ORIGINAL ARTICLE

# Non-HDL cholesterol: A valid surrogate marker to apolipoprotein b100 in type 2 diabetes mellitus patients in assessment of risk of cardiovascular diseases

*Vidyashree[1], Lakshmi D[2], Prashanth D[3], Indushree T[3*], Hamsaveena[2]*

*[1]Department of General Medicine, Sapthagiri Institute of Medical Sciences & Research Centre, Bangalore-560090 (Karnataka) India, [2]Department of Biochemistry, [3]Department of Pharmacology, Siddaganga Medical College & Research Institute, Tumkur-572102 (Karnataka) India*

**Abstract**

*Background:* Considering the cardiovascular complications in Type 2 Diabetes Mellitus (T2DM) despite normal Total Cholesterol (TC) and Low Density Lipoprotein (LDL) levels, Non High Density Lipoprotein (non-HDL-C) and Apolipoprotein B (ApoB) were added as secondary targets and have been increasingly acknowledged as measures of risk estimation for developing atherosclerotic Cardiovascular Disease (CVD). *Aim and Objectives:* To study the correlation between ApoB100 and non-HDL-C in assessing the risk of cardiovascular complications in T2DM patients. *Material and Methods:* A total of 90 T2DM patients attending Medicine and Cardiology outpatient departments were recruited for the study. They were categorised into 2 groups -T2DM with CVD and T2DM without CVD. Three ml of fasting venous blood sample was taken from each patient and fasting blood sugar, TC, triglycerides, HDL-C and ApoB were estimated on VITROS 5600 Integrated Autoanalyser. LDL-C was calculated using Friedewald formula and non-HDL-C was calculated by subtracting HDL-C from TC. *Results:* The levels of ApoB and non-HDL-C in T2DM patients with CVD and without CVD were found to be significant with $p < 0.001$. There was a significant correlation between ApoB and non-HDL-C values in both the groups as analysed by Karl Pearson's correlation coefficient($p < 0.001$). After linear regression analysis, non-HDL-C was found to have a strong correlation with ApoB100 in both the groups with $r^2$ of 0.753 and 0.215 respectively ($p < 0.001$). *Conclusion:* The present study has demonstrated a strong correlation between ApoB and non-HDL-C in T2DM patients with or without CVD and hence can substitute for each other.

**Keywords**: ApoB100, Atherosclerosis, Cardiovascular disease, Diabetes mellitus, Non HDL cholesterol

## Introduction

Non-communicable diseases are now a massive challenge in reducing morbidity and mortality as they are known to cause major cardiovascular changes. One among non-communicable diseases is Type II Diabetes Mellitus (T2DM) in which patients tend to be soft targets for deadly Cardiovascular Diseases (CVD) due to impaired lipid metabolism besides enduring multiple complications of chronic hyperglycaemia [1].

CVD are the leading cause of premature death and disability in humans and also generate a high socio-economic burden in the general population [1]. Hyperglycaemia in T2DM leads to the formation of several Advanced Glycation End Products (AGEs). Apolipoprotein B100 (ApoB) is one such glycated protein that causes the formation of proinflammatory products by stimulating receptors on various cells related to the development of atherosclerosis and thus playing a vital role in the formation of atherosclerotic plaque [2].

Measurement of serum ApoB reflects total Low Density Lipoprotein (LDL-C), Intermediate Density Lipoproteins (IDL), Very Low Density Lipoprotein (VLDL-C), and Lipoprotein (a) [Lp(a)] particle concentrations because each particle contains exactly one molecule of ApoB100. Thus, ApoB can be considered a powerful tool for assessment of atherogenic lipid status.

Dyslipidemiais found to be one of the significant risk factors for CVD [3]. The pattern of dyslipidemia in diabetes is characterized by elevated triglyceride, low levels of High Density Lipoprotein (HDL-C), and increased prevalence of LDL-C particles. National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) has recognised hypertriglyceridemia as a risk factor for coronary artery disease. Small dense (sd) LDL are highly significant risk biomarkers for premature CVD and many studies have shown that it can predict CVD occurrence independent of traditional lipid parameters. However, it is not being widely used due to the limited availability of specialised equipment for its estimation, time-consuming and high cost. It has also been stated that LDL-C is not a valid basis for the therapeutic purpose at triglyceride levels over 200 mg/dL; rather non-HDL-C is designated as"atherogenic cholesterol" and is identified as a therapeutic target at very high levels of Triglycerides (TG) [3].

