ORIGINAL ARTICLE ASIAN JOURNAL OF MEDICAL SCIENCES

Comparative analysis between fasting and postprandial apolipoprotein B and total cholesterol/high-density lipoprotein-cholesterol ratio for atherosclerosis risk prediction in type 2 diabetes mellitus subjects of central India

Rajesh K Jambhulkar[1], Suryabhan L Lokhande[2], Praveen K Salame[3], Ashok K Ahirwar[4]

[1]Associate Professor, Department of Biochemistry, Government Medical College, Jalgaon, Maharashtra, [2]Assistant Professor, Department of Biochemistry, All India Institute of Medical Sciences, Bhopal, Madhya Pradesh, [3]Asssitant Professor, Department of General Medicine, IGMC, Nagpur, Maharashtra, [4]Assistant Professor, Department of Biochemistry, All India Institute of Medical Sciences, New Delhi, India

Submission: 14-12-2022 Revision: 03-02-2023 Publication: 01-03-2023

ABSTRACT

Background: Type 2 diabetes mellitus (Type 2 DM), characterized by a relative insulin deficiency or insulin resistance, is associated with a cluster of metabolic abnormalities, which includes glucose intolerance, hypertension, a unique dyslipidemia, a procoagulant state, and an increase in macrovascular diseases. **Aims and Objectives:** The present study was conducted to correlate between fasting and postprandial apolipoprotein B and total cholesterol/high-density lipoprotein-cholesterol (TC/HDL-C) ratio for predicting the risk of atherosclerosis in T2DM subjects. **Materials and Methods:** Sixty clinically diagnosed cases of T2DM (age group of 35–65 years and duration of diabetes of more than 5 years) were included in the study and sixty age- and sex-matched healthy subjects were taken as the controls. In both the study groups, we measured postprandial as well as fasting lipid profile, which comprised serum TC, triglycerides (TGs), HDL-C, low-density lipoprotein cholesterol (LDL-C), and apolipoprotein B. Ratio is calculated as TC/HDL-C in both fasting and postprandial state. The statistical analysis was done using the students unpaired'-test. **Results:** The results of the present study showed significantly increased levels of postprandial serum TC, TGs, LDL-C, and apo B as compared to those in the fasting state ($P<0.05$). The serum HDL-C level was significantly lower in the postprandial state as compared to that in the fasting state ($P<0.05$). Postprandial TC/HDL-C and apo B are increased significantly as compared to fasting condition in type 2 diabetes subjects. **Conclusion:** The findings suggest that postprandial lipid profile, apolipoprotein B, and TC/HDL-C ratio as a cardiovascular risk factor can be utilized as simple marker utilizing simple parameters in lipid profile that significantly increased in the postprandial state.Thus, Apolipoprotein B, the marker of the number of LDL particles (for atherosclerosis) in the circulation and TC/HDL-C ratio can be routinely estimated for the atherosclerosis risk assessment in type 2 diabetes subjects in the postprandial state.

Key words: Type 2 diabetes mellitus; Postprandial; Total cholesterol; High-density lipoprotein ratio; Apolipoprotein B; Cardiovascular diseases

Access this article online

Website: http://nepjol.info/index.php/AJMS

DOI: 10.3126/ajms.v14i3.50196

E-ISSN: 2091-0576

P-ISSN: 2467-9100

INTRODUCTION

Diabetes mellitus (DM) is a group of metabolic diseases characterized by chronic hyperglycemia resulting from defects in insulin secretion, insulin action, or both. The basis of the abnormalities in carbohydrate, fat, and protein metabolism in diabetes is incomplete action of insulin on target tissues. Type 2 DM, the most prevalent form of

