

A Comparison of Atherogenic Indices among Untreated Hypertensives in a Tertiary Hospital in Southern Nigeria

Akpa Maclean Romokere¹, Unamba Ndubuisi Norbert^{2,*}

¹Department of Medicine, Faculty Clinical Sciences, University of Port Harcourt, Port Harcourt, Nigeria

²Department of Medicine, University of Port Harcourt Teaching Hospital, Port Harcourt, Nigeria

Abstract BACKGROUND: Cardiovascular disease (CVD) is the major cause of deaths worldwide because of increasing prevalence of hypertension, the major risk factor. Clustering of hypertension and dyslipidemia increases cardiovascular morbidity and mortality. Several atherogenic indices have been derived from standard lipid profiles measurement in an attempt to enhance predictive capacity of the lipid profile in CVD management. **OBJECTIVES:** To measure plasma lipids of untreated hypertensive patients and determine if the lipid ratios were superior in identifying dyslipidemia compared to the use of conventional lipid parameters. **METHODS:** Hypertensive subjects were consecutively enrolled from our cardiac clinic and had fasting lipid profile measured using standard procedures. The following atherogenic indices were then calculated from the lipid profile: Castelli Risk Index-I (total cholesterol/high-density lipoprotein), Castelli Risk Index-II (low-density lipoprotein/high-density lipoprotein), and the atherogenic index of plasma (AIP) [Log Triglyceride/HDL]. Descriptive statistics and correlation studies were used to determine the relationship between lipid parameters, atherogenic indices and gender. The area under the curve (AUC) of the receiver operator curve (ROC) and the 95% confidence intervals (CIs) were used to determine which index showed the highest accuracy in screening for dyslipidemia in the studied population. **RESULTS:** Two hundred and forty-eight subjects (108 males and 140 females) aged 20 to 86 years were recruited, mean age was 55.94 ± 15.29 and 53.46 ± 13.99 years for males and females respectively ($p=0.186$). Mean total cholesterol (TCH) and low-density lipoprotein (LDL-C) levels were significantly higher in the females ($p=0.005$ and $p=0.021$) respectively. Mean high-density lipoprotein (HDL-C) levels were higher in the females ($p=0.003$). 10.3% of patients had increased TCH 2.1% had elevated triglyceride, 37.3% had reduced HDL-C and 30.3% had elevated LDL-Cholesterol. AIP showed a strong inverse correlation with HDL-C ($r = -0.447$, $p < 0.001$), LDL-C ($r = -0.130$, $p = 0.045$), and TCH ($r = -0.209$, $p = 0.001$). CRI-I showed an inverse correlation with HDL-C ($r = -0.404$, $p < 0.0001$) and a positive correlation with TCH and LDL-C ($r = 0.146$, $p = 0.02$) and ($r = 0.128$, $p = 0.04$) respectively. CRI-II showed a strong inverse correlation with HDL-C ($r = -0.406$, $p < 0.0001$) and a weak positive correlation with LDL ($r = 0.284$, $p < 0.0001$). Using the Receiver Operating Curve (ROC), the CRI-II most sensitive in identifying the hypertensive individuals with dyslipidemia (AUC= 0.882). **CONCLUSIONS:** Disorders of lipid is common amongst untreated hypertensive patients in Port Harcourt and CRI –II is most sensitive for identifying dyslipidemia in the population studied.

Keywords Hypertension, Atherogenic indices, Dyslipidemia, Port Harcourt

1. Introduction

Cardiovascular disease is the leading cause of death globally and appears to have overtaken infectious diseases as a leading cause of death in developing countries [1]. Hypertension is the number one cardiovascular disease risk factor in addition to others such as dyslipidemia, obesity, tobacco, diabetes. In addition the risk of cardiovascular disease is increased several folds when several risk factors are clustered in the individual patient. Dyslipidemia is the

