Original Research Article

A study on lipid profile level and cardiovascular markers intype 2 diabetes mellitus(T2DM) patients

.

ABSTRACT

Aim: To assess lipid profile level and cardiovascular markers intype 2 diabetes mellitus(T2DM) patients.
Study design: Cross-sectional Study.
Place and Duration of Study: Port-Harcourt, Nigeria, InqabaBiotec(molecular analysis) andEvashalom Medical Diagnostic services, between November 2022 and April2023.
Methodology: Ninety (90) subjects between aged between 18- 70years among which 54 were females and 26 males were recruited. Blood glucose, total cholesterol (TCHOL), triglycerides (TG) and high-density lipoprotein (HDL-c) were measured by enzymatic colorimetric methods and low-density lipoprotein cholesterol (LDL-c), HOMA_IR, HOMA-β were calculated for each sample.
Results: This study found that females had an increased HDL-C and Lipo-A values, a decreased value in TG and AIP when compared with their male counterparts. A correlation with Insulin/TG (r=0.367, p=0.0275), HOMA-IR/FBS (r=0.426, p=0.0096), HOMA-IR/HbAIc (r=0.484. p=0.0028), Lipo A/TG (r=0.359, p=0.0315), Lipo A/insulin (r=0.380, p=0.0223), Lipo A/HOMA-IR (r=0.393, p=0.0176) was observed only in males. About 43.3% (n=39) had controlled T2DM while 56.7%(n=51) had uncontrolled T2DM.
Conclusion: This study also found that females had an increased HDL-C and Lipo-A values, a decreased value in TG and AIP when compared with their male counterparts.

Keywords: lipid profile, cardiovascular markers, T2DM patients

1. INTRODUCTION

This is a metabolic disorder which is non-communicable and chronic, its development is as a consequence of two major factors: insulin resistance and defective secretion of insulin by islet β cells produced by the pancreas or a combination of both [1]. Regulated molecular mechanisms are involved in insulin synthesis, release and action on tissues as its goal meets the metabolic demand of the body. Therefore, abnormality in any mechanism can give rise to ta metabolic imbalance that eventually births the pathogenesis of T2DM.

There are many medical conditions which can potentially give rise to or exacerbate type 2 diabetes mellitus. These include obesity, hypertension, elevated cholesterol (combined hyperlipidaemia), and with the condition often termed metabolic syndrome also known as Syndrome X, Reaven's syndrome. Other causes include acromegaly, Cushing's syndrome, thyrotoxicosis, pheochromocytoma, chronic pancreatitis, cancer, and drugs. Additional factors found to increase the risk of type 2 diabetes mellitus include aging, high-fat diets, and a less active lifestyle [2].

Type 2 diabetes mellitus, as a multisystem disease exhibits a strong correlation with the development of cardiovascular disease which leads to an increase rate of mortality in adults from stroke and heart disease associated with vascular complications(Gast et al., 2012) [3].which consists of accelerated atherosclerosis resulting in increased risk of cerebrovascular disease, premature coronary artery disease and severe peripheral vascular diseases. Type 2 diabetes mellitus is considered as a significant risk factor for cardiovascular disease through the activities of several molecular mechanisms and

pathological pathways including the role of insulin resistance in oxidative stress, vascular function, hypertension, atherosclerosis, inflammation and macrophage accumulation [4].

