Relationship between circulating serum omentin-1 levels and nascent metabolic syndrome in patients with hypertension

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Accepted 20 October 2021
Published Online First 2 December 2021

To cite: Cetin Sanlialp S, Nar G, Nar R. *J Investig Med* 2022;**70**:780–785.

ABSTRACT

The prevalence of metabolic syndrome (MetS) is more common in patients with hypertension and is associated with an increased risk of target organ damage and/or cardiovascular disease (CVD). Omentin-1 is a beneficial adipokine considered to play a role in MetS and MetS-related states such as obesity, diabetes, and coronary artery disease. The aim of this study was to determine the relationship between circulating omentin-1 levels and MetS uncomplicated by diabetes or CVD (nascent MetS) in patients with hypertension. In this study, 110 patients (54 men, 49%; average age: 49.72±11.32 years) treated for hypertension but without overt diabetes and/or CVD were enrolled. 66 patients were stratified into MetS (+) (group 1) and 44 patients into MetS (–) (group 2) according to the American Heart Association/National Heart, Lung, and Blood Institute criteria. The triglyceride glucose (TyG) index was used to assess insulin resistance. Circulating omentin-1 levels in venous blood samples were measured by an ELISA kit. Circulating omentin-1 levels in patients with MetS were significantly lower than in patients without MetS (46.35 ng/mL (42.70–57.70 ng/mL) vs 130.95 ng/mL (62.83–236.48 ng/mL), p<0.001). Omentin-1 was inversely correlated with TyG index (r=–0.204, p=0.033). In a multivariate logistic regression analysis, omentin-1, TyG index, and body mass index were independent predictors of MetS. A receiver operating characteristic curve analysis determined that the best cut-off value for omentin-1 in predicting MetS was 62.20 ng/mL and the area under the curve was 0.880 (95% CI 0.817 to 0.942, p<0.001). The findings of this study suggest that circulating omentin-1 levels are inversely related to the presence of MetS and may be a reliable marker to predict the development of MetS in patients with hypertension.

Significance of this study

What is already known about this subject?

- Omentin-1 is a new adipokine with a molecular weight of 35 kD secreted by the visceral adipose tissue, endothelial cells, and visceral fat stromal vascular cells.
- Omentin-1 plays a critical role in regulating glucose metabolism and increasing insulin sensitivity.
- Previous studies have reported that circulating omentin-1 levels were lower in obesity, diabetes, metabolic syndrome (MetS), and cardiovascular disease (CVD).

What are the new findings?

- Despite growing evidence on suppression of omentin-1 secretion in MetS, the findings of recent studies conducted on patients with MetS uncomplicated by diabetes and/or CVD (nascent MetS) are conflicting.
- The number of studies investigating its role in specific patient populations at high risk of MetS is also limited.

How might these results change the focus of research or clinical practice?

- The prevalence of MetS is higher in patients with hypertension and its presence usually indicates target organ damage (TOD) and/or CVD in this population.
- Future cardiovascular events and TOD should be prevented by identifying high-risk candidates for MetS.
- In this study, the presence of MetS clearly decreased circulating omentin-1 levels in patients with hypertension, and omentin-1 was an independent predictor of MetS.
- The study findings suggest that omentin-1 may play a role in further promoting MetS in patients with hypertension and may be a reliable marker to identify patients at high risk of MetS.

INTRODUCTION

Metabolic syndrome (MetS) is a cluster of metabolic abnormalities induced by cardiometabolic, proinflammatory, and prothrombotic factors.[1] It has become a major health problem today, affecting approximately 20%–30% of the adult population, and is associated with an increased risk of diabetes, cardiovascular disease (CVD), and mortality.[2] Although the exact mechanisms responsible for the etiopathogenesis are not fully understood, inflammation, ectopic fat accumulation, oxidative stress, and insulin resistance (IR) seem to be key players in this process.[3] Recently, Jialal *et al*[4] used the term nascent MetS to describe patients with MetS without diabetes and/or CVD complications

and reported abnormal secretion of adipokines, chemokines, and cytokines in this population.[5]

