

Study of Postprandial Triglyceridemia as Novel Marker of Carotid Intima-media Thickness in Patients with Type 2 Diabetes Mellitus

Archana A Aher¹, Sagar A Khandare², Mukund G Upadhyay³, Saransh G Barai⁴

Received on: 21 April 2025; Accepted on: 24 February 2026; Published on: xxxx

ABSTRACT

Introduction: Individuals with type 2 diabetes mellitus (T2DM) may have several forms of dyslipidemia. The most common form of diabetic dyslipidemia is hypertriglyceridemia and reduced high-density lipoprotein (HDL) levels. Postprandial lipid measurement, especially triglyceride (TG) measurement, gives better insight regarding the lipemic status of an individual. Measurement of carotid intima-media thickness (CIMT) of the common carotid artery by B-mode ultrasound has proven to be a noninvasive, quantitative, and easily repeatable method for assessing early-onset atherosclerosis. In the present study, the correlation of postprandial TG (PPTG) levels and CIMT in T2DM patients is seen.

Materials and methods: A total of 80 individuals with T2DM were enrolled in the study based on inclusion and exclusion criteria. Patients were divided into 3 groups, namely normo-normo group (NN)—with normal fasting TG <150 mg/dL and PPTG level <200 mg/dL; normo-hyper group (NH)—individuals with normal fasting (<150 mg/dL) and elevated PPTG level (>200 mg/dL); and hyper-hyper group (HH) group—elevated fasting (>150 mg/dL) and elevated postprandial level (>200 mg/dL). CIMT was measured using ultrasound.

Results: The mean CIMT among patients with NN group was 0.66 ± 0.15 . The mean CIMT among patients with NH group was 1.01 ± 0.15 . The mean CIMT among patients with HH group was 1.26 ± 0.19 . This indicates PPTG level has significant correlation with CIMT. Linear regression analysis showed that PPTG is more significantly correlating with CIMT than FTG level ($r = 0.7569$ vs 0.5585).

Conclusion: CIMT correlates better with PPTG levels; hence, postprandial hypertriglyceridemia can be labeled as an independent predictor of accelerated atherosclerosis in diabetics.

Keywords: Cerebrovascular disease, Coronary artery disease, Diabetes mellitus, Dyslipidemia, Hyperglycemia, Postprandial hypertriglyceridemia, Ultrasound.

The Journal of Medical Sciences (2026): 10.5005/jp-journals-10045-00441

INTRODUCTION

Diabetes mellitus (DM) refers to a group of common metabolic disorders that share the phenotype of hyperglycemia.¹ T2DM is the predominant form of diabetes worldwide, accounting for 90% of cases globally. India is home to 77 million diabetics and is the second largest worldwide, with a prevalence of ~10.4% among the adult population.² Diabetes is responsible for the majority of morbidity and mortality associated with the disease. Macrovascular complications of diabetes include cardiovascular disease, cerebrovascular disease, and peripheral vascular disease. Individuals with type 2 diabetes mellitus (T2DM) may have several forms of dyslipidemia. The most common form of diabetic dyslipidemia is hypertriglyceridemia and reduced high-density lipoprotein (HDL) levels.³ Dyslipidemia that occurs in diabetes mellitus plays an important role in accelerated atherosclerosis. Dyslipidemia is strongly correlated with insulin resistance and hyperinsulinemia, and it persists even after treatment of hyperglycemia.⁴ Traditionally, lipid profile is tested in the fasting state, which represents the lowest value over 24 hours. Postprandial lipid measurement, especially triglyceride (TG) measurement, gives better insight regarding the lipemic status of an individual.⁵ Serum TG level generally increases 3–6 hours after a meal. Once postprandial hypertriglyceridemia occurs, it is exacerbated by the next meal and persists for the entire day. Once elevated, the serum TG level is further stimulated by the next meal, and therefore this lipemic status persists throughout the day (Fig. 1). The vascular tree is exposed to postprandial metabolic

^{1,3,4}Department of Medicine, Government Medical College and Hospital, Nagpur, Maharashtra, India

²Department of Medicine, Government Medical College, Gondia, Maharashtra, India

Corresponding Author: Sagar A Khandare, Department of Medicine, Government Medical College, Gondia, Maharashtra, India, Phone: +91 7719920823, e-mail: sagar.khandare@gmail.com

How to cite this article: Aher AA, Khandare SA, Upadhyay MG, *et al.* Study of Postprandial Triglyceridemia as Novel Marker of Carotid Intima-media Thickness in Patients with Type 2 Diabetes Mellitus. *J Med Sci* 2026;12(1–4):00441.

