



Research Article

Comparison of Fasting, Two-Hour and Six-Hour Postprandial Lipid Profiles in Patients with Controlled Type 2 Diabetes Mellitus

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Abstract: Background: Postprandial lipid changes may influence the interpretation of lipid profiles in patients with type 2 diabetes mellitus. Understanding variation at defined intervals after a meal can help clarify the relationship between fasting and postprandial measurements. **Objective:** To compare fasting, two-hour and six-hour postprandial lipid profiles in patients with controlled type 2 diabetes mellitus. **Methods:** This hospital-based prospective comparative observational study included 50 patients aged 35-65 years with controlled type 2 diabetes mellitus. Blood samples were collected after an overnight fast and at two and six hours after a meal. Total cholesterol, triglycerides and HDL-C were measured, while LDL-C and VLDL-C were calculated. Results were summarised as mean (SD), and differences across sampling times were assessed using repeated-measures ANOVA. A p value below 0.05 was considered statistically significant. **Results:** Mean triglycerides increased from 144.72 (73.41) mg/dL during fasting to 167.12 (85.76) mg/dL at two hours and 175.12 (79.46) mg/dL at six hours (overall p = 0.0001). HDL-C decreased from 56.00 (10.78) to 52.42 (9.63) and 51.10 (9.27) mg/dL, respectively (p = 0.0002). Reported VLDL-C increased from 28.94 (14.68) to 33.41 (17.16) and 34.95 (15.96) mg/dL (p = 0.0003). Overall differences in total cholesterol (p = 0.063) and reported LDL-C (p = 0.081) were not statistically significant. At six hours, the triglyceride group mean was 21.01% above fasting, while HDL-C was 8.75% lower. Pairwise significance could not be established from the available summary data, and calculated lipid values required verification. **Conclusion:** Postprandial changes were evident in triglycerides, HDL-C and reported VLDL-C, whereas total cholesterol and reported LDL-C showed smaller changes. Six-hour measurements did not uniformly approximate fasting concentrations. These findings support considering sampling time during interpretation but do not establish interchangeability between fasting and six-hour postprandial lipid profiles.

Keywords: Type 2 Diabetes Mellitus Postprandial Lipemia, Lipid Profile

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INTRODUCTION

Type 2 diabetes mellitus is associated with disturbances in carbohydrate and lipid metabolism that contribute to increased cardiovascular risk. Diabetic dyslipidaemia commonly includes elevated triglycerides, reduced high-density lipoprotein cholesterol (HDL-C), and an increased proportion of small, dense low-density lipoprotein particles. These abnormalities reflect changes in lipoprotein production, composition and clearance associated with insulin resistance. Athyros *et al.* [1] described the quantitative, qualitative and kinetic disturbances underlying diabetic dyslipidaemia and emphasised their contribution to cardiovascular disease. Consequently, assessment of lipid abnormalities remains important even in patients whose diabetes is considered controlled.

Lipid profiles have traditionally been measured after an overnight fast to minimise meal-related variation, particularly in triglyceride concentrations. However, fasting requirements can create practical difficulties for patients and laboratories. Nordestgaard *et al.* [2] concluded that fasting was not routinely necessary for lipid assessment because average changes after habitual meals were generally modest. Their consensus statement supported nonfasting lipid testing for most routine assessments while retaining fasting measurements for selected clinical circumstances. Nevertheless, population-level evidence does not establish that every lipid fraction remains unchanged at specific postprandial intervals in patients with diabetes.

Postprandial lipid measurements provide information about the handling of dietary lipids following a meal. Kolovou *et al.* [3] discussed postprandial hypertriglyceridaemia as a potentially relevant marker of cardiovascular risk and highlighted the influence of meal composition, obesity, physical activity, medications and other individual characteristics. Comparing fasting measurements with those obtained two and six hours after a meal can therefore characterise early and later postprandial changes within the same patient. Such comparisons are particularly relevant when assessing whether lipid concentrations have approached fasting values by six hours.

