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Original Article

Study of Fasting and Postprandial Lipid Abnormality in Type 2 Diabetes Mellitus and Its Correlation with Vascular Complications of Diabetes

P Punekar[1], J K Jatav[1], D S Bantewad[2]

[1]Associate Professor, Department of Medicine, NSCB Medical College, Jabalpur, Madhya Pradesh, India,
[2]Senior Resident, Department of Medicine, NSCB Medical College, Jabalpur, Madhya Pradesh, India

Abstract

Introduction: High cardiovascular mortality which is associated with Type 2 diabetes mellitus (T2DM) is due to a prolonged, exaggerated postprandial (PP) state. The abnormal lipid profile in the PP state is more significant than that in the fasting state in causing atherosclerotic complications in T2DM.

Aims and Objectives: The objective of this study is to find fasting and postprandial lipid abnormality in T2DM patients and its correlation with vascular complications of T2DM.

Material and Methods: Fasting and PP lipids were measured in 150 T2DM patients. On the basis of normal and abnormal fasting lipid profiles, diabetic patients were divided into controls and cases, respectively, and evaluation for various complications was done and statistically analyzed.

Results: The result of this study showed a significant elevation in mean values of fasting blood glucose, serum cholesterol (SCH), low-density lipoproteins (LDL), very LDL, and triglycerides in the PP state in cases as compared to controls ($P < 0.05$). Waist-to-hip ratio in cases (mean 0.96 ± 0.02) was significantly higher ($P = 0.00$) as compared to that in controls (mean 0.93 ± 0.03). Microvascular complications such as retinopathy, nephropathy, and neuropathy were found significantly more in cases (46%, 55%, and 63%, respectively) as compared to controls ($P < 0.05$). Most common macrovascular complication observed in cases were IHD (24.6%) and least common were Peripheral Vascular Disease (10.6%). while there was no significant difference (P>0.05) found in occurrence of macrovascular complications in between cases and controls.

Conclusion: Since we are in the PP phase for most of the day, it is important to estimate the PP lipid profile along with fasting lipid profile, as atherosclerosis is a PP phenomenon with respect to lipids, Thus correction in early phase can prevent complications. So in routine practice PP lipid profile along with fasting is warranted.

Key words: Type 2 diabetes mellitus, Lipid abnormality, Dyslipidaemia, Vascular complications of diabetes

INTRODUCTION

The growing incidence of Type 2 diabetes mellitus (T2DM) is a major problem in the modern world. DM is a group of metabolic diseases, which is characterized by chronic hyperglycemia, which results from the defects in the insulin secretion, insulin action, or both.[1] Diabetic dyslipidemia contributes to the excess morbidity and mortality in T2DM.[2]

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. The high cardiovascular mortality which is associated with T2DM is due to a prolonged, exaggerated, postprandial state.[3] Persistent postprandial hypertriglyceridemia may result in proatherogenic environment leading to atherosclerosis and macrovascular disease in T2DM. It is being increasingly believed that atherosclerosis is a postprandial phenomenon as at least with respect to lipids, as we are in the postprandial phase for most of the day.[4]

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Corresponding Author: Dr. Devidas Bantewad, At Post Barbada Tq. Naigaon Dist Nanded, Maharashtra, India – 431602.
Phone: 7387323787. E-mail: dr.ds.bantewad@gmail.com

It is not clearly known whether diabetic patients with macrovascular disease have greater abnormalities of postprandial lipid metabolism than those without. Hence, this study is being carried out to find the characteristics of postprandial lipid levels in patients with T2DM and its impact on vascular complications.

Aims and Objectives

The aims of this study are to find fasting and postprandial lipid abnormality in T2DM patients and to find the significance of postprandial dyslipidemia and its correlation with vascular complications of T2DM.

