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Original Article

Serum levels of Pentraxin-3, Vaspin, Apelin and troponin in decompensated heart failure patients

Serum pentraxin-3, vaspin, apelin ve troponinin dekompanze kalp yetersizliği hastalarındaki seviyeleri

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ABSTRACT

Aim: The aim of the present study is to evaluate the levels of these biomarkers in decompensated heart failure (HF) patients.

Material and Methods: 44 decompensated HF patients and 32 healthy individuals were enrolled in the study as patient and control groups. Transthoracic echocardiography and serum troponin I, vaspin, apelin, and PTX-3 levels besides routine laboratory analysis were performed for both groups.

Results: Troponin I and vaspin were higher, apelin was lower in patient group (for all, $p<0.05$).PTX-3 levels were higher in patient group, but it was not statistically significant ($p=0.133$). Troponin I and PTX-3 levels were significantly decreased (for both, $p<0.05$) with in-hospital-HF treatment. Vaspinand apelin levels did not show any significant change ($p=0.938$, $p=0.121$, respectively). Vaspin had an independent relationship with troponin, apelin had an independent relationship with PTX-3, troponin and apelin had independent relationship with vaspin, and finally troponin,PTX-3, and vaspin had independent relationship with apelin in decompensated HF patients.

Conclusion: Troponin I and vaspin levels were elevated,and serum apelin levels were reduced in decompensated HF patients. PTX-3 levels were higher in HF patients, but it was not statistically significant. Vaspin and apelin levels did not change with HF stabilization in HF patients, but troponin I and PTX-3 levels were significantly decreased.

Keywords: troponin I; vaspin; apelin; pentraxin-3; biomarker; decompensated heart failure

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ÖZ

Amaç: Vaspin, apelinve Pentraxin-3 (PTX-3),kardiyovasküler fonksiyonların, sıvı hemostazının, vasküler oluşumun, hücre proliferasyonunun, glukoz metabolizmasının ve inflamasyonun düzenlenmesinde etkili olduğu gösterilmiş biyomarkerlardır. Bu çalışmanın amacı, dekompanse kalp yetersizliği (KY) hastalarında bu biyobelirteçlerin düzeylerini değerlendirmektir.

Gereç ve Yöntemler: Çalışmaya 44 dekompanse KY hastası ve 32 sağlıklı birey, hasta ve control grubu olarak dahil edildi. Her iki grup için de transtorasik ekokardiyografi ve rutin laboratuvar analizlerinin yanında serum troponin I, vaspin, apelinve PTX-3 düzeyleri analiz edildi.

Bulgular: Troponin I ve vaspin hasta grubunda daha yüksekti, apelin hasta grubunda daha düşüktü (hepsi için, $p<0.05$). PTX-3 düzeyleri hasta grubunda daha fazlaydı, ancak istatistiksel olarak anlamlı değildi ($p=0.133$). Troponin I ve PTX-3 düzeyleri hastaneiçi KY tedavisi ile anlamlı olarak azaldı (her ikisi için de, $p<0.05$). Vaspin ve apelin düzeyleri anlamlı bir değişiklik göstermedi (sırasıyla $p=0.938$, $p=0.121$). Vaspin troponin ile bağımsız bir ilişkiye sahipti, apelinin PTX-3 ile bağımsız bir ilişkisi vardı, troponin veapelin vaspin ile bağımsız bir ilişkisi vardı ve son olarak troponin, PTX-3 ve vaspin dekompanse KY hastalarında apelin ile bağımsız bir ilişkiye sahipti.

Sonuç: Troponin I ve vaspin düzeyleri dekompanse KY hastalarında daha yüksekti serum apelin düzeyleri daha düşüktü. PTX-3 düzeyleri KY hastalarında daha yüksekti, ancak istatistiksel olarak anlamlı değildi. KY hastalarında vaspin ve apelin düzeyleri KY stabilizasyonu ile değişmedi, ancak troponin I ve PTX-3 düzeyleri anlamlı olarak azaldı.

