Association of Visceral Adiposity Index, Lipid Profile, and Serum Leptin with Glucose Intolerance Risks in Iraqi Obese Patients: A Cross-sectional Study

Wael Waleed Mustafa1, Samer Shukur Moahammed2, Wathiq Mohammed Al-Jewari1, Hussein Saad Abdulrahman2, Saad Abdulrahman Hussain1

Aims: The aim of this study was to evaluate the possibility of using visceral adiposity index (VAI), serum leptin, and lipid profile as indicators of impaired glucose tolerance in Iraqi obese patients. Subjects and Methods: A cross-sectional study was performed in Iraqi obese patients of both sexes. Body mass index (BMI), waist circumference, hip circumference, triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), VAI, waist-to-hip ratio (WHR), serum leptin, and 2-h glucose tolerance test (2-h GT) were determined and compared with those of healthy non-obese control group. A correlation analysis was performed to determine the strength of association between the studied markers. Data were adjusted to determine gender differences in this regard. Statistical Analysis: Kolmogorov–Smirnov, Shapiro–Wilk analyses, Mann–Whitney U test, and unpaired t test were used for the two-group comparisons once applicable. Pearson’s and Spearman’s correlation analyses were used to measure the relationship levels between the studied variables. Results: A total of 144 obese patients were included; the mean age was 37.11 ± 8.2 years and 92 (63.9%) were females. Compared with non-obese subjects, the participants had significantly higher levels of BMI, WC, WHR, VAI, TG, leptin, and 2-h GT. Obese male subjects had significantly higher values of body weight, WC, HC, VAI, and TG compared with obese females. Elevated 2-h GT was significantly associated with VAI (r = 0.291, P = 0.0004), TG (r = 0.319, P = 0.0001), and LDL-C/HDL-C ratio (r = 0.435, P < 0.0001) in the obese patients only. Conclusions: The results provide evidence that VAI, TG, and LDL-C/HDL-C ratio can be suggested as potential markers for the risk assessment of impaired glucose tolerance in Iraqi obese patients.

Keywords: Glucose tolerance, leptin, lipid profile, obesity, visceral adiposity index

Introduction

Obesity is considered as one of the most important global health concerns. It reflects the outcomes of changing lifestyles, limited physical activity, and consumption of high energy diet, probably with the association of genetic factors.[1] Although adipose tissues are distributed throughout the human body, excessive visceral adiposity can lead to “unhealthy” obesity which is correlated with metabolic abnormalities,[2] and considered as a risk factor for many disorders including diabetes mellitus and cardiovascular diseases (CVD) among others.[3,4] Although the amount of deposited
fat is higher in females, males have significantly higher percentage of visceral adipose tissue than females.\(^6\) Visceral fat is an active tissue that releases many types of bioactive chemical mediators, including leptin, resistin, TNF-\(\alpha\), and interleukins. In this regard, leptin regulates body weight and many other metabolic functions.\(^6\) In obese patients, increased level of serum leptin is evident and mostly associated with tissue resistance to the effects of this mediator.\(^7\) Additionally, many reports addressed the relationship between obesity, elevated serum leptin, and oxidative stress which are suggested as potential risk factors for atherosclerosis and other cardiovascular disorders.\(^8,9\) Currently, various methods are utilized to evaluate the amount and distribution pattern of body fat, though they demonstrate the difference in the accuracy, cost, and the required time to perform the test. However, it has been shown that waist circumference (CW) and waist-to-hip ratio (WHR) are widely accepted as markers of CVD risk assessment.\(^10\) Additionally, body mass index (BMI) and visceral adiposity index (VAI) became interested markers and widely used in clinical practice to evaluate the risk of obesity in different diseases, in addition to the previously mentioned methods;\(^11,12\) they may have an add-on value for assessment of CVD risk in obese patients. The present study aims to evaluate the relationship between the currently used obesity-related indexes of the amount and distribution of body fat and the biochemical parameters in obese patients that have no marked clinical disorders.

