Original Article

A Pilot Assessment of Dyslipidaemia and Risk of Arteriosclerosis among Men in Oredo Local Government Area of Edo State, Nigeria

Idemudia, JO[1]. and Atoe K [2,3]

[1]Department of Chemical Pathology, University of Benin Teaching Hospital, Benin City, Nigeria.
[2]Department of Chemical Pathology, College of Medical sciences, Edo University Iyamho, Edo State.
[3]Applied Environmental Biosciences and Public Health Research Group, Department of Microbiology, University of Benin, Benin City.

* ***Correspondence***: *Dr Idemudia JO. Department of Chemical Pathology, University of Benin Teaching Hospital, Benin City, Nigeria.* ***E-mail***: *osagiep2001@yahoo.com*

For reprints contact: *chsjournals@gmail.com or editor@jrbcs.org*

ABSTRACT

The burden of dyslipidemia in Nigeria has been poorly characterized. This metabolic abnormality leads to a persistent increase of plaques in the arteries, which eventually narrows or blocks the arteries, thereby leading to cardiovascular anomalies.This cross-sectional study, which was carried out during a medical outreach programme for male individuals resident within Oredo Local Government Area of Edo State, Nigeria, was set out to investigate the prevalence of dyslipidaemia. Results showed that 45out of 156 participants (28.85%) recruited for the study had a greater artherogenic coefficient-determined cut off value of 4.28, and as such were thought to be at the risk of developing atherosclerosis. The age group mostly implicated in the development of arteriosclerosis was the 40 - 49 years category. The study further showed relevance of total cholesterol, triglycerides, low density lipoprotein cholesterol, high density lipoprotein cholesterol and Castelli Risk Indices as indicators for the prevalence of dyslipidemia ($p<0.001$). It is suggested therefore that individuals above 30 years of age should be evaluated regularly for lipid abnormalities as this may promote early diagnosis of lipid abnormalities.

Keywords: *Artherosclerosis, Cholesterol, Cardiovascular, Dyslipidaemia.*

INTRODUCTION

Currently, the increasing prevalence of dyslipidaemia is a worldwide public health concern.[1]In Nigeria, the frequency of stroke-related hospitalization caused by dyslipidaemia ranges from 1 to 4 %, 0.5 to 45% of neurological admissions[2]and about 4% of emergency medical admission. It was found to be the eighth leading cause of death.[3] Formerly, dyslipidaemia was thought to be uncommon among black people compared to the western world but recent reports have it that this disease is highly prevalent in Nigeria with a consistent pattern of low high density lipoprotein cholesterol (HDL-C) and elevated low density lipoprotein cholesterol (LDL-C).[4] Dyslipidaemia is the presence of abnormal levels of lipids or lipoproteins in the blood, which includes elevated total cholesterol (TC), triglycerides (TG), andLDL-C.[5]Elevatedserum levels of LDL-C is a major risk factor for cerebral infarction. This promotes the formation of atherosclerosis.[6] Apart from hypertension, diabetes mellitus, physical inactivity, carotid stenosis, and transient ischemic attack, dyslipidaemia is also adaptable risk factors that predispose to stroke.[7] Dyslipidaemia is one of the most important risk factors for developing atherosclerosis, which itself is the most important pathologic incident among cardiovascular (CV) diseases.[8,9]It is a manifestation that arises as a result of

anomalies in the level of plasma lipids.[10] This condition is a leading contributor to cardiovascular diseases and mortality globally.[11]Atherosclerosis, which is a condition in which an artery wall thickens because of buildup of fatty materials, is initiated by the inflammatory reaction to dyslipidemia.[12] In 2016, the Global Burden of Disease study reported that high concentrations of total cholesterol caused about 4.4 million deaths and 93.8 million Disability-Adjusted Life Years (DALYs).[1]This represented the seventh and eighth leading risk factor in terms of attributable DALYs globally for women and men, respectively.[13]Definite indication exists that effective treatment of dyslipidaemia will evidently reduce cardiovascular disease (CVD) morbidity and premature mortality.[14,15]

Atherosclerosis causes severe stenosis in arteries, leading to stroke, and many other fatal clinical expressions[16] sPrior to becoming clinically evident, atherosclerosis progresses silently for years. Medical personnel therefore must not only address clinical manifestations of the disease but also identify targets for prevention and early treatment in individuals that may be incubating this deceptive disease. Both these tasks require an understanding of the pathogenesis of atherosclerosis.[16] Dyslipidaemia often coexists with diabetes mellitus (DM), hypertension and obesity.[17,18,19,20] This makes the management of dyslipidaemia crucial in attempting to decrease cardiovascular risk. The absolute risk associated with dyslipidemia rises substantially with advancing age.[21]Aging is associated with undesirable changes in body composition that expose older adults to a host of metabolic complications. It is well known that body fat increases with age and is preferentially accumulated in the abdominal region, thereby increasing the risk of cardiovascular diseases and diabetes in older adults.[19]Elevatedcholesterol levels in early adulthood especially in men increase the risk of developing coronary heart disease later in life.[22]

