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Evaluation of omentin levels in patients with unstable angina pectoris, non-ST elevated myocardial infarction (NSTEMI) and STEMI

Kararsız angina pektoris, ST elevasyonlu miyokard enfarktüsü (STEMI) ve Non-STEMI olan hastalarda omentin düzeylerinin değerlendirilmesi

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Abstract

Aim: Acute coronary syndrome (ACS) is an ischemic cardiac disease that could result in myocardial necrosis with prolonged ischemia. Omentin (intelectin-1) is a new biomarker that is released from adipose tissue. It is associated with coronary artery disease (CAD) and has an acute ischemic injury-reducing effect. This study aimed to assess the omentin levels in patients with unstable angina pectoris (USAP), Non-ST segment elevation myocardial infarction (NSTEMI), and ST-segment elevated myocardial infarction (STEMI).

Methods: This case-control prospective study included 59 patients with ACS and 22 healthy subjects. MB fraction of creatine kinase (CKMB), troponin, myoglobin, and omentin levels were measured from venous blood obtained from each patient within six hours after the onset of symptoms. Plasma omentin levels were determined with an omentin enzyme-linked immunosorbent assay kit.

Results: The patient group was older than the control group (P<0.05) but there was no difference between the groups in terms of gender (P>0.05). The rate of smoking was higher in the patient group, and the patient group was heavier than the control group (P>0.05). Omentin levels were similar in ACS patients and control subjects (6.0 (1.7) vs. 6.3 (1.3), P=0.40). There was no significant correlation among CKMB, troponin, myoglobin, and omentin levels. Moreover, omentin levels were similar in ACS subgroups (P=0.58). There was no significant correlation between body mass index and omentin levels (r =-0.186, P=0.09).

Conclusion: This study revealed that there was no significant relationship between omentin and myoglobin levels in ACS patients. The potential usefulness of blood concentrations of omentin levels in understanding the relationship with ACS warrants further studies.
Keywords: Omentin, Acute coronary syndrome, CKMB, Troponin, Myoglobin

Öz

Amaç: Çalışma vaka-kontrol şeklinde dizayn edildi. Akut koroner sendrom (AKS), uzun süreli iskemi sonrası miyokard nekrozu ile sonuçlanabilen iskemik bir kalp hastalığıdır. Omentin (intelectin-1), yağ dokusundan salınan yeni bir biyobelirteçtir. Omentin koroner arter hastalığı (CAD) ile ilişkilidir ve akut iskemik hasarı azaltıcı etkiye sahiptir. Bu çalışma, kararsız anjina pektoris (USAP), ST elevasyonsuz miyokard enfarktüsü (NSTEMI) ve ST elevasyonlu miyokard enfarktüsü (STEMI) olan hastalarda omentin düzeylerini değerlendirmeyi amaçlamıştır.

Yöntemler: Bu çalışmaya prospektif olarak 59 AKS hastası ve 22 sağlıklı birey dahil edildi. Semptomların başlamasından sonraki altı saat içinde her hastadan alınan venöz kandan kreatin kinaz MB formu (CKMB), troponin, miyoglobin ve omentin düzeyleri ölçüldü. Plazma omentin seviyeleri, bir omentin enzimine bağlı immünosorban test kiti ile belirlenmiştir.

Bulgular: Hasta grubu kontrol grubundan daha yaşlıydı (P<0,05) ancak cinsiyet açısından gruplar arasında fark yoktu (P>0,05). Hasta grubunda sigara içme oranı daha fazlaydı ve hasta grubu kontrol grubuna göre daha ağırdı (P>0,05). AKS hastalarında ve kontrol grubunda omentin düzeyleri benzerdi (6,0 (1,7) ve 6,3 (1,3); P=0,40). CKMB, troponin, miyoglobin ve omentin düzeyleri arasında anlamlı bir ilişki yoktu. CKMB, troponin, miyoglobin ve omentin düzeyleri arasında anlamlı bir korelasyon yoktu, ayrıca AKS alt gruplarında omentin düzeyleri benzerdi (P=0,58). Vücut kitle indeksi ile omentin düzeyleri arasında anlamlı bir ilişki yoktu (r = -0,186, P=0,09).

