Gender-Based Assessment of Haematological Parameters and Acute Phase Reactants of Hypertensives in Port Harcourt, Nigeria

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Abstract

High blood pressure, also called as hypertension is the most prevalent cardiovascular risk factor and a significant contributor to global mortality and morbidity. Hypertension is a multifaceted condition, accounting for around 90% of cases falling into the category of essential hypertension where the exact underlying cause remains unknown. This study was aimed at assessing the haematological parameters and acute phase reactants of hypertensives based on gender in Port Harcourt, Nigeria. A case-control study involving 160 hypertensive individuals and 100 agematched normotensive controls was carried out in Port Harcourt. Aseptic venipuncture technique was employed to collect 10 milliliters (10mls) of venous blood from the participants, which was then distributed into various vacutainer tubes. Three milliliters (3mls) were allocated to EDTA tubes for a full blood count using the Sysmex Kx-21N haematological autoanalyzer and ESR measurement using the Westergren method. Additionally, 4mls were dispensed into sodium citrate tubes for determining fibrinogen levels and 3mls were placed in plain tubes. The latter underwent centrifugation and the resulting separated serum was utilized for assessing CRP and albumin levels. For the analysis of serum CRP and plasma fibrinogen, the Sandwich ELISA method was employed while serum albumin levels were determined using the Bromocresol green (BCG) binding method. The female hypertensives had statistically higher SBP (mm/Hg) (p=<0.001), DBP (mm/Hg) (p=<0.001), BMI (kg/m2) (p=<0.001), WBC (p=<0.001), LYM (p=0.006), NEU (p=0.008), ESR (mm/hr) (p=<0.001), CRP (mg/L) (p=<0.001) and fibringen (mg/dL) (p=<0.001)compared to the female control subjects. However, the ALB (g/dl) (p=<0.005) was statistically lower among female hypertensive compared to the female control subjects. The male hypertensives had statistically higher SBP (mm/Hg) (p=<0.001), DBP (mm/Hg) (p=<0.001), ESR Citation: Chukwu PH, Christian SG, Eze EM, Ken-Ezihuo SU, Moore-Igwe BW, Okey-Omunakwe C, Obeagu EI. Gender-Based Assessment of Haematological Parameters and Acute Phase Reactants of Hypertensives in Port Harcourt, Nigeria. Elite Journal of Haematology, 2024; 2(6): 16-34

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(mm/hr) (p=0.001), CRP (mg/L) (p=<0.001) and fibrinogen (mg/dL) (p=0.001) compared to the male control subjects. However, the ALB (p=0.022) was statistically lower among male hypertensives compared to the male control subjects when compared respectively. The WBC (×109/L) (p=0.874), LYM (×109/L) (p=0.107), NEUT (×109/L) (p=0.491), RBC (×1012/L) (p=0.550), HGB (g/dl) (p=0.106), HCT (%) (p=0.265), MCV (fL) (p=0.392), MCH (pg) (p=0.815), MCHC (g/dl) (p=0.060), RDW-SD (fL) (p=0.707), RDW-CV (%) (p=0.594), PLT (×109/L) (p=0.236), MPV (fL) (p=0.666), PDW (fL) (p=0.441), plateletcrit (%) (p=0.222), PLR (p=0.875), NLR (p=0.750), ESR (mm/hr) (p=0.765), ALB (g/l) (p=0.601), CRP (mg/L) (p=0.452) and fibrinogen (mg/dL) (p=0.453) were not statistically significant by sex. This study has shown that sex has no effect on the haematological parameters and acute phase reactants of hypertensives in Port Harcourt.

Keywords: Sex, Haematological parameters, Hypertension, Port Harcourt.

1. Introduction

Adults with hypertension or those on blood pressure-lowering medications at the age of 30 have about a 40% higher risk of experiencing a cardiovascular disease event compared to their age and sex-matched counterparts with lower blood pressure [1]. Moreover, individuals with hypertension tend to experience cardiovascular disease events approximately five years earlier than those with lower blood pressure [1]. In the 40-69 age group, a 20 mmHg increase in systolic blood pressure or a 10 mmHg increase in diastolic blood pressure, regardless of baseline values, is associated with more than a twofold increase in the risk of stroke or ischemic heart disease mortality. Conversely, a reduction of 5 mmHg in systolic blood pressure can lead to a 14% decrease in stroke mortality and a 9% decrease in cardiovascular disease mortality [2]. Among individuals aged 80 years or older, the relative risk associated with blood pressure changes is slightly lower, but the absolute risk is significantly higher than at younger ages. For instance, a 20 mm Hg difference in systolic blood pressure between 120 and 140 mmHg is associated with an annual absolute risk difference nearly ten times larger in those aged 80-89 years compared to those aged 50-59 years [2]. Haematological parameters are useful in determining the physiological status of humans [3]. The commonly used haematological parameters are white blood cell count and differentials, haemoglobin concentration (Hb), packed cell volume (PCV), platelets and platelet indices, red blood cell count and red cell indices [3]. The cellular components of blood play crucial roles in maintaining blood pressure by influencing its viscosity, volume and coagulability [4]. When hypertension, or high blood pressure, is present, it can disrupt various hematological parameters in the body, leading to functional disturbances in multiple systems. Specifically, hypertension can cause an increase in the white blood cell (WBC) count, a decrease in red blood cell deformability, and an increase in platelet activation [5]. These changes in blood cells may have detrimental

effects, particularly in the microcirculation and can contribute to damage in various organs and tissues [6].

Inflammation significantly participates in the onset of high blood pressure and the damage it can cause to specific organs. Inflammation is linked to heightened blood vessel leakiness and the discharge of potent substances of powerful agents including substances like reactive oxygen species, cytokines, metalloproteinases and nitric oxide [7]. Acute phase reactants (APRs) play a crucial role in the inflammatory response and may contribute to the pathophysiology of hypertension.

