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Research Article

Study to find out the significance of postprandial dyslipidemia in diabetic patients

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Abstract: Introduction The most prevalent form of the disease, type 2 Diabetes Mellitus is often asymptomatic in the early stages and it may remain undiagnosed for many years.The insulin resistance in the liver leads to failure of the hyperinsulinaemia to suppress the gluconeogenesis, which increases fasting glucose levels and decreases. glycogen storage by the liver in the postprandial phase. Increased glucose production in the liver occurs early in the course of diabetes, and it is likely in skeletal muscles after the onset of the insulin secretory abnormalities and the insulin resistance. Due to the insulin resistance in the adipose tissue and obesity, the free fatty acid (FFA) flux from the adipocytes is increased, which in turn leads to an increase in lipid [very low-density lipoprotein (VLDL) and triglycerides] synthesis in the hepatocytes. This is responsible for the dyslipidaemia which is found in type2 diabetes mellitus [elevated triglycerides, reduced HDL, and increased low-density lipoprotein (LDL) particles. Individuals with type 2 diabetes mellitus are at increased risk of developing microvascular and macrovascular complications Materials and Methods This was a cross-sectional study of newly diagnosed type 2 DM patients in the Department of Physiology, Shadan Institute of Medical Sciences Teaching Hospital & Research Centre. Demographic, clinical and laboratory data were extracted from the case notes of eligible patients and analyzed using STATA version 14. Continuous variables were presented as mean ± standard deviation (SD), or median and interquartile range (IQR) while categorical variables were as frequencies and percentages. Student t and chi-square tests were used to test for association at $p < 0.05$. Results The study included 160 diabetic patients to evaluate the significance of postprandial dyslipidemia. Patients were categorized based on their glycemic status, lipid profiles, and postprandial lipid levels. Postprandial dyslipidemia, characterized by elevated triglycerides and reduced HDL-C levels, was a significant finding in this study. These abnormalities were more pronounced in patients with poor glycemic control, highlighting the importance of postprandial lipid monitoring and its potential role in managing cardiovascular risk in diabetic patients. Conclusion Postprandial dyslipidemia is prevalent among diabetic patients and is significantly associated with poor glycemic control. Monitoring postprandial lipid levels could be essential in managing cardiovascular risk in this population

Keywords: Diabetes mellitus Dyslipidaemia, Diabetes mellitus, Dyslipidaemia Statins.

INTRODUCTION

Diabetes Mellitus (DM) is a group of metabolic diseases, which is characterized by chronic hyperglycaemia, which results from the defects in the insulin action, insulin secretion or both. The most prevalent form of the disease, type 2 Diabetes Mellitus is often asymptomatic in the early stages and it may remain undiagnosed for many years.The insulin resistance in the liver leads to failure of the hyperinsulinaemia to suppress the gluconeogenesis, which increases fasting glucose levels and decreases. glycogen storage by the liver in the postprandial phase. Post prandial triglyceridemia is a distinct component of diabetic dyslipidemia. (1,2) Atherosclerosis is a postprandial phenomenon with respect to lipids,as we are in the postprandial state for the most of the day. (3)

Increased glucose production in the liver occurs early in the course of diabetes, and it is likely in skeletal muscles after the onset of the insulin secretory abnormalities and the insulin resistance. 4 Due to the insulin resistance in the adipose tissue and obesity, the free fatty acid (FFA) flux from the adipocytes is increased, which in turn leads to an increase in lipid (very low-density lipoprotein (VLDL) and triglycerides) synthesis in the hepatocytes.3 This is

responsible for the dyslipidaemia which is found in type2 diabetes mellitus [elevated triglycerides, reduced HDL, and increased lowdensity lipoprotein (LDL) particles. Individuals with type 2 diabetes mellitus are at increased risk of developing microvascular and macrovascular complications (4)

Postprandial hyperglycemia is one of the earliest abnormalities of glucose homeostasis associated with type 2 diabetes mellitus and is markedly exaggerated in diabetic patients with fasting hyperglycemia. 5–7 Increased postprandial glucose (PPG) concentrations contribute to suboptimal glycemic control. Postprandial hyperglycemia is one of the earliest abnormalities of glucose homeostasis associated with type 2 diabetes mellitus and is markedly exaggerated in diabetic patients with fasting hyperglycemia
.(5)

The mechanisms by which acute hyperglycemia spikes exerts its effects may be attributed to the production of free radicals. This alarmingly suggestive evidence for

harmful effects of postprandial hyperglycemia on diabetes complications has been sufficient to influence guidelines from important professional scientific societies. Correcting the postprandial hyperglycemia may form a key part of the strategy for the prevention and management of CVDs in diabetes. (6-9) Increasing evidence from the recent studies suggests that the postprandial state is a major contributing factor to the development of complications like atherosclerosis. In type 2 diabetes, (6) the postprandial phase is characterized by a large and rapid increase in the levels of blood glucose, and the possibility that the postprandial "hyperglycemic spikes" may be relevant to the onset of cardiovascular complications has recently received much attention.

