ORIGINAL ARTICLE

Selected adipokines and metabolic profiles in normal-weight women with abdominal obesity

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KEY WORDS

abdominal obesity, adiponectin, adiposity, visfatin

ABSTRACT

INTRODUCTION Abdominal obesity (AO) is a risk factor of insulin resistance and its metabolic consequences.

OBJECTIVES The aim of the study was to assess the associations between adiponectin and visfatin levels, body fat (BF), abdominal and hip fat depots, blood lipid profile, insulin sensitivity surrogates, and AO.

PATIENTS AND METHODS The study included 145 healthy, premenopausal (aged 20–40 years), normal-weight women. Using the cut values of 80 cm waist circumference (WC) and 0.8 waist-to-hip ratio, we identified 38 and 68 women with AO, respectively. We assessed visfatin, adiponectin, blood lipid, glucose, and insulin levels. The body composition was assessed by dual-energy X-ray absorptiometry.

RESULTS Regardless of the criteria used to diagnose AO, we found that women with AO were heavier ($P = 0.01$), had more deliveries ($P = 0.03$), and had lower high-density lipoprotein (HDL) cholesterol levels ($P = 0.01$) than women without AO. Serum visfatin and adiponectin levels, triglycerides, low-density lipoprotein (LDL) cholesterol, glucose, insulin, and indices of insulin sensitivity and resistance were comparable between the groups. AO was associated with higher diastolic blood pressure and higher total, abdominal (android), and hip (gynoid) fat as well as the android/BF ratio (all $P < 0.01$). There was a positive correlation between glucose and WC ($r = 0.206$; $P = 0.02$). Adiponectin was positively associated with HDL cholesterol ($r = 0.248$; $P = 0.008$) and inversely with the android/BF ratio ($r = –0.218$; $P = 0.009$) and android/gynoid ratio ($r = –0.201$; $P = 0.04$). Visfatin inversely correlated with total ($r = –0.251$; $P = 0.01$) and LDL cholesterol ($r = –0.181$; $P = 0.042$).

CONCLUSIONS Normal-weight women with AO have normal adiponectin and visfatin levels, higher diastolic blood pressure, and lower HDL cholesterol levels. The android/gynoid ratio and android/BF ratio are inversely correlated with adiponectin levels.

INTRODUCTION Obesity, defined as an increase in the body mass index (BMI) above 30 kg/m^2, is a chronic and complex condition and a risk factor for many comorbidities, including cardiovascular diseases, hypertension, type 2 diabetes, and malignancy. The BMI, as a simple measure of body weight in relation to height, is routinely applied to estimate body fat (BF), despite warnings that it is not a very accurate measure of adiposity. However, although increased BF is supposed to be accompanied by increased total body mass, not only adiposity but also unfavorable fat distribution within the body (especially abdominal, visceral, fat accumulation), it may play a crucial role in the development of metabolic consequences of obesity.

Visceral obesity is widely assessed by surrogate methods such as waist circumference (WC) and waist-to-hip ratio (WHR). The former index is also the basic component of the metabolic syndrome.[1-3] It has been documented that visceral fat depot is a main contributor to insulin resistance and chronic low-grade inflammation.[4] Although direct mechanisms that might link visceral fat and the development of metabolic syndrome have not been fully elucidated, accumulating evidence suggests that it might be mediated, at least partially, by dysregulated production or secretion of adipokines.

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Adiponectin has been the most extensively studied adipokine. It has been shown that adiponectin levels are often decreased in obesity, type 2 diabetes, and insulin resistance, while high circulating adiponectin levels appear to protect against obesity-related metabolic comorbidities.[5,6] However, it is still unknown whether low adiponectin level is a cause or a consequence of insulin resistance.[6,7] Another adipokine, visfatin, exerts an insulin-like effect and its level is increased in type 2 diabetes, gestational diabetes, and impaired glucose tolerance.[8-10] However, studies evaluating the relationship between visfatin and visceral fat depot have yielded conflicting results.[8,11,12] Nevertheless, there is strong evidence that serum visfatin increases with obesity as demonstrated in a prospective cohort study, in which visfatin levels were augmented in morbidly obese subjects compared with normal-weight individuals but normalized after bariatric surgery and subsequent weight loss.[13]