Sonuç: Bu çalışma AKS hastalarında omentin ve miyoglobin düzeyleri arasında anlamlı bir ilişki olmadığını ortaya koymuştur. Omentin düzeylerinin kan konsantrasyonlarının AKS ile ilişkisinin anlaşılmasındaki potansiyel faydası daha fazla çalışmaya ihtiyaç duyar.
Anahtar kelimeler: Omentin, Akut koroner sendrom, CKMB, Troponin, Miyoglobin

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Introduction

Ischemic heart disease is the main instigator of mortality worldwide [1]. Acute Coronary Syndrome or ACS is described as a conglomeration of various signs and symptoms that results due to an imbalance between myocardial oxygen supply and demand [2]. ACS can also be identified as an ischemic cardiac situation that can lead to both myocardial damage and necrosis in correlation with a protracted duration of ischemia. ACS represents the most damaging clinical expression of coronary artery disease (CAD), in which the pathophysiological mechanism is initiated by plaque rupture. ACS can be categorized into three types: ST elevated myocardial infarction (STEMI), unstable angina, and non-ST elevated myocardial infarction (NSTEMI). The most frequently used biomarkers in the diagnosis of ACS are CKMB, troponin, and myoglobin [3].

Many adipokines such as leptin, adiponectin, and visfatin are released from adipocytes in adipose tissue and function as an endocrine organ. These adipokines play physiological and pathophysiological roles in many systems in the body, including the cardiovascular system [4]. Omentin (intelectin-1) is a hydrophilic protein that is released from the adipose tissue, consists of 313 amino acids, has a molecular weight of 35kDa, and it is also a new biomarker in the good adipokine category. It is released from vascular stromal cells in adipose tissue, as well as from epicardial adipose tissue and endothelial cells [5]. Its anti-inflammatory effects have been reported in the literature [6]. These effects of omentin occur with different cellular signal pathways such as cyclooxygenase-2 (COX-2), endothelial nitric oxide synthase (eNOS), and nitric oxide (NO) [7]. Omentin levels significantly decrease in obesity, insulin resistance, and diabetes mellitus [8].

It is known that omentin plays a protective role against arterial calcification, and that low omentin levels are related to the development of atherosclerosis [9, 10]. In a previous study, it has been reported that omentin is strictly related to coronary artery disease (CAD), and it is a new biomarker which determines the presence of CAD [11]. In their study, Zhong et al. [12] showed that their patients in their study group, who were diagnosed with stable angina pectoris (SAP) and acute coronary syndrome (ACS), had lower serum omentin levels compared to the control group. Omentin levels during admission are observed to correlate with the severity of CAD in patients who have metabolic syndrome presenting with AMI [13]. Moreover, in a study, it has been demonstrated that omentin reduces acute ischemic damage in myocardial tissue by preventing myocyte apoptosis [14].

In the current study, our aim was to analyze and assess the omentin levels in patients diagnosed with unstable angina pectoris (USAP), Non-ST segment elevation myocardial infarction (NSTEMI), and ST-segment elevation. Our second objective was to analyze the correlation between omentin levels and myoglobin levels in patients with ACS. By a comprehensive literature review, we surmise that this is the first time that the relationship between omentin levels and myoglobin in ACS patients has been investigated in a study.

Materials and methods

The current case-control and prospective study included 59 patients who were admitted to Kocaeli University, Medical Faculty, Emergency Services with angina and diagnosed with ACS between December 2018 and December 2019. Twenty-two healthy individuals were selected to serve as a control group for this study. A cardiologist examined all of the subjects, and information on medical histories was obtained via a questionnaire. All of the enrolled patients had to undergo electrocardiography (ECG) within one hour of their admission. The exclusion criteria included patients with valvular heart disease, coronary artery bypass graft surgery, hematological disorders, malignant disease, acute or chronic infection, chronic heart failure (class III and IV), pharmacological immunosuppressive or glucocorticoid therapy, and decreased systolic function (ejection fraction <45%).

ACS patients were categorized into three distinct groups: STEMI, NSTEMI, and UAP. Twenty-nine patients were diagnosed with STEMI based on their clinical symptoms, including electrocardiogram evidence (ST elevation in two or more leads), coronary angiography findings (occlusion of a main coronary artery branch), significantly increased serum troponin-I levels (more than twice the upper limit of normal), and CKMB. Eighteen patients were diagnosed with STEMI based on their clinical symptoms including ST-segment depression, or marked T wave inversion on ECG, and with biomarkers of myocardial necrosis above the normal levels. Twelve patients were diagnosed with UAP based on having ischemic chest pain at rest within the preceding 48 hours or within the past month, with transient ST-T segment depression, or T wave inversion, and normal serum levels of CKMB and troponin.

