Role of Carbohydrate Food in Pathophysiology of Nonobese Type 2 Diabetic Subjects

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ABSTRACT

Dietary nutrients have shown overriding influence on insulin sensitivity and secretory status of diabetic subjects due to rapid lifestyle changes and urbanization. A case control study was conducted with 98 type 2 diabetic subjects and 97 healthy controls with BMI below obesity range to explore the association of dietary carbohydrate and body insulin status of nonobese diabetic subjects. Nutrient profile was taken by dietary recall method. Homeostasis Model Assessment (HOMA) was applied for measuring insulin status. Laboratory analysis and anthropometric measurements were done by standard methods. Statistical analysis was performed. Significant insulin secretory defect was found in diabetic group [Median (range), %; Control, 153 (65-285); DM, 50 (4-269)]. In diabetic group total carbohydrate [Median (range), gm/d; control, 235 (102-422); DM, 292 (151-454)], and fiber [Median (range), gm/d; control, 4.5 (2-18); DM, 6.2 (1.5-21)] consumption were found to be significantly higher than control. But no significant association was established between insulin secretions (HOMA%B) with carbohydrate consumption among the case group. So carbohydrate consumption does not influence β-cell secretory dysfunction in nonobese T2 DM patients. The possible potential effect of carbohydrate in insulin depression is not established.

Key Words: Dietary Carbohydrate, Insulin Secretion, Nonobese Type 2 Diabetics

Introduction

Diabetes mellitus is a fast expanding global health problem. The WHO Report on diabetes prevalence alarmed that diabetes is posing a serious threat to developing countries with respect to their existing health care services. Insulin resistance and impaired insulin secretion are usually present in patients with classic type 2 diabetes.

Basal and total 24 hour rates of insulin secretion are 3 to 4 times higher in obese subjects than in lean controls. Again good glycemic control and duration of diabetes also influence insulin secretion in both obese and nonobese groups. Prando and his co-workers found that in well controlled groups obese persons displayed higher insulin secretion than nonobese patients especially in the early years of disease; while in poorly controlled groups obese, when compared with nonobese, displayed a similar low insulin secretion and possibly an increased hepatic insulin resistance.

It has recently been demonstrated that a marked reduction in insulin sensitivity and a preserved insulin release characterized obese diabetic patients, while nonobese patients have a reduction in insulin release and normal insulin sensitivity.

Despite possible influences of genetic and perinatal factors diet is likely to have greater and overriding influence in generation of type 2 diabetes due to rapid lifestyle changes and urbanization.

There is conflicting evidence on the influence of total carbohydrate intake on insulin sensitivity and in fact a recent dietary intervention found that after six months on a low carbohydrate diet, insulin sensitivity improved among obese individuals. However, the source and quality of dietary carbohydrates may differentially optimize insulin action and thereby affect the degree of
insulin resistance, which is a key underlying feature of metabolic syndrome. It has been found that in subjects consuming high sucrose diet, fasting insulin concentration was higher than those consuming starch rich diet. Studies in rats generally demonstrated that high intake of sucrose (18-70% of energy) or fructose (15-60% of energy) produce a decline in insulin sensitivity in the liver and later in peripheral tissues. In case of complex carbohydrates, studies done in laboratory rats showed that high amylase diets appear to have beneficial effects on insulin sensitivity compared with high amylepectin diets as it is more rapidly digested than amylase.

Ingestion of foods high in dietary fibre content appears to be associated with modest beneficial effects on insulin sensitivity. Fasting insulin concentrations are found lower among individuals reporting higher dietary fibre. Some studies report a beneficial effect on insulin sensitivity with a high consumption of dietary fibre, whereas other showed no effect.

The glycemic index, a measure of the glycemic response to carbohydrate containing foods, has been used to physiologically classify dietary carbohydrates. High glycemic index carbohydrates are associated with components of metabolic syndrome. Some clinical studies have demonstrated that low glycemic index carbohydrates improve glycemic control.

This study is an attempt to set up a causal relationship between dietary carbohydrates and the pathophysiology of diabetes in nonobese patients.

**Subjects and Methods**

This is a case-control study conducted in BIRDEM from July 2007 to January 2008 with 195 subjects out of which 98 were diagnosed patients of type 2 DM (case) taken from outpatient department and the remaining 97 were normal healthy subjects (control) with a BMI less than 30 and within the age limit of 30 to 50 years. The subjects were explained about the nature and purpose of the study. Their written consent, medical history was taken adequately. Clinical examination was done methodically.

Height (m), weight (kg) of the subjects was measured by standard scales and BMI was measured by dividing the weight with square of height. Fasting plasma glucose and insulin level were measured by enzymatic method (glucose oxidase method) and ELISA respectively. β-cell secretion (HOMA B) and insulin sensitivity (HOMA S) were derived from these two parameters by Homeostasis Model Assessment (HOMA) using a particular software (HOMA-CIGMA software). Information regarding dietary carbohydrate was calculated using recall method where a questionnaire, prepared by nutritionist was introduced which dealt with dietary habits, frequency of eating and the type of cooking medium. Intakes of fibers are also calculated. The consumption was recorded on a weekly, monthly and more than a month basis. Standard sets of common utensils, utilized in our households, were used to assess the portion of food articles. Data analysis of dietary parameters was carried out by a special software. Statistical analysis was performed using SPSS-12 software.

