

Determination of Serum Lipid Profile and Atherogenic Indices of Subjects with Diabetic -Hypertensive Comorbidity in Some Tertiary Institutions in Bayelsa State

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ABSTRACT

Introduction: Diabetic- hypertensive comorbidity poses significant health risk to the development or complications in cardiovascular disease (CVD). **Aim:** This study aims to evaluate the influence of diabetes and hypertension on serum lipid profiles and atherogenic indices among subjects attending some tertiary health institutions in Bayelsa State. **Method:** A cross-sectional research design was employed with 200 subjects, 50 apparently healthy adults, 50 subjects with diabetes, 50 with hypertension and 50 with diabetic-hypertensive comorbidity. Serum lipid levels; total cholesterol, triglycerides, high density lipoprotein, low density lipoprotein were measured using standard colorimetric method. Atherogenic indices, including the Castelli Risk index and the Atherogenic Coefficient (AC) were calculated. Data obtained were analyzed using Graphpad Prism Version 9.0.0. (121). **Results:** Results revealed significantly elevated total cholesterol and LDL levels among diabetic-hypertensive subjects compared to other groups ($p < 0.001$). HDL levels were significantly lower in the diabetic- hypertensive group, indicating profound dyslipidemia. Atherogenic indices including the Castelli risk index and Atherogenic Coefficient were significantly higher in the diabetic-hypertensive subjects reflecting an increased risk for cardiovascular disease or abnormalities. HTN/DM group demonstrated significantly higher CRI-I, CR-II and AC compared to the hypertensive group, suggesting a greater imbalance between atherogenic and protective lipoproteins. Duration-based comparisons revealed that subjects with longer disease duration (6–10 years) generally exhibited more adverse lipid patterns than those with shorter duration (1–5 years), especially among hypertensive and HTN/DM participants. **Conclusion:** This study emphasize the importance of regular lipid monitoring and integrated clinical strategies to manage subjects with diabetic hypertensive comorbidity, thus reducing adverse health outcomes.

Keywords: Diabetes, Hypertension, Lipid Profiles, Atherogenic Indices, Cardiovascular Risk, Nigeria.

INTRODUCTION

Diabetes mellitus and hypertension are two prevalent chronic diseases that often coexist, exacerbating cardiovascular risks. Diabetes is characterized by impaired glucose regulation due to insufficient insulin production or action, while hypertension involves consistently elevated arterial blood pressure (1,2). The relationship between these conditions enhances the risk of cardiovascular diseases (CVD), with studies showing that patients with both conditions face significantly higher morbidity and mortality rates (3, 4).

Although diabetes is a global issue, its rise is particularly pronounced in low- and middle-income settings such as Nigeria, where prevalence rates continue to climb (5, 6). Hypertension commonly accompanies diabetes, and extensive research identifies elevated blood pressure as a major contributor to both the development and the severity of diabetic complications, underscoring the close association between these two chronic diseases (7, 8). Recent global statistics highlight the growing health impact of diabetes. Diabetic conditions contribute to about 1.6 million deaths annually (9). Data from the

International Diabetes Federation indicate that, in 2021, approximately 537 million adults, representing 10.5% of the world's adult population, were living with diabetes, with type 2 diabetes accounting for the overwhelming majority of cases. Projections show a continued rise, forecasting that by 2045 the number of adults affected will reach roughly 783 million, or one in every eight people globally, an estimated 46% increase from current levels (10,11).

It has been estimated that 35-75% of diabetic complications can be attributed to hypertension. In contrast, the absence of hypertension is the usual finding in long-term survivors of diabetes. Thus, the coexistence of these two diseases likely contributes substantially to overall mortality in industrialized societies (4, 12). Without timely treatment, diabetes can predispose individuals to a wide range of complications, particularly those affecting cardiovascular function (13).

Hypertension is a long-term medical condition characterized by consistently elevated arterial blood pressure (14). It significantly increases the likelihood of serious health complications, including stroke, coronary artery disease, heart failure, atrial fibrillation, peripheral artery disease, vision impairment, chronic kidney disease, and cognitive decline (15). Globally, high blood pressure is a leading contributor to premature mortality (16).

Individuals with diabetes are particularly susceptible, with the prevalence of hypertension in this population roughly twice that of non-diabetics (4). Studies indicate that among people with type 2 diabetes, which comprises over 90% of all diabetes cases, between 50% and 80% develop hypertension. In contrast, around 30% of individuals with type 1 diabetes experience elevated blood pressure during their lifetime (5, 17). This close relationship underscores the need for careful cardiovascular monitoring in diabetic patients.

