Plasma visfatin level in adult Egyptians with android obesity

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Context
Android obesity is considered an important predictor of increased mortality and morbidity from diabetes mellitus and cardiovascular disorders.
Aim
Our research was conducted to assess the level of serum visfatin in android obesity and its relation to anthropometric and biochemical parameters in adults with android obesity.
Patients and methods
This study was conducted on 136 patients recruited from the outpatient obesity clinic of Specialized Medical Hospital, Mansoura University. The patients were divided into two groups: group I consisted of 65 control nonobese individuals, and group II consisted of 71 obese individuals with android obesity. The obese patients were subdivided into obese diabetic patients (35 patients) and obese nondiabetic patients (36 patients). All participants were subjected to thorough history taking, full clinical examination, and anthropometric measurements to assess body mass index and waist circumference. The changes that appeared in the carbohydrate metabolism were interpreted according to the criteria from the glycometabolic classification of WHO. Plasma glucose levels, lipid profile, and serum visfatin were measured.
Results
Our study demonstrated a significant elevation of visfatin ($P \leq 0.001$) in obese individuals (37.6; 20.7–65.9) compared with that in lean patients (15.3; 7.5–20). Additionally,visfatin levels were higher in the diabetic obese subgroup (45.5; 33.7–65.9) than in the nondiabetic obese subgroup (32; 20.7–46.5). Furthermore, visfatin was positively correlated to blood glucose in the obese group, which suggests a role of visfatin in glycemic control. A significant correlation between visfatin and lipid profile was demonstrated, which may suggest a role for visfatin in lipid homeostasis.
Conclusion
Serum visfatin was elevated in patients with android obesity, with more significant elevation in patients with android obesity with type 2 diabetes mellitus. It showed potential to be used as a marker of metabolic syndrome as it had a strong relation to android obesity.

Keywords:
android obesity, diabetes mellitus, plasma visafatin

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Introduction

Obesity has been officially recognized as a disease by the American Medical Association, an action that could focus more effort by physicians and insurance companies to prevent or minimize its effects [1].

Android obesity has been associated with an increased risk for mortality and morbidity from cardiovascular disorders, metabolic syndrome (MS), diabetes mellitus, and some types of cancer [2]. Android obesity, defined as increased waist circumference (WC), is one of the components of metabolic abnormalities collectively called the MS. The latest definition of MS by the International Diabetes Federation has included android obesity as one of the essential components [3]. However, it is still unclear whether the visceral (intra-abdominal) or the subcutaneous component of abdominal fat is more harmful from the metabolic point of view. Elevated visceral adipose tissue (VAT), in particular, is associated with insulin resistance, dyslipidemia, systemic inflammation, diabetes, hypertension, myocardial infarction, and all-cause mortality [4].

Adipocytokines are mainly adipocyte-derived cytokines regulating metabolism and are key regulators of insulin resistance. Several adipocytokines such as leptin and adiponectin affect immune and inflammatory functions. Visfatin has recently been identified as a new adipocytokine and corresponds to a protein identified

previously as pre-B-cell colony-enhancing factor, a 52-kDa cytokine expressed in lymphocytes that bind to the insulin receptor affecting insulin resistance [5].

Numerous publications have reported various effects and correlations of visfatin with different medical conditions. In this way, increase in visfatin level can be observed in atherosclerosis, endothelial dysfunctions, MS, and renal insufficiency. The relationship with diabetes mellitus and visceral fat is controversial [6].

This adipokine has physiological influences on the control of glucose homeostasis through its insulin mimetic action; however, its association with obesity is not clearly established [7]. Another function of visfatin is the regulation of inflammatory and immunomodulating processes [5]. It was demonstrated that serum levels of visfatin were independently correlated withC-reactive protein and interleukin-6 in obese patients [8]. Molecular screening, epidemiological survey, and pharmacological studies have indicated that visfatin/Nampt may be an attractive diagnostic and drug target for cancer therapy [9].

So far, only a few studies have assessed visfatin in VAT and plasma visfatin concentrations in humans, and their results are divergent.

This study was proposed to assess serum visfatin level as well as its relation to select anthropometric and biochemical parameters in adults with android obesity.