**Subjects and Methods**

**Study design and sample selection**

In the present cross-sectional multicenter study, 144 subjects (52 males and 92 females) aged 39.0 ± 5.9 years, with a mean BMI value of 33.8 ± 2.3 kg/m\(^2\) attending the outpatient clinics for personalized obesity treatment protocol have participated. They are metabolically obese with no clinically marked illness and recruited consecutively from September 2017 to April 2019 on the bases of their BMI value and clinical examination results. The inclusion criteria were BMI in the range of 30–35 kg/m\(^2\), non-users of medications for obesity treatment during the last 3 months before the study, non-diabetics, no history of impaired glycemic control and/or lipid profile, non-smokers (for at least 2 years) and subjects follow lifestyle modification combined exercise and diet approach for bodyweight control. Meanwhile, the exclusion criteria included pregnancy, lactation, impaired renal or hepatic function, endocrine disorders (e.g., Cushing’s syndrome, polycystic ovary syndrome), autoimmune diseases, cancer, and ischemic heart diseases. The study protocol was approved by the local Research Ethics Committee at the Faculty of Pharmacy, Al-Rafidain University College. Moreover, 100 healthy non-obese subjects (37 males and 63 females) matched for age (36.4 ± 7.6 years) with the obese group were recruited and utilized for the comparison of data. All participants accepted to participate in the study provided signed informed consent before enrollment.

**Evaluation of anthropometric and biochemical markers**

All the participants in both groups were clinically evaluated including their physical condition, medical history, and the assessment of anthropometric parameters, such as body weight, body height, waist circumference (WC), and hip circumference (HC), utilizing standard methods routinely followed in the outpatients’ clinics and obesity centers. After 12 h fasting, venous blood samples were obtained from all subjects, left to clot, and the resulted serum was utilized for the analysis of total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), triglycerides (TG), serum leptin, and fasting serum glucose level (FSG) using ready-made kits based on standard procedures. Moreover, 2-h glucose tolerance test was conducted to all subjects and the change in blood glucose levels were analyzed during 2 h after ingestion of 200 g of glucose. Based on the obtained biochemical and anthropometric data the following parameters were calculated: BMI, WHR, and VAI.

\[
\text{BMI} = \frac{\text{body mass}}{\text{height}}^2 \text{ (kg/m}^2) \text{ and the BMI range of 25.0–29.9 is commonly used to characterize overweight and a BMI value >30.0 to diagnose obesity.}
\]

\[
\text{WHR} = \frac{\text{WC (cm)}}{\text{HC (cm)}} \text{ and WHR >1 in men and >0.8 in women is commonly used to characterize the enhanced accumulation of abdominal adipose tissue.}
\]

VAI was calculated for women and men according to the following formulas:

\[
\text{VAI for men} = \left[\frac{\text{WC}}{39.68} + (1.88 \times \text{BMI})\right] \times \left(\frac{\text{TG}}{1.03}\right) \times \left(\frac{1.31}{\text{HDL}}\right)
\]

\[
\text{VAI for women} = \left[\frac{\text{WC}}{36.58} + (1.89 \times \text{BMI})\right] \times \left(\frac{\text{TG}}{0.81}\right) \times \left(\frac{1.52}{\text{HDL}}\right)
\]

**Statistical analysis**

Statistical analyses of data were performed using the Statistical Package for the Social Sciences software version 22.0 (SPSS, Chicago, Illinois) and in all analyses, a \(P\) value <0.05 was considered statistically significant. Continuous variables and categorical
variables were presented in the form of mean ± SD and frequency and percentage, respectively. Among descriptive statistics and continuous data distribution tests, the Kolmogorov–Smirnov and Shapiro–Wilk analyses were used in single groups. The Mann–Whitney U test and unpaired t test were used for the two-group comparisons once applicable. Pearson’s and Spearman’s correlation analyses were used to measure the relationship levels between the studied variables.

