Original Research Article

Comparison of Fasting and Postprandial Lipid Profile in Diabetic Patient and Healthy Individuals

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Abstract

Objective: To compare fasting and postprandial lipid profile in healthy individuals as well as in diabetic patients. **Materials and Methods:** The study will be conducted which includes 30 type-2 diabetic patients diagnosis based on American Diabetes Association Guidelines and 30 healthy nondiabetic control inpatients of medicine department. Fasting and Postprandial lipid levels will be estimated in all cases and controls and blood sample will be collected from patients after an overnight (12hours) fasting and four hours (4hours) postprandial after giving fixed diet. Statistical analysis will be done on collected data. **Results:** Type 2 diabetes has significantly deranged fasting and postprandial lipid profile when compared with controls. In the type 2 diabetes postprandial lipid parameter were significantly increased as compared to the fasting lipid parameters, and the postprandial HDL level were significantly decreased as compared to the fasting HDL level. **Conclusion:** This study concludes the fasting and post-prandial lipid profile is significantly deranged in Diabetics as compared to non-diabetics. Hence diabetics patients are at increased risk of atherogenic event as compared to non-diabetics and requires rigorous control of lipid profile.

Keywords: HDL, LDL, VLDL, DM, TG.

Introduction

Diabetes mellitus (DM) includes a group of metabolic disturbances characterized by hyperglycemia for an extended period. Various etiology contributing to hyperglycemia may include decreased insulin secretion, decreased glucose metabolism, and increased glucose production[1]. It may lead to disturbances in the metabolism of Lipid, carbohydrates, and protein.

Insulin deficiency or resistance to insulin may result in lipid abnormalities by affecting the vital enzymes and pathways involved in metabolism of lipid in DM[2]. Type2 diabetes is associated with a cluster of interrelated plasma lipid and lipoprotein abnormalities, including elevated triglycerides and reduced HDL cholesterol[3]. Insulin resistance has striking effects on lipoprotein particle concentrations for VLDL, LDL and HDL[4,5].

Cholesterol and triglycerides are the significant fats in diet, cholesterol and triglycerides that are taken up by the body.

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Esterification of fat which is primarily done by insulin is the one affected in diabetes and additionally assist the glucose uptake and subsequent conversion into fat in the periphery and hormone-sensitive lipase inhibition. Hence, in type 2 DM, the entire downstream process is affected by hyperinsulinemia that results in an abnormality of lipids especially increased cholesterol and triglycerides (TG) in the bloodstream, i.e., diabetic dyslipidemia[6].

Indian population is genetically predisposed to higher adiposity in upper limbs, higher body fat and higher level of insulin resistance. Along with higher risk of environmental insults, there is higher risk of diabetes and its complication in Indians[7].

Meta-analysis of six large prospective studies by Hokanson J.E and Austin, showed that fasting triglyceride rise was associated with CVD[8]. In contrast, one of the earliest studies done on dyslipidemia showed a substantial increase of developing CVD with postprandial lipid levels abnormality[9]. Additionally, Higher CVD mortality associated with postprandial dyslipidemia in Type 2[10].

In past two decades there has been an alarming rise in the prevalence of CVD in India accounting for 24% of all mortality among adults aged 25–69 years and develop CVD at a younger age than other populations[11,12]. The probable causes for the rise includes lifestyle modification with urbanization and the epidemiologic and nutritional transitions accompanying economic development[13]. Hypertriglycedemia has been consistently shown to be associated with a greater risk for atherosclerosis in those with type 2 diabetes mellitus. In persons with prolonged increases of plasma triglycerides,

either fasting or post prandial, the process of lipid exchange would enrich the triglyceride rich particles in cholesteryl ester and thereby make these particles more atherogenic[14].

Methods and materials

The study will be conducted with 30 type-2 diabetic patients and 30 healthy nondiabetic control will be taken from inpatients of medicine department at MMMC&H Kumarhatti, Solan. To diagnose diabetes American Diabetes Association Guidelines will be followed.

American Diabetes Association Guidelines

- A1C>6.5%. The test should be performed in a laboratory using a method that is National Glycosylated haemoglobin Standardized Program (NGSP) certified and standardized to the Diabetes Control and Complication Trial (DCCT) assay.
- FPG >126 mg/dL (7.0 mmol/L). Fasting Plasma Glucose is defined as no caloric intake for at least 8 h.
- Two-hour plasma glucose>200 mg/dL (11.1 mmol/L) during an Oral Glucose Tolerance Test (OGTT). The test should be performed as described by the World Health Organization, using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water.
- In a patient with classic symptoms of hyperglycemia or hyperglycemic crisis, a random plasma glucose >200 mg/dL (11.1 mmol/L).
- **Fasting and Postprandial Lipid Levels**
- Fasting and Postprandial lipid levels will be estimated in all diabetic patients and controls. Blood sample will be collected from patients after an overnight (12hours) fasting and four hours (4hours) postprandial. This study and controls group will be given fixed diet containing appox. 750 kcal/m^2 and 45gm of fat for lipid profile measurements. The diet will consist of 4 white bread slices, 50 gm amul butter and 250 ml full cream milk.
- Lipid profile which comprises of the total cholesterol (TC), triglycerides (TGs), high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C).

