compliance and were willing to cooperate with the follow-up visit.

Exclusion criteria: Patients with severe heart failure, severe liver and kidney insufficiency, acute myocarditis, cardiomyopathy, valvular heart disease, severe acute and chronic infectious diseases, autoimmune diseases, malignant tumors, and previous percutaneous coronary intervention, or coronary artery bypass graft.

This study was reviewed and approved by the Medical Ethics Committee of our hospital (2016-21-3). The patients and their families had signed an informed consent form.

Diagnosis and grouping of CHD

Coronary angiography was performed by the Seldinger puncture through the radial artery. Patients with a stenosis degree ≥50% detected at any of the four following branches, namely left main trunk, anterior descending artery, circumflex artery, and right coronary artery were distributed into the CHD group. Healthy individuals with a stenosis degree <50% by coronary angiography were assigned to the normal group. The CHD group was divided into an MS group (50%~75%) and a SS group (≥75%) in accordance with the degree of coronary artery stenosis. According to the number of coronary artery lesions, it was also

categorized into an SVD (single-vessel disease) group, a DVD (double-vessel disease) group, and an MVD (multi-vessel disease) group.

Sample collection and detection

5 mL of venous blood was taken from the patients immediately after admission and placed to stand for 60 minutes after Ethylene Diamine Tetraacetic Acid (EDTA) anticoagulation. It was then centrifuged at 3000 r/min for 15 minutes. The upper plasma was collected and stored at -80°C. After admission, the ELISA method was used to detect Gal-3 and Lp-PLA2 at a unified time.

The Gal-3 kit was provided by MultiScience of the United States (lot No.: 70-EK1126-96). The Lp-PLA2 kit was provided by Wuhan Fine Biotechnology Co., Ltd., (lot No.: EH3302). All operations were performed in strict accordance with the instructions.

Scores of the vascular stenosis degree

Based on the Gensini [7] method, the diameter measurement method was employed to select the blood vessel with the most severe stenosis to calculate the stenosis ratio. The coefficient was determined according to different lesions. The scores of Gensini = scores of different stenosis ratios of lesion × the total of coefficients in the same lesion branch. The scores of different degrees of stenosis and different branch coefficients are shown in **Table 1**.

Follow-up visit

All patients were followed up for 30 days by phone calls or outpatient visits. The occurrence of MACE events in patients with CHD, in-

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Table 2. Comparison of general information between the two groups

	Normal group (n=54)	MS group (n=68)	SS group (n=62)	F/ χ	P
Age ($\bar{x} \pm s$, year)	57.63 $\pm$ 13.65	56.26 $\pm$ 14.15	58.89 $\pm$ 15.52	0.506	0.604
Gender (male/female)	31/23	38/30	36/26	1.810	0.167
BMI ($\bar{x} \pm s$, kg/m ²)	27.69 $\pm$ 5.21	26.35 $\pm$ 3.56	27.25 $\pm$ 3.18	0.067	0.967
Smoking (yes/no)	29/25	36/32	32/30	0.053	0.974
Hypertension (yes/no)	18/36	20/48	26/36	2.313	0.315
Diabetes (yes/no)	8/46	10/58	12/50	0.638	0.727
Dyslipidemia (yes/no)	9/47	8/60	9/53	0.496	0.780

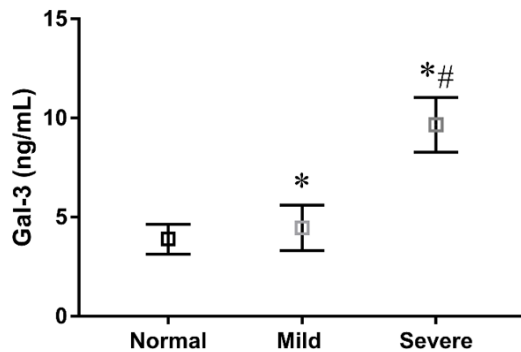


Figure 1. Comparison of plasma Gal-3 concentration among groups. The concentrations of Gal-3 in the normal group, MS group, and SS group were (3.88 $\pm$ 0.76) ng/mL, (4.46 $\pm$ 1.15) ng/mL, and (9.65 $\pm$ 1.38) ng/mL, respectively. * represents the comparison with the normal group, $P < 0.05$; # represents the comparison with the MS group, $P < 0.05$.

cluding angina pectoris, myocardial infarction, heart failure, and cardiac death was recorded.

