Influence of BMI on Serum Adiponectin, Resistine, and FBG among Overweight and Obese Females Diabetic Patient Type2

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Abstract

BACKGROUND: Obesity is a frequent comorbidity in patients with type 2 diabetes mellitus (T2DM) and it has been estimated that at least 90% of these patients are overweight or obese [4]. The risks of many complications and comorbidities (e.g., cardiovascular disease and chronic kidney disease) are considerably increased in patients with T2DM who have concomitant obesity.

AIM: This study aims to evaluate the level of Resistine, Fasting Blood Glucose and Adiponectin in overweight, obese versus normal weight females and compare with the BMI.

METHODS: A descriptive cross-sectional – case–control study, the target population, is a total 204 Sudanese females that were recruited to participate in this study (102 overweight, obese versus 102 non-obese, and non-obese), aged 20–50 years and venous blood samples were collected. Plasma levels of blood glucose were measured using the particle-enhanced immunoturbidimetric assay method Cobas C-311®, while adiponectin and resistine estimated by ELISA Kits. Anthropometric measurements, including height and weight, were taken using standard protocols. BMI was calculated as weight (in kilograms) divided by height (in meters squared).

RESULTS: According to these, results observed significant difference between the means of FBG, adiponectin, resistine, and BMI compare with normal weight, overweight, and obese respectively. (FBG in normal weight (127 ± 16.7), FBG in overweight (153 ± 19.2), FBG in obese (189 ± 22.7), P = 0.037, adiponectin in normal weight (11.2 ± 1.9), adiponectin in overweight (9.7 ± 1.6), adiponectin in obese (6.1 ± 1.5), P = 0.043, resistine in normal weight (12.8 ± 1.1), resistine in overweight (14.2 ± 2.7), resistine in obese (18.2 ± 2.9), P = 0.021, BMI in normal weight (18.3 ± 1.7), BMI in overweight (26.1 ± 2.2), BMI in obese (30.4 ± 3.7), P = 0.006. In this study, the results show strong negative correlation between the levels of serum adiponectin and FBG, adiponectin, and resistine (p = 0.013, r = −7.9) (p = 0.019, r = −6.6), while showing in the other side strong positive correlation between the levels of serum resistine and FBG (p = 0.015, r = 6.0).

CONCLUSION: This study concluded that the level of Resistine and Fasting Blood Glucose increased and Adiponectin was reduced in obese compared to non-obese females, Diabetic Patient Type2.

Introduction

Obesity has emerged as a public health crisis in many populations including Sudanese. Adipose tissue produces several adipokines, one of them is adiponectin which has attracted much attention due to its anti-diabetic and anti-atherogenic effects. Adipose tissue is a key endocrine organ that communicates with brain, muscle, liver, and pancreas, thereby maintaining energy homeostasis. The communication between adipose tissue and other organs is mainly mediated by multiple endocrine substances secreted by adipose tissue, referred to as “adipocytokines” [1]. Changes in the levels of adipocytokines are suspected to be indicators of dysfunction in adipose tissue [2], [3]. Adiponectin [4] and resistin [5] hormones are thought to link with obesity and MS with cardiovascular risk. Adipose tissue is no longer considered an inactive organ, in which only stores lipids and serves as an energy reservoir. These chemical messengers, known as “adipocytokines” or “adipokines,” include tumor necrosis factor α, adiponectin, leptin, resistin, and visfatin [6]. Adiponectin is an adipocyte-secreted polypeptide hormone with molecular weight 30 kDa (244 amino acids) which modulates several metabolic processes and regulates insulin sensitivity and energy homeostasis, as well as glucose and lipid metabolism [7]. The hormone plays a principal role in the suppression of the metabolic derangements that may result in insulin resistance, obesity, MS, and cardiovascular disease [5], [7], [8]. Adiponectin is a protective protein with anti-diabetic, anti-inflammatory, and anti-atherogenic effects [6]. Reduced plasma adiponectin levels have been reported in obese individuals, particularly in those with visceral obesity, and have been negatively correlated with insulin resistance. Recent evidence has also suggested the role of adiponectin in the regulation of insulin action, energy homeostasis, obesity, and insulin resistance. Circulating adiponectin levels and adiponectin gene expression in adipose tissue are reduced in patients with type 2 diabetes [9], [10]. Available data suggest that adiponectin might reduce hepatic glucose production and increase muscle glucose utilization, perhaps by
increasing fat oxidation and thereby reducing circulating NEFA levels and intramyocellular accumulation [11]. Studies of adipocytokines in populations with different propensity for obesity, insulin resistance, type 2 diabetes, and atherosclerosis are needed and are interesting in this respect, because they have very high incidence of insulin resistance, central obesity, type 2 diabetes, and cardiovascular disease [12], [13]. In a previous study by Valsmakis et al. [14], South-Asians have lower adiponectin levels compared to Caucasians. It was not clear; however, if this feature of the South-Asians is related to aspects of glucose metabolism. No published data have been found related to adipocytokines, resistin, and obesity in Sudanese population that this study aims to determine the relationship between body fat distribution and adipocytokines.