Globally, cardiovascular diseases represent a leading health issue [5]. Studies have shown that diabetics have two to four- fold likelihood to develop myocardial infarction and coronary artery disease and about 70% above the age of 65years would die from it [6]. It has been established that T2DM is a risk factor for heart disease and stroke. The close relationship between these two conditions has resulted in the popular soil hypothesis which postulates that both conditions have common environmental and genetic factors which influence their association. However, common risk factors such as Insulin resistance, inflammation, oxidative stress, hypercoagulability, high blood pressure, dyslipidemia, obesity and thrombophilia are common physiological features shared among cardiovascular disease and T2DM and can be identified in many patients although little is known about the mechanisms by which these factors have their influence on T2DM and cardiovascular disease. Particularly, hyperglycaemia linked with a low-grade inflammation and insulin resistance as well as chronic enhancement of oxidative stress which promotes atherogenesis and triggers endothelial dysfunction. It is also a fact that T2DM is associated with hemostatic activities and enhancement of platelet [7]. Presently, it has been revealed that the interaction of T2DM and its associated cardiovascular risk supports the advancing nature of vascular damage resulting in atherosclerosis [8], however, it is also evident that modifications of lifestyle such as weight loss and physical activity oppose cardiovascular disease risk factors in pre-diabetic individuals [9].
Some common single nucleotide polymorphisms (SNPs) have been linked with an increased risk of cardiovascular disease and T2DM. It has been shown that non-coding Ribonucleic acids (RNA) have emerged as key players of the pathophysiology underlying both T2DM and cardiovascular disease [10]. The most common forms of T2DM and cardiovascular disease are polygenic. However, Mendelian forms have been demonstrated for both conditions in which the disease can be triggered by a single gene mutation [11]. In this regard, familial forms of cardiovascular disease risk factors including T2DM, hypertension and hypercholesterolemia could be as a result of heterozygous mutations in candidate genes however, these genes do not predispose individuals to these conditions automatically for instance, paraoxonase is one of the candidate genes involved in the pathway pathophysiologically related to both conditions, it synthesizes an enzyme bound to high density lipoprotein (HDL) protecting the low density lipoprotein (LDL) from proatherogenic, oxidative modifications. Other candidate genes are represented by adiponectin and its pathway. Adiponectin is an adipokin having anti-inflammatory and antiatherogenic actions. A reduced level as observed in obesity correlates with increased risk for T2DM and cardiovascular disease whereas an increased level protects from the risk of cardiovascular disease in diabetes [3].

Over the past few years, the related metabolic disorder of T2DM and obesity globally is certainly driven by behavioural and environmental factors, since there is no change of genetic components in an appreciable manner over a short period of time.Firstly, it has been suggested that DNA sequence variation contributes to T2DM risk. It has also been observed that T2DM is greater for monozygotic twins than for dizygotic twins. Secondly, the incidence of this disease is much higher in certain ethnic/racial groups, despite a relatively comparable environment to that of neighbouring populations. Thirdly, in population studies, family history, however, is an independent risk factor for diabetes and fourthly, mutations in single genes that cause rare familial forms of diabetes prove that single base pair changes occur in the key genes especially in the coding regions leading to alterations in the protein sequence and function sufficient to hyperglycemia in diabetes [11]. Consistent with this notion, T2DM heritability is estimated as high as 72% in a large international meta-analysis of twin studies [12]. Considering all these, it is obvious that the rapid changes in the epidemiology of T2DM

globally are likely caused by behavioural and environmental factors laying on a background of genetic predisposition which is likely to vary across populations, partially due to their divergent genetic history and unequal selection pressures in specific geographical regions. Genetic exploration is relevant such that the identification of genetic variants linked with T2DM illuminates pathogenic mechanisms from which therapeutic windows may emerge. This is important because germline genetic variation predates disease onset, thus genetic approach provides unique opportunity to shed light on the pathophysiology of diabetes and helps in unravelling its clinical heterogeneity and strongly refine therapeutic strategies [12]. The aim of this study was to assess lipid profile level and cardiovascular markers in type 2 diabetes mellitus(T2DM) patients.

2. MATERIAL AND METHODS

2.1 Study Area

The study area was in Port Harcourt, capital of Rivers State, Southern Nigeria. It lies along the Bonny River and coordinates 4·49'27"N 7·2'1"E. It has an area of 369km2 and an estimated population of 1,865,000 as at 2016 [13]. It is a major industrial center having large numbers of multinational firms as well as other industrial concerns, particularly business related to the petroleum industry.