RESULTS

The anthropometric and biochemical characteristics of the obese and non-obese subjects participated in the study are presented in Table 1. A total of 144 obese patients, 92 (63.9%) females and 52 (36.1%) males have participated in the current study, and their results were compared with 100 healthy non-obese subjects (63% females and 37% females). The mean age of the obese patients (37.11 ± 8.2) was not significantly different from that of the non-obese (P = 0.49). Table 1 also showed that the values of HC and TC of the two groups were not significantly different (P > 0.05); while BMI, WHR, and VAI were significantly higher in the obese patients. Besides, serum levels of TG, LDL-C, leptin, FSG, and 2-h GT were also significantly elevated in obese patients (P < 0.05) compared with the non-obese group. Obese patients showed a significantly lower mean HDL-C value (48.6 ± 8.7; P = 0.001) compared with the non-obese control group. In Table 2, the data of the obese patients indicate the highly positive and significant association of VAI with serum TG (r = 0.922, P < 0.0001) and the LDL-C/HDL-C ratio (r = 0.601, P = 0.0001). In this regard, the BMI value demonstrates a significantly weak association with serum TG levels (r = 0.215, P = 0.01). In the whole obese group (males and females), a significant negative correlation between serum leptin content and LDL-C/HDL-C ratio (r = -0.228, P = 0.006) was recognized but not with other measured parameters. In Table 2, correlation analysis revealed that the risk of impaired 2-h GT in obese patients was significantly associated with serum TG and LDL-C/HDL-C ratio (r = 0.319; P = 0.0001 and 0.435; P < 0.0001, respectively).

In non-obese subjects, analysis of VAI relationships demonstrated a significantly negative association with BMI value (r = -0.299, P = 0.003), and significant positive associations with TG and LDL-C/HDL-C ratio (r = 0.911; P < 0.0001 and 0.413; P < 0.0001, respectively [Table 3]. BMI was found to be negatively associated with TG (r = -0.23, P = 0.02). Table 3 also showed that serum leptin level was negatively and significantly associated with WHR only (r = -0.495, P < 0.0001) but not with other parameters. However, the risk of impaired glycemic control (2-h GT) did not

Table 1: The comparison of study parameters between obese patients and non-obese subjects

| Variables               | Obese patients (n = 144) | Non-obese subjects (n = 100) | P value |
|------------------------|--------------------------|-------------------------------|---------|
| Gender, n (%)          |                          |                               |         |
| Male                   | 52 (36.1)                | 37 (37)                       | 0.49    |
| Female                 | 92 (63.9)                | 63 (63)                       |         |
| Age (year)             | 37.11 ± 8.2              | 36.4 ± 7.6                    | 0.49    |
| Bodyweight (kg)        | 95.32 ± 10.9             | 67.37 ± 6.3                   | 0.0001  |
| BMI (kg/m²)            | 33.8 ± 2.3               | 23.9 ± 1.01                   | 0.0001  |
| WC (cm)                | 104.7 ± 10.03            | 89.9 ± 6.2                    | 0.001   |
| HC (cm)                | 99.8 ± 9.9               | 100.4 ± 4.9                   | 0.54    |
| WHR                    | 1.1 ± 0.04               | 0.89 ± 6.1                    | 0.001   |
| VAI                    | 4.13 ± 2.2               | 3.14 ± 1.1                    | 0.0001  |
| TC (mg/dL)             | 172.8 ± 36.3             | 164.6 ± 24.6                  | 0.05    |
| TG (mg/dL)             | 146.1 ± 59.02            | 121.6 ± 37.7                  | 0.002   |
| LDL-C (mg/dL)          | 112.5 ± 30.5             | 101.7 ± 21                    | 0.002   |
| HDL-C                  | 48.6 ± 8.7               | 53.3 ± 6.6                    | 0.001   |
| LDL-C/HDL-C            | 2.43 ± 1.2               | 1.92 ± 0.43                   | 0.001   |
| TC/HDL-C               | 3.71±1.18               | 3.13 ± 0.6                    | 0.01    |
| TG/HDL-C               | 3.17±1.5                | 2.32 ± 0.74                   | 0.001   |
| Leptin (ng/mL)         | 54.1 ± 16.4              | 14.6 ± 4.8                    | 0.001   |
| FSG (mg/dL)            | 90.4 ± 15.7              | 83.6 ± 10.4                   | 0.02    |
| 2-h GT (mg/dL)         | 108.3 ± 18.6             | 84.5 ± 6.8                    | 0.001   |

