Content available at: https://www.ipinnovative.com/open-access-journals

Journal of Preventive Medicine and Holistic Health

Journal homepage: https://www.jpmhh.org/

Original Research Article

Study of fasting and post prandial lipid abnormalities in type 2 diabetes mellitus in comparison to controls

Ram Chaitanya K[1], Ayyali Ambresh[2,*]

[1]*Dept. of Neurology, Ramaiah Medical College, Bengaluru, Karnataka, India*
[2]*Dept. of General Medicine, Shri Atal Bihari Vajpayee Medical College & Research Institute, Bengaluru, Karnataka, India*

ARTICLE INFO

Article history:
Received 01-03-2021
Accepted 05-04-2021
Available online 05-07-2021

Keywords:
T2DM
IHD
CVA
PVD
LDL
TG
Total Cholesterol.

ABSTRACT

Introduction: In the modern world, the growing incidence of type 2 diabetes mellitus (T2DM) is a critical problem. In type 2 DM, abnormal lipid profile in the postprandial state has more significance than abnormal lipid profile in fasting state in causing atherosclerotic complications. The high cardiovascular morbidity and mortality in type2 DM are due to prolonged postprandial hyperglycemia and triglyceridemia. Postprandial hypertriglyceridemia results in a proatherogenic environment which leads to atherosclerosis and macrovascular complications in type 2 diabetes mellitus. It is believed that atherosclerosis is a postprandial phenomenon with respect to lipids, as we are in the postprandial state for most of the day.It is not clearly known whether diabetic patients with macrovascular disease have greater abnormalities of postprandial TG metabolism than those without.Hence this study is being carried out to find out the characteristics of post prandial lipid levels in patients with type 2 diabetes mellitus and itsimpact on macrovascular complications.

Objective: To study fasting & postprandial lipid abnormality in type 2 DM patients in comparison to controls.

Materials and Methods: This is a cross-sectional study, wherein written informed consent was taken after giving detailed information to the participants regarding the study. Patients who were in the age group of 35-65 years, admitted in the Department of Medicine, RRMCH from November 2017 for next 18 months with Diabetes Mellitus who met a predefined inclusion and exclusion criteria were studied. The study was initiated after obtaining clearance from the institution's ethical committee.

Results: There was a significant elevation of mean values of total serum cholesterol, LDL and TG of cases and controls in the postprandial state compared to their fasting state, statistical significance was found ($P<0.05$). Peripheral vascular disease in our study was found in 25 patients i.e.25% of the study group. In cases, it was found in 20 patients and in controls, it was found in 5 patients i.e. 40% and 10% respectively. The occurrence of PVD was more in cases compared to controls with statistical significance($p<0.05$). Stroke in our study is found in 7 patients i.e. 7% of the study group. In cases, it was found in 6 patients and in controls, it was found in 1 patient i.e. 12% and 2% respectively. The occurrence of CVA was more in cases compared to controls with statistical significance($p<0.05$). Ischemic strokes were more common than haemorrhagic strokes.

Conclusion: Patients on irregular treatment (63%) were more in the study group(cases) compared to controls. So it could be said that patient not on regular treatment are more prone to have dyslipidemia. (fasting as well as postprandial). There was a significant increase in total serum cholesterol, LDL and TG in postprandial states of cases compared to that in controls, so it could be said that diabetic patients with fasting dyslipidemia are more prone to have dyslipidemia in the postprandial state. This study concluded that all macrovascular complications (IHD, CVA, PVD) were found more in the case compared to controls with statistical significance. So it could be said that there is an increase in the occurrence of macrovascular complications with an increase in postprandial dyslipidemia.

© This is an open access article distributed under the terms of the Creative Commons Attribution License (https://creativecommons.org/licenses/by/4.0/) which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

https://doi.org/10.18231/j.jpmhh.2021.010
2454-6704/© 2021 Innovative Publication, All rights reserved.