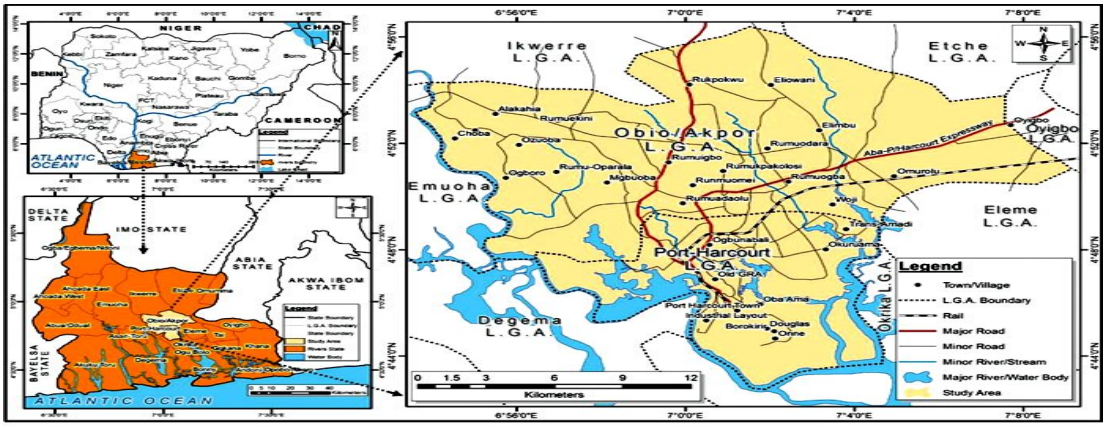


Figure 1: Map of Port Harcourt [13]

2.2 Research Design

This is a cross sectional study involving the evaluation of some genetic variants associated with glycemic response and to evaluate body mass index (BMI), blood pressure (SBP and DBP) and some biochemical markers such as glycated haemoglobin (HbAIc), insulin, homeostatic model assessment of β function (HOMA-β), homeostatic model assessment for insulin resistance (HOMA-IR¬), fasting blood sugar (FBS), Lipoproteins such as total cholesterol (TCHOL), high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C), very low density lipoprotein cholesterol (VLDL-C), Non density lipoprotein cholesterol (Non-HDL-C), Lipoprotein A and atherogenic ratios such as Castelli Risk Index 1(CRI-1), Castelli Risk Index 11 (CRI-11), atherogenic index of plasma (AIP) and atherogenic coefficient (AC) in male and female type 2 diabetes mellitus subjects.. The biodata and medical history of the subjects were obtained using a questionnaire.

2.3 Validity of Instruments

Using manufacturer's specifications, all sensitive equipment was calibrated. Sample bottles were pre-treated accordingly before use. Appropriate labelling and all pre-analytical precautions were adhered to, so as to prevent errors.

2.4 Sample Size Calculation

A total of 90 adults were enrolled for this study. The incident rate of adult with T2DM in Nigeria is 3.84% cases[14]. Making prevalence rate a total of 0.038%. Using the formular,

$$N=Z2 \times p (1-p) / d2$$

Where N= Minimum sample size
d = Desired level of significance (0.05)
z = Confidence interval (1.96)
p = Prevalence rate or proportion of occurrence (0.038%)

Therefore, $N = 3.8416 \times 0.038 (1-0.038)/ 0.0025$
Minimum participants required =56

2.5 Eligibility Criteria

2.5.1 Inclusion Criteria

Individuals with diagnosed T2DM of at least 1 year, aged between 18–70 years and on continuous metformin/sulphonylureas combination therapy for at least six (6) months prior to the study were included.

2.5.2 Exclusion Criteria

Individuals treated with insulin in the last six months or on any other drug for T2DM prior to the study were excluded. Individuals who were also critically ill or pregnant were also excluded from study.

2.6 Sample Collection

Ten (10.0) mls of overnight fasting blood sample was obtained from each subject. This was after completing the questionnaire. Their body weight in kilograms, height in meters was also measured and recorded.

2.6.1 Order of Dispensing and Volume of the Blood Sample

About 3.0 ml into vacutainer type plain tubes, 4.0 ml into vacutainer type EDTA K3 (1st Tube) & 3.0ml into vacutainer type EDTA K3 (2nd Tube). All the tubes were appropriately labelled. The sample in the plain tube was allowed to retract, then centrifuged at 3000 rpm. The serum was separated into two cryo tubes (one for enzyme-linked immunosorbent assay (ELISA). Insulin and one for Lipid profiles and Lipoprotein A), labelled and stored at -150C to -200C until analysis. The first EDTA tube was transported in cold box to Molecular Laboratory for DNA extraction and genotyping (Sequencing). The second EDTA tube for Glycated haemoglobin was stored at 2-80C until analysis. The fasting blood sugar was performed immediately using a controlled Kiptrack glucometer machine.

