

A Comparative Study of Biochemical Indices between Control and Diabetic Patients

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Abstract:

Introduction: Diabetes is a major risk factor for number of noncommunicable diseases. Novel factors like lipid profiles have contributed as risk factors for coronary artery disease along with abdominal obesity. So a comparative study is done to know the extent of altered lipid metabolism in NIDDM.

Methods: Lipid profiles along with blood sugar levels were done to know the effect of dyslipidemia. Available NIDDM patients with history of diabetes without any complication were taken and equal no. of age & sex matched normal healthy control subjects were recruited in the study.

Results: TC showed a highly significant more value ($P < 0.001$) in both male & female NIDDM patients when compared to control group. TG showed higher values in male NIDDM patient than control groups but not statistically significant. But female NIDDM patients showed statistically significant when compared to controls. HDL-C level was lower ($P < 0.05$) in both male & female NIDDM patients when compared to control group. LDL-C showed significant higher value ($P < 0.01$) in male NIDDM patients and ($P < 0.001$) in female NIDDM patients when compared to control group. VLDL-C showed statistically significant higher value ($P < 0.01$) in males & ($P < 0.001$) in females NIDDM patients when compared to control group.

Interpretation and Conclusions: Lipid profiles like, TC, LDL-C & TG are more in female NIDDM than male NIDDM patients & controls.

Keywords: Noninsulin dependent diabetes mellitus (NIDDM), Total cholesterol (TC), Very low density lipoprotein cholesterol (VLDL-C), Low density lipoprotein cholesterol (LDL-C), Fasting blood sugar (FBS), Post prandial blood sugar (PPBS)

I. Introduction

India alone would have 57 million diabetes mainly of type – 2 diabetes constituting 90% of the diabetic population. Worldwide cases of diabetes is 150 million, and number is to be doubled by 2025 prevalence rate of about 5.4 per cent (1). Increase is due to the dramatic up surge in obesity between age group 25 & 84 years, & higher in males. Lipid abnormalities in NIDDM are secondary consequences of insulin resistance. The most important defect in insulin deficient subjects appears to be a deficiency of lipoprotein lipase which is responsible for the removal of the triglyceride rich lipoproteins. In NIDDM the defect is over production of VLDL, triglyceride, cholesterol & HDL-cholesterol levels are decreased in obese NIDDM (30,31). Wide spread biochemical abnormalities are present but the fundamental defect to which most of the abnormalities can be traced are as follow- reduced entry of glucose into various peripheral tissues & increased liberation of glucose into the circulation from the liver. Therefore there is an extracellular glucose deficiency which, a situation that has been called as “starvation in due the midst of plenty” The entry of amino acid into muscle is decreased & lipolysis increased (18).

Increased catabolism of protein & fat produces the consequences of increased fat catabolism, that is ketosis. Principal abnormalities of fat metabolism in diabetes are acceleration of lipid catabolism with increased formation of ketone bodies and decreased synthesis of fatty acids & triglycerides (2), (9).

The manifestations of the disordered lipid metabolism are so prominent that diabetes has been called “more a disease of lipid than of carbohydrate metabolism” (4).

In uncontrolled diabetes the plasma concentration of triglycerides & chylomicrons as well as FFA is increased & the plasma is often lipemic. The rise in the constituents is due to mainly decreased removal of triglycerides into the fat depots. The decreased activity of lipoprotein lipase contributes to this decreased removal (14).

II. Materials And Methods

In our study all available cases of NIDDM patients aged more than 40 years attending Out –Patient-Department of Medicine were included in the study as “ Study group”. Inclusion Criteria for the study group included history of diabetes mellitus less than 5 years. They were on diet control, and without insulin treatment. “Control Group” had no history suggestive of any chronic illness like diabetes mellitus, hypertension etc. Informed consent was taken from the both study and control groups. Our study had Ethical clearance from our institutional ethical committee. Biochemical parameters like, FBS, PPBS, Serum CH, TG, and HDL-C were estimated by semi-Auto analyzer. Serum VLDL-C and LDL-C were calculated.

In our study we included 30 males and 15 females aged more than 40 years as study group and similarly control too. They were instructed to come fasting for 8-10 hours to the laboratory for investigation at next morning. With aseptic precaution 5ml of fasting blood sample was taken analysed for lipid profile and blood sugar.