The majority of previous studies evaluating adiponectin and visfatin have been performed in overweight/obese subjects or patients with type 2 diabetes or other conditions associated with insulin resistance.[8-10,13] In this study, we hypothesized that abdominal obesity (AO) in healthy, normal-weight individuals might represent a relatively early stage of metabolic dysregulation, which initiates excess visceral fat-derived metabolic consequences, including impaired glucose tolerance, dyslipidemia, or arterial hypertension. Therefore, in a homogenous sample of healthy, normal-weight, premenopausal Caucasian women with AO, we sought to determine the associations between abdominal and hip fat depots, BF, lipid profiles, insulin sensitivity surrogates, adiponectin, visfatin, and AO assessed by WC and WHR.

PATIENTS AND METHODS

Study population

The study was performed in 145 Caucasian women aged from 20 to 40 years. The inclusion criteria were as follows: normal weight defined by the BMI value ranging from 18.6 to 25.0 kg/m^2, regular menstruations, no medical conditions that required pharmacological treatment, and no apparent abnormalities on physical examination. We excluded women with a history of malignancy and prior hypertension, abnormal lipid profiles, abnormal glucose tolerance (including gestational diabetes), and rapid weight changes within the last 12 months. AO was diagnosed by an 80 cm WC cut value[1] and, additionally, by a 0.8 WHR cut value. Women without AO served as the control group.

Anthropometric measurements

Height, WC, and hip circumference were measured to the nearest 0.5 cm. WC was determined at the midpoint between the bottom of the rib cage and the iliac crest. Hip circumference was measured as the maximum circumference over the buttocks.

Assessment of body composition

Body composition including BF, regional fat, and lean mass was measured by dual-energy X-ray absorptiometry (DXA; GE Medical Systems Lunar Prodigy Advance, Madison, Wisconsin, United States; software version enCORE 10.1) using automatic total body scan mode. The regions of interest (ROIs) for regional fat were defined using the software provided by the manufacturer. The abdominal ROI (android) extended from the pelvis cut (lower boundary) to above the pelvis cut by 20% of the distance between the pelvis and neck cuts (upper boundary). The hip ROI (gynoid) was defined superiorly below the pelvis cut line by 1.5 times the height of the android ROI, inferiorly below the superior line by 2 times the height of the android ROI and laterally at the outer leg cut lines. The coefficient of variation of total body measurements was below 1.0%. A high correlation between consecutive measurements was observed for all 3 compartments of the body composition, including bone mineral content, lean mass, and BF (standard error, 0.99; all R^2 = 0.99). All scans were analyzed by a single technician.

Assays

Biochemical assessments included fasting blood glucose, insulin, lipid profile, visfatin, and total adiponectin. Serum glucose was measured by the glucose oxidase method (Glucose Konelab/T Series, Fisher Scientific, Poland). Insulin was measured by an immunoenzymatic method (DPC Biermann GmbH, Bad Neuheim, Germany). From fasting glucose and insulin measurements, the insulin resistance and insulin sensitivity were assessed using the following indices: 1) homeostasis model assessment of insulin resistance (HOMA-IR), 2) homeostasis model assessment of β-cell function (HOMA-B), and 3) quantitative insulin sensitivity check index (QUICKI). Serum total cholesterol, high-density lipoprotein (HDL) cholesterol, low-density lipoprotein (LDL) cholesterol, and triglycerides were determined by enzymatic colorimetric methods using Roche Diagnostics assays. Total adiponectin was measured using commercially available enzyme-linked immunosorbent assay kits (ALPCO Diagnostics, Salem, New Hampshire, United States). Visfatin was measured by an immunoenzymatic assay using monoclonal human antibodies (Visfatin EIA; ALPCO Diagnostics).

