

# Comparative study on fasting and postprandial lipid profile in type 2 diabetes mellitus

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## ABSTRACT

**Context:** Postprandial dyslipidemia plays a vital role in the pathogenesis of atherosclerosis and possible macrovascular complications in type 2 diabetes mellitus (DM). **Aims:** To assess and compare the fasting and postprandial lipid profiles in type 2 DM patients. **Settings and Design:** This case-control study was conducted in the Medicine department of a tertiary care teaching hospital. **Methods and Materials:** The study included 100 subjects; 50 type 2 diabetic patients and 50 healthy age- and gender-matched controls. Fasting and postprandial lipid levels were estimated in all the subjects and compared. **Statistical Analysis Used:** The Student's *t*-test and the analysis of variance (ANOVA) test were used for comparison between two and more than two groups, respectively, for normally distributed data. **Results:** Mean total cholesterol (TC), triglyceride (TG), low density lipoprotein (LDL), and very low density lipoprotein (VLDL) levels were significantly higher and high density lipoprotein (HDL) level was significantly lower in the diabetics in comparison to the controls in both fasting (200.82, 172.59, 126.20, 37.63, and 40.74 mg/dL in diabetics versus 179.90, 98.03, 109.54, 19.60, and 50.46 mg/dL in controls) and postprandial states (223.75, 232.99, 139.19, 46.52, and 40.54 mg/dL in diabetics versus 185.36, 102.20, 110.36, 20.24, and 48.96 mg/dL in controls). The mean postprandial TC and TG levels (223.75, 232.99 mg/dL) in diabetics were significantly higher when compared to their fasting values (200.82, 172.59 mg/dL) in these patients. **Conclusions:** Type 2 DM patients show significant postprandial lipid abnormalities particularly postprandial hypertriglyceridemia. Raised postprandial lipid parameters highlight that estimating lipids in the postprandial state is equally important as is estimation of lipids in the fasting state in type 2 DM.

**Keywords:** Fasting lipid profile, postprandial lipid profile, Type 2 diabetes mellitus

## Introduction

Type 2 diabetes mellitus (DM) is characterized by insulin resistance; a syndrome which includes glucose intolerance, dyslipidemia, and hypertension, and results in an increased predisposition to atherosclerotic vascular disease. The increased prevalence of cardiovascular disability in type 2 DM is believed to be because of a prolonged and exaggerated

postprandial dysmetabolism, most notably hyperglycemia and hypertriglyceridemia, which induce endothelial dysfunction and oxidative stress.<sup>[1]</sup> Thus, postprandial dyslipidemia is as significant as fasting dyslipidemia in causing atherosclerotic complications in type 2 DM.<sup>[2]</sup>

Diabetic dyslipidemia is believed to be a vital factor contributing to an increased cardiovascular risk in type 2 DM. However, postprandial hypertriglyceridemia in spite of normal fasting triglyceride (TG) levels may independently contribute to early atherosclerosis in type 2 DM.<sup>[3]</sup> Diabetic dyslipidemia includes quantitative as well as qualitative and kinetic lipoprotein derangements, all of which contribute to accelerated

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atherosclerosis.<sup>[4]</sup> The notable quantitative abnormalities are increased TG and decreased high density lipoprotein (HDL) levels. Qualitative abnormalities include an increase in small, dense low density lipoproteins (LDLs) and large, very low-density lipoprotein subfraction 1 (VLDL<sub>1</sub>). Other qualitative lipoprotein derangements include an increase in the TG content of LDL and HDL, glycation of apolipoproteins and heightened susceptibility of LDL to oxidation. The important kinetic lipoprotein abnormalities are characterized by an elevated VLDL<sub>1</sub> production, reduced VLDL catabolism, and increased HDL catabolism. LDL-cholesterol (LDL-C) levels may be normal in type 2 diabetics; however, LDL particles exhibit decreased catabolism, which contributes to atherogenesis in type 2 DM. Even though the pathogenesis of diabetic dyslipidaemia is not completely elucidated, the insulin resistance and relative insulin deficiency seen in type 2 DM are believed to be key factors leading to dyslipidemia, as insulin has a major role in regulation of lipid metabolism. Certain adipocytokines such as retinol-binding protein 4 and adiponectin may also be involved in the pathogenesis of diabetic dyslipidaemia.<sup>[4]</sup>

