Is galectin-3 a biomarker, a player—or both—in the presence of coronary atherosclerosis?

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ABSTRACT

Atherosclerosis is a complex process mediated by leukocytes, macrophages and various inflammatory markers. Galectin-3 is secreted by activated macrophages and is involved in cardiac fibrosis, cardiac remodeling, and inflammation. The present study aimed to determine the relationship between the presence and severity of coronary artery disease (CAD) and serum galectin-3 levels. The study included 82 patients with CAD confirmed via coronary angiography and 82 healthy participants as control group. Angiographic CAD was defined as ≥50% luminal diameter stenosis of at least one major epicardial coronary artery. The severity of CAD was determined by the Gensini score; and the serum galectin-3 levels were measured via ELISA. Serum galectin-3 levels were significantly higher in the patient group with CAD than in the control group (12.96±4.92 vs 5.52±1.9 ng/mL, p<0.001). In the correlation analysis, serum galectin-3 showed significant correlation with the Gensini score (r=0.715, p<0.001), number of diseased vessels (r=0.752, p<0.001) and serum hs-CRP level (r=0.607, p<0.001). In addition, multivariate logistic regression analysis showed that the serum galectin-3 levels were significant and independent predictors of the presence of angiographic CAD (OR=3.933, 95% CI 2.395 to 6.457; p<0.001). In the present study, the serum galectin-3 levels were higher in the patients with CAD than in healthy controls. Also, serum galectin-3 levels showed a significant positive correlation with the severity of CAD. An increased serum galectin-3 level may be considered an important activator and a marker of the atherosclerotic inflammatory process in CAD.

Significance of this study

What is already known on this subject?

- In recent studies, its been shown that galectin-3, which is secreted from activated macrophages and takes part in cardiac remodeling and pathogenesis of cardiac fibrosis, has a role in formation of atherosclerotic plaques and their progression.
- It has been reported that galectin-3 was found to be at high levels in acute coronary syndrome patients and proposed to be associated with the destabilization of plaques.
- No data are available in the literature, about the relation between extent and severity of coronary artery disease (CAD) and galectin-3 levels.

What are the new findings?

- In this study, we detected galectin-3 levels to be higher in patients with CAD than in non-CAD patients.
- Additionally, galectin-3 levels had significant correlation with number of diseased vessels and Gensini score, and with the extent and severity of CAD.

How might it impact on clinical practice in the foreseeable future?

- Galectin-3 may have value as a marker in the assessment of CAD.
- High galectin-3 levels may serve a role in risk classification of patients with CAD and in building treatment strategies for this disease.

INTRODUCTION

Atherosclerotic disease is a major cause of morbidity and mortality worldwide. Currently, atherosclerosis is considered to be a complex inflammatory process that involves various inflammatory markers.[1] Macrophages play an important role in the formation and progression of atherosclerotic plaque.[2] Macrophages uptaking cholesterol-rich low-density lipoprotein (LDL) and turning it into lipid-loaded foam cells in the arterial intima layer is a critical step in the formation of atherosclerotic lesions.[3]

Galectin-3, a β-galactoside-binding lectin, is secreted by activated macrophages.[4] It is involved in proliferation, macrophage chemotaxis, phagocytosis, neutrophil extravasation, oxidative stress, apoptosis, angiogenesis, fibroblast proliferation, and deposition of type-1 collagen in the extracellular matrix (ECM), resulting in adverse matrix remodeling.[5 6] Several studies have reported an association between the serum galectin-3 level, cardiac

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fibrosis and remodeling. It has also been reported that galectin-3 might be a biomarker in determining the severity and prognosis of heart failure (HF).[7–11] A study investigating the relationship between galectin-3 and atherosclerosis reported that there was an increase in the level of macrophage- and foam cell-originated galectin-3 in human atherosclerotic lesion specimens.[12] In another study, the galectin-3 levels were found to be higher in unstable angina patients than in stable angina patients; and it was emphasized that they might also be involved in plaque destabilization.[13] Nonetheless, currently, there are insufficient data to establish a link between the serum galectin-3 levels and the severity of coronary atherosclerosis.