1. Introduction

In the modern world, the growing incidence of type 2 diabetes mellitus (T2DM) is a critical problem. Most subjects with T2DM have insulin resistance and are at increased risk of developing cardiovascular disease (CVD). Excess morbidity and mortality in T2DM are mainly due to dyslipidemia. Postprandial triglyceridemia is a distinct component of diabetic dyslipidemia.[1,2]

In type 2 DM, abnormal lipid profile in the postprandial state has more significance than abnormal lipid profile in fasting state in causing atherosclerotic complications. The high cardiovascular morbidity and mortality in type2 DM are due to prolonged postprandial hyperglycemia and triglyceridemia. Elevated - total triglycerides (TGs), very low-density lipoproteins (VLDL) and a decreased high-density lipoproteins (HDL) concentration in the serum are the predominant lipid abnormalities seen in diabetes mellitus. Postprandial hypertriglyceridemia results in a proatherogenic environment which leads to atherosclerosis and macrovascular complications in type 2 diabetes mellitus.

It is believed that atherosclerosis is a postprandial phenomenon with respect to lipids, as we are in the postprandial state for most of the day[3] 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. 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. This is responsible for the dyslipidaemia which is found in type2 diabetes mellitus [elevated triglycerides, reduced HDL, and increased low-density lipoprotein (LDL) particles.[4]

Individuals with type 2 diabetes mellitus are at increased risk of developing microvascular and macrovascular complications. 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[5–7] 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, 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.[8] Many epidemiological and preliminary intervention studies have shown that postprandial hyperglycemia is an independent and direct risk factor for the development of cardiovascular diseases (CVD). Most of the cardiovascular risk factors are modified in the postprandial state in diabetics affected by an acute rise in blood glucose levels. 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.[5–7,9,10]

2. Objective of The Study

To study fasting & postprandial lipid abnormality in type 2 DM patients in comparison to controls

3. Material and Methods

This is a descriptive cross-sectional study, wherein written informed consent was taken after giving detailed information to the participants regarding the study. Patients who were in the age group of 35-65 years, admitted in the Department of Medicine, RRMCH from November 2017 for next 18 months with Diabetes Mellitus who met a predefined inclusion and exclusion criteria were studied. The study was initiated after obtaining clearance from the institution's ethical committee.

3.1. Inclusion criteria

All type 2 DM patients who were in the age group of 35-65 years on regular treatment with OHA which had a duration of diabetes of more than 5 years in medicine OPD, diabetic clinic and wards.

3.2. Exclusion criteria

1. Type I DM patients
2. Patients with congenital hyperlipidemia
3. Diabetic patient on the hypolipemic drug
4. Patients on insulin therapy
5. Gestational Diabetic patients
6. Patients with thyroid disease

* *Corresponding author*.
E-mail address: ambrish7799@gmail.com (A. Ambresh).

7. Patients not willing for the study

3.3. Sample

50 cases of type 2 diabetes mellitus with abnormal fasting lipid profile(one or more lipid parameter) and 50 controls of type 2 diabetes mellitus with normal fasting lipid profile.

3.4. Operational definitions

1. Type 2 Diabetes mellitus patients were classified as having type 2 DM using clinical criteria such as a present/prior history of usage of OHAs or usage of a combination of insulin and the OHAs.