**Results**

Both cases and control have BMI below obesity range (Table-I). The insulinaemic status of the cases show significant secretory defect as HOMA% B is significantly low in cases (Table-II, p=<0.001). Intake of both carbohydrate food and dietary fiber are significantly high in patients (Table-III). The consumptions have no effect on their insulinaemic status as HOMA% B shows no correlation with carbohydrate and fiber intake (Table-IV).

**Table I:** BMI of the study subjects

<table>
<thead>
<tr>
<th>Variables</th>
<th>Control (n=97)</th>
<th>T2 DM (n=98)</th>
<th>t/p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI (kg/m²)</td>
<td>22.96±2.78</td>
<td>23.42±2.5</td>
<td>1.063/0.289</td>
</tr>
</tbody>
</table>

Results are expressed as Mean ± SD. Independent ‘t’ test is performed as the test of significance at 5% level of significance n=number of subjects. BMI=Body mass index.
Role of Carbohydrate Food in Pathophysiology of Nonobese

<table>
<thead>
<tr>
<th>Variables</th>
<th>Control (n=97)</th>
<th>T2 DM (n=98)</th>
<th>z/p value Control vs. T2 DM</th>
<th>F. insulin (pmol/L)</th>
<th>HOMA % B</th>
<th>HOMA % S</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>75 (21-212)</td>
<td>76 (20-634)</td>
<td>0.456/0.648</td>
<td></td>
<td>153 (65-285)</td>
<td>69 (1-247)</td>
</tr>
<tr>
<td></td>
<td>(range)</td>
<td>(range)</td>
<td></td>
<td></td>
<td>50 (4-269)</td>
<td>67 (9-256)</td>
</tr>
</tbody>
</table>

Results are expressed as Median (range). Mann-Whitney ‘u’ test was performed as the test of significance at 5% significance level n= number of subjects. F. insulin= Fasting insulin. HOMA % B= β cell function assessed by homeostasis model assessment. HOMA % S = insulin sensitivity by homeostasis model assessment.

Table III: Carbohydrate intake of study subjects

<table>
<thead>
<tr>
<th>Variables</th>
<th>Control (n=97)</th>
<th>T2 DM (n=98)</th>
<th>z/p value Control vs. T2DM</th>
<th>CHO (g/d)</th>
<th>Fiber (g/d)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>235 (102-454)</td>
<td>292 (151-454)</td>
<td>3.008/.003</td>
<td>4.5 (2-18)</td>
<td>6.2 (1.5-21)</td>
</tr>
<tr>
<td></td>
<td>(range)</td>
<td>(range)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Results are expressed as Median (range). Mann-Whitney ‘u’ test was performed as test of significance at 5% significance level. n= number of subjects. CHO= Carbohydrate.

Table IV: Spearman’s rho correlation coefficient (r) analysis of HOMA % B with carbohydrate intake of study subjects

<table>
<thead>
<tr>
<th>Variables</th>
<th>Control (n=97)</th>
<th>T2 DM (n=98)</th>
<th>r</th>
<th>p</th>
<th>r</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHO (g/d)</td>
<td>0.097</td>
<td>-0.150</td>
<td>0.972</td>
<td>0.472</td>
<td>0.140</td>
<td></td>
</tr>
<tr>
<td>Fiber (g/d)</td>
<td>0.215</td>
<td>0.095</td>
<td>0.109</td>
<td>0.354</td>
<td>0.095</td>
<td></td>
</tr>
</tbody>
</table>

Spearman’s rho correlation coefficient (r) was performed for analysis. n= number of subjects. r= correlation coefficient. CHO= Carbohydrate.

Discussion

In this study, both carbohydrate and fiber intake was found to be significantly higher in diabetic subjects compared with the control group but no association was found between carbohydrate and dietary fiber intake and insulin secretion.

It is now evident that non obese subjects have pathophysiology of depressed insulin secretion rather insulin resistance. Their total carbohydrate and fiber consumptions are higher but not potentiate the disease pathology.

This finding claims detailed information of carbohydrate consumption on the basis of their biochemical classification or glycemic index. This will provide an organized dietary guideline for diabetic subjects regarding carbohydrate food.

The usual pathophysiological events in type 2 DM is the presence of insulin resistance followed by compensatory hyperinsulinemia but eventually β cell failure due to exhaustion. In contrast to this scenario the nonobese diabetic population in the present study did not show any hyperinsulinemia or insulin resistance (table 2). Rather they showed a highly significant reduction in insulin secretory capacity. This conforms to the previous studies in Bangladeshi population where a predominant secretory defect has been found in T2 DM.

The influence of carbohydrate intake on insulin status is controversial as its type and source determine its metabolic effect. The glycemic index (GI), is considered to predict the potential of carbohydrate foods to influence insulin action. Some studies showed that diets having a low GI may improve insulin secretion. In contrast, an inverse picture was found between low GI carbohydrates and the onset of frank diabetes. Another observational study also showed that total carbohydrate intake is unrelated to fasting insulin.

A study done by raising carbohydrates resulted in improved glucose tolerance as a result of an increase in insulin secretion in older subjects and mild diabetic subjects.

Dietary fibers are claimed to have a unique role in determining insulin secretion or sensitivity. A study on middle aged and older men, and postmenopausal women showed no effect of dietary fiber on insulin secretion. Some observational studies reported a beneficial effect on insulin sensitivity with a high consumption of dietary fiber.

Conclusion

The study is concluded with the finding that high intake of carbohydrate food does not aect decreased insulin secretion in non obese T2 diabetic patients.
References


