The Relationship Between Obesity and Plasma Omentin Levels in Newly Diagnosed and Untreated Type 2 Diabetic Patients

Yeni Tanı Konmuş ve Tedavi Almamış Tip 2 Diyabetik Hastalarda Plazma Omentin Seviyeleri ile Obezite Arasındaki İlişki

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ABSTRACT Objective: In addition to its role in energy storage, adipose tissue produces several hormones and cytokines termed adipokines. These adipokines have widespread effects on carbohydrate and lipid metabolism. Omentin is a newly identified adipokine that is highly and selectively expressed in visceral adipose tissue relative to subcutaneous adipose tissue. Omentin was shown to be decreased in obese, insulin resistant and diabetic patients. In our study, we intended to show the relation of omentin with obesity in newly diagnosed type 2 diabetic patients. **Material and Methods:** The study included 84 type 2 diabetic female patients, 11 of them had body mass index <25, 35 had body mass index between 25-30 and 38 of them had body mass index >30. Healthy 40 age matched females with body mass index <25, between 25-30 and >30 served as control group. As well as making physical and antropometric examinations, fasting plasma glucose and insulin, post prandial plasma glucose, lipid profile, and omentin levels were measured in all female subjects. **Results:** All the diabetic patients with body mass index <25, 25- 30 and body mass index>30 had lower omentin levels than the controls whose body mass index were similar. **Conclusion:** In conclusion, we may speculate that omentin has an important role in diabetes and obesity and studies about omentin may lead us to new approaches about diagnoses or therapy of diabetes mellitus or obesity or insulin resistance.

Key Words: Insulin resistance; obesity; diabetes mellitus, type 2

ÖZET Amaç: Adipöz doku enerji deposu olarak görev yapmasının yanı sıra adipokin adı verilen bazı hormon ve sitokinleri salgılar. Adipokinlerin karbonhidrat ve lipid metabolizmasında yaygın etkileri vardır. Omentin, subkütanöz adipöz dokuya oranla, visseral adipöz dokudan daha çok miktarda ve selektif olarak salgılanan yeni tanınmış bir adipokindir. Obez, insülin rezistan ve diyabetik insanların serumlarında omentin seviyelerinin düştüğü gösterilmiştir. Çalışmamızda yeni tespit tip 2 diabetes mellitus hastalarında omentinin obezite ile ilişkisini göstermeyi planladık. **Gereç ve Yöntemler:** Çalışmamıza 84 tip 2 diyabetik kadın hasta alındı, 11'inin vücut kitle indeksi <25, 35'inin vücut kitle indeksi 25- 30 arası, 38'inin >30 idi. Ayrıca kontrol grubu için 40 normal kadın bireyi vücut kitle indekslerine göre aynı şekilde sınıfladık. Tüm kadın hastalarda fizik muayene ve antropometrik ölçümlere ek olarak açlık kan şekeri, açlık insülin, tokluk kan şekeri, lipid profili, ve omentin seviyelerine bakıldı. **Bulgular:** Vücut kitle indeksi <25, 25-30 arası olan ve >30 olan tüm tip 2 diyabetik hastalar aynı vücut kitle indeksine sahip kontrol kişilerden daha düşük omentin seviyelerine sahiptiler. **Sonuç:** Sonuç olarak, omentinin diyabet ve obezitede önemli bir rolü olduğunu ve omentin ile ilgili gelecekteki çalışmaların bizi diabetes mellitus veya obezite veya insülin rezistansının tanı ve tedavisinde yeni yaklaşımlara götürebileceğini ileri sürebiliriz.

