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POSTPRANDIAL LIPID PROFILE ABNORMALITIES IN TYPE 2 DIABETES MELLITUS

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ABSTRACT

Background: Dyslipidemia is commonly seen as diabetes. Type 2 DM is one of the most common secondary causes of hyperlipidemia. Type 2 diabetes mellitus is associated with the development of premature arteriosclerosis and higher cardiovascular morbidity and mortality. Diabetic dyslipidemia is believed to play an important role in the pathogenesis of accelerated atherosclerosis in this condition. The predominant lipid abnormalities seen in diabetes mellitus are an elevated serum triglyceride (Tg) level and a low HDL-C level. While several studies have found a significant association of fasting hypertriglyceridemia and coronary artery disease (CAD) in diabetes mellitus, the relationship is not consistent particularly after adjusting for fasting HDL-C Levels.9 It is being increasingly believed that atherosclerosis is a postprandial phenomenon as at least with respect to lipids, we are in the postprandial phase for nearly 2/3rd of the day. High postprandial triglycerides have shown a strong and independent association with CAD. Earlier studies of postprandial lipids in diabetes mellitus have suggested abnormalities of Tg metabolism secondary to insulin resistance (15) although results have not been consistent.
Aims: To assess the postprandial lipid abnormalities in type 2 diabetic patients attending PRH Loni.
Objective: To study postprandial lipid profile abnormalities in Type 2 diabetes patients as compared to non diabetics.
Methodology: The present study was a prospective case-control type of study that was conducted in Pravara Rural Hospital, Loni. (PRH) with a sample size of 187 (137 cases and 50 controls).
Results: The mean age of cases was 57.78 ±12.79 years and of controls was 60.50 ±14.39 years. The fasting bsl in cases was 204.61 ±105.01mg/dl and controls were 98.52±30.03 mg/dl. Postprandial bsl was 235.13 ±114.85 mg/dl in cases and 141.92 ±62.47 mg/dl in controls

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(P= 0.0001; P<0.05 is significant). Total cholesterol level in cases was 157.52 ±53.08 mg/dl and in controls was133.06 ±34.22 (P=0.002). Triglycerides level was 185.23 ±104.34 mg/dl in cases and 123.70 ±47.83 mg/dl in controls (P=0.0001). VLDL value in cases was 37.14 ±21.41mg/dl and in controls was 25.14 ±10.46mg/dl (P=0.0002).

INTRODUCTION

Diabetes mellitus (DM) is the most common metabolic disorder affecting people worldwide. Even though diabetes has been known since antiquity, only in the last few decades new discoveries have provided great hopes to minimize morbidity and mortality. Type 2 Diabetes Mellitus is caused by decreased insulin sensitivity at the end receptor level in the body. Hence the insulin production, to begin with, is normal but increases gradually due to the insensitivity of receptors leading to a state of hyperinsulinemia which, still, paradoxically causes a state of hyperglycemia. Type 2 Diabetes Mellitus is characterized by insulin resistance associated with glucose intolerance, hypertension, dyslipidemia and a procoagulant state and an increase in the micro vascular and macrovascular disease. Dyslipidemia is commonly seen as diabetes. Type 2 DM is one of the most common secondary causes of hyperlipidemia. Type 2 diabetes mellitus is associated with the development of premature arteriosclerosis and higher cardiovascular morbidity and mortality.[1-3] Diabetic dyslipidemia is believed to play an important role in the pathogenesis of accelerated atherosclerosis in this condition[4-5]. The predominant lipid abnormalities seen in diabetes mellitus are an elevated serum triglyceride (Tg) level and a low HDL-C level[6]. While several studies have found a significant association of fasting hypertriglyceridaemia[5,7,8] and coronary artery disease (CAD) in diabetes mellitus, the relationship is not consistent particularly after adjusting for fasting HDL-C Levels[9]. It is being increasingly believed that atherosclerosis is a postprandial phenomenon as at least with respect to lipids, we are in the postprandial phase for nearly 2/3rd of the day. High postprandial triglycerides have shown a strong and independent association with CAD.

Earlier studies of postprandial lipids in diabetes mellitus have suggested abnormalities of Tg metabolism[13,14] secondary to insulin resistance[15] although results have not been consistent[(16)].

Therefore, the present study was done to assess the postprandial lipid abnormalities in type 2 diabetic patients.