Omentin-1 is an adiponectin secreted mainly from the visceral adipose tissue, endothelial cells, and visceral fat stromal vascular cells and has a beneficial effect on glucose metabolism. It has been suggested that abnormalities in omentin-1 secretion may be associated with IR, inflammation, endothelial dysfunction, and CVD.[6 7] Previous studies have demonstrated lower circulating omentin-1 levels in patients with obesity, diabetes, impairment of endothelial function, carotid atherosclerosis, and CVD.[8–13] In light of clinical study findings, it can be thought that omentin-1 has a critical role in MetS and MetS-related states. However, there are limited data on the relationship between circulating omentin-1 levels and nascent MetS, defined as the early stage of MetS.[4 14] The effects of circulating omentin-1 levels on nascent MetS and on adverse changes in adipose tissue biology should be clearly elucidated in specific populations at high risk of MetS, such as patients with hypertension, since reports support that target organ damage (TOD) or CVD is more common in hypertensive patients with MetS compared with those without.[15 16] Thus, in this study, we aimed to investigate the relationship between circulating omentin-1 levels and nascent MetS in patients with hypertension.

MATERIALS AND METHODS

Study population

This cross-sectional study was conducted in a population of 110 patients treated for hypertension in our cardiology outpatient clinic. The diagnosis of hypertension was made according to the 2018 hypertension European Society of Cardiology and the European Society of Hypertension guidelines.[17] The American Heart Association/National Heart, Lung, and Blood Institute criteria (waist circumference (WC) ≥102 cm in men or ≥88 cm in women; systolic blood pressure ≥130 mm Hg and/or diastolic blood pressure ≥85 mm Hg or being on antihypertensive drug treatment; triglycerides (TG) ≥150 mg/dL or being on drug treatment; high-density lipoprotein cholesterol (HDL-C) <40 mg/dL in men or <50 mg/dL in women or being on drug treatment; fasting plasma glucose ≥100 mg/dL or being on drug treatment) were used to diagnose MetS.[1] Patients meeting at least three of these criteria were included in group 1 (MetS+) and those not meeting the criteria in group 2 (MetS−). Prior CVD or cerebrovascular disease, severe kidney or liver disease, history of type 1 or type 2 diabetes, left ventricular systolic dysfunction (ejection fraction <50%), malignancy, acute or chronic infection, and antidiabetic/fibrate/steroid drug use were defined as the exclusion criteria. At admission, patients with systolic blood pressure ≥180 mm Hg or diastolic blood pressure ≥110 mm Hg, fasting glucose ≥126 mg/dL or glycated hemoglobin >6.5%, and TG >500 mg/dL were also excluded from the study.

All patients were informed before participating in the study and a written consent form was obtained from each.

Clinical data and laboratory assay

Demographic and clinical characteristics of each patient were recorded in the hospital registry system. Anthropometric measurements, including height, weight, and WC, were performed by the same clinician blinded to patient data during the physical examination. Weight and height measurements were performed while patients were wearing light clothes and no shoes. Body mass index (BMI) was calculated using the formula (weight/height^2). WC was measured in standing position using a plastic tape measure at the level midway between the lower rib margin and the iliac crest. The WC to height ratio (WHtR) was obtained by dividing the WC by the height. After a 5–10 min rest period in sitting position, blood pressure was measured two times on the right arm at the heart level with an automated monitor, and the average of the two blood pressures taken at a 5 min interval was recorded.

Venous blood samples were collected after 8–12 hours of fasting. Blood glucose, lipid parameters, glomerular filtration rate (GFR), C reactive protein (CRP), and complete blood count were evaluated by standard laboratory techniques. The triglyceride glucose (TyG) index, a novel marker of IR, was calculated using the following formula: ln [fasting TG (mg/dL)×fasting blood glucose (mg/dL)/2].[18] The blood samples reserved for omentin-1 were centrifuged at 3000× *g* for 10 min and stored at −80°C. An ELISA kit (Sunred, cat. 201-12-0156; Shanghai Technology, China) was used to analyze serum omentin-1 concentrations. Absorbance was measured at 450 nm with Biotek Elx800 microplate reader (measurable range: 6–1500 ng/mL). All samples were tested in duplicate and omentin-1 concentrations were determined according to the standard curve obtained via the five-parameter curve-fitting equation.