Anahtar kelimeler: troponin I; vaspin; apelin; pentraxin-3; biyobelirteç; dekompanse kalp yetersizliği

Introduction

Despite improvements in diagnosis and treatment of heart failure (HF), it still has high morbidity and mortality. Ventricular dysfunction, hemodynamic overload and increased neurohumoral activation occur in HF. Increased neurohumoral activation leads to increased renal-angiotensin system activation, increased adrenergic activity, and cytokine release [1].Medical history, physical examination, electrocardiography, telegraphy, and biochemical parameters are the first step diagnostic tests which should be evaluated in HF patients. Biochemical parameters have a pivotal role in early diagnosis, early treatment and prognosis. Troponin levels are known to increase in acute HF or acute exacerbations of chronic HF [2]. There is also an increment in both BNP and ANP release due to increased myocardial tension and stress [3].

Pentraxin-3 (PTX-3) is an acute phase reactant member of the C-reactive protein (CRP) family and expressed by a variety of cell types in response to various inflammatory stimuli. PTX-3 levels were found to be higher in patients with normal ejection fraction of diastolic heart failure, even though the brain natriuretic peptide was within normal limits. It has also been emphasized that PTX-3 may be used as an indication of significant inflammation in heart failure [4].

Vaspin and apelin are found in the adipocytokine family. Apelin has been shown to be effective in regulation of cardiovascular functions, fluid hemostasis, vascular formation and cell proliferation [5]. Other studies in this area suggest that vaspin may be related to metabolic syndrome and coronary artery disease[6].

According to the current literature, there is no study in which vaspin was studied in heart failure patients. In addition, PTX-3 and apelin were found to have different results. In this study, troponin I, vaspin, apelin, and PTX-3 levels in acute exacerbations of heart failure and their changes before and after treatment were investigated.

Materials and Methods

Study population

44 patients who were admitted to cardiology clinic of our university with the diagnosis of HF (New York Heart Association [NYHA] class II-IV) and 33 healthy individuals as control group were enrolled in the study between the date of January 2009 and December 2010. This prospective case-control study has been approved by the ethics committee of the faculty of medicine. Informed consent was received from both patients and control groups included in the study. This study was conducted according to the principles of the World Medical Association Declaration of Helsinki "Ethical Principles for Medical Research Involving Human Subjects".

All patients' and control groups' medical history, anthropometric

measurements, physical examinations, transthoracic echocardiographic measurements, and laboratory tests were recorded. Additionally, they were recorded for age, sex and smoking. HF patients recieved standart medical treatmentincluding ACE inhibitors, angiotensin receptor and aldosterone blockers, and loop diuretics for the stabilization while hospitilazion. Since these patients were in the decompensated period, furosemide infusion (50 mg-100 mg/hour infusion) and beta-blocker dose titration(metoprolol 50-200 mg/day) were provided to compensate. Hypertension (HT) was defined as 140 mmHg and above for SBP, 90 mmHg and above for DBP, or usage of antihypertensive medications. Diabetes mellitus (DM) was defined as a fasting glucose value >126 mg/dL, with or without hemoglobin A1c >6.5 %, or current use of medication for DM.

Patients with reduced or preserved left ventricular ejection fraction presented with acute HF or acute exacerbation of the chronic HF were accepted in the patient group according to the current HF guidelines [7, 8]. The patients included in the study were our follow-up patients and their coronary angiography was performed at their previous hospitalization. During this hospitalization, a possible acute coronary syndrome was excluded considering the findings of electrocardiography, creatinin kinase, creatinin kinase-MB and physical examination. The control group was defined as no history ofcoronary artery disease (CAD), HT, hyperlipidemia, DM, smoking, and other causes of HF. These individuals were diagnosed as normal by physical examinations, telegrams, electrocardiography, and transthoracic echocardiography. Patients with acute coronary syndrome, uncontrolled HT, renal failure, stroke, intermittent claudication, chronic obstructive pulmonary disease, obstructive sleep apnoea syndrome,severe systemic inflammatory and hematological diseases, oral contraceptive and estrogen replacement therapy, insulin treatment, malignancy, and inadequate echocardiographic image quality were excluded from the study.

