



## **Atherogenic Triad in Overweight and Obese Adults in Benin-City, South-South Nigeria**

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### **Author's contribution**

*The sole author designed, analyzed, interpreted and prepared the manuscript.*

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### **ABSTRACT**

**Context:** Atherogenic triad is a constellation of lipid abnormalities characterised by elevated small, dense low-density lipoprotein particles, decreased high-density lipoprotein particles and hypertriglyceridaemia.

**Aim:** This study determined the prevalence of atherogenic triad among the overweight and obese adults in Benin-City, South-South, Nigeria.

**Setting and Design:** Study was conducted among overweight and obese adults in Benin City. It is a cross-sectional study.

**Materials and Methods:** This study was conducted among 138 overweight and obese adults in Benin-City, Nigeria between July and September 2016. The concentration of small, dense LDL-cholesterol was determined using precipitation method involving sodium-heparin and manganese II chloride as described by Hirano *et al.* Subjects were categorised into three groups (ideal weight, overweight and obese) based on their body mass index.

**Results:** The mean small dense LDL-cholesterol levels in the three groups were  $0.79 \pm 0.16$ ,  $0.96 \pm 0.15$  and  $1.33 \pm 0.26$  mmol/l respectively which were statistically different, triglyceride levels were  $1.41 \pm 0.73$ ,  $1.33 \pm 0.61$  and  $1.31 \pm 0.73$  mmol/L in the ideal weight, overweight and obese categories respectively, while plasma HDL-cholesterol were  $0.85 \pm 0.20$ ,  $1.05 \pm 0.34$  and  $0.94 \pm 0.30$  mmol/L in the

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ideal weight, overweight and obese groups respectively. Elevated LDL-cholesterol was seen in more than 80% of the normal weight and the obese subjects and; 57.8% of the overweight subjects. Two (3.6%) obese subjects had elevated sdLDL-C with LDL-C within the reference interval. Reduced HDL-C was found more in the normal weight and obese subjects while atherogenic triad was detected in 7 (15.2%) of the obese.

**Conclusion:** The prevalence of atherogenic triad among the obese adult was 15.2%. The prevalence of the atherogenic triad components was higher among the obese individuals than the overweight. There is an urgent need for the introduction of therapeutic lifestyle modification among overweight and obese individuals in Nigerian population.

**Keywords:** Atherogenic triad; overweight; obesity; small dense LDL-cholesterol; lipids.

**Key message:** Prevalence of atherogenic triad is high among the obese Nigerians. They are at the risk of cardiovascular diseases.

## 1. INTRODUCTION

Atherogenic triad, otherwise known as dyslipidaemic triad or lipid triad comprises of elevated blood concentration of small, dense low-density lipoprotein (sdLDL) particles, decreased high-density lipoprotein particles and increased triglyceride [1]. These lipid abnormalities are the typical features of obesity, metabolic syndrome, insulin resistance and type-2 diabetes mellitus [2]. The involvement of lipid triad in the aetiopathogenesis of cardiovascular diseases (CVD) has been well documented [3]. CVD is the major cause of morbidity and mortality worldwide with approximately 17.5 million deaths in 2005, among them, 7.6 million deaths from coronary heart disease and 5.7 million deaths from stroke. This figure has been projected to rise to 20 million deaths in 2015 [4].

Low-density lipoproteins are primary plasma lipid carriers in humans comprising of two distinct subfractions, namely, large buoyant LDL and small dense LDL particles [5] that vary in their size, density, physicochemical composition, metabolic-behavior and atherogenicity. SdLDL is lipoprotein fraction with a density ranging from 1.019 to 1.063 g/ml. sdLDL particle has recently emerged as an important risk factor for myocardial infarction and cardiovascular diseases [2,6]. Generally, LDL particles are classified into four subclasses. These include large (LDL I), intermediate (LDL II), small (LDL III) and very small (LDL IV) LDL subfractions. The LDLIII and LDLIV constitute the small dense LDL particle commonly referred to as phenotype pattern B with a peak diameter of  $\leq 25.5$  nm. Increased atherogenicity of sdLDL is based on their specific biochemical and biophysical properties. Its small size favours their penetration into the arterial wall where they serve as a

source of cholesterol and lipid storage [7]. The association between lipids and coronary heart disease (CHD) has been well documented [5].

