ORIGINAL RESEARCH

To compare the fasting and postprandial lipid profile in diabetic patients

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ABSTRACT
Aim: A comparison of lipid profiles in type 2 diabetic patients while they are fasting and after they have eaten is the goal of this research. **Material and methods:** Patients with type 2 diabetes mellitus (DM) and healthy controls of similar age and gender were recruited from the outpatient and in-patient wards of the Medicine department of a large, urban academic medical centre for this case-control research. The serum sample of each subject was analyzed using a semiautomatic analyzer for the biochemical parameters: Serum TC and serum TG by enzymatic methods. Serum HDL-C by phosphotungstate precipitation, followed by enzymatic method. Serum LDL-C and VLDL-C by using Friedewald's formula. **Results:** The average number of years that the patients had been diabetic was 5.85, with a standard deviation of 1.66, and their mean HbA1C was 8.74, with a standard deviation of 1.74. In the condition of fasting, it was shown that diabetic patients had considerably higher total cholesterol and triglyceride levels, as well as LDL-C and VLDL-C levels, while control subjects had significantly lower HDL-C levels ($P<0.001$). In a similar manner, all of the parameters of lipid profile, including TC, TG, LDL-C, and VLDL-C, were considerably greater in the postprandial state in cases as compared to the controls. 32 (53.33%) diabetic subjects had hypertriglyceridemia in the fasting phase while in the postprandial phase, 45 (75%) diabetic subjects had hypertriglyceridemia. Thus, there was a significant elevation in the number of cases having hypertriglyceridemia in the postprandial state. **Conclusions:** We concluded that the patients with diabetes are more likely to be in the postprandial state than the fasting state, and the cumulative effects of hyperlipidemia and hyperglycemia in this condition contribute to the development of atherosclerosis.
Keywords: Fasting lipid profile, postprandial lipid profile, Type 2 diabetes mellitus

INTRODUCTION

Insulin resistance is the hallmark of type 2 diabetes mellitus (DM), a condition that also includes glucose intolerance, dyslipidemia, and hypertension and raises the risk of atherosclerotic vascular disease. Endothelial dysfunction and oxidative stress are thought to have a role in the higher incidence of cardiovascular impairment in type 2 DM due to a prolonged and intensified postprandial dysmetabolism, especially hyperglycemia and hypertriglyceridemia. [1] Hence, atherosclerotic problems due to type 2 DM are just as likely to be caused by postprandial dyslipidemia as they are by dyslipidemia during fasting. [2]

It is well accepted that diabetic dyslipidemia is a significant contributor to the elevated cardiovascular risk seen in people with type 2 diabetes. Nevertheless, even in the presence of normal fasting triglyceride (TG) levels, postprandial hypertriglyceridemia may independently contribute to early atherosclerosis in type 2 DM. [3] In addition to quantitative changes, diabetic dyslipidemia causes qualitative and kinetic changes in lipoproteins, all of which hasten the development of atherosclerosis. Increased triglyceride (TG) and reduced high density lipoprotein (HDL) values are the most noteworthy quantitative abnormalities [4]. An rise in tiny, dense low density lipoproteins (LDLs) and huge, extremely low-density

lipoprotein subfraction 1 are examples of qualitative abnormalities (VLDL1). Increased triglyceride (TG) content in low-density lipoprotein (LDL) and high-density lipoprotein (HDL), apolipoprotein glycation, and LDL's increased susceptibility to oxidation are all examples of other qualitative lipoprotein derangements. Increased synthesis of very low density lipoprotein 1, decreased VLDL catabolism, and enhanced HDL catabolism are the major kinetic lipoprotein abnormalities. While levels of LDL cholesterol (LDL-C) may be normal in type 2 diabetics, the impaired catabolism of LDL particles leads to atherogenesis in this disease. While the exact cause of diabetic dyslipidemia is unknown, it is thought that insulin resistance and relative insulin insufficiency are primary contributors to the condition. Insulin plays a crucial role in the control of lipid metabolism. The pathophysiology of diabetic dyslipidaemia may also include certain adipocytokines, such as retinol-binding protein 4 and adiponectin. [4]