Before conducting a primary percutaneous coronary intervention (PCI), STEMI patients were treated with clopidogrel 600 mg, aspirin 300 mg, and intravenous heparin (70 – 100 U/kg). Beta-blockers and statins were administered to the STEMI patients after PCI. NSTEMI patients were treated with aspirin (300 mg), low-molecular-weight heparin, statins, clopidogrel (initially 300 mg and subsequently 75 mg), and beta-blockers at the time of admission. Coronary angiography was performed with the Judkins technique within three hours from the initial chest pain symptoms in patients with STEMI by experienced cardiologists, according to the guidelines for coronary angiography and percutaneous coronary intervention of the American Heart Association. Angiography was performed within three days in NSTEMI patients. UAP patients were followed up medically, and angiography was evaluated according to the exercise test or myocardial scintigraphy results. All angiography was conducted utilizing a Philips (Integris BH 5000, Philips, Netherlands) coronary angiography equipment. Selective coronary angiograms, conducted with the femoral approach by utilizing 6-French (F) and 7-F Judkins catheters, were evaluated by quantitative analysis (AET-met S.P.A. Italy QCA). Luminal stenosis, which was more than 50% in any of the coronary vessels, was considered CAD.

Hypertension (HT) was defined as having systolic blood pressure greater than or equal to 140 mmHg, and diastolic blood pressure greater than or equal to 90 mmHg, or a condition requiring the use of any antihypertensive medications. The

diagnosis of Type 2 Diabetes Mellitus (DM) was made according to the criteria set by the American Diabetes Association. Smoking was defined as an active or prior use of tobacco for greater than 10 pack/years. Body mass index (BMI) was calculated by dividing the body weight in kilograms by the square of the height value. Before the initiation of the study, all of the patients signed their informed consent forms. This study was conducted according to the principles of the Declaration of Helsinki and approved by the local Ethics Committee of the Kocaeli University Hospital (Ethic no: 053, Date: 18/05/2012).

The levels of troponin I, MB fraction of creatine kinase (CKMB), myoglobin, creatinine, low-density lipoprotein-cholesterol (LDL), high-density lipoprotein-cholesterol (HDL), total cholesterol (TC), triglyceride, C-reactive protein (CRP), sedimentation rate, and whole blood count were measured using the venous blood samples that were obtained from each patient within 6 hours after the onset of the first symptoms. The cubital vein was used for obtaining the peripheral venous blood samples and the samples were collected into heparinized tubes, and subsequently, for the separation of the plasma, the cells were centrifuged at a rate of 3000 rpm for a duration of 10 minutes and then stored at a temperature of −80°C until the day of biochemical analysis. CK-MB and cardiac TnI levels were measured in the serum by Simens ADVIA Centaur Cp analyzers in the emergency laboratory. The measurement of the Myoglobin levels was conducted with a sandwich ELISA immunoassay using an anti-myoglobin monoclonal antibody and an anti-myoglobin polyclonal antibody. The determination of the plasma omentin levels was achieved with the use of an omentin enzyme-linked immunosorbent assay (ELISA) kit (Bio Vendor, NC, USA). The intra-assay and inter-assay coefficients of variation of this kit were 4.1% and 4.8%, respectively.

Statistical analysis

Data were analyzed with SPSS software version 25.0 for Windows (SPSS Inc, Chicago, Illinois). Kolmogorov-Smirnov test was used to verify that continuous variables were normally distributed. Normally distributed variables were expressed as mean (standard deviation (SD)), while non-normally distributed variables were expressed as median with interquartile range (IQR). The categorical variables were presented as percentages. The differences between the two groups were evaluated with Student's unpaired t-test for parameters with a normal distribution. In multiple comparisons, one-way analysis of variance (ANOVA) test followed by the Tukey post hoc test was used for the normally distributed continuous data, while the nonparametric Kruskal-Wallis test was used to analyze continuous, non-normally distributed data. The frequencies of nominal variables were compared using Fisher's exact test or chi-square test. Pearson and Spearman's tests were used to examine correlations between continuous variables. Statistical significance was defined as $P<0.05$.

