Galectin 3 in patients with metabolic syndrome and atrial fibrillation

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Abstract

Objective. To evaluate serum galectin 3 level in patients with metabolic syndrome (MS) and atrial fibrillation (AF) and to reveal the significance of this marker of fibrosis in MS. **Design and methods.** We examined 100 patients with MS (50 with paroxysmal or persistent AF and 50 without arrhythmia) and 50 healthy persons. Serum galectin 3 was measured by ELISA method, all examined subjects underwent echocardiography. **Results.** Galectin 3 was higher in patients with MS and AF compared to patients with MS without arrhythmia and much higher than in healthy persons [0.72 (0.44; 1.36); 0.44 (0.42; 1.22 and 0.32 (0.28; 0.42) ng/ml, $p < 0.05$; $p < 0.001$ and $p < 0.001$, respectively]. Galectin 3 in patients with 5 components of MS was higher, than in patients with 4 and 3 components of MS [2.01 (0.52; 4.59); 0.54 (0.44; 1.37) and 0.42 (0.32; 0.42) ng/ml, $p < 0.05$; $p < 0.001$ and $p < 0.001$, respectively]. Correlations between galectin 3 and waist circumference, blood pressure, left and right atrium volume, triglyceride level were found ($r = 0.57$; $r = 0.51$; $r = 0.45$; $r = 0,40$; $r = 0.41$; $p < 0.001$). **Conclusion.** Galectin 3, a marker of fibrosis in patients with MS and atrial fibrillation was higher than in patients with MS without arrhythmia and significantly higher than in healthy persons.

Key words: galectin 3, marker of fibrosis, metabolic syndrome, atrial fibrillation.

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Галектин 3 у пациентов с метаболическим синдромом и фибрилляцией предсердий

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Резюме

Цель исследования — изучить уровень галектина 3 в сыворотке крови у пациентов с метаболическим синдромом (МС), в том числе в сочетании с фибрилляцией предсердий (ФП) для определения значимости этого маркера фиброза при метаболическом синдроме. **Материалы и методы.** Обследовано 100 пациентов с МС (IDF, 2005), из них 50 с больных пароксизмальной и персистирующей формами ФП, а также 50 практически здоровых людей сопоставимого возраста. Уровень галектина 3 в сыворотке крови оценивался методом иммуноферментного анализа ELISA. Всем обследованным выполнена трансторакальная эхокардиография. **Результаты**. Уровень галектина 3 в сыворотке крови у больных с МС в сочетании с ФП выше, чем у пациентов с МС без ФП и выше, чем у здоровых [0,72 (0,44; 1,36); 0,44 (0,42; 1,22) и 0,32 (0,28; 0,42) нг/мл соответственно, $p < 0,05$; $p < 0,001$ и $p < 0,001$]. У больных с 5 компонентами МС уровень галектина 3 выше, чем у пациентов с 4 и 3 составляющими [2,01 (0,52; 4,59); 0,54 (0,44; 1,37) и 0,42 (0,32; 0,42) нг/мл соответственно, $p < 0,05$; $p < 0,001$ и $p < 0,001$]. По результатам корреляционного анализа выявлены связи между уровнем галектина 3 и окружностью талии, уровнем артериального давления, объемом левого предсердия, объемом правого предсердия и уровнем триглицеридов ($r = 0,57$; $r = 0,51$; $r = 0,45$; $r = 0,40$; $r = 0,41$ соответственно, $p < 0,001$). **Выводы.** Маркер фиброза галектин 3 у больных МС выше, чем у здоровых, а у больных с МС в сочетании с ФП выше, чем у пациентов без данной аритмии.

Ключевые слова: галектин 3, маркер фиброза, метаболический синдром, фибрилляция предсердий.

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Introduction

The prevalence of the metabolic syndrome (MetS) and abdominal obesity in the last decade has been increasing progressively. According to WHO, worldwide 35% of people aged 20 years old and older were overweight and 11% were obese in 2008. In accordance with the criteria of the International Diabetes Federation, MetS includes a cluster of factors: abdominal obesity, hypertension, hyperglycemia, hypertriglyceridemia and decreased level of high density lipoprotein cholesterol (IDF, 2005) [1].