Non-HDL-C has been found to be strongly associated with the risk of CVD when defined based on Total Cholesterol (TC)/HDL ratio. Non-HDL-C provides information on the concentrations of all atherogenic lipoproteins [i.e., LDL, VLDL, IDL, and Lp(a)] and is determined by subtracting HDL-C from TC. The levels of non-HDL-C should be under 130 mg/dl for anyone aged 20 years or older and the higher the levels, higher the risk of heart diseases [4].

Many large prospective studies like Apolipoprotein -related MOrtalityRISk (AMORIS) have proposed the relation between Apolipoproteins and the risk of cardiovascular diseases in the general population [5-6]. Normal ApoB values range from 60 to 130 mg/dL, when exceeded indicate a high risk of cardiovascular events.

INTERHEART Modifiable Risk Score (IHMRS) has been developed by the more recent INTERHEART studies whichwere conducted in 52 countries worldwide and the Apolipoprotein ratio independently topped the list in predicting risk of future CVDs [7]. A study by Hermans *et al*. demonstrated non-HDL-C represents not only a metabolic surrogate but is close to a true biological equivalent of ApoB in a specific population [8]. Diabetes mellitus is defined as plasma glucose ≥ 126 mg/dL in at least two measurements, Glycated Hemoglobin (HbA1c) ≥ 6.5%, or prescription of any antidiabetic medication [9]. CVDs are predominantly of atherosclerotic origin where the patient has a history of coronary artery disease or cerebrovascular disease or venous thromboembolism or peripheral vascular diseases [10]. Hence this study was undertaken to establish the correlation between Apo-B100, an established risk factor and non HDL-C, a cost-effective and reliable parameter in assessing the risk of developing cardiovascular complications in patients of T2DM in the general population.

**Material and Methods**

This was a cross sectional study conducted in a tertiary care hospital at Tumkur. The ethical clearance from Institutional Ethics Committee was obtained before the initiation of the project. Ninety

participants > 18 years of age of both the gender who were ready to give informed consent, diagnosed with diabetes mellitus according to American Diabetes Association (ADA) criteria [9], and attending Medicine and Cardiology Outpatient Departments (OPDs) were included in the study through convenient sampling method. While subjects with history of acute/chronic infections, history of acute/chronic inflammations, neoplasms, previous history of myocardial infarction, and previous history of coronary artery intervention were excluded.

After obtaining informed consent in the local language, baseline characteristics of participants such as age, gender, vitals, comorbidities and their duration and concomitant medications were recorded in a predesigned proforma. Under aseptic precautions, 3 ml of fasting venous blood was collected from each of the study participants and the following parameters were estimated. Fasting blood glucose was estimated by colorimetric Glucose Oxidase Peroxidase (GOD-POD) method [11]. TC was estimated by cholesterol oxidase method (colorimetric), TG levels by Lip/Glycerol kinase method (colorimetric), HDL-C by non-HDL precipitation method (colorimetric) and ApoB100 by Electrochemiluminescence Immunoassay (ECLIA) method [12-13]. All the parameters were analysed on the VITROS 5600 integrated auto-analyzer. LDL-C was calculated using Friedewald formula [14] and non-HDL-C was calculated by subtracting HDL-C from TC.

**Statistical analysis**

Statistical analysis was performed using IBM SPSS® version 20.0 and a *p*-value below 0.05 was considered statistically significant. Results were presented as mean value ± standard deviation. Student t-test for independent variables was used to compare continuous variables with normal distribution between groups. Karl Pearson correlation was used to analyze the correlation between non-HDL-C and ApoB100, and $r^2$ was calculated using linear regression analysis.