Koba et al. reported that small dense LDL is more atherogenic than large buoyant (lbLDL). They readily penetrate the arterial wall, have higher affinity for the intimal proteoglycans, prolonged plasma half-life than buoyant LDL, low affinity for LDL receptor, low resistance to oxidative stress than buoyant LDL and they are easily engulfed by macrophages [8]. SdLDL-C is elevated in patients with family history of premature coronary heart disease (CHD), hyperapobetalipoproteinaemia, familial combined hyperlipidaemia, familial dyslipidaemic hypertension, subclinical hypothyroidism and metabolic syndrome [9]. From genome-wide association studies, proprotein convertase subtilisin kexin type-7 (PCSK7) gene has been found to be associated with sdLDL-C and other lipid factors [10].

Highly atherogenic sdLDL-C are generated from VLDLI particles by the action of lipoprotein lipase, cholesterylester transport protein (CETP) and hormone-sensitive lipase [11]. Environmental factors, abdominal adiposity, oral contraceptive use, diabetes mellitus and high carbohydrate diets are associated with elevated sdLDL-C [11]. Gazi et al. reported the acceptance of the sdLDL-cholesterol as an emerging CVD risk factor by National Cholesterol Education Program (NCEP)-Adult Treatment Panel III (ATP-III). But no definite causal relationship was established properly due to the close association between sdLDL-C, triglycerides and another risk factor [12]. Small, dense LDL-C is now seen as a valuable screening and preventive tool in the assessment of CVD most especially in patients with metabolic syndrome

[13] and also featuring in some clinical guidelines for the management of dyslipidaemia [14]. Prevalence of dyslipidaemia has been reported to be high in all the six geopolitical zones in Nigeria most especially reduced HDL-cholesterol and elevated LDL-cholesterol [15]. In apparently healthy and diabetic Nigerians, dyslipidaemia has been reported as high as 60% and 89% respectively. Dyslipidaemia was also documented among obese women in Southeastern Nigeria most especially elevated triglyceride, reduced HDL-cholesterol and elevated LDL-cholesterol [16].

This study assessed the pattern of dyslipidaemia as it relates to atherogenic triad among overweight and obese adults in Benin City, Nigeria.

## 2. MATERIALS AND METHODS

A total of 138 subjects were recruited and categorised equally as ideal weight, overweight and obese using the National Institute of Health criteria for body mass index classification (NIH, 2000). The study population consisted of male and female subjects between the ages of 18-65 years. Subjects were selected using a systematic sampling method. They were recruited from clients who came for routine investigations at the Centre for Disease Control and Prevention (CDC Unit) of the Department of Community Health, UBTH, where apparently healthy individuals are routinely screened for common diseases; the Metabolic Clinic of the Department of Chemical Pathology in UBTH, where patients with metabolic diseases are evaluated and managed. The controls (ideal weight individuals) with BMI of 18.5-25.0 kg/m<sup>2</sup> were both male and female individuals between the ages of 18-65 years recruited from the hospital workers. Subjects taking lipid-lowering agents, contraceptive and other steroid were excluded. Pregnant and breastfeeding mothers were also excluded.

Ethical clearance was obtained from the Ethical Committee of the University of Benin Teaching Hospital (ADM/E 22/A/VOL.VII/1078). Written consent was obtained from the subjects.

Height was measured (to the nearest 0.1cm) using stadiometer (RGZ-120) with the subjects standing erect against the wall on a horizontal floor without shoes. The head was positioned in such a way that the external auditory meatus and the angle of the eye in horizontal plane [17].

