ORIGINAL ARTICLE

Serum visfatin levels in obese and non-obese individuals; A comparative cross-sectional study.

Shama Iqbal1, Irfan Younus2, Muhammad Shahid3, Hamid Hassan4


ABSTRACT… Objective: To compare serum visfatin levels in obese and non-obese individuals. Study Design: Cross-sectional study. Setting: District Head Quarter Hospital, Gujrat. Period: July 2020 to May 2021. Material & Methods: A total of 52 subjects were included fulfilling the inclusion criteria. They were divided in two groups, 26 non-obese subjects were placed in Group A and 26 obese subjects in Group B. The serum visfatin level, Low-Density Lipoprotein (LDL), High-Density Lipoprotein (HDL), triglycerides (TG) were calculated in both groups. The statistical analysis was carried out using SPSS version 22.0. Results: The obese subjects had higher serum visfatin levels than the non-obese i.e. 3.87 ± 0.90ng/mL vs. 1.08 ± 0.56 ng/mL (p=0.001). A significant elevation was observed in the serum LDL level among obese subjects, 224.6 ± 55.0 mg/dl compared to 114.2 ± 22.9 mg/dl among non-obese subjects. Similarly, the serum TG levels were also significantly high among obese subjects (p=0.001). The intergroup comparison showed that the serum visfatin levels were higher among obese subjects with dyslipidemia (3.97 ± 0.93 ng/dl) than non-obese (3.46 ± 0.86 ng/dl). Conclusion: It is concluded that the serum visfatin levels are significantly high among obese individuals than non-obese counterparts.

Key words: Lipid Profiles, Obesity, Visfatin.

INTRODUCTION

Obesity, a preventable yet prevailing medical condition associated with unusual accumulation of adipose tissues, has emerged as an epidemic in Pakistan, but still, it remains unrecognized.1,2 Statistically, the increasing ratio of visceral fat and central obesity among Asians contribute to the prevalence of related comorbidities like Diabetes Mellitus (DM) and Coronary Artery Disease (CAD).1,3 The adipose tissues were previously considered only therpository for the fats and energy, but according to the recent literature it has been included among the major endocrine organs, secreting adipocytokines.4 These Adipocytokines primarily regulates the body weight, food intake, insulin resistance, immunity and inflammatory activities.5

Visfatin is a proinflammatory adipocytokine, also known as pre-ß cell colony-enhancing factor 1(pBEF-1).6 This cytokine is a protein comprising nearly 500 amino acids, predominantly produced by the human visceral adipose tissues (VAT). It exerts insulin-mimetic actions through insulin receptors. Hypoxia triggers the release of Visfatin from the macrophages of adipose tissue. The hypoxia-inducible factor-1a (HIF-1a) binds to hypoxia response elements (HREs) after stabilization as it moves into the nucleus. The transcription of Visfatin initiates within the target genes.3 In addition to insulin sensitivity, visfatin also regulates lipid homeostasis. It is involved in triglyceride metabolism and adipocyte proliferation.5,7

Several studies have reported a significant association between serum visfatin level and obesity.1,3,7 A study involving the Taiwanese population indicated that serum visfatin levels are closely linked with abdominal obesity and type 2 DM.8

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The direct parameters of obesity, including waist to hip circumference, body mass index (BMI), Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) scores, dyslipidemia and insulin concentration, are found to be positively correlated with serum visfatin level. A few European studies do not suggest any such significant correlation between the serum visfatin level and visceral fat. In this context, it is essential to determine the association between serum visfatin levels with adiposity by comparing the visfatin levels among obese and non-obese subjects.

**MATERIAL & METHODS**

This comparative cross-sectional observational study was conducted on 52 individuals who visited District Head Quarter Hospital, Gujrat from July 2020 to May 2021. They were recruited through non-probability consecutive sampling after taking informed consent. The patients with medical conditions like hypothyroidism, Cushing syndrome and End-Stage Renal Disease (ESRD) and liver disease were excluded from the study. The ethical approval was obtained from the ethical review board (NSMC/25/6).