Results

The study population's demographic and clinical data are provided in Table 1. In the study, there were 22 healthy individuals (14 males, 8 females) who constituted the control group, and 59 ACS patients who constituted the patient group (41 males, 18 females). Patients were older than the control group, but the two groups were similar in terms of gender distribution. The rate of smoking was higher in the patient group, and the patient group was heavier in weight compared to the control group. While TC, LDL, triglyceride levels were higher in the patient group, HDL level was higher in the control group. CRP levels and sedimentation rates, both of which are markers of inflammation, were higher in the patient group. While hemoglobin was higher in the control group, WBC was higher in the patient group. However, no significant difference was observed in platelet values between the groups. Omentin levels were similar in the control and patient groups (6.3 (1.3) vs. 6.0 (1.7); $P=0.40$). Figure 1 shows the values of omentin between groups. Troponin, CKMB, and myoglobin parameters are provided in Table 1.

Table 1: Demographic and clinical data of the study population

	Control (n=22)	Patients (n=59)	*P*-value
Age (years)	31.2(13.1)	59.9(12.2)	<0.001
Male/Female, n	14/8	41/18	0.60
BMI (kg/m^2)	24.2(2.2)	27.7(3.4)	<0.001
Smoking n(%)	3(13%)	26(44%)	0.001
Hypertension n(%)	-	46(77%)	-
Diabetes mellitus n(%)	-	21(35%)	-
Total cholesterol (mg/dl)	135.9(25.2)	183.8(36.6)	<0.001
Low density lipoprotein (mg/dl)	76.3(16.7)	118.6(33.3)	<0.001
High density lipoprotein (mg/dl	42.9(5.9)	37.5(8.7)	0.009
Triglyceride (mg/dl)	111.6(21.1)	137.6(58.3)	0.04
C-reactive protein (mg/L)	1.2(0.7)	2.3(0.7-5.4)	0.02
Creatinine (mg/dl)	0.7(0.2)	1.0(0.7-1.2)	0.001
Sedimentation rate (mm/hr)	10.0(5.5)	23.4(16.6)	<0.001
White blood cell count (10^3/mm^3)	5.7(1.4)	9.1(2.6)	<0.001
Platelet count (10^3/mm^3)	239.3(42.4)	265.5(72.8)	0.11
Hemoglobin (g/dL)	13.9(0.9)	12.8(1.7)	0.007
Omentin (ng/ml)	6.3(1.3)	6.0(1.7)	0.40
Troponin (ng/ml)	-	5.7(8.0)	-
CKMB (ng/ml)	-	83.0(9.0-234.0)	-
Myoglobin (ng/mL)	-	268.0(91-656)	-

BMI: Body mass index, CKMB: Creatine kinase-MB

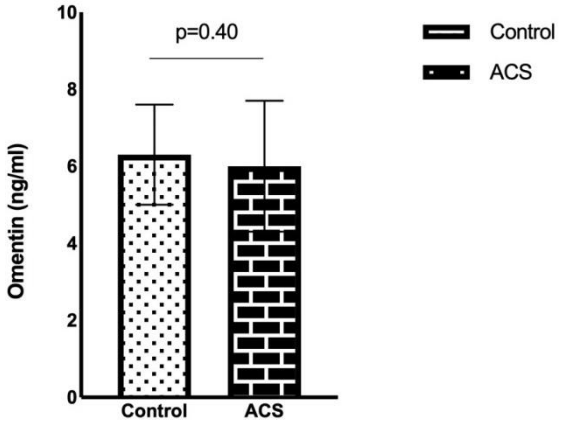

Figure 1: Omentin levels between study groups

A comparison of ACS subgroups and the control group are shown in Table 2. No statistically significant difference was observed in age, gender, BMI, HT, DM, and smoking rates within the ACS subgroups. Smoking rates were significantly higher in the STEMI group compared to the control group. While TC and LDL levels were similar in ACS subgroups, they were higher compared to the control group. The lowest HDL levels were observed in the NSTEMI group, and it was significantly different from the control group. Triglyceride levels were similar among ACS subgroups and the control group. While the sedimentation rate was higher in ACS subgroups, CRP levels were similar among the study groups. WBC, hemoglobin, and platelet counts were similar in the ACS subgroups.

The levels of omentin were similar in the USAP, NSTEMI, STEMI, and control groups ($P=0.58$). Figure 2 shows the levels of omentin in the ACS subgroups and the control

group. Troponin, CKMB, and myoglobin levels were highest in the STEMI group and lowest in the USAP group. In correlation analysis, no significant relationship was observed among omentin, troponin, CKMB, and myoglobin levels (Table 3).