The study fully complied with all applicable institutional and governmental regulations concerning the ethical use of human volunteers and with the terms of the Helsinki Declaration. The Pomeranian Medical University Ethics Committee approved the study protocol, and all the recruited subjects gave their written informed consent.

Statistical analysis

Variables were presented as the mean ± standard deviation or as the number of subjects and percentage. Differences among groups in normally and non-normally distributed variables were evaluated by the Mann-Whitney *U* test, paired *t* test, or the Wilcoxon signed

TABLE 1 Characteristics of women with waist circumference ≥80 cm and <80 cm

Characteristic	Waist circumference		*P*
	≥80 cm, n = 38	<80 cm, n = 107	
age, y	31.89 ±4.42	31.33 ±5.11	0.52
parity, n	1.26 ±0.92	0.93 ±1.11	0.03
systolic blood pressure, mmHg	118.60 ±12.72	116.06 ±12.9	0.11
diastolic blood pressure, mmHg	79.55 ±8.35	75.52 ±9.41	<0.01
heart rate, beats/min	74.68 ±7.84	71.99 ±8.48	0.07
height, cm	168 ±6.18	164.37 ±5.77	<0.01
weight, kg	65.82 ±5.59	57.62 ±5.8	<0.01
body mass index, kg/m²	23.30 ±1.2	21.30 ±1.61	<0.01
waist circumference, cm	83.84 ±2.83	72.06 ±4.23	<0.01
hip circumference, cm	100.03 ±4.83	93.57 ±5.84	<0.01
waist-to-hip ratio	0.84 ±0.04	0.77 ±0.04	<0.01
visfatin, ng/ml	1.63 ±1.04	1.57 ±1.30	0.30
adiponectin, µg/ml	12.96 ±3.84	13.85 ±4.15	0.18
glucose, mmol/l	4.90 ±0.88	4.96 ±1.0	0.86
total cholesterol, mmol/l	4.86 ±0.86	4.87 ±0.86	0.69
triglycerides, mmol/l	0.97 ±0.71	0.80 ±0.31	0.15
HDL cholesterol, mmol/l	1.47 ±0.34	1.67 ±0.33	0.01
LDL cholesterol, mmol/l	2.87 ±0.56	2.83 ±0.79	0.75
insulin, µIU/ml	6.74 ±2.87	6.63 ±3.77	0.45
HOMA-IR	1.51 ±0.76	1.51 ±0.99	0.56
HOMA-B	152.95 ±177.6	147.55 ±172.5	0.54
QUICKI	0.37 ±0.03	0.37 ±0.04	0.56
body composition			
android fat, %	39.40 ±5.83	29.12 ±8.18	<0.01
android fat, kg	2.09 ±0.39	1.26 ±0.47	<0.01
gynoid fat, %	43.92 ±4.37	42.08 ±5.06	0.05
gynoid fat, kg	6.55 ±1.05	5.73 ±1.17	<0.01
body fat, %	35.91 ±4.13	30.68 ±5.57	<0.01
body fat, kg	22.57 ±3.00	17.06 ±4.15	<0.01
android/body fat ratio	0.09 ±0.01	0.07 ±0.01	<0.01
lean mass, kg	40.34 ±4.52	38.14 ±4.09	0.01

Data are presented ± SD.

Abbreviations: HDL – high-density lipoprotein, HOMA-B – homeostasis model assessment of β-cell function, HOMA-IR – homeostasis model assessment of insulin resistance, LDL – low-density lipoprotein, QUICKI – quantitative insulin sensitivity check index, SD – standard deviation, WC – waist circumference

rank test as appropriate. Linear Spearman's rank correlation coefficients and regression or non-parametric regression analyses were used to determine the relationships between continuous variables.