MATERIALS AND METHODS

The present study was carried out on 150 T2DM patients from the diabetic clinic or the indoor medicine wards in Government Medical College and Hospital, Jabalpur, M.P., India; on the basis of fasting lipids, we divided patients into cases and control. Cases were those with abnormal fasting lipid profile and controls were those with normal fasting lipid profile. Further evaluation for various complications was done according to protocol and statistically analyzed. The study was approved by the Institutional Ethics Committee 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.

Inclusion Criteria

Cases

All T2DM patients who were in the age group of 35–65 years on treatment with oral hypoglycemic agents (OHA) and were attending medicine Outpatient Department (OPD), diabetic clinic, and admitted in medicine wards with abnormal fasting lipid profiles were selected as cases.

Controls

All Type 2 diabetic mellitus patients which were in the age group of 35–65 years on treatment with OHA attending medicine OPD, diabetic clinic and admit in wards having normal fasting lipid profile were selected as controls.

Exclusion Criteria

The following criteria were excluded from the study**:**

- Type I DM patients
- Diabetic patient on hypolipemic drug
- Patients on insulin therapy
- Gestational diabetic patients
- Patients with thyroid disease
- Patient not willing for study.

Laboratory Assays

Under aseptic conditions, blood samples were drawn in the morning after an overnight (i.e., after 12 h) fast and 2 h after meals. The serum was separated from the blood cells by centrifugation within 30 min of the collection of the blood. The separated serum was analyzed for lipid abnormalities.

Statistical Analysis

The data of the present study were recorded into the computers and after its proper validation, checked for errors and data were analyzed using the software SPSS 20 for windows. Appropriate univariate and bivariate analysis was carried out using the Student *t*-test for the continuous variable (age) and Chi-square (χ^2) test for categorical variables. All means are expressed as mean ± standard deviation for continuous data, while qualitative information is expressed in proportion with a percentage. The critical levels of significance of the results were considered at 0.05 levels, i.e., $P < 0.05$ was considered statistically significant.

RESULTS

A total of 100 cases and 50 controls were included in the final analysis. Mean age of cases and controls were 53.9 ± 9.9 years and 51 ± 7.9 years respectively, on applying unpaired *t* test no significant difference was found (P=0.073). On applying unpaired *t*-test, there were significantly higher value of hemoglobinA1c (HbA1c) (8.70 ± 1.5 vs. 6.58 ± 1.1) and waist–to-hip ratio (WHR) (0.96 ± 0.02 vs. 0.93 ± 0.03) value found to be more in cases as compared to controls ($P < 0.05$). We observed a significant increase in both the fasting and the postprandial blood glucose levels in cases, as compared to those of their controls. Furthermore, the postprandial blood glucose level was significantly increased as compared to that in the fasting state in cases.

On applying paired *t*-test [Tables 1-3], there was a significant increase in SCH, low-density lipoproteins (LDL), very LDL (VLDL), and triglycerides (TG) postprandial state as compared to fasting state in both cases as well as controls ($P < 0.05$), there were significantly higher values of postprandial SCH, LDL, VLDL, and TGs in cases as compared to respective values in controls ($P < 0.05$), while high-density lipoproteins (HDL) was not found higher ($P > 0.05$).

On applying Chi-square test [Table 4], all three microvascular complications (peripheral neuropathy, diabetic retinopathy, and diabetic nephropathy) were found statistically more prevalent in cases as compared to controls ($P < 0.05$), while none of three macrovascular complications (ischemic heart disease [IHD], Peripheral vascular disease [PVD], and cerebrovascular accident [CVA]) in our study [Table 5] was not found statistically significant ($P > 0.05$).