3.5. Diabetes is diagnosed by world health organization (WHO) criteria

1. Fasting plasma glucose (FPG)-126 mg/dl(7.0 mmol/l)or,
2. 75 g oral glucose tolerance test (OGTT) with fasting plasma glucose>126 mg/dl (7.0i. mmol/l) and/or 2 hours plasma glucose >200 mg/dl (11.1 mmol/l) or,
3. Glycated hemoglobin (HbA1c) > 6.5mg/dl or 48 mmol/mol, or
4. Random plasma glucose >200 mg/dl (11.1 mmol/l) in the presence of classical diabetes symptom.
5. Plasma glucose is measured by Randox autoanalyser using the colorimetric method without deproteinisation using glucose oxidase enzyme.
6. Dyslipidaemia: Abnormal lipid profile includes the following either singly or in combination, triglyceride (TG) levels >150 mg/dl, high density lipoprotein cholesterol (HDL-C) (for men < 40 mg/dl and women > 50 mg/dl), low density lipoprotein cholesterol (LDL-C) >100mg/dl. Lipid profile using RANDOX autoanalyser. All parameters expressed in milligram /decilitre. Cut-off values according to NCEP ATP III guidelines.[7]
7. Dyslipidaemia: Abnormal lipid profile includes the following either singly or in combination, triglyceride (TG) levels >150 mg/dl, high density lipoprotein cholesterol (HDL-C) (for men < 40 mg/dl and women > 50 mg/dl), low density lipoprotein cholesterol (LDL-C) >100mg/dl. Lipid profile using RANDOX autoanalyser. All parameters expressed in milligram /decilitre. Cut-off values according to NCEP ATP III guidelines.[7]

3.6. NCEP ATP III criteria for Lipids

3.6.1. Cholesterol (mg/dL)

3.6.2. Total Cholesterol (mg/dL)

3.6.3. HDL Cholesterol (mg/dL)

Also, considered abnormal is an elevated total serum cholesterol level $^{>200mg/dl}$.

Table 1:

<100	Optimal
100-129	Near optimal/above optimal
130-159	Borderline high
160-189	High
>190	Very high

Table 2:

<200	Desirable
200-239	Borderline high
>240	High

Table 3:

<40	Low
>60	High

3.7. Study protocol

Subjects fulfilling inclusion criteria were selected for the study. Each subject made two visits to the study hospital during which medical history, physical examination, systemic examinations and relevant investigations were carried out and they were advised to take a low fat diet consisting of 60% carbohydrates,25% fats and 15% proteins divided into 3 meals. Complete clinical history including name, age, sex, occupation, residence, presenting symptoms, duration of diabetes was noted. The general examination includes vitals i.e. pulse, supine and standing blood pressure at 3 minutes, general body survey, waist to hip ratio will be recorded. Systemic examination of cardiovascular, respiratory, gastrointestinal and CNS was done. Patients were examined, investigated and evaluated for specific complications of diabetes.

3.8. Following selection, the following was done

1. History, (including family history to judge the likelihood of a familial lipid disorder
2. Complete clinical examination (including fundus
3. CBC, ESR, Peripheral smear
4. Renal function test, Liver function test, TSH, Serum Electrolytes
5. Fasting and Postprandial blood sugars, HbA1C
6. Urine Routine,
7. BT, CT, PT/INR, aPTT
8. ECG and 2D Echo
9. CT scan / MRI scan of the brain
10. Chest X-ray (PA view)
11. USG Abdomen
12. Fasting Lipid Profile, Fasting LDL, LDL after 2 hours, 4 hours

3.9. Total cholesterol

1. Measured using enzymatic endpoint method. Cholesterol is degraded in the presence of enzyme cholesterol esterase. Indicator quinone imine is formed in presence of hydrogen peroxide & 4- aminoptyrine level of which is determined.
2. Chylomicrons, VLDL & HDL are eliminated by cholesterol esterase and oxidase then the specific measurement of LDL cholesterol is made after the release of LDL by detergents.

3.10. LDL- Cholesterol

1. Triglycerides:- GPO-PAP method. Triglycerides are determined after enzymatic hydrolysis with lipases and indicator quinonimine is formed is measured by colourimetric method.
2. HDL-Cholesterol:- Chylomicrons, VLDL & LDL are eliminated by cholesterol esterase and oxidase then the specific measurement of HDL cholesterol is made after the release of HDL by detergents.
3. VLDL cholesterol:- Calculated from total cholesterol –(LDL+ HDL cholesterol).

The data was collected in pre-prepared proforma and then transferred to a master chart for analysis.

4. Sample Size Estimation Calculation

4.1. Sample size

This is a descriptive cross-sectional study which was done in the Department of Medicine, RRMCH, Bangalore.

