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Haematological Profiles and Acute Phase Reactants among Individuals with Hypertension in Port Harcourt, Nigeria

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

Background: Hypertension or high blood pressure affects numerous individuals worldwide and is associated with cardiovascular issues. Recent studies have examined the connection between haematological parameters and hypertension.
Aim: This study was aimed at assessing the haematological profiles and acute phase reactants of hypertensive individuals in Port Harcourt, Nigeria.
Methods: A case control study was conducted among 160 hypertensives and 100 age-matched normotensives in Port Harcourt. Ten milliliters (10mls) of venous blood was collected aseptically by venipuncture technique from the subjects into vacutainer tubes. Three milliliters (3mls) was dispensed into EDTA tubes for full blood count using haematological autoanalyser, Sysmex Kx-21N and ESR using Westergren method while 4mls was dispensed into sodium citrate tubes for the determination of fibrinogen levels and 3mls into plain tubes; this was spun and the separated serum

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was used for the determination of CRP and albumin levels. The serum CRP and plasma fibrinogen were determined by Sandwich ELISA method while serum albumin was determined using Bromocresol green (BCG) binding method.
Results: The hypertensives had significantly higher WBC (p≤0.001), LYM (p=0.044), NEU (p=0.009) and RDW-SD (p=0.004) compared to normotensives. The hypertensives had statistically higher SBP (mm/Hg) (p≤0.001), DBP (mm/Hg) (p≤0.001) and BMI (kg/m^2) (p<0.001) compared to the normotensives. The hypertensives also had statistically higher CRP (mg/L) (p≤0.001) and fibrinogen (mg/dL) (p=<0.001) compared to the normotensives. However, the ALB (g/dl) (p≤0.001) was statistically lower among hypertensives compared to the normotensives.
Conclusion: This study has shown that some haematological parameters may possibly indicate heightened immune system activity and chronic inflammation associated with hypertension.

Keywords: Haematological profiles; acute phase reactants; hypertension; Port Harcourt.

1. INTRODUCTION

Hypertension, often termed as high blood pressure is a medical condition marked by increased levels of blood pressure [1]. About 90% of cases falls into the category of essential hypertension where the exact underlying cause remains unknown [1]. A minority of individuals with hypertension experience secondary hypertension where a specific factor is identified as the primary cause of elevated blood pressure. Various factors can result in secondary hypertension including conditions like primary aldosteronism, renovascular disease and obstructive sleep apnea [2]. The link between hypertension and inflammation is firmly established, yet it remains unclear whether inflammation serves as the primary cause of hypertension or is a result of the condition.

Present treatments for hypertension in humans encompass inhibitors of angiotensin II (Ang II) type 1 receptor (AT1R), diuretics, angiotensin converting enzyme (ACE), beta-blockers and calcium channel blockers [3]. The use of commonly prescribed antihypertensive medications minimizes the likelihood of major cardiovascular occurrences and it is noteworthy that a greater decrease in blood pressure corresponds to a more substantial decrease in cardiovascular risk. Nevertheless, despite the effectiveness of the aforementioned therapies in lowering the pressure of blood for majority of patients, there exists a subset of individuals who do not respond well to these treatments. Additionally, even when blood pressure targets are met, a considerable number of hypertensive patients remain susceptible to cardiovascular events, possibly attributable to underlying inflammation [1].

Inflammation is a natural defense mechanism in response to infection or injury. It is an intricate procedure involving several stages, including the identification of damaged tissue by inflammatory cells, recruitment of leukocytes to the affected area, removal of the harmful agent, and subsequent tissue repair. This process relies on interactions between proinflammatory mediators, the extracellular matrix and cell surfaces [4]. Excessive inflammation can have adverse consequences and function in the advancement and evolution of persistent or long-lasting health conditions like rheumatoid arthritis, systemic lupus erythematosus and atherosclerosis [5].

The acute phase protein known as C-reactive protein (CRP) plays a crucial role in innate immune reactions with functions that encompass triggering the complement system and boosting phagocytosis [6]. CRP can also trigger monocytes to discharge proinflammatory cytokines like tumor necrosis factor alpha (TNF-α), interleukin-1 beta (IL-1) and interleukin-6 (IL-6) [7]. Additionally, it prompts endothelial cells to manifest vascular cell adhesion molecule (VCAM-1) and intracellular adhesion molecule (ICAM-1), actions that additionally facilitate an inflammatory response [8]. CRP is recognized as an indicator of inflammation in conjunction with the most robust connection to hypertension.

Consistent findings from clinical trials have demonstrated that individuals with hypertension often exhibit increased concentrations of CRP in the bloodstream [6]. Prehypertensive individuals typically have higher plasma CRP levels in contrast to individuals with regular blood pressure and [9] reported that higher baseline CRP levels were linked to an elevated risk of developing overt hypertension. This observation aligns using the idea low-level inflammation

throughout the body might precede the onset of high blood pressure.

Thrombosis represents a complex and serious complication for individuals with hypertension, potentially leading to damage in various organs. The stability of circulatory homeostasis relies on an equilibrum between vasodilatory and vasoconstrictive forces governing the pressure of blood as well as the balance between fibrinolytic and procoagulant factors influencing blood rheology [9]. Arterial disease serves as the primary underlying factor behind most clinically significant cardiovascular events often attributed to the development of a blood clot at the location of an atherosclerotic plaque. Fibrinogen, acting as both a coagulation factor and an acute-phase reactant has been identified as a significant standalone risk factor for coronary artery problems [10]. Prior investigations have linked levels of fibrinogen and various coagulation factors with hypertension and other conditions. Elevated fibrinogen levels have been seen as a significant risk factor for cardiovascular disease that is not dependent on other factors as well as assessing fibrinogen levels might offer benefits in preventing hypertension-related complications [11-13].

Serum albumin which is a negative acute phase reactant is found in the blood plasma helps in the regulation of osmotic pressure between the tissues and blood vessels [14]. Decrease in serum albumin levels indicates inflammation, liver disease and malnutrition which has been linked with elevated cardiovascular disease mortality and morbidity [15]. Meanwhile, higher serum albumin levels are associated with cardiovascular risk factors which include cholesterol levels and blood pressure[16].
Erythrocyte Sedimentation Rate (ESR) is a vital biomarker employed in the detection of systemic inflammation. The ESR is not a disease-specific marker rather it is utilized alongside other diagnostic tests to ascertain the presence of heightened inflammatory activity. However, hypertension can contribute to other health issues that may cause inflammation leading to an elevated ESR [17].

