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Original article

Postprandial Dyslipidemia: Emerging Lipid Profile for Cardiovascular Disease risk in Type 2 Diabetes Mellitus Subjects: A Case Control Study

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ABSTRACT:

Background and Objectives: Type 2 Diabetes Mellitus (Type 2 DM), characterized by a relative insulin deficiency or insulin resistance is associated with a cluster of metabolic abnormalities, which includes glucose intolerance, hypertension, a unique dyslipidemia, a procoagulant state, and an increase in macrovascular diseases. The present study was conducted to assess the significance of postprandial dyslipidemia with respect to fasting dyslipidemia, in the pathogenesis of athero-sclerotic changes and possible cardiovascular diseases.

Methods and Statistical Analysis: Fifty clinically diagnosed cases of Type 2 DM (age group of 34-68 years, duration of diabetes of more than five years), were in¬cluded in the study and 50 age and sex matched healthy subjects were taken as the controls. In both the study groups, we measured postprandial as well as fasting lipid profile, which comprised of serum total Cholesterol (TC), triglycerides (TGs), high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C) and the waist-hip ratio (WHI) as the cardiovascular risk factors. The statistical analysis was done by using the Students unpaired't'-test.

Results: The results of present study showed significantly increased levels of postprandial serum total cholesterol, TGs, LDL-C and VLDL-C as compared to those in the fasting state ($p<0.001$). The serum HDL-C level was significantly lower in the postprandial state as compared to that in the fasting state ($p<0.001$).

Conclusion: The findings of the study indicated that postprandial lipid profile, as a cardiovascular risk factor, was significantly elevated as compared to lipid profile in fasting state. This signifies that the routine estimation of the postprandial lipid profile, in addition to the fasting lipid parameters is mandatory in the cardiovascular disease risk assessment in Type 2 Diabetes Mellitus subjects.

KEYWORDS: Cardiovascular disease (CVD);Diabetes Mellitus (DM); postprandial blood glucose(PBG); Waist-hip ratio(WHR).

INTRODUCTION

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Diabetes Mellitus (DM) is a group of metabolic diseases characterized by chronic hyperglycemia resulting from defects in insulin secretion, insulin action, or both. The basis of the abnormalities in carbohydrate, fat, and protein metabolism in Diabetes is incomplete action of insulin on target tissues. Type 2 Diabetes Mellitus (Type 2 DM), the most prevalent form of the disease, is often asymptomatic in its early stages and can remain undiagnosed for many years[1,2].

Type 2 Diabetes Mellitus (DM) is characterized by insulin resistance which is associated with glucose intolerance, hypertension, dyslipidemia, a procoagulant state, and an increase in the microvascular and the macrovascular disease. Diabetics are frequently hyperlipidemic and they are at a high risk for coronary heart disease[3]. The high cardiovascular mortality which is associated with Type 2 DM is due to a prolonged, exaggerated, postprandial state[4,5]. The abnormal lipid profile in the postprandial state is more significant than the abnormal lipid profile in the fasting state in causing atherosclerotic complications in Type 2 diabetics[6-9]. Very few studies are available on the estimation of the postprandial lipid profile in Type 2 diabetic patients.

MATERIALS ANDMETHODS

The study population and its design: The present study was carried out on fifty Type 2 DM patients from the diabetic clinic or the indoor medicine wards and on fifty age and sex matched healthy controls, in the Department of Biochemistry, in collaboration with the Department of Medicine, Government Medical College and Hospital, Nagpur Maharashtra, India. The study group comprised of diagnosed Type 2 DM patients. The patients who were on oral hypoglycaemic drugs, who had a duration of diabetes of more than five years and who were in the age group of 34-68 years, were only included in the study.

The patients with Type 1 DM, ages of less than 35 years and more than 65 years, renal failure, hepatic diseases, acute illnesses, recurrent myocardial infarction, unstable angina and a drug therapy that interfered with the serum lipid levels, were excluded from the study. This study was approved by the local ethical committee and before their participation; the patients and the volunteers were fully informed about the nature and the purpose of the study. Written consents were obtained from each of them. A majority of the patients had similar diets and lifestyles with regards to their daily exercise. The body weight and the height were recorded. The waist circumference and the hip circumference were measured and the WHR was calculated. A clinical examination, a urine examination, and a fundus examination were performed to assess the diabetic target organ damage.

LABORATORY ASSAYS

Under aseptic conditions, blood samples were drawn in the morning after an overnight (i.e. after 12 hours) fast for fasting lipid profile and 6 hours after meals for postprandial lipid profile. The serum was separated from the blood cells by centrifugation within 30 minutes of the collection of the blood. The separated serum was analyzed for the following biochemical parameters:

1.Serum total cholesterol (TC) by an enzymatic method

2.Serum triglycerides (TGs) by an enzymatic method.