This study was aimed at assessing the haematological parameters and acute phase reactants of hypertensives based on gender in Port Harcourt, Nigeria.

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

The research was conducted in Port Harcourt, which is the capital city of Rivers State in Nigeria. Port Harcourt is situated in the Niger Delta region of Nigeria and is located between Latitude 4°53'N and Latitude 4°23'N, and Longitude 6°54'E and Longitude 6°18'E. It is worth noting that Rivers State consists of approximately 23 local government areas and is situated along the Bonny River [8].

2.3 Study Population

A total of 260 individuals comprising 160 hypertensives on antihypertensive medications and 100 non-hypertensive controls between the ages of 30-89 years who gave informed and oral consents were recruited from Port-Harcourt and used for this study.

2.4 Inclusion Criteria

Hypertensive individuals who had been attending the hypertension clinic of the same tertiary healthcare facility for a minimum of 2 years.

2.5 Exclusion Criteria

Individuals currently suffering from diabetes, stroke or haematologic conditions that could affect the investigated parameters. Individuals 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. Female participants who were not pregnant and were not using hormone therapy or hormonal contraception.

2.6 Subject Selection

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

Aseptic venipuncture technique was used to collect venous blood samples from the subjects with a volume of 10 milliliters (10mls). The blood was collected into vacutainer tubes. For the analysis of erythrocyte sedimentation rate (ESR), 4 milliliters (4mls) of blood was dispensed into tubes containing EDTA. To determine fibrinogen levels, 3 milliliters (3mls) of blood was dispensed into tubes containing sodium citrate. Additionally, 3 milliliters (3mls) of blood was collected into plain tubes, which were then spun to separate the serum. The serum was used for the determination of C-reactive protein (CRP) and albumin levels.

2.8 Laboratory Analysis

The full blood count estimation was performed using the Sysmex Kx-21N Haematology Analyzer, following the methodology described by Cheesbrough [9]

2.8.1 Procedure for using Sysmex Kx-21N Haematology Analyzer

The EDTA bottles containing the samples were appropriately labeled with unique numbers and placed in a mixer. The mixer was connected to an electrical outlet, allowing the blood samples to mix thoroughly. The Sysmex equipment underwent cleaning and quality control checks. Each sample number was entered into the equipment, and the caps of the corresponding samples were opened. The equipment's probe tube was positioned, and the 'Start Switch' was activated. Each sample was securely held beneath the probe, and the probe was inserted into the sample until it aspirated the required amount, signaled by a 'beep' sound. Subsequently, the sample was removed from the probe, and within 60 seconds, the results were obtained in a printed format.

2.9 Method

The estimation of erythrocyte sedimentation rate (ESR) was performed using the Westergren method, as described by Cheesbrough [9].

2.9.1 Procedure for using the Westergren Method: To perform the ESR measurement using the Westergren method, $400\mu L$ of trisodium citrate solution was pipetted into a Westergren bucket. Additionally, $1600\mu L$ of mixed blood was transferred into the same bucket and gently mixed. Using a clean Westergren tube, the blood was drawn up to the zero mark on the tube. The tube was then vertically placed in a Westergren stand and allowed to stand for 1 hour.

2.10. Method:

The estimation of serum albumin was performed using the Bromocresol Green method described by Garcia et al. [10].

2.10.1 Procedure:

The appropriately labeled test tubes were pipetted with the necessary components for the serum albumin estimation.

2.11 Method:

The plasma fibrinogen estimation was performed using a latex particle-enhanced immunoturbidometric ELISA, as described by Pletsch-Borba et al. [11].

2.11.1 Procedure for plasma fibrinogen estimation using ELISA: Reagent volumes were prepared by mixing 1 mL of Reagent 2 with 4 mL of Reagent 1. After **Citation**: Chukwu PH, Christian SG, Eze EM, Ken-Ezihuo SU, Moore-Igwe BW, Okey-Omunakwe C, Obeagu EI. Gender-Based Assessment of Haematological Parameters and Acute Phase Reactants of Hypertensives in Port Harcourt, Nigeria. *Elite Journal of Haematology*, 2024; 2(6): 16-34

preparing the working reagent, it was brought to room temperature. Two test tubes were labeled as "Test" and "Standard." Approximately 7 μ L of the sample was transferred into the test tube labeled "Test," and approximately 7 μ L of the fibrinogen standard was transferred into the test tube labeled "Standard." Then, about 1 mL of the working reagent was added to both test tubes. The tubes were mixed thoroughly and allowed to stand for 10 minutes. After 10 minutes, the absorbance was measured at 540nm.

2.11 Method: Estimation of plasma fibrinogen using Latex Particle-enhanced Immunoturbidometric ELISA as described by Pletsch-Borba et al. [11].

2.11.2 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 μ L of the sample was transferred into the test tube labeled "Test", about 7 μ L 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 Method:**

The CRP (C-reactive protein) estimation was conducted using a latex particle-enhanced immunoturbidimetric assay, as described by Dupuy et al. [12].

2.12.1 Procedure for CRP estimation using ELISA: Reagent volumes were prepared by mixing 1 mL of Reagent 2 with 4 mL of Reagent 1. The Reagent 2 vial was gently mixed before pipetting. The working reagent, after preparation, was brought to room temperature. Two test tubes were labeled as "Test" and "Standard." 7μ L of the sample was transferred into the test tube labeled "Test," and 7μ L of the CRP standard was transferred into the test tube labeled "Standard." Then, 1 mL of the working reagent was added to both tubes, mixed thoroughly, and allowed to stand for 10 minutes. After 10 minutes, the absorbance was measured at 540nm.

