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Elevated high-sensitivity C-reactive protein and dyslipidaemia in type 2 diabetes mellitus: implications for cardiovascular risk prediction in Nigerian patients

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Abstract

Background Cardiovascular disease (CVD) is a leading cause of morbidity and mortality among individuals with type 2 diabetes mellitus (T2DM). Inflammation, marked by elevated high-sensitivity C-reactive protein (hs-CRP) levels, and dyslipidaemia, are critical contributors to atherosclerosis and cardiovascular risk. In Nigeria, where T2DM prevalence is rising, there is a need for more comprehensive risk prediction tools, incorporating both traditional and newer biomarkers such as hs-CRP. This study aimed to investigate the association between elevated hs-CRP levels and dyslipidaemia in Nigerian patients with T2DM and to explore the potential implications for cardiovascular risk prediction.

Methods A hospital-based cross-sectional study was conducted among 150 T2DM patients and 150 age-matched controls. Data on socio-demographics, medical history, clinical characteristics, and laboratory parameters, including lipid profiles and hs-CRP levels, were collected. The relationship between hs-CRP levels and lipid parameters was assessed using Pearson's correlation coefficient and independent t-tests.

Results T2DM patients exhibited significantly higher hs-CRP levels (2.2 ± 1.8 mg/L vs. 1.2 ± 1.0 mg/L, $p < 0.001$), dyslipidaemia ($p < 0.001$), and blood pressure (SPB– 127.6 ± 12.4 mmHg, DBP– 77.6 ± 6.6 mmHg vs. SBP– 119.6 ± 10.8 mmHg, DBP– 72.1 ± 8.0 mmHg; $p = 0.001$) compared to controls. However, no significant correlation was found between hs-CRP levels and lipid parameters.

Conclusion Although no direct association was found between elevated hs-CRP levels and dyslipidaemia, hs-CRP remains an important marker of cardiovascular risk possibly through non-lipid pathways, such as inflammation-driven endothelial dysfunction. Further research is needed to evaluate its potential role in refining cardiovascular risk assessment in the Nigerian T2DM population.

Clinical trial number Not applicable.

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Keywords Cardiovascular risk, Dyslipidaemia, High-sensitivity C-reactive protein, Hypertension, Systemic inflammation, Nigerian patients, Type 2 diabetes

Introduction

Cardiovascular disease (CVD) remains a leading cause of morbidity and mortality among individuals with type 2 diabetes mellitus (T2DM) globally, particularly in low- and middle-income countries like Nigeria [1]. The interrelationship between diabetes and cardiovascular risk is well-established, with multiple pathophysiological mechanisms contributing to the increased risk observed in diabetic patients. Among these, chronic low-grade inflammation and dyslipidaemia are recognized as pivotal factors in the development and progression of atherosclerosis, the underlying cause of most cardiovascular events [2]. Considering the high burden of diabetes and its complications in Nigeria, accurately identifying those at increased risk of CVD is essential to guide effective preventive measures and mitigate the overall impact of the disease.

High-sensitivity C-reactive protein (hs-CRP), a marker of systemic inflammation, has garnered considerable attention as a potential predictor of cardiovascular events, with elevated levels correlating strongly with increased CVD risk, independent of traditional risk factors [3]. Conversely, dyslipidaemia characterized by elevated triglycerides, low high-density lipoprotein cholesterol (HDL-C), and the presence of small dense low-density lipoprotein cholesterol (sdLDL-C) particles is a common metabolic disturbance in T2DM patients that accelerates atherogenesis [4]. These lipid abnormalities not only exacerbate cardiovascular risks but are also influenced by the inflammatory state indicated by elevated hs-CRP levels. Thus, this temporal relationship between systemic inflammation and dyslipidaemia in T2DM creates a synergistic effect, further amplifying the development of atherosclerosis and consequently increasing cardiovascular risk in diabetic individuals [5, 6].

In Nigeria, the prevalence of T2DM is on the rise, accompanied by a concomitant increase in CVD-related morbidity and mortality [7, 8]. However, the current state of CVD risk management in Nigeria, especially among diabetic patients, presents significant challenges [9]. The most commonly used traditional risk prediction models, are often based on Western populations and may not be entirely applicable to Nigerian patients due to differences in genetic, environmental, and socio-economic factors. Consequently, there is a notable lack of locally adapted risk prediction tools designed specifically for the Nigerian population. The rising prevalence of CVD risk factors such as diabetes and hypertension, combined with limited integration of advanced biomarkers like hs-CRP into the commonly used risk assessment frameworks, highlights the urgent need for more comprehensive and culturally relevant prediction approaches.