Transthoracic echocardiography

A two-dimensional transthoracic echocardiography was performed to both patient and control groups with a GE Vivid 7 device (GE Healthcare Inc. Milwaukee, Wisconsin, USA) with 6T, 5 MHz probe lying supine in the left lateral position. LVEF was calculated using Simpson's biplane method. All the patients had undergone to echocardiographic evaluation in the same hospitalization.

Laboratory and biomarker analysis

We obtained venous 12-hour fasting blood samples, and then centrifuged at 3000 rpm for 10 minutes at room temperature, processed the serum and ethylene diaminetetra-acetic acid plasma aliquots within 2 hours. Samples were stored at -80 ^{0}C for later biomarker measurements. Total cholesterol, high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C), triglyceride were measured by the technique described by Friedewald et al[9]. Urea and creatinine were studied in the same day of blood samples received. The parameters were measured in the automatic chemistry analyzer (Aeroset, Abbott Labs.).

Commercially available enzyme-linked immunosorbent assays (ELISA) were used for measuring pentraxin-3 (Human PTX-3ELISA system, Adipobioscience, USA), apelin (Apelin-36,Phonex Pharma Ceuticals, Belmond, USA), and vaspin (Vaspin, Adipobioscience, USA).Stability value of apelin was 0.05 ng/mL, the intra-assay variability was <5%, the inter-assay variability value was <14% and normal range was between 0 and 100 ng/mL.The sensitivity was determined as 0.156 ng/mL, the intra-assay variability was 4-6%, the inter-assay variability was 8-10% and the normal range was 0.312-20 ng/mL for vaspin; the intra-assay variability value was 4-6%, the inter-assay variability value was 8-10%, the normal range was 0.219-14 ng/mL, and the sensitivity was 0.02 ng/mL for PTX-3.

Statistical analysis

SPSS software 21.0 for Windows (SPSS Inc., Chicago, IL, USA) was used for all statistical analysis. Categorical variables are presented as counts and percentages. Continuous variables were evaluated for normal distribution assumption using the Kolmogorov-Smirnov and Shapiro-Wilks tests and were reported as mean plus standard deviation in brackets or median with interquartile range.

Fastingblood glucose and triglyceridewere not normally distributing. These parameters were compared by usingMann-Whitney Utest. Other parameters were compared by using Student-t test. Categorical variables such as gender, HT, CAD, smoking, DMwere compared byusing chi-square test.Troponin I, PTX-3, vaspin, and apelin were compared with first day of the hospitalization and just before discharge by using the paired-two test.

Spearman Rank correlation test was used for correlation analysis.Linear regression analysis was used to determine independent predictors in multivariate analysis. Variables with a p value <0.10 in univariate analysis were included in multivariate regression analysis. All p-values were two-sided and considered statistically significant when they were <0.05.

Results

In the patient group, 9 (20%) patients were NYHA class II, 28 (64%) patients were NYHA class III, and 7 (16%) patients were class IV. 34 (77%) patients were ischemic cardiomyopathy and 10 (23%) patients were non-ischemic cardiomyopathy. The patient group consisted of older and more male gender individuals compared with control group. BMI was higher in the control group. HDL-C, triglyceride, LDL-C, total cholesterol levels were lower in the patient group. LVEF was lower and systolic PAB was higher in the patient group. Troponin land vaspin werehigher, but apelin was lower in the patient group (for all, p<0.05).Additionally, PTX-3 levels were higher in the patient group, but it was not statistically significant (p=0.133). Other demographic, clinical and laboratory findings of the patient and control groups are shown in Table 1.

