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Original Research Article

CHROMOSOMAL ABERRATIONS IN ACUTE MYELOID LEUKEMIA IN NORTH INDIAN POPULATION

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ABSTRACT

Background: Acute Myeloid Leukemia (AML) is a hematological malignancy characterized by genetic and chromosomal abnormalities that significantly influence its prognosis and therapeutic outcomes. Cytogenetic analysis plays a crucial role in identifying these aberrations and stratifying patients for risk-adapted therapy. **Aim:** To evaluate the spectrum and distribution of chromosomal aberrations in AML patients in the North Indian population and to analyze their association with age and gender.

Materials and Methods: This descriptive, laboratory-based study was conducted at the Cytogenetic Laboratory, Department of Anatomy, King George's Medical University (KGMU), Lucknow. AML patients from the Departments of Pediatrics and Medicine were enrolled following informed consent. Bone marrow and peripheral blood samples were collected and processed for conventional karyotyping using standard G-banding techniques. Karyograms were analyzed with Cytovision software, and chromosomal abnormalities were identified based on established cytogenetic criteria.

Results: Out of 34 AML cases, karyotyping was successful in 22 patients, of which 12 (54.5%) exhibited chromosomal abnormalities. Pediatric patients (≤10 years) constituted the majority (73.53%), and chromosomal aberrations were more prevalent in this age group. Structural abnormalities, primarily translocations such as t (8;21), t(9;11), and t(9;22), were the most common (40.9%), while numerical abnormalities such as +21 and +8 were also frequently observed. Males showed a higher incidence of abnormal karyotypes (66.7%) compared to females (40%).

Conclusion: This study underscores the predominance of chromosomal abnormalities in pediatric AML cases within the North Indian population. Translocations and trisomies (+21, +8) were the most frequent findings. Cytogenetic profiling remains indispensable for early diagnosis, risk stratification, and personalized management in AML.

Keywords: Acute Myeloid Leukemia, Cytogenetic Analysis, Chromosomal Abnormalities, Pediatric AML, Translocations.

INTRODUCTION

Acute myeloid leukaemia (AML), also known as acute myelogenous leukaemia, is a malignant disorder of the myeloid line of blood cells, characterized by the clonal proliferation and accumulation of immature myeloblasts in the bone marrow, which interferes with normal haematopoiesis.[1] It is the most common type of acute leukaemia in adults, and its incidence increases with age, with the average age of onset being around 63 years.[1] The disease originates from a malignant transformation of myeloid progenitor cells, which leads to the rapid expansion of poorly differentiated myeloblasts that fail to mature into functional blood cells.[2]

AML typically arises in the bone marrow—the central site of blood cell formation—and rapidly infiltrates the bloodstream. In many cases, it may spread to other tissues, including the liver, spleen, lymph nodes, central nervous system, and testes in males.[1] The proliferation of leukemic cells results

in the suppression of normal blood cell production, leading to symptoms such as fatigue, pallor, infections, bruising, and bleeding.[1]

AML is a biologically and clinically heterogeneous disease that encompasses a variety of subtypes, each with distinct cytogenetic, molecular, and morphological features.[3] According to the French-American-British (FAB) classification, AML subtypes are designated as M0 to M7, depending on the degree of differentiation and the type of lineage involved.[3] These include granulocytic maturation (M1–M3), monocytic differentiation (M4, M5), erythroid predominance (M6), and megakaryoblastic features (M7).[4] Identification of subtypes is achieved through a combination of cellular morphology, cytochemistry, immunophenotyping, and cytogenetic or molecular analysis.[4]

Cytochemical staining remains a valuable diagnostic tool in distinguishing AML from acute lymphoblastic leukaemia (ALL) and in defining AML subtypes.[4] In the most common forms of AML such as acute promyelocytic leukaemia (APL), the differentiation process is arrested at specific stages, such as the promyelocyte stage, with the leukemic blasts acquiring self-renewal capacity[5] . These blocked immature cells continue to multiply, ultimately overcrowding the bone marrow and suppressing normal haematopoiesis.[5]

Diagnostic criteria for AML include the presence of more than 20% blasts in the bone marrow. However, certain chromosomal abnormalities, such as t(8;21)(q22;q22), inv(16)(p13q22), and t(15;17)(q22;q12), are considered definitive for diagnosis, even if blast percentage is below this threshold.[6] Cytogenetic analysis thus plays a critical role not only in diagnosis but also in prognosis and therapeutic stratification. Specific translocations such as t(15;17), which is associated with APL, and t(8;21) or inv(16), typically indicate a favourable prognosis.[6]

Despite significant advances in understanding the biology of AML, the disease remains challenging to treat. Although 75–85% of patients may achieve complete remission with induction chemotherapy, long-term survival remains limited, particularly in older individuals or those with adverse cytogenetic profiles.[6] Additionally, patients with normal karyotypes—who comprise about 45–50% of cases—show intermediate outcomes and require further molecular stratification to predict response and relapse risk.[6]

MATERIALS AND METHODS

The present study was a descriptive, laboratory-based investigation conducted at the Cytogenetic Laboratory, Department of Anatomy, King George's Medical University (KGMU), Uttar Pradesh, Lucknow. Ethical clearance was obtained from the Institutional Review Board of KGMU prior to the initiation of the study. Patients diagnosed with acute myeloid leukaemia (AML) were identified through the Departments of Pediatrics and Medicine. Inclusion criteria included diagnosed cases of AML of any age and gender, with informed consent obtained from each patient or guardian. Patients who did not provide consent were excluded from the study. Bone marrow aspirate and peripheral venous blood samples were collected from eligible patients through the Department of Pathology, KGMU. The collected specimens were processed in the Cytogenetic Laboratory for karyotyping analysis.