**Results**

A total of 90 patients diagnosed with T2DM were recruited for the study. All the patients were middle aged adults (Table 1). They were divided into 2 groups as patients with CVD (n = 45) and without CVD (n = 45). Their demographic profile and the biochemical values have been presented in Table 1. A significant difference in serum levels of TC, TG and VLDL among cases and controls was observed with $p < 0.001$. The ApoB100 levels in cases were found to be 108.74 ± 22.22 and in controls 81.84 ± 23.75 with a significant difference ($p < 0.001$). Non HDL-C levels in cases were 150.71 ± 30.69 and in controls 118.20 ± 30.42 with a difference ($p < 0.001$). There was no significant age difference in both groups. After applying Pearson's correlation coefficient, ApoB100 showed a positive correlation with non-HDL-C with r value of 0.868 and was statistically significant ($p < 0.001$) as shown in Table 2. The findings were strongly supported by the scatter plot with positive correlation between ApoB100 and non-HDL-C in patients with CVD (Figures 1 and 2). Receiver Operating Characteristic (ROC) for both the parameters were plotted. ApoB100 had a sensitivity of 73% and specificity of 74% with a cut-off value of 99.01 whereas non-HDL-C had sensitivity of 76% and specificity of 61% with a cut off value of 125.5 (Table 4, Figure 3).

**Table 1: Comparison of various parameters between patients with or without CVD**

| Type | Patients without CVD | Patients with CVD | *p** |
|---|---|---|---|
| **Age** | 55.48 ± 11.18 | 54.67 ± 11.84 | 0.737 |
| **FBS/RBS** | 144.63 ± 40.38 | 198.42 ± 71.82 | <0.001 |
| **TC** | 150.89 ± 37.21 | 184.07 ± 29.44 | <0.001 |
| **HDL** | 31.61 ± 8.55 | 39.04 ± 11.05 | 0.001 |
| **LDL** | 83.61 ± 26.56 | 100.36 ± 27.32 | 0.004 |
| **VLDL** | 32.89 ± 16.01 | 51.49 ± 22.60 | <0.001 |
| **TG** | 148.07 ± 56.38 | 253.49 ± 108.50 | <0.001 |
| **ApoB100** | 81.84 ± 23.75 | 108.74 ± 22.22 | <0.001 |
| **Non-HDL** | 118.20 ± 30.42 | 150.71 ± 30.69 | <0.001 |

**Independent sample t-test*

**Table 2: Karl Pearson's correlation coefficient of ApoB100 with Non-HDL**

| | Correlation | *p* |
|---|---|---|
| **Patients with CVD** | | |
| Non HDL | 0.868 | <0.001 |
| **Patients without CVD** | | |
| Non HDL | 0.464 | 0.001 |

**Table 3: Linear regression equation for estimation of ApoB100 using Non-HDL**

| Regression Equations | SSE | $r^2$ | *p* |
|---|---|---|---|
| **Equation for Diabetes with CVD** | | | |
| 1.778 + 0.677 (Non-HDL) | 11.944 | 0.753 | <0.001 |
| **Equation for Diabetes without CVD** | | | |
| 58.127 + 0.336 (Non-HDL) | 19.914 | 0.215 | 0.001 |

**Table 4: Sensitivity, specificity, AUC and cut off values OD ApoB100 and non-HDL among patients with and without CVD**

| Parameter | Sensitivity | Specificity | AUC | Cut-off |
|---|---|---|---|---|
| **ApoB100** | 73% | 74% | 0.80 | 99.01 |
| **Non-HDL** | 76% | 61% | 0.76 | 125.5 |