Blood glucose was estimated by Trinders method .Lipid profiles are estimated from fasting 3 ml of blood in plane bulb made to stand still for 1/2 hour. Later centrifuge for 3,000 revolutions per minute. Serum cholesterol estimated by using Roeschlan method. (Normal value is 140-250 mg /dl) . Serum Triglyceride is estimated by Mc. GOWAN method. Normal value is less than 160 mg / dL. Serum HDL is estimated by BURSTEIN method. Normal values males 30-65 mg/dl & females 35-80 mg/dl. Analysis of result was done by using Unpaired Student “ t” test.

III. Results

All the subjects age, height, weight, and biochemical parameters were analysed. Average age of diabetic group was 59.23 years and in control group is 56.93 years . (table 1).FBS was very highly significant in male diabetic patients than female diabetic patients and controls. PPBS was highly significant in female diabetic patients compared to male diabetic patients and control subjects.

Serum CH, LDL and TG was more significant in female diabetic patients than male diabetics and control subjects. Serum HDL levels was lower in diabetic patients (male and female) than control subjects. Serum VLDL levels was more significant in male diabetic patients when compared to female diabetic patients. FBS was statistically very highly significant ($P<0.001$) but in females it was significant ($P<0.05$) (Graph No. 1 & 2). PPBS was statistically in males it is significant ($P<0.05$) in females it is highly significant ($P<0.01$). Females have increased PPBS than males (Graph no. 1 & 2) . Serum CH of control group males was 191.27 ± 26.47 mg/dl & in females was 177.67 ± 25.51 mg/dl, but in diabetic males was 260.2 ± 17.79 mg/dl. & in females was 274.8 ± 17.58 mg/dl. It was statistically highly significant in males ($P<0.01$) & in females it was very highly significant ($P<0.001$). Females had increased serum cholesterol levels compare to males. (Graph no. 3 & 4) Serum TG The control group males had 155.43 ± 32.52 mg/dl & in females 154.43 ± 28.18 mg/dl where as in diabetic group males had 201.1 ± 24.47 mg/dl & in females had 233.33 ± 29.35 mg/dl. The statistically in males it was not significant but in females it was significant. Females had hypertriglyceridemia than male (Graph No. 3& 4) .

Serum HDL-c control male had 65.2 ± 12.76 mg/dl but in females 65.6 ± 13.63 mg/dl where as is diabetic male was 39.43 ± 10.66 mg/dl & in females 38.2 ± 9.31 mg/dl. In both males & females it was statistically significant ($P<0.05$). (Graph No. 3 &4) .Serum LDL -C control male had 95.39 ± 31.85 mg/dl but in females 81.19 ± 24.92 mg/dl, where as in diabetic males it was 179.97 ± 20.03 mg/dl. The statistically in males it was highly significant ($P<0.01$) but in females it was very highly significant ($P<0.001$). Serum LDL was increased more in females compare to males. (Graph No. 3&4)

Serum VLDL : The control male had 30.41 ± 3.33 mg/dl but in females was 30.88 ± 5.64 mg/dl. Where as in diabetic groups in males was 41.43 ± 4.49 mg/dl & in females was 46.67 ± 5.87 mg/dl. The statistically in males it was highly significant & in females it was significant. Serum VLDL was increased in males compare to females. (Graph No. 3 &4)

IV. Discussion

Diabetes mellitus is a global problem, with approximately 150 million diabetic patients (26). This chronic condition poses a five times greater risk of developing micro vascular complications, mainly nephropathy and it has become the leading cause of end stage renal disease. NIDDM is a chronic degenerative disease and poses major challenges to the public health (31).

In our present study of both diabetic patients and controls highlights the facts about the presence of dyslipidemia associated with hyperglycemia .Confirming the presence of diabetes in our study groups there was

no difference in age between two groups ,therefore the results obtained in the study are due to impact of age on the biochemical profiles .

Hyperglycemia was confirmed by estimating FBS and PPBS levels in both diabetic and control groups which were significantly higher . Lipid profile showed raised in serum CH, TG ,and LDL levels but reduced HDL – C levels constitute the key feature of the insulin resistance syndrome which was similarly seen in other studies (15).

Insulin resistance as a result of increasing obesity, aging and glucose intolerance concomitantly. As response to increasing glycemia, insulin secretion increases, limiting increase in plasma glucose concentrations. The insulin secretory response declines and hepatic glucose production and plasma glucose concentrations increases in parallel with the decline in plasma insulin. Concentration response may be the effect of aging or prolonged mild hyperglycemia so called glucose toxicity.