The main feature of MetS is considered to be abdominal obesity because this factor and underlying insulin resistance determine the development of other components of the Mets. There is no doubt that MetS is a risk factor for many pathological conditions, e. g. type 2 diabetes mellitus, coronary heart disease, congestive heart failure, and others. According to a cohort study in St Petersburg employees aged 30–55 years old the most common components of MetS are abdominal obesity and hypertension [2]. There is evidence that MetS often leads to the development of atrial fibrillation (AF). Thus, a prospective study ARIC (Atherosclerosis Risk in Communities Study) demonstrated that MetS increases the risk of AF by 67%, and there is a 4.4-fold increase in AF risk in the presence of five components of the MetS compared to the subjects without MetS [3].

AF is one of the most common sustained cardiac rhythm disorders, and its incidence is 1.5% in adult population in developed countries [4]. O. V. Listopad and colleagues (2013) demonstrated that there was a significant increase in number of patients with AF admitted to one of therapeutic clinic of St Petersburg in period from 1985 and 2010 along with the increase in the frequency of hypertension, obesity and overweight in hospitalized patients with AF [5]. AF is an independent mortality risk factor and often leads to complications such as stroke, thromboembolism, heart failure and dementia [6–7]. Thus, the causes and underlying mechanisms are topical subjects of the research.

It is known that the following conditions contribute to the development of AF: hemodynamic disturbances, atrial structural changes, myocardial electric instability. Myocardial fibrosis is the key factor in the development and progression of AF.

A meta-analysis involving 123 249 people, residents of the United States and Europe, from the clinical trials published between 1966 and 2007

in the databases PubMed and Cochrane, showed that obesity is associated with an increase in AF risk by 49 % (HR: 1.49) [8]. This largely explains the high prevalence of AF in relatively young people with abdominal obesity and hypertension and with no evidence of coronary heart disease.

Markers of myocardial fibrosis found in AF is a topical issue due to the potential use for AF prevention. One of these markers is galectin 3 — a protein belonging to the lectin family, activating fibroblasts and collagen synthesis, involved in the development of fibrosis in the heart, lungs, kidneys, liver leading to the disease onset. Experimental studies demonstrate that intrapericardial injection of galectin 3 leads to the more than 3-fold increase in left ventricular mass due to the synthesis of collagen types I and III compared to placebo [9].

Clinical studies showed that high serum levels of galectin 3 are associated with chronic heart failure and is a predictor of mortality [10]. The data published in 2014 evidence higher level of galectin 3 in patients with AF than in the general population. Moreover, during 10-year follow-up of the 3 306 participants from the Framingham study, AF episodes were registered in 250 (7.8 %), and the higher level of circulating galectin 3 was associated with the increased risk of AF (HR: 1.19, 95-% CI 1.05–1.36, p = 0.09) [11].

The relationship between adipose tissue and galectin 3 synthesis was studied in a number of experimental and clinical studies. A group of scientists from the University of Illinois (Chicago) and the University of Queen Mary (London) showed that galectin 3 level in adipose tissue is higher than in the stroma, particularly in mice with genetically determined or diet-induced obesity. Moreover, they also found that galectin 3 level is higher in visceral adipose tissue than in the subcutaneous fat [12].

However, no studies assessed the role of galectin 3 in patients with AF and MetS. The search of myocardial fibrosis markers and predictors of AF in patients with MetS is highly relevant as it will enable risk stratification and AF primary and secondary prevention.

Thus, the objective of our study was to investigate serum level of galectin 3 in patients with MetS combined with AF in order to determine the role of this fibrosis marker for MetS.

Design and methods

Altogether 150 patients 35–65 years old were studied, 80 % patients were younger than 60 years old. Among them 50 patients had MetS without AF, and 50 patients had MetS and AF. Also 50 apparently healthy patients without cardiac diseases and without MetS were included in a control group. MetS components were assessed according to the International Diabetes Federation (IDF, 2005) criteria: waist circumference in men ≥ 94 cm, in women ≥ 80 cm; systolic blood pressure (SBP) ≥ 130 mmHg and/or diastolic BP (DBP) ≥ 85 mmHg, or previously diagnosed hypertension (HTN); increased triglyceride level (TG) ≥ 1.7 mmol/l; decreased level of high density lipoproteins (HDL) in men < 1.03 mmol/l and in women < 1.3 mmol/l, increased fasting plasma glucose level ≥ 5.6 mmol/l or previously diagnosed type 2 diabetes mellitus (DM). AF was diagnosed when the episodes were registered at electrocardiography (ECG) or 24-hour ECG monitoring.

