Study of Serum Visfatin Level in Impaired Glucose Tolerance Subjects

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ABSTRACT

The aim of study was to measure serum level of visfatin in impaired glucose tolerance subjects and compare with healthy control subjects. In this study 120 IGT and 120 healthy subjects (age and gender matched) were enrolled. BMI, Glucose, HbA1c, Insulin and Visfatin were assessed. Serum visfatin levels were higher in impaired glucose tolerance subjects compared with healthy controls ($4.95±1.81$ ng/ml vs $2.91±1.12$ ng/ml, respectively, P<0.0001). Increased levels of visfatin can assess diabetes at an early stage.

Keywords : Visfatin, IGT, Diabetes.

INTRODUCTION

Diabetes Mellitus is one of the main threat to human health in the twenty- first century and therefore should be controlled and managed early to avoid its complication. Type 2 diabetes is preceded by a pre-diabetes (PD) is a dysmetabolic state of glucose level between diabetes mellitus and normal glucose tolerance. This asymptomatic phase may last for years, and about one third of individuals with pre-diabetes finally develop type 2 diabetes.

According to American Diabetes Association (2018), Impaired glucose tolerance (IGT) is defined as two-hour glucose levels of 140 to 199 mg per dL (7.8 to 11.0 mmol/L) on the 75g oral glucose tolerance test. These glucose levels are above normal but below the level that is diagnostic prevention for diabetes. IGT is commonly associated with both obesity and disturbances in insulin secretion and/or insulin resistance. It is characterized by reduced peripheral insulin sensitivity, near normal hepatic insulin sensitivity, and progressive loss of beta cell function.

Visfatin, an adipocytokine has important role in regulation of glucose levels in humans. It has been shown that visfatin has insulin-mimetic effects on glucose metabolism by activating the insulin signal transduction pathway through the direct binding to the insulin receptor at a site different from that of insulin. Recent studies also demonstrated that serum visfatin levels were significantly higher in diabetic compared with non-diabetic group.

Although there are many evidences linking obesity, serum visfatin and type 2 diabetes. Data about serum Visfatin concentration in Impaired Glucose Tolerance is limited. Therefore, present study was undertaken to evaluate serum Visfatin levels in patients with IGT and to compare it with healthy controls.

MATERIALS & METHODS

120 impaired glucose tolerance subjects were recruited in this study from the OPD of Department of Medicine, Jawahar Lal Nehru Medical College and Associated group of Hospitals, Ajmer. The subjects were considered as IGT based on the ADA guidelines (2018). Age and gender matched 120 healthy subjects without a family history of diabetes were also recruited in this study to serve as the controls. On a prescheduled morning, the subjects were requested to arrive after overnight fast (at least 10 hour) to provide a fasting blood sample. After collecting fasting blood samples, the subjects were given 75g of glucose dissolved in 250ml of water. The blood was drawn via venepuncture 2h after glucose load. After 30 mins of collection, the blood sample was centrifuged for 10-15 mints at 3000rpm to obtain the serum.

BMI was determined following standard procedures. Glucose was measured using the glucose oxidase method, (HbA1c) glycosylated hemoglobin was measured by ion exchange resin method (Trivelli et al;1971), Insulin and serum Visfatin were measured using an enzyme linked immunosorbent assay (ELISA) technique.

The quantitative variables were expressed as the Mean ± SD (Standard deviation) median (range). The baseline characteristic between IGT and healthy subjects were assessed using student’s t-test for continuous variables (as applicable). All P-values were based on a two sided test of statistical significance. Significance was accepted at the level of p<0.05.

RESULTS AND OBSERVATION

In this study, 120 cases of impaired glucose tolerance were compared with 120 healthy controls.

Table 1- Anthropometric parameters of IGT subjects & Healthy controls

<table>
<thead>
<tr>
<th>Parameters</th>
<th>IGT Cases (Mean ± SD)</th>
<th>Healthy Controls (Mean ± SD)</th>
<th>P-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>AGE (yrs)</td>
<td>40.18 ± 2.8</td>
<td>40.35 ± 2.9</td>
<td>0.644 (NS)</td>
</tr>
<tr>
<td>WEIGHT (kg)</td>
<td>79.99 ± 3.05</td>
<td>51.58 ± 4.9</td>
<td>-</td>
</tr>
<tr>
<td>HEIGHT (cm)</td>
<td>1.67 ± 0.05</td>
<td>1.55 ± 4.39</td>
<td>-</td>
</tr>
</tbody>
</table>
Basic anthropometric parameters of IGT subjects and healthy subjects are summarized in table-1. There was no significant difference between IGT subjects and healthy subjects regarding mean age (40.18±2.8 vs. 40.35±2.9 yrs.). BMI mean ± SD in kg/m² in IGT and healthy subjects was (23.4 ± 1.5 vs. 21.34 ± 1.5) and it was highly significant. Biochemical parameters of IGT subjects and healthy subjects are presented in table-2. IGT subjects had higher fasting plasma glucose levels compared to healthy subjects (4.95 ± 1.81 vs. 2.91 ± 1.12, P<0.0001). Serum insulin, HbA1c were significantly higher in IGT subjects compared with healthy controls.

<table>
<thead>
<tr>
<th>Parameters</th>
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<th>Healthy Controls (Mean ± SD)</th>
<th>P-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fasting Plasma Glucose (mg/dl)</td>
<td>117.2 ± 4.5</td>
<td>87.4 ± 11.4</td>
<td>&lt;0.0001 (HS)</td>
</tr>
<tr>
<td>HbA1c (%)</td>
<td>6.4 ± 0.4</td>
<td>5.1 ± 0.3</td>
<td>&lt;0.0001 (HS)</td>
</tr>
<tr>
<td>Serum Insulin (µIU/ml)</td>
<td>11.9 ± 3.1</td>
<td>9.8 ± 2.2</td>
<td>&lt;0.0001 (HS)</td>
</tr>
<tr>
<td>Serum Visfatin (ng/ml)</td>
<td>4.95 ± 1.81</td>
<td>2.91 ± 1.12</td>
<td>&lt;0.0001 (HS)</td>
</tr>
</tbody>
</table>

P value <0.0001 is considered highly significant while p<0.01 is considered significant.

DISCUSSION

In the present study, IGT subjects have significantly higher levels of visfatin as compared to healthy control subjects. A number of articles have reported increased levels of serum visfatin in type 2 diabetes and obese patients, but impaired glucose tolerance subjects have not been studied extensively to know whether the increase in the circulating visfatin levels begin before the onset of diabetes. Increased levels of serum visfatin suggests that hyperglycemia and development of type 2 diabetes is delayed through hypersecretion of adipose tissue derived visfatin as it possesses insulin mimetic effects (A. Kaminska et al.; 2015) therefore, increased serum visfatin may be a compensatory mechanism or part of pathophysiology of diabetes mellitus. Wajchenberg et al; 2000 has seen increased visfatin in newly diagnosed type 2 diabetics as a result of β cell deterioration. This study was also supported by Kowalska et al; 2013 who found insulin’s inability to suppress visfatin production in insulin resistant conditions. However, Dogru et al; 2007 indicated that hyperglycemia causes an increase in plasma visfatin levels in people with type 2 DM but not with impaired glucose tolerance. This discrepancy can be explained by different subjects characteristics. F. Kabir et al; 2015 reported that fasting serum visfatin level was significantly higher in IFG, IGT & IIFG-IGT subjects compared with healthy controls.

Limitations of Study

Our sample size was relatively small.