Research Article

Comparison of lipid profile and new atherogenic indices among the coronary artery disease (CAD)-negative and -positive diabetic dyslipidemia subjects

Pusapati Madan Ranjit[1,2], Girija Sankar Guntuku[3], Ramesh Babu Pothineni[4]

[1,2]Department of pharmaceutical biotechnology, School of Pharmacy, Jawaharlal Nehru Technological University, Kakinada, Andhra Pradesh, India.
[3]Department of pharmaceutical biotechnology, University College of Pharmaceutical Sciences, Andhra University, Visakhapatnam, Andhra Pradesh, India.
[4]Department of Cardiology, Dr. Ramesh Cardiac and Multispecialty Hospital, Vijayawada, Andhra Pradesh, India.
Correspondence to: Pusapati Madan Ranjit, E-mail: madanranjit@gmail.com

Received May 11, 2015. Accepted May 16, 2015.

Abstract

Background: Type 2 diabetes mellitus (T2DM) is associated with a cluster of interrelated plasma lipid and lipoprotein abnormalities including reduced HDL cholesterol.

Objective: To compare the lipid profile and new atherogenic indices among the coronary artery disease (CAD)-negative and -positive diabetic dyslipidemia subjects and determine the use of atherogenic indices in the early prediction of CAD in diabetic subjects.

Materials and Methods: A total number of 194 subjects participated in our study, of which 65 people with diabetic dyslipidemia were considered cases and 129 nondiabetic people with normal lipid profile were considered control subjects based upon their fasting blood glucose (FBG) and lipid profiles. Furthermore, among the 65 cases, 38 subjects were identified as CAD negative and rest of the 27 subjects were CAD-positive subjects.

Result: Lipid profiles of both CAD-negative and -positive diabetic subjects showed significantly different values than the control subjects except for high-density lipoprotein cholesterol (HDL-c) value (41.89 ± 1.49; $p > 0.05$) of CAD-negative subjects. The comparison between the CAD-negative and -positive diabetic dyslipidemia subjects observed significantly higher values of triglycerides (339.59 ± 52.24; $p < 0.001$) and very low-density lipoprotein cholesterol (VLDL-c) (67.91 ± 10.44; $p < 0.001$) and lower values of HDL-c (35.59 ± 1.40; $p < 0.001$) in CAD-positive subjects. When compared with control subjects, both CAD-negative and -positive subjects showed higher values of atherogenic indices that were significantly ($p < 0.01$ and $p < 0.001$, respectively) different.

Conclusion: Our results indicate that atherogenic indices may be useful for identifying individuals at higher risk of cardiovascular disease in the clinical practices, especially, not markedly deranged or in centers with insufficient resources.

KEY WORDS: Diabetic dyslipidemia, coronary artery disease, atherogenic indices

Access this article online	
Website: http://www.ijmsph.com	Quick Response Code:
DOI: 10.5455/ijmsph.2015.11052015326	

Introduction

Diabetes mellitus (DM) is one of the most important diseases in modern society; according to the World Health Organization (WHO) study experts, an increase in the incidence of DM up to 300 million can occur, in 2025, among the persons aged older than 25 years.[1] India alone would have 57 million diabetic patients, particularly of type 2 diabetes mellitus (T2DM), constituting 90% of the DM population.[2]

Patients with T2DM are at a larger risk of developing vascular disorders: coronary artery disease (CAD)/coronary heart disease (CHD), stroke, peripheral arterial disease (PAD), cardiomyopathy, and congestive cardiac failure,[3] for the reason that they are associated with a cluster of interrelated plasma lipid and lipoprotein abnormalities, including reduced high-density lipoprotein-cholesterol (HDL-c) and predominantly higher levels of small-dense low-density lipoprotein-cholesterol (LDL-c) particles, very low-density lipoprotein-cholesterol (VLDL-c), and elevated triglycerides (TGs).[4,5] Irrespective of the ethnic background, the risk of CAD among the diabetic patients is greater by a factor of 2 to 4 times when compared with nondiabetic subjects.[3,6] Individuals with T2DM are at a higher risk of developing CAD than are non-T2DM patients. In addition, 75% of T2DM patients die as a consequence of cardiovascular diseases including CAD.[7,8] The aim of this study was to compare the lipid profiles and new atherogenic indices among the CAD-negative and -positive diabetic dyslipidemia subjects and determine the use of atherogenic indices in the early prediction of CAD in diabetic subjects.