Exclusion criteria were the following: clinically relevant coronary heart disease (CHD) or CHD suspected based on the exercise test results, chronic heart failure (NYHA functional class II–IV), heart valve disease, systemic diseases, oncological diseases, acute and chronic inflammatory diseases, chronic kidney disease, liver and lung diseases with functional impairment, thyroid diseases, history of stroke, cardiosurgical and other heart interventions.

We evaluated clinical, anthropometrical, and laboratory parameters, and instrumental examination: ECG, 24-hour Holter ECG monitoring, echocardiography. Transthoracic echocardiography (TTE) was performed ("Vivid 7", Norway) by two professionals blinded to clinical data.

Serum galectin-3 level was evaluated in venous blood samples, taken in the morning after overnight fasting, by the enzyme-linked immunosorbent assay (ELISA) with Human Galectin-3 ELISA kit (eBioscience, Bender MedSystems GmbH, Campus Vienna), with lower detection limit of 0.12 ng/ml. NT-proBNP level was assessed by ele ctrochemiluminescence immunoassay kit (ECLIA, Roche Diagnostics GmbH, Mannheim), with lower detection limit of 5 pg/ml. Estimation of glomerular filtration rate (eGFR) was performed

using CKD-EPI formula. Thyrotrophic hormone was assessed in all patients.

Numerical data are presented as mean ± standard deviation or median with interquartile limits. Student t-test was used to compare parameters with normal distribution; parameters with non-normal distribution were compared using nonparametric Mann-Whitney U-test. Non-parametric Spearman test was used to determine correlations between parameters. Statistical analysis was carried out using software SPSS 17.0.

Results

The treatment groups were comparable by age and gender distribution. There were differences in body weight, waist and hip circumferences, body mass index, lipid profile and fasting plasma glucose between healthy individuals and patients with MetS. Among patients with AF there were no difference in these parameters between subjects with and without AF. NT-proBNP level was higher in patients with AF compared to healthy individuals and patients with MetS without arrhythmia, however, the median was within the population reference values in all groups. Detailed characteristics are presented in Table 1.

According to the echocardiography atrial volumes were higher in patients with MetS and AF than in MetS patients without AF, and significantly higher than in healthy subjects. Left ventricular ejection fraction did not differ between the groups (Table 2).

There was a more than 2-fold increase in galectin 3 level in MetS patients and AF compared to healthy subjects (0.72 [0.44; 1.36] and 0.32 [0.28; 0.42] ng/ml, respectively, $p < 0.001$) and higher than that in MetS patients without AF (0.72 [0.44; 1.36] and 0.44 [0.42; 1.22] ng/mL, $p < 0.05$). Galectin

Table 1

BASELINE CHARACTERISTICS OF STUDIED GROUPS

	Healthy individuals (n = 50) Group 1	**MetS (n = 50) Group 2**	**MetS + AF (n = 50) Group 3**	**P-value**
Age, years	52.1 ± 8.6	53.7 ± 7.6	53.7 ± 7.2	$p_{1,2} > 0.05$; $p_{1,3} > 0.05$; $p_{2,3} > 0.05$
Male/Female, n	24/26	21/29	30/20	$p_{1,2} > 0.05$; $p_{1,3} > 0.05$; $p_{2,3} > 0.05$
Body mass, kg	66.6 ± 11.1	91.4 ± 16.9	91.9 ± 19.0	$p_{1,2} < 0.001$; $p_{1,3} < 0.001$; $p_{2,3} > 0.05$
Waist circumference, cm	81.9 ± 8.1	108.3 ± 11.5	107.4 ± 13.5	$p_{1,2} < 0.001$; $p_{1,3} < 0.001$; $p_{2,3} > 0.05$
Hip circumference, cm	92.7 ± 8.4	111.8 ± 11.5	109.7 ± 15.9	$p_{1,2} < 0.001$; $p_{1,3} < 0.001$; $p_{2,3} > 0.05$
BMI, kg/m^2	23.4 ± 2.8	32.7 ± 5.6	31.2 ± 5.6	$p_{1,2} < 0.001$; $p_{1,3} < 0.001$; $p_{2,3} > 0.05$
TC, mmol/l	4.8 ± 0.9	5.9 ± 1.1	5.8 ± 1.2	$p_{1,2} < 0.001$; $p_{1,3} < 0.001$; $p_{2,3} > 0.05$
HDL, mmol/l	1.6 ± 0.3	1.1 ± 0.3	1.1 ± 0.4	$p_{1,2} < 0.001$; $p_{1,3} < 0.001$; $p_{2,3} > 0.05$
TG, mmol/l	0.9 ± 0.3	2.3 ± 0.8	2.1 ± 1.2	$p_{1,2} < 0.001$; $p_{1,3} < 0.001$; $p_{2,3} > 0.05$
TH, mIU/L	1.9 ± 0.8	2.2 ± 0.9	2.2 ± 1.1	$p_{1,2} > 0.05$; $p_{1,3} > 0.05$; $p_{2,3} > 0.05$
eGFR, ml/min	96.0 ± 8.0	96.8 ± 9.4	96.0 ± 6.8	$p_{1,2} > 0.05$; $p_{1,3} > 0.05$; $p_{2,3} > 0.05$
Glucose, mmol/l	4.7 ± 0.6	5.9 ± 1.2	5.9 ± 1.4	$p_{1,2} < 0.001$; $p_{1,3} < 0.001$; $p_{2,3} > 0.05$
NT-proBNP pg/ml	41.0 [22.8; 53.0]	47.5 [20.8; 72.0]	112.0 [34.0; 170.0]	$p_{1,2} > 0.05$; $p_{1,3} < 0.001$; $p_{2,3} < 0.05$

