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Original Research Article

A Study of Correlation of Postprandial Lipid Profile with Carotid Intima Media Thickness in Type 2 Diabetes Mellitus Patients

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Conflict of interest: Nil

Abstract

Background: Study of Correlation of Postprandial Lipid Profile with Carotid Intima Media Thickness in Type 2 Diabetes Mellitus patients.

Methods: In this comparative study in 150 type 2 diabetic patients with good control, duration of disease less than 5 years, with age from 30 to 50 years, body mass index less than 30 and non-smokers, in Department of Medicine, JLN Medical College, Ajmer (Rajasthan) between September 2021 September 2022 with an aim to estimate the fasting Lipid Profile and postprandial Lipid Profile levels Carotid intima media thickness (CIMT) by Doppler ultrasonography and the correlation between variability of fasting Lipid Profile and postprandial Lipid Profile with Carotid intima media thickness (CIMT) in patients with type-2 Diabetes mellitus with duration of diabetes less than 5 years, were studied. They were categorized into three groups namely NN group; NA group and AA group. **NN Group** included whose both fasting and postprandial lipid profile is normal, **NA Group** included whose fasting lipid profile is normal and post-prandial lipid profile is abnormal and finally **AA Group** included whose both fasting and post-prandial lipid profile is abnormal.

Results: The highest number of cases with high CIMT were found in Group AA ie (37) 50% followed by Group NA (29) 72.50%, and Group NN (7)19.44%. High. Significant A mean SD difference was observed among the groups. In groups AA, AN, and NN, the values were 1.27±0.16, 1.01±0.16, and 0.75±0.07, respectively. A significantly positive correlation was found between CIMT and postprandial serum lipid profile parameters viz. CIMT and postprandial Total Cholesterol; CIMT and postprandial LDL; CIMT and postprandial TG; CIMT and postprandial VLDL and negative correlation was found between CIMT and postprandial HDL.

Conclusion: There is a strong correlation between carotid intima media thickness and both fasting and postprandial lipid levels. However, postprandial lipid levels have a greater connection with carotid intima media thickness than fasting lipid levels do. Significant

postprandial lipid abnormalities, notably postprandial hypertriglyceridemia, are present in type 2 DM patients. Age, duration of disease, fasting triglycerides and postprandial triglycerides have a significant impact on the CIMT, whereas other variables do not have any significant impact on CIMT. Therefore, we recommend measurement of postprandial lipid profile and CIMT as a part of routine investigatory workup in type 2 diabetes mellitus patients.

Keywords: Type-DM, CIMT(Carotid Intima Media Thickness), RBS, Postprandial Lipid Profile.

Introduction

Diabetes mellitus comprises a group of common disorders that share the phenotype of hyperglycemia. Diabetes has emerged as a main contributor to coronary heart disease (CHD). Cardiovascular disease appears to be the major cause of morbidity and mortality in diabetes. Approx 80% of mortality in these patients are due to macrovascular complications caused by DM. It increases the risk of coronary artery disease by 2-3 folds. [1]

Studies initially showed LDL is recognized as important factor for premature macrovascular changes. Many studies show and association of fasting triglycerides and increased coronary risk but only few studies are there on the role of postprandial triglycerides. Diabetes is mainly a postprandial metabolic disorder. Serum triglycerides found to rise 3-6 hours after a meal, more so in diabetics. Once postprandial triglyceridemia occurs it is further exacerbated by the next meal. As we stay in postprandial state most of the day due to frequent meal therefore postprandial triglyceridemia is sustained. Therefore, circulation of the human body is exposed to the elevated triglycerides for a major part of the day. Therefore, post-prandial triglycerides may have a greater role in the premature atherosclerotic process than the fasting triglycerides. Patients with early atherosclerotic changes are usually asymptomatic. Only when the vascular occlusion reaches a significant level, they will become symptomatic. [2]

An early sign of atherosclerosis is hypertrophy of the arterial wall. Carotid intima-media thickness (CIMT) is a non-invasive, inexpensive, rapid and reproducible measure and increased CIMT, a proxy for carotid atherosclerosis, is a significant determinant of CVD and stroke risks. Increased CIMT has been accepted as a marker of early atherosclerosis Type 2 diabetes is related to an increased risk of atherosclerotic diseases and an increased cardiovascular risk has been observed in individuals with elevated glucose levels below the diabetic range. Finally, previous studies showed that impaired glucose tolerance was associated with increased CIMT. [3]

Material and Methods

The study was designed as comparative study conducted at Department of Medicine, Jawahar Lal Nehru Medical College, Ajmer, Rajasthan and 150 Type 2 diabetic mellitus patients were studied during September 2021 to September 2022.

In our study, to reduce confounding in correlation, we took certain demographic and clinical criteria as inclusion criteria.

These Inclusion criteria inter alia includes duration of type-2 Diabetes mellitus less than 5 years, HbA_{1c} less than 7.5 (ie DM with good control, age 30-50 years and patients without macrovascular complications.