Statistical analysis

All statistical data were analyzed using SPSS V.21.0 software. Kolmogorov-Smirnov test was used to control distribution normality. Mean±SD or median (Q1–Q3 quartiles) was used to express continuous variables. Categorical variables were presented as numbers and percentages. Continuous variables were compared using either Student's t-test or Mann-Whitney U test. Differences between categorical variables were determined using χ^2 test. The relationship between omentin-1 and other continuous variables was evaluated by Pearson's correlation analysis. A multivariate logistic regression analysis was performed using a backward stepwise method to identify independent variables for MetS. A receiver operating characteristic (ROC) curve analysis was done to determine the best cut-off value for omentin-1 in predicting MetS. A two-tailed $p<0.05$ was considered statistically significant.

RESULTS

Basic demographic and clinical data of the study population are presented in table 1. There were no statistically significant differences between the groups with regard to age, gender, current smoking, and ejection fraction (all $p>0.05$). Antihypertensive drugs used by patients in group 1 were as follows: renin-angiotensin system (RAS) blockers, 42 patients; calcium channel blockers, 18 patients; beta-blockers, 9 patients; and diuretics, 6 patients. In group 2, 27 patients were receiving RAS blockers, 13 patients were receiving calcium channel blockers, 8 patients were receiving beta-blockers, and 5 patients were receiving diuretic therapy. In anthropometric measurements, BMI was significantly higher in group 1 than in group 2 ($p=0.049$). On the other hand, WC and WHtR measurements did not show significant difference between the groups. Both systolic and diastolic blood pressure levels were significantly

Table 1 Basic demographic and clinical characteristics of the study population

Variables	All population (N=110)	Group 1 (n=66)	Group 2 (n=44)	P value
Age (years)	49.72±11.32	49.08±10.56	50.68±12.45	0.469
Male, n (%)	54 (49)	33 (50)	21 (47.7)	0.815
BMI, kg/m^2	28.40±4.82	29.18±5.20	27.24±3.96	0.049
WC (cm) (IQR)	92 (84–102)	93.5 (84.75–104.25)	90 (84–99.50)	0.153
WHtR	0.56±0.07	0.57±0.08	0.54±0.06	0.132
Current smoking, n (%)	37 (33.6)	22 (33.3)	15 (34)	0.934
EF (%) (IQR)	65 (60–65)	65 (60–65)	65 (60.25–65)	0.374
SBP (mm Hg)	124.27±5.02	128.98±5.39	115.95±4.92	0.032
DBP (mm Hg)	76.96±3.89	80.48±5.52	74.50±3.43	0.046

BMI, body mass index; DBP, diastolic blood pressure; EF, ejection fraction; SBP, systolic blood pressure; WC, waist circumference; WHtR, waist circumference to height ratio.

elevated in group 1 compared with group 2 (p=0.032 and p=0.046, respectively).

In laboratory parameters, significant differences were found between the groups in serum TG, HDL-C levels, and TG:HDL-C ratio (all p<0.05). However, serum blood glucose, GFR, total cholesterol, low-density lipoprotein cholesterol (LDL-C), CRP, hemoglobin levels, LDL-C:HDL-C ratio, and white cell count were similar (table 2). Serum omentin-1 concentrations were significantly lower in group 1 compared with group 2 (46.35 ng/mL (42.70–57.70 ng/mL) vs 130.95 ng/mL (62.83–236.48 ng/mL), p<0.001) (table 2 and figure 1). When the entire population was divided into three groups (normal, overweight, and obese) according to BMI, serum omentin-1 concentrations showed a significant difference between normal and obese patients in both groups (group 1, p=0.03; group 2, p=0.048, respectively). On the other hand, serum omentin-1 concentrations were not affected by gender (p=0.485 and p=0.760, respectively). Additionally, the TyG index, a novel marker of IR, was significantly higher in group 1 patients (8.96±0.53 vs 8.62±0.45, p=0.001, respectively) (figure 1).