Source of support: Nil

Conflict of interest: None

milieu throughout the day. Therefore, measuring postprandial lipid profile along with fasting lipid profile is also important in patients with diabetes mellitus. Atherosclerosis, unless severe, is asymptomatic, so direct examination of the blood vessel wall is required to detect the affected individual in the early stages. Measurement of carotid intima-media thickness (CIMT) of the common carotid artery by B-mode ultrasound has proven to be a noninvasive, quantitative, and easily repeatable method for assessing early-onset atherosclerosis.⁶ CIMT acts as a surrogate marker for atherosclerosis. An increase in CIMT is associated with a higher risk of cerebrovascular and cardiovascular events (Fig. 2).⁷

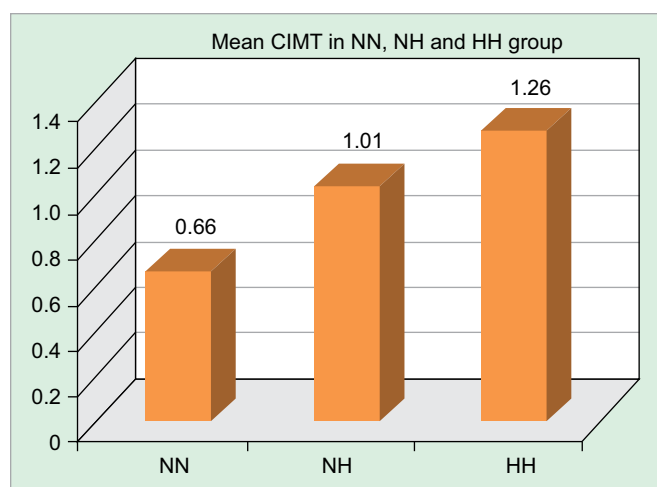


Fig. 1: Bar diagram of mean CIMT in NN, NH, and HH groups

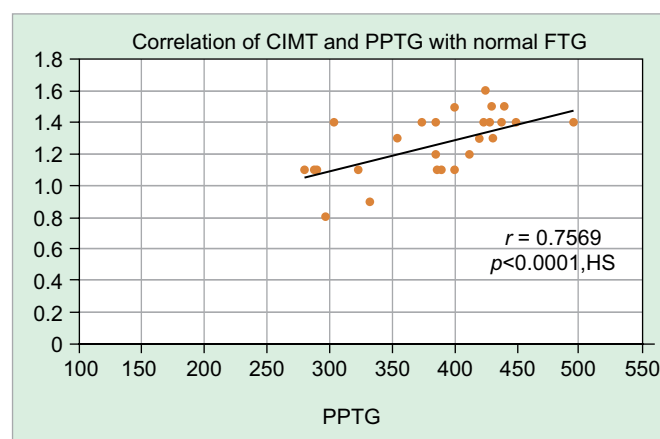


Fig. 2: Linear regression analysis depicting the relation between CIMT and PPTG in subjects with normal FTG

Although several studies have shown fasting TG levels to be associated with coronary artery disease in both diabetic and nondiabetic subjects, relatively little attention has been given to postprandial TGs (PPTG) in this regard, especially in diabetic subjects. To look for the role of postprandial dyslipidemia in accelerating atherosclerosis, we can thus examine the correlation between PPTG and CIMT. In the present study, the correlation of PPTG levels (4 hours after a regular meal) and CIMT in T2DM patients is seen.

MATERIALS AND METHODS

This prospective, observational cohort study was carried out at a tertiary care center in central India from November 2018 to October 2020 over a period of 2 years. All diagnosed cases of T2DM as per ADA diagnostic criteria were included. Patients with type 1 diabetes mellitus, ischemic heart disease, cerebrovascular disease, peripheral vascular disease, chronic kidney disorder, and thyroid disorder were excluded from the study. A total of 80 individuals with T2DM were enrolled in the study based on inclusion and exclusion criteria. All the subjects were interviewed, and a detailed history was taken. It included patient information, duration of diabetes, treatment modality they were on, such as insulin therapy, oral antidiabetic agents, or a combination of both, and any history suggestive of complications of diabetes was elicited. Detailed clinical examination

