Usefulness of Serum Omentin-1 Levels for the Prediction of Adverse Cardiac Events in Patients with Hypertrophic Cardiomyopathy

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Significance of the Study

- This study investigated the association between serum omentin-1 levels and adverse cardiac events in patients with hypertrophic cardiomyopathy (HCM). Serum omentin-1 levels were lower in patients with HCM than in healthy controls and were associated with adverse cardiac events during the 2-year follow-up. Therefore, the serum omentin-1 level could serve as a biochemical marker to predict the prognosis of patients with HCM.

Keywords

Omentin · Hypertrophic cardiomyopathy · Adverse cardiac events

Abstract

Objective: To investigate the association between serum omentin-1 levels and adverse cardiac events in patients with hypertrophic cardiomyopathy (HCM). **Subjects and Methods:** This prospective, observational study included 87 patients with HCM and 50 age- and sex-matched control subjects. Serum omentin-1 and brain natriuretic peptide (BNP) levels were measured in all subjects, using enzyme-linked immunosorbent assay and electrochemiluminescence, respectively. Patients with HCM were divided into 2 groups ac-

cording to their omentin levels, i.e., low: ≤ 291 ng/mL ($n = 48$) and high: > 291 ng/mL ($n = 39$). Cardiac mortality, hospitalization due to heart failure, and implantable cardioverter-defibrillator (ICD) implantation were considered adverse cardiac events. Statistical analysis included uni- and multivariate logistic regression, receiver-operating characteristic (ROC) analysis, and the Kaplan-Meier method. **Results:** Serum omentin-1 levels were significantly lower in the obstructive (253.9 ± 41.3 ng/mL) and nonobstructive (301.9 ± 39.8 ng/mL) HCM groups than in the control group (767.1 ± 56.4 ng/mL), $p < 0.001$, respectively. The BNP levels were higher in the obstructive and nonobstructive HCM groups than in the control group (269.5 ± 220 , 241.0 ± 227 , and 24.0 ± 18.9 pg/mL, respectively, $p < 0.001$). The Kaplan-Meier analysis indicated that patients with low omentin-1 levels showed a

significantly higher (48.2%) 2-year cumulative incidence of overall adverse cardiac events than those with high omentin-1 levels (16.2%) (log-rank test, $p = 0.001$). In the multivariate logistic regression analysis, omentin-1, interventricular septum (IVS) thickness, and male gender were independent predictors of adverse cardiac events in the follow-up. **Conclusion:** Omentin-1 levels were lower in patients with HCM than in the control group, and this was associated with worse cardiac outcomes.

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Introduction

Hypertrophic cardiomyopathy (HCM) is the most common genetic cardiovascular disease, affecting approximately 1 in every 500 adults (0.2%) [1]. It is inherited in an autosomal dominant manner, and is characterized by a wall thickness ≥ 15 mm in ≥ 1 left ventricular (LV) myocardial segments (that cannot be explained by abnormal overloading conditions) [2]. HCM is a myocardial disease that presents with LV hypertrophy, myocardial fibrosis, and myocardial irregularity [3]. In previous studies, it has been shown that chronic inflammation decreases myocardial contractility and induces hypertrophy, apoptosis, and fibrosis [4, 5]. The natural course of HCM is highly variable, ranging from being asymptomatic throughout life, to progressive heart failure (HF), and premature sudden cardiac death (SCD) early in life [6].

Schaffler [7] identified omentin as an anti-inflammatory adipokine that is predominantly expressed and secreted by visceral adipose tissue rather than by subcutaneous adipose tissue. Omentin plays a pivotal role in adipose tissue differentiation, maturation, metabolism, immune response regulation, inflammation, and insulin resistance [8, 9]. Negative and positive correlations between serum levels of omentin-1 and various conditions have been reported [10–12]. Huang et al. [13] reported a negative correlation between nonischemic dilated cardiomyopathy and circulating levels of omentin-1. Narumi et al. [14] reported that decreased serum levels of omentin-1 were associated with poor cardiac outcome in patients with HF.

However, whether or not serum omentin-1 levels are associated with clinical outcomes in patients with HCM remains unclear. Therefore, the objective of this study was to investigate the relationship between serum levels of omentin-1 and cardiac prognosis in patients with HCM.