The present study aimed to determine the relationship between the presence and severity of coronary artery disease (CAD), and serum galectin-3 levels.

MATERIALS AND METHODS

Study population

This was a prospective study in which patients who underwent diagnostic coronary angiography (CAG) for suspected CAD between May, 2012 and July, 2014, were enrolled. Among the enrolled participants, 82 had CAD, while 82 were healthy controls with normal coronary artery.

Patients with acute coronary syndrome (ACS), previously documented CAD, suspected myocarditis or pericarditis, left ventricular ejection fraction ≤50%, significant valvular disease, renal insufficiency (creatinine-based estimated glomerular filtration rate <90 mL/min/1.73 m^2 calculated by the Cockcroft-Gault formula), known malignant disease, and systemic inflammatory or autoimmune disease, were excluded from the study. The study protocol was approved by the Ethics Committee and informed consent was obtained from all the participants.

CAG analysis

All the patients underwent CAG by Judkins technique via a femoral approach. During the procedure, the images were recorded at a speed of 15 square/s on a digital angiographic system (ACOM.PC; Siemens AG, Germany). Iopromide (Ultravist 370, Schering AG, Berlin, Germany) was used as the contrast agent. The recordings were examined by two independent cardiologists. Angiographic CAD was defined as ≥50% luminal diameter stenosis of at least one major epicardial coronary artery. The severity of CAD was determined base on the Gensini score, which is a measure of the extent of coronary stenosis according to degree and location. As per the Gensini scoring system, the larger segments are more heavily weighted, ranging from 0.5 to 5.0. The narrowing of the coronary artery lumen is rated 2 for 0–25% stenosis, 4 for 26–50%, 8 for 51–75%, 16 for 7690%, 32 for 91–99%, and 64 for 100% stenosis. The Gensini Index is the sum of the total weights for each segment.[14] Moreover, scores of the number of diseased vessels ranged from 0 to 3. The criteria for 1, 2 or 3-vessel disease is a ≥50% reduction in the internal diameter of the left anterior descending (LAD), right or left circumflex coronary artery. A ≥50% reduction in the internal diameter of the left main coronary artery (LMCA) is defined as a 2-vessel disease.

Standard echocardiography

Echocardiographic examinations were performed using a GE Vingmed Vivid 7 (GE Vingmed Ultrasound, Horten, Norway) echocardiography device in the patient and control groups by a cardiologist experienced in echocardiography, while the patients were in the left lateral decubitus position. Parasternal long axis, short axis, apical four chamber and two chamber images were obtained, and evaluation by M-mode, two-dimensional continuous wave Doppler and pulse wave Doppler methods was performed in accordance with the criteria of the American Echocardiography Society.[15] The values were measured on three separate beats and then averaged for all the parameters.

Biochemical measurements

Biochemical parameters were measured in an Abbott ARCHITECT c8000 (Abbott Laboratories, Illinois, USA) autoanalyzer using commercial kits. Hematological parameters were analyzed using an Abbott CellDyn 3700 (Abbott Laboratories) device via laser and impedance methods. Biochemical and hematological parameters were studied on the same day. Peripheral blood sampling was performed in enrolled patients at the time of admission (before coronary angiography) for galectin-3 measurements. Serum samples were stored at −80°C until galectin-3 was studied by ELISA. Serum galectin-3 levels were determined by the ELISA method in accordance with the manufacturer's instructions, using Multiwash (TriContinent Scientific, USA) and Synergy 4 Microplate Reader (Biotek, USA) devices and the Human Galectin-3 Platinum ELISA kit (eBioscience Inc, San Diego, USA). A standard curve was drawn by implementing the five-parameter curve fit method and the results were presented in ng/mL. High sensitive C reactive protein (hs-CRP) (CardioPhase, hs-CRP) was quantitatively measured by a BN II System Nephelometer (Dade Behring, Marburg, Germany) using a immunonephelometric method from patients' serum and the results were reported in mg/L.

