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ORIGINAL RESEARCH

Adiposity, Proinflammatory Indices, Apolipoproteins and Antioxidants' Activities in Relation to the Severity of Hypertension

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Abstract

Background: Hypertension is an important risk for several chronic illnesses, notably heart failure, stroke, chronic renal disease and others. In these chronic disease conditions, promising biochemical parameters could be of immense diagnostic value.
Objectives: To investigate adiposity indices, lipid Profile, apolipoproteins, proinflammatory markers and antioxidant status among subgroups of hypertensive patients.
Methods: The study involved 150 participants aged 18-65 years, consisting of 50 normotensives as control subjects and 100 hypertensive individuals as the test group. The blood pressures (BP) of the participants were measured. Their body mass index and waist-to-hip ratio were calculated. Serum lipid profile, serum levels of apolipoprotein-A, apolipoprotein-B, glutathione, catalase, superoxide dismutase, high sensitivity C-reactive protein, tumour necrosis factor-α and interleukin-6 were also measured.
Results: There was a significant increase ($p<0.001$) in adiposity measures, Apo-B ($p<0.001$), IL-6 ($p<0.001$), hs-CRP levels ($p<0.001$), and dyslipidaemia among hypertensive patients. Apo A ($p<0.001$), glutathione ($p<0.001$) and SOD ($p<0.001$) showed significantly lower levels in the test group. A strong positive correlation was observed between BP and anthropometric profiles (BMI, WC; $r = 0.754$; $p = 0.001$), serum lipids (TC, triglycerides; $r = 0.417$; $p = 0.01$), Apo-B ($r = 0.726$; $p = 0.01$) and proinflammatory markers (TNF-α, IL-6; $r = 0.845$; $p = 0.01$). Also, a strong negative but significant correlation existed between BP and antioxidants (GSH, SOD; $r = -0.632$; $p = 0.01$), and Apo-A ($r = -0.838$; $p = 0.01$).
Conclusion: The study showed that adiposity indices, serum lipid parameters, serum apolipoproteins, proinflammatory markers and antioxidants could serve as diagnostic tools for complications of hypertension.

Keywords: Anthropometry, Antioxidants, Dyslipidaemia, Hypertension, Serum lipid profile, Proinflammatory markers.

Introduction

Hypertension is an important cardiovascular disease that is associated with significant mortality and morbidity all over the world. It has been defined and classified using the recommendations of the Seventh Joint National Committee for the Prevention, Detection, Evaluation and Treatment of High Blood Pressure. [1] It is defined as blood pressure higher than normal; normal (systolic < 120mmHg, diastolic < 80mmHg). Hypertension is classified into mild or pre-hypertension (systolic 120-139mmHg, diastolic 80-89mmHg), moderate or Stage 1 (systolic 140-159, Diastolic 90-99mmHg) and severe or Stage 2 (Systolic 160mmHg and above, Diastolic 100mmHg and above). Globally, it was reported in the year 2010 that the prevalence of hypertension in the world was estimated to be 31% of adults (1.39 billion) [2]. This has increased tremendously and is projected to reach about 1.56 billion in 2025 [2]. In Africa, the cumulative estimated prevalence of hypertension is 30.8%. [3] In Nigeria, hypertension prevalence has been increasing over the past several decades, with recent estimates ranging from 20% to 40%, and varies by region. [4]

Notable biomarkers of hypertension that are of diagnostic value in predicting and diagnosing hypertension have been identified; these include plasma renin, aldosterone, albumin/creatinine ratio, C-reactive protein, brain natriuretic peptide, fibrinogen, homocysteine, and plasminogen activator inhibitor-1.[5] Microalbuminuria has been identified in diabetic hypertensives. [6]

Hypertension affects all socio-economic and income strata as a result of its many complications which include myocardial infarction, heart failure, chronic kidney disease, stroke, peripheral artery disease (PAD), metabolic syndrome, retinopathy and sexual dysfunction. Progression of hypertension and development of associated complications reflect structural and functional cardiovascular abnormalities and resultant damage to organs, leading to premature morbidity and mortality. [7, 8]

