Prevalence of Metabolic syndrome among the offspring of individuals with type 2 diabetes mellitus

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Abstract
There is high prevalence of Mets among patients with type 2 diabetes mellitus. However, it is not clear whether there is increased prevalence of Mets among their offspring. Therefore a study was undertaken to determine the prevalence of Metabolic Syndrome (Mets) among the offspring of individuals with type 2 diabetes. Otherwise healthy, forty eight offsprings of diabetic patients in the age group of 15 - 45 years were selected randomly. Data regarding patient characteristics were recorded using a pre-designed questionnaire. Measurement of body weight and blood pressure and investigations including blood glucose and lipid profile were done. Prevalence of Mets was determined by the ATP111 criteria. Five out of forty eight (11%) offsprings of diabetic individuals fulfilled criteria for Mets. The mean age of individual with Mets was 39.2 years (±10.4) and those without Mets were 28.7 years (±9.4). The prevalence of Mets in females was 21.7% whereas no male had Mets in this cohort. The commonest factor that contributes for Mets was HDL less than 40mg/dl (29.2%) followed by TG more than 150mg/dl (22.9%). At baseline 34% (10/29) of patients with type 2 diabetes had Mets which was much higher than that of their offspring. The prevalence of Mets in otherwise healthy offspring of diabetic patients was similar to those reported in general population with a female preponderance. However, the prevalence of Mets in offspring was much lower than the prevalence of Mets in their diabetic parents. Environmental factors more than genetic background may be involved in the pathogenesis of metabolic syndrome.

Key words- Metabolic syndrome, offspring of diabetic patients, prevalence

Introduction
In South East Asia, diabetes mellitus is spreading as an epidemic. The reasons are complex, and include adoption of a western lifestyle and soaring prevalence of obesity in this region. Since diabetes mellitus occurs in substantial number of obese patients and since obesity is also commonly seen in patients with type 2 diabetes, it is clear that these two conditions go hand in hand. Obesity of any configuration, but particularly abdominal obesity, is associated with resistance to the effects of insulin on peripheral glucose utilization, often leading to type 2 diabetes mellitus. Furthermore, the associated hyperinsulinemia may then lead to hypertension and an abnormal lipid profile, both of which promote the development of atherosclerosis. This constellation of abdominal obesity, hypertension, diabetes, and dyslipidemia is called the metabolic syndrome. It has been shown that the occurrence of type 2 diabetes in patients with metabolic syndrome can be prevented by early identification and interventions such as exercises and dietary modifications (Knowler & Barrett-Connor, 2002). Since it is not clear whether the genetic predisposition affect the likelihood that a given person will become obese and develop metabolic syndrome, in this study, we tried to identify the prevalence of metabolic syndrome among the off-spring of diabetic patients.

Methodology
This study was a cross sectional study aimed at detecting the prevalence of metabolic syndrome in healthy offsprings of type 2 diabetes mellitus. Men and women in the age group 15- 45 years, without a history of diabetes mellitus, represented the study population. The study was carried out in the Centre for Diabetes and Lipid disorders, Faculty of Medicine, Karapitiya and the Diabetic Clinic, Teaching Hospital, Karapitiya. Sixty healthy offsprings of patients with type 2 diabetes mellitus were invited to participate in the study on a first come basis to screen for the presence of metabolic syndrome. After informed written consent was obtained, data regarding patient characteristics were recorded using a pre-designed questionnaire and detailed examination including measurement of body weight and blood pressure was carried out and documented. 10 ml of blood was drawn by a medical officer for investigations including plasma glucose and lipid profile. Concentrations of plasma glucose, serum total triglycerides, total cholesterol and HDL cholesterol were measured using standard
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analytical methods. Using the above results, total fatness and abdominal fatness was determined and the metabolic risk profile was assessed by the use of serum lipids, glucose and blood pressure measurements. Prevalence of metabolic syndrome was determined by the criteria laid down by the National Cholesterol Education Program (NCEP) Adult Treatment Panel (ATP) III. According to the NCEP/ATP III report, participants who had three or more of the following criteria were defined as having the metabolic syndrome (NCEP-ATP III 2002): abdominal obesity: waist circumference >102 cm in men and >88 cm in women; hypertriglyceridemia: ≥ 150 mg/dl 1.695 mmol/l); low HDL cholesterol: < 40 mg/dl; high blood pressure: ≥ 130/85 mmHg; and high fasting glucose: ≥ 110 mg/dl. Because of a revision of the waist circumference criterion for people in South East Asia (Grundy et al., 2004), in this study, the prevalence of the metabolic syndrome was measured using a waist circumference cutoff point of >90 cm in men and >80 cm in women. Recently, the American Diabetes Association changed the definition of impaired fasting glucose by lowering the glucose threshold to 100 mg/dl from 110 mg/dl (Genuth et al. 2003). This change was incorporated into the NCEP/ATP III definition of the metabolic syndrome (Grundy et al., 2004) and therefore we used the new glucose threshold of 100 mg/dl for our study.