The Platelet-to-lymphocyte ratio (PLR) and the Neutrophil-to-Lymphocyte ratio (NLR) are predictors of cardiovascular mortality and hypertension linked with different malignancies and poor clinical outcomes in cardiac diseases [18]. The PLR and NLR are easy and dependable biomarkers of an inflammatory response which can be obtained from a full blood count [18].

Body mass index (BMI) is strongly connected with both systolic blood pressure (SBP) and diastolic blood pressure (DBP). The accumulation of excess adipose tissue sets off a sequence of events that result to elevated blood pressure. Adipose tissue is unique in its susceptibility to lipolysis and its capacity to produce high levels of inflammatory cytokines which contribute to increased blood pressure and damage to organs as a result of the inflammatory response. Moreover, it could be that excess adipose tissue influences a range of factors linked to vascular tone and the inhibition of the growth of smooth muscle in blood vessels [19].

The present study was aimed at evaluating the haematological profiles and acute phase reactants among individuals with hypertension in Port Harcourt.

2. MATERIALS AND METHODS

2.1 Study Design

A case-control study was employed to assess the haematological parameters of hypertensive individuals in Port Harcourt, Nigeria.

2.2 Study Area

This study was carried out in Port Harcourt, Rivers State, Nigeria. Port Harcourt is the capital of Rivers state which has about 23 local government areas and lies along the Bonny River, in the Niger Delta region of Nigeria located between Latitude 4º53'N and Latitude 4º23'N, and Longitude 6º54'E and Longitude 6º18'E [20].

2.3 Study Population

A total of 160 hypertensives comprising of 94 females and 66 males attending the Rivers State University Teaching Hospital (RSUTH) and University of Port Harcourt Teaching Hospital (UPTH) as well as 100 apparently healthy individuals comprising of 73 females and 27 males between the ages of 30-89 years were recruited for this study.

2.4 Inclusion Criteria

Hypertensives who has been attending the hypertension clinic at the same tertiary health

care facility for a minimum of 2 years, female participants who were not pregnant and were not using hormone therapy or hormonal contraception.

2.5 Exclusion Criteria

Individuals currently suffering from a previous history of diabetes, participants with hormonal diseases like hypothyroidism and hyperthyroidism, orthopaedic and surgical conditions, stroke or haematologic conditions that could affect the investigated parameters, below the age of 30 and those who were above the age of 89 in order to narrow down the age group under investigation and ensure consistency in the study population. Also, participants not residing in Port Harcourt.

2.6 Sample Size

Sample size was determined using G-power 3.1.9.2 at power of 0.95. This gave a sample size of 76. However, this study used sample size of 160 hypertensive subjects and 100 control subjects.

2.7 Sample Collection and Processing

Ten milliliters (10mls) of venous blood was collected aseptically by venipuncture technique from the subjects into vacutainer tubes. Four milliliters (4mls) was dispensed into EDTA tubes for the analysis of ESR while three millilitres (3mls) was dispensed into sodium citrate tubes for the determination of fibrinogen levels and three millilitres (3mls) into plain tubes; this was spun and the serum separated was used for the determination of CRP and albumin levels.

2.8 Estimation of Full Blood Count

Estimation of full blood count was analysed using Sysmex Kx-21N Haematology Analyzer as described by Cheesbrough [21].

Principle: This device conducts hematology analyses through several methods, including hydro-dynamically focused impedance measurement, flow cytometry employing a semiconductor laser, and the sodium lauryl sulphate hemoglobin (SLS-HGB) method. Specifically, it counts and sizes red blood cells (RBC) and platelets (PLT) using hydrodynamic impedance counting. Simultaneously, it calculates the hematocrit (HCT) through the RBC pulse height detection method. The device also employs cytometry to examine the physiological and chemical characteristics of cells and other biological particles, with flow cytometry being the method used to analyze these entities as they pass through extremely small flow cells.

Procedure for using Sysmex Kx-21N Haematology Analyzer: The samples in EDTA bottles were numbered appropriately and placed in a mixer. The mixer was plugged to an electric socket, which allows the blood to properly mix together. The Sysmex equipment was then cleaned and quality control checked. Each sample number was inputted into the equipment, followed by opening of the cap of each sample to be run. The tube of the equipment's probe was set and 'Start Switch' put on. Each of the samples was held firmly beneath the probe which was inserted into the sample until it aspirated the sample, which was indicated by a 'beep' sound. After this, the sample was removed from the probe, and with within 60 seconds, the result was obtained in a printed format.

2.9 Estimation of Erythrocyte Sedimentation Rate (ESR)

Estimation of erythrocyte sedimentation rate (ESR) using Westergren method as described by Cheesbrough [21]

Principle: This is based on the fact that blood is essentially a suspension of formed elements such as red blood cells and white blood cell capsules in plasma and as such red blood cells will settle out of suspension in blood plasma measured under standard conditions.

Procedure for using Westergren Method: 400µL of trisodium citrate solution was pipetted into a westergren bucket, 1600µL of mixed blood was also transferred into the same bucket and then mixed gently. By the application of a clean westergren tube, the blood is drawn to the zero mark. It was then allowed to stand vertically in the westergren stand for 1 hour.

2.10 Estimation of CRP

Estimation of CRP using Latex particle enhanced immunoturbidimetric assay as described by Dupuy et al. [22]

Principle: Serum C-reactive protein (CRP) causes agglutination of the latex particles coated with anti- human C-reactive protein. The agglutination of the latex particles is proportional

to the CRP concentration and can be measured by turbidometry.

Procedure for using ELISA for C-reactive protein estimation: Reagent volumes were prepared by mixing 1 mL of Reagent 2 and 4 mL of Reagent 1. Reagent 2 vial was gently mixed before pipetting. The working reagent after preparation of was brought to room temperature. Two test tubes were labelled “Test” and “Standard”. 7µL of the sample was transferred into the test tube labelled “Test”, 7µL of the CRP standard was transferred into the test tube labelled “Standard”. 1mL of the working reagent was transferred into both tubes and mixed properly and allowed to stand for 10 minutes. After 10 minutes the absorbance was measured at 540nm.