Statistical analysis

The IBM SPSS Statistics V.22 (IBM SPSS, Turkey) program was employed for statistical analyses. Appropriateness of the parameters for normal distribution was assessed by a Shapiro Wilk test. The study data were evaluated by descriptive statistical methods, such as mean, SD and frequency. The Student's t test was used to compare two groups of values demonstrating normal distribution, while groups of values without normal distribution were compared using the Mann-Whitney U test. The non-parametric Kruskal-Wallis test was used to compare the mean of the galectin-3 measurements in patients with CAD categorized according to the number of diseased vessels. χ^2 Test and Continuity (Yates) Correction were used to compare qualitative data. Logistic analysis was used for multivariate analysis. The relationship between the parameters showing consistency with normal distribution was analyzed by Pearson's correlation analysis, while the association between the parameters not showing any consistency with normal distribution was analyzed by Spearman's r correlation analysis. The level of statistical significance was set at

Table 1 Baseline clinical and biochemical characteristics of the study population

Variables	CAD group (N=82)	Control group (N=82)	p Value
Age (years)	61.9±7.8	58.5±7.1	0.003
Male sex, n (%)	51 (62.1)	47 (57.3)	0.289
BMI (kg/m^2)	27.89±2.2	27.6±2.1	0.407
Hypertension, n (%)	36 (43.9)	22 (26.8)	0.034
Diabetes mellitus, n (%)	16 (19.5)	15 (18.3)	1.000
Smoking, n (%)	49 (59.8)	36 (43.9)	0.061
Heart rate (bpm)	73.4±10.6	70.6±9.6	0.086
Systolic blood pressure (mm Hg)	125.1±14.2	121.2±12.3	0.066
Diastolic blood pressure (mm Hg)	79±10.5	75±9.1	0.022
White cell count (×10^3/mm^3)	11.42±4.1	11.1±5.17	0.449
Hemoglobin (g/dL)	14.34±1.4	14.05±1.7	0.231
Platelet (×10^3/mm^3)	244.41±48.3	234.04±60.3	0.066
Serum glucose (mg/dL)	108.72±28.9	109.79±39.7	0.234
Serum creatinine (mg/dL)	0.9±0.2	0.85±0.2	0.120
HbA1C (%)	5.4±1.1	5.2±0.9	0.456
Triglyceride (mg/dL)	176.16±129.4	171.49±80.3	0.425
Total cholesterol (mg/dL)	182.68±38	178.85±42.03	0.542
LDL-C (mg/dL)	112.17±29.5	105.15±39.1	0.197
HDL-C (mg/dL)	33.37±7.72	39.6±8.52	<0.001
LVEF (%)	57.57±6.7	57.13±8.6	0.983
Galectin-3 (ng/mL)	12.96±4.9	5.52±1.9	<0.001
Hs-CRP (mg/L)	2.83±1.47	1.11±0.56	<0.001
Gensini score	63.37±23.9 (26–123)		
Number of diseased vessels			
0		82 (100%)	
1	37 (45.1%)		
2	30 (36.6%)		
3	15 (18.3%)		
Location of stenosis			
LMCA	4 (4.9%)		
LAD	51 (62.2%)		
LCx	35 (42.7%)		
RCA	41 (50%)		
Medical treatments			
ACE inhibitor/ARB, n (%)	22 (26.8)	19 (23.2)	0.718
B-blocker, n (%)	15 (18.3)	11 (13.4)	0.521
Statin, n (%)	11 (13.4)	9 (11)	0.811
ASA	21 (25.6)	16 (19.5)	0.231

ARB, angiotensin receptor blocker; ASA, acetylsalicylic acid; BMI, body mass index; CAD, coronary artery disease; HbA1C, glycated haemoglobin HDL-C, high density lipoprotein cholesterol; Hs-CRP, high sensitive C reactive protein; LAD, left anterior descending coronary artery; LCx left circumflex coronary artery; LDL, low density lipoprotein cholesterol; LMCA, left main coronary artery; LVEF, left ventricular ejection fraction; RCA, right coronary artery.

p<0.05. Bonferroni's correction was performed when statistical comparisons of four groups were made as <0.001.