Statistical processing

Data analysis was conducted by using SPSS 22.0 software, and GraphPad 8.0 was employed for plotting the graphics. The measurement data were represented by ($\bar{x} \pm s$). The mean of the paired samples was analyzed by t-test, the comparison of the means among multiple groups was analyzed by One-Way ANOVA. The multiple comparisons between groups were by least significant difference (LSD) test. The count data was expressed as [n (%)]. The comparison between groups was performed by using the χ^2 test. Pearson correlation analysis was employed for the correlation analysis between the indicators. Logistic regression analysis was used for multivariate analysis. The difference was considered statistically significant when $P < 0.05$.

Results

Comparison of clinical data

As shown in **Table 2**, the two groups did not present any statistical difference regarding their general information ($P > 0.05$).

Comparison of plasma Gal-3 concentration of patients

The concentrations of Gal-3 of the normal group, the MS group, and the SS group were (3.88 $\pm$ 0.76) ng/mL, (4.46 $\pm$ 1.15) ng/mL, and (9.65 $\pm$ 1.38) ng/mL respectively, with a sequence from the high to low concentration in the SS group. The MS group, and the normal group were determined by LSD test (**Figure 1**, all $P < 0.001$).

Comparison of plasma Lp-PLA2 concentration of patients

Results of One-Way ANOVA emphasized a prominent difference among the three groups, with a sequence from the high to low Lp-PLA2 concentration of (236.65 $\pm$ 30.25) ng/mL in the SS group, (199.366 $\pm$ 25.14) ng/mL in the MS group, and (158.36 $\pm$ 16.82) ng/mL in the normal group ($P < 0.001$). See **Figure 2**.

Comparison of Gensini scores

Higher Gensini scores of (32.48 $\pm$ 5.36) were observed in the SS group as compared with the (32.48 $\pm$ 5.36) in the MS group (**Figure 3**, $P < 0.001$).

Comparison of plasma Gal-3 and Lp-PLA2 concentrations in patients with different diseased branches

The differences among Gal-3 and Lp-PLA2 concentrations of the three groups are

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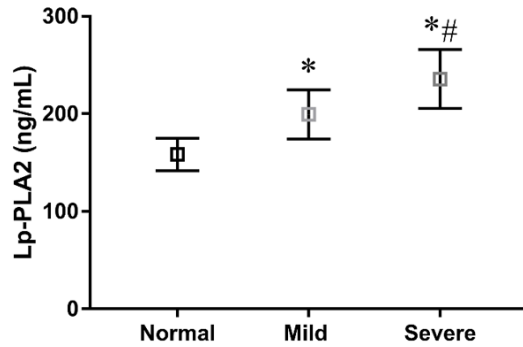


Figure 2. Comparison of plasma Lp-PLA2 concentration among groups. The concentrations of Lp-PLA2 in the normal group, MS group and SS group were (158.36±16.82) ng/mL, (199.366±25.14) ng/mL, and (236.65±30.25) ng/mL, respectively. * represents $P < 0.05$ when compared with the normal group; # represents the comparison with the MS group, $P < 0.05$.

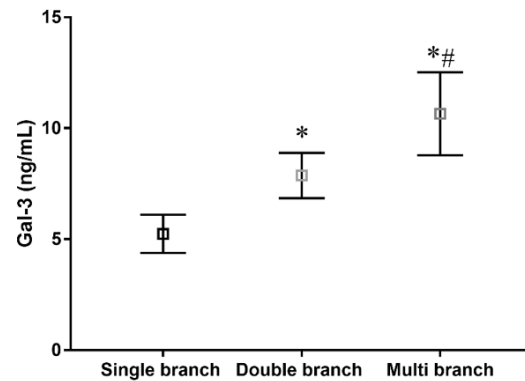


Figure 4. Comparison of Gal-3 concentration among patients with different diseased branches. The concentration of Gal-3 in SVD, DVD, and MVD group were (5.24±0.86) ng/mL, (7.87±1.02) ng/mL, and (10.65±1.87) ng/mL, respectively. * represents $P < 0.05$ compared with SVD group; # represents the comparison with DVD group, $P < 0.05$.