50 cases of type 2 diabetes mellitus with abnormal fasting lipid profile (one or more lipid parameter) and 50 controls of type 2 diabetes mellitus with normal fasting lipid profile.

4.2. Data and statistical analysis

The data collected was analyzed using mean, mode for demographic data and frequency percentage for the analysis of the clinical data.

Statistical Analysis was done using SPSS software version 23.0. A ‘p’ value less than 0.05($p<0.05$) is considered significant.

The various measures of central tendencies and graphical representations were used to analyze the data.

The data was analyzed using SPSS version 20. Mean and standard deviation will be applied between two groups. ’t’ test was applied to know the mean difference between two groups.

5. Observation and Results

Most common disease found in the past history was hypertension ,44% in cases and 16% in controls , followed by PVD (20% in cases and 4 % in controls) and IHD (16% and 2% in cases and controls respectively) .Past history of these diseases were more in the cases compared to controls. Statistical significance was found($P< 0.05$).

Table 4: Distribution according to past history among cases and control

	Htn	**Ihd**	**Pvd**	**Cva**	**Nil**
Cases	22	8	10	5	15
Control	8	1	2	1	38
P value	< 0.05	< 0.05	< 0.05	< 0.05	

Table 5:

Treatment	**Number**			**Percentage**			P value
	Cases	Controls	Total	Cases	Controls	Total	
OHA single	38	49	87	76.00%	98.00%	87.00%	
OHA comb-inations	12	1	13	24.00%	2.00%	13.00%	
Total	50	50	100	100.00 %	100.00 %	100.00 %	<0.0001

Most of the patients in both cases and controls were on treatment with a single OHA. 76.00% and 98.00% among of cases and controls respectively. This was statistically significant $p < 0.05$.

Table 6: Distribution based on regularity of treatment

	Number			**Percentage**		
Regularity Treatment	Cases	Controls	Total	Cases	Controls	Total
No treatment	0	0	0	0.00%	0.00%	0.00%
Irregular treatment	15	2	17	30.00 %	4.00%	17.00%
Regular treatment	35	48	83	70.00 %	96.00 %	83.00%
Total	50	50	100	100.00%	100.00 %	100.00 %

Most of the patients were on regular treatment (83 out of 100 patients). 35 patients (70.00%) and 48 patients (96.00%) of cases and controls respectively were on regulartreatment.15 patients in cases and 2 patients in controls were on irregular treatment.

5.1. Descriptive statistics

Table 7: Comparison of mean fasting lipid parameters

Group	N	M	Maximum	Mean	Std. deviation
cases					
Ldl	50	196	299	229.70	19.810
Hdl	50	20	68	30.12	7.553
TG	50	97	410	218.10	66.565
Total cholestrol	50	218	326	259.82	19.888
Controls	50	79	137	110.16	13.365
Ldl					
Hdl	50	27	67	47.74	9.705
TG	50	74	245	165.20	40.069
Total	50	127	194	157.90	17.081
Fasting		LDL	HDL	TG	Total
P value		<0.001	<0.001	<0.001	<0.001

There is a statistically significant increase intotal cholesterol, LDL and Triglycerides (P value <0.05) in Fasting state of the casescompared to controls.

5.2. Descriptive statistics

Table 8: Comparison of meanpostprandial lipid parameters

Group	N	Minimun	Maximum	Mean	Std. deviation
Cases					
LDL-pp	50	216	319	259.50	19.431
HDL-PP	50	17	65	27.12	7.553
Total Cholesterol-pp	50	245	343	286.62	19.727
TG-pp	50	115	428	236.10	66.565
Controls					
LDL-pp	50	110	168	141.76	13.540
HDL-pp	50	30	70	50.74	9.705
Total	50	150	229	192.50	17.210
Cholesterol-pp	50	92	263	183.20	40.659
TG-pp					
Post prandial		LDL	HDL	TG	Total
P Value		<0.001	<0.001	<0.001	<0.001

There is a statistically significant increase intotal cholesterol, LDL and triglycerides(P value <0.05) in Postprandial state of the casescompared to controls.