Usually, hypertension occurs without any identifiable cause (primary or essential hypertension). Still, it may also be secondary to an underlying condition such as congenital abnormalities involving blood vessels, sleep apnea, medications and drugs (steroids, birth control pills, cocaine and amphetamines), chronic kidney disease, thyroid gland disease, and adrenal gland tumours. The risk factors include age (> 60 years), race (common among blacks and people of African heritage), genetics, overweight or obesity, tobacco (smoking or chewing), excessive sodium salt consumption, excessive alcohol consumption, and stress (mental, economic, job, domestic). Hypertension and other factors such as tobacco smoking, diabetes mellitus, obesity, physical inactivity, dyslipidemia, and hypercholesterolemia are classified as modifiable cardiovascular risk factors while age, sex, and race are non-modifiable risk factors. [7, 9]

Inflammation protects tissues by involving inflammatory cells that phagocytose infecting agents and assist in tissue repair through proinflammatory mediators such as cytokines and prostaglandins. [10, 11] Excessive increased intravascular inflammatory processes and oxidative stress are of aetiological importance and therapeutic target in prognosticating hypertension, though the link between the two

processes has not been clarified. Factors such as pathogens, trauma, surgery, burns, cancer, damaged cells and toxic compounds induce inflammatory responses in the affected organs, leading to tissue damage or disease. Such diseases produce some proinflammatory markers of diagnostic and therapeutic importance in managing inflammatory diseases. The most commonly used inflammatory markers in clinical practice are C-reactive protein (CRP), erythrocyte sedimentation rate, plasma viscosity, and procalcitonin. Other promising biochemical parameters could be diagnostic values in chronic disease conditions that are yet to be used as markers. Hence, this study determined adiposity indices, serum apolipoproteins, and antioxidant status across varying levels of severity in hypertension.

Methods

Study location

This study was carried out at the General Out-patient Department (GOPD) of Olabisi Onabanjo University Teaching Hospital (OOUTH), Sagamu between 19 June and 21 July 2023.

Study design and sample size

The research was a cross-sectional and case-control study involving participants enlisted via a systematic random sampling method who attended the GOPD of OOUTH within four weeks of the study. The sample size was determined using the Yamane formula based on the hospital record of the average GOPD monthly attendance of adults (age range 18-65 years) over the past six months. [12] One hundred and fifty patients were purposefully recruited, comprising one hundred (100) newly diagnosed hypertensive patients (test group) and fifty (50) non-hypertensive subjects (control group). The test group were newly diagnosed hypertensive patients attending or referred to OOUTH from nearby villages/towns who had not commenced antihypertensive medications. The control group comprised individuals who came to the hospital with patients and some normotensive hospital workers. Participants' socio-demographic characteristics such as age, sex, and social habits (cigarette smoking and alcohol intake) were extracted using structured study proforma.

Inclusion and exclusion criteria

Included in the study were consenting newly diagnosed patients with hypertension who were yet to be commenced on antihypertensive drugs (to avoid the likely influence of these drugs on the studied parameters) and non-hypertensive control subjects, male and female genders, aged 18 to 65 years. Excluded from the study were hypertensive patients on medication, active cigarette smokers, chronic alcoholic users, obese individuals, individuals on steroids, those with diabetes mellitus and chronic kidney diseases, and other cardiovascular diseases.

Biophysical measurements

Anthropometric data (height, weight, hip circumference (HC), and waist circumference (WC) were measured using standard procedures. Body Mass Index (BMI), waist-to-hip ratio (WHR), and percentage body fat (%BF) were computed for tests and controls. BMI = weight /height^2, WHR = WC/HC; and %BF = [(1.2 x BMI) + (0.23 x age) – 16.2]. The tests and controls were categorised based on BMI as healthy (18.5–24.9 kg/m^2), overweight (25.0-29.9 kg/m^2), and obese (≥ 30.0 kg/m^2) respectively. Blood pressure was measured using a mercurial sphygmomanometer while participants were seated with their arms rested. Two recordings were made at 30 minutes intervals, and the average value was recorded. The systolic and diastolic blood pressure was determined using the first and fifth phases of Korotkof sounds, respectively. Hypertension was defined as systolic blood pressure (SBP) and diastolic blood pressure (DBP) greater than 120 mmHg and 80 mmHg respectively. The hypertensive patients

were further categorised as mild (SBP 120-139; DBP 80-89), moderate (SBP 140-159; DBP 90-99), and severe (SBP ≥ 160; DBP ≥ 100) based on established criteria. [13] A WHR value of ≥ 0.90 in males and ≥ 0.85 in females showed heightened susceptibility to diseases. [14]

Sample collection and storage

Ten millilitres (10 mL) of fasting venous blood (8-10 hours overnight fast) was aseptically drawn from each participant through the antecubital vein into plain bottles. The blood samples were allowed to be clotted and centrifuged at 5000 rpm for five minutes to obtain the serum, which was separated into plain sterile bottles. Two millilitres (2 mL) of serum sample were used to determine high sensitivity C-Reactive Protein (hs-CRP) immediately after separation while the remaining sample was stored at -20°C until assayed.