Mora *et al.* [4] found that fasting and no fasting lipid concentrations showed similar associations with subsequent cardiovascular events, with high agreement in cardiovascular risk classification. However, interpretation also depends

on the method used to determine individual lipid fractions. Sampson *et al.* [5] demonstrated the importance of the calculation method for LDL-C, particularly in patients with elevated triglyceride concentrations. Thus, changes in calculated LDL-C must be interpreted alongside changes in triglycerides and the limitations of the calculation used. The present study was undertaken to compare fasting, two-hour and six-hour postprandial lipid profiles in patients with controlled type 2 diabetes mellitus and to describe the magnitude and pattern of within-patient variation across these sampling times.

Aim

To compare fasting, two-hour and six-hour postprandial lipid profiles in patients with controlled type 2 diabetes mellitus.

Objectives

- To determine total cholesterol, triglycerides, HDL-C, calculated LDL-C and calculated VLDL-C concentrations in fasting, two-hour and six-hour postprandial samples
- To compare the lipid parameters across the three sampling times within the same patients
- To quantify the absolute and percentage changes in lipid parameters at two and six hours after a meal relative to fasting values

MATERIALS AND METHODS

Source of Data

Data were obtained from patients with previously diagnosed type 2 diabetes mellitus attending the outpatient department of the study hospital. Clinical information was collected through patient interviews, examination and available medical records. Laboratory data were obtained from fasting, two-hour postprandial and six-hour postprandial blood samples.

The original study included 50 patients with controlled type 2 diabetes mellitus and 50 nondiabetic controls. For the present title, the study population comprised the 50 diabetic patients; the nondiabetic comparison group was outside the scope of the within-patient analysis.

Study Design

The study was conducted as a hospital-based, prospective comparative observational study with repeated measurements. Each participant underwent lipid assessment at three time points and served as their own comparator. Consequently, the three sets of measurements represented related observations rather than independent groups.

Study Location

The study was conducted in the Department of Biochemistry at Grant Government Medical College and Sir J. J. Group of Hospitals, Mumbai, with participants recruited from the outpatient department.

Study Duration

The study was conducted for 12 months.

Sample Size and Formula

The study included 50 patients with controlled type 2 diabetes mellitus.

Inclusion Criteria

Patients were eligible when they met the following criteria:

- Age between 35 and 65 years
- Either sex
- Previously diagnosed type 2 diabetes mellitus
- Controlled diabetes according to the study's stated operational criterion of HbA1c below 6.4%
- Dyslipidaemia, as specified in the original eligibility criteria
- Provision of informed consent

Exclusion Criteria

Patients were excluded if they had any of the following:

- Age outside the specified range of 35-65 years

- Type 1 diabetes mellitus
- Hypothyroidism
- A history of smoking or alcoholism, as described in the original protocol
- A family history of dyslipidaemia
- Hypertension or hepatic, renal or cardiac disease
- Pregnancy or lactation
- Use of medications considered likely to interfere with lipid assessment, including lipid-lowering agents, beta blockers and corticosteroids

Procedure and Methodology

Patients attending the outpatient department were screened against the eligibility criteria. The purpose of the study and the requirement for repeated blood collection were explained, and informed consent was obtained.

Clinical assessment included demographic information, duration of diabetes, treatment history, relevant medical and family history, and examination findings. Height and weight were recorded.

Fasting blood samples for lipid estimation were collected after an overnight fast of 10-12 hours. Subsequent samples were collected from the same participants at two and six hours after a meal. Total cholesterol, triglycerides and HDL-C were measured at each time point, while VLDL-C and LDL-C were calculated. HbA1c was measured to assess the study-defined glycaemic control status. Plasma glucose was also measured in the original study.

The source did not adequately describe meal composition, caloric content, intervening food intake or the timing of antidiabetic medications. These details require verification from the study records before describing the meal as standardised or attributing all observed differences solely to elapsed postprandial time.