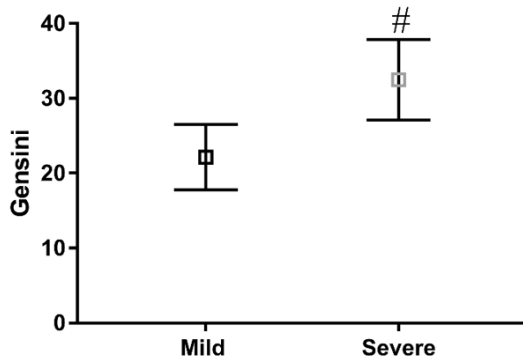


Figure 3. Comparison of Gensini scores. Gensini scores for MS group and SS group were (22.15±4.36) and (32.48±5.36), respectively. # represents the comparison with the MS group, $P < 0.05$.

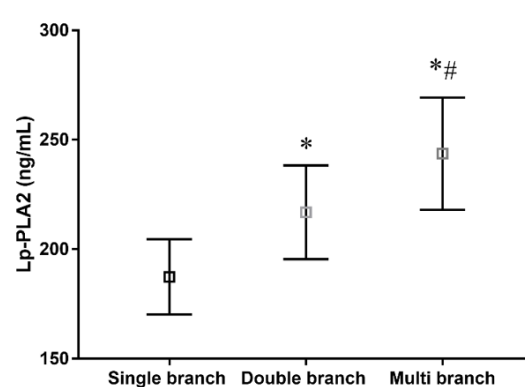


Figure 5. Comparison of Lp-PLA2 concentration among patients with different number of diseased branches. The concentrations of Lp-PLA2 in SVD, DVD, and MVD group were (187.36±17.20) ng/mL, (216.85±21.38) ng/mL, and (243.69±25.63) ng/mL, respectively. * represents the comparison with SVD group, $P < 0.05$; # represents the comparison with DVD group, $P < 0.05$.

highlighted in **Figures 4 and 5** by One-Way ANOVA, with Gal-3 concentrations of (5.24±0.86) ng/mL, (7.87±1.02) ng/mL, and (10.65±1.87) ng/mL and Lp-PLA2 concentrations of (187.36±17.20) ng/mL, (216.85±21.38) ng/mL, (243.69±25.63) ng/mL in the SVD, DVD, and MVD group, respectively. A rising trend of the Gal-3 and Lp-PLA2 concentrations along with the increasing diseased branches was observed in LSD results ($P < 0.001$).

Correlation analysis of Gal-3 and Gensini scores in MS group and SS group

Pearson correlation analysis obtained a positive correlation between the plasma Gal-3 level with the Gensini scores ($r = 0.765, 0.876$; all $P < 0.05$) in the MS group and the SS group. See **Table 3**.

Correlation analysis between Lp-PLA2 and Gensini scores in MS group and SS group

The plasma Lp-PLA2 in the MS group and the SS group was proved to be positively related with the Gensini scores by Pearson correlation analysis ($r = 0.547, 0.683$; all $P < 0.05$). See **Table 4**.

Concentrations of Gal-3 and Lp-PLA2 in MACE and non-MACE patients

As shown in **Tables 5, 6**, the 30-day follow-up visit recorded 26 cases of MACE, among which 24 cases were from the SS group and 2

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Table 3. Correlation analysis between Gal-3 and Gensini scores in MS group and SS group

	n	Gal-3	Gensini	r	P
MS group	68	4.46±1.15	22.15±4.36	0.765	0.011
SS group	62	9.65±1.38	32.48±5.36	0.876	0.001

Table 4. Correlation analysis between Lp-PLA2 and Gensini scores in MS group and SS group

	n	Lp-PLA2	Gensini	r	P
MS group	68	199.366±25.14	22.15±4.36	0.547	0.021
SS group	62	236.65±30.25	32.48±5.36	0.683	0.003

Table 5. The incidence of MACE events during the 30-day follow-up visit [n (%)]

	n	Angina pectoris	Myocardial infarction	Heart failure	Cardiogenic death	MACE incidence
MS group	68	2 (2.94)	0	0	0	2 (2.94)
SS group	62	15 (24.19)	5 (8.06)	3 (4.84)	1 (1.61)	24 (38.71)
χ^2						25.932
P						<0.001

Table 6. Concentrations of Gal-3 and Lp-PLA2 in MACE patients and non-MACE patients ($\bar{x} \pm s$, ng/mL)

	n	Gal-3	Lp-PLA2
Non-MACE	134	7.38±1.42	243.68±15.67
MACE	26	16.39±3.46	354.38±53.67
t		22.19	20.07
P		0.001	0.002

