Cureus
Part of SPRINGER NATURE

Open Access Original Article

Karyotyping Patterns in Adolescent and Adult Patients With Acute Leukemia: A Cross-Sectional Study From a Tertiary Care Hospital in Bangladesh

Review began 10/29/2026
Review ended 11/14/2026
Published 11/22/2026

© **Copyright** 2026
Kabir et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.120104

Nadia Kabir [1], Fatema Tuj Juhara Khanam [2], Nahid Sultana [2], Sajuti Humaira [3], Akhil Ranjan Biswas [4], MD Foys Ullah [5]

1. Department of Haematology and Stem Cell Transplant, Evercare Hospital Dhaka, Dhaka, BGD 2. Department of Haematology, Dhaka Medical College Hospital, Dhaka, BGD 3. Department of Medicine, Dinajpur Medical College Hospital, Dinajpur, BGD 4. Department of Haematology, Chittagong Medical College Hospital, Dhaka, BGD 5. Department of Haematology and Bone Marrow Transplant, Continental Hospital, Dhaka, BGD

Corresponding author: MD Foys Ullah, foys.sbmc@gmail.com

Abstract

Background

Karyotyping refines diagnosis and risk assignment in acute leukemia, although induction therapy is started based on morphology and immunophenotype without waiting for the result. Cytogenetic services are not available in Bangladesh, and local data are scarce. We evaluated the karyotypic pattern of acute myeloid leukemia (AML) and acute lymphoblastic leukemia (ALL) in adolescents and adults at a national referral center.

Methods

This descriptive cross-sectional study enrolled 92 patients aged 12 years and above with newly diagnosed or relapsed AML or ALL at Dhaka Medical College and Hospital, Dhaka, Bangladesh, between July 2024 and December 2025. Bone marrow aspirates were karyotyped by Giemsa trypsin banding at accredited reference laboratories abroad and described according to the International System for Human Cytogenomic Nomenclature. Fluorescence in situ hybridization and molecular testing were not performed. Cytogenetic risk was assigned with disease-specific criteria, and AML and ALL were reported separately. Groups were compared with the Mann-Whitney U, chi-square, and Fisher exact tests, with Benjamini and Hochberg adjustment for multiple comparisons.

Results

Of the 92 patients, 48 (52.2%) had AML and 44 (47.8%) had ALL, 52 (56.5%) were male and the median age was 28.5 years. An abnormal karyotype was present in 24 patients (26.1%), comprising 13 of 48 with AML (27.1%) and 11 of 44 with ALL (25.0%, p = 0.820). Translocation was the most common abnormality (15 of 24, 62.5%). In AML, t(8;21) predominated (n = 6), followed by t(15;17), trisomy 8, inv(16), t(9;22) and add(1) with monosomy 17, and no patient had a complex karyotype. In ALL, t(9;22) predominated (n = 4), followed by complex karyotype (n = 3), triploidy and near tetraploidy. Nine patients (9.8%) had adverse or poor risk karyotypes. A lower leukocyte count with an abnormal karyotype (p = 0.027) and a lower hemoglobin with adverse or poor risk karyotypes (p = 0.026) did not remain significant after adjustment for multiple comparisons.

Conclusions

About one in four adolescents and adults with acute leukemia at this single referral center carried a chromosomal abnormality detectable by conventional karyotyping, with translocation dominant in both AML and ALL. The detection rate was lower than in regional series, probably reflecting sample transport abroad, the age range studied, and the absence of complementary testing. Developing cytogenetic, fluorescence in situ hybridization, and molecular testing within Bangladesh would make genetic risk information available early in treatment.

Categories: Hematology, Genetics, Oncology
Keywords: acute leukemia, acute lymphoblastic leukemia, acute myeloid leukemia, bangladesh, cytogenetics, karyotype, risk stratification

Introduction

Acute leukemia is a clonal malignancy of hematopoietic precursor cells in which immature blasts accumulate in the bone marrow, displace normal hematopoiesis and produce rapidly progressive anemia,

neutropenia and thrombocytopenia. The two principal entities, acute myeloid leukemia (AML) and acute lymphoblastic leukemia (ALL), differ in age distribution, underlying biology and treatment, yet both behave as medical emergencies in which the interval between presentation and correctly targeted therapy determines survival [1,2]. Leukemia remains a leading cause of cancer death before the age of forty, and its global burden has risen over the past three decades, with steep increases reported from South and Southeast Asia [3,4].

Cytogenetic analysis of the leukemic clone is the single most powerful independent prognostic variable in acute leukemia and remains the foundation on which every modern classification is built. The fifth edition of the World Health Organization classification and the International Consensus Classification both define several disease entities by chromosomal rearrangement alone, so that a patient cannot be fully classified without a karyotype or equivalent genetic study [5,6]. The European LeukemiaNet risk model, which guides the decision to proceed to allogeneic transplantation in first remission, is likewise anchored on the karyotype [7]. In ALL, the presence of BCR::ABL1 converts a historically dismal disease into one treated with tyrosine kinase inhibitors, whereas hypodiploidy or a complex karyotype identifies patients who need intensified therapy [8,9]. In acute promyelocytic leukemia, all-trans retinoic acid is started as soon as the diagnosis is suspected on morphological and coagulation findings, and detection of t(15;17) confirms rather than triggers this treatment [10]. Because conventional cytogenetic reports usually take one to two weeks even in well-equipped centers, induction therapy is begun on morphology and immunophenotype, and the karyotype is then used to confirm the diagnosis, refine risk and guide postinduction decisions such as consolidation, the addition of tyrosine kinase inhibitors and allogeneic transplantation [7,9].

Despite this, karyotyping is not routinely available to patients with acute leukemia in Bangladesh. No public cytogenetic laboratory currently processes leukemic bone marrow, and samples must be transported abroad at considerable cost and with delays that jeopardize cell viability. Clinicians therefore treat many patients on morphology and immunophenotype alone, assign risk by proxy and defer transplantation decisions that should have been taken within the first weeks of therapy [11,12]. Published data on the chromosomal profile of acute leukemia in Bangladesh are scarce [13], so clinicians have no local reference for the expected frequency of risk-defining abnormalities in their own patients. Generating such data within the local population is a necessary first step toward building national cytogenetic capacity that can serve these patients directly.

The frequency and spectrum of chromosomal abnormalities vary appreciably between populations. Reported rates of abnormal karyotype in acute leukemia range from roughly 28 to 64 percent in studies from Pakistan, India, Iran, Egypt and Sweden, and the relative frequency of individual rearrangements such as t(8;21), t(15;17) and t(9;22) differs in parallel [14-19]. These differences are attributed to ethnicity, environmental exposure and the age composition of the cohort, and also to laboratory factors such as culture failure and low metaphase yield [20]. Extrapolating risk estimates from one population to another is therefore unsafe, and country-specific data are needed before any risk-adapted protocol can be adopted with confidence. Data from other countries can thus provide context only, and the evidence that guides local practice has to come from local patients.