Recent investigations have shown that postprandial hypertriglyceridemia also contribute to endothelial dysfunction and atherosclerosis, despite the fact that traditional risk assessments for atherosclerosis only include fasting TG levels. Because to the frequent meal and snack eating typical of humans, they are seldom in a fasted state and instead spend most of their time in a fed condition. Several meals may cause postprandial hypertriglyceridemia, even in those whose fasting TG levels are normal or nearly so. [5] After eating, TG levels remain increased for up to 3-4 hours in healthy people, but for 6-10 hours in those with prediabetes or diabetes. [6] As a result, the vascular endothelium is more likely to experience the negative effects of TGs postprandially than during the fasting state. As a result, the post-meal period is when endothelial damage manifests the most severely. [7] Primary care doctors may play a crucial role in lowering cardiovascular morbidity and mortality in diabetes patients by recognising and treating substantial postprandial dyslipidemia as early as possible. To successfully treat postprandial lipid abnormalities in these patients by dietary and pharmaceutical therapies, it may be prudent for primary care doctors to collect lipid profile in both fasting and postprandial state in type 2 diabetes.

The purpose of this research was to evaluate the importance of postprandial lipid profile in the cardiovascular risk stratification of type 2 DM patients by comparing their fasting and postprandial lipid profiles.

MATERIAL AND METHODS

Patients with type 2 diabetes mellitus (DM) and healthy controls of similar age and gender were recruited from the outpatient and in-patient wards of the Medicine department of a large, urban academic medical centre for this case-control research. Each participant gave his or her written informed permission after approval from the Institutional Ethics Committee.

INCLUSION CRITERIA

Over 33-year-old men and women with ADA-diagnosed type 2 DM were included in the research.

EXCLUSION CRITERIA

Participants were not included if they had a history of taking medications that alter lipid metabolism, such as lipid lowering drugs, oestrogens (oral contraceptive pills), thiazides, glucocorticoids, beta blockers, etc., or if they had type 1 diabetes, coronary artery disease, hypertension, cerebrovascular disease, liver disorders, hypothyroidism, Cushing's syndrome, nephrotic syndrome, a smoking or drinking habit, or a history.

METHODOLOGY

The research followed the guidelines laid forth in the Helsinki Declaration. All participants were informed of the study's goals and were assured of complete anonymity. Each participant had a complete medical history, physical, and any other required tests. All participants had their lipid profiles taken both before and after a meal. The participants fasted for 12 hours before giving blood, and then gave blood again 4 hours after eating. [8] Lipid profiles were taken when both the study and control groups were fed a fixed diet of around 750 kcal/m2 and 45 g fat. Four pieces of white bread were consumed together with 50 grammes of Amul butter and 250 millilitres of full cream milk. The serum sample of each subject was analyzed using a semiautomatic analyzer for the following biochemical parameters:

- Serum TC and serum TG by enzymatic methods.
- Serum HDL-C by phosphotungstate precipitation, followed by enzymatic method.
- Serum LDL-C and VLDL-C by using Friedewald's formula.[9]

Blood glucose was measured both before and after a meal, as was glycosylated haemoglobin (HbA1C), renal function, liver function, an electrocardiogram (ECG), and a regular check of the patient's urine. The criteria for the diagnosis of dyslipidemia were established by the National Cholesterol Education Program Adult Treatment Panel III (NCEP-ATP III). [10]

STATISTICAL ANALYSIS

Information was put into Microsoft Excel spreadsheets and double-checked for mistakes. For the quantitative data, we used means and standard deviations, whereas for the qualitative data, we used straightforward percentages. We used normality tests to make sure our interval and ratio data were distributed normally. For normally distributed data, the t-test was used to compare two groups and the ANOVA test was used to compare more than two groups. When the P-value for an observation was less

than 0.05 (P 0.05), we regarded it to be statistically significant.

