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Original Article

Micronuclei Frequency and White Blood Cell Functional Indices among Patients with Leukaemia and Multiple Myeloma Attending University of Ilorin Teaching Hospital, Ilorin

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Abstract

Background: Leukaemia represents a heterogeneous group of blood cancers frequently linked to immune dysfunction and genomic instability.

Objective: This study evaluated micronuclei frequency and white blood cell functional indices among leukaemic patients attending the University of Ilorin Teaching Hospital, Ilorin, Kwara State, Nigeria**.**

Materials and Methods: An analytical case–control design was employed involving consented 43 leukaemic subjects and age-matched healthy controls recruited. Venous blood samples were analyzed for White blood cell indices using an automated haematology analyzer, and Micronuclei Frequency using the cytokinesis-block method (CBMN).

Results: White blood cell functional indices (Relative lymphocyte count) were significantly elevated in patients across all types of cancer, AML (44.50 ± 1.29) compared to control (33.83 ± 6.34), ALL (35.75 ± 1.26) Control (33.83 ± 6.34), CLL (68.38 ± 1.77) Control (33.83 ± 6.34) and Multiple Myeloma (38.00 ± 14.77) Control (33.83 ± 6.34). Micronuclei frequency parameters and proliferation index were significantly higher in ALC and ANC relative to control in all participants ($p < 0.01$).

Conclusion: Leukaemia is associated with increased genomic instability and altered immune response. Micronuclei frequency and white blood cell functional indices may serve as useful adjuncts to leukaemic cells in evaluating disease progression and prognostic potential.

Keywords: 'Leukaemia', 'Micronuclei Frequency', and 'White blood cell indices'

Introduction

Micronuclei (MN) are small extranuclear bodies containing whole chromosomes or chromosome fragments that fail to segregate into daughter nuclei during mitotic cell division (Sommer *et al.,* 2020; Zhang *et al.,* 2020). They represent a direct cytological readout of chromosomal instability, DNA damage, and whole-chromosome loss (Fenech, 2020; Di and Bakhoum, 2024). Increased micronuclei frequency is widely recognized as a validated biomarker of genetic damage and has been linked to cancer susceptibility, disease progression, treatment response, and overall prognosis (Almeida *et al.,* 2022; Rowland *et al.,* 2024).

Leukaemia and Multiple myeloma encompass a broad spectrum of malignancies originating from blood-forming tissues in the bone marrow, including leukemias and multiple myeloma (Santos *et al.,* 2023; Mulatie *et al.,* 2025). The etiology of these malignancies is multifactorial, involving genetic mutations (such as chromosomal translocations, point mutations, and epigenetic alterations), environmental exposures (ionizing radiation, benzene, cytotoxic agents), and viral infections (Coyle *et al.,* 2017; Kipps *et al.,* 2017; Pedersen *et al.,* 2018). In Nigeria, Leukaemia and Multiple myelomaaccount for approximately 2% to 10% of all diagnosed cancers (Abudu *et al.,* 2018). Hospital-based reviews at the University of Ilorin Teaching Hospital (UITH) indicate that Chronic Lymphocytic Leukemia accounts for (20-25%), Acute Leukemias (15-20%) and Multiple Myeloma (10-12%) (Akinbami *et al.,* 2014; Anifowoshe *et al.,* 2018; Babatunde *et al.,* 2020).

Although standard peripheral blood investigations such as Complete Blood Count (CBC) and differential counts reveal quantitative abnormalities (leukocytosis, lymphocytosis, neutropenia), they do not reflect underlying genomic instability or DNA damage dynamics (Kantarjian *et al.,* 2015; Terwilliger and Abdul-Hay, 2017). Evaluating micronuclei frequency alongside white cell functional indices (neutrophils, lymphocytes, monocytes) provides critical insights into cytogenetic integrity and immune status (Tan *et al.,* 2018; Fenech and Bonassi, 2019). Therefore, this study assessed white cell functional indices and micronuclei frequency in patients with Leukaemia and Multiple myeloma at the University of Ilorin Teaching Hospital, Kwara State, Nigeria.

Materials And Methods

Study Site: The study was conducted at the University of Ilorin Teaching Hospital (UITH), Oke-Oyi, Kwara State. Blood samples were equally collected at the Haematology Clinic of UITH. Laboratory culture, cytogenetic preparation, micronuclei scoring, and cellular proliferation assays were performed in the Department of Medical Laboratory Science Research Laboratories, Al-Hikmah University, Ilorin, Kwara State, Nigeria (Akinbami *et al.,* 2014).