Subjects and Methods

Patients and Study Protocol

This prospective, observational study included 87 patients with HCM and 50 age- and sex-matched controls without HCM admitted to the Cardiology Outpatient Clinic, Bagcilar Training and Research Hospital, Istanbul, Turkey, in the period March 2012 to January 2015. The patients with HCM were followed for 2 years.

HCM was diagnosed based on the definition of the European Society of Cardiology, i.e., a wall thickness of ≥ 15 mm in ≥ 1 LV myocardial segments, as measured by any imaging technique that is not explained solely by loading conditions [2]. Exclusion criteria were coronary artery disease ($n = 4$), a symptoms-consistent New York Heart Association (NYHA) class of >3 ($n = 4$), cardiac valvular disease ($n = 4$), uncontrolled hypertension (a blood pressure of $>140/90$ mm Hg with or without medications; $n = 7$), active or chronic inflammatory disease ($n = 3$), aortic valve stenosis ($n = 2$), and severe renal failure (estimated glomerular filtration rate of <30 mL/min/m²; $n = 1$). Thirty-seven subjects were enrolled in the obstructive HCM group, 50 in the nonobstructive HCM group, and 50 in the control group. The medical history, risk factors, a family history of SCD, medication use, and NYHA class of all the subjects involved in the study were recorded. Body mass index (BMI) was calculated (by G.C., G.A., and S.S.). Each patient was assessed based on physical examinations and electrocardiography done every 3 months, and 24-h ambulatory electrocardiography was performed at least once (by S.S.Y., İ.S., S.S., S.C., and İ.İ.A.). Patients who reported symptoms of chest pain, dyspnea, palpitation, dizziness, and syncope were reevaluated using echocardiography and 24-h ambulatory electrocardiography; based on the outcome, treatment was planned in an outpatient or inpatient setting. An implantable cardioverter-defibrillator (ICD) was implanted (by S.S.Y., G.A., and K.K.) in patients who had survived cardiac arrest due to ventricular fibrillation or sustained ventricular tachycardia that caused syncope or hemodynamic compromise. In the follow-up, 2 cardiologists (H.K. and G.C.), who were blinded to the data of blood biomarkers, reviewed the medical records of the patients and made phone calls to patients or their relatives to find out if cardiovascular events had occurred. Rate of hospitalization, syncope, status according to NYHA class, mortality, and arrhythmic events were recorded. Hospitalizations due to HF, ICD implantation, and cardiac death were considered as adverse cardiac events.

The study was performed in accordance with the recommendations of the Declaration of Helsinki on Biomedical Research Involving Human Subjects. The Bagcilar Training and Research Hospital Ethics Committee approved the study protocol and written informed consent was obtained from each patient.

Echocardiography Study

Two-dimensional (2-D), M-mode, and Doppler echocardiographic studies based on the recommendations of the American Society of Echocardiography/European Association of Echocardiography [15] were performed using an ultrasonography machine (IE33; Philips Medical Systems, Andover, MA, USA). The maximum wall thickness measured at any border of the LV was considered the maximal thickness. The diameter of the left atrium (LA), diastolic interventricular septum (IVS), and LV posterior wall thickness were recorded from the parasternal short- and long-axis views. The peak velocity of the early (E) and late (A) waves and

Table 1. The clinical, demographic, and biochemical characteristics of the study population

	Obstructive HCM (n = 37)	Nonobstructive HCM (n = 50)	Control (n = 50)	p value
Age, years	40.9±12.1	36.6±13	37.7±9.6	0.35
Male/female, n	20/17	25/25	24/26	0.81
Hypertension	5 (13.5)	3 (10.3)	3 (8.3)	0.3
Hypercholesterolemia	4 (10.8)	5 (10.0)	4 (8.0)	0.3
Family history of HCM	7 (18.9)*	9 (18.0)*	0 (0)	0.005
Family history of SCD	2 (5.4)*	2 (4.0)*	0 (0)	0.1
Previous AF	5 (13.5)	6 (12.0)	0 (0)	0.13
BMI	25.7±3.4	25.2±3.2	24.0±3.4	0.1
Fasting blood glucose, mg/dL	98.3±24.6	95.9±15.7	89.9±13.6	0.32
HbA _{1c} , %	5.6±0.7	5.5±0.4	5.3±0.3	0.26
Creatinine, mg/dL	0.85±0.2	0.82±0.2	0.81±0.1	0.08
TG, mg/dL	135.0±18.0	140.0±18.0	136.0±17.0	0.09
LDL-C, mg/dL	130.0±21.0	136.0±25.0	129.0±21.0	0.4
HDL-C, mg/dL	45.0±8.0	43.0±8.0	47.0±9.0	0.08
BNP level, pg/mL	269.5±224.4*	241.0±217.6*	24.0±18.9	<0.001
Omentin-1 level, ng/mL	253.9±41.3*, #	301.9±39.8*	767.1±56.4	<0.001
Medication				
Amiodarone	9 (24.3)*, #	5 (10.0)*	0 (0)	0.005
Warfarin	4 (10.8)	4 (8.0)	0 (0)	0.13
β-Blocker	31 (83.8)*, #	29 (58.0)*	0 (0)	<0.001
ACE-I/ARB	4 (14.3)	2 (6.9)	2 (5.6)	0.09