6. Discussion

CVD events are four times more common in individuals with diabetes, occur at a younger age, and have a much higher case fatality rate, this is more so in Indians. Coronary Artery Disease in Indians (CADI) is a phenomenon by itself.

Table 9: Comparison of meanlipid parameters within cases

Fasting	Minimum	Maximum	Mean	Std.deviation
LDL	196	229	229.7	19.81
HDL	20	68	30.12	7.533
TG	97	410	218.1	66.565
Total	218	326	259.82	19.888
Postprandial	Minimum	Maximum	Mean	Std.deviation
LDL-pp	216	319	256.5	19.431
HDL-pp	17	65	27.12	7.553
TG-pp	115	428	236.1	66.565
Total	245	343	286.62	19.727
P Value	LDL	HDL	TG	Total
	<0.01	>0.05	<0.01	<0.01

There is a statistically significant increase in total cholesterol, LDL, Triglycerides (P value <0.05) in Postprandial state of the cases compared to their fasting state.

Table 10: Comparison of meanlipid parameters within controls

Fasting	Minimum	Maximum	Mean	Std.deviation
LDL	79	137	110.16	21.816
HDL	27	67	47.74	10.61
TG	74	245	165.20	40.069
Total	127	194	157.90	21.833
Postprandial	Minimum	Maximum	Mean	Std.deviation
LDL-pp	110	168	141.76	12.915
HDL-pp	30	70	50.74	9.697
TG-pp	92	263	183	39.445
Total	150	229	192	16.80
	LDL	HDL	TG	Total
PValue				
	<0.01	>0.05	<0.01	<0.01

There isa statistically significant increase in total cholesterol, LDL, Triglycerides,(P value <0.05) in Postprandial state of thecontrols compared to their fasting state.

Table 11: Comparison of fbs andppbs within controls

	Cases	Controls	P value
FBS	142.09	122.17	0.00
	+45.86	+33.42	
PPBS	212.35	181.96	0.00
	+67.97	+33.77	
P value	0.00	0.00	

There was a statistically significant increase in PPBS value in both cases and controls compared to their FBS values and also there is a significant increase in PPBS values in cases compared to PPBS values in controls (p = 0.00).

Table 12:

Group	N	Minimum	Maximum	Mean	Std.deviation
Cases					
HBA1C	50	5.7	8.4	7.142	0.8318
Control					
HBA1C	50	4.6	6.2	5.554	0.4367

In fact, people with diabetes and no history of vascular disease have the same risk of having a heart attack or dying of vascular disease as non-diabetic individuals with a prior history of cardiovascular disease.Lipid and lipoprotein abnormalities are common in the diabetic population due to the effects of insulin deficiency and insulin resistance on key metabolic enzymes. In a study carried out by Angelo Avogaro, et al they found that the age-standardized incidence rate (per 1,000 person/years) of first CHD event was 28.8 in men and 23.3 in women.

In a study carried out by R.P. Agrawal, et al in 4067 patients of DM 2 they found that the prevalence of CHD was 12.4% and the risk of CHD in known Type 2 diabetic patients was higher in men than in women. Of the diabetic men 60% showed high (20–40%) or very high (>40%) risk of CHD, while 56% of the women had values compatible with moderate (10–20%), mild (5–10%) or low (<5%) risk of CHD ($p<0.05$)

In a study carried out by Nafisa C Vaz, et al to determine the prevalence of diabetes mellitus (DM) and its associated diabetic complications in rural Goa, India, they found that among 1,266 study participants about 130 (10.3%) were diabetics. The prevalence of CHD (32.3%) was higher among the diabetics compared to non- diabetics (3.3%). In the study carried out by Deepa D. V., et al in 100 newly diagnosed type 2 DM patients they found prevalence of CAD as 26 % and ECG findings were normal in 74 cases, myocardial infarction (MI), left bundle branch block, left ventricular hypertrophy (LVH) in three cases each, old MI in seven cases, ischemic heart disease in six cases and arrhythmias in two cases. 2D- echocardiography showed regional wall motion abnormality in 23 cases, hypertensive heart disease in eight cases, concentric LVH and ischemic dilated cardiomyopathy in one caseeach.