Anahtar Kelimeler: İnsülin direnci; şişmanlık; diabetes mellitus, tip 2

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Adipose tissue is closely associated with insulin resistance, diabetes mellitus and cardiovascular disease, but the underlying pathophysiological mechanisms are unknown. Regional distribution of adipocytes appears to be an important indicator for metabolic disturbances.[1,2] Adipokines such as leptin, adiponectin, osteopontin and visfatin are currently investigated as potential future drug targets in T2DM, lipid metabolism, endothelial dysfunction and inflammatory diseases.[3,4]

In 2003 a new adipokine, omentin (also named omentin-1, intelectin, intelectin-1, endothelial lectin and intestinal lactoferrin receptor) was described and reported to be expressed specifically in human omental tissue.[5] In 2006 Yang et al. demonstrated that omentin is capable of enhancing insulin mediated glucose uptake in adipocytes, furthermore they found that omentin is predominantly expressed in visceral but not in subcutaneous adipose tissue.[2,5]

Lean subjects had significantly higher plasma omentin levels than obese and overweight subjects.[6-8] Decreased plasma omentin levels were reported in type 1 diabetes mellitus and type 2 diabetes.[9,10] Keeping in mind that visceral obesity may be more pathogenic than subcutaneous obesity in promoting insulin resistance, type 2 diabetes, and cardiovascular disease and omentin is a visceral adipose tissue specific adipocytokine we planned to analyse the relationship of plasma omentin with some parameters of adiposity, insulin resistance and plasma lipid profile in newly diagnosed, untreated type 2 diabetic patients.

MATERIAL AND METHODS:

PATIENTS

A total of 84 female newly diagnosed, untreated type 2 diabetics patients aged from 45-65 years, were recruited from the outpatient Clinic of Ankara Education and Research Hospital from February 2009 to June 2009. Fourteen of them had BMI <25, 35 female had BMI 25-30, and 38 female had BMI >30. Fourty aged matched female subjects formed the control group. Fourteen of the control females had BMI <25, 14 had BMI 25 - 30, and 12 had BMI > 30. This study was performed according to Helsinki decleration 2008.

Patients with male gender, conditions which may effect metabolic parameters (such as polycystic ovary syndrome or thyroid dysfunctions in history or nowadays), pregnancy, chronic diseases, infection, coronary artery disease were excluded. None of the women were on any medications for at least 6 months before the study including oral contraceptives, glucocorticoids, ovulation induction agents, antidiabetic and antiobesity drugs, estrogenic, antiandrogenic, antihypertensive or antihyperlipidemic medication.

After detailed physical examination, in all subjects body weight and height were measured. Body mass index (BMI) was calculated as weight in kilograms divided by the square of height in meters. Body fat were estimated by Tanita body composition analayser TBF -300 after the subjects rested 30 minutes.

Blood pressure was measured after a 5 min rest in the semi-sitting position with a sphyngmomanometer. Blood pressure was determined at least three times at the right upper arm, and the mean was used in the analysis. The patients who were taking antihypertensive drugs or patients whose determined mean blood pressure levels ≥140/90 mmHg were assumed to be hypertensive and excluded.

Blood was withdrawn after 12 h of overnight fasting, at 08.30 a.m. for fasting plasma glucose (FPG), insulin (FI), hemoglobin A1c (HbA1c) serum total and high density lipoprotein cholesterol (HDL-C), triglyceride (TG), and omentin levels. Another blood sample was taken for postprandial plasma glucose (PPPG) 2 h after breakfast.

The local ethics committee approved this study and all the subjects gave written informed consent.

LABORATORY METHODS

Plasma glucose, total cholesterol, triglyceride (TG) and HDL cholesterol concentrations were determined by enzymocalorimetric spectrophotometric method in a Roche/Hitachi molecular PP auto-

analyser. Low density lipoprotein cholesterol (LDL-C) was calculated by the Friedewald Formula (LDL: Total cholesterol –HDL-TG/5). Insulin was measured by means of DRG Diagnostics (DRG Instruments GmbH, Germany) ELISA kits. HbA1c level was measured by TOSOH G7 HPLC system.

The estimate of insulin resistance by homeostasis model assessment insulin resistance index (HOMA-IR) was calculated as fasting plasma insulin (μunite / ml) x fasting plasma glucose (mmol /l)/22. 5.