Table 7. Multivariate analysis of prognosis of coronary artery disease

Index	β	SE	Wald	P	OR	95% CI
Gal-3	0.49	0.08	37.40	<0.01	1.63	1.40~1.92
Lp-PLA2	0.46	0.13	12.44	<0.01	1.54	1.26~2.07

were from the MS group, indicating that SS patients were of a higher possibility to suffer from MACE than those with MS. Higher Gal-3 and Lp-PLA2 concentrations of (16.39±3.46) ng/mL and (354.38±53.67) ng/mL in MACE patients were observed, as compared to those of (7.38±1.42) ng/mL and (243.68±15.67) ng/mL in non-MACE patients ($P<0.05$).

Multivariate regression analysis

Taking Gal-3 and Lp-PLA2 as the independent variable and Gensini scores as the dependent variable, the *Logistic* regression analysis re-

sults showed that the increase in Gal-3 and Lp-PLA2 was a negative factor of prognosis of coronary artery disease. See **Table 7**.

Discussion

Traditional concepts believe that CHD is mainly triggered by myocardial insufficiency due to coronary artery stenosis [8]; yet consensus statements in recent studies have been developed specifically to turn down the direct relationship between myocardial hypoperfusion and coronary artery stenosis. Though the pathological mechanism of CHD remains elusive, inflammation, endothelial dysfunction, and microvascular dysfunction have received extensive attention, among which inflammation exerts a tremendous fascination on academia [9-12]. Gal-3 is one of the members of the galectin family, which can regulate various inflammatory cells such as T lymphocytes, B lymphocytes, macrophages, and centrioles-[13]. It is synthesized by macrophages, which is the activation of macrophages and capable of promoting the secretion of pro-inflammatory factors such

as tumor necrosis factor α , C-X-C Motif Chemokine Ligand 8 (CXCL-8), C-C Motif Chemokine Ligand 5 (CCL5), and intensifying tissue inflammatory response [14]. Gal-3 can activate reduced coenzyme II, stimulate neutrophils to produce superoxide, give rise to oxidative stress, and further aggravate vascular damage [15]. Lp-PLA2, as one of the subtypes of the phospholipase superfamily, can hydrolyze oxidized low-density lipoproteins to produce oxidized free fatty acids and lysolecithin, enhance monocyte migration and foam cell proliferation, cause endothelial dysfunction, and induce chronic inflammation, accelerating

the occurrence and development of atherosclerosis [16].

Remarkably higher plasma Gal-3 and Lp-PLA2 concentrations in the CHD group were collected in comparison with the normal group. The SS group obtained a more positive result regarding plasma Gal-3 and Lp-PLA2 concentrations than the MS group ($P<0.05$). The concentrations of plasma Gal-3 and Lp-PLA2 also witnessed a growing trend along with the increase of diseased branches. Prior studies conducted by Tsai et al. obtained higher plasma Gal-3 concentrations of patients with ST-segment elevation myocardial infarction than that of normal people [17]. Chung H et al. proposed that the plasma Lp-PLA2 concentrations in patients with acute angina pectoris were significantly higher than that of normal people [18]. It has also been stated in previous studies that in atherosclerotic plaques, especially vulnerable plaques, the plasma levels of Gal-3 and Lp-PLA2 were elevated, which is in conformity with the results of this study. The relevancy between Gal-3 and Lp-PLA2 and the degree of coronary artery disease was also proved in this study from the higher results of the plasma levels of Gal-3 and Lp-PLA2 concentrations in the SS group than those in the MS group. To evaluate the relationship between Gal-3 and Lp-PLA2 and the severity and prognosis of CHD, this study employed Pearson correlation analysis to detect the correlation of plasma Gal-3 and Lp-PLA2 concentrations with Gensini scores which is a widely used method to assess the severity of CHD. The higher the Gensini scores, the more severe the disease [19]. A positive correlation of plasma Gal-3 and Lp-PLA2 concentrations with the Gensini scores was collected from the results ($P<0.05$), indicating that the plasma Gal-3 and Lp-PLA2 concentrations were proportional to the severity of the coronary artery, which implied a latent application value for CHD risk assessment. It was also found in the 30-day follow-up results that higher plasma Gal-3 and Lp-PLA2 levels indicated a greater risk of MACE events. Similar conclusions have been drawn by prior studies that Gal-3 and Lp-PLA2 are one of the independent and effective markers for risk stratification of patients with suspected ACS. They can be used as one of the indicators of ACS risk assessment with a positive correlation with the occurrence of MACE events in ACS patients [20].