Cardiovascular disease mortality rates are well known to be lower in women than men and to increase with age[23] A report on gender differences in coronary heart disease and mortality assessment of the effect of ageing between 1980 and 2010 provided evidence that coronary heart disease mortality rates were consistently higher in men than in women, though the magnitude of the ratio varied by age.[23]Thus men were assessed in the present study for risk to atherosclerosis. It is also often thought that cardiovascular disease develops 7 to 10 years later in women than in men.[24] This thought is based on the assumption that exposure to endogenous estrogens during the fertile period of life delays the manifestation of atherosclerotic disease in women.[25] In Nigeria, there have been several assessments of lipid levels among patients in health centers across various communities[4,9,26,27] but the rate of occurrence of dyslipidaemia and risk of arteriosclerosis especially among the male population continue to increase with the growing population, sedentary lifestyle and unhealthy dietary habits of most Nigerian people. The aim of this study therefore was to assess the prevalence of dyslipidaemia in a population of men without clinical symptoms so as to possibly prevent the onset of cardiovascular diseases and reduce the growing mortality trend caused by these diseases..

PARTICIPANTS AND METHODS

A total of 156 male participants were recruited into this study during a medical outreach programme organized in Oredo Local Government Area, Benin City, Edo state, Nigeria. Participant weights were measured in light indoor clothing without shoes, using a calibrated balance beam scale and height measured using a calibrated standiometer. BMI was calculated as body weight in kilogram divide by height in meters square (Kg/M^2). BMI was categorized in the study as underweight (<18 kgm^{-2}), normal (18 - 24.9 kgm^{-2}), overweight (25 – 29.9 kgm^{-2}), mild obese (30 – 34.9kgm^{-2}), moderate obese (35 – 39.9kgm^{-2}), and severe obese (>40kgm^{-2}). Blood pressure of participants was taken in the sitting position with a mercury sphygmomanometer after 10 minutes rest. Venipuncture was done and about 5mls of blood was collected and shared into an EDTA and fluoride oxalate containing tubes. The whole blood was centrifuged and the plasma collected into 5mls plain tubes with a Pasteur pipette and stored at about -20^{o}C until they were subjected to biochemical analysis. Glucose oxidase method previously used by Alikor *et al*. (2015)[28]was used in this study to detect the fasting plasma Glucose. The lipid analysis was done using spectrophotometer, the reagents were manufactured by Randox Laboratory Limited and the standard operating

assay procedure was employed. The blood lipid indexes were calculated using the following equations;

Friedewald equation for LDL Chol. = TC – TG/5 – HDL Chol. (mg/dL)

$$\text{Atherogenic index of plasma, AIP} = \text{Log}\ \frac{\text{TG}}{\text{HDLChol.}}$$

$$\text{Atherogenic coefficient, AC} = \frac{\text{Non HDL Chol}}{\text{HDL Chol.}}$$

Non-HDL cholesterol, NHDL = Total Chol – HDL Chol

$$\text{Very low density lipoprotein, VLDL} = \frac{\text{TRIGLYCERIDES}}{5}$$

$$\text{Castelli Risk Index I,(CRI I)} = \frac{\text{TC}}{\text{HDL Chol.}}$$

$$\text{Castelli Risk Index II, (CRI II)} = \frac{\text{LDLC}}{\text{HDLC}}$$

Data obtained in the study were subjected to statistical analyses using SPSS® version-21. Results were presented in percentages as well as mean of replicated data. Correspondence analyses was used to show association between selected parameters and the participant lipid status..

RESULTS

The mean age of the study group was 42yrs, with a mean height of 1.725m and a body mass index (BMI) of 26.4 (Table 1). The mean blood pressure of the study group was around the region of 122/80 mm Hg. As per BMI categorization, 3 out of 156 were underweight representing 1.92%, whereas 60 (38.5%) were of the normal category, while 25.64% were generally obese and 33.97% were overweight. The blood pressure of the study group increased significantly ($p<0.001$) with age from 117/73 mmHg in the 20 – 29 year to 149/93 mmHg beyond 60yrs. However, the fasting Plasma Glucose (FPG) of the study participants did not significantly Sinfluence their respective blood pressures ($p>0.05$). Table 2 presents the biochemical characteristics of the study group on the basis of BMI, age and FPG respectively. FPG increased with BMI ($p<0.05$) from 81mg/dL in the underweight to 114 mg/dL in the severely obese individuals.FPG was higher in the aged (106.8 mg/dL) than in the younger (90.2 mg/dL) ($p<0.05$). However, total cholesterol, triglyceride concentration, HDL cholesterol, LDL cholesterol as well as very low density lipoprotein cholesterol concentrations were not significantly separated on the basis of ages of the respondents (Table 2). Atherogenic index of plasma (AIP), Atherogenic coefficient (AC), as well as the Castelli Risk Indices (CRI)were used in this study as the risk-determining ratios for dyslipidemia and arteriosclerosis (Table 3). Mean AIP was 0.05, whereas CRI ranged from 4.28 – 4.38.There were minimal decreases in AC and CRI with increasing BMI ($p<0.05$). However, these ratios did not significantly change within the group when the respondents were reordered on the basis of age.