The integration of hs-CRP into CVD risk prediction approaches largely reliant on lipid profiles could be particularly valuable in the Nigerian setting, where early identification of high-risk individuals is crucial for effective intervention. Despite the recognized potential, there is a paucity of data on the utility of hs-CRP as a cardiovascular risk predictor in Nigerian patients with T2DM, especially in relation to dyslipidaemia. This limits the development of tailored risk assessment tools and targeted preventive strategies. This study was aimed at investigating the association between elevated hs-CRP levels and dyslipidaemia in Nigerian patients with T2DM and reviewing the potential implications of these findings for cardiovascular risk prediction in the study population. By evaluating these relationships, this study aims to enhance cardiovascular risk prediction, develop more targeted interventions, and improve patient outcomes through more effective CVD risk assessment and management strategies.

Materials and methods

Study design and setting

This hospital-based study was conducted between October 2020 and October 2021 (13 months) and employed an analytical non-interventional cross-sectional and controlled design to investigate the association between elevated hs-CRP levels and dyslipidaemia in Nigerian patients with T2DM attending the endocrinology clinic of the medical out-patient clinic at National Hospital, Abuja (NHA). The control participants were recruited from the general outpatient clinic, among patients who present for routine check-ups and are non-diabetic with FPG < 5.6 mmol/L. The study was conducted in compliance with the Declaration of Helsinki [10]. Number codes were allotted to each recruited participant, and their clinical data and test results were locked out in secured spaces to ensure confidentiality throughout the study.

The sample size was determined using a prevalence of T2DM and that of dyslipidaemia in T2DM from previous studies in the North-central region of Nigeria [11, 12], a margin of error of 0.05, and a confidence interval of 95%. Using the formula for calculating sample size in cross-sectional studies [13], and adjusting for a 10% non-response rate, a minimum sample size of 62 was determined to be necessary for this study. However, a sample size of 150 was chosen to ensure sufficient statistical power for detecting associations between hs-CRP

levels and dyslipidaemia and allow for sub-group analyses, while also accounting for potential variability in the study population. A convenience sampling technique was employed and patients who met the inclusion criteria were consecutively recruited until the target sample size was achieved.

Inclusion and exclusion criteria

This study's participants included T2DM patients between the ages of 30 and 65 years on oral hypoglycaemic medications with age-matched non-diabetic patients with fasting plasma glucose (FPG) levels < 5.6 mmol/L serving as controls. Patients with comorbidities such as cardiovascular, renal, and liver diseases were excluded because these conditions independently influence hs-CRP and lipid metabolism, potentially confounding the study outcomes. Similarly, individuals with acute or chronic inflammatory disorders (e.g., rheumatoid arthritis, systemic lupus erythematosus) were excluded to prevent misinterpretation of elevated hs-CRP levels.

Participants using medications known to affect hs-CRP and lipid levels (e.g., steroids, oral contraceptives, NSAIDs, aspirin) were also excluded to eliminate pharmacological influences on inflammatory and lipid parameters. Additionally, cigarette smokers, and individuals with significant alcohol consumption, defined as seven standard drinks per week or more than three standard drinks per day [14], were also excluded as both are known to alter systemic inflammation and lipid metabolism, which could impact study findings.

Data collection and interpretation

Data on participant's socio-demographic parameters, medical history, medications, and clinical characteristics were collected via the use of a structured research proforma, and trained research assistants guided participants in completing the proforma to ensure accuracy and completeness. The blood pressure was measured in mmHg with AccuSure Mercury Sphygmomanometer BP Monitor. An average of two readings of both systolic blood pressure (SBP) and diastolic blood pressure (DBP) taken at 5-minute intervals was recorded and entered into the data collection form for each participant. The participant's weight in kilogram (kg) and height in meters (m) were measured using the SECA weighing balance with height attachment to the nearest decimal, wearing light clothing without shoes and the body mass index (BMI) was calculated as weight (kg)/height in meters squared (m^2).

