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Research Article

Lipid Profile and Blood Sugar Variations Among Hypertensive Subjects in Yenagoa, Nigeria

Simon Akpomemera Erho[1], Wadioni Aduema[1], Allen Erighanyoyefa[1], Mao Ebimobotei Bunu[1], Blessing Elohor Poripo[1], Ugorji Nnaemeka Ogbonna[2], Chimburuoma Nath-Abraham[3], Bruno Chukwuemeka Chinko[3, *]

[1]Department of Human Physiology, Faculty of Basic Medical Sciences, Bayelsa Medical University, Yenagoa, Nigeria

[2]Department of Medical Biochemistry, Faculty of Basic Medical Sciences, University of Port Harcourt, Port Harcourt, Nigeria

[3]Department of Human Physiology, Faculty of Basic Medical Sciences, University of Port Harcourt, Port Harcourt, Nigeria

Abstract

Hypertension is the leading risk factor for death and disability worldwide and a major contributor to premature mortality. Individuals with hypertension often exhibit alterations in lipid and glucose metabolism, further increasing their risk of severe cardiovascular complications. The present study evaluated the lipid profile and blood sugar among hypertensive individuals in Yenagoa, Nigeria. A total of 246 subjects (31-60 years) were recruited for the study. They consisted of 172 hypertensives attending the Cardiology Clinic of Niger Delta University Teaching Hospital (NDUTH) and 74 normotensive control subjects drawn from the staff of NDUTH. Body mass index, fasting blood sugar and lipid profile were measured following standard protocols. The result of this study shows a significantly elevated mean levels of fasting blood sugar (FBS), total cholesterol (TC), triglycerides (TG), and low-density lipoproteins (LDL) among hypertensive subjects compared to the normotensive controls ($p<0.05$). Fasting blood sugar showed sex and age-dependent variations among the hypertensives. Male hypertensive subjects had a significantly higher fasting blood sugar compared to the female hypertensives ($p<0.05$). Also, fasting blood sugar increased with age for both hypertensive and normotensive subjects regardless of gender. The current evidence has identified elevated BMI, FBS, TC, TG, and LDL among hypertensive patients receiving medical care at a tertiary healthcare facility in Yenagoa, Nigeria. These findings underscore the significant association between hypertension and metabolic risk factors and recommend routine assessments of these metabolic parameters among hypertensive patients.

Keywords

Hypertension, Body Mass Index, Lipid Profile, Fasting Blood Sugar, Cardiovascular Risk

*Corresponding author: bruno.chinko@uniport.edu.ng (Bruno Chukwuemeka Chinko)

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1. Introduction

The global epidemic of cardiovascular diseases (CVDs) is on the rise, encompassing a spectrum of conditions such as hypertension, acute coronary syndromes, stroke, and heart failure [1]. Hypertension, or high blood pressure, is a chronic CVD characterized by persistently elevated arterial pressure (≥140/90 mmHg) when measured on multiple occasions. [1-4]. Hypertension is the leading risk factor for death and disability worldwide, surpassing other major health risks, including tobacco use, obesity, high blood sugar and lipid disorders. It is the single most significant contributor to premature mortality. Globally, approximately 33% of adults aged 30 to 79 are affected by hypertension. The number of individuals living with the condition has doubled from 650 million in 1990 to 1.3 billion in 2009, highlighting its rapid and widespread increase. This trend is projected to continue as populations grow and age, with age-adjusted hypertension being more prevalent in males than in females [4-7]. Notably, 78% of those affected reside in low and middle-income countries, where access to diagnosis, treatment, and management remains a significant challenge [6, 8]. In low and middle-income countries, variations in blood pressure between rural and urban populations serve as a key indicator of different stages of epidemiological transition. This transition is driven by significant socioeconomic changes, which, in turn, influence shifts in risk factors and disease patterns within affected communities [3, 9]. Several studies have highlighted the rising prevalence of hypertension across Africa with Nigeria being a significant contributor [6, 10, 11], A systematic review and meta-analysis estimated that in 2010, approximately 20.8 million Nigerians aged 20 years and older had hypertension, corresponding to a prevalence rate of 28.0% [12]. This prevalence has steadily increased over the decades, with age-adjusted rates rising from 8.5% in 1995 to 32.5% in 2020 [13]. Recent studies have reported varying prevalence rates across different regions and populations within the country. For example, a study conducted in a rural community in southeastern Nigeria found a hypertension prevalence of 27.6%, with a notably low awareness rate of 2.8% among those affected [14]. Another study reported an overall hypertension prevalence of 38.1%, with higher rates observed in women (41.8%) compared to men (31.8%). Interestingly, the prevalence among rural dwellers (37.5%) was comparable to that of urban residents [15] although evidence suggests that prevalence and management are largely influenced by socio-demographic factors [3, 13].

