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Original Research Article

Assessment of Some Haematological Parametrs in Cardiovascular Patients Attending Federal Teaching Hospital, Owerri

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Abstract

Cardiovascular diseases are heavy burden in the society. This study was carried out to assess the haematological parameters in cardiovascular disease subjects at federal teaching hospital owerri, Imo state, Nigeria. A total of 200 subjects were recruited for the study out of which 140 were people suffering from cardiovascular disease while 60 were apparently healthy control subjects. The levels of haematological parameters were determined was assayed by sysmex KY2IN haematology analyzer. The data generated from these researches were analyzed using SPSS statistical software version 20.0. The result showed that the full blood count, of the subjects and controls were analyzed and there were haematological alterations. In CVD, Rbc count, Hb, PCV and MCHC were significantly reduced when compared with the control (P= 0.001, p=0.036, p=0.002 and 0.050 respectively) and their mean ±SD values 4.37±/5.2±×10^{12}/L, 13.19±/13.86±G/DL, 39.70±/42.18±%, and 33.38±/34.22±g/dl respectively. Platelet count did not differ significantly between groups. (p=0.125) when compared to the control (mean ±SD 253.49±/283.97±×10^{9}/l). There was increased wbc count, Mpv and PLCR in cardiovascular disease (P= 0.027, p=0.001, p= 0.001 respectively) with mean ±SD 8.40±/7.43±×10^{9}/l, 10.53±/9.14± FL, also increased significantly in CVD (P=0.001 p=0.005. There was no statistically significant difference in MCV values among the CVD subjects (mean ±SD 88.94±/87.06±FL, p=0.188). There was no significant gender-based concentration values observed in all other parameters measured in the diseases under study. These levels did not differ significantly between sexes. From the findings, the inclusion of full blood count(FBC), in the routine laboratory investigations of conditions related to cardiovascular disease, will be useful in the prediction, management and risk assessment of the disease and may subsequently improve treatment outcome.

Keywords: *haematological parametrs, cardiovascular, owerri.*

INTRODUCTION

Any condition affecting the heart or blood arteries is referred to as cardiovascular disease (CVD). It falls into one of four categories, such as coronary heart disease (CHD) or coronary artery disease (CAD). Angina, myocardial infarction (MI), and/or heart failure are brought on by a reduction in myocardial perfusion. Between one-third and half of CVD cases are caused by it [1]. Cerebrovascular disease ncludes stroke, which can be either hemorrhagic or ischaemic. Peripheral artery disease (PAD), especially limb-related vascular disease that can cause claudication Atherosclerosis of the arteries. Abdominal and thoracic aneurysms are among them [2].

Worldwide, cardiovascular disease (CVD) is the leading cause of mortality. Primary prevention is characterised by actions intended to stop the advancement of atherosclerosis. The burden of cardiovascular illnesses on society is significant. These illnesses affect around 750 million people globally [3].

Despite the existence of several research on homocystein, haematological markers, and their correlation with cardiovascular disease (CVD), there is a dearth of published data in this region of the nation. Hyperhomocysteinemia, or elevated homocystein, is linked to a higher risk of renal disease, heart disease, and prostate cancer, among other illnesses. Additionally, it has been connected to autoimmune illnesses, neurological diseases, and some types of cancer. Haematological indicators and blood cell count can be impacted by cardiovascular problems [4].

Because of this assumption, it becomes essential to evaluate the haematological parameters linked to these illnesses. We can determine the risk or advancement of various diseases with the help of this study. [5]

Due to their high rates of morbidity and mortality, cardiovascular diseases are a major public health problem and a global health burden. Inflammation, oxidative stress, and metabolic abnormalities are among the risk factors that frequently overlap between these illnesses. The presence of systemic disorders can cause major changes in haematological parameters, which include haemoglobin levels, red and white blood cell counts, and haemotocrit. These parameters are crucial indicators of the body's ability to carry oxygen, immunological function, and general health. Although these biomarkers have been extensively studied separately, little is known about how haematological alterations and status relate to each of the three main disease groups. Early detection, risk assessment, and focused nutritional or therapeutic intervention are all hampered by this knowledge gap. Cost-effective biomarker profiling has the potential to greatly enhance patient management and results in settings with limited resources and a lack of sophisticated diagnostic tools. Investigating the trends and interactions of haematological markers in people with cardiovascular disease and contrasting them with those in healthy populations is therefore crucial. This will make it clearer if these markers may be used as easily accessible early indicators for the prognosis, progression, or risk of disease [6].

MATERIALS AND METHODS

STUDY AREA

The study was carried out at Federal teaching hospital (FETHO), Owerri, Imo state, Nigeria. Owerri is located in the South East of Nigeria. It is the capital of Imo State. It lies on latitude 5° 28¹ 59`` north Longitude 7° 01¹ 49`` north east and 159 meters above sea level.

