

Packed Cell Volume, Red Blood Cell Count and Erythrocyte Indices in Adults with Chronic Myeloid Leukaemia in Owerri Nigeria

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Abstract

Background: Chronic myeloid leukaemia (CML) is a BCR::ABL1-positive myeloproliferative neoplasm in which clonal myeloid expansion and its treatment may disturb normal erythropoiesis. Routine erythrocyte measurements are widely available, but their pattern among patients receiving care in south-eastern Nigeria has not been adequately described. **Objective:** To compare packed cell volume (PCV), red blood cell (RBC) count, mean cell volume (MCV), mean corpuscular haemoglobin (MCH), mean corpuscular haemoglobin concentration (MCHC) and red cell distribution width (RDW) between adults with CML and apparently healthy controls in Owerri, and to examine variation by age and sex among the patients. **Methods:** This comparative cross-sectional study recruited 30 adults with CML and 30 age-matched apparently healthy controls at Imo State Specialist Hospital, Owerri, from February to April 2024. Five milliliters of venous blood was collected into ethylenediaminetetraacetic acid tubes. The haematological parameters were measured using a Mindray BC-5180 haematology analyser. Data were summarised as mean $\pm$ standard deviation and compared using independent-samples t tests or one-way analysis of variance, with statistical significance set at $p < 0.05$. **Results:** Participants with CML had lower PCV ($24.60 \pm 4.50\%$ versus $37.50 \pm 3.22\%$; $p < 0.001$) and RBC count (2.54 ± 0.43 versus $3.82 \pm 0.64 \times 10^{12}/L$; $p < 0.001$) than controls. MCV (86.13 ± 10.56 versus 73.10 ± 16.00 fL), MCH (29.73 ± 2.88 versus 25.77 ± 4.49 pg) and RDW (14.70 ± 2.74 versus $12.10 \pm 1.58\%$) were higher in the CML group (all $p < 0.001$), whereas MCHC did not differ (34.47 ± 1.74 versus 34.37 ± 2.75 g/dL; $p = 0.884$). No sex-related difference was detected among patients. MCV varied across age groups ($p = 0.024$), but the other indices did not. **Conclusion:** Adults with CML in this setting showed a marked reduction in red cell mass accompanied by greater erythrocyte size and size heterogeneity. Routine interpretation of the complete blood count should therefore extend beyond haemoglobin or PCV alone. Because disease phase, treatment exposure, nutritional status and molecular

response were not measured, the findings are descriptive and require confirmation in larger longitudinal studies.

Keywords: *Anaemia, BCR::ABL1, chronic myeloid leukaemia, erythrocyte indices, packed cell volume, red cell distribution width, Nigeria*

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1 Introduction

Chronic myeloid leukaemia is a clonal myeloproliferative neoplasm defined by the Philadelphia chromosome or its molecular counterpart, the BCR::ABL1 fusion gene. The resulting constitutively active tyrosine kinase promotes survival and proliferation of the malignant haematopoietic clone, producing the characteristic expansion of granulocytic cells and their precursors. CML represents about 15% of newly diagnosed adult leukaemias and typically occurs at an incidence of one to two cases per 100,000 adults each year, although the measured burden varies with population structure, diagnostic access and cancer registration.¹ The disease usually begins in chronic phase and may progress through accelerated phase to blast phase when treatment is unavailable or ineffective.^{1,2}

The introduction of BCR::ABL1 tyrosine kinase inhibitors transformed CML from a frequently fatal disease into a chronic condition for many patients. Contemporary care depends on confirmation of BCR::ABL1, selection of an appropriate tyrosine kinase inhibitor and serial molecular monitoring on the International Scale.¹⁻³ Despite this therapeutic progress, the complete blood count remains central to diagnosis, early assessment and day-to-day clinical review. It provides immediate information about leucocytosis, basophilia, thrombocytosis or thrombocytopenia and anaemia, and it may reveal

treatment-related marrow suppression before a molecular result becomes available.^{1,2} In settings where molecular testing is intermittent because of cost or access, careful use of routine haematological measurements becomes even more important.

Anaemia is common in malignant and chronic inflammatory disorders, but its mechanisms are rarely singular. In CML, reduced red cell mass may reflect replacement or functional suppression of normal marrow erythropoiesis by the malignant clone, inflammatory restriction of iron availability, shortened erythrocyte survival, hypersplenism, nutritional deficiency, bleeding, renal dysfunction or adverse effects of therapy. Disease phase and tumour burden may also influence the severity of anaemia. Nigerian cohorts have described frequent anaemia and marked leucocytosis at presentation, while also demonstrating substantial clinical heterogeneity.⁴⁻⁶ A low PCV or RBC count therefore identifies an important abnormality but does not, by itself, establish the mechanism.