When we compared the biomarker levels of the HF patients between the first day of the hospitalization (pre-treatment) and just before the discharge (post-treatment), troponin I and PTX-3 levels were significantly decreased (for both, p<0.05) with the HF treatment. Vaspin levels did not show any significant change. Apelinlevels were increased, but it was not statistically significant (p=0.938, p=0.121, respectively) (Table 2, Figure 1).

Parameters effecting pre-treatment troponin I levels in HF patients are presented in Table 3. Vaspin, apelin, age, smoking, LVEF, FBG, total cholesterol, triglyceride, HDL-C, and LDL-C levels were significantly correlated with troponin I in the binary correlation analysis. Vaspin, smoking, and LVEF showed independent relationship with troponin I in the linear regression analysis (Table 3).

Table 1. Demographic, clinical, and laboratory findings of the patient and control groups

Variables	Patient group (n=44)	Control group (n=33)	P
Male, n (%)	25 (57)	8 (24)	<0.001
Age (years)	64.2±12.5	55.4±5.7	<0.001
BMI (kg/m2)	27.1±3.3	28.9±3.8	0.014
SBP (mmHg)	122.4±19.6	125.9±8.3	0.433
DBP (mmHg)	80.5±15.7	81.3±4.8	0.569
Heart rate (bpm/min)	84.2±15.1	78.3±9.6	0.058
CAD, n (%)	33 (75)	0	<0.001
DM, n (%)	19 (43)	0	<0.001
Hyperlipidemia, n (%)	12 (27)	0	<0.001
HT, n (%)	30 (68)	0	<0.001
Smoking, n (%)	28 (57)	0	<0.001
FBG (mg/dL)	136.5 (90.0-239.0)	93.6 (70.0-124.0)	<0.001
Total Cholesterol (mg/dL)	154.3±41.4	207.1±31.2	<0.001
HDL-C (mg/dL)	32.5±10.4	41.9±9.8	0.001
LDL-C (mg/dL)	97.0±34.5	135.7±26.4	<0.001
Triglyceride (mg/dL)	123.2 (41.2-169.6)	153.1 (33.0-175.2)	0.033
LVEF (%)	39.9±13.4	62.3±14.5	<0.001
Systolic PAB (mmHg)	44.7±18.1	22.1±11.6	<0.001
Troponin I (ng/mL)	3.15±0.95	0.02±0.01	<0.001
Pentraxin-3 (ng/mL)	5.50±2.64	4.51±1.94	0.133
Vaspin (ng/mL)	2.04±0.46	0.84±0.07	0.001
Apelin (ng/mL)	0.77±0.43	1.13±0.35	0.001

BMI: body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure; CAD: coronary artery disease; DM: diabetes mellitus; HT: hypertension; FBG: fasting blood glucose; HDL-C: high density lipoprotein cholesterol; LDL: low density lipoprotein cholesterol; LVEF: left ventricular ejection fraction; PAB: pulmonary artery pressure
Data are presented as noun (percentile), mean±standard deviation, median (interquartile range)
Mann Whitney U and Student t test were used for statistical analysis

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Table 2. Comparison of the biomarker levels between first day of the hospitalization (pre-treatment) and before the discharge (post-treatment)

Variables	Pre-treatment (n=44)	Post-treatment (n=44)	P
Troponin I (ng/mL)	3.15±0.95	2.78±0.43	0.023
Pentraxin-3 (ng/mL)	5.50±2.64	4.49±1.94	0.043
Vaspin (ng/mL)	2.04±0.46	2.06±0.53	0.938
Apelin (ng/mL)	0.77±0.43	0.95±0.64	0.121

Data are presented as mean±standard deviation, Paired two tests was used for statistical analysis

