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Original article

The comparison of serum vaspin and visfatin concentrations in obese and normal weight women

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ABSTRACT

Background and objectives: There is evidence based studies which show that plasma level of visfatin and vaspin in patients with type 2 diabetes mellitus elevate in comparison with healthy people. But there is no consistency in plasma visfatin and vaspin concentration between studies done on obese people. For this reason, the aim of this study is to investigate the serum level concentrations of visfatin and vaspin in obese women compared to normal weight women.
Materials and methods: The participants of this study consist of 43 women aged 20–50, and 43 healthy women with normal weight as a control group. They were matched for age and physical activity. 24 h food recall was used to collect dietary information from subjects. Moreover, blood sampling was taken to measure the blood levels of sugar, lipid profile, vaspin and visfatin.
Results: The mean serum level of visfatin was not statistically different between obese and normal weight women. But, the obese women had statistically higher mean serum level of vaspin than normal women ($p = 0.04$). We found no relations between serum levels of vaspin with serum concentration of visfatin. Also, serum levels of these two adipokines were not related to the serum concentrations of fasting glucose, total cholesterol, low-density lipoprotein cholesterol and triglyserides and high-density lipoprotein cholesterol. Also, there was a significant positive relationship between carbohydrate intake and serum visfatin level in women participating to this study ($p = 0.018$, $r = 0.257$).
Conclusion: The results of this study demonstrated that the level of serum vaspin was significantly higher in obese women. But there were no differences in serum levels of visfatin in comparison to normal weight women. Meanwhile this study demonstrated a positive relationship between serum levels of visfatin with dietary intake of carbohydrate, but no relationship between serum level of visfatin and vaspin in women participating in this study.

1. Introduction

Today, the adipose tissue considered as an endocrine organ which releases many adipokines such as leptin, Acylation Stimulating Protein (ASP), chemerin, adiponectin, vaspin and visfatin [1].

Visfatin, which is an adipocytokine, that was recently identified by Fukuhara et al. [2]. Visfatin has a potential insulin-mimic action that may increases insulin sensitivity [2]. Visfatin is also called NAMPT because of its significant sequence and functional homology with nicotinamide phosphoribosyltransferase (NAm-PRTase), an enzyme involved in nicotinamide adenine dinucleotide (NAD) biosynthesis from nicotinamide [3].

Visfatin is mainly secreted from visceral adipose tissue, so the plasma concentration of visfatin correlates with the amount of visceral fat in humans [2].

Vaspin, another adipokine which is secreted from adipose tissue, is a member of serin protease family. This adipokine was first extracted from adipose tissue of OLETF [4]. Otsuka–Long–Evans–Tokushima Fatty (OLETF) rat is an animal model of type 2 diabetes mellitus which has characteristics of abdominal obesity, insulin resistance, hypertension and dislipidemia [5]. Vaspin has insulin sensitizing property and there is sufficient evidence to suggest that the expression of visceral vaspin in humans had significant relations with their BMI, body fat percentage and plasma level of 2 h glucose [6].

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There are some studies showing that plasma visfatin concentrations in patients with type 2 diabetes mellitus is elevated compared to healthy people [7], but however, there is no consistency in plasma visfatin concentration between studies done on obese people.

Some studies showed that the plasma visfatin concentration in obese people increases [8–10], however, there is one study showing reduced plasma visfatin levels in obese subjects [11]. Also, the role of vaspin in obesity development is not entirely clear.

It is interesting that both weight reduction [12,13] as well as over-nutrition resulted in reduced circulating visfatin concentrations in humans [14]. According to these observations in humans and animals, the regulation of visfatin and vaspin production under the conditions of obesity is not completely clear and also seems different between people with different genetic background which may be related to different polymorphism forms of these adipokine genes in different people [15].

For this reason, the purpose of this study is investigating the serum levels of visfatin and vaspin in obese women in comparison with normal weight women.

2. Subjects and methods

The participants of this study consist of 43 women aged 20–50, and 43 healthy women with normal weight as a control group. They were matched for age and had a low physical activity level. These women were selected from clients recruited to nutrition clinic of Amir-al Momenin Hospital of Qazvin University of medical sciences, Qazvin, Iran. Subjects who met the following criteria were eligible to participate in this study: having $30 \leq \text{BMI} \leq 39.9\ \text{kg/m}^2$ for obese group and having $18.5 \leq \text{BMI} \leq 24.9\ \text{kg/m}^2$ for normal weight group. Also, the exclusion criteria for this study include: excessive obesity ($\text{BMI} \geq 40\ \text{kg/m}^2$) and overweight ($25 \leq \text{BMI} \leq 29.9\ \text{kg/m}^2$), history of intensive liver, biliary and pancreatic disease, diabetes, hypertension, hypothyroidism, secondary obesity, menopause, pregnancy and breast feeding, and consuming any drug or supplement.