Against this background, we evaluated the karyotypic pattern of acute leukemia in adolescents and adults at a national referral center in Bangladesh. Because AML and ALL differ in biology, risk classification and treatment, the two diseases were analyzed and reported separately wherever the numbers allowed. The primary objective was to describe the frequency and spectrum of chromosomal abnormalities detected by conventional karyotyping in patients with AML and, separately, in patients with ALL. The secondary objectives were to classify patients into established cytogenetic risk categories using disease-specific criteria and to explore whether karyotype status and cytogenetic risk group were associated with age, sex or the presenting hematological profile. Our purpose was to provide local evidence that can inform the development of cytogenetic services for patients treated in Bangladesh.

Materials And Methods

Study design and setting

This was a descriptive cross-sectional study carried out in the Department of Haematology and Bone Marrow Transplant Unit of Dhaka Medical College and Hospital, Dhaka, Bangladesh, which serves as a national referral center for hematological malignancy. The study was conducted, and participants were recruited, over an 18-month period from July 2024 to December 2025.

Participants

Patients were recruited from the inpatient and outpatient services of the department. The inclusion criteria were (i) age of 12 years or above, (ii) newly diagnosed or relapsed AML or ALL, with the diagnosis established according to World Health Organization criteria [5] by combining clinical data, bone marrow morphology and immunophenotyping by flow cytometry, and (iii) written informed consent, together with written assent and parental or guardian consent for participants younger than 18 years. The lower age limit of 12 years was set in the approved protocol, which was restricted to adolescents and adults attending the Department of

Haematology and Bone Marrow Transplant Unit. The exclusion criteria were (i) a concomitant solid malignancy, (ii) a known inherited chromosomal disorder, (iii) mixed phenotype acute leukemia, and (iv) refusal to participate. Mixed phenotype acute leukemia was excluded because it is classified as a distinct entity in the World Health Organization and International Consensus classifications [5,6] and cannot be assigned to either the AML or the ALL cytogenetic risk framework used in this study. A purposive sampling technique was applied, and every eligible patient who gave written informed consent during the recruitment period from July 2024 to December 2025 was included. Both newly diagnosed and relapsed patients were eligible under the approved protocol. Disease status at the time of sampling was not recorded as a separate variable in the study dataset, so newly diagnosed and relapsed patients could not be analyzed separately, and for relapsed patients, the karyotype describes the leukemic clone at relapse rather than at first presentation.

Sample size

The sample size was calculated using the standard formula for estimating a single proportion [21], $n = \frac{Z^2 pq}{d^2}$, where Z was 1.96 at the 95 percent confidence level, p was 0.39 based on the proportion of abnormal karyotype reported by Kulkarni et al. [15], q was 0.61, and the allowable error was 10 percent. The calculation yielded 91.4, and 92 patients were therefore recruited.

Data collection and cytogenetic analysis

A semi-structured, interviewer-administered questionnaire recorded demographic and occupational details. The presenting complete blood count and the peripheral blood blast percentage were taken from the diagnostic workup. Bone marrow aspiration was performed under aseptic precautions, and 2 mL of aspirated material was collected into a heparinized tube. Samples were labeled, held at a controlled temperature, and transported to accredited reference laboratories outside the country for cell culture, namely Core Diagnostics Pvt. Ltd., New Delhi, India (College of American Pathologists accredited, AU-ID-2010258) and SN GeneLab Pvt. Ltd., Surat, Gujarat, India (National Accreditation Board for Testing and Calibration Laboratories accredited to ISO 15189:2012). Each sample was processed at one of these two laboratories, both of which operate under accreditation standards that require internal quality control and participation in external proficiency testing. The transit time in hours and the temperature during transport were not logged in the study records. At the reference laboratories, cytogenetic analysis was performed by conventional karyotyping with the Giemsa trypsin banding (GTG banding) technique. In brief, the bone marrow cells were set up in short-term unstimulated culture, dividing cells were arrested in metaphase with colcemid, treated with hypotonic potassium chloride solution and fixed in methanol and glacial acetic acid, and the fixed cell suspension was dropped onto glass slides. The slides were treated with trypsin and stained with Giemsa to produce the banding pattern. Banded metaphases were examined under light microscopy, and karyotypes were identified and described according to the International System for Human Cytogenomic Nomenclature (ISCN) 2020 [22], with the chromosome bands given for the recurrent rearrangements. All 92 analyzed samples yielded a reportable karyotype. The number of cultures established for each sample, the number of metaphases analyzed in each case, and culture failures among other eligible patients were not recorded in the study records. Fluorescence in situ hybridization and molecular genetic studies were not performed because these tests were not available within Bangladesh and were outside the scope of the approved protocol. Abnormalities identified by karyotyping were therefore not confirmed by an independent method.

Classification of cytogenetic abnormalities

A chromosomal abnormality was regarded as clonal when the same structural rearrangement or the same extra chromosome was present in at least two metaphases, or the same missing chromosome was present in at least three metaphases, in accordance with ISCN 2020 [22]. A karyotype was classified as normal when no clonal abnormality was detected among the metaphases analyzed and as abnormal when at least one clonal abnormality was present. Abnormalities were further classified as numerical or structural. Numerical abnormalities included gain of a whole chromosome (trisomy), loss of a whole chromosome (monosomy), and changes in ploidy (hypodiploidy, hyperdiploidy, triploidy, and near tetraploidy). Structural abnormalities included translocation (t), deletion (del), inversion (inv), and additional material of unknown origin (add). For the descriptive analysis, each abnormal karyotype was assigned to one or more of the categories translocation, deletion, inversion, trisomy, complex karyotype, triploidy, and near tetraploidy. Loss of chromosomal material, whether a partial deletion or the loss of a whole chromosome, was grouped in the deletion category. A karyotype meeting the definition of a complex karyotype was counted only in the complex karyotype category, whereas a noncomplex karyotype with more than one abnormality was counted in each applicable category.

Definitions and cytogenetic risk stratification

A complex karyotype was defined as three or more independent chromosomal abnormalities in AML [7]. For descriptive consistency, the same threshold of three or more abnormalities was applied in ALL, and the stricter threshold of five or more abnormalities used in the UKALLXII and ECOG E2993 analyses [23] is reported separately. In AML, favorable risk was defined by t(8;21), inv(16), or t(15;17); adverse risk by t(6;9),