Evaluation of lipid profile has been considered as a useful tool to assess the risk of developing cardiovascular disease (CVD) in patients including diabetic patients as dyslipidemia is one of the major risk factors for cardiovascular disease in Type 2 Diabetes mellitus and hypertension comorbidity, characterized by elevated Total cholesterol (TC), Triglycerides (TG), Low density lipoprotein (LDL) and decreased High density lipoprotein (HDL) (18). Since the detection and treatment of dyslipidemia play a crucial role in reducing cardiovascular disease (CVD) risk, assessing serum lipid levels in individuals with diabetes has become a standard component of diabetes care (19). Research findings indicate that among body composition factors, excess body fat is the primary contributor to the increasing prevalence of dyslipidemia in diabetes (20, 21).

Type 2 diabetes is associated with a cluster of interrelated lipid and lipoprotein abnormalities,

including reduced HDL cholesterol, an increased proportion of small, dense LDL particles, and elevated triglycerides (22, 23). Evidence suggests that each of these abnormalities contributes to an increased risk of cardiovascular disease, which remains the leading cause of mortality among patients with diabetes and hypertension (23,24).

Beyond traditional cardiovascular risk markers in diabetic-hypertensive patients such as lipid profile and the acute-phase protein C-reactive protein (CRP), the identification of additional independent markers could enhance early detection and risk assessment. Given the rising prevalence and mortality associated with this comorbidity, particularly among high-risk individuals, there is a pressing need for more sensitive and early biomarkers to improve diagnosis and management (25). Monitoring these lipid parameters can provide critical insights into cardiovascular risk and guide preventive or therapeutic interventions. Monitoring and managing dyslipidemia is essential for minimizing the risk of cardiovascular disease (CVD), making the evaluation of serum lipid levels a routine aspect of diabetes management (26).

In diabetes, dyslipidemia commonly presents as a combination of elevated triglycerides, reduced levels of high-density lipoprotein (HDL) cholesterol, and an abundance of small, dense low-density lipoprotein (LDL) particles (19, 27). These lipid disturbances arise primarily from insulin resistance or insulin deficiency, which interfere with the normal function of enzymes and metabolic pathways that regulate lipid homeostasis (28).

Elevated blood glucose levels, excess body fat, and impaired insulin regulation contribute markedly to the acceleration of atherosclerotic processes in individuals with diabetes (29). In type 2 diabetes, lipid and lipoprotein abnormalities often occur together, including lower levels of HDL cholesterol, a higher proportion of small, dense LDL particles, and increased triglyceride concentrations (23). These interrelated disturbances play a significant role in enhancing cardiovascular risk among affected patients. Importantly, these lipid abnormalities may be present even in patients whose LDL cholesterol levels are within the normal range. Research indicates that each of these dyslipidemia changes independently elevates the risk of cardiovascular disease, which continues to be the primary cause of death among individuals with both diabetes and hypertension (23).

MATERIALS AND METHODS

Study Area- The study was conducted at Niger Delta University Teaching Hospital, Okolobiri, Yenagoa LGA, Bayelsa State, Nigeria and Federal Medical Centre, Yenagoa, Bayelsa State. Yenagoa Local Government Area is geographically located in the Niger Delta region of Southern Nigeria approximately between within

longitude 6° 07'35" E and 6° 24'00" E and the latitude is 4° 47'15"N and 5° 11;55" N. It has an area of 706 km² (30) with a projected population of approximately 524,400 persons (31).

Study Population

The subjects in this study comprised of apparently healthy male and female adults between the ages of 30 to 69 years. A total of 200 participants were recruited for the study. Fifty (50) participants were apparently healthy subjects without diabetes, hypertension or diabetes and hypertension comorbidity, another 50 participants were patients with diabetes without hypertension. Moreso, 50 participants were patients with hypertension without diabetes, while another 50 participants were diabetic-hypertensive patients all residing in Bayelsa State, Nigeria and either attending NDUTH Okolobiri and FMC Yenagoa.

Sample Size Determination

Simple randomized method was used in selection of subjects. The sample size for this study was determined using G*Power statistical software. The parameters were set as follows:

Effect size (f) = 0.30, -Power (1 – β err prob) = 0.95, - α error probability = 0.05, Number of groups = 4. Based on these inputs, G*Power calculated a total sample size of 196 participants. To enhance the statistical robustness and allow for potential data loss, the sample size was increased to 200 participants, with 50 participants assigned to each group in the study.

Inclusion Criteria

- known diabetic and hypertensive patients currently attending diabetic and cardiovascular clinic in Niger Delta University Teaching Hospital (NDUTH), Okolobiri, Bayelsa State, Nigeria and FMC Yenagoa with a fasting blood sugar level above 10 mmol/L, HbA1c >5.5% and blood pressure of over 150/90mmHg for the comorbidity group.
- Only known diabetics attending the diabetic clinic with a fasting blood glucose above 10mmol/L and HbA1c >6.5% were included into the diabetic group.
- Only known hypertensive patents attending the clinic with a blood pressure of ≥ 150/90mmHg were included into the hypertensive group.