Sample Processing

Blood was collected under aseptic precautions into plain tubes for lipid estimation, fluoride-containing tubes for plasma glucose estimation and EDTA tubes for HbA1c measurement.

Serum and plasma were separated by centrifugation at 3,000 rpm for 3-5 minutes, as reported in the study. EDTA-anticoagulated whole blood was used for HbA1c estimation. Chemistry samples were processed within two hours of collection using an Erba XL 640 automated analyser. HbA1c was estimated separately by capillary electrophoresis using the CAPILLARYS 2 Flex-Piercing system.

Calculated LDL-C and VLDL-C require cautious interpretation in postprandial samples. In particular, calculated VLDL-C is mathematically determined by triglycerides and does not provide an independent measurement of postprandial lipoprotein metabolism. The Friedewald equation should not be used when triglycerides are at least 400 mg/dL; the handling of such observations must be verified from the records Table 1.

Statistical Methods

Data were entered into Microsoft Excel and analysed using IBM SPSS Statistics version 25. Continuous variables were summarised as mean and standard deviation, and categorical variables were expressed as frequencies and percentages. Repeated-measures analysis of variance was used to compare lipid concentrations across the fasting, two-hour and six-hour postprandial measurements. A two-sided p value below 0.05 was considered statistically significant.

For the final analysis, the following additions are recommended and should be described as completed only after they have been performed: distributions and model residuals should be assessed; sphericity should be examined, with Greenhouse-Geisser correction where required; and pairwise comparisons should account for multiple testing. Markedly skewed measurements may require transformation or a Friedman test with adjusted paired comparisons.

Mean differences should be reported with 95% confidence intervals, alongside test statistics and p values. A nonsignificant difference should not be interpreted as proof that fasting and postprandial measurements are equivalent.

Table 1: The Biochemical Methods

Parameter	Method
Total cholesterol	Cholesterol oxidase-peroxidase method
Triglycerides	Glycerol phosphate oxidase enzymatic method
HDL-C	Direct homogeneous assay
VLDL-C	Calculated as triglycerides divided by 5
LDL-C	Calculated using the Friedewald equation
Plasma glucose	Glucose oxidase-peroxidase method
HbA1c	Capillary electrophoresis

Data Collection

Information was collected using the study case-record form. Recorded variables included age, sex, medical history, duration of diabetes, treatment history, family history, anthropometric measurements, examination findings and biochemical results. Lipid and glucose measurements were recorded separately for fasting, two-hour and six-hour postprandial samples, while HbA1c was recorded as an indicator of glycaemic control.

RESULTS

Among the 50 patients with controlled type 2 diabetes mellitus, mean triglyceride concentrations increased from 144.72 (73.41) mg/dL during fasting to 167.12 (85.76) mg/dL at two hours and 175.12 (79.46) mg/dL at six hours postprandially, with a statistically significant overall difference across sampling times ($p = 0.0001$). Mean HDL-C decreased progressively from 56.00 (10.78) mg/dL to 52.42 (9.63) mg/dL and 51.10 (9.27) mg/dL, respectively ($p = 0.0002$). Reported VLDL-C increased from 28.94 (14.68) mg/dL during fasting to 33.41 (17.16) mg/dL at two hours and 34.95 (15.96) mg/dL at six hours ($p = 0.0003$). Total cholesterol increased slightly at two hours before approaching its fasting concentration at six hours, without a statistically significant overall difference ($p = 0.063$). Reported LDL-C followed a similar pattern, with no significant overall difference across the three measurements ($p = 0.081$). These overall comparisons did not identify which specific pairs of sampling times differed significantly Table 2.