t(9;22), t(8;16), inv(3), monosomy 5 or del(5q), monosomy 7, an abnormality of 17p, or a complex karyotype; and intermediate risk by all remaining karyotypes, including a normal karyotype [7]. Because molecular testing for NPM1, CEBPA, FLT3, TP53, RUNX1, ASXL1, and the other genes required by the 2022 European LeukemiaNet classification was not available, this assignment reflects only the cytogenetic component of that classification and does not constitute full genetic risk stratification. Patients with a normal karyotype or trisomy 8 were therefore placed in the intermediate category by default, and their true risk could be favorable or adverse depending on molecular findings [7]. Acute promyelocytic leukemia with t(15;17) is managed outside the European LeukemiaNet risk scheme and was grouped with the favorable karyotypes for descriptive purposes only. In ALL, standard risk comprised hyperdiploidy, t(12;21), t(1;19), and all karyotypes that did not meet poor risk criteria, whereas poor risk comprised hypodiploidy, near triploidy, del(17p), t(9;22), monosomy 7, and a complex karyotype [2,8,23]. These are the cytogenetic risk groups described for adult ALL in the UKALLXII and ECOG E2993 trials and in recent reviews [2,8,23]. As in AML, a normal karyotype in ALL was placed in the standard risk group by default. For the comparative analysis, adverse risk AML and poor risk ALL were merged into a single adverse or poor risk group, and the remaining patients formed the favorable, intermediate, or standard risk group. For the analysis of presenting blood counts, hemoglobin was grouped as below 8.0, 8.0 to 10.9, 11.0 to 12.0, and above 12.0 g/dL on the basis of the World Health Organization thresholds for severe, moderate, and mild anemia [24], and the total leukocyte count (below 4.0, 4.0 to 11.0, and above 11.0 × 10^9/L) and the platelet count (below 50, 50 to 99, 100 to 150, and above 150 × 10^9/L) were grouped around the conventional adult reference limits [25].

Statistical analysis

Data were checked and edited manually, then coded and entered into IBM SPSS Statistics for Windows, Version 26.0 (Released 2018; IBM Corp., Armonk, NY, USA). Categorical variables were summarized as frequency and percentage, n (%). The proportion of patients with an abnormal karyotype was reported with its exact (Clopper-Pearson) 95 percent confidence interval (CI). Continuous variables were summarized as median with interquartile range (IQR) because all of them were skewed on inspection, and they were compared between two groups with the Mann-Whitney U test. Categorical variables, including the distribution of normal and abnormal karyotypes and of cytogenetic risk groups by type of acute leukemia, sex, age group, and categorized blood counts, were compared with the Pearson chi-square test, and the Fisher exact test was used instead when any expected cell count was below 5 or when more than 20 percent of cells had an expected count below 5. The frequency of individual abnormality categories in AML and ALL was compared with the Fisher exact test. All tests were two-sided, and a p-value below 0.05 was taken as statistically significant. Figures were drawn in Python with the matplotlib library, version 3.10.9.

Ethics

The Ethical Review Committee of Dhaka Medical College, Dhaka, Bangladesh, which serves as the institutional review board, approved the protocol before the study began (approval number ERC-DMC/ECC/2024/181). Each adult participant gave written informed consent in their preferred language after an explanation of the study aims, procedures, risks, and benefits, including the processing of the bone marrow sample at an accredited reference laboratory outside Bangladesh. Participants younger than 18 years gave written assent alongside written consent from a parent or legal guardian. Participation was voluntary, withdrawal carried no consequence for clinical care, personal identifiers were removed from the dataset, and confidentiality was maintained throughout. The study conformed to the Declaration of Helsinki.

Results

A total of 92 patients were analyzed, of whom 48 (52.2 percent) had AML and 44 (47.8 percent) had ALL. Among the participants, 52 patients (56.5 percent) were male, giving a male-to-female ratio of 1.3 to 1. The median age of the whole cohort was 28.5 years, with an IQR of 18.8 to 40.0 years and a range of 12 to 75 years. Patients with AML were significantly older than patients with ALL (median 35.0 versus 22.0 years, p = 0.001), and the distribution across age groups differed accordingly (p = 0.003). Anemia was profound, with 50 patients (54.3 percent) presenting with a hemoglobin below 8.0 g/dL, and 63 patients (68.5 percent) had a platelet count below 50 × 10^9/L. Peripheral blood blasts were present in 64 patients (69.6 percent). None of the presenting hematological variables differed significantly between AML and ALL. An abnormal karyotype was identified in 24 of 92 patients (26.1 percent, 95 percent CI 17.5 to 36.3), whereas 68 patients (73.9 percent) had a normal karyotype. The proportion was 13 of 48 (27.1 percent) in AML and 11 of 44 (25.0 percent) in ALL, and the difference between the two diseases was not significant (p = 0.820) (Table *1*).

Characteristic	AML (n = 48), n (%)	ALL (n = 44), n (%)	p-value
Sex			
Male	25 (52.1)	27 (61.4)	0.370*
Female	23 (47.9)	17 (38.6)	
Age, years, median (IQR)	35.0 (23.8, 49.0)	22.0 (16.8, 30.5)	0.001†

Age group			
Up to 20 years	10 (20.8)	20 (45.5)	0.003*
21 to 30 years	8 (16.7)	13 (29.5)	
31 to 40 years	14 (29.2)	7 (15.9)	
Above 40 years	16 (33.3)	4 (9.1)	
Hemoglobin, g/dL, median (IQR)	7.6 (6.6, 9.2)	7.8 (7.4, 9.1)	0.390†
Hemoglobin category			
Below 8.0 g/dL	27 (56.2)	23 (52.3)	0.978‡
8.0 to 10.9 g/dL	16 (33.3)	16 (36.4)	
11.0 to 12.0 g/dL	3 (6.2)	3 (6.8)	
Above 12.0 g/dL	2 (4.2)	2 (4.5)	
Total leukocyte count, × 10^9/L, median (IQR)	13.4 (3.8, 34.2)	7.5 (3.0, 41.8)	0.566†
Total leukocyte count category			
Below 4.0	14 (29.2)	19 (43.2)	0.375*
4.0 to 11.0	7 (14.6)	5 (11.4)	
Above 11.0	27 (56.2)	20 (45.5)	
Platelet count, × 10^9/L, median (IQR)	40.5 (20.0, 63.0)	29.5 (12.0, 46.0)	0.083†
Platelet count category			
Below 50	29 (60.4)	34 (77.3)	0.078‡
50 to 99	8 (16.7)	7 (15.9)	
100 to 150	5 (10.4)	3 (6.8)	
Above 150	6 (12.5)	0 (0.0)	
Peripheral blast, percent, median (IQR)	35.0 (5.2, 80.0)	70.0 (0.0, 80.0)	0.763†
Peripheral blast			
Absent	12 (25.0)	16 (36.4)	0.237*
Present	36 (75.0)	28 (63.6)	
Karyotype			
Normal	35 (72.9)	33 (75.0)	0.820*
Abnormal	13 (27.1)	11 (25.0)	

TABLE 1: Baseline demographic, hematological and cytogenetic characteristics of the patients by type of acute leukemia (n = 92).

Values are presented as n (%), with percentages calculated within each column, unless shown as median (IQR). p-values were obtained with the Pearson chi-square test (*), the Mann-Whitney U test (†), or the Fisher exact test (‡).

