Original Article

Fasting and postprandial lipid parameters: A comparative evaluation of cardiovascular risk assessment in prediabetes and diabetes

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Abstract

Context: Dyslipidemia plays a crucial role in atherogenesis, in both prediabetes and diabetes. There persists a lacuna in the evaluation of postprandial lipid parameters in prediabetes. **Aims:** To comparatively evaluate fasting and postprandial blood lipid parameters and atherogenic lipid ratios for cardiovascular risk assessment, in prediabetes and diabetes. **Materials and Methods:** Fifty-one patients diagnosed with diabetes mellitus and thirty-two with prediabetes were selected for the study. Lipid profile and blood glucose were analyzed in fasting and postprandial blood samples. **Statistical Analysis Used:** Kolmogorov–Smirnov test, Shapiro–Wilk test, one-way ANOVA, and Pearson's regression analysis were applied. **Results:** Postprandially, triglycerides (TG) was increased significantly in diabetes compared to controls ($P < 0.01$) and prediabetics ($P < 0.05$). Among the lipid ratios, triglyceride/high density lipoprotein (TG/HDLc) was significantly increased postprandially in diabetes compared to controls ($P < 0.05$). A comparative analysis of fasting and postprandial parameters within each group showed a significant increase in postprandial TG/HDLc compared to the fasting state in prediabetes ($P < 0.001$) and diabetes ($P < 0.001$). Postprandial TG ($P < 0.01$) and TG/HDLc ($P < 0.01$) showed a stronger correlation with HbA_{1c} compared to fasting TG ($P < 0.05$) and TG/HDLc ($P > 0.05$). The prevalence of dyslipidemia and insulin resistance was higher in postprandial state than the fasting state in prediabetes and diabetes. **Conclusions:** Postprandial TG and the TG/HDLc reflect lipid abnormalities than the corresponding fasting variables in diabetes and prediabetes. Postprandial TG and TG/HDLc are better reflectors of cardiovascular status in prediabetes and diabetes.

Keywords: Diabetes, dyslipidemia, fasting lipid profile, postprandial lipid profile, prediabetes

Introduction

Cardiovascular disease is a major contributor to morbidity and mortality in diabetes mellitus.[1] An array of cardiovascular risk factors such as insulin resistance, glucose intolerance, and abnormal lipid profile are the key contributors of atherogenesis in diabetes.[2-4] In recent years, there has been an increased awareness about the prevalence of prediabetes,[5] a high-risk state for developing diabetes. The end-organ target damage in prediabetes and the associated adverse outcome is indistinguishable to that of diabetes.[6]

Dyslipidemia plays a crucial role in atherogenesis, in both prediabetes and diabetes, characterized by elevated

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triacylglycerol (TG), reduced high-density lipoprotein (HDLc), and predominant small density-low density lipoprotein.[7,8] Several lines of evidence have shown exaggerated postprandial lipid derangements, evoking functional abnormalities of the vascular endothelium and atherosclerosis in diabetes.[9,10] India being a hub of diabetes population also presents significant cases of dyslipidemia and consequent morbidity and mortality. If the cardiovascular risk assessment can be done by a routine parameter, the same can be detected at the level of primary care physicians.

The nonfasting lipid parameters bestow an edge over fasting lipid profile (FLP) in cardiovascular risk prediction and diabetes evaluation.[11-13] There persists a lacuna in the evaluation of postprandial lipid parameters in prediabetes. Fasting dyslipidemia has been observed in prediabetes similar to abnormalities in diabetic fasting profile.[14,15] However, studies on evaluation of postprandial lipid parameters are very few and with conflicting results.[16] Moreover, there are no reports documented on the utility of both fasting and postprandial blood lipid parameters and lipid ratios for cardiovascular risk assessment in these patients. Hence, the present study was taken up to compare the fasting and postprandial blood lipid parameters and lipid ratios for evaluation of cardiovascular risk assessment in prediabetes and diabetes.

Subjects and Methods

The present study was carried in a tertiary care hospital from September 2017 to February 2018 02-08-2017. Fifty-one cases of type-2 diabetes mellitus and thirty-two cases of prediabetes were selected after necessary clearances and informed consent for the study. Twenty-eight, age- and sex-matched subjects, were taken as controls.

