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Circulating Omentin-1 in Obesity and Metabolic Syndrome Status Compared to Control Subjects

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Abstract

Background and aim: The goal of this study was to investigate the association between omentin-1 and different biochemical and anthropometric parameters in obese and non-obese, also in patients with metabolic syndrome compared to the healthy control group.

Methods: A total of 81 women were included in the current case-control study. Based on their BMI they were divided into obese and non-obese groups and according to WHO criteria for metabolic syndrome they were divided in to metabolic syndrome group and healthy subjects. Fasting blood sample was collected to determine biochemical indicators and insulin resistance and sensitivity indices (HOMA-IR and Quicki). Omentin-1 plasma level was assessed by ELISA. Association of omentin-1 with biochemical markers was studied. Body composition was measured using Body composition analyzer BC-418MA- Tanita.

Results: Levels of omentin-1 were lower in obese than non-obese subjects. It was also lower in patients with metabolic syndrome than in healthy ones. In correlation analysis, omentin-1 was associated with waist circumference, fat percent and fat mass and nearly visceral fat. But there was no significant relation between omentin and any insulin resistance indices.

Conclusions: In conclusion, omentin-1 is an adipokine closely associated with visceral obesity, but not with insulin resistance, and glucose metabolism; its serum concentration is decreased in the state of obesity and the regulation of omentin-1 production in adipose tissue is probably multifactorial. Future analysis of omentin's biological actions, and measurement of omentin-1 levels in the omental depot as well as in the circulation of humans with or without obesity and its co morbidities, will help to define its role in the pathogenesis of these diseases.

Keywords: Omentin-1; Metabolic syndrome; Obesity; Insulin resistance

Introduction

Obesity is associated with an array of health problems in adult and pediatric populations. Adipose tissue represents an active endocrine organ that, in addition to regulating fat mass and nutrient homeostasis, by releasing the large number of bioactive mediators (adipokines) plays an important role in modulating hemostasis, blood pressure, lipid and glucose metabolism, and inflammation [1-3]. Although anatomical location and vascularization of visceral and subcutaneous adipose tissue depots are clearly different [4], the molecular basis of differences in metabolism and secretory profile between visceral and subcutaneous adipose tissues and their impact on whole-body physiology are still not totally understood [5].

Increased visceral fat is shown to be associated with insulin resistance and Metabolic Syndrome (MS) which is a state of hyperinsulinemia with altered gonadal and adrenal steroid levels [6]. Insulin resistance should be conceptualized in a very broad manner that takes into account the interplay between nutrition, glucose, insulin and adipokines in various metabolic important tissues. Furthermore, it is apparent that accumulation of visceral adipose tissue poses a greater metabolic risk than subcutaneous adipose tissue [7], as removal of visceral rather than subcutaneous adipose tissue has been shown to improve insulin sensitivity [8].

Omentin is a newly identified secretory protein that relative to subcutaneous adipose tissue is highly and selectively expressed in visceral adipose tissue. This fat depot-specific protein is synthesized by visceral stromal vascular cells, but not adipocytes [9-11]. Omentin has been identified in other tissues at lower expression levels and named intelectin [10], intestinal lactoferrin receptor [11], and endothelial lectin [12]. This protein is also expressed in intestinal Paneth cells [13] and endothelial cells [12]. Omentin identified in other tissues at lower expression levels is named intelectin [12], intestinal lactoferrin receptor [13], or endothelial lectin [14].

In vitro studies have shown that omentin increases insulin signal transduction by activating the protein kinase Akt/protein kinase B and enhancing insulin-stimulated glucose transport in isolated human adipocytes [5,9]. Indeed decreased levels of plasma omentin and omentin gene expression levels in visceral adipose tissues of obese subjects has been reported [15]. Using homeostasis model assessment (HOMA), an inverse correlation between plasma omentin-1 levels, obesity and insulin resistance and a positive correlation with adiponectin and HDL levels was reported [16,17]. *In vitro* administration of glucose and insulin to human omental adipose tissue resulted in a dose-dependent reduction of omentin-1 expression. Furthermore, prolonged insulin-glucose infusion in healthy individuals significantly decreased the plasma omentin-1 levels [18]. These findings point to a paracrine or endocrine role of omentin in modulation of insulin sensitivity and obesity.

