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Omentin and chemerin and their association with obesity in women with polycystic ovary syndrome

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Abstract

We aimed to investigate whether overweight/obesity is associated with omentin and chemerin. The study group consisted of 81 women with Polycystic ovarian syndrome (PCOS) (41 lean, BMI $< 25\,kg/m^2$ and 40 overweight or obese, BMI $> 25\,kg/m^2$) and 61 healthy subjects (31 lean, BMI $< 25\,kg/m^2$ and 30 overweight or obese, BMI $> 25\,kg/m^2$; control group). The clinical, endocrine, metabolic parameters, plasma omentin and chemerin levels were measured in patients and compared to control. In all subjects with PCOS ($n = 80$), serum chemerin levels were higher compared with those of the controls ($n = 58$) (7.71 ± 1.78 ng/mL versus 6.94 ± 0.82 ng/mL, $p = 0.003$). However, serum omentin levels were not significantly different between the PCOS subjects and the controls (1.55 ± 0.43 ng/mL versus 1.69 ± 0.37 ng/mL, $p = 0.056$). The mean chemerin concentrations were significantly elevated in the obese PCOS group compared with the obese control subjects (8.98 ± 1.45 ng/mL versus 7.02 ± 0.67 ng/mL, $p = 0.000$) and the nonobese PCOS group compared with the obese control subjects (6.57 ± 1.17 ng/mL versus 7.02 ± 0.67 ng/mL, $p = 0.000$). In conclusion, fat mass seems to be the main determinant factor of increased chemerin and decreased omentin in women with PCOS.

Keywords

Androgen, chemerin, insulin resistance, omentin, polycystic ovary syndrome

History

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Introduction

Polycystic ovarian syndrome (PCOS) is one of the most common endocrine diseases in women of reproductive age [1]. Women with PCOS are characterized by insulin resistance (IR) [2,3], an increased risk of glucose intolerance, type 2 diabetes [4–6], and an increased prevalence of lipid-related abnormalities [7,8]. Insulin resistance plays a pivotal role in the pathogenesis of PCOS, although the mechanisms underlying PCOS are not completely understood. The presence of hyperinsulinemia in patients with PCOS, independent of obesity, was previously confirmed [9,10].

Adipose tissue is now considered an active organ that secretes substances, which may play a role in the pathogenesis of insulin resistance [11]. In recent years, numerous studies pointed out that so-called true adipkines, secreted only by fat cells, as well as other adipocytokines, which can also be secreted by stromal cells in adipose tissue, play a significant role in the regulation of insulin sensitivity [12–14].

Omentin is a novel adipokine that is preferentially produced by visceral adipose tissue compared with subcutaneous tissue [15,16]. Omentin also plays a key role in glucose metabolism because *in vitro* experiments revealed that treatment with recombinant omentin-1 enhanced insulin-stimulated glucose uptake in human subcutaneous and omental adipocytes, triggering Akt signaling in both the presence and absence of insulin [16].

Besides omentin, chemerin is a recently identified adipokine that is highly expressed in liver and adipose tissue and is associated with adiposity, insulin resistance, metabolic syndrome risk factors and the degree of nonalcoholic fatty liver [17–19]. In addition, recent studies have associated chemerin with obesity and type 2 diabetes [20, 21].

In the current study, due to limited reports of omentin and chemerin in PCOS, our aim was to investigate whether overweight/obesity is associated with these novel adipokines.

Materials and methods

Subjects and data collection

The study group consisted of 81 women with PCOS (41 lean, BMI $< 25\,kg/m^2$ and 40 overweight or obese, BMI $> 25\,kg/m^2$) and 61 healthy subjects (31 lean, BMI $< 25\,kg/m^2$ and 30 overweight or obese, BMI $> 25\,kg/m^2$; control group). The patients with PCOS and the healthy subjects were recruited from the outpatient endocrinology and gynecology clinics of Namik Kemal University Hospital, Turkey, between 2011 and 2012. The study was approved by the institutional review board of the hospital, and all participants signed informed consent.