Dyslipidemia in diabetes mellitus ie is high triglyceride, low HDL cholesterol. (4, 6).

The total LDL cholesterol changes ie is a shift to smaller LDL denser particles & an increased suability to oxidation. Oxidized LDL promote atherosclerotic complication in diabetes mellitus. Raised triglycerides, a marker of insulin resistance syndrome seen in ,NIDDM diabetes subject than raised LDL cholesterol.(3,5,6,14)

Diabetes mellitus is a disorder characterized by impaired carbohydrate, fat & proteins metabolism. NIDDM is characterized by impaired pancreatic β -Cell function & insulin resistance or by decreased tissue uptake of glucose in response to insulin. Insulin level may be normal decreased or increased, but secretion of insulin is impaired in relation to the degree of hyperglycemia. It is usually diagnosed after by age 30-40 years and 75% of individuals with type II are obese or have a history of obesity(28) &(16).

NIDDM is characterized by insulin resistance at the receptor & post receptor level leading to the elevation of plasma insulin concentration. Obesity is very common in NIDDM is characterized with insulin resistance & high insulin levels obese diabetic patients suffer from endogenous hyperinsulinemia, where liver is exposed to high insulin concentration. The degree of hyperinsulinemia plays a central role in the development of lipid & lipoproteins abnormalities in NIDDM. So obese diabetic have lower levels of HDL cholesterol & higher levels of total triglycerides & VLDL triglycerides than the corresponding non-diabetic controls subjects (7).

Moderate lipoprotein lipase deficiency is found in NIDDM, lead to the subnormal formation of HDL from triglyceride rich lipoproteins. Insulin is the major stimulant of lipoprotein lipase but hyperinsulinemia seen in NIDDM does not lead to an increased level of HDL which is probably due to its role in stimulating production in the liver.

Total triglycerides & VLDL levels were elevated in the diabetic patients as compared to those in the non-diabetic control subjects. Disturbances of triglyceride metabolism may be partly explained by the high prevalence of obesity in these patients. Hypertriglyceridemia seen in NIDDM is caused by excessive secretion of VLDL into plasma & may be explained on the basis of hyperinsulinemia, hyperglycemia, or increased influx of free fatty acids into the liver. Over production of VLDL in the major cause of hypertriglyceridemia in NIDDM, although defective removal due to a decrease in the lipoprotein lipase activity may contribute to a high level of VLDL triglycerides(12,13).

Adipose tissue is less resistant to insulin in obesity than NIDDM due to cellular alterations. Thus as long as adipose tissue responds to insulin action, LDL size remains unaffected due to adipose tissue lipoprotein lipase activity & levels respond to insulin is conceivable that Lipoprotein lipase may have a role in shaping LDL. And also alternative way is abnormal lipoproteins especially triglyceride rich lipoproteins should induce insulin resistance & elevated fasting insulin levels by impairing insulin mediated glucose uptake (21).

The reasons for the impaired efficiency of VLDL apo-B conversion to LDL in the upper lipemic is as follows - the longer transit time necessary to remove the VLDL TG load prosecuted to the Lipolytic system may effect the partial delipidation of a greater fraction of plasma VLDL, which their re-enters the circulation as remnant VLDL. It also lead to impairment in the lipolytic cascade, stuce in patient with moderately severe diabetes the efficiency of this conversion is much lower than in mild diabetes, despite of equal magnitudes of VLDL secretion & hypertriglyceridemia. Thus increased supply supply of cholesterol to peripheral tissues, whether derived from LDL ot remnant VLDL may contribute to increased cardiovascular risk in diabetes. (22)

The cause for obesity are sedentary life style and high - fat energy dense diet. The rising epidemic of obesity reflects preformed changes in society and in the behavioral patterns of communities, because of genetic or other biological predisposition to gain weight more readily when they are exposed to an unfavorable environment (17).Central obesity which predicts NIDDM, as significant risk factor when percentage of body fat and insulin resistance were taken into account. Insulin resistance lead to decreased rate of glycogen synthesis in skeletal muscle, insulin resistance in skeletal muscle is predictive of NIDDM (27).

In the present study, elevated levels of total cholesterol and triglycerides and decreased HDL levels were observed in the cases with complications. This was also observed in the studies which were carried out by Tuttle et al (32) and Ravid et al (33) .

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