Study Design

This study was a comparative case-control study that was carried on leukaemia and Multiple myeloma patients who met the inclusion criteria at the University of Ilorin Teaching Hospital, Ilorin.

Study Population and Selection Criteria

The study population comprised of confirmed patients presenting with various Leukaemia and Multiple myeloma (AML, ALL, CLL, and MM) attending the UITH Haematology Clinic. The controls were Age- and sex-matched apparently healthy individuals recruited from voluntary blood donors and hospital staff who gave consent were recruited.

Sample Size Determination

Sample size was calculated using the Cochrane formula $n = \frac{z^2 p (1-p)}{d^2}$

where: n = sample size, z = the standard deviation at a 95% confidence interval (1.96), P = proportion of the population with the desired factor (adopted from Haemtolyphoid neoplasm) in Ilorin = 2.9% = 0.029 as reported by (Abudu *et al.,* 2018), q = 1-p (0.971), while d is the maximum allowable error (5%). Thus, a total of 43 estimated participants were recruited for this study, 22 of which were leukaemia and Multiple myeloma, while 21 were lymphoma Patients. A corresponding group of healthy controls was recruited for comparative evaluation.

Data Collection tools

Data were collected using well-structured, self-administered questionnaires which were specifically designed to obtain information that was used to either include or exclude individuals from this study. A questionnaire was given to the participants through an interviewer-assisted administration method, which served as the major source of primary data for this study. The participants were informed of the study's goals and objectives. The consent forms were submitted with the questionnaire, and informed consent from the participants was sought. The gathered data were kept on a safe, password-protected I-drive for future reference.

Sample collection

From each participant, 7 mL of peripheral venous blood was collected aseptically via venipuncture. Three milliliters (3 mL) were dispensed into Ethylenediaminetetraacetic acid (EDTA) anticoagulant tubes for automated full blood count and leukocyte differential analysis. Four milliliters (4 mL) were collected in lithium heparinized tubes for leukocyte isolation, lymphocyte culture, and cytokinesis-block micronucleus assay.

Laboratory Procedures and Protocols

Full Blood Count and Differential Indices

Full blood counts and differential white cell parameters were performed within 2 hours of sample collection using a 5-part automated hematology analyzer (Mindray BC-5300) operating on electrical impedance, flow cytometry, and laser light scatter principles. Relative neutrophil count (RNC %), relative lymphocyte count (RLC %), relative monocyte count (RMC %), absolute neutrophil count (ANC x10^9/L), absolute lymphocyte count (ALC x10^9/L), and absolute monocyte count (AMC x10^9/L) were recorded. Peripheral blood smears were stained with Leishman stain and evaluated under oil immersion (x100 objective) for morphological verification.

Cytokinesis-Block Micronucleus (CBMN)

The CBMN assay was conducted according to the standard protocol defined by (Fenech *et al.,* 2020). with slight modifications:
Leukocyte Isolation: Equal volumes (1.5 mL) of fresh whole blood and Hank's Balanced Salt Solution (HBSS) were layered over 1.0 mL of Ficoll-Paque density gradient media and centrifuged at 2000 rpm for 30 minutes at room temperature. The leukocyte layer was aspirated, washed twice in HBSS, and resuspended in RPMI 1640 culture medium.
Culture and Cytokinesis Block: Isolated lymphocytes were cultured in RPMI 1640 supplemented with 10% Fetal Bovine Serum (FBS), 2 mM L-glutamine, 1 mM sodium pyruvate and stimulated with Phytohemagglutinin (PHA, 2.25 mg/mL). Cultures were incubated at 37°C in a humidified 5% CO2 incubator for 44

hours. At 44 hours, Cytochalasin-B (6 ug/mL) was added to block cytokinesis and arrest dividing cells at the binucleated stage, followed by an additional incubation of 28 hours (total culture duration = 72 hours).

Harvesting and Slide Preparation: Cultured cells were harvested by gentle centrifugation, fixed with cold methanol, and smeared onto clean, grease-free glass slides. Slides were air-dried, stained with Giemsa stain, and cover slipped with DPX. Scoring was conducted under high-power microscopy (x1000 magnification). Cytome endpoints mononucleated cells, binucleated (BN) cells, acentric fragments/micronuclei per 1000 cells, nucleoplasmic bridges, and nuclear buds were scored according to strict international criteria (Fenech *et al.,* 2020).

