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A study on fasting and postprandial lipid profile in patients in type 2 diabetes mellitus

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Abstract

The triglyceride levels in serum generally remain elevated for about 3 to 6 hours after a meal. This postprandial hypertriglyceridemia (more than 2.72 mmol/l or 201 mg/dl) is exacerbated by the next routine meal and thus the lipemic milieu persists throughout the day. Therefore, measuring and documenting postprandial dyslipidemia in diabetic patients is vital in addition to measuring the fasting lipid levels. Blood samples were drawn before and 4 hours after the standardized test meal. The HbA1c, fasting and postprandial lipid profile levels and glucose levels were estimated. Fasting and postprandial triglycerides, total Cholesterol, HDL, VLDL were measured by standard laboratory technique. The mean serum HDL cholesterol in the NN group was 40.57 ± 5.23.The mean serum HDL cholesterol in the NH group was 35.43 ± 6.33.The mean serum HDL cholesterol in the HH group was 32.87 ± 6.30. The mean serum fasting triglyceride in the NN group was 107.29 ± 20.69.The mean serum fasting triglyceride in the NH group was 132.57 ± 7.52.The mean serum fasting triglyceride in the HH group was 230.13 ± 48.96.

Keywords: Fasting and Postprandial lipid, Type 2 diabetes mellitus, lipemic milieu

Introduction

Hypertriglyceridemia and low HDL cholesterol levels constitute the most common dyslipidemic pattern in type 2 diabetic patients. Traditionally we measure only the fasting lipid values. In fact these values represent the nadir of the lipid values over 24 hours. Postprandial lipid measurements, especially triglyceride levels provide a more meaningful insight regarding the lipemic status of a individual [1].
The triglyceride levels in serum generally remain elevated for about 3 to 6 hours after a meal. This postprandial hypertriglyceridemia (more than 2.72 mmol/l or 201 mg/dl) is exacerbated by the next routine meal and thus the lipemic milieu persists throughout the day. Therefore, measuring and documenting postprandial dyslipidemia in diabetic patients is vital in addition to measuring the fasting lipid levels [2].
A prospective study by Koskinen P *et al*. has revealed that hypertriglyceridemia is the most significant risk factor for coronary artery disease (CAD). In fact elevated triglyceride levels may be a harbinger of future IHD than high LDL cholesterol levels. Hypertriglyceridemia (more than 1.7 mmol/l or 150

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mg/dl) often precedes hypercholesterolemia in diabetic patients with early-onset IHD. Acute coronary events occur more frequently in patients with fasting triglyceridemia (more than 1.13 mmol/l or100 mg/dl) [3]. While fasting hypertriglyceridemia may be a potential risk factor for atherosclerosis, particularly in the presence of diabetes mellitus, this association has not been consistently strong. However, when TGs are studied in postprandial state, they emerge as stronger and independent coronary risk factor. Postprandial dyslipidemia has been linked with asymptomatic and symptomatic macrovascular disease in subjects with normal fasting values. The increased risk of atherosclerosis among them, might therefore be related to the higher postprandial lipemia.

It is hypothesized that elevated postprandial lipids levels may lead to an alteration in oxidative stress and consequent endothelial dysfunction that may finally lead to atherosclerosis and macrovascular disease in diabetic patients. Several mechanisms have been hypothesized to cause postprandial triglyceride abnormalities in type 2 diabetes subjects. There is significant delay in triglyceride clearance in diabetic patients, more so in individuals with macrovascular disease [4].

Chronic hyperglycemia results in impaired endothelial function. Recent studies done in both healthy as well as type 2 diabetic individuals indicate that even acute hyperglycaemia can cause profound endothelial dysfunction. The circulating ICAM-1 levels in plasma significantly increased, following an oral glucose tolerance test in diabetic and normal subjects which is an indirect indicator of endothelial cell activation. HbA1c level is a reliable measurement of chronic hyperglycemia in a given patient. A Chinese population based study done by Hung CS *et al*. demonstrated that glycated haemoglobin levels have a significant correlation with carotid intima-media thickness [5].