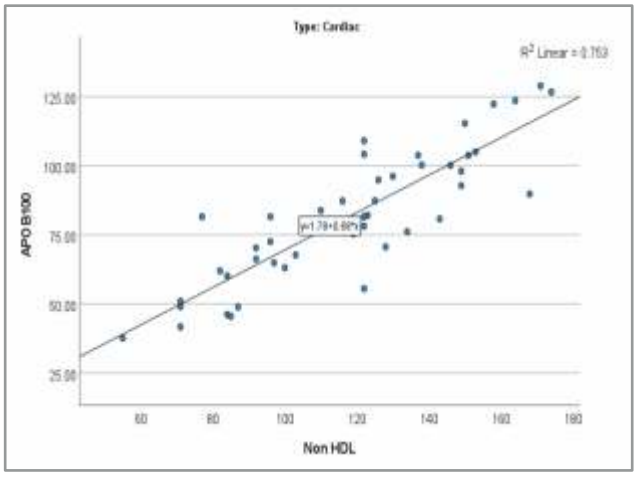


**Figure 1: Scatter plot for correlation between ApoB100 and Non-HDL in patients with CVD**

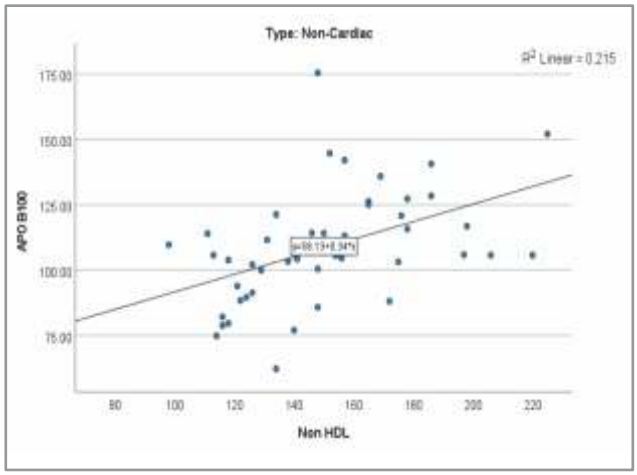


**Figure 2: Scatter plot for correlation between ApoB100 and Non-HDL in patients without CVD**

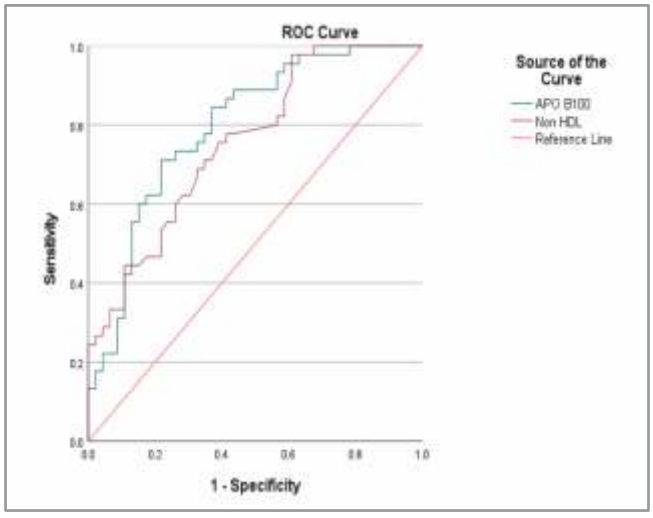


**Figure 3: Receiver Operating Characteristics**

**Discussion**

CVD is the leading cause of death in T2DM patients, thus prioritizing cardiovascular risk reduction becomes of utmost importance in these individuals [15-16]. T2DM patients with LDL-C within the target range suffer from cardiovascular events. It suggests that there is residual CV risk that is unaccounted for by LDL-C assessment. Hence, the 2016 ESC/EAS guidelines had highlighted the importance of ApoB and non-HDL-C determination in patients with diabetes [17].