In study carried out by Pekka Koskinen, et al they found compared with nondiabetic subjects, non insulin-dependent diabetic patients (NIDDM) had higher triglyceride concentration ($P< 0.0001$), lower HDL cholesterol ($P < 0.001$), and greater BMI ($P < 0.001$), there were more hypertensive patients among them ($P < 0.001$). The incidence of myocardial infarction and cardiac deaths were significantly higher among diabetic than nondiabetic participants (7.4 vs. 3.3%, respectively($P < 0.02$). In the present study, PVD was found in 25 out of 100 patients, 20 patients out of 50 in cases i.e. 40% and in controls it was found in 5 out of 50 patients i.e.10%. Evidence of PVD was found more in cases as compared to controls with statistical significance($p<0.05$).

In study carried out by Hiren P Pandya, et al they concluded On analysis of ABI among patients with diabetes mellitus, out of 50 patients, 9 (18%) were found not to have PAD (ABI < 1) and their lipid profile was within normal range, whereas 41 (82%) were found to have ABI of >1, and on comparative analysis of lipid profile, their TC, triglyceride, and LDL were significantly higher than those of patients having ABI of <1 ($P < 0.05$). In a study carried out by Samson Okello, et al they found the prevalence of PAD (ABI of < 0.9) was 24%. Among the patients with PAD, 87% had mild PAD (ABI 0.71-0.90) while 13% had moderate to severe PAD (ABI < 0.70). In this study we found CVA in 7 out of 100 patients, in cases evidence of CVA was found in 6 patients out of 50 i.e. 12% and in controls it was found in 1 patient out of 50 patients i.e. 2%. Evidence of CVA is found more in cases compared to controls with statistical significance. ($p<0.05$).The overall prevalence of stroke found was 7 out of 100 patients (7%) out of which 6 patients had an ischemic stroke and 1 had a hemorrhagic stroke.

In a study carried out by Nafisa C Vaz, et al To determine the prevalence of diabetes mellitus (DM) and its associated diabetic complications in rural Goa, India they found Among the total 1,266 study participants about 130 (10.3%) were diabetics And prevalence of cerebrovascular accidents (CVAs) was 6.9%. Carlo Bruno Giorda et al in a study prospectively followed-up 14 432 type 2 DM patients, aged 40 to 97 years. During a 4-year follow-up, they found 296 incident stroke events were recorded. In subjects with no history of cardiovascular disease, the age-standardized incidence of stroke was 5.5 in men and 6.3 in women (per 1000 person-years). In subjects with a history of cardiovascular disease, it was 13.7 in men and 10.8 in women. The ratios of incidence of stroke varied according to the age, sex, and history of cardiovascular disease. Among men with no history of CVD, HbA1c and smoking were the predictors of stroke. Among subjects with history, the risk factors in men were, therapy with oral agents plus insulin, treated high total cholesterol and a low HDL cholesterol, whereas in women microvascular complications were a risk factor. The previous history of CVA was a strong predictor of stroke in both sexes.

In a study carried out by Seppo Lehto, et al they concluded that the risk of stroke innon-insulin dependent diabetes mellitus(NIDDM) men was about threefold and in NIDDM women fivefold higher than that in corresponding nondiabetic individuals. Low levels of HDL cholesterol (<0.90 mmol/L), high levels of total TGs (>2.30 mmol/L), and the presence of hypertension(HTN) were associated with a twofold increase in the risk of stroke mortality or morbidity. In a study carried out by Jasmina Djelilovic-Vranic, et al Ischemic stroke was confirmed in 78.0%, of which 32% were lacunar infarcts and 22% hemorrhagic. The most common risk factors were hypertension 85%, then smoking in 65%, diabetes mellitus in 39.0%, in 27.38% dyslipidemia, the previous stroke in 26.69%, in 23.57% arrhythmia. In the baseline sample, 30.06% of patients had previously diabetes mellitus and in 8.94% the diabetes was diagnosed during hospitalization, while dyslipidemia was known from earlier in 22.0% and in 5.38% cases was

detected during the hospitalization.