Body mass index (BMI) was calculated as weight divided by height metre square (kg/m<sup>2</sup>) [18]. BMI of >30 kg/m<sup>2</sup> was considered as obese and 25-29.9 kg/m<sup>2</sup> as overweight while among the ideal weight controls; BMI was 18-24.9 kg/m<sup>2</sup> [17]. Waist circumference was measured midway between the inferior margin of the last rib and the iliac crest in a horizontal plane with the measuring tape not compressing the soft tissue and subject standing with feet 25-30 cm apart to ensure the weight is evenly distributed [19]. Blood pressure (systolic and diastolic) was measured using sphygmomanometer and Litmann® stethoscope (in mmHg). The blood pressure was taken using Accousson® sphygmomanometer with the subject in a relaxed sitting position [10,9]. The cuff was wrapped around the left mid- upper arm; systolic blood pressure was detected initially by palpation and later by the Korotkov sounds I and V to determine systolic and diastolic pressures respectively.

Five millilitres (5 ml) of venous blood was collected under aseptic procedure from the antecubital vein of all the subjects. Blood was dispensed into potassium ethylene diamine tetraacetic acid (EDTA) specimen bottle for fasting plasma lipids profile (FPLP) and sdLDL-cholesterol. Blood was centrifuged at 3000 revolutions per minute for 10 minutes, and plasma separated and stored in -80°C freezer not later than 2 weeks before they were analysed.

### 2.1 Estimation of Small Dense LDL-Cholesterol

Small dense LDL-Cholesterol was determined by simple precipitation method. This was done by heparin-manganese precipitation according to the method of Hirano et al. in 2003 [20]. Thirty (30) mmol/l of manganese II chloride, MnCl<sub>2</sub> was prepared by dissolving 1.485 g in 250 ml of distil water while 40 IU/ml sodium-heparin was prepared by adding 0.2 g of the salt into 500 ml of distil water (20,000 IU in 500 ml of distil water) since 100 IU sodium-heparin is equivalent to 1 mg of the salt [20].

### 2.2 Procedure for sdLDL-cholesterol Estimation

1. The precipitation reagent (0.1 ml), containing 150 IU/ml heparin-sodium salt (Elabscience, LOT: AKOO17MAY13042) and 90 mmol/L Manganese II chloride

tetrahydrate (Loba Chemie, LOT:L157011601) were added to each serum sample (0.1 ml), mixed and incubated for 10 minutes at 37°C.

2. The samples were placed in an ice bath and allowed to stand for 15 minutes. This was followed by centrifugation at 15,000 rpm for 15 minutes at 4°C using a cold centrifuge (Rotina 380R, Singapore).
3. An aliquot of the supernatant was taken for routine cholesterol analysis.
4. The cholesterol content was determined using the enzymatic method (CHOD-PAP assay) for total cholesterol as described by Allain et al. [21].
5. Small dense LDL-Cholesterol was calculated as  $\text{sdLDL-C} = \text{Measured cholesterol} - \text{HDL-C}$ .
6. Percentage sdLDL (%sdLDL) =  $(\text{sdLDL-C} / \text{LDL-C}) \times 100$

Total cholesterol and triglyceride were determined using the enzymatic methods [21] while HDL-cholesterol was determined using the precipitation method as described by Burstein et al. [22]. Calculated LDL-cholesterol was estimated using the Friedewald equation [23] ( $\text{LDL-C mmol/L} = \text{Total cholesterol} - \text{HDL-C} - \text{triglyceride}/2.2$ ). Non-HDL-cholesterol was estimated as the difference between total cholesterol and HDL-cholesterol. Control sera from Randox® (UK) (levels 1 and 3) were assayed for each run. Controls and standards were analysed 10 times to determine the mean, standard deviation and the coefficient of variation ( $\text{CV} = \text{mean}/\text{standard deviation} \times 100$ ) were calculated to determine the precision. The estimated CV for the various assays was sdLDL-C (2.1%), total cholesterol (3%), and triglyceride (4%). These CVs were in agreement with values stated by the kit manufacturers. Their precisions are sdLDL-C (97.7%), total cholesterol (97%) and triglyceride (96%). The reference intervals for various biochemical parameters were stated as plasma sdLDL-C (0.197 – 1.347 mmol/l), total cholesterol (2.38 – 4.65 mmol/l), LDL – C (1.99 – 3.36 mmol/l), HDL – C (0.75 – 1.55 mmol/l), Triglyceride (0.22 – 0.87 mmol/l).