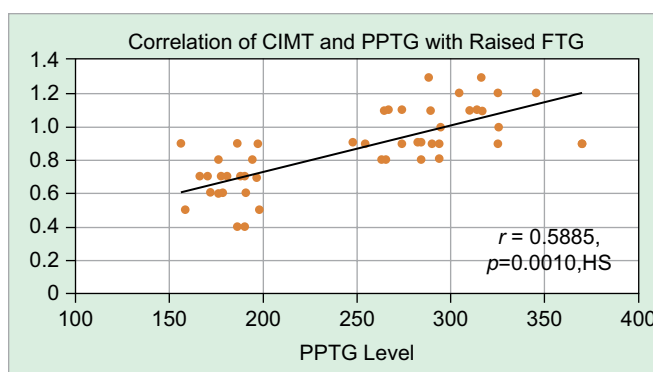


Fig. 3: Linear regression analysis depicting the correlation between CIMT and PPTG in subjects with raised FTG

of patients, including systolic and diastolic blood pressure, was performed. Complete systemic examination was done. Routine blood investigations were performed. Electrocardiography was done for every patient, and patients with ECG changes suggestive of CAD were excluded from the study. Routine blood investigations, which included CBC, LFT, RFT, and urine microscopy, were done. Blood samples were collected for fasting and postprandial blood sugar and lipid profile along with routine investigations. Fasting and PPTG were measured after overnight fast and 4 hours after a meal, respectively (Fig. 3).

Patients were divided into 3 groups, namely normo-normo group (NN)—with normal fasting TG <150 mg/dL and PPTG level <200 mg/dL; normo-hyper group (NH)—individuals with normal fasting (<150 mg/dL) and elevated PPTG level (>200 mg/dL); and hyper-hyper group (HH) group—elevated fasting (>150 mg/dL) and elevated postprandial level (>200 mg/dL).

Carotid intima-media thickness was measured in the radiology department using ultrasound. Carotid artery Doppler was done by B-mode (bright mode) ultrasound using a 7.5 MHz transducer at the right common carotid artery 10 mm distal to the carotid bifurcation. The ultrasonic image formed by the arterial wall is characterized by 2 parallel echogenic lines separated by a hypoechoic space—the “double-line” pattern. CIMT is calculated from the distance between echogenic lines of the arterial wall that represent lumen-intima and media-adventitia borders, and it was quantitatively measured using an electronic caliper. A cutoff point of 1 mm was chosen to identify subjects as having arterial wall thickening. Obvious plaques were excluded from measurement. Values of CIMT were correlated in all 3 groups. In all patients, levels of FTG and PPTG among various groups were correlated with CIMT.

Data were entered in Windows Excel format. Frequency tables, measures of central tendency (mean), and measures of dispersion (standard deviation) were obtained by using the statistical package SPSS-20. Variation between the 3-group means was studied using analysis of variance (ANOVA), and test statistic *F* values were obtained with appropriate degrees of freedom, and *p*-values were obtained with appropriate level of significance. To compare means of 2 groups, Student's *t*-test was used with appropriate degrees of freedom and levels of significance. Linear regression analysis of CIMT and PPTG among patients with normal FTG and raised FTG was done, and graph was prepared. Ethical approval for this study was provided by the ethics committee, GMCH Nagpur. Permission was taken from the head of the concerned department. Informed written consent in patients in vernacular language was taken before enrollment.

Table 1: Distribution of diabetic patients based on FTG and PPTG

Group	Criteria	No. of cases	Percentage
NN group	FTG ≤ 150 PPTG ≤ 200	23	28.75
NH group	FTG ≤ 150 PPTG ≥ 200	29	36.25
HH group	FTG ≥ 150 PPTG ≥ 200	28	35.00

Table 2: Correlation of CIMT in NN, NH, and HH group

	NN	NH	HH	p-value
Mean	0.66	1.01	1.26	
SD	0.15	0.15	0.19	$F = 83.37$ $p < 0.0001$, HS
Range	0.4–0.9	0.8–1.3	0.8–1.6	

Table 3: Correlation of FTG and PPTG level with CIMT

		Mean $\pm$ SD	Range	p-value
FTG	<150	0.85 $\pm$ 0.23	0.4–1.3	$F = 36.85$
	150–200	1.06 $\pm$ 0.21	0.8–1.3	$p < 0.0001$, HS
	>200	1.31 $\pm$ 0.16	1.1–1.6	
PPTG	<200	0.66 $\pm$ 0.15	0.4–0.90	$F = 48.20$
	200–250	0.90 $\pm$ 0	0.9–0.9	$p < 0.0001$, HS
	>250	1.13 $\pm$ 0.21	0.8–1.6	

RESULTS

In this study entitled study of correlation between postprandial triglyceridemia and CIMT in patients with T2DM, a total of 80 patients were enrolled based on the inclusion and exclusion criteria. The study was conducted between November 2018 and October 2020. At the end of the study, data were analyzed systematically and arranged in tables and figures as below.