RESULTS

The average age of the participants in this research was found to be 50.85 years, with a standard deviation of 6.39 years. The mean age of the controls was found to be 50.99 years, with a standard deviation of 5.85 years. Cases (those with diabetes) had a higher body mass index (BMI) in comparison to the controls (29.01±2.97 kg/m2 vs. 24.15±2.14 kg/m2, P <0.001) and a higher waist to hip ratio (WHR) in comparison to the controls (0.98±0.11 in men, 0.99±0.14 in females vs. 0.89±0.05 in males, 0.88±0.05 The average number of years that the patients had been diabetic was 5.85, with a standard deviation of 1.66, and their mean HbA1C was 8.74, with a standard deviation of 1.74. [Table 1]

Table 1: Basic parameter

Parameters	Cases	Controls	*P*
Age (years)	50.85± 6.39	50.99 ± 5.85	0.45
Duration of Diabetes (years)	5.85 ± 1.66	-	-
BMI (Kg/m^2)	29.01 ± 2.97	24.15 ± 2.14	0.001
Waist to hip ratio			
Males	0.98 ± 0.11	0.99 ± 0.14	0.001
Females	0.89 ± 0.05	0.88 ± 0.05	0.001
HbA1C (gm/dl)	8.74 ± 1.74	4.98±0.44	0.001

The lipid profiles of the patients and the controls when they were fasting are shown in Table 2. In the condition of fasting, it was shown that diabetic patients had considerably higher total cholesterol and triglyceride levels, as well as LDL-C and VLDL-C levels, while control subjects had significantly lower HDL-C levels (P<0.001). In a similar manner, all of the parameters of lipid profile, including TC, TG, LDL-C, and VLDL-C, were considerably greater in the postprandial state in cases as compared to the controls [Table 3]. HDL, on the other hand, was significantly lower in the postprandial state in cases.

Table 2: Fasting lipid profile

Parameters	Cases	Controls	*P*
Fasting total cholesterol (mg/dl)	199.98±25.63	180.36±9.87	0.001
Fasting triglycerides (mg/dl)	169.81±25.37	99.85±7.81	0.001
Fasting HDL Cholesterol (mg/dl)	41.25±6.63	51.22±7.58	0.001
Fasting LDL cholesterol (mg/dl)	127.55±12.22	111.20±11.29	0.001
Fasting VLDL cholesterol (mg/dl)	38.01±8.88	20.02±3.36	0.001

Table 3: Postprandial lipid profile

Parameters	Cases	Controls	P
Postprandial total cholesterol (mg/dl)	224.29±15.63	186.37±14.36	0.001
Postprandial Triglycerides (mg/dl)	237±22.58	101.58±6.31	0.001
Postprandial HDL-cholesterol (mg/dl)	39.85±5.55	49.27±6.71	0.001
Postprandial LDL-cholesterol (mg/dl)	141.22±12.27	109.58±9.63	0.001
Postprandial VLDL-cholesterol (mg/dl)	47.14±11.25	21.57±2.03	0.001

As compared to their respective fasting values, the postprandial levels of TC and TG in diabetics (cases) were considerably greater than their respective fasting values (P< 0.001). The postprandial levels of LDL-C and VLDL-C in diabetics rose from their respective fasting values, however the rise did not reach statistical significance (P = 0.066, P = 0.33). In contrast, HDL-C levels dropped in the postprandial state compared to their values in the fasting state in certain patients; however, the observed drop did not meet the criteria for statistical significance (P = 0.74 [Table 4]).