The pathophysiology of hypertension remains unclear as a small fraction of hypertensive individuals (between 2% and 5%) have an undiagnosed renal or adrenal disease which may have resulted in their elevated blood pressure [16, 17]. The rest have no clear or single cause of the hypertension and are hence referred to as “essential hypertension”. Normal blood pressure is regulated by multifaceted physiological mechanisms and disruptions in these processes lead to the pathogenesis of essential hypertension. Among hypertensive patients, it is more likely that there is an interplay of various factors that leads to the development and sustenance of elevated blood pressure. Key factors that have been extensively studied include salt intake, obesity, insulin resistance, the renin-angiotensin system, and the sympathetic nervous system [2, 16]. In recent years, additional factors have been explored, including genetic influences, endothelial dysfunction, low birth weight, intrauterine nutrition, neurovascular abnormalities, dyslipidemia, and impaired blood sugar regulation [16, 18-22]. Our previous studies observed abnormal iron metabolism and elevated white blood cell parameters and platelets in hypertensive individuals [4, 23].

Hypertensive subjects often exhibit alterations in lipid metabolism and glucose homeostasis, predisposing them to severe cardiovascular complications [1, 24, 25]. Given the increasing burden of hypertension in Nigeria, particularly in urbanizing regions like Yenagoa, it is essential to evaluate the patterns of lipid and blood sugar variations among affected individuals. However, data on the specific patterns of lipid profile and blood sugar variations among hypertensive individuals in Yenagoa, Nigeria, remain limited. The absence of localized studies on these metabolic alterations creates a knowledge gap that hinders targeted interventions and effective management strategies. This study evaluates lipid profile and blood sugar variations among hypertensive individuals in Yenagoa, Nigeria. This will serve to generate, region-specific data that can enhance clinical decision-making, optimize treatment approaches, and contribute to more effective public health interventions.

2. Materials and Method

2.1. Study Population

A total of 246 subjects (31-60 years), comprising 172 hypertensive and 74 normotensive subjects were recruited for the study. The hypertensive subjects were confirmed hypertensive individuals attending the cardiology clinic of the Niger Delta University Teaching Hospital (NDUTH) in Okolobiri, Yenagoa, Bayelsa State, Nigeria. Inclusion criteria for hypertensive subjects comprised adults aged 18–65 years with confirmed hypertension, residing in Yenagoa for at least 4 years, and in stable health without recent acute illness or hospitalization in the past 5 months. Participants were excluded if they were pregnant, lactating, had known diabetes, were on anti-dyslipidemic or antidiabetic drugs, or had recent surgery, trauma, or acute illness within the last 3 months. The normotensive subjects were selected from the staff population of NDUTH. For normotensive controls, inclusion criteria included apparently healthy adults aged 18–65 years with no history of hypertension or diabetes, who had resided in Yenagoa for at least 4 years. They were also confirmed to

have normal blood pressure levels upon screening. Exclusion criteria involved pregnancy, lactation, recent illness, trauma, or any chronic metabolic disorder. All participants were informed about the study's objectives, procedures, and potential implications. They voluntarily agreed to participate by providing written informed consent following ethical research guidelines.

2.2. Data Collection and Laboratory Analysis

This study employed a cross-sectional descriptive design. A structured questionnaire was administered to obtain vital demographic and clinical information from the participants.