3.Serum HDL cholesterol (HDL-C) by phosphotungstate precipitation and enzymatic method.

4.Serum LDL Cholesterol and VLDL Cholesterol by using Friedewald's formula [10].

All parameters were analyzed by using a semiautomatic analyzer (Transasia ERBA Chem-5 Plus).

STATISTICAL ANALYSIS

In this case control study, all the statistical analyses were performed by using the "Graph Pad Prism 5" Software. The data was expressed as Mean ± SD. By using the Students unpaired 't'-test, the statistical analysis was carried out to assess whether the differences between the Type 2 DM patients and the controls were significant and P values of <0.05 were considered as statistically significant.

RESULTS

We observed that the waist to hip ratios of the diabetic males and females were found to be statistically significant ($p<0.05$) as compared to those of their respective controls (Table 1). We observed a significant increase in both fasting as well as postprandial blood glucose levels in the Type 2 Diabetic subjects, as compared to those of their respective controls. Also, the postprandial

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blood glucose level was significantly increased as compared to that in the fasting state in the Type 2 Diabetic subjects (Table 2).

Table 1. Comparison of waist hip ratio between Diabetic males and females.

Subjects	Waist Hip ratio (Mean ± S.D.)		p value
	CONTROLS (n=50)	DIABETICS (n=50)	
Males	**0.92 ± 0.035**	0.96 ± 0.048	0.000*
Females	**0.83 ± 0.044**	0.86 ± 0.048	0.036*

*Statistically significant in both diabetic males and females compared to controls.

Table 2. Values of Fasting and Postprandial blood glucose levels in study group (Type 2).

Blood Glucose levels	Type 2 DM subjects (N=50) (Mean ± SD)	Controls (N=50) (Mean ± SD)	P value
Fasting	154.60 ± 16.14	104.60 ± 12.14	0.000*
Postprandial	244.70 ± 21.14	124.60 ± 12.68	0.000*
P value	0.000*	0.000*	

We observed a significant increase in the serum total cholesterol (TC), triglycerides (TGs), the LDL-cholesterol levels in fasting as well as postprandial state in the Type 2 DM patients as compared to their respective control subjects (*p<0.001*). The HDL-cholesterol level was significantly decreased in fasting as well as postprandial state in the Type 2 DM patients as compared to that of control subjects (*p<0.001*) (Table 3 & 4 and fig. 1 & fig.2).

Table 3. Values of various parameters of fasting lipid profile in study group (Type 2 DM) and control group.

FASTING SERUM LIPIDS (mg/dl)	Normal Values (mg/dl)	CONTROLS (n=50) (Mean ± SD)	DIABETICS (n=50) (Mean ± SD)	p value by unpaired t test
Total Cholesterol	200	156.50 ± 32.92	208.10 ± 53.18	0.000*
Triglycerides	160	114.70 ± 34.17	171.70 ± 71.71	0.000*
HDL Cholesterol	40	50.82 ± 6.05	45.72 ± 8.82	0.001#
LDL Cholesterol	100	83.06 ± 33.58	128.80 ± 51.02	0.000*
VLDL Cholesterol				

*Significantly higher (*p<0.001*) as compared to control.
#Significantly lower (*p<0.001*) as compared to control.

Table 4. Values of various parameters of postprandial lipid profile in study group (Type 2 DM).

POSTPRANDIAL SERUM LIPIDS (mg/dl)	Normal Values (mg/dl)	CONTROLS (n=50) (Mean ± SD)	DIABETICS (n=50) (Mean ± SD)	P value by unpaired t test
Total Cholesterol	200	191.50 ± 36.38	238.90 ± 56.77	0.000*
Triglycerides	160	139.50 ± 34.32	209.50 ± 74.48	0.000*
HDL Cholesterol	40	41.88 ± 4.62	35.30 ± 7.25	0.000#
LDL Cholesterol	100	121.50 ± 36.71	162.10 ± 53.26	0.000*
VLDL Cholesterol				

*Significantly higher (*p<0.001*) as compared to control.
#Significantly lower (*p<0.001*) as compared to control.