RESULTS In the whole group of 145 women, WC and WHR were highly correlated (r = 0.664; P <0.001). Using the WC cut value of 80 cm or higher, AO was identified in 38 women (26.2%; TABLE 1), while using the WHR cut value of 0.8 or higher, it was identified nearly 2-fold more frequently (68 women; 46.9%; TABLE 2). Regardless of the method used for diagnosis, women with AO were heavier, had more deliveries, and had lower HDL cholesterol levels than controls. Serum visfatin and adiponectin levels, TG, LDL cholesterol, glucose, insulin and the indices of insulin sensitivity and resistance were comparable between the groups. AO was associated with higher BF, android fat, and the android/BF ratio. Interestingly, lean mass was similar in women with and without AO. Women with AO had higher diastolic blood pressure than controls. Moreover, women with AO defined by WHR (but not by WC) had also higher systolic blood pressure.

As expected, both WC and WHR were positively correlated with BF and regional fat depots (TABLE 3), although overall correlations with body composition parameters were higher for WC than for WHR. We also found a positive correlation between glucose and WC (but not WHR). HDL cholesterol was inversely associated with AO diagnosed by both methods. Both visfatin

TABLE 2 Characteristics of women with waist-to-hip ratio ≥0.8 and <0.8

Characteristic	Waist-to-hip ratio		*P*
	≥0.8, n = 68	<0.8, n = 77	
age, y	31.31 ±4.69	31.62 ±5.14	0.82
parity, n	1.15 ±0.93	0.91 ±1.17	0.03
systolic blood pressure, mmHg	119.23 ±13.47	114.52 ±11.98	0.02
diastolic blood pressure, mmHg	79.25 ±9.55	74.22 ±8.43	<0.01
heart rate, beats/min	73.38 ±8.60	72.09 ±8.18	0.31
height, cm	165.31 ±5.39	165.34 ±6.65	0.95
weight, kg	60.83 ±6.43	58.83 ±6.97	0.09
body mass index, kg/m²	22.22 ±1.67	21.48 ±1.75	0.01
waist circumference, cm	78.87 ±5.95	71.87 ±5.05	<0.01
hip circumference, cm	94.72 ±5.88	95.74 ±6.52	0.50
waist-to-hip ratio	0.83 ±0.03	0.75 ±0.03	<0.01
visfatin, ng/ml	1.70 ±1.29	1.49 ±1.18	0.36
adiponectin (µg/ml)	13.19 ±3.76	14.00 ±4.33	0.29
glucose, mmol/l	4.96 ±0.93	4.94 ±1.02	0.38
total cholesterol, mmol/l	4.71 ±0.84	4.96 ±0.87	0.07
triglycerides, mmol/l	0.90 ±0.57	0.80 ±0.31	0.33
HDL cholesterol, mmol/l	1.52 ±0.36	1.70 ±0.32	0.01
LDL cholesterol, mmol/l	2.78 ±0.78	2.89 ±0.83	0.43
insulin, µIU/ml	6.59 ±3.09	6.72 ±3.91	0.69
HOMA-IR	1.50 ±0.83	1.52 ±1.02	0.68
HOMA-B	140.26 ±162.7	156.79 ±182.9	0.84
QUICKI	0.37 ±0.03	0.37 ±0.03	0.68
body composition			
android fat, %	34.4 ±9.51	29.54 ±7.61	<0.01
android fat, kg	1.69 ±0.62	1.292 ±0.48	<0.01
gynoid fat, %	42.24 ±4.95	42.85 ±4.95	0.47
gynoid fat, kg	5.89 ±1.10	5.99 ±1.27	0.66
body fat, %	32.93 ±5.95	31.28 ±5.39	0.05
body fat, kg	19.32 ±4.6	17.79 ±4.39	0.03
android/body fat ratio	0.08 ±0.02	0.07 ±0.01	<0.01
lean mass, kg	38.83 ±3.90	38.61 ±4.39	0.65

Data are presented ± SD.

Abbreviations: see **TABLE 1**

and adiponectin were not associated with total fat and regional fat depots.