Advocacy, Mobilization and Pre survey contacts

This research was carried out in agreement with the international guiding principles for research involving humans.

STUDY DESIGN

A cohort study design was used to study the Haematological parameters in cardiovascular disease subjects

STUDY POPULATION

The populations of the study were people with cardiovascular disease, attending Federal teaching hospital owerri (FETHO). Control subjects were apparently healthy individuals, who met the inclusion criteria.

Selection criteria

Inclusion criteria

Those included in the study were:

i. Male and female subjects with cardiovascular disease
ii. Male and female that are apparently healthy without cardiovascular disease
iii. Men that are apparently healthy (not diagnosed of prostate cancer) (control subjects)

Exclusion criteria

i. Men that have undergone prostate surgery
ii. Patients diagnosed of malignant disease other than prostate cancer.

Sample collection and preparation

Samples were collected from two hundred (200) subjects who met the inclusion criteria and also consented to the study. Sixty of these subjects were the healthy control whereas one hundred and forty (140) subjects were the test group.

LABORATORY PROCEDURES

All reagents were commercially purchased and the manufacturer's standard operating procedures were strictly adhered to.

Determination of Full blood count analysis (Using sysmex haematology analyzer KY2IN Model)

Parameters assayed are: White blood cell count (WBC), red blood cell count (RBC), Haemoglobin (HB), Haemtocrit (PCV), mean cell volume (MCV), Mean cell haemoglobin concentration (MCHHC), Red cell distribution with width (RDW), platelet count (PET), Mean platelets volume (MPV), Platelet distribution width (PDW) and Platelet large cell ratio (P-LCR).

The operating principle of haematology analyzers uses the coulter impendence method. This method counts cells by detecting and measuring changes in electrical impendence when a particle in a conducting liquid pass through the aperture. For each cell passing through the aperture, there is constant current flowing between the external and internal electrodes and causes some changes in the impendence in the conductive blood cell suspension. These changes are

recorded as increases in the voltage between the electrodes. The number of pulses is proportional to the number of particles the intensity of each pulse is proportional to the volume of that particle.

For the haemoglobin measurement the sample is lysed, diluted and measured photometrically. The reagent lyses the red blood cells which releases haemoglobin. The chemical process for a stable form of methaemoglobin and in the chamber and the result is displayed on the screen.

Procedure:
Start up was run at the beginning of the first session each operational day and the date and fine displayed were checked if they are correct, the sample was mixed gently, the sample tube was opened and the aspirator tip was immersed in the sample. The sample bar was pressed and released; The sample was then removed when the instrument beeped. The results were displayed on the screen.

STATISTICAL ANALYSIS

Data were analysed using statistical package for social science (SPSS) statistical software (Version 20) (SPSS Inc, Chicago. USA). Values obtained were expressed as mean ± standard deviation and results presented in tables and charts. Compact letters display (CLD) were used to compare all parameters among various diseases. The study also employed T-test to analyse values between two group at 95% confidence limit/interval.

RESULTS

Table 1: Mean ± SD values of Hematological Parameters between CVD Subjects and Controls
White Blood Cell (WBC) count was significantly higher in CVD subjects ($8.40 \pm 1.69 \times 10^9$/L) compared to controls ($7.43 \pm 1.90 \times 10^9$/L), $p = 0.027$. Red Blood Cell (RBC) count was significantly lower in CVD subjects ($4.37 \pm 0.74 \times 10^{12}$/L) than in controls ($5.21 \pm 0.33 \times 10^{12}$/L), $p = 0.001$, indicative of possible anemia associated with cardiovascular complications. Hemoglobin (HB) levels were also lower in CVD subjects (13.19 ± 1.23 g/dL) compared to controls (13.86 ± 1.37 g/dL), $p = 0.036$, supporting mild anemia. Packed Cell Volume (PCV) was significantly reduced in the CVD group ($39.70 \pm 3.13\%$) compared to controls ($42.18 \pm 3.29\%$), $p = 0.002$, corroborating the hemoglobin findings. Mean Corpuscular Volume (MCV) did not show a statistically significant difference between CVD (88.94 ± 5.90 fL) and controls (87.06 ± 4.61 fL), $p = 0.188$. Mean Corpuscular Hemoglobin (MCH) was similar in both groups: 30.22 ± 3.04 pg in CVD and 30.33 ± 2.52 pg in controls, $p = 0.878$. Mean Corpuscular Hemoglobin Concentration (MCHC) was slightly lower in CVD (33.38 ± 1.53 g/dL) compared to controls (34.22 ± 2.16 g/dL), with marginal significance, $p = 0.050$. Red Cell Distribution Width (RDW) was significantly higher in CVD ($15.03 \pm 1.18\%$) versus controls ($12.89 \pm 1.16\%$), $p = 0.001$, suggesting increased red cell size variation often associated with chronic disease states. Platelet count (PLT) did not differ significantly between groups (CVD: $253.49 \pm 79.22 \times 10^9$/L vs Control: $283.97 \pm 76.64 \times 10^9$/L), $p = 0.125$. Mean Platelet Volume (MPV) was significantly higher in CVD subjects (10.53 ± 0.90 fL) than in controls (9.14 ± 1.21 fL), $p=0.001$, indicating increased platelet reactivity. Platelet Distribution Width (PDW) was elevated in CVD ($14.29 \pm 1.92\%$) compared to controls ($12.96 \pm 1.50\%$), $p = 0.005$, reinforcing the evidence of platelet morphological changes. Platelet Large Cell Ratio (PLCR) was significantly higher in CVD subjects ($34.76 \pm 7.03\%$) than in controls ($28.77 \pm 5.37\%$), $p = 0.001$.