Traditionally, only the fasting TG levels are focussed upon while assessing the risk for atherosclerosis but recent studies have demonstrated that postprandial hypertriglyceridemia also contribute to endothelial dysfunction and atherosclerosis. Humans tend to be in the fed state most of the time than in the fasting state because of consumption of multiple meals and snacks in-between. Individuals with normal or near-normal fasting TG levels often show postprandial hypertriglyceridemia as a result of multiple meals.<sup>[5]</sup> TGs tend to be elevated for upto 3–4 h after a meal in normal individuals but upto 6–10 h in pre-diabetics and diabetics.<sup>[6]</sup> Thus, the vascular endothelium is exposed to the harmful effect of TGs mostly in the postprandial rather than the fasting phase. Consequently, most of the endothelial damage occurs during the postprandial phase.<sup>[7]</sup> Early recognition and appropriate treatment of significant postprandial dyslipidemia in diabetics by primary care physicians can be of paramount importance in reducing cardiovascular morbidity and mortality. Hence, it may be advisable that primary care physicians obtain lipid profile in both fasting and postprandial state in type 2 diabetics so as to address postprandial lipid abnormalities in these patients effectively by dietary and pharmacological interventions.

**Table 1: Baseline characteristics of the study population**

Parameters	Cases (Mean±S.D)	Controls (Mean±S.D)	P
Age (years)	51.86±10.25	51.20±9.66	0.79
Duration of Diabetes (years)	6.06±4.50	-	-
BMI (Kg/m <sup>2</sup> )	28.70±3.46	23.93±1.84	<0.001*
Waist to hip ratio			
Males	0.97±0.07	0.88±0.04	<0.001*
Females	0.98±0.22	0.87±0.04	<0.001*
HbA1C (gm/dl)	8.39±1.60	5.10±0.36	< 0.001*

This study was conducted to compare the fasting and postprandial lipid profiles in type 2 DM patients and to assess the significance of postprandial lipid profile in the cardiovascular risk stratification of these patients.

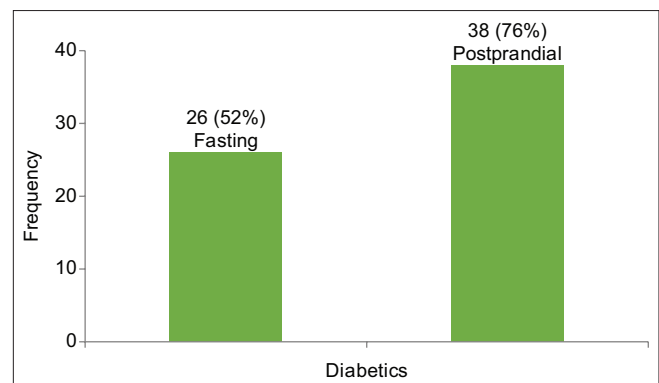
### Subjects and Methods

This case-control study, carried out in the Medicine department of a tertiary care teaching hospital, enrolled 50 diagnosed patients of type 2 DM {according to American Diabetes Association (ADA) guidelines} and 50 healthy age- and gender-matched controls attending the Medicine outpatient and in-patient departments. Permission was obtained from the Institutional Ethics Committee and written informed consent was obtained from each subject before enrolling him/her for the study (Date of approval: 07-11-2014).

**Inclusion criteria:** Both males and females aged more than 35 years having type 2 DM as per the ADA criteria were enrolled for the study.

**Exclusion criteria:** Those with type 1 DM, history of inherited disorders of lipid metabolism, coronary artery disease, hypertension, cerebrovascular disease, liver disorders, hypothyroidism, Cushing’s syndrome, nephrotic syndrome, history of smoking or alcoholism, history of taking drugs that affect lipid metabolism like lipid lowering drugs, estrogens (oral contraceptive pills), thiazides, glucocorticoids, beta blockers, etc., were excluded from the study.

