

Alterations of Haemoglobin, Total and Differential White Blood Cell Counts in Patients Diagnosed with Chronic Myeloid Leukemia in Owerri, Nigeria

¹Onwubiko Goodness E., ²Aloy-Amadi Oluchi C., ³Akogu Okechukwu and ⁴Okpara Lydia T.

¹Student ²Senior Lecturer Department of Medical Laboratory Science, Imo State University, Owerri, Nigeria, ³Senior Lecturer Department of Optometry, Imo State University, Owerri, Nigeria, ⁴Lecturer Whitetouch Diagnostic Centre, Lagos, Nigeria

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Abstract

Leukemia can be described as a malignant progressive disease in the blood from excessive or abnormal immature white blood cell. Which results to acute or chronic leukemia. Chronic myeloid leukemia (CML) is one of the most common leukemias occurring in the adult population. It is a type of leukemia which starts in the myeloid cells of the bone marrow. In CML, the disorder is characterized by translocation t(9;22)(q34;q11), resulting in the fusion of BCR and ABL1 genes into the pathogenic BCR-ABL1 oncogene, with many subsequent effects on downstream pathways. The study was aimed at deterring the levels of alteration of haemoglobin, Total and different white blood cell counts in patients attending Federal Teaching Hospital, Owerri. A total of 60 subjects (30 patients and 30 controls) were recruited for the study. Participants completed an informed consent form and questionnaire. Two milliliters (2 mls) of venous blood sample was collected at the ante-cubital vein aseptically and 2 mls was dispensed into ethylenediamine tetraacetic acid containers, and used for the analysis of hemoglobin using cyanmethemoglobin method, white blood cell count (WBCS) using manual WBC and differential white cell counting method. The mean values of haemoglobin (8.78±1.82)g/dl and neutrophils(28.47±10.27)%, in patients with leukemia were significantly reduced when compared to controls. (11.89±1.04)g/dl, and (48.83±12.86)%, (t=8.08,P=0.000) and (t=6.78,P=0.000). On the other hand, The mean values of TWBC (30.43±29.19)×10⁹/L, lymphocytes (57.90±11.16)%, monocytes (11.07±6.87)% and Eosinophils (2.20±1.32)% in patients with chronic myeloid leukaemia were significantly raised when compared to the controls (7.14±2.27) ×10⁹/L, (48.83±12.86)%, (44.50±13.86)%, (4.93±3.05)% and (1.53±0.94)%. (t=4.36, p=0.000, t=4.13, p=0.000); (t=4.47, p=0.000); and (t=2.25, p=0.028). The mean values of Hb (9.82±1.34)g/dl, Neutrophils (30.35±10.70)% and lymphocytes (58.06±12.70)%, in males with chronic myeloid leukemia were non-significantly higher compared to the females(7.42±1.46)g/dl,(26.00±9.53)%,(57.69±9.25)%, respectively(t=4.69,p=0.178;t=6.78,p=0.257) and (t=4.13,p=0.931). The mean values of TWBC (27.74±23.21)×10⁹/L, monocytes (9.76±5.21)% and eosinophils(1.29±0.85)% were significantly raised in males with chronic myeloid leukaemia when compared to females (33.94±36.29)×10⁹/L, (12.77±8.51)%, and (1.85±0.98)%, respectively(t=4.36,p=0.574;t=4.47,p=0.242) and t=2.25,p=0.111).There was a non-significant negative correlation of haemoglobin with TWBC, Neutrophils, Lymphocytes, Monocytes and Eosinophils in

Corresponding Author: Onwubiko Goodness E. Department of Medical Laboratory Science, Imo State University, Owerri, Nigeria.

E-mail: goodnessonwubiko5@gmail.com

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Chronic Myeloid Leukaemia Patients. ($r=-0.16$, $p=0.41$, $r=-0.15$, $p=0.432$, $r=-0.03$, $p=0.868$; $r=-0.06$, $p=0.728$ and $r=-0.29$, $p=0.110$). CML is associated with alteration in the levels of haemoglobin, total and differential WBC counts, resulting in anaemia and leukocytosis. Therefore complete blood count (CBC) is recommended as a routine test in the diagnosis, management and treatment of CML.

Keywords: Chronic Myeloid Leukemia, Haemoglobin, White Blood Cell, Differential White Cells.