Spectrophotometric Cellular Proliferation Assay

Cellular proliferation was measured using a modified spectrophotometric approach (Akinbami *et al.,* 2014). Following 28 hours of cytochalasin-B incubation, cell culture turbidity and optical density (OD) were measured at 550 nm wavelength using a calibrated spectrophotometer to quantify cellular proliferation capacity.

Statistical Analysis

Data were entered, cleaned, and analyzed using IBM SPSS Statistics for Windows, Version 27.0 (IBM Corp., Armonk, NY). Descriptive statistics were expressed as frequencies and percentages for categorical variables, and as Mean ± Standard Deviation (SD) for continuous parameters. Independent sample t-tests and Analysis of Variance (ANOVA) with post-hoc tests were used to evaluate statistical differences between patient subgroups and healthy controls. Pearson correlation analysis was conducted to establish bivariate relationships between white cell functional indices and micronuclei cytome endpoints. Statistical significance was set at $p < 0.05$.

Ethical Considerations

Ethical approval for the study was granted by the Ethical Review Committee of the University of Ilorin Teaching Hospital (Approval Protocol Number: ERC PAN/2025/07/0618). Written informed consent was obtained from all adult participants or guardians of pediatric participants prior to recruitment. All procedures strictly adhered to the ethical standards outlined in the Declaration of Helsinki. Confidentiality and data security were strictly maintained.

Results

Table 1 shows the socio-demographic characteristics of the participants. A total of 43 participants with Leukaemia and Multiple myelomawere evaluated. As detailed in Table 1, the majority of the participants belonged to the 50–69 years age bracket (n = 24, 55.8%), followed by those under 30 years (n = 9, 20.9%), 30–49 years (n = 5, 11.6%), and >=70 years (n = 5, 11.6%). Females represented 53.5% (n = 23) of the cohort, while males constituted 46.5% (n = 20). Most participants were married (79.1%), engaged in business/trading (67.4%). All participants were undergoing active systemic chemotherapy regimens. Hypertension was present as a co-morbidity in 9.3% (n = 4) of patients. More than half of the study population (n = 24, 55.8%) had a history of prior blood transfusions.

Fig 1 shows the classification of haematological malignancy in the study population. Chronic Lymphocytic Leukemia (CLL) was the most prevalent malignancy (n = 8, 18.6%), followed by Multiple Myeloma (MM, n = 6, 14.0%) Acute Myeloid Leukemia (AML, n = 4, 9.3%). Acute Lymphoblastic Leukemia (ALL, n = 4, 9.3%).

Table 1: Socio-demographic and Clinical Characteristics of the study Participants

Parameter	Frequency	Percentage
Age		
< 30 years	9	20.9%
30–49 years	5	11.6%
50–69 years	24	55.8%
>= 70 years	5	11.6%
Sex		
Female	23	53.5%
Male	20	46.5%
Marital Status		
Married	34	79.1%
Single	9	20.9%
Occupation		
Civil Servant	2	4.7%
Business	29	67.4%
Unemployed	12	27.9%
Treatment Regimen		
Chemotherapy	22	100.0%
Co-morbidity Status		
None	39	90.7%
Hypertension	4	9.3%
Blood Transfusion		
Previously Transfused	24	55.8%
Never Transfused	19	44.2%

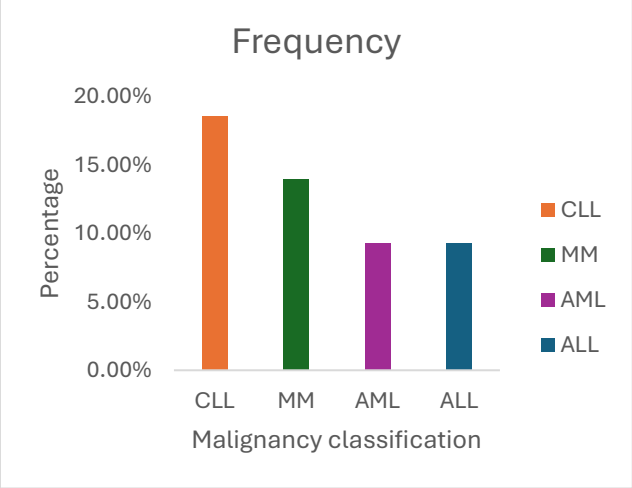

Fig 1: Prevalence of haematological malignancies in the study participants.