AML, acute myeloid leukemia; ALL, acute lymphoblastic leukemia; IQR, interquartile range; n, number of patients; %, percentage of patients; g/dL, grams per deciliter; × 10^9/L, × 10^9 cells per liter

The individual karyotypes are shown in Table 2. In AML, t(8;21) was the single most frequent abnormality and was seen in six patients (12.5 percent of all patients with AML), two of whom also had loss of the X chromosome. It was followed by t(15;17) in two patients, trisomy 8 in two, inv(16) in one, t(9;22) in one, and add(1) with monosomy 17 in one. In ALL, t(9;22) was seen in four patients (9.1 percent of all patients with ALL), of whom three carried it as an isolated translocation and one carried it within a complex karyotype. The other abnormalities in ALL were t(1;6) in two patients and t(2;12) in one, while three patients had a

complex karyotype, one had triploidy, and one had a near tetraploid clone with 90 to 94 chromosomes. Hypodiploidy was not observed in either group, and monosomy 7 occurred only as a component of a complex karyotype in one patient with ALL. No patient with AML had a complex karyotype, and the two AML karyotypes with more than one abnormality, 45,X,-X,t(8;21) and 45,XY,add(1),-17, each carried two abnormalities. Of the three complex karyotypes in ALL, one contained seven abnormalities and met the stricter threshold of five or more, whereas the other two contained three abnormalities each.

Karyotype	AML (n = 48), n (%)	ALL (n = 44), n (%)
Normal karyotype	35 (72.9)	33 (75.0)
Abnormal karyotype	13 (27.1)	11 (25.0)
46,XX,t(8;21)(q22;q22.1)	3 (6.2)	0 (0.0)
46,XY,t(8;21)(q22;q22.1)	1 (2.1)	0 (0.0)
45,X,-X,t(8;21)(q22;q22.1)	2 (4.2)	0 (0.0)
46,XY,t(15;17)(q24.1;q21.2)	2 (4.2)	0 (0.0)
47,XY,+8	2 (4.2)	0 (0.0)
46,XX,inv(16)(p13.1q22)	1 (2.1)	0 (0.0)
46,XY,t(9;22)(q34.1;q11.2)	1 (2.1)	1 (2.3)
45,XY,add(1),-17	1 (2.1)	0 (0.0)
46,XX,t(9;22)(q34.1;q11.2)	0 (0.0)	2 (4.5)
46,XX,t(1;6)	0 (0.0)	2 (4.5)
46,XY,t(2;12)	0 (0.0)	1 (2.3)
46,XY,t(2;9),del(7p),t(9;22)(q34.1;q11.2)	0 (0.0)	1 (2.3)
47,XX,+8/47,idem,t(9;19),del(17)	0 (0.0)	1 (2.3)
46,XY,del(6),-7,-9,-11,+mar1,+mar2,+mar3	0 (0.0)	1 (2.3)
69,XXY	0 (0.0)	1 (2.3)
90~94<4n>	0 (0.0)	1 (2.3)

TABLE 2: Karyotypes observed in patients with acute myeloid leukemia and acute lymphoblastic leukemia (n = 92).

Values are presented as n (%), with percentages calculated within the respective disease group. This table is descriptive, and no statistical test was applied. Karyotypes are written according to ISCN 2020, with the breakpoints of the recurrent rearrangements shown in parentheses. Breakpoints of the nonrecurrent rearrangements were not available in the study records and are shown without band designation. In the karyotype 47,XX,+8/47,idem,t(9;19),del(17), the stemline carries trisomy 8, and the sideline carries the additional rearrangements. 90~94 denotes a near tetraploid clone with 90 to 94 chromosomes.

AML, acute myeloid leukemia; ALL, acute lymphoblastic leukemia; ISCN, International System for Human Cytogenomic Nomenclature; n, number of patients; %, percentage of patients; t, translocation; inv, inversion; del, deletion; add, additional material of unknown origin; mar, marker chromosome; idem, the same karyotype as the stemline; p, short arm of a chromosome. A plus sign before a chromosome indicates gain and a minus sign indicates loss of that chromosome.

Translocation was the commonest abnormality overall and was present in 15 of the 24 patients with an abnormal karyotype (62.5 percent). Among the 13 patients with AML and an abnormal karyotype, nine (69.2 percent) had a translocation, three (23.1 percent) a deletion, two (15.4 percent) a trisomy, and one (7.7 percent) an inversion, and two patients carried both a translocation and a deletion. Among the 11 patients with ALL and an abnormal karyotype, six (54.5 percent) had a translocation, three (27.3 percent) a complex karyotype, one (9.1 percent) triploidy, and one (9.1 percent) near tetraploidy (Figure *1*). Because one of the four patients with t(9;22) in ALL carried it within a complex karyotype, only three of them are counted in the translocation category of this figure, and the fourth is counted in the complex karyotype category. The frequency of translocation did not differ significantly between AML and ALL (69.2 versus 54.5 percent, p = 0.675, Fisher exact test), and complex karyotypes were confined to ALL (3 of 11 versus 0 of 13, p = 0.082, Fisher exact test).

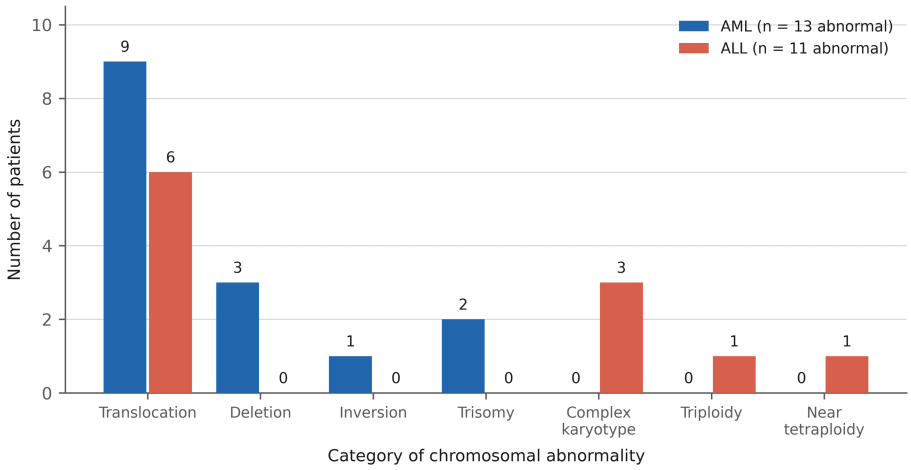

FIGURE 1: Categories of chromosomal abnormality among patients with an abnormal karyotype, shown separately for acute myeloid leukemia and acute lymphoblastic leukemia.

Categories are not mutually exclusive because two patients with acute myeloid leukemia carried both a translocation and a deletion. The x-axis shows the category of chromosomal abnormality, and the y-axis shows the number of patients, with the count printed above each bar.

AML, acute myeloid leukemia; ALL, acute lymphoblastic leukemia; n, number of patients.