Of the total 80 patients studied, the mean age was 49.81 years with a standard deviation of 10.08. Of the total 80 patients studied, 44 (55%) were female, and 36 (45%) were male. The female-to-male ratio was 1.22. Out of 80 patients included in the study, 9 patients (11.25%) were hypertensive, and 71 (88.75%) patients were nonhypertensive.

Patients were classified into 3 groups based on their serum TG level.

Patients with fasting serum TG level <150 mg/dL and PPTG level <200 mg/dL were classified as NN group. Patients with fasting TG level <150 mg/dL and postprandial level >200 mg/dL were classified as NH group. Patients with fasting serum TG level >150 mg/dL and PPTG level >200 mg/dL were classified as HH group.

Out of 80 patients included in the study, 23 (28.75%) were in NN group, 29 (36.25%) patients were in NH group, and 28 (35%) patients were in HH group (Table 1).

The mean CIMT among patients with NN group was 0.66 $\pm$ 0.15. The mean CIMT among patients with NH group was 1.01 $\pm$ 0.15. The mean CIMT among patients with HH group was 1.26 $\pm$ 0.19. This indicates PPTG level has significant correlation with CIMT (Table 2).

The mean CIMT value of patients with FTG level <150 mg/dL was 0.85 $\pm$ 0.23. The mean CIMT value of patients with FTG level 150–200 mg/dL was 1.06 $\pm$ 0.21. The mean CIMT value of patients with FTG level >200 mg/dL was 1.31 $\pm$ 0.16. The CIMT increased

with increase value of FTG level, which was statistically significant with p -value < 0.0001.

The mean CIMT value of patients with PPTG level <200 mg/dL was 0.66 $\pm$ 0.15. The mean CIMT value of patients with PPTG level 200–250 mg/dL was 0.90 $\pm$ 0. The mean CIMT value of patients with PPTG level >250 mg/dL was 1.13 $\pm$ 0.21. The CIMT increased with increase value of PPTG level, which was statistically significant with p -value < 0.0001 (Table 3).

Linear regression analysis was done to depict the relationship between CIMT and PPTG among patients with normal FTG and raised FTG. A positive correlation was observed between CIMT and PPTG. This graph depicts that PPTG is more significantly correlating with CIMT than FTG level ($r = 0.5585$ vs 0.7569) (Table 4).

DISCUSSION

Although several studies have shown fasting TG levels to be associated with coronary artery disease in both diabetic and nondiabetic subjects, relatively little attention has been given to PPTGs in this regard, especially in diabetic subjects. In this study, the CIMT value is assessed according to FBS and PPBS levels (p -value < 0.001). It was found that values of fasting and postprandial blood sugar levels correlate with CIMT. This was similar to that observed by Pathak et al.,⁸ who also reported that CIMT values had significant correlation with FBS as well as PPBS. In our study, levels of LDL, HDL, and total cholesterol had significant relationship with CIMT. A similar relationship was observed in the study of Amruth Rao et al. and Kirbhakaran et al.,⁹ indicating higher levels of these lipids play an important role in development of atherosclerosis.

In the present study, mean CIMT in NN, NH, and HH groups were 0.66 $\pm$ 0.15, 1.01 $\pm$ 0.15, and 1.26 $\pm$ 0.19 mm, respectively. The values of CIMT were higher in NH and HH group as compared to NN