BMI = body mass index, WC = waist circumference, HC = hip circumference, WHR = waist/hip ratio, VAI = visceral adiposity index, TC = total cholesterol, TG = triglyceride, LDL-C = low-density lipoprotein-cholesterol, HDL-C = high-density lipoprotein-cholesterol, FSG = fasting serum glucose; GT = glucose tolerance

Data were expressed as mean ± SD; nonparametric data were presented as numbers and percentages
show a significant correlation with any of the studied parameters [Table 3].

When the characteristics of the studied obese patients were adjusted according to their gender [Table 4], it was found that although body weights were significantly higher in men, their BMI values were not significantly different (34.1 ± 3.0 vs. 33.6 ± 1.7; P = 0.22). However, the mean values of WC, HC, VAI, TG, and TG/HDL-C were significantly higher in men compared with women (P < 0.05). Meanwhile, the mean values of the other markers (WHR, TC, LDL-C, LDL-C/HDL-C, TC/HDL-C) were not significantly different between the two groups (P > 0.05). The risk of impaired glycemic control in the obese patients (2-h GT) was not significantly different between the males and females (107.9 ± 17.5 vs. 115.3 ± 19.2; P = 0.85) regardless of the elevated values of VAI and TG in males. Additionally, the mean values of serum leptin levels were not significantly different between the different genders (53.7 ± 16.3 vs. 54.3 ± 16.4; P = 0.83) regardless of the differences in the anthropometric markers and triglycerides body contents [Table 4].

**DISCUSSION**

Currently, abdominal obesity is characterized as an important risk factor of many cardiovascular and metabolic disorders including metabolic syndrome and ischemic heart diseases. The traditionally used marker for diagnosis of obesity, the BMI, is not a gender-specific marker, cannot represent the actual metabolic condition of the subject, and distinguish the influence of non-adipose tissues in athletic individuals, in addition to its low sensitivity.[13,14] In the present study, obese patients demonstrated significantly higher BMI, WC, and WHR values compared with non-obese subjects; however, the HC values were comparable. This finding clearly supported the idea that BMI does not accurately reflect the distribution of body fat, and other more sensitive anthropometric indicators like VAI should be utilized to accurately characterize visceral adiposity.[15] Amato et al.[16] showed that VAI value >1.9 was associated with increased risks of metabolic syndrome; meanwhile, our study reported a significant association of VAI with the risk of disturbing glycemic control (2-h GT) in obese patients, which supports the utility of VAI as a risk predictor of impaired carbohydrate metabolism with a normal metabolic profile. Accordingly, VAI can be suggested as a surrogate indicator of adipose tissue dysfunction and its association with impaired lipid profile and insulin resistance; it can be utilized in daily clinical practice to evaluate the cardiometabolic risk factors in obese patients. This idea was also supported by Stepień et al.[17] who reported the association of elevated VAI

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**Table 2: Correlation (r value) between the obesity-related markers of obese patients**

| Markers | VAI | BMI | WHR | Leptin | TG | LDL-C/HDL-C | 2-h GT |
|---------|-----|-----|-----|--------|----|-------------|--------|
| VAI     | 0.197<sup>a</sup> | 0.009 | 0.089 | 0.922<sup>a</sup> | 0.601<sup>a</sup> | 0.291<sup>a</sup> |
| BMI     | 0.197<sup>a</sup> | -0.009 | -0.147 | 0.078 | -0.228<sup>a</sup> | 0.429<sup>a</sup> |
| WHR     | 0.009 | 0.111 | -0.147 | 0.013 | 0.103 | -0.066 |
| Leptin  | 0.089 | -0.009 | -0.147 | 0.078 | -0.228<sup>a</sup> | 0.429<sup>a</sup> |
| TG      | 0.922<sup>a</sup> | 0.215<sup>a</sup> | 0.013 | 0.078 | 0.429<sup>a</sup> | 0.319<sup>a</sup> |
| LDL-C/HDL-C | 0.601<sup>a</sup> | 0.148 | 0.103 | -0.228<sup>a</sup> | 0.429<sup>a</sup> | 0.435<sup>a</sup> |
| 2-h GT  | 0.291<sup>a</sup> | 0.068 | -0.066 | -0.031 | 0.319<sup>a</sup> | 0.435<sup>a</sup> |

