Prevalence of Metabolic Syndrome and Its Related Factors Among Adult Population Karikal District a Cross-Sectional Study

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Abstract: Lifestyle and behavioral changes that have occurred during the last century, such as an increasing obesity, sedentary lifestyle and excessively energy-rich nutrition, have contributed to a dramatic increase in the prevalence of metabolic syndrome (MetS) and type 2 diabetes. The prevalence of metabolic syndrome which is a group of atherosclerotic risk factors is rising at an alarming rate among young adults of urban India. Central obesity which represents the visceral adipose tissue deposits is the most prevalent manifestation of metabolic syndrome. Adipose tissue, a dynamic endocrine organ secretes pro-inflammatory factors called adipokines. This causes Meta inflammation. Prevalence of metabolic syndrome and its related factors among adult population karikal district. Using a cross sectional analytical study design, a total of 60 healthy subjects of Karikal district were enrolled in this study. Sixty healthy subjects were further divided into non-obese, group I (n=30) and obese, group II (n=30) based on their waist circumference Blood pressure, waist circumference, height and bodyweight were measured according to standard procedures and were reported in detail previously. Blood pressure Plasma glucose .Blood samples were obtained after 8 hours of fasting to determine blood chemistry parameters. Serum total cholesterol, triglycerides and HDL, LDL .

Results: In this study we found that obese of the study population of 20 adults had metabolic syndrome. 87.4% of the obese group had increased fasting glucose, and elevated triglycerides and LDL levels. The change in the prevalence of metabolic syndrome. Among our student population was not statistically significant. Low HDL cholesterol and hypertriglyceridemia were the main risks factors in the population less than 20 years of age, while in the population of 20–30 years, low HDL cholesterol and high blood pressure were the main risk factors (low HDL cholesterol affected two thirds of the population). Therefore, it is important the study of young populations in order to make a timely intervention in individuals at risk, given the high prevalence of metabolic abnormalities at an early age.

Keywords: Metabolic Syndrome, Elevated Triglycerides, Adult Obesity.

INTRODUCTION

Metabolic syndrome (MetS) is defined as a constellation of risk factors, including abdominal obesity, hypertriglyceridaemia, low levels of high-density lipoprotein cholesterol (HDL-C), elevated serum levels of fasting glucose and elevated blood pressure. These factors tend to cluster together, suggesting a common aetiology, and place individuals at increased risk for diabetes mellitus and cardiovascular disease (CVD) [1]. Recently, the prevalence of MetS has increased substantially in many developing Asian countries with the improved economic environment and the resulting sedentary lifestyles and changes in diet. Obesity is one of the most significant contributors of morbid conditions like metabolic syndrome [2]. The prevalence of metabolic syndrome in obese adolescents has been reported to be between 18% and 42% depending on the country of origin, suggesting an ethnic based association between obesity and metabolic syndrome. The worldwide epidemic of obesity is related to the increase in the prevalence of MetS, which is currently highly prevalent in the general population. In most countries 20-30% of the adult population can be characterized as having MetS. Different expert panels have provided various definitions for MetS to enable a clinical diagnosis and treatment of patients at risk of CVD [3]. However, MetS has proved to have only a limited practical utility as it is a pre-morbid condition rather than a clinical diagnosis. During the past 5 years, there has been increasing controversy regarding the concept of MetS and especially its usefulness as a diagnostic tool [4].