In this study, dyslipidaemia was defined by one or more of the following criteria: total cholesterol level ≥ 5.17 mmol/L (≥ 200 mg/dL), plasma LDL cholesterol ≥ 2.6 mmol/L (≥ 100 mg/dL), plasma triglyceride ≥ 1.7 mmol/L (≥ 150 mg/dL), and/or plasma HDL cholesterol < 1 mmol/L (< 40 mg/dL) for men or < 1.3 mmol/L (< 50 mg/dL) for women [15]. Well-controlled type 2 diabetes mellitus (T2DM) was defined as having an HbA1c level < 7%, while poorly controlled T2DM was defined as an HbA1c level ≥ 7% [16]. Also, hs-CRP levels > 3 mg/L were categorized as high [17], and obesity was defined as a body mass index (BMI) of ≥ 30 kg/m^2 [18].

Sample collection and assays

Blood samples were drawn from participants using aseptic techniques after an overnight fast of at least 8 h into five-millilitre vacutainers. To ensure accurate and reliable results, samples for FPG were collected into a fluoride oxalate container and HbA1c into EDTA container, while samples for lipid profile and hs-CRP were collected into lithium heparin containers. The samples were centrifuged at 5000 revolutions per minute (rpm) for 5 min after collection and the plasma/serum samples were aliquoted in plain cryovial tubes.

Glucose, Total cholesterol, HDL-cholesterol, and Triglyceride were measured immediately in the fluoride oxalate plasma and lithium heparin plasma, respectively while LDL-cholesterol was calculated using the Friedewald formula. The EDTA sample and aliquot of heparinised plasma (for HbA_{1c} and hs-CRP) were stored at -25^{0}C, for three months until the analyses were performed. All assays were performed using the Cobas c311® automated random-access analyzer for clinical chemistry and homogenous immunology assay (HIA) (Roche Diagnostics, Mannheim, Germany).

Statistical analysis

Data were analyzed using the Statistical Package for the Social Sciences (SPSS) version 25 (IBM Corp., Armonk, NY, USA). Descriptive statistics were used to summarize the demographic and clinical characteristics of the study population. The normality of the distribution was assessed using the Shapiro-Wilk test. Continuous variables were expressed as mean ± standard deviation (SD) while categorical variables were presented as frequencies and percentages. For comparisons between T2DM patients and control individuals, independent t-tests were used for continuous variables, while the chi-square test (or Fisher's exact test, where appropriate) was employed to compare categorical variables between groups.

The relationship between hs-CRP levels and lipid parameters was evaluated using Pearson's correlation coefficient while the point biserial correlation was used to assess its relationship with sex. Participants were stratified into two groups based on their hs-CRP levels: elevated hs-CRP (≥ 3 mg/L) and normal hs-CRP (< 3 mg/L), and the differences in lipid parameters between these groups were analyzed using independent t-tests. Subgroup analyses were conducted to explore the potential

effect modifications by gender, BMI, glycaemic control (HbA1c ≥ 7% vs. <7%), and duration of diabetes (< 5 years vs. ≥5 years). All statistical tests were two-sided and a *p*-value of < 0.05 was considered statistically significant for all analyses.

Results

A total of 300 individuals participated in this study, of whom 150 were known T2DM patients while 150 served as healthy age-matched controls. The baseline socio-demographic and clinical characteristics of the study participants presented in Table 1 revealed several significant differences between the two groups. Participants with T2DM had notably higher levels of systemic inflammation, as indicated by elevated hs-CRP levels (2.2 ± 1.8 mg/L versus 1.2 ± 1.0 mg/L; $p < 0.001$), alongside higher systolic and diastolic blood pressure (SPB– 127.6 ± 12.4 mmHg, DBP– 77.6 ± 6.6 mmHg versus SBP– 119.6 ± 10.8 mmHg, DBP– 72.1 ± 8.0 mmHg; $p = 0.001$). In addition, the T2DM group exhibited significant dyslipidaemia, with increased total cholesterol (183.6 ± 50.4 mg/dL versus 165.8 ± 24.8 mg/dL, $p < 0.001$), triglycerides (113.9 ± 48.2 mg/dL versus 100.9 ± 35.0 mg/dL, $p = 0.008$), and LDL-cholesterol (111.0 ± 41.7 mg/dL versus 96.0 ± 22.4 mg/dL, $p < 0.001$) compared to the controls. Markers of glycaemic control also differed significantly, with the T2DM group showing higher fasting blood glucose (7.6 ± 2.6 mmol/L versus 4.9 ± 0.3 mmol/L, $p < 0.001$) and HbA1c levels (7.6 ± 1.9% versus 4.9 ± 0.2%, $p < 0.001$), reflecting poor glucose regulation.