### 2.3 Data Analysis

Data were analysed using the Statistical Package for Social Science (SPSS) version 20 (IBM Corp., Amonk, NY; released 2011). Data were tested for normality using the Kolmogorov-Smirnov test and were found to be normally distributed. Results were expressed as mean  $\pm$

standard deviation; student t-test and Analysis of Variance (ANOVA) were used for comparison of means between two groups and multiple groups respectively. Statistical significance was set at  $p < 0.05$ .

### 3. RESULTS

One hundred and thirty-eight subjects constituting 88 females and 50 males were recruited for this study. They included 46 ideal weight, 46 overweight and 46 obese. Their mean ages were  $55.57 \pm 10.48$ ,  $48.91 \pm 8.33$  and  $45.11 \pm 7.61$  years respectively. Thirty-eight (82.6%) of the ideal weight subjects were females, 22 (47.8%) of the overweight subjects were males while 30 (65.2%) of the obese were females. Their mean ages were  $55.57 \pm 10.48$ ,  $48.91 \pm 8.33$  and  $45.11 \pm 7.61$  years respectively. Thirty-eight (82.6%) of the normal weight subject were females, 22 (47.8%) of the overweight subjects were males while 30 (65.2%) of the obese were females. There were no statistical differences in their age distribution ( $p=0.000$ ). Their anthropometric, clinical and biochemical parameters were measured and documented in Table 1.

#### 3.1 Anthropometric and Clinical Parameters

The mean weight, height, BMI, waist circumference, hip circumference and waist-hip ratio are shown in Table 1. Using one-way ANOVA across the 3 groups, there was a significant difference ( $p= 0.000$ ) in the anthropometric parameters except for the height ( $p= 0.054$ ) of the participants. Also, there was a significant difference ( $p= 0.000$ ) in systolic blood pressure, respiratory and pulse rates but no difference was found in diastolic blood pressure ( $p= 0.064$ ).

#### 3.2 Biochemical Profile of the Subjects

The average serum total cholesterol levels across the groups were  $5.68 \pm 0.99$ ,  $5.36 \pm 1.25$  and  $6.28 \pm 1.05$  mmol/L respectively ( $F= 2.9$ ;  $p= 0.000$ ). Serum triglyceride levels across the groups were not statistically different ( $p= 0.354$ ). There was reduced HDL-cholesterol among the normal weight subjects. There was no statistical difference between LDL-cholesterol levels between the normal weight and obese subject ( $p= 0.520$ ). Dyslipidaemia was observed across the three groups, most especially elevated LDL-cholesterol, and elevated triglyceride, occurring

in more than 70% of all the subjects. Elevated total cholesterol was seen in 95.7% of the obese subjects.

Highest mean values for serum total cholesterol, LDL-cholesterol and small dense LDL-cholesterol were recorded among the obese participants. Highest level of HDL-cholesterol was found among the overweight category while the mean triglyceride level was the highest among the normal weight participants. The average sdLDL-cholesterol levels were found to be  $0.79 \pm 0.16$ ,  $0.96 \pm 0.15$  and  $1.33 \pm 0.26$  mmol/l among the normal weight, overweight and obese participants respectively, with a significant difference existing between the three groups ( $F=7.3$ ,  $p=0.000$ ). Increased levels of % of sdLDL were seen among the overweight and obese subjects. There was no significant difference in the serum levels of triglycerides among the groups ( $F=1.1$ ;  $p=0.354$ ). There was a significant difference in the means of all the biochemical parameters except serum