Data are expressed as mean ± SD or n (%), unless otherwise indicated. ACE-I, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; AF, atrial fibrillation; BNP, brain natriuretic peptide; HCM, hypertrophic cardiomyopathy; HbA_{1c}, glycated hemoglobin; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; SCD, sudden cardiac death; TG, triglycerides. * $p < 0.01$ versus control group, # $p < 0.05$ versus group 2.

the E/A ratio were calculated from the transmitral flow velocity, using an apical 4-chamber view and positioning the sample volume at the tip of the mitral leaflets during diastole. The septal early diastolic velocity (e') of the mitral annulus from the apical 4-chamber view was measured using tissue Doppler imaging. The LV diastolic function was defined as the ratio of E to e' velocity (E/ e'). The LV volume and ejection fraction were computed using the biapical Simpson rule. The dynamic LV outflow tract (LVOT) pressure gradient was measured using continuous-wave Doppler in the apical views, either while resting or while being provoked like in the Valsalva maneuver. LVOT obstruction was defined as the presence of a peak instantaneous LVOT gradient of ≥ 30 mm Hg. All echocardiographic examinations were recorded and analyzed at the end of the study by 2 independent, experienced echocardiographers (I.I.A. and S.S.), who were blinded to the subjects' clinical characteristics and levels of serum omentin-1 and brain natriuretic peptide (BNP).

Laboratory Measurements

Peripheral venous blood samples were collected from the antecubital veins of all the subjects after 12 h of fasting, and centrifuged at 3,000 rpm for 10 min. The serum was then separated and stored at -80°C until analysis. Levels of serum fasting blood glucose, creatinine, blood urea nitrogen, triglycerides (TG), total cholesterol

(TC), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C) were measured using standard laboratory techniques (Hitachi 7600 Automatic Biochemical Analyzer, Hitachi Co., Japan). Serum levels of omentin-1 were determined using a commercially available enzyme-linked immunosorbent assay (ELISA) kit with high sensitivity and specificity for detecting human omentin-1 (Sunred Biotechnology Co., Shanghai, China). Serum BNP levels were assessed using the electrochemiluminescence ECLIA method (Roche Cobas 8000 Modular Analyzer System, Roche Diagnostics, UK).

Statistical Analysis

Statistical analysis was performed using SPSS v21 software (SPSS Inc., Chicago, IL, USA). Data are reported as mean ± SD for continuous variables. Categorical variables are reported as percentages. The normality assumption was evaluated using the Kolmogorov-Smirnov test. Continuous variables in the 3 groups were compared using a one-way analysis of variance or the Kruskal-Wallis test. Continuous variables were compared between 2 groups using the independent-samples t test or the Mann-Whitney U test. Categorical data were compared using the χ^2 or Fisher exact test. Univariate logistic regression analysis was performed, and the variables with a p value < 0.1 were then entered into a backward stepwise multivariate logistic regression model to assess the

Fig. 1. In the ROC curve analysis, an omentin-1 level ≤ 291 ng/mL had a sensitivity of 69% and a specificity of 75% in predicting adverse events in patients with hypertrophic cardiomyopathy (AUC 0.780, 95% CI 0.56–0.85, $p = 0.009$).