Findings in the present study showed a statistically significant difference in the ApoB levels in patients with CVD and those without CVD individuals which is concordant with the study by Jiang *et al*. in which diabetic patients with CVD had ApoB values of 111.1 ± 22.2 and without CVD had 104 ± 24.6 with a significant *p* value [17]. Non-HDL-C levels in patients with CVD were 150.71 ± 30.69 and in patients without CVD were 118.20 ± 30.42 with a significant $p < 0.001$ which is also similar to the study by Jiang *et al*. in which diabetic patients with CVD had non-HDL-C values of 181.1 ± 36 and without CVD had 168 ± 39.4 with a significant *p* value [17]. There was a significant correlation between ApoB and non-HDL-C values in both the groups as analyzed by Karl Pearson's correlation coefficient ($p < 0.001$) as shown in Table 2 which is similar to a study done by Wagner *et al*. in which both non-HDL and ApoB when compared with LDL-C had given positive correlation regarding the occurrence of CVD [12]. This may be due to the characteristics of Apo B and non-HDL-C where, ApoB is essential for the binding of LDL particles to the LDL receptor, allowing cells to internalize LDL, thus absorbing cholesterol and an excess of Apo B-containing particles is a main trigger in the atherogenic process [18-19]. Non-HDL-C is a calculated parameter and its measurement is representative of all atherogenic ApoB containing lipoproteins which includes LDL-C, VLDL-C, IDL-C, and Lp(a) [19]. After linear regression analysis, non-HDL was found to have a strong correlation with ApoB100 in both the groups with $r^2$ of 0.753 and 0.215 respectively ($p < 0.001$) which is similar to a study by Hermans *et al*. [8] which demonstrated, non-HDL-C and ApoB100 with the uncorrected linear regression of ApoB (mg/dl) = 0.60 [non-HDL-C (mg/dl)] + 12.0 mg/dl which signified that both performed equally well to discriminate patients according to their atherogenic cholesterol values or atherogenic particles number and the underlying correlation between these two continuous variables reached unity [20-21]. The scatter plot as shown in Figure 1 showed a stronger linear correlation with $r^2$ value of 0.723 of ApoB 100 and non-HDL-C with cardiovascular events which indicates both the variables are interrelated and slight change in non-HDL-C can cause changes in values of ApoB100 whereas as in Figure 2, though there is a linear correlation but $r^2$ value is 0.215 which is very weak relationship among non-cardiac patients. This shows that ApoB100 and non-HDL-C can be major predictors of cardiovascular events.

ApoB is less sensitive than non-HDL-C (73% vs. 76%) but specificity was higher for ApoB than non-HDL-C (74% vs. 61%) which is in contrast to a study by Aditya *et al*. where ApoB was more sensitive and non-HDL-C was more specific [22]. The area under ROC curve was greater for Apo B

than non-HDL-C (0.80 vs. 0.76). Though ApoB and non-HDL-C theoretically often equally reflect the atherogenic burden, ApoB was a more discriminating factor for ACS cases than non-HDL-C which is similar to Sniderman *et al*. [23] which found that ApoB is superior over non-HDL-C and in contrast to Aditya *et al*. [22] in which non-HDL-C ($r = 0.138$, $p < 0.001$) was slightly superior in predicting cardiovascular events. Villalpando *et al*. [9] in their study showed a positive association between ApoB/ApoA1 ($r^2$=0.187, $p$=0.001), and non-HDL-cholesterol/HDL-cholesterol ($r^2 = 0.269$, $p = 0.0001$) ratios and between ApoB/ApoA1 ratio and ischemic cardiomyopathy ($r^2 = 0.160$, $p = 0.032$) after adjusting for comorbidities and risk factors in women with MS.

Though ApoB and non-HDL-C are considered to be the secondary targets for lipid-lowering therapy according to the 2016 European Society of Cardiology/European Atherosclerosis Society (ESC/EAS Guidelines), several studies have demonstrated the role of these 2 parameters concerning the occurrence of cardiovascular disease in T2DM [23-24].

Sondermeijer *et al*. [25] concluded that both non-HDL-C, as well as Apo-B, are equivalent in predicting the future CAD risk. Our study is in line with many studies and demonstrated that in diabetic patients, the results confirm the equivalence of both measurements from normal to frank dyslipidemia with elevated cholesterol (non-HDL-C) and/or atherogenic particles (ApoB). Unlike Apo-B, non-HDL-C can be easily determined from the standard lipid profile panel and requires no additional expense and thus, is readily available for clinical decision making [26-28]. Also, the mean time to report for Apo-B is about four times longer than that of non-HDL-C. Additionally, non-HDL- C seems able to predict CVD over a wider range of TG concentrations surpassing Friedewald formula limitation, which is affected by TG concentrations [29-30]. The limitations of our study are that sample size was small and it was a cross-sectional study conducted at a single centre. Follow-up of the patients was required for assessing risk of cardiovascular complications.

## Conclusion

Though there are many studies which prove the role of ApoB in the CVD complications in T2DM, it is costly and a developing country like India cannot afford it to all segments of population. Both the Apo-B and non-HDL-C are being considered as parameters of cardiovascular risk stratification beyond LDL-C. Considering pathophysiological reliability, easy to determine and low cost, it can be proposed that non-HDL-C can be used as an atherogenic index instead of ApoB in T2DM patients in a developing countries.