When adjusting for BMI and SBP among T2DM patients, hs-CRP showed no significant correlation with total cholesterol ($r = 0.001$, $p = 1.000$), triglycerides ($r = 0.067$, $p = 0.416$), LDL-cholesterol ($r = 0.021$, $p = 0.799$), or HDL-cholesterol (r = -0.051, $p = 0.536$). However, further analysis within the T2DM cohort revealed a weak but significant positive correlation between hs-CRP and BMI ($r = 0.296$, $p = 0.000$) in T2DM patients, while no significant correlations were observed with sex, HbA1c, or duration of diabetes.

In the comparison of clinical and biochemical characteristics of T2DM patients based on hs-CRP categories (Table 2), those with elevated hs-CRP levels (> 3 mg/L) had a significantly higher BMI compared to those with normal hs-CRP levels (29.9 ± 6.3 kg/m² versus 27.9 ± 4.4 kg/m², $p = 0.027$). Additionally, SBP was notably

Table 1 Socio-demographic, clinical and biochemical characteristics of the study participants

Participant Characteristics	T2DM group (*N* = 150) Mean ± SD or *n* (%)	Control group (*N* = 150) Mean ± SD or *n* (%)	*p*-Value
Age (years)	52.8 ± 9.1	49.6 ± 9.6	0.101
Sex:	-	-	0.204
Male	66 (44.0)	77 (51.3)	
Female	84 (56.0)	73 (48.7)	
Occupation:	-	-	0.119
Civil Servants	57 (38.0)	89 (59.3)	
Traders	46 (30.7)	27 (18.0)	
Unemployed	15 (10.0)	24 (16.0)	
Retiree	30 (20.0)	4 (2.7)	
Others	2 (1.3)	6 (4.0)	
Educational status:	-	-	0.309
Primary	16 (10.6)	21 (14.0)	
Secondary	81 (54.1)	72 (48.0)	
Tertiary	53 (35.3)	57 (38.0)	
Marital Status:	-	-	0.222
Single	37 (24.7)	47 (31.3)	
Married	70 (46.7)	65 (43.3)	
Divorced	43 (28.6)	38 (25.4)	
BMI (kg/m^2)	28.6 ± 5.1	27.8 ± 6.7	0.283
Blood Pressure (mmHg):	-	-	0.001*
Systolic	127.6 ± 12.4	119.6 ± 10.8	
Diastolic	77.6 ± 6.6	72.1 ± 8.0	
hs-CRP (mg/L)	2.2 ± 1.8	1.2 ± 1.0	0.000*
Total Cholesterol (mg/dL)	183.6 ± 50.4	165.8 ± 24.8	0.000*
HDL-Cholesterol (mg/dL)	49.5 ± 3.4	50.4 ± 7.4	0.477
Triglyceride (mg/dL)	113.9 ± 48.2	100.9 ± 35.0	0.008*
LDL-Cholesterol (mg/dL)	111.0 ± 41.7	96.0 ± 22.4	0.000*
FBG (mmol/L)	7.6 ± 2.6	4.9 ± 0.3	0.000*
HbA1c (%)	7.6 ± 1.9	4.9 ± 0.2	0.000*

**p-value significant < 0.05; N = Number of participants in the group, n = Number of participants within the group, SD = Standard deviation*

Table 2 Comparison of clinical and biochemical characteristics of T2DM patients based on categories of hs-CRP

Participant Characteristics	Normal (< 3 mg/L) hs-CRP (*N* = 96) Mean ± SD or *n* (%)	Elevated (> 3 mg/L) hs-CRP (*N* = 54) Mean ± SD or *n* (%)	*p*-Value
Age (years)	51.7 ± 9.6	53.9 ± 8.3	0.155
Sex:	-	-	0.865
Male	43 (44.8)	23 (42.6)	
Female	53 (55.2)	31 (57.4)	
BMI (kg/m^2)	27.9 ± 4.4	29.9 ± 6.3	0.027*
Blood Pressure (mmHg):	-	-	-
Systolic	126.0 ± 12.1	130.4 ± 12.6	0.040*
Diastolic	77.2 ± 6.3	78.5 ± 7.4	0.244
Duration of Diabetes (years)	6.7 ± 5.9	8.1 ± 5.7	0.164
Anti-Diabetic drugs:	-	-	0.160
Yes	92 (95.8)	54 (100.0)	
No	4 (4.2)	0 (0.0)	
Anti-hypertensive drugs:	-	-	0.000*
Yes	45 (46.9)	45 (83.3)	
No	51 (53.1)	9 (16.7)	
Anti-hyperlipidemic drugs:	-	-	0.604
Yes	84 (87.5)	49 (90.7)	
No	12 (12.5)	5 (9.3)	
FPG (mmol/L)	7.5 ± 2.3	7.7 ± 3.2	0.649
HbA1c (%)	7.6 ± 1.9	7.7 ± 1.8	0.694
Total Cholesterol (mg/dL)	186.2 ± 55.5	179.1 ± 40.0	0.408
HDL-Cholesterol (mg/dL)	50.6 ± 12.2	47.6 ± 15.4	0.199
Triglyceride (mg/dL)	110.1 ± 44.3	119.5 ± 54.6	0.295
LDL-Cholesterol (mg/dL)	113.0 ± 46.4	107.6 ± 40.0	0.450