Fig. 2. In the Kaplan-Meier curve analysis, the rate of cardiac adverse events was significantly higher in the low omentin-1 group than in the high omentin-1 group (log-rank test, $p < 0.001$).

independent predictors of adverse cardiac events in HCM. Event-free survival curves were generated using the Kaplan-Meier method. Differences in survival curves among the low and high omentin-1 subgroups were assessed using the log-rank test. Receiver-operating characteristics (ROC) analysis was used to determine the sensitivity, specificity, and positive and negative predictive values of serum levels of omentin-1 for adverse cardiac events during the long-term follow-up. Based on the ROC analysis, we divided the patients into 2 groups according to cut-off values as follows: a low omentin (≤ 291 ng/mL) and high omentin (> 291 ng/mL). A post hoc power analysis was performed with a sample size of 137, using the mean values and SDs of the omentin levels of the subjects in the patient and control groups and an α level set at $p < 0.05$. The effect size of the study population was 0.80, and the power of the study was 0.93. $p < 0.05$ was considered statistically significant.

Results

The clinical, demographic, and laboratory findings of the patients are given in Table 1. No significant differences were found among the groups regarding BMI, hypertension, and hypercholesterolemia. Levels of creatinine, glycated hemoglobin, LDL-C, HDL-C, TG, and fasting glucose were similar in both groups. Serum levels of omentin-1 levels were significantly lower in the obstructive

and nonobstructive HCM groups than in the control group (253.9 ± 41.3 , 301.9 ± 39.8 , 767.1 ± 56.4 ng/mL, respectively, $p < 0.001$). Furthermore, the obstructive HCM group had the lowest serum levels omentin-1 between groups (253.9 ± 41.3 vs. 301.9 ± 39.8 vs. 767.1 ± 56.4 ng/mL, $p < 0.001$). The level of BNP was significantly higher in the obstructive and nonobstructive HCM groups than in the control group (269.5 ± 224.4 , 241.0 ± 217.6 , and 24.0 ± 18.9 pg/mL, respectively, $p < 0.001$). However, no significant difference in the serum level of BNP was found between obstructive and nonobstructive HCM.

Using ROC curve analysis, omentin levels of ≤ 291 ng/mL were predictive of adverse cardiac events in HCM patients, with 75% specificity and 69% sensitivity (area under the curve [AUC] 0.780, 95% confidence interval [CI] 0.56–0.85, $p = 0.009$; Fig. 1). Characteristics of patients with low and high omentin levels are shown in Table 2. At baseline, no difference in LV systolic and diastolic diameters, posterior wall thickness, LVOT peak gradient, and LV ejection fraction were found between the low and high omentin groups (Table 2). The patients with low serum levels of omentin-1 had a thicker IVS (2.8 ± 0.5 vs. 2.0 ± 0.6 cm, $p < 0.001$) and larger LA (3.9 ± 0.4 vs. 3.3 ± 0.5 cm, $p < 0.001$) than the high omentin group. In addition, the E/e' ratio was significantly higher ($p < 0.001$) and

Table 2. The clinical, demographic, and echocardiographic characteristics of the study population according to low and high serum omentin-1 levels

	Low omentin ≤291 ng/mL (<i>n</i> = 48)	High omentin ≥291 ng/mL (<i>n</i> = 39)	<i>p</i> value
Age, years	40.3±12.4	36.5±10.5	0.12
Male/female, <i>n</i>	28/20	22/17	0.92
Body mass index	26.4±4.1	25.8±3.4	0.16
Family history of SCD	3 (6.2)	1 (2.5)	0.01
Atrial fibrillation	6 (12.5)	5 (12.8)	0.68
NYHA class >1	14 (29.2)	4 (10.3)	0.004
Syncope	10 (20.8)	7 (18.0)	0.07
Heart failure	14 (29.2)	6 (15.4)	0.02
Nonsustained VT	11 (22.9)	9 (23.1)	0.9
Sustained VT	4 (8.3)	3 (7.7)	0.6
Hospitalization	18 (37.5)	5 (12.8)	<0.01
ICD implantation	9 (18.7)	3 (7.7)	0.02
Mortality	3 (6.2)	1 (2.5)	0.04
Adverse cardiac events	23 (47.9)	7 (17.9)	<0.001
Ejection fraction, %	62.3±4.2	61.3±3.8	0.38
IVS thickness, cm	2.8±0.5	2.0±0.6	<0.001
PW thickness, cm	1.9±0.3	1.9±0.4	0.9
LA diameter, cm	3.9±0.4	3.3±0.5	<0.001
LV diastolic diameter, cm	4.5±0.4	4.6±0.4	0.43
LV systolic diameter, cm	3.07±0.4	3.13±0.4	0.63
LVOT peak gradient, mm Hg	36±18	32±20	0.5
E wave, cm/s	68.9±16.6	75.7±6.5	<0.001
A wave, cm/s	77.4±16.3	63±14	<0.001
Septal e' wave, cm/s	6.0±2.4	9.2±2.5	<0.001
E/e' ratio	13.2±3.9	9.0±3.0	<0.001