Paired *t*-test was applied in between fasting and postprandial lipid profile of patients with various

Table 1: Comparison of mean postprandial lipid parameters

Parameters (mg/dl)	SCH	HDL	LDL	VLDL	TG
Cases (*n*=100)	215.30±55.01	39.53±9.50	129.52±37.68	42.02±16.8	207.87±70.32
Controls (*n*=50)	171.68±20.84	39.25±16.5	109.50±19.08	33.22±10.1	144.14±21.92
P value	<0.05	>0.05	<0.05	<0.05	<0.05

All values in mg/dl, mean±SD. SD: Standard deviation, SCH: Serum cholesterol, HDL: High-density lipoproteins, LDL: Low-density lipoprotein, VLDL: Very low-density lipoproteins, TG: Triglycerides

Table 2: Comparison of mean lipid parameters within cases (*n*=100)

Parameters (mg/dl)	SCH	HDL	LDL	VLDL	TG
Fasting	133.46±23.83	42.34±3.06	93.07±16.5	26.58±8.00	102.04±16.09
Postprandial	171.68±20.84	39.25±16.5	109.50±19.08	33.22±10.1	144.14±21.92
P value	<0.05	>0.05	<0.05	<0.05	<0.05

All values in mg/dl, mean±SD. SD: Standard deviation, SCH: Serum cholesterol, HDL: High-density lipoproteins, LDL: Low-density lipoprotein, VLDL: Very low-density lipoproteins, TG: Triglycerides

Table 3: Comparison of mean lipid parameters within controls (*n*=50)

Parameters (mg/dl)	SCH	HDL	LDL	VLDL	TG
Fasting	166.17±38.77	39.13±8.25	103.17±28.43	31.36±12.9	135.96±41.29
Postprandial	215.30±55.01	39.53±9.50	129.52±37.68	42.02±16.8	207.87±70.32
P value	<0.05	>0.05	<0.05	<0.05	<0.05

SCH: Serum cholesterol, HDL: High-density lipoproteins, LDL: Low-density lipoprotein, VLDL: Very low-density lipoproteins, TG: Triglycerides

Table 4: Comparison of fasting and postprandial lipid profile in patients with microvascular complications

Lipid parameters (mg/dl)	Peripheral neuropathy (*n*=75)			Diabetic retinopathy (*n*=60)			Diabetic nephropathy (*n*=70)		
	Fasting	PP	*P*	Fasting	PP	*P*	Fasting	PP	*P*
SCH	160.0±42.6	205.9±63.3	<0.05	159.5±42.6	204.0±54.0	<0.05	160.1±43.9	208.4±61.4	<0.05
HDL	39.9±6.9	40.1±11.0	>0.05	41.0±7.6	37.8±9.2	<0.05	40.3±7.5	40.7±16.5	>0.05
LDL	99.27±30.0	125.5±42.5	<0.05	100.8±30.3	124.2±41.6	<0.05	98.9±31.5	122.7±38.6	<0.05
TG	128.3±48.9	199.5±78.1	<0.05	124.6±45.4	192.8±79.1	<0.05	126.3±45.9	193.8±75.9	<0.05

All values in mg/dl, mean±SD. SD: Standard deviation, SCH: Serum cholesterol, HDL: High-density lipoproteins, LDL: Low-density lipoprotein, TG: Triglycerides

Table 5: Comparison of fasting and postprandial lipid profile with macrovascular complications among study group

Lipid parameters (mg/dl)	IHD (*n*=37)			PVD (*n*=16)			CVA (*n*=29)		
	Fasting	PP	*P*	Fasting	PP	*P*	Fasting	PP	*P*
SCH	149.0±42.6	199.3±54.0	<0.05	172.0±47.4	213.5±49.9	<0.05	171.1±36.7	218.8±55.6	<0.05
HDL	39.2±9.5	40.5±20.4	>0.05	39.3±6.7	39.2±7.1	>0.05	41.1±5.5	43.9±20.3	>0.05
LDL	96.1±28.6	123.4±39.1	<0.05	97.4±32.2	122.9±45.9	>0.05	111.7±29.2	134.2±30.5	<0.05
TG	118.0±42.4	183.4±69.7	<0.05	138.0±53.9	219.6±94.6	<0.05	132.6±53.7	192.9±65.2	<0.05