Anthropometric measurements were taken using standardized equipment. Body weight was measured with a FULLMEDI adult weighing scale (FM-S120), while height was recorded using a wall-mounted meter rule. Body mass index (BMI) was subsequently calculated from these measurements. Blood pressure readings were obtained using an aneroid sphygmomanometer (Wuxi Yuqing Medical, China) and a Littmann stethoscope (USA) after the participants had rested for five minutes in a seated position. Three consecutive measurements were taken at one-minute intervals, and the average reading was recorded.

Fasting blood sugar (FBS) levels were measured following an overnight fast using a Finetest auto-coding premium blood glucose meter (DOOSAN, South Korea). To assess the lipid profile, approximately 3 ml of venous blood was collected into a plain sample bottle. The lipid profile [total cholesterol (TC), triglycerides (TG), high-density lipoproteins (HDL), and low-density lipoproteins (HDL)] were analysed using a standard laboratory test kit (Agappe, Switzerland) following established spectrophotometric procedures.

2.3. Ethical Consideration

All procedures in this study were carried out in strict adherence to the highest ethical standards outlined in the World Medical Association (WMA) Helsinki Declaration, originally established in 1964 and revised in 2024 [26]. Before their inclusion in the study, each prospective participant provided informed consent by signing a consent form. The research design and protocol received formal approval from the Ethics Committee Board of the Niger Delta University Teaching Hospital (NDUTH/REC/2023/040821).

2.4. Statistical Analysis

The data were analyzed using SPSS version 25. The mean and standard deviation were computed for each parameter. Differences between hypertensive and normotensive subjects were assessed using the student's t-test, while variations across age groups were evaluated using analysis of variance (ANOVA). A p-value of less than 0.05 (<0.05) was considered statistically significant for all comparisons.

3. Results

Table 1. *Age, BMI and blood pressure parameters characteristics of the study population.*

Parameters	Normotensive n=74	Hypertensive n=172	T-test p-value
Age (Yrs)	45.15±8.08	45.97±6.71	0.412
Body mass index (kg/m^2)	25.27±5.08	28.66±5.54*	0.001
Systolic blood pressure (mmHg)	113.27±9.65	133.84±22.15*	0.001
Diastolic blood pressure (mmHg)	74.92±8.24	83.46±14.65*	0.001
Mean arterial pressure (mmHg)	86.42±8.14	98.57±16.18*	0.001

Results are given as mean ±standard deviation.
*Significantly higher compared to the normotensive subjects

Table 1 presents the age, body mass index (BMI), and blood pressure parameters of the study population. The data indicate that both the normotensive and hypertensive groups were within a similar age range, with no significant difference observed in age between the two groups ($p > 0.05$). However, the study found a significant increase in body mass index (BMI), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) among hypertensive subjects compared to the normotensive controls ($p<0.05$).

Table 2. *Fasting blood sugar and lipid profile characteristics of the study population.*

Parameters	Normotensive n=74	Hypertensive n=172	T-test p-value
Fasting blood sugar (mg/dL)	91.80 ±17.83	113.17 ±25.14*	0.01
Total cholesterol (mmol/L)	5.15 ±1.46	6.14 ±2.09*	0.01
Triglycerides (mmol/L)	1.23 ±0.75	1.44 ±0.79*	0.04
High-density lipoproteins (mmol/L)	1.61 ±0.38	1.83 ±0.7	0.06
Low-density lipoproteins (mmol/L)	3.02 ±1.33	3.67 ±2.0*	0.01

Results are given as mean ±standard deviation.
*Significantly higher compared to the normotensive subjects

Table 2 presents the mean fasting blood sugar and lipid profile of the study population. The data revealed significantly higher mean levels of fasting blood sugar (FBS), total cholesterol (TC), triglycerides (TG), and low-density lipoproteins (LDL) in hypertensive subjects compared to the normotensive controls ($p<0.05$).