Materials and Methods

Study Design

This cohort study was carried out at Dr. Ramesh Cardiac and Multispecialty Hospital, Ltd., Vijayawada, Andhra Pradesh, India. The study subjects were selected randomly, who were on a visit to the hospital for their general health checkup. The study protocol was approved by the Institutional Ethical Committee and was conducted during the period from 2012 to 2014. The selection of subjects was carried out by implementing certain inclusion criteria such as estimation of fasting blood glucose (FBG) and lipid profile, based upon selecting both, the cases and healthy subjects. Likewise, certain exclusion criteria followed were: subjects with hepatic, metabolic, and renal diseases and those who were on exogenous hormones supplements, on hormone replacement therapy, or on the use of lipid lowering drugs were excluded from the study. An informed written consent was obtained from all the study subjects who participated in our study.

Data Collection and Selection of Subjects

Systemic examination of each subject was carried out; it included their name, age, address, type of diet, occupation, physical exercise, present and past medical illness, and family history. Anthropometric assessments such as height in meter (m), weight in kilogram (kg), and body mass index (BMI) were done. The BMI was calculated by weight in kilograms divided by the square of the height in meter (kg/m^2). The selection of subjects was based upon the plasma lipid abnormalities and FBG cutoff values given by the expert panel of the National Cholesterol Education Program (NCEP).[9] A total number of 194 subjects participated in our study, of which 65 people with diabetic dyslipidemia were considered as cases and 129 nondiabetic subjects with normal lipid profile selected as control subjects. Among the 65 subjects, 38 subjects were considered as CAD-negative subjects, enrolled individuals with normal clinical, cardiologic, and resting and stress electrographic assessments. The rest of the 27 subjects were considered as CAD-positive subjects, enrolled individuals with abnormal clinical, cardiologic, and precordial pain and characteristic electrocardiographic changes.[10]

Collection of a Blood sample and Estimation of Lipid Profile

Fasting blood samples were collected in the morning between 7 a.m. and 8 a.m. by venepuncture of antecubital vein with all aseptic precautions, using a dry disposable syringe under sterile conditions. Fresh plasma and serum were used for the estimation of FBG and total cholesterol (TC), TGs, and HDL-c, respectively. The tests were carried out in an automated clinical auto analyzer. Furthermore, LDL-c, VLDL-c, and non-HDL-c were calculated by using Friedewald's formula.[11] In addition, atherogenic indices such as, Castelli's Risk Index (CRI)-I = TC/HDL-c, CRI-II = LDL-c/HDL-c, atherogenic coefficient (AC) = (TC − HDL-c)/HDL-c, TGs/HDL-c ratio, and atherogenic index of plasma (AIP) = log (TGs/HDL-c) were calculated from the individuals.

Statistical Analysis

The collected data were analyzed by using Graph Pad Prism, version 6. The differences in the groups were determined by performing the one-way analysis of variance (ANOVA) and the Tukey–Kramer multiple comparisons test; data were expressed either as mean ± standard error mean (SEM). The statistical significance was set at the p values of $p < 0.05$, $p < 0.01$, and $p < 0.001$, and $p > 0.05$ was considered as nonsignificant.

Result

Table 1 shows the mean ± SEM values of the age, weight, and BMI of the diabetic dyslipidemia and control subjects. Diabetic dyslipidemia subjects were further divided into CAD-negative and -positive subjects. Table 2 shows the mean ± SEM values of the FBG levels and lipid profiles of the diabetic and control subjects. The mean values of the FBG of CAD-negative and -positive group showed significantly higher values than the control nondiabetic subjects but did not show significantly different values when CAD-negative subjects were compared with CAD-positive subjects. Table 2 also shows the mean ± SEM values of the lipid profiles of both CAD-negative and -positive diabetic subjects, which were significantly different when compared with those of the control subjects, except the HDL-c value of CAD-negative subjects. The comparison between the CAD-negative and -positive diabetic dyslipidemia subjects revealed significantly higher values of TGs and VLDL-c and lower values of HDL-c in CAD-positive subjects.