**p-value significant < 0.05; N = Number of participants in the group, n = Number of participants within the group, SD = Standard deviation*

higher in the elevated hs-CRP group (130.4 ± 12.6 mmHg versus 126.0 ± 12.1 mmHg, $p = 0.040$). A significantly higher proportion of patients with elevated hs-CRP were on antihypertensive medication (83.3% vs. 46.9%, $p < 0.001$). Other variables, including age, sex, duration of diabetes, fasting plasma glucose (FPG), HbA1c, and lipid profile parameters, showed no significant differences between the two categories.

Discussion

In this study, the higher levels of systemic inflammation, hypertension, significant dyslipidaemia, and poor glucose control observed in patients with T2DM are reflective of the complex and interconnected pathophysiological mechanisms underlying the disease and its associated cardiovascular risks. Chronic low-grade inflammation, characterized by increased levels of inflammatory cytokines, is a hallmark of T2DM and plays a key role in endothelial dysfunction, atherogenesis, and insulin resistance [19]. Inflammation promotes the activation of the renin-angiotensin-aldosterone system, leading to vasoconstriction and increased blood pressure [20, 21]. This explains the significant association between elevated hs-CRP levels and hypertension among T2DM patients as observed in the study. Also, insulin resistance (IR), a core defect in T2DM, disrupts normal lipid metabolism, leading to an overproduction of very low-density lipoprotein (VLDL) and sd-LDL particles, which are more atherogenic [22]. This dyslipidaemia is exacerbated by poor glucose control leading to an increase in free fatty acid flux from adipose tissue to the liver, further promoting the production of VLDL which contributes to the development of atherosclerosis and perpetuates a vicious cycle [23]. Previous and recent studies have shown that elevated hs-CRP levels are strongly associated with an increased risk of CVD, independent of traditional risk factors such as dyslipidaemia, and that tight glycaemic control reduces cardiovascular events in T2DM patients [24–26].

This study aimed to explore the association between systemic inflammation (elevated hs-CRP) and dyslipidaemia in Nigerian patients with T2DM, and the lack of an association observed may be due to the intricate and multifactorial nature of dyslipidaemia in diabetes. Dyslipidaemia is primarily driven by insulin resistance, with inflammation playing a potentially secondary role [27]. Other factors unique to the Nigerian population such as genetics and diet might also influence lipid levels in T2DM, diluting the impact of inflammation on lipid metabolism. Additionally, hs-CRP might reflect increased CVD risk through pathways unrelated to dyslipidaemia, such as endothelial dysfunction and plaque instability. This finding diverges from some previous

studies, which have reported a significant association between elevated hs-CRP levels and dyslipidaemia in T2DM patients [5, 28], while agreeing with a few others where no association was found [29, 30]. The variability in findings across different populations suggests that the relationship between inflammation and dyslipidaemia may be modulated by other factors. For instance, variations between previous studies and the present study were observed in study design, population demographics, and baseline inflammatory status. Notably, many studies reporting a significant association included participants with higher inflammatory states, whereas the mean hs-CRP levels in our study were below the 3 mg/L threshold. Additionally, differences in genetic predisposition, lifestyle factors, and healthcare access across populations may further contribute to these discrepancies.