Inclusion criteria

The prediabetic group was selected on the basis of the following criteria: Fasting blood glucose (FBG) of 100–125 mg/dl or/and 2-h postprandial blood glucose (PPBG) of 140–199 mg/dl or/and HbA_{1c} of 5.7% to 6.4%. Diabetic group was selected as per American Diabetic Association guidelines.[17] The cut-off for defining dyslipidemia in FLP was as per the National Cholesterol Education Program, Adult Treatment Panel III guidelines.[18] The cut-off for defining dyslipidemia in postprandial lipid profile (PPLP) was based on European Atherosclerosis Society and the European Federation of Clinical Chemistry and Laboratory Medicine.[19]

Exclusion criteria

Patients with a history of type-I diabetes, obesity, diabetes mellitus on treatment, renal impairment (serum creatinine >1.5 mg/dl), hepatic diseases, acute illnesses, thyroid disorders, alcoholics, pregnancy, and those on drug therapy that interfered with the serum lipid levels were excluded from the study.

Biochemical parameters

Under aseptic conditions, overnight fasting blood samples were used for analysis of FLP, FBG, and glycated haemoglobin (HbA_{1c}). Two-hour postprandial blood samples were analyzed for PPLP and PPBG. FBS, PPBS, total cholesterol (TC), HDLc, and TG were analyzed in an automated chemistry analyser (BC1200 clinical chemistry analyser, Snibe diagnostics) using commercial assay kits from Biolabo, Germany. HbA_{1c} was analyzed by immunoturbidimetric assay in BC 1200 clinical chemistry analyser, Snibe diagnostics. The atherogenic lipid ratios TC/HDLc, LDLc/HDLc, and TG/HDLc (marker of insulin resistance),[20] were calculated in FLP and PPLP. Low-density lipoprotein (LDLc) was calculated by Friedwald's formula.

Statistical analysis

Data was analyzed with the statistical package; SPSS-20. Mean and standard deviation were used to assess the level of the study parameters. The normality of data was tested using Kolmogorov–Smirnov and Shapiro–Wilk tests. Differences between mean among the groups were compared by one-way ANOVA. Pearson's regression analysis was applied to find the association between the study parameters. P value < 0.05 was considered statistically significant.

Results

Baseline characteristics

Of the total 83 study subjects, 53% were males and rest 47% were females as shown in Table 1. The mean age of the cases in the prediabetes group was observed to be 54.41 ± 13.23 years, whereas that of diabetics was 55.53 ± 9.83 years. No significant difference in the age was observed between cases and controls ($P > 0.05$) [Table 1].

Fasting glycemic indices and fasting lipid parameters

Table 2 highlights the mean ± SD of glycemic indices, lipid profile indices along with the atherogenic ratios in fasting state among the different study groups. HbA_{1c} was significantly different in the 3 study groups ($P < 0.01$ in case of controls versus prediabetes, $P < 0.001$ in case of controls versus diabetes; prediabetes versus diabetes). FBG in diabetes was found to be significantly raised when compared to controls and prediabetes group ($P < 0.001$). Diabetes group were found to have significantly higher serum TC when compared to controls ($P < 0.01$) and prediabetes ($P < 0.05$).

Postprandial glycemic indices and postprandial lipid parameters

Table 3 depicts the mean ± SD of glycemic indices, lipid profile along with the atherogenic ratios in 2-h postprandial state among the different study groups. PPBG was increased significantly as one moved from the control group to the diabetic group. Serum TG was found to be increased significantly in diabetes compared to controls ($P < 0.01$) and prediabetes group ($P < 0.05$). In the diabetes group, TG/HDLc was also significantly increased ($P < 0.05$) compared to controls.