Table 3. *Gender-based variation in FBS and lipid profile of the study population.*

	Male		Female	
Parameters	**Normotensive (n=31)**	**Hypertensive (n=69)**	**Normotensive (n=43)**	**Hypertensive (n=103)**
FBS (mg/dL)	86.39 ±12.81	122.59 ±28.12*	95.70 ±19.96	106.85 ±20.78*
TC (mmol/L)	5.24 ±1.44	5.03 ±1.87	5.09 ±1.48	6.89 ±1.89*
TG (mmol/L)	1.32 ±0.89	1.30 ±0.95	1.17 ±0.63	1.54 ±0.65*
HDL (mmol/L)	1.47 ±0.26	1.58 ±0.60	1.71 ±0.42	2.00 ±0.66*
LDL (mmol/L)	3.17 ±1.03	2.86 ±1.71	2.92 ±1.52	4.22 ±2.01*

Results are given as mean ±standard deviation.
*Significantly higher compared to the normotensive subjects

Table 3 displays gender-based variations in fasting blood sugar and lipid profile parameters within the study population. Among male participants, only fasting blood sugar was significantly higher in hypertensive subjects compared to the normotensive controls ($p < 0.05$). In contrast, among female participants, hypertensive subjects exhibited significantly higher levels of fasting blood sugar, total cholesterol, triglycerides, high-density lipoprotein (HDL), and low-density lipoprotein (LDL) compared to the normotensive controls ($p<0.05$).

Table 4. *Age-based variation in FBS and lipid profile of the study population.*

Age (years)	Participants	FBS (mg/dL)	TC (mmol/L)	TG (mmol/L)	HDL (mmol/L)	LDL (mmol/L)
31 – 40	Normotensive (n=31)	78.81 ±13.20	5.63 ±0.77	1.07 ±0.49	1.59 ±0.32	3.55 ±0.6
	Hypertensive (n=38)	105.32 ±24.53*	6.85 ±2.02*	1.37 ±0.46*	1.98 ±0.71	4.26 ±1.89*
41 – 50	Normotensive (n=31)	94.21 ±12.80	5.29 ±1.64	1.25 ±1.03	1.56 ±0.37	3.16 ±1.12
	Hypertensive (n=74)	111.96 ±22.13*	5.62 ±2.00*	1.49 ±0.81*	1.81 ±0.66	3.17 ±1.97
51 – 60	Normotensive (n=24)	106.67 ±13.81	4.42 ±1.72	1.42 ±0.75	1.68 ±0.45	2.23 ±1.77

Age (years)	Participants	FBS (mg/dL)	TC (mmol/L)	TG (mmol/L)	HDL (mmol/L)	LDL (mmol/L)
	Hypertensive (n=60)	113.17 ±25.14*	6.34 ±2.11*	1.43 ±0.93	1.77 ±0.66	3.93 ±1.98*

Results are given as mean ±standard deviation.
*Significantly higher compared to the normotensive subjects

Table 4 presents age-based variations in fasting blood sugar and lipid profile parameters within the study population. The data reveal that among young adults (31–40 years), FBS, TC, TG and LDL were significantly higher in hypertensive subjects compared to the normotensive controls ($p < 0.05$). Similarly, among middle-aged adults (41–50 years), significantly elevated levels of FBS, TC and TG were observed in hypertensive subjects relative to the normotensive controls ($p < 0.05$). Finally, in late middle-aged adults (51–60 years), hypertensive subjects exhibited significantly higher FBS, TC and LDL levels compared to the controls ($p < 0.05$).

4. Discussion

Hypertension is a significant global public health concern due to its high prevalence and associated risks of cardiovascular and kidney diseases and remains the leading risk factor for death and disability worldwide [6]. The aetiology and pathogenesis of hypertension have been linked to various factors, including age, genetic influences, socioeconomic conditions, dyslipidaemia, and insulin resistance. Individuals with hypertension often experience alterations in lipid and glucose metabolism, increasing their risk of severe cardiovascular complications. This study examines the lipid profile and blood sugar variations among hypertensive individuals in Yenagoa, Nigeria.