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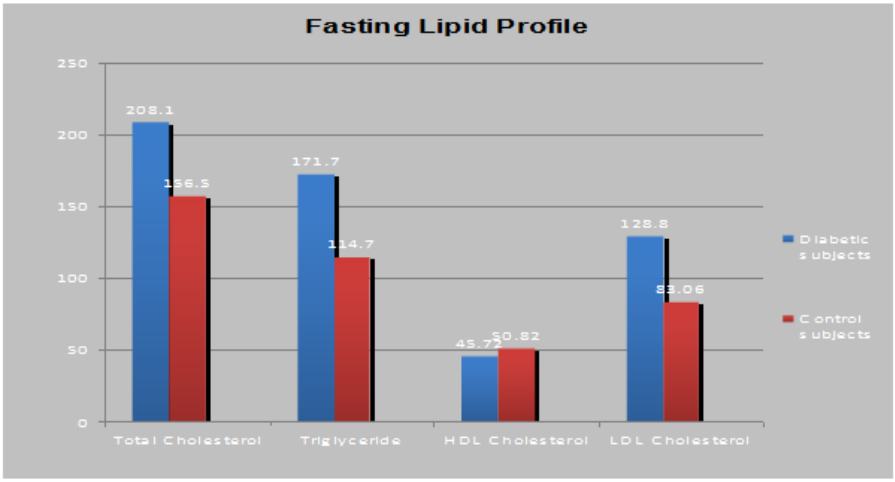

Figure 1. Pattern of fasting lipid profile.

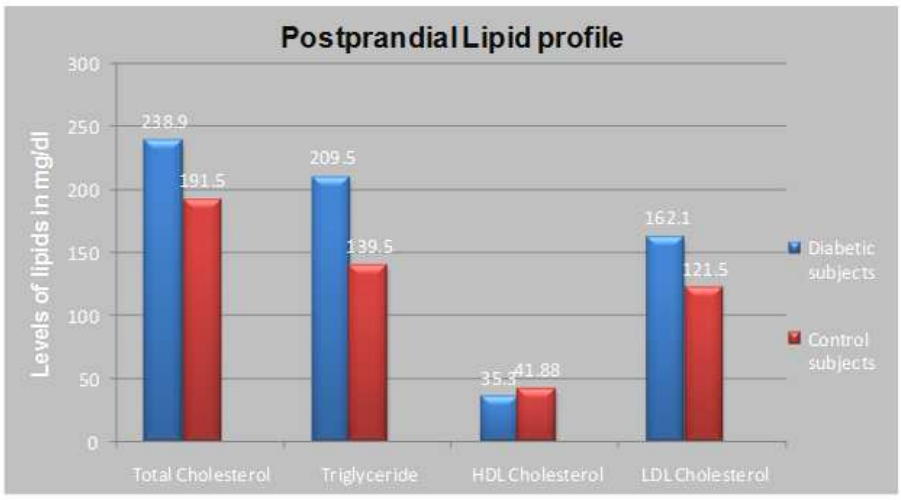

Figure 2. Pattern of postprandial lipid profile.

We observed a significant increase in the serum total cholesterol (TC), triglycerides (TGs), the LDL-cholesterol levels in the postprandial state in the Type 2 DM patients as compared to their serum levels in the fasting state (*p<0.001*). The HDL-cholesterol level was significantly decreased in the postprandial state as compared to that in the fasting state in the Type 2 DM patients (*p<0.001*) (Table 5 and Fig. 3).

Table 5. Values of various parameters of fasting and postprandial lipid profile in study group.

Type 2 Diabetes Mellitus Subjects (n=50)	Normal Values (mg/dl)	Postprandial lipid Profile (Mean ± SD)	Fasting lipid Profile (Mean ± SD)	P value by unpaired t test
Total Cholesterol	200	238.90 ± 56.77	208.10 ± 53.18	0.000*
Triglycerides	160	209.50 ± 74.48	171.70 ± 71.71	0.000*
HDL Cholesterol	40	35.30 ± 7.25	45.72 ± 8.82	0.000#
LDL Cholesterol	100	162.10 ± 53.26	128.80 ± 51.02	0.000*
VLDL Cholesterol				

*Significantly higher (*p<0.001*) as compared to control.
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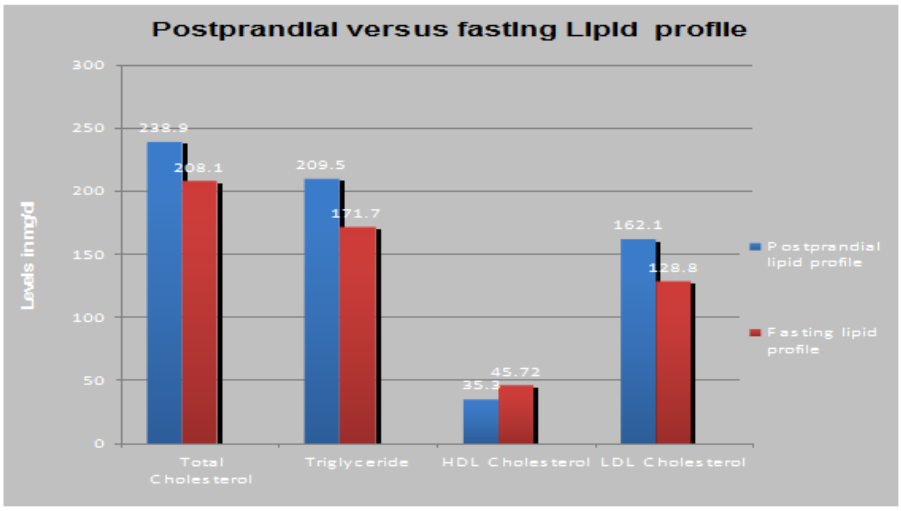