The confidence intervals illustrated the precision of the mean lipid concentrations at each sampling time. Mean total cholesterol was 178.02 mg/dL (95% CI: 168.55-187.49) during fasting, 182.18 mg/dL (95% CI: 173.71-190.65) at two hours and 178.24 mg/dL (95% CI: 169.64-186.84) at six hours. Mean triglycerides increased from 144.72 mg/dL (95% CI: 123.86-165.58) to 167.12 mg/dL (95% CI: 142.75-191.49) and 175.12 mg/dL (95% CI: 152.54-197.70), respectively. Conversely, mean HDL-C decreased from 56.00 mg/dL (95% CI: 52.94-59.06) to 52.42 mg/dL (95% CI: 49.68-55.16) and 51.10 mg/dL (95% CI: 48.47-53.73). Reported LDL-C means were 96.86, 99.42 and 96.30 mg/dL, with corresponding 95% confidence intervals of 89.48-104.24, 92.65-106.19 and 89.82-102.78 mg/dL. Reported VLDL-C increased from 28.94 mg/dL (95% CI: 24.77-33.11) to 33.41 mg/dL (95% CI: 28.53-38.29) and 34.95 mg/dL (95% CI: 30.41-39.49). These confidence intervals described individual time-point means and did not establish the significance of paired differences Table 3.

The pairwise differences calculated from the reported means showed that triglycerides increased by 22.40 mg/dL at two hours and 30.40 mg/dL at six hours relative to fasting, with a further increase of 8.00 mg/dL between two and six hours. HDL-C decreased by 3.58 mg/dL at two hours and 4.90 mg/dL at six hours' relative to fasting, including a

Table 2: Comparison of Fasting, Two-Hour and Six-Hour Postprandial Lipid Profiles in Patients with Controlled Type 2 Diabetes Mellitus (N = 50)

Lipid parameter (mg/dL)	Fasting, Mean (SD)	Two-hour Postprandial, Mean (SD)	Six-hour Postprandial, Mean (SD)	Test of Significance	F statistic	Overall p value*
Total cholesterol	178.02 (33.31)	182.18 (29.81)	178.24 (30.27)	Repeated-measures ANOVA	Not reported	0.063
Triglycerides	144.72 (73.41)	167.12 (85.76)	175.12 (79.46)	Repeated-measures ANOVA	Not reported	0.0001
HDL-C	56.00 (10.78)	52.42 (9.63)	51.10 (9.27)	Repeated-measures ANOVA	Not reported	0.0002
LDL-C†	96.86 (25.97)	99.42 (23.83)	96.30 (22.79)	Repeated-measures ANOVA	Not reported	0.081
VLDL-C†	28.94 (14.68)	33.41 (17.16)	34.95 (15.96)	Repeated-measures ANOVA	Not reported	0.0003

Table 3: Lipid Concentrations and 95% Confidence Intervals at Fasting, Two-Hour and Six-Hour Postprandial Sampling (N = 50)

Lipid parameter	Sampling time	Mean (SD), mg/dL	95% CI for the mean, mg/dL	Overall p value*
Total cholesterol	Fasting	178.02 (33.31)	168.55-187.49	0.063
	Two hours	182.18 (29.81)	173.71-190.65	
	Six hours	178.24 (30.27)	169.64-186.84	
Triglycerides	Fasting	144.72 (73.41)	123.86-165.58	0.0001
	Two hours	167.12 (85.76)	142.75-191.49	
	Six hours	175.12 (79.46)	152.54-197.70	
HDL-C	Fasting	56.00 (10.78)	52.94-59.06	0.0002
	Two hours	52.42 (9.63)	49.68-55.16	
	Six hours	51.10 (9.27)	48.47-53.73	
LDL-C†	Fasting	96.86 (25.97)	89.48-104.24	0.081
	Two hours	99.42 (23.83)	92.65-106.19	
	Six hours	96.30 (22.79)	89.82-102.78	
VLDL-C†	Fasting	28.94 (14.68)	24.77-33.11	0.0003
	Two hours	33.41 (17.16)	28.53-38.29	
	Six hours	34.95 (15.96)	30.41-39.49	

