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Evaluation of Endothelial Functions in Essential Hypertensive Subjects in Ekiti State.

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Abstract

Background: Endothelial dysfunction plays an important role in vascular complications and pathogenesis of essential hypertension marked by an imbalance between vasodilatory and vasoconstrictive factors, increased inflammatory activation, and pro-atherogenic modifications. This study assessed endothelial functions in essential hypertensive subjects in Ekiti State, Nigeria.

Methodology: A total of 126 participants (63 hypertensives and 63 controls) were recruited. Social demographic information was obtained using a structured questionnaire. Height and weight were measured with a stadiometer and ZT-120 scale, and blood pressure was assessed using a digital sphygmomanometer. ET-1 and P-selectin were quantified using ELISA; nitric oxide and lipid profile using spectrophotometry, with lipid indices calculated. Data were statistically analyzed at $p<0.05$.

Result: The results revealed that body mass index, family history of hypertension, systolic and diastolic blood pressure, Endothelin-1, Total cholesterol, Triglyceride, Low Density Lipoprotein and P-selectin levels were significantly higher in the essential hypertensive subjects compared to the healthy controls; however, Nitric oxide and High-Density Lipoprotein were significantly lower in the essential hypertensive subjects compared to healthy controls. There was no statistically significant difference in all parameters studied based on age and sex. The results also revealed that Endothelin-1 and P-selectin levels were significantly higher in drug-naive hypertensive subjects compared with those on treatment.

Conclusion: The study highlights the role of endothelial biomarkers in the pathophysiology of essential hypertension, particularly ET-1 and P-selectin, which showed greater sensitivity and specificity statistically may serve as an early indicator of vascular dysfunction and potential targets for risk classification and disease management.

Keywords: Essential hypertension, Endothelial dysfunction, Endothelin-1, P-selectin, Nitric oxide, Lipid profile.

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INTRODUCTION

Hypertension is a significant public health issue because of its widespread occurrence and correlation with cardiovascular problems. About 95% of hypertension cases are categorized as essential hypertension (EH), a multifaceted condition defined by elevated systolic blood pressure (SBP) of 140 mmHg and/or diastolic blood pressure (DBP) of approximately 90 mmHg, resulting from a confluence of environmental and hereditary influences[2].

The vascular endothelium is essential to cardiovascular physiology since it establishes an interface between blood and adjacent tissues, facilitates the movement of nutrients and metabolites, and engages with hormones, cytokines, and circulating cells[3]. Endothelial dysfunction is characterized by diminished vasodilation, increased cell proliferation, enhanced platelet adhesion and activation, with a proinflammatory and prothrombotic condition[3].

Nitric oxide (NO) regulates several biological processes, including vascular tone, blood pressure, neurotransmission, coagulation, immunological response, and oxidative mechanisms. Selectins, a kind of cell adhesion molecule, are essential for endothelial function and platelet activation and are linked to the pathophysiology of heart failure (HF). P-selectin exhibits a poor link with outcomes in several cardiovascular disorders, such as coronary artery disease and hypertension[5]. Endothelin-1 (ET-1) is a peptide essential for the regulation of several physiological systems in the body. ET-1 induces vasoconstriction of blood vessels owing to its powerful vasoconstrictive characteristics[6].

Dyslipidemia, defined by irregular blood lipid concentrations, can increase the risk of cardiovascular diseases. Dyslipidemia may induce endothelial dysfunction, which is characterized by the endothelium's inability to respond to physiological stimuli and maintain vascular homeostasis. Dyslipidemia can impact the structure and function of cardiac muscle, leading to arrhythmias, fibrosis, and ventricular hypertrophy[7].
Atherogenic indices are biochemical markers indicating the association among various lipid fractions in the blood, utilized to evaluate the risk of atherosclerosis and cardiovascular events[8]. These indices provide a more thorough evaluation of lipid profiles compared to traditional techniques of assessing HDL, LDL, and total cholesterol separately. [9]

The Atherogenic Index of Plasma (AIP) and the Castelli Risk Index (CRI) I and II are simple ratios calculated from a person's lipid profile, employed as screening tools for identifying increased cardiovascular risk. CRI-II is determined by the ratio of total cholesterol (TC) to high-density lipoprotein (HDL) cholesterol, while CRI-I is computed from the ratio of low-density lipoprotein (LDL) to HDL cholesterol[10]. The atherogenic coefficient (AC) is a crucial metric for assessing the degree of atherosclerosis, calculated as (TC – HDL)/HDL[11].