Table 1: Comparison of age and BMI by Tukey–Kramer multiple comparisons test of controls with CAD-negative (CADN) and CAD-positive (CADP) diabetic dyslipidemia subjects

Parameter	Control (*n* = 129), mean ± SEM	Subjects with dyslipidemia and diabetes (*n* = 65)		*p*, CADN vs. CADP
		CADN, mean ± SEM (*n* = 38)	CADP, mean ± SEM (*n* = 27)	
Age	45.77 ± 1.36	54.71 ± 1.64**	51.37 ± 1.78ns	>0.05
BMI	24.26 ± 0.39	26.48 ± 0.72ns	26.23 ± 0.87ns	>0.05

**$p < 0.01$, significant; ns, nonsignificant.

Table 2: Comparison of FBG and lipid profile by Tukey–Kramer multiple comparisons test of controls with CAD-negative (CADN) and CAD-positive (CADP) diabetic dyslipidemia subjects

Parameter	Control (*n* = 129) mean ± SEM	Subjects with dyslipidemia and diabetes (*n* = 65)		*p*, CADN vs. CADP
		CADN, mean ± SEM (*n* = 38)	CADP, mean ± SEM (*n* = 27)	
FBG	87.96 ± 0.47	156.61 ± 7.48***	162.00 ± 8.63***	>0.05
TC	153.71 ± 1.46	204.68 ± 8.56***	199.52 ± 10.25***	>0.05
TGs	110.53 ± 2.32	166.61 ± 12.21*	339.59 ± 52.24***	<0.001
LDL-c	87.99 ± 1.26	204.68 ± 8.56***	199.52 ± 10.25***	>0.05
VLDL-c	22.10 ± 0.46	33.32 ± 2.44*	67.91 ± 10.44***	<0.001
HDL-c	43.61 ± 0.46	41.89 ± 1.49ns	35.59 ± 1.40***	<0.001
Non-HDL-c	110.10 ± 1.38	162.79 ± 7.33***	163.93 ± 9.28***	>0.05

*$p < 0.05$, significant; **$p < 0.01$, significant; ***$p < 0.001$, significant; ns, nonsignificant.

Table 3: Comparison of atherogenic indices by Tukey–Kramer multiple comparisons test of controls with CAD-negative (CADN) and CAD-positive (CADP) diabetic dyslipidemia subjects

Parameter	Control (*n* = 129), mean ± SEM	Subjects with dyslipidemia and diabetes (*n* = 65)		*p*, CADN vs. CADP
		(CADN), mean ± SEM (*n* = 38)	(CADP) mean ± SEM (*n* = 27)	
CRI-I	3.55 ± 0.04	4.89 ± 0.10***	5.62 ± 0.19***	<0.001
CRI-II	2.03 ± 0.03	3.07 ± 0.12***	3.21 ± 0.25***	>0.05
TG/HDL-c	2.59 ± 0.06	4.05 ± 0.28**	9.50 ± 1.25***	<0.001
AIP	0.39 ± 0.01	0.57 ± 0.02***	0.92 ± 0.04***	<0.001
AC	2.55 ± 0.04	3.89 ± 0.10***	4.62 ± 0.19***	<0.001

**$p < 0.01$, significant; ***$p < 0.001$, significant.

Table 3 shows the mean ± SEM values of atherogenic indices of diabetic dyslipidemia and control subjects. When compared with control subjects, both CAD-negative and -positive subjects showed higher values of indices that are significantly different. Among the CAD-negative and -positive subjects, CAD-positive subjects showed all indices that were significantly different, except CRI-II.