Address for Correspondence:
Suryabhan L Lokhande, Assistant Professor, Department of Biochemistry, All India Institute of Medical Sciences, Bhopal - 462 020, Madhya Pradesh, India. **Mobile:** +91-8888865479. **E-mail:** suryabhanbiochem@gmail.com

the disease, is often asymptomatic in its early stages and can remain undiagnosed for many years.[1] Diabetes is a syndrome of multiple etiologies characterized by chronic hyperglycemia with disturbance of carbohydrate, protein, fat metabolism resulting from defects in insulin secretion, action, or both. This disorder is often associated with long term complications involving organs such as eyes, kidney, nerve, heart, and blood vessels.[2]

According to the data published by international diabetes federation diabetes Atlas 2021, an estimated 537 million adults aged 20–79 years worldwide (10.5% of all adults in this age group) have diabetes. By 2030, 643 million, and by 2045, 783 million adults aged 20–79 years are projected to be living with diabetes. Thus, while the world's population is estimated to grow 20% over this period, the number with diabetes is estimated to increase by 46% with increasing prevalence of diabetes by age.[3]

The most common etiological factor of insulin resistance in T2DM is central abdominal obesity. In T2DM, insulin resistance in the liver indicates the failure of hyperinsulinemia to suppress gluconeogenesis, which results in fasting hyperglycemia and decreased glycogen storage by the liver in the postprandial state. Increased hepatic glucose production occurs early during diabetes, though likely after the onset of insulin secretory abnormalities and insulin resistance in skeletal muscle. This is responsible for the dyslipidemia found in T2DM.[4]

Diabetics are frequently hyperlipidemia and they are at a high risk for coronary heart disease.[5] The high cardiovascular disease (CVD) morbidity and mortality associated with type 2 diabetes is at least partly due to a prolonged and exaggerated postprandial state in type 2 diabetes patients. However, controlled randomized intervention studies showing that postprandial both glucose and triglyceride (TG) lowering results in amelioration of clinically relevant endpoints are lacking.[6,7] The abnormal lipid profile in the postprandial state is more significant than the abnormal lipid profile in the fasting state in causing atherosclerotic complications in type 2 diabetics.[8-11] Some studies have been conducted so far regarding analysis between fasting and postprandial total cholesterol/high-density lipoprotein-cholesterol (TC/HDL-C) ratio and their association with apo B in the subjects of T2DM in some parts of India. However, very few such studies have been conducted so far in the central Indian population. Hence, this study was proposed to analysis fasting and postprandial (TC/HDL-C) ratio and apo B for predicting the risk of atherosclerosis in type 2 diabetes subjects of Central Indian population.

Aims and objectives

The aims of this study were as follows:

1. To estimate the fasting and postprandial lipid profile and apolipoprotein B in type 2 diabetes cases as well as controls
2. To compare the fasting and postprandial apo B and TC/HDL-ratio in T2DM subjects and its association with the atherosclerosis process.

MATERIALS AND METHODS

Study design

The present case–control study was conducted in the department of biochemistry in collaboration with Department of Medicine, Government Medical College, Nagpur. The study was pre-approved by the Institutional Ethics Committee for the final permission. Informed consent was taken while enrolling the participants and a patient information sheet was given to the individuals participants.

Sample size

The present case–control study was commenced by enrolling 120 participants which included 60 cases (age group of 35–65 years and duration of diabetes of more than 5 years) and 60 age and gender matched controls recruited from wards and outpatient department (OPD) of the Department of Medicine, Government Medical College, Nagpur. The participants who were enrolled in the study were diagnosed according to Classification and Diagnosis of Diabetes: Standards of Medical Care in Diabetes (2021)/WHO criteria (FPG ≥126 mg/dl (7.0 mmol/L). Fasting was defined as no caloric intake at least for 8 h. In a patient with classic symptoms of hyperglycemia or hyperglycemic crisis, a random plasma glucose ≥200 mg/dl (11.1 mmol/L) and 2-h plasma glucose ≥200 mg/dl (11.1 mmol/L) during an OGTT is considered as the diagnostics of Diabetes Mellitus. The test should be performed as described by the World Health Organization using a (glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water).[12]