Adiponectin was positively associated with HDL cholesterol (r = 0.248; P = 0.008) and inversely with the android/BF ratio (r = -0.218; P = 0.009) and android/gynoid ratio (r = –0.201; P = 0.04). Visfatin was inversely correlated with total cholesterol (r = –0.251; P = 0.01) and LDL cholesterol (r = –0.181; P = 0.042). In the multiple regression analysis, the android/gynoid ratio (β = –0.190; P = 0.028) and HDL cholesterol (β = 0.202; P = 0.021) were the most significant determinants of adiponectin levels. Visfatin concentrations were associated with BF (β = 0.191; P = 0.029) and LDL cholesterol (β = –0.262; P = 0.004).

DISCUSSION In this study, we assessed AO by surrogate methods using WC and WHR. These methods, similarly to the assessment of abdominal fat by DXA, do not discriminate subcutaneous and visceral fat in the abdominal area. However, studies suggest that the measurement of AO by WC, WHR, DXA, and computed tomography (the latter method calculates subcutaneous and visceral fat masses separately) may be equivalent in predicting obesity-related metabolic risk factors and metabolic syndrome.[14,15] AO evaluated by WC is considered as one of the components of metabolic syndrome and refers to the increased risk of type 2 diabetes and cardiovascular diseases predominantly in overweight or obese subjects. However, a subset of normal-weight individuals (with the BMI <25 m/kg²) may also display a cluster of metabolic changes typical for obesity. The metabolically obese but normal-weight (MONW) phenotype, which is associated with excess abdominal (visceral) fat, is known to predispose to insulin

TABLE 3 Correlations between waist-to-hip ratio, waist circumference, and study variables

	Waist-to-hip ratio		Waist circumference (cm)	
	r	*P*	*r*	*P*
visfatin, ng/ml	0.059	0.483	0.111	0.185
adiponectin, µg/ml	–0.113	0.177	–0.088	0.294
glucose, mmol/l	0.007	0.936	0.206	0.019
total cholesterol, mmol/l	–0.151	0.074	–0.044	0.604
triglycerides, mmol/l	0.080	0.344	0.138	0.104
HDL cholesterol, mmol/l	–0.294	<0.001	–0.287	<0.001
LDL cholesterol, mmol/l	–0.077	0.364	0.006	0.940
insulin, µIU/ml	0.001	0.988	0.049	0.563
HOMA-IR	–0.053	0.549	0.093	0.292
HOMA-B	0.004	0.959	–0.067	0.450
QUICKI	0.053	0.549	–0.093	0.292
body composition				
android fat, %	0.327	<0.001	0.603	<0.001
android fat, kg	0.354	<0.001	0.734	<0.001
gynoid fat, %	–0.031	0.711	0.281	<0.001
body fat, %	0.206	0.013	0.532	<0.001
body fat, kg	0.207	0.012	0.7	<0.001
android/body fat ratio	0.419	<0.001	0.557	<0.001
lean mass, kg	0.003	0.968	0.327	<0.001

r – Spearman's rank correlation coefficient

Abbreviations: see **TABLE 1**

resistance and its clinical consequences, including metabolic syndrome.[16,17] The prevalence of AO in the general population varies from 13% to 66% and depends on age, ethnicity, socio-economic status, eating habits, and applied diagnostic criteria.[18,19] It has been estimated that the prevalence of AO among young, normal-weight women ranges from 12% to 23%.[20-22] In this study, we assessed subjects who did not yet have MONW, but having AO they seemed to be at risk for metabolic abnormalities. Indeed, we demonstrated that women with increased WC (or WHR) had frequently higher BF and blood pressure but lower HDL cholesterol levels than the age-matched controls. Moreover, both WC and WHR were inversely correlated with HDL. These results suggest that this group of women may be at risk for arterial hypertension, especially diastolic, as well as abnormal lipid profile.