2.13 Measurement of height and weight was conducted following the method described by Ezuizo et al. [13].

Participants' height was measured using a standard scale (seca model) and recorded in meters (m), while their weight was measured in kilograms (kg).

2.14 Measurement of Body Mass Index (BMI) was performed according to the guidelines provided by the World Health Organization (WHO) [14].

BMI is calculated by dividing an individual's weight in kilograms by the square of their height in meters. Based on their BMI values, participants were classified into different weight categories, including underweight, normal weight, overweight, and obese.

2.15 Measurement of blood pressure were taken following the method described by Williams et al. [15].

A standard mercury sphygmomanometer with an appropriate cuff size was used. Participants were given a 30-minute rest period in a comfortable chair before the measurements were taken. With **Citation**: Chukwu PH, Christian SG, Eze EM, Ken-Ezihuo SU, Moore-Igwe BW, Okey-Omunakwe C, Obeagu EI. Gender-Based Assessment of Haematological Parameters and Acute Phase Reactants of Hypertensives in Port Harcourt, Nigeria. *Elite Journal of Haematology*, 2024; 2(6): 16-34

their left arm resting on a table at heart level, both systolic blood pressure (SBP) and diastolic blood pressure (DBP) were recorded from the left upper arm. Multiple readings were obtained 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. Hypertension was categorized according to the criteria outlined in the ESC/ESH document, with different grades based on specific ranges of SBP and DBP values [15].

2.16 Data Analysis

Data analysis was performed using SPSS version 23, a statistical software package. The results were presented in tables, with the mean \pm standard deviation (SD) used to express the findings. Statistical significance was considered when the p-value was less than 0.05, with a confidence level of 95%.

3.0 RESULTS

3.1 Demographic Characteristics Female and Male 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 hypertensive while 73 females and 27 males were apparently healthy controls. The mean values of Age(years), Height(m) and Weight(kg) for the hypertensive were 57±10, 1.63±0.44, 81.70±4.10 for the control were 51±5, 1.64±0.90, 69.88±5.35 respectively as shown in Table 1.

3.2 Comparison of Blood Pressures and Body Mass Index (BMI) of Female Hypertensives and Female Control Subjects

The comparison of the mean and standard deviation values for female hypertensives were SBP (mm/Hg) (146.35 ± 15.46), DBP (mm/Hg) (100.36 ± 7.96) and BMI (kg/m2) (31.31 ± 7.94) for female control subjects were SBP (mm/Hg) (118.93 ± 6.14) DBP (mm/Hg) (79.96 ± 6.07) and BMI (kg/m2) (25.42 ± 7.17) respectively as shown in Table 2.

The female hypertensives had statistically higher SBP (mm/Hg) (p=<0.001), DBP (mm/Hg) (p=<0.001) and BMI (kg/m2) (p=<0.001) compared to the female control subjects.

3.3 Comparison of Haematological Parameters of Female Hypertensives and Female Control Subjects

The comparison of the mean and standard deviation values for female hypertensives were WBC ($\times10^9$ /L) (8.16±4.97), LYM (x 10⁹/L) (2.77±1.58), NEUT ($\times10^9$ /L) (4.81±0.47), RBC (x 10¹²/L) (4.19±1.22), HGB (g/dl) (11.44±2.70), HCT (%) (33.87±7.42), MCV (fL) (82.01±9.19), MCH (pg) (28.26±6.78), MCHC (g/dl) (33.80±2.02), RDW-SD (fL) (46.44±10.08), RDW-CV (%) (14.88±6.41), PLT (x 10⁹/L) (239.43±113.79), MPV (fL) (9.91±1.13), PDW (fL) (13.21±2.65), plateletcrit (%) (0.51±0.27), PLR (113.60±10.61), NLR (2.47±0.34) and ESR (mm/hr) (48.62±39.03) for female control subjects were WBC ($\times109$ /L) (5.92±2.55), LYM (x 109/L) (2.27±0.66), NEUT ($\times109$ /L) (3.32±0.28), RBC (x 1012/L) (4.33±1.09), HGB (g/dl) (12.15±3.15), HCT (%) (34.23±5.49), MCV (fL) (81.34±6.65), MCH (pg) (28.11±3.13), MCHC (g/dl) (35.20±6.31), RDW-SD (fL) (42.47±7.76), RDW-CV (%) (14.86±5.14), PLT (x 109/L) (226.24±87.71), MPV (fL) (10.17±1.17), PDW (fL) (13.99±2.91), plateletcrit (%) (1.99±0.86), Citation: Chukwu PH, Christian SG, Eze EM, Ken-Ezihuo SU, Moore-Igwe BW, Okey-Omunakwe C, Obeagu EI. Gender-Based Assessment of Haematological Parameters and Acute Phase Reactants of Hypertensives in Port Harcourt, Nigeria. *Elite Journal of Haematology*, 2024; 2(6): 16-34

PLR (107.31 \pm 5.94), NLR (1.68 \pm 0.24) and ESR (mm/hr) (25.36 \pm 18.26) respectively as shown in Table 2.

The female hypertensive had statistically higher WBC ($\times 10^3/\mu L$) (p=<0.001), LYM ($\times 10^3/\mu L$) (p=0.006), NEU ($\times 10^3/\mu L$) (p=0.008) and ESR (mm/hr) (p=<0.001) compared to the female control. However, the RBC (x 106/ μL) (p=0.437), HGB (g/dl) (p=0.120), HCT (%) (p=0.716), MCV (fL) (P=0.603), MCH (pg) (p=0.857), MCHC (p=0.072), RDW-SD (fL) (p=0.239), RDW-CV (p=0.985), PLT (0.399), MPV (fL) (p=0.147), PDW (fL) (p=0.071), plateletcrit (%) (p=0.105), PLR (0.606) and NLR (0.071) were not statistically significant when compared respectively.