2.7 Methodologies for Laboratory Determinants

2.7.1 Fasting Blood Glucose (FBG)

FBG was performed using Kiptrack blood glucose monitoring system by Taidoc Technology Corporation, Taiwan [15].

2.7.1.1 Principle

The glucose testing is based on the measurement of electric current generated by the reaction of glucose in the sample (blood) with the enzyme glucose oxidase on the test strip. When glucose undergoes a chemical reaction in the presence of the enzymes, electrons are produced. These electrons (i.e, the charges passing through the electrodes) are measured and this is proportional to the concentration of glucose in the sample.

2.7.2 Glycosylated Haemoglobin (HbA1c)

Quantitative determination of glycosylated Haemoglobin in blood was done using the modified Ion Exchange Resin method with kit from INTECO Diagnostics, UK [16].

2.7.2.1 Principle

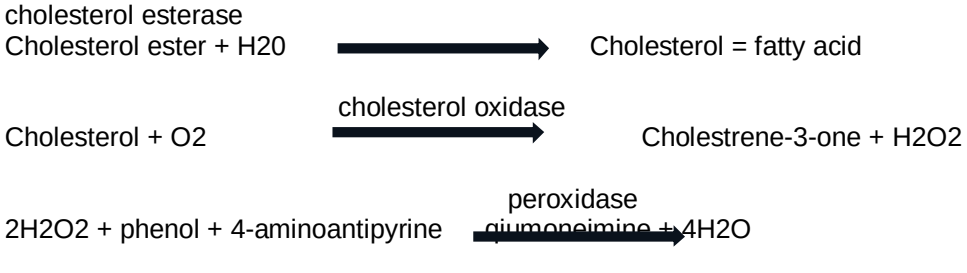
Glycosylated Haemoglobin has been defined operationally as the fast fraction of Haemoglobin A1 which elute first during column chromatography. A haemolysed preparation of whole blood is mixed continuously for 5 minutes with a weakly binding cation-exchange resin. The labile fraction is eliminated during the haemolysate preparation and during binding. During this mixing, the non-glycosylated binds to the ion exchange resin leaving the glycosylated Haemoglobin fraction free in the supernatant. After the mixing period, a filter separator is used to remove the resin from the supernatant. The percent glycosylated Haemoglobin is determined by measuring the absorbance of the ratio of the glycosylated Haemoglobin and the total Haemoglobin fraction [16].

2.7.3 Serum Total Cholesterol

Serum fasting cholesterol was performed by the cholesterol oxidase-peroxidase, enzymatic endpoint method using Erba Chem V5 Semi-Autoanalyzer and kit from Randox [17].

2.7.3.1 Principle

Cholesterol is determined after enzymatic hydrolysis and oxidation. The indicator quinoneimine is formed from hydrogen peroxide and 4-aminoantipyrine in the presence of phenol and peroxidase.

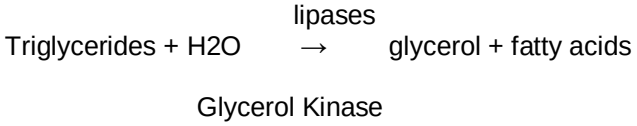


2.7.4 Serum Triglyceride

Serum fasting triglyceride was performed by a modified enzymatic colourimetric method using Erba Chem v5 Semi-auto analyzer and kit from Randox[18].

2.7.4.1 Principle

Triglycerides are determined after enzymatic hydrolysis with lipases. The indicator is a quinoneimine formed from hydrogen peroxide, 4-aminophenazone and 4-chlorophenol under catalytic influence of peroxidase.



Glycerol + ATP → glycerol-3-phosphate + ATP

Glycerol-3-phosphate Oxidase

Glycerol-3-phosphate → dihydroxyacerone + phosphate + H_2O_2

Peroxidase

$2H_2O_2$ + 4-aminophenazone → quinoneimine + HCl + $4H_2O$

2.7.5 Serum HDL-Cholesterol

Quantitative in vitro determination of HDL-Cholesterol in serum was performed using phosphotungstic acid precipitation and the cholesterol oxidase-peroxidase method [19].