Ceriello and co-workers demonstrated an additive effect of hypertriglyceridemia and hyperglycaemia in hampering endothelial function. A single metabolic component inadequately accounts for the real life postprandial dysmetabolic milieu. Thus postprandial dyslipidemia and hyperglycemia act as independent and cumulative factors in causing postprandial endothelial dysfunction. Hence both these values should be targeted by treatment protocols to prevent the development of macrovascular complications [6].

Methodology

Inclusion criteria

All patients with type 2 diabetes mellitus with normal ECG and normal echocardiogram.

Exclusion criteria

1. Prior history of Ischemic heart disease as determined by history and ECG.
2. Patients on lipid lowering drugs, thiazides, beta blockers.
3. Patients with history of CVA/TIA.
4. Patients with clinical or imaging evidence of Peripheral vascular disease or history of limb amputation.
5. Patients with history of bariatric surgery.
6. Known cases of hypothyroidism.
7. Patients with chronic complications of diabetes like nephropathy, retinopathy.
8. Patients with history or clinical findings suggestive of familial hyperlipidemias.
9. Patients with known hepatic disease.

Patients on oral hypoglycemic agents, antihypertensive drugs (ACE inhibitors or AT II antagonists) were not excluded from the study.

The following investigations were done in all the patients prior to entering into the study

- Haemoglobin.

- Total Leukocyte Count, Differential Leukocyte Count.
- ESR.
- Random Blood Sugar.
- Blood Urea.
- Serum Creatinine.
- Liver function tests.
- Urine Microscopy for sugar and albumin.
- ECG or 2D Transthoracic Echocardiogram.

Standardized meal test

A standardized mixed meal was given to all the patients after an overnight fast. The meal consisted of three idlis and a standard serving of sambar and a standard serving of coconut chutney. The total energy content of the standard meal was 9 Kcal/kg, with 60% of the total energy from carbohydrates, 20% of the energy from fat and 20% of the energy from proteins. Blood samples were drawn before and 4 hours after the standardized test meal. The HbA1c, fasting and postprandial lipid profile levels and glucose levels were estimated. Fasting and postprandial triglycerides, total Cholesterol, HDL, VLDL were measured by standard laboratory technique. The Friedwald's formula was used to calculate LDL cholesterol.

Friedwalds Formula: LDL cholesterol = Total Cholesterol – (HDL + Triglycerides/5)

Results

Table 1: Comparison between PPTC with Groups

			Groups			Total
			NN	NH	HH	
PPTC	<= 200	Count	8	6	1	15
		%	57.1%	26.1%	4.3%	25.0%
	> 200	Count	6	17	22	45
		%	42.9%	73.9%	95.7%	75.0%
Total		Count	14	23	23	60
		%	100.0%	100.0%	100.0%	100.0%

- The mean serum total cholesterol in the NN group was 141.19 ± 20.
- The mean serum total cholesterol in the NH group was 162.67 ± 30.41.
- The mean serum total cholesterol in the HH group was 176.42 ± 30.69.

Table 2: Comparison between PPLDL with Groups

			Groups			Total
			NN	NH	HH	
PPLDL	< 100	Count	8	9	2	19
		%	57.1%	39.1%	8.7%	31.7%
	100 - 130	Count	6	8	10	24
		%	42.9%	34.8%	43.5%	40.0%
	> 130	Count	0	6	11	17
		%	0.0%	26.1%	47.8%	28.3%
Total		Count	14	23	23	60
		%	100.0%	100.0%	100.0%	100.0%

- The mean serum LDL cholesterol in the NN group was 85.98 ± 19.12.
- The mean serum LDL cholesterol in the NH group was 100.41 ± 26.98.

- The mean serum LDL cholesterol in the HH group was 97.84 ± 31.3.

Table 3: Comparison between PPHDL with Groups

			Groups			Total
			NN	NH	HH	
PPHDL	<= 40	Count	13	18	21	52
		%	92.9%	78.3%	91.3%	86.7%
	> 40	Count	1	5	2	8
		%	7.1%	21.7%	8.7%	13.3%
Total		Count	14	23	23	60
		%	100.0%	100.0%	100.0%	100.0%

- The mean serum HDL cholesterol in the NN group was 40.57 ± 5.23.
- The mean serum HDL cholesterol in the NH group was 35.43 ± 6.33.
- The mean serum HDL cholesterol in the HH group was 32.87 ± 6.30.