Erythrocyte indices help to characterise that abnormality. MCV describes average red cell volume, MCH estimates the average mass of haemoglobin per erythrocyte, and MCHC expresses haemoglobin concentration within the packed red cell volume. RDW quantifies heterogeneity of erythrocyte size and therefore provides information that can be missed when an average value such as MCV is considered alone. A patient may have an

apparently normal mean cell volume while simultaneously carrying microcytic and macrocytic populations, producing a high RDW. Interpretation of the indices together can support a more focused assessment of iron deficiency, vitamin B12 or folate deficiency, haemolysis, marrow stress and mixed anaemia.^{7,8}

RDW has also attracted interest as a marker of biological stress beyond the morphological classification of anaemia. Oxidative stress, inflammation, disordered iron handling and ineffective erythropoiesis can broaden the circulating red cell size distribution. Across haematological malignancies, higher RDW has been associated with adverse outcomes, although its prognostic meaning depends on the underlying disease and study design.⁹ In chronic-phase CML treated with tyrosine kinase inhibitors, Iriyama and colleagues reported poorer event-free and transformation-free survival among patients with higher pretreatment RDW.¹⁰ Subsequent CML studies likewise found that increased RDW was associated with less favourable clinical characteristics or outcomes.^{11,12} These associations do not make RDW a disease-specific marker, but they support examining it as part of the routinely generated blood count.

MCV and MCH require equally cautious interpretation in CML. Macrocytosis may arise from reticulocytosis, folate or vitamin B12 deficiency, hepatic disease, alcohol exposure, hypothyroidism or drug effects; a higher group mean cannot automatically be attributed to the leukaemic process. Some tyrosine kinase inhibitors can alter erythropoiesis, and treatment duration may

influence the observed indices. Conversely, iron deficiency or chronic blood loss may lower MCV and MCH. Consequently, any cross-sectional comparison must be understood as a description of the combined effects of disease, treatment and patient-level factors unless these influences are measured directly.^{7,8}

Evidence from Nigeria remains valuable because the timing of diagnosis, access to molecular monitoring, nutritional exposures and treatment continuity may differ from those in high-income settings. Earlier Nigerian reports have focused mainly on presenting symptoms, total white cell count, platelet count, splenomegaly, cytogenetics and survival.⁴⁻⁶ Fewer reports have presented PCV, RBC count, MCV, MCH, MCHC and RDW together or explored their distribution by age and sex. Local data may therefore help laboratory professionals and clinicians recognise the erythrocyte pattern seen in routine practice and identify questions that deserve prospective investigation.

The present study compared PCV, RBC count and red cell indices between adults diagnosed with CML and apparently healthy controls in Owerri, Nigeria. It also examined whether these measurements differed by age group or sex among the participants with CML. The working null hypotheses were that the mean erythrocyte measurements would not differ between CML and control groups and would not vary by age or sex within the CML group.

2 Materials and Methods

2.1 Study design and setting

A comparative cross-sectional study was conducted

from February to April 2024 at Imo State Specialist Hospital, Umuguma, Owerri, Imo State, south-eastern Nigeria. The hospital provides secondary-level clinical services and receives patients from Owerri and surrounding communities. The design allowed erythrocyte measurements obtained from adults with an established diagnosis of CML to be compared with those of apparently healthy individuals during the same study period.

2.2 Study population

Sixty participants were enrolled: 30 adults with CML attending the medical outpatient clinic and 30 age-matched apparently healthy controls. The CML group comprised 13 males and 17 females. The source document reported an identical sex distribution for the control group. Participants were adults, and the age categories used for the within-CML analysis were 33-43, 44-54, 55-65 and at least 66 years. Each potential participant received information about the study, provided written informed consent and completed a structured questionnaire before specimen collection.

2.3 Eligibility criteria

Eligible cases were adults with a diagnosis of CML who consented to participate. Apparently healthy age-matched adults were enrolled as controls. The study excluded people who did not consent and those reported to have tested positive for human immunodeficiency virus, hepatitis B surface antigen, hepatitis C virus or syphilis.

2.4 Sample size

We calculated a minimum sample using the single-proportion expression $n = Z^2 p(1-p)/d^2$, with a stated prevalence estimate of 1.35%, 95% confidence and 5% precision, and then recruited 30 cases. Thirty

controls were added, giving a total sample of 60.