strongest risk factor for atherosclerotic vascular disease after age [2] which underlies all cardiovascular disease. Thus, hypertension and dyslipidemia must be seen as the most potent risk factor for cardiovascular diseases. Adequate management for hypertension must include identification and treatment of all other known risk factors for cardiovascular disease. The identification of hypertension is easy but the determination of what level of dyslipidemia to treat may not be very easy. Dyslipidemia is the strongest risk factor for myocardial infarction in addition to others such as hypertension, diabetes and tobacco. Thus a proper assessment of dyslipidemia in hypertensive patients is appropriate. Plasma lipids have both pro-atherogenic components (triglyceride and LDL cholesterol) and anti-atherogenic components, HDL cholesterol [3]. While

* Corresponding author:

norbertunamba2@gmail.com (Unamba Ndubuisi Norbert)

Published online at <http://journal.sapub.org/ajmms>

Copyright © 2017 Scientific & Academic Publishing. All Rights Reserved

guidelines have recommended threshold values for treatment of dyslipidemia [4] in developed countries with high prevalence of coronary artery disease, the determination of absolute levels of lipid that require treatment in resource limited setting with a low prevalence of coronary disease may be challenging. Studies have also shown that the risks of cardiovascular disease events attributable to dyslipidemia are better assessed using different ratios of the different components of the plasma lipid profile [5, 6]. The Castelli Risk Index I (CRI – I) assesses the ratio of total cholesterol to high density lipoprotein cholesterol (TCH/HDL-C) and sets healthy value at 3.5 or less [7], Castelli Risk Index II assesses the ratio of Low density lipoprotein cholesterol LDL -C to High Density Lipoprotein cholesterol –C (LDL –C/HDL –C) [8] while atherogenic index of plasma (AIP) is the logarithm of molar ratio of triglyceridemia to high-density lipoprotein cholesterol ($\text{Log}_{10}\text{TG}/\text{HDL-cholesterol}$) and is said to reflect the true relationship between protective and atherogenic lipoprotein and as well as being associated with the size of pre- and anti- atherogenic lipoprotein particle [9-11].

We reviewed the lipid profile of treatment naïve hypertensive patients seen in the cardiology clinic of UPTH over a one year period July 2013 to June 2014 and calculated various indices of atherogenicity with a view to determining the degree of dyslipidemia and which of the parameters best reflects the degree of dyslipidemia in our hypertensive population.

The aim of the analysis was to determine the prevalence of dyslipidemia among newly diagnosed and treatment naïve hypertensive patients, the sensitivity of various measures of dyslipidemia and which best identify the risk of cardiovascular disease.

2. Methods

The study population consisted of all the hypertensive patients referred to the cardiology clinic of the University of Port Harcourt Teaching Hospital over a two year period from July 2013 to June 2015. All the patients were treatment naïve and referred to the clinic for proper evaluation and treatment.

All patients had routine biodata collected, clinical evaluation and routine cardiovascular risk assessment including renal function, full lipid profile, 12-lead electrocardiogram and echocardiogram. All the data were entered into the clinic data base. Fasting cholesterol and triglyceride levels were measured using the enzymatic method with a reagent from Atlas Medical Laboratories. Fasting HDL was measured with the precipitation method. LDL cholesterol values were calculated using the Friedewald equation when the triglyceride level was less than 4.0mmol/l: $\text{LDL} = \text{TC} - (\text{HDL} + \text{TG} / 2.2)$ [12].

Definition of abnormal lipid profile [13]:

- Elevated triglyceride= $\text{TG} > 1.7 \text{ mmol/l}$.
- Hypercholesterolemia= $\text{TC} > 5.2 \text{ mmol/l}$.

- Low high density lipoprotein cholesterol= $\text{HDL-C} < 1.03 \text{ mmol/l}$
- Elevated low density lipoprotein cholesterol = $\text{LDL-C} > 3.0 \text{ mmol/l}$.