All values in mg/dl, mean±SD. SD: Standard deviation, SCH: Serum cholesterol, HDL: High-density lipoproteins, LDL: Low-density lipoprotein, TG: Triglycerides, IHD: Ischemic heart disease, PVD: Peripheral vascular disease, CVA: Cerebrovascular accident

complications [Table 6], and we observed that there were significant ($P < 0.05$) higher mean values of postprandial SCH, LDL, and TG as compared to fasting SCH, LDL, and TG, respectively, while HDL was observed not significant in patients with peripheral neuropathy and diabetic nephropathy ($P > 0.05$). There were significant higher mean values of postprandial SCH, LDL, and TG in patients with IHD and CVA as compared to their fasting SCH, LDL, and TG ($P < 0.05$), while LDL in peripheral vascular disease and HDL in all three macrovascular complications (IHD, peripheral vascular disease, and CVA) was not significant ($P > 0.05$).

DISCUSSION

Diabetes is one of the leading health problems in modern world. Diabetic dyslipidemia contributes to excess morbidity and mortality in T2DM, and the abnormal lipid profile in postprandial state is more significant than lipid profile in fasting state as most in

Table 6: Comparison of various parameters with lipid profile

Parameters	Cases (Mean±SD)								Controls (Mean±SD)							
	Fasting (mg/dl)				Postprandial (mg/dl)				Fasting (mg/dl)				Postprandial (mg/dl)			
	SCH	HDL	LDL	TG	SCH	HDL	LDL	TG	SCH	HDL	LDL	TG	SCH	HDL	LDL	TG
Age (years)																
<50	157.9±34.1	37.5±7.5	96.4±21.3	133.1±41.2	212.9±59.6	39.3±9.7	123.1±34.0	218.7±86.6	131.3±18.2	41.9±1.7	88.7±11.8	99.9±13.4	172.1±20.4	39.8±21.2	109.6±20.1	149.2±17.8
>50	170.7±40.6	40.0±8.5	106.8±31.2	135.5±42.9	216.6±52.8	39.7±9.5	133.0±39.4	202.1±59.7	135.2±27.1	42.9±4.1	89.8±14.0	104.6±18.7	171.2±21.8	38.6±8.8	109.4±18.3	138.2±25.1
Sex																
M	155.0±27.5	49.1±74.0	94.5±26.5	133.4±42.5	209.2±50.1	37.0±7.8	126.2±37.1	211.2±76.1	132.4±20.8	42.4±2.3	90.2±11.0	101.0±19.1	172.3±21.6	36.9±7.3	113.0±20.7	144.7±23.8
