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Relation of visfatin to cardiovascular risk factors and adipocytokines in patients with impaired fasting glucose

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ABSTRACT

Objective: The discovery of visfatin has great potential to significantly enhance our understanding of impaired fasting glucose and diabetes mellitus. The aim of the present study was to explore the relation of visfatin concentrations to cardiovascular risk factors and serum adipocytokine concentrations in patients with impaired fasting glucose.
Methods: A sample of 55 patients with impaired fasting glucose was analyzed in a prospective way. All patients with a 2-wk weight-stabilization period before recruitment were enrolled. Weight, blood pressure, basal glucose, lipoprotein(a), C-reactive protein, insulin, total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triacylglycerols, blood, and adipocytokines (visfatin, leptin, adiponectin, resistin, tumor necrosis factor-α [TNF-α], and interleukin-6) levels were measured. Tetrapolar impedancometry, indirect calorimetry, and prospective serial assessment of nutritional intake with 3-d written food records were performed.
Results: Fourteen men (25.5%) and 41 women (74.5%), with a mean age of 57.3 ± 11.7 y and mean body mass index of 35.8 ± 3.6 kg/m^2, were included. Patients were divided in two groups by median visfatin value (18.2 ng/mL): group I had low values and group II had high values. Patients in group I had greater weight, body mass index, fat mass, fat-free mass, and adiponectin than patients in group II. Patients in group II had higher total cholesterol, low-density lipoprotein cholesterol, resistin, and TNF-α levels than patients in group I. In the multivariate analysis with age- and sex-adjusted basal visfatin concentration as a dependent variable, only TNF-α remained an independent predictor in the model ($F = 8.4$, $P < 0.05$), with an inverse correlation. Visfatin concentration decreased 7.33 ng/mL (95% confidence interval 2.10–12.58) for each nanogram per milligram of TNF-α increase.
Conclusion: Only TNF-α is related in an independent way to serum visfatin levels.
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Introduction

The current view of adipose tissue is that of an active secretory organ, sending out and responding to signals that modulate appetite, insulin sensitivity, energy expenditure, inflammation, and immunity. The association between accumulation of visceral adipose tissue and insulin resistance is well established in obesity and type 2 diabetes mellitus (DM2), and visceral fat and insulin resistance are associated with increased cardiovascular risk [1].

Visfatin was recently identified as a protein preferentially expressed in visceral adipose tissue compared with subcutaneous adipose tissue [2]. Interestingly, visfatin expression is regulated by cytokines that promote insulin resistance, such as tumor necrosis factor-α (TNF-α), interleukin-6 (IL-6), and lipopolysaccharide [3]. Fukuhara et al. [2] clearly suggested an endocrine role for visfatin; it cannot be excluded that visfatin might also have a paracrine effect on the visceral adipose tissue through its pro-adipogenic and lipogenic actions. Investigations of visfatin levels in patients with diabetes mellitus are contradictory. Decreased concentrations of visfatin have been observed in subjects with gestational diabetes mellitus [4]. However, other investigators [5] have demonstrated elevated levels of visfatin in women with gestational diabetes mellitus. Some studies have observed a lack of effect on visfatin concentrations by thiazolidinediones [6]. Recently, Tsiotra et al. [7] detected that visfatin, TNF-α, and IL-6 mRNA expressions are increased in mononuclear cells from subjects with DM2. As far as we know, patients with impaired fasting glucose have not been evaluated in these

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previous studies. The discovery of this curious new adipokine has great potential to significantly enhance our understanding of impaired fasting glucose and diabetes mellitus.

The aim of the present study was to explore the relation of visfatin concentrations to cardiovascular risk factors and serum adipocytokine concentrations in patients with impaired fasting glucose.

Materials and methods

Subjects

A sample of 55 patients with impaired fasting glucose was analyzed in a prospective way (no probabilistic sample). These patients were studied in a nutrition clinic unit after signed informed consent.

Procedure

All patients with a 2-wk weight-stabilization period before recruitment were enrolled. Weight, blood pressure, basal glucose, C-reactive protein, insulin, total cholesterol, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein cholesterol, triacylglycerols, blood, and insulin resistance (homeostatic model assessment) levels were measured after informed consent was signed.

Assays

Serum total cholesterol and triacylglycerol concentrations were determined by an enzymatic colorimetric assay (Technicon Instruments, Ltd., New York, NY, USA), and high-density lipoprotein cholesterol was determined enzymatically in the supernatant after precipitation of other lipoproteins with dextran sulfate-magnesium. LDL cholesterol was calculated using Friedewald's formula. Plasma glucose levels were determined by using an automated glucose oxidase method (Glucose Analyser 2, Beckman Instruments, Fullerton, CA, USA). Insulin was measured by enzymatic colorimetry (Insulin, Wako Pure Chemical Industries, Osaka, Japan) and the homeostasis model assessment for insulin sensitivity was calculated using these values [8].

Adipocytokines

Resistin was measured by an enzyme-linked immunosorbent assay (ELISA; Biovendor Laboratory, Inc., Brno, Czech Republic) with a sensitivity of 0.2 ng/mL and a normal range of 4–12 ng/mL. Leptin was measured by an ELISA (Diagnostic Systems Laboratories, Inc., Houston, TX, USA) with a sensitivity of 0.05 ng/mL and a normal range of 10–100 ng/mL. Adiponectin was measured by an ELISA (R&D Systems, Inc., Minneapolis, MN, USA) with a sensitivity of 0.246 ng/mL and a normal range of 8.65–21.43 ng/mL. IL-6 and TNF-α were measured by an ELISA (R&D Systems, Inc.) with sensitivities of 0.7 and 0.5 pg/mL, respectively. Normal values were 1.12–12.5 pg/mL for IL-6 and 0.5–15.6 pg/mL for TNF-α.