Furthermore, no statistically significant correlation was found between Omentin levels and BMI. The correlation between omentin and myoglobin was shown in Figure 3 (r=-0.017 *P*=0.881).

Table 2: Clinical and demographic data of the control and acute coronary syndrome subgroups

	Control (n=22)	USAP (n=12)	NSTEMI (n=18)	STEMI (n=29)	*P*-value
Age (years)	31.2(13.1)	62.3(10.1)[a]	61.7(12.6)[a]	57.8(12.7)[a]	<0.001
Male/Female, n	14/8	8/4	12/6	21/8	0.92
BMI (kg/m^2)	24.2(2.2)	28.0(3.7)[a]	27.9(3.9)[a]	27.4(3.2)[a]	0.001
Smoking n(%)	3(13%)	4(33%)	5(28%)	17(56%)[a]	0.007
Hypertension n(%)	-	10(83%)	16(89%)	20(69%)	0.24
Diabetes mellitus n(%)	-	5(41%)	8(44%)	8(27%)	0.44
Total cholesterol (mg/dl)	135.9(25.2)	172.9(31.7)[a]	176.9(37.6)[a]	192.5(37.0)[a]	<0.001
Low density lipoprotein (mg/dl)	76.3(16.7)	116.8(32.4)[a]	111.8(35.1)[a]	123.6(33.0)[a]	<0.001
High density lipoprotein (mg/dl	42.9(5.9)	39.4(11.)2	34.9(8.4)[a]	38.3(7.7)	0.02
Triglyceride (mg/dl)	111.6(21.1)	131.1(55.9)	147.6(67.9)	134.1(54.1)	0.17
C-reactive protein (mg/L)	1.2(0.7)	1.6(0.4-5.3)	3.4(3.6)	2.7(1.1-4.9)	0.07
Creatinine (mg/dl)	0.7(0.2)	1.2(0.5)[a]	1.2(0.8-1.4)[a]	0.9(0.3)[b,c]	0.001
Sedimentation rate (mm/hr)	10.0(5.5)	17.5(10.0-27.5)[a]	17.0(12.3-30.0)[a]	23.0(10.5-36.5)[a]	0.001
White blood cell count (10^3/mm^3)	5.7(1.4)	8.5(1.9)[a]	8.5(2.9)[a]	9.7(2.7)[a]	<0.001
Platelet count (10^3/mm^3)	239.3(42.4)	291.6(87.2)	274.6(62.0)	249.0(71.0)	0.09
Hemoglobin (g/dL)	13.9(0.9)	12.8(2.0)	12.4(1.4)[a]	13.1(1.8)	0.02
Omentin (ng/ml)	6.3(1.3)	5.7(0.8)	5.8(1.1)	6.2(2.2)	0.58
Troponin (ng/ml)	-	0.10(0.10-0.19)	0.80(0.2-3.2)[b]	6.9(2.4-17.5)[b,c]	<0.001
CKMB (ng/ml)	-	2.3(1.1)	33.0(14.8-129.5)[b]	169.0(97.0-408.5)[b,c]	<0.001
Myoglobin (ng/mL)	-	28.0(13.0-54.5)	278.0(136.8-403.8)[b]	543.0(216.5-900.0)[b,c]	<0.001

USAP: Unstable angina pectoris, NSTEMI: Non-ST segment elevation myocardial infarction, STEMI: ST segment elevation myocardial infarction, BMI: Body mass index, CKMB: Creatine kinase-MB, [a] Control vs Other groups, [b] USAP vs. NSTEMI/STEMI, [c] NSTEMI vs. STEMI

Table 3: Correlation analyses between Omentin and CKMB, Troponin and Myoglobin

	Omentin	
	r	*P*-value
CKMB	-0.083	0.463
Troponin	-0.095	0.397
Myoglobin	-0.017	0.881
BMI	-0.186	0.09