Figure3. Comparison of postprandial versus fasting lipid profile pattern.

In fasting state, isolated hypercholesterolemia and isolated hypertriglyceridaemia were found to be highly significant in cases as compared to controls (*p< 0.001*). Isolated HDL dyslipidemia in fasting state was found to be non significant in cases as compared to controls (*p> 0.05).* No association was found between fasting mixed dyslipidemia of cases and fasting mixed dyslipidemia of control subjects. Isolated hypercholesterolemia, isolated hypertriglyceridaemia, isolated HDL dyslipidemia and mixed dyslipidemia in the postprandial state were found to be highly significant in cases as compared to controls (*p< 0.001*). The numbers of subjects having isolated dyslipidemia are higher in postprandial state when compared with that of in fasting state in Type 2 Diabetes Mellitus patients.

DISCUSSION

In the present study, the postprandial lipid parameters i.e. TC, TGs and LDL-C were significantly increased in the Type 2 DM subjects as compared to the fasting lipid parameters and the postprandial HDL-C level was significantly decreased as compared to the fasting HDL-C level (*p<0.001*) (Table 5 and Fig. 3). Also, the postprandial lipid parameters i.e. TC, TGs and LDL-C were significantly increased in the Type 2 DM subjects as compared to those in the control subjects (Table 4 and Fig. 2), which was in accordance with the results of previous studies by Axelsen M et al. (1999)[6], Ferreira AC (2004)[9], Tushuizen MF et al. (2005)[17] and Kumar V et al. (2010)[21]. The pattern of isolated dyslipidemia in Type 2 Diabetes Mellitus subjects in postprandial state is statistically significant when compared with the pattern of isolated dyslipidemia in fasting state as well as control subjects (Table 7 and 8).

Table 6. Frequency of complications in study group (Type 2 DM patients).

S. no	Complications	Number of Subjects	n (%)
1	Retinopathy a. Background b. Proliferative c. Mixed	 16 03 --	19 (38)
2	Peripheral Neuropathy	11	11 (22)
3	Microalbuminuria	20	20 (40)

Table 7. Pattern of isolated dyslipidemia in postprandial state in Type 2 Diabetes Subjects.

	POSTPRANDIAL DYSLIPIDAEMIA	INCIDENCE (%)	
		DIABETES (n=50)	CONTROLS (n=50)
1	Isolated hypercholesterolemia *(Cholesterol > 200 mg/dl)*	42(84%)	18(36%)
2	Isolated hypertriglyceridaemia *(TG > 160 mg/dl)*	40(80%)	16(32%)

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3	Isolated HDL dyslipidemia *(HDL < 35 mg/dl in males and < 45 mg/dl in females)*	40(80%)	19(38%)
4	Mixed dyslipidemia: *(TC> 200 mg/dl + TG > 160 mg/dl + HDL < 35 mg/dl in males & <45mg/dl in females)*	34(68%)	06(12%)

Table 8. Pattern of isolated dyslipidemia in fasting state in Type 2 Diabetes Subjects.

	FASTING DYSLIPIDAEMIA	INCIDENCE (%)	
		DIABETES (n=50)	CONTROLS (n=50)
1	Isolated hypercholesterolemia *(Cholesterol > 200 mg/dl)*	22(44%)	4(8%)
2	Isolated hypertriglyceridaemia *(TG > 160 mg/dl)*	26(52%)	5(10%)
3	Isolated HDL dyslipidemia *(HDL < 35 mg/dl in males and < 45 mg/dl in females)*	10(20%)	03(6%)
4	Mixed dyslipidemia *(TC> 200 mg/dl + TG > 160 mg/dl + HDL < 35 mg/dl in males & <45mg/dl in females)*	4(8%)	00(0%)