**Data collection procedure:** The study conformed to the Helsinki Declaration. All the subjects were briefed about the purpose of the study and were ensured strict confidentiality. All the subjects underwent detailed history, clinical examination, and necessary investigations. Fasting and postprandial lipid profiles were obtained for all the subjects. Blood samples were collected after an overnight (12 h) fasting and 4 h postprandial.<sup>[8]</sup> The study and control groups were given a fixed diet containing approximately 750 kcal/m<sup>2</sup> and 45 g fat for lipid profile measurements. The diet consisted of four white bread slices, 50 gm Amul butter, and 250 ml full cream milk. The serum sample of each subject



**Figure 1:** Comparison of number of diabetics with hypertriglyceridemia in fasting and postprandial state

**Table 2: Fasting lipid profile of cases and controls**

Parameters	Cases (Mean±S.D)	Controls (Mean±S.D)	P
Fasting total cholesterol (mg/dl)	200.82±31.81	179.90±14.10	<0.001*
Fasting triglycerides (mg/dl)	172.59±84.33	98.03±10.67	<0.001*
Fasting HDL Cholesterol (mg/dl)	40.74±10.37	50.46±10.57	<0.001*
Fasting LDL cholesterol (mg/dl)	126.20±25.793	109.54±11.11	<0.001*
Fasting VLDL cholesterol (mg/dl)	37.63±26.75	19.60±2.13	<0.001*

**Table 3: Postprandial lipid profile of cases and controls**

Parameters	Cases (Mean±S.D)	Controls (Mean±S.D)	P
Postprandial total cholesterol (mg/dl)	223.75±29.40	185.36±15.43	<0.001*
Postprandial Triglycerides (mg/dl)	232.99±97.67	102.20±9.51	<0.001*
Postprandial HDL-cholesterol (mg/dl)	40.54±12.24	48.96±8.73	<0.001*
Postprandial LDL-cholesterol (mg/dl)	139.19±31.80	110.36±15.13	<0.001*
Postprandial VLDL-cholesterol (mg/dl)	46.52±28.07	20.24±1.90	<0.001*

**Table 4: Comparison of fasting and postprandial lipid profile in diabetics**

Serum lipid profile in cases	Fasting (Mean±S.D)	Postprandial (Mean±S.D)	P
Total Cholesterol (mg/dl)	200.82±31.81	223.75±29.40	<0.001*
Triglycerides (mg/dl)	172.59±84.33	232.99±97.67	=0.001*
HDL Cholesterol (mg/dl)	40.74±10.37	40.54±12.24	0.945
LDL Cholesterol (mg/dl)	126.20±25.73	139.19±31.80	0.087
VLDL Cholesterol (mg/dl)	37.63±26.75	46.52±28.07	0.214

**Table 5: Comparison of fasting and postprandial lipid profile in controls**

Serum lipid profile in controls	Fasting (Mean±S.D)	Postprandial (Mean±S.D)	P
Total Cholesterol (mg/dl)	179.90±14.10	185.36±15.43	0.067
Triglycerides (mg/dl)	98.03±10.67	102.20±9.51	0.091
HDL Cholesterol (mg/dl)	50.46±10.57	48.96±8.73	0.441
LDL Cholesterol (mg/dl)	109.54±11.11	110.36±15.13	0.750
VLDL Cholesterol (mg/dl)	19.60±2.13	20.24±1.90	0.116

was analyzed using a semiautomatic analyzer for the following biochemical parameters:

- Serum TC and serum TG by enzymatic methods.
- Serum HDL-C by phosphotungstate precipitation, followed by enzymatic method.
- Serum LDL-C and VLDL-C by using Friedewald's formula.<sup>[9]</sup>

Other laboratory investigations done were fasting and postprandial blood glucose, glycosylated hemoglobin (HbA1C), renal function tests, liver function tests, electrocardiogram (ECG), and urine routine examination. Dyslipidemia was defined according to National Cholesterol Education Programme - Adult treatment panel III (NCEP-ATP III) guidelines.<sup>[10]</sup>

Statistical Analysis: The collected data was entered into Microsoft Excel spread sheets and checked for possible errors. Quantitative data was presented as mean and standard deviation whereas qualitative data was represented as simple proportions. Tests

for normality were applied to check the normal distribution of interval/ratio data. *t*-test and ANOVA test were applied for comparison between 2 and more than 2 groups, respectively, for normally distributed data. Observation was considered statistically significant when *P* value was less than 0.05 ( $P < 0.05$ ).