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Recent evidence has shown that MetS does not predict CVD events or disease progression any better than the sum of its components. However, MetS as a concept of risk factor clustering can be observed both in childhood and in adulthood, and exposure to metabolic risk factors, especially those related to obesity is associated with increased risk of atherosclerosis which can be measured by increased carotid intima-media thickness (cIMT). MetS and the components of MetS are strongly linked to the presence of obesity and, in particular, to waist circumference as a measure of intra-abdominal obesity [5]. This relation may not be due to obesity itself, but rather excess adiposity causes a decrease in insulin-mediated glucose uptake and increases likelihood of insulin resistance. Nevertheless, not all overweight/obese individuals are insulin resistant and carry metabolic abnormalities. For example, a substantial portion of these overweight/obese persons are insulin sensitive, without any of the metabolic abnormalities alternatively, insulin resistance and other metabolic abnormalities are not uncommon in normal-weight persons especially in persons of South Asian ethnicity [6]. Approximately 5,000 participants from the NHANES (1999-2004) were divided using BMI into normal weight, overweight and obese categories and NCEP criteria, HOMA-IR (90th percentile) and CRP (90th percentile) were used to define participants as metabolically normal (≤1 abnormality) or metabolically abnormal (≥2 abnormalities). Based on these parameters 51% of overweight and 32% of obese participants were classified as metabolic healthy and on the contrary 24% of normal-weight participants were metabolically abnormal [7].

**MATERIALS AND METHOD**

We performed a prospective, descriptive and nonrandomized study in karikal district during the period of 2015 June -2016 June. Pregnant subjects were excluded. The study design was approved by the institution’s ethics committee and informed consent was obtained from all the participants. Anthropometric measurements were taken, blood samples to perform blood chemistry and lipid profile were drawn, and blood pressure was measured. Blood pressure, waist circumference, height and bodyweight were measured according to standard procedures. And were reported in detail previously. Blood pressure measurements were the means of two separate measurements of systolic and diastolic blood pressure taken at an interval of 5 min. Blood samples were obtained after 8 hours of fasting to determine blood chemistry parameters. Serum total cholesterol, triglycerides and HDL-C were determined by enzymatic methods using chemistry analyzer. Plasma glucose was measured using the glucose oxidase peroxidase method. To determine metabolic syndrome, International Diabetes Federation criteria were used. Abdominal obesity was defined as a waist circumference ≥ 90 cm in men and ≥ 80 cm in women, plus two of the following fasting plasma glucose greater than 100 mg/dL or a prior diagnosis of DM2, triglycerides ≥ 150 mg/dL, HDL cholesterol ≤ 40 mg/dL in men or ≤ 50 mg/do in women, and a systolic blood pressure ≥ 130 mmHg or a diastolic blood pressure ≥ 85 mmHg. Economic status, hometown and parents job were not evaluated in this study population.

**RESULTS**

Table 1 shows the variations in biochemical parameters among non obese group I and obese II subject’s. Data are shown as Mean ± standard deviation BMI body mass index; BP blood pressure; LDL low-density lipoprotein cholesterol; HDL high-density lipoprotein cholesterol. Student’s t test was used for the analysis with a p <0.05 being considered statistically significant.

**Table 1: Variations in biochemical parameters among non obese group I and obese II subject’s**

<table>
<thead>
<tr>
<th>VARIABLES</th>
<th>NON OBESE(GROUP I)</th>
<th>OBESE II(GROUP II)</th>
<th>PVALUE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>18±6.8</td>
<td>17.76±9.3</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>weight (kgs)</td>
<td>63±7.14</td>
<td>72.89±4.83</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>waist (cm)</td>
<td>63.76</td>
<td>78.95</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Fasting glucose (mg/dl)</td>
<td>72.89±0.4</td>
<td>52.8±7.3</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Systolic BP (mm Hg)</td>
<td>110±0.8</td>
<td>128±7.2</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Diastolic BP (mm Hg)</td>
<td>78±6.2</td>
<td>85±8.5</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Triglycerides (mg/dL)</td>
<td>112.82±2.4</td>
<td>131±6.8</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>HDL (mg/dL)</td>
<td>45.12±6.8</td>
<td>39±2.9</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>LDL (mg/dL)</td>
<td>112.54±5.9</td>
<td>120.02±6.7</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Total Cholesterol (mg/dL)</td>
<td>129±7.3</td>
<td>144±3.4</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