2.11 Estimation of Plasma Fibrinogen

Estimation of plasma fibrinogen using Latex Particle-enhanced Immunoturbidometric ELISA as described by Pletsch-Borba et al. [23].

Principle: Plasma fibrinogen induces the clustering of latex particles that have been covered with anti-human fibrinogen. The degree of particle is proportional to the concentration of fibrinogen and can be quantified using turbidometry.

Procedure for using ELISA for plasma Fibrinogen estimation: Reagent volumes were prepared by mixing 1 mL of Reagent 2 with 4 mL of Reagent 1. After preparation of the working reagent, it was brought to room temperature. Two test tubes were labeled “Test” and “Standard”. About 7 µL of the sample was transferred into the test tube labeled “Test”, about 7 µL of the fibrinogen standard was transferred into the test tube labeled “Standard”. Then, about 1 mL of the working reagent was transferred into both test tubes; they were mixed properly and allowed to stand for 10 minutes, after which the absorbance was measured at 540nm.

2.12 Estimation of Serum Albumin

Estimation of serum albumin Bromocresol Green by Garcia et al. [24] was employed.

Principle: Bromocresol Green functions as an indicator that appears yellow within the pH range of 3.5 to 4.3. When exposed to acidic conditions, serum selectively associates with bromocresol green, causing a transition in the indicator's color from yellow-green to blue-green. The intensity of this color change is directly related to the concentration of albumin in the sample. The absorbance is read at 640nm.

Procedure: The following was pipetted into the appropriately labeled test tubes

2.13 Determination of Height and Weight as described by Ezuizo et al. [25] was employed

Participants’ height and weight were measured using a standard scale (seca model). Height was measured in meter (m) and weight in kilogram (kg).

2.14 Determination of Body Mass Index (BMI) as described by WHO [26] was employed

Individuals in the study were sorted into various weight categories using the Body Mass Index (BMI). BMI is determined by dividing a person's weight in kilograms by the square of their height in meters. Based on their BMI values, study participants were then classified into categories such as underweight, normal weight, overweight and obese.

2.15 Determination of Blood Pressure as described by Williams et al. [27] was employed

Blood pressure measurements were taken manually using a standard mercury sphygmomanometer with a cuff size suitable for each participant whereby individuals were provided with a 30-minute rest period in a comfortable chair before taking the measurements. While seated with their left arm resting on a table at heart level, both the diastolic blood pressure (DBP) and systolic blood pressure (SBP) from the left upper arm were recorded. Multiple readings were acquired until two consecutive measurements closely matched. SBP was determined based on the initial phase of the Korotkoff sound, while DBP was determined from the fifth phase. To categorize hypertension, the ESC/ESH document criteria were used with Grade 1 hypertension defined as 140-159 mmHg for SBP and 90-99 mmHg for DBP, Grade 2 as 160-179 mmHg for SBP and 100-109 mmHg for DBP and Grade 3 as SBP ≥180 mmHg and DBP ≥110 mmHg [35].

List 1. Study protocol

	Test	Standard	Blank	QC
BCG Reagent	1mL	1mL	1mL	1mL
Plasma	10µL	-	-	-
Standard	-	10µL	-	-
QC	-	-	-	10µL

***Calculation**:*

Albumin conc. in g/L = Reading of test x conc. of std. in g/L. / Reading of std.

2.16 Data Analysis

Data derived from this study were analyzed using SPSS version 23, a statistical software package. The findings were then displayed in tables. Results were expressed as mean ±SD, and statistical significance was established when the p-value was less than 0.05 with a confidence level of 95%.

3. RESULTS

3.1 Demographic Characteristics of Hypertensives and Controls

A total of 260 samples consisting of 167 females and 93 males were recruited for this study. 94 females and 66 males were hypertensives while 73 females and 27 males were apparently healthy controls. The mean values of age(years), height(m) and weight(kg) for the hypertensives were 57±10, 1.63±0.44, 81.70±4.10 for the controls were 51±5, 1.64±0.90, 69.88±5.35 respectively as shown in table 1.

3.2 Comparison of Haematological Parameters of Hypertensives and Controls

The comparison of the mean and standard deviation values for hypertensives were WBC ($\times10^9$/L) (8.10±5.27), LYM ($\times10^9$/L) (2.61±1.50), NEUT ($\times10^9$/L) (4.63±0.32), RBC ($\times10^{12}$/L) (4.24±1.69), HGB (g/dl) (12.43±8.19), HCT (%) (34.41±7.31), MCV (fL) (81.48±9.46), MCH (pg) (28.18±5.67), MCHC (g/dl) (34.04±1.97), RDW-SD (fL) (46.07±10.53), RDW-CV (%) (14.73±5.82), PLT (x 10^9/L) (231.21±105.25), MPV (fL) (9.87±1.18), PDW (fL) (13.34±2.62), plateletcrit (%) (0.23±0.09) and ESR (mm/hr) (47.84±3.09) for controls were WBC ($\times10^9$/L) (6.13±2.52), LYM ($\times10^9$/L) (2.28±0.82), NEUT ($\times10^9$/L) (3.45±2.96), RBC ($\times10^{12}$/L) (4.24±1.01), HGB (g/dl) (12.69±7.82), HCT (%) (33.87±5.80), MCV (fL) (81.50±6.70), MCH (pg) (30.87±2.74), MCHC (g/dl) (35.03±5.45), RDW-SD (fL) (42.57±7.52), RDW-CV (%) (15.00±6.24), PLT (x 10^9/L) (239.30±119.02), MPV (fL) (10.15±1.15), PDW (fL) (13.97±2.71), plateletcrit (%) (0.22±0.15) and ESR (mm/hr) (25.27±1.88) respectively as shown in Table 2.

The hypertensives had statistically higher WBC ($\times10^3$/µL) (p≤0.001), LYM ($\times10^9$/L) (p=0.044), NEU ($\times10^9$/µL) (p=0.009), RDW-SD (fL) (p=0.004) and ESR (mm/hr) (p≤0.001) compared to the controls. However, the RBC (x 10^{12}/L) (p=0.976), HGB (g/dl) (p=0.796), HCT (%) (p=0.538), MCV (fL) (p=0.983), MCH (p=0.227), MCHC (p=0.084), RDW-CV (p=0.072), PLT ($\times10^9$/L) (0.568), MPV (fL) (p=0.067), PDW (fL) (p=0.066) and plateletcrit (%) (p=0.121) were not statistically significant when compared.