The diagnosis of PCOS was based on Rotterdam criteria, in which two of the following three features were

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present: (1) oligoovulation and/or anovulation, (2) clinical and/or biochemical signs of hyperandrogenism, and (3) polycystic ovaries on ultrasound examination (the presence of 12 or more follicles measuring 2–9 mm in diameter and/or ovarian volume $>10\,cm^3$) [22].

None of the controls had signs or symptoms of the Rotterdam criteria. Patients who had hyperprolactinemia, congenital adrenal hyperplasia, thyroid disorders, Cushing's disease, hypertension, hypercholesterolemia, a history of neoplasm, and those using medication (e.g. insulin-sensitizing drugs, oral contraceptives, antiandrogens, statins, aspirin, corticosteroids and GnRH agonists and antagonists) during the period of 90 days prior to enrollment were excluded.

Hirsutism was evaluated using the modified Ferriman-Gallwey score [23]. The waist-to-hip ratio (WHR) was calculated by dividing the minimal waist circumference by the hip circumference at the level of greater trochanters. The BMI was calculated as weight (kg)/height squared (m^2).

Biochemical analysis and hormone assays

Fasting blood samples were drawn in the morning during the follicular phase (cycle days 2–8) in the patients with a cycle length shorter than three months. Blood samples from patients with a cycle length greater than 3 months were drawn on a random cycle day. Blood tests included the measurement of androgens (17-hydroxyprogesterone, dehydroepiandrosterone sulphate (DHEA-S), total testosterone, luteinizing hormone (LH), follicle stimulating hormone (FSH) and prolactin).

An OGTT was performed at 8:00 am on a random day of the cycle. Insulin and capillary blood glucose levels were measured at baseline and at 30, 60 and 120 minutes after oral ingestion of a glucose load containing an equivalent of 75 g anhydrous glucose dissolved in water.

Insulin resistance, defined by the homeostasis model assessment insulin resistance index (HOMA-IR), was calculated with following equation: HOMA-IR = fasting insulin (IU/mL) × fasting glucose (mmol/L)/22.5 [24].

Chemerin measurement

Chemerin levels in the serum were quantified by an enzyme-linked immunosorbent assay (ELISA) (BioVendor, Laboratomi Medicina, Karasek, Czech Republic). The detection limit of this assay was 0.061 ng/mL. Intra and inter assay variabilities were less than 10% and 12%, respectively.

Omentin measurement

Serum omentin levels were determined with an omentin ELISA kit (BioVendor, Laboratomi Medicina, Karasek, Czech Republic). The intra-assay and inter-assay coefficients of variation of this kit were 4.1% and 4.8%, respectively.

Statistical analysis

All data were analyzed with the use of the Statistical Package for the Social Sciences for Windows software (Version 16.0 SPSS, Chicago, IL). Data are presented as mean and SD or percentage. The Shapiro–Wilk W test was used to identify whether the variables were normally distributed. The differences between groups were assessed by using unpaired *t*-tests for parametric data and Mann–Whitney U-test for nonparametric data. Fisher's exact test was used to compare differences in rates. Correlations between variables were evaluated with the use of Pearson's correlation coefficient. Subsequently, where individual correlations achieved statistical significance, variables were entered into a linear regression model. Statistical significance was defined as $p < 0.05$.

Results

In all subjects with PCOS ($n = 80$), serum chemerin levels were higher compared with those of the controls ($n = 58$) (7.71 ± 1.78 ng/mL versus 6.94 ± 0.82 ng/mL, $p = 0.003$). However, serum omentin levels were not significantly different between the PCOS subjects and the controls (1.55 ± 0.43 ng/mL versus 1.69 ± 0.37 ng/mL, $p = 0.056$). The PCOS group had higher LDL cholesterol and triglyceride than the control group (111.72 ± 30.58 mg/dL versus 102 ± 20.83 mg/dL, $p = 0.041$; 107.19 ± 73.20 mg/dL versus 84.28 ± 37.2 mg/dL, $p = 0.031$). In contrast, the control group had higher serum HDL cholesterol levels than the PCOS group (53.26 ± 12.76 mg/dL versus 48.01 ± 12.35 mg/dL, $p = 0.018$). The mean insulin and HOMA levels were higher in the PCOS group than in the control group (9.76 ± 7.6 IU/mL versus 6.86 ± 4.37 IU/mL, $p = 0.0011$; 3.97 ± 3.29 versus 2.84 ± 1.99, $p = 0.0023$).