Patients with a normal karyotype and patients with an abnormal karyotype did not differ with respect to sex, age group, hemoglobin category, leukocyte category, platelet category, peripheral blast status, or type of acute leukemia, and all p-values exceeded 0.05 with the Pearson chi-square or Fisher exact test. When the blood counts were analyzed as continuous variables, the presenting total leukocyte count was significantly lower in patients with an abnormal karyotype (median 4.9 versus 15.3 × 10^9/L, p = 0.027, Mann-Whitney U test), whereas hemoglobin and platelet count did not differ significantly (Table *3*). After Benjamini and Hochberg adjustment for the 11 comparisons in Table *3*, this difference was no longer significant (adjusted p = 0.295). Within each disease, the leukocyte count was lower in patients with an abnormal karyotype in ALL (median 3.1 versus 12.2 × 10^9/L, p = 0.042) but not in AML (median 5.2 versus 16.0 × 10^9/L, p = 0.260).

Characteristic	Normal karyotype (n = 68), n (%)	Abnormal karyotype (n = 24), n (%)	p-value
Sex			
Male	40 (58.8)	12 (50.0)	0.453*
Female	28 (41.2)	12 (50.0)	
Age, years, median (IQR)	28.5 (18.8, 40.0)	28.0 (18.8, 39.0)	0.929†
Age group			
Up to 20 years	23 (33.8)	7 (29.2)	0.940*
21 to 30 years	16 (23.5)	5 (20.8)	
31 to 40 years	15 (22.1)	6 (25.0)	
Above 40 years	14 (20.6)	6 (25.0)	
Hemoglobin, g/dL, median (IQR)	8.1 (7.0, 9.2)	7.2 (6.5, 8.1)	0.073†
Hemoglobin category			
Below 8.0 g/dL	33 (48.5)	17 (70.8)	0.105‡
8.0 to 10.9 g/dL	28 (41.2)	4 (16.7)	
11.0 to 12.0 g/dL	5 (7.4)	1 (4.2)	
Above 12.0 g/dL	2 (2.9)	2 (8.3)	

Total leukocyte count, × 10⁹/L, median (IQR)	15.3 (3.4, 45.8)	4.9 (2.0, 14.6)	0.027†
Total leukocyte count category			
Below 4.0	23 (33.8)	10 (41.7)	
4.0 to 11.0	7 (10.3)	5 (20.8)	0.225‡
Above 11.0	38 (55.9)	9 (37.5)	
Platelet count, × 10⁹/L, median (IQR)	31.0 (20.0, 60.2)	33.0 (11.5, 50.0)	0.357†
Platelet count category			
Below 50	46 (67.6)	17 (70.8)	
50 to 99	12 (17.6)	3 (12.5)	0.776‡
100 to 150	5 (7.4)	3 (12.5)	
Above 150	5 (7.4)	1 (4.2)	
Peripheral blast			
Absent	19 (27.9)	9 (37.5)	0.382*
Present	49 (72.1)	15 (62.5)	
Type of acute leukemia			
AML	35 (51.5)	13 (54.2)	0.820*
ALL	33 (48.5)	11 (45.8)	

TABLE 3: Comparison of demographic and hematological characteristics between patients with a normal and an abnormal karyotype (n = 92).

Values are presented as n (%), with percentages calculated within each column, unless shown as median (IQR). p-values were obtained with the Pearson chi-square test (*), the Mann-Whitney U test (†), or the Fisher exact test (‡). p-values are unadjusted. After Benjamini and Hochberg adjustment for the 11 comparisons in this table, no p-value remained below 0.05 (adjusted p for the total leukocyte count = 0.295).

AML, acute myeloid leukemia; ALL, acute lymphoblastic leukemia; IQR, interquartile range; n, number of patients; %, percentage of patients; g/dL, grams per deciliter; × 10⁹/L, × 10⁹ cells per liter.

Among the 48 patients with AML, nine (18.8 percent) were assigned to the favorable risk group, 37 (77.1 percent) to the intermediate risk group, and two (4.2 percent) to the adverse risk group. Among the 44 patients with ALL, 37 (84.1 percent) were standard risk, and seven (15.9 percent) were poor risk (Table *4*). Overall, 9 of 92 patients (9.8 percent) carried adverse or poor risk cytogenetics, comprising two patients with AML and seven patients with ALL.

Leukemia type	Risk category	Defining cytogenetic findings, n (%)	Patients, n (%)
AML (n = 48)	Favorable	t(8;21), 4 (8.3); t(8;21) with loss of X, 2 (4.2); t(15;17), 2 (4.2); inv(16), 1 (2.1)	9 (18.8)
	Intermediate	Normal karyotype, 35 (72.9); trisomy 8, 2 (4.2)	37 (77.1)
	Adverse	t(9;22), 1 (2.1); add(1) with monosomy 17, 1 (2.1)	2 (4.2)
ALL (n = 44)	Standard	Normal karyotype, 33 (75.0); t(1;6), 2 (4.5); t(2;12), 1 (2.3); near tetraploidy, 1 (2.3)	37 (84.1)
	Poor	t(9;22), 3 (6.8); complex karyotype, 3 (6.8); triploidy, 1 (2.3)	7 (15.9)

TABLE 4: Cytogenetic risk stratification of patients with acute myeloid leukemia and acute lymphoblastic leukemia (n = 92).

Values are n (%) within each disease group (AML, n = 48; ALL, n = 44), and no statistical test was applied. Risk categories follow the definitions given in the Methods and are based on the karyotype alone, so the AML intermediate category is provisional. t(15;17) is grouped with the favorable karyotypes for descriptive purposes. One patient with ALL carrying both t(9;22) and a complex karyotype is counted once, under complex karyotype. No patient with AML had a complex karyotype.

AML, acute myeloid leukemia; ALL, acute lymphoblastic leukemia; t, translocation; inv, inversion; add, additional material of unknown origin.

No categorical variable differed significantly between the combined favorable, intermediate, or standard risk group and the adverse or poor risk group. The proportion of patients with ALL was higher in the adverse or poor risk group than in the remaining patients (77.8 versus 44.6 percent), which corresponds to a higher frequency of adverse or poor risk cytogenetics in ALL than in AML (15.9 versus 4.2 percent), but the difference did not reach statistical significance (p = 0.081, Fisher exact test). When analyzed as a continuous variable, hemoglobin was significantly lower in the adverse or poor risk group (median 6.8 versus 7.9 g/dL, p = 0.026, Mann-Whitney U test) (Table 5). After adjustment for the 11 comparisons in Table 5, this difference was no longer significant (adjusted p = 0.284). Within ALL, the difference in hemoglobin between the poor risk and standard risk groups did not reach significance (median 7.1 versus 8.1 g/dL, p = 0.050), and the AML adverse risk group contained only two patients and was not tested separately.