3.4 Comparison of Acute Phase Reactants of Female Hypertensive and Female Control Subjects

The mean values for female hypertensives were ALB (g/dl) (36.51 ± 16.46), CRP(mg/L) (12.21 ± 8.64) and fibrinogen (mg/dL) (398.79 ± 108.45) for female control subjects were ALB (g/dl) (42.71 ± 9.95), CRP(mg/L) (5.13 ± 4.17) and fibrinogen (mg/dL) (292.04 ± 85.63) respectively as shown in Table 4.

The female hypertensives had statistically higher CRP (mg/L) (p=<0.001) and fibrinogen (mg/dL) (p=<0.001) compared to the female control subjects. However, the ALB (g/dl) (p=<0.005) was statistically lower among female hypertensive compared to the female control subjects.

3.5 Comparison of Blood Pressures and Body Mass Index (BMI) of Male Hypertensives and Male Control Subjects

The mean and standard deviation for male hypertensives were SBP (mm/Hg) (149.88 \pm 15.50), DBP (mm/Hg) (99.61 \pm 8.08) and BMI (kg/m²) (29.24 \pm 6.44) for male control subjects were SBP (mm/Hg) (121.15 \pm 5.47), DBP (mm/Hg) (80.85 \pm 6.37) and BMI (kg/m²) (27.38 \pm 4.53) in respectively as shown in Table 5. The male hypertensives had statistically higher SBP (mm/Hg) (p=<0.001) and DBP (mm/Hg) (p=<0.001) compared to the male control subjects. The BMI (kg/m²) (p=0.175) was not statistically significant when compared respectively.

3.6: Comparison of Haematological Parameters of Male Hypertensive and Male Control Subjects

The mean and standard deviation for male hypertensives were WBC ($\times 10^9$ /L) (8.03±5.72), LYM ($\times 10^9$ /L) (2.38±1.36), NEUT ($\times 10^9$ /L) (4.37±3.00), RBC ($\times 10^{12}$ /L) (4.30±1.10), HGB (g/dl) (12.42±4.90), HCT (%) (35.18±7.14), MCV (fL) (80.71±9.85), MCH (pg) (28.05±3.56), MCHC (g/dl) (34.38±1.86), RDW-SD (fL) (45.54±11.21), RDW-CV (%) (14.52±4.89), PLT ($\times 10^9$ /L) (219.38±91.12), MPV (fL) (9.82±1.25), PDW (fL) (13.53±2.59), plateletcrit (%) (0.22±0.01), PLR (116.02±10.17), NLR (2.32±0.26), ESR (mm/hr) (46.73±39.94) for male control subjects were WBC ($\times 10^9$ /L) (6.69±2.41), LYM ($\times 10^9$ /L) (2.31±1.15), NEUT ($\times 10^9$ /L) (3.82±2.02), RBC ($\times 10^{12}$ /L) (4.00±0.77), HGB (g/dl) (14.13±2.72), HCT (%) (32.90±6.83), MCV (fL) (81.91±6.92), MCH (pg) (38.20±10.00), MCHC (g/dl) (34.57±1.62), RDW-SD (fL) (42.83±6.98), RDW-CV (%) (15.39±8.61), PLT ($\times 10^9$ /L) (274.15±33.69), MPV (fL) (10.09±1.13), PDW (fL) (13.90±2.15), plateletcrit (%) (2.86±1.55), PLR (152.52±30.47), NLR (2.34±0.43), ESR (mm/hr) (25.04±20.35) respectively as shown in Table 6.

The male hypertensives had statistically higher ESR (mm/hr) (p=0.001) compared to the male control subjects. However, the WBC ($\times10^9$ /L) (p=0.245), LYM ($\times10^9$ /L) (p=0.805), NEU ($\times10^9$ /L) (p=0.391), RBC (x 10^{12} /L) (p=0.195), HGB (g/dl) (p=0.435), HCT (%) (p=0.161), MCV (fL) (p=0.563), MCH (p=0.318), MCHC (g/dl) (p=0.638), RDW-SD (fL) (p=0.246), RDW-CV (%) (p=0.539), PLT ($\times10^9$ /L) (p=0.133), MPV (fL) (p=0.338), PDW (fL) (p=0.516), plateletcrit (%) (p=0.101), PLR (p=0.149) and NLR (p=0.973) were not statistically significant when compared respectively.

3.7 Comparison of Acute Phase Reactants of Male Hypertensives and Male Control Subjects The mean and standard deviation for male hypertensives were ALB (g/dl) 35.38±6.97, CRP(mg/L) (11.20±8.04) and fibrinogen (mg/dL) (385.82±106.26) for male control subjects were 39.59±9.95, 4.18±3.83, 300.56±95.81 respectively as shown in Table 4.10. The male hypertensives had statistically higher CRP (mg/L) (p=<0.001) and fibrinogen (mg/dL) (p=0.001) compared to the male control subjects. However, the ALB (p=0.022) was statistically lower among male hypertensives compared to the male control subjects when compared respectively.

3.8 Comparison of Blood Pressures and Body Mass Index (BMI) between Female and Male Hypertensives

The comparison of the mean and standard deviation for female hypertensives were SBP (mm/Hg) (146.35±7.95), DBP (mm/Hg) (100.36±7.95) and BMI (kg/m2) (31.31±7.93) for male hypertensives were SBP (mm/Hg) (149.88±15.50), DBP (mm/Hg) (99.61±8.08) and BMI (kg/m2) (29.24±6.44) respectively as shown in Table 8. The SBP (mm/Hg) (p=0.156), DBP (mm/Hg) (p=0.558) and BMI (p=0.081) were not statistically significant when compared respectively.