We extracted the relevant data and lipid profile for this report. The lipid profile results were used to calculate three atherogenic index of plasma (AIP), the Castelli Risk Index I (CRI-I) and Castelli Risk Index II (CRI –II). The Atherogenic ratios were calculated as follows: Atherogenic Index of Plasma (AIP) = $\log \text{TG}/\text{HDL-C}$; Castelli Risk Index (CRI-I) = $\text{TC}/\text{HDL-C}$; Castelli Risk Index (CRI-II) = $\text{LDL-C}/\text{HDL-C}$. According to previous studies, AIP was further classified into three groups: low (< 0.11) risk, intermediate ($0.11-0.21$), and increased (> 0.21) risk [14, 15].

Descriptive statistics and correlation studies were used to determine the relationship between these atherogenic indices and were then compared across gender and the general population. Finally, the screening ability of various atherogenic indices to identify individuals with dyslipidemia among the hypertensives was explored using receiver operating characteristic curve (ROC) analysis. The area under the curve (AUC) of the ROC which have a high discriminating power were used to determine the highest accuracy in screening for dyslipidemia in this hypertensive population. The AUC of 0.5, $0.6 \leq \text{AUC} < 0.7$, $0.7 \leq \text{AUC} < 0.8$, $0.8 \leq \text{AUC} < 0.9$, and ≥ 0.9 corresponded to no discrimination, poor, acceptable, excellent, and outstanding discrimination, respectively [16]. All analyses were performed by SPSS statistical software (version 19.0, SPSS Inc). *P* values of < 0.05 were considered statistically significant.

3. Results

Two hundred and forty-eight untreated (248) hypertensive patients (108 males and 140 females) aged 20 to 86 years were consecutively enrolled in the register over a two years period. The mean (\pm SD) age of the patients in the study population was 55.94 ± 15.29 years for the males and 53.46 ± 13.99 years for the females ($p=0.186$). The mean systolic blood pressure was $142.0 \pm 23.8 \text{ mmHg}$ for males and $137.46 \pm 24.37 \text{ mmHg}$ for females while mean diastolic blood pressure was $86.28 \pm 16.23 \text{ mmHg}$ for males and $84.43 \pm 13.74 \text{ mmHg}$ for females (See Table 1). The mean body mass index (BMI) was $26.67 \pm 4.49 \text{ Kg/m}^2$ for the males and the mean BMI was $29.40 \pm 6.80 \text{ Kg/m}^2$ for the females ($p < 0.0001$). About 27.1% of all the subjects had normal BMI, 27.1% were overweight, and 45.7% were obese.

The mean total cholesterol (TCH) was $4.69 \pm 1.36 \text{ mmol/L}$, mean low-density lipoprotein cholesterol (LDL-C) was $3.52 \pm 1.09 \text{ mmol/L}$, mean high density lipoprotein cholesterol (HDL –C) was $0.92 \pm 0.56 \text{ mmol/L}$ and mean triglycerides (TG) was $0.90 \pm 0.62 \text{ mmol/L}$. The mean TCH and LDL-C levels were significantly higher in the females than in the males ($p=0.005$ and $p=0.022$) respectively. The mean

HDL-C levels were higher in the females ($p=0.003$) (See Table 1). The mean AIP, CRI-I and mean CRI-II were comparable between sexes ($p=0.199$, $p=0.979$, and $p=0.742$ respectively).

Table 1. Baseline Anthropometric and Biochemical Parameters across Gender

Variables	Males (n=108) Mean±SD	Females (n=140) Mean±SD	Student t-test p-value
Age (years)	55.94±15.29	53.46±14.00	0.186
BMI (Kg/m ²)	26.67±4.49	29.40±6.80	<0.0001
SBP (mmHg)	142.00±23.80	137.46±24.37	0.143
DBP (mmHg)	86.28±16.23	84.43±13.74	0.333
TCH (mmol/L)	4.41±1.18	4.91±1.45	0.005
TG (mmol/L)	0.85±0.59	0.94±0.64	0.293
HDL-C (mmol/L)	0.80±0.24	1.02±0.71	0.003
LDL-C (mmol/L)	3.34±0.79	3.66±1.15	0.022
AIP	0.19±0.20	0.14±0.30	0.199
CRI-I	6.39±4.10	6.37±6.13	0.979
CRI-II	4.84±3.48	5.04±5.43	0.742