METHODOLOGY

This research was a case-control study including two participant groups: individuals with essential hypertension and a control group of apparently healthy individuals. A total of 126 participants were recruited for this study, including 63 individuals in the test group and 63 in the control group, from Ekiti State University Teaching Hospital, Ado-Ekiti, and Federal Teaching Hospital, Ido-Ekiti, Ekiti State. This study comprised individuals aged 18-50 years diagnosed with essential hypertension (cases) and normotensive individuals devoid of a hypertension history (controls), while excluding patients with secondary hypertension, diabetes, chronic renal disease, and pregnant women.

Informed permission was obtained from all participants before their inclusion in the study. A questionnaire was created to collect socio-demographic information. Anthropometric and blood pressure assessments were performed in accordance with recognized protocols. The weight and height of each participant were measured using a stadiometer in conjunction with a ZT-120 health scale. Measurements were conducted with individuals standing upright, attired in lightweight garments, and barefoot. Height was measured to the nearest 0.01 meter (m) and weight to the nearest 0.5 kilogram (kg). The body mass index (BMI) was determined by dividing weight (in kilograms) by the square of height (in meters) using the formula: BMI = Weight (kg) / Height (m^2). The resulting value is denoted in kg/m^2. The blood pressure measurements were taken with a digital sphygmomanometer, namely the OMRON M7 Basic Automatic Upper Arm Blood Pressure Monitor (Model code: M7 Intelli IT (HEM-736IT-EBK)) produced by OMRON HEALTHCARE Co., Ltd. in Japan. Blood samples were obtained through venipuncture from all subjects and placed in a sterile

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container to evaluate the various biochemical markers. Endothelin-1 and P-selectin were measured using a sandwich Enzyme-Linked Immunosorbent Assay (ELISA). Nitric oxide and lipid profile were quantified using a spectrophotometer. Atherogenic indices were calculated using the values from the lipid profile as follows:

The atherogenic index of plasma (AIP) = log (TGL/HDL-c)[12].
Castelli's risk index I (CRI-I) = TC / HDL-c[11].
Castelli's Risk Index II (CRI-II) = LDL-c / HDL-c[11].
Atherogenic coefficient (AC) = (TC–HDL-c) / HDL-c[13].
Ethical approval for this study was obtained from the Research and Health Ethics Committee of Afe Babalola University, Ado-Ekiti; Ekiti State University Teaching Hospital, Ado-Ekiti; and Federal Teaching Hospital, Ido-Ekiti, Ekiti State.

Data analysis was conducted using version 26.0 of the Statistical Package for the Social Sciences (SPSS) for Windows, with findings expressed as mean ± standard deviation (SD). Statistical techniques such as the Student's t-test, Pearson correlation, and chi-square test were employed. A significance threshold of $p < 0.05$ was considered appropriate. The Receiver Operating Characteristic was used to evaluate the sensitivity, specificity, and diagnostic accuracy of the biomarkers.

RESULTS

Table 1 showed the socio-demographic characteristics of the subjects and control. The results obtained showed that a family history of hypertension was significantly higher in hypertensive subjects compared to control ($p>0.05$). There was no significant difference in other demographic variable studied between the hypertensive subjects and controls respectively.

Table 1: Socio-demographic characteristics of the subjects and control

Variable	Subjects (n=63)	Control (n=63)	Statistical Analysis
Gender			
Male	25 (45.0%)	30 (46.7%)	$X^2 = 1.034$
Female	38 (55.0%)	33 (53.3%)	$p = 0.798$
Age (years)			
20 – 30	4 (6.3%)	31 (49.2%)	$X^2 = 1.457$
31 – 40	3 (4.8%)	13 (20.6%)	$p = 0.321$
41 – 50	56 (88.9%)	19 (30.2 %)	
Family history			
Yes	41 (65.1%)	14 (22.2%)	$X^2 = 6.432$
No	22 (34.9%)	49 (77.8%)	$p = 0.016$*
Exercise per week			
Daily	4 (6.3%)	13 (20.6%)	
Less than 1 hour	11 (17.5%)	2 (3.2%)	$X^2 = 1.540$
1-2 hours	28 (28.6%)	21 (33.3%)	$p = 0.462$
3-4 hours	21 (33.3%)	27 (42.9%)	
Never	9 (14.3%)	0 (0%)	
Sleep hours			
Less than 4 hours	10 (15.9%)	0 (0%)	
4-6 hours	44 (69.8%)	51 (81.0%)	$X^2 = 1.398$
7-8 hours	7 (11.1%)	12 (19.0%)	$p = 0.504$
More than 8 hours	2 (3.2%)	0 (0%)	
Alcohol consumption			
Yes	9 (14.3%)	2 (3.2%)	$X^2 = 1.765$
No	54 (85.7%)	61 (96.8%)	$p = 0.373$
Smoking			
Yes	2 (3.2%)	0 (0%)	$X^2 = 1.631$
No	61 (96.8%)	63 (100%)	$p = 0.389$