Characteristic	Favorable, intermediate or standard risk (n = 83), n (%)	Adverse or poor risk (n = 9), n (%)	p-value
Sex			
Male	46 (55.4)	6 (66.7)	0.727‡
Female	37 (44.6)	3 (33.3)	
Age, years, median (IQR)	28.0 (18.0, 40.0)	37.0 (22.0, 38.0)	0.430†
Age group			
Up to 20 years	29 (34.9)	1 (11.1)	0.551‡
21 to 30 years	18 (21.7)	3 (33.3)	
31 to 40 years	18 (21.7)	3 (33.3)	
Above 40 years	18 (21.7)	2 (22.2)	
Hemoglobin, g/dL, median (IQR)	7.9 (6.9, 9.2)	6.8 (5.7, 7.6)	0.026†
Hemoglobin category			
Below 8.0 g/dL	43 (51.8)	7 (77.8)	0.387‡
8.0 to 10.9 g/dL	30 (36.1)	2 (22.2)	
11.0 to 12.0 g/dL	6 (7.2)	0 (0.0)	
Above 12.0 g/dL	4 (4.8)	0 (0.0)	
Total leukocyte count, × 10^9/L, median (IQR)	11.4 (3.3, 42.3)	13.7 (1.6, 19.1)	0.247†

Total leukocyte count category			
Below 4.0	29 (34.9)	4 (44.4)	
4.0 to 11.0	12 (14.5)	0 (0.0)	0.522‡
Above 11.0	42 (50.6)	5 (55.6)	
Platelet count, × 10^9/L, median (IQR)	31.0 (17.5, 60.0)	32.0 (9.0, 36.0)	0.563†
Platelet count category			
Below 50	56 (67.5)	7 (77.8)	
50 to 99	15 (18.1)	0 (0.0)	0.586‡
100 to 150	7 (8.4)	1 (11.1)	
Above 150	5 (6.0)	1 (11.1)	
Peripheral blast			
Absent	24 (28.9)	4 (44.4)	0.447‡
Present	59 (71.1)	5 (55.6)	
Type of acute leukemia			
AML	46 (55.4)	2 (22.2)	0.081‡
ALL	37 (44.6)	7 (77.8)	

TABLE 5: Comparison of demographic and hematological characteristics between patients with favorable, intermediate or standard risk cytogenetics and patients with adverse or poor risk cytogenetics (n = 92).

Values are presented as n (%), with percentages calculated within each column, unless shown as median (IQR). p-values were obtained with the Pearson chi-square test (*), the Mann-Whitney U test (†), or the Fisher exact test (‡). p-values are unadjusted. After Benjamini and Hochberg adjustment for the 11 comparisons in this table, no p-value remained below 0.05 (adjusted p for hemoglobin = 0.284).

AML, acute myeloid leukemia; ALL, acute lymphoblastic leukemia; IQR, interquartile range; n, number of patients; %, percentage of patients; g/dL, grams per deciliter; × 10^9/L, × 10^9 cells per liter.

The two blood count variables that differed significantly on continuous analysis before adjustment for multiple comparisons are displayed in Figure *2* for descriptive purposes. The presenting total leukocyte count was lower in patients with an abnormal karyotype than in patients with a normal karyotype (panel a), and hemoglobin was lower in patients with adverse or poor risk cytogenetics than in patients with favorable, intermediate, or standard risk cytogenetics (panel b). Neither association remained significant after adjustment, and both should be regarded as exploratory.

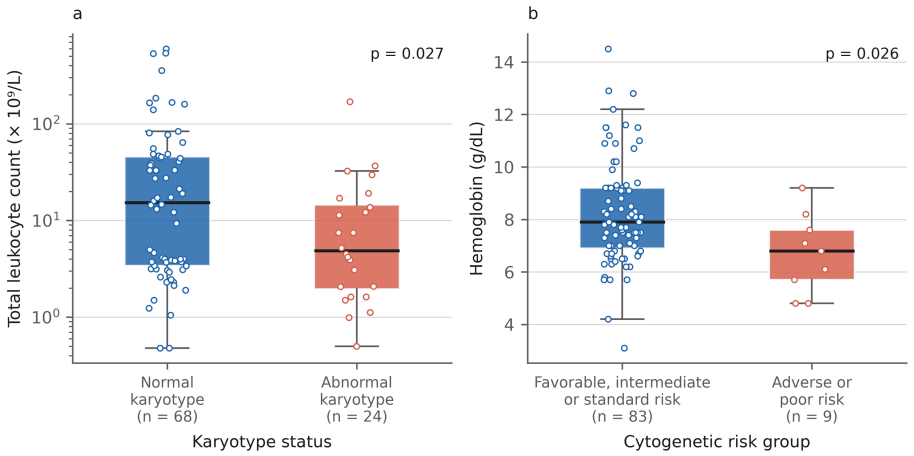

FIGURE 2: Presenting blood counts by cytogenetic finding: (a) Total leukocyte count in patients with a normal and an abnormal karyotype, plotted on a logarithmic scale. (b) Hemoglobin concentration in patients with favorable, intermediate, or standard risk cytogenetics and in patients with adverse or poor risk cytogenetics.

(a) Total leukocyte count by karyotype status, on a logarithmic scale. (b) Hemoglobin by cytogenetic risk group.

Boxes show the median and interquartile range, whiskers extend to 1.5 times the interquartile range, and dots represent individual patients. p-values are unadjusted Mann-Whitney U test values, and neither remained significant after adjustment for multiple comparisons.

n, number of patients.

Discussion

This study describes the karyotypic pattern of acute leukemia in adolescents and adults treated at a single referral center in Bangladesh. An abnormal karyotype was demonstrated in 26.1 percent of 92 patients, and the proportion was almost identical in AML and ALL. Translocation was the dominant structural abnormality in both diseases, and only nine patients carried cytogenetics that would be classified as adverse or poor risk.

The overall rate of abnormal karyotype was lower than in most published series. Studies from Pakistan, India, Iran, Egypt, and Sweden have reported abnormal karyotypes in 28 to 64 percent of patients with acute leukemia [14-19]. Three explanations are plausible. First, our inclusion criterion of age 12 years and above excluded young children, in whom hyperdiploid and translocation-positive ALL is concentrated [26]. Second, all cultures were established after the samples had been transported to laboratories in another country, and delayed transport is a recognized cause of culture failure and low metaphase yield that biases results toward normal [20,27]. Third, we relied on conventional banding alone, so cryptic rearrangements that require fluorescence in situ hybridization or sequencing were not detectable [28]. For these reasons, and because the sample came from a single center, the rate we observed should not be taken as representative of all patients with acute leukemia in Bangladesh.

Within AML, the abnormalities we observed were the expected ones. Translocation of chromosomes 8 and 21 was the commonest finding, followed by t(15;17), trisomy 8, inv(16), and t(9;22). This ordering matches the Pakistani series of Khan et al. and the large Indian cohort of Srivastava et al., in which t(8;21) and t(15;17) were also the leading structural changes [14,16]. We found no complex karyotype and no monosomy 5 or monosomy 7 in AML, which explains the small adverse risk group and probably reflects both the young median age of our cohort and the modest number of analyzable metaphases.