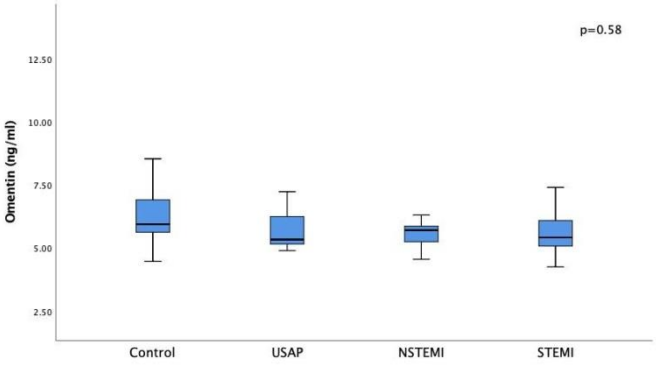

Figure 2: Omentin levels between acute coronary syndrome subgroups and control group

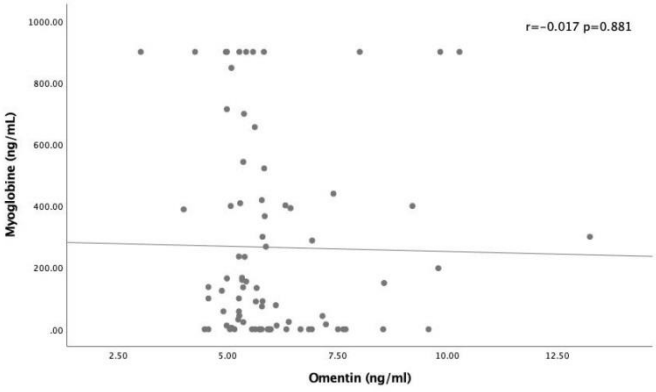

Figure 3: The correlation relationship between omentin and myoglobin levels

Discussion

In the current study, omentin levels were similar among ACS patients and healthy subjects. There was no significant correlation between the omentin and ischemic biomarkers such as CKMB and troponin. As far as we know, this is the first study in the literature to investigate the relationship between omentin levels and myoglobin in ACS patients. Moreover, we presented that no association exists between omentin and myoglobin levels in patients with ACS.

Recent studies have determined that there is an association between omentin levels and inflammatory responses. Turkcu et al. [15] reported that while serum omentin levels decrease, the TNF-α/omentin ratio and TNF-α levels increase in patients with Behcet's disease. The low levels of serum omentin values can predict adverse cardiac events in patients with heart failure (HF). It has also been suggested that omentin is a new prognostic marker in the risk classification of patients with HF. Serum omentin levels have been shown to decrease in cases of oxidative stress and chronic inflammation in HF patients [16]. Moreover, low serum omentin levels are associated with many metabolic risk factors such as high blood pressure, glucose tolerance, dyslipidemia, and increased navel environment [17]. The inflammation-reducing effects of omentin occur through signaling pathways such as Cyclooxygenase-2 (COX-2), endothelial nitric oxide synthase (eNOS), nitric oxide (NO), and nuclear factor kappa B (NF-κB) [18]. Omentin activates the AMP-activated protein kinase (AMPK), which reduces endothelial inflammation through E-selectin inhibition [7]. AMPK also activates eNOS, which has a vasodilating effect. Activated eNOS blocks the c-Jun N-terminal protein kinases (JNK) activation, which plays a role in the activation of inflammation through TNF-α-mediated COX2 induction in the cell [18]. Omentin has an antiangiogenic effect by inhibiting Akt and NF-κB signaling pathways. So, AMPK- and Akt-dependent mechanisms show acute ischemic damage reducing effects in the myocardial tissue [7]. A recent study demonstrated that omentin activated the Akt – eNOS signaling pathway in order to improve the revascularization and endothelial cell function in response to ischemia [19].

The production of pro- and anti-inflammatory adipocytokines from epicardial adipose tissue (EAT) could change in certain pathological situations [20]. Adiponectin expression decreases, and proinflammatory adipocytokine expressions such as IL-6 and TNF-alpha increases in patients with CAD [21]. So, it is accepted that these adipocytokines released from the EAT via vasocrine and paracrine routes regulate the atherosclerotic process [22]. Endothelial dysfunction is associated with subclinical atherosclerosis. Omentin shows anti-atherogenic effects through increasing endothelial NO and by reducing inflammation and oxidative stress. In a study by Yamawaki et al. [23], it was reported that omentin can inhibit vasoconstriction by endothelium-dependent mechanisms in mice aorta. Moreno-Navarrete et al. [24] suggested that the levels of plasma omentin are a marker indicating endothelial function due to their relationship with endothelial vasodilation. Omentin is inversely associated with CAD, and it was reported that omentin can be utilized as a biomarker to diagnose CAD [10, 11].

