Journal Of Cardiovascular Disease Research
ISSN: 0975-3583,0976-2833 VOL15, ISSUE 6, 2024

Original Research

Evaluation Of Fasting And Postprandial Lipid Profile In Diabetic Patients And Healthy Subjects

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Received: 30 May 2024 Accepted: 27 June 2024

ABSTRACT
Background: Diabetes mellitus (DM) is the term used to describe a collection of metabolic diseases marked by persistently elevated blood sugar levels. The present study was conducted to assess fasting and postprandial lipid profile in diabetic patients and healthy individuals.
Materials & Methods: 74 type II diabetes patients of both genders were kept in group I and 74 healthy subjects in group II. Parameters such as HbA1c, FBS, PBS, total cholesterol (TC), triglycerides (TG), high density lipoprotein – cholesterol (HDL-C), very-low-density lipoprotein cholesterol (VLDL) and low-density lipoprotein cholesterol (LDL) were compared.
Results: Group I had 44 males and 30 females and group II had 37 males and 37 females. In group I and group II, mean HbA1c level was 9.8% and 4.7%, PBS was 185.2 mg/dl and 134.6 mg/dl, TG was 194.6 mg/dl and 152.6 mg/dl, TC was 210.3 and 172.2, HDL- C was 43.7 mg/dl and 59.4 mg/dl, VLDL was 37.9 mg/dl and 31.5 mg/dl and LDL was 168.4 mg/dl and 84.2 mg/dl. The difference was significant (P< 0.05). The mean fasting and postprandial TG value in group I was 214.2 and 248.6, TC was 182.2 and 196.1, HDL- C was 43.2 and 38.3, VLDL was 36.5 and 37.2 and LDL was 169.5 and 178.2 respectively. The mean fasting and postprandial TG value in group I was 152.6 and 180.4, TC was 172.2 and 181.2, HDL- C was 59.4 and 37.2, VLDL was 31.5 and 32.8 and LDL was 84.2 and 92.6 respectively. The difference was significant (P< 0.05).
Conclusion: Patients with type 2 diabetes had a considerably higher postprandial lipid profile compared to their fasting lipid profile.
Key words: diabetes mellitus, lipid profile, LDL

Introduction

Diabetes mellitus (DM) is the term used to describe a collection of metabolic diseases marked by persistently elevated blood sugar levels. Hyperglycemia is the result of elevated blood sugar levels brought on by insufficient insulin secretion, action, or both.[1] It might result in issues with how protein, carbs, and lipids are metabolized. Type 2 diabetes, also known as non-insulin dependent diabetes mellitus (NIDDM), is becoming more common worldwide and is particularly prevalent in developing nations like India.[2,3]

One of the main factors thought to be responsible for the elevated cardiovascular risk in type 2 diabetes is diabetic dyslipidemia. Even though fasting triglyceride (TG) levels are acceptable, postprandial hypertriglyceridemia may independently contribute to early atherosclerosis in type 2 diabetes. Faster atherosclerosis is caused by kinetic, qualitative, and quantitative lipoprotein abnormalities, all of which are present in diabetic dyslipidemia.[4] The two main quantitative anomalies are lower levels of high- density lipoprotein (HDL) and elevated TG. Increases in large, very low-density lipoprotein subfraction 1 (VLDL1) and small, dense low-density lipoproteins (LDLs) are examples of qualitative abnormalities. Glycation of apolipoproteins, elevated susceptibility of LDL to oxidation, and a rise in the TG content of LDL and HDL are further qualitative lipoprotein abnormalities.[5] The important kinetic lipoprotein abnormalities are characterized by an elevated $VLDL_1$ production, reduced VLDL catabolism, and increased HDL catabolism. LDL-cholesterol (LDL-C) levels may be normal in type 2 diabetics; however, LDL particles exhibit decreased catabolism, which contributes to atherogenesis in type 2 DM.[6] The present study was conducted to assess fasting and postprandial lipid profile in diabetic patients and healthy individuals.

Materials & Methods

The present study comprised of 74 type II diabetes patients of both genders. All patients gave their written consent for participation in the study.

Data such as name, age, gender etc. was recorded. Patients were kept in group I and 74 healthy subjects in group II. Parameters such as HbA1c, FBS, PBS, total cholesterol (TC), triglycerides (TG), high density lipoprotein – cholesterol (HDL-C), very-low-density lipoprotein cholesterol (VLDL) and low-density lipoprotein cholesterol (LDL) were compared. Data thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

Results

Table: I Distribution of patients

Groups	Group I (Type II DM)	Group II (Healthy)
M:F	44:30	37:37

Table I shows that group I had 44 males and 30 females and group II had 37 males and 37 females.