Note: MetS — metabolic syndrome; MetS + AF — metabolic syndrome with atrial fibrillation; BMI — body mass index; TC — total cholesterol; HDL — high-density lipoprotein; TG — triglycerides; TH — thyrotrophic hormone; eGFR — estimated glomerular filtration rate; NT-proBNP — N-terminal brain natriuretic propeptide.

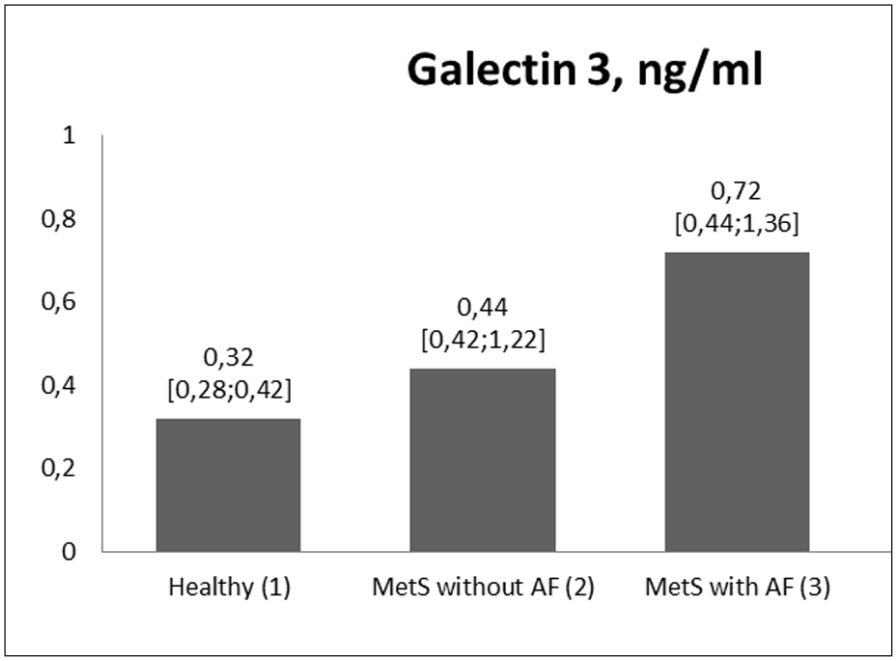
Table 2

ECHOCARDIOGRAPHY

		Healthy individuals (n = 50) Group 1	MetS (n = 50) Group 2	MetS + AF (n = 50)	p- value
LA Diam, cm		35.7 ± 2.7	Group 3	P-value	$p_{1,2} < 0.001$; $p_{1,3} < 0.001$; $p_{2,3} > 0.05$
LAV mean, mm^3		42.8 ± 9.4	69.3 ± 16.6	82.0 ± 19.4	$p_{1,2} < 0.001$;$p_{1,3} < 0.001$; $p_{2,3} < 0.001$
LAVi, mm^3/m^2		24.2 ± 4.9	36.2 ± 9.7	42.3 ± 11.2	$p_{1,2} < 0.001$; $p_{1,3} < 0.001$; $p_{2,3} < 0.05$
RAV mean, mm^3		42.0 ± 8.9	58.6 ± 14.4	64.4 ± 14.7	$p_{1,2} < 0.001$; $p_{1,3} < 0.001$; $p_{2,3} < 0.05$
RAVi, mm^3/m^2		23.8 ± 4.3	30.1 ± 7.3	33.2 ± 7.8	$p_{1,2} < 0.001$; $p_{1,3} < 0.001$; $p_{2,3} < 0.05$
RAS, cm^2		13.5 ± 2.4	18.2 ± 4.0	21.4 ± 4.1	$p_{1,2} < 0.001$; $p_{1,3} < 0.001$;$p_{2,3} < 0.001$
LVMi, g/m^2	male	83 ± 17	115 ± 30	107 ± 26	$p_{1,2} < 0.001$; $p_{1,3} < 0.001$; $p_{2,3} > 0.05$
	female	68 ± 11	104 ± 20	101 ± 18	
EF, %		65 ± 7	65 ± 6	62 ± 6	$p_{1,2} > 0.05$; $p_{1,3} < 0.05$; $p_{2,3} < 0.05$

Note: LA Diam — left atrium diameter; LAV — left atrium volume; LAVi — left atrium volume index; RAV — right atrium volume; RAVi — right atrium volume index; RAS — right atrium square area; LVMi — left ventricle mass index; EF — ejection fraction..

Figure. Galectin 3 level in patients with metabolic syndrome and atrial fibrillation



Note: MetS — metabolic syndrome; AF — atrial fibrillation; $p_{1,2} < 0.001$, $p_{1,3} < 0.001$, $p_{2,3} < 0.05$.

3 was 1.5-fold higher in patients with MetS without AF than in healthy subjects (0.44 [0.42; 1.22] and 0.32 [0.28; 0.42] ng/ml, p < 0.001) (Fig.).