Results

Out of the total number (60) of subjects selected, fifty two attended the morning examination. four were found to have diabetes mellitus and therefore excluded from the study. Of the forty eight participants, five (11%) were found to have the metabolic syndrome. However, there were 20 subjects (42%) with no risk factor for metabolic syndrome and nine (19%) and fourteen (29%) subjects had one and two risk factors respectively. (Table 1) There were twenty five (5%) males and twenty three (5%) females. Baseline characteristics of these two groups (male and female) were normal. However, the prevalence of metabolic syndrome in females was 21.7% whereas no male had metabolic syndrome in this cohort.

<table>
<thead>
<tr>
<th>No of risk factors</th>
<th>Frequency</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>No risk factors</td>
<td>20</td>
<td>41.7%</td>
</tr>
<tr>
<td>One risk factors</td>
<td>09</td>
<td>18.8%</td>
</tr>
<tr>
<td>Two risk factors</td>
<td>14</td>
<td>29.2%</td>
</tr>
<tr>
<td>Three risk factors</td>
<td>05</td>
<td>10.6%</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>48</strong></td>
<td><strong>100%</strong></td>
</tr>
</tbody>
</table>

The commonest factor that contributed for metabolic syndrome was HDL less than 40mg/dl (29.2%) followed by TG more than 150mg/dl (23%). The least common risk factor, which contributed for metabolic syndrome, was fasting blood sugar more than 100 mg/dl (4/48) (Table 2). In females the commonest risk factor which contributed for metabolic syndrome was waist circumference >80 cm (10/23) whereas in male it was HDL less than 40mg/dl (8/25).

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Number</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>HDL&lt; than 40mg/dl</td>
<td>14</td>
<td>29.2</td>
</tr>
<tr>
<td>TG &gt; than 150mg/dl</td>
<td>12</td>
<td>23</td>
</tr>
<tr>
<td>Waist circumference</td>
<td>11</td>
<td>20.9</td>
</tr>
<tr>
<td>High blood pressure</td>
<td>07</td>
<td>14.6</td>
</tr>
<tr>
<td>FBS&gt;100</td>
<td>04</td>
<td>8.3</td>
</tr>
</tbody>
</table>

The mean age of individual with metabolic syndrome was 39.2 years (±10.4) and those without metabolic syndrome were 28.7 years (±9.4). This indicates that the prevalence of the metabolic syndrome increased with age. This can be explained by the increase prevalence of risk factors like blood pressure in older people compared to young. We assessed the prevalence of metabolic syndrome in the respective diabetic patients to find out whether there was a relationship between these two groups. Out of the 29 diabetic patients, 10 (34%) had metabolic syndrome. This figure is much higher than that of their offspring.
Discussion
The frequent simultaneous presence of obesity, dyslipidemia, diabetes, and hypertension was first described in the late 1960s (NCEP-ATP 111 2002). This association (i.e., diabetes, hypertension, and obesity with dyslipidemia) was subsequently highlighted in the late 1970s by a number of German researchers (Meigs et al., 2003) They coined the term "metabolic syndrome" and described its association with atherosclerosis. However, the clinical significance of the metabolic syndrome remains controversial, and much remains to be learned about it. It is not clear if the syndrome is a disease or simply a constellation of risk factors (Reaven 2002). The aetiology of the metabolic syndrome is also controversial with obesity, insulin resistance and other aetiologies being advocated. By focusing on the metabolic syndrome, the NCEP/ATP III reinforced the need for health care professionals to take a more comprehensive approach to their patients and to address all relevant risk factors that increase the risk for type 2 diabetes and cardiovascular events. Our study did not reinforce the hypothesis of genetic transmission of this syndrome. But further analysis revealed that those who have metabolic syndrome were comparatively older than subjects without metabolic syndrome. The low prevalence of metabolic syndrome among offspring may be a result of the relatively young study population. Therefore, further study is necessary with comparatively older offspring of diabetic patients.

Conclusions
Prevalence of metabolic syndrome in the offspring of diabetic patients according to this study was 11% and was similar to that reported in the general population (Ford et al., 2002). However, the prevalence of metabolic syndrome in offspring was much lower than the prevalence of metabolic syndrome in their diabetic parents (34%). This may indicate that the environmental factors more than genetic background may be involved in the pathogenesis of metabolic syndrome.

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References