Materials and Methods

This is a descriptive cross-sectional and analytical case–control study. The study was done in Khartoum state. The target population is a total 204 Sudanese females that were recruited to participate in this study (102 overweight, obese versus 102 non-overweight, and non-obese), aged 20–50 years. The sample size calculations were based on the formula for unmatched case–control studies. Open EPI-INFO statistical package version 7 was used with 99 % two-sided confidence level, 80% power and 40% of controls exposed, 57% of cases exposed, and odds ratio >2. The ratio of controls to cases is 1:1. Venous blood sample (5 ml) was drawn by a well-trained medical technologist into vacutainer tubes from overweight and obese, and non-overweight and non-obese. Blood collected in anticoagulant fluoride oxalate and then plasma samples were obtained by centrifugation at room temperature at 3000 rpm/10 min. Obesity was defined by both body mass index (BMI) ≥30 kg/m$^2$ and overweight subject was those with BMI ≥25. Anthropometric measurements, including height and weight, were taken using standard protocols. BMI was calculated as weight (in kilograms) divided by height (in meters squared).

**Estimation of fasting blood glucose (FBG)**

Serum levels of blood glucose were measured using the particle-enhanced immunoturbidimetric assay method Cobas C-311®. Human glucose agglutinates with latex particles coated with monoclonal anti-glucose antibodies, and then, the precipitate was determined turbidimetrically.

**Estimation adiponectin and resistin**

For adiponectin and resistine used, ELISA Kits-(ab222508)/(ab222403) are a single-wash 90 mins sandwich ELISA designed for the quantitative measurement of adiponectin and resistin in plasma. Simple step ELISA® technology employs capture antibodies conjugated to an affinity tag that is recognized by the monoclonal antibody used to coat our Simple step ELISA® plates.

**Statistical analysis**

Finally, the result analyzed by SPSS version 24. The mean and SD were obtained, and "t"-test and one-way analysis of variance (ANOVA) are used for comparison. Linear regression was also use for correlation. p-value was obtained to assess the significance of the results (p < 0.05 was significant).

**Results**

The study was done on 100 Female Diabetic Patient Type 2 with different body mass index BMI (normal weight, over weight and obese) as shown in Table 1.

According to the statistical test, one-away ANOVA used to determine the mean difference between groups (normal weight, overweight, and obese), observed from this study significant difference between the means of FBG, adiponectin, resistine, and BMI compare with normal weight, overweight, and obese, respectively. Table 2 shows the comparison between means of adiponectine, resistin, FBG, and BMI, among obese and overweight versus normal body weight (FBG in normal weight (127 ± 16.7), FBG in over weight (153 ± 19.2), FBG in obese (189 ± 22.7), P = 0.037, adiponectin in normal weight (11.2 ± 1.9), adiponectin in over weight (9.7 ± 1.6), adiponectin in obese (6.1 ± 1.5), P = 0.043, resistin in normal weight (12.8 ± 1.1) , resistin in over weight (14.2 ± 2.7), resistin in obese (18.2 ± 2.9), P = 0.021,BMI in normal weight (18.3 ± 1.7) , BMI in over

Figure 1: A scatter plot shows strong negative correlation between the levels of serum adiponectin and fasting blood glucose (p = 0.013, r = −7.9)
weight (26.1 ± 2.2), BMI in obese (30.4 ± 3.7), P = 0.006). Person t-test was used for correlation, after statistical analysis, the results show strong negative correlation between the levels of serum adiponectin versus FBG and resistine (Adiponectin vs. FBG: p = 0.013, r = −7.9) and (Adiponectin vs. Resistine: p = 0.019, r = −6.6) (Figures 1 and 2), while the other side of hand shows, moderate positive correlation between the levels of serum resistine and FBG (p = 0.015, r = 6.0) (Figure 3).