Table 1: Physical characteristics of the study group

	Age (yrs)	Height (m)	Weight (kg)	BMI	SBP (mm Hg)	DBP (mm Hg)
Statistical Summary (n=156)						
Mean	42.4	1.725	76.2	26.4	122.1	80.46
Std. Deviation	9.87	0.081	14.7	6.69	21.69	15.36
Distribution by BMI						
Underweight (n= 3)	34	1.7	52.0	-	116.7	73.33
Normal (n= 60)	38.95	1.73	65.22	-	117.3	77
Overweight (n= 53)	43.868	1.74	79.09	-	126.2	82.47
Mild obesity (n= 30)	45.8	1.72	88.26	-	122.3	81.33
Moderate obesity (n= 4)	43.5	1.68	89.5	-	132.5	90
Severe obesity (n= 6)	51.167	1.69	103.2	-	126.7	90
p-value	0.001	0.651	<0.001	-	0.292	0.126
Distribution by Age						
20 - 29yrs (n= 14)	-	1.73	64.92	23.2	117	73
30 - 39yrs (n= 43)	-	1.74	74.37	25.5	114	74
40 - 49yrs (n= 69)	-	1.72	78.35	26.8	122	82
50 - 59yrs (n= 21)	-	1.75	83.18	29	129	87
>60 yrs (n= 9)	-	1.68	71.63	26.6	149	93
p-value		<0.001	0.02	<0.001	0.28	<0.001
Distribution by Fasting Plasma Glucose						
Normal (n=119)	41.7	-	-	25.9	123	81
Impaired (n=25)	44.7	-	-	27.99	116	78
Diabetic (n=12)	44.8	-	-	28.16	120	83
p-value	0.27	-	-	0.237	0.3	0.5

Table 2: Biochemical characteristics of the study group

	FPG (mg dl-1)	CHOL	TRYG	HDL	LDL	NHDL	VLDL
Statistical Summary (n=156)							
Mean	95.33	169.8	112	41.5	105	128.3	22.4
Std. Deviation	17.85	35.33	89.24	12.51	37.81	36.44	17.8
Distribution by BMI							
Underweight (n= 3)	81.03^{a}	162.5^{ab}	120.4^{a}	30.70^{a}	107.7^{a}	131.77^{a}	24.08^{a}
Normal (n= 60)	94.12^{ab}	163.6^{ab}	101.8^{a}	42.30^{a}	98.18^{b}	121.25^{ab}	20.36^{a}
Overweight (n= 53)	93.99^{ab}	177.9^{a}	114.9^{a}	39.25^{a}	118.09^{a}	138.63^{a}	22.97^{a}
Mild obesity (n= 30)	99.20^{ab}	178.1^{a}	105.2^{a}	44.61^{a}	108.8^{ab}	133.52^{a}	21.03^{a}
Moderate obesity (n= 4)	84.88^{a}	131.9^{b}	227.9^{b}	33.48^{a}	53.08^{c}	98.45^{b}	45.49^{a}
Severe obesity (n= 6)	114.1^{b}	148.7^{b}	142.5^{a}	48.42^{a}	71.72^{bc}	100.23^{b}	28.50^{a}
p-value	0.034	0.019	0.134	0.087	0.001	0.018	0.134
Distribution by Age							
20 - 29yrs (n= 14)	90.2^{a}	156.1^{a}	84.95^{b}	38.06^{a}	100.2^{a}	118.9^{a}	17.0^{a}
30 - 39yrs (n= 43)	90.03^{a}	166.9^{a}	103.6^{ab}	40.23^{a}	104.7^{a}	125.8^{a}	20.7^{a}
40 - 49yrs (n= 69)	97.08^{a}	171.8^{a}	112.3^{ab}	42.46^{a}	103.3^{a}	129.4^{a}	22.5^{a}
50 - 59yrs (n= 21)	100.1^{a}	184.2^{a}	141.3^{a}	43.71^{a}	118.5^{a}	140.5^{a}	28.3^{a}
60 - 69yrs (n= 9)	136.8^{b}	168.5^{a}	138.1^{a}	40.74^{a}	103.5^{a}	127.8^{a}	23.6^{a}
p-value	0.002	0.066	0.137	0.601	0.843	0.385	0.62
Distribution by FPG							
Normal (n=119)	-	169.24^{a}	106.12^{a}	40.82^{a}	107.12^{a}	128.40^{a}	21.22^{a}
Impaired (n=25)	-	172.65^{a}	109.20^{a}	42.89^{a}	103.68^{a}	129.75^{a}	21.84^{a}
Diabetic (n=12)	-	169.67^{a}	176.48^{b}	45.19^{a}	86.15^{a}	124.47^{a}	35.29^{a}
p-value	-	0.909	0.032	0.431	0.185	0.918	0.032

NHDL non-high density lipoprotein cholesterol, VLDL very low density lipoprotein cholesterol, CHOL Total cholesterol, TRYG Triglycerides. Means within each category qith the alphabetic superscript on same column do not differ from each other (p>0.05)

Table 3: Risk determining ratios for dyslipidemia and arteriosclerosis among the study group

