Association between serum insulin and uric acid concentrations in type 2 diabetic subjects in Nigeria

Y P Mamza, B R Aladeusi, R M Gali, D S Mshelia, R Y Genesis, and S A Habu

Abstract
Diabetic patients who are hyperuricaemic appear to be at increased risk for developing diabetic complications, renal disease, and cardiovascular disease. The present study was undertaken to determine the association between serum insulin and uric acid concentrations in individuals with type 2 diabetes and control subjects attending the University of Maiduguri Teaching Hospital (UMTH) in Nigeria. One hundred and sixty (160) subjects with an age range of 30–75 years participated in the study: 100 confirmed type 2 diabetes subjects and 60 non-diabetic controls. A significantly (p<0.05) high mean serum insulin was observed in type 2 diabetes subjects as compared with controls (9.3±2.0 vs 5.1±0.6 µlU/L). No significant difference (p>0.05) was observed in the mean serum uric acid of diabetic and control subjects (358±89 vs 334±66 µmol/L). There was a positive and significant correlation (r = 0.410; p<0.05) between serum insulin and uric acid levels in type 2 diabetes subjects. This may relate to the insulin resistance that characterises type 2 diabetes.

Introduction
Diabetes mellitus, one of the most common diseases worldwide is considered a major health problem with increasing prevalence, and is the leading cause of morbidity, mortality, and complications.1,2 Diabetes is a metabolic disorder characterised by hyperglycaemia and insufficiency of secretion or action of endogenous insulin.

Type 2 diabetes is a heterogeneous disease associated with variable degrees of insulin resistance, impaired insulin secretion, and increased glucose production. Insulin resistance occurs when cells become less sensitive to the effects of insulin.3 The ‘insulin resistance syndrome’ consists of a group of metabolic abnormalities that increase the risk of cardiovascular disease (CVD) and diabetes.

Elevated serum uric acid levels or hyperuricaemia is a risk factor for insulin resistance, peripheral arterial disease, and other components of the metabolic syndrome.4 For some time, it has been recognised that serum uric acid is positively associated with serum glucose levels in healthy subjects.5 Recent studies have demonstrated that uric acid levels are higher in subjects with prediabetes and early type 2 diabetes, than in healthy controls.6 Furthermore, hyperuricaemia was found to increase the risk of developing type 2 diabetes in individuals with impaired glucose tolerance (IGT).7 An elevated uric acid level often precedes the development of obesity,8,9 and diabetes.10,11

However, hyperuricaemia is not always found in diabetic patients, and conflicting data exist regarding uric acid in type 2 diabetes, as low levels may be found in diabetic patients, while elevated levels are a feature of IGT.12 Although several studies have implicated the role of uric acid in the progress of pre-diabetes to diabetes, this remains controversial and therefore deserves further analysis. The main purpose of this study was to examine the association of serum insulin and uric acid levels in type 2 diabetic subjects in Maiduguri, Nigeria.

Table 1: Comparison of biochemical parameters in subjects with type 2 diabetes and controls (means±SD)

<table>
<thead>
<tr>
<th>Biochemical parameters</th>
<th>Type 2 diabetes subjects (n=100)</th>
<th>Controls (n=60)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>FPG (mmol/L)</td>
<td>8.3±0.5</td>
<td>4.4±0.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Insulin (µlU/L)</td>
<td>9.3±2.0</td>
<td>5.1±0.6</td>
<td>0.025</td>
</tr>
<tr>
<td>Uric acid(µmol/L)</td>
<td>358±89</td>
<td>334±66</td>
<td>0.069</td>
</tr>
</tbody>
</table>

Table 2: Correlation between serum insulin, uric acid, and FPG in diabetic subjects

<table>
<thead>
<tr>
<th>Biochemical parameters</th>
<th>r value</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>FPG and insulin</td>
<td>-0.038</td>
<td>0.707</td>
</tr>
<tr>
<td>FPG and uric acid</td>
<td>-0.206</td>
<td>0.040</td>
</tr>
<tr>
<td>Uric acid and insulin</td>
<td>0.410</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Patients and methods
A total of 160 subjects were recruited for the study. They were 100 known diabetic subjects attend-
ing the University of Maiduguri Teaching Hospital (UMTH) Endocrinology Clinic and General Outpatient Department, as well as 60 non-diabetic individuals as controls. All were within the age range of 30–75 years. Creatinine estimation was carried out to rule out kidney disease in all subjects. Those with type 1 diabetes, liver diseases, or kidney diseases were excluded from the study. Informed consent was sought from the selected individuals using standard guidelines. Ethical approval was obtained from the Ethical Committee of the UMTH. Plasma glucose was measured using the glucose oxidase peroxidase enzymatic method as described by Trinder. Insulin was measured by enzyme-linked immunosorbent assay (ELISA), as described by Gerbitz et al. Serum uric acid concentration was measured by the uricase-peroxidase method as described by Trivedi et al.