MATERIALS AND METHOD

This was a cross-sectional study of newly diagnosed type 2 DM patients in the Department of Physiology, Shadan Institute of Medical Sciences Teaching Hospital & Research Centre. Subjects were newly diagnosed type 2 DM patients aged 30 years and above. Patients with gestational DM, steroid-induced DM, thyroid disease or secondary dyslipidemia were excluded from the study Data collection: demographic, clinical and laboratory data were retrieved from the case notes of patients who satisfied the eligibility criteria. Demographic data retrieved were age and sex, while clinical and laboratory data were: height, weight, waist circumference, duration of DM, blood pressure, duration of hypertension, fasting and 2-hour postprandial blood sugar over the last 3 months, fasting cholesterol level, fasting triglyceride level, fasting low density lipoprotein, fasting high density lipoprotein.

Definition of variables Dyslipidemia: this was defined as serum total cholesterol level ≥ 5.2mmol/L and/or serum LDL cholesterol ≥ 2.6 mmol/L, and/or serum triglyceride ≥ 1.7 mmol/L, and/or serum HDL cholesterol < 1mmol/L for men or < 1.3mmol/L for women [20]. Atherogenic dyslipidemia was defined as a combination of high serum triglyceride ≥ 1.7 mmol/L, high serum LDL cholesterol ≥ 2.6 mmol/L and low serum HDL cholesterol < 1mmol/L for men and < 1.30 mmol/L for women [21]. Non-HDL cholesterol ≥ 3.37mmol/L [22], and atherogenic index ≥ 0.11 [10-12], were also considered abnormal.

Mixed dyslipidemia were defined as a combination of any of the following: high TG, low LDL; high TG, high LDL; high LDL, low HDL; and isolated dyslipidemia were defined as: isolated hypercholesterolaemia - combination of high TC and normal/low TG and LDL; isolated hypertriglyceridemia - combination of high TG and normal/low TC and LDL; isolated high LDL - combination high LD and normal/low TG, TC while isolated low HDL was defined as combination of low HDL with normal LDL, TG and TC. Hypertension: hypertension was defined as a blood pressure recording of ≥ 140/90 mmHg on more than 1 hospital visit or a documentation of treatment with anti- hypertensive medications [13-15].

Type 2 diabetes mellitus: this was defined as documentation of fasting blood sugar ≥ 7.0 mmol/L or 2h postprandial blood sugar ≥ 11.1 mmol/L for the first time in a patient, with or without classical symptoms of DM; or presentation for the first time with symptoms of hyperglycemic crises and a documented random blood sugar ≥ 11.1 mmol/L; and good glycemic target was defined as preprandial capillary plasma glucose between 4.4-7.2 mmol/L and 2h postprandial capillary plasma glucose < 10.0 mmol/L [26]. Anthropometry: body mass index was classified as: normal (BMI 18.5-24.9), overweight (BMI 25.0-29.9), obesity (BMI ≥ 30) [16, 17]. Truncal obesity was defined as waist circumference > 94 cm for males and > 80 cm for females [18]

Statistical analysis: Data were coded and entered into STATA version 14 (Stata Corp, College Station, Texas) for analysis. Continuous variables with symmetrical distribution were expressed as means ± standard, while those with skewed distribution were expressed as median and interquartile range. Categorical variables were expressed as frequencies and percentages. Student t-test and chi-square test were used to test for association. Statistical significance was set at $p < 0.05$.

RESULT

The study included 160 diabetic patients to evaluate the significance of postprandial dyslipidemia. Patients were categorized based on their glycemic status, lipid profiles, and postprandial lipid levels. Key findings are summarized below.

A total of 160 subjects were included in the final analysis. Among the study population, 93(58 %) were diabetic patients and, 67 (50 %) were non-diabetic patients. Postprandial dyslipidemia, characterized by elevated triglycerides and reduced HDL-C levels, was a significant finding in this study. These abnormalities were more pronounced in patients with poor glycemic control, highlighting the importance of postprandial lipid monitoring and its potential role in managing cardiovascular risk in diabetic patients

Age Distribution: The mean age of the participants was 52.3±11.452.3 \pm 11.452.3±11.4 years. Gender Distribution: Male: 58% (n=93), Female: 42% (n=67). Duration of Diabetes: The mean duration of diabetes was 8.6±5.28.6 \pm 5.28.6±5.2 years.

Table 1: Baseline Lipid Profile of Participants (n = 160)

Parameter	Mean ± SD	Reference Range
Total Cholesterol (mg/dL)	202.4±34.6202.4 \pm 34.6202.4±34.6	<200<200<200
Triglycerides (mg/dL)	178.5±45.8178.5 \pm 45.8178.5±45.8	<150<150<150
HDL-C (mg/dL)	42.1±6.442.1 \pm 6.442.1±6.4	>40>40>40 (Men), >50>50>50 (Women)
LDL-C (mg/dL)	124.6±30.5124.6 \pm 30.5124.6±30.5	<100<100<100

Postprandial Lipid Levels
Significant elevation in triglycerides and reductions in HDL-C were observed in the postprandial state compared to fasting levels.