Introduction

Chronic myeloid leukemia (CML) is a clonal myeloproliferative neoplasm that originates in the bone marrow and is characterized by the uncontrolled proliferation of myeloid cells in the peripheral blood and bone marrow. It accounts for about 15–20% of adult leukemias worldwide and typically affects middle-aged and older adults, though it can also occur in younger individuals^{1,2}. The hallmark genetic abnormality associated with CML is the presence of the Philadelphia chromosome, which results from a reciprocal translocation between chromosomes 9 and 22 [t(9;22)(q34;q11)]. This rearrangement produces the BCR-ABL1 fusion gene, a constitutively active tyrosine kinase that promotes continuous cell division and inhibits apoptosis^{3,4}.

CML typically progresses through three phases: the chronic phase, the accelerated phase, and the blast crisis. Most patients are diagnosed during the chronic phase, which may be asymptomatic or present with mild symptoms such as fatigue, night sweats, weight loss, and splenomegaly⁵. As the disease advances, it alters hematopoiesis, leading to hematological abnormalities such as anemia (low red blood cell count), leukocytosis (elevated white blood cell count), and varying shifts in the differential white blood cell distribution⁶. Monitoring these hematological changes is central to both diagnosis and disease management, as they provide insights into disease progression and treatment response.

Despite advances in targeted therapy with tyrosine kinase inhibitors (TKIs), hematological monitoring remains essential in resource-limited settings where molecular testing may not be readily available. In Nigeria and other sub-Saharan African countries, many health facilities still rely primarily on hematological profiles for diagnosis, prognosis,

and monitoring of hematological malignancies, including CML⁷. However, there is a paucity of published data on the specific hematological alterations associated with CML in Nigerian populations, particularly in southeastern Nigeria.

While numerous studies in Europe, Asia, and North America have extensively documented hematological changes in CML patients, there is limited region-specific evidence from Nigeria. Variations in genetic, environmental, and healthcare factors may influence disease patterns and outcomes across populations. This study therefore aims to provide valuable insight into the hematological alterations, particularly hemoglobin concentration and white blood cell distribution, among CML patients in Federal Teaching Hospital, Owerri. Such data could enhance clinical decision-making, improve disease monitoring, and contribute to the growing body of knowledge on CML in sub-Saharan Africa.

Materials and Methods

Study Area

The research was conducted at the Federal Teaching Hospital Owerri (FTHO), located in Imo State, Nigeria.

Study Design

This was a hospital-based case-control study involving patients with confirmed CML and healthy individuals as controls, which was carried out from the month of February to May, 2024. All eligible subjects who gave their informed consent for the study were sampled to investigate the alterations of haemoglobin, total white blood cell count and differential count in chronic myeloid leukemia patients. The study population consisted

of 60 subjects (30 Patients and 30controls). The procedure was carried out at Federal Teaching Hospital Owerri. The data was analyzed using SPSS version 25.

Participants

Sixty people were included 30 patients diagnosed with CML and 30 healthy controls matched by age and sex. All participants gave their informed consent and completed a short questionnaire.

Method of Recruitment

Patients were recruited from the hematology unit of the hospital based on medical diagnosis and ongoing follow-up. Healthy individuals were selected from hospital workers and visitors who met the inclusion criteria.

Ethical Approval

Ethical approval was obtained from the hospital's Ethics Committee (FTH/OW/HREC/VOL.1/046), and all participants gave written informed consent before being included in the study.

Sample Collection

Two milliliters of blood were drawn from each participant using sterile techniques and placed into EDTA tubes to prevent clotting.

Laboratory Procedures

Haemoglobin Estimation: Measured using the cyanmethemoglobin method, a standard technique for determining haemoglobin concentration.

Total WBC Count: Performed manually using a microscope and specialized counting chamber.

Differential Count: A stained blood smear was used to count individual types of white blood cells (neutrophils, lymphocytes, monocytes, and eosinophils).

Statistical Analysis

The data were analyzed using SPSS version 25. Differences between patients and controls were assessed using independent t-tests. Pearson's correlation was used to explore the relationship between haemoglobin and WBC counts. A p-value less than 0.05 was considered statistically significant.