The correlation between circulating omentin-1 levels and other variables is shown in table 3. Circulating omentin-1 levels were significantly associated with BMI (r=−0.169, p=0.038), systolic blood pressure (r=−0.284, p=0.024), diastolic blood pressure (r=−0.194, p=0.043), TG (r=−0.189, p=0.048), HDL-C (r=0.265, p=0.046), TG:HDL-C ratio (r=−0.201, p=0.035), and TyG index (r=−0.204, p=0.033).

A multivariate logistic regression analysis performed to predict the independent predictors of MetS and a backward stepwise method were used. Variables with p<0.25 between groups in the univariate analysis but not showing multicollinearity were included in the model (omentin-1, TyG index, BMI, TG:HDL-C ratio, CRP). Variables used directly as MetS diagnostic criteria were also excluded. In the final analysis, circulating omentin-1 levels, TyG index, and BMI were determined to be independent predictors of MetS (table 4). ROC curve analysis revealed that the best cut-off value for circulating omentin-1 levels in predicting MetS was 62.20 ng/mL, with 82% sensitivity and 80% specificity, and the area under the curve was 0.880 (95% CI 0.817 to 0.942, p<0.001) (figure 2).

DISCUSSION

We summarize our study findings as follows: (1) Patients with MetS had significantly lower circulating omentin-1 levels compared with those without MetS. (2) Omentin-1 showed a significant inverse correlation with IR estimated through the TyG index. (3) In patients with hypertension,

Table 2 Laboratory findings of the study population

Variables	All population (N=110)	Group 1 (n=66)	Group 2 (n=44)	P value
Glucose, mg/dL	96.16±10.12	97.17±10.65	94.66±9.19	0.234
GFR, mL/min/1.73 m^2	94.46±14.95	93.27±15.10	96.25±14.71	0.306
TG, mg/dL	160.02±85.01	179.09±92.74	131.41±62.62	0.002
TChol, mg/dL	190.56±33.67	192.98±28.62	186.54±40.84	0.271
LDL-C, mg/dL	111.30±29.90	111.72±27.14	110.63±34.34	0.637
HDL-C, mg/dL	48.10±10.38	45.85±9.48	51.48±10.87	0.006
CRP, mg/dL	0.32±0.37	0.34±0.36	0.31±0.38	0.107
Hemoglobin, g/L	146.80±18.60	147.50±17.70	145.70±20.10	0.621
WCC, cells/µL	8.12±2.04	8.22±1.94	7.97±2.19	0.618
Omentin-1, ng/mL (IQR)	55.05 (44.20–121.53)	46.35 (42.70–57.70)	130.95 (62.83–236.48)	<0.001
TyG index	8.82±0.53	8.96±0.53	8.62±0.45	0.001
TG:HDL-C	3.70±2.60	4.26±2.86	2.85±1.87	0.001
LDL-C:HDL-C (IQR)	2.34 (1.95–2.81)	2.36 (1.94–3.04)	2.29 (1.91–2.67)	0.333

CRP, C reactive protein; GFR, glomerular filtration rate; HDL-C, high-density lipoprotein cholesterol; TyG index, triglyceride glucose index; LDL-C, low-density lipoprotein cholesterol; TChol, total cholesterol; TG, triglycerides; WCC, white cell count.