Discussion

In our study, higher BMI value was observed in diabetic patients than control subjects but was not significantly different. BMI has been widely used as an indicator of total adiposity; its limitations are clearly recognized by its dependence on race.[12] Epidemiologic studies done earlier have shown that increasing BMI is associated with higher TC and LDL-c. However, these studies were limited by underrepresentation of obese subjects.[13] Higher levels of FBG and lipid profiles in both CAD-positive and -negative diabetic subjects were observed when compared with control subjects. Research studies have explained that the most common and serious effects of diabetes in adults is cardiovascular disorders (CVDs)[14] and the leading cause of death in patients with diabetes is CVDs than individuals without diabetes.[15,16] Research studies have also explained that, in diabetes, elevated levels of TGs are observed, owing to alterations in metabolism that include increased hepatic secretion of VLDL-c, impaired clearance of VLDL-c, and intestinally derived chylomicrons. These remnants include intermediate density lipoproteins (IDL-c) that are particularly involved in the development of atherogenesis in humans and in a number

of animal models[17,18]; these remnants are also responsible for the increased production of precursors of small-dense LDL-c particles.[19] Previous studies have also explained that plasma VLDL-c levels correlate with the increased density and decreased size of LDL-c particles.[20,21] In addition, LDL-c particles size and density are inversely related to the plasma levels of HDL-c.[22] Moreover, several studies have also suggested that development of insulin resistance was seen in patients with diabetic dyslipidemia[23–25]; furthermore, it was associated with hypertriglycerides, higher levels of VLDL-c, and low levels of HDL-c cholesterol.[26–28] These higher levels of triglycerides and VLDL-c particles may impair insulin action by inhibiting insulin binding to its receptor.[29,30] Garg et al.[31] proved and suggested that insulin resistance is the underlying mechanism in patients with hypertriglyceridemia and not vice versa. Lower levels of HDL-c in a diabetic person are explained by several mechanisms. First, diminished activity of lipoprotein lipase (LPL) may result in the excessive transfer of TGs from triglyceride-rich chylomicrons and VLDL-c particles in exchange for cholesterol esters from HDL-c particles, thus reducing the levels of HDL-c. Second, decreased activity of LPL resulting in reduced hydrolysis of TGs in chylomicrons particles may curtail the contribution of chylomicrons-derived nascent HDL-c particles. Further reduction of LPL activity and increased hepatic triglyceride lipase (HTGL) activity may explain markedly the lower levels of HDL-c when compared with nondiabetic subjects and patients with noninsulin-dependent diabetes mellitus (NIDDM).[32] In our study, results were concurrent with the earlier studies, such as elevated levels of TGs and VLDL-c and lower levels of HDL-c in CAD-positive subjects than in CAD-negative subjects, which may be owing to the early development of insulin resistance.

Plasma lipids can be divided into the proatherogenic lipoproteins and antiatherogenic HDL-c. Assessment of the relative proportions of cholesterol in these two fractions can be valuable than the individual lipid measurements. One method is to compare the levels of HDL-c and non–HDL-c.[9] Another method is the use of atherogenic indices; these are powerful indicators of the risk assessment of CADs. The higher the values, the higher are the risks of developing CVDs and vice versa.[33] Atherogenic ratios such as, CRI-I, CRI-II, AC,[34–36] TGs/HDL-c ratio,[37] and AIP[38] are calculated. We applied these indices for predicting the cardiovascular risks in diabetic dyslipidemia subjects. The average ratio of TC to HDL-c (CRI-I) of healthy individuals was about 3.5 or lower,[39,40] and in the case of LDL-c/HDL-c ratio (CRI-II), it was three or lower.[40,41] Another research study explained the association of TC/HDL-c with coronary plaques formation.[42] In the PROCAM study, it was observed that subjects with LDL-c/HDL-c (CRI-II) > 5 showed six times higher rate of coronary events.[43] In our study, higher values of CRI-I and CRI-II in both CAD-negative and -positive subjects than the control subjects were observed, and higher value of CRI-I in CAD-positive subjects than CAD-negative subjects was also observed. CAD-positive group was confirmed by electrocardiographic changes and other clinical characteristics; so, the above-mentioned results indicate and support this index, which may be very useful in the prediction for CAD. In the case of CRI-II, both the subjects were near to normal values owing to the normal levels of TC in both CAD-negative and -positive subjects. da Luz et al.[37] explained that the ratio of TGs to HDL-c was found to be a powerful independent indicator of extensive coronary disease. The ratio TG/HDL-c, initially proposed by Gaziano et al.,[44] is an atherogenic index that has proven to be a highly significant independent predictor of myocardial infarction, even stronger than TC/HDL-c and LDL-c/HDL-c.[44] Bampi et al.[45] reported that TGs/HDL-c ratio is possible to approximately determine the presence and extent of CAD by noninvasive methods. The above-mentioned results indicate and support TGs/HDL-c ratio as a very useful predictor for the assessment of CVDs.