MATERIALS AND METHODS:

Research Design: Case-Control Study

Duration of the Study: October 2017 to October 2019

Study Setting: The study was conducted in Pravara Rural Hospital, Loni, a tertiary care teaching hospital situated in the rural area of Ahmednagar district.

Sample Size: A total of 187 individuals were included in the study. This included 137 subjects with Type 2 Diabetes Mellitus and 50 age and gender-matched non-diabetic patients.

Informed written Consent: Was taken in the mother tongue of the patient and the participants were assured of their confidentiality.

Participants were then interviewed in their mother tongue or the language they best understood (Marathi, Hindi or English).

Samples for blood sugar levels were collected in fluoride bulbs, one after an 8 hour fast and another, 2 hours after dinner. Blood samples for lipid profiles were taken 2 hours after dinner in bulbs containing clot activator. The samples were processed within 2 hours of collection. Samples were processed in the Vitros 5600 machine manufactured by Ortho Clinical Diagnostics.

RESULTS:

As shown in Figure 1 and Table 1 blood sugar levels of diabetics were compared against age and gender-matched controls. There was a significant difference in fasting and postprandial blood sugar levels of diabetics and non-diabetics.

Table 1: Comparison of blood sugar profile among two groups

Blood sugar	Diabetes group	Control group	P-value
Fasting	204.61 ±105.01	98.52 ±30.03	<0.0001
Postprandial	235.13 ±114.85	141.92 ±62.47	<0.0001

(P<0.05 statistically significant)

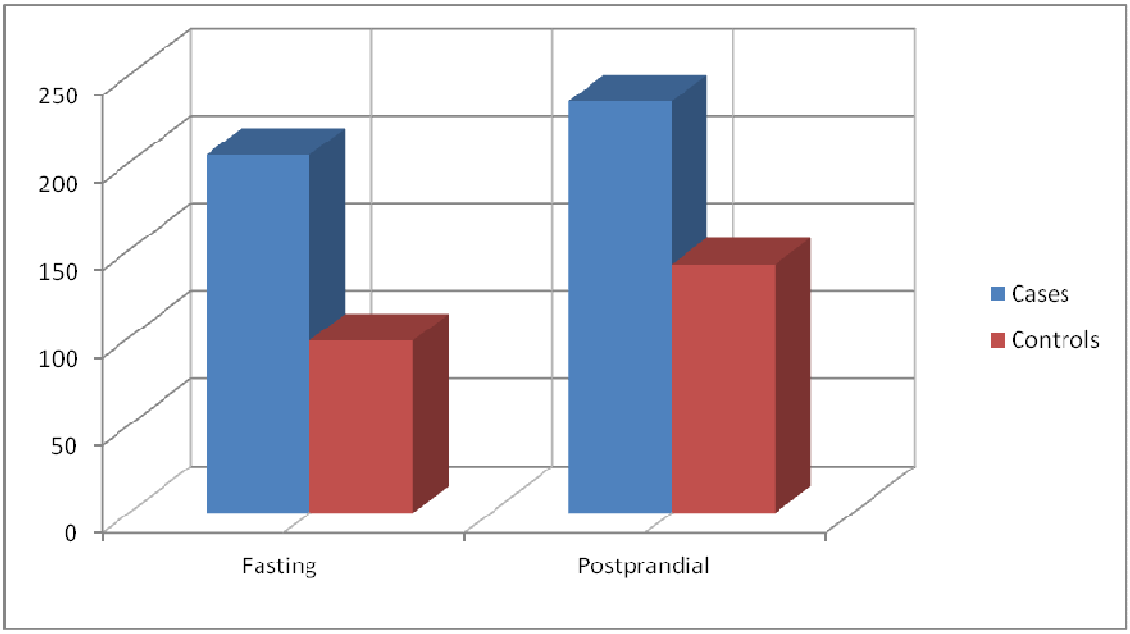

Figure 5: Figure showing a comparison of blood sugar levels (fasting and postprandial) among cases and controls.

The postprandial lipid profiles of diabetics were compared against non-diabetics and there was significant derangement in lipid profiles of diabetics. As shown in Figure 2 and Table 2, there was a significant difference in postprandial lipid profiles of diabetics as compared to non-diabetics.