Table 4: Within-Patient Pairwise Comparisons of Lipid Parameters Across the Three Sampling Times (N = 50)

Lipid parameter	Comparison: later minus earlier	Mean difference, mg/dL
Total cholesterol	Two hours - fasting	+4.16
	Six hours - fasting	+0.22
	Six hours - two hours	-3.94
Triglycerides	Two hours - fasting	+22.40
	Six hours - fasting	+30.40
	Six hours - two hours	+8.00
HDL-C	Two hours - fasting	-3.58
	Six hours - fasting	-4.90
	Six hours - two hours	-1.32
LDL-C†	Two hours - fasting	+2.56
	Six hours - fasting	-0.56
	Six hours - two hours	-3.12
VLDL-C†	Two hours - fasting	+4.47
	Six hours - fasting	+6.01
	Six hours - two hours	+1.54

Table 5: Absolute and Percentage Changes in Lipid Concentrations at Two and Six Hours Relative to Fasting (N = 50)

Lipid parameter	Two-hour absolute change, mg/dL	Two-hour change, %	Six-hour absolute change, mg/dL	Six-hour change, %
Total cholesterol	+4.16	+2.34	+0.22	+0.12
Triglycerides	+22.40	+15.48	+30.40	+21.01
HDL-C	-3.58	-6.39	-4.90	-8.75
LDL-C†	+2.56	+2.64	-0.56	-0.58
VLDL-C†	+4.47	+15.45	+6.01	+20.77

further reduction of 1.32 mg/dL between the postprandial measurements. Total cholesterol increased by 4.16 mg/dL at two hours and subsequently decreased by 3.94 mg/dL, leaving a six-hour concentration only 0.22 mg/dL above fasting. Reported LDL-C increased by 2.56 mg/dL at two hours before decreasing by 3.12 mg/dL, resulting in a six-hour concentration 0.56 mg/dL below fasting. Reported VLDL-C increased by 4.47 and 6.01 mg/dL at two and six hours, respectively. Paired confidence intervals, test statistics and p values were unavailable; therefore, these differences represented descriptive comparisons without confirmed pairwise statistical significance Table 4.

Relative to fasting, the triglyceride group mean increased by 22.40 mg/dL (15.48%) at two hours and 30.40 mg/dL (21.01%) at six hours. Reported VLDL-C increased by 4.47 mg/dL (15.45%) and 6.01 mg/dL (20.77%), respectively. HDL-C decreased by 3.58 mg/dL (6.39%) at two hours and 4.90 mg/dL (8.75%) at six hours. Total cholesterol increased by 4.16 mg/dL (2.34%) at two hours but was only 0.22 mg/dL (0.12%) above fasting at six hours. Reported LDL-C increased by 2.56 mg/dL (2.64%) at two hours and decreased by 0.56 mg/dL (0.58%) relative to fasting at six hours. These percentages represented changes in group means rather than average individual percentage changes. Their statistical significance could not be assessed because confidence intervals and paired test results were unavailable Table 5.

DISCUSSION

The present study evaluated fasting, two-hour and six-hour postprandial lipid profiles in 50 patients with controlled type 2 diabetes mellitus. The principal findings were progressively higher triglyceride and reported VLDL-C concentrations and lower HDL-C concentrations across the sampling times. Total cholesterol and reported LDL-C showed comparatively small changes, without statistically significant overall differences. These findings suggest that individual components of the lipid profile responded differently to the postprandial state. Athyros *et al.* [1] described diabetic dyslipidaemia as a combination of quantitative, qualitative and kinetic lipoprotein abnormalities associated with insulin resistance. Accordingly, satisfactory glycaemic control should not be assumed to indicate normal postprandial lipid metabolism.