*Statistically significant at $p < 0.05$

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Table 2 shows the anthropometric characteristics of the subjects and controls. The results obtained showed that weight, BMI, SBP and DBP were significantly higher in subjects compared to control ($p<0.05$). However, there was no significant difference in the height of the subjects compared to controls ($p>0.05$).

Table 2. Anthropometric status of subjects and controls

Parameters	Subjects (n=63)	Control (n=63)	t-value	p-value
Weight (kg)	71.67±10.84	62.11±8.30	4.790	0.000*
Height (m)	1.64±0.06	1.64±0.08	0.671	0.505
BMI (kg/m²)	26.76±4.79	23.13±2.99	5.638	0.000*
SBP (mmHg)	142.02±9.74	109.33±7.57	4.482	0.001*
DBP (mmHg)	85.44±10.89	70.33±7.57	6.009	0.000*

*Statistically significant at $p < 0.05$

Keys: **BMI** – Body mass index; **SBP** – Systolic blood pressure; **DBP** – Diastolic blood pressure

Table 3 shows the biochemical parameters of the subjects compared with control. The results obtained showed that TC, TG, LDL, P-selectin and ET-1 were significantly higher in hypertensive subjects ($p<0.05$) compared to normotensive controls. On the hand, HDL and NO were significantly lower in hypertensive subjects ($p<0.05$) compared to normotensive control. There was no significant difference in the AIP, AC, CRI-I and CRI-II of the subjects compared to control ($p>0.05$).

Table 3. Biochemical parameters of subjects and controls

Parameters	Subjects (n=63)	Control (n=63)	t-value	p-value
TC (mmol/L)	5.54±1.29	4.99±0.89	2.806	0.006*
TG (mmol/L)	1.30±0.44	1.16±0.26	2.240	0.029*
HDL (mmol/L)	0.71±0.13	1.00±0.14	2.769	0.007*
LDL (mmol/L)	3.97±1.15	3.50±0.96	2.555	0.013*
NO (µmol/L)	36.64±9.01	67.35±12.86	16.806	0.000*
ET-1 (pg/ml)	5.44±1.16	3.76±0.77	7.115	0.000*
P-selectin (ng/ml)	137.84±31.61	89.36±28.50	8.697	0.000*
AIP	0.21±0.10	0.20±0.08	0.593	0.437
AC	6.17±1.53	6.18±1.40	0.398	0.513
CRI-I	7.16±1.54	7.18±1.41	0.469	0.501
CRI-II	5.15±1.48	5.03±1.43	0.524	0.601

*Statistically significant at $p < 0.05$

Keys: **TC** – Total cholesterol, **TG** – Triglycerides, **HDL** – High density lipoprotein,
LDL – Low density lipoprotein, **NO** – Nitric oxide, **ET-1** – Endothelin-1,
AIP – Atherogenic index of plasma, **AC** – Atherogenic coefficient, **CRI-I**- Castelli risk index I, **CRI-II** – Castelli risk index II

Figure 1 and 2 showed the biochemical parameters of the subjects according to gender. The results obtained showed that there was no significant difference in the biochemical parameters studied with respect to gender ($p>0.05$). There was no significant difference in the AIP, AC, CRI-I and CRI-II of the subjects according to gender ($p>0.05$).