F	177.0±44.8	39.4±8.5	111.5±28.0	136.0±42.1	221.1±59.3	42.0±10.4	132.7±38.3	204.7±64.9	133.9±24.8	42.3±3.7	88.1±14.5	103.2±12.4	171.0±20.5	41.8±22.6	105.7±16.7	143.5±20.2
Locality																
Rural	167.9±42.0	46.7±63.5	104.3±30.0	139.3±45.7	219.6±54.0	40.1±10.1	132.4±38.8	219.3±80.9	135.0±19.6	41.2±1.3	90.9±7.6	104.5±14.3	164.2±19.1	37.5±6.5	101.6±18.3±	142.3±23.4
Urban	162.7±31.4	38.9±6.5	100.9±25.2	125.6±32.5	206.6±56.8	38.3±8.2	123.7±35.2	184.7±31.2	132.4±23.8	42.8±3.3	88.6±14.1	101.2±16.8	174.3±21.0	39.9±18.9	112.3±18.8	144.8±21.7
Treatment history																
Regular	160.7±26.7	39.6±8.2	97.7±24.5	138.0±46.6	207.0±43.1	38.1±6.1	127.9±38.1	224.0±71.4	128.9±24.9	42.3±3.6	87.5±15.4	102.1±16.5	170.4±18.7	41.2±22.1	111.0±17.3	144.2±22.8
Irregular	168.4±42.7	39.0±8.3	105.4±29.8	133.4±40.4	218.7±59.1	40.1±10.6	130.2±37.8	201.3±69.3	137.2±19.6	42.5±2.4	90.9±9.4±	102.0±16.0	173.0±23.1	37.4±7.9	108.0±21.0	144.1±21.5
Duration of DM (years)																
<5	165.2±41.3	54.3±90.3	102.2±32.9	136.6±49.9	203.1±65.0	38.4±10.2	114.2±32.5	222.6±102.6	130.5±23.2	42.4±3.3	88.8±11.4	102.6±16.0	167.3±21.3	40.9±19.4	108.0±21.4	144.8±20.4
>5	166.7±37.8	39.1±6.7	103.6±26.2	133.8±38.2	221.3±48.8	40.1±9.2	137.1±38.0	200.6±46.4	138.6±21.0	42.4±2.4	90.1±15.6	100.8±16.7	180.9±17.1	35.8±6.4	112.7±12.9	142.7±25.6
HbA1C																
<6.5	183.1±41.9	39.3±6.6	101.3±37.6	161.7±55.8	201.5±89.9	42.5±11.0	117.2±38.5	236.2±111.4	130.5±23.6	42.0±2.1	91.7±8.6	106.7±12.7	165.3±21.8	35.3±6.0	108.0±18.0	143.1±25.0
>6.5	164.3±38.2	44.7±54.9	103.4±27.5	131.7±39.5	216.8±50.3	39.2±9.3	130.9±37.6	204.7±64.4	137.4±20.8	43.0±4.1	85.1±17.0	94.5±18.4	182.0±14.4	45.8±24.7	111.9±21.0	145.9±16.1
WHR																
<0.95	162.1±39.8	38.2±6.7	94.1±26.3	144.7±47.0	222.3±59.0	40.9±8.5	124.2±37.8	233.9±83.2	135.5±19.8	42.3±2.2	90.8±9.1	102.7±16.7	172.4±20.7	41.0±19.9	111.8±20.0	145.3±21.9
>0.95	168.5±38.3	47.4±64.9	108.3±28.5	129.0±38.3	211.4±52.7	38.8±10.0	132.5±37.6	193.3±57.6	128.8±27.0	42.5±4.2	86.4±17.4	100.8±15.3	170.3±21.6	36.1±6.9	105.4±17.2	142.1±22.5