Nordestgaard *et al.* [2] reported that the maximal average changes following habitual meals were approximately +26 mg/dL for triglycerides and -8 mg/dL for total cholesterol and LDL-C, whereas HDL-C was largely unaffected. In the present study, triglycerides increased by 22.40 mg/dL at two hours and 30.40 mg/dL at six hours. These changes were broadly comparable in magnitude to the consensus estimates, although the six-hour increase was somewhat greater. In contrast, total cholesterol increased slightly rather than decreasing, and HDL-C showed a more pronounced decline. These differences indicate that population-average findings should not be applied directly to every diabetic cohort or sampling protocol.

Kolovou *et al.* [3] emphasised that postprandial triglyceride responses depend on meal composition, age, adiposity, physical activity, alcohol consumption and medication use. Such factors could contribute to differences between the present findings and published studies. However, the available study documentation did not adequately characterise meal composition or intervening food intake. Therefore, the higher six-hour triglyceride concentration cannot be

attributed specifically to delayed clearance. Mora *et al.* [4], in their analysis of 8,270 ASCOT-LLA participants, found similar associations of fasting and nonfasting lipid concentrations with cardiovascular events and high agreement in risk classification. Their findings support the clinical usefulness of nonfasting testing, but they do not establish numerical equivalence between fasting and fixed two-hour or six-hour measurements.

Interpretation also depends on how lipid fractions were determined. Sampson *et al.* [5] demonstrated that LDL-C estimation is influenced by the calculation method, particularly when triglycerides are elevated. This is relevant because LDL-C and VLDL-C in the present study were described as calculated parameters. The apparent stability of calculated LDL-C cannot independently establish that LDL particle concentration, composition or atherogenicity remained unchanged after a meal. Furthermore, discrepancies between the reported component means and the stated calculation equations require resolution before definitive interpretation of these fractions.

Comparison of Triglyceride Responses

In Table 2, mean triglycerides increased from 144.72 (73.41) mg/dL during fasting to 167.12 (85.76) mg/dL at two hours and 175.12 (79.46) mg/dL at six hours, with a reported overall p value of 0.0001. Hirano [6] described increased hepatic production and impaired removal of triglyceride-rich lipoproteins as central features of diabetic dyslipidaemia. This provides a plausible biological explanation for a sustained postprandial triglyceride elevation, although the present study did not directly measure lipoprotein production or clearance.

Nordestgaard [7] noted that the nonfasting state includes intestinally derived lipoproteins in addition to those originating from the liver and therefore represents much of an individual's usual daily metabolic exposure. Langsted and Nordestgaard [8] similarly reported that ordinary food intake generally produces modest lipid changes and that nonfasting lipid concentrations retain cardiovascular prognostic value. The present triglyceride findings are consistent with the expected direction of postprandial change. Nevertheless, the observed increase between two and six hours does not locate the actual triglyceride peak because intermediate and later measurements were unavailable.

Farukhi and Mora [9] highlighted the convenience of nonfasting testing and its potential to reduce fasting-related difficulties in patients with diabetes. A directly relevant comparison was provided by Chahal *et al.* [10], who studied 50 patients with type 2 diabetes and 50 controls. In their diabetic group, mean triglycerides increased from 172.59 mg/dL during fasting to 232.99 mg/dL postprandially, an absolute increase of 60.40 mg/dL. This exceeded both postprandial increases in the present study. Although both studies demonstrated postprandial triglyceride elevation, differences in baseline lipid concentrations, participant characteristics and sampling protocols prevent direct attribution of the difference to glycaemic control alone.

Ginsberg *et al.* [11] described the role of triglyceride-rich lipoproteins and their remnants in atherosclerotic cardiovascular disease. Their consensus supports the biological relevance of studying postprandial lipaemia. However, the present study measured conventional lipid concentrations rather than remnant particle number or cardiovascular outcomes. Consequently, the triglyceride rise represents a biochemical observation and cannot be translated into a quantified increase in cardiovascular risk.