	AIP	AC	CRI.I	CRI.II
Statistical Summary (n=156)				
Mean	0.05	3.38	4.38	2.765
Std. Deviation	0.02	1.39	1.39	1.305
Distribution by BMI				
Underweight (n= 3)	0.07[a]	4.59[a]	5.59[a]	3.83[a]
Normal (n= 60)	0.05[a]	3.17[a]	4.17[a]	2.56[a]
Overweight (n= 53)	0.05[a]	3.73[a]	4.73[a]	3.17[a]
Mild obesity (n= 30)	0.05[a]	3.38[a]	4.38[a]	2.74[a]
Moderate obesity (n= 4)	0.07[a]	3.56[a]	3.86[a]	1.56[a]
Severe obesity (n= 6)	0.05[a]	3.17[a]	3.77[a]	2.62[a]
p-value	0.062	0.061	0.072	0.054
Distribution by Age				
20 - 29yrs (n= 14)	0.053[a]	3.29[a]	4.29[a]	2.79[a]
30 - 39yrs (n= 43)	0.053[a]	3.48[a]	4.48[a]	2.89[a]
40 - 49yrs (n= 69)	0.051[a]	3.34[a]	4.34[a]	2.66[a]
50 - 59yrs (n= 21)	0.049[a]	3.52[a]	4.52[a]	2.99[a]
60 - 69yrs (n= 9)	0.055[a]	3.27[a]	4.27[a]	2.64[a]
p-value	0.273	0.94	0.87	0.77
Distribution by FPG				
Normal (n=119)	0.052[a]	3.45[a]	4.45[a]	2.87[a]
Impaired (n=25)	0.049[a]	3.27[a]	4.27[a]	2.63[a]
Diabetic (n=12)	0.050[a]	2.89[a]	3.89[a]	2.03[a]
p-value	0.07[a]	4.59[a]	5.59[a]	3.83[a]

AIP artherogenic index plasma, AC artherogenic coefficient, CRI Castelli Risk Index I and II, Mean ratios within each category qith the alphabetic superscript on same column do not differ from each other (p>0.05)

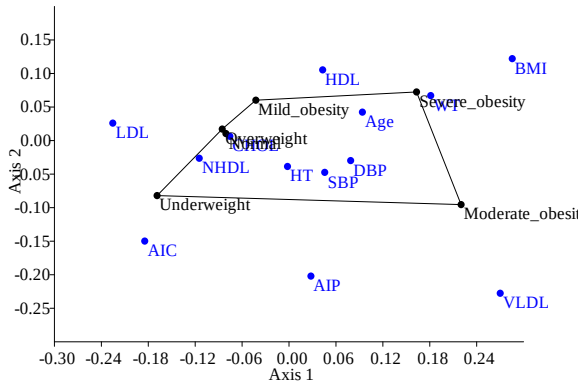

Figure 1: Correspondence analyses showing association between parameters and participant BMI status.

keys; *AIP artherogenic index of plasma, AC artherogenic coefficient, NHDL non-high density lipoprotein cholesterol, VLDL very low density lipoprotein cholesterol, LDL low density lipoprotein, CHOL cholesterol, ht height, BMI body mass index, HDL high density lipoprotein HDL, SBP systolic blood pressure, DBP diastolic blood pressure.*

A correspondence analyses showing association between parameters and participant BMI status have been presented on Fig. 1. There was association between total cholesterol concentration and the overweight group. Similarly, the closest association between the measured parameters and the BMI classes was that between weight and severe obesity. Being a factor of BMI, weight was more related to any of the BMI classes as presented in Fig. 1 than height. This therefore underscores the significance of weight in the determination of BMI as a risk factor for heart-related ailments, rather than height. Similarly, when the correspondence analyses was presented to show association between the measured biochemical characteristics of the study group against their age classes, BMI was seen to be most associated with the 40 – 49yrs age category more than weight was associated with the 30-39yrs category (Fig. 2). The close relatedness between the 30-39yr and 40-49yr categories have been further shown in the dendrogram from cluster analysis that showed similarities on the basis of age (Fig. 3).Euclidean distances were used to show similarity and distance indices among the three classes based on FPG (Table 4a). The distance between normal and impaired

group, determined from a conglomeration of all physical and biochemical parameters, was 13.3, compared to 75.2 which was the distance between normal and diabetic classes.

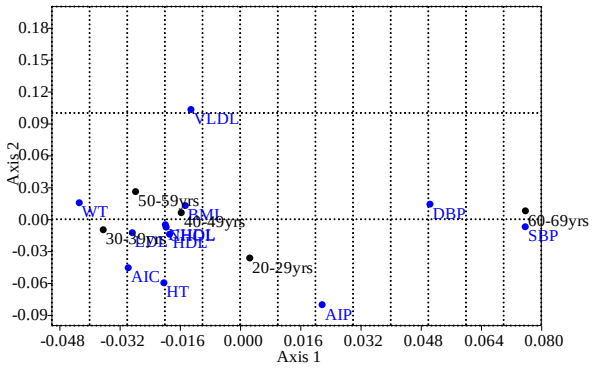

Figure 2: Correspondence analyses showing association between parameters and participant age grouping.

keys; *AIP artherogenic index of plasma, AC artherogenic coefficient, WT weight, NHDL non-high density lipoprotein cholesterol, VLDL very low density lipoprotein cholesterol, LDL low density lipoprotein, CHOL cholesterol, HT height, BMI body mass index, HDL high density lipoprotein HDL, SBP systolic blood pressure, DBP diastolic blood pressure*