## Acknowledgements

The authors would like to acknowledge the services provided by the faculty of Medicine and Cardiology department in recruiting the patients and controls. We would also thank Mr. Riyaz Ahmed for helping us in statistical analysis of the data.

## References


1. Grundy SM, Benjamin IJ, Burke GL, Chait A, Eckel RH, Howard BV, *et al*. Diabetes and cardiovascular disease: a statement for healthcare professionals from the American Heart Association. *Circulation* 1999; 100(10):1134-46.
2. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, And Treatment of High Blood Cholesterol In Adults (Adult Treatment Panel III). *JAMA* 2001; 285(19):2486-97.
3. Pallavi B, Krishnamurthy U, Radhika K. Association of Non HDL cholesterol with risk of cardiovascular disease defined by total cholesterol/HDL cholesterol ratio. *Int J Pharm Bio Sci* 2016;7(1):285-288.
4. Walldius G, Jungner I, Holme I, Aastveit AH, Kolar W, 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.
5. Walldius G, Jungner I. Apolipoprotein B and apolipoprotein A-I: risk indicators of coronary heart disease and targets for lipid-modifying therapy. *J Intern Med* 2004;255(2):188-205.
6. Walldius G, Aastveit AH, Jungner I. Stroke mortality and the apoB/apoA-I ratio: results of the AMORIS prospective study. *J Intern Med* 2006;259(3):259-266.
7. McGorrian C, Yusuf S, Islam S, Jung H, Rangarajan S, Avezum A, *et al*. INTERHEART Investigators. Estimating modifiable coronary heart disease risk in multiple regions of the world: the INTERHEART Modifiable Risk Score. *Eur Heart J* 2011;32(5):581-589.
8. Hermans MP, Sacks FM, Ahn SA, Rousseau MF. Non-HDL-cholesterol as valid surrogate to apolipoprotein B100 measurement in diabetes: Discriminant Ratio and unbiased equivalence. *Cardiovasc Diabetol* 2011; 10:20.
9. Reynoso-Villalpando GL, Sevillano-Collantes C, Valle Y, Moreno-Ruiz I, Jorge Ramón Padilla-Gutiérrez JR *et al*. ApoB/ApoA1 ratio and non-HDL-cholesterol/ HDL-cholesterol ratio are associated to metabolic syndrome in patients with type 2 diabetes mellitus subjects and to ischemic cardiomyopathy in diabetic women. *Endocrinol Diabetes Nutr (EnglEld)* 2019; 66(8):502-511.
10. Richardson TG, Sanderson E, Palmer TM, Ala-Korpela M, Ference BA, Davey Smith G, *et al*. Evaluating the relationship between circulating lipoprotein lipids and apolipoproteins with risk of coronary heart disease: A multivariable Mendelian randomisation analysis. *PLoS Med* 2020; 17(3):e1003062.
11. Fonseca L, Paredes S, Ramos H, Oliveira JC, Palma I. Apolipoprotein B and non-high-density lipoprotein cholesterol reveal a high atherogenicity in individuals with type 2 diabetes and controlled low-density lipoprotein-cholesterol. *Lipids Health Dis* 2020; 19(1): 127.
12. Wägner AM, Pérez A, Zapico E, Ordóñez-Llanos J. Non-HDL cholesterol and apolipoprotein B in the dyslipidemic classification of type 2 diabetic patients. *Diabetes Care* 2003;26(7):2048-51.
13. American Diabetes Association. 2. Classification and Diagnosis of Diabetes: Standards of Medical Care in Diabetes-2019. *Diabetes Care* 2019; 42(Suppl 1): S13-S28.
14. 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(6):499-502.
15. Catapano AL, Graham I, De Backer G, Wiklund O, Chapman MJ, Drexel H, *et al*. 2016 ESC/EAS Guidelines for the Management of Dyslipidaemias. *Eur Heart J* 2016; 37(39):2999-3058.
16. Paredes S, Fonseca L, Ribeiro L, Ramos H, Oliveira JC, Palma I. Novel and traditional lipid profiles in metabolic syndrome reveal a high atherogenicity. *Sci Rep* 2019;9:1-7.
17. Jiang R, Schulze MB, Li T, Rifai N, Stampfer MJ, Rimm EB, Hu FB. Non-HDL cholesterol and apolipoprotein B predict cardiovascular disease events among men with type 2 diabetes. *Diabetes Care* 2004;27(8):1991-1997.
18. Mach F, Baigent C, Catapano AL, Koskina KC, Casula M, Badimon L, *et al*. 2019 ESC/EAS guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk. *Eur Heart J* 2020; 41(1): 111-118.
19. Holewijn S, den Heijer M, Swinkels DW, Stalenhoef AF, de Graaf J. Apolipoprotein B, non-HDL cholesterol and LDL cholesterol for identifying individuals at increased cardiovascular risk. *J Intern Med* 2010; 268(6):567-577.