Intragroup analysis of lipid parameters and blood glucose

Table 4 represents a comparative analysis of fasting and postprandial glycemic indices, lipid profile indices, and

Table 1: Demographic data in the study groups

		Number	Percentage	Age in years (gender-wise)	Average age in years	*P*
Control	Male	13	46.4%	53	49.43	*P*>0.05
	Female	15	53.5%	46		
Prediabetics	Male	16	50%	55	54.41	
	Female	16	50%	53		
Diabetics	Male	28	54.9%	52	55.53	
	Female	23	45.1%	58		

Table 2: Fasting glycemic indices and fasting lipid parameters in the study groups

	Controls	Prediabetes	Diabetes	*P*
HbA1c (%)	5.1±0.35	5.7±0.47	7.0±0.88	<0.001 (P: D, C:D) <0.01 (C:P)
FBG (mg/dl)	85.7±9.2	106.7±11.9	167.5±73.6	<0.001 (P: D, C: D)
TC (mg/dl)	169.8±35.4	173.6±35.0	196.7±41.2	<0.01 (C: D) <0.05 (P: D)
TG (mg/dl)	113.5±68.9	119.6±51.7	147.7±65.9	Not significant
HDLc (mg/dl)	44.8±8.6	46.5±8.7	47.7±8.3	Not significant
LDLc (mg/dl)	102.3±33.9	103.0±33.3	119.4±38.9	Not significant
TC/HDLc	3.9±0.91	3.82±0.95	4.2±1.09	Not significant
LDLc/HDLc	2.31±0.79	2.30±0.92	2.59±1.01	Not significant
TG/HDLc	2.9±2.9	2.62±1.3	3.2±1.3	Not significant

C=controls, P=prediabetes, D=diabetes

Table 3: Postprandial glycemic indices, postprandial lipid parameters in the study groups

	Controls	Prediabetes	Diabetes	*P*
PPBG (mg/dl)	120.7±21.3	155.9±28.8	256.1±73.2	<0.001 (P: D, C: D) <0.05 (C: P)
TC (mg/dl)	171.5±35.4	175.1±39.0	190.0±48.4	Not significant
TG (mg/dl)	136.2±74.7	150.5±56.0	191.8±77.0	<0.01 (C: D) <0.05 (P: D)
HDLc (mg/dl)	49.6±11.7	47.8±9.2	47.3±10.1	Not significant
LDLc (mg/dl)	94.6±34.9	97.2±37.8	104.3±46.7	Not significant
TC/HDLc	3.6±1.02	3.8±1.02	4.1±1.15	Not significant
LDLc/HDLc	2.0±0.83	2.1±0.97	2.3±1.08	Not significant
TG/HDLc	3.1±2.68	3.3±1.54	4.3±1.84	<0.05 (C: D)

C=controls, P=prediabetes, D=diabetes

atherogenic ratios within the study groups. Except TC and TG/HDLc, all the other parameters were significantly different in the fasting and postprandial state in the control group. TG and TG/HDLc were significantly increased in postprandial state when compared with the fasting state in prediabetes and diabetes ($P < 0.001$).

Correlation analysis of fasting and postprandial lipid parameters with HbA_{1c}

Table 5 represents the correlation between fasting and postprandial lipid with the glycemic status (HbA_{1c}). Postprandial TG ($P < 0.01$) and TG/HDLc ($P < 0.01$) showed a stronger correlation with HbA_{1c} than fasting TG ($P < 0.05$) and TG/HDLc (TG/HDLc $P > 0.05$). Both fasting and postprandial HDLc, LDLc and HDLc/LDLc did not correlate significantly with HbA_{1c}.

Prevalence of dyslipidemia and insulin resistance

Table 6 evaluates the prevalence of fasting and postprandial dyslipidemia (as per increased TG) and insulin resistance (as per TG/HDLc ≥3.5) in the study groups. The prevalence of dyslipidemia and insulin resistance was higher in the postprandial state than the fasting state.

Discussion

Dyslipidemia in diabetes is a risk factor for coronary heart disease. The performance of traditional lipid measurements has provided substantial information in the prediction of cardiovascular disease in diabetes.[21] However, this is the first study to report on the comparison of the fasting and postprandial lipid variables, for cardiovascular risk evaluation, in prediabetes and diabetes, in a single setting.