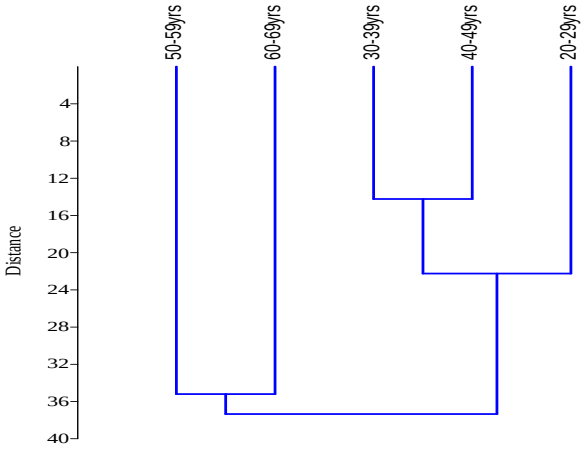

Figure 3: Dendrogram from cluster analysis showing similarity and distance indices (Euclidean distances provided) on the basis of age.

Table 4a: Similarity and distance indices (Euclidean distances provided)

	Normal	Impaired	Diabetic
Normal	0	13.3	75.2
Impaired	13.3	0	71.7
Diabetic	75.2	71.7	0

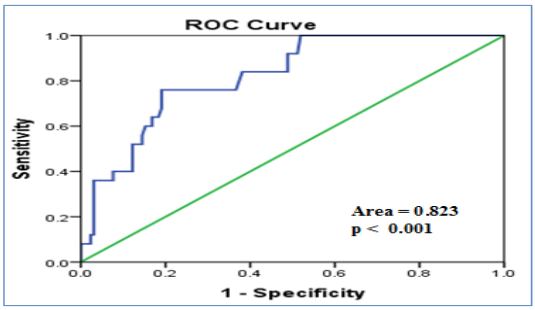

Fig. 4: Reciever Operator Curve for Artherogenic Coefficient as a predictor of artherosclerosis

Table 4b : Coordinates of the receiver operator characteristic (ROC) Curve

Coordinates of the Curve		
Positive if Greater Than or Equal To	Sensitivity	1 – Specificity
3.5400	0.760	0.321
3.5900	0.760	0.313
4.2000	0.760	0.206
4.2150	0.760	0.198
4.2750	0.760	0.191
4.3450	0.720	0.191
7.2250	0.040	0.000
8.2700	0.000	0.000

An attempt was made to determine cut-off for atherogenic coefficient (AC) using cholesterol levels of the study group since this analyte was used in the computation of AC. An ROC curve was presented (Fig. 4) with an area under the curve of 0.823 ($p<0.05$). The cut-off extrapolated was 4.275. On the basis of this cut-off, the 156 study participants were classed into normal (lower AC) and abnormal (>AC) (Table 5). A total of 45 out of 156 participants had an AC value greater than cut-off and as such were thought to be at the risk of developing atherosclerosis. The age group that was mostly implicated in the development of arteriosclerosis was those between 30 and 50 years old, accounting for a total of 61% of cases attributed to abnormal AC. This further strengthens the observation on Fig. 3 above. Prediction of atherogenic coefficient using BMI personated a sensitivity value of 40.4% and specificity of 64.3%. Reliance on total cholesterol presented a 93.0% sensitivity but a 40.5% specificity (Table 7).

Table 5: Distribution atherogenic coefficients of participants by age categories.

			Age Category					Total
			20-29yrs	30-39yrs	40-49yrs	50-59yrs	>60yrs	
AIC	Normal	N	11	29	49	14	8	111
		(%)	78.60	67.40	71.00	66.70	88.90	71.20
	Abnormal	N	3	14	20	7	1	45
		(%)	21.40	32.60	29.00	33.30	11.10	28.80
Total		Count	14	43	69	21	9	156

Table 6: Distribution on the basis of atherogenic coefficient

	Age	BMI	SBP	DBP	FPG	CHL	TRYG	HDL	LDL	AIP	CRI.I	CRI.II	NHDL	VLDL
Normal (n=111)	43	26.6	123	81	95.7	158.9	109.6	45.16	91.55	0.046	3.67	2.14	113.7	21.9
Abnormal (n=45)	42	25.8	120	79	94.5	196.7	118	32.46	138	0.064	6.13	4.31	164.3	23.6
p-value	0.6	0.49	0.56	0.3	0.72	<0.001	0.599	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.6

AIP artherogenic index plasma, AIC artherogenic coefficient, CRI Casterlly Risk Index I and II, NHDL non-high density lipoprotein cholesterol, VLDL very low density lipoprotein cholesterol, CHOL Total cholesterol, TRYG Triglycerides, SBP systolic blood pressure, DBP diastolic blood pressure, FPG fasting blood glucose.