From the medicine OPD, after the diagnosis is made, participants were taken to the blood collection area of clinical biochemistry laboratory, GMC Nagpur. Venous samples were withdrawn following all the aseptic procedures and conditions, while the samples were withdrawn. Tourniquet was applied properly, and the sample was withdrawn by syringe. Participants fasted overnight before the collection of blood in case of fasting blood and postprandial blood sample was collected in the fluoride containing gray vacutainers for glucose estimation, kidney function test, and liver function tests and for lipid profile low-density lipoprotein (TC, TG, low-density lipoprotein

[LDL], very LDL [VLDL], and HDL) and apo B, blood samples were collected in yellow plain vacutainers. Samples were processed in the clinical biochemistry laboratory, Department of Biochemistry, GMC Nagpur, following standard operating procedure using a commercial kit with standard enzymatic method in fully automated analyzer. Serum was separated from cells with centrifugation after 30 min of sample collection and was processed immediately for estimation of fasting blood sugar, postprandial sugar, lipid profile, urea, and creatinine. Proper calibration is done for the individual test that is to estimate and quality control is performed every day under 2 SD which was analyzed using Levy Jennings chart. Participants were incorporated in this study after considering following.

Inclusion criteria

The following criteria were included in the study:

1. A clinical diagnosis of T2DM
2. T2DM presenting with symptoms of weight loss, polyuria, and polydipsia
3. Use of hypoglycemic drugs/insulin in a patient with age more than 40 years
4. Age 35–65 years (both cases as well as age- and sex-matched controls).

Exclusion criteria

The following criteria were excluded from the study:

1. Type 1 DM
2. DM with history of cardiovascular episode
3. T2DM below the age of 35 years and above the age of 65 years
4. Liver diseases
5. Kidney disorders.

Laboratory assays

Under aseptic conditions, blood samples were drawn in the morning after an overnight (i.e., after 12 h) fast for fasting lipid profile and 6 h after meals for postprandial lipid profile. The serum was separated from the blood cells by centrifugation within 30 min of the collection of the blood. The separated serum was analyzed for the following biochemical parameters:

1. Serum TC by an enzymatic method
2. Serum TGs by an enzymatic method
3. Serum HDL-C by phosphotungstate precipitation and enzymatic method
4. Serum LDL cholesterol (LDL-C) and VLDL-cholesterol using Friedewald's formula[13]
5. Apolipoprotein B measured by rate nephelometry method.

Statistical analysis

In this case–control study, all the statistical analyses were performed using the GraphPad Software. The data were expressed as Mean±SD. Using the students unpaired "t"-test, the statistical analysis was carried out to find whether the differences between the T2DM patients and the controls were significant and $P<0.05$ was considered as statistically significant. Microsoft Excel 2019 and Statistical Package for the Social Sciences Version 22.0 software was used for data entry and analysis.

RESULTS

We observed that body mass index was found to be statistically non-significant in diabetic subjects as compared to controls (Table 1).

Fasting lipid parameters, that is, serum TC, TGs, and LDL-C were found to be significantly increased in the Type 2 diabetic subjects as compared to healthy controls ($P<0.05$), while HDL-C (which was found to be significantly decreased in type 2 diabetic subjects as compared to controls $P<0.05$) (Table 2 and Figure 1).

The mean values of postprandial lipid parameters, that is, serum TC, TGs, LDL-C, and apo B were found to be significantly increased in the type 2 diabetic subjects as compared to healthy controls ($P<0.05$), while postprandial HDL-C (which was found to be significantly decreased in type 2 diabetic subjects as compared to controls $P<0.05$) (Table 3 and Figure 2).

There is significant increase in the TC/HDL-C in the postprandial state when compared with the fasting state, as shown in Table 4, which is in accordance with the some of the studies conducted so far.[14-16]

DISCUSSION

The present study is conducted in the Department of Biochemistry, Government Medical College, Nagpur. In the present study, the postprandial lipid parameters low-density lipoprotein cholesterol (i.e., TC, TGs, LDL-C, and apo B) were significantly increased in the T2DM subjects as compared to the fasting lipid parameters and the postprandial HDL-C level was significantly decreased as compared to the fasting HDL-C level ($P<0.05$).