3.3 Comparison of Acute Phase Reactants of Hypertensives and Controls

The comparison of the mean and standard deviation values for hypertensives were ALB (g/dl) (41.86±10.00), CRP(mg/L) (4.54±3.35) and fibrinogen (mg/dL) (294.36±88.10) for controls were ALB (g/dl) (36.04±13.39), CRP (mg/L) (10.99±7.25) and fibrinogen (mg/dL) (393.47±107.41) respectively as shown in Table 3.

The hypertensives had statistically higher CRP (mg/L) (p≤0.001) and fibrinogen (mg/dL) (p=<0.001) compared to the control subjects. However, the ALB (g/dl) (p≤0.001) was statistically lower among hypertensives compared to the control subjects.

3.4 Haematological Parameters According to Hypertension Grades

The comparison of the mean and standard deviation according to hypertension grades for pre-hypertension were WBC ($\times10^9$/L) (9.65±7.94), LYM ($\times10^9$/L) (2.59±1.52), NEUT ($\times10^9$/L) (5.82±0.88), RBC (x 10^{12}/L) (4.03±1.25), HGB (g/dl) (11.50±3.57), HCT (%) (34.05±8.93), MCV (fL) (81.16±11.66), MCH (pg) (29.15±8.81), MCHC (g/dl) (34.02±1.77), RDW-SD (fL) (45.44±9.72), RDW-CV (%) (14.73±4.21), PLT (x 10^9/L) (229.37±103.27), MPV (fL) (9.92±1.34), PDW (fL) (13.32±2.82), plateletcrit (%) (0.22±0.09), PLR (110.59±75.72[a]), NLR (2.98±0.47) and ESR (mm/hr) (57.48±6.61) for grade 1 were WBC ($\times10^9$/L) (7.56±3.66), LYM ($\times10^9$/L) (2.78±1.65), NEUT ($\times10^9$/L) (3.96±2.67),

Table 1. Demographic characteristics of hypertensives and controls

	Hypertensive	Control
Female	94	73
Male	66	27
Age (years)	57±10	51±5
Height (m)	1.63±0.44	1.64±0.90
Weight (kg)	81.70±4.10	69.88±5.35
SBP	147.80±15.52	119.53±6.13
DBP	100.05±7.98	80.20±6.13
BMI	30.05±5.20	25.58±4.45

Table 2. Comparison of blood pressures and body mass index (BMI) of hypertensives and controls

	Hypertensive Mean±SD (n = 160)	Control Mean±SD (n = 100)	P –value	t – value	Remark
SBP (mm/Hg)	147.80±15.52	119.53±6.13	<0.001	17.317	S
DBP (mm/Hg)	100.05±7.98	80.20±6.13	<0.001	21.180	S
BMI (kg/m^2)	30.05±5.20	25.58±4.45	<0.001	7.034	S

Abbreviations: SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; BMI: Body Mass Index

Table 3. Comparison of haematological parameters of hypertensives and controls

	HYP (n = 160) Mean±SD	Control (n = 100) Mean±SD	P –value	t – value	Remark
WBC (x10^3/µL)	8.10±5.27	6.13±2.52	<0.001	4.040	S
LYM (x10^3/µL)	2.61±1.50	2.28±0.82	0.044	2.026	S
NEUT (x10^3/µL)	4.63±0.32	3.45±2.96	0.009	2.629	S
RBC (x10^6/µL)	4.24±1.69	4.24±1.01	0.976	0.031	NS
HGB (g/dl)	12.43±8.19	12.69±7.82	0.796	0.259	NS
HCT (%)	34.41±7.31	33.87±5.80	0.538	0.617	NS
MCV (fL)	81.48±9.46	81.50±6.70	0.983	0.021	NS
MCH (fL)	28.18±5.67	30.87±2.74	0.227	1.212	NS
MCHC (g/dl)	34.04±1.97	35.03±5.45	0.084	1.74	NS
RDW-SD (fL)	46.07±10.53	42.57±7.52	0.004	2.887	S
RDW-CV (%)	14.73±5.82	15.00±6.24	0.720	0.358	NS
PLT (x10^3/µL)	231.21±105.25	239.30±119.02	0.568	0.572	NS
MPV (fL)	9.87±1.18	10.15±1.15	0.067	1.838	NS
PDW (fL)	13.34±2.62	13.97±2.71	0.066	1.847	NS
PLATELETCRIT(%)	0.23±0.09	0.22±0.15	0.121	12.233	NS
ESR (mm/hr)	47.84±3.09	25.27±1.88	<0.001	5.350	S

Abbreviations: WBC: White Blood Cell; LYM: Lymphocytes; NEU: Neutrophils, RBC: Red Blood Cell; HGB: Hemoglobin; HCT: Hematocrit; MCV: Mean Corpuscle Volume, MCH: Mean Corpuscle Hemoglobin; MCHC: Mean Corpuscle Hemoglobin Concentration, RDW-SD: Red Cell Distribution Width -Standard Deviation, RDW-CV: Red Cell Distribution Width – Coefficient of Variation; PLT: Platelet count, MPV: Mean Platelet Volume; PDW: Platelet Distribution Width; ESR: Erythrocyte Sedimentation Rate.

