ABSTRACT

Background: Visfatin is an adipokine secreted mainly by visceral adipose tissue and has been linked to obesity. Visfatin has insulin mimetic properties. Visfatin also play an important role in the development of several chronic diseases and inflammation.

Aim of the study: To evaluate serum visfatin concentrations in women of different body weights to determine the relationships with obesity and diabetes mellites in women.

Methodology: In this study, 58 women of different body weights. Anthropometric measurements were recorded for all participants. Blood samples were collected to assay the biochemical parameters, including the levels of visfatin, insulin, glucose and lipid profile.

Results: The results were showed that diabetic women exhibited significantly higher visfatin and leptin levels than lean women. Furthermore, diabetic obese women showed significant increase of total cholesterol (T.C), triglyceride (T.G) and low-density lipoprotein-cholesterol (LDL-C) than lean women. However, diabetic women had significantly lower high-density lipoprotein-cholesterol (HDL-C) than lean women. Whereas, no significant differences of adiponectin and insulin found between groups.

Conclusions: The results of this study revealed that visfatin levels were increased in diabetic obese women. This suggests that visfatin levels strongly associated with obesity and diabetes.

Key words: Visfatin, Obesity, Diabetes, Adipose tissue, Adipokines.

I. INTRODUCTION

Obesity is chronic medical condition characterized by excessive fat accumulation in body [1]. It is one of the most important factors that lead to many metabolic complications include type 2 diabetes, insulin resistance and cardiovascular disease [2].

The World Health Organization (WHO) reports that in 2016, about 2 billion people were overweight, with 650 million meeting the obesity criterion [3].

Obesity is measured by using Body Mass Index (BMI) that determined by dividing the weight of the person in kilograms by their square height in meters, therefore, individuals can be classified into three categories, normal (BMI= 18-24.9 kg / m2), overweight (BMI= 25-29.9 kg / m2) and obese (BMI= 30 kg / m2) [4].

The main source of fatty acids (FFA) in the fasting state is adipose tissue which is used for energy use and heat production. Adipose tissue also recognized as large endocrine and paracrine organ in human body which is secretes hundreds of bioactive molecules called adipokines [5]. These molecules are proteins secreted mainly by adipocytes and have role in several function in the body including energy metabolism, glucose homeostasis, inflammation, insulin resistance, immunity, appetite and satiety [6].
Visfatin hormone, is one of the important adipokines secreted from adipose tissue. Visfatin has insulin mimic properties, its play an important role in the homeostasis of energy, glucose metabolism and inflammation by regulation the production of some inflammatory cytokines including tumor necrosis factor-a (TNF-a) and interleukin-6 [7]. Visfatin is also implicated in the pathogenesis of multiple metabolic disorders such as obesity, diabetes mellitus (DM), blood pressure and insulin resistance (IR) [8].

II. THE AIM OF THE STUDY
Evaluate visfatin levels in diabetic obese women and study the relationship between visfatin and diabetes mellitus.

III. METHODS

Study population
In this study, a total of 58 women samples aged from 25 - 55 years. A brief explanation of the project was explained to the participants before sample collecting. Written informed consent has been obtained from all participants before their inclusion. The medical histories of the study population and some required data such age and geographical area was obtained by direct interview with women by using a questionnaire.

Study design
The participants were divided into two main groups according to their BMI. The first group I including 30 lean women with BMI range (18-24.9), the second group including 28 diabetic obese women with BMI more than 30. The exclusion criteria for were the presence of any chronic diseases except diabetes, endocrine diseases, treatment with any medication except medications of diabetes, pregnancy, and irregular menstrual cycle.

Anthropometric measurements
Anthropometric measurements, including body weight, height, and waist and hip circumferences, were measured. BMI values were calculated by dividing the person’s weight in kilograms to height in meters square, and the waist-to-hip ratio (WHR) was calculated by dividing the waist circumference to the hip circumference in centimeters.

Serum preparation
Five ml of venous blood was collected in the morning between 8:00 and 10:00 after an overnight fasting, and placed in sterilized serum separation tube (gel tube). Leave it for a period (about 10 minutes) until the clot formation is occurred. After clot formation, the samples were placed in centrifuge (3500 rpm for 10 minutes at room temperature) to obtain the serum. The serum obtained were withdraw and placed in Eppendorf safe-lock tubes (1ml) which used for dividing the samples before storage in deepfreeze at (-20°) until the time of assay.