SBP=Systolic blood pressure; DBP=Diastolic blood pressure; TG=Triglyceride; LDL-C=Low density lipoprotein cholesterol; HDL-C=High density lipoprotein cholesterol; AIP=Atherogenic index of plasma; CRI-I=Castelli risk index-I; CRI-II=Castelli risk index-II

Based on absolute value of serum total cholesterol greater than 5.20mmol/L 10.3% had hypercholesterolemia, 2.1% had hypertriglyceridemia ($TG > 1.7\text{mmol/L}$) and 37.3% had reduced HDL-C ($< 1.03\text{mmol/L}$) and 30.3% had elevated LDL-C greater than 3.0mmol/L.

Dyslipidemia as assessed by AIP was 63.3%, when assessed by CRI-I was 52.4% and when assessed by CRI-II was 66.4%. Further analysis revealed that with respect to gender, more females had hypercholesterolemia and reduced LDL-C than males ($p=0.037$ and $p=0.010$; Table 2). This study however showed that AIP was able to delineate more males with dyslipidemia ($X^2=13.991$, $p<0.0001$; Table 3). The mean AIP was 0.12 ± 0.26 , mean CRI-I was 6.38 ± 5.33 , and mean CRI-II was 4.95 ± 4.67 . The mean AIP values in both females and males were higher than 0.1 conferring at least intermediate cardiovascular risk on the individuals.

Dyslipidemia when assessed by AIP was 63.3% and in the presence of hypertension increases the CV risk significantly. Furthermore, when AIP was used to risk-stratify the subjects; 48.1% of the male hypertensives were low risk, 23.6% were intermediate risk, and 28.3% were high risk. Over 62% of the female hypertensives were low risk, 12.7% were intermediate risk, while 24.6% of the females were high risk ($X^2=6.556$, $p=0.038$).

AIP showed an inverse correlation with HDL-C ($r = -0.447$, $p<0.0001$), LDL-C ($r = -0.130$, $p=0.045$), and TC ($r = -0.209$, $p=0.001$).

CRI-I showed an inverse correlation with HDL-C ($r = -0.404$, $p<0.001$) and a positive correlation with TC and LDL-C ($r = 0.146$, $p=0.02$) and ($r = 0.128$, $p=0.04$) respectively.

CRI-II showed a strong inverse correlation with HDL-C ($r = -0.406$, $p<0.0001$) and a positive correlation with LDL-C ($r = 0.284$, $p<0.0001$).

Using the Receiver Operating Curve (ROC), the lipid ratios were more sensitive than the individual components of the lipid profile in identifying dyslipidemia in the study population (Table 4). The CRI-II had a significant area under the curve ($AUC = 0.882$, $CI: 0.840-0.927$, $p<0.0001$). This study revealed that with a cut-off value of 0.15, CRI-II had a sensitivity of 98.9% and a specificity of 99.3% for dyslipidemia, and as such CRI-II is a better marker in identifying dyslipidemia in hypertensive, non-diabetic individuals. We also found that when also compared to the other atherogenic indices, CRI-II had the highest AUC in males ($AUC=0.884$) and females ($AUC=0.881$).

Table 2. Sex-related Prevalence of Dyslipidemia with respect to the Components of the Lipid Profile

Category	Dyslipidemia		X^2	p-value
	Yes	No		
TCH	(%)	(%)		
Males	5.7	94.3	4.363	0.037
Females	13.6	86.4		
TG				
Males	1.0	99.9	1.100	0.294
Females	2.9	97.1		
HDL-C				
Males	39.6	60.4	0.420	0.517
Females	35.6	64.4		
LDL-C				
Males	21.7	78.3	6.610	0.010
Females	37.0	63.0		