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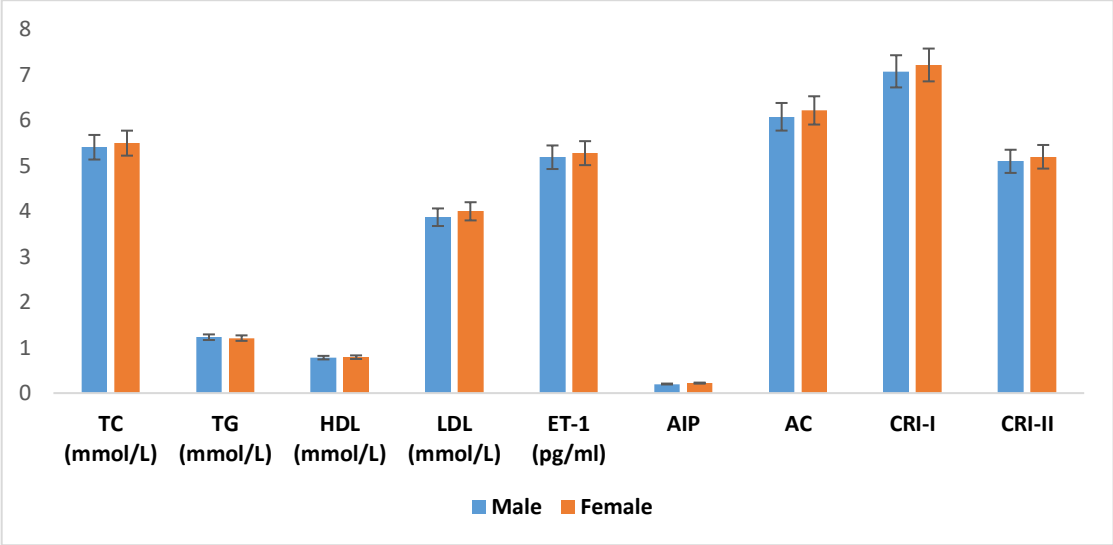

Figure 1: Biochemical parameters of the subjects according to gender

Keys: **TC** – Total cholesterol, **TG** – Triglycerides, **HDL** – High density lipoprotein, **LDL** – Low density lipoprotein, **ET-1** – Endothelin-1, **AIP** – Atherogenic index of plasma, **AC** – Atherogenic coefficient, **CRI-I**- Castelli risk index I, **CRI-II** – Castelli risk index II

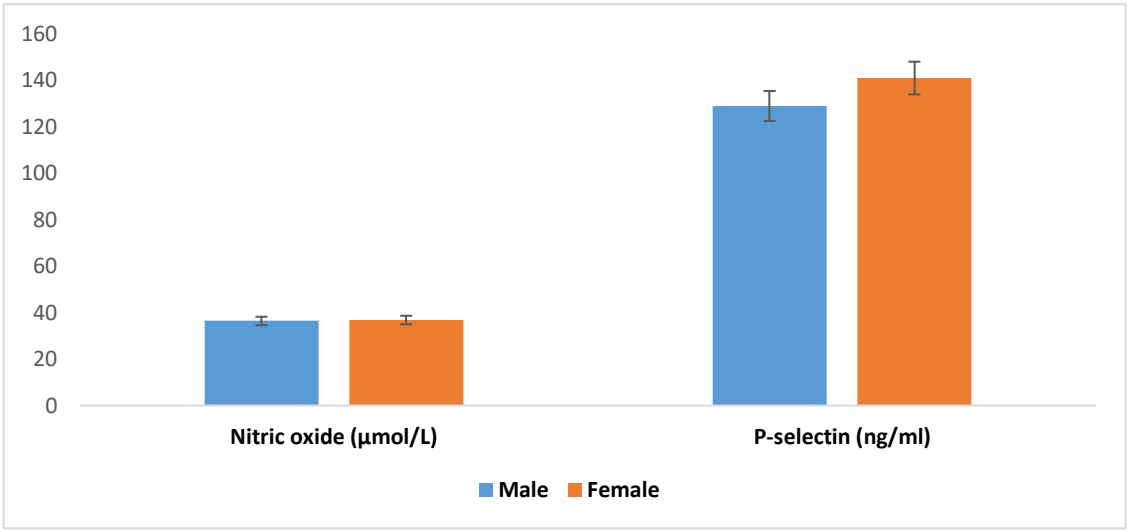

Figure 2: Biochemical parameters of the subjects according to gender

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Figure 3 and 4 showed the biochemical parameters of the subjects according to age. The results obtained showed that there was no statistically significant difference in the biochemical parameters of the subjects with respect to age ($p>0.05$). There was no significant difference in the AIP, AC, CRI-I and CRI-II of the subjects with respect to age ($p>0.05$).