The clinical characteristics and biochemical data of the women diagnosed with PCOS and the control subjects are shown in Table 1. When the subjects with PCOS and the controls were

Table 1. Metabolic, clinical characteristics and omentin, chemerin levels for healthy controls and PCOS.

Parameters	Obese PCOS ($n = 40$)	Non-obese PCOS ($n = 40$)	Non-Obese Controls ($n = 27$)	Obese Controls ($n = 30$)
Age (years)	28.11 ± 5.63*	23.35 ± 5.49	25.46 ± 4.69	24.37 ± 3.87
BMI (kg/m^2)	31.47 ± 5.08*	20.99 ± 2.37	21.29 ± 2.16	27.47 ± 3.55
Waist:hip ratio	0.85 ± 0.0065	0.78 ± 0.06	0.76 ± 0.06	0.91 ± 0.51
2 hr Glucose (mg/dL)	111.05 ± 27.32	93.27 ± 20.12¶	82.81 ± 21.05	110.90 ± 30.36
Fasting glucose (mg/dL)	91.50 ± 7.48	88.51 ± 7.34	88.59 ± 6.32	93.66 ± 8.43
Insulin (IU/mL)	12.93 ± 8.2*	6.83 ± 5.66	5.43 ± 2.81	8.29 ± 5.17
HOMA	5.29 ± 3.43*	2.71 ± 2.63	2.15 ± 1.12	3.53 ± 2.41
Triglycerides (mg/dL)	138.72 ± 84.11	78.81 ± 47.04	72.27 ± 25.02	96.1 ± 43.4
Total Cholesterol (mg/dL)	193.8 ± 32.78*	166.54 ± 32.94	176.24 ± 21.19	168.09 ± 46.61
HDL (mg/dL)	44.45 ± 12.5	51.21 ± 11.45¶	96.23 ± 19.1	46.90 ± 9.43
LDL (mg/dL)	123.41 ± 25.81*	101.21 ± 31	59.84 ± 12.54	107.58 ± 21.23
Omentin (pg/mL)	1.50 ± 0.4†	1.61 ± 0.45	1.74 ± 0.47	1.64 ± 0.22
Chemerin ng/mL	8.98 ± 1.45*,‡,†	6.57 ± 1.17	6.86 ± 0.96	7.02 ± 0.67
DHEA-S (μmol/L)	198.26 ± 112.36	224.20 ± 96.92	226.17 ± 112.89	192.38 ± 66.7

*$p < 0.05$ Obese PCOS versus Obese control
¶$p < 0.05$ Lean- PCOS versus lean control
‡$p < 0.05$ Lean- PCOS versus obese PCOS
†$p < 0.05$ Lean- PCOS versus obese control

Table 2. Correlation values omentin and chemerin and metabolic, hormonal parameters in women with PCOS.

	Omentin (r)	Chemerin (r)
Age (Years)	0.077	0.301*
BMI	−0.173	0.612*
WHR	0.007	0.524*
2 h Glucose	−0.128	0.538*
Fasting glucose	0.013	0.208
HOMA	−0.152	0.299*
Triglycerides	−0.005	0.506*
Total Cholesterol	−0.090	0.211
HDL	−0.098	−0.260
LDL	0.069	0.174
LH	0.073	0.023
FSH	0.064	−0.150
E_2	−0.003	0.107
DHEA-S	−0.109	−0.169

*$p < 0.05$.

Table 3. Correlations of serum chemerin levels with six factors by linear regression analysis.