WC and WHR are widely applied in clinical settings, although both indices do not identify the same metabolic risks. Many studies have demonstrated that WHR is superior to WC in predicting cardiovascular risk[23,24] because it is composed of hip circumference, which is inversely associated with the risk of dyslipidemia, type 2 diabetes, and hypertension.[25-27] On the other hand, some studies have shown that WC better than WHR relates to the risk of metabolic abnormalities, including type 2 diabetes.[14,28] Our results seem to indirectly support the superiority of WC over WHR because we found a positive correlation between WC (but not WHR) and fasting blood glucose. Moreover, WC was better correlated with total and regional fat than the WHR. Interestingly, although both indices of AO were highly correlated and identified similar metabolic profiles (except for systolic blood pressure), the incidence of WHR equal or above 0.8 in the studied group of women was nearly 2-fold higher than the incidence of WC equal or above 80 cm.

In conclusion, normal-weight women with AO have normal adiponectin and visfatin levels, higher diastolic blood pressure, and lower HDL cholesterol level. The android/gynoid and android/BF ratios are inversely associated with adiponectin levels.

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ARTYKUŁ ORYGINALNY

Wybrane adipokiny i profil metaboliczny u kobiet z prawidłową masą ciała i otyłością brzuszną

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SŁOWA KLUCZOWE

adiponektyna, otyłość, otyłość brzuszna, wisfatyna

STRESZCZENIE

WPROWADZENIE Otyłość brzuszna (*abdominal obesity* – AO) jest czynnikiem ryzyka insulinooporności i jej następstw metabolicznych.

CELE Celem badania była ocena powiązań między stężeniami adiponektyny i wisfatyny, tłuszczem całkowitym (*body fat* – BF), depozytami tłuszczu brzusznego i biodrowego, profilem lipidów krwi, pośrednimi wskaźnikami wrażliwości na insulinę oraz AO.

PACJENCI I METODY Do badania włączono 145 zdrowych kobiet przed menopauzą (w wieku 20–40 lat), z prawidłową masą ciała. Za pomocą kryterium 80 cm dla obwodu talii (*waist circumference* – WC) oraz 0,8 dla wskaźnika talia–biodra rozpoznano AO odpowiednio u 38 i 68 kobiet. Oznaczano stężenia wisfatyny, adiponektyny, lipidów krwi, glukozy i insuliny. Skład ciała oceniano za pomocą absorpcjometrii z wykorzystaniem podwójnej energii promieniowania X.

WYNIKI Bez względu na zastosowane kryteria rozpoznania AO, stwierdzono, że kobiety z AO były cięższe ($p = 0,01$), częściej rodziły ($p = 0,03$) i miały mniejsze stężenie cholesterolu lipoprotein dużej gęstości (*high-density lipoprotein* – HDL; $p = 0,01$) w porównaniu z grupą kobiet bez AO. Stężenia wisfatyny, adiponektyny, triglicerydów, cholesterolu frakcji lipoprotein o małej gęstości (*low-density lipoprotein* – LDL), glukozy i insuliny oraz wskaźników oporności i wrażliwości na insulinę były porównywalne w obu grupach. AO wiązała się z wyższym rozkurczowym ciśnieniem tętniczym, większą masą BF oraz tłuszczu brzusznego (*android*) i biodrowego (*gynoid*), a także wyższą wartością wskaźnika *android*/BF ($p < 0,01$ dla wszystkich porównań). Stwierdzono dodatnią korelację między stężeniem glukozy i WC ($r = 0,206$; $p = 0,02$). Adiponektyna korelowała dodatnio z cholesterolem HDL ($r = 0,248$; $p = 0,008$), a ujemnie ze wskaźnikami *android*/BF ($r = –0,218$; $p = 0,009$) i *android*/*gynoid* ($r = –0,201$; $p = 0,04$). Wisfatyna korelowała ujemnie z cholesterolem całkowitym ($r = –0,251$; $p = 0,01$) i cholesterolem LDL ($r = –0,181$; $p = 0,042$).

WNIOSKI Kobiety z prawidłową masą ciała i AO mają prawidłowe stężenie adiponektyny i wisfatyny, wyższe rozkurczowe ciśnienie tętnicze i mniejsze stężenie HDL-C. Wskaźniki *android*/*gynoid* i *android*/BF są ujemnie skorelowane ze stężeniami adiponektyny.

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