Table 1: Body mass index (BMI) in type 2 diabetic subjects and controls

Subjects	Body mass index (kg/m²) (mean±SD)		P-value
	Diabetics (n=60)	Controls (n=60)	
Males	23.80±2.94	24.60±1.99	0.240
Females	24.16±3.16	23.10±2.33	0.210

Table 2: Fasting lipid profile and apo B in type 2 diabetic subjects and controls

Fasting lipid profile (mg/dl)	Diabetes (n=60) (Mean±SD)	Controls (n=60) (Mean±SD)	P-value
Total cholesterol	208.10±53.18	156.50±32.92	0.05
Triglycerides	171.70±71.71	114.70±34.17	0.05
HDL cholesterol	45.72±8.82	50.82±6.05	0.05
LDL cholesterol	128.80±51.02	83.06±33.58	0.05
Apo B (65–105 mg/dl)	140.30±39.16	82.12±12.16	0.05

Table 3: Postprandial lipid profile and apo B in type 2 diabetic subjects and controls

Postprandial serum lipids (mg/dl)	Diabetics (n=60) (Mean±SD)	Controls (n=60) (Mean±SD)	P-value
Total cholesterol	238.90±56.77	191.50±36.38	<0.05
Triglycerides	209.50±74.48	139.50±34.32	<0.05
HDL cholesterol	35.30±7.25	41.88±4.62	<0.05
LDL cholesterol	162.10±53.26	121.50±36.71	<0.05
Apo B (65–105 mg/dl)	182.60±52.09	101.00±11.58	<0.05

Table 4: Comparison between TC/HDL-C ratio between fasting and postprandial state in type 2 diabetes mellitus

TC/HDL ratio	Diabetes	Controls
Fasting	4.55	3.07
PP	6.76	4.57
Type 2 DM	PP	Fasting
TC/HDL ratio	6.76	4.55

Diabetes is a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both. The chronic hyperglycemia of diabetes is associated with long-term damage, dysfunction, and failure of different organs, especially the eyes, kidneys, nerves, heart, and blood vessels.[17] T2DM reduces life expectancy by as much as 10 years, and the main cause of death for patients with T2DM is CVD and people with T2DM are disproportionately affected by CVD compared with non-diabetic subjects.[18] In T2DM, the insulin resistance in the liver reflects the failure of the hyperinsulinemia to suppress the gluconeogenesis, which results in fasting hyperglycemia and a decreased glycogen storage by the liver in the postprandial state.[19]

This chronic hyperglycemia of diabetes is associated with a long-term damage, dysfunction, and failure of various organs, especially the eyes, kidneys, nerves, heart, and the blood vessels. The risk of the chronic complications increases as a function of the duration of the hyperglycemia. Since T2DM, often, has a long asymptomatic period of hyperglycemia, many individuals with T2DM have complications at the time of their diagnosis.[20] The macrovascular complications such as coronary heart disease and cerebrovascular disease are two to 4 times greater in the patients with T2DM. Other factors (dyslipidemia and hypertension) also play important roles in the macrovascular complications.[21]

Postprandial hypertriglyceridemia has been linked to asymptomatic and symptomatic macrovascular diseases in both normo- and hypertriglyceridemic subjects and such abnormalities have been reported in the type 2 diabetics. The increased risk of atherosclerosis among them, therefore, may be related to the higher postprandial lipaemia in them. The earlier studies clearly demonstrate the presence of postprandial hypertriglyceridemia among the diabetic subjects, irrespective of the fasting TG levels.[22]