Table 4: Comparison of fasting and postprandial lipid profile in diabetics

Serum lipid profile	Fasting	Postprandial	P
Total Cholesterol (mg/dl)	199.98±25.63	224.29±15.63	0.001
Triglycerides (mg/dl)	169.81±25.37	237±22.58	0.001
HDL Cholesterol (mg/dl)	41.25±6.63	39.85±5.55	0.74
LDL Cholesterol (mg/dl)	127.55±12.22	141.22±12.27	0.066
VLDL Cholesterol (mg/dl)	38.01±8.88	47.14±11.25	0.33

While there was a rise in total cholesterol, total fat, low-density lipoprotein cholesterol, and very low-density lipoprotein cholesterol levels in the controls, the increases in TC, TG, LDL-C, and VLDL-C levels, as well as HDL-C, were not statistically significant [Table 5].

Table 5: Comparison of fasting and postprandial lipid

Serum lipid profile in controls	Fasting	Postprandial	*P*
Total Cholesterol (mg/dl)	180.36±9.87	186.37±14.36	0.07
Triglycerides (mg/dl)	99.85±7.81	101.58±6.31	0.052
HDL Cholesterol (mg/dl)	51.22±7.58	49.27±6.71	0.36
LDL Cholesterol (mg/dl)	111.20±11.29	109.58±9.63	0.47
VLDL Cholesterol (mg/dl)	20.02±3.36	21.57±2.03	0.22

32 (53.33%) diabetic subjects had hypertriglyceridemia in the fasting phase while in the postprandial phase, 45 (75%) diabetic subjects had hypertriglyceridemia. Thus, there was a significant elevation in the number of cases having hypertriglyceridemia in the postprandial state.

DISCUSSION

The participants in this research had a mean age of 50.85 years, with a standard deviation of 6.39 years, and 50% of them were male. There were sixty healthy controls that were of the same age and gender. The cases had a greater body mass index and waist-to-hip ratio than the controls did. WHR was also found to be higher in cases of type 2 diabetes than in the controls in a previous study that was conducted by Punekar et al. (0.98± 0.11 in males, 0.99± 0.14 in females vs. 0.89± 0.05 in males, 0.88± 0.05 in females, P< 0.001) the WHR was also found to be higher in cases of type 2 diabetes than in the controls. [11] Studies conducted by Lokhande et al.[2] and Puthenveedu et al. likewise came to comparable conclusions about the topic at hand. [12] The patients had an average duration of diabetes of 5.85 years (standard deviation: 1.66) and their mean HbA1C was 8.74 (standard deviation: 1.74) g/dl. In a research that was quite comparable to this one, the mean HbA1C level of the diabetes patients was 8.70±1.50, while the level of the controls was 6.58±1.10. [11] According to the findings of this research, both fasting and postprandial lipid parameters (TC, TG, LDL-C, and VLDL-C) were higher in cases by statistically significant margins when compared to the controls. On the other hand, HDL-C was significantly lower in both the fasting and postprandial phases in the diabetic cases. Previous research have produced conclusions that were comparable to this one. [2,11,12,13]

Increased insulin resistance is one of the causes of type 2 diabetes, which leads to an increase in the flow of free fatty acids (FFA) from adipocytes. This causes an increase in the supply of FFA to the liver, which in turn leads to an increase in the production of lipids (both VLDL and TGs) inside the hepatocytes. This factor, together with impaired lipoprotein clearance in the liver, plays a significant part in the pathogenesis of dyslipidemia that is associated with type 2 diabetes. It is well known that diabetes may cause dyslipidemia, which can then lead to atherogenesis and macrovascular disease. [14,15] The postprandial state is associated with a further deterioration of diabetic dyslipidemia, which is due to the additively harmful impact of postprandial hyperglycemia. Postprandial accumulation of triglyceride-rich lipoproteins promotes the creation of small dense LDLs, which are essential contributors to the development of oxidative stress, inflammation, and endothelial dysfunction. Triglyceride-rich lipoproteins accumulate postprandially. In diabetic individuals, all of these factors contribute to a faster progression of atherosclerosis, which ultimately results in macrovascular problems. [15,16]