	Regression coefficient	Standard error	p Value
PCOS	0.172	0.233	0.023
Fasting glucose (mg/dL)	0.119	0.005	0.150
Body mass index (kg/m^2)	0.404	0.026	0.000
Triglycerides (mg/dL)	0.315	0.002	0.000
HDL (mg/dL)	0.191	0.011	0.030
Insulin (IU/mL)	−0.019	0.021	0.830

A Linear regression analysis was used to examine correlations of serum chemerin level with six factors that had a significant correlation by a simple regression analysis.

categorized by BMI, there were four groups: Group 1 obese PCOS ($n = 40$); Group 2 non-obese PCOS ($n = 40$); Group 3 obese controls ($n = 30$); and Group 4 non-obese controls ($n = 27$). The mean chemerin concentrations were significantly elevated in the obese PCOS group compared with the obese control subjects (8.98 ± 1.45 ng/mL versus 7.02 ± 0.67 ng/mL, $p = 0.000$) and the non-obese PCOS group compared with the obese control subjects (6.57 ± 1.17 ng/mL versus 7.02 ± 0.67 ng/mL, $p = 0.000$). Subgroup analyzes did not reveal a statistically significant difference in the mean omentin levels between the groups.

As shown in Table 2, chemerin was significantly correlated with age ($r = 0.301$; $p < 0.05$), BMI ($r = 0.612$; $p < 0.05$), WHR ($r = 0.524$; $p < 0.05$), 2 h glucose ($r = 0.538$; $p < 0.05$), HbA1c ($r = 0.371$; $p < 0.05$), HOMA-IR ($r = 0.299$; $p < 0.05$), and triglycerides ($r = 0.506$; $p < 0.05$).

Stepwise regression analysis was used to assess the individual contributing factors. Among these contributing factors, chemerin levels showed the change in PCOS ($r^2 = 0.172$), BMI ($r^2 = 0.404$), Triglycerides ($r^2 = 0.315$) and HDL ($r^2 = 0.191$) (Table 3).

Discussion

In the present study, we examined omentin and chemerin concentrations in PCOS women compared to healthy controls and their relationships with indices of metabolic parameters. We found that chemerin levels were significantly higher in women with PCOS than those without PCOS but not significantly different between non-obese women with and without PCOS after considering the effect of obesity. Furthermore, we found the omentin levels were significantly lower in patients with PCOS compared to the healthy subjects.

Individuals with high chemerin serum levels coupled with low circulating adiponectin had a significantly increased risk of dyslipidemia and metabolic syndrome [25]. It is well established that an altered adipokine profile leads to profound changes in insulin sensitivity and other biochemical alterations of metabolites, causing an individual to be more prone to metabolic disorders. Omentin levels are known to be lower in patients with glucose intolerance and diabetes [16]. However, previous studies of omentin and chemerin levels in patients with PCOS were limited. Tan et al., reported that chemerin levels were increased and that omentin levels were significantly lower in women with PCOS [26,27].

Some studies showed a relationship between chemerin and the presence of metabolic syndrome and coronary artery disease [28,29]. Another study suggested that chemerin is a novel negative regulator of FSH-induced follicular steroidogenesis and that it may contribute to the pathogenesis of PCOS [30]. The elevated chemerin levels in patients with PCOS in the current study may indicate early signs of cardiovascular and metabolic risk in these women compared to healthy subjects.

The adipokine profile is linked to different parameters of adiposity (total body fat, percentage body fat and fat distribution). Visceral fat accumulation is more important than whole body adiposity in the development of diabetes, lipid disorders and atherosclerosis [31].

In our study, we found that chemerin levels were positively associated with an increased BMI in women with PCOS. We did not observe any significant difference in chemerin and omentin levels in patients with and without PCOS, regardless of the presence of obesity. It is clear that adiposity plays an important role in maintaining and generating PCOS. It is also evident that obesity worsens the phenotype (metabolic and reproductive) in many women [32]. As previously described, circulating omentin increases after weight loss-induced improvements in insulin sensitivity [33,34]. Therefore, the control of obesity in patients with PCOS might also help to control IR and improve metabolic parameters.

There are a number of limitations in our study. The study was a cross-sectional design and did not include age-weight matched groups.

In conclusion, fat mass rather than a PCOS diagnosis *per se* seems to be the main determinant of increased chemerin and decreased omentin in women with PCOS. Thus, this study raises the possibility that strategies that can modulate concentrations of omentin and chemerin would improve glucose tolerance and metabolic disarrangements in women with PCOS.

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Decleration of interest

We certify that there are no conflicts of interest with any financial organization regarding the material discussed in the manuscript.

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