Table 4: Patient's datasheet

S. no.	Age	Sex	Duration	OHA	Insulin	HTN	UP	HR	SBP	DBP	HB%	FBS	PPBS	FTG	PPTG	Cholesterol	HDL	LDL	CIMT (mm)	S. creatinine	TSH
1	52	M	8	1	0	0	1+	90	130	80	14	108	224	129	265	188	46	78	0.8	1.1	1.68
2	40	M	4	1	0	0	0	84	120	70	13	98	189	124	177	120	49	80	0.7	0.9	2.34
3	44	M	7	1	0	0	0	68	118	72	13.5	90	165	134	180	170	48	88	0.7	1.3	1.45
4	42	F	6	1	0	0	1+	88	114	76	10	200	325	212	390	220	40	106	1.1	0.7	3.67
5	47	M	5	1	0	0	0	78	110	70	14	105	228	100	186	96	50	39	0.4	1	0.98
6	68	F	13	1	1	0	1+	70	130	90	9.6	255	356	198	412	242	28	132	1.2	1.2	2.54
7	54	F	9	1	0	0	0	84	124	80	11	180	270	114	178	67	45	128	0.7	1.1	4.87
8	63	F	18	1	0	0	0	82	130	88	13.2	162	218	186	332	116	48	88	0.9	1	3.24
9	49	M	5	1	0	0	0	92	126	78	13.8	221	320	265	386	200	34	96	1.1	0.9	1.28
10	45	M	3	1	0	0	0	88	114	72	14.6	187	278	223	431	186	46	78	1.3	1.2	1.88
11	64	F	10	0	1	0	1+	80	140	90	13.2	227	338	265	438	213	35	110	1.4	1.3	2.44
12	35	F	1	1	0	0	0	110	112	70	12.6	199	322	154	297	154	40	86	0.8	0.7	3.58
13	39	M	3	1	0	0	0	94	116	74	14.2	91	176	108	190	108	60	65	0.4	1	2.84
14	67	M	22	0	0	0	1+	76	148	86	15	250	452	218	288	179	32	99	1.1	1.4	5.14
15	35	F	2	1	0	0	0	98	120	70	11.2	126	212	124	198	132	55	50	0.5	1.1	0.95
16	44	M	8	1	0	0	0	76	110	82	13.2	153	190	121	178	100	56	65	0.6	1	1.54
17	62	F	12	1	0	0	0	72	130	88	11.6	215	287	129	265	190	45	94	1.1	1.1	2.86
18	36	M	5	1	0	0	0	88	112	70	15.2	134	198	139	197	140	48	66	0.9	0.8	3.94
19	55	F	10	1	0	0	0	84	130	84	13.6	172	267	190	430	212	38	78	1.3	1.4	4.57
20	48	M	4	1	0	0	0	68	118	76	14	208	366	298	428	230	35	104	1.4	1.1	2.56
21	40	M	2	1	0	0	0	88	120	80	15.2	119	218	145	284	126	49	65	0.8	1	1.88
22	54	F	7	0	1	0	0	94	146	86	11.8	260	428	320	450	245	37	130	1.4	1.1	2.98
23	67	M	14	0	1	1	0	74	140	90	12.6	251	398	312	426	198	30	112	1.6	1.3	3.54
24	49	F	8	1	0	0	0	88	120	72	10.2	179	275	140	310	150	40	83	1.1	1.2	1.94
25	43	F	4	1	0	0	0	76	118	68	11.8	127	245	138	294	176	48	65	0.8	0.9	2.85
26	40	F	1	1	0	0	0	66	114	70	9.1	125	228	127	325	164	45	45	0.9	1.1	3.64
27	56	M	12	1	0	0	0	78	130	78	13.8	107	267	131	266	143	50	76	1.1	1.4	1.95
28	59	F	13	1	1	0	0	88	140	88	14.2	202	340	241	385	187	29	88	1.4	1.3	2.65
29	38	F	2	1	0	0	0	110	130	76	9.6	189	378	196	323	200	50	116	1.1	1	4.57
30	55	M	1	1	0	0	0	78	124	72	12.8	181	332	241	496	188	47	98	1.4	0.8	3.74
31	62	F	17	0	1	1	0	72	130	86	13.2	246	410	227	440	246	28	120	1.5	1.3	2.85
32	42	M	2	1	0	0	0	70	110	74	15.4	123	254	130	248	176	48	76	0.9	1	1.88
33	35	M	1	1	0	0	0	68	104	70	13.2	112	178	117	190	104	50	60	0.7	0.9	2.94
34	38	F	3	1	0	0	0	88	96	70	10.7	170	292	132	306	115	46	98	1	0.7	3.84
35	39	F	3	1	0	0	0	70	100	72	11.2	226	385	277	420	165	40	132	1.3	1.1	1.44
36	36	M	1	1	0	0	0	88	102	70	9.2	146	220	138	290	212	40	93	0.9	1	2.54
37	35	F	9	1	0	0	0	98	94	86	10.6	210	296	142	325	187	38	110	1.2	1.1	3.57
38	46	M	4	1	0	0	0	86	120	80	14.2	173	269	205	280	146	50	88	1.1	0.8	2.86
39	53	F	6	1	0	0	0	78	126	84	10.2	132	210	146	254	120	49	53	0.9	0.9	5.14
40	42	M	5	1	1	0	0	68	114	70	14.5	204	370	140	314	188	42	100	1.1	0.7	2.66
41	37	M	1	1	0	0	0	88	110	70	13.8	102	189	120	282	98	54	54	0.9	1	3.45
42	38	F	4	1	0	0	0	76	100	68	8.8	90	156	131	158	112	52	47	0.5	1.1	2.72
43	44	F	3	1	0	0	0	72	102	74	9.5	120	197	126	172	130	52	76	0.6	0.9	3.45
44	39	F	2	1	0	0	0	70	110	70	10.2	160	194	123	316	110	51	50	1.1	1.1	1.98
45	66	M	15	1	0	0	0	86	130	88	14.2	116	210	138	304	146	42	83	1.2	1.2	3.24
46	52	M	12	1	0	0		88	116	74	13.2	120	201	124	293	136	54	80	0.9	1	2.65
47	42	F	4	1	0	0	0	65	110	70	10.5	166	230	138	263	167	50	65	0.8	0.9	3.75
48	52	F	10	1	1	1	0	87	124	72	13.6	243	330	250	400	214	34	98	1.1	1.4	4.65
49	40	M	7	1	0	0	0	88	110	70	15.6	145	194	140	274	158	38	64	0.9	1.4	1.96
50	63	M	14	0	1	0	1	104	128	86	12.5	206	338	280	374	188	32	100	1.4	1.6	4.88
51	38	M	2	1	0	0	0	76	96	70	14.6	148	212	128	370	130	48	75	0.9	1	2.85
52	51	F	7	1	0	0	0	82	110	80	12.8	150	244	130	274	156	46	82	1.1	0.9	6.65

