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OBESITY AN INDIVIDUAL RISK FACTOR FOR LIPOPROTEIN ABNORMALITIES IN PATIENTS WITH TYPE-2 DIABETES MELLITUS

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ABSTRACT

Objectives: To compare body mass index and lipid profile in type-2 diabetic patients and control subjects.

Material and Methods: The study was conducted in tertiary care hospitals of Peshawar from July 2005 to July 2008. Four hundred diabetic patients and hundred control subjects were included in this study. Blood glucose levels and lipid profiles were determined by enzymatic colorimetric method.

Results: The body mass index, fasting and random blood glucose, triglycerides are significantly increased ($p<0.001$), whereas the High density lipoprotein Cholesterol (HDL-C) concentration is significantly decreased ($p<0.001$) in type 2 diabetic patients as compared to control group.

Conclusions: Majority of diabetic patients are obese. Obesity leads to insulin resistance, which is evident before the onset of hyperglycemia and predictive of the subsequent development of diabetes. Insulin resistance leads to lipoprotein abnormalities.

Key Words: Obesity, type 2 diabetes, dyslipidemia, body mass index.

INTRODUCTION

Type 2 Diabetes Mellitus is characterized by four major metabolic abnormalities: obesity, impaired insulin action, insulin secretory dysfunction and increased endogenous glucose output, although there is substantial evidence that the first three of these abnormalities are present in most individuals before the onset of diabetes.[1]

Obesity is the most frequent nutritional dysfunction and at present is one of the greatest social problems. Obesity is an excess of body fat. It results due to an imbalance of energy. Excessive fat may be deposited due to some diseases or it may occur without any underlying disease. Obesity is a risk factor for chronic diseases such as diabetes mellitus, hypertension, cardiovascular disease, hyperlipidemia, some type of cancer and articular pathologies.[2]

The prevalence of overweight and obesity continues to increase in Pakistan, with more than half of all adults currently either are overweight or obese. In general, people become obese because of a combination of inherited genes, a life style consisting of low levels of physical activity and consumption of excess calories.[3]

In Pakistan the overall prevalence of obesity and over weight in educated population is 8 and 29.6% respectively which is sufficiently high.[4]

The operational definitions of obesity and overweight however are based on body mass index (BMI), which is closely correlated with body fatness. These BMI cut-off points are arbitrary along a continuum of increasing risk with increasing BMI. Cut-offs used for the definition of obesity can be based on statistical data from reference populations or on the excess morbidity and mortality associated with increasing body fat content.[5] The objective of the present study was to determine and compare BMI, fasting and random blood glucose and lipid profile concentration amongst type-2 diabetic patients and control subjects.

MATERIAL AND METHODS

This cross sectional, questionnaire based study was conducted in physiology department of Khyber Medical College Peshawar from July 2005 to July 2008. Four hundred type 2 diabetic patients were selected from three tertiary care hospitals of Peshawar i.e. Khyber Teaching Hospital, Lady Reading Hospital and

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Hayatabad Medical Complex. The age group was 40 to 60 years.

Patients with impaired glucose tolerance test or known diabetics taking oral hypoglycemic drugs, or managed with diet for control of diabetes were included in the study.

Patients using insulin, cardiovascular and hypertension medication, having previous history of angina, severe vascular disease, nephropathy or other life threatening disease, and patients taking corticosteroids or any drug affecting lipid profile or diabetic status, were excluded from the study.

Participants of the study were divided into three groups on the basis of body mass index, for Asian the normal body mass index is from 18.5 to 22.9 kg/m^2, body mass index considered overweight is from 23 to 24.9 kg/m^2 and obesity is considered when body mass index is > 25kg/m^2.

For laboratory purpose the facility of Pakistan Medical Research Council, Khyber Medical College was availed. The patients underwent a clinical assessment which included history (a questionnaire) and clinical examination. The variables of questionnaire were age, sex, marital status, personal history (occupation, education, socio economic status) eating pattern, nutritional status, exercise history, smoking status, menstrual history and family history (including family history of diabetes).

Fasting blood sugar and lipid profile was done for all the patients and controls. Participants were asked to come to the laboratory after an overnight fasting for at least 6 to 8 hours. Random blood sugar samples were taken after 2 hours of taking routine breakfast.

Determination of triglycerides was done by kit method (triglycerides escape), cholesterol determination by Elitech Cholesterol pap, and LDL, HDL cholesterol was determined by Friedewald formula.

RESULTS

Data was analyzed statistically. Two tailed "t" test was applied and p value was calculated in order to compare the different groups. $P<0.05$ was considered as significant. Out of 400 patients 376 (94%) had BMI >23 and 24 (6%) patients had a BMI < 23 whereas on the other hand in the control subjects 88 (88%) had BMI > 23 and 12 (12%) control subjects had BMI < 23.The results supported the importance between increased BMI and development leading towards the insulin resistance syndrome.