Data from our study show that hypertensive subjects had significantly higher BMI (Table 1), among the hypertensive individuals compared to the normotensive controls ($p < 0.05$). Higher BMI as seen in overweight and obesity is independently and positively linked to an increased risk of morbidity and mortality from hypertension, cardiovascular disease, type II diabetes mellitus, and other chronic conditions [6, 27]. Several pathophysiological mechanisms underline the link between higher BMI and hypertension. Obesity is associated with overactivation of the sympathetic nervous system, which leads to increased vasoconstriction, heart rate, and cardiac output, all of which elevate blood pressure [28-30]. Moreso, it has also been associated with impaired endothelial function due to reduced nitric oxide (NO) bioavailability and increased oxidative stress, leading to vasoconstriction and increased peripheral resistance, contributing to hypertension [31, 32]. Similarly, adipose tissues secrete pro-inflammatory cytokines like leptin, tumour necrotic factor -α (TNF-α) and interleukin-6 (IL-6) which promote, oxidative stress, and endothelial dysfunction, all of which contribute to hypertension [30, 33]. Previous studies have observed elevated BMI among hypertensive subjects in Yenagoa, Nigeria [34-36]. This elevated BMI among hypertensives as observed in the present study serves as a critical risk factor for hyperlipidaemia, predisposing individuals to cardiovascular complications such as atherosclerosis and hypertension.

Our data indicate significantly elevated levels of total cholesterol (TC), triglycerides (TG), and low-density lipoprotein (LDL) among hypertensive subjects compared to the control group ($p<0.05$) (Table 2). This pattern of lipid profile variation was particularly evident among female hypertensive individuals, who exhibited higher levels of TC, TG, HDL, and LDL compared to non-hypertensive females (Table 3). Additionally, when analyzed across different age groups, regardless of gender, hypertensive subjects consistently showed increased TC, TG, and LDL levels compared to the control group ($p<0.05$). Dyslipidaemia is characterized by abnormal levels of triglycerides, low-density lipoprotein (LDL), and/or reduced high-density lipoprotein (HDL), and is closely linked to the development and progression of hypertension via interconnected and multifocal mechanisms [37]. It has been shown to impair endothelial function by reducing the bioavailability of nitric oxide (NO), a potent vasodilator, leading to hypertension [38, 39]. Also, oxidized LDL (ox-LDL) and other lipid abnormalities promote oxidative stress and vascular inflammation, smooth muscle cell proliferation, and arterial stiffness, contributing to hypertension [40, 41]. It has also been shown that elevated lipids contribute to hypertension by activating the renin-angiotensin, aldosterone system (RAAS), leading to increased angiotensin II production, which causes vasoconstriction, sodium retention, and vascular remodelling, all of which elevate blood pressure [42, 43]. Furthermore, dyslipidaemia is associated with insulin resistance, sodium retention, sympathetic nervous system activation, vascular smooth muscle cell proliferation, microvascular thrombosis and impaired blood flow, all of which lead to the worsening of existing hypertension [30, 44-46]. Previous studies have observed elevated TG, TC and reduced HDL as a predictive risk factor in the development of hypertension in Yenagoa, Nigeria [36, 47, 48] and could predispose hypertensive persons to further cardiovascular complications like atherosclerosis [6, 29].

The present study found that hypertensive subjects had significantly higher fasting blood sugar levels compared to normotensive controls ($p<0.05$), as detailed in Table 2. Notably, hypertensive males exhibited higher mean fasting blood