TG=Triglyceride; LDL-C=Low density lipoprotein cholesterol; HDL-C=High density lipoprotein cholesterol

Table 3. Sex-related Prevalence of Dyslipidemia with respect Lipid Ratios

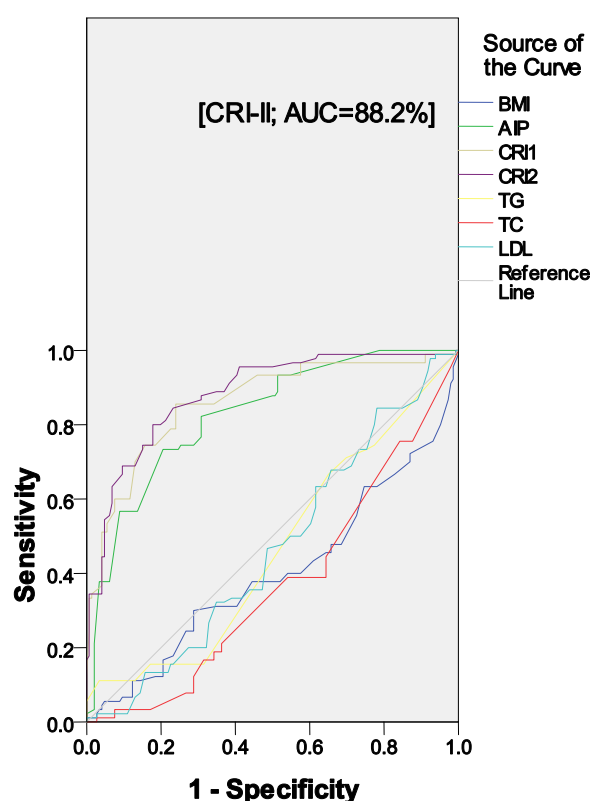
Category	Dyslipidemia		X^2	p-value
	Yes	No		
AIP	(%)	(%)		
Males	76.4	23.6	13.991	<0.0001
Females	53.0	47.0		
CRI-I				
Males	58.1	41.9	2.000	0.156
Females	48.9	51.1		
CRI-II				
Males	66.0	34.0	0.110	0.918
Females	66.7	33.3		

AIP=Atherogenic index of plasma; CRI-I=Castelli risk index-I; CRI-II=Castelli risk index-II

Table 4. Area under the Curve for Dyslipidemia in Hypertensive individuals

Variables	Area	Std. Error	p-value	Confidence Interval	
				Lower Bound	Upper Bound
BMI	0.400	0.039	0.010	0.323	0.477
AIP	0.827	0.027	<0.0001	0.774	0.880
CRI1	0.861	0.026	<0.0001	0.811	0.911
CRI2	0.882	0.023	<0.0001	0.838	0.926
TG	0.464	0.039	0.349	0.388	0.539
TC	0.369	0.037	0.001	0.298	0.441
LDL-C	0.466	0.038	0.380	0.392	0.540
HDL-C	0.016	0.012	<0.0001	0.000	1.000

BMI=Body mass index; AIP=Atherogenic index of plasma; CRI-I=Castelli risk index-I; CRI-II=Castelli risk index-II; TG=Triglyceride; LDL-C=Low density lipoprotein cholesterol; HDL-C=High density lipoprotein cholesterol.

**Figure 1.** ROC Curve for Dyslipidemia in Hypertensives subjects

4. Discussion

Lipid profile refers to some routinely done biochemical tests to assess the atherogenic status of individuals at risk of cardiovascular disease (CVD). It includes serum triglycerides (TG), serum total cholesterol (TCH) and its sub fractions like HDL-C and LDL-C. Various studies have established the role of deranged lipid profile in the progression of CVD and deranged LDL-C levels are the primary target for treatment [13]. Calculating certain ratios using these parameters may increase the identification of

at-risk individuals. Hence, this study aimed at evaluating the role and contribution of the ratios like AIP, CRI-I, and CRI-II in identifying dyslipidemia in drug-naïve newly-diagnosed hypertensive individuals.