The study subjects were divided into Group A (non-obese individuals) and Group B (obese individuals). Obesity was labeled on the basis of the subject’s BMI, which was calculated using the international standard formula. Subjects with a BMI > 25 kg/m² were considered obese.

Fasting Lipid Profile including Serum Triglyceride (TG), High Density Lipoprotein (HDL), cholesterol and Low density lipoprotein (LDL) cholesterol was calculated using the Friedewald formula. The serum visfatin levels were estimated through commercial enzyme immunoassay kits; 10 ml peripheral venous blood samples were collected and stored at -70°C for biochemical estimation.

The collected data was analyzed using SPSS version 23.0. The demographic, clinical and biochemical characteristics of the two groups were compared using independent sample t-test and chi-square test. P-value < 0.05 was considered significant.

**RESULTS**

In the present study, there were 52 cases, 26 in each of the study groups. Of the total, there were 13 males in group A and 12 in group B. The mean age of the subjects was somewhere around 48 years in both groups. The mean BMI was significantly high among the obese individual (Group B) than the normal (Group A) (p=0.001). The mean serum LDL and TG were significantly high in subjects of group B than group A (p=0.001). The serum HDL was significantly low among obese subjects compared to normal healthy ones (non-obese) (p=0.001). The Serum Visfatin levels were also found to be significantly elevated among obese individuals (Group B), i.e. 3.87±0.90 ng/ml as compared to 1.08±0.56ng/ml among subjects of group A.

![Table-I. Demographic, clinical and biochemical characteristics of the studied groups.](image)

<table>
<thead>
<tr>
<th>Variables</th>
<th>Study Groups</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender; n (%)</td>
<td>Group A</td>
<td>Group B</td>
</tr>
<tr>
<td>Male</td>
<td>13(50)</td>
<td>12(46.15)</td>
</tr>
<tr>
<td>Female</td>
<td>13(50)</td>
<td>14(53.85)</td>
</tr>
<tr>
<td>Mean±SD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>48.12±4.83</td>
<td>48.50±4.22</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>21.83±0.67</td>
<td>30.32±4.07</td>
</tr>
<tr>
<td>Serum LDL (mg/dl)</td>
<td>114.2±22.9</td>
<td>224.6±55.0</td>
</tr>
<tr>
<td>Serum HDL (mg/dl)</td>
<td>45.8±6.3</td>
<td>29.3±6.1</td>
</tr>
<tr>
<td>Serum Triglycerides (mg/dl)</td>
<td>117.2±47.9</td>
<td>209.5±96.5</td>
</tr>
<tr>
<td>Serum Visfatin level</td>
<td>1.08±0.56</td>
<td>3.87±0.90</td>
</tr>
</tbody>
</table>

*Group A (normal healthy individuals); Group B (obese individuals).* *p*-value < 0.05—significant

The variations in the serum visfatin levels among the subjects of both groups were also compared concerning age, gender and presence of dyslipidemia. The mean serum visfatin levels were significantly elevated among both male and female obese subjects (p<0.05). A similar trend was observed among subjects of both age groups, with slight high serum visfatin levels detected among obese and non-obese subjects ≥ 40 years of age than those < 40 years. A
significant elevation in the visfatin level was observed among obese subjects with or without dyslipidemia (p<0.05) than the normal ones.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Study Groups</th>
<th>Mean±SD</th>
<th>P-Value</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Group A</td>
<td>Group B</td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>Male</td>
<td>1.21±0.63</td>
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<tr>
<td></td>
<td>Female</td>
<td>1.05±0.53</td>
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<tr>
<td>Age Groups</td>
<td>&lt;40 years</td>
<td>1.02±0.53</td>
<td>3.83±0.89</td>
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<tr>
<td></td>
<td>≥40 years</td>
<td>1.13±0.58</td>
<td>3.96±0.92</td>
</tr>
<tr>
<td>Dyslipidaemia</td>
<td>Yes</td>
<td>1.02±0.52</td>
<td>3.97±0.93</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>1.23±0.59</td>
<td>3.46±0.86</td>
</tr>
</tbody>
</table>