## Results

In this study, the mean age of the enrolled cases was  $51.86 \pm 10.25$  years and of the controls was  $51.20 \pm 9.66$  years; 22 (44%) cases were males and 23 (46%) controls were males. Cases (diabetics) had higher body mass index (BMI) as compared to the controls ( $28.70 \pm 3.46$  kg/m<sup>2</sup> vs.  $23.93 \pm 1.84$  kg/m<sup>2</sup>,  $P < 0.001$ ) and higher waist to hip ratio (WHR) as compared to the controls ( $0.97 \pm 0.07$  in males,  $0.98 \pm 0.22$  in females vs.  $0.88 \pm 0.04$  in males,  $0.87 \pm 0.04$  in females,  $P < 0.001$ ) suggestive of central obesity in diabetic patients. The average duration of diabetes among the cases was  $6.06 \pm 4.50$  years and their mean HbA1C was  $8.39 \pm 1.60$  g/dl [Table 1].

The fasting lipid profiles of cases and controls are depicted in Table 2. It was observed that cases (diabetics) had significantly higher TC, TG, LDL-C, and VLDL-C in the fasting state and significantly lower HDL-C in comparison to the controls ( $P < 0.001$ ). Likewise, all the parameters of lipid profile (TC, TG, LDL-C, and VLDL-C) were also significantly higher and HDL was significantly lower in the postprandial state in cases as compared to the controls [Table 3].

The postprandial TC and TG levels in diabetics (cases) were significantly higher in comparison to their corresponding fasting values ( $P < 0.001$ ). The postprandial LDL-C and VLDL-C levels in diabetics also increased from their corresponding fasting values but the increase was not significant statistically ( $P = 0.087$ ,  $P = 0.214$ ). On the contrary, the HDL-C levels reduced in the postprandial state from their fasting values in cases, however, the fall observed was statistically insignificant ( $P = 0.945$ ) [Table 4]. In controls, there was an increase in TC, TG, LDL-C, and VLDL-C levels and a

reduction in HDL-C but these changes were not statistically significant [Table 5].

Twenty-six (52%) diabetic subjects had hypertriglyceridemia in the fasting phase while in the postprandial phase, 38 (76%) diabetic subjects had hypertriglyceridemia. Thus, there was a significant elevation in the number of cases having hypertriglyceridemia in the postprandial state [Figure 1].

## Discussion

The mean age of cases enrolled in this study was  $51.86 \pm 10.25$  years and 44% were males. 50 healthy controls were age and gender matched. Cases had higher BMI and WHR as compared to the controls. In a previous study by Puneekar *et al.*, WHR was also found to be more in cases of type 2 DM than in the controls ( $0.96 \pm 0.02$  vs.  $0.93 \pm 0.03$ ,  $P < 0.05$ ).<sup>[11]</sup> Similar results were also seen in studies by Lokhande *et al.*<sup>[2]</sup> and Puthenveedu *et al.*<sup>[12]</sup> The average duration of diabetes among the cases was  $6.06 \pm 4.50$  years and their mean HbA1C was  $8.39 \pm 1.60$  gm/dl, while mean HbA1C of controls was  $5.10 \pm 0.36$ . In a similar study, mean HbA1C of the diabetic patients was  $8.70 \pm 1.50$  compared to  $6.58 \pm 1.10$  of that of the controls.<sup>[11]</sup>

This study found that both fasting and postprandial lipid parameters (TC, TG, LDL-C, and VLDL-C) were higher in cases by statistically significant margins as compared to the controls, while HDL-C was significantly lower in both fasting and postprandial phases in the diabetic cases. Similar findings were reported by previous studies.<sup>[2,11-13]</sup>