Various studies have shown that postprandial dyslipidemia is more important in the pathogenesis of the vascular changes and atherosclerosis and that it increases the risk of the cardiovascular events.[23] Although the importance of LDL-C in the development of atherosclerosis has long been recognized, the increasing research attention over the past decades has been devoted to the heterogeneity of the LDL particles and the atherogenicity of the lipids and the lipoproteins which are other than LDL.[6] The postprandial dysmetabolism and the associated oxidative stress may link the insulin resistance and the T2DM to the disproportional incidence of CVD.[24] The high CVD morbidity and the mortality in T2DM is associated at least partly caused by a prolonged and an exaggerated postprandial state in these patients.[25] Persistent postprandial hypertriglyceridemia may result in a pro-atherogenic environment, leading to atherosclerosis and macrovascular disease in type 2 diabetes subjects.[19] LDL oxidation in the postprandial state seems to be affected by an acute increase in glycemia. Thus, oxidative modification of LDL may contribute to higher CVD risk among diabetic patients, and elevated levels of TG may contribute to the rapid LDL oxidation seen in T2DM.[26]

There are significant correlations that were found between fasting blood glucose (FBG) with TC, TG, LDL-C, non-HDL-C, LDL/HDL ratio, and TC/HDL ratio; TC/HDL

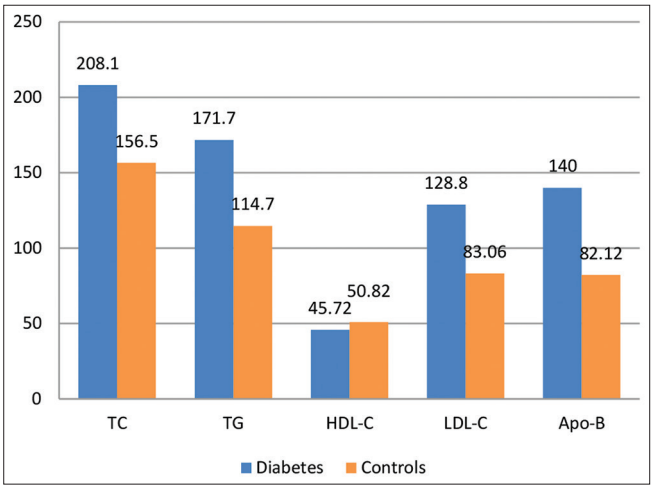

Figure 1: Fasting lipid profile and apo B (mg/dl) in type 2 diabetic subjects and controls

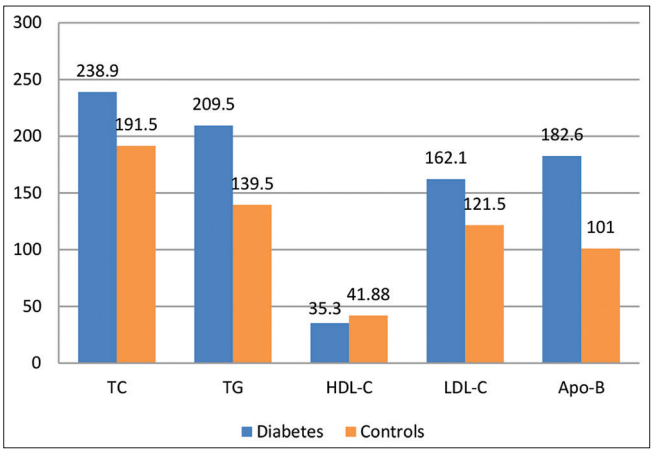

Figure 2: Postprandial lipid profile and apo B in type 2 diabetic subjects and controls

ratio and HbA1c were significantly lower in patients on statin treatment than non-treated patients ($P<0.05$).[27] FBG and TC: HDL ratios were increased in the patient group ($P\leq0.001$). Comparison of FBG and TC: HDL ratios revealed a highly significant rise ($P\leq0.001$) in the patient group. FBG with TC: HDL ratios between both groups showed a positive correlation ($r=0.554$).[28] The results of the present study suggest an association between TC: HDL ratio and apo B, which may be a contributory factor to the increased prevalence of coronary artery disease in patients with T2DM.