The results of this study revealed that dyslipidemia was high in our hypertensive population. Reduced HDL-C levels were the most common form of dyslipidemia (37.3%). This is consistent with the findings by Akintunde *et al* [17] and Ojji *et al* [18], but in contrast to Ajayi *et al* [19] where elevated LDL-C was the most common lipid abnormality. This disparity in findings may be explained by the difference in the study populations. We also found that hypertriglyceridemia was the least prevalent dyslipidemic pattern. This study also revealed that hypercholesterolemia and elevated LDL-C levels were more common among the female hypertensives (See Table 2). This was consistent with the findings by Russo *et al* [20]. This sex-related difference in the level of LDL-C and TCH could be due to the fact that the female population had significantly more total body fat than their male counterparts (See Table 1). This alone could be responsible for the changes in lipid homeostasis because increased whole-body adiposity and central fat accumulation and decreased insulin sensitivity are associated with increased plasma TG and LDL-C concentrations [21] which were all evident in the female population in this study.

In this study, dyslipidemia as assessed by AIP was 63.3%, when assessed by CRI-I was 52.4% and when assessed by CRI-II was 66.4%. These values were greater than those reported by Ajayi *et al* [19] in South-western Nigeria. The importance of this finding hinges on the fact that lipid ratios have been found to indicate atherogenic risk and are said to be better predictors of coronary artery disease than lipids alone [22] because they these lipid parameters have a combination of pro-atherogenic and anti-atherogenic lipids and lipoproteins. We also found that although the mean values of AIP was comparable across gender, AIP was able to discriminate for atherogenicity in the males ($p<0.0001$). The association between TG and HDL-C reflected by AIP depicts the balance between atherogenic and protective lipoproteins. The clinical implication of sex-related difference in AIP values reported in this study is that because of statistically significant difference in HDL-C levels (See Table 1), the male hypertensive may have a higher predilection to atherosclerotic vascular disease (ASVD). This is in concert with previous studies that had shown that validated difference exists in disease presentation, progress, and outcome between the sexes, even though the mechanism(s) behind this is still unclear [23, 24]. Also, when AIP was used to risk-stratify the subjects by gender, we found that more of the male subjects were classified as high ($p=0.038$). Also, the AIP is known to have a positive correlation with the HDL esterification rate and an inverse correlation with LDL size [25]. This finding was also reported in this study, with AIP showing inverse correlations with LDL-C, HDL-C, and TCH. AIP is therefore able to define abnormal levels of HDL-C, LDL-C and TCH which also includes TG and may therefore be considered as

mirroring the derangement of spectrum of lipids.

The CRI-I and CRI-II appear to be comparable in significance in a gender-related sense (See Table 3). The role played by LDL-C in atherogenicity is well-documented. CRI-I and CRI-II are lipid ratios in which LDL-C plays a central role. So it was not surprising that CRI-I and CRI-II showed positive correlations with LDL-C ($P=0.02$ and $p<0.0001$), while showing inverse associations with HDL-C. The CRI-I appears to be as useful as CRI-II. Their similarity can be explained by the fact that approximately two-thirds of plasma cholesterol is found in LDL and, consequently, total and LDL-C are closely related [26]. It has been reported that individuals with high CRI-I or CRI-II have a greater cardiovascular risk owing to the imbalance between the cholesterol carried by atherogenic and protective lipoproteins. This may be due to an increase in the atherogenic component contained in the numerator, a decrease in the anti-atherosclerotic trait of the denominator, or both [27]. The evidence derived from the Framingham study [28] suggests that the TCH/HDL-C ratio is a more powerful coronary risk predictor than independently-used TCH and HDL-C.