Previous studies reported that low omentin levels existed in coronary endothelium, epicardial adipose tissue, and serum in patients with CAD [25, 26]. Omentin levels positively correlated with BMI, serum interleukin-6, systolic blood pressure, hemoglobin-A1c (glycosylated hemoglobin), and total cholesterol levels, and negatively correlated with HDL and adiponectin levels [12, 27]. It has been determined that omentin levels predict adverse cardiac events regardless of the severity of angiographic lesions [17].

Previous studies presented that omentin levels were significantly lower in DM patients with ischemic heart disease. Another study also suggests that ACS patients have lower omentin levels [12]. Omentin has a reducing effect on myocyte apoptosis in ischemic tissue by suppressing AMPK- and Akt-dependent mechanisms [14]. Omentin also decreases the vascular smooth muscle cell proliferation and neointimal formation after arterial injury [28]. Moreover, it improves myocardial damage and myocardial functions in patients with STEMI [14]. Time-dependent kinetic changes in omentin levels have been associated with early improvement of ejection fraction and negative remodeling in patients with anterior STEMI. Because omentin ameliorates the myocardial damage, it was considered a cardioprotective acute phase reactant in ACS patients. High omentin levels and low CKMB levels were observed in ACS patients at the time of admission [29]. Du et al. [25] demonstrated that omentin levels were lower in CAD, and the amount of omentin expression was lower in adipose tissue around the stenotic coronary artery than the adipose tissue surrounding the non-stenotic coronary artery. However, there was no difference in the expression of adiponectin levels in the adipose tissue around the stenotic and non-stenotic coronary arteries. Omentin level was significantly lower, and IL-6 level was significantly higher in ACS patients compared to stable angina pectoris (SAP) group and the control group. Furthermore, omentin was a CAD predictor, and it also negatively correlated with IL-6 [12].

In the present study, omentin levels were similar between ACS patients and healthy subjects.

Several reports indicated that omentin levels were lower in ACS patients. These reports suggest that the principal reason for low omentin levels was the reduced release of omentin into plasma [25]. Harada et al. [30] presented that a low omentin level was associated with EAT volume in CAD patients, but this relationship was not significant. In this study, we thought that omentin levels were similar in study groups due to the similar EAT thickness, volume, or mRNA expression of omentin in EAT. However, we did not evaluate EAT thickness, volume, and mRNA expression of omentin. It has previously been shown in patients with ACS that there was an inverse correlation between omentin levels and BMI [31]. In our study, no significant correlation was observed between omentin levels and BMI, and it could explain the similar omentin levels between the study groups. Furthermore, no statistically significant relationship was observed among omentin, CKMB, troponin, and myoglobin levels. These non-significant correlations could be explained by the timing of the blood analyses, which were conducted within six hours after the observation of the initial symptoms. If serial measurements were made, a significant correlation could be observed. So, further studies need to be conducted to verify these findings.

Limitations

One of the significant limitations of our study was that the study group was limited to patients who were diagnosed with ACS. Hence, the findings of this study cannot be generalized to all patients with atypical symptoms, stable angina pectoris, and CAD. The relatively limited number of patients could limit the strength of results and conclusions obtained from this study. Although we measured the CKMB, troponin, myoglobin, and omentin levels within 6 hours of admission, we did not perform serial measurements of these parameters. Future investigations with more number of patients are needed to evaluate the CKMB, troponin, myoglobin, and omentin measurement levels for patients presenting with ACS. The other limitation of the study was that the ischemic modified albumin, which is an ischemia biomarker, was not measured and compared with omentin levels. Moreover, the effect of omentin on prognosis in ACS patients was not evaluated in this study. Despite these limitations, our study is still essential as it provides significant results for further studies about CKMB, troponin, myoglobin, and omentin levels in patients with ACS.

Conclusion

In conclusion, our data reveal that levels of omentin were similar in patients with ACS and healthy subjects. In the study, no statistically significant correlation was found between Omentin, CKMB, and Troponin levels. This is thought to be the first study in the literature to investigate the relationship between omentin levels and myoglobin in ACS patients and hence it may shed light on for future ACS diagnosis options. For ACS patients, no significant relationship was observed between omentin and myoglobin levels. Hence, the potential usefulness of blood concentrations of omentin levels in understanding the relationship with ACS needs to be examined with further studies.

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