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A REPORT ON DIABETES AND RISK FOR CARDIOVASCULAR DISEASE

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ABSTRACT
Patients with diabetes mellitus are at high risk of cardiovascular disease and the risk is further increased when they are complicated with nephropathy, neuropathy or Retinopathy. 33 patients were recruited into the study (patient group) along with 33 healthy individuals (control group). The control group was recruited from the people attending the master health checkup program of the hospital and staff of the department. In the present study we have evaluated the diabetes and risk for CVD in rural population of India. We found increased fasting glucose and total cholesterol in test subjects compared to control, but these are statistically not significant. An increase in glucose and lipid profile they are well with in the normal range. But these finding are crucial because they are at the higher normal range. This could be due to due to small number of population and age factor of the participants. Further studies which include more number of subjects from rural area will through more light on Prevalence of diabetes and risk for cardiovascular disease.

INTRODUCTION

Diabetes mellitus is syndrome of multiple etiologies. It is characterized by hyperglycemia with disturbances of carbohydrate, fat and protein metabolism. It is mainly characterized by insulin resistance, hypertension and polycystic ovarian disease. The prevalence of diabetes is rapidly rising all over the globe at an alarming rate1. Over the past 30 yr, the status of diabetes has changed from being considered as a mild disorder of the elderly to one of the major causes of morbidity and mortality (Wild *et al.,* 2004). More common form of diabetes, namely type 2 diabetes, which accounts for more than 90 per cent of all diabetes cases. Nowhere is the diabetes epidemic more pronounced than in India as the World Health Organization (WHO) reports show that 32 million people had diabetes in the year 2002 (Ahuja, 1979). The International Diabetes Federation (IDF) estimates the total number of diabetic subjects to be around 40.9 million in India and this is further set to rise to 69.9 million by the year 2025.

Future projections have estimated the prevalence of diabetes to exceed 300 million cases by 2030, with the majority of growth occurring in developing countries. It is well known that diabetes is associated with significant morbidity and mortality. For these reasons considerable resources have been invested to improve diabetes management (Wild *et al.,* 2004).

There have been several studies over the past 15 years documenting the impact of acute mental stress on blood lipid concentrations. The majority of experiments have demonstrated that acute stress elicits small but significant increases in the concentration of total and low-density lipoprotein (LDL) cholesterol, with less consistent increases in high-density lipoprotein (HDL) cholesterol Atherosclerotic lesions in men and in animals appear to be related to elevated total cholesterol (TC), LDL-cholesterol (LDL-C), decreased HDL-cholesterol (HDL-C) and excess fat consumption. Plasma lipid levels are not only influenced by the amount of fat consumed but by its nature as well. Hegsted *et al.,* 1965; Hegsted,1981 and Keys *et al.,*1965 demonstrated that the saturated fatty acids were twice as cholesterol rising as the polyunsaturated (PUFA). Various prospective epidemiological studies across several population groups indicate that type 2 diabetes progresses over a continuum of worsening insulin action, beginning with peripheral insulin resistance and ending with a loss of insulin secretion (Sridhar *et al.,* 2002). In most patients, insulin resistance can be detected long before the deterioration of glucose intolerance occurs. Insulin resistance is a quite common state, associated with aging and a sedentary lifestyle, as well as a genetic predisposition. The state seems to be fueled by, or perhaps to a certain extent the result of, obesity. The ensuing dysregulation of carbohydrate and lipid metabolism that occurs as a consequence of insulin resistance further exacerbates its progression (Alberti and Zimmet, 1998). Beta cells of the pancreas normally compensate for the insulin resistant state by increasing basal and postprandial insulin secretion. At some point, the b cells can no longer compensate,

failing to respond appropriately to glucose. This ultimately leads to the deterioration of glucose homeostasis and the development of glucose intolerance. Approximately 5 to 10% of glucose-intolerant patients in a given year progress to frank diabetes, which continues to worsen as insulin resistance increases.

Many studies suggest that genetic factors influence insulin resistance, but nearly all of these have assessed family resemblance, which can result from shared environmental influences rather than the effects of genes per se (Deo *et al.,* 2006). For example, persistent maternal smoking could cause siblings to resemble one another in having below average birth weights, and, since low birth weight is associated with insulin resistance in adult life such resemblance could lead to sibling resemblance in insulin resistance as adults. Such a shared environmental influence would simulate the action of shared genes. Nevertheless, taken in aggregate, the studies of family resemblance have generally been interpreted as supporting a genetic basis for the observed resemblance in various indicators of insulin resistance. Even in the absence of diabetes, insulin resistance is a key feature of other human disease states. Impaired insulin action coupled with hyperinsulinemia leads to a variety of abnormalities, including elevated triglycerides, low levels of HDL, enhanced secretion of VLDL, disorders of coagulation, increased vascular resistance, changes in steroid hormone levels, attenuation of peripheral blood flow, and weight gain (Varillas and Roche, 2005). Thus, insulin resistance is often associated with central obesity, hypertension, polycystic ovarian syndrome, dyslipidemia, and atherosclerosis. This constellation of symptoms is often referred to as syndrome X, or insulin resistance syndrome. Whether impaired insulin action is directly responsible for all of the symptoms in these patients remains unclear. However, the broad prevalence of insulin resistance and its association with profound metabolic abnormalities are widely accepted. Even after intense research for decades the molecular basis of insulin resistance, diabetes and its associated complications like CVDs are not clearly known (Varillas and Roche, 2005). This study is undertaken to study the prevalence of diabetes and risk for cardiovascular disease in rural population of India.

