Section: Cardiology

Original Research Article

POSTPRANDIAL HYPERTRIGLYCERIDEMIA - A HIDDEN ELEPHANT IN ATHEROSCLEROSIS

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Abstract

Background: Postprandial hypertriglyceridemia contributes to atherosclerosis, as most of the day is postprandial, and fasting lipid profiles miss these variations. Routine postprandial lipid profiling is essential for early detection of cardiovascular risk in patients with type 2 Diabetes Mellitus. This study aimed to determine the prevalence of postprandial hypertriglyceridemia in patients with type 2 diabetes and to compare fasting and postprandial lipid levels between patients and controls, correlating them with postprandial glucose levels. **Materials and Methods:** This cross-sectional study included 160 participants, 80 patients with type 2 Diabetes Mellitus, and 80 controls, and was conducted in the Department of Biochemistry and Diabetology. Fasting and postprandial blood sugar and lipid profiles were measured, with postprandial lipid level cutoff values determined based on guidelines from the European Atherosclerosis Society and the European Federation of Clinical Chemistry and Laboratory Medicine. **Result:** Patients and age-matched controls, postprandial hypertriglyceridemia were in 83.75% of patients and 78.75% of controls. Significant differences in fasting and postprandial blood glucose levels ($p<0.001$) but no significant differences in fasting and postprandial lipid profiles were observed. Postprandial HDL ($p=0.64$), fasting total cholesterol ($p=0.59$), fasting triglycerides ($p=0.75$), fasting HDL ($p=0.15$), postprandial total cholesterol ($p=0.26$), and postprandial triglycerides ($p=0.61$) levels were not significant. **Conclusion:** This study found higher fasting and postprandial triglyceride levels in patients than in controls, highlighting the need for routine postprandial lipid monitoring to detect dyslipidaemia early. Treating hypertriglyceridemia with statins and fenofibrate may reduce the risk of diabetes, hypertension, and coronary artery disease in patients and controls.

INTRODUCTION

Diabetes mellitus is a metabolic and vascular syndrome of multiple aetiology characterized by a chronic hyperglycaemic state due to deficiency of insulin secretion or insulin resistance or both interfering with the metabolism of glucose, proteins, and lipids leading to complications including micro-angiopathy & macro-angiopathy.[1] According to WHO, about 422 million people have diabetes worldwide, most of the diabetics living in middle-income countries. South Asian populations are more prone to acquire type 2 Diabetes mellitus, which has an increasing incidence throughout the world, and 1.5 million deaths are directly attributed to diabetes each year.[2]

Insulin resistance or deficiency alters key enzymes and pathways in lipid metabolism, leading to lipid abnormalities in DM.[3] The term 'DIABETIC DYSLIPIDEMIA' comprises a triad of increased triglycerides (TGL), decreased high-density lipoprotein cholesterol (HDL-C), and excess of small, dense low-density lipoprotein cholesterol (LDL-C) particles. The dyslipidaemia in type 2 DM is different than in non-diabetics, as it has been proposed that the composition of lipid particles in diabetic dyslipidaemia is more atherogenic than other types of dyslipidemia.[4] Epidemiological studies, such as DECODE (Diabetic Epidemiology: Collaborative Analysis of Diagnostic Criteria in Europe), reported that elevated postprandial blood glucose is strongly associated with increasing incidence of cardiovascular diseases.[5] The high cardiovascular mortality associated with type 2 Diabetes Mellitus is due to prolonged, exaggerated, postprandial dyslipidemia.[6,7]

Prolonged and exaggerated postprandial hypertriglyceridemia and associated oxidative stress have been linked to macrovascular diseases in both regular and hypotriglyceridaemic patients with type 2 Diabetes Mellitus. Hypertriglyceridemia is attributable to abnormalities in the synthesis and

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catabolism of triglyceride-rich lipoproteins (TGL), such as very low-density lipoprotein (VLDL) and chylomicrons.[8] Under normal conditions, insulin controls the intracellular degradation of apoB100. In the insulin resistance state, overproduction of apoB-100-VLDL occurs, resulting in hypertriglyceridemia, which is related indirectly to atherosclerotic progression through an increase in small dense LDL and lowered HDL.[9]

Many studies have been conducted on dyslipidaemia in type 2 DM; however, most have included only fasting lipid levels. Very few studies have included postprandial lipid levels in patients with type 2 DM. Hence, the present study aimed to compare postprandial lipid levels with fasting lipid levels in individuals with type 2 diabetes mellitus, and their correlation with postprandial glucose levels.

Aim

This study aimed to determine the prevalence of postprandial hypertriglyceridemia in patients with type 2 diabetes mellitus and to compare fasting and postprandial lipid levels in both patients and controls, correlating with their postprandial glucose levels.

MATERIALS AND METHODS

This cross-sectional study included 160 study patients, including 80 patients with type 2 diabetes mellitus and 80 controls, who were subjected to estimated fasting and postprandial blood sugar and lipid profile levels in the Department of Biochemistry and Department of Diabetology at Government Stanley Medical College and Hospital, Chennai between 2 Months (September 2022 and October 2022). The Institutional Ethics Committee approved this study before initiation, and informed consent was obtained from all patients.

Inclusion criteria

Patients with type 2 diabetes, aged 30–60 years, who had a condition for < 5 years and were taking oral diabetes medications were included.

Exclusion criteria

Patients with a history of hypertension; kidney, liver, or heart disorders; smokers; alcoholics; those with type 1 diabetes; a family history of abnormal cholesterol levels; or those taking steroids, medication for cholesterol issues, insulin therapy, or gestational diabetes were excluded.