Results

Table 1. Mean Values of Haemoglobin, TWBC, Neutrophils, Lymphocytes and Eosinophils in Chronic Myeloid Leukemia and Control Subjects.

| Parameter | Test | Control | t-value | p-value |
|----------------------------|-------------|-------------|---------|---------|
| Hb (g/dl) | 8.78±1.82 | 11.89±1.04 | 8.08 | 0.000* |
| TWBC (x10 ⁹ /L) | 30.43±29.19 | 7.14±2.27 | 4.36 | 0.000* |
| Neutrophils (%) | 28.47±10.27 | 48.83±12.86 | 6.78 | 0.000* |
| Lymphocytes (%) | 57.90±11.16 | 44.50±13.86 | 4.13 | 0.000* |
| Monocytes (%) | 11.07±6.87 | 4.93±3.05 | 4.47 | 0.000* |
| Eosinophils (%) | 2.20±1.32 | 1.53±0.94 | 2.25 | 0.028* |

Key

Hb: Haemoglobin

TWBC: Total White Blood Cell

*: Significant p value

D:Standard deviation

The mean value of haemoglobin (8.78±1.82)g/dl and neutrophils (28.47±10.27)%, in patients with leukemia were significantly reduced when compared to controls. (11.89±1.04)g/dl, and (48.83±12.86)%, (t=8.08,P=0.000) and (t=6.78,P=0.000). On the other hand, The mean value of TWBC (30.43±29.19)x10⁹/L,

lymphocytes (57.90±11.16)%, monocyte (11.07±6.87)% and Eosinophils (2.20±1.32)% in patients with chronic Myeloid Leukaemia were significantly raised when compared to the controls (7.14±2.27)

×10⁹/L, (48.83±12.86)%, (44.50±13.86)%, (4.93±3.05)% and (1.53±0.94)%. (t=4.36, p=0.000, t=4.13, p=0.000); (t=4.47, p=0.000); and (t=2.25, p=0.028).

Table 2. Mean Value of Haemoglobin, TWBC, Neutrophils, Lymphocytes, Monocytes and Eosinophils in Male and Female Patients with Chronic Myeloid Leukaemia.

| Parameter | Male n=15 | Female n=15 | t-value | p-value |
|----------------------------|--------------|----------------|---------|---------|
| Hb (g/dl) | 9.82±1.34 | 7.42±1.46 | 4.69 | 0.178* |
| TWBC (×10 ⁹ /L) | 27.74±23.21 | 33.94±36.29 | 4.36 | 0.574* |
| Neutrophils (%) | 30.35±10.70 | 26.00±9.53 | 6.78 | 0.257 |
| Lymphocytes (%) | 58.06±12.70 | 57.69±9.25 | 4.13 | 0.931 |
| Monocytes (%) | 9.76±5.21 | 12.77±8.51 | 4.47 | 0.242 |
| Eosinophils (%) | 1.29±0.85 | 1.85±0.98 | 2.25 | 0.111 |

Key

Hb: Haemoglobin

TWBC: Total White Blood Cell

S.D: Standard Deviation

The mean values of Hb (9.82±1.34)g/dl, Neutrophils (30.35±10.70)% and Lymphocytes (58.06±12.70)%, in males with chronic myeloid leukemia were non-significantly increased compared to the females (7.42±1.46)g/dl, (26.00±9.53)%, (57.69±9.25)%, respectively (t=4.69, p=0.178; t=6.78, p=0.257) and (t=4.13, p=0.931).

The mean values of TWBC (27.74±23.21)×10⁹/L, monocytes (9.76±5.21)% and eosinophils (1.29±0.85)% in males with chronic myeloid leukaemia were insignificantly lowered when compared to females (33.94 36.29)×10⁹/L, (12.77±8.51)%, and (1.85±0.98)%, respectively (t=4.36, p=0.574; t=4.47, p=0.242) and t=2.25, p=0.111).

Table 3. Correlation of Haemoglobin with TWBC and Differential Count in Patients with Chronic Myeloid Leukaemia.

| Variable | N | r | p-value |
|-------------|----|-------|---------|
| TWBC | 30 | -0.16 | 0.411 |
| Neutrophils | 30 | -0.15 | 0.432 |
| Lymphocytes | 30 | -0.03 | 0.868 |
| Monocytes | 30 | -0.06 | 0.728 |
| Eosinophils | 30 | -0.29 | 0.110 |

Key

Hb: Haemoglobin

TWBC: Total White Blood Cell

r: correlation coefficient

There was a non - significant negative correlation of haemoglobin with TWBC, Neutrophils, Lymphocytes, Monocytes and Eosinophils in patients with Chronic Myeloid Leukaemia (r=-0.16, p=0.411, r=-0.15, p=0.432, r=-0.03, p=0.868; r=-0.06, p=0.728 and r=-0.29, p=0.110).

Discussion

The present study demonstrated that patients with CML had significantly reduced hemoglobin concentrations compared to healthy individuals. This finding is consistent with the well-established understanding that CML disrupts normal hematopoiesis in the bone marrow, leading to suppression of erythropoiesis and the development of anemia⁸. Previous studies in India and Ethiopia similarly reported lower hemoglobin levels among CML patients, attributing this to both bone marrow infiltration and chronic disease burden^{9,10}.