Data are expressed as mean ± SD or *n* (%), unless otherwise indicated. A wave, peak late transmitral filling velocity; E' wave, peak early diastolic mitral annulus velocity on tissue Doppler imaging; E wave, peak early transmitral filling velocity; ICD, implantable cardioverter defibrillator; LA, left atrium; LV, left ventricle; LVOT, left ventricular outflow tract; IVS, interventricular septum; NYHA, New York Heart Association; PW, posterior wall; SCD, sudden cardiac death; VT, ventricular tachycardia.

the septal e'-wave ($p < 0.001$) and E/A ratio ($p < 0.001$) significantly lower in the low omentin group than in the high omentin group.

During 24 months of follow-up, sustained and non-sustained ventricular tachycardia (detected using Holter monitoring) as well as syncope were found to be similar in the low omentin and high omentin groups (4 [8.3%], 3 [7.7%], $p = 0.6$; 11 [22.9%], 9 [23.1%], $p = 0.9$; 10 [20.8%], 7 [18.0%], $p = 0.07$, respectively; Table 2). The number of patients with NYHA class >1 and the rate of HF were higher in the low omentin group than in the high omentin group (14 [29.2%], 4 [10.2%], $p < 0.01$, and 14 [29.2%], 6 [15.4%], $p = 0.02$, respectively). Hospitalizations due to HF, ICD implantation, and cardiac mortality rates were higher in the low omentin group than in the high omentin group (18 [37.5%], 5 [12.8%], $p < 0.01$; 9 [18.7%], 3 [7.7%],

$p = 0.02$; and 3 [6.2%], 1 [2.5%], $p = 0.04$; respectively). The composite end point of hospitalization due to HF, ICD implantation, and cardiac mortality occurred in 23 (47.9%) patients in the low omentin group and 7 (17.9%) patients in the high omentin group ($p < 0.001$). Kaplan-Meier curves revealed that the low omentin group had a significantly higher prevalence (48.2%) of adverse cardiac events than the high omentin group (16.2%) (log-rank test, $p < 0.001$; Fig. 2).

The univariate logistic regression analysis revealed that serum levels of BNP, the LVOT peak gradient, the IVS thickness, male sex, a family history of SCD, and serum levels of omentin-1 showed a trend ($p < 0.1$) towards an association with adverse cardiac events in HCM. The multivariate logistic regression analysis demonstrated that serum levels of omentin-1, IVS thickness, and male

Table 3. Univariate and multivariate analysis of HCM patients for predicting adverse cardiac events

	Univariate		Multivariate	
	HR (95% CI)	<i>p</i> value	HR (95% CI)	<i>p</i> value
Omentin-1 level	14.3 (4.96–35.1)	<0.001	5.10 (3.5–9.9)	<0.01
BNP level	5.1 (1.81–8.06)	0.03		
Creatinine	1.78 (0.54–5.83)	0.34		
Hypertension	0.69 (0.32–1.83)	0.43		
LVOT gradient	4.6 (2.03–9.1)	0.03		
IVS thickness	11.1 (3.9–30.9)	<0.001	3.85 (2.2–8.1)	0.02
NYHA class >1	0.94 (0.20–4.32)	0.93		
Male gender	7.36 (1.2–15.7)	0.01	2.57 (1.8–7.7)	0.02
A family history of SCD	4.04 (1.38–11.8)	0.01		
Age	0.99 (0.96–1.04)	0.93		

HR, hazard ratio; CI, confidence interval; BNP, brain natriuretic peptide; HCM, hypertrophic cardiomyopathy; IVS, interventricular septum; LVOT, left ventricular outflow tract; NYHA, New York Heart Association; SCD, sudden cardiac death.

sex were independent predictors of a poor prognosis for HCM (hazard ratio [HR] 5.10, 95% CI 3.5–9.9, $p < 0.01$; HR 3.85, 95% CI 2.2–8.1, $p = 0.02$; and HR 2.57, 95% CI 1.8–6.7, $p = 0.02$; respectively; Table 3).