VAI = visceral adiposity index, BMI = body mass index, WHR = waist/hip ratio, TG = triglyceride, LDL-C = low-density lipoprotein cholesterol, HDL-C = high-density lipoprotein cholesterol, GT = glucose tolerance

<sup>a</sup>Significantly different (P < 0.05)

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**Table 3: Correlation (r value) between the obesity-related markers of healthy non-obese subjects**

| Markers | VAI | BMI | WHR | Leptin | TG | LDL-C/HDL-C | 2-h GT |
|---------|-----|-----|-----|--------|----|-------------|--------|
| VAI     | -0.299<sup>a</sup> | 0.194 | -0.155 | 0.911<sup>a</sup> | 0.413<sup>a</sup> | 0.069 |
| BMI     | 0.194 | -0.071 | 0.088 | -0.230<sup>a</sup> | 0.026 | -0.132 |
| WHR     | -0.155 | 0.088 | -0.495<sup>a</sup> | 0.037 | 0.079 | 0.189 |
| Leptin  | 0.911<sup>a</sup> | -0.230<sup>a</sup> | 0.037 | -0.154 | 0.102 | 0.085 |
| TG      | 0.413<sup>a</sup> | 0.026 | 0.079 | 0.102 | 0.371<sup>a</sup> | 0.194 |
| LDL-C/HDL-C | 0.069 | -0.132 | 0.189 | -0.085 | 0.069 | 0.194 |

VAI = visceral adiposity index, BMI = body mass index, WHR = waist/hip ratio, TG = triglyceride, LDL-C = low-density lipoprotein cholesterol, HDL-C = high-density lipoprotein cholesterol, GT = glucose tolerance

<sup>a</sup>Significantly different (P < 0.05)
with insulin resistance in obese patients. Moreover, our study showed a poor association between VAI and risk of impaired glycemic control (2-h GT) in non-obese subjects [Table 3]; this finding confirms the previously reported data regarding the negative association of VAI with insulin sensitivity in healthy non-obese subjects. The present study reported that obese patients had elevated levels of serum leptin compared with a non-obese group (3.7 folds), and demonstrated an only poor negative association with the LDL-C/HDL-C ratio. However, no significant difference was reported between males and females in this regard [Table 4]. Previous reports indicated that serum leptin level was higher in obese patients (males and females) and seems to be proportional to the degree of obesity despite the anti-obesity action of leptin. Meanwhile, many other studies disagreed with our findings in that obese female presented with higher leptin levels than males. The current finding can be partly explained by the relatively comparable obesity rate of the included subjects.

Many clinical studies demonstrated non-conclusive data regarding the association of serum leptin with the lipid profile in obese patients. However, a positive association was reported between leptin and TG levels in Mexican obese patients but not with other markers of lipid profile. In contrast, Iraqi and Korean obese males have shown the positive correlation of leptin with TC and LDL-c levels. The results of the present study failed to confirm similar findings and demonstrated an only weak negative correlation between leptin and LDL-C/HDL-C ratio in obese patients, and significant negative association with WHR in the non-obese group. This might be attributed to sampling differences and that the subjects in the present study have a relatively normal metabolic profile.

In the present study, we demonstrated the association of various common anthropometric indices (BMI and VAI), lipid profile, and serum leptin with the risk of impaired glucose tolerance (2-h GT). Central obesity is mostly attributed to the accumulation of visceral adipose tissue. Meanwhile, visceral and subcutaneous fat have been evaluated as independent components and correlated with cardiometabolic risk factors. The VAI is a recently developed parameter utilized to achieve a better assessment of adipose mass and the degree of visceral adiposity. The current study confirmed the significant association of VAI and TG with an increase in the risks of impaired 2-h GT in healthy obese patients.