In addition, our study revealed markedly elevated total white blood cell counts among CML patients. This finding aligns with the pathophysiological hallmark of CML, which involves uncontrolled

proliferation of myeloid cells driven by the BCR-ABL1 fusion protein³. Elevated leukocyte counts have been widely reported in CML patients across various populations, including studies in Pakistan and South Africa^{11,12}.

Interestingly, differential white blood cell distribution showed reduced neutrophil percentages but increased lymphocytes, monocytes, and eosinophils. These abnormalities reflect the disruption in myeloid maturation and expansion of abnormal cell lineages characteristic of CML. Similar patterns were described in studies from Egypt and Ghana, where elevated eosinophil and basophil counts were highlighted as potential markers of disease progression^{13,14}.

When gender differences were analyzed, the study did not find significant variations in hematological parameters between male and female patients. This suggests that the hematological impact of CML is largely independent of sex, consistent with findings from previous reports in Nigeria and India^{10,15}.

Furthermore, correlation analysis showed negative but statistically insignificant relationships between hemoglobin levels and white cell subtypes. Although anemia and leukocytosis often coexist in CML, the lack of strong correlation indicates that the severity of anemia may not directly predict leukocyte proliferation. This finding mirrors the results of a study in Ethiopia, which found no significant association between hemoglobin concentration and total leukocyte count in CML patients⁹.

Our results are comparable with those of Nwabuko et al.⁷, who reported anemia and leukocytosis as dominant features among CML patients in Nigeria. Similarly, a study in India observed significantly higher total leukocyte counts and lower hemoglobin in CML cases compared to controls¹⁰. However, unlike some studies that reported male predominance in CML-related anemia¹⁵, our study did not observe gender-related differences. This could be due to regional, genetic, or sample size variations.

The consistent alterations in hemoglobin concentration and leukocyte distribution underscore

the clinical utility of complete blood counts in the diagnosis and monitoring of CML, especially in low-resource settings. Regular hematological assessment can serve as an affordable surrogate marker for disease progression and treatment response where advanced molecular diagnostics are not available.

Conclusion

This study demonstrated that chronic myeloid leukemia (CML) significantly alters hematological parameters, with patients presenting with markedly reduced hemoglobin levels, elevated total white blood cell counts, and abnormal distributions of neutrophils, lymphocytes, monocytes, and eosinophils. These findings are consistent with the pathophysiology of CML, where the BCR-ABL1 fusion gene drives uncontrolled proliferation of myeloid cells and suppresses normal erythropoiesis.

Importantly, no gender-based differences were observed, suggesting that the hematological impact of CML is largely similar in males and females. The weak and statistically insignificant correlations between hemoglobin concentration and white blood cell parameters indicate that while anemia and leukocytosis commonly coexist, they do not necessarily predict each other's severity.

In comparison with studies from other regions, our findings reinforce the global patterns of hematological abnormalities in CML while adding context-specific data for Nigerian patients. This emphasizes the continued relevance of complete blood counts in the diagnosis and monitoring of CML, particularly in resource-limited settings where molecular testing may be inaccessible.

Future research should focus on integrating hematological monitoring with molecular diagnostics to better understand disease progression and treatment outcomes among Nigerian CML patients. Additionally, larger multicenter studies will help validate these findings and provide a more comprehensive picture of the hematological profile of CML across different populations in sub-Saharan Africa.

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Studies on Some Haemostatic Parameters in Patients with Myasthenia Gravis Attending Imo State Teaching Hospital, Orlu, Nigeria

¹Uzodinma Janet A., ²Okoroiwu L. I., ³Aloy-Amadi Oluchi C., ⁴Uzodinma Emmanuel E., ⁵Anokwute Michael U., ⁶Okpara Lydia T.

¹Senior Medical Laboratory Scientist, ²Professor, ³Senior Lecturer Department of Medical Laboratory Science, Imo State University, Owerri, Nigeria, ⁴Associate Professor Department of Psychology, University of Agriculture and Environmental Sciences, Owerri, Nigeria, ⁵Director Department of Haematology, Nnamdi Azikiwe University Teaching Hospital, Nnewi, Nigeria, ⁶Lecturer Whitetouch Diagnostic Centre, Lagos, Nigeria

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Abstract

Background: Myasthenia gravis (MG) is a chronic autoimmune neuromuscular disorder characterized by fluctuating muscle weakness and fatigue, which can lead to severe complications such as myasthenic crisis. This study aimed to evaluate specific haemostatic parameters in MG patients attending the Neurology Clinic at Imo State Teaching Hospital, Orlu, Nigeria.