Methods: Relevant data were collected using a validated semi-structured format to ensure patient confidentiality. Personal details and a brief history of present and past illnesses were recorded, followed by detailed clinical examination. Venous blood (5 mL) was collected under sterile conditions, and serum was separated for the analysis of fasting and postprandial glucose levels and lipid parameters. Blood collection followed specific criteria: fasting glucose and lipid profiles were assessed after 12 h of fasting, postprandial glucose was measured 2 h post-meal, and postprandial lipid profiles were measured 3 h post-meal following a diet of staple food for two days. The European Atherosclerosis Society and the European Federation of Clinical Chemistry and Laboratory Medicine referenced cutoff values for postprandial lipid levels. Biochemical analyses, including fasting and postprandial glucose, total cholesterol, triglycerides, and high-density lipoproteins, were performed using a random-access, fully automated chemistry analyser.

Statistical analysis: Statistical analyses were performed using the SPSS software version 16.0. For variables with normal distribution, data were expressed as mean ± standard deviation (SD) or standard error of the mean (SEM). Group comparisons were conducted using an independent samples t-test for equality of means. Homogeneity of variances was assessed using the Levene's test. Statistical significance was set at $p < 0.05$.

RESULTS

In our study, 16% of the patients were aged 30-40 years, 56% were aged 41-50 years, and 100% were aged 51-60 years. This study examined the relationship between fasting and postprandial lipid profiles in patients with type 2 diabetes and controls, showing a higher occurrence of increased postprandial triglycerides than fasting triglycerides. The average fasting blood sugar, postprandial blood sugar, fasting triglyceride, and postprandial triglyceride levels were 181.84, 276.40, 239.39, and 262.53, respectively. For the controls, the averages were 80.33, 111.96, 235.25, and 254.44, respectively. The prevalence of postprandial hypertriglyceridemia was 83.75% and 78.75% in the patient and control groups, respectively.

Table 1: Comparison of biochemical parameters between groups.

	Mean±SD		P-value
	Control	Case	
PPHDL	37.24±6.681	37.81±8.673	0.639
FBS	80.33±7.729	181.84±54.22	<0.0001
PPBS	111.96±9.765	276.4±78.593	<0.0001
TC	208.09±45.259	204.01±48.645	0.584
TGL	235.25±92.182	239.39±89.634	0.774
HDL	34.58±7.575	36.35±7.973	0.151
PPTC	203.26±44.695	195.11±46.306	0.259
PPTGL	254.44±99.612	262.53±101.522	0.612

Statistical analysis using Levene's test for equality of variance showed significant differences in postprandial HDL (p=0.004), fasting blood sugar (p=0.000), and postprandial blood sugar (p=0.000).

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The t-test for equality of means showed significant differences in fasting blood sugar (p=0.000) and postprandial blood sugar (p=0.000), whereas postprandial HDL was not substantial (p=0.64).
The comparison of fasting and postprandial lipid profiles between patients with type 2 diabetes and age-matched controls revealed no significant differences in other variables, including fasting total cholesterol, fasting triglycerides, fasting HDL, postprandial total cholesterol, and postprandial triglycerides. However, the fasting and postprandial blood sugar levels were significantly different (p=0.000) [Table 1].

DISCUSSION

In our study, the prevalence of postprandial hypertriglyceridemia was higher in the patients than in the controls, although the difference was smaller than expected. This finding highlights that dyslipidaemia is a significant concern, both with and without diabetes, as even the controls showed elevated lipid levels. This suggests that diabetes may worsen dyslipidaemia or vice versa.
We could not achieve statistical significance for postprandial hypertriglyceridemia between patients and controls, likely because of several factors. Key contributors included prolonged postprandial states caused by eating habits in this demographic, absence of a standardized reference meal, and inconsistent timelines for postprandial sample collection. Among these, prolonged postprandial states due to cultural shifts, irregular meal timings, and lifestyle patterns appear to be the most plausible explanations. Frequent late-night eating and irregular meal schedules lead to repeated postprandial glycaemic spikes, accelerating the onset of dyslipidaemia.
Although this study yielded a null result, the data underscores the importance of postprandial dyslipidaemia, particularly postprandial hypertriglyceridemia, as a reliable predictor of cardiovascular risk. This is particularly relevant in modern eating patterns that involve frequent meals throughout the day.

Limitation of the study

Limitations include a small sample size, lack of a standardized reference meal, and inconsistent timing for postprandial sample collection, making it difficult to establish causation. Future research should address these factors to better understand the relationship between postprandial dyslipidaemia and cardiovascular risk.

CONCLUSION

In postprandial dyslipidaemia, the chylomicron remnant concentration is elevated compared to the fasting state. Atherosclerosis is a process that is active during the postprandial state because of our prolonged postprandial surge in triglyceride and glucose levels due to our 3+2 meal pattern. Biochemical investigations, such as lipid profile and glucose, are simple and economical tests for lower socioeconomic diabetic patients to predict their risk and prevent them from acquiring cardiovascular disease at an earlier stage.
The fasting lipid profile is a mandatory screening test for patients with type 2 DM. A postprandial lipid profile is advisable as a mandatory screening test for patients with type 2 DM and normal individuals, as most individuals are in a postprandial state throughout the day. Potential pharmacological interventions can be made to treat early onset type 2 diabetes mellitus patients to protect them from cardiovascular complications and dyslipidaemia, preventing them from acquiring further complications.

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