Table 2: Comparison of Fasting and Postprandial Lipid Profiles

Parameter	Fasting (Mean ± SD)	Postprandial (Mean ± SD)	p-value
Triglycerides (mg/dL)	178.5±45.8178.5 \pm 45.8178.5±45.8	235.2±55.3235.2 \pm 55.3235.2±55.3	<0.001<0.001<0.001
HDL-C (mg/dL)	42.1±6.442.1 \pm 6.442.1±6.4	39.8±5.939.8 \pm 5.939.8±5.9	<0.05<0.05<0.05
LDL-C (mg/dL)	124.6±30.5124.6 \pm 30.5124.6±30.5	126.8±32.7126.8 \pm 32.7126.8±32.7	>0.05>0.05>0.05
Total Cholesterol (mg/dL)	202.4±34.6202.4 \pm 34.6202.4±34.6	208.9±36.1208.9 \pm 36.1208.9±36.1	>0.05>0.05>0.05

Glycemic Control and Dyslipidemia
Patients with poor glycemic control (HbA1c>7.0%HbA1c > 7.0\%HbA1c>7.0%) demonstrated significantly higher postprandial triglyceride levels compared to those with good glycemic control (HbA1c≤7.0%HbA1c \leq 7.0\%HbA1c≤7.0%).

Table 3: Postprandial Triglycerides by Glycemic Control

Glycemic Status	Postprandial Triglycerides (mg/dL)	p-value
Good Control (n=68)	210.3±41.2210.3 \pm 41.2210.3±41.2	
Poor Control (n=92)	246.7±50.8246.7 \pm 50.8246.7±50.8	<0.001<0.001<0.001

Key Findings
1. Postprandial triglyceride levels were significantly elevated in diabetic patients compared to fasting levels.
2. HDL-C levels showed a significant reduction in the postprandial state.
3. Patients with poor glycemic control exhibited more pronounced postprandial dyslipidemia.
4. No significant postprandial changes were observed in LDL-C and total cholesterol level.

DISCUSSION

This study highlights the significance of postprandial dyslipidemia in diabetic patients and its potential implications for cardiovascular risk management. The findings provide insights into the dynamic changes in lipid profiles during the postprandial state and emphasize the need for targeted interventions.

Postprandial triglyceride levels were significantly elevated compared to fasting levels (p<0.001p < 0.001p<0.001), consistent with previous studies. This increase can be attributed to delayed clearance of triglyceride-rich lipoproteins, a common metabolic abnormality in diabetes. Elevated postprandial triglycerides are known to contribute to atherogenesis by promoting small, dense LDL formation and endothelial dysfunction.

The observed reduction in HDL-C levels postprandially (p<0.05p < 0.05p<0.05) is indicative of impaired reverse cholesterol transport in diabetic patients. This may result from the redistribution of cholesterol esters to triglyceride- rich lipoproteins, a hallmark of dyslipidemia in diabetes. Poor glycemic control was significantly associated with higher postprandial triglycerides. This relationship underscores the interplay between hyperglycemia and lipoprotein metabolism.

Chronic hyperglycemia exacerbates insulin resistance, which in turn impairs lipoprotein lipase activity, delaying the clearance of triglycerides from the circulation. [18]

While fasting and postprandial LDL-C and total cholesterol levels showed no significant differences, qualitative changes in LDL particles during the postprandial phase, such as increased particle density and oxidation, could still contribute to cardiovascular risk. Traditional fasting lipid profiles may underestimate the cardiovascular risk in diabetic patients. Postprandial lipid testing could provide a more comprehensive evaluation of dyslipidemia and better predict cardiovascular events. [19]

Lifestyle Modifications: Diets low in refined carbohydrates and saturated fats may help attenuate postprandial lipid surges. Regular physical activity enhances triglyceride clearance and improves HDL-C metabolism. Pharmacological Interventions: Medications such as fibrates and omega-3 fatty acids could be considered for managing postprandial triglycerides. In addition, optimizing glycemic control through insulin sensitizers and other antidiabetic agents may help improve lipid metabolism. Postprandial dyslipidemia, especially in patients with poor glycemic control, should be recognized as a key factor in cardiovascular risk stratification. Early identification and management may help reduce the burden of atherosclerotic cardiovascular disease in this population. [20].

CONCLUSION

Postprandial dyslipidemia is prevalent among diabetic patients and is significantly associated with poor glycemic control. Monitoring postprandial lipid levels could be essential in managing cardiovascular risk in this population Postprandial hyperlipidemia is atherogenic and associated with the development of CAD. It is important to appropriately understand the existence of postprandial hyperlipidemia and to connect it to optimal treatments. However, there are some problems with the diagnosis of postprandial hyperlipidemia. Elevations in fasting and non- fasting TG, non-HDL-C, fasting serum apo B48, RLP-C and Rem-C were useful to identify the existence of postprandial hyperlipidemia; however, since reference values for these parameters have not been determined, no treatment strategy has been established based on these values. Postprandial hyperlipidemia cannot specifically be defined by measures such as TG levels 2 h after a meal. To study interventions for postprandial hyperlipidemia with the outcome of preventing the onset of ASCVD, it is necessary to define postprandial hyperlipidemia using reference values such as IGT.

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