Table 2: Comparison of lipid profile among two groups

Lipid profile	Diabetes group	Control group	P-value
Total cholesterol	157.52 ±53.08	133.06 ±34.22	0.002
Triglycerides	185.23 ±104.34	123.70 ±47.83	<0.0001
HDL	31.57 ±13.49	31.90 ±11.70	0.874
LDL	88.61 ±44.29	75.40 ±29.56	0.052
VLDL	37.14 ±21.41	25.14 ±10.46	0.0002

(P<0.05 statistically significant)

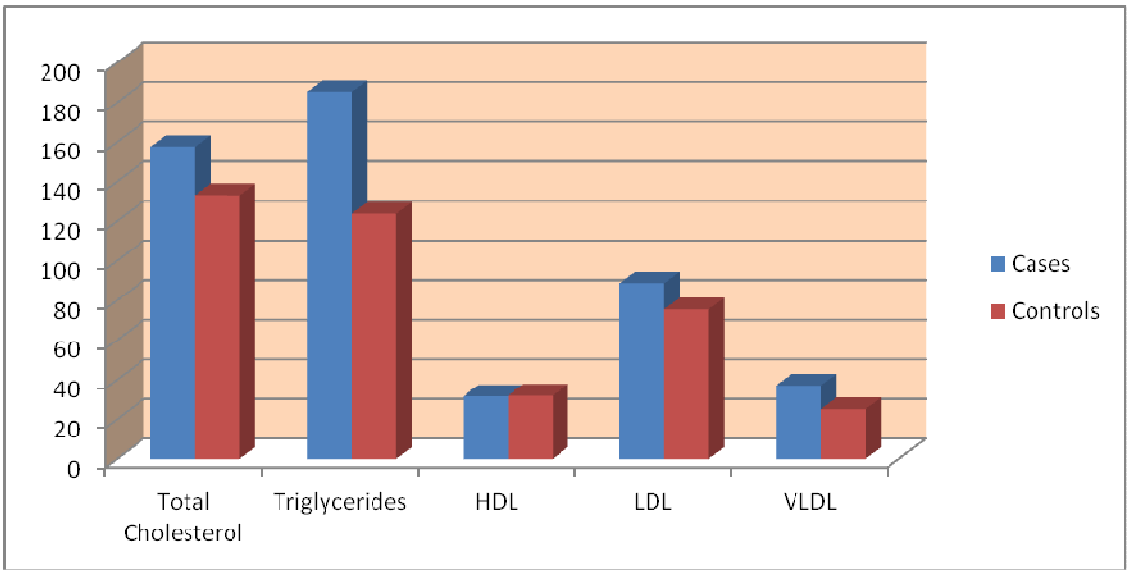

Figure 6: Figure showing comparison Lipid profiles amongst cases and controls.

Blood sugar levels in patients and controls:

DISCUSSION:

In our present study, the mean fasting blood sugar in diabetic group was 204.61 ±105.01 mg/dl and mean postprandial blood sugar was 235.13 ±114.85 mg/dl and in the controls group the fasting blood sugar level was 98.52 ±30.03 mg/dL and postprandial blood sugar level was 141.92 ±62.47 mg/dL.

Lipid Profile abnormalities:

i) Serum Cholesterol comparison: In the present study the serum cholesterol value in diabetics was 157.52 ±53.08 mg/dL and in the non-diabetics was 133.06 ±34.22 mg/dL.

ii) Serum Triglyceride comparison: In the present study the postprandial serum triglycerides in the diabetic group were 185.23 ±104.34 mg/dL and in the controls was 123.70 ±47.83 mg/dL.

iii) Serum HDL comparison: In the present study the postprandial serum HDL in the diabetic group was 31.57 ±13.49 mg/dL and in the controls was 31.90 ±11.70 mg/dL.

iv) Serum LDL comparison: In the present study the postprandial serum LDL in the diabetic group was 88.61 ±44.29 mg/dL and in the controls was 75.40 ±29.56 mg/dL.

v) Serum VLDL comparison: In the present study the postprandial serum VLDL in the diabetic group was 37.14 ±21.41 mg/dL and in the controls was 25.14 ±10.46 mg/dL.

There were significantly higher blood sugar levels in diabetics as compared to non-diabetics. There was significantly deranged cholesterol, triglycerides and VLDL in diabetics as compared to non-diabetics.

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