3.9: Comparison of Haematological Parameters Between Female and Male Hypertensives The comparison of mean and standard deviation for female hypertensives were WBC (×109/L) 8.16±4.97), LYM (×109/L) (2.77±1.58), NEUT (×109/L) (4.81±0.47), RBC (×1012/L) (4.19±1.22), HGB (g/dl) (11.44±2.70), HCT (%) (33.87±7.42), MCV (fL) (82.01±9.19), MCH (pg) (28.26±6.78), MCHC (g/dl) (33.80±2.02), RDW-SD (fL) (46.44±10.08), RDW-CV (%) (14.88±6.41), PLT (×109/L) (239.43±113.79), MPV (fL) (9.91±1.13), PDW (fL) 13.21±2.65, plateletcrit (%) (0.24±0.09), PLR (113.60±10.61), NLR (2.47±0.36) and ESR (mm/hr) (48.62±4.00) for male hypertensives were WBC (×109/L) (8.03±5.72), LYM (×109/L) (2.38±1.36), NEUT (×109/L) (4.37±3.00), RBC (×1012/L) (4.30±1.10), HGB (g/dl) (12.42±4.90), HCT (%) (35.18±7.14), MCV (fL) (80.71±9.85), MCH (pg) (28.05±3.56), MCHC (g/dl) (34.38±1.86), RDW-SD (fL) (45.54±11.21), RDW-CV (%) (14.52±4.89), PLT (×109/L) (219.38±91.12), MPV (fL) (9.82±1.25), PDW (fL) (13.53±2.59), plateletcrit (%) (0.22±0.08), PLR (116.02±10.17), NLR (2.33±0.26) and ESR (mm/hr) (46.73±4.92) respectively as shown in Table 9.

The WBC $(\times 109/L)$ (p=0.874), LYM $(\times 109/L)$ (p=0.107), NEUT $(\times 109/L)$ (p=0.491), RBC $(\times 1012/L)$ (p=0.550), HGB (g/dl) (p=0.106), HCT (%) (p=0.265), MCV (fL) (p=0.392), MCH (pg) (p=0.815), MCHC (g/dl) (p=0.060), RDW-SD (fL) (p=0.707), RDW-CV (%) (p=0.594), PLT

 $(\times 109/L)$ (p=0.236), MPV (fL) (p=0.666), PDW (fL) (p=0.441), plateletcrit (%) (p=0.222), PLR (p=0.875), NLR (p=0.750) and ESR (mm/hr) (p=0.765) were not statistically significant.

3.10 Comparison of Acute Phase Reactants Between Female and Male Hypertensives

The comparison of the mean and standard deviation for female hypertensives were ALB (g/dl) 36.51 ± 16.46 , CRP (mg/L) (12.21 ± 8.64) and fibrinogen (mg/dL) (398.79 ± 108.45) for male hypertensives were ALB (g/dl) (35.38 ± 6.97), CRP (mg/L) (11.20 ± 8.04) and fibrinogen (mg/dL) (385.82 ± 106.26) respectively as shown in Table 10. The ALB (g/l) (p=0.601), CRP (mg/L) (p=0.452) and fibrinogen (mg/dL) (p=0.453) were not statistically significant when compared respectively.

Table 1: Demographic Characteristics of Hypertensives and Control Subjects

	Female	Male	Age (years)	Height (m)	Weight (kg)
Hypertensive	94	66	57±10	1.63±0.44	81.70±4.10
Control	73	27	51±5	1.64±0.90	69.88±5.35

Table 2: Comparison of Blood Pressures and Body Mass Index (BMI) of Female Hypertensives and Female Control Subjects

Typer tensives and remain control subjects								
	Female Hypertensive	Female Control (n=73)	P –value	t – value	Remark			
	(n=94)	Mean±SD						
	Mean±SD							
SBP (mm/Hg)	146.35±15.46	118.93±6.14	< 0.001	15.724	S			
DBP (mm/Hg)	100.36±7.96	79.96±6.07	< 0.001	18.805	S			
$BMI (kg/m^2)$	31.31±7.94	25.42 ± 7.17	< 0.001	4.948	S			

Abbreviations: SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; BMI: Body Mass Index; S: Significant; NS: Not Significant

Table 3: Comparison of Haematological Parameters of Female Hypertensives and Female Control Subjects

	Female Hypertensive (n=94) Mean±SD	Female Control (n=73) Mean±SD	P –value	t – value	Remark
WBC $(x10^3/\mu L)$	8.16±4.97	5.92 ± 2.55	< 0.001	3.768	S
LYM $(x10^3/\mu L)$	2.77 ± 1.58	2.27 ± 0.66	0.006	2.791	S

NEUT $(x10^3/\mu L)$	4.81 ± 0.47	3.32 ± 0.28	0.008	2.694	S
RBC $(x10^6/\mu L)$	4.19 ± 1.22	4.33±1.09	0.437	0.780	NS
HGB (g/dl)	11.44 ± 2.70	12.15 ± 3.15	0.120	1.564	NS
HCT (%)	33.87 ± 7.42	34.23 ± 5.49	0.716	0.365	NS
MCV (fL)	82.01±9.19	81.34±6.65	0.603	0.521	NS
MCH (fL)	28.26 ± 6.78	28.11±3.13	0.857	0.181	NS
MCHC (g/dl)	33.80 ± 2.02	35.20 ± 6.31	0.072	1.820	NS
RDW-SD (fL)	46.44±10.08	42.47±7.76	0.239	1.185	NS
RDW-CV (%)	14.88 ± 6.41	14.86 ± 5.14	0.985	0.018	NS
PLT $(x10^3/\mu L)$	239.43±113.79	226.24 ± 87.71	0.399	0.846	NS
MPV (fL)	9.91±1.13	10.17 ± 1.17	0.147	1.458	NS
PDW (fL)	13.21 ± 2.65	13.99 ± 2.91	0.071	1.818	NS
PLATELETCRIT	0.51 ± 0.27	1.99 ± 0.86	0.105	1.636	NS
(%)					
PLR	113.60±10.61	107.31 ± 5.94	0.606	0.517	NS
NLR	2.47 ± 0.34	1.68 ± 0.24	0.071	1.819	NS
ESR (mm/hr)	48.62±39.03	25.36±18.26	< 0.001	5.116	S
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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; S: Significant; NS: Not Significant