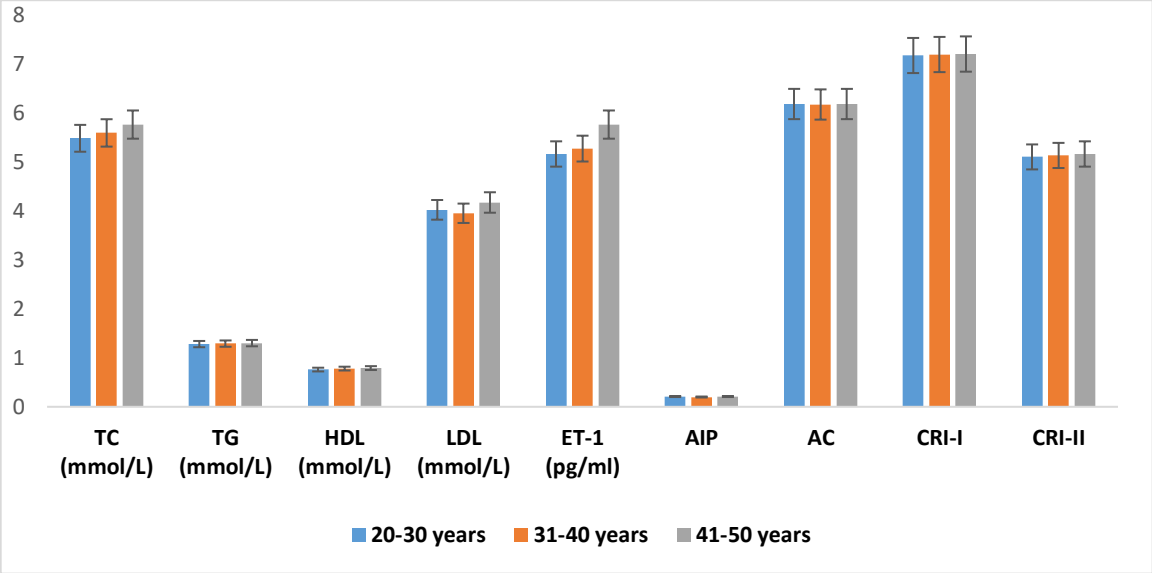

Figure 3: Biochemical parameters of the subjects according to age

Keys: **TC** – Total cholesterol, **TG** – Triglycerides, **HDL** – High density lipoprotein, **LDL** – Low density lipoprotein, **ET-1** – Endothelin-1, **AIP** – Atherogenic index of plasma, **AC** – Atherogenic coefficient, **CRI-I**- Castelli risk index I, **CRI-II** – Castelli risk index II

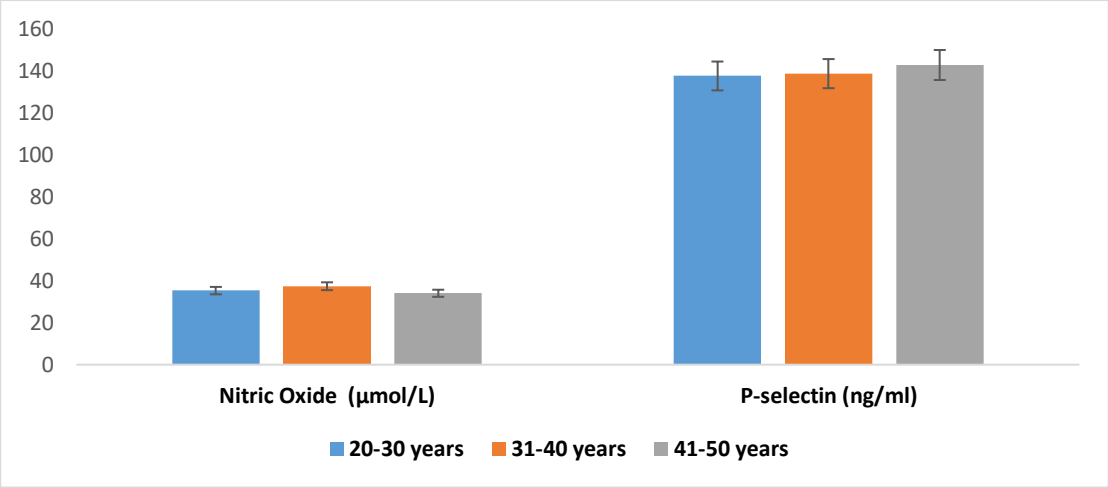

Figure 4: Biochemical parameters of the subjects according to age

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Table 4 shows the biochemical parameters of drug naive subjects compared to those on treatment. The results obtained showed that ET-1 and P-selectin were significantly higher in drug naive hypertensive subjects compared with those on treatment ($p<0.05$). However, there was no significant difference in the lipid profile and nitric oxide of drug naive subjects compared to those on treatment ($p>0.05$). There was no significant difference in the AIP, AC, CRI-I and CRI-II of the subjects' drug naive compared to those on antihypertensive drugs ($p>0.05$).