Further, in order to reduce confounding and find true correlation between lipid profile parameters and CIMT we have taken

umpteen of factors in exclusion criteria. This included all the factors which directly or indirectly affect either the process of atherosclerosis or cause anomalous presentation of lipid profile.

These Exclusion criteria inter alia includes Patients with Type-1 diabetes mellitus; Patients receiving lipid lowering agents, thiazides, beta blockers, oral contraceptive drugs (OCP), steroids etc.; Patients who are known cases of hypothyroidism, Patients with liver disease, chronic alcoholism as determined by history and investigations; Patients who will have history/clinical findings/investigations suggestive of familial Hyperlipidemias; Chronic Smokers; Diagnosed cases of cerebrovascular accident, ischemic heart disease, peripheral vascular disease etc.; Cases of secondary diabetes mellitus; Cases of hypertension mediated organ disease (HMOD), chronic kidney disease (CKD), gestational diabetes mellitus, uncontrolled hypertension, nephrotic syndrome, Cushing's syndrome etc. ; Obese patients ie. whose BMI (Body Mass Index) is more than 30 kg/ m^2 ; and Patients not willing for study (not given consent).

Methodology

It is a comparative study in 150 type 2 diabetic patients of age less than 50 years. With duration of diabetes less than 5 years, were studied. This study was done at Department of Medicine, JLN Medical College, Ajmer between September 2021 September 2022.

Patients receiving Oral hypoglycemic agents, antihypertensives (ACE inhibitors or AT II antagonists etc.) and antiplatelet drugs were not excluded from the study. Maximum Body Mass index (BMI) was 30 Kg/ M^2. They were categorized into three groups.

Group I were included whose both fasting and postprandial lipid profile is normal (i.e. NN Group);

Group II were included whose fasting lipid profile is normal and post-prandial lipid profile is abnormal.(i.e. NA group)

Group III were included whose both fasting and post-prandial lipid profile is abnormal.(i.e. AA Group)

Data analysis and Results

Table 1: Distribution of cases according to status of diabetes

Variable	Group AA	Group AN	Group NN	Total
Diabetes prevalence	74(49.33%)	40(26.67%)	36(24.00%)	150
Age in years	44.15±3.77	43.40±3.862	42.67±3.906	43.59±3.853
Male: female	54:20	29:11	19:17	48:102
BMI in kg/mt^2	27.281±2.50	25.018±1.25	23.351±1.32	25.734±2.56
Random Blood Sugar	214.89±39.08	215.725±42.19	195.86±29.90	210.45±38.63
Fasting	181.73±38.09	178.73±33.32	172.08±28.87	178.61±34.82
PPBS	226.31±39.68	225.30±40.00	213.22±29.00	222.90±37.65
Random Blood Sugar	214.89±39.08	215.725±42.19	195.86±29.90	210.45±38.63
HDL	29.42±6.86	32.78±4.32	42.53±5.76	33.55±7.74
LDL	180.21±48.53	121.74±6.44	109.50±10.40	155.97±48.68
TG	253.57±64.44	143.18±16.69	117.81±14.96	191.55±77.54
VLDL	50.71±9.06	28.64±3.34	23.56±2.99	38.31±15.51
TG/HDL	9.06±3.44	4.50±0.75	2.83±0.61	6.35±3.68
HDL	29.42±6.86	32.78±4.32	42.53±5.76	33.55±7.74
CIMT	1.27±0.16	1.01±0.16	0.75±0.07	1.07±0.25

Table 2: Comparative Analysis of B/L Carotid Artery Doppler measuring Carotid Intima Media Thickness CIMT with Various Variable Under Study

CIMT With	R	Sig. (2-tailed)
AGE	0.134	0.103
Waist :Hip Ratio	0.647**	<0.001S
Blood Sugar		
Random Blood Sugar	0.072	0.383
Fasting Blood Sugar	0.000	0.996
PPBS Blood Sugar	-0.011	0.898
HbA1c	0.672**	<0.001S
Fasting lipid profile		
SCH	0.830**	<0.001S
HDL	-0.658**	<0.001S
LDL	0.775**	<0.001S
TG	0.809**	<0.001S
VLDL	0.809	<0.001S
TG/HDL	0.796	<0.001S
Postprandial lipid profile		
SCH	0.833**	<0.001S
HDL	-0.633**	<0.001S
LDL	0.787**	<0.001S
TG	0.816**	<0.001S
VLDL	0.816	<0.001S
TG/HDL	0.799	<0.001S

Discussion

In the present study, correlation of CIMT with blood sugar were studied that the Pearson correlation (r = 0.072, P = 0.38 NS) was found to be weakly positive but not significant. With fasting blood sugar (0.99 NS) and with PPBS blood sugar (P = 0.898 NS). CIMT and random blood sugar were 1.0740.25 and 210.41337.65 units, respectively. The Pearson correlation (r = 0.072, P = 0.38 NS) was found to be weakly positive but not significant. With fasting blood sugar (0.99 NS) and with PPBS blood sugar (P = 0.898 NS), our result is in concordance with R Pathak et al. (2018) [4] where there was no statistical correlation between the FBS and CIMT (r = 0.04; P 0.05) but it was observed that the PPBS correlated significantly with the CIMT (r = 0.442; P 0.01S).