Since diabetes and hypertension are considered high-risk factors for CHD and the attribute of Gensini scores reflect the degree of coronary artery stenosis seen. It can be inferred that Plasma Lp-PLA2, which is involved in the development of coronary atherosclerotic plaques [21], can be used as an indicator to assess the relationship among CHD, diabetes, and hypertension. Wang et al. [22] stated a higher level of the Lp-PLA2 level and higher Gensini scores of the CHD combined with diabetes group, CHD combined with hypertension group, CHD combined with diabetes and hypertension group than those of the CHD group without combined diseases, which indicated a certain correlation between human plasma lipoprotein-related Lp-PLA2 and CHD with diabetes and hypertension. It is reasonable that the severity of coronary artery lesions can be evaluated from the level of human plasma lipoprotein-related Lp-PLA2 and Gensini scores, which is conducive to accurately understanding the coronary artery lesion and the severity of it. Increased levels of Gal-3 and Lp-PLA2 are unfavorable factors for the prognosis of coronary artery disease. Gal-3 and Lp-PLA2 levels are closely related to atherosclerosis, and the expression of Lp-PLA2 levels in arterial lesions is significantly increased. Lp-PLA2 levels can also be used as a serum marker of heart failure. Clinical monitoring of Gal-3 level can reflect the degree of heart failure in real-time, which is helpful for the diagnosis, treatment, and prognosis of cardiovascular diseases, and is consistent with the study results conducted by Zhang et al. [23].

This study provided a basis for the assessment of the severity of CHD and MACE events by the detection of plasma Gal-3 and Lp-PLA2. This study was limited by the small sample size, the limited time of follow-up visits, and the absence of further analysis of the reasons behind the correlations. Corresponding basic research with a larger sample size and a long-term follow-up clinical research should be conducted to provide stronger evidence for the application of plasma Gal-3 and Lp-PLA2 in the future.

In conclusion, plasma Gal-3 and Lp-PLA2 concentrations in patients with CHD are strongly related to the severity of coronary artery disease and MACE events. A new promising therapeutic target for the treatment of CHD is now

revealed that Gal-3 and Lp-PLA2 are considered as key biochemical markers in patients with CHD at the early stage.

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Disclosure of conflict of interest

None.