Total cholesterol, LDL-C and VLDL-C

Total cholesterol increased from 178.02 mg/dL during fasting to 182.18 mg/dL at two hours and returned to 178.24 mg/dL at six hours. The overall difference was not statistically significant ($p = 0.063$). This contrasted with Chahal *et al.* [10], who reported a significant postprandial increase from 200.82 to 223.75 mg/dL. The comparatively small change in the present cohort may reflect differences in baseline metabolism or meal exposure, but these explanations remain untested. Reported LDL-C similarly changed little, from 96.86 mg/dL during fasting to 99.42 mg/dL at two hours and 96.30 mg/dL at six hours ($p = 0.081$).

The absence of a statistically significant LDL-C difference does not imply absence of atherogenic risk. Borén *et al.* [12] emphasised the causal role of LDL in atherosclerosis and the importance of particle retention and modification within the arterial wall. Varbo *et al.* [13] also demonstrated the cardiovascular relevance of elevated nonfasting remnant cholesterol in a large general-population investigation. These publications provide context for assessing atherogenic lipoproteins beyond changes in conventional LDL-C concentration, but neither establishes the prognostic significance of the short-term changes observed in this cohort.

Reported VLDL-C increased from 28.94 mg/dL during fasting to 33.41 mg/dL at two hours and 34.95 mg/dL at six hours ($p = 0.0003$). However, when VLDL-C is calculated as triglycerides divided by five, its changes are mathematically determined by triglycerides. It therefore cannot be regarded as an independent confirmation of altered lipoprotein metabolism. Moreover, exact scaling of triglyceride values would yield identical repeated-measures ANOVA statistics for triglycerides and VLDL-C. Their different reported p values indicate that the calculations or transcription should be checked.

HDL-C Changes

Mean HDL-C declined from 56.00 mg/dL during fasting to 52.42 mg/dL at two hours and 51.10 mg/dL at six hours, with a reported overall p value of 0.0002. This decline differed from the relative stability described by Nordestgaard *et al.* [2]. It was also larger than the change reported by Chahal *et al.* [10], whose diabetic participants had fasting and postprandial HDL-C means of 40.74 and 40.54 mg/dL, respectively. The present findings therefore suggest a different HDL-C response, although analytical variation, fluid intake and participant characteristics require consideration before assigning a biological explanation.

Vergès [14] described altered HDL metabolism and lipoprotein remodelling in diabetes, providing a mechanistic context for changes in HDL-C. Nevertheless, a short-term reduction in HDL-C concentration does not establish impaired HDL function. Sniderman *et al.* [15] emphasised the importance of apolipoprotein B particle burden in assessing atherogenic risk. Because neither HDL functionality nor apolipoprotein B was measured, the present study cannot determine whether the observed HDL-C decline was accompanied by a corresponding change in functional protection or atherogenic particle number.

Confidence Intervals and Within-Patient Changes

Table 2 showed substantial variability in triglyceride concentrations. The fasting mean was 144.72 mg/dL, with a 95% CI of 123.86-165.58 mg/dL, while the six-hour mean was 175.12 mg/dL, with a 95% CI of 152.54-197.70 mg/dL. These intervals describe uncertainty around the separate time-point means. Their overlap does not contradict the reported repeated-measures result because paired analysis incorporates the relationship between measurements from the same individual. Conversely, these intervals cannot be used to derive the confidence interval for the fasting-to-six-hour difference without information about within-patient covariance.

Langlois *et al.* [16] highlighted the importance of consistent analytical methods and the limitations of calculated LDL-C in hypertriglyceridaemia. Wilson *et al.* [17] similarly discussed the usefulness of non-HDL-C and the need to account for reduced accuracy of some LDL-C equations. These recommendations are relevant to future analyses of this cohort: non-HDL-C could be derived from total cholesterol and HDL-C, while validated LDL-C estimation or direct measurement could help clarify the reported calculation discrepancies. Such additional analyses should complement, rather than replace, verification of the original measurements.