In type 2 DM, as a consequence of insulin resistance, the free fatty acid (FFA) flux from the adipocytes is increased. This leads to an increased supply of FFA to liver, and therefore increased lipid (VLDL and TGs) synthesis within the hepatocytes. Together with defective hepatic clearance of lipoproteins, this plays a key role in the causation of dyslipidemia seen in type 2 DM (elevated TGs, low HDL-C, and increased small dense oxidized LDL particles). Diabetic dyslipidemia is an established trigger for atherogenesis and macrovascular disease.<sup>[14,15]</sup> Diabetic dyslipidemia further worsens in the postprandial state with additive adverse effect of postprandial hyperglycemia. Triglyceride-rich lipoproteins accumulate postprandially and promote the formation of small dense LDLs, which are key contributors to the development of oxidative stress, inflammation, and endothelial dysfunction. All of these lead to accelerated atherosclerosis in diabetic patients, culminating in macrovascular complications.<sup>[15,16]</sup>

In this study, the postprandial TC and TG levels in diabetics (cases) were significantly greater than their corresponding fasting values ( $P < 0.001$ ). The postprandial LDL-C and VLDL-C levels in diabetics also increased from their fasting values but the increase was not statistically significant. HDL-C level reduced in the postprandial state compared to its fasting value in cases, however, the fall was not statistically significant. On the contrary, no significant changes were observed in any of the lipid

parameters in the postprandial state among the controls. Studies by Lokhande SL,<sup>[2]</sup> Raghavendra S *et al.*,<sup>[17]</sup> and Wali VV *et al.*<sup>[18]</sup> reported that postprandial lipid parameters significantly increased from their fasting levels in type 2 diabetics, while the postprandial HDL significantly reduced in comparison to the fasting HDL level. Another study reported that serum TC and LDL-C levels decreased ( $P > 0.05$ ) in the postprandial stage while TG and VLDL levels increased ( $P < 0.001$ ). Serum HDL also increased ( $P > 0.05$ ), however, the increase was not statistically significant.<sup>[19]</sup> A recent study observed that postprandial TG ( $P = 0.003$ ) and TG/HDL-C ratio ( $P = 0.006$ ) showed a stronger correlation with HbA1c than fasting TG ( $P = 0.017$ ) and TG/HDLc ( $P = 0.292$ ), thereby suggesting the prominence of postprandial pro-atherogenic milieu in individuals with deranged glycaemic status.<sup>[13]</sup>

In addition, this study found that the number of diabetic subjects having hypertriglyceridemia increased from 26 (52%) in the fasting state to 38 (76%) in the postprandial state. Thus, there was a significant elevation in the number of diabetic cases having hypertriglyceridemia in the postprandial state.

Thus, the present study highlights an altered postprandial response of serum lipids especially triglycerides in type 2 diabetics following an oral fat meal. Exaggerated postprandial hypertriglyceridemia have also been reported in diabetics in previous studies.<sup>[16,20-23]</sup> Besides, postprandial hypertriglyceridemia in spite of normal fasting TG level has been found to be an independent risk factor for early atherosclerosis in type 2 DM.<sup>[24]</sup>

Previous studies have demonstrated that postprandial dyslipidemia plays a vital role in the development of atherosclerotic plaques, leading to increased risk of cardiovascular events.<sup>[25]</sup> The postprandial dysmetabolism including postprandial dyslipidemia, with associated endothelial dysfunction and oxidative stress may link the insulin resistance in type 2 DM patients to a higher incidence of cardiovascular diseases in these patients.<sup>[26,27]</sup> Previous literature has also reported that the duration and magnitude of postprandial lipidaemia is directly related to pathogenesis and progression of cardiovascular diseases.<sup>[28]</sup>

Besides, the fasting prerequisite for lipid profile assessment possibly makes blood sampling unnecessarily difficult for numerous patients worldwide, especially for diabetics. The above observations highlight that lipid estimation in postprandial phase is as important as fasting lipid profile in type 2 DM. In fact, it has been proposed that postprandial lipid profile may be a better indicator of deranged lipid metabolism and therefore of cardiovascular disease in type 2 DM.