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Although the data clearly support the regulation of omentin by obesity, omentin may also be regulated by inflammation. Other studies have shown that omentin 1 expression is altered in inflammatory states [10,19]. Obesity itself is associated with low levels of chronic inflammation, which this may contribute to alteration of omentin expression and production [20,21]. Consequently, weight loss and different inflammatory states could modulate the omentin expression and function. However how different inflammatory status changes the serum levels of omentin-1 and also the relation between omentin-1 and inflammatory cytokines is not well known [22].

Taking together, all these data indicate that omentin is a potential candidate to play a role in the pathogenesis of obesity and insulin resistance. However, its circulating levels in obesity have not been adequately studied and its correlation with insulin resistance or obesity is still controversial. Therefore, based on the presence of omentin in the circulation and its potential role as insulin sensitizer the current study was designed to determine the circulating levels of omentin in obese subjects compared with normal weight control and to study its relationship with other biochemical and anthropometric parameters. We also compared its circulating levels in healthy subjects and MS patients.

Methods

Study population

A total of 81 women were included in this case-control study. Subjects were selected according to our defined inclusion criteria which was: age 22-52 years. Exclusion criteria were defined as: having the history of any condition that affects inflammatory markers such as known cardiovascular diseases, thyroid diseases, malignancies, current smoking, known case of diabetes mellitus who were on oral or insulin therapy, known case of hypertension who were on anti hypertensive therapy, heart failure, acute or chronic infections, acute or chronic inflammatory disease, hepatic or renal diseases, alcohol or drug abuse, and being pregnant. The study had the approval of the local ethics committee of Endocrinology and Metabolism Research Institute of Tehran University of Medical Sciences. Body mass index (BMI) of all subjects was calculated as weight (kg)/height (m^2) and subjects with BMI equal or more than 30 kg/m^2 were considered as obese subjects and placed in obese group. The control group was consisted of women with BMI lower than 30 $kg/m^{2.}$ To evaluate the role of omentin in insulin resistance, the recruited subjects were divided into two groups of MS patients (according to WHO criteria) and healthy subjects

HOMA and QUICKI calculations

Insulin resistance (IR) was calculated by homeostasis model assessment (HOMA). The HOMAIR was calculated according to HOMAIR equation= [Fasting Plasma Glucose (mmol/L) × Fasting Plasma Insulin (mIU/L)] /22.5 [23]. Quantitative insulin sensitivity check index (QUICKI) was calculated by (ISQUICKI= 1/ [log (fasting insulin) + log (fasting glucose)] [24].

Complete body composition analysis

We assessed the body composition of subjects with use of the body composition analyzer BC-418MA - Tanita (United Kingdom). This equipment is designed to send out a very weak electric current (50kHz, 500μA) and measures the impedance (electrical resistance) of the body. Based on this, device can calculate the complete body composition profile including weight, body fat percentage, body fat mass, fat free mass, estimated muscle mass, total body water and basal metabolic rate. The device predicts muscle mass on the basis of data obtained by Dual Energy X-ray Absorptiometry (DXA) using Bioelectrical Impedance Analysis (BIA). On time of assessment subjects were barefoot and we made sure that the soles of the feet were free from excessive dirt or crust; as this may also act as a barrier to the mild current. Since impedance fluctuates in accordance with distribution of body fluids, we followed all the following instructions for obtaining an accurate measurement. To prevent a possible discrepancy in measured values, we avoided taking measurements after vigorous exercise and waited until the subject sufficiently rested. Measurement was done when both arms were holding straight down, but not touching patient's side. The inner thighs also did not touch each other during the measurements; if necessary, we placed a dry towel between their arms and sides and/or between their thighs. As changes in body-water distribution and body temperature can have a major impact on measurements, all the measurements were performed in the morning in a fasting condition after urination. We didn't take measurements while transmitters such as mobile phones were in use. To obtain the measurements, 8 electrodes were positioned in a way that electric current was supplied from the electrodes on the tips of toes of both feet and fingertips of both hands. The voltage was measured on the heel of both feet and the thenar side of both hands. This method allows obtaining five different impedance measurements (whole body, right leg, left leg, right arm, and left arm) by switching the part of the body in which the current is flowing and the location where the voltage is measured.