Contd...

Contd...

S. no.	Age	Sex	Duration	OHA	Insulin	HTN	UP	HR	SBP	DBP	HB%	FBS	PPBS	FTG	PPTG	Cholesterol	HDL	LDL	CIMT (mm)	S. creatinine	TSH
53	59	M	14	1	0	0	0	80	124	90	13.6	168	278	132	288	188	40	64	1.3	1.2	3.45
54	58	F	18	0	1	1	1+	76	140	88	12.8	220	395	280	446	230	36	110	1.1	1.3	2.86
55	44	M	4	1	0	1	0	88	110	70	14.1	123	234	114	190	166	44	73	0.4	1	3.48
56	44	F	6	1	0	0	0	76	114	86	9.8	124	180	121	170	100	53	48	0.7	0.9	2.78
57	59	F	11	0	1	1	0	68	130	90	8.2	257	398	274	430	198	29	130	1.5	0.7	0.95
58	63	M	18	1	0	0	1+	72	140	88	10.7	226	298	227	354	176	26	114	1.3	1.4	1.72
59	41	M	6	1	0	0	0	88	120	70	11.3	126	198	122	194	135	42	89	0.8	1.2	2.68
60	40	M	1	1	0	0	0	78	126	70	14.2	184	265	123	325	144	50	65	1	0.9	3.55
61	57	F	12	1	0	0	0	82	114	84	10.2	102	255	122	176	120	47	86	0.8	1.2	4.25
62	62	F	24	1	0	0	0	80	132	90	13.1	94	176	130	186	110	39	43	0.9	1	1.86
63	61	F	14	1	0	0	0	92	110	80	12.8	131	217	124	196	164	45	64	0.7	1.1	2.87
64	41	M	2	1	0	0	0	90	120	70	14.2	138	275	120	190	136	35	77	0.6	1.2	3.94
65	66	F	15	0	1	1	1+	82	128	90	11.8	226	321	240	290	180	42	112	1.1	1.6	1.33
66	39	M	3	1	0	0	0	68	120	70	14.2	226	287	130	284	156	42	88	0.9	1.1	5.44
67	53	F	8	0	1	0	0	92	118	88	13.9	207	366	265	423	178	40	134	1.4	1.2	1.38
68	48	F	4	1	0	0	0	80	130	76	12.7	103	188	126	170	124	37	102	0.7	1.2	3.92
69	62	F	12	1	0	1	0	88	140	82	10.8	94	176	113	156	120	40	55	0.9	1	1.86
70	66	M	4	1	0	0	0	98	130	90	13.4	102	188	122	290	98	41	78	1.1	1.1	2.67
71	48	M	9	0	1	0	1+	78	120	84	11.8	210	393	260	385	216	32	98	1.2	1.4	4.22
72	63	F	10	0	1	0	0	86	118	90	8.6	220	355	286	400	230	28	104	1.5	1.3	3.84
73	63	M	18	1	0	0	1	76	150	94	13.2	184	280	144	316	186	33	75	1.3	1	1.68
74	66	F	20	1	0	0	0	68	130	90	14.2	142	210	117	166	140	42	64	0.7	0.9	2.75
75	50	M	8	1	0	0	0	88	124	84	12.4	153	215	138	289	128	40	60	1.1	1.1	3.68
76	42	M	4	1	0	0	0	82	114	78	13.6	177	280	137	345	136	46	88	1.2	1.2	4.38
77	56	F	12	1	0	1	0	76	126	90	9.4	114	210	130	176	114	40	76	0.6	1	2.94
78	52	F	10	1	0	0	0	73	124	86	10.2	117	196	140	294	106	38	65	1	1.1	1.98
79	47	M	7	0	1	0	1+	92	110	82	14.8	251	380	260	304	210	42	116	1.4	1.4	3.65
80	55	M	15	1	0	0	0	80	120	76	13.5	115	220	136	188	146	45	75	0.7	1	2.45

