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Correlation of fasting and postprandial triglyceride levels and carotid artery intimal medial thickness in type 2 diabetic patients: A cross sectional study

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***Abstract*---**Background: Although the prevalence of both type 1 and type 2 diabetes mellitus is increasing worldwide, the prevalence of type 2 DM is rising more rapidly, because of sedentary lifestyle, increasing obesity and increased life expectancy. Objectives: To study correlation between fasting and postprandial triglyceride levels and Carotid artery intimal medial thickness (CIMT) in Type 2 Diabetic Patients. Material and Methods: This cross sectional observation study was done in Department of General Medicine, Mysore Medical College and Research Institute, Mysore, Karnataka, India during January 2019 to June 2020. Sample size was 50 and Simple Random sampling method was used. Results: The mean carotid IMT of subjects with postprandial triglycerides < 200 with normal fasting triglyceride was 0.92±0.33. The mean carotid IMT of subjects with postprandial triglyceride 200-299 with normal fasting triglyceride was 1.52±0.57. The mean carotid IMT of subjects with postprandial triglycerides ≥300 with normal fasting triglycerides was 1.66±0.45. Conclusion: postprandial hypertriglyceridemia, despite normal fasting triglyceride levels, may be an independent risk factor for early

atherosclerosis in type 2 diabetes. Hence evaluating not only for FTG but also PPTG level during clinical assessment of patients with type 2 diabetes is important.

***Keywords*---**Type 2 Diabetes Mellitus, Fasting triglyceride level, Postprandial triglyceride level.

Introduction

Diabetes mellitus is a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action or both. Chronic hyperglycemia of diabetes is associated with long term damage, dysfunction and failure of various organs especially eyes, kidneys, nerves, heart and blood vessels.[1]

T2D exerts a huge toll in human suffering and economy. A recent evaluation using a computerized generic formal disease model revealed that the total number of people with diabetes is projected to rise from 171 million in 2000 to 366 million in 2030, with India, China and USA being the top 3 countries estimated to have the highest numbers of people with diabetes. [2]

The pathogenesis of T2DM is complex and involves the interaction of genetic and environmental factors. From a pathophysiologic standpoint, persons with T2DM consistently demonstrate eight cardinal abnormalities:

Type 2 diabetes is associated with development of premature atherosclerosis. Diabetic dyslipidemia is believed to play an important role in the pathogenesis of accelerated atherosclerosis. The most important components of this dyslipidemia are elevated very low-density lipoproteins (VLDL), total triglycerides (TGs) and a decreased high-density lipoproteins (HDL) concentration in the serum. Several studies have proved that in type 2 diabetes, elevated triglyceride levels may be a better predictor of ischemic heart disease (IHD) than elevated low-density lipoprotein (LDL) cholesterol levels. Fasting hypertriglyceridemia has been consistently shown to be associated with a greater risk for coronary artery disease and atherosclerosis in those with type 2 diabetes.

Atherosclerosis, unless in a severe form, is often asymptomatic, so that a direct examination of the vessel wall is necessary to detect affected individuals in the early stages. Measurement of the intimal-medial thickness (IMT) of the common carotid artery (CCA) by B-mode ultrasound was found to be a suitable noninvasive method to visualize the arterial walls and to monitor the early stages of the atherosclerotic process. Carotid intima media thickness is considered as a surrogate marker of atherosclerosis. An increased carotid IMT was observed in type 2 diabetic patients. This increase in carotid intima media thickness is associated with an increased risk of Ischemic heart disease (IHD) and cerebrovascular disease (CVD) in diabetics. [3] Hence this study was conducted to study the correlation between fasting and postprandial triglyceride levels and Carotid artery intimal medial thickness (CIMT) in Type 2 Diabetic Patients.

Materials and Methods

This cross sectional observation study was done in Department of General Medicine, Mysore Medical College and Research Institute, Mysore, Karnataka, India during January 2019 to June 2020. The patients of type 2 DM of age between 35-75 years with duration > 1 year admitted to KR Hospital, Department of General Medicine, Mysore Medical College and Research Institute, Mysore, Karnataka, India. Sample size was 50 and Simple Random sampling method was used. Permission for the study was obtained from the College authorities prior to commencement.