triglyceride ( $p=0.354$ ). There was no significant difference between LDL-C levels in normal weight and obese subjects ( $p=0.520$ ). However, a statistically significant difference was found in sdLDL-C between normal weight and obese subjects ( $p=0.000$ ). Dyslipidaemia was observed across the three groups; most especially elevated LDL-cholesterol elevated triglyceride occurring in more than 70% of all the subjects while elevated total cholesterol was seen in 95.7% of the obese subjects. Elevated sdLDL-C was seen in 10 (12.7%) and 20 (43.5%) of the overweight and obese subjects respectively, while atherogenic triad (a constellation of elevated triglyceride, reduced HDL-C and elevated sdLDL-cholesterol) was observed in 7 (15.2%) of the obese subjects. There was no statistical difference between hypertriglyceridaemia across the three groups ( $p=0.713$ ) unlike other abnormalities which were statistically significant ( $p<0.05$ ). Also, 2 (3.6%) of the obese subjects had elevated sdLDL-C with LDL-C within the reference interval.

**Table 1. Anthropometric and clinical characteristics of the study subjects**

Variables	Ideal weight mean $\pm$ SD	Overweight mean $\pm$ SD	Obese mean $\pm$ SD	F-value	p-value
Weight (Kg)	63.3 $\pm$ 9.48	75.54 $\pm$ 7.78	88.93 $\pm$ 10.65	7.6	0.000
Height (m)	1.68 $\pm$ 1.06	1.65 $\pm$ 0.08	1.61 $\pm$ 0.07	1.6	0.054
BMI (Kg/m <sup>2</sup> )	22.19 $\pm$ 1.61	27.82 $\pm$ 1.16	34.28 $\pm$ 3.21	26.5	0.000
HC (cm)	86.99 $\pm$ 10.20	98.87 $\pm$ 7.94	98.56 $\pm$ 15.42	4.6	0.000
WC (cm)	90.48 $\pm$ 8.18	105.13 $\pm$ 7.39	117.0 $\pm$ 19.72	14.2	0.000
Age (years)	55.57 $\pm$ 10.48	48.91 $\pm$ 8.33	45.11 $\pm$ 7.61	46.3	0.000
Meal frequency	2.56 $\pm$ 0.65	2.52 $\pm$ 0.66	2.48 $\pm$ 0.75	1.14	0.310
SBP (mmHg)	110 $\pm$ 9.35	137.26 $\pm$ 19.58	132.87 $\pm$ 18.48	4.8	0.000
DBP (mmHg)	83.26 $\pm$ 9.74	82.0 $\pm$ 11.50	87.87 $\pm$ 10.07	1.6	0.004
PR (bpm)	74 $\pm$ 12.0	74.13 $\pm$ 11.81	75.96 $\pm$ 11.93	1.7	0.040
RR (cpm)	13.35 $\pm$ 1.34	16.17 $\pm$ 10.33	14.3 $\pm$ 1.40	123.4	0.000

*BMI: Body mass index; WC: Waist circumference; HC: Hip – circumference; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; PR: Pulse rate; RR: Respiratory rate*

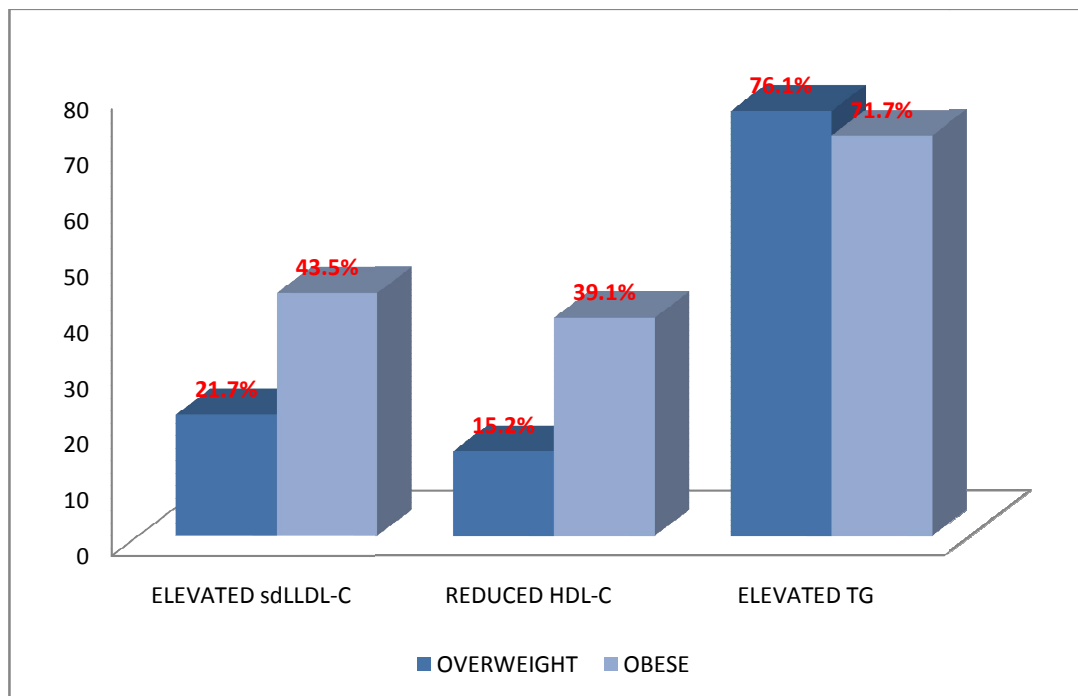
**Table 2. Biochemical profile of study subjects**