2.5 Ethical considerations

Approval was obtained from the relevant authority and ethics committee of Imo State Specialist Hospital after submission of the research proposal and an introductory letter from the Department of Medical Laboratory Science, Imo State University. Recruitment and specimen collection were arranged with clinic staff. Participation was voluntary, written informed consent was obtained, and study information was handled confidentially.

2.6 Specimen collection and laboratory analysis

Five millilitres of venous blood were collected aseptically from an antecubital vein into an ethylenediaminetetraacetic acid tube, labelled with a study identifier and collection date, and mixed appropriately. The blood dispensed into the EDTA container was used for the estimation of haematological parameters.

2.7 Statistical analysis

Data were analysed using IBM SPSS Statistics version 27. Continuous variables were reported as mean±standard deviation. Independent-samples Student's t tests were used for comparisons between CML and control groups and between males and females with CML.

3 Results

3.1 Comparison of participants with CML and controls

The CML group had substantially lower PCV and RBC count than the control group. Mean PCV was $24.60 \pm 4.50\%$ in participants with CML and $37.50 \pm 3.22\%$ in controls ($t=14.73$, $p<0.001$). Mean RBC count was $2.54 \pm 0.43 \times 10^{12}/L$ in the CML group compared with $3.82 \pm 0.64 \times 10^{12}/L$ in controls

($t=9.73$, $p<0.001$). The direction and magnitude of these differences indicate a pronounced reduction in circulating red cell mass among the patients.

MCV, MCH and RDW were higher in the CML group. Mean MCV was 86.13 ± 10.56 fL among patients and 73.10 ± 16.00 fL among controls ($t=4.05$, $p<0.001$). Mean MCH was 29.73 ± 2.88 pg and 25.77 ± 4.49 pg, respectively ($t=4.73$, $p<0.001$), while RDW was $14.70\pm2.74\%$ and $12.10\pm1.58\%$, respectively ($t=4.59$, $p<0.001$). MCHC was similar in both groups (34.47 ± 1.74 versus 34.37 ± 2.75 g/dL; $t=0.147$, $p=0.884$).

Parameter	CML n=30	Control n=30	Test statistic	p value
PCV (%)	24.60±4.50	37.50±3.22	14.73	<0.001*
RBC (×10 ¹² /L)	2.54±0.43	3.82±0.64	9.73	<0.001*
MCV (fL)	86.13±10.56	73.10±16.00	4.05	<0.001*
MCH (pg)	29.73±2.88	25.77±4.49	4.73	<0.001*
MCHC (g/dL)	34.47±1.74	34.37±2.75	0.147	0.884
RDW (%)	14.70±2.74	12.10±1.58	4.59	<0.001*

Table 1. Erythrocyte measurements in participants with CML and controls. Values are mean±SD. *Statistically significant at $p<0.05$.

3.2 Variation by age among participants with CML

Across the four age categories, MCV declined from 95.10 ± 9.89 fL in those aged 33-43 years to 88.50 ± 7.47 fL at 44-54 years, 79.80 ± 9.68 fL at 55-65 years and 82.00 ± 14.11 fL among those aged at least 66 years. This variation was statistically significant ($F=3.724$, $p=0.024$).

Parameter	33-43 n=6	44-54 n=9	55-65 n=12	≥66 n=3	F	p value
PCV (%)	21.67±4.90	25.70±4.99	24.90±3.84	26.67±2.52	1.30†	0.293
RBC (×10 ¹² /L)	2.35±0.45	2.60±0.49	2.55±0.46	2.67±0.15	0.49	0.682
MCV (fL)	95.10±9.89	88.50±7.47	79.80±9.68	82.00±14.11	3.724	0.024*
MCH (pg)	31.17±3.76	30.00±1.49	28.80±3.05	27.67±2.52	1.511	0.236

Parameter	33-43 n=6	44-54 n=9	55-65 n=12	≥66 n=3	F	p value
MCHC (g/dL)	33.30±1.63	34.50±2.01	35.10±1.37	34.67±2.08	1.309	0.294
RDW (%)	13.17±3.25	15.30±3.23	15.00±1.94	13.67±1.52	0.962	0.426

Table 2. Erythrocyte measurements by age group among participants with CML. Values are mean±SD. * $p<0.05$. †The source table printed 7.30 for PCV, but this is inconsistent with $p=0.293$ and the displayed means; the statistic requires verification against the original SPSS output before submission.

3.3 Variation by sex among participants with CML

None of the measured variables differed significantly between males and females with CML. PCV and RBC count were almost identical across sex groups. Males had a slightly higher mean MCV, whereas females had slightly higher MCH, MCHC and RDW, but each difference was small relative to its within-group variation.