In ALL, t(9;22) was the single most frequent abnormality and was present in 9.1 percent of ALL patients. This is below the 13.4 percent reported among Pakistani adolescents and young adults and the 26 percent seen in a Swedish population-based series [19,29]. Complex karyotype, triploidy, and near tetraploidy accounted for the remainder. Neither hypodiploidy nor high hyperdiploidy was identified, a notable difference from Indian reports and one that again points toward limited metaphase recovery rather than biological absence.

Cytogenetic risk assignment placed 18.8 percent of AML patients in the favorable group, a proportion close

to the frequency of core binding factor disease reported elsewhere [30], and only 4.2 percent in the adverse group, while 15.9 percent of ALL patients were poor risk. The intermediate and standard categories were therefore very large, but this reflects a normal karyotype defaulting into them rather than favorable biology. A normal karyotype obtained without molecular confirmation is a finding of exclusion, and such patients now require targeted sequencing before their risk can be considered established [5,7,8]. Under the 2022 European LeukemiaNet framework, risk cannot be assigned from the karyotype alone, so the AML risk groups in this study should be read as cytogenetic risk groups only.

Karyotype status and risk group were not associated with sex, age, platelet count, or peripheral blast status. Two continuous associations reached nominal statistical significance before adjustment. Patients with an abnormal karyotype presented with a lower total leukocyte count, and patients with adverse or poor risk cytogenetics presented with a lower hemoglobin concentration. Neither association remained significant after adjustment for multiple comparisons, and both derive from a modest sample, so they should be regarded as exploratory and hypothesis generating rather than confirmatory.

The karyotype also identified findings with potential management relevance. Nine patients had a favorable risk AML karyotype, including the two patients with t(15;17), in whom the result confirmed the need for differentiation therapy, and in this group, deferral of transplantation in first remission would ordinarily be considered once molecular findings and treatment response were known [7,10]. A further five patients, one with AML and four with ALL, carried t(9;22), a finding that identifies candidates for tyrosine kinase inhibitors [9]. Because these two groups do not overlap, 14 of 92 patients had a karyotypic finding that could inform management. Treatment and outcome data were not collected, however, so we could not determine whether these results actually changed therapy or survival.

These findings also carry practical implications for risk stratification and management of acute leukemia in Bangladesh. Treatment of acute leukemia should not be delayed while cytogenetic results are awaited, and induction is appropriately started on morphology and immunophenotype. Because karyotyping currently depends on samples sent abroad, the result often becomes available only after induction has started, so the first treatment decisions are frequently made without cytogenetic information [11,12]. In this setting, patients with suspected acute promyelocytic leukemia should continue to receive all trans retinoic acid as soon as the diagnosis is suspected on clinical and morphological grounds, without waiting for confirmation of t(15;17) [10]. Rapid targeted testing for BCR::ABL1 by fluorescence in situ hybridization or reverse transcription polymerase chain reaction at diagnosis would allow tyrosine kinase inhibitors to be added to induction for patients with Philadelphia chromosome-positive ALL without delay [9]. The large group of patients with a normal karyotype, who fell by default into the intermediate or standard risk category, cannot be assigned a reliable risk without molecular testing for markers such as NPM1, CEBPA, and FLT3 in AML [7], and until such testing is available, decisions about consolidation and referral for allogeneic transplantation in these patients will have to rely on clinical features and response to induction. A pragmatic local strategy would therefore be to perform karyotyping in every patient, to add a small panel of targeted molecular tests for those with a normal karyotype or culture failure, and to direct scarce transplantation resources toward patients with adverse or poor risk cytogenetics. Establishing cytogenetic and molecular testing within the country would shorten turnaround time and make such risk-adapted treatment achievable for most patients.

Several limitations should be considered. The study was conducted at a single center with a modest purposive sample, so the estimates should not be taken as representative of all patients with acute leukemia in Bangladesh. The sample size was calculated to estimate the overall proportion of abnormal karyotypes with a precision of 10 percent, and the study was not powered for separate estimates within AML and ALL or for comparisons between subgroups. All bone marrow samples were transported abroad for culture, and the delay inherent in that process can reduce metaphase yield and shift results toward an apparently normal karyotype; the number of metaphases analyzed, the number of cultures per sample, the transit time, the temperature during transport, and the culture failure rate were not recorded systematically and could not be adjusted for. Only conventional banding was available, so cryptic rearrangements detectable by fluorescence in situ hybridization or by sequencing were missed, and patients with a normal karyotype could not be characterized further. Abnormalities were not confirmed by an independent method, and risk was assigned on the karyotype alone, which does not meet the 2022 European LeukemiaNet requirement for integrated molecular testing, so the intermediate and standard risk groups in particular are provisional. Children below 12 years of age were excluded, which introduces selection bias and removes the age band in which ALL, and particularly hyperdiploid ALL, is most frequent, so our findings apply to adolescents and adults only. Patients at relapse were included alongside newly diagnosed patients, and because clonal evolution may add abnormalities at relapse, the spectrum may differ from that of de novo disease. Patients with mixed phenotype acute leukemia were excluded, so the findings do not apply to that entity. Finally, the cross-sectional design carried no treatment or survival follow-up, so the prognostic value of the observed abnormalities could not be tested directly, and the two nominally significant continuous associations arose from multiple comparisons and did not remain significant after adjustment.

Conclusions

In this single-center series of adolescents and adults with acute leukemia in Bangladesh, conventional banding detected an abnormal karyotype in 26.1 percent of patients, with a nearly identical yield in AML

and ALL. Translocation was the dominant abnormality; t(8;21) led in AML and t(9;22) in ALL, no complex karyotype was found in AML, and 9.8 percent had adverse or poor risk karyotypes. The lower detection rate than in regional series may reflect the age range, sample transport abroad, and the absence of complementary testing. Because risk was assigned without molecular data and outcomes were not collected, these findings describe cytogenetic patterns rather than prognosis. Cytogenetic, fluorescence in situ hybridization, and molecular testing within Bangladesh would make genetic risk information available early in treatment, alongside prompt induction therapy.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: MD Foys Ullah, Nadia Kabir, Fatema Tuj Juhara Khanam, Nahid Sultana, Sajuti Humaira, Akhil Ranjan Biswas

Critical review of the manuscript for important intellectual content: MD Foys Ullah, Nadia Kabir, Fatema Tuj Juhara Khanam, Nahid Sultana, Sajuti Humaira, Akhil Ranjan Biswas