Variables	Ideal weight mean $\pm$ SD	Overweight mean $\pm$ SD	Obese mean $\pm$ SD	F-value	p-value
TC (mmol/L)	5.68 $\pm$ 0.99	5.36 $\pm$ 1.25	6.28 $\pm$ 1.05	2.9	0.000
TG (mmol/L)	1.41 $\pm$ 0.73	1.33 $\pm$ 0.61	1.31 $\pm$ 0.73	1.1	0.354
HDL-C mmol/L Male	0.89 $\pm$ 0.20	1.02 $\pm$ 0.34	0.90 $\pm$ 0.30	3.01	0.000
HDL-C mmol/L Female	0.94 $\pm$ 0.26	1.00 $\pm$ 0.30	0.94 $\pm$ 0.32	3.4	0.000
LDL-C (mmol/L)	4.61 $\pm$ 1.09	3.70 $\pm$ 1.09	4.75 $\pm$ 1.04	2.8	0.000
sdLDL-C (mmol/L)	0.79 $\pm$ 0.16	0.96 $\pm$ 0.15	1.33 $\pm$ 0.26	7.3	0.000
%sdLDL	18.05 $\pm$ 5.22	27.74 $\pm$ 10.88	28.94 $\pm$ 9.35	5.66	0.002

*TC: Total cholesterol; TG: Triglyceride; HDL-C: High-density lipoprotein-cholesterol; LDL-C: Low-density lipoprotein-cholesterol, sdLDL-C: small dense low density lipoprotein-cholesterol*

**Table 3. Frequencies of elevated biochemical parameters and lipid atherogenic triad in normal weight, overweight and obese subjects**

Abnormalities	Ideal weight (n=46)	Overweight (n=46)	Obese (n=46)	$\chi^2$	p-value
Elevated TC	40 (87%)	32 (69.6%)	44 (95.7%)	10.12	0.006
Elevated TG	35 (76.1%)	35 (76.1%)	33 (71.7%)	0.676	0.713
Elevated LDL-C	39 (84.8%)	27 (58.7%)	41 (89.1%)	20.52	0.000
Elevated sdLDL-C	0 (0%)	10 (21.7%)	20 (43.5%)	46.78	0.000
Reduced HDL-C	18 (39.1%)	7 (15.2%)	18 (39.1%)	12.31	0.000
Atherogenic triad	0 (0%)	0 (0%)	7 (15.2%)	-	-

**Fig. 1. Prevalence of components of atherogenic triad in overweight and obese subjects**

#### 4. DISCUSSION

Atherogenic triad is characterised by elevated sdLDL cholesterol, increased triglyceride and reduced HDL-cholesterol. This has been reported to be of higher clinical significance than LDL-cholesterol as atherogenic triad is commonly found in a patient with coronary artery disease, diabetes mellitus, obesity and metabolic syndrome [24]. Overweight or pre-obesity is defined as having more body fat than is optimally healthy [25] while obesity is a medical condition in which excess body fat has accumulated to the extent of having a negative effect on the health of the individual [26]. Obesity arises from the combination of excessive food intake, lack of physical activity and genetic susceptibility [27].