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References

- [1] Zhu KF, Wang YM, Zhu JZ, Zhou QY and Wang NF. National prevalence of coronary heart disease and its relationship with human development index: a systematic review. *Eur J Prev Cardiol* 2016; 23: 530-543.
- [2] Bentzon JF, Otsuka F, Virmani R and Falk E. Mechanisms of plaque formation and rupture. *Circ Res* 2014; 114: 1852-66.
- [3] Bivona G, Bellia C, Lo Sasso B, Agnello L, Scuzzone C, Novo G and Ciaccio M. Short-term changes in Gal 3 circulating levels after acute myocardial infarction. *Arch Med Res* 2016; 47: 521-525.
- [4] Madrigal-Matute J, Lindholt JS, Fernandez-Garcia CE, Benito-Martin A, Burillo E, Zalba G, Beloqui O, Llamas-Granda P, Ortiz A, Egido J, Blanco-Colio LM and Martin-Ventura JL. Galectin-3, a biomarker linking oxidative stress and inflammation with the clinical outcomes of patients with atherothrombosis. *J Am Heart Assoc* 2014; 3: e000785.
- [5] Maiolino G, Bisogni V, Rossitto G and Rossi GP. Lipoprotein-associated phospholipase A2 prognostic role in atherosclerotic complications. *World J Cardiol* 2015; 7: 609-620.
- [6] Chang JG. PLAC test for Lp-PLA2 activity to predict coronary heart disease. *Am Fam Physician* 2020; 101: 44-46.
- [7] Rampidis GP, Benetos G, Benz DC, Giannopoulos AA and Buechel RR. A guide for Gensini Score calculation. *Atherosclerosis* 2019; 287: 181-183.
- [8] Marfella R, Sardu C, Calabrò P, Siniscalchi M, Minicucci F, Signoriello G, Balestrieri ML, Mauro C, Rizzo MR, Paolisso G and Barbieri M. Non-ST-elevation myocardial infarction outcomes in patients with type 2 diabetes with non-obstructive coronary artery stenosis: effects of incretin treatment. *Diabetes Obes Metab* 2018; 20: 723-729.
- [9] Wirtz PH and von Känel R. Psychological stress, inflammation, and coronary heart disease. *Curr Cardiol Rep* 2017; 19: 111.
- [10] Brainin P, Frestad D and Prescott E. The prognostic value of coronary endothelial and microvascular dysfunction in subjects with normal or non-obstructive coronary artery disease: a systematic review and meta-analysis. *Int J Cardiol* 2018; 254: 1-9.
- [11] Karoli NA and Rebrov AP. Endothelial dysfunction in patients with chronic obstructive pulmonary disease in combination with coronary heart disease. *Ter Arkh* 2019; 91: 22-26.
- [12] Taqueti VR and Di Carli MF. Coronary microvascular disease pathogenic mechanisms and therapeutic options: JACC state-of-the-art review. *J Am Coll Cardiol* 2018; 72: 2625-2641.
- [13] Volarevic V, Markovic BS, Bojic S, Stojanovic M, Nilsson U, Leffler H, Besra GS, Arsenijevic N, Paunovic V, Trajkovic V and Lukic ML. Gal-3 regulates the capacity of dendritic cells to promote NKT-cell-induced liver injury. *Eur J Immunol* 2015; 45: 531-543.
- [14] Gao P, Gibson PG, Baines KJ, Yang IA, Upham JW, Reynolds PN, Hodge S, James AL, Jenkins C, Peters MJ, Zhang J and Simpson JL. Anti-inflammatory deficiencies in neutrophilic asthma: reduced galectin-3 and IL-1RA/IL-1 β . *Respir Res* 2015; 16: 5.
- [15] Gao Z, Liu Z, Wang R, Zheng Y, Li H and Yang L. Galectin-3 is a potential mediator for atherosclerosis. *J Immunol Res* 2020; 2020: 5284728.
- [16] Tellis CC and Tselepis AD. Pathophysiological role and clinical significance of lipoprotein-associated phospholipase A₂ (Lp-PLA₂) bound to LDL and HDL. *Curr Pharm Des* 2014; 20: 6256-6269.
- [17] Tsai TH, Sung PH, Chang LT, Sun CK, Yeh KH, Chung SY, Chua S, Chen YL, Wu CJ, Chang HW, Ko SF and Yip HK. Value and level of galectin-3 in acute myocardial infarction patients undergoing primary percutaneous coronary intervention. *J Atheroscler Thromb* 2012; 19: 1073-1082.
- [18] Chung H, Kwon HM, Kim JY, Yoon YW, Rhee J, Choi EY, Min PK, Hong BK, Rim SJ, Yoon JH, Lee SJ, Park JK, Kim MH, Jo M, Yang JH and Lee BK. Lipoprotein-associated phospholipase A₂ is related to plaque stability and is a potential biomarker for acute coronary syndrome. *Yonsei Med J* 2014; 55: 1507-1515.
- [19] Hu LH, Zhang LJ, Jin ZT, Yang W, Zhang LN and Lu CY. Analysis of the clinical characteristics of patients with acute coronary syndrome in dif-

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- ferent states of renal function. *West Indian Med J* 2015; 64: 357-361.
- [20] Eliaz I, Weil E, Dutton JA, McCalley AE, Nolte B and Moriarty PM. Lipoprotein apheresis reduces circulating galectin-3 in humans. *J Clin Apher* 2016; 31: 388-392.
- [21] Bonnefont-Rousselot D. [Lp-PLA2, a biomarker of vascular inflammation and vulnerability of atherosclerosis plaques]. *Ann Pharm Fr* 2016; 74: 190-197.
- [22] Wang ZJ, Deng ZM, Li XH and Kuang YH. Association of plasma lipoprotein-related phospholipase A2 with coronary heart disease and diabetes mellitus and hypertension. *Clin Lab J* 2018; 7: 203-205.
- [23] Zhang XY, Qu XB and Peng XL. Association of lipoprotein-associated phospholipase A2 levels with coronary artery disease, extent of angiographic coronary artery lesions and risk factors in the elderly. *Chin J Geriatrics* 2009; 28: 904.