In the present study, FLP in prediabetes did not differ significantly from the healthy individuals, in contrast to an earlier study that showed a deranged FLP in prediabetes.[15] Postprandially, triglycerides and TG/HDLc were significantly increased in diabetes compared to prediabetes, following regular meal intake. But similar changes were not reflected in prediabetes compared to the controls. Diabetic dyslipidemia is due to an increased supply of fatty acids to the liver and defective hepatic clearance of lipoproteins, which are further exaggerated postprandially with an additional adverse effect of meal-induced hyperglycaemia.[22] Triglyceride-rich lipoproteins accumulated in the postprandial state promote the formation of small dense low-density lipoproteins, closely related to the development of the inflammation, oxidative stress, and endothelial dysfunction, all of which attribute to cardiovascular disease. An earlier study displayed significant postprandial hypertriglyceridemia in diabetes, and not in prediabetes, after a standard oral fat meal challenge.[16] In the fasting samples, the hypertriglyceridemia was not seen in diabetes as well. This is in accordance with earlier studies that demonstrated significant lipid abnormalities, particularly of triglycerides in postprandial state and not in fasting state.[23,24] Postprandial hypertriglyceridemia despite normal fasting triglycerides has shown to be an independent risk factor for early atherosclerosis in type-2 diabetes.[25] In the present study, the stronger correlation of postprandial TG and TG/HDLc with HbA_{1c} compared to the respective fasting parameters suggests

the presence of postprandial pro-atherogenic environment with altered glycaemic status. An earlier study documented that postprandial lipaemia seen in diabetes was independent of glycaemic control in diabetes.[23]

Postprandial evaluation of TG and TG/HDL suggested deranged lipid abnormalities in diabetes. However, its estimation could not distinguish the prediabetes from healthy individuals. As glycaemia has an impact on the lipid abnormalities and cardiovascular disease, an attempt was taken up to compare the FLP and PPLP within each of the individual groups. Postprandially, after normal food intake, TG was increased significantly in healthy individuals, prediabetes, and diabetes. Similar changes were seen in an earlier study in diabetes.[26] Our study took into account even prediabetes subjects. As HDLc did not vary significantly with glycaemic status, TG/HDLc became significant lipid ratio parameter, in comparing FLP and PPLP within prediabetes and diabetes. Moreover, the stronger correlation between TG and TG/HDLc postprandially with glycaemic status further emphasize the importance of postprandial dyslipidemia in the pathogenesis of the disease.

Table 4: Intragroup analysis of lipid parameters and blood glucose

		Controls	Prediabetes	Diabetes
Blood glucose	Fasting	85.7±9.2	106.7±11.9	167.5±73.6
	Postprandial	120.7±21.3	155.9±28.8	256.1±73.2
	P	0.000***	0.000***	0.000***
TC	Fasting	169.8±35.4	173.6±35.0	196.7±41.2
	Postprandial	171.5±35.4	175.1±39.0	190.0±48.4
	P	0.438	0.699	0.089
TG	Fasting	113.5±68.9	119.6±51.7	147.7±65.9
	Postprandial	136.2±74.7	150.5±56.0	191.8±77.0
	P	0.000***	0.000***	0.000***
HDLc	Fasting	44.8±8.6	46.5±8.7	47.7±8.3
	Postprandial	49.6±11.7	47.8±9.2	47.3±10.1
	P	0.008**	0.321	0.605
LDLc	Fasting	102.3±33.9	103.0±33.3	119.4±38.9
	Postprandial	94.6±34.9	97.2±37.8	104.3±46.7
	P	0.002**	0.157	0.000***
TC/HDLc	Fasting	3.9±0.91	3.82±0.95	4.2±1.09
	Postprandial	3.6±1.02	3.8±1.02	4.1±1.15
	P	0.020*	0.577	0.246
LDLc/HDLc	Fasting	2.31±0.79	2.30±0.92	2.59±1.01
	Postprandial	2.0±0.83	2.1±0.97	2.3±1.08
	P	0.000***	0.053	0.000***
TG/HDLc	Fasting	2.9±2.9	2.62±1.3	3.2±1.3
	Postprandial	3.1±2.68	3.3±1.54	4.3±1.84
	P	0.325	0.000***	0.000***