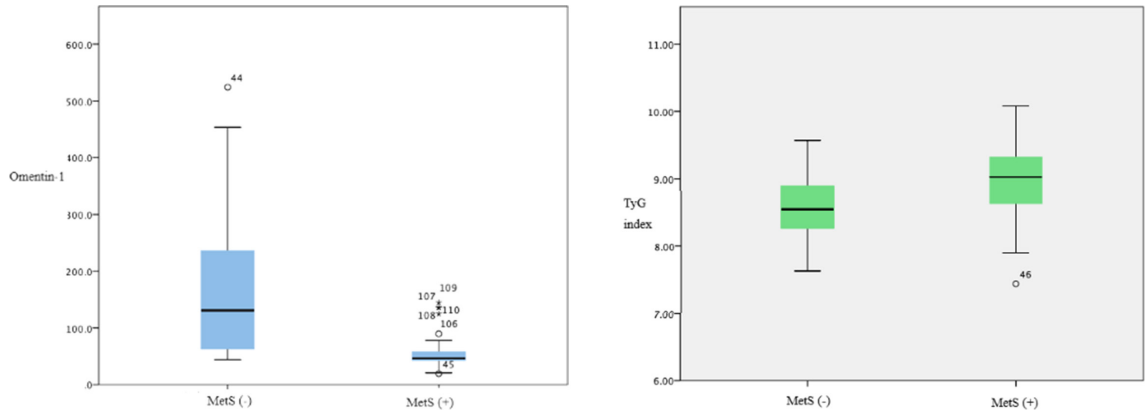

Figure 1 Comparison of circulating omentin-1 levels and TyG index between the groups. MetS, metabolic syndrome; TyG index, triglyceride glucose index.

omentin-1 was an independent predictor of MetS. (4) Circulating omentin-1 levels were affected by BMI, but not by gender.

Elevated blood pressure plays an important role in the MetS process related to diabetes and CVD, and MetS is more common in patients with hypertension.[19] Therefore, candidates at high risk of developing MetS should be identified in this population to prevent future cardiovascular events or TOD. Omentin-1 is a novel adipokine with a molecular weight of 35 kD, first described in 2005, and plays a role in insulin-dependent glucose reuptake and regulation of insulin sensitivity.[6 20 21] Apart from this role, the effects of omentin-1 on endothelial functions have been demonstrated in in vitro and in vivo studies.

Table 3 Correlation analysis of omentin-1 with other variables

Variables	r	P value
Age	−0.203	0.809
BMI	−0.169	0.038
WC	−0.232	0.115
WHtR	−0.116	0.226
EF	−0.112	0.244
SBP	−0.284	0.024
DBP	−0.194	0.043
Glucose	−0.013	0.895
GFR	0.048	0.615
TG	−0.189	0.048
TChol	−0.027	0.797
LDL-C	−0.047	0.657
HDL-C	0.265	0.046
CRP	−0.030	0.756
Hemoglobin	−0.064	0.507
WCC	−0.135	0.160
TyG index	−0.204	0.033
TG:HDL-C	−0.201	0.035
LDL-C:HDL-C	−0.016	0.187

BMI, body mass index; CRP, C reactive protein; DBP, diastolic blood pressure; EF, ejection fraction; GFR, glomerular filtration rate; HDL-C, high-density lipoprotein cholesterol; TyG index, triglyceride glucose index; LDL-C, low-density lipoprotein cholesterol; SBP, systolic blood pressure; TChol, total cholesterol; TG, triglycerides; WC, waist circumference; WCC, white cell count; WHtR, waist circumference to height ratio.

One study showed that omentin-1 induced vasodilation by increasing endothelial nitric oxide synthase in rat-isolated blood vessels.[22] Another study found that omentin-1 may have a role in endothelial dependent and independent vasodilation measured from the brachial artery.[10] Considering the available data, it can be thought that omentin-1 has not only favorable effect in glucose metabolism, but also an endothelial protective effect by regulating endothelial cell functions. It is well known that endothelial dysfunction is also a hallmark of hypertension related to a defect in the L-arginine-nitric oxide pathway,[23] and omentin-1 seems to play a critical role in blood pressure regulation using the same biological pathways. Kazama *et al*'s study[24] supported this view by demonstrating blood pressure decrease in rats injected with intravenous omentin-1. Afterwards, Çelik *et al*[25] confirmed the relationship between lower omentin-1 levels and the presence and severity of hypertension. In line with these studies, it may be considered that abnormal omentin-1 secretion promotes hypertension and this abnormality may be more pronounced in the presence of MetS. As a matter of fact, previous data reported that omentin-1 was significantly associated with MetS and MetS complications such as diabetes and CVD.[26–28]