AIP shows an inverse relationship that exists between TGs and HDL-c and that the ratio of TGs to HDL-c is a strong predictor of infarction, and it was used by some practitioners as a significant predictor of atherosclerosis.[46] Other researchers have suggested that, AIP is a highly sensitive marker of difference of lipoprotein in patients. AIP values of –0.3 to 0.1 are associated with low, 0.1 to 0.24 with medium, and above 0.24 with high cardiovascular risks.[46] In our study, high values ($p < 0.001$) of AIP in diabetic subjects were observed. The CAD-positive subjects were already confirmed by electrocardiographic changes and other clinical characteristics; so the aforementioned result indicates and supports that this index may be very useful in the prediction for CAD. Atherogenic coefficient (AC) is a measure of cholesterol in LDL-c, VLDL-c lipoprotein fractions with respect to good cholesterol, or HDL-c. It reflects the atherogenic potential of the entire spectrum of lipoprotein fractions. The higher the values, the higher are the risks of developing CVDs and vice versa.[33] In our study, we observed high values of AC in both CAD-negative and -positive subjects than those in control subjects; we also observed higher values of AC in CAD-positive subjects than in CAD-negative subjects.

Conclusion

Our conclusion is that BMI is not an important predictor for the assessment of CVDs in diabetic subjects; we also observed the elevated levels of TGs, LDL-c, and VLDL-c and the reduced levels of HDL-c are important risk factors for the development of CVDs in diabetic subjects. We also observed the development of cardiovascular signs in CAD-positive group that may be owing to the reduced levels of HDL-c. On the basis of earlier studies and our results, we conclude, these atherogenic indices are powerful indicators to predict the risk of CADs based on the higher values of atherogenic indices observed in CAD-positive subjects than the CAD-negative subjects. These results indicate that atherogenic indices may be useful for identifying individuals at a higher risk of CVDs in the clinical practices, especially, and not markedly deranged or in centers with insufficient resources.