Visfatin was analyzed using a commercially available ELISA kit (Phoenix Peptides, Belmont, CA, USA). The assay sensitivity was 2 ng/mL and the interassay and intra-assay coefficients of variation were less than 10% and less than 5%, respectively.

Anthropometric measurements

Body weight was measured to an accuracy of 0.1 kg and body mass index (BMI) was computed as body weight (kilograms)/height (meters) squared. Waist (narrowest diameter between the xiphoid process and the iliac crest) and hip (widest diameter over the greater trochanters) circumferences to derive the waist-to hip ratio were measured. Tetrapolar body electrical bioimpedance was used to determine body composition [9]. Electric currents of 0.8 mA and 50 kHz were produced by a calibrated signal generator (Biodynamics Model 310e, Seattle, WA, USA) and applied to the skin using adhesive electrodes placed on right-side limbs. Resistance and reactance were used to calculate total body water, fat, and fat-free mass.

Blood pressure was measured twice after a 10-min rest with a random zero mercury sphygmomanometer and averaged.

Indirect calorimetry

Indirect calorimetry (MedGem, Health Tech, Golden, CO, USA) was performed in a standard way (fasting conditions and 8 h of previous resting). Resting metabolic rate (kilocalories per day) and oxygen consumption (milliliters per minute) were calculated [10].

Dietary intake

Patients received a prospective serial assessment of weight and nutritional intake with 3-d written food records. All enrolled subjects received instructions to record their daily dietary intake for 3 d including a weekend day. Handling of the dietary data was by means of a personal computer equipped with personal software, incorporating the use of food scales and models to enhance portion-size accuracy. Records were reviewed by a registered dietitian and analyzed with a computer-based data evaluation system. National composition food tables were used as a reference [11].

Statistical analysis

The results were expressed as average ± standard deviation. The distribution of variables was analyzed with the Kolmogorov-Smirnov test. Qualitative variables were analyzed with the chi-square test, with Yates's correction as necessary, and Fisher's test. Quantitative variables with normal distribution were analyzed with a two-tailed, unpaired Student's *t* test. Non-parametric variables were analyzed with the Mann-Whitney U test. A multiple regression model (step by step backward) was used to study the dependent variable (visfatin) adjusted by age and sex. $P < 0.05$ was considered statistically significant. The statistical computer software package used was SPSS 15.0 (SPSS, Inc., Chicago, IL, USA).

Results

Fifty-five patients (14 men, 25.5%, and 41 women, 74.5%) gave informed consent and were enrolled in the study (approved by the ethical committee of our hospital). Their mean age was 57.3 ± 11.7 y and their mean BMI was 35.8 ± 3.6 kg/m^2. Table 1 lists the baseline characteristics of the patients. Table 2 lists the average circulating levels of adipocytokines.

All subjects had a stable weight during the 2-wk period preceding the study (body weight change 0.2 ± 0.1 kg). Anthropometric measurements showed average waist circumference (120.3 ± 11.6 cm), waist-to hip ratio (0.96 ± 0.07), and average weight (95.6 ± 17.4 kg). Tetrapolar body electrical bioimpedance showed a fat-free mass of 52.2 ± 14.5 kg and a fat mass of 42.5 ± 16.2 kg. Indirect calorimetry showed a resting metabolic rate of 2001.3 ± 603.4 kcal/d and an oxygen consumption of 298.1 ± 109 mL/min.

Serial assessment of nutritional intake with 3-d written food records showed a caloric intake of 1958 ± 861 kcal/d, a carbohydrate intake of 192.1 ± 87.3 g/d, a fat intake of 82.2 ± 41.2 g/d, and a protein intake of 90.5 ± 18.4 g/d.

Patients were divided in two groups by median visfatin value (18.2 ng/mL): group I had low values (average value 12.1 ± 3.5) and group II had high values (average value 46.6 ± 8.7). Table 3 presented the statistical differences between the groups in epidemiologic and biochemical parameters. Patients in group I

Table 1
Clinical characteristics of study population ($n = 55$)

Characteristics	Mean ± SD	Normal range
BMI (kg/m^2)	37.8 ± 6.3	<20
Systolic BP (mmHg)	137.8 ± 18.2	<130
Diastolic BP (mmHg)	85.5 ± 16.4	<85
Glucose (mg/dL)	119.5 ± 24	<110
Total cholesterol (mg/dL)	213.4 ± 40	<200
LDL cholesterol (mg/dL)	143.3 ± 37	<100
HDL cholesterol (mg/dL)	52.2 ± 10.7	<55
Triacylglycerols (mg/dL)	147.7 ± 89	<150
Insulin (mUI/L)	23.7 ± 20	<6
HOMA	7.8 ± 7.6	<2.5
CRP (mg/dL)	7.3 ± 7.3	<3

BMI, body mass index; BP, blood pressure; CRP, C-reactive protein; HDL, high-density lipoprotein; HOMA, homeostatic model assessment; LDL, low-density lipoprotein

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