RBC (x 10^{12}/L) (4.03±1.24), HGB (g/dl) (11.99±4.34), HCT (%) (34.28±7.04), MCV (fL) (81.75±8.73), MCH (pg) (27.88±3.70), MCHC (g/dl) (33.94±1.81), RDW-SD (fL) (46.07±11.31), RDW-CV (%) (14.92±6.59), PLT (x 10^9/L) (222.08±96.52), MPV (fL) (9.95±1.16), PDW (fL) (13.43±2.56), plateletcrit (%) (0.53±0.30), PLR (105.42±82.94^b), NLR (1.99±0.32) and ESR (mm/hr) (46.78±3.86) for grade 2 were WBC (×10^9/L) (7.49±3.67), LYM (×10^9/L) (2.28±0.75), NEUT (×10^9/L) (4.46±3.36), RBC (x 10^{12}/L) (4.39±0.90), HGB (g/dl) (12.12±2.19), HCT (%) (35.59±6.06), MCV (fL) (81.99±8.05), MCH (pg) (28.02±4.27), MCHC (g/dl) (37.56±4.38), RDW-SD (fL) (49.12±11.62), RDW-CV (%) (14.73±6.51), PLT (x 10^9/L) (239.24±115.65), MPV (fL) (9.58±0.83), PDW (fL) (13.28±2.41), plateletcrit (%) (0.25±0.10), PLR (111.03±62.45^a), NLR (2.22±1.77) and ESR (mm/hr) (32.29±8.96) for grade 3 were WBC (×10^9/L) (6.90±2.74), LYM (×10^9/L) (1.92±0.80), NEUT (×10^9/L) (5.10±3.62), RBC (x 10^{12}/L) (4.40±0.41), HGB (g/dl) (11.73±1.51), HCT (%) (35.03±3.49), MCV (fL) (80.00±7.58), MCH (pg) (26.78±2.97), MCHC (g/dl) (33.43±2.43), RDW-SD (fL) (44.18±4.65), RDW-CV (%) (13.40±4.59), PLT (x 10^9/L) (292.33±42.05), MPV (fL) (9.55±1.15), PDW (fL) (12.82±2.88), plateletcrit (%) (0.26±0.11), PLR (200.72±58.19^c), NLR (3.51±1.04) and ESR (mm/hr) (40.58±11.18) respectively as shown in table 4.

The PLR (p=0.012) statistically increased from pre-hypertension to grade 3 hypertension.

Table 4. Comparison of acute phase reactants of hypertensives and controls

	Hypertensive Mean±SD (n = 160)	Control Mean±SD (n = 100)	P –value	t – value	Remark
ALB (g/dl)	36.04±13.39	41.86±10.00	<0.001	3.728	S
CRP (mg/L)	10.99±7.25	4.54±3.35	<0.001	9.730	S
FIBRINOGEN (mg/dL)	393.47±107.41	294.36±88.10	<0.001	7.720	S

Abbreviations: ALB: Albumin; CRP: C-Reactive Protein

Table 5. Haematological parameters of hypertensives according to hypertension grades

	Pre-hypertension (n=46) Mean±SD	Grade1 (n=85) Mean±SD	Grade 2 (n=17) Mean±SD	Grade 3 (n=12) Mean±SD	P - value	F– value	Remark
WBC (x 109/L)	9.65±7.94	7.56±3.66	7.49±3.67	6.90±2.74	0.127	1.930	NS
LYM (x 109/L)	2.59±1.52	2.78±1.65	2.28±0.75	1.92±0.80	0.212	1.578	NS
NEU (x 109/L)	5.82±0.88	3.96±2.67	4.46±3.36	5.10±3.62	0.087	2.225	NS
RBC (x 1012/L)	4.03±1.25	4.03±1.24	4.39±0.90	4.40±0.41	0.547	0.707	NS
HGB (g/dl)	11.50±3.57	11.99±4.34	12.12±2.19	11.73±1.51	0.895	0.202	NS
HCT (%)	34.05±8.93	34.28±7.04	35.59±6.06	35.03±3.49	0.882	0.220	NS
MCV (fL)	81.16±11.66	81.75±8.73	81.99±8.05	80.00±7.58	0.927	0.153	NS
MCH (pg)	29.15±8.81	27.88±3.70	28.02±4.27	26.78±2.97	0.507	0.779	NS
MCHC (g/dl)	34.02±1.77	33.94±1.81	37.56±4.38	33.43±2.43	0.131	0.654	NS
RDW-SD (fL)	45.44±9.72	46.07±11.31	49.12±11.62	44.18±4.65	0.581	0.654	NS
RDW-CV (%)	14.73±4.21	14.92±6.59	14.73±6.51	13.40±4.59	0.871	0.237	NS
PLT (x 109/L)	229.37±103.27	222.08±96.52	239.24±115.65	292.33±42.05	0.187	1.621	NS
MPV (fL)	9.92±1.34	9.95±1.16	9.58±0.83	9.55±1.15	0.505	0.783	NS
PDW (fL)	13.32±2.82	13.43±2.56	13.28±2.41	12.82±2.88	0.898	0.198	NS
PLATELETCRIT (%)	0.22±0.09	0.53±0.30	0.25±0.10	0.26±0.11	0.170	1.674	NS
PLR	110.59±75.72[a]	105.42±82.94[b]	111.03±62.45[a]	200.72±58.19[c]	0.012	3.762	S
NLR	2.98±0.47	1.99±0.32	2.22±1.77	3.51±1.04	0.170	0.266	NS
ESR (mm/hr)	57.48±6.61	46.78±3.86	32.29±8.96	40.58±11.18	0.116	2.003	NS

Abbreviations: WBC: White Blood Cell; LYM: Lymphocytes; NEU: Neutrophils, RBC: Red Blood Cell; HGB: Hemoglobin; HCT: Hematocrit; MCV: Mean Corpuscle Volume, MCH: Mean Corpuscle Hemoglobin; MCHC: Mean Corpuscle Hemoglobin Concentration, RDW-SD: Red Cell Distribution Width-Standard Deviation, RDW-CV: Red Cell Distribution Width – Coefficient of Variation; PLT: Platelet count, MPV: Mean Platelet Volume; PDW: Platelet Distribution Width; Abbreviations: NLR: Neutrophil/Lymphocyte Ratio; PLR: Platelet to Lymphocyte Ratio; ESR: Erythrocyte Sedimentation Rate;S: Significant; NS: Not Significant. Values with different superscripts are significantly different from each other (p<0.05).