Sample size

Sample size calculated is 44 with level of significance (α) 5% and absolute error(d) 14% with prevalence of hypertriglyceridemia in T2DM as 66% using the formula $4PQ/d^2$. Where P is prevalence, Q is 1-P, d = 14% (margin of error). Sample size was rounded to 50. Simple Random sampling method was used.

Inclusion Criteria: Patients of type 2 DM of age between 35-75 years with duration > 1year.

Exclusion Criteria:

- Patients with familial hypertriglyceridemia
- Patients who are alcoholics and smokers
- Those patients with history or clinical evidence of IHD
- Hypertenion, cerebrovascular accident and peripheral vascular disease.
- Patients who are on lipid lowering therapy
- Patients who are on drugs affecting lipid metabolism

Study tools and Data collection procedure:

Detailed history, clinical examination and following investigations was done.

Investigations

- Fasting Triglycerides.
- Postprandial Triglycerides.

Blood samples were obtained after an overnight fasting and then the patients ate a standard meal that had a total energy of 9Kcal/Kg with 60-65% of energy being supplied by carbohydrate, 15-20% by protein and 20% by fat after taking insulin or oral hypoglycaemic agents. Blood samples were taken again 4 hrs after the meal.

Carotid Artery Intimal Thickness was estimated by Carotid Artery Doppler by B-mode ultrasound using 7.5 MHZ transducer with annular array ultrasound imaging system

Patients were divided into three groups.

The first group is normo-hyper (NH) group i.e. patients having normal fasting but high postprandial triglyceride levels.

Second group is normo-normo (NN) group i.e. patients having normal fasting and postprandial triglyceride levels.

Third group is hyper-hyper (HH) group i.e. patients with high fasting and postprandial triglyceride levels.

Statistical Analysis

The data was collected, compiled and compared statistically by frequency distribution and percentage proportion. Quantitative data variables were expressed by using Descriptive statistics (Mean ± SD). Qualitative data variables were expressed by using frequency and Percentage (%).The level of significance is estimated by applying Chi-square test , p value < 0.05 is taken as significant and <0.01 taken as highly significant..

Result

The age of patients varied from a minimum of 35 years to a maximum of 75 years. Mean age of patients in NH, NN, HH group were 55.12±9.13, 57.63±9.13 and 58.43±10.98 respectively.

Among the total of 50 patients 29 were males and 21 were females. In NH group 12 were males (63.2%) and 7 were females (36.8%). In the NN group 8 were males (47.1) and 9 were females (52.9%). In the HH group 9 were males (57.15%) and 5 were females (42.85%).

The mean duration of diabetes in NH group was 6.85±3.85. The mean duration of diabetes in NN group was 4.94±2.30. The mean duration of diabetes in HH group was 8.21±4.37.

In the NN group 8 patients were on OHA (47.05%), 1 patient was on insulin (5.90%) and 8 patients were on both OHA and insulin (47.03%). In the NH group 7 patients were on OHA (36.84%), 4 patients were on insulin (21.05%) and 8 patients were on both insulin and OHA (42.11%). In the HH group 7 patients were on OHA (50%), 3 patients were on insulin (21.42%) and 4 patients were on both OHA and insulin (28.58%).

The mean serum fasting triglyceride in the NH group was 116.63± 21.6. The mean serum fasting triglyceride in the NN group was 100.41± 22. The mean serum fasting triglyceride in the HH group was 193.71± 42.27. Table 1

Table 1: Distribution of subjects by serum fasting triglyceride in different groups

Serum fasting triglycerides (mg/dl)	NH group		NN group		HH group	
	No.	%	No.	%	No.	%
<150	19	100	17	100	0	
150-249	0	0	0	0	12	85.7
≥250	0	0	0	0	2	14.3
Total	19	100	17	100	14	100
Mean (NH)	116.63		100.41		193.71	
Std.Dev.	21.16		22.00		42.27	