Patients and methods

This study was conducted on 136 patients who were divided into two groups. Group I included 65 healthy nonobese patients with BMI less than 25 kg/m^2, and group II included 71 obese patients with android obesity with BMI greater than 30 kg/m^2. All patients gave their formal consent. The protocol was approved the Ethical Committee of the Department of Mansoura Faculty of Medicine, Mansoura University, Mansoura, Egypt. The diagnosis of android obesity was made as per The International Diabetes Federation guidelines for WC cutoff points for Egyptians (80 cm for women and 94 cm for men). Group II was subdivided into 35 obese diabetic patients and 36 obese nondiabetic patients. All participants were recruited from the outpatient obesity clinic of Specialized Medical Hospital, Mansoura University. All of them underwent thorough history taking and clinical assessment using anthropometric examinations to assess body mass index and WC.

The changes that appeared in the carbohydrate metabolism were interpreted according to the criteria from the glycometabolic classification of WHO. The basal plasma glucose was determined through enzymatic method with glucose-6 phosphate. The normal values of plasma glucose were considered as values below 6.1 mmol/l. Plasma lipids were determined from a blood sample collected in the morning, after an overnight fast of a minimum 10 h. The total cholesterol and high-density lipoprotein (HDL)-cholesterol and triglycerides were determined through an enzymatic photometric method. Low-density lipoprotein (LDL)-cholesterol was calculated by the Friederwald formula: LDL-cholesterol=Total cholesterol–HDL-cholesterol–(triglycerides/2.2).

We determined the ratios between total cholesterol/HDL-cholesterol and LDL-cholesterol/HDL-cholesterol, the values over five being considered as indicating high atherogenic risk. The determination of visfatin was done from blood samples collected in the morning, in a fasting state, through enzyme-linked immunosorbent assay (R&D System), according to the reagents' working protocol attached to the kit.

Statistical analysis

Data were statistically analyzed using the statistical package for the social sciences (SPSS, version 16; SPSS Inc.).

(1) Qualitative data were explained in numbers and percentages. The χ^2-test was used for comparison between groups.
(2) Quantitative data were described as medians or means (SD), as appropriate.

They were tested for normality by means of the Kolmogorov–Smirnov test. The independent sample *t*-test was used for normally distributed variables and the Mann–Whitney test for non-normally distributed variables in the comparison between groups.

(1) Odds ratios and their 95% confidence interval were calculated. *P*-values less than or equal to 0.05 were considered to be statistically significant.

Results

This study was conducted on 71 obese individuals and 65 healthy controls.

Discussion

In recent times, many research studies have been working on different types of obesity and have been

Table 1 Comparison of demographic characters of the studied participants

	Obese patients (*n*=71)	Control (*n*=65)	Significance	Odds ratio (95% CI)
Age (mean±SD)	42.48±11.07	39.5±10.7	*t*=1.6 *P*=0.11*	
Sex [*N* (%)]				
Male	20 (28.2)	18 (27.7)	χ^2=0.004 *P*=0.95*	1.024 (0.484–2.17)
Female	51 (71.8)	47 (72.3)		

CI, confidence interval. **P*-value is significant at ≤0.05, ≤0.001.

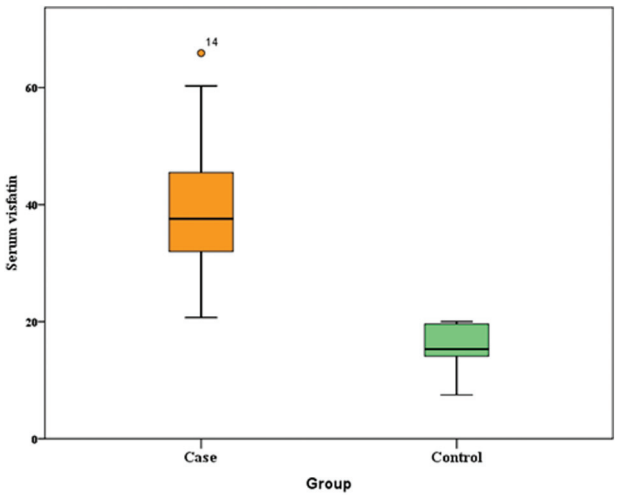
Table 2 Comparison of serum visfatin level, lipid profile, blood sugar level, and anthropometric measurements among obese patients and controls