**DISCUSSION**

The clustering of metabolic risk factors has been recognized for more than 80 years. Kylie, a Swedish physician, described a clustering of hypertension, hyperglycemia and gout. MetS has been in the scientific spotlight since Raven reintroduced the concept of Syndrome X in 1988 and proposed that insulin resistance is the primary underlying reason behind MetS, as it strongly associates with other risk factors [15] and correlates with cardiovascular risk.
Obesity was not originally included in the definition [8]. First of all, Syndrome X was a pathophysiological construct, attempting to explain why minor degrees of cardio metabolic abnormalities tend to cluster in the same individual. A considerable amount of research has been conducted in attempt to define the epidemiology of MetS. The existence of several different definitions has led to confusion especially when comparing MetS prevalence’s [9]. The prevalence of MetS varies widely among countries and ethnic groups, ranging from approximately 1% to 40%. However, in all countries and ethnic groups, the prevalence of MetS increases rapidly with age. Several prospective studies have indicated that MetS predicts future CVD and type 2 diabetes [10]. Moreover, individuals with MetS are susceptible to other conditions such as fatty liver, gallstones, asthma, obstructive sleep apnea, hypogonadism in men and polycystic ovarian syndrome in women, a clinical syndrome including an ovulation, androgen excess and insulin resistance. MetS is shown to be a strong predictor of incident type 2 diabetes [11]. According to previous studies, the relative risk of incident type 2 diabetes in participants with MetS ranges from 2 to 5. Impaired fasting glucose has tended to be the abnormality most strongly associated with incident type 2 diabetes. However, in the EPIC-Potsdam study; abdominal obesity was more strongly associated with incident type 2 diabetes than impaired fasting glucose [12].

Children and adolescents are becoming more overweight and the first traits of MetS can be found in these obese children. There are no widely accepted criteria available to diagnose MetS in childhood. Most commonly, modifications of the adult definitions with age- and sex-specific percentiles have been used in pediatric research [13]. Several study groups have investigated the prevalence of MetS in childhood and adolescence, although it is known that the clinical utility of the diagnosis may be reduced especially in young people because of changing growth patterns and the effects of puberty on insulin sensitivity and lipid profile. Moreover, the definition of MetS has undergone significant debate, especially focusing on which components and which component cut points should be part of the definition. The dichotomization of MetS risk factors has been particularly criticized [14]. Report of a WHO Expert Consultation have recently discussed potential limitations of MetS in detail and highlighted many important issues. Although persons with MetS need not be clinically obese, they commonly have an abnormal fat distribution that is characterized by excess upper body fat, which can be accumulated either intraperitoneally (visceral fat) or subcutaneously. There is substantial evidence that visceral fat is more strongly associated with insulin resistance and MetS than other adipose tissue compartments in obese participants. Alternatively, although waist circumference correlates well with the amount of total abdominal fat, it cannot distinguish between visceral adiposity, an important correlate of metabolic abnormalities, and subcutaneous abdominal fat. The relation between insulin resistance and hypertension is well established and is related to several different mechanisms in an insulin-resistant, non-diabetic individual not all tissues are equally insulin-resistant. For example the kidney is not resistant to the ability of insulin to promote the renal reabsorption of sodium and altering cell electrolyte composition. Similarly, compensatory hyperinsulinemia may raise blood pressure by increasing sympathetic nervous system activation [15].

CONCLUSION
MetS is common among young adults and increases substantially with age. Significant secular trend in MetS was observed for individuals aged 24 years between years 1986-2001 and that was driven mostly by an increase in the prevalence of obesity. Childhood predictors of adult MetS included obesity, family history of hypertension, family history of type 2 diabetes, high triglycerides, high insulin and high CRP. Identifying these risk factors in children and adolescents could be helpful in pediatric metabolic risk assessment.

REFERENCES
6. Barker DJ, Hales CN, Fall CH, Osmond C, Phipps K, Clark PM; Type 2 (non-insulin-dependent) diabetes mellitus, hypertension and