Table 2 shows the association between white blood cell functional indices and micronuclei frequency (mononucleated cells, binucleated cells, acentric fragments, and cellular proliferation) in acute myeloid leukaemia patients and healthy controls. Mononucleated cells (345.00±12.91 vs 186.67±18.62, p = 0.000) and acentric fragments (97.50±17.08 vs 28.33±9.83, p = 0.000) were significantly elevated compared to controls, indicating severe genomic fragmentation.

Table 3 shows the Association between white blood cell functional indices and Micronuclei frequency in Acute Lymphoblastic Leukemia (ALL) and healthy controls. Mononucleated cells exhibited a massive and highly significant elevation (705.00±12.91 vs 186.67±18.62, p = 0.000), reflecting intense cell division and arrest in the mononucleated stage.

Table 4 shows the association between white blood cell functional indices and Micronuclei frequency in Chronic Lymphocytic Leukemia (CLL) and healthy controls. Patients demonstrated statistically significant elevations in Absolute Lymphocyte Count (5.14±1.06 vs 1.83±0.74 x10^9/L, p = 0.001), Absolute Monocyte Count (0.96±0.19 vs 0.39±0.12 x10^9/L, p = 0.000), Mononucleated cells (p = 0.000), Binucleated cells with micronuclei (58.75±18.17 vs 0.00±0.00, p = 0.000), and Acentric fragments (76.25±12.82 vs 28.33±9.83, p = 0.000).

Table 5 shows the association between white blood cell functional indices and Micronuclei frequency in Multiple Myeloma (MM). Micronuclei frequency is significantly higher in MM at a p-value ≤ 0.05

Table 2: Association between white blood cell functional indices and Micronuclei frequency in Acute Myeloid Leukemia (AML)

White Cell / Cytome Index	Control (Mean±SD)	AML (Mean±SD)	Mean Diff.	p-value
Relative Neutrophil Count (RNC %)	57.50 ± 7.12	44.00 ± 1.41	13.50	1.000
Relative Lymphocyte Count (RLC %)	33.83 ± 6.34	44.50 ± 1.29	-10.67	1.000
Relative Monocyte Count (RMC %)	6.67 ± 1.51	8.75 ± 0.96	-2.08	1.000
Absolute Neutrophil Count (ANC x10^9/L)	3.06 ± 0.73	2.48 ± 0.03	0.57	1.000
Absolute Lymphocyte Count (ALC x10^9/L)	1.83 ± 0.74	2.29 ± 0.26	-0.46	1.000
Absolute Monocyte Count (AMC x10^9/L)	0.39 ± 0.12	0.49 ± 0.01	-1.10	1.000
Mononucleated Cells (/1000 cells)	186.67 ± 18.62	345.00 ± 12.91	-158.33	0.000*
Binucleated Cells (/1000 cells)	0.00 ± 0.00	20.00 ± 8.17	-20.00	0.351
Acentric Fragments (/1000 cells)	28.33 ± 9.83	97.50 ± 17.08	-69.17	0.000*
Cellular Proliferation (OD 550nm)	182.50 ± 21.90	190.25 ± 2.22	-7.75	1.000

Table 3: Association between white blood cell functional indices and Micronuclei frequency in Acute Lymphoblastic Leukemia (ALL)

White Cell / Cytome Index	Control (Mean±SD)	ALL (Mean±SD)	Mean Diff.	p-value
Relative Neutrophil Count (RNC %)	57.50 ± 7.12	54.50 ± 1.73	3.00	1.000
Relative Lymphocyte Count (RLC %)	33.83 ± 6.34	35.75 ± 1.26	-1.92	1.000
Relative Monocyte Count (RMC %)	6.67 ± 1.51	7.00 ± 0.82	-0.33	1.000
Absolute Neutrophil Count (ANC x10^9/L)	3.06 ± 0.73	5.62 ± 0.02	-2.57	1.000
Absolute Lymphocyte Count (ALC x10^9/L)	1.83 ± 0.74	3.80 ± 0.03	-1.97	0.439
Absolute Monocyte Count (AMC x10^9/L)	0.39 ± 0.12	0.74 ± 0.03	-0.35	1.000
Mononucleated Cells (/1000 cells)	186.67 ± 18.62	705.00±12.91	-518.33	0.000*
Binucleated Cells (/1000 cells)	0.00 ± 0.00	20.00 ± 8.17	-20.00	0.351
Acentric Fragments (/1000 cells)	28.33 ± 9.83	20.00 ± 8.17	8.33	1.000
Cellular Proliferation (OD 550nm)	182.50 ± 21.90	183.00 ± 4.97	-0.50	1.000