Parameters	Obese patients (*n*=71) (mean±SD)	Control (*n*=65) (mean ±SD)	Significance
Serum visfatin	37.6 (20.7–65.9)	15.3 (7.5–20)	Mann–Whitney *U* *P*≤0.001*
BMI	36.65±4.16	23.4±1.05	*t*=24.92*P*≤0.001*
Weight	95.79±18.16	64.62±8.79	*t*=12.56*P*≤0.001*
Height	162.37±7.85	159.77±30.17	*t*=0.7*P*=0.485*
Waist circumference	138 (120–130)	82 (54–100)	Mann–Whitney *UP*≤0.001*
Hip circumference	123.2±8.13	118.08±12.31	*t*=2.894*P*=0.004*
Waist hip ratio	1±0.01	0.64±0.049	*t*=62.6*P*≤0.001*
Fasting blood sugar	99.62±11.8	78.75±6.72	*t*=12.5 *P*<0.0001
Postprandial blood sugar	161.03±14.93	114.62±50.07	*t*=7.5 *P*<0.0001
Serum cholesterol level	224.23±51.16	160.06±17.58	*t*=9.6 *P*≤0.001*
Serum TGS	125 (50–327)	75 (50–160)	Mann–Whitney *U* *P*≤0.001*
Serum LDL	99.46±36.97	80.02±16.46	*t*=3.9 *P*≤0.001*
Serum HDL	46.49±15.21	50.18±13.35	*t*=1.49 *P*=0.136

HDL, high-density lipoprotein; LDL, low-density lipoprotein; *t*, independent test; TGS, triglycerides. **P*-value is significant at ≤0.05, ≤0.001.

searching for new markers or predictors for comorbidities of obesity. The aim of the present study was to assess serum visfatin level and its relation to some biochemical parameters and anthropometric measurements in adult android obesity. The study was conducted on 71 patients with android obesity and 65 healthy individuals matched for age and sex who served as the control group. All participants were subjected to full history taking, clinical examination, assessment of anthropometric parameters [body wieght, BMI, WC, and hip circumference (HC)], and biochemical assessment of lipid profile (serum total cholesterol, triglycerides, fasting blood glucose, random blood sugar). In addition, serum visfatin was assessed.

Figure 1



Comparison of serum visfatin between cases (orange) and controls (green).

The present study included 51 (71.85%) women and 20 (28.2%) men (Table 1) as the majority of individuals attending the obesity clinic at Mansoura Internal Medicine Specialized Hospitals were female, which agrees with the finding of Patidar [10]. Other studies have shown different results [11].

In this study the group of individuals with android obesity had significantly higher serum visfatin levels compared with the control group (*P*≤0.001) (Table 2 and Fig. 1). This finding agrees with the data of Zahorska-Markiewicz [12]. In contrast, some research studies found no significant difference in visfatin levels between patients with android obesity and lean individuals [13]. The reasons for such conflicting findings may be related to different population characteristics such as age and sex, as well to ethnic heterogeneity, or other confounding factors like diabetes mellitus [14]. Another possible reason may be qualitative and quantitative discrepancies such as laboratory measurements obtained using different techniques [14]. Diabetes as a systemic disease is considered an inflammatory process, especially when the glycemic state is poor. Under these conditions the plasma visfatin levels are

Table 3 Comparison of serum visfatin level between obese diabetic versus obese nondiabetic of the studied patients

	Chronic disease [median (min.–max.)]		
Clinical parameter	DM (*n*=35)	No DM (*n*=36)	Significance
Serum visfatin	45.5 (33.7–65.9)	32 (20.7–46.5)	Mann–Whitney *UP*≤0.001*

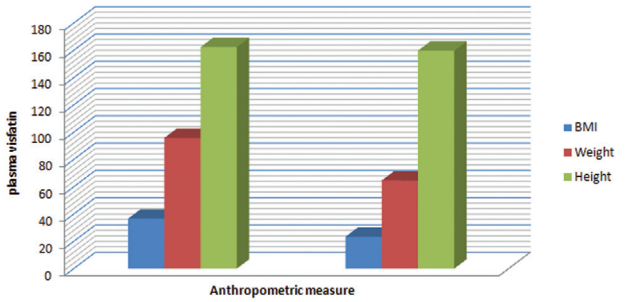
DM, diabetes mellitus; max., maximum; min., minimum. **P*-value is significant at ≤0.05, ≤0.001.