Also, in this study, Figure 4 shows a negative relationship between the mean history of the patient compare with the body mass index.

According to the results, this study observed that significant difference in resistin concentration was identified between patients with T2DM who had normal BMI and those who were obese. Mabrouk et al. found that resistin concentrations were higher in obese patients with diabetes than in obese non-diabetic participants; moreover, they were higher in obese patients with diabetes and obese non-diabetic participants than in non-obese healthy controls [1], [5], [7]. This disparity in findings may be due to differences in the study populations. Serum adiponectin concentrations have been shown to be inversely correlated with the severity of insulin resistance in patients with T2DM [3].

Consistent with the findings of previous studies [9], [10], the present study showed that a lower level of adiponectin was present in patients with newly diagnosed T2DM who were obese than in those who had normal BMI. Adiponectin is considered to have anti-diabetic and anti-inflammatory effects; therefore, it is reasonable to presume that patients

### Discussion

Obesity is a frequent comorbidity in patients with type 2 diabetes mellitus (T2DM) and it has been estimated that at least 90% of these patients are overweight or obese [4]. The risks of many complications and comorbidities (e.g., cardiovascular disease and chronic kidney disease) are considerably increased in patients with T2DM who have concomitant obesity. In this study, we have shown that circulating adipocytokine concentrations differed based on the degree of obesity in patients with newly diagnosed T2DM, we demonstrated that higher plasma concentrations of FBG, resistine, and lower plasma concentrations of adiponectin were present in patients diagnosed T2DM who were overweight and obese than in patients diagnosed T2DM who had normal BMI.
with T2DM who obese exhibit that more severe insulin resistance status are than patients with T2DM who have normal BMI. Some of the studies have suggested that adiponectin/resistin ratios are more closely related to the severity of insulin resistance [11], [12], [13], [14]; therefore, we calculated these ratios for the patients with newly diagnosed T2DM. According to this, result observed positive correlation between the levels of serum resistine and FBG. Serum resistin was also positively correlated with FBG, while adiponectin was negatively correlated with these same parameters. Serum resistin was positively correlated with FBG, but adiponectin negatively correlated with resistin and FBG. In conclusion, serum resistin and adiponectin, levels are correlated with the occurrence of T2DM and microvasculopathy complications.

**Conclusion**

Our findings suggest that, in patients diagnosed T2DM, adipocytokine concentrations (resistin and adiponectin) differed between patients who had normal BMI and those who were obese. This study concluded that the level of Resistine and Fasting Blood Glucose increased and Adiponectin was reduced in obese compared to non-obese female Diabetic Patient Type 2.

**Acknowledgment**

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**References**


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**Table 2: Comparison between means of adiponectine, resistine, FBG, and BMI, among obese and overweight versus normal body weight**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Normal weight (mean SD) (n = 57)</th>
<th>Overweight (mean SD) (n = 27)</th>
<th>Obese (mean SD) (n = 16)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>FBG – mg/dl</td>
<td>122 (10.4)</td>
<td>115 (10.2)</td>
<td>109 (22.7)</td>
<td>0.003</td>
</tr>
<tr>
<td>Range</td>
<td>125–137</td>
<td>149–167</td>
<td>178–244</td>
<td></td>
</tr>
<tr>
<td>Adiponectin – U/l</td>
<td>11.2 (1.9)</td>
<td>9.7 (1.6)</td>
<td>6.1 (1.5)</td>
<td>0.043</td>
</tr>
<tr>
<td>Range</td>
<td>9.8–15</td>
<td>6.8–10.5</td>
<td>5.1–7.3</td>
<td></td>
</tr>
<tr>
<td>Resistine – U/l</td>
<td>12.8 (1.1)</td>
<td>14.2 (2.7)</td>
<td>18.2 (2.9)</td>
<td>0.021</td>
</tr>
<tr>
<td>Range</td>
<td>11–17</td>
<td>15.4–27</td>
<td>18.4–29</td>
<td></td>
</tr>
<tr>
<td>BMI – kg/m²</td>
<td>18.3 (1.7)</td>
<td>20.1 (2.2)</td>
<td>30.4 (3.7)</td>
<td>0.006</td>
</tr>
<tr>
<td>Range</td>
<td>19–24</td>
<td>25.2–32.8</td>
<td>30.1–33.7</td>
<td></td>
</tr>
</tbody>
</table>

BMI: Body mass index, FBG: Fasting blood glucose.