Table 4: Biochemical parameters of newly diagnosed subjects compared to those on treatment

Parameters	Drug Naïve (n=31)	On Treatment (n=32)	t-value	p-value
TC (mmol/L)	5.69±1.27	5.34±1.36	1.071	0.293
TG (mmol/L)	1.34±0.49	1.25±0.37	0.663	0.512
HDL (mmol/L)	0.78±0.13	1.0±0.15	0.190	0.851
LDL (mmol/L)	4.09±1.07	3.82±0.96	0.989	0.331
NO (µmol/L)	36.82±8.97	36.24±9.35	0.248	0.806
ET-1 (pg/ml)	6.59±1.01	4.38±1.17	6.541	0.000*
P-selectin (ng/ml)	171.33±28.26	107.21±26.15	8.010	0.000*
AIP	0.22±0.10	0.20±0.09	0.765	0.561
AC	6.19±1.50	6.18±1.38	0.423	0.610
CRI-I	7.18±1.42	7.18±1.40	0.789	0.357
CRI-II	5.16±1.36	5.03±1.38	0.612	0.529

*Statistically significant at $p < 0.05$

Keys: **TC** – Total cholesterol, **TG** – Triglycerides, **HDL** – High density lipoprotein,
LDL – Low density lipoprotein, **NO** – Nitric oxide, **ET-1** – Endothelin-1, **AIP** – Atherogenic index of plasma, **AC** – Atherogenic coefficient, **CRI-I**- Castelli risk index I, **CRI-II** – Castelli risk index II

Table 5 showed the correlations of the biochemical parameters studied. The results obtained showed that TC had significant positive correlation with TG (r=0.562, p=0.000), HDL (r=0.534, p=0.000), LDL (r=0.943, p=0.000) and P-selectin (r=0.248, p=0.050) respectively. There was significant positive correlation between TG and HDL (r=0.570, p=0.000), LDL (r=0.435, p=0.000), and NO (r=0.269, p=0.033) respectively. Similarly, there was significant positive correlation between HDL and LDL (r=0.450, p=0.000), and between ET-1 and P-selectin (r=0.770, p=0.000) respectively.

Table 5: Correlations of the biochemical parameters studied

		TC	TG	HDL	LDL	NO	ET1	P-sel
TC	Pearson Correlation		0.562**	0.534**	0.943**	0.244	0.166	0.248*
	Sig. (2-tailed)		0.000	0.000	0.000	0.054	0.193	0.050
TG	Pearson Correlation	0.562**		0.570**	0.435**	0.269*	0.205	0.207
	Sig. (2-tailed)	0.000		0.000	0.000	0.033	0.106	0.104
HDL	Pearson Correlation	0.534**	0.570**		0.405**	0.190	0.126	0.199
	Sig. (2-tailed)	0.000	0.000		0.001	0.135	0.325	0.117
LDL	Pearson Correlation	0.943**	0.435**	0.405**		0.288*	0.109	0.206
	Sig. (2-tailed)	0.000	0.000	0.001		0.022	0.394	0.105
NO	Pearson Correlation	0.244	0.269*	0.190	0.288*		0.040	-0.003
	Sig. (2-tailed)	0.054	0.033	0.135	0.022		0.754	0.980
ET1	Pearson Correlation	0.166	0.205	0.126	0.109	0.040		0.770**
	Sig. (2-tailed)	0.193	0.106	0.325	0.394	0.754		0.000
Psel	Pearson Correlation	0.248*	0.207	0.199	0.206	-0.003	0.770**	
	Sig. (2-tailed)	0.050	0.104	0.117	0.105	0.980	0.000	

**. Correlation is significant at the 0.01 level (2-tailed).
*. Correlation is significant at the 0.05 level (2-tailed).

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Table 6 showed the Area Under Curve (AUC) of all parameters studied. The test result variable(s): TC, TG, HDL, LDL, Nitric oxide, ET-1 and P-selectin has at least one tie between the positive actual state group and the negative actual state group. From the results obtained, the AUC, sensitivity and specificity of TC were 0.659, 74% and 71%, TG were 0.576, 88% and 78%, HDL was 0.636, 87% and 80%, LDL was 0.633, 76% and 72%, NO was 0.011, 55% and 56%, ET-1 was 0.815, 86% and 89%, while P-selectin was 0.807, 79% and 82% respectively.