Exclusion Criteria

Participants who did not give informed consent were excluded from this study. Participants were also excluded if they are alcoholics, suffering from any other coronary heart disease such as ischemic heart disease and myocardial infarction that can interfere with lipid

parameters and at the time of the blood sampling, were on medication or were found to be drug addicts.

Sample Collection, Transportation, Processing and Preservation

Venous blood sample were collected using fluoride oxalate tube for fasting blood glucose analysis and EDTA tube for glycated haemoglobin estimation while plane bottle was for fasting lipid profile The samples in plain bottles were allowed to clot, then spun using a centrifuge (NewLife bucket centrifuge, Model: 800D) at 3000rpm for 5minutes. The serum was collected into another plain bottle for analysis. The processed samples were preserved using ice pack in a thermo-cool container and then transported to the laboratory for analysis

Estimation of Lipid Profile Parameters

All samples for lipid profile were analysed using semi-automated clinical chemistry analyzers (Model: MAP LAB plus, Cod. RM2060). Reagents from Spectrum Diagnostics were used for estimation of all parameters. Total cholesterol present in the samples were estimated using enzymatic colorimetric method. HDL cholesterol present in the samples were estimated using Precipitation Method. Triglycerides present in the samples were estimated using Enzymatic Colorimetric Method while the LDL was calculated from the data of total cholesterol, triglyceride and HDL cholesterol above according to Friedewald's equation (32) as follows;

$$LDL (mmol/L) = Total Cholesterol - \left(\frac{Triglyceride}{2.2} + HDL Cholesterol \right)$$

Calculation of Atherogenic Indices

Each value for Atherogenic Indices was calculated accordingly as described by Abid *et al.* (33). Atherogenic indices were calculated using the lipid profile values obtained from the assays as follows:

Atherogenic Index of Plasma (AIP) was calculated according to the formula:

$$AIP = \log_{10} \left(\frac{Triglyceride (mmol/L)}{HDL - Cholesterol (mmol/L)} \right)$$

Castelli Risk Index I (CRI-I) was calculated using the formula:

$$CRI - I = \left(\frac{Total Cholesterol (mmol/L)}{HDL - Cholesterol (mmol/L)} \right)$$

Castelli Risk Index II (CRI-II) was calculated using the formula:

$$CRI - II = \left(\frac{LDL - Cholesterol (mmol/L)}{HDL - Cholesterol (mmol/L)} \right)$$

Atherogenic Coefficient (AC) was calculated as:

$$AC = \left(\frac{Total Cholesterol (mmol/L) - HDL - Cholesterol (mmol/L)}{HDL - Cholesterol (mmol/L)} \right)$$

RESULTS

Demographic Details of Subjects

The demographic characteristics of the study subjects are showed on Table 1.

Comparison of Mean Levels of Blood Pressures among the Study Subjects

There were significant differences in the mean levels of diastolic pressure and systolic pressure among the different groups (Table 2).

Comparison of Mean Levels of Glycemic Parameters among Study Groups

Comparison of mean levels of fasting blood sugar (FBS) and glycated hemoglobin (HbA1c) among the study subjects revealed that there was a significant difference across the groups (Table 3)

Comparison of Mean Levels of Lipid Profile among the Study Groups

Comparison of mean levels of lipid parameters among the study subjects revealed a significant difference across the group (Table 4).

Comparison of Mean Levels of Atherogenic Indices among the Study Groups

Comparison of mean levels of cardiovascular risk indices among the study subjects revealed that Castelli Risk Index 1 (CR1) was significantly higher in diabetic (9.08 ± 1.75) and hypertensive/ diabetic (6.59 ± 4.55) subjects compared to hypertensive (3.88 ± 2.09) and control (4.66 ± 2.81) subjects ($p = 0.011$). Castelli Risk Index 2 (CR2) was also significantly higher in diabetic subjects (1.80 ± 0.36) compared to hypertensive (0.86 ± 0.41), hypertensive/diabetic (0.79 ± 0.54), and control (1.22 ± 0.56) subjects ($p = 0.010$). In the case of the triglyceride-glucose index (TYG), control subjects (0.99 ± 0.13) and hypertensive subjects (1.21 ± 0.48) had significantly higher levels compared to diabetic (0.57 ± 0.12) and hypertensive/diabetic (0.61 ± 0.21) subjects ($p < 0.0001$). Similarly, the atherogenic coefficient (AC) was markedly elevated in diabetic (8.09 ± 1.75) and hypertensive/diabetic (5.89 ± 0.64) subjects compared to hypertensive (2.88 ± 0.30) and control (3.66 ± 0.40) subjects ($p = 0.013$) (Table 5).