7. Summary

This study was conducted in the department of medicine, Rajarajeswari medical college and hospital Bangalore. In the present study 100 type 2 diabetic patients, divided into 50 cases and 50 controls satisfying inclusion criteria were considered.

The findings are summarized as follows:-

1. A maximum number of diabetic individuals belonged to the age group 56-65 years(39 out of 100patients i.e. 41%). A maximum number of cases were in the age group of 56-65 years(22 out of 50 patients i.e.44%), while the maximum number of controls belonged to 46-55years age group (21 out of 50patients i.e. 42%).
2. The maximum number of patients belonged to urban areas (73 out of 100 patients i.e.73%). Both in control and cases maximum no of patients were from urban areas(38 and 35 patients in controls and cases respectively).
3. Past history of HTN, IHD, PVD and CVA were significantly more in cases as compared to controls, statistical significance was found (P<0.05).
4. More patients in cases were on irregular anti-diabetic treatment as compared to controls (15 patients in cases and 2 in controls).
5. Abnormal ECG findings in our study were mainly IHD changes and LVH by voltage criteria.
6. IHD changes were found in 8 cases and 3 controls, LVH by voltage criteria was found in 15 cases and 8 controls. IHD changes found in cases and controls are 16% and 6% respectively. LVH changes found in cases and controls are 30% and 16% respectively.
7. Abnormal 2D Echo findings in our study were mainly IHD and LVH. IHD was found in 11 cases and 3 controls, LVH was found in 18 cases and 10 controls. IHD changes found in cases and controls are 22% and 6% respectively. LVH changes found in cases and controls are 36% and 20% respectively.
8. There was a significant elevation of mean values of total serum cholesterol, LDL and TG of cases and controls in the postprandial state compared to their fasting state, statistical significance was found (P<0.05).
9. Mean values of total serum cholesterol, LDL and TG of cases in fasting state were significantly more compared to controls in fasting state, statistical significance was found (P<0.05).
10. Mean values of total serum cholesterol, LDL and TG of cases in the postprandial state were significantly more compared to controls in the postprandial state, statistical significance was found (P<0.05).
11. Mean values of both FBS and PPBS in cases were higher compared to controls (P<0.05). Mean values of PPBS in cases and controls were significantly more compared to their respective FBS values (P<0.05).
12. The mean HbA1c values in cases were higher(7 142 compared to controls (5 554
13. Ischemic Heart disease was found in 14 patients i.e.14% of the study group. In cases, it was found in 11 patients and in controls, it was found in 3 patients i.e. 22% and 6% respectively. The occurrence of IHD was more in cases compared to controls with statistical significance(p<0.05).
14. Peripheral vascular disease in our study was found in 25 patients i.e.25% of the study group. In cases, it was found in 20 patients and in controls, it was foundin 5 patients i.e. 40% and 10% respectively. The occurrence of PVD was more in cases compared to controls with statistical significance(p<0.05).
15. Stroke in our study is found in 7 patients i.e. 7% of the study group. In cases, it was found in 6 patients and in controls, it was found in 1 patient i.e. 12% and 2% respectively. The occurrence of CVA was more in cases compared to controls with statistical significance(p<0.05).Ischemic strokes were more common than haemorrhagic strokes.

