

## ASSESSMENT OF CARDIOVASCULAR MARKERS AND RISK-FACTORS AMONG NEWLY DIAGNOSED HYPERTENSIVE PATIENTS IN BENIN CITY

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**Abstract.** Hypertension represents a major cardiovascular risk factor and quite often co-exists with dyslipidemia, further increasing the cardiovascular risk. The present study was done with the aim of studying some cardiovascular markers and risk factors in newly diagnosed hypertensive subjects in Benin City. In this present study, a total of 104 participants comprising 52 hypertensive patients and 52 normotensive controls aged between 20 and 80 years were enrolled. Lipid profile, for the following components, namely TC, LDL-C, HDL-C, and TG, was assayed using standard analytic procedures. Demographic data and anthropometric measures were collected. This analysis indicated that most of the parameters of the lipid profile were significantly associated with the risk factors of CVDs. Hypertensive subjects revealed significantly higher mean systolic and diastolic blood pressure values when compared with normotensive subjects ( $P < 0.001$ ). Comparing hypertensive patients with controls revealed a statistically significant increase in the mean  $\pm$  SEM values of TC and LDL-C to  $240.35 \pm 8.11$  and  $166.98 \pm 7.50$ , respectively, with  $P < 0.001$ . Besides, TG was significantly elevated; its value was  $112.15 \pm 6.96$ , with  $P = 0.018$ , while HDL-C failed to show any statistical difference in its values, with  $46.38 \pm 2.53$  in this series, with  $P = 0.365$ . AIP, CRR, and AC were higher with  $P < 0.001$ . This study has estimated the prevalence of dyslipidemia and a high atherogenic index among hypertensive patients. Results thus provide support for lipid profile monitoring in hypertensive subjects for reduction in cardiovascular events. Earlier detection and timely treatment of dyslipidemia might definitely render better health to these patients.

**Keywords:** *hypertension, dyslipidemia, atherogenic index, cardiovascular markers*

### Introduction

Hypertension, a non-communicable disease characterized by blood pressure that is persistently too high, presents as a major global health problem due to its very high prevalence and strong association with CVDs, such as coronary artery disease, stroke, and heart failure (WHO, 2013). This chronic, non-communicable disease is one of the leading causes of deaths due to cardiac events and stroke worldwide (Correction Naghavi et al., 2015). Hypertension tends to be painless during its early development

period and thus is difficult to detect and treat. If not treated, it could lead to kidney, eye, heart, and brain impairment (Bonow et al., 2002). Whereas primary hypertension accounts for 90% to 95% of the cases, which is usually transmitted genetically together with hormonal imbalances and changes in the volume of blood plasma, aside from environmental factors such as stress and sedentary lifestyle, secondary hypertension accounts for 2% to 10% and is related to disorders in the vascular system, kidneys, and endocrine system (Chobanian et al., 2003). Hypertension is a condition that highly burdens the world population, currently estimated at 1.13 billion people, and is projected to rise to 1.56 billion by the year 2025 (Kearney et al., 2005). Dyslipidemia is one of the major risk factors for CVDs among hypertensive patients, described as a condition of abnormal blood lipid levels, typical of elevated blood levels of TC, LDL-C, and triglycerides, along with reduced levels of HDL-C (Grundy et al., 2006). Dyslipidemia originates and aggravates atherosclerosis, while the latter process is one of the key mechanisms responsible for the development of cardiovascular diseases in general (Chapman et al., 2011).

Evidence from studies indicates that dyslipidemia occurs more frequently in subjects suffering from hypertension compared to those with normal blood pressure, and that usually, lipid levels increase with the rise in blood pressure (Borghetti, 2002; Neaton and Wentworth, 1992). Dyslipidemia is variable; most studies, however, have found that some lipid components such as total cholesterol, triglycerides, and lipoproteins may show marked abnormalities (Taylor and Agbedana, 1983). Dyslipidemia as a modifiable risk factor for cardiovascular diseases was documented by Kannel et al. (1971) hypertension, when combined with dyslipidemia, has additive effects; the overall effect of such combination gets to be multiplicative rather than additive, causing manifold increases in the risks of cardiovascular complications (Grundy et al., 2001). For instance, for every 20/10 mmHg rise in blood pressure throughout the 40-90 years of life has been blamed for a doubling of the risk for fatal coronary events (Olafiranye et al., 2011). Though there exists a strong correlation between the two, hypertension and dyslipidemia, results are divergent for different population subgroups (Castelli and Anderson, 1986).