## Strength of the study

Few studies have focused on postprandial dysmetabolism associated with type 2 DM, particularly postprandial dyslipidemia. Postprandial hyperlipidemia especially postprandial hypertriglyceridemia is a public health problem as it predisposes to accelerated atherosclerosis in diabetics. Its common yet



significant occurrence in the diabetics is highlighted by this study and calls for incorporation of estimating postprandial lipid profile routinely, in addition to the fasting lipid profile in the clinical assessment of type 2 diabetic patients.

### Limitations of the study

Limited sample size, single-center study.

### Conclusions

Type 2 DM patients show significant postprandial metabolic abnormalities especially postprandial dyslipidemia. Atherosclerosis is predominantly a postprandial phenomenon with additive effects of hyperlipidemia and hyperglycemia in diabetics, as the patients tend to be in the postprandial state most of the time than in the fasting state. The duration and magnitude of postprandial lipidemia is directly related to pathogenesis and progression of cardiovascular diseases. The present study suggests that postprandial lipid profile estimation is as important as estimating fasting lipid profile in the cardiovascular risk stratification of type 2 diabetics. Early recognition and appropriate treatment of significant postprandial dyslipidemia is of paramount importance in diabetics so as to reduce impending complications of atherosclerotic vascular disease.

### Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient (s) has/have given his/her/their consent for his/her/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.

### Key Message

Estimating lipids in the postprandial phase is as important as the estimation of lipids in the fasting phase in type 2 diabetics. Atherosclerosis is believed to be a postprandial phenomenon with additive effects of hyperlipidemia and hyperglycemia in type 2 DM. Early recognition and appropriate treatment of significant postprandial dyslipidemia is of paramount importance in diabetics so as to reduce cardiovascular morbidity and mortality.

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Nil.

### Conflicts of interest

There are no conflicts of interest.

### References

1. Tushuizen M, Diamant M, Heine R. Postprandial dysmetabolism and cardiovascular disease in type 2 diabetes. *Postgrad Med J* 2005;81:1-6.
2. Lokhande SL, Iyer CM, Nandedkar PD. A comparative study on the fasting and the postprandial dyslipidaemia in type 2 diabetes mellitus. *J Clin Diagn Res* 2013;7:627-30.
3. Shinichi T, Yuko U, Hirotaka N, Yasuhiro E, Yasuhiko I. Association of postprandial hypertriglyceridemia and carotid intima-media thickness in patients with Type 2 Diabetes. *Diabetes Care* 2000;23:1401-6.
4. Verges B. Pathophysiology of diabetic dyslipidaemia: Where are we? *Diabetologia* 2015;58:886-99.
5. Dubios C, Armand M, Azais-Braesco V, Portugal H, Pauli AM, Bernad PM, *et al.* Effects of moderate amounts of emulsified dietary fat on postprandial lipemia and lipoproteins in normolipidemic adults. *Am J Clin Nutr* 1994;60:374-82.
6. Lewis GF, O' Meara NM, Soltys PA, Blackman JD, Iverius PH, Pugh WL, *et al.* Fasting hypertriglyceridemia in non insulin dependent diabetes mellitus is an important predictor of postprandial lipid and lipoprotein abnormalities. *J Clin Endocrinol Metab* 1991;72:934-44.
7. Madhu SV, Mittal V, Krishnam B, Srivastava DK, Kaur J, Sethi K. Postprandial triglyceride metabolism in obese and non-obese patients with non-insulin dependent diabetes mellitus. *Diabetes Res Clin Pract* 2000;50:317.
8. Mihas C, Kolovou GD, Mikhailidis DP, Kovar J, Lairon D, Nordestgaard BG, *et al.* Diagnostic value of postprandial triglyceride testing in healthy subjects: A meta-analysis. *Curr Vasc Pharmacol* 2011;9:271-80.
9. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin Chem* 1972;18:499-502.
10. Third Report of the National Cholesterol Education Programme (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult treatment Panel III) final report. *Circulation* 2002;106:3143-421.
11. Puneekar P, Jatav JK, Bantewad DS. Study of fasting and postprandial lipid abnormality in type 2 diabetes mellitus and its correlation with vascular complications of diabetes. *Int J Sci Stud* 2018;5:128-33.
12. Puthenveedu DK, Ravindran S. A comparative study on the fasting and post prandial lipid levels as a cardiovascular risk factor in patients with type 2 diabetes mellitus. *IAIM* 2017;4:72-6.
13. Chakraborty M, Singh P, Dsouza JM, Pethusamy K, Thatkar PV. Fasting and postprandial lipid parameters: A comparative evaluation of cardiovascular risk assessment in prediabetes and diabetes. *J Family Med Prim Care* 2020;9:287-92.
14. Blann AD, McCollum CN. Circulating endothelial cell/leukocyte adhesion molecules in atherosclerosis. *Thromb Haemost* 1994;72:151-4.
15. Taskinen MR. Diabetic dyslipidemia: From basic research to clinical practice. *Diabetologia* 2003;46:733-49.
16. Kumar V, Madhu SV, Singh G, Gambhir JK. Postprandial hypertriglyceridemia in patients with type 2 diabetes mellitus with and without macrovascular Disease. *J Assoc Physicians India* 2010;58:603-7.
17. Raghavendra S, Dutta TK, Tumbanatham A, Sethuraman KR, Jayasingh K, Pyadala N. Fasting and postprandial lipid profile in type 2 diabetes mellitus: A comparative study. *International Journal of Contemporary Medicine Surgery and Radiology* 2018;3:161-5.
18. Wali VV, Patil SS. A Comparative study on the fasting and