Acquisition, analysis, or interpretation of data: Nadia Kabir

Drafting of the manuscript: Nadia Kabir

Supervision: Akhil Ranjan Biswas

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. Ethical Review Committee of Dhaka Medical College issued approval ERC-DMC/ECC/2024/181. **Animal subjects:** All authors have confirmed that this study did not involve animal subjects or tissue. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. DiNardo CD, Erba HP, Freeman SD, Wei AH: Acute myeloid leukaemia. Lancet. 2023, 401:2073-86. 10.1016/S0140-6736(23)00108-3
2. Malard F, Mohty M: Acute lymphoblastic leukaemia. Lancet. 2020, 395:1146-62. 10.1016/S0140-6736(19)33018-1
3. Daltveit DS, Morgan E, Colombet M, et al.: Global patterns of leukemia by subtype, age, and sex in 185 countries in 2022. Leukemia. 2025, 39:412-9. 10.1038/s41375-024-02452-y
4. Zhang N, Wu J, Wang Q, et al.: Global burden of hematologic malignancies and evolution patterns over the past 30 years. Blood Cancer J. 2023, 13:82. 10.1038/s41408-023-00853-3
5. Khoury JD, Solary E, Abla O, et al.: The 5th edition of the World Health Organization classification of haematolymphoid tumours: myeloid and histiocytic/dendritic neoplasms. Leukemia. 2022, 36:1703-19. 10.1038/s41375-022-01613-1
6. Arber DA, Orazi A, Hasserjian RP, et al.: International consensus classification of myeloid neoplasms and acute leukemias: integrating morphologic, clinical, and genomic data. Blood. 2022, 140:1200-28. 10.1182/blood.2022015850
7. Döhner H, Wei AH, Appelbaum FR, et al.: Diagnosis and management of AML in adults: 2022 recommendations from an international expert panel on behalf of the ELN. Blood. 2022, 140:1345-77. 10.1182/blood.2022016867
8. Passet M, Kim R, Clappier E: Genetic subtypes of B-cell acute lymphoblastic leukemia in adults . Blood. 2025, 145:1451-63. 10.1182/blood.2023022919
9. Abou Dalle I, Moukalled N, El Cheikh J, Mohty M, Bazarbachi A: Philadelphia-chromosome positive acute lymphoblastic leukemia: ten frequently asked questions. Leukemia. 2024, 38:1876-84. 10.1038/s41375-024-02319-2
10. Odetola O, Tallman MS: How to avoid early mortality in acute promyelocytic leukemia . Hematology Am Soc Hematol Educ Program. 2023, 2023:248-53. 10.1182/hematology.2023000477
11. Rujkijyanont P, Inaba H: Diagnostic and treatment strategies for pediatric acute lymphoblastic leukemia in low- and middle-income countries. Leukemia. 2024, 38:1649-62. 10.1038/s41375-024-02277-9
12. Morcos-Sandino M, Quezada-Ramírez SI, Gómez-De León A: Advances in the treatment of acute myeloid leukemia: implications for low- and middle-income countries. Biomedicines. 2025, 13:1221. 10.3390/biomedicines13051221

13. Khan ER, Begum F, Islam A, Ahmed KMA: Coagulopathy in pediatric acute promyelocytic leukemia in Bangladesh: a single-center, prospective study. Pediatr Hematol Oncol J. 2024, 9:114-9. 10.1016/j.phoj.2024.03.012
14. Khan M, Altaf C, Malik HS, Naeem MA: Cytogenetic profile of acute myeloid leukemia and acute lymphoblastic leukemia in northern Pakistan. Pak J Med Sci. 2023, 39:1440-5. 10.12669/pjms.39.5.6405
15. Kulkarni NV, Shetty VV, Prasad Hl K, et al.: Evaluation of the cytogenetic profile in patients with acute leukaemia. Pol J Pathol. 2023, 74:89-97. 10.5114/pjp.2023.129425
16. Srivastava VM, Nair SC, Sappani M, et al.: Cytogenetic profile of 1791 adult acute myeloid leukemia in India . Mol Cytogenet. 2023, 16:24. 10.1186/s13039-023-00653-1
17. Shakeri S, Ayatollahi H, Sadeghian MH, Shams SF: A bone marrow study; report of chromosomal variations in hematologic malignancies including acute myeloid leukemia, acute lymphoid leukemia, and myelodisplastic syndrome (Northeast Iran). Middle East J Cancer. 2021, 12:391-8. 10.30476/mejc.2020.82641.1092
18. Elnaggar MG, Mosad E, Makboul A, Shafik EA: Cytogenetic profile of adult acute myeloid leukemia in Egypt: a single-center experience. Mol Cytogenet. 2022, 15:43. 10.1186/s13039-022-00621-1
19. Bergfelt Lennmyr E, Engvall M, Barbany G, Fogelstrand L, Rhodin H, Hallböök H: Cytogenetic aberrations in adult acute lymphoblastic leukemia - a population-based study. EJHaem. 2021, 2:813-7. 10.1002/jha2.300
20. Javed S, Hassan J, Naz M, Shan S, Abid M, Shamsi TS: Cytogenetic culture failure and its causes in hematological disorders; a single centre perspective. Mol Cytogenet. 2023, 16:4. 10.1186/s13039-022-00635-9
21. Charan J, Biswas T: How to calculate sample size for different study designs in medical research? . Indian J Psychol Med. 2013, 35:121-6. 10.4103/0253-7176.116232
22. McGowan-Jordan J, Hastings RJ, Moore S: An International System for Human Cytogenomic Nomenclature . Karger, Karger; 2020. 10.1159/isbn.978-3-318-06867-2
23. Moorman AV, Harrison CJ, Buck GA, et al.: Karyotype is an independent prognostic factor in adult acute lymphoblastic leukemia (ALL): analysis of cytogenetic data from patients treated on the Medical Research Council (MRC) UKALLXII/Eastern Cooperative Oncology Group (ECOG) 2993 trial. Blood. 2007, 109:3189-97. 10.1182/blood-2006-10-051912
24. Haemoglobin concentrations for the diagnosis of anaemia and assessment of severity . (2011). Accessed: October 12, 2026: http://who.int/publications/i/item/WHO-NMH-NHD-MNM-11.1.
25. Bain BJ, Bates I, Laffan MA: Dacie and Lewis Practical Haematology. Elsevier, London; 2017.
26. Haas OA, Borkhardt A: Hyperdiploidy: the longest known, most prevalent, and most enigmatic form of acute lymphoblastic leukemia in children. Leukemia. 2022, 36:2769-83. 10.1038/s41375-022-01720-z
27. Behrens YL, Pietzsch S, Antić Ž, Zhang Y, Bergmann AK: The landscape of cytogenetic and molecular genetic methods in diagnostics for hematologic neoplasia. Best Pract Res Clin Haematol. 2024, 37:101539. 10.1016/j.beha.2024.101539
28. Chebly A: Cytogenetics in the genomics era: why karyotyping still matters . BMC Med Genomics. 2026, 19:10. 10.1186/s12920-025-02296-0
29. Brohi S, Shaikh IA, Tariq F, et al.: Spectrum of cytogenetic abnormalities in adolescent and young adult patients of B-cell acute lymphoblastic leukemia in a tertiary care hospital in Pakistan. Cureus. 2025, 17:e83122. 10.7759/cureus.83122
30. Zhai Y, Wang Q, Ji L, et al.: Clinical characteristics and prognostic factors analysis of core binding factor acute myeloid leukemia in real world. Cancer Med. 2023, 12:21592-604. 10.1002/cam4.6693