Table 7: Prediction of atherogenic coefficient using BMI, SBP, DBP, and CRI values

	SBP	DBP	Total cholesterol	HDL
Sensitivity (%)	40.4	60.5	93.0	38.6
Specificity	64.3	31.0	40.5	54.8
PPV	75.41	70.41	80.91	69.84
NPV	28.42	22.41	68.0	24.73
Disease prevalence	73.08	73.08	73.08	73.08
Likelihood Ratio	0.279	0.971	22.69	0.559
	(p = 0.57)	(p = 0.32)	(p <0.001)	(p=0.455)
Fisher's Exact Test	p = 0.71	p = 0.37	p <0.001	p=0.468
Linear-by-Linear Association	0.275	0.948	25.37	0.559
	(p = 0.60)	(p = 0.33)	(p <0.001)	(p=0.455)

HDL high density lipoprotein, SBP systolic blood pressure, DBP diastolic blood pressure, NPV negative predictive value, PPV positive predictive value

DISCUSSION

There are generally several indicators for ascertaining the level of risk of seemingly healthy individuals to dyslipidaemia and atherosclerosis. Many people between 35 and 50 years of age are moderately at risk of dyslipidaemia.[29] This risk is usually further modulated by obesity. Subjects in this study were on the average in the35 to 50 years category and overweight based on the mean BMI of the study population.

It is often thought that total cardiovascular risk estimation means the likelihood of a person developing an atherosclerotic cardiovascular event over a defined period of time[30] However, there is no ideal target blood level for LDL-C. The 2018 guidelines on the management of blood cholesterol recognizes that total cholesterol below 150mg/dL and LDL-C lower than 100 mg/dL is better.[31] Adults with LDL-C at 100 mg/dL have lower rates of heart disease and stroke.[29]Findings from the study population suggests total cholesterol levels higher than the 150mg/dL and LDL levels higher than 100mg/dL. This trend was observed in participants between the ages of 40-69 years. It may be inferred therefore that individuals within this age bracket are more at risk of heart disease. Although on the basis of BMI, the moderately obese and severely obese

participants had lower levels of total cholesterol and LDL-C (Table 2). Some evaluators opined that age was the most prominent factor in the prevalence of cardiovascular issues.[1,11,32] This probably ranks this study participants above 39 years at risk of atherosclerosis. However, the report of Shawar *et al*. 2012 showed a relatively high percentage of hypercholesterolemia among young university students of ages 16-30 years in Arabian Gulf University.[21] The early hypercholesterolemia observed by Shawar and colleagues may have been contributed by other factors like obesity, fatty diet and the epigenetics of their target subjects.

The present study provided evidence of close similarity among participants that were between the ages of 30 and 49years (Figure 3). There were very close association between these two age brackets with respect to most of the parameters evaluated (Figure 2). This may be because most of the participants were between the ages of 30 and 49 years and people within this age bracket generally have similar life styles. A cross sectional research of the prevalence of dyslipidaemia carried out in China stated that the peak prevalence in men was among those within 30 and 39 years old (48.2 %) though, declined gradually with increasing age.[36]

This research study group was also characterized by fasting plasma glucose (FPG). About 119 participants were normal but had high total cholesterol (TC) and LDL-C (169.24 and 107.12mg/dL respectively). This was similar to the TC and LDL-C levels in the participants with impaired fasting glucose. The similarity and distance indices of impaired and normal participants was 13.3 compared to the 75.2 difference between normal and diabetics (Table 4). The implication is that the normal group was more closely related than they were with the diabetics. The diabetics were also not related with the impaired group. This further strengthens the argument that an impaired individual was more likely to become normal upon simple positive life changes than become diabetic.

A study done by kesteloot and others in Benin, Nigeria[35] reported low prevalence of dyslipidemia among black people. There have recently been reports of 59.3% prevalence of dyslipidemia in Katstina, northern Nigeria [26] and 60% in Owerri, also in Nigeria.[35]These reports show patterns of consistently low HDL-C and high LDL-C. There were reports of elevated cholesterol of 31.7% among another study population in the southern part of Nigeria.[27]This trend was similar to what was observed in the present study after the determination of cut off for atherogenic coefficient (AC) using cholesterol levels. There was 28.8 % risk of developing atherosclerosis in this study based on age distribution (see Table 5). These 28.8% were regarded as abnormal based on the AC and they had very high TC level of 196.7mg/dL and high LDL-C level of 138mg/dL both significant at $P < 0.001$ (see Table 6).

Lipid ratios such as CRI,AC and the AIP are diagnostic alternatives shown to predict the risk of cardiovascular events.[36]In this study, minimal decrease ($p>0.05$) was observed in the AC and CRI with increasing BMI; the ratios also did not significantly change within the group when the respondents were examined on the basis of age. The study of Olamoyegun and colleagues in 2016 reported that the CRI predicted the highest prevalence of predisposition to cardiovascular diseases at 47.8%[9]. The present study recommends that high risk individuals; men between the ages of 35-50 years and overweight persons require regular screening for lipid abnormalities and where necessary therapy initiated.

CONCLUSION

The possibility for prediction of atherogenic coefficient using BMI, SBP, DBP, and CRI values were determined. These parameters either presented a weak sensitivity or specificity or both, and as such were not good instruments for predicting AC of individuals. The results in this study also highlighted the need for extensive routine screening for dyslipidaemia among those at risk of atherosclerosis. Regular screening will help to provide status and risk factors on dyslipidaemia and atherosclerosis, promote early diagnosis, therapy and ultimately reduce the occurrence of cardiovascular diseases.