Table 6: Receiver operating characteristic curve analysis of continuous variables for hypertension

Variables	AUC	p-value	Sensitivity	Specificity
TC	0.659	0.000	74%	71%
TG	0.576	0.000	88%	78%
HDL	0.636	0.000	87%	80%
LDL	0.633	0.000	76%	72%
NO	0.011	0.000	55%	56%
ET-1	0.815	0.000	86%	89%
P-selectin	0.807	0.000	79%	82%

Keys: **TC** – Total cholesterol, **TG** – Triglycerides, **HDL** – High density lipoprotein, **LDL** – Low density lipoprotein, **NO** – Nitric oxide, **ET-1** – Endothelin-1

DISCUSSION

The prevalence of hypertension is influenced by genetic and environmental factors, and its adverse effects stem not only from hemodynamic stress but also from various cardiovascular risk factors, including metabolic syndrome (MetS), which frequently coexists in hypertensive individuals[14]. Essential or primary hypertension is characterized by elevated blood pressure without other causes, such as pheochromocytoma, renal failure, renovascular disease, or aldosteronism[15]. Ninety-five percent of hypertension cases are classified as essential hypertension, characterized by distinct causative factors for increased blood pressure across different people. This research assessed endothelial markers in individuals with essential hypertension.

This study demonstrated a statistically significant elevation ($p<0.05$) in systolic blood pressure (SBP) and diastolic blood pressure (DBP) in hypertensive individuals compared to healthy controls. Essential hypertension significantly increases the risk of morbidity and mortality linked to coronary heart disease, stroke, and kidney disease, and is characterized by a persistent rise in systolic and diastolic blood pressure. Increased systolic blood pressure may lead to renal impairment by intensifying oxidative stress and influencing myogenic contractility in patients with hypertension. This result corresponds with previous research[16-19] that confirmed the significant increase in SBP and DBP in persons with hypertension.

This study observed a substantial rise in BMI in hypertensive individuals, irrespective of treatment, as compared to healthy controls. This research suggests that fat and overweight may both induce and exacerbate the onset of essential hypertension. This finding supports prior research that has shown a markedly increased BMI in persons with hypertension compared to healthy controls[19-21].

Our study found that individuals with essential hypertension had significantly increased blood levels of ET-1 compared to normotensive controls. Elevated ET-1 levels in hypertensive patients correspond with previous clinical studies demonstrating that serum ET-1 concentrations were significantly increased in persons with high blood pressure[22-24].

The study revealed that P-selectin levels were significantly higher in essential hypertensive subjects ($p<0.05$) in comparison to normotensive controls. This research demonstrates that the expression levels of P-selectin are modulated by the exposure of endothelial cells, platelets, or both to mechanical stress induced by blood pressure. This research corroborates previous studies demonstrating that P-selectin blood levels increase in individuals with hypertension relative to healthy controls[25,26]. In individuals with essential hypertension, plasma P-selectin levels have been shown to correlate with reduced vascular resistance, a marker of hypertension-related vascular damage[27].

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This study indicated that nitric oxide levels significantly decreased in individuals with hypertension compared to healthy normotensive controls. This finding is consistent with previous studies. Barbadoro et al. (2021) noted that patients with hypertension displayed markedly reduced amounts of nitric oxide in comparison to normotensive individuals[28]. Zhang et al. (2020) reported a reduction in nitric oxide levels in hypertensive persons, ascribed to elevated reactive oxygen species and oxidative stress[29]. An altered balance between nitric oxide (NO) and reactive oxygen species (ROS) may restrict the bioavailability of NO, resulting in reduced endothelium-dependent vasodilation, which ultimately induces or worsens hypertension. Moreover, oxidized LDL-C[30] may diminish the synthesis of endothelial nitric oxide synthase (eNOS).

This investigation revealed significantly higher serum total cholesterol, triglycerides, and low-density lipoprotein levels in individuals with hypertension compared to normotensive controls. This corresponds with prior observations in several worldwide locations and in other parts of Nigeria[31-34]. Increased levels of cholesterol in the blood have been found to elevate the chance of macrovascular outcomes, such as coronary heart disease (CHD) and stroke[35]. This hypertriglyceridemia may be due to hypercoagulability. The increased triglyceride levels observed in hypertension are likely to concentrate in certain vulnerable arteries, therefore worsening endothelial dysfunction and promoting the production of LDL cholesterol, as evidenced by this study, which indicates an increase in serum LDL-C levels. Dyslipidaemia adversely affects the vascular endothelium and modulates blood pressure; hence, vascular endothelial function is likely the link between hypertension and hyperlipidaemia. Abnormal lipids may indirectly affect arterial elasticity by influencing vascular endothelial regulation, hence leading to hypertension[36].