F = 4; p < 0.01

The mean serum postprandial triglyceride in NH group was 276.10± 61.80. The mean serum postprandial triglyceride in NN group was 153.64 ± 31.15. The mean serum postprandial triglyceride in HH group was 291.21 ± 63.90. Table 2

Table 2: Distribution of postprandial triglyceride among subject groups

Serum postprandial triglycerides (mg/dl)	NH group		NN group		HH group	
	No.	%	No.	%	No.	%
< 200	0	-	17	-	0	-
200-299	13	68.4	0	-	9	-
300-399	5	26.3	0	-	4	28.6
≥400	1	5.3	0	-	1	7.1
Total	19	100	17	100	14	100
Mean	276.10		153.64		291.21	
Std.Dev.	61.80		31.15		63.90	

F =6, P <0.01

The mean serum fasting triglyceride in the NH group was 116.63± 21.6. The mean serum fasting triglyceride in the NN group was 100.41± 22.00. The mean serum fasting triglyceride in the HH group was 193.71 ± 42.27. Table 3

Table 3: Distribution of subjects by serum fasting triglyceride in different groups

Serum fasting triglycerides (mg/dl)	NH group		NN group		HH group	
	No.	%	No.	%	No.	%
<150	19	100	17	100	0	
150-249	0	0	0	0	12	85.7
≥250	0	0	0	0	2	14.3
Total	19	100	17	100	14	100
Mean (NH)	116.63		100.41		193.71	
Std.Dev.	21.16		22.00		42.27	

F = 4; p < 0.01

The mean serum postprandial triglyceride in NH group was 276.10 ± 61.80. The mean serum postprandial triglyceride in NN group was 153.64 ± 31.15. The mean serum postprandial triglyceride in HH group was 291.21 ± 63.90. Table 4

Table 4: Distribution of postprandial triglyceride among subject groups

Serum postprandial triglycerides (mg/dl)	NH group		NN group		HH group	
	No.	%	No.	%	No.	%
< 200	0	--	17	--	0	--
200-299	13	68.4	0	--	9	--
300-399	5	26.3	0	--	4	28.6
≥400	1	5.3	0	--	1	7.1

Total	19	100	17	100	14	100
Mean	276.10		153.64		291.21	
Std. Dev.	61.80		31.15		63.90	

F =6; p < 0.01

The mean carotid IMT of subjects with fasting triglyceride < 150 was 1.21±0.48. The mean carotid IMT of subjects with fasting triglyceride 150-249 was 1.78±0.47. The mean carotid IMT of subjects with fasting triglyceride 250-349 was 1.83±0.34. The mean carotid IMT of subjects with fasting triglyceride ≥ 350 was 1.37±0.46. Table 5

Table 5: Distribution of fasting triglycerides with CIMT

Fasting triglycerides (mg/dl)	Subjects	
	Number	CIMT (mean) in mm
<150	36	1.21±0.48
150-249	11	1.78±0.47
250-349	3	1.83±034
Total	50	1.37±0.46

F = 2.78; p < 0.05

The mean carotid IMT of subjects with postprandial triglyceride < 200 was 0.98 ±0.41. The mean carotid IMT of subjects with postprandial triglyceride 200-299 was 1.60 ± 0.61. The mean carotid IMT of subjects with postprandial triglyceride 300-399 was 1.71 ± 0.49. The mean carotid IMT of subjects with postprandial triglyceride ≥ 400 was 1.40 ± 0.59. Table 6

Table 6: Distribution of postprandial triglycerides with CIMT

Postprandial triglycerides (mg/dl)	Number	CIMT (Mean) (mm)	Standard Deviation
< 200	17	.9859	.41836
200-299	22	1.6009	.61237
300-399	9	1.7133	.49066
>400	2	1.4650	.33234
Total	50	1.4066	.59599

F = 5.66; p < 0.01

The mean carotid IMT of subjects with postprandial triglycerides < 200 with normal fasting triglyceride was 0.92±0.33. The mean carotid IMT of subjects with postprandial triglyceride 200-299 with normal fasting triglyceride was 1.52±0.57. The mean carotid IMT of subjects with postprandial triglycerides ≥300 with normal fasting triglycerides was 1.66±0.45 Table 7