Table 1 shows the comparison of body mass index, fasting and random blood glucose, and the lipid profile amongst the control group and the type-2 diabetic patients. Body mass index, fasting and random blood glucose and triglycerides were significantly increased ($p<0.001$), whereas the HDL-C concentrations were significantly decreased ($p<0.001$) in type-2 diabetic patients as compared to the control group.

Table 1: Body Mass Index, Glucose And Lipid Profile In Control Group And Type-2 Diabetes Mellitus

	Control Group (n=100)	Type-2 Diabetes mellitus (n=400)
BMI	27.48 ± 4.02	29.82 ± 4.47*
Fasting blood glucose	85.20 ± 15.73	182.23 ± 70.90*
Random blood glucose	117.08 ± 23.57	246.99 ± 85.04*
Total Cholesterol	195.44 ± 45.61	183.37 ± 44.53**
Triglycerides	149.57 ± 66.24	193.72 ± 109.64*
HDL-C	49.63 ± 9.50	45.09 ± 9.37*
LDL-C	115.16 ± 40.25	98.51 ± 37.84*

Values are presented as mean + SD.

** $P<0.01$ as compared to control group.

* $P<0.001$ as compared to control group.

DISCUSSION

The obesity epidemic is an enormous problem associated with a significantly increased risk of type 2 diabetes, cardiovascular diseases and premature deaths[6]. Obesity is associated with increased mortality especially when it co-exists with diabetes.[7]

Overweight and obese individuals are at increased risk for developing type 2 diabetes.[8] There is an inverse relationship between BMI and the age of diabetes onset in obese adults who are younger then 70 years old.[9] The duration and degree of obesity, central distribution of weight, and recent weight are all independent risk factors for type 2 diabetes.[10] Geol. PK et al concluded that worsening of glycemic control and obesity deteriorates lipid and lipoprotein abnormalities.[11] This is evident in our study as well, as among 400 diabetic patients the total obese patients were 87.8% the lipoprotein abnormalities were significantly increased with increase in body mass index. The characteristic lipoprotein abnormality is marked triglyceridemia with low HDL-C levels.

The incidence of diabetes mellitus in the human population has reached epidemic proportions worldwide, and it is increasing at a rapid rate. In 2000, there were an estimated 150 million cases in the world, and this number is projected to be increased to 221 million by 2010. 90% of the present cases are type 2 diabetes, and most of the increase will be in type 2, paralleling the increase in the incidence of obesity.[12]

As the body weight increases, insulin resistance increases, i.e. there is a decreased ability of insulin to move glucose into fat and muscle and so shut off glucose release from the liver. Weight reduction decreases insulin resistance. Associated with obesity there is hyper insulinemia and dyslipidemia characterized by high circulating triglycerides and low HDL-C and accelerated development of atherosclerosis. This combination of finding is commonly called the metabolic syndrome or syndrome X. Some of the patients with syndrome are prediabetic, whereas others have type 2 diabetics.[13] It has not been proved but it is logical to assume that the hyper insulinemia is a compensatory response to the increased insulin resistance and that frank diabetes develops in individuals with reduced B cell reserves.

These observations and the other data strongly suggest that fat produces a chemical signal or chemical signals that act on muscles and the liver to increase insulin resistance. Insulin is glycogenic, antigluconeogenic, antilipolytic, and antiketotic in its action. It thus favors storage of absorbed nutrients and is a "hormone of energy storage". Glucagon, on the other hand, is glycogenolytic, gluconeogenic, lipolytic, and ketogenic. It mobilizes energy stores and is a "hormone of energy release".[14]

Because of their opposite effects, the blood levels of both hormones must be considered in any given situation. It is convenient to think in terms of molar ratio of these hormones. The insulin glucagon molar ratio fluctuates markedly because the secretion of glucagon and insulin are both modified by the conditions that preceded the application of any given stimulus for example the insulin-glucagon molar ratio at balance diet is approximately 2:3. The link between diabetes and obesity is such that obesity increases the odds of developing insulin resistance, a precursor to diabetes. An obese body requires much more insulin then a healthy weight body to maintain normal blood sugar levels. As body fat increases, the numbers and sensitivity of insulin receptors decline. In many cases, the body is eventually unable to produce enough insulin to maintain blood glucose levels within a normal range, and type 2 diabetes develops.[15]

CONCLUSION

Obesity is the most common and important cause of insulin resistance, leading to interrelated lipid and lipoprotein abnormalities.

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