Atherogenic triad has been reported as a feature of obesity and predisposes the obese to cardiovascular diseases [28]. Alteration in lipoprotein metabolism leads to the elevation of lipid particles rich in triglycerides remnants such as chylomicrons very low-density lipoprotein (VLDL) and intermediate density lipoprotein (IDL). Like LDL, these remnant particles readily penetrate the intima of the arterial wall to cause atherosclerosis [29].

Presence of elevated triglyceride contributes immensely to the formation [6] of sdLDL particles from VLDL 1. HDL-C protects the arterial vessels from atherosclerosis as it promotes reverse-cholesterol transport from cells in the vessel wall to the liver for elimination [30].

This study showed that the prevalence of atherogenic triad in the obese subjects is 15.7%. This prevalence is a bit reduced from the prevalence (24.08%) reported among Germans population [30]. Dietary habits among the Germans could account for a higher prevalence of atherogenic triad seen in their population [30].

However, a lower prevalence of atherogenic triad was reported among the Spanish working population [31]. Elevated sdLDL-Cholesterol has been reported as one of the additional cardiovascular risk factors in the most population.

This study observed an elevated sdLDL-C among the overweight (21.7%) and obese (43.5%) subjects. The only study pointing to elevated sdLDL-Cholesterol among diabetics in Nigeria was reported by Inaku *et al*; where the percentage sdLDL (%) among the diabetic patients and control group were  $45 \pm 17.79$  and  $32 \pm 15.93\%$  respectively [32]. The present study is also in agreement with elevated sdLDL-C reported in adult Thai population. This implies that obese and diabetic Nigerians are at the risk of cardiovascular diseases.

Elevated triglyceride observed in more than seventy percent of overweight and obese in this study further supports the previous studies that reported prevalence of hypertriglyceridaemia as 34.1% among women in South-Eastern Region, Nigeria [33]. However, Iloh *et al.* reported the prevalence of hypertriglyceridaemia as 4.7% among obese adult Nigerians [34].

The reduced HDL-C observed in this study also corroborates the findings reported by Oguejiofor *et al.* who reported a high prevalence of dyslipidaemia across the six geopolitical zones in Nigeria, most especially reduced HDL-cholesterol and elevated LDL-cholesterol among apparently healthy Nigerians [15]. The present study is also in agreement with the reduced HDL-C levels reported by Ilo *et al.* among the adult population in Nigeria [16].

Abnormalities in blood lipids have been well reported among the overweight and obese individuals. Elevated LDL-cholesterol, triglyceride and low HDL-cholesterol are risk factors for the development of cardiovascular diseases. In this study; dyslipidaemia was observed across the three groups most especially elevated LDL-cholesterol which cut across the three groups. It was also observed that there is no significant

difference between LDL-C levels in normal weight and obese subjects but a statistically significant difference seen in the sdLDL-C levels in both groups. These abnormalities may be a pointer to increased prevalence of metabolic syndrome and dyslipidaemia among normal weight, overweight and obese subjects in the larger population.

## 5. LIMITATION

The reference interval for sdLDL-C used in the interpretation of the results is from The Caucasian population as local reference interval for sdLDL-C is not available yet.

## 6. CONCLUSION

Atherogenic triad is 15.7% in the obese subjects while the prevalence of the components of the atherogenic triad was high in the study population, most especially in the obese. These findings necessitate institutionalising preventive measures such as therapeutic lifestyle modification among the overweight and obese individuals to prevent the cardiovascular diseases.

## ETHICAL APPROVAL AND CONSENT

Ethical clearance was obtained from the Ethical Committee of the University of Benin Teaching Hospital (ADM/E 22/A/VOL.VII/1078). Written consent was obtained from the subjects.

## COMPETING INTERESTS

Author has declared that no competing interests exist.

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