Table 1: Mean ± SD values of Hematological Parameters between CVD Subjects and Controls

Parameter	CVD Subjects (N = 80)	Controls (N = 20)	*t-value*	*p*-value
WBC ($\times 10^9$/L)	8.40 ± 1.69	7.43 ± 1.90	2.24	0.027*
RBC ($\times 10^{12}$/L)	4.37 ± 0.74	5.21 ± 0.33	-4.93	< 0.001*
HB (g/dL)	13.19 ± 1.23	13.86 ± 1.37	-2.12	0.036*
PCV (%)	39.70 ± 3.13	42.18 ± 3.29	-3.14	0.002*
MCV (fL)	88.94 ± 5.90	87.06 ± 4.61	1.33	0.188
MCH (pg)	30.22 ± 3.04	30.33 ± 2.52	-0.15	0.878
MCHC (g/dL)	33.38 ± 1.53	34.22 ± 2.16	-1.99	0.050
RDW (%)	15.03 ± 1.18	12.89 ± 1.16	7.32	< 0.001*
PLT ($\times 10^9$/L)	253.49 ± 79.22	283.97 ± 76.64	-1.55	0.125
MPV (fL)	10.53 ± 0.90	9.14 ± 1.21	5.77	< 0.001*
PDW (%)	14.29 ± 1.92	12.96 ± 1.50	2.90	0.005*
PLCR (%)	34.76 ± 7.03	28.77 ± 5.37	3.56	0.001*

Keys: Wbc= White Blood Cell count, Rbc = Red Blood Cell count, HB= Haemoglobin, Pcv= Packed Cell Volume, MCV= Mean Corpuscular Volume, MCH= Mean Corpuscular Hemoglobin, MCHC= Mean Corpuscular Hemoglobin

Concentration, RDW= Red Cell Distribution Width, PLT=Platelet count, MPV = Mean Platelet Volume, PDW = Platelet Distribution Width, PLCR = Platelet Large Cell Ratio

Key:*** = significant p value**

DISCUSSION

Several haematological markers in cardiovascular individuals were evaluated in this study. A major global health concern, cardiovascular disease (CVD) is frequently linked to complicated haematologic abnormalities that reflect oxidative stress, endothelial dysfunction, systemic inflammation, and subclinical nutritional deficits [7]. As compared to healthy controls, the current study demonstrates that while white blood cell (WBC) count, platelet large cell ratio (PLCR), platelet distribution width (PDW), red cell distribution width (RDW), and mean platelet volume (MPV) were significantly increased ($p = 0.001$ for all), RBC count, haemoglobin (Hb), packed cell volume (PCV), mean corpuscular haemoglobin concentration (MCHC), and platelet count were significantly decreased in CVD subjects. These results have diagnostic and prognostic implications and provide insight into the proinflammatory, prothrombotic, and anaemic states that commonly accompany CVD.

It is becoming more widely acknowledged that anaemia is a poor prognostic indicator in people with CVD. Anaemia of chronic inflammation or anaemia of chronic illness (ACD), which is frequently seen in heart failure, ischaemic heart disease, and other chronic cardiovascular conditions, is indicated by a decrease in RBC count, Hb, and PCV [8]. This haematologic phenotype is caused by a number of processes, such as chronic inflammation, which reduces erythropoiesis and impedes iron utilisation by downregulating erythropoietin synthesis and iron availability through interleukin-6-induced hepcidin production [9].

Additionally, erythropoietin production is decreased due to reduced renal perfusion in congestive heart failure, which further suppresses RBC output [10]. Iron, vitamin B12, and folate deficits are common in chronic cardiovascular disease (CVD) and can lead to hypoproliferative anaemia [11]. Pseudoanemia may result from hemodilution, especially in congestive heart failure [12]. The decrease in MCHC indicates hypochromia, which supports the idea that an iron deficit, whether functional or absolute, is a contributing factor to anaemia [13].