sugar levels (122.59 ± 28.12 mg/dL) than hypertensive females (106.85 ± 20.78 mg/dL), as shown in Table 3. Furthermore, when analyzed across age groups, hypertensive subjects demonstrated an age-dependent increase in fasting blood sugar levels, irrespective of gender. The mean values were 105.32 ± 24.53 mg/dL for young adults (31–40 years), 111.96 ± 22.13 mg/dL for middle-aged adults (41–50 years), and 113.17 ± 25.14 mg/dL for late middle-aged adults (51–60 years), as presented in Table 4. Elevated fasting blood sugar (hyperglycemia) is a key indicator of prediabetes and diabetes and is closely linked to the onset of and sustenance of high blood pressure [49, 50]. Just like obesity and dyslipidaemia, the mechanisms by which elevated fasting blood sugar contributes to hypertension are multifactorial and interconnected. Hyperglycemia impairs endothelial function by reducing the bioavailability of nitric oxide (NO), a potent vasodilator. This occurs due to increased oxidative stress which damages the endothelium and increases vascular resistance, hence contributing to high blood pressure [51, 52]. It has also been shown that hyperglycemia activates the RAAS, leading to increased angiotensin II production which causes vasoconstriction, sodium retention, and vascular remodelling, all of which elevate blood pressure [53]. Hyperglycaemia reduces the responsiveness of body cells to insulin (insulin resistance), the hormone responsible for regulating blood sugar. In response, the pancreas increases insulin production (hyperinsulinemia) to compensate and help maintain normal blood glucose levels [54, 55]. Also, insulin promotes sodium reabsorption in the kidneys, increases sympathetic nervous system activity, and stimulates vascular smooth muscle growth, all of which contribute to hypertension [30, 44, 52, 54-56]. Hyperglycaemia triggers the non-enzymatic modification of proteins, lipids, and nucleic acids by sugars through glycation, leading to the formation of advanced glycation end products (AGEs). These compounds accumulate in blood vessels and tissues, promoting inflammation, oxidative stress, and vascular stiffness, which further contribute to hypertension [57-59]. The higher fasting blood glucose levels observed in male hypertensive compared to female hypertensive could be attributed to the ability of estrogen to improve insulin sensitivity in premenopausal females by enhancing insulin signalling pathways and promoting glucose uptake in skeletal muscle and adipose tissue [60-62]. It has also been observed that insulin resistance is sex-dependent, being higher in males compared to females [63]. Conversely, higher testosterone levels in males are associated with increased secretion of pro-inflammatory adipokines and reduced secretion of adiponectin, which promotes insulin resistance and higher fasting blood sugar [64]. Furthermore, the higher fasting blood sugar levels with age are attributable to age-related physiological changes in glucose metabolism. Factors such as insulin resistance and impaired insulin secretion [65-67], increased visceral fat [68, 69], decreased physical activities [70, 71], and low-grade inflammation [72, 73]. This goes to show that observed elevated fasting blood sugar is a predictive risk factor in the development of sustenance of hypertension and could predispose hypertensive persons to further metabolic complications like diabetes [6, 28].

5. Conclusion

The present study identified elevated levels of body mass index, fasting blood sugar, total cholesterol, triglycerides, and low-density lipoprotein among hypertensive patients receiving medical care at a tertiary healthcare facility in Yenagoa, southern Nigeria. These findings underscore the significant association between hypertension and metabolic risk factors, highlighting the need for comprehensive management strategies. Given the implications for hypertension control in Nigeria, across Africa, and beyond, it is recommended that routine evaluations for hypertensive patients include fasting blood sugar testing, lipid profile assessment, and weight management interventions. Integrating these measures into standard care protocols could improve outcomes and reduce the burden of cardiovascular disease in this population.

Abbreviations

NDUTH	Niger Delta University Teaching Hospital
FBS	Fasting Blood Sugar
TC	Total Cholesterol
TG	Triglycerides
LDL	Low-density Lipoprotein
HDL	High-density Lipoprotein
BMI	Body Mass Index
CVD	Cardiovascular Diseases
WMA	World Medical Association
ANOVA	Analysis of Variance
NO	Nitric Oxide
TNF-α	Tumour Necrotic Factor α
IL-6	Interleukin 6
ox-LDL	Oxidized Low-Density Lipoprotein
RAAS	Renin Agiontensin Aldosterone System
AGE	Advanced Glycation End Product

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Author Contributions

Simon Akpomemera Erho: Conceptualization, Funding acquisition, Investigation, Methodology, Project administration, Writing – original draft

Wadioni Aduema: Funding acquisition, Investigation,

Methodology

Allen Erighanyoyefa: Funding acquisition, Investigation, Methodology

Mao Ebimobotei Bunu: Funding acquisition, Investigation, Methodology

Blessing Elohor Poripo: Funding acquisition, Investigation, Methodology

Ugorji Nnaemeka Ogbonna: Investigation, Methodology, Project administration, Validation

Chimburuoma Nath-Abraham: Investigation, Methodology, Project administration, Validation

Bruno Chukwuemeka Chinko: Conceptualization, Data curation, Formal Analysis, Investigation, Methodology, Supervision, Validation, Writing – review & editing

Conflicts of Interest

The authors declare no conflicts of interest.

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