RESULTS

Baseline clinical characteristics

The study included 82 patients with CAD (group I, 51 males; mean age 61.9±7.8 years) and 82 healthy controls with normal coronary artery (group II, 47 males; mean age 58.5±7.1 years). Demographic characteristics, as well as clinical and laboratory findings of the patients, are shown in table 1. Age (p=0.003), the prevalence of hypertension (p=0.034), and diastolic blood pressure (DBP) (p=0.022) were significantly higher in the patient group with CAD; high-density lipoprotein (HDL) cholesterol (p<0.001) was significantly lower in the patient group with CAD than in the control group (table 1). Furthermore, the serum hs-CRP value was also higher in the patient group with CAD than in the control group (2.83±1.47 vs 1.11 ±0.56 mg/L, p<0.001). In the patient group with CAD, 37 patients (45.1%) had one-vessel disease, 30 patients (36.6%) had two-vessel disease, and 15 patients (18.3%) had three-vessel disease. Four patients (4.9%) had LMCA stenosis, 51 patients (62.2%) had LAD coronary artery stenosis, 35 patients (42.7%) had left circumflex coronary artery stenosis, and 41 patients (50%) had right coronary artery stenosis. Besides, no significant difference was observed between the patient groups, in terms of medical treatments they were receiving (p>0.05).

Serum galectin-3 levels

Serum galectin-3 levels were significantly higher in the patient group with CAD than in the control group (12.96

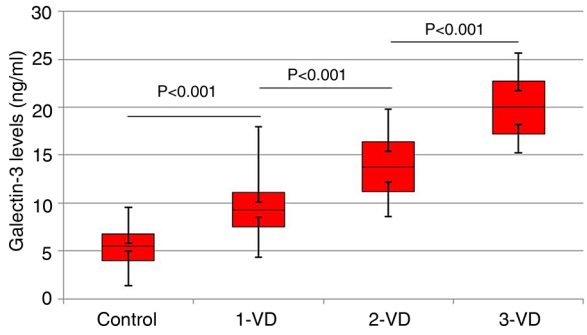

Figure 1 Serum galectin-3 levels in the control and the patient groups with CAD categorized according to the number of diseased vessels. CAD, coronary artery disease; VD, vessel disease.

±4.92 vs 5.52±1.9 ng/mL, p<0.001) (table 1). Moreover, the serum galectin-3 levels were higher in the patients with three-vessel disease than in those with two-vessel disease (19.94±3.32 vs13.74±3.13 ng/mL, p<0.001). The serum galectin-3 levels were also considerably higher in the patients with two-vessel disease than in those with one-vessel disease (13.74±3.13 vs 9.48±2.96 ng/mL, p<0.001) (figure 1).

Association of serum galectin-3 levels with clinical characteristics, laboratory findings and angiographic risk score

Based on correlation analysis, the serum galectin-3 level in the patient group with CAD showed positive correlation with age (r=0.540, p<0.001), fasting blood glucose (r=0.280, p=0.011), serum creatinine (r=0.280, p=0.011), and serum hs-CRP levels (r=0.607, p<0.001) (figure 2), and the number of diseased vessels (r=0.752, p<0.001), whereas it showed significantly negative correlation with the serum HDL-C levels (r=-607, p<0.001) (table 2). In addition, a significantly positive correlation was determined between the serum galectin-3 levels and the Gensini score (r=0.715, p<0.001) (figure 3).

Table 2 Correlation analyses between serum galectin-3 levels and various parameters in patients with coronary artery disease

Variables	r Value	p Value
Age*	0.540	<0.001
BMI*	0.001	1.000
SBP†	0.068	0.543
DBP†	0.048	0.668
Fasting glucose†	0.280	0.011
HDL-C†	−0.607	<0.001
Serum creatinine†	0.280	0.011
White cell count†	0.005	0.966
LVEF†	−0.136	0.224
Gensini score†	0.715	<0.001
Number of diseased vessels†	0.752	<0.001
Hs-CRP†	0.607	<0.001

*Pearson Correlation test.
†Spearman Correlation Test.
BMI, body mass index; DBP, diastolic blood pressure; HDL-C, high-density lipoprotein cholesterol; Hs-CRP, high-sensitive C reactive protein; LVEF, Left ventricular ejection fraction; SBP, systolic blood pressure.