group with p -value < 0.0001 , which is statistically highly significant. This was in concordance with the study done by Teno et al.,¹⁰ and carotid IMT was 0.73 ± 0.13 mm in the NN group, 0.86 ± 0.13 mm in the NH group, and 0.85 ± 0.12 mm in the HH group. In the Amruth Rao et al.¹¹ study, the mean carotid IMT in the NN, NH, and HH group were 0.52 ± 0.13 , 1.78 ± 0.81 , and 2.06 ± 0.08 mm, with a range between 0.3–0.7, 0.7–2.7, and 1–3.2, respectively. It was observed from this study that the mean CIMT in patients with postprandial hypertriglyceridemia HH group was significantly higher than that in patients with normal FTG and normal PPTG levels (NN group) (1.26 vs 0.66 mm, $p < 0.0001$).

In our study, CIMT showed a significant correlation with FTG and PPTG levels. Correlation between CIMT and FTG levels ($F = 36.85$, $p < 0.0001$) reflected that CIMT increased with increase in the FTG levels. There was a significant correlation between CIMT and PPTG (F statistic = 48.20, $p < 0.0001$). It was also seen that CIMT increased with increase in the PPTG levels. F statistic of CIMT vs PPTG (48.20) is greater than F statistic of CIMT vs FTG (36.80), which shows that PPTG level is a better predictor of CIMT than FTG. Correlation coefficient r -value when compared between FTG and PPTG with CIMT were 0.7614 vs 0.8500, predicting the higher correlation between PPTG with CIMT as compared to fasting TG levels ($p < 0.0001$ HS). Similar correlation was noted by Bendwal et al.¹² They observed that although both FTG and PPTG levels were significantly correlating with CIMT, the PPTG levels ($r = 0.67$) were more significantly correlating with CIMT as

compared to the FTG levels ($r = 0.45$). In a study done by Ahmad et al.,¹³ the mean CIMT in the NN, NH, and HH group were 0.59 ± 0.09 , 0.79 ± 0.09 ($p < 0.001$ vs NN group), and 0.82 ± 0.06 mm ($p < 0.001$ vs NN group), respectively. So, in their study, it was observed that CIMT was increased in patients with postprandial hypertriglyceridemia despite normal FTG levels, and the PPTG levels showed the strongest influence on CIMT. These results were similar to our study.

In our study, out of total 80 patients with type 2 diabetes, 59 patients had normal LDL level. We compared value of CIMT in patients among NN, NH, and HH group who had normal level of LDL, and it was observed that patients with NH and HH group had significantly high level of CIMT as compared to NN group (p -value < 0.0001). In study conducted by Mohan et al.,¹⁴ it was found that LDL cholesterol was not significantly correlating with CIMT in diabetics. Study of Bendwal et al. also showed that LDL cholesterol had no significant relationship with CIMT. Hence, evaluation of TG level is more important in diagnosis of diabetic dyslipidemia.