Conflict of Interest

None declared

REFERENCES

1. Noubiap, J. J., Bigna, J. J., Nansseu, J. R., Nyaga, U. F., Balti, E. V., Echouffo-Tcheugui, J. B., Kengne, A. P. Prevalence of dyslipidaemia among adults in Africa: A systematic review and meta-analysis. Lancet Glob Health, 2018;6: e998–1007.
2. Ojini, F. I. and Danesi, M. A. Pattern of neurological admissions at the Lagos University Teaching Hospital. Nigeria Journal of Clinical Practice, 2013; 5(1): 38 - 41.
3. World Health Organisation, WHO. (2002).Country mortality Fact Sheet 2006 Nigeria. Death and DALY estimates by cause, Available at http://www.who.int/entity/healthinfo/statistics/bodgbddeathdalyestimates.xis Accessed on March 10, 2015.
4. Oguejiofor, O. C., Onwukwe, C. H. and Odenigbo, C. U. Dyslipidemia in Nigeria: prevalence and pattern. Annals of African Medicine, 2012; 11(4): 197-202.
5. Mohiuddin, S. M., Pepine, C. J., Kelly, M. T.,Buttler S.M., Setze, CM.,Sleep.D.J. et al. Efficacy and safety of ABT-335 (fenofibric acid) in combination with simvastatin in patients with mixed dyslipidaemia: a phase 3, randomized, controlled study. American Heart Journal, 2009;157(1):195-203.
6. Paciaroni, M., Hennerici, M., Angelli, G . Bogousslavsky J. Statin and stroke prevention. Cerebrovascular disorders, 2007;24(2-3):170-182.
7. Goldstein, L. B., Bushnell, C. D., Adams, R. J, Appel, L. J, Braun, L.T., Chaturvedi, S. *et al*. Guidelines for the primary prevention of stroke: A guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke.2011; 42(2):517-584.
8. Xing, X. Y., Li, G. W., Yao, C. H. *et al*. The association of stroke high plasma low density lipoprotein cholesterol level and metabolic syndrome in Chinese adults. Chinese Journal Interntionl Medicine, 2009;48(5):388-391.
9. Olamoyegun, M. A., Oluyombo, R. and Asaolu, S. O. Evaluation of dyslipidemia, lipid ratios and atherogenic index as cardiovascular risk factors among semi- urban dwellers in Nigeria. Annals African medicine, 2016; 5(4): 194-199.
10. Garces, M. F., Guarin, Y., Carrero, Y., Stekman, H., Hernández, C.,Núñez, M. L., Apitz, R. and Camejo, G. Fat intolerance in apparently healthy individuals with normal fasting lipoproteins is associated with markers of cardiovascular risk. JALM, 2016; 1(3): 250–259.
11. Murray, C.J., Vos, T., Lozano, R., Naghavi, M., Flaxman, A.D., Michaud, C., Ezzati, M., Shibuya, K., Salomon, J.A., Abdalla, S., et al. Disability adjusted life years (DALYs) for 291 diseases and injuries in 21 regions, 1990 - 2010: a systematic analysis for the global burden of disease study 2010. Lancet, 2012;380: 2197–2223.
12. Dessi, M., Noce, A., Bertucci, P., Villahermosa, S. M., Zenobi, R., Castagnola, V., Addessi, E. and Daniele, N. D. Atherosclerosis, Dyslipidemia, and Inflammation: The significant role of polyunsaturated fatty acids. ISRN Inflammation, 2013;191823:13pp. http://dx.doi.org/10.1155/2013/191823
13. Noubiap, J. J, Nansseu, J. R., Bigna, J. J., Jingi, A. M., Kengne, A. P. Prevalence and incidence of dyslipidaemia among adults in Africa: A systematic review and meta-analysis protocol. BMJ Open, 2015;5: e007404.
14. Robinson, J. G., Smith, B., Maheshwari, N. *et al*. Pleiotropic effects of statins: benefit beyond cholesterol reduction? J Am CollCardiol, 2005; 46: 1855 – 1862.
15. Collins, R., Reith, C., Emberson, J., *et al*. Interpretation of the evidence for the efficacy and safety of statin therapy. Lancet, 2016;388: 2532–2561.
16. Libby, P., Lichtman, A. H. and Hansson, G. K.Immune effector mechanisms implicated in atherosclerosis: From mice to humans Immunity, 2013;38: 1092- 1104.
17. Fletcher GF, Balady G, Blair SN. Statement on exercise: Benefits and recommendations for physical activity programs for all Americans. A statement for health professionals by the Committee on Exercise and Cardiac Rehabilitation of the Council on Clinical Cardiology, American Heart Association. Circulation.1996;94(4):857–862.