Parameter	Male n=13	Female n=17	t	p value
PCV (%)	24.62±5.20	24.59±3.95	0.016	0.987
RBC (×10 ¹² /L)	2.59±0.56	2.51±0.32	0.450	0.656
MCV (fL)	87.31±12.66	85.29±8.87	0.513	0.612
MCH (pg)	29.69±3.17	30.12±3.02	0.374	0.711
MCHC (g/dL)	34.39±1.85	34.53±1.69	0.223	0.825
RDW (%)	14.31±2.32	15.00±3.05	0.673	0.503

Table 3. Erythrocyte measurements by sex among participants with CML. Values are mean±SD.

4 Discussion

The principal finding was a coherent pattern of reduced red cell mass and altered erythrocyte morphology among adults with CML. PCV and RBC count were markedly lower than in apparently healthy controls, while MCV, MCH and RDW were

higher. MCHC did not differ. This pattern suggests that anaemia in the CML group was accompanied by larger and more heterogeneous erythrocytes, rather than a uniform reduction in the haemoglobin concentration of individual cells. The study was not designed to determine causation, and the absence of disease-phase, treatment and nutritional data means that the pattern should not be attributed to CML biology alone.

The reduction in PCV from 37.50% in controls to 24.60% in the CML group is clinically substantial. A parallel decrease in RBC count strengthens the interpretation that the patients had a genuine reduction in circulating red cell mass rather than an isolated analytical change. Anaemia is a recognised feature of CML, particularly with increasing disease burden, marrow dysfunction or advanced phase.^{1,2} Nigerian studies have also described anaemia among patients presenting with CML, although the reported frequency and severity vary across centres and eras.⁴⁻⁶ Differences in referral patterns, diagnostic delay, treatment exposure and case mix can readily account for some of that variation.

Several mechanisms could have contributed to the low PCV and RBC count. Expansion of the malignant myeloid clone may impair effective erythropoiesis by altering marrow architecture and access to supportive stromal signals. Inflammatory cytokines can restrict iron availability and blunt the erythropoietin response. Splenomegaly may increase sequestration or destruction of circulating cells, while bleeding, infection, renal disease and nutritional deficiencies may add further effects. Tyrosine kinase inhibitors can also cause cytopenias, particularly early in treatment or in

patients with limited normal marrow reserve.¹⁻³ Without reticulocyte count, iron studies, vitamin B12, folate, renal indices and treatment history, the present data cannot separate these possibilities.

Mean MCV was higher in participants with CML than in controls. However, the mean value in the CML group, 86.13 fL, remained within the usual adult reference interval in many laboratories. The statistical difference should therefore not be described automatically as frank macrocytosis in every patient. Rather, it indicates a rightward shift in average red cell volume at group level. The unusually low and variable control mean, 73.10 ± 16.00 fL, also deserves attention because it may reflect a high background burden of microcytosis, unrecognised iron deficiency, haemoglobinopathy or pre-analytical variation among individuals considered healthy. Screening controls only by apparent health may not exclude these conditions.

The age-stratified analysis adds another caution. MCV was highest in the youngest CML category and generally lower in older groups, with an overall p value of 0.024. This direction is not the usual simple effect expected from ageing and may instead reflect uneven distribution of treatment, disease phase, nutritional status or other unmeasured factors. The oldest group contained only three participants, and the four groups were unequal. A single omnibus p value in this context does not identify which groups differ, and no post hoc comparisons were reported. The result is therefore hypothesis-generating rather than evidence of a stable age effect.

MCH was also higher in the CML group. Because

MCH is mathematically and biologically related to cell volume, larger cells generally contain more total haemoglobin even when their intracellular haemoglobin concentration is unchanged. The absence of a group difference in MCHC is consistent with this explanation: the erythrocytes may have contained more haemoglobin per cell primarily because they were larger, while the concentration of haemoglobin within the cells remained comparable. This linked interpretation is more plausible than treating MCV, MCH and MCHC as independent processes.

RDW was higher among the patients, indicating greater anisocytosis. RDW can rise when erythropoiesis produces cells of differing sizes, when a reticulocyte response introduces larger young cells, when nutritional deficiencies coexist, or when transfused and native erythrocyte populations circulate together. Inflammation and oxidative stress may further disturb erythrocyte maturation and survival.⁹ Because the mean RDW in the CML group approached common upper reference limits and its standard deviation was wide, the group likely included patients with quite different erythropoietic states.