Table-II. Show the variation in the serum visfatin level with respect to age, gender and presence of dyslipidemia in the studied groups.
*Group A (normal healthy individuals); Group B (obese individuals).
*p-value < 0.05-significant

DISCUSSION
Our study intended to investigate the comparative serum visfatin levels among obese and non-obese subjects. The increasing prevalence of obesity has become a public health concern as it imposes various complications.12-14 As VAT is the primary source of visfatin, research focusing on the alterations in the serum visfatin levels helps in understanding the obesity complications. In the present study, a significantly high serum visfatin level was observed among obese subjects as compared to non-obese, i.e.3.87±0.90ng/ml vs. 1.08±0.56ng/ml (p=0.001). Several other studies1,7,15 support these findings. A study from Turkey revealed that the serum visfatin levels were significantly elevated in the obese group than the non-obese group (p=0.014).11 This can be explained by the factor that obese subjects have increased fat deposition, which in turn triggers the visfatin synthesis by adipocytes. Contrary to our results, a few also report no correlation between adiposity parameters and elevated visfatin levels.16,17

A significant difference in the serum visfatin level among the obese individuals was also reported by Mahaweerawar et al18, Araki et al19 and Tascilar et al.20 They further revealed a positive correlation between the serum visfatin level and the individual’s BMI, higher levels of visfatin were observed among cases with increased visceral fat than those with subcutaneous fat. Although the correlation of BMI with serum visfatin wasn’t studied. However, a significant increase in the mean BMI was observed among obese subjects (30.32 ± 4.07 kg/m²) than the normal ones (21.83 ± 0.67 kg/m²) (p=0.001).

However, the studies correlating circulating visfatin levels to adiposity have produced variable results. Elevated plasma visfatin levels were found in positive correlation with body weight (BW), BMI, waist circumference and WHR among obese children.16 Berndt et al. also supported, suggesting a positive correlation of visfatin mRNA and concentration in VAT with BMI and body fat among obese subjects.21

Moreover, the mean serum LDL and TG were significantly raised among obese subjects as compared to the counterparts (p<0.05). A similar study also reported increased LDL and TG levels among obese individuals, but the differences were insignificant.22 Moreover, the serum visfatin level was also significantly high among obese subjects with dyslipidemia 3.97 ± 0.93 ng/ml vs. 3.46 ± 0.86ng/ml among obese subjects without dyslipidemia, indicating a significant effect of lipid metabolism on visfatin levels. Chen et al. observed that the TG, HDL-C, Total Cholesterol (TC) and LDL-C were significantly affecting the serum visfatin level.8 In contrast, Pagano et al. found a negative correlation between plasma visfatin level and BMI. They surprisingly observed reduced plasma visfatin levels among obese adults.23

CONCLUSION
In conclusion, we report that serum visfatin levels are significantly high among obese subjects. Certainly, our results are based on cross-sectional data and relatively a small sample size. Further prospective studies with a large sample size are required to clarify visfatin’s role and the mechanism behind its overexpression among obese subjects.

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**AUTHORSHIP AND CONTRIBUTION DECLARATION**

<table>
<thead>
<tr>
<th>No.</th>
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<th>Contribution to the paper</th>
<th>Author(s) Signature</th>
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<tr>
<td>1</td>
<td>Shama Iqbal</td>
<td>Concept and study design, Revision and final approval of the study.</td>
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<tr>
<td>2</td>
<td>Irfan Younus</td>
<td>Data collection and literature review, Critical review.</td>
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<tr>
<td>3</td>
<td>Muhammad Shahid</td>
<td>Data analysis and interpretation and drafting of the manuscript.</td>
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<td>4</td>
<td>Hamid Hassan</td>
<td>Literature review, Concept and study design.</td>
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