MATERIALS AND METHODS

33 patients were recruited into the study [patient group] along with 33 healthy individuals [control group]. The control group was recruited from the people attending the master health checkup programmed of the hospital and staff of the department. All the members were recruited with informed consent. The age differences were evaluated statistically and found to be matching. This was a case control study. All the procedures followed in the study were approved by the Institute research council and informed consent was obtained from the participants. 32 age and sex matched controls subjects with any acute infections or with co-existing systemic diseases such as coronary artery disease, hypertension or chronic renal failure were excluded. None of the subjects was receiving any form of drugs. Plasma glucose (Glucose oxidase and Peroxidase method) was estimated in fasting samples and Lipid profile was estimated using standard commercial kits.

Statistical analysis

Results were shown as M ± SD. Statistical significance of difference between the groups was evaluated using student's t-test. A p value of < 0.05 was considered as significant. Statistical analysis was performed using SPSS for Windows – Version 11.5.

RESULTS AND DISCUSSION

In the present study we have evaluated the diabetes and risk for CVD in rural population of India. We found an increased fasting glucose and total cholesterol in test subjects compared to control, but these are statistically not significant. A study was done in western India showed prevalence of 8.6 per cent in urban population (Mohan *et al.,* 2006). A more recent study reported a high prevalence (9.3%) in rural Maharashtra. The Amrita Diabetes and Endocrine Population Survey (ADEPS, a community based cross-sectional survey done in urban areas of Ernakulam district in Kerala has revealed a very high prevalence of 19.5 per cent (Menon *et al.,* 2006).

Indians across the globe have shown that Asian Indians have an increased risk for developing type 2 diabetes and related metabolic abnormalities compared to other ethnic groups. Although the exact reasons are still not clear, certain unique clinical and biochemical characteristics of this ethnic group collectively called as the "Asian Indian phenotype" is considered to be one of the major factors contributing to the increased predilection towards diabetes (Deepa *et al.,* 2006). Despite having lower prevalence of obesity as defined by body mass index (BMI), Asian Indians tend to have greater waist circumference and waist to hip ratios thus having a greater degree of central obesity (MC Keigue *et al.,* 1991). Again, Asian Indians have more total abdominal and visceral fat for any given BMI and for any given body fat they have increased insulin resistance. Moreover, they have lower levels of the protective adipokine and have increased levels of adipose tissue metabolites. Studies on neonates suggested that Indian babies are born smaller but relatively fatter compared to Caucasian babies and are referred to as "the thin fat Indian baby". A recent study confirmed this finding and suggested that the "thin fat phenotype" in neonates persisted in childhood (Mohan *et al.,* 2006). Even in the absence of diabetes, insulin resistance is a key feature of other human disease states. Impaired insulin action coupled with hyperinsulinemia leads

Table 1: Blood levels of the parameters studied (Mean ± SD)

Group	Plasma Glucose-F (mg/dL)	Serum Cholesterol (mg/dL)	Serum Triglycerides (mg/dL)	Serum HDL (mg/dL)	Serum LDL (mg/dL)
Controls(n = 33)	88.3 ± 12.70	161± 22.2	145.0± 15.3	42.03± 8.82	74.87± 18.2
DM(n = 33)	106.8±11.9	210.3±29.1	144.3± 20.7	38.66± 11.3	100.8± 13.0
p Value	< 0.001	< 0.05	NS	<0.05	<0.05

NS: Not Significant

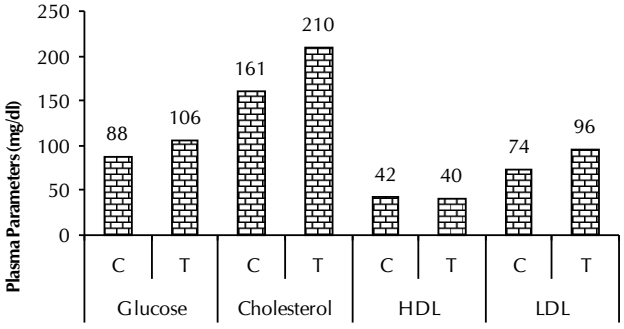

Figure 1: Levels of Glucose, cholesterol and LDL in patients. A significant increase in the levels was observed in Diabetes mellitus patients when compared to controls C = Control Group, T = Test Group

to a variety of abnormalities, including elevated triglycerides, low levels of HDL, enhanced secretion of VLDL, disorders of coagulation, increased vascular resistance, changes in steroid hormone levels, attenuation of peripheral blood flow, and weight gain(Radha and Mohan, 2007). Thus, insulin resistance is often associated with central obesity, hypertension, polycystic ovarian syndrome, dyslipidemia, and atherosclerosis.

In the present study even we get an increase in glucose and lipid profile they are well with in the normal range. But these finding are crucial because they are at the higher normal range. This could be due to due to small number of population and age factor of the participants. Further studies which include more number of subjects from rural area will through more light on Prevalence of diabetes and risk for cardiovascular disease in rural population of India.

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