Previous data have indicated that VAI increased in various stages of impaired glucose tolerance; meanwhile, another study reported that a cut-off value of VAI 2.3 gave the sensitivity of 61.2% and specificity of 59.7% to detect impaired glucose tolerance. Additionally, a strong correlation was reported between elevated VAI and increased fasting serum insulin levels and impaired sensitivity.

Furthermore, it has been reported that VAI is better than WC, WHR, and BMI as a surrogate marker for

| Variables      | Obese Males (n = 52) | Obese Females (n = 92) | P value |
|----------------|----------------------|------------------------|---------|
| Age (year)     | 37.31 ± 8.2          | 37.0 ± 8.1             | 0.83    |
| Body weight (kg)| 101.2 ± 13.4         | 91.98 ± 7.6            | <0.0001 |
| BMI (kg/m²)    | 34.1 ± 3.0           | 33.6 ± 1.7             | 0.22    |
| WC (cm)        | 107.4 ± 11.5         | 103.2 ± 8.8            | 0.015   |
| HC (cm)        | 102.1 ± 10.9         | 98.4 ± 9.1             | 0.032   |
| VAI            | 1.05 ± 0.04          | 1.05 ± 0.03            | 0.662   |
| TC (mg/dL)     | 176.6 ± 33.8         | 170.6 ± 37.7           | 0.35    |
| TG (mg/dL)     | 166.5 ± 70.7         | 134.5 ± 47.9           | 0.002   |
| LDL-C (mg/dL)  | 112.5 ± 30.8         | 110.3 ± 33.7           | 0.69    |
| HDL-C (mg/dL)  | 49.1 ± 10.4          | 48.4 ± 7.6             | 0.61    |
| LDL-c/HDL-C    | 2.49 ± 1.11          | 2.36 ± 0.9             | 0.47    |
| TG/HDL-C       | 3.8 ± 1.3            | 3.65 ± 1.1             | 0.36    |
| Leptin (ng/mL) | 53.7 ± 16.3          | 54.3 ± 16.4            | 0.83    |
| FSG (mg/dL)    | 89.2 ± 13.1          | 91.02 ± 17.1           | 0.51    |
| 2-h GT (mg/dL) | 107.9 ± 17.5         | 115.3 ± 19.2           | 0.85    |

BMI = body mass index, WC = waist circumference, HC = hip circumference, WHR = waist/hip ratio, VAI = visceral adiposity index, TC = total cholesterol, TG = triglyceride, LDL-C = low-density lipoprotein-cholesterol, HDL-C = high-density lipoprotein-cholesterol, FSG = fasting serum glucose; GT = glucose tolerance

Data were expressed as mean ± SD
the detection of type 2 diabetes mellitus risks in young adults,[21] though this finding has not been supported by other studies.[32-34] Besides, Yang et al. demonstrated the superiority of VAI over TG levels to predict type 2 diabetes mellitus risk,[28] which is not supported by the findings of the current study.

The current cross-sectional study cannot be regarded as ideal and comprehensive; however, it showed the correlation (for the first time) between many selected anthropometric and biochemical markers in Iraqi obese patients. The major limitations of the current study are the relatively small sample size that does not permit sub-classification of the included subjects according to age and BMI values; in addition to that, the cross-sectional design of the study does not permit exact and clear conclusions on the etiology of VAI, WHR, and TG in terms of future progression to impaired GT in the obese healthy subjects.

**Conclusion**

Our data reported that VAI, TG, and LDL-C/HDL-C ratio, as surrogate markers of obesity and adiposity, can predict the risk of impaired glucose tolerance among Iraqi obese patients, while WHR and serum leptin did not show similar reliability for the risk assessment of impaired GT in those subjects.

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**Conflicts of interest**

There are no conflicts of interest.

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