Although different studies have been conducted on cardiovascular indices, risk factors, and hypertension in the population of this country, very few focused studies have been done among newly diagnosed hypertensive subjects in Benin City. This, therefore, forms the lacuna which this study is trying to fill since this association would be enhanced among this particular group.

## **Materials and Methods**

### ***Study design and participants***

University of Benin Teaching Hospital Benin City, Edo State, Nigeria was used to conduct this study. A total of one hundred and four (104) participants enrolled for the study (fifty-two (52) hypertensive patients diagnosed in Benin City, alongside with fifty-two (52) control group of apparently healthy individuals from the same location). Participants were selected using a convenient sampling method, meeting specific inclusion criteria. Informed consent was obtained from all participants prior to enrollment. Ethical approval was obtained from the Research and Ethics Committee of the University of Benin Teaching Hospital. Hypertensive patients were included if they had a confirmed diagnosis of hypertension without commencement of treatment and

provided consent. Control group participants were included if they were free from hypertension history and consented to participate.

### ***Laboratory analysis of parameters***

Lipid profile parameters; high-density lipoprotein cholesterol, total cholesterol and triglyceride were accessed using standard methods, while low-density lipoprotein cholesterol was calculated using the formula:  $LDL = \text{Total Cholesterol} - (\text{HDL Cholesterol} + \text{Triglyceride}/5)$

### ***Questionnaire and sample collection***

The study has used a questionnaire targeting capturing all data on background information such as age, sex, state of origin, occupation, and marital status. Information is captured on medical history, for example, family history of hypertension, present illness, and type of diabetes mellitus. Other lifestyle determinants for this questionnaire were also determined to ascertain the quantity of tobacco smoked and alcohol consumed, nutrient supplements intake, eating behavior, and physical activities/exercise engagement. Ethical approval for this study was provided by the Health Research Ethics Committee, University of Benin Teaching Hospital, Benin City, Edo State, Reference No: ADME/E 22/A/VOL.VII/14838152172. Sample collection involved 5 mL of fasting venous blood in each group using a sterile syringe and needle under aseptic precautions. The blood samples are drawn into plain containers and allowed to clot. After clotting, the samples undergo centrifugation at 5000 rpm for 5 minutes to separate the serum. This was pipetted out into a sterilized dry container and reserved for the analysis of HDL-C, total cholesterol, and triglycerides. All the serum samples were preserved at -20°C until analysis.

### ***Laboratory investigation***

#### ***Assessment of the level: Cholesterol, Triglycerides, high density lipoprotein***

Assessment of cholesterol levels of the participants were carried out using colorimetric method according to Allain et al. (1974). The reagent was commercially purchased from Randox Limited, United Kingdom, according to the manufacturer's instructions. Level of triglycerides of the participants were determined by colorimetric method according to Bucolo and David (1973). The reagent was commercially purchased from Randox Limited, United Kingdom, according to the manufacturer's instructions. In accordance with Nauck et al. (2002), the colorimetric approach was used to determine the High Density Lipoprotein level by following the manufacturer's directives. The reagent was commercially acquired from Randox Limited, United Kingdom.

#### ***Calculation: Low density lipoprotein, Atherogenic Index of Plasma (AIP), Cardiac Risk Ratio (CRR), Atherogenic Correlation (AC)***

Level of Low Density Lipoprotein Level of the participant was calculated using Friedewald's formula (Friedewald et al., 1972):

$$LDL_{\text{cholesterol}} = \text{Total Cholesterol (TC)} - (\text{HDL}_{\text{cholesterol}} + \text{Triglyceride}/5) \quad \text{Eq. (1)}$$

Values of AIP were derived from the formula (Kanthé et al., 2012):

$$\text{AIP} = \text{Log (TG/HDL-C)} \quad \text{Eq. (2)}$$

Cardiac Risk Ratio was calculated according to the formula:

$$\text{CRR} = \text{TC/HDL-C} \quad \text{Eq. (3)}$$

Atherogenic correlation was calculated according to the formula:

$$\text{AC} = \text{TC-HDL-C/HDL-C} \quad \text{Eq. (4)}$$

### ***Statistical analysis***

Statistical analysis including descriptive statistics and correlation was carried out using the statistical package for social scientist (SPSS) version 20.0. All values were expressed as  $\pm$  standard error of the mean. Means were compared in both groups independent t-test. Subgroup means were compared using analysis of variation test (ANOVA). Pearsons's correlation test was used for correlation study. The P-value ( $< 0.05$ ) was taken as the cut off level of significance.