* *P*<0.05, ** *P*<0.01, *** *P*<0.001

An earlier study reported a modest variation in triglycerides and no variations in other lipid parameters with timely postprandial measurements, following regular food intake in healthy individuals and diabetes.[26] In the present study, lipid concentrations were analyzed at 2 h postprandially, after the ingestion of the last ordinary meal. This is because the postprandial triglycerides reach a peak at 3–4 h after the meal and slowly return to initial levels at 6–8 h. With regular food habits and dietary timings, the blood level of triglycerides at 2 h postprandially is maintained throughout the maximum period of the day.[27] The most obvious advantage of sample collection postprandially at 2 h over FLP measurements in diabetes is the simplification of blood-sampling process for both glucose and lipid parameters, preventing an extra prick to the patient, thus improving the compliance.

The postprandial TG and TG/HDLc indicated a higher prevalence of dyslipidemia and insulin resistance in both prediabetes and diabetes than the respective fasting parameters. The findings of this study suggest postprandial TG and TG/HDLc to be superior to other fasting or postprandial lipid parameters under study in the evaluation of the presence of dyslipidemia in prediabetes and diabetes. This signifies that, in the evaluation of cardiovascular risk assessment, PPLP, particularly postprandial TG and TG/HDLc should be used adjunct to the conventional fasting lipid parameters, in both prediabetes and diabetes. However, similar studies with larger sample size need to be assessed for the validation of the postprandial TG/HDLc in a clinical setting. Postprandial time is not known to affect the associations between lipid concentrations or influence cardiovascular disease risk in patients with diabetes.[13] Studies on the association between postprandial lipids and risk of cardiovascular disease in diabetes are few with conflicting results. A significant association of triglycerides and carotid intima media thickness postprandially and not in fasting state was documented earlier.[25] In contradiction to the present study, researchers have failed to find an association between nonfasting triglycerides and cardiovascular disease in diabetes.[28-30]

There were some limitations in the present study. The timing of the last meal was self-reported. The data regarding the intake of the regular meals was based on questionnaire and not based on direct supervision. Emerging lipid markers were not evaluated in

Table 5: Pearson's correlation analysis of fasting and postprandial lipid parameters with HbA_{1c}

	TC	TG	HDLC	LDLC	TC/HDLC	TG/HDLC	LDLC/HDLC
Fasting profile							
R	0.189	0.226	0.091	0.105	0.111	0.101	0.079
P	0.046*	0.017*	0.343	0.273	0.246	0.292	0.412
Postprandial profile							
r	0.094	0.280	-0.129	0.029	0.161	0.260	0.076
P	0.324	0.003**	0.178	0.761	0.090	0.006**	0.429

* *P*<0.05, ** *P*<0.01

Table 6: Prevalence of dyslipidemia and insulin resistance in the study groups

	Controls	Prediabetes	Diabetes
Prevalence of dyslipidemia			
Fasting TG (≥150 mg/dl)	5 (17.8%)	7 (22.5%)	21 (41.1%)
Postprandial TG (≥175 mg/dl)	5 (17.8%)	9 (29.03%)	28 (54.9%)
Prevalence of insulin resistance			
TG/HDLc (Fasting≥3.5)	3 (10.7%)	4 (12.5%)	17 (33.3%)
TG/HDLc (Postprandial≥3.5)	6 (21.4%)	10 (31.25%)	30 (58.8%)

the present study. A comparative evaluation of the lipid profile fasting and postprandially in both prediabetes and diabetes, in a single setting, is the strength of the present study. The evaluation of postprandial samples at 2 h is uniqueness, giving scope for future validation in a hospital and clinical setting, where serial postprandial sampling becomes cumbersome to the patient in day-to-day practice.

Conclusion

Postprandial TG and the TG/HDLc reflect lipid abnormalities than the corresponding fasting variables in diabetes and prediabetes. Postprandial TG and TG/HDLc are better reflectors of cardiovascular status in prediabetes and diabetes.

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Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patients have given their consent for their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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