We have found a wide range of concentrations of galectin 3 in the groups, especially in patients with MetS. Assuming that it might be related to the number of MetS components, we conducted additional analysis and found that galectin 3 level significantly varies depending on the number of MetS components, both in AF group and in

patient without arrhythmia. Among patients with MetS and AF the highest level of galectin 3 was detected in patients with five MetS components, and it was 5-fold higher than in patients with three components of MetS (2.87 [1.14; 6.94] and 0.53 [0.44; 0.82] ng/ml, p < 0.001). A similar pattern is found in MetS patients without AF, as shown in Table 3.

Correlation analysis showed a positive relationship between galectin 3 level and waist circumference (r = 0.57, p < 0.001), triglycerides and fasting glucose (r = 0.41; r = 0.38, p < 0.001) and a negative correlation with the level of HDL-cholesterol (r = –0.4, p < 0.001). Galectin 3 also correlated with both SBP and DBP (r = 0.51; r = 0.39, respectively, p < 0.001).

We also found a positive correlation between galectin 3 and left atrial diameter (r = 0.51, p < 0.001) and left atrial volume (r = 0.45, p < 0.001), square area (0.45, p < 0.001) and right atrium volume (r = 0.40, p < 0.001). The data are presented in Table 4.

Discussion

AF is a multifactorial disease. Risk factors for non-valvular AF are numerous, and include older

Table 3

УРОВЕНЬ ГАЛЕКТИНА 3 В СЫВОРОТКЕ КРОВИ У ПАЦИЕНТОВ С РАЗНЫМ ЧИСЛОМ КОМПОНЕНТОВ МЕТАБОЛИЧЕСКОГО СИНДРОМА

	3 components of MetS group 1	4 components of MetS group 2	5 components of MetS group 3	P-value
MetS without AF	0.42 [0.32; 0.42] N = 20	0.54 [0.44; 1.37] N = 20	2.01 [0.52; 4.59] N = 10	$p_{1,2} < 0.001$, $p_{1,3} < 0.001$ $p_{2,3} < 0.05$
MetS with AF	0.53 [0.44; 0.82] N = 20	0.63 [0.44; 1.39] N = 20	2.87 [1.14; 6.94] N = 10	$p_{1,2} > 0.05$, $p_{1,3} < 0.01$ $p_{2,3} < 0.001$

Note: MetS — metabolic syndrome; AF — atrial fibrillation.