Classification of hypertension (mmHg) was based on the ESC/ESH document, as follows: Grade 1: 140-159/90-99, Grade 2: 160-179/100-109, Grade 3: Grade 3, ≥180/110 (Williams et al.*, 2018) [27].*

Table 6. Comparison of acute phase reactants of hypertensives according to hypertension grades

	Pre-hypertension (n=46) Mean±SD	Grade1 (n=85) Mean±SD	Grade 2 (n=17) Mean±SD	Grade 3 (n=12) Mean±SD	P - value	F– value	Remark
ALB (g/dl)	39.00±3.17	34.27±8.08	35.24±5.61	38.58±9.70	0.237	1.428	NS
CRP (mg/L)	13.18±9.53	11.99±8.08	10.38±7.96	7.16±4.44	0.141	1.847	NS
Fibrinogen (mg/dL)	391.41±105.52	396.78±101.94	379.47±117.39	397.50±149.65	0.941	0.133	NS

Classification of hypertension (mmHg) was based on the ESC/ESH document, as follows:Grade 1: 140-159/90-99, Grade 2: 160-179/100-109, Grade 3: Grade 3, ≥180/110 (Williams et al.*, 2018)[27]. Abbreviations:ALB: Albumin; CRP: C-Reactive Protein.*

However, the WBC (×109/L) (p=0.127), LYM (×109/L) (p=0.212), NEUT (×109/L) (p=0.087), RBC (×1012/L) (p=0.549), HGB (g/dl) (p=0.895), HCT (%) (p=0.882), MCV (fL) (p=0.927), MCH (pg) (p=0.507), MCHC (g/dl) (p=0.131), RDW-SD (p=0.581), RDW-CV (p=0.871), PLT (×109/L) (p=0.189), MPV (p=0.505), PDW (fL) (p=0.898), plateletcrit (%) (p=0.170), NLR (p=0.170) and ESR (mm/hr) (p=0.116) were not statistically significant when compared respectively.

3.5 Comparison of Acute Phase Reactants of Hypertensives According to Hypertension Grades

The comparison of the mean and standard deviation for according to hypertension grades for pre-hypertension were ALB (g/dl) (39.00±3.17), CRP (mg/L) (13.18±9.53) and fibrinogen (mg/dL) (391.41±105.52) for grade 1 were ALB (g/dl) (34.27±8.08), CRP (mg/L)

(11.99±8.08) and fibrinogen (mg/dL) (396.78±101.94) for grade 2 were ALB (g/dl) (35.24±5.61), CRP (mg/L) (10.38±7.96) and fibrinogen (mg/dL) (379.47±117.39) for grade 3 were ALB (g/dl) (38.58±9.70), CRP (mg/L) (7.16±4.44) and fibrinogen (mg/dL) (397.50±149.65) respectively as shown in table 5. The ALB (g/dl) (p=0.237), CRP (mg/L) (p=0.141) and fibrinogen (mg/dL) (p=0.941) were not statistically significant when compared respectively.

4. DISCUSSION

Hypertension or high blood pressure affects numerous individuals universally and this study evaluated the haematological profiles among individuals with hypertension in Port Harcourt. Findings from this study revealed significantly increased systolic blood pressure (p≤0.001) among hypertensives as compared to control subjects. This may be due to increased peripheral resistance caused by narrowed and stiffened arteries which makes it difficult for the heart to pump blood effectively during systole, an increased cardiac output leading to a greater volume of blood being forced into the arteries during each contraction and endothelial dysfunction which can impair vasodilation. This finding is consistent with the findings of another author [9] which stated that hypertensives had significantly higher systolic blood pressure than normotensives.

This study revealed significantly higher diastolic blood pressure (p≤0.001) among hypertensives as compared to control subjects. This may result from increased resistance to blood flow in peripheral arteries making it difficult for arteries to relax and expand during diastole, arterial stiffness, endothelial dysfunction as well as increased blood volume which could require the arteries to accommodate more blood during diastole. This finding is in line with the report of another author [9].

Findings from this study revealed significantly increased body mass index (p=<0.001) among hypertensives compared to control subjects which could be caused by chronic inflammation and obesity. This reflects an increase in body fat which has been identified as an independent risk factor for hypertension. However, the exact mechanisms connecting visceral fat and hypertension are not fully understood. Inflammatory processes could play a significant role in the development of hypertension. Fat cells are sensitive to fat breakdown and can produce inflammatory cytokines in substantial quantities contributing to elevated blood pressure. Findings from this study is in consonance with the study of other authors [28,9].

Findings from this study revealed elevated total white blood cell count (p≤0.001) among hypertensives when compared to control subjects. This may be due to the fact that hypertension is frequently associated with persistent, mild inflammation in the body that disrupts the endothelial function. This is similar to the findings of other authors [29-32].

Significantly higher absolute lymphocyte levels (p=0.044) in hypertensives compared to control subjects as revealed in this study. This could be triggered by inflammation and this inflammatory response can affect the immune cell counts, leading to a rise in the production and circulation of lymphocytes, which are integral to immune defense mechanisms. This is in line with the study of another author [33]. This finding is not in consonance with the study reported by another author [25] which stated that there was no significant difference in the absolute lymphocyte count among hypertensive subjects compared to non-hypertensive controls. This could be attributed to the smaller sample size and age range of the subjects used.

Significantly increased absolute neutrophil count (p=0.009) among hypertensives compared to control subjects was observed in this study. This may be attributed to the fact that neutrophils could contribute to the immune response during sterile inflammation, which occurs in the absence of infection. This finding is consistent with the study of another author [25].

Significantly higher random distribution width standard deviation (RDW-SD) levels (p=0.004) in hypertensives compared to control subjects as revealed in this study may be due to inflammation which could contribute to heightened immune system activity, possibly resulting in greater variability in measurement. This finding is not consistent with the study of another author [34] who reported that there was no significant difference in RDW-SD of hypertensives compared to normotensives. This could be attributed to the geographical location carried out in Southern Nigeria and study design used.

Findings in this study revealed significantly increased levels of erythrocyte sedimentation

rate (ESR) (p≤0.001) among hypertensives compared to normotensives which may be caused by chronic inflammation as a result of a heightened inflammatory response which could play a significant role in the pathophysiology leading to the onset of hypertension. This is not in agreement with the findings of another author [35] who reported that there was no significant difference in erythrocyte sedimentation rate between the hypertensive and normotensive subjects. This is attributed to the study population and sample size used in this study.

Significantly reduced levels of serum albumin (p≤0.001) among hypertensives in comparison with normotensives was observed in this study which could be attributed to the fact that albumin has anti-inflammatory characteristics and this can trigger the body's reaction to inflammation and vascular damage. This finding is similar to the findings of another author [36].