2.7.5.1 Principle

Low Density Lipoproteins (LDL and VLDL) and chylomicron fractions are precipitated quantitatively by the addition of phosphotungstic acid in the presence of magnesium ions. After centrifugation, the cholesterol concentration in the HDL (High Density Lipoprotein) fraction, which remains in the supernatant, is determined.

2.7.6 Serum LDL-Cholesterol

Quantitative in vitro determination of LDL-Cholesterol in serum was performed using Polyvinyl Sulphate/Polyethyleneglcol precipitation and the cholesterol oxidase-peroxidase method [20].

2.7.6.1 Principle

Low Density Lipoproteins (LDL) in the samples is precipitated with polyvinyl sulphate. The concentration is calculated from the difference between the serum total cholesterol and the cholesterol in the supernatant after centrifugation. The cholesterol is spectrophotometrically measured by means of the coupled reactions described below.

Cholesterase

Cholesterol ester + H_2O → cholesterol + fatty acid

Cholesterol oxidase

Cholesterol + O¬2 + H_2O → cholesterone + H_2O_2

Peroxidase

$2H_2O_2$ + 4-aminoantipyrine + phenol l → Quinoneimine + $4H_2O$

2.7.7 Non-HDL-C and VLDL

Non-HDL-C and VLDLwere calculated using values of lipid profile parameters in the following way;

Non-HDL-C =Total cholesterol- HDL-C

VLDL-C = TG/2.2.

2.7.8 Atherogenic Indices

Atherogenic indices [Atherogenic Index of Plasma (AIP), Cardiac Risk Ratio (CRR) & Atherogenic Coefficient (AC)] would be calculated using the values of lipid profile parameters in the following way:

AIP = Log (TG/HDL-C)

Where, the concentration of TG and HDL are in mmol/L.

Calculation of AIP was done using CZECH online calculator of atherogenic risk [21].

CR1 = TC/HDL-C
CR11 =LDL-C/HDL-C
AC = (TC-HDL-C)/HDL-C

2.7.9 Lipoprotein a (Bio-Inteco ELISA Kit) Catalog No: E-EL-H0160

2.7.9.1 Principle

This Bio-Inteco catalogue kit uses the Sandwich-ELISA principle. The micro-ELISA plate provided in this kit has been pre-coated with an antibody specific to Human LP-a. Samples (or Standards) are added to the micro-ELISA plate wells and combined with the specific antibody. Then a biotinylated detection antibody specific for Human LP-a and Avidin Horseradish Peroxidase (HRP) conjugate are added successively to each micro plate well and incubated. Free components are washed away. The substrate solution is added to each well. Only those wells that contain Human LP-a, biotinylated detection antibody and Avidin-HRP conjugate will appear blue in color. The enzyme-substrate reaction is terminated by the addition of stop solution and the color turns yellow. The optical density (OD) is measured spectrophotometrically at a wavelength of 450 nm ± 2 nm. The OD value is proportional to the concentration of Human LP-a. The concentration of Human LP-a in the samples can be calculated by comparing the OD of the samples to the standard curve [18].

2.7.10 Serum Insulin (Bio-Inteco ELISA Kit) Catalog No: IN3745

2.7.10.1 Principle

The Insulin quantitative test is based on a solid phase enzyme-linked immunosorbent assay. The system utilizes one insulin antibody for solid phase (microtiter wells) immobilization and another anti-insulin antibody-enzyme (horseradish peroxidase) conjugate solution. The standards and test specimen (serum) are added to the insulin antibody coated microtiter wells. Then anti-insulin antibody labeled with horseradish peroxidase (conjugate) is added. If human insulin is present in the specimen, it will combine with the antibody on the well and the enzyme conjugate resulting in the insulin molecules being sandwiched between the solid phase and the enzyme-linked antibodies. After a 1hour incubation at room temperature, the wells are washed to remove unbound labeled antibodies. A solution of 3,3′,5,5′-Tetramethylbenzidine (TMB) is added incubation and incubated for 20 minutes, resulting in the development of a blue colour. The colour development is stopped with the addition of stop solution. The colour is changed to yellow and measured spectrophotometrically at 450 nm. The concentration on insulin is directly proportional to the colour intensity of the test sample [22].