Association of serum galectin-3 levels with presence of CAD

Simple logistic regression analysis revealed that the age (OR=1.065, 95% CI 1.020 to 1.112; p=0.004), DBP (OR=1.042, 95% CI1.009 to 1.077; p=0.013), HDL-C (OR=0.907, 95% CI 0.867 to 0.948; p<0.001), hypertension (OR=2.134, 95% CI 1.109 to 4.108; p=0.023), smoking (OR=1.897, 95% CI 1.020 to 3.529; p=0.043), serum galectin-3 (OR=2.304, 95% CI 1.765 to 3.008; p<0.001), and hs-CRP levels (OR=1.872, 95% CI1.371 to 2.265; p<0.001) showed an association with the presence of angiographic CAD in all the patients. These variables were entered into a backward stepwise multivariate logistic regression model. The multivariate logistic regression analysis demonstrated that hypertension, smoking, serum galectin-3, and hs-CRP levels, were significant and independent predictors of angiographic CAD (OR=4.145,

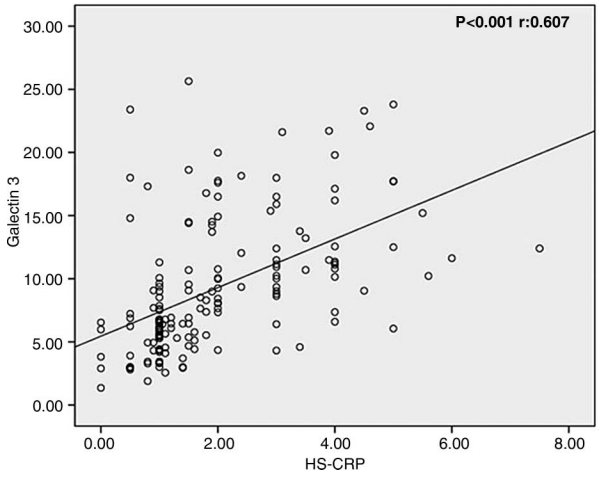

Figure 2 Correlation between serum galectin-3 levels and high sensitive C reactive protein (hs-CRP).

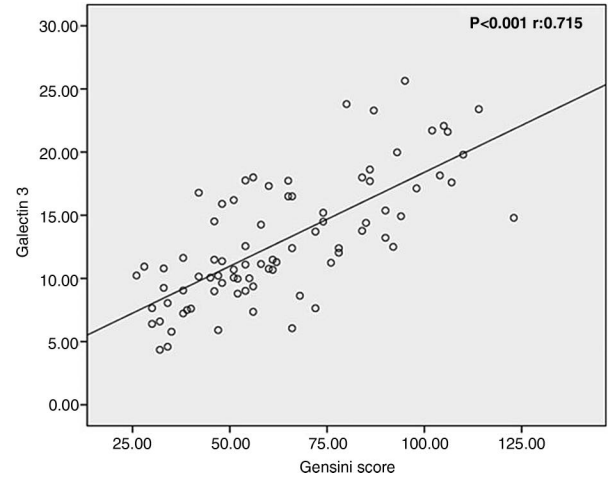

Figure 3 Correlation between serum galectin-3 levels and Gensini score.

Table 3 Logistic regression analysis for the presence of CAD in all patients

	Univariate logistic regression			Multivariate logistic regression analysis		
	OR	95% CI	p Value	OR	95% CI	p Value
Age (years)	1.065	1.020 to 1.112	0.004			
Sex (male)	2.419	0.980 to 5.974	0.055			
BMI (kg/m^2)	1.061	0.923 to 1.219	0.405			
DBP (mm Hg)	1.042	1.009 to 1.077	0.013			
LDL-C (mg/dL)	1.006	0.997 to 1.015	0.198			
HDL-C (mg/dL)	0.907	0.867 to 0.948	<0.001			
Fasting glucose (mg/dL)	0.999	0.990 to 1.008	0.843			
Serum creatinine	2.112	0.651 to 6.849	0.213			
Hypertension	2.134	1.109 to 4.108	0.023	4.145	0.86 to 19.888	0.046
Smoking	1.897	1.020 to 3.529	0.043	1.023	0.003 to 1.156	<0.001
Galectin-3	2.304	1.765 to 3.008	<0.001	3.933	2.395 to 6.457	<0.001
Hs-CRP	1.872	1.371 to 2.265	<0.001	2.256	1.456 to 2.963	<0.001

BMI, body mass index; CAD, coronary artery disease; DBP, diastolic blood pressure; HDL-C, high-density lipoprotein cholesterol; Hs-CRP, high sensitive C reactive protein; LDL-C, low-density cholesterol.