Biochemical analysis

Fasting serum triglycerides, serum total cholesterol, high-density lipoprotein (HDL), Apo-A, Apo-B, vitamin A, C, E, catalase, glutathione, superoxide dismutase (SOD), hs-CRP, interleukine-6 (IL-6), and tumour necrosis factor-α (TNF-α) were determined using standard methods. [15-26] The low-density lipoprotein (LDL), atherogenic index (AI: LDL/HDL), coronary risk index (CRI: total cholesterol/HDL), and Apo-B/Apo-A ratio were calculated. [15, 27, 28]

Statistical analysis

The Statistical Package for Social Sciences (SPSS) version 25 was used for data analysis. Descriptive statistics were used to describe and represent variables, and the differences in mean between the groups were assessed using one-way analysis of variance (ANOVA). *P* values less than 0.05 defined statistical significance.

Ethical considerations

Ethical approval was obtained from the Health Research Ethics Committee of Olabisi Onabanjo University Teaching Hospital before the study was commenced (OOUTH/HREC/586/2023AP). Written informed consent to participate in the study was obtained from each subject using an informed consent form.

Results

The hypertension group comprised 53 females and 47 males (Figure 1). Table I shows a significant relationship between central adiposity, as measured by waist circumference, waist-to-hip ratio, and hypertension.

From Table II, the direct relationship between dyslipidaemia and hypertension was indicated in this study: mean levels of serum Total Cholesterol, Triglycerides, Low-Density Lipoprotein and very Low-Density Lipoprotein were significantly higher in hypertensive patients compared with the control. The mean high-density lipoprotein level was significantly lower in hypertensive patients than in the control group. The Atherogenic Index was also significantly higher in hypertensive patients compared with the control. Table III shows there was a significantly lower mean serum glutathione and SOD levels but non-significant lower mean levels of serum catalase, vitamins A, C, and E levels among hypertensive patients compared with the control group. Furthermore, Table III shows significantly higher mean proinflammatory cytokine levels among hypertensive patients than the control group. The mean C-reactive protein levels were significantly higher in the hypertensive group than in the control group.

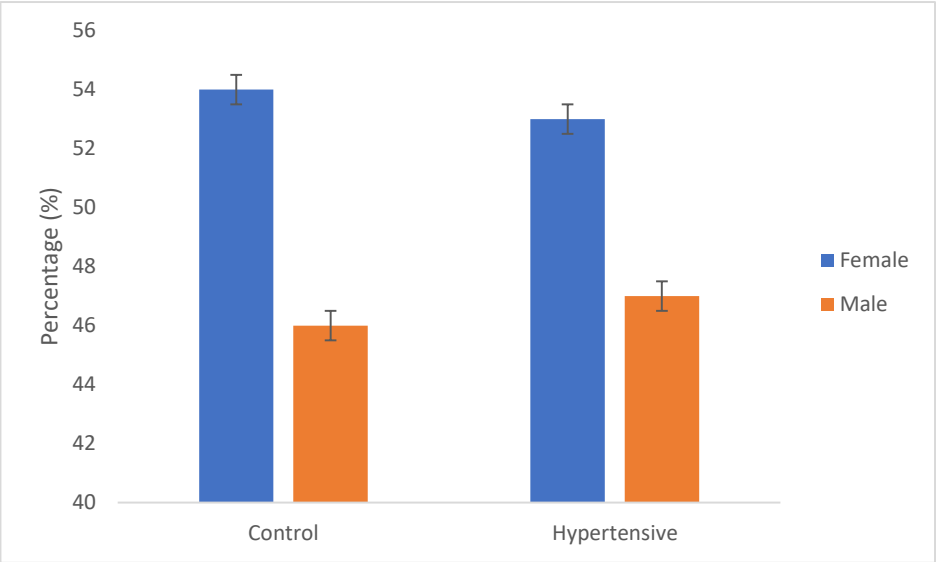

Figure I: Gender distribution among hypertensive patients and the controls

Table I: Mean anthropometric parameters of the hypertensive group and the control group