Table: II Comparison of parameters

Parameters	Group I	Group II	P value
HbA1c (%)	9.8	4.7	0.02
PBS (mg/dl)	185.2	134.6	0.02
TG (mg/dl)	194.6	152.6	0.01
TC (mg/dl)	210.3	172.2	0.05
HDL- C (mg/dl)	43.7	59.4	0.05
VLDL (mg/dl)	37.9	31.5	0.04
LDL (mg/dl)	168.4	84.2	0.01

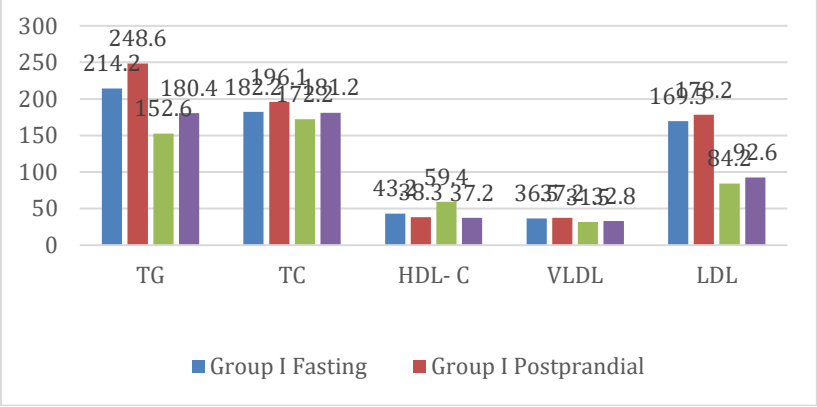
Table II shows that in group I and group II, mean HbA1c level was 9.8% and 4.7%, PBS was 185.2 mg/dl and 134.6 mg/dl, TG was 194.6 mg/dl and 152.6 mg/dl, TC was 210.3 and 172.2, HDL- C was 43.7 mg/dl and 59.4 mg/dl, VLDL was 37.9 mg/dl and 31.5 mg/dl and LDL was 168.4 mg/dl and 84.2 mg/dl. The difference was significant (P< 0.05).

Table: III Comparison of fasting and postprandial lipid profile in both groups

Parameters	Group I		Group II		P value
	Fasting	Postprandial	Fasting	Postprandial	
TG	214.2	248.6	152.6	180.4	0.02
TC	182.2	196.1	172.2	181.2	0.72
HDL- C	43.2	38.3	59.4	37.2	0.04
VLDL	36.5	37.2	31.5	32.8	0.57
LDL	169.5	178.2	84.2	92.6	0.01

Table III, graph I shows that mean fasting and postprandial TG value in group I was 214.2 and 248.6, TC was 182.2 and 196.1, HDL- C was 43.2 and 38.3, VLDL was 36.5 and 37.2 and LDL was 169.5 and 178.2 respectively. The mean fasting and postprandial TG value in group I was 152.6 and 180.4, TC was 172.2 and 181.2, HDL- C was 59.4 and 37.2, VLDL was 31.5 and 32.8 and LDL was 84.2 and 92.6 respectively. The difference was significant (P< 0.05).

Graph: I Comparison of fasting and postprandial lipid profile in both groups



Discussion

Insulin resistance, a syndrome that combines glucose intolerance, dyslipidemia, and hypertension that increases the risk of atherosclerotic vascular disease, is a hallmark of type 2 diabetes mellitus (DM).[7] It is thought that a prolonged and heightened postprandial dysmetabolism, primarily hyperglycemia and hypertriglyceridemia, which promote endothelial dysfunction and oxidative stress, is the reason of the elevated prevalence of cardiovascular impairment in type 2 diabetes. For type 2 diabetes, postprandial dyslipidemia contributes just as much to atherosclerosis consequences as fasting dyslipidemia does.[8]

One of the main factors thought to be responsible for the elevated cardiovascular risk in type 2 diabetes is diabetic dyslipidemia. Even though fasting triglyceride (TG) levels are acceptable, postprandial hypertriglyceridemia may independently contribute to early atherosclerosis in type 2 diabetes.[9] Faster atherosclerosis is caused by kinetic, qualitative, and quantitative lipoprotein abnormalities, all of which are present in diabetic dyslipidemia. The two main quantitative anomalies are lower levels of high- density lipoprotein (HDL) and elevated TG.[10] A rise in large, very low-density lipoprotein subfraction 1 (VLDL1) and small, dense low-density lipoproteins (LDLs) is one of the qualitative abnormalities. Glycation of apolipoproteins, elevated susceptibility of LDL to oxidation, and a rise in the TG content of LDL and HDL are further qualitative lipoprotein abnormalities.[11] The present study was conducted to assess fasting and postprandial lipid profile in diabetic patients and healthy individuals.