We also found that when the individual lipid parameters were compared with the lipid ratios to determine their degree of discrimination for atherogenicity in the entire population, the CRI-II had the highest AUC value ($AUC=0.882$), followed by CRI-I, AIP, LDL-C, TG, TCH, BMI, and HDL-C (See Table 4 and Figure 1). We also found that when also compared to the other atherogenic indices, CRI-II had the highest AUC in males ($AUC=0.884$) and females ($AUC=0.881$). This finding highlights the excellent discriminating ability of CRI-II in identifying dyslipidemia in newly-diagnosed, drug-naïve hypertensives.

5. Conclusions

In this present study, the ROC curve analysis revealed significant AUC for the lipid ratios of TCH/HDL-C, LDL-/HDL-C and AIP. These lipid ratios were found to have high sensitivity and specificity for atherogenicity compared to absolute lipid values. Hence, the lipid ratios, especially CRI-II could be applied as a marker for dyslipidemia even in the presence of insignificant changes in the individual lipid parameters in hypertensives in the Nigerian environment.

REFERENCES

- [1] Fabris F, Zancocchi M, Bo M, Fonte G, Poli L, et al. Carotid plaque, aging and risk factors. *Stroke* 1994; 25:1133-1140.
- [2] Castelli WP, Abbott RD, McNamara PM. Summary of cholesterol used to predict coronary heart disease. *Circulation* 1983; 67: 730-734.
- [3] Castelli WP, Garrison J, Wilson PW, Abbott RD, Kalousdian S et al. Incidence of coronary artery disease and lipoprotein cholesterol levels The Framingham Study. *JAMA* 1986; 256: 2835 -2838.
- [4] Reiner Z, Catapano AL, De Backer G, Graham I, Taskinen MR, et al. ESC/EAS Guidelines for the management of dyslipidaemias: the Task Force for the management of dyslipidaemias of the European Society of Cardiology (ESC) and the European Atherosclerosis Society (EAS). *Eur Heart J* 2011; 32:1769-1818.
- [5] Hsia SH, Pan D, Berookim P, Lee ML. A population-based, cross-sectional comparison of lipid-related indexes for symptoms of atherosclerotic disease. *Am J Cardiol*, 2006; 98: 1047-1052.
- [6] Kastelein JJ, van der Steeg WA, Holme I, Gaffney M, Nilo BC, et al. Lipids, apolipoproteins, and their ratios in relation to cardiovascular events with statin treatment. *Circulation*, 2008; 117: 3002-3009. doi: 10.1161/CIRCULATIONAHA.107.713438.
- [7] Castelli WP, Abbott RD and McNamara PM. Summary estimates of cholesterol used to predict coronary heart disease. *Circulation* 1983; 67: 730-734.
- [8] Brehm A, Pfeiler G, Pacini G, Vierhapper H, Roden M. Relationship between Serum Lipoprotein Ratios and Insulin Resistance in Obesity. *Clin. Chem* 2004; 50: 2316-2322.
- [9] Sova MD. Atherogenic Index of Plasma [Log (Triglycerides/HDL Cholesterol)]: Theoretical and Practical Implications: *Clinical Chemistry* 2004; (50)7: 1113-1115.
- [10] Gaziano JM, Hennekens CH, O'Donnell CJ, Breslow JL, Buring JE. Fasting triglycerides, high density lipoprotein, and risk of myocardial infarction. *Circulation* 1997; 96: 2520-2525.
- [11] Dobiášová M, Frohlich J, Šedová M, Cheung MC, Brown BG. Cholesterol esterification and atherogenic index of plasma correlate with lipoprotein size and findings on coronary angiography. *J Lipid Res.* 2011; 52(3): 566-571.
- [12] Friedewald WT, Levy RI, Fredrickson DS. Estimation of the Concentration of Low Density Lipoprotein Cholesterol in Plasma without Use of Preparative Ultracentrifugation. *Clin. Chem* 1972; 18:499-502.
- [13] 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) Final Report. *Circulation* 2002; 106: 3143.
- [14] Raslova K, Dobiasova M, Hubacek J.A, Bencova D, Sivakova, et al. Association of metabolic and genetic factors with cholesterol esterification rate in HDL plasma and atherogenic index of plasma in a 40 years old Slovak population. *Physiol. Res.* 2011, 60, 785-795.
- [15] Akbas E.M, Timuroglu A, Ozcicek A, Ozcicek F, Demirtas L, et al. Association of uric acid, atherogenic index of plasma and albuminuria in diabetes mellitus. *Int. J. Clin. Exp. Med.* 2014, 7, 5737-5743.
- [16] Zhang X-H, Zhang M, He J, Yan Y-Z, Ma J-L, et al. Comparison of Anthropometric and Atherogenic Indices as Screening Tools of Metabolic Syndrome in the Kazakh Adult Population in Xinjiang. *Int. J. Environ. Res. Public Health* 2016, 13, 428; doi: 10.3390/ijerph13040428.