The RDW finding is consistent with published CML literature. Iriyama et al. found that higher pretreatment RDW identified chronic-phase patients with poorer event-free and transformation-free survival during tyrosine kinase inhibitor therapy.¹⁰ Li et al. also reported higher RDW as an adverse prognostic factor, while Mao et al. described associations between RDW and clinical response variables.^{11,12} A broader meta-analysis concluded that elevated RDW has prognostic

relevance across several haematological malignancies.⁹ Nevertheless, the present study measured group differences at one time point and collected no outcome data. It therefore supports the descriptive relevance of RDW but does not establish a prognostic threshold for patients in Owerri.

The lack of a sex difference should be interpreted in light of sample size. Although physiological haemoglobin and PCV reference intervals often differ between adult men and women, the disease-associated reduction in red cell mass may have outweighed the smaller sex-related contrast in this cohort. In addition, only 13 male and 17 female patients were analysed. The non-significant results do not prove equivalence; they show that the study did not detect a difference of the observed magnitude. A larger study could estimate sex-specific effects with narrower confidence intervals and adjust for menopausal status, bleeding, nutritional variables and treatment.

The findings reinforce the value of reading the complete erythrocyte profile rather than relying on PCV alone. A low PCV in a patient with CML should prompt review of RBC count, MCV, MCH, MCHC, RDW and, where available, reticulocyte measures and the peripheral blood film. A high RDW with an average MCV may signal mixed deficiency or a recovering marrow. A higher MCV should prompt review of reticulocytosis, vitamin B12 and folate status, liver and thyroid function, alcohol exposure and relevant medicines. These tests do not replace BCR::ABL1 monitoring, which remains fundamental to assessment of CML response, but they can identify concurrent problems

that are clinically actionable.¹⁻³

The study also highlights the importance of analytical quality. PCV was measured manually, whereas RBC count and RDW came from an automated analyser and calculated indices combined values from different methods. Agreement depends on proper mixing, timely analysis, centrifuge performance, capillary sealing, analyser calibration and internal quality control. Delayed analysis of EDTA blood may cause cell swelling and alter MCV or RDW. Future studies should specify the time from venepuncture to testing, record two or three levels of daily control material, participate in external quality assessment and use a single validated automated platform where practicable.^{7,8}

A further issue is the interpretation of the control group. Age matching was reported, and the sex distribution was identical, which improves comparability. However, apparently healthy volunteers were not characterised with ferritin, C-reactive protein, haemoglobin electrophoresis, renal indices or parasitological testing. In south-eastern Nigeria, asymptomatic iron deficiency, inherited haemoglobin traits and low-grade infection may affect red cell indices. The low mean control MCV makes this more than a theoretical concern. Better phenotyping of controls would reduce the risk that the observed differences reflect unmeasured conditions on either side of the comparison.

From a research perspective, a longitudinal design would be more informative than another small cross-sectional comparison. Newly diagnosed, treatment-naïve participants could be assessed before therapy and at predefined molecular

monitoring points. Disease phase, spleen size, BCR::ABL1 transcript level, ELTS risk, treatment, adherence, transfusion, reticulocyte count, blood film, ferritin, transferrin saturation, vitamin B12, folate, bilirubin, lactate dehydrogenase, haptoglobin and renal function could be recorded. Such data would clarify whether changes in RDW or MCV track molecular response, treatment toxicity, nutritional correction or disease progression.^{2,3}

Overall, the internal pattern of the main comparison is biologically coherent: lower PCV and RBC count demonstrate anaemia, higher RDW demonstrates greater size heterogeneity, and higher MCV and MCH with stable MCHC describe relatively larger cells without a meaningful difference in intracellular haemoglobin concentration. The results are useful as a local baseline..

5 Conclusion

Adults with CML attending Imo State Specialist Hospital, Owerri, had substantially lower PCV and RBC count than apparently healthy controls, together with higher MCV, MCH and RDW. MCHC was similar between groups. No sex-related difference was detected, while MCV varied across age categories in an exploratory analysis. The findings support comprehensive interpretation of routine erythrocyte indices in CML, particularly because a mean value alone may conceal substantial size heterogeneity. They do not establish that RDW or MCV is independently prognostic. Larger prospective studies incorporating disease phase, treatment exposure, molecular response, nutritional status and laboratory quality-control data are required.

6 Recommendations

1.Clinicians should review the complete erythrocyte profile and peripheral blood film when assessing anaemia in CML rather than relying on PCV or haemoglobin alone.

2.Patients with high RDW or increased MCV should be evaluated for reversible causes, including iron, vitamin B12 or folate deficiency, haemolysis, bleeding, renal or hepatic dysfunction, thyroid

disease, alcohol exposure and treatment effects.

3.Future local studies should enrol larger, treatment-defined cohorts and relate serial erythrocyte indices to BCR::ABL1 molecular response and patient outcomes.

Declarations

Competing interests: No competing interest.

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