8. Conclusion

1. Prevalence of diabetes was highest in the age group 56-65 years in our hospital
2. As the duration of diabetes increases, there is an increased prevalence of dyslipidemia in the cases.
3. Past history of HTN, IHD, PVD and CVA were found significantly more in subjects with fasting and postprandial dyslipidemia (cases) compared to those without(controls).
4. Patients on irregular treatment (63%) were more in the study group(cases) compared to controls. So it could be said that patient not on regular treatment are more prone to have dyslipidemia. (fasting as well as postprandial).
5. There was a significant increase in total serum cholesterol, LDL and TG in postprandial states of cases compared to that in controls, so it could be said that diabetic patients with fasting dyslipidemia are more prone to have dyslipidemia in the postprandial state.
6. Cases were having significantly higher FBS and PPBS compared to the controls, so it could be said that as blood sugar increases the occurrence of dyslipidemia increases.
7. HbA1c was significantly higher in cases compared to controls, so it could be said that as HbA1c increases there is an increase in the occurrence of dyslipidemia.
8. It could be said that there is an increase in the occurrence of postprandial dyslipidemia with

increasing age, irregular treatment, increase in HbA1c, FBS ,PPBS and with the past history of HTN, CVA, PVD and IHD.

9. All macrovascular complications (IHD, CVA, PVD) were found more in the case compared to controls with statistical significance. So it could be said that there is an increase in the occurrence of macrovascular complications with an increase in postprandial dyslipidemia.
10. It could be said that there is an increase in the occurrence of macrovascular complications with an increase in postprandial dyslipidemia, increasing age, irregular treatment and increase in HbA1c, FBS and PPBS

9. Source of Funding

None.

10. Conflict of Interest

None.

References

1. Voukali M, Kastrinelli I, Stragalinou S, Tasiopoulou D, Paraskevopoulou P, Katsilambros N, et al. Study of Postprandial Lipaemia in Type 2 Diabetes Mellitus: Exenatide versus Liraglutide. *J Diab Res*. 2014;2014:1–7. doi:10.1155/2014/304032.
2. Suryabhan L, Chandrashekhar MI, Ratnendra RS. A Comparative Study on the Fasting and the Postprandial Dyslipidaemia in Type 2 Diabetes Mellitus. *J Clin Diagn Res*. 2013;7(4):627–30. doi:10.7860/JCDR/2013/4845.2868.
3. Das S. Current understanding of risk factors and mechanisms in the pathogenesis of macrovascular disease in diabetes mellitus. *Indian Acad Clin Med*. 2001;2(3):214–21.
4. Diagnosis and classification of diabetes mellitus. *Am Diab Assoc*. 2013;36(1):67–74. doi:10.2337/dc13-S067.
5. Raj S, Rajasekharan C, Jayakumar B. Postprandial hypertriglyceridaemia in type 2 diabetic subjects. *Int J Diab Dev* . 2006;26(4):160. doi:10.4103/0973-3930.33182.
6. Madhu SV, Mittal V, Ram K, Srivastava B, K D. Postprandial lipid abnor malities in type 2 diabetes mellitus. *J Assoc Physicians India*. 2005;53:1043–6.
7. Alvin C. Chapter 396: Diabetes Mellitus: Diagnosis, Classification, and Pathophysiology; 2021. Available from: https://accessmedicine.mhmedical.com/book.aspx?bookid=2129.
8. Polonsky KS. The Past 200 Years in Diabetes. *New England Journal of Medicine*. 2012;367(14):1332–1340. Available from: https://dx.doi.org/10.1056/nejmra1110560. doi:10.1056/nejmra1110560.
9. Poretsky L. Principles of Diabetes Mellitus; 2009. p. 978. Available from: https://www.springer.com/gp/book/9780387098418.
10. Schofield JD, Liu Y, Balakrishna PR, Malik RA, Soran H. Diabetes Dyslipidemia. *Diabetes Ther*. 2016;7(2):213–19. doi:10.1007/s13300-016-0167-x.

Author biography

Ram Chaitanya K, Senior Resident

Ayyali Ambresh, Assistant professor

Cite this article: Chaitanya K R, Ambresh A. Study of fasting and post prandial lipid abnormalities in type 2 diabetes mellitus in comparison to controls. *J Prev Med Holistic Health* 2021;7(1):45-52.