- [17] Akintunde AA, Ayodele EO, Akinwusi OP, Opadijo GO. Dyslipidemia among newly-diagnosed hypertensives: Pattern and clinical correlates. *J Natl Med Assoc* 2010; 102: 403-407.
- [18] Ojji DB, Ajayi SO, Mamven MH, Antherton J. Prevalence of dyslipidemia in normoglycemic subjects with newly diagnosed high blood pressure in Abuja Nigeria. *J Clin Lipid* 2009; 3: 51-56.
- [19] Ajayi EA, Ajayi AO, Adeyeye VO. Pattern of abnormal serum lipids and lipoprotein ratios in Nigerian Hypertensive patients seen in clinical practice. *BJMMR* 2015; 6(5): 500-508.
- [20] Russo G, Pintauro B, Giorda C, Lucisano G, Nicolucci A, et al. Age- and Gender-Related Differences in LDL-Cholesterol Management in Outpatients with Type 2 Diabetes Mellitus. *International Journal of Endocrinology* Volume 2015, Article ID 957105, 8 pages <http://dx.doi.org/10.1155/2015/957105>.
- [21] Ferrannini E, Balkau B, Coppock SW, Dekker JM, Mari A, et al. Insulin resistance, insulin response, and obesity as indicators of metabolic risk. *J Clin Endocrinol Metab* 2007; 92: 2885-2892.
- [22] Nimmanapalli HD, Kasi AD, Devapatla PK, Nuttakk V. Lipid ratios, atherogenic coefficient and atherogenic index of plasma as parameters in assessing cardiovascular risk in type 2 diabetes mellitus. *Int J Res Med Sci.* 2016 Jul; 4(7): 2863-2869.
- [23] Hendel RC, Chen MH, L'Italien GJ, Newell JB, Paul SD, et al. Sex differences in perioperative and long-term cardiac event-free survival in vascular surgery patients. An analysis of clinical and scintigraphic variables. *Circulation.* 1995; 91: 1044-1051.
- [24] Roger VL, Go AS, Lloyd-Jones DM, Adams RJ, Berry JD, et al; American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Heart disease and stroke statistics-2011 update: a report from the American Heart Association. *Circulation.* 2011; 123: e18-e209.
- [25] Dobiasova M, Frolich J. The plasma parameter log (TG/HDL-C) as an atherogenic index: correlation with lipoprotein particle size and esterification rate in apoB-lipoprotein-depleted plasma (FERHDL). *Clin Biochem.* 2001; 34: 583-588.
- [26] Millán J, Pintó X, Muñoz A, Zúñiga M, Rubiés-Prat J, et al. Lipoprotein ratios: Physiological significance and clinical usefulness in cardiovascular prevention. *Vascular Health and Risk Management* 2009; 5 757-765.
- [27] Criqui MH, Golom BA. Epidemiologic aspects of lipid abnormalities. *Am J Med.* 1998; 105(Suppl 1A):48S-57S.
- [28] Castelli WJ, Garrison RJ, Wilson PWF, Abbot RD, Kaulousian S, et al. Incidence of coronary heart disease and lipoprotein cholesterol levels. The Framingham Study. *JAMA.* 1986; 256: 2835-2838.