### **Results and Discussion**

The *Table 1* represents the mean $\pm$ SEM of anthropometric measurements of hypertensive patients and control subjects. This means that in the hypertensive patients, there is a highly significant increase in diastolic and systolic blood pressure with a  $P < 0.001$ , when compared to that in the control group. However, the weight, height, and the BMI in the two groups of patients remained similar, as  $P > 0.05$ . Mean $\pm$ SEM value of total cholesterol, triglycerides, HDL-C, and LDL-C in hypertensive patients and the control subjects. It was manifested that the levels of total cholesterol, triglycerides, and LDL-C in hypertensive patients were apparently higher than those in controls ( $P < 0.05$ ); however, HDL-C did not show any statistical difference between the two groups ( $P = 0.365$ ) (*Table 2*). *Table 3* shows mean $\pm$ SEM of atherogenic indices. There is a significant increment in atherogenic indices,  $P < 0.001$ , in hypertensive patients as compared to control subjects reflecting very high cardiovascular risk. *Table 4* shows relationship of lipid profile with DBP. Total cholesterol, LDL-C, and triglycerides in the hypertensive patients were significantly higher as compared to that of the control subjects ( $P < 0.05$ ), whereas HDL-C showed no significant difference between the two groups ( $P = 0.617$ ). Parameters of lipid profile according to the SBP. According to the data analysis (*Table 5*), the levels of total cholesterol, LDL-C, and triglycerides in the hypertensive group were significantly higher as compared with normotensive controls,  $P < 0.05$ , but the level of HDL-C was not significantly different in both groups,  $P = 0.125$ . *Table 6* presents a correlation table on the levels of lipid profiles with regard to age, weight, height, body mass index, and blood pressure for the hypertensive patients. No significant relation can be found in the above-mentioned parameters.  $P > 0.05$  is considered. *Table 7* shows lipid profile parameters and atherogenic indices by gender in hypertensive subjects. From this table, it was observed that the values for total cholesterol and HDL-C are significantly higher in females compared to the males,

P<0.05, and the levels of LDL-C, total cholesterol, and atherogenic indices were not significantly different from the male to the female respondents.

**Table 1.** Anthropometric measurements of the hypertensive subjects and control subjects.

Parameters	Control (N=52)	Hypertensive patients (N=52)	T value	P value
Weight (kg)	67.21±1.40	69.04±1.58	0.393	0.357
Height (meter)	1.69±0.01	1.67±0.01	-0.703	0.484
BMI (kg/m <sup>2</sup> )	24.42±0.87	24.90±0.42	0.496	0.621
DBP (mmHg)	67.87±0.60	84.98±1.55	10.30	<0.001*
SBP (mmHg)	116.02±1.11	150.65±2.18	14.18	<0.001*

Note: Value are expressed in mean±SEM; SBP=Systolic Blood Pressure; DBP=Diastolic Blood Pressure, BMI=Body Mass Index, \*Significant.

**Table 2.** Levels of lipid profile parameters in hypertensive subjects and control subjects.

Parameters	Control (N=52)	Hypertensive patients (N=52)	T value	P value
T. CHOL (mg/dl)	182.56±6.04	240.35±8.11	5.713	<0.001*
HDL-C (mg/dl)	49.17±1.72	46.38±2.53	-0.911	0.365
LDL-C (mg/dl)	112.42±4.95	166.98±7.50	6.072	<0.001*
TRIG (mg/dl)	92.65±4.17	112.42±4.95	2.403	0.018*

Note: Value are expressed in mean±SEM; TRIG=Triglyceride; LDL-C=Low Density Lipoprotein Cholesterol; HDL-C=High Density Lipoprotein Cholesterol; T.CHOL=Total Cholesterol; \* Significant.

**Table 3.** Atherogenic indices of hypertensive subjects and control subjects.

Parameters	Control (N=52)	Hypertensive patients (N=52)	T value	P value
AIP	0.27±0.02	0.38±0.03	3.676	<0.001*
CRR	3.83±0.14	5.91±0.37	5.194	<0.001*
AC	2.83±0.14	4.91±0.37	5.194	<0.001*

Note: Value are expressed in mean±SEM; AC=Atherogenic Coefficient; AIP=Atherogenic Index of Plasma; CRR=Cardiac Risk Ratio; \*Significant.