For the measurements of omentin, after fasting blood samples were drawn, they were santrifuged 4000 cycle / min in 30 minutes. Plasma was then stored at - 75˚C, in two different tubes. Plasma omentin levels were assayed by a commercial USCNLIFE (Chinese) ELISA kit.

Statistical analysis

Calculations were performed using SPSS version 10,1. Data were presented as mean ± SD. Student t-test was used to compare the groups in a parametric way (For homogen distributed data). Non parametric Mann Whitney U test was used for non homogen distributed data. For determining the correlations within the parameters Pearson correlation analysis was made. A p value of < 0.05 was considered as statistically significant.

RESULTS

We performed this study with female type 2 diabetic patients and control females. In Table 1 all the characteristics of type 2 diabetic patients and control subjects with BMI <25 were demonstrated.

In the T2DM patient and control female groups with BMI <25 age, SBP, DBP, BMI, body fat, T-Chol, TG, LDL-C, HDL-C levels were statistically insignificant (Table 2). FBG, PPBG, HbA1c, FI HOMA-IR was found to be elevated in diabetic patients with BMI <25 (p<0.01 p<0.02 p<0.02 p<0.01 p<0.008 respectively). The diabetic patients had statistically lower omentin levels than the control females with BMI <25 (p<0.01) (Table 1).

In Table 2 all the characteristics of T2DM and control groups with BMI 25-30 were demonstrated.

TABLE 1: Characteristics of diabetic and control females with BMI <25

	T2 DM (n=11)	Control (n= 14)	P
Age (year)	53.0 ± 12.2	54.5 ± 9.6	NS
SBP (mm Hg)	120.8 ± 13.1	124.0 ± 20.4	NS
DBP (mm Hg)	77.2 ± 6.9	81. 8 ± 26.0	NS
BMI (kg/m2)	23.2 ± 1.7	23.2 ± 1.3	NS
Body fat (%)	23.8 ± 12.2	23.6 ± 6.0	NS
FBG (mg/dl)	114.3 ± 63.7	85.2 ± 9.1	<0.001
PPBG (mg/dl)	210.3 ± 85.1	111.9 ± 18.7	<0.002
HbA1c (%)	9.9 ± 2.0	5.7 ± 0.2	<0.002
FI(μÜ/ml)	8.1 ± 2.9	8.1 ± 1.1	<0.001
HOMA-IR	4.5 ± 2.3	1.6 ± 0.2	<0.008
T-Chol (mg/dl)	187.8 ± 0.2	169.5 ± 7.9	NS
TG (mg/ dl)	172.7 ± 72.2	159.7 ± 51.4	NS
LDL-C	113.7 ± 36.7	97.3 ± 32.0	NS
HDL-C	40.5 ± 14.0	46.5 ± 0.7	NS
Omentin(ng/ml)	317.0 ± 97.5	513.4 ± 109.9	<0.01

T2 DM: Type 2 diabetes mellitus, SBP: systolic blood pressure, DBP: diastolic blood pressure, BMI: Body mass index, FBG: Fasting blood glucose, PPBG: Post prandial blood glucose, HbA1c: Hemoglobin A1c, FI: Fasting insulin, HOMA-IR: Homeostasis model assessment insulin resistance index, T-Chol, TG: trigyceride, LDL-C: Low density lipoprotein cholesterol, HDL-C: High density lipoprotein cholesterol, NS: Non- significant.

When we compared the T2DM patients and control females with BMI 25 - 30 we found that FBG, PPBG, HbA1c, FI, HOMA-IR levels of the diabetic patients with BMI 25 - 30 were statistically higher than the control females with similar BMI (p< 0.001 p< 0.001 p< 0.001, p< 0.001, p< 0.008 respectively). Age, SBP, DBP, BMI, body fat, T-Chol, TG, LDL-C, HDL-C levels of the groups were similar. Omentin levels were found to be lower in the T2DM group than the control group (p<0.01) (Table 2).

In Table 3 all the characteristics of T2DM and control groups with BMI>30 were demonstrated.