Recently, Jialal *et al*[4] pointed out irregularity in adipose tissue, which is the main source of adipokine secretions, in nascent MetS without concomitant diabetes or CVD. To our knowledge, we are the first to evaluate the relationship between circulating omentin-1 levels and nascent MetS in a relatively large population of patients with hypertension and we showed that lower omentin-1 levels were an independent predictor of nascent MetS. Similar to our study, a

Table 4 Multivariate logistic regression analysis for presence of metabolic syndrome

	P value	OR	95% CI	
Constant	**0.004**	**0.000**	**Upper**	**Lower**
Omentin-1	0.000	0.962	0.945	0.979
TyG index	0.005	6.813	1.783	26.040
BMI	0.020	1.161	1.023	1.318

Variables entered at step 1: omentin-1, TyG index, BMI, TG:HDL-C, and CRP. BMI, body mass index; CRP, C reactive protein; HDL-C, high-density lipoprotein cholesterol; TyG index, triglyceride glucose index; TG, triglycerides.

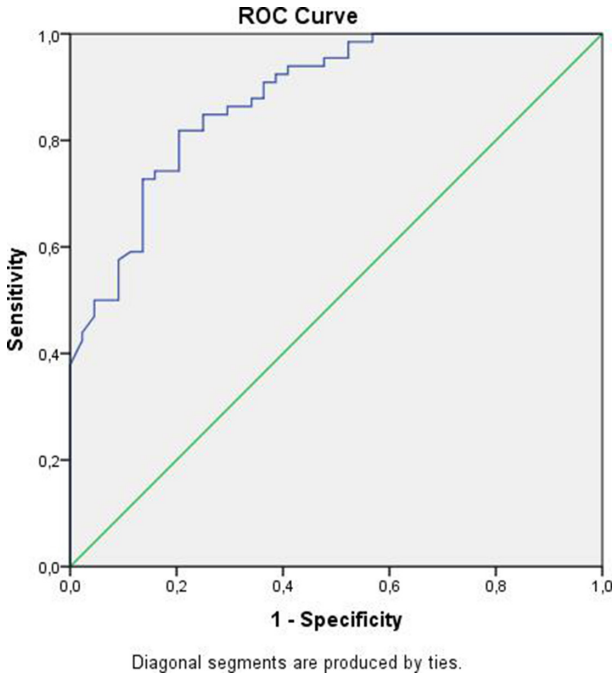

Figure 2 Receiver operating characteristic (ROC) curve of omentin-1 in predicting metabolic syndrome.

study demonstrated lower circulating omentin-1 levels in patients with nascent MetS compared with those without.[29] In another study, there was an inverse relationship between omentin-1 and nascent MetS in obese patients.[30] In addition, compared with these studies, decreased circulating omentin-1 levels in patients with MetS in our study were more pronounced. The fact that hypertension and MetS have similar pathophysiological mechanisms and synergistic interactions through changes at the molecular and cellular basis explains the significant difference in circulating omentin-1 levels between the groups in our study.[31] On the other hand, Kilic *et al*[32] and Vu *et al*[33] failed to show the relationship between circulating omentin-1 levels and nascent MetS. However, study findings may differ depending on age, study design, inclusion and exclusion criteria, heterogeneity of MetS components in the study populations, ethnic diversity, drug use, and measurement techniques. For example, the study of Vu *et al*[33] included subjects of different ethnic origins. However, we included only Turkish population in our study. In addition, while the entire population in our study consisted of patients with hypertension, 32 patients with MetS had hypertension in the study of Vu *et al*.[33] In the study of Kılıç *et al*,[32] the patient population was younger and the sample was relatively small compared with our study. Furthermore, no information was reported on the incidence of hypertension in the groups in this study.