Biochemical analysis
Glucose and lipid profile concentrations were measured by enzymatic colorimetric method, by using commercial Kit (COBAS INTEGRA 400 plus, Roche, Germany). Enzyme-linked immunosorbent assay kits (ELISA) were used to determine serum visfatin and insulin levels.

Statistical analysis
The data were statistically analyzed using SSPS software and the significance of the observed differences, associations, or calculations was determined at p-value <0.05. Chi² statistical test was used to investigate the significance of associations, Kruskal-Wallis and Mann-Whitney tests were used for differences between the groups of non-parametric data, and Spearman's test to examine nonparametric correlations.

IV. RESULTS
Table (1) was recorded that BMI, WHR, glucose, T.G and VLDL had higher significant increase (p=0.000) in diabetic group than control. However, the level of T.C (p=0.036) and LDL (p=0.016) had significantly increase in diabetic than control. In addition, HDL concentration had significant decrease in diabetic group than control (p=0.001). The study results as illustrated in table (2), that visfatin (p=0.002) was significantly increase in diabetic than control. Furthermore, the leptin level was high significant increase (p=0.000) in diabetic group than control. On other hand, adiponectin (p=0.243) and insulin (p=0.050) were showed no significant difference between two groups.
Table (1) Comparison between group I and group III regarding BMI, WHR, glucose and lipid profile regardless age group.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Group I Control n=30</th>
<th>Group III Obese diabetic n=28</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>Median</td>
<td>Mean ± SD</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>23.38 ± 1.6658</td>
<td>24.050</td>
<td>35.104 ± 4.3485</td>
</tr>
<tr>
<td>WHR</td>
<td>0.7857 ± 0.0606</td>
<td>0.8000</td>
<td>0.9639 ± 0.08478</td>
</tr>
<tr>
<td>Glucose (mmol/l)</td>
<td>93.963 ± 6.2184</td>
<td>92.800</td>
<td>201.075 ± 118.07</td>
</tr>
<tr>
<td>Total cholesterol (mmol/l)</td>
<td>180.063 ± 31.748</td>
<td>182.000</td>
<td>208.032 ± 49.467</td>
</tr>
<tr>
<td>Triglycerides (mmol/l)</td>
<td>82.157 ± 37.2205</td>
<td>78.800</td>
<td>172.614 ± 69.189</td>
</tr>
<tr>
<td>HDL-C (mmol/l)</td>
<td>48.210 ± 8.8651</td>
<td>48.050</td>
<td>39.615 ± 10.5173</td>
</tr>
<tr>
<td>LDL-C (mmol/l)</td>
<td>92.967 ± 23.552</td>
<td>95.450</td>
<td>111.046 ± 28.583</td>
</tr>
<tr>
<td>VLDL (mmol/l)</td>
<td>16.433 ± 7.4358</td>
<td>15.750</td>
<td>34.521 ± 13.8346</td>
</tr>
</tbody>
</table>

* Significant; P-value <0.05
NS; not significant

Table (2) Comparison between group I and group III according to hormones (visfatin and insulin) regardless age group.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Group I Control n=30</th>
<th>Group III Obese diabetic n=28</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>Median</td>
<td>Mean ± SD</td>
</tr>
<tr>
<td>Visfatin (ng/ml)</td>
<td>6.083 ± 1.7046</td>
<td>5.950</td>
<td>7.754 ± 2.4479</td>
</tr>
<tr>
<td>Insulin (μIU/ml)</td>
<td>14.897 ± 5.1885</td>
<td>13.400</td>
<td>20.550 ± 13.9523</td>
</tr>
</tbody>
</table>

* Significant; P-value <0.05
NS; not significant

V. DISCUSSION

Obesity is characterized by low-grade inflammatory responses and increased oxidative stress [9]. Obesity has developed into a hazardous condition that involves a variety of interventions, treatments, and preventions. Adipokines are small polypeptide growth factors released primarily by white and brown adipose tissue adipocytes such as visfatin, adiponectin, leptin and resistin [10]. Adipokines are hormones that can influence a variety of physiological and pathological processes, particularly those linked to immune and inflammatory activities. Skeletal muscle, kidney, pancreas and immune systems, can all benefit from adipokines. Adipokines' extensive impacts may explain (at least in some part) the systemic issues that are commonly linked with obesity [11].