In the comparison of T2DM patients and the control females with BMI >30). Age, DBP, BMI, body fat, TG, HDL-C levels of the groups were similar, but SBP, FBG, PPBG, HbA1c, FI, HOMA-IR, T-Chol, LDL-C levels of T2 diabetic group were higher than the control group (p<0.002, p<0.001, p<0.001 p<0.001, p<0.001, p<0.001 p<0.01, p<0.03, respectively). In the T2DM group omentin levels were statistically lower than the control group (p< 0.006) (Table 3).

TABLE 2: Characteristics of diabetic and control females with BMI 25-30

	T2 DM (n= 35)	Control (n= 14)	P
Age (year)	52.8 ± 10.3	56.3 ± 6.3	NS
SBP (mm Hg)	131.2 ± 17.5	119.2 ± 16.3	NS
DBP (mm Hg)	83.4 ± 12.7	75.1 ± 9.3	NS
BMI (kg/m2)	29.1 ± 1.3	28.9 ± 1.3	NS
Body fat (%)	29.3 ± 7.8	28.5 ± 5.9	NS
FBG (mg/dl)	195.6 ± 83.2	95.5 ± 8.0	<0.001
PPBG (mg/dl)	305.3 ± 46.9	111.5 ± 14.6	<0.001
HbA1c (%)	8.7 ± 2.0	5.5 ± 0.3	<0.001
FI (µÜ/ml)	13.1 ± 6.8	8.5 ± 4.1	<0.001
HOMA-IR	6.3 ± 1.3	2.9 ± 0.3	<0.008
T-Chol (mg/dl)	213.8 ± 64.2	198.0 ± 36.3	NS
TG (mg/ dl)	238.8 ± 72.2	147.7 ± 92.8	NS
LDL-C	120.6 ± 42.7	121.5 ± 30.1	NS
HDL-C	44.6 ± 10.7	47.5 ± 8.5	NS
Omentin (ng/ml)	310.4 ± 97.4	430.0 ± 94.8	<0.01

T2 DM: Type 2 diabetes mellitus, SBP: systolic blood pressure, DBP: diastolic blood pressure, BMI: Body mass index, FBG: Fasting blood glucose, PPBG: Post prandial blood glucose, HbA1c: Hemoglobin A1c, FI: Fasting insulin, HOMA-IR: Homeostasis model assessment insulin resistance index, T-Chol: Total cholesterol, TG: triglyceride, LDL-C: Low density lipoprotein cholesterol, HDL-C: High density lipoprotein cholesterol, NS: Non- significant.

TABLE 3: Characteristics of diabetic and control females with BMI>30

	T2 DM (n= 38)	Control (n= 12)	P
Age (year)	52.8 ± 11.0	53.1 ± 8.6	NS
SBP (mm Hg)	143.7 ± 19.3	124.1 ± 15.0	<0.002
DBP (mm Hg)	90.5 ± 12.17	79.5 ± 9.1	NS
BMI (kg/m^2)	34.2 ± 4.2	35.3 ± 4.7	NS
Body fat (%)	38.3 ± 9.1	40.3 ± 4.9	NS
FBG (mg/dl)	199.0 ± 80.4	82.5 ± 25.3	<0.001
PPBG (mg/dl)	289.0 ± 45.6	110.2 ± 19.2	<0.001
HbA1c (%)	8.5 ± 2.3	5.7 ± 0.4	<0.001
FI (µÜ/ml)	20.3 ± 13.3	10.5 ± 4,0	<0.001
HOMA-IR	9.8 ± 3.4	2.1 ± 0.9	<0.001
T-Chol (mg/dl)	246.3 ± 72.5	188.9 ± 32.6	<0.01
TG (mg/ dl)	261.6 ± 82.5	195.3 ± 53.0	NS
LDL-C	147.4 ± 49.2	114.5 ± 30.6	<0.03
HDL-C	42.6 ± 8.7	43.8 ± 8.5	NS
Omentin (ng/ml)	300.7 ± 97.9	416.1 ± 90.8	<0.006

T2 DM: Type 2 diabetes mellitus, SBP: systolic blood pressure, DBP: diastolic blood pressure, BMI: Body mass index, FBG: Fasting blood glucose, PPBG: Post prandial blood glucose, HbA1c: Hemoglobin A1c, FI: Fasting insulin, HOMA-IR: Homeostasis model assessment insulin resistance index, T-Chol: Total cholesterol, TG: Triglyceride, LDL-C: Low density lipoprotein cholesterol, HDL-C: High density lipoprotein cholesterol, NS: Non- significant.