Because LDL particles differ substantially from one another in the amount of cholesterol that they contain, TC and LDL-C are imprecise measures of the number of apo B particles. Measuring apo B, therefore, provides a direct estimate of the total number of atherogenic particles. The measurement of plasma apo B is standardized, automated, and inexpensive, it can be performed on non-fasting samples, and population reference values are now available.[29] Serum apo B is a good predictor of CVD, a more reliable one than LDL or non-HDL-C. Serum apo B (as a marker of small and dense LDL particles) strongly determines the development of atherogenic dyslipidemia in both sexes among Turks.[30,31]

Lipid profiles LDL-C and lipid ratios (LDL-C/HDL-C and TC/HDL-C ratio) show potential markers that can be used in predicting glycemic control in patients with T2DM.[32] Thus, the present case control study shows that TC/HDL-C ratio and apo B can be utilized as a simple, non-invasive, and readily available parameter to predict the future risk of diabetes related complications associated with prolonged T2DM.

Limitations of the study

1. We could not estimate Apo A, which is the important parameter for the calculation of apo-A/apo B ratio
2. We could not analysis HbA1C for assessment of the long-term glycemic status in the subjects of T2DM subjects.

CONCLUSION

Atherosclerosis is a postprandial phenomenon with respect to lipids, as we are in the postprandial phase for most of the day, with an additional adverse effect of the meal induced hyperglycemia. The present study suggests that it is important and beneficial to routinely estimate the postprandial lipid profile and apo B, in addition to the fasting lipid parameters, in the cardiovascular risk assessment in T2DM. Thus, by rectifying the abnormal postprandial lipid parameters early during diabetes, we can prevent the hazardous complication associated with T2DM, the most common one being atherosclerotic coronary artery disease.

Thus, for future complication risk prediction, apo B and TC/HDL-C ratio can be utilized as simple marker utilizing simple parameters in lipid profile that significantly increased in the postprandial state in T2DM.

ACKNOWLEDGMENT

Authors are thankful to the Heads of the department of Biochemistry and General Medicine for the support to carry out this research work.

REFERENCES

1. American Diabetes Association. Screening for Type 2 diabetes. Diabetes Care. 2004;27 Suppl 1:S11-S14. https://doi.org/10.2337/diacare.27.2007.S11