In this study, we also demonstrated a significant inverse correlation between TyG index, a novel reliable marker of IR,[34] and circulating omentin-1 levels. It has been suggested that omentin-1 contributes to the modulation of insulin sensitivity by paracrine and endocrine factors through the activation of protein kinase (Akt/protein kinase B). However, it only plays a role in insulin-mediated glucose transport and has no effect on basal glucose transport. In vitro studies showed that increased insulin and glucose levels directly or indirectly decrease omentin-1 messenger RNA (mRNA) expression and protein synthesis in omental adipose tissue.[6 9] In addition, further studies have been performed to reveal the association between omentin-1 and IR in humans. In one study, IR was evaluated using homeostatic model assessment in insulin resistance index (HOMA-IR), and fasting insulin and higher IR were related to decreased circulating omentin-1 levels.[35] Another study reported that weight loss provided higher circulating omentin-1 levels and insulin sensitivity in obese patients.[36] Our study findings confirmed the findings of these studies through the TyG index. Besides, Jialal *et al*[29] previously reported an inverse correlation of circulating omentin-1 levels with glucose and TG, components of the TyG index.

In addition, we found that circulating omentin-1 levels were affected by BMI. Obesity is a chronic low-grade inflammatory condition and may affect circulating adipokine levels by causing adipocyte dysregulation in visceral adipose tissue. Increased visceral adipose tissue has also been proposed to decrease omentin-1 gene expression.[37] However, there are inconsistencies between studies investigating the relationship between omentin-1 and obesity. One study revealed a significant correlation between circulating omentin-1 levels and BMI, similar to our study, but another study failed.[8 32] These contradictions raise the question of whether omentin-1 regulation is affected by obesity itself or by the inflammatory process triggered by obesity. Another remarkable finding in our study was that omentin-1 levels did not differ according to gender in both groups. Similar to obesity, there are conflicting data on this topic. In one study, women had a significantly higher circulating omentin-1 levels compared with men, and in another study gender was an independent risk factor for circulating omentin-1 levels in obese subjects.[8 36] On the other hand, no relationship was found between omentin-1 and gender in some studies.[27 38] However, it has been suggested that the gene expressions and functions of omentin-1 may differ due to the heterogeneity of sex-related body fat distribution and the effect of sex hormones on omentin-1 regulation. This hypothesis was further confirmed by showing an inverse correlation of circulating omentin-1 levels with free testosterone concentrations in a study.[39]

There were some limitations to our study. First, this was a cross-sectional study and was performed with a relatively small population and hence we could not more reliably assess the causal relationship between plasma omentin-1 levels and MetS. Second, we did not directly measure the distribution of fat accumulation. However, evaluation of visceral adipose tissue with MRI may be more helpful in identifying the main cause of change in serum omentin-1 concentration. Third, we did not perform HOMA-IR measurements, the traditional method for IR, and did not reveal the relationship between circulating omentin-1 levels and HOMA-IR as in other studies. Finally, patients with a history of overt diabetes or meeting the criteria for diabetes were not included. However, patients with impaired glucose tolerance may have been included in this study as oral glucose tolerance test was not performed, and this may affect the study results.

CONCLUSION

Our study demonstrated that patients with hypertension had clearly lower circulating omentin-1 levels in the presence of MetS, regardless of the cause. In addition, it can be considered that omentin-1 may have a partial role in the development of MetS and may be a reliable marker to predict MetS. However, prospective large-scale studies are needed to confirm our findings.

Contributors Surgical and medical practices: SCS, GN, RN. Concept: SCS, GN. Design: SCS, GN. Data collection or processing: SCS, GN, RN. Analysis or interpretation: SCS, GN, RN. Literature search: SCS. Writing: SCS. Guarantor: SCS.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent for publication Not required.

Ethics approval The study was performed in accordance with the guidelines of the Declaration of Helsinki and approved by the local hospital ethic committee (Pamukkale University Faculty of Medicine Hospital; protocol no: E-60116787-020-39209).

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement All data relevant to the study are included in the article or uploaded as supplementary information.

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