Omentin and HOMA-IR levels according to BMI in diabetic and control subjects were presented in Figure 1 and 2.

Then we mixed all patient and control females and correlation analysis was made between omentin and all the parameters. Positive weak correlations were obtained between age (r:0.264 p<0.004), HDL-C (r:0.266 p<0.003) and omentin levels, negative weak correlations between BMI (r:-0.216 p<0.017), FBG (r:-0.423 p<0.001) PPBG (r:-0.378 p<0.001), HbA1c (r:-0.392 p<0.001), FI (r:-0.388 p<0.001), HOMA-IR (r:-0.470 p<0.001) and omentin levels (Table 4). Significant correlations were demonstrated in bold within the table.

DISCUSSION

Omentin is newly found protein, shown to be expressed in visceral adipose tissue, 150 times more than in subcutaneous adipose tissue[2]. It was stated that omentin was among the first molecules known to exhibit such a dramatic difference in gene expression between the two major fat depots. As a secretory factor, omentin may be a novel hormone

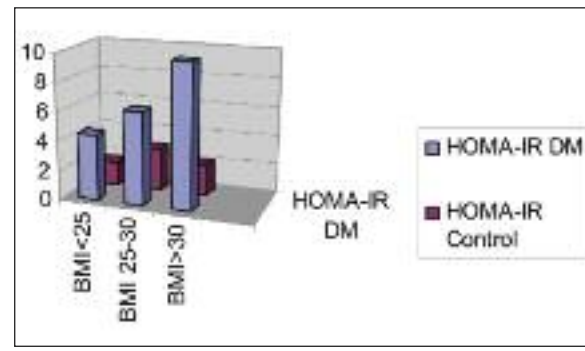

FIGURE 1: Omentin levels according to BMI in diabetic and control subjects.

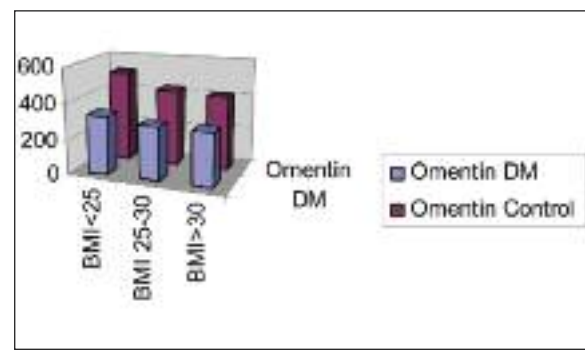

FIGURE 2: HOMA-IR levels according to BMI in diabetic and control subjects.

TABLE 4: A summary of correlation analysis among all females (n: 124).

		AGE	SBP	DBP	BMI	BODY FAT	FBG	PPBG	T-CHOL	TG	LDL-C	HDL-C	HBA1C	FI	HOMA-IR
OMENTIN	R	0.264	-0.036	-0.002	-0.216	0.017	-0.423	-0.378	-0.115	-0.145	-0.096	0.266	-0.392	-0.388	-0.475
	P	0.003	0.696	0.987	0.017	0.857	0.000	0.000	0.211	0.114	0.298	0.003	0.000	0.000	0.000

SBP: Systolic blood pressure, DBP: Diastolic blood pressure, BMI: Body mass index, FBG: fasting blood glucose, PPBG: post prandial blood glucose, TG: triglyceride, LDL-C: low density lipoprotein cholesterol, HDL-C: high density lipoprotein cholesterol, FI: fasting insulin, HOMA-IR: Homeostasis model assessment insulin resistance index, T-Chol: Total cholesterol; HbA1c: Hemoglobin A1c.