The data generated were analysed using the Statistical Package for Social Sciences (SPSS) version 16.0. Student’s t-test was used to compare the means of fasting plasma glucose, serum insulin, and serum uric acid of diabetic and control subjects. The correlations were done by Pearson’s correlation coefficient. Scatter plots were determined by using Microsoft Excel. A p value of less than or equal to 0.05 was considered statistically significant.

Results
The mean age of the diabetic patients (n=100) was 50±1 years compared with 49±1 years for controls (p=0.537). Mean body mass index was 27.8±5.7 (diabetic group) versus 28.9±5.1 (controls), also non-significant (p=0.324). Similarly, systolic blood pressure (BP) and diastolic BP were similar between the two groups (138±2 vs 128±3 mmHg, p=0.898; and 85±1 vs 87±2 mmHg, p=0.158).

The mean fasting plasma glucose (FPG) and serum insulin were significantly (p<0.05) higher in diabetic subjects than in the control subjects. However, there was no significant difference (p>0.05) in serum uric acid levels of diabetic and control subjects as shown in Table 1.

As shown in Table 2, there was no significant correlation between FPG and serum insulin (r = 0.038; p>0.05). The correlation between FPG and serum uric acid was negative and significant (r = 0.206, p<0.05) which is further illustrated by the scatter
plot in Figure 1. A positive and significant correlation was observed between serum uric acid and insulin (r = 0.410; p<0.001), and the scatter plot in Figure 2 also shows this.

**Discussion**

This study was undertaken to determine the association between serum insulin and uric acid concentrations in type 2 diabetes subjects attending the UMTH. As may be expected, FPG and insulin levels were higher in our diabetic group compared with controls, but there was no significant difference in serum uric acid levels (Table 1).

Hyperglycaemia and insulin resistance occurs when the beta cells of the pancreas become less sensitive to the effects of insulin. This results in hyperglycaemia and a drop in energy production. To compensate for the insulin resistance and to attempt to maintain normal blood glucose via elevated plasma concentrations of insulin.

The mechanisms by which uric acid is involved in glucose metabolism by increase in uric acid may be related to insulin resistance. FPG and serum uric acid. The significant decrease in FPG and serum insulin levels was not expected, FPG and insulin levels were higher in our diabetic group compared with controls, but there was no significant difference in serum uric acid levels (Table 1).

Hyperglycaemia and insulin resistance occurs when the beta cells of the pancreas become less sensitive to the effects of insulin. This results in hyperglycaemia and a drop in energy production. To compensate for the insulin resistance and to attempt to maintain normal blood glucose via elevated plasma concentrations of insulin.

A negative and significant correlation was shown between FPG and serum uric acid. The significant decrease in FPG with increase in uric acid may be related to insulin resistance. The mechanisms by which uric acid is involved in glucose concentrations or beta cell function and even the development of type 2 diabetes are uncertain. It is accepted that the most important mechanism may be that of the association between insulin resistance and renal absorption of urates.

In this study we also observed a positive and significant correlation between serum uric acid and insulin in our type 2 diabetes subjects. Whenever there is an increase in insulin level, there appears to be an increase in uric acid level. This may be due to the elevation of serum uric acid which is associated with IGT. Elevated uric acid is also a feature of hyperinsulinaemia and insulin resistance. The result in this study is similar to that reported by Choi et al. and Anju et al.

Based on our research work, it could be deduced that there is a positive correlation between the serum uric acid and insulin levels in type 2 diabetic subjects attending the University of Maiduguri Teaching Hospital. Further work is needed to elucidate the exact cause of this relationship, and its clinical implications.

**Acknowledgements**

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**Author declaration**

Competing interests: none.

Any ethical issues involving humans or animals: none.

Was informed consent required: yes - documentation on file.

**References**