The significantly elevated levels of c-reactive protein (CRP) (p≤0.001) in hypertensives compared to control subjects was observed in this study as a result of persistent inflammatory state and obesity which involves the release of pro-inflammatory cytokines from adipose tissue (fat cells). This finding is consistent with the report of another author [6].

Significantly elevated plasma fibrinogen levels (p≤0.001) in hypertensive subjects compared to normotensive subjects was observed in this study. This phenomenon is likely due to various factors related to the complex pathophysiology of hypertension caused by chronic inflammation, which can stimulate the liver to produce more fibrinogen. This finding in this study is supported the findings of other authors [37-39, 12].

The platelet-to-lymphocyte ratio (PLR) significantly increased (p=0.012) from pre-hypertension to grade 3 hypertension in this study. Grade 3 hypertension is associated with not only heightened blood pressure but also underlying inflammation, stress responses in the body and suggests an increased likelihood of cardiovascular events such as strokes, heart attacks and peripheral artery disease. The finding in this study is similar with the findings of other authors [40,41] Yildiz et al (2015); Projahn and Koenen, 2012.

5. CONCLUSION

Findings from this study revealed that higher body mass index and some haematological parameters indicate heightened immune system activity and chronic inflammation associated with hypertension. Acute phase reactants like fibrinogen, CRP and albumin, which indicate inflammation are often elevated or decreased in hypertensives, providing insights into their inflammatory burden and vascular health. Additionally, erythrocyte sedimentation rate, a non-specific measure of inflammation, can be used to assess the inflammatory state of hypertensive patients. By evaluating these markers, health-care professionals can better manage hypertension and improve patient outcomes.

6. RECOMMENDATION

Sequel to this study, it can be recommended that

1. Individuals should go for regular medical checkup.
2. The body mass index, systolic and diastolic blood pressure should be monitored regularly.

ETHICAL APPROVAL

Ethical approval for this study was obtained from the Research Ethics Committee of the Ministry of Health, State Secretariat Complex with a clearance from Rivers State Hospital Management Board, Port-Harcourt, Rivers State, Nigeria.

CONSENT

As per international standards or university standards, respondents' written consent has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. David MP, Justin PVB, Annet K. The role of inflammation in hypertension: novel concepts. Current Opinion in Physiology. 2021;19:92-98.
2. Funder JW, Carey RM, Mantero F, Murad HM, Reincke, MS, H., Stowasser, M, Young FM. The management of primary aldosteronism: Case detection, diagnosis, and treatment: an Endocrine Society clinical practice guideline. Journal of

Clinical Endocrinology and Metabolism. 2016;101:1889–1916.
3. Lu X, Crowley SD. Inflammation in salt-sensitive hypertension and renal damage. Current Hypertension Reports. 2018;20(12):103.
4. Zenglei Z, Lin Z, Xingyu Z, Xu M, Xianliang Z. Role of inflammation, immunity, and oxidative stress in hypertension: New insights and potential therapeutic targets. Frontal Immunology. 2022;13:1098725.
5. Asanuma YF. Accelerated atherosclerosis and inflammation in systemic lupus erythematosus. Japanese Journal of Clinical Immunology. 2012;35(6):470–480.
6. Hage FG. C-reactive protein and hypertension. Journal of Human Hypertension. 2014;28(7):410-415.
7. Sproston NR, Ashworth JJ. Role of c-reactive protein at sites of inflammation and infection. Frontiers in Immunology. 2018;9,754.
8. Xiao L, Harrison DG. Inflammation in hypertension. Canadian Journal of Cardiology. 2020;36:635–647.
9. Yeldu MH, Yale BM, Akuyam SA, Isezuo SA. Inflammatory biomarkers and their correlates among hypertensive population in two tertiary hospitals in Sokoto, Nigeria. International Journal of Biology and Chemical Sciences. 2018;12(3):1131-1140.
10. Davalos D, Akassoglou K. Fibrinogen as a key regulator of inflammation in disease. Seminars in Immunopathology. 2012; 34:43-62.
11. Ibrahim HM, Abdalla MH. D-Dimer Levels in Sudanese Hypertensive Patients. American Journal of Medicine and Medical Sciences. 2014;4:92-95.
12. Osman SSA, Ali AE, Muddathir ARM. Measurements of Plasma Fibrinogen Level, Total Cholesterol and Triglyceride Levels in Hypertensive Sudanese Patients. American Journal of Medicine and Medical Sciences. 2014;4(5): 161-165.
13. Ali NMA, Gameel FEMH, Elsayid M, Babker AMAAA. Alterations in D-Dimer, Prothrombin time and activated partial thromboplastin time as thrombogenesis activity markers in patients with acute myocardial infarction. Open Journal of Blood Diseases. 2016;6(1):1-5.
14. Arroyo V. Human serum albumin: not just a plasma volume expander. Hepatology. 2009;50:355–357.
15. Ishida S, Hashimoto I, Seike T, Abe Y, Nakaya Y, Nakanishi H. Serum albumin levels correlate with inflammation rather than nutrition supply in burns patients: a retrospective study. The Journal of Medical of Medical Investigation. 2014;61(3-4):361-368.
16. Arques S. Serum albumin and cardiovascular disease: State-of-the-art review. Annales de Cardiologie et D'angeiologie. 2020;69(4):192–200.
17. Vanessa A, Manuela A, Nuria V, Carmen T, Daniel R, Carmen G, Bernardo S, Francisco G, Arturo G. Factors influencing erythrocyte sedimentation rate in adults. Medicine. 2019;98(34):16816.
18. Man MA, Rajnoveanu R, Motoc NS, Bondor CI, Chis AF, Lesan A, Lucaciu S, Dantes E, Gergely-Domokos B, Fira-Mladinescu O. Neutrophil-to-lymphocyte ratio, platelets-to-lymphocyte ratio, and eosinophils correlation with high-resolution computer tomography severity score in COVID-19 patients. Plos One. 2021; 16(6):e0252599.
19. Stelmach-Mardas M, Rodacki T, Dobrowolska-Iwanek J, Brzozowska A, Walkowiak J, Wojtanowska-Krosniak A, Zagrodzki P, Bechthold A, Mardas M, Boeing H. Link between Food Energy Density and Body Weight Changes in Obese Adults. Nutrients. 2016; 8(4):229.
20. Baeka GB, Oloke JK, Opaleye OO. Detection of hepatitis C virus among HIV patients in Port Harcourt, Rivers State. African Health Sciences. 2021;21(3):1010-1015.
21. Cheesbrough M. District Laboratory Practice in Tropical Countries. 2nd ed. Cambridge: Cambridge University Press; 2010.
22. Dupuy AM, Badiou S, Descomps B, Cristol JP. Immunoturbidimetric determination of C-reactive protein (CRP) and high-sensitivity CRP on heparin plasma. Comparison with serum determination. Clin Chem Lab Med. 2003;41(7):948-9.
23. Pletsch-Borba L, Watzinger C, Turzanski FR, Katzke V, Schwingshackl L, Sowah SA, Hüsing A, Johnson T, Grob ML, González MS, Hoffmeister M, Bugert P, Kaaks R, Grafetstätter M, Kühn T. Biomarkers of vascular injury and type 2 diabetes: a prospective study, systematic review and meta-analysis. Journal of Clinical Medicine. 2019;8(12):2075.