It is noteworthy that the platelet count in CVD patients has significantly decreased. Although platelet activation and thrombosis are frequently linked to CVD, thrombocytopenia can also be brought on by endothelial dysfunction, which causes platelet adhesion and aggregation at sites of vascular injury, antiplatelet therapy (such as aspirin or clopidogrel), or consumption coagulopathy in acute coronary syndromes or heart failure [14, 15]. Despite the decreased number of platelets, their morphological and functional traits—discussed below—indicate heightened reactivity.

Low-grade systemic inflammation, a key pathophysiological characteristic of atherosclerosis and CVD, is reflected in the notable increase in WBC count [16]. Leukocytosis has been associated with endothelial activation and dysfunction, plaque instability and rupture as a result of inflammatory cells secreting proteases [17], and elevated oxidative stress, all of which affect erythropoiesis and encourage thrombosis [18]. Particularly in acute coronary syndromes, an elevated WBC count has been found to be an independent predictor of subsequent cardiovascular events [19].

Subjects with CVD showed a marked increase in red cell distribution width (RDW), a measure of RBC size variability. Increased oxidative stress and compromised red cell membrane integrity, which result in fluctuating cell lifespan and morphology [21], are further indicators of inefficient erythropoiesis, which is most likely brought on by iron-restricted erythropoiesis, chronic inflammation, or vitamin B12/folate deficits [20]. Even in patients without overt anaemia, high RDW has been demonstrated to independently predict cardiovascular and all-cause mortality. [22]

MPV (mean platelet volume), PDW (platelet distribution width), and PLCR (platelet large cell ratio) were sharply increased in spite of a decreased platelet count; this is in line with the idea of reactive thrombopoiesis and elevated platelet turnover [23].

MPV is a measure of platelet activity and size. Bigger platelets are more metabolically active, have more thrombotic potential, and have denser granules and thromboxane A2 [24]. Increased MPV is linked to recurrent thrombotic events in coronary artery disease, acute myocardial infarction, and stroke, especially in patients with atherothrombosis [25].

The thromboembolic risk in individuals with cardiovascular disease is further increased by PDW and PLCR, which show heterogeneity in platelet size and suggest accelerated platelet creation, frequently in response to peripheral destruction, inflammatory stimulation of megakaryopoiesis, and platelet activation. Collectively, these indices are helpful indicators for cardiovascular risk stratification and indicate a prothrombotic condition even when thrombocytopenia is present [26].

According to recent research, patients with prostate cancer exhibit severe haematological abnormalities, as evidenced by markedly lower red cell indices (RBC, Hb, PCV, MCHC) and higher leukocyte and platelet volume indices (WBC, RDW, MPV, PDW, PLCR). The complicated interaction of tumor-induced inflammation, bone marrow and nutritional suppression, and increased haematologic activity that frequently accompanies the advancement of malignancy is reflected in these changes [27].

Anaemia of malignancy, a typical paraneoplastic symptom in cardiovascular disorders, is consistent with the reported decrease in haemoglobin (Hb), PCV, MCHC, and RBC count [28]. Chronic inflammation increases hepcidin, which prevents iron absorption and traps iron in macrophages, resulting in functional iron shortage. This anaemia is frequently multifactorial.

A lower MCHC suggests a hypochromic component, which could be caused by chronic blood loss or hepcidin-mediated inhibition of iron-restricted erythropoiesis.

The markedly increased red cell distribution width (RDW) was indicative of anisocytosis, a defining feature of inefficient erythropoiesis. The inflammatory inhibition of erythropoiesis, which results in heterogeneous RBC formation, disrupts normal haematopoiesis, and increases red cell size variability, has been linked to high RDW in CVD. Interestingly, in patients with CVD, high RDW has been linked to poor survival and advanced disease stage [29], probably as a result of its association with malnutrition and systemic inflammation.

The pro-inflammatory condition frequently observed in cancers is reflected in the rise in white blood cell (WBC) count. WBC rise in CVD may be a sign of tumor-induced chronic inflammation, which can lead to poor prognosis and accelerate tumour progression through angiogenesis, immune evasion, and metastasis or leukocytosis as a paraneoplastic phenomenon. Changes in the neutrophil-lymphocyte ratio (NLR), a proven prognostic indicator in CVD, are likewise linked to elevated WBC counts [30].

CONCLUSION

Haematological parameter results shows that anaemia is a hall mark of cardiovascular disease. This study revealed profound haematological derangements in cardiovascular disease. The observed profile of anaemia coupled with leucocytosis and platelet activation supports the notion that cardiovascular disease is a systemic disease

Individuals with cardiovascular disease (CVD) exhibit significant haematological deviations especially in parameters related to erythrocytes indices and platelet activity, consistent with chronic inflammation, anaemia and vascular complications seen in Chronic cardiovascular disease.

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