The lack of association between elevated systemic inflammation (hs-CRP) and dyslipidaemia in this study and previous similar studies may suggest that relying solely on lipid profiles for CVD risk prediction could be insufficient in this population. Hs-CRP, as an independent marker of systemic inflammation, might capture cardiovascular risk through pathways unrelated to lipid metabolism, such as endothelial dysfunction and arterial stiffness, which are also significant contributors to atherosclerosis and CVD. Therefore, hs-CRP may serve as a complementary biomarker, warranting further investigation in larger, longitudinal studies. Further research is needed to determine its potential role in refining cardiovascular risk assessment strategies tailored to Nigerian T2DM patients, particularly in combination with traditional lipid-based measures and other relevant population-specific factors like genetics and environmental influences unique to Nigeria.

Further analysis among T2DM patients revealed a significant positive correlation between elevated hs-CRP levels and BMI, with patients in the elevated hs-CRP group also showing higher SBP, a characteristic phenotype of hypertension in T2DM, and a greater likelihood of using antihypertensive medications. This finding aligns with the established pathophysiological link between obesity, inflammation, and cardiovascular risk, as adipose tissue in obese individuals secretes pro-inflammatory cytokines such as interleukin-6, which stimulates hepatic production of C-reactive protein (CRP), thereby promoting systemic inflammation and endothelial dysfunction [31–34]. Elevated SBP further exacerbates cardiovascular risk by increasing arterial stiffness and promoting atherogenesis [35]. These results are consistent with other studies that highlight the strong association between obesity, inflammation, and hypertension in T2DM populations [31, 36]. However, unlike certain Caucasian populations where dyslipidaemia has been closely associated with systemic inflammation, this study found no such correlation between hs-CRP and lipid parameters [5, 37]. This discrepancy may be attributed to variations in genetic predispositions, differences in access to healthcare, and distinct lifestyle factors across populations.

There are some limitations to the index study. The cross-sectional design limits the ability to draw causal inferences between elevated hs-CRP levels and cardiovascular risk factors in T2DM patients, suggesting the need for longitudinal studies to assess these associations over time. Additionally, being a hospital-based study, the findings may not fully represent the broader Nigerian T2DM population, limiting their generalizability. As such, future research should include larger, more diverse samples from multiple healthcare centres across different regions for a more comprehensive understanding. Lastly, although we excluded patients with conditions known to influence hs-CRP levels, residual confounding from unmeasured factors, such as diet, stress, and genetic predisposition, cannot be ruled out. Incorporating these variables into future studies would provide a clearer picture of the complex relationship between inflammation, dyslipidaemia, and cardiovascular risk in T2DM patients.

Conclusion

This study assessed the relationship between systemic inflammation, dyslipidaemia, and cardiovascular risk in Nigerian patients with T2DM. Despite the absence of a significant association between elevated hs-CRP levels and dyslipidaemia, it suggests that hs-CRP remains an important marker of cardiovascular risk through non-lipid pathways, such as inflammation-driven endothelial dysfunction. This highlights the need for further research to clarify its potential role in refining CVD risk assessment strategies for the Nigerian T2DM population.

Abbreviations

CVD	Cardiovascular disease
T2DM	Type 2 diabetes mellitus
hs-CRP	High-sensitivity C-reactive protein
HDL-C	High-density lipoprotein cholesterol
LDL-C	Low-density lipoprotein cholesterol
sd-LDL-C	Small dense low-density lipoprotein cholesterol
VLDL	Very-low-density lipoprotein
BMI	Body mass index
FPG	Fasting plasma glucose
HbA1c	Glycated hemoglobin
NSAIDs	Non-steroidal anti-inflammatory drugs
SBP	Systolic blood pressure
DBP	Diastolic blood pressure
EDTA	Ethylenediaminetetraacetic acid
SD	Standard deviation
SPSS	Statistical package for the social sciences

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Author contributions
All authors collaborated on this research project. MJA, MJAF and YIA conceived and designed the study, and were involved in data acquisition, analysis, and interpretation. BB and MIN contributed significantly to the study design and data interpretation. AND and LAO contributed to data collection and analysis. All authors participated in drafting and critically revising the manuscript. They collectively approved the final version for publication and accepted responsibility for all aspects of the work.

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Data availability
The datasets from this study will be available upon reasonable request to the corresponding author. This is because the dataset includes additional data that are not relevant to this study and may require exclusion.

Declarations

Ethics approval and consent to participate
This study was approved by the Health Research Ethics Committee of the National Hospital, Abuja with protocol number NHA/EC/076/2019 on 12th September 2019, and was conducted in compliance with the Declaration of Helsinki. Informed and written consent was obtained from all participants before inclusion in the study and data collection.

Consent for publication
Not Applicable in this study.

Competing interests
The authors declare no competing interests.

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