2. Lebovitz HE. Type 2 diabetes mellitus: An overview. Clin Chem. 1999;45(8 Pt 2):1339-1345.
   https://doi.org/10.1093/clinchem/45.8.1339
3. International Diabetes Federation. Diabetes Atlas. 10th ed., Ch. 3. Belgium: International Diabetes Federation; 2021. p. 32-35.
4. Unnikrishnan R, Anjana RM and Mohan V. Diabetes mellitus and its complications in India. Nat Rev Endocrinol. 2016;12(6):357-370.
   https://doi.org/10.1038/nrendo.2016.53
5. Maeda E, Yoshino G and Kasuga M. Diabetes mellitus as a risk factor for arteriosclerosis. Nippon Rinsho. 1993;51(8):2170-2176.
6. Tushuizen ME, Diamant M and Heine RJ. Postprandial dysmetabolism and cardiovascular disease in Type 2 diabetes. Postgrad Med J. 2005;81(951):1-6.
   https://doi.org/10.1136/pgmj.2004.020511
7. Tentolouris N, Stylianou A, Lourida E, Perrea D, Kyriaki D, Papavasiliou EC, et al. High postprandial triglyceridemia in patients with Type 2 diabetes and microalbu-minuria. J Lipid Res. 2007;48(1):218-225.
   https://doi.org/10.1194/jlr.M600367-JLR200
8. Axelsen M, Smith U, Eriksson JW, Jansson PA and Taskinen MR. Postprandial hypertriglyceridemia and insulin resistance in normoglycemic first-degree relatives of patients with Type 2 diabetes. Ann Intern Med. 1999;131(1):27-31.
   https://doi.org/10.7326/0003-4819-131-1-199907060-00006
9. Evans M, Anderson RA, Graham J, Ellis GR, Morris K, Davies S, et al. Ciprofibrate therapy improves endothelial function and reduces postprandial lipaemia and oxidative stress in Type 2 diabetes mellitus. Circulation. 2000;101(15):1773-1779.
   https://doi.org/10.1161/01.cir.101.15.1773
10. Annuzzi G, De Natale C, Iovine C, Patti L, Rivellese AA, Di Marino L, et al. Insulin resistance is independently associated with postprandial alterations of triglyceride-rich lipoproteins in Type 2 diabetes mellitus. Arterioscler Thromb Vasc Biol. 2004;24(12):2397-2402.
    https://doi.org/10.1161/01.atv.0000146267.71816.30
11. Ferreira AC, Peter AA, Mendez AJ, Jimenez JJ, Mauro LM, Chirinos JA, et al. Postprandial hypertriglyceridemia increases circulating levels of endothelial cell micro-particles. Circulation. 2004;110(23):3599-3603.
    https://doi.org/10.1161/01.cir.0000148820.55611.6B
12. American Diabetes Association. Classification and diagnosis of diabetes: Standards of medical care in diabetes-2021. Diabetes Care. 2021;44(Suppl 1):S15-S33.
    https://doi.org/10.2337/dc21-S002
13. Friedwald WT, Levy RL and Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma without use of the preparative ultracentrifuge. Clin Chem. 1972;18(6):499-502.
    https://doi.org/10.1093/clinchem/18.6.499
14. Yan Z, Liu Y and Huang H. Association of glycosylated hemoglobin level with lipid ratio and individual lipids in Type 2 diabetic patients. Asian Pac J Trop Med. 2012;5(6):469-471.
    https://doi.org/10.1016/S1995-7645(12)60080-7
15. Aryal M, Poudel A, Satyal B, Gyawali P, Pokharel BR, Raut BK, et al. Evaluation of non-HDL-c and total cholesterol: HDL-c ratio as cumulative marker of cardiovascular risk in diabetes mellitus. Kathmandu Univ Med J (KUMJ). 2010;8(32):398-404.
    https://doi.org/10.3126/kumj.v8i4.6239
16. Ali F, Jamil H, Anwar SS and Wajid N. Characterization of lipid parameters in diabetic and non-diabetic atherosclerotic patients. J Geriatr Cardiol. 2015;12(1):37-43.
    https://doi.org/10.11909/j.issn.1671-5411.2015.01.005
17. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care. 2013;36(Suppl 1):S67-S74.
    https://doi.org/10.2337/dc13-S067
18. Einarson TR, Acs A, Ludwig C and Panton UH. Prevalence of cardiovascular disease in Type 2 diabetes: A systematic literature review of scientific evidence from across the world in 2007-2017. Cardiovasc Diabetol. 2018;17(1):83.
    https://doi.org/10.1186/s12933-018-0728-6
19. Kumar V, Madhu SV, Singh G and Gambhir JK. Post-prandial hypertriglyceridemia in patients with Type 2 diabetes mellitus with and without macrovascular disease. J Assoc Physicians India. 2010;58:603-607.
20. Kolovou GD, Anagnostopoulou KK, Pilatis N, Kafaltis N, Sorodila K, Psarros E, et al. Low fasting low high-density lipoprotein and postprandial lipaemia. Lipids Health Dis. 2004;3:18.
    https://doi.org/10.1186/1476-511X-3-18
21. Viigimaa M, Sachinidis A, Toumpourleka M, Koutsampasopoulos K, Alliksoo S and Titma T. Macrovascular complications of Type 2 diabetes mellitus. Curr Vasc Pharmacol. 2020;18(2):110-116.
    https://doi.org/10.2174/1570161117666190405165151
22. Rivellese AA, De Natale C, Di Marino L, Patti L, Lovine C, Coppola S, et al. Exogenous and endogenous postprandial lipid abnormalities in Type 2 diabetic patients with optimal blood glucose control and optimal fasting triglyceride level. J Clin Endocrinol Metab. 2004;89(5):2153-2159.
    https://doi.org/10.1210/jc.2003-031764
23. Madhu SV, Mittal V, Ram BK and Srivastava DK. Postprandial lipid abnormalities in Type 2 diabetes mellitus. J Assoc Physicians India. 2005;53:1043-1046.
24. Athyros VG, Tziomalos K, Karagiannis A and Mikhailidis DP. Dyslipidaemia of obesity, metabolic syndrome and Type 2 diabetes mellitus: A case for residual risk reduction after statin treatment. Open Cardiovasc Med J. 2011;5:24-34.
    https://doi.org/10.2174/1874192401105010024
25. Nadia A and Boumediene KM. Effect of overweight and obesity on postprandial lipaemia among the subjects with Type 2 diabetes. J Diabetes Metab. 2012;3(2):1-5.
    https://doi.org/10.4172/2155-6156.1000178
26. Enkhmaa B, Ozturk Z, Anuurad E and Berglund L. Postprandial lipoproteins and cardiovascular disease risk in diabetes mellitus. Curr Diab Rep. 2010;10(1):61-69.
    https://doi.org/10.1007/s11892-009-0088-4
27. Taskinen MR, Barter PJ, Ehnholm C, Sullivan DR, Mann K, Simes J, et al. Ability of traditional lipid ratios and apolipoprotein ratios to predict cardiovascular risk in people with Type 2 diabetes. Diabetologia. 2010;53(9):1846-1855.
    https://doi.org/10.1007/s00125-010-1806-9
28. Hanif R, Sattar A and Qayum I. FBG and TC/HDL ratios in Type 2 diabetes mellitus. J Ayub Med Coll Abbottabad. 2001;13(2):42-44.
29. Sniderman AD, Bergeron J and Frohlich J. Apolipoprotein B versus lipoprotein lipids: Vital lessons from the AFCAPS/TexCAPS trial. CMAJ. 2001;164(1):44-47.
30. Walldius G, Jungner I, Holme I, Aastveit AH, Kolar W and Steiner E. High apolipoprotein B, low apolipoprotein A-I, and improvement in the prediction of fatal myocardial