95% CI 0.86 to 19.888; p=0.046, OR=1.023 95% CI 0.003 to 1.156; p<0.001, OR=3.933, 95% CI 2.395 to 6.457; p<0.001, OR=2.256, 95% CI 1.456 to 2.963; p<0.001, respectively) (table 3).

DISCUSSION

In the present study, the serum galectin-3 levels were higher in patients with CAD than in healthy controls. Moreover, serum galectin-3 level is an independent predictor for the presence of CAD and it showed a significant correlation with the severity of CAD.

Inflammatory process plays an important role in the development of atherosclerosis. Inflammatory activity is reportedly associated with ischemia-forming conditions, such as local proteolysis, plaque rupture and thrombus formation.[2] Formation of foam cells via phagocytosis of oxidized LDL molecules by macrophages and synthesis of extracellular connective tissue (fibrous heading) by means of vascular smooth muscle cells (VSMC), which migrate to intima from media layer, gaining proliferative property, play a critical role in intimal remodeling and atherosclerotic plaque formation.[16] Therefore, the inflammatory markers involved in the formation and progression of atherosclerosis in such inflammatory process have received a great deal of interest among researchers.

Galectin-3 belongs to the family of soluble β-galactoside-binding lectins. Although galectin-3 is primarily secreted by activated macrophages, it is also synthesized by T-lymphocytes, endothelial cells and fibroblasts.[17 18] Earlier studies have demonstrated that galectin-3 contributes to the development of myocardial fibrosis by enhancing myofibroblast proliferation,[19 20] and plays a key role in aldosterone-induced vascular inflammation and fibrosis.[17] Recently, it was reported that galectin-3 is involved in the pathophysiology of HF, where it contributes to cardiac remodeling by means of myocardial fibrosis and inflammation.[21] It was also reported that serum galectin-3 level was an independent predictor of long-term mortality among acute patients with HF.[22] In addition, de Boer *et al*[23] reported that the serum galectin-3 level was higher in patients with HF with preserved left ventricle ejection fraction (LVEF); it was also demonstrated that serum galectin-3 level is an independent and strong predictor of hospitalization for HF and all-cause mortality.

Although galectin-3 has widely been investigated in patients with HF, complete elucidation of its role in the atherosclerotic process remains unclear. Serum galectin-3 plays an important role in the formation of atherosclerotic processes such as chemotaxis and phagocytosis of macrophage,[5] and proliferation of VSMC.[24] Based on immunohistochemical examination of carotid endarterectomy specimens, Nachtigal *et al*[12] observed an increase in the expression of galectin-3 localized in foam cells and macrophages in atherosclerotic lesions. A study investigating the contribution of galectin-3 to the formation of atherosclerotic lesions found that galectin-3 is the main contributor of the pathology of atherosclerotic plaque progression by means of amplification of the proinflammatory molecules.[25] Animal studies have also demonstrated that a decrease in galectin-3 levels by gene inactivation or therapeutic modulation in apolipoprotein-E-deficient mice leads to regression of atherosclerotic lesions by decreasing macrophage activation.[26 27] Arar *et al*[24] did not observe expression of the galectin-3 gene (LGALS3) in quiescent VSMC; nevertheless, an increase in galectin-3 gene expression was noted in VSMC that was activated as a result of hypercholesterolemia or balloon catheter injury. Consequently, it was concluded that galectin-3 is involved in the atherosclerotic process.