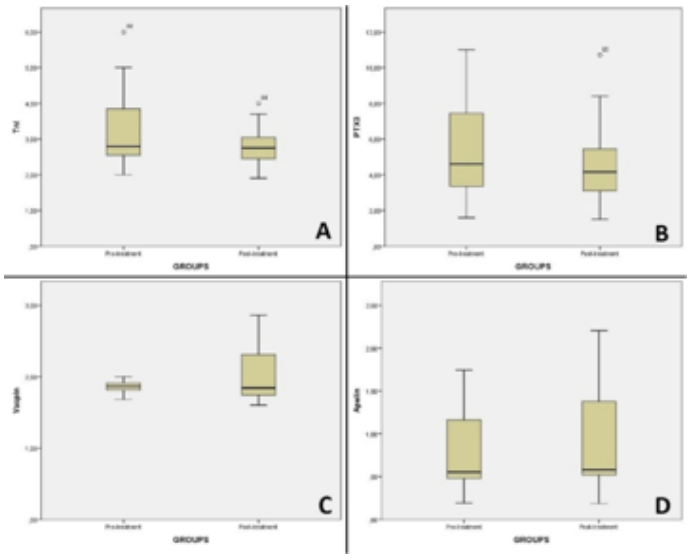

Figure 1: *Box plot analysis of compared biomarker levels of the HF patients between the first day of the hospitalization (pre-treatment) and just before the discharge (post-treatment).A: for Troponin I; B: for pentraxin-3; C: for vaspin; D: for apelin. Abbreviations: TnI: troponin I; PTX-3: pentraxin-3*

Parameters effecting pre-treatment PTX-3 levels in HF patients are presented in Table 4. Only apelin was significantly correlated with PTX-3 in the binary correlation analysis, and only apelin showed an independent relationship with PTX-3 in the linear regression analysis (Table 4).

Parameters effecting pre-treatment vaspin levels in HF patients are presented in Table 5. Troponin I,apelin, age, smoking, LVEF, FBG, HDL-C, and LDL-C were significantly correlated with vaspinin the binary correlation analysis, and these parameters showed independent relationship with vaspin in the linear regression analysis, too (Table 5).

Parameters effecting pre-treatment apelin levels in HF patients are presented in Table 6. Troponin I,PTX-3, vaspin, age, smoking, total cholesterol, HDL-C, and LDL-C were significantly correlated with apelinin the binary correlation analysis, and these parameters showed independent relationship with apelin in the linear regression analysis, too (Table 6).

Table 3. Parameters related with pre-treatment troponin levels

Variables	Rho	P	Multivariate regression	P
Pentraxin-3	-0.179	0.127		
Vaspin	0.830	<0.001	0.560	0.001
Apelin	-0.378	0.001	0.016	0.887
Age	0.394	<0.001	0.010	0.899
BMI	-0.196	0.088		
Smoking	0.389	<0.001	0.222	0.014
SBP	-0.132	0.268		
DBP	-0.137	0.235		
Heart rate	0.151	0.212		
LVEF	-0.687	<0.001	-0.430	0.014
FBG	0.597	<0.001	0.091	0.415
Total cholesterol	-0.522	<0.001	-0.457	0.751
Triglyceride	-0.213	0.063		
HDL-C	-0.309	0.006	0.227	0.544
LDL-C	-0.497	<0.001	0.413	0.716

BMI: body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure; LVEF: left ventricular ejection fraction; FBG: fasting blood glucose; HDL-C: high density lipoprotein cholesterol; LDL: low density lipoprotein cholesterol
Spearmen correlation test and linear regression analysis were used for statistical analysis

Table 4. Parameters related with pre-treatment pentraxin-3 levels

Variables	Rho	P	Multivariate regression	P
Troponin	-0.179	0.127		
Vaspin	-0.185	0.115		
Apelin	0.310	0.017	0.184	0.007
Age	-0.133	0.160		
BMI	0.068	0.564		
Smoking	-0.163	0.166		
SBP	-0.002	0.990		
DBP	-0.019	0.869		
Heart rate	-0.032	0.797		
LVEF	0.025	0.857		
FBG	-0.072	0.559		
Total cholesterol	0.071	0.549		
Triglyceride	-0.089	0.451		
HDL-C	-0.035	0.768		
LDL-C	0.119	0.373		