Table 7: Distribution of postprandial triglyceride levels with CIMT in patients with normal fasting triglyceride level

Postprandial triglycerides (mg/dl)	Subjects	
	Number	CIMT (mean) in mm
<200	17	0.92 ±0.33
200-299	14	1.52±0.57
≥300	5	1.66±0.45

F = 2.83 p <0.05

Discussion

Fasting triglycerides and postprandial triglyceride levels were estimated. Patients were divided into three groups. Although several studies have shown that fasting triglyceride levels to be associated with coronary artery disease in both diabetic and non-diabetic subjects, relatively little attention has been given to postprandial triglycerides in this regard, especially in diabetic subjects. In the present study out of 50 patients, 22 were on OHA, 8 were on insulin and 20 were on both OHA and insulin. In a study done by Shinichi Teno 35 patients were on OHA and 8 patients were on insulin. [4]

In the present study the mean serum fasting triglyceride in the NH, NN, HH groups were 116.63±21.16, 100.41±22.00, 193.71±42.27 respectively. The mean serum postprandial triglycerides in NH, NN and HH group were 276.10 ± 61.80, 153.64±31.15, 291.21 ±63.90 respectively. In a study done by Shinichi Teno The mean serum fasting triglycerides in the NN, NH and HH group were 111.1 ± 39.9, 125.3±21.3 and 270.2 ±110.2 respectively. The mean serum postprandial triglycerides in the NN, NH and HH group were 145.5 ± 44.4, 263.1 ± 42.6 and 391.9 ± 23.7 respectively.

In this study, it was seen that high PPTG levels was significantly associated with CIMT in patients with normal FTG level ($p < 0.05$). Also inspite of normal FTG levels, PPTG levels were significantly associated with CIMT. PPTG levels were more strongly and independently correlated with CIMT than FTG levels. The results were similar to other studies. Therefore, PPTG may be a better predictor of carotid intima media thickness than FTG in patients with type 2 DM.

In a study done by Shinichi Teno, the mean CIMT in the NN, NH and HH group were 0.73±0.13, 0.86±0.13 and 0.85±0.12 mm respectively. In their study although a positive correlation existed between FTG levels and CIMT in patients with fasting triglyceridemia, no correlation existed in patients who had normal FTG levels. Furthermore, PPTG levels were more strongly and independently correlated with CIMT and FTG levels. These observations are similar to our study.

In a study done by Chen Xiang[5], the CIMT in patients with postprandial hypertriglyceridemia was significantly greater than that in patients with normal PPTG levels (0.90 mm vs 0.81 mm, $p < 0.05$), which remained significant after adjustment for FTG and HDL levels. The result was again similar to our study.

In a study done by Jamal Ahmed[6], the mean CIMT in NN, NH and HH group were 0.59±0.09,0.79±0.09 ($p < 0.01$ versus NN group) and 0.82 ±0.06 mm($p<0.001$ versus NN group) respectively. So in their study., it was observed that CIMT was increased in plasma with postprandial hyper-triglyceridemia despite normal FTG, levels and PPTG levels showed strongest influence on CIMT. These results were similar to our study.

In a study done by Susanna Boquist[7], it was observed that in the postprandial state, the plasma triglycerides, total triglycerides under curve, incremental triglyceride are under curve were significantly associated with CIMT ($p < 0.05$). In a study done by Mohan V[8] on intimal-medial thickness of carotid artery in the south Indian diabetic and non-diabetic, it was shown that the mean intimal-medial thickness of diabetic subjects 0.95±0.31 mm were significantly higher than those of non-diabetic subjects 0.74±0.14 mm with $p>0.01$.

Conclusion

Elevated fasting triglyceride level was significantly correlated with carotid intima media thickness, but more higher correlation was found in this study between elevated postprandial triglyceride level and carotid intima media thickness. Postprandial hypertriglyceridemia, despite normal fasting triglyceride levels, may be an independent risk factor for early atherosclerosis in type 2 diabetes. Hence evaluating not only for FTG but also PPTG level during clinical assessment of patients with type 2 diabetes is important.

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Declarations
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