Table 1: Demographic Detail of Subjects

Variables	Hypertensive Subjects	Hypertensive/ Diabetic Subjects	Diabetic Subjects	Apparently Healthy Control Subjects
Sex				
Male	27 (54%)	25 (50%)	25 (50%)	30 (60%)
Female	23 (46%)	25 (50%)	25 (50%)	20 (40%)
Age Range (Years)				
30 – 49	35 (70%)	33 (66%)	36 (72%)	33 (66%)
50 – 69	15 (30%)	17 (34%)	14 (28%)	17 (34%)
Duration of Disease (Years)				
1-5	22 (44%)	32 (64 %)	43 (86%)	
6-10	28 (56%)	18 (36%)	7 (14%)	

Table 2: Comparison of Mean Levels of Blood Pressures among the Study Subjects

Groups/Parameters	DP (mmHg)	SP (mmHg)
HTN (n=50)	83.14± 14.92 ^a	155.86± 12.53 ^a
DM (n=50)	85.11± 11.79 ^a	125.42± 5.56 ^b
HTN/DM (n=50)	90.34± 22.64 ^b	156.06± 13.18 ^a
Control (n=50)	75.13± 9.80 ^c	121.76± 3.46 ^c
P-value	<0.0001	<0.0001
F-value	8.212	188.199
Inference	S	S

N/B: Values in the same row but having different superscripts are significantly different from each other ($p \leq 0.05$). Keys: HTN-Hypertension subjects, DM-Diabetes mellitus subjects, HTN/DM-Subjects with the two conditions, SP-Systolic Pressure, DP-Diastolic Pressure

Table 3. Comparison of Mean Levels of Glycemic Parameters among Study Groups

Groups/Parameters	FBS (mmol/L)	HbA1c (%)
HTN (n=50)	4.40 ± 0.58 ^a	4.55 ± 0.72 ^a
DM (n=50)	8.92 ± 1.47 ^b	6.96 ± 0.58 ^b
HTN/DM (n=50)	9.50 ± 1.95 ^b	7.09 ± 0.66 ^b
Control (n=50)	4.34 ± 0.38 ^a	4.68 ± 0.47 ^a
P-value	<0.0001	<0.0001
F-value	244.63	256.62
Inference	S	S

N/B: Values in the same row but having different superscripts are significantly different from each other ($p \leq 0.05$). Keys: HTN-Hypertension subjects, DM-Diabetes mellitus subjects, HTN/DM-Subjects with the two conditions, FBS – Fasting Blood Sugar, HbA1c – Glycated Haemoglobin

Table 4 Comparison of Mean Levels of Lipid Profile among the Study Groups

Groups/Parameters	TC (mmol/L)	TG (mmol/L)	LDL (mmol/L)	HDL (mmol/L)	VLDL (mmol/L)
HTN (n=50)	5.34 ± 1.81 ^a	1.41 ± 0.30 ^a	1.41 ± 0.55 ^a	1.11 ± 0.51 ^a	3.45 ± 2.20 ^a
DM (n=50)	4.48 ± 0.90 ^b	1.16 ± 0.22 ^b	1.36 ± 0.47 ^b	1.27 ± 0.70 ^b	2.52 ± 1.33 ^b
HTN/DM (n=50)	5.61 ± 1.59 ^a	1.41 ± 0.30 ^a	1.52 ± 0.77 ^c	1.01 ± 0.49 ^a	4.03 ± 2.03 ^c
Control (n=50)	4.27 ± 0.43 ^b	1.24 ± 0.25 ^b	1.24 ± 0.50 ^d	1.28 ± 0.24 ^b	2.57 ± 1.09 ^b
P-value	<0.0001	0.034	0.009	<0.0001	<0.0001
F-value	11.50	2.942	3.939	7.805	8.949
Inference	S	S	S	S	S

Keys: Values in the same row but having different superscripts are significantly different from each other ($p \leq 0.05$). HTN-Hypertension subjects, DM-Diabetes mellitus subjects, HTN/DM-Subjects with the two conditions, TC- Total Cholesterol, TG-Triglyceride, HDL – High Density Lipoprotein, LDL-Low Density Lipoprotein, VLDL- Very Low-Density Lipoprotein.