In the present study, the Pearson correlation between CIMT with HbA1c was found to be positive and significant (r = 0.672, P <0.001S). It is in agreement with study conducted by Jamal Ahmad et al 2005[5] observed that HbA_{1C} was correlated with CIMT. Significant correlation was observed between the total cholesterol levels (P 0.05) with the CIMT. The mean SD of CIMT, fasting and postprandial SCH were 1.074±0.257, 213.27±60.786 and 219.51±60.85 respectively. The Pearson correlation (r = 0.830 and 0.833, respectively, P = 0.001) was found to be significant and strongly positive for CIMT with SCH. R Pathak et al 2018[4] observed that no correlation was observed between the total cholesterol levels, either fasting (p0.05), with the CIMT. Similarly, Katherine Esposito et al 2008[9] also found and CIMT did not correlate with total cholesterol levels (r=-0.02; p=0.82).

This study shows the correlation of CIMT with LDL (fasting and postprandial). The mean SD of CIMT, fasting, and postprandial LDL were 1.074±0.25, 155.97±48.68 and 145.73±51.75,

respectively. The Pearson correlation (r = 0.775 and 0.787 respectively, P = 0.001) was found to be strongly positive and significant. S Teno et al (2000) observed that fasting LDL cholesterol levels was all independently correlated with CIMT. In R Pathak et al 2018 [4] study No statistical correlation was found between the fasting and postprandial LDL levels with the CMIT (p>0.05) However, Jamal Ahmad et al 2005 [5] observed that fasting LDL-cholesterol, was independently correlated with CIMT, had the strongest statistical influence.

This study shows the correlation of CIMT with HDL (fasting and postprandial). The mean SD of CIMT, fasting, and postprandial HDL were 1.074±0.25, 33.55±7.74 and 32.15±7.79, respectively. The Pearson correlation (r = -0.658 and -0.633, respectively, P = 0.001) was found to be strongly negative and significant. HDL levels with the CIMT which was supported by the study of Katherine Esposito et al in 2008 who reported that CIMT did not correlate with high-density lipoprotein (HDL)-cholesterol levels (r=-0.03; p=0.67).

The mean SD of CIMT, fasting, and postprandial TG were 1.074±0.25, 191.55±77.54 and 211.40±75.84 respectively. The Pearson correlation (r = 0.809and 0.816respectively, P = 0.001) was found to be strongly positive significant. In the present study, it was observed that although both FTG and PPTG levels are significantly correlating with CIMT but the PPTG levels (R=0.816) were more significantly correlating with CIMT as compared to the FTG levels (R =0.809). This observation is supported by various authors. Teno S et al. 2000 [6] found that the CIMT of the patients with fasting hypertriglyceridemia was greater than that of the patients with normal FTG levels (P = 0.02). Mala Dharmalingam et al in 2004 [7] also found that fasting triglycerides correlated significantly with carotid intima-media thickness. R Pathak et al 2018 [4] observed that CIMT increased with increase in the PPTG levels and there exists a significant correlation between CIMT and PPTG (P<0.001S) It was also discovered that as FTG levels increased, so did CIMT, with a statistically significant correlation between CIMT and FTG levels (P <0.001S). CIMT was higher in the patients with fasting hypertriglyceridemia as compared to those with normal fasting triglyceride levels. [8,9]

Conclusion

There is a strong correlation between carotid intima media thickness and both fasting and postprandial lipid levels. However, postprandial lipid levels have a greater connection with carotid intima media thickness than fasting lipid levels do. Significant postprandial lipid abnormalities, notably postprandial hypertriglyceridemia, are present in type 2 DM patients. Age, duration of disease, fasting triglycerides and postprandial triglycerides have a significant impact on the CIMT, whereas other variables do not have any significant impact on CIMT.

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Availability of data and materials: From 150 patients studied at Department of General Medicine, JLN Medical college, Ajmer. (Raj) with informed consent.

Contributions: Dr. Siddharth Bharatiya prepared the manuscript with help from all other authors Dr Maniram Kumhar, Dr Harsh Tak ,Dr. Deepak etc under the meticulous guidance of his mentor professor Dr Maniram Kumhar with co-guidance of Dr. Harsh Tak, Dr. VB Singh, Dr. Mayank Srivastav, Dr. Ravi Gaur.

Ethics Declarations

Ethics approval and consent to participate: Written and informed consent was taken from all the participants. An approval from Ethical Committee of the this institution was duly obtained.

Consent for Publication: Written and informed consent was obtained from concerned patients.

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