Inclusion Criteria

- As describe above.

Exclusion Criteria

TYPE 1 DM patients

- Those with history of inherited disorders of lipid metabolism (lipoprotein lipase deficiency, familial apolipoprotein C-II deficiency, familial hypercholesterolemia, familial defective apoB-100)
- Liver disease
- Hypothyroidism
- Cushing syndrome
- Nephrotic syndrome
- H/O Smoking or Alcohol
- H/O taking drugs that affect lipid metabolism like lipid lowering drugs, estrogens (OCP), thiazides, Glucocorticoids, beta blockers etc.

Results

In this study, in the fasting state mean TC level in the controls was 159.87 ± 13.50 mg/dl and a mean TC level of 177.15 ± 43.64 mg/dl in the diabetics. Also, there is significantly increased levels of the TG levels in diabetics (165.42± 62.01) as compared to the controls (95.16 ± 3.3) While, the controls had a mean LDL-C level of 76.36 ± 6.02 mg/dl in the fasting state and the diabetics had a mean LDL-C level of 102.52 ± 6.74 mg/dl in the fasting state, which is also significant. The HDL cholesterol is significantly lower in diabetics (41.99 ± 9.85) as compared to the controls (44.53 ± 3.20). Similarly, the diabetics (29.26 ± 13.57) had higher VLDL cholesterol than their corresponding controls (32.80 ± 12.64).

In present study, in the postprandial state mean TC level in the diabetics was 206.26 ± 29.17 mg/dl which is significantly more than the mean TC level of 153.87 ± 3.07 mg/dl in the controls. Also, there is significantly increased levels of the TG levels in diabetics (212.17 ± 70.90) as compared to the controls (129.6 ± 20.30). While, the controls had a mean LDL-C level of 73.86 ± 3.07 mg/dl in the postprandial state and the diabetics had a mean LDL-C level of 98.90 ± 40.32 mg/dl , which is also significant. The HDL cholesterol is also lower in diabetics (39.74 ± 13.14) as compared to the controls (43.87 ± 3.07). Similarly, the diabetics (41.02 ± 15) had a higher VLDL cholesterol than their corresponding controls (23.26 ± 1.98).

Table 1: Fasting Lipid Profile

Biochemical Study Parameters	Controls N = 30 (Mean ± S.D.)	Diabetics N = 30 (Mean ± S.D.)
Fasting Cholesterol	159.87 ± 13.50	177.15 ± 43.64
Fasting Triglycerides	95.16 ± 3.32	165.42 ± 62.01
Fasting HDL-C	44.53 ± 3.20	41.99 ± 9.85
Fasting LDL-C	76.36 ± 6.02	102.52 ± 6.74
Fasting VLDL-C	29.26 ± 13.57	32.80 ± 12.64

Table 2: Postprandial Lipid Profile

PP - Serum Lipids	Controls N = 30 (Mean ± S.D.)	Diabetics N = 30 (Mean ± S.D.)
Total Cholesterol (TC)	153.87 ± 3.07	206.26 ± 29.17
Triglycerides	129.6 ± 20.30	212.17 ± 70.90
HDL-Cholesterol	43.87 ± 3.07	39.74 ± 13.14
LDL-Cholesterol	73.86 ± 3.07	98.90 ± 40.32
VLDL-Cholesterol	23.26 ± 1.98	41.02 ± 15

Discussion

The study was conducted at MMMC&H Kumarhatti, Solan (H.P) to study correlation of postprandial lipid profile and CIMT in diabetes mellitus patients. 30 diabetic (type II) patients and 30 healthy subjects were selected from OPD or indoor wards and their fasting and postprandial lipid profile levels were estimated

This study suggests that mean fasting cholesterol, mean fasting triglyceride, mean fasting LDL-C, mean fasting VLDL-C were significantly higher in diabetic group as compared to healthy controls while mean fasting HDL-C was lower in diabetic patients as compared to healthy controls.

Also mean postprandial cholesterol, mean postprandial triglyceride, mean LDL-C, mean VLDL-C were found significantly raised in diabetic group as compared to healthy controls, while mean postprandial HDL-C was lower in diabetics signifying abnormal lipid profile derangements in diabetic patients.

Conclusion

This study concludes the fasting and post-prandial lipid profile is significantly deranged in Diabetics as compared to non-diabetics. Hence diabetics patients are at increased risk of atherogenic event as compared to non-diabetics and requires rigorous control of lipid parameters.

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