Biochemical assay

The peripheral blood samples were obtained following 10-12 hrs overnight fasting. Serum was separated, aliquoted and stored at -80°C. All samples were analyzed by means of a single assay. All measurements were performed at the EMRC laboratory of Shariati hospital. GOD/PAP method was used for the measurement of fasting serum glucose and triglyceride levels. Total cholesterol levels were measured by Enzymatic Endpoint method, and direct high-density lipoprotein-cholesterol (HDL) was measured using enzymatic clearance assay. All measurements were done using Randox laboratories kit (Hitachi 902). Serum High sensitive C-reactive protein (hs-CRP) was measured by means of imonoturbidimetric assay (High sensitivity assay, by Hitachi 902). Serum insulin concentrations were measured by ELISA method (Human insulin ELISA kit, DRG Pharmaceuticals, GmbH, Germany) with a minimum detectable concentration of 1.76 μlU/ml, Intra Assay Coefficients of Variability (CV) was 2.19% and Inter assay CV was 4.4%. Serum Omentin 1 was measured by ELISA (human Intelectin-1, Apotech; Enzo Life Sciences) with sensitivity of 0.4 ng/ml, reference range of 0.5 –32 ng/ml, inter-assay CV of 4.61%, and intra-assay CV of 5.2 %.

Statistical analyses

Results are reported as the mean ± standard deviation. All the statistical analyses were performed using SPSS version 16 software. Chi-square test was used for comparison of the frequency of variables between two groups. Student T-test was used to compare quantitative variables. P values less than 0.05 were considered to be statistically significant.

Results

Study population characteristics

A total of 81 subjects were included in this study. 21 subjects (25.9%) had the BMI ≥30 kg/m^2 and were placed in obese group and 60 (74.0%) subjects with BMI <30 kg/m^2 were composed the non-obese group. two groups of obese and non-obese have a significant difference

in regards to their weight, BMI and waist circumference and also all the body fat composition indexes (table 1). In obese group a significantly higher levels of FBS but not fating insulin was observed. Measuring the insulin resistance state of two groups by HOMA IR showed a higher though not significant levels of insulin resistance in obese subjects (p= 0.06) while state of insulin sensitivity in non-obese subjects was significantly higher than obese patients (p=0.001). Two groups have similar levels of serum omentin, however, hs-CRP was significantly higher in obese group.

To evaluate the role of omentin in MS patients, the subjects were divided into two groups of MS (n=14, 17.29%) and healthy subjects (n=67, 82.71%). As it shown in (table 2), age, weight, waist circumference, free fat mass, visceral fat percent, FBS, cholesterol, and HDL cholesterol were the parameters that remarkable were different between two groups. In Pearson correlation, only waist circumference, fat percent and fat mass were significantly associate with omentin levels (table 3).

Discussion

Obesity is a chronic pathological condition and a risk factor for Metabolic Syndrome development, type 2 diabetes and cardiovascular disease [25-28]. Several studies have shown that visceral obesity is strongly associated with insulin resistance, hyperglycemia, dyslipidemia, and hypertension [29-31]. Excess visceral fat accumulation results in altered release of adipokines leading to CNS-mediated skeletal muscle and hepatic insulin resistance [1].

This study was undertaken to better understand the role of plasma omentin in obesity and insulin resistance. In this regard, we determined the serum omentin-1 levels in obese and non- obese subjects and found no difference in circulatory levels of this secretory protein in these two groups. In a study performed by de Souza Batista et al. [5] plasma levels of omentin were measured in lean, overweight, and obese otherwise healthy subjects. The authors found that plasma levels of omentin were highest among the lean subject and these levels were inversely correlated with BMI, waist circumference, and insulin resistance as measured by HOMA. One reason to not see a significant difference in omentin levels between two groups in our study might be due to low number of subjects recruited in this study. On the other hand it is possible that the difference in omentin levels would be more prominent in our study if two groups of normal (BMI< 25 kg/m^2) and obese subjects (BMI>30kg/m^2) were compared together.