**Table 4.** Correlation of lipid profile with diastolic blood pressure in hypertensive subjects and control subjects.

Parameters	Correlation coefficient (r)	P-value
T.CHOL (mg/dl)	0.48	<0.001*
HDL-C (mg/dl)	-0.05	0.617
LDL-C (mg/dl)	0.46	<0.001*
TRIG (mg/dl)	0.30	0.002*

Note: TRIG=Triglyceride; LDL-C=Low Density Lipoprotein Cholesterol; HDL-C=High Density Lipoprotein Cholesterol; T. CHOL=Total Cholesterol; \* Significant.

**Table 5.** Correlation of lipid profile with systolic blood pressure in hypertensive subjects and control subjects.

Parameters	Correlation coefficient (r)	P-value
T.CHOL (mg/dl)	0.503	<0.001*
HDL-C (mg/dl)	-0.152	0.125
LDL-C (mg/dl)	0.522	<0.001*
TRIG (mg/dl)	0.274	0.005*

Note: TRIG=Triglyceride; LDL-C=Low Density Lipoprotein Cholesterol; HDL-C=High Density Lipoprotein Cholesterol; T. CHOL=Total Cholesterol; \* Significant.

**Table 6.** Correlation between lipid profile in hypertensive subjects with anthropometric factors.

Category	Total cholestrol		HDL-C		LDL-C		TRIG	
	R	P-value	R	P-value	R	P-value	R	P-value
Age	0.12	0.415	0.16	0.257	0.07	0.605	0.05	0.737

Weight	-0.30	0.029	-0.23	0.110	-0.17	0.223	-0.19	0.175
Height	-0.17	0.222	-0.26	0.062	-0.46	0.746	-0.08	0.558
BMI	-0.23	0.099	-0.05	0.734	-0.16	0.256	-0.23	0.100
DBP	0.17	0.224	-0.02	0.901	0.12	0.405	0.18	0.210
SBP	0.20	0.150	-0.22	0.115	0.19	0.172	0.11	0.439

Note: SBP=Systolic Blood Pressure; DBP=Diastolic Blood Pressure; BMI=Body Mass Index; *r*=correlation coefficient; TRIG=Triglyceride; LDL-C=Low Density Lipoprotein Cholesterol; HDL-C=High Density Lipoprotein Cholesterol; T.CHOL=Total Cholesterol; \*Significant.

**Table 7.** Levels of lipid profile and atherogenic indices of hypertensive subjects with sex.

Parameters	Female	Male	P-value
AIP	0.32±0.18	0.33±0.15	>0.05
CRR	5.00±2.40	4.70±2.10	>0.05
AC	4.00±2.40	3.70±2.10	>0.05
T.CHOL (mg/dl)	223.00±58.00	195.00±56.00	0.016*
HDL-C (mg/dl)	50.00±17.00	44.00±13.00	0.048*
LDL-C (mg/dl)	147.00±54.00	129.00±52.00	>0.05
TRIG (mg/dl)	52.00±14.00	52.00±15.00	>0.05
AIP	0.32±0.18	0.33±0.15	>0.05

Note: Value are expressed in mean±SEM; AC=Atherogenic Coefficient; AIP=Atherogenic Index of Plasma; CRR=Cardiac Risk Ratio; TRIG=Triglyceride; LDL-C=Low Density Lipoprotein Cholesterol; HDL-C=High Density Lipoprotein Cholesterol; T.CHOL=Total Cholesterol; \*Significant.

Hypertension remains one of the most important public health issues worldwide and is identified as a major risk factor for CVDs, significantly contributing to the global burden of coronary heart disease. Hypertension, coupled with dyslipidemia, makes important contributions to the manifestation of CVDs; together, they account for approximately 30% of global mortality rates (Gupta, 2004). Over time, changes in lipid profiles in patients with hypertension have been reported, showing that hypertension and abnormal lipid profiles often occur together. Many studies have indicated that, in most cases, patients with hypertension also have high LDL-C and triglycerides, with mild elevation in total cholesterol levels (Guo et al., 2012). Our results indicated a significant increase in both systolic and diastolic blood pressure in the hypertensive patients compared to their normal counterparts, as shown in *Table 1*. Although the differences in weight, height, and BMI were insignificant in both groups, the increase in the levels of blood pressure agrees with a report by Palmieri et al. (2001) that said hypertensive and normotensive subjects did not differ significantly in height or BMI. Analysis of the lipid profile shows significantly higher values of total cholesterol, LDL-C, and triglyceride in the hypertensive subjects compared to normotensive subjects (*Table 2*), which is in agreement with previous studies conducted in Nigeria by Asaolu et al. This indeed showed significantly higher levels of these lipids among the hypertensive subjects than the normotensive controls; thus suggesting that these may be related to factors such as impaired endothelial function, physical inactivity regularly, high intake of dietary fat, and genetic predisposition.