infarction (AMORIS study): A prospective study. Lancet. 2001;358(9298):2026-2033.
https://doi.org/10.1016/S0140-6736(01)07098-2

31. Kathiresan S, Otvos JD, Sullivan LM, Keyes MJ, Schaefer EJ, Wilson PW, et al. Increased small low-density lipoprotein particle number: A prominent feature of the metabolic syndrome in the Framingham heart study. Circulation. 2006;113(1):20-29.
https://doi.org/10.1161/circulationaha.105.567107
32. Artha IM, Bhargah A, Dharmawan NK, Pande UW, Triyana KA, Mahariski PA, et al. High level of individual lipid profile and lipid ratio as a predictive marker of poor glycemic control in Type-2 diabetes mellitus. Vasc Health Risk Manag. 2019;15:149-157.
https://doi.org/10.2147/vhrm.S209830

Authors' Contributions:
RKJ- Design of the study and clinical biochemistry laboratory work, contribution in the preparation of final draft; **SLL-** Concept and design of the study, prepared first draft of the manuscript, reviewed the literature and manuscript preparation, preparation of the final draft; **PKS-** Coordination, revision of manuscript, provision of the subjects from clinics for samples; **AKA-** Concept, coordination, statistical analysis and interpretation, revision of the manuscript.

Work attributed to:
Government Medical College and Hospital, Nagpur - 440 003, Maharashtra, India.

Orcid ID:
Dr. Rajesh K Jambhulkar - https://orcid.org/0000-0001-9170-9434
Dr. Suryabhan L Lokhande - https://orcid.org/0000-0002-2571-8735
Dr. Praveen K Salame - https://orcid.org/0000-0003-1198-8870
Dr. Ashok K Ahirwar - https://orcid.org/0000-0003-3371-378X

Source of Support: Nil, **Conflicts of Interest:** None declared.