The results of this study have shown that there were significant increases in visfatin in diabetic obese group in comparison with control group. Several studies agreed with this study results, its clarified that obesity causes increase the release of visfatin from adipocytes. Mabrouk was found significantly higher levels of visfatin in obese diabetics compared to healthy normal weight group [12]. Moreover, another experimental study was conducted that serum visfatin concentration was significantly raised in obese diabetic mice than control [13].

Previously published investigation was appeared that plasma visfatin was increase in patients with diabictic than control group. The elevated in visfatin level in individuals with DM might indicate impaired visfatin signalling in target tissues, biosynthetic dysregulation, or a response to hyperglycemia or hyperinsulinemia in a diabetic condition [14], [15], [16]. Similar results were also recorded in rats, that high levels of fasting serum visfatin was
observed in diabetic rat when comparison with control [17], [18], [19] shown there were significant increase in the mean of visfatin serum in diabetics group than obese and the control group.

Moreover, another research reported that T2D patients showed a significant high levels of serum visfatin than healthy subjects [20], [21]. This may be due to that increase in adipocytes will result in increase in visfatin levels [22]. In addition, high level of visfatin was showed in T2D group than control, and the lowest level of visfatin was recorded in T1D group [18].

The hypothesis that this resulted from a compensatory mechanism developed in response to impaired insulin action, which confirms insulin mimetic effect of visfatin. This theory seems to be confirmed by other studies, which demonstrated that plasma visfatin concentration was dependent on the degree of insulin resistance. However, it should be noted that the relationship between serum visfatin level and insulin resistance remains unclear and studies revealed conflicting results [23], [24], [25].

In addition, although of the statistical analysis of present study had shown no significant differences in insulin level between all groups, the data was recorded elevated in mean of insulin in diabetic obese more than control group (20.550 and 14.897) respectively. This perhaps due to the fact that the type of diabetes was not determined by authors when the participants samples were collected.

This fact has been proven in several studies. Since, the results referred that insulin concentration had significant increase in T2D than T1D and control group [18], [26]. These elevated insulin levels may be due to increased insulin resistance due to insulin over secretion to overcome the tissue resistance [27]. Moreover, in the T1D group the insulin levels were the lowest when compared to the T2D and control group, these findings due to that alloxa treated rats undergone destruction of pancreatic β-cells [28].

Furthermore, the results of this study were appeared a significant increase in the anthropometric measurements (BMI and WHR) in obese diabetic group than control. In addition, fasting glucose, T.C, LDL, T.G and VLDL were highly significant increase in diabetic group in comparison with control group. Additionally, the results revealed a significant decrease of HDL in obese and diabetic group than control.

Many previously studies revealed similar results. BMI and WHR had significant increase in obese diabetics in compared with control group, the levels of T.C, T.G and LDL-C were significant increase in diabetic group than control while HDL-C degrese in diabetic than control group [29]. However, the data results showed that WHR had significant increase in diabetic group than lean group, this result is agreed with several studies [14].

Obesity is global epidemic was associated with dyslipidemia, that is mainly caused by insulin resistance and pro-inflammatory adipocytokines [30]. The typical dyslipidemia of obesity consists of increased triglycerides and FFA, decreased HDL-C with HDL dysfunction and normal or slightly increased LDL-C with increased small dense LDL [31]. Plasma FFA levels are known to be higher in obese individuals as a result of increased fatty acid production from adipose tissue and a decrease in plasma FFA clearance [32]. The increase in FFA and obesity-induced inflammation play a crucial role in the development of insulin resistance [33].

The results of current study also revealed a significant increase in the level of glucose in diabetic subjects than control. This conducted was agreed with several studies, high significant increase of glucose in diabetic subjects than control [12].

Moreover, the results of this study were agreed with many researches, no correlation was found between visfatin and anthropometric measurements (BMI and WHR) in diabetic group [34]. Furthermore, no correlation was conducted between visfatin and insulin, and glucose in diabetic group [35].

REFERENCES

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