Table 5: Comparison of Mean Levels of Atherogenic Indices among the Study Groups

Groups/Parameters	CR 1	CR 11	TYG	AC
HTN (n=50)	3.88 ± 2.09 ^a	0.86 ± 0.41 ^a	1.21 ± 0.48 ^a	2.88 ± 0.30 ^a
DM (n=50)	9.08 ± 1.75 ^b	1.80 ± 0.36 ^b	0.57 ± 0.12 ^b	8.09 ± 1.75 ^b
HTN/DM (n=50)	6.59 ± 4.55 ^c	0.79 ± 0.54 ^a	0.61 ± 0.21 ^b	5.89 ± 0.64 ^c
Control (n=50)	4.66 ± 2.81 ^a	1.22 ± 0.56 ^c	0.99 ± 0.13 ^c	3.66 ± 0.40 ^d
P-value	0.011	0.010	<0.0001	0.013
F-value	5.789	5.979	69.263	5.813
Inference	S	S	S	S

Keys: Values in the same row but having different superscripts are significantly different from each other ($p \leq 0.05$). HTN-Hypertension subjects, DM-Diabetes mellitus subjects, HTN/DM-Subjects with the two conditions, CR I (Castelli Risk Index I) = TC/HDL, CR II (Castelli Risk Index II) = LDL/HDL, TYG = Triglyceride-glucose ratio, AC (Atherogenic Coefficient) = [TC-HDL]/HDL

Comparison of Mean Levels of Lipid Profile parameters and Atherogenic Indices among the Study Subjects Based on Duration of Disease

The comparison of mean levels of the various parameters among the study subjects showed no significant differences between the two duration categories within each disease group. Though there were significant differences in the levels of the respective parameters among the study subjects based on the duration of disease as shown in the following tables;

Table 6a and Table 6b.**Table 6a: Comparison of Mean Levels of Lipid Profile among the Study Groups Based on Duration of Disease**

Parameters	1-5 Yrs HTN (n=22)	6-10 Yrs HTN (n=28)	1-5 Yrs DM (n=43)	6-10 Yrs DM (n=7)	1-5 Yrs HTN/DM (n=32)	6-10 Yrs HTN/DM (n=18)	p-value	F-value
TC (mmol/L)	4.72 ± 1.17 ^a	5.63 ± 2.11 ^b	4.45 ± 0.95 ^a	4.69 ± 1.25 ^a	5.73 ± 1.69 ^b	5.39 ± 1.48 ^b	0.002	4.090

TG (mmol/L)	1.44 ± 0.52 ^a	1.37 ± 0.57 ^a	1.17 ± 0.47 ^b	1.42 ± 0.46 ^a	1.82 ± 0.68 ^c	1.93 ± 0.69 ^c	0.022	2.725
HDL (mmol/L)	1.12 ± 0.45 ^a	1.10 ± 0.69 ^a	1.52 ± 0.73 ^b	1.19 ± 0.49 ^a	1.03 ± 0.48 ^a	0.97 ± 0.50 ^d	0.002	3.945
LDL (mmol/L)	2.92 ± 1.37 ^a	3.86 ± 2.62 ^b	2.46 ± 1.35 ^a	1.25 ± 0.62 ^d	2.13 ± 0.69 ^a	3.98 ± 1.90 ^b	0.002	3.945
VLDL (mmol/L)	0.63 ± 0.24 ^a	0.58 ± 0.25 ^a	0.46 ± 0.20 ^b	0.62 ± 0.19 ^a	0.69 ± 0.34 ^a	0.58 ± 0.30 ^a	0.015	2.921

N/B: Values in the same row but having different superscripts are significantly different from each other ($p \leq 0.05$). Keys: HTN-Hypertension subjects, DM-Diabetes mellitus subjects, HTN/DM-Subjects with the two conditions, TC- Total Cholesterol, TG-Triglyceride, HDL – High Density Lipoprotein, LDL-Low Density Lipoprotein, VLDL- Very Low-Density Lipoprotein.

Table 6b: Comparison of Mean Levels of Atherogenic Indices among the Study Groups Based on Duration of Disease

Parameters	1-5 Yrs HTN (n=22)	6-10 Yrs HTN (n=28)	1-5 Yrs DM (n=43)	6-10 Yrs DM (n=7)	1-5 Yrs HTN/DM (n=32)	6-10 Yrs HTN/DM (n=18)	p-value	F-value
CR I	5.32 ± 0.77 ^a	8.46 ± 1.69 ^b	4.18 ± 0.85 ^b	7.19 ± 3.81 ^b	8.72 ± 1.66 ^b	9.90 ± 2.60 ^c	0.045	3.231
CR II	3.60 ± 0.72	3.73 ± 1.63	2.82 ± 0.85	5.39 ± 3.53	6.92 ± 1.60	8.29 ± 2.52	0.066	2.120
AC	4.32 ± 0.77	7.46 ± 1.70	3.18 ± 0.85	6.19 ± 8.81	7.72 ± 1.66	8.90 ± 2.60	0.054	2.231
TYG	0.34 ± 0.14 ^a	0.32 ± 0.14 ^a	0.13 ± 0.06 ^b	0.15 ± 0.05 ^b	0.17 ± 0.08 ^b	0.14 ± 0.08 ^b	<0.0001	24.348