References

1. Zimnet P. Diabetes care and prevention-around the world in 80 days. In: *Diabetes,* Rifkin H, Colwell. JS, Taylor SI (Eds.) Amsterdam: Elsevier, 1991. pp. 721–9.
2. Ramachandran A, Snehalatha C, Viswanathan V. Burden of type 2 diabetes and its complications—The Indian scenario. Curr Sci 2002;83(12):1471–6.
3. Kannel WB, McGee DI. Diabetes and glucose tolerance as risk factors for cardio-vascular disease. The Framingham study. Diabetes Care 1979;2(2):120–31.
4. Haffner SM; American Diabetes Association. Management of dyslipidemia in adults with diabetes. Diabetes Care 2003; 26(Suppl 1):S83–6.
5. Shaikh MA, Kumar S, Ghouri RA. Type 2 diabetes mellitus and lipid abnormalities. J Liaquat Univ Med Health Sci 2010;9(3):145–7.
6. Deepa R, Arvind K, Mohan V. Diabetes and risk factors for coronary artery disease. Curr Sci 2003;83(12):1497–505.
7. Center for Disease Control and Prevention. *National Diabetes Fact Sheet: National Estimates and General Information on Diabetes and Prediabetes in the US 2011*. Washington, DC: US Department of Health and Human Services, 2011. Available at: http://www.cdc.gov/diabetes/pubs/pdf/ndfs_2011 (last accessed on April 10, 2015).
8. Hammoud T, Tanguay JF, Bourassa MG. Management of coronary artery disease: therapeutic options in patients with diabetes. J Am Coll Cardiol 2000;36(2):355–65.
9. Grundy SM, Cleeman JI, Merz CN, Brewer HB Jr, Clark LT, Hunninghake DB, et al. Implications of recent clinical trials for the National Cholesterol Education Program Adult Treatment Panel III guidelines. Circulation 2004;110(2):227–39.
10. Almeida KA, Strunz CM, Maranhao RC, Mansur AP. The S447X polymorphism of lipoprotein lipase: Effect on the incidence of premature coronary disease and on plasma lipids. Arq Bras Cardiol 2007:88(3):297–303.
11. Freidewald 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.
12. Al-Ajlan AR. Lipid profile in relation to anthropometric measurements among college male students in Riyadh, Saudi Arabia: A cross-sectional study. Int J Biomed Sci 2011;7(2):112–9.
13. Shamai L, Lurix E, Shen M, Novaro GM, Szomstein S, Rosenthal R, et al. Association of body mass index and lipid profiles: Evaluation of a broad spectrum of body mass index patients including the morbidly obese. Obes Surg 2011;21(1):42–7.
14. Boyle JP, Honeycutt AA, Narayan KM, Hoerger TJ, Geiss LS, Chen H, et al. Projection of diabetes burden through 2050: Impact of changing demography and disease prevalence in the U.S. Diabetes Care 2001;24(11):1936–40.
15. National Diabetes Data Group. *Diabetes in America,* 2nd edn. Bethesda, MD: National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Disease, 1995. Available at: http://diabetes.niddk.nih.gov/dm/pubs/america/ (last accessed on April 10, 2015) .
16. Haffner SM, Lehto S, Rönnemaa T, Pyorala K, Laakso M. Mortality from coronary heart disease in subjects with type 2 diabetes and in nondiabetic subjects with and without prior myocardial infarction. N Engl J Med 1998;339(4):229–34.
17. Krauss RM. Atherogenicity of triglyceride-rich lipoproteins. Am J Cardiol 1998;81(4A):13B–17B.
18. Krauss RM. Triglycerides and atherogenic lipoproteins: Rationale for lipid management. Am J Med 1998;105(Suppl 1A): 58S–62S.
19. Blanche PJ, Gong EL, Forte TM, Nichols AV. Characterization of human high-density lipoproteins by gradient gel electrophoresis. Biochim Biophys Acta 1981;665(3):408–19.
20. McNamara JR, Jenner JL, Li Z, Wilson PW, Schaefer EJ. Change in LDL particle size is associated with change in plasma triglyceride concentration. Arterioscler Thromb 1992;12(11):1284–90.
21. McNamara JR, Campos H, Ordovas JM, Peterson J, Wilson PW, Schaefer EJ. Effect of gender, age, and lipid status on low density lipoprotein subfraction distribution: Results from the Framingham Offspring Study. Arteriosclerosis 1987; 7(5):483–90.
22. Krauss RM, Williams PT, Lindgren FT, Wood PD. Coordinate changes in levels of human serum low and high density lipoprotein subclasses in healthy men. Arteriosclerosis 1988;8(2):155–62.
23. Garg A, Helderman JH, Koffler M, Ayuso R, Rosenstock J, Raskin P. Relationship between lipoprotein levels and in vivo insulin action in normal young white men. Metabolism 1988;37(10):982–7.
24. Mykkanen L, Haffner SM, Ronnemaa T, Bergman R, Leino A, Laakso M. Is there a sex difference in the association of plasma insulin level and insulin sensitivity with serum lipids and lipoproteins? Metabolism 1994;43(4):523–8.
25. Boden G. Role of fatty acids in the pathogenesis of insulin resistance and NIDDM. Diabetes 1997;46(1):3–10.
26. Laakso M, Sarlund H, Mykkanen L. Insulin resistance is associated with lipid and lipoprotein abnormalities in subjects with varying degrees of glucose tolerance. Arteriosclerosis 1990;10(2):223–31.
27. Reaven GM, Chen YD, Jeppesen J, Maheux P, Krauss RM. Insulin resistance and hyperinsulinemia in individuals with small, dense low density lipoprotein particles. J Clin Invest 1993; 92(1):141–6.
28. Selby JV, Austin MA, Newman B, Zhang D, Quesenberry CP Jr, Mayer EJ, et al. LDL subclass phenotypes and the insulin resistance syndrome in women. Circulation 1993;88(2):381–7.
29. Steiner G, Vranic M. Hyperinsulinemia and hypertriglyceridemia, a vicious cycle with atherogenic potential. Int J Obes 1982;6(Suppl 1):117–24.
30. Berliner JA, Frank HJ, Karasic D, Capdeville M. Lipoprotein-induced insulin resistance in aortic endothelium. Diabetes 1984;33(11):1039–44.
31. Garg A, Helderman JH, Grundy SM, Koffler M, Unger RH. Insulin resistance underlies primary hypertriglyceridemia (Abstract). Clin Res 1989;37:449A.
32. Garg A. Insulin resistance in the pathogenesis of dyslipidemia. Diabetes Care 1996;19(4):387–9.
33. Usoro CAO, Adikwuru CC, Usoro IN, Nsonwu AC. Lipid profile of postmenopausal women in Calabar, Nigeria. Pak J Nutr 2006;5(1):79–82.
34. Castelli WP, Abbott RD, McNamara PM. Summary estimates of cholesterol used to predict coronary heart disease. Circulation 1983;67(4):730–4.
35. Brehm A, Pfeiler G, Pacini G, Vierhapper H, Roden M. Relationship between serum lipoprotein ratios and insulin resistance in obesity. Clin Chem 2004;50(12):2316–22.
36. Frohlich J, DobiásováM. Fractional esterification rate of cholesterol and ratio of triglycerides to HDL-cholesterol are