Table 4: Comparison of Acute Phase Reactants of Female Hypertensives and Female Control Subjects

	Female Hypertensive (n=94)	Female Control (n=73)	P –value	t – value	Remark
	Mean±SD	Mean±SD			
ALB (g/dl)	36.51±16.46	42.71±9.95	< 0.005	2.828	S
CRP (mg/L)	12.21 ± 8.64	5.13 ± 4.17	< 0.001	6.989	S
FIBRINOGEN (mg/dL)	398.79±108.45	292.04±85.63	< 0.001	6.882	S

Abbreviations: ALB: Albumin; CRP: C-Reactive Protein; S: Significant; NS: Not Significant

Table 5: Comparison of Blood Pressures and Body Mass Index (BMI) of Male Hypertensives and Male Control Subjects

	ond of Subjects				
	Male Hypertensive	Male Control (n=27)	P –value	t – value	Remark
	(n=66)	Mean±SD			
	Mean±SD				
SBP (mm/Hg)	149.88±15.50	121.15±5.47	< 0.001	9.371	S
DBP (mm/Hg)	99.61±8.08	80.85 ± 6.37	< 0.001	11.884	S
$BMI (kg/m^2)$	29.24±6.44	27.38 ± 4.53	0.175	1.367	NS

Abbreviations: SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; BMI: Body Mass Index; S: Significant; NS: Not Significant

Table 6: Comparison of Haematological Parameters of Male Hypertensive and Male Control Subjects

	Male	Male Control	P –value	t – value	Remark
	Hypertensive	(n=27)			
	(n=66)	Mean±SD			
	Mean±SD				
WBC $(x10^3/\mu L)$	8.03 ± 5.72	6.69 ± 2.41	0.245	1.171	NS
LYM $(x10^3/\mu L)$	2.38±1.36	2.31 ± 1.15	0.805	0.248	NS
NEUT $(x10^3/\mu L)$	4.37 ± 3.00	3.82 ± 2.02	0.391	0.863	NS
RBC $(x10^6/\mu L)$	4.30±1.10	4.00 ± 0.77	0.195	1.305	NS
HGB (g/dl)	12.42 ± 4.90	14.13 ± 2.72	0.435	0.79	NS
HCT (%)	35.18±7.14	32.90 ± 6.83	0.161	1.412	NS
MCV (fL)	80.71 ± 9.85	81.91±6.92	0.563	0.580	NS
MCH (fL)	28.05 ± 3.56	38.20 ± 10.00	0.318	1020	NS
MCHC (g/dl)	34.38 ± 1.86	34.57 ± 1.62	0.638	0.47	NS
RDW-SD (fL)	45.54±11.21	42.83 ± 6.98	0.246	1.167	NS
RDW-CV (%)	14.52 ± 4.89	15.39 ± 8.61	0.539	0.617	NS
PLT $(x10^3/\mu L)$	219.38±91.12	274.15±33.69	0.133	1.542	NS
MPV (fL)	9.82 ± 1.25	10.09 ± 1.13	0.338	1.003	NS
PDW (fL)	13.53 ± 2.59	13.90 ± 2.15	0.516	0.651	NS
PLATELETCRIT (%)	0.22 ± 0.01	2.86 ± 1.55	0.101	1.699	NS
PLR	116.02 ± 10.17	152.52 ± 30.47	0.149	1.456	NS

NLR	2.32 ± 0.26	2.34 ± 0.43	0.973	0.034	NS
ESR (mm/hr)	46.73±39.94	25.04 ± 20.35	0.001	3.450	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, NLR: Neutrophil/Lymphocyte Ratio; PLR: Platelet to Lymphocyte Ratio; ESR: Erythrocyte Sedimentation Rate; S: Significant; NS: Not Significant.

Table 7: Comparison of Acute Phase Reactants of Male Hypertensives and Male Control Subjects

	Male Hypertensive (n=66) Mean±SD	Male Control (n=27) Mean±SD	P –value	t – value	Remark
ALB (g/dl)	35.38 ± 6.97	39.59 ± 9.95	0.022	2.324	S
CRP (mg/L)	11.20 ± 8.04	4.18 ± 3.83	< 0.001	5.690	S
FIBRINOGEN (mg/dL)	385.82±106.26	300.56±95.81	0.001	0.634	S

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

Table 8: Comparison of Blood Pressures and Body Mass Index (BMI) between Female and Male Hypertensives

	Female (n = 74) Mean±SD	Male (n = 66) Mean±SD	P –value	t – value	Remark
SBP (mm/Hg)	146.35±7.95	149.88 ± 15.50	0.156	1.424	NS
DBP (mm/Hg)	100.36±7.95	99.61±8.08	0.558	0.586	NS
$BMI (kg/m^2)$	31.31±7.93	29.24 ± 6.44	0.081	1.757	NS

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

Table 9: Comparison of Haematological Parameters Between Female and Male Hypertensives