2.7.11 Homeostatic Model Assessment for Insulin Resistance (HOMA-IR)

HOMA-IR index was calculated using the formula: Fasting Insulin (mU/L) × Fasting Glucose (mmol/L)/22.5 [18].

Healthy Range: 0.5 – 1.4

2.7.12 Homeostatic Model Assessment for Beta Cells (HOMA-β)

HOMA-β index was calculated using the formula: Fasting Insulin (mU/L) × 20/Fasting Glucose (mmol/L)-3.5 [18].

2.8 Statistical Analysis

The general characteristics of the participants were expressed as frequency (percentages), mean (X) and standard deviation (SD). Statistical Analysis was carried out using GraphPad Prism 9.03. Statistical comparisons of the means between groups were made using t-test and oneway analysis of variance (ANOVA).

3. RESULTS AND DISCUSSION

Table 1: Clinical Characteristics of Female and Male Subjects with Type 2 Diabetes Mellitus

Parameter	Female (n=54)	Male (n=36)	Test Statistics	
	Mean ± SD	Mean ± SD	T-Ratio	Prob> |t|
Weight (Kg)	80.70±2.17	83.75±2.66	0.8878	0.3771
Height (m)	1.58±0.01	1.66±0.02	4.3415	<.0001****
BMI (kg/m^2)	32.64±0.83	30.41±1.02	-1.6958	0.0935
SBP (mmHg)	138.33±2.66	138.67±3.26	0.0792	0.9371
DBP (mmHg)	77.39±1.41	81.64±1.73	1.9007	0.0606
Duration of DM (Years)	3.54±0.25	2.86±0.30	-1.7195	0.0890

***Key**: SD: Standard Deviation, BMI: Body Mass Index, SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure, DM: Diabetes Mellitus. Significance Level: ****=p<0.0001.*

Table 2: Biochemical Markers of Subjects with Type 2 Diabetes Mellitus by Sex

	Female (n=54)	Male (n=36)	Test Statistics	
Parameter	Mean ± SD	Mean ± SD	T-Ratio	Prob> |t|
FBS (mmol/L)	7.31±0.31	7.41±0.39	0.2049	0.8381
HbA1c (%)	7.00±0.27	7.31±0.33	0.7251	0.4703
TCHOL (mmol/L)	4.67±0.15	4.46±0.18	-0.8788	0.3819
HDL-C (mmol/L)	1.27±0.04	1.05±0.05	-3.3325	0.0013***
Non-HDL-C (mmol/L)	3.39±0.14	3.41±0.17	0.0853	0.9322
LDL-C (mmol/L)	2.79±0.14	2.65±0.17	-0.6262	0.5328
VLDL-C (mmol/L)	0.60±0.06	0.76±0.08	1.6099	0.1110
TG (mmol/L)	1.29±0.10	1.66±0.13	2.2695	0.0257*
Insulin (mlU/L)	5.30±0.65	3.91±0.80	-1.3513	0.1800
HOMA-IR	1.63±0.20	1.28±0.24	-1.1480	0.2541
HOMA β (%)	13.05±2.24	8.12±2.75	-1.3906	0.1679
LIPOPR A (mmol/L)	7.96±0.44	6.59±0.54	-1.9747	0.0514

***Key:** SD: Standard Deviation, FBS: Fasting Blood Sugar, HbA1c: Glycosylated Hemoglobin, TCHOL: Total Cholesterol, HDL-C: High-density Lipoprotein Cholesterol, LDL-C: Low-density Lipoprotein Cholesterol, VLDL-C: Very Low-density Lipoprotein Cholesterol, Non-HDL-C: Non-High-density Lipoprotein Cholesterol, TG: Triglycerides. HOMA-IR: Homeostatic Model Assessment of Insulin Resistance, HOMA β: Homeostatic Model Assessment of β Cell Function, LIPOPR A: Lipoprotein A. Significance Level: *=p<0.05, **=p<0.01, ***=p<0.001, ****=p<.0001.*

Table 3: Atherogenic Indices of Female and Male Subjects with Type 2 Diabetes Mellitus

	Female (n=54)	Male (n=36)	Test Statistics	
Measure	Mean ± SD	Mean ± SD	T-Ratio	Prob> |t|
AIP	-0.02±0.04	0.15±0.04	2.9631	0.0039**
CRI-1	3.90±0.17	4.35±0.20	1.7232	0.0884
CRI-11	2.32±0.14	2.60±0.17	1.2952	0.1986
AC	2.90±0.17	3.35±0.20	1.7232	0.0884