N/B: Values in the same row but having different superscripts are significantly different from each other ($p \leq 0.05$). Keys: HTN-Hypertension subjects, DM-Diabetes mellitus subjects, HTN/DM-Subjects with the two conditions, CR I (Castelli Risk Index I) = TC/HDL, CR II (Castelli Risk Index II) = LDL/HDL, TYG =Triglyceride-glucose ratio, AC (Atherogenic Coefficient) = [TC-HDL]

DISCUSSION

The demographic characteristics of the study participants showed a relatively balanced gender distribution across all groups for age distribution with most participants within the 30–49 years age bracket. Regarding disease duration, most hypertensive individuals (56%) had lived with the condition for 6–10 years, while 44% had it for 1–5 years. In the HTN/DM group, the majority (64%) had been diagnosed for 1–5 years, with 36% having the condition for 6–10 years. Among diabetic subjects, a larger proportion (86%) reported a disease duration of 1–5 years, while only 14% had lived with diabetes for 6–10 years. These findings indicate a relatively middle-aged population with modest differences in sex distribution and a predominance of shorter disease duration across the study groups (Table 1).

In this study, there was a significant increase in systolic and diastolic blood pressures in the hypertensive subjects compared to the control group confirming the expected hemodynamic profile of hypertension. Elevated arterial pressure in hypertension arises primarily from increased systemic vascular resistance, endothelial dysfunction, and reduced nitric oxide (NO) bioavailability (34). This elevation was also seen in diabetes likely reflecting early-stage vascular dysfunction where endothelial injury and loss of elasticity precede sustained hypertension. Diabetes mellitus induces arterial stiffness and impaired

vasorelaxation through several mechanisms, including the non-enzymatic glycation of vascular proteins, oxidative stress, and low-grade inflammation (35). These processes alter vascular compliance and promote mild elevations in blood pressure. Jia & Sowers, (36) similarly reported that diabetic patients often develop prehypertensive states driven by endothelial dysfunction, enhanced sympathetic nervous system activity, and insulin resistance. Therefore, the significant elevation in blood pressure among diabetic participants in this study may reflect subclinical hypertensive changes secondary to chronic hyperglycemia and endothelial inflammation (36). Blood pressure parameters were highest in the HTN/DM group, significantly exceeding those of the hypertensive and diabetic group alone (Table 2). This finding suggests that diabetes exacerbates the vascular and hemodynamic burden of hypertension. Hyperglycemia promotes renal sodium retention and fluid accumulation, leading to increased plasma volume and cardiac output (37). The coexistence of these mechanisms results in a more severe hypertensive state, consistent with the findings of Schmieder & Ruilope, (38), who observed significantly higher blood pressure indices in comorbid hypertensive-diabetic populations compared to hypertensives alone.

There was a significant increase in fasting blood glucose (FBG) and glycated hemoglobin (HbA1c) levels in the diabetic group compared to the control, confirming the

presence of chronic hyperglycemia and poor glycaemic control. However, no significant difference was observed in FBG and HbA1c levels between the hypertensive and control groups (Table 3). The markedly higher FBG and HbA1c levels in the diabetic group are consistent with persistent hyperglycemia characteristic of diabetes mellitus, where prolonged exposure of erythrocyte hemoglobin to circulating glucose elevates HbA1c - a reliable indicator of average blood glucose over 8–12 weeks (39). These elevated glycaemic indices align with the findings of Ezeani *et al.* (40), who reported mean HbA1c levels above 7.0% among Nigerian type 2 diabetes patients. The absence of a significant increase in FBG and HbA1c levels in the hypertensive group compared to the control could be attributed to the non-diabetic nature of hypertensive participants, implying that their blood glucose metabolism remained largely intact. Moreover, hypertension alone does not directly impair glucose regulation unless accompanied by insulin resistance or pancreatic dysfunction (41). The HTN/DM group, however, showed significantly higher FBG and HbA1c levels than all other groups (Table 3), reflecting aggravated metabolic derangement associated with comorbidity. The coexistence of hypertension and diabetes creates a bidirectional pathogenic loop, vascular dysfunction worsens insulin resistance, while hyperglycemia enhances vascular stiffness and inflammation (35). Chronic hypertension triggers endothelial injury and inflammatory cytokine release, impairing insulin receptor signaling and glucose uptake (41). Conversely, persistent hyperglycemia promotes sodium retention and sympathetic activation, further elevating blood pressure, a phenomenon described as the “metabolic-endothelial axis” (35). However, there is need for integrated cardiovascular and glycemic assessment in hypertension management (42).