Female (n =	Male (n = 66)	Р –	t – Remark
74)	Mean±SD	value	value
Mean±SD			

WBC (x10 ³ /μL)	8.16±4.96	8.03±5.72	0.874	0.159	NS	
LYM $(x10^3/\mu L)$	2.77 ± 1.58	2.38±1.36	0.107	1.621	NS	
NEUT $(x10^3/\mu L)$	4.81 ± 0.48	4.37±0.37	0.491	0.691	NS	
RBC $(x10^6/\mu L)$	4.19 ± 1.22	4.30±1.10	0.550	0.599	NS	
HGB (g/dl)	11.44 ± 2.70	12.42 ± 4.90	0.106	1.625	NS	
HCT (%)	33.87±7.42	35.18 ± 7.14	0.265	1.119	NS	
MCV (fL)	82.01±9.19	80.71 ± 9.85	0.392	0.859	NS	
MCH (fL)	28.27 ± 6.78	28.05 ± 3.56	0.815	0.235	NS	
MCHC (g/dl)	33.80 ± 2.02	34.38 ± 1.86	0.060	1.890	NS	
RDW-SD (fL)	46.44 ± 10.08	45.54±11.21	0.707	0.377	NS	
RDW-CV (%)	14.88 ± 6.41	14.52 ± 4.89	0.594	0.534	NS	
PLT $(x10^3/\mu L)$	239.43±113.79	219.38±91.12	0.236	1.191	NS	
MPV (fL)	9.91±1.13	9.82 ± 1.25	0.666	0.432	NS	
PDW (fL)	13.21±2.65	13.53 ± 2.59	0.441	0.773	NS	
PLATELETCRIT	0.51 ± 0.27	0.22 ± 0.01	0.380	0.881	NS	
(%)						
PLR	113.60±10.61	116.02±10.17	0.875	0.158	NS	
NLR	2.47 ± 0.36	2.33 ± 0.26	0.750	0.319	NS	
ESR (mm/hr)	48.62±4.00	46.73 ± 4.92	0.765	0.300	NS	
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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, NLR: Neutrophil/Lymphocyte Ratio; PLR: Platelet to Lymphocyte Ratio; ESR: Erythrocyte Sedimentation Rate; S: Significant; NS: Not Significant.

Table 10: Comparison of Acute Phase Reactants Between Female and Male Hypertensives

	Female (n = 74) Mean±SD	Male (n = 66) Mean±SD	P-value	t-value	Remark
ALB (g/dl)	36.51±16.46	35.37±6.97	0.601	0.524	NS
CRP (mg/L)	12.21 ± 8.64	11.20 ± 8.04	0.452	0.753	NS
FIBRINOGEN (mg/dL)	398.79 ± 108.45	385.82±106.26	0.453	0.753	NS

Abbreviations: ALB: Albumin; CRP: C-Reactive Protein; S: Significant; NS: Not Significant

4. Discussion

Significantly elevated SBP (p=<0.001) among female hypertensives compared to female normotensive was observed in this study. This could be attributed to augmented peripheral resistance resulting from arteries that have become narrowed and less flexible, thus making it difficult for the heart to efficiently pump blood during systole. Additionally, an increased cardiac output causes a greater volume of blood to be pushed into the arteries with each contraction, and there is also the presence of endothelial dysfunction that can hinder vasodilation. This finding is in consonance with the findings of Vrettos et al. [16]; Eziuzo et al. [13].

This research finding showed a significant elevation in DBP (p=<0.001) among female hypertensives compared to female normotensives. This could be attributed to heightened resistance in peripheral arteries, which hampers the relaxation and expansion of arteries during the diastolic phase. Factors such as arterial stiffness, endothelial dysfunction and an increased blood volume may necessitate arteries to accommodate a greater volume of blood during diastole. This finding is in line with the findings of Vrettos et al. [16]; Eziuzo et al. [13].

Significantly elevated BMI levels (p=0.001) was observed among female hypertensives compared to female normotensives in this study. This indicates a rise in body fat which has been recognized as a distinct risk factor for hypertension. Nevertheless, the precise pathways linking visceral fat and hypertension remain incompletely comprehended. Inflammatory mechanisms might have a substantial role in the onset of hypertension. Adipose cells are responsive to fat metabolism and may generate considerable amounts of inflammatory cytokines, contributing to increased blood pressure. The finding in this study is supported by Eziuzo et al. [13]; Singh et al. [17].

Significantly elevated WBC was observed in this study among female hypertensives (p=<0.001) compared to female normotensives. This might be attributed to the common occurrence of chronic, low-level inflammation in the body associated with hypertension, which can have a detrimental impact on endothelial function. This finding is similar with the findings of Onyema et al [18]; Obeagu *et al.* [19].

Significantly increased LYM was observed in this study among female hypertensives (p=0.006) compared to female normotensives. An increased sympathetic nervous system activity in hypertensive individuals could also influence immune function, contributing to elevated lymphocyte levels. This finding is consistent with the findings of Onyema et al. [18]; Obeagu *et al.* [19]. Further research by Eziuzo et al. [13] showed no significant difference in absolute lymphocyte count among known hypertensive female participants as compared to the normotensive female participants. This may be attributed to the study area and smaller sample size of 150 and age ranging from 31-54 years of the subject used.

Findings from this study revealed significantly higher NEU was observed in this study among female hypertensives (p=0.008) compared to female normotensives. Hypertension being a chronic inflammatory condition may lead to increased neutrophil production as part of the immune response to ongoing inflammation. This finding is in consonance with the findings of Onyema et Citation: Chukwu PH, Christian SG, Eze EM, Ken-Ezihuo SU, Moore-Igwe BW, Okey-Omunakwe C, Obeagu EI. Gender-Based Assessment of Haematological Parameters and Acute Phase Reactants of Hypertensives in Port Harcourt, Nigeria. *Elite Journal of Haematology*, 2024; 2(6): 16-34

al. [18]; Obeagu *et al.* [19]. Further research by Eziuzo *et al.* [13] showed no significant difference in absolute neutrophil count among known hypertensive female participants as compared to the normotensive participants.