We found that group I had 44 males and 30 females and group II had 37 males and 37 females. In group I and group II, mean HbA1c level was 9.8% and 4.7%, PBS was 185.2 mg/dl and 134.6 mg/dl, TG was 194.6 mg/dl and 152.6 mg/dl, TC was 210.3 and 172.2, HDL- C was 43.7 mg/dl and 59.4 mg/dl, VLDL was 37.9 mg/dl and 31.5 mg/dl and LDL was 168.4 mg/dl and 84.2 mg/dl. Madhu et al[12] studied the postprandial lipid abnormalities in patients with type 2 diabetes mellitus. Average duration of diabetes among diabetic was 2.32 +/- 3.03 years. The body mass index (cases 25.84 +/- 4.52; controls 25.74 +/- 5.0; $p > 0.05$) and waist-hip ratio (cases 1.06 +/- 0.13; controls 1.14 +/- 0.2; $p > 0.05$) were similar in both groups. While fasting serum lipids were not significantly different between the two groups, a number of serum lipid abnormalities were noted in type 2 diabetic subjects in the postprandial state. These included a higher triglyceride-area under curve (AUC) (cases 1298.08 +/- 485.2 vs. controls 922.15 +/- 390.47 mg/dl/8h; $p=0.01$), a higher triglyceride-area under incremental curve (AUIC) (cases 549.68 +/- 382.24; control 294.75 +/- 172.6 mg/dl/8h; $p=0.01$), a higher peak triglyceride level (cases 425.2 +/- 204.47 mg%, controls 283.9 +/- 11.6.94 mg%, $p=0.01$), a lower HDL-AUC (cases 130.35 +/- 33.55 vs. controls 168.48 +/- 56.01 mg/dl/8h, $p=0.013$) and a lower HDL nadir (Cases 28.05 +/- 10.94 mg%, controls 37.13 +/- 13.52 mg%, $p < 0.02$). Triglyceride AUC correlated significantly with fasting serum triglyceride ($r=0.62$) and BMI ($r=0.7$), but not with waist hip ratio or fasting serum insulin levels. Postprandial lipaemia did not correlate with age, duration of diabetes, fasting blood glucose or glycosylated hemoglobin.

We observed that the mean fasting and postprandial TG value in group I was 214.2 and 248.6, TC was 182.2 and 196.1, HDL- C was 43.2 and 38.3, VLDL was 36.5 and 37.2 and LDL was 169.5 and 178.2 respectively. The mean fasting and postprandial TG value in group I was 152.6 and 180.4, TC was 172.2 and 181.2, HDL- C was 59.4 and 37.2, VLDL was 31.5 and 32.8 and LDL was 84.2 and 92.6 respectively. Hawa et al[13] studied the effect of fed status on serum lipid profile values in type 2 diabetes patients. A cross-sectional observational study of 110 known cases of type 2 diabetes mellitus. In study serum total cholesterol decreased 1.06% ($p > 0.05$) after 1 hour and decreased 1.65% ($p > 0.05$) after 2 hours of diet. Serum triglycerides increased 1.76% ($p < 0.01$) after 1 hour and 3.81% ($p < 0.001$) after 2 hours of diet. Serum HDL increased 0.06% ($p > 0.05$) after 1 hour and 1.86% ($p > 0.05$) after 2 hours of diet. Serum VLDL increased 1.73% ($p < 0.01$) after 1 hour and 3.80% ($p < 0.001$) after 2 hours of diet and serum LDL decreased 2.43% ($p < 0.05$) after 1 hour and 4.72% ($p < 0.01$) after 2 hours of diet[14-17].

Conclusion

The present study highlights the significant differences in fasting and postprandial lipid profiles between type II diabetes mellitus (T2DM) patients and healthy individuals. T2DM patients exhibited higher levels of HbA1c, postprandial blood sugar (PBS), triglycerides (TG), total cholesterol (TC), very-low-density lipoprotein (VLDL), and low-density lipoprotein (LDL), while having lower high-density lipoprotein (HDL) levels compared to healthy controls. These findings underscore the pronounced dyslipidemia in T2DM patients, characterized by elevated postprandial TG and LDL levels, which are significant contributors to increased cardiovascular risk. The study emphasizes the importance of monitoring and managing lipid profiles in T2DM patients, especially postprandial lipids, to mitigate the risk of atherosclerosis and related cardiovascular complications. Effective management strategies should include lifestyle modifications and pharmacological interventions aimed at improving lipid metabolism and glycemic control to reduce the burden of cardiovascular disease in T2DM patients.

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