BMI: body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure; LVEF: left ventricular ejection fraction; FBG: fasting blood glucose; HDL-C: high density lipoprotein cholesterol; LDL: low density lipoprotein cholesterol
Spearmen correlation test and linear regression analysis were used for statistical analysis

Table 5. Parameters related with pre-treatment vaspin levels

Variables	Rho	P	Multivariate regression	P
Troponin	0.830	0.001	0.467	0.001
Pentraxin-3	0.185	0.115		
Apelin	-0.388	<0.001	-0.013	0.900
Age	0.394	<0.001	0.179	0.087
BMI	-0.193	0.093		
Smoking	0.610	<0.001	0.278	0.010
SBP	-0.032	0.790		
DBP	-0.025	0.826		
Heart rate	0.198	0.100		
LVEF	-0.668	<0.001	-0.218	0.204
FBG	0.617	<0.001	0.157	0.148
Total cholesterol	-0.494	<0.001	0.302	0.319
Triglyceride	-0.207	0.071		
HDL-C	-0.427	<0.001	-0.110	0.405
LDL-C	-0.431	<0.001	-0.230	0.406

BMI: body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure; LVEF: left ventricular ejection fraction; FBG: fasting blood glucose; HDL-C: high density lipoprotein cholesterol; LDL: low density lipoprotein cholesterol
Spearmen correlation test and linear regression analysis were used for statistical analysis

Table 6. Parameters related with pre-treatment apelin

Variables	Rho	P	Multivariate regression	P
Troponin	-0.378	0.001	-0.161	0.502
Pentraxin-3	0.310	0.007	0.150	0.189
Vaspin	-0.388	<0.001	-0.065	0.819
Age	-0.407	<0.001	-0.257	0.057
BMI	0.040	0.732		
Smoking	-0.263	0.021	-0.138	0.356
SBP	0.094	0.433		
DBP	-0.001	0.993		
Heart rate	-0.108	0.372		
LVEF	0.257	0.061		
FBG	-0.130	0.252		
Total cholesterol	0.251	0.028	-0.152	0.731
Triglyceride	0.020	0.824		
HDL-C	0.007	0.954		
LDL-C	0.291	0.010	0.299	0.455

BMI: body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure; LVEF: left ventricular ejection fraction; FBG: fasting blood glucose; HDL-C: high density lipoprotein cholesterol; LDL: low density lipoprotein cholesterol. Spearmen correlation test and linear regression analysis were used for statistical analysis, A p value under 0.05 is accepted as statistical significant

Discussion

The present study found that serum troponin and vaspin levels were higher, serum apelin levels were lower in acute decompensated HF patients than controls. PTX-3 levels were higher in acute decompensated HF patients, but these relationships were not statistically significant. With the treatment of the decompensated HF, troponin and PTX-3 levels were significantly decreased.Vaspinand apelin levels were increased, but these relationships were not statistically significant. Vaspin had an independent relationship with troponin, apelin hadan independent relationship with PTX-3, troponin and apelin had independent relationship with vaspin, and finally troponin,PTX-3, and vaspin had independent relationship with apelin in decompensated HF patients.

Higher cardiac troponin levels are associated with depressed LVEF and HF with high sensitivity and specificity. Elevated troponin levels are especially associated with non-coronary myocardial damage in HF [10]. Troponin levels may be highly traced in both acute decompensation and chronic compensation of HF. Elevated troponin levels may have a predictive value on depressed LVEF, impaired hemodynamic parameters, mortality and morbidity [11]. Left ventricular hypertrophy, myocarditis, concomitant renal insufficiency, ischemia or neurohumoral changes occur during HF process, cause myocyte integrity to be impaired, lead to elevation of troponin [12]. In the present study, troponin levels were significantly higher in acute decompensated HF patients and decreased at the end of the treatment. These findings are consistent with the results previously reported in the literature.