Diabetes Mellitus (DM) is a group of metabolic diseases, which is characterized by chronic hyperglycaemia, which results from the defects in the insulin secretion, insulin action, or both. Type 2 Diabetes Mellitus (Type 2 DM), the most prevalent form of the disease, which is often asymptomatic in its early stages and it can remain undiagnosed for many years[11]. In Type 2 DM, the insulin resistance in the liver reflects the failure of the hyperinsulinemia to suppress the gluconeogenesis, which results in fasting hyperglycaemia and a decreased glycogen storage by the liver in the postprandial state. Increased hepatic glucose production occurs early in the course of diabetes, though it is likely after the onset of the insulin secretory abnormalities and the insulin resistance in the skeletal muscle. As a result of the insulin resistance in the adipose tissue and obesity, the free fatty acid (FFA) flux from the adipocytes is increased, which leads to an increased lipid [very low density lipoprotein (VLDL) and TGs] synthesis in the hepatocytes. This is responsible for the dyslipidemia which is found in Type 2 DM [elevated TGs, reduced HDL-C, and increased small dense low-density lipoprotein (LDL) particles[12].

This chronic hyperglycaemia of diabetes is associated with a long term damage, dysfunction and failure of various organs, especially the eyes, kidneys, nerves, heart and the blood vessels. The risk of the chronic complications increases as a function of the duration of the hyperglycaemia. Since Type 2 DM, often, has a long asymptomatic period of hyperglycaemia, many individuals with Type 2 DM have complications at the time of their diagnosis[13]. The macrovascular complications such as coronary heart disease and cerebrovascular disease are two to four times greater in the patients with Type 2 DM. Other factors (dyslipidemia and hypertension) also play important roles in the macrovascular complications [14].

The postprandial dysmetabolism and the associated oxidative stress may link the insulin resistance and the Type 2 DM to the disproportional incidence of cardiovascular disease. Postprandial hypertriglyceridaemia has been linked to asymptomatic and symptomatic macrovascular diseases in both normo- and hypertriglyceridaemic subjects and such abnormalities have been reported in the type 2 diabetics. The increased risk of atherosclerosis among them therefore, may be related to the higher postprandial lipaemia in them. The earlier studies clearly demonstrate the presence of postprandial hypertriglyceridaemia among the diabetic subjects, irrespective of the fasting triglyceride levels[15].

Various studies have shown that postprandial dyslipidemia is more important in the pathogenesis of the vascular changes and atherosclerosis and that it increases the risk of the cardiovascular events [16]. Though the importance of LDL cholesterol in the development of atherosclerosis has long been recognized, the increasing research attention over the past decades has been devoted to the heterogeneity of the LDL particles and the atherogenicity of the lipids and the lipoproteins which are other than LDL. A particularly atherogenic form of LDL includes the small, dense LDL particles and the oxidized LDL[17,18]. The postprandial dysmetabolism and the associated oxidative stress may link the insulin resistance and the Type 2 DM to the disproportional incidence of cardiovascular

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disease[19]. The high cardiovascular disease morbidity and the mortality in Type 2 DM, is associated at least partly caused by a prolonged and an exaggerated postprandial state in these patients[20]. Persistent postprandial hypertriglyceridemia may result in a pro-atherogenic environment leading to atherosclerosis and macrovascular disease in type 2 diabetes subjects[21]. LDL oxidation in the postprandial state seems to be affected by an acute increase in glycemia. Thus, oxidative modification of LDL may contribute to higher CVD risk among diabetic patients, and elevated levels of TG may contribute to the rapid LDL oxidation seen in Type 2 DM[22].

Hence, it is important and beneficial to estimate the postprandial lipid profile, in addition to the fasting lipid profile, in the cardiovascular risk assessment in the patients with Type 2 DM.

CONCLUSIONS

Atherosclerosis is a postprandial phenomenon with respect to lipids, as we are in the postprandial phase for most of the day, with an additional adverse effect of the meal induced hyperglycaemia. The present study suggests that it is important and beneficial to routinely estimate the postprandial lipid profile, in addition to the fasting lipid parameters, in the cardiovascular risk assessment in Type 2 DM. Thus, by rectifying the abnormal postprandial lipid parameters early in the course of diabetes, we can prevent the hazardous complication associated with Type 2 DM, the most common one being atherosclerotic coronary artery disease.

For lipids, the measurements which need to be used in the routine clinical practice and the clinically meaningful cut off values for the decision making, need to be established and more information regarding their clinical utility is needed. Further large scale studies including large sample size, are needed to elucidate the role of postprandial dyslipidemia in the pathogenesis of accelerated atherosclerotic cardiovascular disease, as well as the microangiopathic complications in the subjects of Type 2 Diabetes mellitus.

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