Discussion

In this study, serum levels of omentin-1 were lower in patients with HCM than in controls. HCM patients with lower serum omentin-1 levels (≤ 291 ng/mL) had a larger LA, higher LVOT gradient, thicker IVS, and greater diastolic dysfunction than HCM patients with higher serum omentin-1 levels (> 291 ng/mL). A serum level of omentin-1 ≤ 291 ng/mL was a predictor of a poor prognosis, and involved cardiac mortality, hospitalization due to HF, and ICD implantation during the 24-month follow-up, with a sensitivity of 69% and specificity of 75%. A negative correlation was observed between serum levels of omentin-1 and BNP in patients with HCM. To our knowledge, there is no study in the literature investigating the association between serum levels of omentin-1 and adverse cardiac events in patients with HCM.

Hypertrophy of the myocardium, diminished coronary blood flow, microvascular dysfunction, and chronic inflammation all act together in the pathophysiology of HCM, ultimately leading to myocardial fibrosis which is the central pillar of the disease [16–18]. In previous studies, C-reactive protein, TNF- α , and IL-6, which are all inflammatory biomarkers in HCM, were found to be

related to the pathogenesis of the disease [17, 19]. In addition, BNP, as a biomarker of wall tension, and myocardial fibrosis have been shown to increase in patients with HCM compared to normal subjects [20].

We did not study inflammatory biomarkers in our study, as our main objectives were to analyze the serum levels of omentin-1 and BNP and investigate whether these levels had an effect on cardiac events. We found that, compared to controls, BNP levels were elevated 11-fold and 10-fold in patients with obstructive and nonobstructive HCM, respectively.

Adipose tissue performs many endocrine functions via its production and secretion of various bioactive peptides, called adipokines [21]. Recent studies have shown that some adipokines play an active role in preventing cardiovascular diseases owing to their anti-inflammatory, antioxidant, and antiapoptotic properties [22, 23]. Omentin-1 belongs to this group of adipokines, and is mainly produced in the stromal vascular cells of visceral adipose tissue [24]. It is negatively correlated with chronic inflammation, endothelial dysfunction, arterial stiffness, atherosclerosis, and calcification [25, 26]. Recently, Narumi et al. [14] showed that low omentin-1 (but not BNP) was an independent predictor of cardiac events in patients with HF due to different etiologies in a 1-year follow-up. Likewise, Wang et al. [27] reported that serum levels of omentin-1 were reduced in patients with ischemic HF, and they found a negative correlation between the serum level of omentin-1 and the severity of HF.

In our study, we also found that decreased omentin-1 was associated with poor prognosis for patients with HCM. Patients with a higher NYHA class had lower serum concentrations of omentin-1 than those with a lower NYHA class, a finding which could indicate that reduced omentin-1 might be associated with HF severity. Although serum levels of omentin-1 were not affected by LVOT gradient, patients with low omentin-1 had greater IVS thickness and worse diastolic dysfunction than the high omentin group.

We speculate that low serum omentin levels correlate negatively with cardiac filling pressures, LV diastolic dysfunction, and abnormal LV wall stress. We also suggest that patients with low serum omentin-1 levels have higher myocardial inflammation and fibrosis. Thus, in our study, serum omentin-1 was an independent predictor of adverse cardiac events. However there was no association between serum BNP and cardiac outcomes. Omentin-1 could contribute to the anti-inflammatory process that leads to the slowing down of myocardial fibrosis and the prevention of future adverse cardiac events in patients with HCM. Therefore, serum omentin levels may be considered for the diagnosis of unfavorable LV remodeling which predicts adverse cardiac events.

The limitations of this study were its relatively small sample size and it being conducted at a single center. In addition, cardiac MRI was not used to detect the presence and the extent of myocardial fibrosis. Also, concentrations of other adipocytokines and inflammatory markers were not measured. Future studies with larger sample sizes are required to elucidate the relationship between circulating levels of omentin-1 and the prognosis of patients with HCM.

Conclusion

In this study, serum levels of omentin were lower in patients with obstructive and nonobstructive HCM, and this was associated with worse cardiac outcomes. Therefore, serum omentin-1 might be a new biomarker for predicting myocardial fibrosis and the development of adverse cardiac events in patients with HCM.

Disclosure Statement

There were no conflicts of interest.

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