powerful predictors of positive findings on coronary angiography. Clin Chem 2003;49(11):1873–80.
37. da Luz PL, Favarato D, Faria-Neto JR Jr, Lemos P, Chagas NCP. High ratio of triglycerides to HDL-cholesterol predicts extensive coronary disease. Clinics 2008;63(4):427–32.
38. Dobiasova M. Atherogenic index of plasma [log (triglycerides/HDL-cholesterol)]: Theoretical and practical implications. Clin Chem 2004;(50)7:1113–5.
39. Reddy Kilim S, Chandala SR. A comparative study of lipid profile and oestradiol in pre-and post-menopausal women. J Clin Diagn Res 2013;7(8):1596–8.
40. Castelli WP, Garrison RJ, Wilson PW, Abbott RD, Kalousdian S, Kannel WB. Incidence of coronary heart disease and lipoprotein cholesterol levels. The Framingham Study. JAMA 1986;256(20):2835–8.
41. Jamil S, Siddiq A. Comparison of CVD risk associated with the long term use of contraceptives in young females. J Appl Pharm Sci 2012;2(11):62–6.
42. Nair D, Carrigan TP, Curtin RJ, Popovic ZB, Kuzmiak S, Schoenhagen P, et al. Association of total cholesterol/high-density lipoprotein cholesterol ratio with proximal coronary atherosclerosis detected by multislice computed tomography. Prev Cardiol 2009;12(1):19–26.
43. Assmann G, Cullen P, Schulte H. The Munster Heart Study (PROCAM). Results of follow-up at 8 years. Eur Heart J 1998;19(Suppl A):A2–11.
44. Gaziano JM, Hennekens CH, O Donnell CJ, Breslow JL, Buring JE. Fasting triglycerides, high density lipoprotein, and risk of myocardial infarction, Circulation 1997;96:2520–5.
45. Bampi ABA, Rochitte CE, Favarato D, Lemos PA, da Luz PL. Comparison of non-invasive methods for the detection of coronary atherosclerosis. Clinics 2009;64(7):675–82.
46. Dobiasova M. AIP—Atherogenic index of plasma as a significant predictor of cardiovascular risk: From research to practice. Vnitr Lek 2006;52(1):64–71.

How to cite this article: Ranjit PM, Guntuku GS, Pothineni RB, Comparison of lipid profile and new atherogenic indices among the coronary artery disease (CAD)-negative and -positive diabetic dyslipidemia subjects. Int J Med Sci Public Health 2015;4:1574-1579

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