24. Garcia MV, Beridze VN, Martinez GMD, Laborda GB, Garcia AS, Fernandez RE. Overestimation of albumin measured by bromocresol green vs bromocresol purple method: influence of acute-phase globulins. Laboratory Medicine. 2018;49(4): 355–361.
25. Eziuzo CI, Chinko BC, Dapper VC. Comparative assessment of some white blood cell and platelet parameters among normotensive and hypertensive subjects in Port Harcourt, Nigeria. Nigerian Medical Journal. 2017;58(4):131-137.
26. World Health Organization. A Healthy Lifestyle - WHO Recommendations; 2010. Available:https://www.who.int/europe/news-room/fact-sheets/item/a-healthy-lifestyle---who-recommendations
27. Williams B, Mancia G, Spiering W, Enrico Agabiti Rosei A E, Michel A, Burnier M, Clement D, Coca A, Simone DG, Dominiczak A, Kahan T, Mahfoud F, Redon J, Ruilope L, Zanchetti A, Kerins M, Kjeldsen S, Kreutz R, Laurent S, Lip HYG, McManus R, Narkiewicz K, Ruschitzka F, Schmieder R, Shlyakhto E, Tsioufis K, Aboyans V, Desormais I. Practice Guidelines for the management of arterial hypertension of the European Society of Cardiology and the European Society of Hypertension. Blood Pressure. 2018;27(6):314–340.
28. Chen Y, Liang X, Zheng S, Wang Y, Lu W. Association of Body Fat Mass and Fat Distribution with the Incidence of Hypertension in a Population-Based Chinese Cohort: A 22-Year Follow-Up. Journal of the American Heart Association. 2018;7:e007153.
29. Fantin F, Giani A, Zoico E, Rossi AP, Mazzali G, Zamboni M. Weight Loss and Hypertension in Obese Subjects. Nutrients. 2019;11:1667.
30. Enawgaw BAN, Terefe B, Asrie F, Melku M. A comparative cross-sectional study of some hematological parameters of hypertensive and normotensive individuals at the University of Gondar hospital, Northwest Ethiopia. BMC Hematology. 2017;17(1):21.
31. Imtiaz A, Nadera, Y. A comparative study of hematological parameters in normal and hypertensive individuals. International Journal of Physiology.2017;5:1.
32. Furuncuoğlu Y, Tulgar S, Dogan AN, Calar S, Tulgar YK, Cakiroglu B. How obesity affects the neutrophil/lymphocyte and platelet/lymphocyte ratio. Systemic immune-inflammatory index and platelet indices: a retrospective study. European Review for Medical and Pharmacological Sciences. 2016;20:1300–1306.
33. Nunez J, Sastre C, D'Ascoli G. Relation of low lymphocyte count to frailty and its usefulness as a prognostic biomarker in patients >65 years of age with acute coronary syndrome. American Journal of Cardiology. 2020;125:1033-1038.
34. Sileshi B, Urgessa F, Wordofa MA. A comparative study of hematological parameters between hypertensive and normotensive individuals in Harar, Eastern Ethiopia. Plos One. 2021;16(12): e0260751.
35. Ighoroje DA, Dapper DV. Haemorrheological parameters in hypertensive Nigerians compared to normotensive controls. Journal of Medicine and Biomedical Research. 2002;1:1.
36. Haile B, Wolde M, Gebregziabiher T. Assessment of fasting blood glucose, serum electrolyte, albumin, creatinine, urea and lipid profile among hypertensive patients and non-hypertensive participants at Wolaita Sodo University Teaching and Referral Hospital, SNNPR, Ethiopia. Journal of Clinical Chemistry and Laboratory Medicine. 2023; 6:261.
37. Pasquale C, Szilvia M, Suvra N, Yu-Hsuan C, Subash CM, Jose CMS, Sachin SD, Lauriane P, Barbara Z, Christian S. Fibrinogen regulates lesion border-forming reactive astrocyte properties after vascular damage. Glia. 2022;70(7):1251-1266.
38. Ahmed AAE, Tarig OK, Hassan MN, Eltayib SMS, Mohieldin E, Asaad MAAB. Fibrinogen levels in hypertensive and normotensive: A cross-sectional study from El-Obied City, Sudan. Journal of Biosciences and Medicines. 2016;4:2.
39. Eldour AAA, Khalafallah TO, Noja HM, Saad ESM, Elsayid M, Babker AMAAA. Fibrinogen levels in hypertensive and normotensive: a cross-sectional study from El-Obied City, Sudan. Journal of Biosciences and Medicines. 2016;04 (02):28–32.
40. Yildiz A, Yuksel M, Oylumlu M, Polat N, Akyuz A, Acet H, Aydin M, Ülgen MS. The Utility of the Platelet-Lymphocyte Ratio for Predicting No Reflow in Patients With ST-Segment

Elevation Myocardial Infarction. Clinical and Applied Thrombosis/ Hemostasis. 2015;21(3):223-228.

41. Projahn D, Koenen RR. Platelets: Key players in vascular inflammation. Journal of Leukoc Biology. 2012;92(6):1167-75.

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