- postprandial dyslipidaemia in type 2 diabetes mellitus. *Int J Clin Biochem Res* 2016;3:177-80.
19. Hawa VP, Garg S, Maheshwar, Singh SS. Studying the effect of fed status on serum lipid profile values in type 2 diabetes mellitus. *JACM* 2019;20:166-9.
  20. Fontbonne A. Relationship between diabetic dyslipoproteinemia and coronary heart disease risk in non- insulin dependent diabetes. *Diabetes Metab Rev* 1991;7:179-89.
  21. Raj S, Rajsekheran C, Jaykumar B. Postprandial hypertriglyceridemia in type 2 diabetic subjects. *Int J Diab Dev Ctries* 2006;26:160-2.
  22. Cooper MB, Tan KC, Hales CN, Betteridge DJ. Postprandial lipid metabolism and beta-cell function in non-insulin-dependent (type 2) diabetes mellitus after a mixed meal with a high fat content. *Diabet Med* 1996;13:816-27.
  23. Cavallero E, Dachet C, Neufcour D, Wirquin E, Mathe D, Jacotot B. Postprandial amplification of lipoprotein abnormalities in controlled type II subjects: Relationship to postprandial lipemia and C-peptide/glucagon levels. *Metabolism* 1994;43:270-8.
  24. Teno S, Uto Y, Nagashima H, Endoh Y, Iwamoto Y, Omori Y, *et al.* Association of postprandial hypertriglyceridemia and carotid intima media thickness in patients with type 2 diabetes. *Diabetes Care* 2000;23:1401-6.
  25. Madhu SV, Mittal V, Ram BK, Srivastava DK. Postprandial lipid abnormalities in Type 2 diabetes Mellitus. *J Assoc Physicians India* 2005;53:1043-6.
  26. Athyros VG, Tziomalos K, Karagiannis A, Mikhailidis DP. Dyslipidaemia of obesity, metabolic syndrome and type 2 diabetes mellitus: The case for residual risk reduction after statin treatment. *Open Cardiovasc Med J* 2011;5:24-34.
  27. Madhu SV, Kant S, Srivastava S, Kant R, Sharma SB, Bhadoria DP. Postprandial lipaemia in patients with impaired fasting glucose, impaired glucose tolerance and diabetes mellitus. *Diabetes Res Clin Pract* 2008;80:380-5.
  28. Chapman MJ, Ginsberg HN, Amarenco P, Andreotti F, Boren J, Catapano AL, *et al.* Triglyceride-rich lipoproteins and high-density lipoprotein cholesterol in patients at high risk of cardiovascular disease: Evidence and guidance for management. *Eur Heart J* 2011;32:1345-61.