Some reports support the contention that galectin-3 is not only involved in the formation of atherosclerotic plaque, but that it also contributes to plaque destabilization. Moreover, studies have also demonstrated its significant correlation with matrix metalloproteinases, which play an important role in plaque destabilization and ECM turnover.[6] One study reported that galectin-3 modulates vascular calcification, which is associated with inflammation and plaque instability.[28] Tsai *et al*[29] observed significantly higher serum galectin-3 levels in ST segment elevation myocardial infarction patients who underwent primary

percutaneous coronary intervention, than in healthy participants, and reported that the galectin-3 level is predictive of 30-day major adverse cardiac events. Furthermore, Falcone *et al*[13] reported higher serum galectin-3 levels in patients with unstable angina pectoris than in those with stable angina pectoris, and observed a significant correlation between galectin-3 and the number of diseased vessels. These findings indicate that galectin-3 contributes to the promotion of macrophage activation and monocyte attraction in atherosclerotic plaque destabilization. In addition, long-term follow-up studies reported that galectin-3 level is an independent predictor for cardiovascular events and mortality in patients with CAD.[30 31]

The strong association between atherosclerosis and inflammation suggests that galectin-3, which contributes to the inflammatory process in the formation of atherosclerotic lesions, might be a potential biomarker for atherosclerosis. A recent study reported that the galectin-3 level was higher in patients with carotid atherosclerosis than those in the control group; moreover, galectin-3 showed a significantly positive correlation with carotid intima media thickness, which could be a predictor of coronary atherosclerosis.[32] In another study, the degree of coronary atherosclerosis was evaluated by coronary CT angiography in patients with type 2 DM; and higher galectin-3 levels were determined in patients with type 2 DM with CAD than in patients without CAD. Furthermore, the serum galectin-3 level has shown significant correlation with total number of diseased vessels, number of plaques and calcified plaque type.[33] A recent study by Kusaka *et al*[34] reported that the plasma galectin-3 level was higher in patients with CAD than in patients without CAD, and that the plasma galectin-3 level was higher in patients with multivessel CAD than in those with single-vessel CAD. Similar to the aforementioned reports, the present study found higher galectin-3 levels in the patient group with CAD than in the group without CAD. In addition, the positive correlation determined in the present study between the Gensini score and Galectin-3 might be due to the fact that a massive plaque burden reflects a kind of intensive inflammatory process. Thereby, high Gensini scores, as observed in the present study, might be associated with a more intensive atherosclerotic inflammatory process. The present study is the first to report a relationship between galectin-3 levels and the severity of CAD. In addition, another striking point of our study is the demonstration that galectin-3 level is an independent predictor for the presence of CAD.

De Boer *et al*[35] investigated the link between galectin-3 with cardiovascular risk factors and mortality in the general population; they demonstrated that galectin-3 levels have a significant positive correlation with age, serum creatinine, glucose, and hs-CRP levels. In another study, it was determined that serum galectin-3 levels show significant positive correlation with white cell count, serum creatinine levels and the number of diseased vessels in patients with ACS.[29] In a similar vein, in this study, the serum galectin-3 levels showed a considerable positive correlation with age, fasting blood glucose, serum creatinine levels, Gensini score, number of diseased vessels, and hs-CRP levels, whereas it showed a significant negative correlation with HDL-C levels. Based on these findings, we believe the observed significant correlation between serum galectin-3 level and hs-CRP level, number of diseased vessels, and Gensini score, could all be markers of the severity of inflammation, supporting the notion that galectin-3 plays a role in the atherosclerotic inflammatory process in patients with stable angina pectoris.

Study limitations

The present study had some limitations. First, this was a single center study that included a small study population. Thus, future studies may include a large patient cohort to overcome these limitations. Second, we were not able to completely evaluate the prognostic value of serum galectin-3 levels in the patients who were diagnosed with CAD.

Conclusion

Our findings revealed that serum galectin-3 levels are higher in patients with CAD than in healthy controls, and this fact is significantly correlated with the severity of CAD. The increase in serum galectin-3 levels might be an important activator that could be taken as a crucial marker of an atherosclerotic inflammatory process in CAD.

Competing interests None declared.

Patient consent Obtained.

Ethics approval Ondokuz Mayıs University Ethics Committee.

Provenance and peer review Not commissioned; externally peer reviewed.

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