Table 4

THE CORRELATION BETWEEN GALECTIN 3, METABOLIC SYNDROME COMPONENTS AND CARDIAC STRUCTURAL REMODELING

	r	P-value
Waist circumference	0.57	p < 0.001
Hip circumference	0.49	p < 0.001
Body mass index	0.51	p < 0.001
Total cholesterol	0.28	p < 0.001
High-density lipoprotein cholesterol	–0.40	p < 0.001
Triglycerides	0.41	p < 0.001
Glucose	0.38	p < 0.001
Systolic blood pressure	0.51	p < 0.001
Diastolic blood pressure	0.39	p < 0.001
NT-proBNP	0.31	p < 0.001
Left atrium diameter	0.51	p < 0.001
Left atrium volume	0.45	p < 0.001
Left atrium volume index	0.40	p < 0.001
Right atrium volume	0.40	p < 0.001
Right atrium volume index	0.30	p < 0.001
Right atrium square	0.45	p < 0.001
Left ventricle mass index	0.35	p < 0.001

age, male sex, thyroid disease, HTN, obesity, type 2 diabetes mellitus, obstructive sleep apnea, chronic heart failure, smoking, alcohol abuse, chronic obstructive pulmonary disease, and others. Most of these risk factors were excluded in our patients. Most of them were younger than 60 years old. Thyroid disease and chronic heart failure were excluded due to the absence of clinical manifestations and normal laboratory parameters (thyrotrophic hormone, NT-proBNP). The main difference between Mets groups was the presence of ECG verified episodes of AF. Patients with MetS, as opposed to the comparison group, had 3 or more components of MetS, the most common were HTN, abdominal obesity, and dyslipidemia.

MetS is a risk factor for AF and HTN and abdominal obesity are the most important components for the development of AF [3]. HTN and abdominal obesity are associated with the structural and hemodynamic changes leading to the development and progression of AF. HTN causes left ventricular hypertrophy and diastolic dysfunction, as well as left atrial dilation.

Left atrium volume is the most significant echocardiographic predictor of AF. We found that the volume and diameter of the atria in patients with MetS is higher than in healthy subjects. Undoubtedly, HTN and left ventricular diastolic dysfunction play the major role in the left atrium remodeling. Abdominal obesity and associated plasma volume expansion are the most important for the development of right atrium dilatation, as well as of the left one. However, AF often occurs in patients with relatively small size of the left atrium. We can assume that remodeling verified by imaging techniques is not the only factor determining the risk of AF.

Myocardial fibrosis, including atrial fibrosis, causes heterogeneity and promotes re-entry onset and AF development. Echocardiography does not enable atrial fibrosis visualization.

Galectin 3 is the indirect marker of myocardial fibrosis, it also indicates fibrosis in lungs and other internal organs. Previously, it has been shown that galectin 3 is increased in chronic heart failure. In 2014 an increase of galectin 3 in patients with AF was reported [13]. In our study we investigated the role of galectin 3 as a possible marker of myocardial fibrosis in the development of AF in patients with MetS, who are at high risk for AF development. We found that Mets patients have significantly higher level of galectin 3 compared to healthy individuals, and it is higher in those with the combination of MetS and AF compared to patients with MetS without arrhythmia. Moreover, galectin 3 level seems to be dependent on the number of MetS components: the highest levels were recorded in patients with 5 components of MetS. We also found a correlation between galectin 3 level and MetS components, such as abdominal obesity, HTN, dyslipidemia, and glucose metabolism. Galectin 3 level also correlates with left atrial volume and diameter, as well as right atrial area and volume. We assume that galectin 3 plays a role in cardiac remodeling at the molecular and cellular level that has been previously demonstrated in experimental animal models [12].

Our data suggest that galectin 3 is not only a marker of myocardial fibrosis, but may also serve as a predictor of AF occurrence in patients with MetS. This hypothesis requires a prospective study of patients with MetS without AF and high serum levels of galectin 3.

Conclusions

1. Galectin 3 serum level is higher in patients with metabolic syndrome compared to healthy individuals.

2. Patients with 5 components of MetS have higher serum levels of galectin 3 than those with less components of MetS.

3. Patients with MetS and AF have higher serum levels of galectin 3 than patients with MetS without AF.

Conflict of interest

Authors declare no conflict of interest.

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