18. Knoops, K. T., de Groot, L. C., Kromhout, D., *et al*. Mediterranean diet, lifestyle factors, and 10-year mortality in elderly European men and women: the HALE project. JAMA.2004; 292 (12):1433–1439.
19. Shanmugasundaram, M., Rough, S. J., Alpert, J. S. Dyslipidemia in the elderly: Should it be treated? Clinical Cardiology, 2010;33: (1) 4–9.
20. Chang, Y., Kim, B., Yun, K. E., Cho, J., Zhang, Y., Rampal, S., Zhao, D., Jung, H., Choi, Y., João, J. A., Lima, A. C., Shin, H.,Guallar, E. and Ryu, S. Metabolically-healthy obesity and coronary artery calcification. Journal of the American College of Cardiology, 2014;63(24): 2679–2686.
21. Shawar, S. M., Al-Bati, N. A., Al-Mahameed, A., Nagalla, D. S., Obeidat, M.Hypercholesterolemia among apparently healthy university students. Oman MedicalJournal, 2012;27(4): 274-280.
22. Lee, J. S., Chang, P., Zhang, Y., Kizer, J. R., Best, L. G. and Howard, B. V.Triglyceride and HDL-C dyslipidemia and risks of coronary heart disease and ischemic stroke by glycemic dysregulation status: The strong heart study. Diabetes Care, 2017;40:529–537.
23. Maas, A. H. E. M. and Appelman, Y. E. A.Gender differences in coronary heart disease. Netherlands Heart Journal, 2010;18 (12); 598 – 603.
24. George, J., Rapsomaniki, E., Pujades-Rodriguez, M., et al.How does cardiovascular disease first present in women and men? Incidence of 12 cardiovascular diseases in a contemporary cohort of 1,937,360 people. Circulation, 2015;132:1320–1328.
25. Bots, S. H., Peters, S. A. E., Woodward, M. Sex differences in coronary heart disease and stroke mortality: a global assessment of the effect of ageing between 1980 and 2010. BMJ Global Health 2:e000298. Doi: 10.1136/bmjgh-2017- 000298.
26. Sani, M. U., Wahab, K. W., Yusuf, B. O., Gbadamosi, M., Johnson, O. V., Gbadamosi. A. Modifiable cardiovascular risk factors among apparently healthy adult Nigerian population - A cross sectional study. BMC Research Notes, 2010;3:11 http://www.biomedcentral.com/1756-0500/3/11
27. Okaka, E. I and Eiya, B. O. Prevalence and pattern of dyslipidemia in a rural community in Southern Nigeria. African Journal of Medical Health Science, 2013;12: 82 - 86.
28. Alikor, C. A. and Emem-Chioma, P. C. Epidemiology of diabetes and impaired fasting glucose in a rural community of Nigerian Niger delta region. Niger Journal of Medicine, 2015;24(2); 114-124.
29. Reiner, Z. E., Catapano, A. L., Backer, G. D., Graham, I. Taskinen, M., Wiklund, O., Agewall, S., Alegria, E., Chapman, M. J., Durrington, P., Erdine, S., Halcox, J., Hobbs, R., Kjekshus, J., Filardi, P. P., Riccardi, G., Storey, R. F., Wood, D. European Society of Cardiology (ESC) and the European Atherosclerosis Society (EAS) Guidelines for the management of dyslipidaemias. European Heart Journal, 2011;32: 1769–1818.
30. Le, J., Zhang, D., Menees, S., Chen, J., Raghuveer, G. "Vascular Age" Is advanced in children with atherosclerosis promoting risk factors Circ Cardiovasc Imaging, 2010; 3:8-14
31. Grundy, S. M., Stone, N. J., Bailey, A. L., *et al*. Check, change, control cholesterolAHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA guideline on the management of blood cholesterol: A report of the American college of cardiology/American heart association task force on clinical practice guidelines [published online November 10, 2018]. Circulation.
32. Jacobson, T. A., Ito, M. K., Maki, K. C., Orringer, C. E., Bays, H. E., Jones, P. H., McKenney, J. M., Grundy, S. M., Gill, E. A., Wild, R. A., Wilson, D. P. and Brown, W. V. National lipid association recommendations for patient-centered management of dyslipidemia: Part 1 - Full Report. Journal of Clinical Lipidology, 2015; In press
33. Qi, L., Ding, X., Tang, W., Li, Q., Mao, D. and Wang, Y. Prevalence and risk factors associated with dyslipidemia in Chongqing, China. International Journal of Environmental Research for Public Health,2015;12: 13455 – 13465.
34. Jellinger, P S., Handelsman, Y., Rosenblit, P. D., Bloomgarden, Z. T., Fonseca, V. A., Garber, A. J., Grunberger, G., Guerin, C. K.,Bell, D. S. H., Mechanick, J. I., Pessah-Pollack, R., Wyne, K., Smith, D., Brinton, E. A., Fazio, S. and Davidson. M. American association of clinical endocrinologists and American college of endocrinology guidelines for management of dyslipidemia and prevention of cardiovascular disease. Endocrine Practice, 2017;23(2): 1-87
35. Osuji, C. U., Omejua, E. G., Onwubuya, E. I. and Ahaneku, G. I. Serum Lipid Profile of Newly Diagnosed Hypertensive Patients in Nnewi, South-East Nigeria. International Journal of Hypertension, 2012; 710486: 7pp. doi:10.1155/2012/710486
36. Akpinar, O., Bozkurt, A., Acarturk, E., Seydaoglu, G. A new index (CHOLINDEX) in detecting coronary artery disease risk. Anadolu Kardiyol Derg, 2013; 13:315-319.