In this study, HDL-C levels were significantly lower in hypertension participants compared to healthy normotensive controls. This discovery aligns with other research investigations [32-34]. Moreover, it has been established that a low HDL-C level correlates with the presence of other atherogenic risk factors, some of which are novel risk variables not assessed separately in prevalence evaluations. Mackness et al. (2020) suggest that alterations in lipid metabolism, particularly a decrease in HDL-C, might result in endothelial damage and increased blood pressure, thereby elucidating its substantial prognostic value for coronary heart disease (CHD)[37].

This study demonstrated no significant difference in atherogenic indices between individuals with essential hypertension and normotensive controls, supporting a previous study that similarly found no notable difference in the distribution of atherogenic indices across comorbidities, including diabetes mellitus, hypertension, coronary artery disease, or statin use. Contrary to current research, several writers have shown that atherogenic indices, or lipid ratios, have emerged as significant diagnostic and conceptual tools for predicting cardiovascular disease risk in recent years[39]. This investigation revealed no significant changes in the lipid profile and endothelial markers of hypertensive people with respect to age and gender. These findings validate previous research by Kostov et al. (2021), which demonstrated that no significant alterations in lipid profiles and endothelial markers were seen in relation to age or gender.

ET-1 and P-selectin concentrations were significantly higher in drug-naive hypertensive subjects relative to those on treatment. However, there was no significant difference in the lipid profile and nitric oxide levels between drug-naive patients and those receiving treatment. The findings suggest that the timely detection and management of hypertension may be crucial in avoiding or mitigating endothelial dysfunction. The findings indicate the potential role of ET-1 and P-selectin in the initiation and progression of hypertension[23].

The results demonstrated a significant positive correlation between TC and TG, HDL, LDL, and P-selectin, respectively. A notable positive correlation was seen between TG and HDL, LDL, and NO, respectively. A notable positive link existed between HDL and LDL, as well as between ET-1 and P-selectin. In hypertensive individuals, increased lipid levels, namely total cholesterol, triglycerides, and LDL, correlate with impaired endothelial function, as shown by raised levels of endothelial markers such as ET-1 and P-selectin. This connection signifies that abnormal lipid profiles lead to endothelial dysfunction, a critical factor in hypertension and cardiovascular disease[40]. This

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observation supports previous studies[23,24]. Furthermore, increased ET-1 levels are associated with increased P-selectin and reduced nitric oxide (NO) production. This connection suggests that ET-1 may contribute to vascular dysfunction and elevated blood pressure in hypertension. ET-1 is directly linked to endothelial dysfunction, leading to increased P-selectin expression and reduced nitric oxide generation[41]. ET-1 can directly affect nitric oxide synthesis by suppressing the expression of endothelial nitric oxide synthase (eNOS).

Limitation

The limitation of this study may be due to the number of subjects recruited in this study, which were the subjects who presented to the medical clinic during the period of study

CONCLUSION

In conclusion, our findings indicate that serum Endothelin-1 (ET-1), P-selectin, Total Cholesterol (TC), Triglycerides (TG), and Low-Density Lipoprotein (LDL) levels are significantly elevated, whereas High-Density Lipoprotein (HDL) and Nitric Oxide (NO) levels are diminished in patients with essential hypertension, potentially facilitating the advancement of the hypertensive condition. No significant changes were observed in the lipid profile and endothelium markers of hypertensive persons with respect to age and gender; however, levels of ET-1 and P-selectin were significantly increased in drug-naive hypertensive subjects compared to those receiving treatment. Increased production of endothelial markers in the vascular wall may provoke oxidative stress, dyslipidemia, and low-grade inflammation, resulting in endothelial dysfunction and increased vasoconstrictor activity.

DECLARATION

Conflict of Interest:

The authors declare no conflict of interest.

Ethical Approval:

Ethical approval for this study was obtained from the Health Research Ethics Committees of:

i. Afe Babalola University, Ado-Ekiti (PROTOCOL NUMBER: ABUADHREC/27/02/2025/615)
ii. Ekiti State University Teaching Hospital, Ado-Ekiti (PROTOCOLNUMBER: EKSUTH/A67/2025/04/039)
iii. Federal Teaching Hospital, Ido-Ekiti (POTOCOL NUMBER: ERC/2025/03/24/1228B)

All procedures involving human participants were conducted in accordance with the ethical standards of the institutional review boards and with the 1964 Declaration of Helsinki. Written informed consent was obtained from all individual participants involved in the study.

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