Table 4: Distribution of the Study Population According to Fasting Triglyceride Values

Fasting Triglycerides (mg/dl)	Subjects (NN Group)		Subjects (NH Group)		Subjects (HH Group)	
	No	%	No	%	No	%
<150	18	100	11	100	-	
150-249	-		-		18	58.06
250-349	-		-		13	41.93
≥350	-		-			
TOTAL	18		11		31	

- The mean serum fasting triglyceride in the NN group was 107.29 ± 20.69.
- The mean serum fasting triglyceride in the NH group was 132.57 ± 7.52.
- The mean serum fasting triglyceride in the HH group was 230.13 ± 48.96.
- The mean serum postprandial triglyceride in the NN group was 129.21 ± 14.21.
- The mean serum postprandial triglyceride in the NH group was 218.26 ± 44.42.
- The mean serum postprandial triglyceride in the HH group was 314.48 ± 65.21.

Table 5: Comparison between PPTG with Groups

			Groups			Total
			NN	NH	HH	
PPTG	< 200	Count	14	10	0	24
		%	100.0%	43.5%	0.0%	40.0%
	200 - 299	Count	0	13	11	24
		%	0.0%	56.5%	47.8%	40.0%
	300 - 399	Count	0	0	9	9
		%	0.0%	0.0%	39.1%	15.0%
	>= 400	Count	0	0	3	3
		%	0.0%	0.0%	13.0%	5.0%
Total		Count	14	23	23	60
		%	100.0%	100.0%	100.0%	100.0%

Discussion

Table 6: Distribution of FTG in different Studies

Study		Amruth Rao *et al*. [7]	Kavitha Bendal *et al*. [8]	R Pathak *et al*. [9]	Khamesh ME *et al*. [10]	Our Study
FTG	NN	130.1±13.55	112.9±14.2	89.7±29	146.1±63.5	98±14.93
	NH	133.91±11.37	124.4±16.29	107.4±24.7		129.83±17.92
	HH	252.17±79.52	206.8±28.44	237.3±81.9		218.7±44.82

Distribution of FTG in our study in three groups were comparable with Kavtha bendal *et al*. and R Pathak *et al*.

Table 7: Distribution of PPTG in different Studies

Study		Amruth Rao *et al*. [7]	Kavitha Bendal *et al*. [8]	R Pathak *et al*. [9]	Khamesh ME *et al*. [10]	Our Study
PPTG	NN	180.4±135.84	182.9±24.2	114.9±28.2	194±106.4	129.21±14.21
	NH	285.55±37.73	242.4±26.29	192.7±38.3		218.21±44.2
	HH	378.5±10.24	306.8±29.44	229.1±87.9		314.48±65.21

PPTG in our study group distributed in three groups is comparable with R Pathak et all in case PPTG in NN group, PPTG in NH is comparable with Kavitha Bendal *et al*. and PPTG in HH group is comparable with Kavitha Bendal *et al*.

Ashwini Kumar *et al*.: This study was done amongst 50 North Indian patients, between the age group of 35-75 years suffering from Type II Diabetes Mellitus for less than 1 year duration. Both PPTG and FTG showed a strong correlation with CIMT, the correlation coefficients being 0.879 and 0.764 respectively.

Kavita Bendwal *et al*.: 120 patients of T2DM with no features peripheral vascular disease, IHD or stroke with age group between 30 to 70 years were included. In the present study, it was observed that although both FTG and PPTG levels were significantly correlating with CIMT but the PPTG levels (r=0.67) were more significantly correlating with CIMT as compared to the FTG levels (r=0.45).

Conclusion

- Postprandial total cholesterol and postprandial triglycerides were significantly elevated in diabetic patients compared to normal individuals.
- Postprandial triglycerides were significantly elevated compared to fasting triglycerides in diabetic patients.

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