For dyslipidemia and hypertension, there was adequate work within the Nigerian populace to say most of them pointed out a significant relationship between the two variables under study (Osuji et al., 2012; Asaolu et al., 2010; Idemudia et al., 2009). This relationship runs differently under different populations and trends. Otsuka et al. (2021) indicated that, out of the 14,215 working-age Japanese males, increased levels of

total cholesterol, low-density lipoprotein cholesterol, and triglycerides were those that developed hypertension. In another study by He et al. comprising 9,540 Chinese aged above 40 years, triglyceride levels and reduced high-density lipoprotein cholesterol were seen to be related to the development of hypertension in the subjects studied by Alberti et al. (2005) as well as He and MacGregor (2003), these may be based on the general way of life, genetic situation, and dietary conditions in different populations. Very recently, one Chinese study was conducted by Yang et al. after adjustment for BMI and presented positive association of HDL-C level with the risk of hypertension in sharp contrast to its classic status of "good cholesterol". Regarding this, it may give a clue to the functional role of HDLC independent of its level. It has been demonstrated that changes in HDL-C alone do not confer any additional cardiovascular benefit Cannon et al. (2010). In our study, there was a significant positive correlation of SBP with HDL-C and a negative correlation of DBP with HDL-C, reflecting a complex relationship. It is also possible that HDL-C increases the bioavailability of nitric oxide in endothelial cells, with the result of vasodilation; the action thus may raise systolic blood pressure but lower peripheral resistance and diastolic pressure. This might be because the majority of the hypertensive patients were suffering from insulin resistance, endothelial dysfunction, inflammatory responses, genetic predisposition, and medications which could result in a high level of total cholesterol, triglycerides, and LDL-C. Furthermore, the majority of our respondents showed sedentary lifestyles and dietary behaviors which might have caused dyslipidemia. Most participants revealed a family history of hypertension which again gives significance to the role of genetic predisposition in these disorders.

Sensitive markers for atherogenic dyslipidemia have been sought in research studies with the hope of increasing predictability of CVD risk and monitoring of responses to treatment. The AIP in this respect has been considered by Sami Khaza (2013), Tan et al. (2004) as well as Dobiášová and Frohlich (2001) as a strong predictor of the risk for atherosclerosis and coronary heart disease. The Atherogenic Index of Plasma is a more sensitive indicator of the balance between protective and atherogenic lipoproteins, representing the balance between the protective and atherogenic lipid particles (Dobiášová et al., 2011). Our study showed a significant elevation of atherogenic indices, including AIP, CRR, and AC, among hypertensives (*Table 3*). These findings align with those of Cheng et al. (2022) who reported higher atherogenic indices among hypertensive patients compared to normotensive controls, regardless of gender, age, or BMI. The increase in the atherogenic indices in hypertensive patients is an interaction between dyslipidemia, chronic low-grade inflammation, insulin resistance, and oxidative stress; all these usually can be overlapping and interacting with each other to create a very promoting milieu of atherosclerosis development and enhance the risk of cardiovascular events. Thus, comprehension of these will be important in devising a strategy for treatment and mitigation of risks associated with hypertension and dyslipidemia. The case study discusses the regular follow-up of the lipid profile in hypertensive patients and signifies life modifications with diet and exercise in order to counteract dyslipidemia and avoid cardiovascular complications. Early identification and proactive management of these risk factors would result in a radical improvement in the outcomes, and one would suffer very little from cardiovascular diseases.

## Conclusion

In fact, the new diagnoses of hypertension in Benin City had significantly high levels of total cholesterol, low-density lipoprotein cholesterol, and triglycerides, with increased atherogenic indices such as atherogenic index of plasma, cardiac risk ratio, and atherogenic coefficient, hence an increased cardiovascular risk when compared with normotensive subjects. Again, this finding calls for regular lipid profile evaluation among hypertensive patients. These early changes in lifestyle, like adequate nutrition and exercises, besides proper management will help reduce the chances of cardiovascular complications. In addition, more genetic and environmental research into dyslipidemia will no doubt complement these better preventive measures and enable the patients to result in improved outcomes with reduced cardiovascular diseases risks.

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## Conflict of interest

The authors confirm that there is no conflict of interest involve with any parties in this research study.

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