Table 4: Association between white blood cell functional indices and Micronuclei frequency in Chronic Lymphocytic Leukemia (CLL)

White Cell / Cytome Index	Control (Mean±SD)	CLL (Mean±SD)	Mean Diff.	p-value
Relative Neutrophil Count (RNC %)	57.50 ± 7.12	44.00 ± 2.14	13.50	0.297
Relative Lymphocyte Count (RLC %)	33.83 ± 6.34	46.13 ± 2.17	-12.30	0.198
Relative Monocyte Count (RMC %)	6.67 ± 1.51	8.63 ± 0.74	-1.96	1.000
Absolute Neutrophil Count (ANC x10^9/L)	3.06 ± 0.73	4.88 ± 1.02	-1.82	0.315
Absolute Lymphocyte Count (ALC x10^9/L)	1.83 ± 0.74	5.14 ± 1.06	-3.31	0.001*
Absolute Monocyte Count (AMC x10^9/L)	0.39 ± 0.12	0.96 ± 0.19	-0.57	0.000*
Mononucleated Cells (/1000 cells)	186.67 ± 18.62	468.75 ± 90.47	-282.08	0.000*
Binucleated Cells (/1000 cells)	0.00 ± 0.00	58.75 ± 18.17	-58.75	0.000*
Acentric Fragments (/1000 cells)	28.33 ± 9.83	76.25 ± 12.82	-47.92	0.000*
Cellular Proliferation (OD 550nm)	182.50 ± 21.90	210.00 ± 11.34	-27.50	0.720

Table 5: Association between white blood cell functional indices and Micronuclei frequency in Multiple Myeloma (MM)

White Cell / Cytome Index	Control (Mean±SD)	MM (Mean±SD)	Mean Diff.	p-value
Relative Neutrophil Count (RNC %)	57.50 ± 7.12	52.50 ± 2.14	5.00	1.000
Relative Lymphocyte Count (RLC %)	33.83 ± 6.34	39.00 ± 1.67	-5.17	1.000
Relative Monocyte Count (RMC %)	6.67 ± 1.51	7.33 ± 0.61	-0.66	1.000
Absolute Neutrophil Count (ANC x10^9/L)	3.06 ± 0.73	3.42 ± 0.14	-0.36	1.000
Absolute Lymphocyte Count (ALC x10^9/L)	1.83 ± 0.74	2.55 ± 0.12	-0.72	1.000
Absolute Monocyte Count (AMC x10^9/L)	0.39 ± 0.12	0.48 ± 0.03	-0.09	1.000
Mononucleated Cells (/1000 cells)	186.67 ± 18.62	363.33 ± 34.42	-176.66	0.002*
Binucleated Cells (/1000 cells)	0.00 ± 0.00	35.00 ± 8.06	-35.00	0.007*
Acentric Fragments (/1000 cells)	28.33 ± 9.83	76.67 ± 11.45	-48.34	0.000*
Cellular Proliferation (OD 550nm)	182.50 ± 21.90	183.33 ± 2.11	-0.83	1.000

Discussion

This study evaluated white blood cell functional indices and micronuclei frequency across diverse Leukaemia and Multiple myeloma at the University of Ilorin Teaching Hospital, Kwara State, Nigeria. The spectrum of malignancies observed in this study was dominated by Chronic Lymphocytic Leukemia (18.6%) and Multiple Myeloma (14.0%), which aligns closely with previous epidemiological reviews in North-Central Nigeria and broader Sub-Saharan Africa

(Abudu *et al.,* 2018; Anifowose *et al.,* 2018; Tan *et al.,* 2018). The peak age distribution in the 50–69 years group (55.8%) reflects the age-dependent cumulative risk of somatic mutations, DNA repair decay, and environmental genotoxic exposures in adult hematological oncogenesis (Mulatie *et al.,* 2025).