Parameters	*Controls*	*Hypertension Group*			*F*	*p-value*
	n = 50	**Mild, n = 11**	**Moderate, n = 62**	**Severe, n = 27**		
Age (years)	55.44±8.69	54.73±8.04	59.37±7.97	49.96±10.37	3.666	0.014
Weight (kg)	71.08±12.25	73.54±1.80	76.29±0.78	78.97±1.03	12.191	<0.001
Height (m)	1.68±0.06	1.56±0.03	1.56±0.05	1.57±0.05	63.116	<0.001
BMI (kg/m^2)	23.79±2.89	28.90±1.49	30.94±1.44	31.01±2.16	100.23	<0.001
WC (cm)	32.66±1.51	36.36±1.96	36.84±1.64	38.00±1.54	89.167	<0.001
HC (cm)	37.92±2.16	39.91±0.83	41.18±1.37	41.93±1.52	47.52	<0.001
WHR	0.86±0.06	0.91±0.06	0.90±0.05	0.91±0.04	5.613	0.001
% Body Fat (%)	25.10±3.84	31.06±3.18	33.50±2.34	34.89±2.65	91.864	<0.001
Systolic (mmHg)	112.24±7.35	135.00±0.00	143.21±2.02	163.22±2.39	823.365	<0.001
Diastolic (mmHg)	76.00±5.35	91.64±1.57	94.44±3.04	97.96±4.10	250.989	<0.001

BMI – Body Mass Index, WC – Waist Circumference, HC – Hip circumference, WHR – Waist-Hip Ratio.

In Table IV, Pearson correlation showed a strong, positive and significant correlation between blood pressure, anthropometric parameters, serum lipid profile (Total Cholesterol and Triglycerides) and inflammatory markers. On the other hand, a strong, negative but significant correlation existed between blood pressure and antioxidant activities (GSH, SOD) among hypertensive patients when compared with the control group.

Table II: Mean serum fasting lipids levels and lipoproteins levels among the hypertensive group and the control group

Parameters	*Controls*	*Hypertension Group*			*F*	*p-value*
	n = 50	*Mild, n = 11*	*Moderate, n = 62*	*Severe, n = 27*		
Cholesterol (mg/dL)	157.80±24.93	180.18±31.23	194.10±43.26	200.56±48.55	10.763	<0.001
Triglycerides(mg/dL)	90.84±31.37	105.64±31.06	110.50±28.71	119.59±33.96	6.217	0.001
HDL (mg/dL)	58.62±13.90	56.91±7.66	50.24±7.10	48.44±8.37	9.02	<0.001
LDL (mg/dL)	81.02±27.23	102.15±37.12	121.76±45.59	128.19±49.34	12.06	<0.001
VLDL (mg/dL)	18.17±6.27	21.13±6.21	22.10±5.74	23.92±6.79	6.217	0.001
AI	1.53±0.78	1.88±0.91	2.50±1.07	2.76±1.28	12.188	<0.001
CRI	2.86±0.88	3.26±0.93	3.95±1.10	4.27±1.32	14.117	<0.001
Apo-A (g/L)	2.13±0.47	0.90±0.08	0.40±0.14	0.33±0.07	383.876	<0.001
Apo-B (g/L)	0.83±0.52	0.91±0.10	1.97±0.38	2.12±0.21	101.902	<0.001
Apo-B/Apo-A	0.41±0.27	1.01±0.14	5.70±2.67	6.89±1.91	104.111	<0.001

AI - Atherogenic Index, CRI - Coronary Risk Index, Apo-A - Apolipoprotein A, Apo-B - Apolipoprotein B.

Table III: Mean serum antioxidant and proinflammatory markers among the hypertensive group and the control group

Parameters	**Control**	**Hypertensive patients**			**F**	**p-value**
	n = 50	**Mild, n = 11**	**Moderate, n = 62**	**Severe, n = 27**		
Vitamin A (mcg/mL)	4.28±1.38	4.00±1.71	3.61±1.06	3.78±1.38	5.019	0.003
Vitamin C (ng/mL)	49.32±16.98	47.00±6.09	43.86±15.54	42.37±13.86	1.681	0.174
Vitamin E (mg/mL)	6.15±2.59	5.85±1.90	5.72±2.02	5.70±1.86	0.424	0.736
Glutathione (IU/gHb)	0.205±0.19	0.017±0.02	0.004±0.001	0.003±0.001	34.322	<0.001
SOD (IU/gHb)	3.29±0.72	3.05±0.24	2.94±0.37	2.81±0.32	6.841	<0.001
Catalase (U/mgHg)	2.13±0.11	0.23±0.23	0.15±0.14	0.10±0.08	1.005	0.393
hsCRP (mg/L)	1.02±0.12	1.63±0.05	2.15±0.12	2.98±0.08	2060.754	<0.001
TNF-α (pg/mL)	12.43±0.61	15.46±0.62	16.06±0.56	16.14±0.47	454.025	<0.001
IL-6 (pg/mL)	2.50±0.09	3.51±0.36	3.74±0.27	4.80±0.35	518.335	<0.001