One interesting finding of this study was the higher levels of hs-

Characteristics	Obese	Non-Obese	P value
Age(years)	38.71±12.1	33.8±11.6	<0.1
Weight(kg)	88.51±13.23	63.75±11.04	*< 0.001
Height (cm)	159.57±8.5	162.93±7.7	<0.09
Waist circumference (cm)	105.84±8.72	84.73±9.9	*<0.006
Waist-hip ratio	0.87±0.5	0.86±0.05	<0.4
Fat percent	40.80±6.23	29.47±7.14	*< 0.001
fat mass	36.11±10.01	17.55±6.15	*< 0.001
Free fat mass	53.15±9.34	46.63±7.90	*< 0.001
Visceral fat	9.80±2.90	3.87±2.45	*< 0.001
BMI(kg/m^2)	34.71±4.0	23.94±3.25	*< 0.001
FBS (mg/dl)	101.83±17.05	87.20±12.68	*< 0.001
LDL Cholesterol (µg/dl)	103.11±25.02	93.62±26.38	<0.18
HDL Cholesterol (µg/dl)	40.0±6.47	42.63±10.62	<0.32
Total cholesterol (µg/dl)	176.61±30.10	170.08±38.81	<0.51
Triglyceride(µg/dl)	119.16±41.37	107.03±66.16	<0.46
Fasting Insulin (µlU/ml)	15.92±16.43	8.79±5.27	<0.08
hs-CRP	7.76±5.79	1.71±1.32	<0.01
Omentin(pg/ml)	16±20.0	50±27.14	<0.19
HOMA	4.13±2.64	1.91±1.30	<0.16
QUICKI	0.32 ±0.027	0.36 ±0.043	*< 0.001

Data are presented as mean ± SD, *P value < 0.05 was considered significant.
BMI: Body Mass Index; FBS: Fasting Blood Sugar; LDL: Low Density Lipoprotein; HDL: High Density Lipoprotein; hs-CRP: High Sensitive C- reactive Protein; HOMA: Homeostatic Model Assessment; QUICKI: Quantitative Insulin Sensitivity Check index.

Table 1: Demographic and clinical characteristics of the obese and non-obese group

Characteristics	Metabolic syndrome	Control	P value
Age(years)	47.50±13.98	33.74±11.09	*<0.02
Weight(kg)	89.8±15.19	68.64±14.44	*<0.00
Height (cm)	163±8.98	162.16±7.93	<0.83
Waist circumference (cm)	104.78±8.21	91±13.74	*<0.00
Waist-hip ratio	0.89±0.04	0.86±0.05	<0.09
Fat percent	33.92±13.19	30.42±8.9	<0.63
fat mass	30.92±15.66	21.73±10.35	<0.09
Free fat mass	58.90±13.31	46.90±7.21	*<0.00
Visceral fat	11.75±4.34	4.96±3.36	*< 0.001
BMI(kg/m^2)	34.05±7.39	26.15±5.43	<0.07
FBS (mg/dl)	124.4±28.32	88.94±13.79	*<0.01
LDL Cholesterol (µg/dl)	103.11±25.02	93.85±25.07	<0.18
HDL Cholesterol (µg/dl)	31.0±6.74	42.78±9.57	*<0.00
Total cholesterol (µg/dl)	219.2±45.0	168.28±34.14	*<0.00
Triglyceride(µg/dl)	225.4±134.26	101.77±44.26	<0.10
Fasting Insulin (µlU/ml)	14.9±8.96	10.17±9.59	<0.2
hs-CRP	8.22±8.64	2.79±5.65	<0.2
Omentin(pg/ml)	24.27±26.74	25.43±18.81	<0.9
HOMA	4.31±2.68	2.305±2.62	<0.1
QUICKI	0.31±0.02	0.35±0.04	<0.05

Data are presented as mean ± SD, *P value < 0.05 was considered significant
BMI: Body Mass Index; FBS: Fasting Blood Sugar; LDL: Low Density Lipoprotein; HDL: High Density Lipoprotein; hs-CRP: High Sensitive C-reactive Protein. HOMA: Homeostatic Model Assessment. QUICKI: Quantitative Insulin Sensitivity Check Index

Table 2: Demographic and clinical characteristics of the metabolic syndrome and control group

Characteristic	r	P value
Weight(kg)	0.07	<0.764
Height (cm)	0.18	<0.482
Waist circumference (cm)	-0.31	*<0.01
Waist-hip ratio	-0.07	<0.57
Fat percent	-0.16	*<0.04
fat mass	-0.17	*<0.03
Visceral fat	-0.15	<0.07
BMI(kg/m^2)	-0.2	<0.1
FBS (mg/dl)	-0.16	<0.21
HDL Cholesterol (µg/dl)	-0.215	<0.09
Triglyceride(µg/dl)	0.026	<0.84
Fasting Insulin (µlU/ml)	-0.19	<0.14
hs-CRP	0.05	<0.70
HOMA	0.03	<0.64

P value < 0.05 was considered significant
BMI: Body Mass Index; FBS: Fasting Blood Sugar; HDL: High Density Lipoprotein; hs-CRP: High Sensitive C-reactive Protein; HOMA: Homeostatic Model Assessment.