Findings in this study revealed significantly increased levels of ESR (<0.001) in female hypertensives compared to female normotensives. This could be the consequence of persistent inflammation, stemming from an intensified inflammatory reaction, which may play a substantial role in the underlying mechanisms contributing to the development of hypertension. This is not in agreement with the findings of Ighoroje and Dapper [20] who reported that there was no significant difference in erythrocyte sedimentation rate between the hypertensive females and normotensive females. This could be attributed to the study area and population used.

This study observed a notable decrease in ALB levels (p=<0.005) in female hypertensives when compared to female normotensives. This may be linked to the anti-inflammatory properties of albumin, which can influence the body's response to inflammation and vascular injury. The finding in this study is consistent with the findings of Berhanu *et al.* [21].

CRP levels (p=<0.001) were significantly elevated in female hypertensives compared to female normotensives in this study. This is possibly attributed to an ongoing inflammatory condition and obesity, which entails the release of pro-inflammatory cytokines from adipose tissue (fat cells). This finding is not in consonance with the study of Orji *et al.* [22].

Fibrinogen levels (p=<0.001) were significantly higher among female hypertensives compared to female control subjects in this study. This occurrence is probably linked to multiple factors within the intricate pathophysiology of hypertension driven by chronic inflammation, which can stimulate the liver to increase its production of fibrinogen. This finding is in consonance with the findings of Eldour *et al.* [23]; Shankar *et al.* [24].

Elevated levels of SBP (p=<0.001) in male hypertensives compared to male normotensives was observed in this study. This may be ascribed to an increased resistance in the peripheral arteries, which has occurred due to their narrowing and reduced flexibility, making it challenging for the heart to effectively pump blood during systole. Furthermore, an elevated cardiac output results in a larger volume of blood being ejected into the arteries during each contraction and there is also the occurrence of endothelial dysfunction which could impede the dilation of blood vessels. This finding is consistent with the findings of Eziuzo *et al.* [13]; Kazushi et al. [25].

Significantly increased DBP (p=0.001) in male hypertensives compared to male normotensives was observed in this study. This could be linked to increased resistance in peripheral arteries which impedes the ability of arteries to relax and expand during the diastolic phase. Various factors including arterial stiffness, endothelial dysfunction and a higher blood volume may require arteries to handle a greater volume of blood during diastole. This finding is consistent with the findings of Eziuzo *et al.* [13]; Kazushi *et al.* [25].

ESR levels (p=0.001) were significantly higher in male hypertensives compared to male normotensives in this study. This might result from prolonged inflammation originating from an escalated inflammatory response which could have a significant role in the mechanisms Citation: Chukwu PH, Christian SG, Eze EM, Ken-Ezihuo SU, Moore-Igwe BW, Okey-Omunakwe C, Obeagu EI. Gender-Based Assessment of Haematological Parameters and Acute Phase Reactants of Hypertensives in Port Harcourt, Nigeria. *Elite Journal of Haematology*, 2024; 2(6): 16-34

contributing to the onset of hypertension. This finding is not in agreement with the study of Ighoroje and Dapper [20] who reported that there was no significant difference in ESR between the hypertensive males and non-hypertensive male subjects. This could be attributed to the sample size used in the study.

ALB were significantly lower (p=0.022) in male hypertensives compared to male normotensives in this study. This might be associated with the anti-inflammatory characteristics of albumin, which can impact how the body responds to inflammation and vascular damage. This may be caused by endothelial dysfunction and inflammation. The finding in this study is consistent with the findings of Berhanu *et al.* [21].

Significantly elevated levels of CRP (p=<0.001) in male hypertensives compared to male normotensives was observed in this study. This increase in CRP among hypertensive subjects is linked to their enduring state of inflammation and obesity, which leads to the release of proinflammatory cytokines from adipose tissue (fat cells). This finding is not in line with the study of Orji *et al.* [22].

Higher plasma fibrinogen levels (p=0.001) in male hypertensives compared to male normotensives was observed in this study. This is likely associated with various elements in the complex pathophysiology of hypertension, which is influenced by chronic inflammation that can stimulate the liver to produce higher amounts of fibrinogen. This is in consonance with the findings of Shiradhonkar *et al.* [26]; Machlus *et al.* [27].

No significant differences in haematological parameters between males with hypertension and males with normal blood pressure was observed in this study. Hypertension itself is not typically associated with significant differences in most haematological parameters. However, other factors such as medications, co-existing health conditions and individual variations can influence these parameters [28]. This is not in consonance with the findings of Sileshi et al. [29] who reported that white blood cell count, mean cell haemoglobin, mean cell haemoglobin concentration and mean cell volume were significantly different between the two groups.

Findings in this study revealed that both male and female individuals with hypertension did not exhibit significant differences in their haematological parameters. The underlying causes and mechanisms of hypertension are generally similar in both genders, resulting in comparable effects on blood characteristics. Diagnostic criteria and treatment approaches for hypertension are not sexspecific and do not have a substantial impact on haematological parameters differently in men and women. This is not in consistence with the report by Obeagu *et al.* [19] that significant differences in lymphocyte count was observed based on gender.

5. Conclusion

Findings from this study revealed that there were no sex-dependent risks associated with blood inflammatory markers, components and blood pressure in hypertensive male and female subjects.

6. Recommendation

Sequel to this study, it can be recommended that the BMI, SBP and DBP of hypertensives should be monitored regularly, at least once daily to reduce risks of cardiovascular complications.

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.

Competing Interests

Authors have declared that no competing interests exist.

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