SOD – Superoxide dismutase; hsCRP – High sensitivity C-Reactive Protein; TNF-α - Tumour Necrosis Factor-Alpha; IL – interleukin.

Discussion

Generally, the prevalence of hypertension increases with age, occurring more in males than females. Females experience a higher rise in blood pressure from the third decade of life, thus having a comparatively accelerated prevalence of hypertension with age and a higher risk of developing adverse cardiovascular outcomes, even at relatively lower blood pressure thresholds. [29] Premenopausal women, as they go through the transition into menopause, may exhibit a higher prevalence of hypertension. The findings in this study suggest a higher prevalence of hypertension among women (53%) compared to men (47%). However, this only reflects the presence of more females in the subjects studied. This finding corroborates the report of a previous

study in this respect. [29] This observation may reflect variations in blood levels of sex hormones and their influence on the renin-angiotensin-aldosterone system (RAAS). Other reasons why females may predominate in the hypertension group include sustained effects of preconceptual birth control pills, sustained preeclampsia-induced vascular damage, socioeconomic-induced psychologic effects, exhibition of high genetic predisposition among women, or high prevalence of comorbidities with vascular damaging effects among women.

Table IV: Correlations among anthropometric measures, blood pressure, antioxidants and inflammatory markers

	BMI	WC	SBP	DBP	TC	TG	Apo A	Apo B	GSH	SOD	TNF	IL-6
BMI	1											
WC	.619**	1										
SBP	.754**	.777**	1									
DBP	.720**	.747**	.878**	1								
TC	.392**	.323**	.417**	.410**	1							
Trig	.356**	.239**	.309**	.253**	0.03	1						
Apo A	-.771**	-.736**	-.838**	-.849**	-.424**	-.313**	1					
Apo B	.632**	.594**	.726**	.714**	.290**	.216**	-.722**	1				
GSH	-.542**	-.524**	-.632**	-.576**	-.319**	-.263**	.631**	-.498**	1			
SOD	-.405**	-.321**	-.322**	-.333**	-.218**	-.174*	.342**	-.261**	.192*	1		
TNF	.771**	.776**	.845**	.854**	.402**	.313**	-.893**	.707**	-.607**	-.342**	1	
IL-6	.727**	.733**	.927**	.831**	.354**	.346**	-.817**	.690**	-.536**	-.312**	.800**	1

BMI – Body Mass Index; WC – Waist Circumference; SBP – Systolic Blood Pressure; DBP – Diastolic Blood Pressure; TC – Total Cholesterol; Trig – Triglycerides; Apo A – Apoprotein A; Apo B – Apoproteins B; GSH – Glutathione; SOD – Superoxide dismutase; TNF – Tumour Necrosis Factor; IL - Interleukin

The present study also shows the relationship between adiposity and hypertension. While BMI identifies individuals with an increased risk of having adiposity-related adverse health outcomes, WC (HC and WHR) is often used as a surrogate of subcutaneous and visceral fast deposit. [30] In other words, WC (HC or WHR) measures central adiposity, which is directly linked to hypertension as a progressive increase in blood pressure and increases extracellular matrix and adipose tissue. [31] The findings in this study reveal a trend of increasing mean SBP and DBP with hypertension severity in the test group with the mean SBP values of 135.00±0.00mmHg, 143.21±2.02mmHg and 163.22±2.39mmHg for mild, moderate and severe hypertension. Similarly, mean DBP values were 91.64±1.57mmHg, 94.44±3.04mmHg, and 97.96±4.10mmHg for mild, moderate, and severe hypertension. A similar trend was observed concerning obesity in the hypertension group, with the mean BMI values progressively increasing with increasing severity of hypertension. The direct relationship between hypertension and obesity in this study is in agreement with a previous report. [31] The mechanism responsible for this observation is explained by changes in adipose-derived cytokines, which have been reported to be a causative factor in hypertension. [32]

On the pattern of lipid profile in the present study, the mean serum levels of total cholesterol, triglycerides, LDL, and VLDL increased with a decrease in serum HDL concentrations in a severity-dependent manner were observed among hypertensive patients when compared with the controls. The pattern of alterations in the

lipid profile is such that the mean serum levels progressively increased in relation to the severity of hypertension.