Hypertensive subjects demonstrated significantly elevated levels of total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and triglycerides (TG), alongside a significant reduction in high-density lipoprotein cholesterol (HDL-C) compared to the control group (Table 4). These findings indicate the presence of hypertensive dyslipidemia, a metabolic disturbance frequently observed in essential hypertension (43). The pathophysiological mechanism underlying this lipid alteration involves increased sympathetic activation, oxidative stress, and altered adipocyte metabolism. Elevated catecholamine levels in hypertension activate hormone-sensitive lipase, promoting lipolysis and the release of free fatty acids (FFAs) into circulation (44). The influx of FFAs to the liver enhances hepatic very-low-density lipoprotein (VLDL) production, thereby increasing plasma TG and LDL-C concentrations (45). Reduced HDL-C levels suggest impaired reverse cholesterol transport, wherein the capacity of HDL to

remove cholesterol from peripheral tissues and macrophages is diminished, predisposing to atherogenesis (43). This finding is consistent with the reports of Akintunde *et al.* (44) who documented elevated LDL-C and TG levels with concomitant HDL-C reduction in untreated hypertensive patients. Likewise, Osuji *et al.* (46) and Adamu *et al.* (47) reported significantly higher TC and LDL-C levels among hypertensive subjects compared to normotensive controls. Also, the Castelli's Risk Index II (CRI-II), triglyceride-glucose (TYG) ratio, and atherogenic coefficient (AC) were significantly lower in the hypertensive group compared to the control group (Table 5) as observed in this study. This finding may reflect the influence of antihypertensive medications, particularly statins and ACE inhibitors, which are known to modulate lipid metabolism and improve insulin sensitivity (48). Moreover, it is possible that dietary modifications and medical interventions in hypertensive patients contributed to this reduction, indicating partial management of atherogenic risk despite elevated LDL-C and TG levels. The lowered TYG ratio suggests improved insulin sensitivity or reduced hepatic triglyceride production in treated hypertensives, which aligns with reports that certain antihypertensive therapies exert beneficial effects on glucose–lipid metabolism (49). Consistent with these findings, Carey *et al.* (50) demonstrated that long-term pharmacological management of hypertension led to an improved lipid ratio profile despite elevated absolute lipid concentrations, supporting the notion that medical intervention may attenuate atherogenic risk indices.

Amongst the diabetic subjects, there was a significant increase in LDL-C compared to the control, while no significant differences were observed in TC, TG, HDL-C, and VLDL levels (Table 4). Elevated LDL-C in diabetes mellitus (DM) is a hallmark of diabetic dyslipidemia and reflects impaired hepatic clearance of LDL particles due to glycation of apolipoprotein B and LDL receptors (51). The absence of significant changes in other lipid fractions may be due to variations in glycaemic control, duration of disease, or ongoing use of lipid-lowering agents among the subjects. Additionally, the increased LDL-C level, even in the absence of hypertriglyceridemia or low HDL-C, still poses a major atherogenic threat, as small dense LDL particles in diabetics are more susceptible to oxidative modification and arterial deposition (52). Similarly, there was also a significant increase in the levels of Castelli's Risk Index I (CRI-I), CRI-II, and Atherogenic Coefficient (AC) in diabetic subjects compared to the control, indicating heightened cardiovascular and atherogenic risk (Table 5). These indices, derived from ratios of atherogenic to anti-atherogenic lipoproteins, provide a more sensitive measure of dyslipidemia-linked vascular risk than

absolute lipid concentrations (53). The observed elevation suggests an imbalance in lipid homeostasis that favors cholesterol deposition in arterial walls, contributing to diabetic macrovascular complications such as coronary artery disease and atherosclerosis (54). Interestingly, the TYG ratio was significantly lower in diabetic subjects compared to controls, which may reflect a paradoxical effect of glycaemic control interventions or pharmacotherapy aimed at reducing insulin resistance. Consistent with this study, Gowda *et al.* (53) observed increased Castelli indices and atherogenic coefficient in type 2 diabetics compared with controls, indicating a high predisposition to atherosclerosis.

Considering the hypertensive-diabetic (HTN/DM) subjects, there was a significant increase in TC, TG, LDL-C, and VLDL-C, with a concurrent decrease in HDL-C compared to the diabetic group (Table 4). This pattern reflects the combined metabolic and vascular burden of coexisting hypertension and diabetes, which synergistically aggravate dyslipidemia through oxidative stress, chronic inflammation, and hormonal dysregulation (36). The simultaneous presence of insulin resistance and renin-angiotensin-aldosterone system (RAAS) activation enhances lipolysis and hepatic VLDL production, increasing TG and LDL-C levels, while impairing HDL synthesis and reverse cholesterol transport (25). Hyperglycemia-induced glycation of apolipoproteins also reduces hepatic clearance of LDL-C, further worsening lipid imbalance (55). The observed reduction in HDL-C suggests diminished antioxidant and anti-inflammatory capacity of HDL particles, predisposing to accelerated atherogenesis in comorbid patients (44). Additionally, LDL-C and VLDL-C levels were significantly higher in the HTN/DM group compared to hypertensive subjects alone (Table 4), indicating that the addition of diabetes to hypertension amplifies hepatic lipid synthesis and impairs lipoprotein metabolism. This could result from persistent hyperinsulinemia, which increases hepatic acetyl-CoA carboxylase activity and promotes triglyceride-rich lipoprotein formation (56). The marked dyslipidemia observed in HTN/DM patients aligns with the findings of Akintunde *et al.*, (44) who reported more severe lipid derangements in hypertensive-diabetic subjects compared to patients with either condition alone.