SD: Standard deviation, SCH: Serum cholesterol, HDL: High-density lipoproteins, LDL: Low-density lipoprotein, TG: Triglycerides, DM: Diabetes mellitus, HbA1C: HemoglobinA1c, WHR: Waist-to-hip ratio

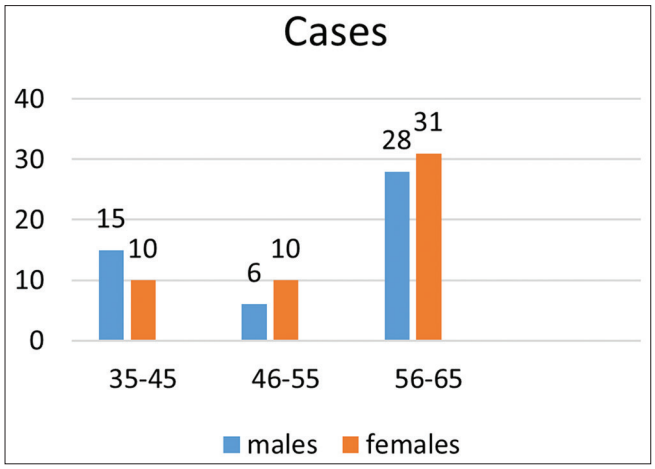

Chart 1: Distribution of cases according to age and sex

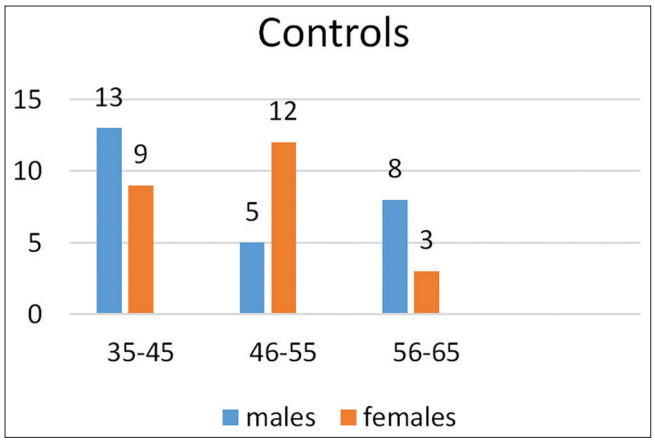

Chart 2: Distribution of controls according to age and sex

causing atherosclerotic complications. 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 hyperglycemia.

In the present study, the postprandial lipid parameters (SCH, LDL, VLDL, and TG) were significantly increased in both cases as well as controls in comparison with their respective fasting parameters ($P < 0.05$). There were significantly increased postprandial lipid parameters (SCH, LDL, VLDL, and TG) of cases as compared to that of controls ($P < 0.05$). We also observed that there were significantly higher values of postprandial SCH, LDL, VLDL, and TG in patients with most of the various micro- as well as macro-vascular complications as compared to their respective fasting values ($P < 0.05$).

T2DM is metabolic disorder characterized by insulin resistance associated with proatherogenic cardiovascular risk profile which includes impaired glucose regulation, abdominal obesity, hypertension, atherogenic dyslipidemia, and an increase in the microvascular and the macrovascular disease.[5,6] As a result of the insulin resistance in the adipose tissue and obesity, the free fatty acid flux from the adipocytes is increased, which leads to an increased lipid synthesis in the hepatocytes. This is responsible for the dyslipidemia which is found in T2DM (elevated TGs, reduced HDL-cholesterol, and increased small dense LDL particles).[7] 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.[8] There are few studies that have reported that postprandial dyslipidemia is more important in the pathogenesis of the vascular changes and atherosclerosis and it increases the risk of the cardiovascular events.[9-11]

The postprandial dysmetabolism and the associated oxidative stress may have a link with insulin resistance and T2DM, thereby increasing the incidence of cardiovascular disease disproportionately.[12] Another study has proposed that cardiovascular disease morbidity and mortality associated with T2DM showed prolonged and exaggerated postprandial state.[13]

CONCLUSION

In the present study, we found that there was very high occurrence of fasting and postprandial dyslipidemia in patients having diabetes, and when we compared postprandial dyslipidemia with fasting dyslipidemia in diabetic patients, we found that postprandial dyslipidemia was significantly higher than fasting dyslipidemia.

When we studied postprandial dyslipidemia in diabetic patients, we found a statistically significant association of postprandial dyslipidemia with increasing age, prolonged duration of disease, patients belonging to rural area, patients on irregular treatment, patients having other comorbid illness, and patients having higher HbA1c and increased WHR.

When we compared postprandial dyslipidemia and fasting dyslipidemia in diabetic patients having various vascular complication, we observed that there was a significant association between postprandial dyslipidemia and various complications including microvascular complications (peripheral neuropathy, diabetic retinopathy, and diabetic nephropathy) and macrovascular complications (IHD, CVA, and PVD). Hence, it can be concluded that postprandial dyslipidemia has the major contributory effect on various micro- and macro-vascular complications.

Hence, routine follow-up of postprandial dyslipidemia in diabetes may help in early diagnosis and prompt

management of various vascular complications which in turn can lead to improved quality of life.

Limitations of Study

1. Since our hospital being a tertiary center, more of the complicated diabetic patients were referred to medical college hospital so a community-based study might had given better idea about the prevalence of various complications.
2. We had included 150 diabetic patients which were a small number considering the high prevalence of diabetes in our society.
3. Since we had not included normal population as control group, it might had been the reason for variation in the findings observed in our study as compared to previous studies.
4. Fundus photography which is a gold standard for diagnosis of retinopathy was not available at our center.

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