that is likely to act as both an endocrine factor to modulate systemic metabolism, including insulin action in subcutaneous adipocytes, and an autocrine and paracrine factor to regulate visceral adipose biology locally.[2,11]

After Vague mentioned the concepts of android and gynoid obesity in 1947, it was understood that obesity was not a homogenous situation and distribution of adipose tissue was very important in pointing out the complex relations between lipid, glucose metabolism and obesity[12]. In various studies it was demonstrated that excessively increased adipose tissue in the upper part of the body was a risk factor in mortality and morbidity about diabetes, hyperlipidemia, hypertension and atherosclerosis.[13,14]

Omentin was shown to enhance insulin stimulated glucose uptake but systemic levels were not correlated to postprandial blood glucose[15]. Also omentin was demonstrated to trigger Akt signalling in both the absence and presence of insulin[6,7]. Furthermore omentin plasma levels and gene expression in visceral adipose tissue were low in obesity[7,9]. Decreased omentin levels were found patients with impaired glucose regulation[16] and type 2 diabetic[9,16] and type 1 diabetic patients.[8]

In our study we found lower omentin levels in all our T2 diabetic patients than in all control females with similar BMI levels. In our diabetic patients as well as control females, as BMI increased omentin levels decreased more. After the correlation analysis we showed negative correlation with BMI and omentin levels. In all the groups with BMI <25, BMI 25- 30 and BMI >30, FBG, PPBG, Hb A1c, HOMA-IR levels were higher in diabetics as anticipated. In the groups with BMI <25 and BMI 25 - 30 the lipid levels were similar, but in the group with BMI>30 the diabetic females had significantly high levels, this result made us think that the morbid obese females did not bother diabetic, as well as lipid diets.

When we chose our cases, we decided not to add male subjects in our study, in order to produce an homogenous group. Furthermore, in order to eliminate the interferance of any drug, we included our study only newly diagnosed, untreated type 2 diabetic females. In order to demonstrate the effects of adipokines in metabolic disorders such as diabetes, obesity and insulin resistance, it may be useful to examine the patients with higher diabetic age, patients having different therapeutical modalities and with different sexes.

Our evaluation with BMI reminds us the studies supporting the idea that increased fat mass which was thought to be responsible for obesity related insulin resistance will not always be an increased inflammation marker in the adipose tissue. It was demonstrated that Asian, especially Indians had higher risk of diabetes and cardiovascular diseases than other populations in spite of their lower weights.[17,18] Indians living in America had higher insulin resistance than body fat matched other Americans, so it was speculated that they had more visceral fat amount.[19] Studies showing inconsistent relation between body fat and insulin resistance in Asian populations are emerging.[17, 18] Lean but having insulin resistance subjects supports the thought of metabolically obese, but with normal weight subjects. In these patients it may be important to diagnose early and treat early before complications are emerged, but the definition of obesity we use nowadays makes it diffucult. Perhaps the fundemental principle is to focus on the functional structure not the amount of adipose tissue. In this point adipocytokines secreted from the adipose tissue gain importance.

The hypothesis of the presence of subjects metabolically obese but having normal weight and metabolically normal but obese, also came to light in our study. In the comparison of the groups of T2DM and control with BMI >30, BMI was similar but HOMA-IR was higher in the diabetic than the control group. In summary, our control subjects with BMI >30, were not insulin resistant. When we compare our diabetic and control females who have BMI <25, BMI was similar but HOMA-IR was also high in the diabetics, briefly our lean diabetics had insulin resistance. In summary we supported the hypothesis of metabolically obese, with normal weight and metabolically normal, obese subjects.[20] We can also ask a new question; can we add HOMA-IR and also the decrease of omentin into the definition of metabolic obesity.

In conclusion we found that omentin levels decreased in diabetes mellitus and omentin levels decreased further when obesity worsened in diabetes.

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