Tables 4 and 5 provided descriptive estimates of the magnitude of change. At six hours, triglycerides were 30.40 mg/dL above fasting, corresponding to a 21.01% increase in the group mean, while HDL-C was 4.90 mg/dL lower, corresponding to an 8.75% decrease. Total cholesterol and reported LDL-C differed from fasting by only +0.22 and -0.56 mg/dL, respectively. These observations suggest greater meal-related variation in triglycerides and HDL-C than in total cholesterol or reported LDL-C. However, the percentages were calculated from group means and were not averages of individual percentage changes. Missing paired confidence intervals and adjusted pairwise p values prevent identification of statistically significant individual contrasts.

Clinical Relevance and Interpretation

Grundy *et al.* [18] and Mach *et al.* [19] placed lipid measurements within an overall cardiovascular risk assessment rather than interpreting a single lipid result in isolation. In that context, the present findings support recording sampling conditions and considering postprandial variation when interpreting serial lipid profiles. They do not establish that every patient requires both fasting and postprandial testing, nor do they demonstrate that six-hour samples are interchangeable with fasting samples.

Clinical outcome trials also show why biochemical changes should not be equated automatically with treatment benefit. Bhatt *et al.* [20] reported cardiovascular benefit with icosapent ethyl in a selected high-risk population with elevated triglycerides. In contrast, Das Pradhan *et al.* [21] found that pemafibrate did not reduce cardiovascular events in patients with type 2 diabetes despite lowering triglycerides and related lipid fractions. These trials were not studies of meal timing, but they demonstrate that the clinical implications of triglyceride concentrations depend on the intervention and patient population. The present observational findings cannot establish a treatment indication or treatment effect.

Finally, Wasserstein and Lazar [22] emphasised that statistical significance does not measure effect size or clinical importance. Thus, the small p values for triglycerides, HDL-C and reported VLDL-C should be interpreted alongside the magnitude of change and the limitations of the available analysis. Similarly, p values of 0.063 and 0.081 for total cholesterol and reported LDL-C do not prove equivalence across sampling times. The study demonstrates differing descriptive postprandial patterns among lipid fractions, but patient-level reanalysis, verification of calculated lipid values and documentation of meal conditions are necessary before making firm conclusions about pairwise differences or the suitability of six-hour testing as a substitute for fasting assessment.

CONCLUSION

Among 50 patients with controlled type 2 diabetes mellitus, triglyceride and reported VLDL-C concentrations increased, while HDL-C decreased across fasting, two-hour and six-hour postprandial measurements, with statistically significant overall differences. Total cholesterol and reported LDL-C showed no statistically significant overall differences. Triglyceride concentrations remained above fasting values at six hours, indicating that the lipid profile had not uniformly returned to fasting levels. These findings highlight the importance of considering sampling time when interpreting lipid measurements. However, the absence of paired confidence intervals and unresolved discrepancies in calculated lipid values preclude concluding that six-hour postprandial measurements are interchangeable with fasting measurements.

Limitations of The Study

The study was limited by its small sample of 50 patients and single-centre setting, which restricted the generalisability and precision of the findings. Selection of patients with controlled diabetes and the exclusion of several common comorbidities and lipid-altering medications further limited applicability to the broader diabetic population. Meal composition, caloric intake, intervening food consumption, physical activity and medication timing were insufficiently documented, making their influence on postprandial lipid changes difficult to assess. Measurements at only three time points could not establish the actual peak or complete duration of the postprandial response. LDL-C and VLDL-C were calculated rather than directly measured, and inconsistencies between the reported values and calculation equations required verification. Patient-level paired data, ANOVA F statistics, adjusted pairwise comparisons and confidence intervals for changes were unavailable, limiting interpretation of specific time-point differences. Furthermore, cardiovascular outcomes, apolipoprotein B, lipoprotein particle characteristics and HDL functionality were not evaluated. The study therefore could not establish the prognostic significance of the observed changes or demonstrate equivalence between fasting and postprandial testing.

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