More than half of the patient cohort (55.8%) had a history of prior blood transfusions. Chronic blood transfusion introduces foreign cellular antigens, minor histocompatibility mismatches, and residual donor leukocytes into the recipient, driving immune activation, alloimmunization, and oxidative stress (Kareem *et al.,* 2024). Repetitive transfusion therapy can elevate systemic inflammatory signaling, which further exacerbates genomic instability in multi-transfused oncological patients (Nava *et al.,* 2019).

A central finding of this investigation is the significant elevation of micronuclei cytome endpoints, specifically mononucleated cells, binucleated (BN) cells containing micronuclei, and acentric chromosome fragments across all groups relative to healthy controls. The Cytokinesis-Block Micronucleus (CBMN) assay provides a comprehensive cytome evaluation of chromosomal breakage, chromosome loss, and mitotic failure (Nava *et al.,* 2019).

Micronuclei originate from acentric fragments or whole lagging chromosomes that fail to integrate into daughter nuclei during anaphase due to defects in double-strand break repair (such as non-homologous end joining and homologous recombination) or mitotic spindle checkpoints (Sommer *et al.,* 2020: Zhang *et al.,* 2020).

In this study, Chronic Lymphocytic Leukemia (CLL) demonstrated the highest frequency of binucleated cells with micronuclei (58.75±18.17 per 1000 cells) and acentric fragments (76.25±12.82 per 1000 cells). These findings are consistent with international studies reporting profound genomic instability, deletion of 13q14, TP53 aberrations, and complex karyotypic alterations in mature B-cell malignancies.

Furthermore, Multiple Myeloma (MM) exhibited significantly elevated binucleated and micronucleated cells and acentric fragments compared to controls ($p < 0.01$). In Multiple Myeloma, ongoing DNA damage driven by immunoglobulin gene hypermutation, genomic translocations involving the IGH locus, and bone marrow microenvironmental stress contribute to elevated micronuclei formation. In Acute Myeloid Leukemia (AML) and Acute Lymphoblastic Leukemia (ALL), mononucleated cell counts were markedly elevated (345.00 ± 12.91 and 705.00 ± 12.91, respectively), reflecting rapid blast proliferation and mitotic arrest. All participants in this cohort were undergoing active chemotherapy regimens. Cytotoxic chemotherapeutic agents, such as alkylating agents, antimetabolites, and anthracyclines, kill malignant cells primarily by inducing DNA double-strand breaks, cross-linking, or spindle poisoning. Consequently, while chemotherapy targets neoplastic clones, it simultaneously induces systemic genotoxicity in non-neoplastic lymphocytes, compounding the baseline micronuclei burden.

Conclusion

This study concludes that leukaemic patients exhibited profound genomic instability, characterized by significantly elevated micronuclei frequency, acentric chromosome fragments, and altered white cell functional indices. The Cytokinesis-Block Micronucleus assay serves as a highly reliable, accessible, and cost-effective biomarker for genotoxicity assessment and therapeutic monitoring in resource-limited clinical settings.

Recommendations

1. Institutional Integration of CBMN Assay: Diagnostic haematology laboratories in tertiary hospitals should adopt the CBMN assay as a routine, low-cost adjunctive test for monitoring baseline genomic instability and chemotherapy-induced genotoxicity in cancer patients.
2. Multi-Center Longitudinal Studies: Multi-center longitudinal studies across regional tertiary centers should be commissioned to evaluate dynamic micronuclei fluctuations before, during, and after specific chemotherapy and immunotherapy regimens.
3. Enhanced Blood Transfusion Compatibility: For multi-transfused leukaemic patients, advanced extended antigen matching and leukoreduced blood products should be prioritized to minimize immune alloimmunization and oxidative stress.

Contributions To Knowledge

1. Validation of CBMN as a Cost-Effective Genotoxic Biomarker: This study establishes the CBMN assay as a practical, highly informative biomarker for evaluating chromosomal damage in African leukaemic patients, providing a viable alternative to high-cost molecular cytogenetics in resource-constrained settings.
2. Baseline Epidemiological Data for North-Central Nigeria: The study provides comprehensive baseline clinical, sociodemographic, and cytogenetic data on leukaemic patients in Kwara State, supporting future regional oncology research and health policy planning.

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