The lipoproteins also showed variations with significantly higher mean levels of apolipoprotein-B (Apo B) but significantly lower mean levels of apolipoprotein-A (Apo A) in the same severity-dependent manner. The dyslipidaemia observed in the hypertensives, with severity in direct relation to the severity of hypertension, is a result of abnormalities of lipoprotein metabolism. [33] The atherogenic index (AI) and coronary risk index (CRI) were observed to be higher among the hypertensive patients in a severity-dependent manner. The pattern of the atherogenic index, coronary risk index and lipoproteins observed in this study reflects the changes and extents of CVD complicating hypertension, according to the severity of hypertension, as these indices are markers of CVD. [34]

The determination of antioxidant levels is an indirect reflection of oxidative stress. Oxidative stress has been described as a unifying factor linking blood pressure regulatory systems involving the cardiovascular system, kidneys, brain, and immune cells to each other. [35] The findings from this study suggest significantly lower levels of serum glutathione and SOD but non-significant lower levels of serum catalase, vitamins A, C, and E levels in the hypertensive group, in a severity-dependent manner when compared to the controls.

The observed lower mean serum values of antioxidants (enzymic and non-enzymic) concentrations in the hypertension group reflect the involvement of oxidative stress in hypertension, worse in severe hypertension and least in mild hypertension. This finding corroborates the reported decrease in blood antioxidant enzyme activities (SOD, glutathione peroxidase, and catalase) among hypertensive patients in a similar study. [36] The observed lowering of serum antioxidant levels depicts the increased generation of free radicals and reflects the involvement of antioxidants in mopping up excess systemic free radicals. This is responsible for some degree of imbalances between pro-oxidants and antioxidants' activities in the body, suggesting oxidative stress as a pathogenic factor and, thus, antioxidants as therapeutic in hypertension. Serum levels of pro-oxidants are, therefore, highest among individuals with severe hypertension and lowest among patients with mild hypertension.

In response to inflammatory conditions, there is increased local or systemic production of pro-inflammatory cytokines (IL-1β, IL-6, TNF-α), which induce the production of CRP by the liver as an acute-phase reactant. High-sensitivity C-Reactive Protein could be a dependable biomarker of chronic inflammation and predictor of cardiovascular events among healthy subjects and during inflammatory conditions due to its long half-life, stable circulating levels, and minimal circadian variation. This study revealed a significantly higher serum hs-CRP among hypertensive patients in a severity-dependent manner when compared to the controls. Similar elevation of hs-CRP had earlier been reported in patients with cardiometabolic risk factors including metabolic syndrome, obesity, dyslipidaemia, diabetes mellitus, and hypertension. [37]

Interleukin-6 is a major proinflammatory cytokine, having a pivotal role in inflammatory processes involved in inflammatory disease. In the present study, a significantly higher mean serum IL-6 level was observed in a severity-dependent manner among hypertensive patients when compared to the controls. This corroborates an earlier report of increased IL-6 levels in hypertensive patients. [38] Several other studies have reported that inflammation contributes to the pathogenesis of hypertension. [39] Therefore,

the finding observed in this study suggests that inflammation plays an essential role in hypertension.

Strong, positive correlations were observed between blood pressure (SBP, DBP) and anthropometric indices (BMI, WC), serum lipid profile (total cholesterol, triglycerides), Apo-B and proinflammatory markers (TNF-α, IL-6). Also, a strong negative correlation exists between blood pressure and serum levels of antioxidants (GSH, SOD) and Apo-A.

Conclusion

Based on the findings in this study, hypertension may be associated with adiposity and dyslipidaemia, reduced anti-oxidants and increased proinflammatory activities. The biochemical parameters are useful for diagnostic, prognostic, and therapeutic monitoring. Patterns of dyslipidaemia, reduced antioxidant concentrations and high concentrations of proinflammatory entities vary with the severity of hypertension. These biomarkers are useful in the clinical management of hypertension.

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