20. Grundy SM. Low-density lipoprotein, non-high-density lipoprotein, and apolipoprotein B as targets of lipid-lowering therapy. *Circulation* 2002; 106(20): 2526-2529.
21. Bittner V. Non-HDL Cholesterol: Measurement, interpretation and significance. *John Hopkins Advance Studies Med* 2007; 7: 8-11.
22. Aditya GP, Bari MA. Apolipoprotein B versus non-high density lipoprotein cholesterol as a discriminating factor for acute coronary syndrome in young people. *Mymensingh Med J* 2016; 25(3):458-64.
23. Sniderman AD. Non-HDL cholesterol versus apolipoprotein B in diabetic dyslipoproteinemia: alternatives and surrogates versus the real thing. *Diabetes Care* 2003; 26(7):2207-2208.
24. Sniderman AD, Williams K, Contois JH, Monroe HM, McQueen MJ, de Graaf J, *et al*. A meta-analysis of low-density lipoprotein cholesterol, non-high-density lipoprotein cholesterol, and apolipoprotein B as markers of cardiovascular risk. *Circ Cardiovasc Qual Outcomes* 2011; 4(3):337-345.
25. Sondermeijer BM, Rana JS, Arsenault BJ, Shah PK, Kastelein JJ, Wareham NJ, *et al*. Non-HDL cholesterol vs. apo B for risk of coronary heart disease in healthy individuals: the EPIC-Norfolk prospective population study. *Eur J Clin Invest* 2013; 43(10):1009-1015.
26. Lu W, Resnick HE, Jablonski KA, Jones KL, Jain AK, Howard WJ, *et al*. Non-HDL cholesterol as a predictor of cardiovascular disease in type 2 diabetes: the strong heart study. *Diabetes Care* 2003; 26(1):16-23.
27. Boekholdt SM, Arsenault BJ, Mora S, Pedersen TR, LaRosa JC, Nestel PJ, *et al*. Association of LDL cholesterol, non-HDL cholesterol, and apolipoprotein B levels with risk of cardiovascular events among patients treated with statins: a meta-analysis. *JAMA* 2012; 307(12):1302-1309.
28. Hariharan V, Namrithaa S. A study on high ApoB and LDL levels in people attending a tertiary hospital in Coimbatore, India. *J Krishna Inst Med Sci Univ* 2024; 13(2):98-104.
29. Sarah JM, John S, Madhanagopal R. Risk factors contributing to dyslipidemia among female school teachers. *J Krishna Inst Med Sci Univ* 2018; 7(3):48-58.
30. Marjani A, Poursharifi N, Sajedi A, Tatari M. Age and gender related paraoxonase I gene polymorphisms rs662 and rs854560 and serum activity in subjects with metabolic syndrome in fars ethnic group. *J Krishna Inst Med Sci Univ* 2023; 12(4):68-80.

***Author for Correspondence:***
*Dr. Indushree T, Department of Pharmacology, Siddaganga Medical College & Research Institute, Tumkur-572102, Email: indushri1509@gmail.com Cell: 9663791855*

**How to cite this article:**
Vidyashree, Lakshmi D, Prashanth D, Indushree T, Hamsaveena. Non-HDL cholesterol: A valid surrogate marker to apolipoprotein b100 in type 2 diabetes mellitus patients in assessment of risk of cardiovascular diseases. *J Krishna Inst Med Sci Univ* 2024; 13(3):146-154.

***Submitted:*** *17-Apr-2024* ***Accepted:*** *21-June-2024* ***Published:*** *01-July-2024*