Compared to the diabetic group, the HTN/DM patients demonstrated higher TC, TG, LDL-C, VLDL-C and lower HDL-C levels (Table 4), alongside significantly lower CRI, CR11 and AC indices (Table 5). Also, there was a significant increase in the levels of LDL-C and VLDL-C in the HTN/DM group compared to the HTN group (Table 4). Meanwhile, TYG level was significantly lower in HTN/DM group compared to the HTN group while CR1 and AC levels were significantly

higher in HTN/DM groups compared to the HTN group (Table 5).

Regarding atherogenic indices, the HTN/DM group demonstrated significantly higher CRI-I, CR-II and AC compared to the hypertensive group, suggesting a greater imbalance between atherogenic and protective lipoproteins, and consequently, a higher cardiovascular risk (Table 5). These elevated indices indicate that the lipid disturbances in comorbid patients favor cholesterol deposition within arterial walls, increasing susceptibility to atherosclerosis and ischemic heart disease (54). Conversely, the lower Triglyceride-Glucose (TYG) index observed in the HTN/DM group compared to hypertensives alone may reflect the influence of antidiabetic therapy, such as metformin or insulin, which improves glucose utilization and reduces triglyceride accumulation (57). It may also indicate partial glycaemic control among some HTN/DM participants, mitigating hepatic lipogenesis despite ongoing dyslipidemia (58).

Duration-based comparisons revealed that subjects with longer disease duration (6–10 years) generally exhibited more adverse lipid patterns than those with shorter duration (1–5 years), especially among hypertensive and HTN/DM participants (Table 6a). Similarly, diabetics with longer duration demonstrated higher TG and VLDL levels, reflecting progressive insulin resistance and hepatic overproduction of VLDL particles due to continuous glucose and free fatty acid flux (59). The reduction in HDL among long-duration diabetic and HTN/DM subjects supports previous evidence that chronic hyperglycaemia and glycoxidation degrade HDL structure and function, impairing its antioxidant role (60). The progressive rise in CRI-I with disease duration across HTN and HTN/DM groups (Table 6b) confirms that the atherogenic burden increases over time, in line with reports that longer exposure to metabolic abnormalities compounds vascular risk and endothelial damage (61). However, the absence of significant duration-related differences in CRI-II and AC may suggest partial therapeutic modulation by lipid-lowering or antihypertensive therapy (62) but still validates the usefulness of these lipid ratios for early identification of those at risk of cardiovascular issues (63).

Strengths of the Study

The assessment of lipid profile and atherogenic ratios provide valuable insights into the early identification of diabetic - hypertensive comorbidity subjects in Bayelsa State at increased risk of cardiovascular events. This was shown by the combined metabolic and vascular burden of coexisting hypertension and diabetics which synergistically increased dyslipidemia and lipid derangements in the comorbidity subjects compared to diabetic or

hypertensive subjects alone. as reflected in the lipid profiles. The study also showed a progressive rise in CR1 with disease duration across the hypertensive and comorbidity group indicating that atherogenic burden increases over time. This finding demonstrates that metabolic imbalance and cardiovascular risk are markedly amplified when hypertension coexist with diabetes mellitus.

Limitations of the Study

In this study, the sample size was relatively small, with 50 subjects in each group not considering age differences and sex, which may limit the generalizability of the findings. Again, the subjects were only accessed at the hospital, there is need to move into demographic regions. Evaluation of the progress of endothelial dysfunction was not monitored

CONCLUSION

The concurrent disturbances in glycemic regulation and lipid metabolism further indicate that the coexistence of these conditions creates a compounding effect that aggravates atherogenic and cardiometabolic risk. Overall, the study highlights the critical need for early identification and integrated management of subjects with diabetes and hypertension to reduce cardiovascular burden, and improve long-term outcomes.

DECLARATIONS

Ethics Approval

Ethical clearance was gotten from the ethical committee of Federal Medical Centre, Yenagoa (FMCY/REC/ECC/2024/AUG/776) and Niger Delta University Teaching Hospital (NDUTH), Okolobiri, Bayelsa State (NDUTH/REC/0078/2024).

Competing Interests

The authors declare that they have no competing interests.

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Authors' Contributions

All authors contributed to the conception and design of the study, data collection, analysis and interpretation of the findings, manuscript preparation, critical revision of the manuscript, and approved the final production.

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AI Use Disclosure

The authors declare that no generative artificial intelligence tools were used to generate or substantially modify the scientific content of this manuscript.

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