Key: *AIP:Atherogenic index of plasma, CRI-1: Castelli Risk Index 1, CRI-11: Castelli Risk Index 11, and AC: Atherogenic coefficient.*
*Significance Level: **=p<0.01.*

It has been proposed that Lipid profile pose a risk for the development of atherosclerosis, suggesting it to be a potential criterion for subclinical detection of atherosclerosis since the accumulation of cholesterol observed in atherosclerotic lesion originates basically from plasma lipoproteins, especially LDL. On the other hand, dyslipidemia has been recognized as an important risk factor associated with coronary artery disease. It is worth to note that beneficial changes of atherogenic lipid profile occur with optimal management of T2DM. Recently, the function of lipid ratios as risk indices involving the lipids/lipoproteins has been eminent. More than half of the subjects reached a HbAIc target of <6.5% whereas 16% had HbAIc target of ≥6.5%. It was also observed in this study that age, sex and BMI lacked effect on HbA1c level. An introspection of the interpretations from clinical assessment and perspectives, shows that total cholesterol (TC), low-density lipoprotein cholesterol fraction (LDLc), triglycerides (TG) and high-density lipoprotein cholesterol fraction (HDLc) were all normal and less than 5.17mmol/l, 3.5mmol/l, 1.7mmol/l and greater than 0.9mmol/L respectively in the entire population. HDL-C (0.00131) and TG (0.0257) levels observed a significant difference between both sexes, with the females having a higher HDL-C and lower TG levels than males, the significant increase of triglycerides could be attributed to overproduction of VLDL through a process of exchange enhanced by cholesterol ester transfer protein and increased hepatic triglyceride lipase activity further resulting in lower levels of HDL-c. Furthermore, a study of lipid profile levels of T2DM did not observe any difference in lipid profile parameters between both sex and attributed it to the degree of insulin resistance observed between both sex and an effect of hormone on enzymes implicated in lipid protein metabolism [23]. However, no significance difference was observed in both sex for FBS, HbAIc, TCHOL, Non-HDL-C, LDL-C, VLDL-C, insulin, HOMA-IR, HOMA-β and Lipoprotein A (Table 2). In Table 3, AIP, was significantly reduced in females when compared to males. AIP in predicting cardiovascular risk revealed that the female group was within low risk (<0.1) while the males were in intermediate risk level (0.1-0.24) thereby placing the males at a higher risk of developing coronary heart disease, this finding is consistent with that of Niroumand et al. [24]. AIP has been shown to be a strong marker to predict atherosclerosis and coronary heart disease. It serves as an easy method to identify patients within a populace of increased risk of coronary artery disease, insulin resistance and T2DM with sophisticated cardiometabolic risk.

4. CONCLUSION

There was an increased HDL-C and Lipo-A values in the female subjects and a decreased levels in TG and AIP when compared with their male counterparts.

ACKNOWLEDGEMENTS

Authors would like to sincerely thank Adele Uyota Anthony, AyibatonyeOrutugu and Dr(Mrs) Ibiene Sarah Kalio for helping with the sample collection.Thank you for your support, enthusiasm and passion for research that has made this an amazing experience. I sincerely appreciate the support of Dr Nwachukwu O.Ekpete for the laboratory analysis and also grateful for the invaluable input of Dr ElekimaIbioku for his help with data analysis and for his continued support in the project

COMPETING INTERESTS

Authors have declared that no competing interests exist.

AUTHORS' CONTRIBUTIONS

Author BES, NEO and TDG designed the study and wrote the protocol, author RI wrote the first draft of the manuscript and managed the analyses of the study and managed the literature searches. All authors read and approved of the final manuscript.

CONSENT

All authors declare that 'written informed consent was obtained from the patient (or other approved parties) for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editorial office/Chief Editor/Editorial Board members of this journal.

ETHICAL APPROVAL

Ethical approval and permission were sought and obtained from the Rivers State Health Research Ethics Committee of Rivers State Health Management Board. Informed consent of the participants involved was obtained and anthropometric data was also obtained via a questionnaire.

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