CONCLUSION

In this study, it is shown that CIMT is higher in people with postprandial hypertriglyceridemia despite normal levels of fasting TGs. CIMT correlates better with PPTG levels when compared to fasting TG levels and hence can predict early

atherosclerosis. Postprandial hypertriglyceridemia can be labeled as an independent predictor of accelerated atherosclerosis in diabetics. Therefore, PPTG level estimation along with fasting TG level should be done for early detection of diabetic dyslipidemia and prevention of macrovascular complications.

Clinical Significance

Usually, only fasting lipid levels are measured in patients, but our study showed that PPTG has significant correlation with CIMT. So, by measuring PPTG, we can detect macrovascular complications in type 2 diabetes patients in early stages.

REFERENCES

1. Powers AC. Diabetes mellitus. In: Kasper DL, Fauci AS, Hauser SL, et al. editors. *Harrison's Principles of Internal Medicine*. 20th ed. New York: McGraw-Hill; 2018. pp. 2850–2860.
2. International Diabetes Federation. *IDF Diabetes Atlas*. 9th ed. Brussels: International Diabetes Federation; 2019 [cited 3 Feb 2021]. Available from: <https://diabetesatlas.org/en/>
3. Wu L, Parhofer KG. Diabetic dyslipidemia. *Metabolism* 2014;63(12):1469–1479. DOI: 10.1016/j.metabol.2014.08.010
4. Betteridge D. Lipid control in patients with diabetes mellitus. *Nat Rev Cardiol* 2011;8:278–290. DOI: 10.1038/nrcardio.2011.23
5. Madhu SV, Mittal V, Krishnaram B, et al. Postprandial triglyceride metabolism in obese and non-obese patients with non-insulin dependent diabetes mellitus. *Diabetes Res Clin Pract* 2000;50:5318. DOI: 10.1016/S0168-8227(00)81083-9
6. Qu B, Qu T. Causes of changes in carotid intima-media thickness: a literature review. *Cardiovasc Ultrasound* 2015;13:46. DOI: 10.1186/s12947-015-0041-4
7. Lorenz MW, Markus HS, Bots ML, et al. Prediction of clinical cardiovascular events with carotid intima-media thickness. Systematic review and meta-analysis. *Circulation* 2007;115:459–467. DOI: 10.1161/CIRCULATIONAHA.106.628875
8. Pathak R, Adhikari P, Pathak U. Role of fasting and postprandial hypertriglyceridemia and its association with carotid intima-media thickness in type II diabetes patients. *J Adv Intern Med* 2018;7(1):17–22. DOI: 10.3126/jaim.v7i1.19578
9. Kirubhakaran K, Rangabashyam RS, Shankar R. Correlation of blood sugar and lipid parameters with carotid intima-media thickness among patients with type II diabetes mellitus. *Int J Med Res Rev* 2019;7(2):54–60. DOI: 10.17511/ijmrr.2019.i02.01
10. Teno S, Uto Y, Nagashima H, et al. Association of postprandial hypertriglyceridemia and carotid intima-media thickness in patients with type 2 diabetes. *Diabetes Care* 2000;23(9):1401–1406. DOI: 10.2337/diacare.23.9.1401
11. Amruth Rao P, Ramulu P, Paul Marx K, et al. Association of post prandial hyper triglyceridemia and carotid intima-media thickness in patients with type-2 diabetes mellitus. *Int J Diabetes Res* 2016;5(5):87–91. DOI: 10.5923/j.diabetes.20160505.01
12. Bendwal K, Bendwal S, Dhawale RG. Correlation between triglyceride level and carotid intima-media thickness in patients with type 2 diabetes mellitus. *Int J Clin Trials* 2018;5(2):97. DOI: 10.18203/2349-3259.ijct20181051
13. Ahmad J, Hameed B, Das G, et al. Postprandial hypertriglyceridemia and carotid intima-media thickness in north Indian type 2 diabetic subjects. *Diabetes Res Clin Pract* 2005;69(2):142–150. DOI: 10.1016/j.diabres.2004.11.012
14. Mohan V, Ravikumar R, Shanthi Rani S, et al. Intimal medial thickness of the carotid artery in South Indian diabetic and non-diabetic subjects: The Chennai Urban Population Study. *Diabetologia* 2000;43:494–499. DOI: 10.1007/s001250051334