Table 4 Correlations between serum visfatin level and anthropometric measures, blood sugar level, and lipid profile among obese patients

Serum visfatin	BMI	Waist circumference	Hip circumference	FBS	RBS	Cholesterol	LDL	HDL
Pearson's correlation	0.287	0.168	0.373	0.303	0.039	–0.058	0.165	0.165
P	0.015	0.014	0.001	0.010	0.747	0.05	0.015	0.169
N	71	71	71	71	71	71	71	71

FBS, fasting blood sugar; HDL, high-density lipoprotein; LDL, low-density lipoprotein; RBS, random blood sugar.

Figure 2



Comparison between BMI (blue), weight (red), and height (green).

even higher [15]. Serum visfatin may be a promising predictor for diabetes mellitus and insulin-resistant state in individuals with obesity [16].

Our study demonstrated that serum visfatin was significantly higher in obese diabetic patients than in obese patients without diabetes (*P*≤0.001) (Table 3). This result was in agreement with those of Palin *et al.* [17]. In contrast, Taşkesen *et al.* [18] demonstrated no significant difference.

Regarding the anthropometric parameters, our study demonstrated that BMI, WC, and HC were significantly higher in obese patients when compared with the control group (*P*≤0.001, *P*≤0.001, and *P*=0.004, respectively) (Table 2 and Fig. 2).

Our study demonstrated that there was a significant positive correlation between serum visfatin level and anthropometric parameters such as BMI (*P*=0.015) and WC (*P*=0.014) in obese patients. Additionally, the data obtained in the present study demonstrated a significant positive correlation between serum visfatin and HC (*P*=0.001) (Table 4). These results might suggest that elevated serum visfatin levels that were detected in these individuals are determined by the subcutaneous adipose tissue located in the thighs and buttocks rather than by the VAT [19].

In contrast Ersoy *et al.* [20] found no correlation between serum visfatin serum level and different anthropometric parameters.

In our study, fasting blood glucose levels and random blood glucose were significantly higher in obese patients compared with the control group (*P*<0.0001 and <0.0001, respectively). In addition there was a significant positive correlation between serum visfatin level and fasting blood glucose (*P*=0.01) and random blood glucose (*P*=0.01) (Table 4). These findings were in agreement with those of Legakis *et al.* [21]. Other investigators suggested that serum visfatin levels might compensate for the impairment of insulin, whereas others found no relation [22]. Furthermore, combined components of MS, as central obesity with either insulin resistance or hyperinsulinemia, were associated with higher circulating serum visfatin levels [23].

Regarding the serum lipid profile, our study demonstrated that there were higher levels of serum cholesterol level, serum triglycerides, and serum LDL in obese patients than in control lean patients (*P*≤0.001, ≤0.001, and ≤0.001, respectively) (Table 2).

In addition, our study revealed a significant positive correlation between visfatin serum levels and different parameters of lipid profile such as serum cholesterol and serum LDL (*P*=0.05 and 0.015, respectively) and no significant correlation with serum HDL (*P*=0.629) (Table 4). These findings were in agreement with the findings of De Luis *et al.* [22], who reported the association between cholesterol-related profile and visfatin level in female patients. Another study by Catalán *et al.* [24] reported a positive association between circulating visfatin and triglycerides and total cholesterol, suggesting a role of visfatin in lipid

homeostasis, which, according to the authors, remains to be further investigated. Although the connection between serum visfatin and lipid profile may be through nicotinamide adenine dinucleotide (NAD) metabolism given its extracellular and intracellular NAD biosynthetic properties, nicotinic acid increases HDL-cholesterol and reduces triglyceride-rich lipoproteins [15]. Different results were demonstrated by Zahorska-Markiewicz [12], as they found no relation between visfatin and lipid profile parameters.

The conflicting data regarding the relation of visfatin to lipid profile was suggested to be due to different recruitment criteria in previous studies or confounding factors such as sex, age, lifestyle, duration of diabetes, and degree of obesity that may affect visfatin concentrations [22].

Conclusion

From our study, it could be concluded that serum visfatin was elevated in obese individuals with android obesity with more elevation in type 2 diabetes mellitus, which may support the hypothesis that serum visfatin may be associated with impaired glucose metabolism. Moreover, the high sensitivity and specificity of visfatin that predicts MS suggests its pathophysiological role, which might be useful in therapeutic intervention in future.

Limitations of the study

The first limitation of our study is the small number of patients. The second limitation is that we conducted our study in one center, the internal medicine hospital (diabetes clinic and diabetes inpatient department), Mansoura University. The reason for these limitations are the high cost associated with including large numbers of patients and conducting the study in different centers.

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Conflicts of interest

There are no conflicts of interest.

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