Methods: A cross-sectional comparative study was conducted involving 50 participants: 25 diagnosed MG patients and 25 age- and gender-matched healthy controls. After obtaining informed consent, fasting blood samples were collected and analyzed for prothrombin time (PT), activated partial thromboplastin time (APTT), and fibrinogen levels using the Sysmex CA-50 coagulation analyzer. Data were analyzed using SPSS version 27, with significance set at $p < 0.05$.

Results: MG patients showed significantly higher mean values of PT (15.30 ± 1.95 s), APTT (35.60 ± 3.74 s), and fibrinogen (442.52 ± 95.45 mg/dL) compared to controls ($p < 0.0001$). No significant differences in these parameters were found when stratified by gender or age.

Conclusion: The study demonstrates that MG is associated with elevated haemostatic markers such as fibrinogen, PT, and APTT, indicating possible subclinical coagulation disturbances. Age and gender had no significant effect on these parameters in MG patients. Further research on inflammatory markers may improve management and prognosis in MG.

Keywords: Myasthenia gravis, haemostasis, fibrinogen, prothrombin time, APTT, autoimmune disorders.

Introduction

Myasthenia gravis (MG) is a rare but well-characterized autoimmune disorder that affects

the neuromuscular junction, resulting in impaired transmission of nerve impulses to skeletal muscles. The disease is marked by fluctuating muscle weakness that worsens with activity and improves

Corresponding Author: Uzodinma Janet A. Department of Medical Laboratory Science, Imo State University, Owerri, Nigeria.

E-mail: okparajanet8@gmail.com

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with rest. It predominantly affects voluntary muscles such as those controlling the eyes, mouth, throat, and limbs¹.

Pathophysiologically, MG results from autoantibodies targeting acetylcholine receptors (AChRs) or associated proteins such as muscle-specific tyrosine kinase (MuSK), leading to compromised synaptic transmission². Though traditionally considered a neurological condition, MG also triggers systemic immune responses that may affect haematological and haemostatic parameters³. Dysregulation in coagulation markers such as fibrinogen, PT, and APTT have been documented in autoimmune and inflammatory disorders⁴.

Existing literature suggests that chronic inflammation, common in autoimmune diseases, can disrupt coagulation by activating endothelial cells and platelets, increasing fibrinogen production, and altering clotting times⁵. Elevated fibrinogen has been linked to both acute and chronic inflammatory states, while prolonged PT and APTT may suggest liver dysfunction or the presence of lupus anticoagulant, both possible in autoimmune conditions like MG⁶.

Previous studies such as those by Giannoccaro et al.⁷ and Punga et al.⁸ have highlighted abnormalities in various haematological parameters in MG patients, including anemia and changes in platelet function. However, limited data exist on haemostatic profiles in African populations with MG. This study addresses that gap by evaluating coagulation parameters in MG patients attending a tertiary hospital in South-Eastern Nigeria.

Materials and Methods

Study Area

The research was conducted at the Imo State University Teaching Hospital (IMSUTH), Orlu, a tertiary healthcare facility in South-Eastern Nigeria. The hospital provides specialized care, including a dedicated neurology unit for autoimmune and neuromuscular disorders.

Study Design

A descriptive cross-sectional study was employed to assess and compare haemostatic parameters between diagnosed MG patients and healthy controls.

Study Population

The study population consisted of adults (aged 20–75 years) attending the neurology clinic at IMSUTH. Participants were grouped into:

Test group: 25 clinically diagnosed MG patients

Control group: 25 age- and sex-matched healthy individuals with no history of autoimmune or bleeding disorders.

Method of Recruitment

Participants were selected using convenience sampling. A structured questionnaire was administered after obtaining informed consent. Demographic information, medical history, lifestyle factors, and exclusion criteria (e.g., recent infections, known coagulopathies, or anticoagulant therapy) were documented.

Ethical Consideration

Approval for this study was obtained from the Institutional Research Ethics Committee of IMSUTH. All participants gave written informed consent in line with the Declaration of Helsinki.

Sample Collection

Five milliliters of fasting venous blood were collected aseptically into trisodium citrate tubes. Samples were transported immediately to the hospital laboratory for analysis.

Laboratory Analysis

Haemostatic parameters including: Prothrombin Time (PT), Activated Partial Thromboplastin Time (APTT), Fibrinogen concentration were measured