In this particular investigation, the postprandial levels of total cholesterol and triglyceride (TG) in diabetics (cases) were considerably higher than their respective fasting values (P 0.001). In diabetics, the levels of postprandial LDL-C and VLDL-C rose from their fasting values; however, the rise was not statistically significant. In some instances, the HDL-C level was lower in the postprandial state when compared to its value in the fasting state; nevertheless, the drop did not meet the criteria for statistical significance. On the other hand, in the postprandial state among the controls, we did not find any significant alterations in any of the lipid measures. Postprandial lipid parameters significantly increased from their fasting levels in type 2 diabetics, according to research conducted by Lokhande SL, Raghavendra S et al., and Wali VV et al. [2, 17, 18]. On the other hand, postprandial HDL significantly decreased in comparison to the fasting HDL level. According to the findings of another research, blood levels of TC and LDL-C dropped after eating (P values more than 0.05), but levels of TG and VLDL rose significantly (P values less than 0.001). Even though there was an increase in serum HDL (P > 0.05), the rise was not significant according to the statistics. [19]

The number of diabetic subjects who had hypertriglyceridemia increased from 32 (53.33%) diabetic subjects having hypertriglyceridemia in the fasting phase to 45 (75%) diabetic subjects having hypertriglyceridemia in the postprandial phase, according to the findings of this study. This increase occurred between the fasting phase and the postprandial phase. As a result, there was a discernible increase in the total number of patients that presented with hypertriglyceridemia in the postprandial stage.

As a result, the current research sheds insight on an altered postprandial response of blood lipids, particularly triglycerides, in type 2 diabetics after they ate a meal that included oral fat. Other investigations have also found an extreme case of post-meal hypertriglyceridemia in diabetic patients. [16,20,-23] In addition, it has been shown that type 2 diabetics who have postprandial hypertriglyceridemia while having normal fasting levels of triglycerides are at an

increased risk for developing early atherosclerosis. [24]

Postprandial dyslipidemia has been shown to play a significant part in the formation of atherosclerotic plaques, which in turn leads to an increased risk of cardiovascular events, as was proven in previous research.[25] Insulin resistance in type 2 diabetes patients may be linked to a greater prevalence of cardiovascular illnesses in these individuals because postprandial dysmetabolism, which may include postprandial dyslipidemia, is connected with endothelial dysfunction and oxidative stress. [26,27] The length of time postprandial lipidaemia persists for and the severity of postprandial lipidaemia are both closely associated to the aetiology and progression of cardiovascular illnesses, according to previous research. [28] In addition, the need that patients fast before undergoing blood sample for lipid profile analysis might make the process more difficult than it has to be for a great number of patients all over the globe, particularly diabetics. According to the data made above, determining lipid levels in the postprandial period is just as significant as determining lipid levels during the fasting phase in patients with type 2 diabetes. In point of fact, it has been hypothesised that the postprandial lipid profile may be a more accurate indication of an abnormal lipid metabolism and, as a result, of cardiovascular disease in people with type 2 diabetes.

CONCLUSIONS

Significant postprandial metabolic abnormalities, in particular postprandial dyslipidemia, are seen in individuals with type 2 diabetes. Patients with diabetes are more likely to be in the postprandial state than the fasting state, and the cumulative effects of hyperlipidemia and hyperglycemia in this condition contribute to the development of atherosclerosis. The development and progression of cardiovascular illnesses are linked to the length and severity of postprandial lipidemia. This work adds to the growing body of evidence that estimates of postprandial lipid profiles are just as useful as estimates of fasting lipid profiles for determining cardiovascular risk stratification among people with type 2 diabetes. Reducing the risk of atherosclerotic vascular disease and its implications requires prompt diagnosis and treatment of substantial postprandial dyslipidemia in diabetics.

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