Inflammation, oxidative stress, and extracellular matrix remodeling have been considered to play a key role in the pathophysiology of the HF. Elevations in the blood levels of the biomarkers such as tumor necrosis factor alpha (TNF-α), C-reactive protein, interleukin 6 (IL-6) are correlated with HF severity and prognosis [13]. Recently, PTX-3 has been thought to be a marker of vascular inflammation and was found to facilitate risk stratification of HF patients. Additionally, it was considered that PTX-3 might have a pathophysiological role in the development of acute decompensated HF[14]. It was observed in the present study that PTX-3 levels did not significantly differ between patient and control groups. But, the levels of the PTX-3 were significantly decreased after the treatment of HF.

Vaspin (visceral adipose tissue-derived serine protease inhibitor) is a unique insulin-sensitizing adipokine that has been demonstrated to improve glucose tolerance and

insulin resistance in obese mice [15].A relation has been found between visceral vaspin expression in humans and their body mass indexes, body fat percentage, and plasma glucose levels [16]. Vaspin was firstly isolated from OLETF guinea pigs with type 2 DM animal models characterized abdominal obesity, insulin resistance, HT, and dyslipidemia[17].Previous studies have supposed that the effects of the vaspin were inhibiting inflammatory factors secretion from vascular smooth muscle cells, and antagonizing endothelial apoptosis induced by free fatty acid [18]. It is accepted as vaspin has anti-inflammatory and anti-atherosclerotic properties via the enhancement of eNOS expression and activity, decrease of ADMA level, increase expression of dimethylarginine dimethylamino hydrolase 2, decrease of TNF-alpha, increase migration and proliferation of EPCs [19]. Actually, vaspin protects endothelial cells from inflammation and apoptosis. In our study, serum vaspin levels were significantly higher in DCHF patients than controls, and there was no significant change with the stabilization treatment. Our study may be the first to demonstrate that HF patients exhibited significant higher vaspin levels compared to healthy controls. As a result of previous studies data, it may be thought that vaspin release may be increased in order to suppress inflammation as a contra-regulated mechanism in HF characterized by chronic inflammation, to decrease increased blood glucose level and insulin resistance.

Apelin is an adipocyte affecting the APJ receptor system found in the heart, lung, kidney, vascular endothelium, and fatty tissue. The apelin/APJ system is a novel nuerohormonal pathway that is widely represented in the heart and is an important regulator of cardiovascular hemostasis. Apelin is a strong endogenous inotrope that causes nitric oxide dependent vasodilatation, reduces ventricular preload and afterload, increases coronary blood flow and cardiac contractility [20]. Moreover, it has an antagonistic effect on the renin angiotensin aldosterone system which plays an important role in the development and progression of HF [21]. In a previous study, apelin level found to be increased at early and acute stage of HF, but progressively decreased in the advanced stages of the HF. Additionally, its predictive role has been studied in several studies [22, 23]. In our study, lower serum levels of apelin were observed in the HF patients in parallel with the previous studies.

Our study has some limitations. Relatively small number of patients was consisted in the study. Comparison of the serum markers vaspin and apelin levels with the known markers of HF such as brain natriuretic peptide, or other inflammation parameters such as C-reactive protein were not done. Baseline clinical differences of the groups may be another limitation. Additionally, diuretic tolerance and baseline significant dosage differences in the usage of the medical treatment (ACE inhibitors, angiotensin receptor blockers, aldosterone blockers, and loop diuretics) could have independently influenced biomarker levels. However, the medical treatment in the hospital for stabilization was standard to all HF patients.

Conclusion

We measured the serum troponin I and vaspin levels elevated,and serum apelin levels reduced in decompensated HF patients. Decompensated HF patients tended to have higher levels of troponin I and PTX-3. No significant change was observed in vaspin and apelin levels with the acute HF stabilization. Finally, more multicenter studies with a large population derived from different countries must be performed to confirm that these parameters could be considered as a universal risk factor or marker like natriuretic peptides in HF patients, especially with vaspin and PTX-3.

Declaration of conflict of interest

The authors received no financial support for the research and/or authorship of this article. There is no conflict of interest.

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