Table 3: Correlations of demographic and clinical characteristics with plasma omentin 1 levels

CRP in obese subjects despite of lack having any chronic or acute inflammation. Obesity is considered as a state of chronic low-grade inflammation and the serum omentin-1 concentration may be related to inflammatory states so obesity itself , may contribute to the regulation of the role of omentin in human physiology [21,32]. Although the data clearly support regulation of omentin by obesity, it may also be regulated by inflammatory mediators. Other studies have shown that omentin 1 expression is altered in inflammatory states [10,19]. Inflammatory cytokines may contribute to regulation of protein biosynthesis in adipose tissue. Levels of circulating inflammatory cytokines TNF-α and IL-6 are increased in obese and T2DM subjects [33]. Schaffler A et al. [10] reported that omentin levels were altered by inflammation [10]. In this study, hsCRP serum levels were elevated in the obese group compared with those subjects in the control group but serum omentin-1 concentrations were not significantly correlated with hsCRP serum levels. The interrelation between omentin and inflammation could be further clarified through investigating the association between omentin and other proinflammatory markers such as nuclear factor–κB and IL-6.

In this study we found a higher state of insulin resistance in obese subjects as was manifested by lower levels of QUICKI. QUICKI value less than 0.33 has been previously used as cut-off for diagnosis of insulin resistance. In this study, the mean of QUICKI value in obese subjects was reported to be 0.32 which clearly indicate a state of insulin resistance in this group. This finding is more confirmed with higher levels of HOMA IR (though not significant) in obese group, a finding that has bee reported by previous studies. Tan et al. [34] found that plasma omentin-1 levels were decreased in female subjects with type 1 diabetes [34] suggesting that omentin is important for glucose metabolism. *In vitro*, omentin increases insulin signal transduction by activating the protein kinase B and enhances insulin-mediated glucose transport in adipocytes [5]. We expected that the decreased serum omentin-1 levels observed in obese group (though not significant) might cause a reduction of insulin-stimulated glucose uptake in visceral and subcutaneous adipocytes or other insulin sensitive tissues and contributing, at least partially, to insulin resistance. Moreover, insulin and glucose significantly and dose-dependently decreased omentin mRNA expression and omentin protein production in omental adipose tissue explants and that hyperinsulinemia significantly reduced plasma omentin-1 levels in healthy subjects [17].

We should note that in our study we did not found an association between omentin and insulin resistance in term of HOMA IR. This might be caused by our exclusion criteria that excluded the subjects with documented diabetes and hypertension, two main characteristic of MS and insulin resistance state. It is plausible that our subjects were in early stages of insulin resistance development and could not reach to a remarkable association levels with leptin. There are also controversy in the association of omentin levels with insulin resistance in previous studies which might be due to different patient populations in these studies or other currently undefined factors that may affect omentin [35].

In a study done by Moreno-Navarrete**,** omentin levels were found to be correlated with some markers of lipid metabolism such as TG, TC, and to a lesser extent with LDL-C, which indicates that omentin may play a role in lipid metabolism or diabetic dyslipidemia as a compensatory mechanism [36]. But in our study there was no significant correlation with any of the mentioned lipid metabolism markers.

However, it is important to point out that the current study is an epidemiologic study, capable of identifying correlations between variables and not direct cause and effect. Accordingly, further experimental studies are required to unravel the molecular mechanism of the observed associations between omentin with various metabolic parameters.

Conclusion

In conclusion, omentin is an adipokine that has been reported to have an association with visceral obesity, insulin resistance, and glucose metabolism, however the exact mechanism and physiological role of omentin in glucose metabolism is not very well understood. In this study, we did not find a relation between omentin and BMI as an indicator of obesity and HOMA IR and QUICKI as an indicator of insulin resistance. Further study with larger scale and more precise selection of study population is recommended. Further investigation is needed to determine whether omentin can act on other insulin-sensitive tissues such as liver and skeletal muscle and to elucidate the relevant signal transduction pathways.

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