All subjects provided written consent, and the Ethical Committee of the Tehran University of Medical Sciences, approved the study.

From each participant a 3-day food recall was filled and blood samples were taken after 12–14 h fasting overnight. After centrifuge, serums were stored at −80 °C for subsequent analyses. Height was measured to the nearest centimeter, weight to the nearest kilogram. BMI was calculated as the weight divided by the square of height. The waist circumference was measured at the narrowest part of the torso, the hip circumference was measured in a horizontal plane at the level of the maximal extension of the buttocks. Then, the waist-to-hip circumference ratio (WHR) was determined by dividing waist circumference to the measure of hip circumference.

Biochemical parameters including glucose, triglycerides (TG), total cholesterol (TC) and high-density lipoprotein cholesterol (HDL-C) were measured by Behring RXL autoanalyser (Germany). And LDL-C was calculated by the Friedwald formula, LDL-C (mg/dl): TC − [HDL-C + TG/5] [16].

Serum vaspin was measured by Human visceral adipose-specific serine protease inhibitor (vaspin) ELIZA kit (CUSABIO BIOTECH, Wuhan, China), with the sensitivity of 0.8 pg/ml and an intra-assay and inter-assay variability of 1.3–3.8 and 3.3–9.1%, according to the manufacturer.

Plasma visfatin was measured by human visfatin ELIZA kit (CUSABIO BIOTECH, Wuhan, China) with the sensitivity of 0.16 ng/ml and an intra-assay and inter-assay variability of 1.3–3.8 and 3.3– 9.1%, according to the manufacturer.

Statistical analysis was performed using SPSS version (11.5). All data were expressed as means ± standard deviation (SD). Comparisons of the groups were examined by Student's *t*-test. Pearson correlation test was used to determine the relationship between continuous variables. P value < 0.05 was considered statistically significant for all analyses.

3. Results

Biochemical and anthropometric characteristics of obese and control subjects are illustrated in Table 1.

There was no significant difference in mean age of two groups. The obese group had significantly higher weight, BMI, waist and hip circumferences and WHR compared with healthy controls. Also, obese women had significantly higher fasting glucose ($p = 0.026$), total cholesterol (<0.001), low-density lipoprotein cholesterol ($p = 0.002$) and triglycerides (<0.001) but lower high-density lipoprotein cholesterol (0.004) than control subjects. Serum vaspin concentration in obese women was significantly higher than normal weight women (84.9 ± 37.6 vs. 70.6 ± 25.5 pg/ml, $p = 0.043$, respectively). Visfatin values in obese women were not significantly different from control subjects (4.01 ± 6.70 ng/ml vs. 4.70 ± 8.96 ng/ml, $p = 0.69$) (Table 1).

Table 2 shows some dietary intakes of two groups. The mean intake of energy, protein and fat in the obese women were higher than normal weight women, but dietary intake of carbohydrate between two groups was not statistically different. The mean % of dietary intake from each macronutrient was not different between two groups.

In this study we found no relation between serum level of vaspin with serum concentration of visfatin. Also, serum level of

Table 1
Anthropometric and biochemical parameters of obese subjects and controls (means ± SD).

	Obese ($n = 43$)	Control ($n = 43$)	*P*-value
Age (yr)	41.7 ± 6.4	43.5 ± 5.8	ns
Weight (kg)	81.1 ± 7.6	55.0 ± 5.7	<0.001
Height (cm)	156.7 ± 6.4	157.6 ± 6.6	ns
BMI (kg/cm^2)	32.99 ± 2.0	22.15 ± 1.67	<0.001
Waist circumference (cm)	98.63 ± 4.98	83.51 ± 3.77	<0.001
Hip circumference (cm)	115.63 ± 3.52	100.12 ± 5.93	<0.001
WHR	0.85 ± 0.04	0.83 ± 0.032	0.026
Fasting glucose (mg/dl)	98.67 ± 23.03	89.81 ± 11.30	0.026
Total cholesterol (mg/dl)	192.12 ± 29.05	170.95 ± 18.77	<0.001
High-density lipoprotein cholesterol (mg/dl)	44.09 ± 7.01	48.35 ± 6.25	0.004
Low-density lipoprotein cholesterol (mg/dl)	115.23 ± 32.81	96.69 ± 18.17	0.002
Triglycerides (mg/dl)	163.95 ± 37.99	126.58 ± 38.66	<0.001
Visfatin (ng/ml)	4.01 ± 6.70	4.70 ± 8.96	ns
Vaspin (pg/ml)	84.9 ± 37.6	70.6 ± 25.5	0.043

Data are expressed as mean ± SD. ns, not significant.