Cytogenetic Analysis and Karyogram Preparation

Bone Marrow Cell Culture and Harvesting

Bone marrow aspirates were collected in BD Vacutainer tubes containing sodium heparin as an anticoagulant. In a sterile environment under laminar airflow, 0.5 ml of bone marrow was added to 5 ml of RPMI-1640 culture medium in a sterile test tube. Cultures were incubated at 37°C with 85% humidity and 5% CO_2 concentration in a slanting position for 24 hours.

Post-incubation, five drops of KaryoMAX colcemid solution were added and the tubes were incubated for an additional hour to arrest metaphase cells. The samples were then centrifuged at 1000 rpm for 10 minutes, and the supernatant was carefully discarded.

The resulting cell pellet was resuspended in 5 ml of pre-warmed hypotonic solution (0.075 M KCl and sodium citrate in a 3:1 ratio) and incubated at 37°C for 30 minutes. Following a second centrifugation at 1000 rpm for 10 minutes, the supernatant was discarded.

Cells were then fixed by adding 5 ml of chilled fixative (methanol:acetic acid, 3:1) dropwise while vortexing gently. This fixation step was repeated 2–3 times until the cell button turned white, indicating adequate fixation. The final cell suspension was used for slide preparation.

Peripheral Blood Cell Culture and Harvesting

Venous blood was similarly collected in heparinized vacutainers. Under sterile conditions, 0.5 ml of blood was added to 5 ml of RPMI-1640 medium supplemented with 0.1 ml of phytohemagglutinin (PHA) to stimulate cell division. Cultures were incubated at 37°C in a CO_2 incubator (5% CO_2 , 85% humidity) for 72 hours in a slanting position.

After 72 hours, five drops of colchicine (0.1 µg/ml) were added, and the cultures were returned to the incubator for 1 hour. Cells were then harvested using the same centrifugation, hypotonic treatment, and fixation protocol as for bone marrow specimens.

Slide Preparation and Karyotyping

Prepared cell suspensions were dropped onto clean glass slides using the dropping method and air-dried. The slides were pre-treated with trypsin for optimal banding and stained using Giemsa stain.

Microscopic analysis was performed using a computer-assisted microscope equipped with Cytovision software. Karyograms were prepared both digitally and manually, and each was analyzed

for chromosomal abnormalities according to standardized cytogenetic criteria (Ram S. Verma and Arvind Babu, 1st edition).

RESULTS

Table 1: Age Distribution of AML Cases
The analysis of age-wise distribution among the 34 patients diagnosed with AML reveals a significant predilection for the pediatric age group. A striking 73.53% (n=25) of cases were identified in children aged up to 10 years, while only 26.47% (n=9) were seen in individuals older than 10 years. This suggests a higher burden of AML in younger patients in this study cohort, indicating the importance of early diagnostic vigilance in pediatric populations.

Table 2: Gender-wise Distribution of Chromosomal Abnormalities
Among the 34 total AML cases, karyotyping was successfully obtained in 22 patients. Out of these, 12 were males and 10 were females. The gender-wise breakdown shows that abnormal karyotypes were more common in males (8 out of 12; 66.7%) compared to females (4 out of 10; 40%). Conversely, normal karyograms were observed more frequently in females (6 out of 10; 60%) than in males (4 out of 12; 33.3%). This pattern may suggest a potential gender-related biological difference in the chromosomal aberration profiles associated with AML.

Table 3: Types of Structural Chromosomal Abnormalities
Structural chromosomal abnormalities were observed in 12 of the 22 karyotyped cases. Translocations were the most common type of structural abnormality, with three recurrent translocations—t(9;11), t(8;21), and t(9;22)—each found in two patients, accounting for 9.09% per translocation type. Less frequently observed abnormalities included t(15;17), t(3;9), and t(4;7), each in one patient (4.54%). Additionally, p-arm additions like add(19p+) and deletions like del(16q-) were noted in one case each. The diversity of these structural alterations reflects the genetic heterogeneity characteristic of AML.

Table 4: Numerical Chromosomal Abnormalities
Out of the 22 karyotyped cases, numerical chromosomal abnormalities were present in 7 patients, corresponding to 31.8% of the cohort. Trisomy 21 (+21) was the most prevalent numerical abnormality, identified in three cases (13.63%), followed by trisomy 8 (+8) in two cases (9.09%). Less commonly, trisomy 11 (+11) and monosomy 8 (-8) were each seen in one patient (4.54%). The predominance of +21 and +8 aligns with known associations of these abnormalities in AML pathogenesis and prognosis.

Table 5: Age and Gender-wise Distribution of Abnormal Karyotypes
Detailed case-wise analysis of 12 patients with chromosomal abnormalities revealed that most cases occurred in males (8 out of 12; 66.7%), with ages ranging from 2 to 45 years. The most common chromosomal aberrations were +21, t(9;22), t(9;11), and t(8;21), with several patients exhibiting multiple complex rearrangements. Notably, three cases involved patients under 10 years of age, and five more were aged between 10 to 22 years, reaffirming the high burden of chromosomal abnormalities in younger patients. Females were represented in four cases, primarily showing hyperploidy (+8, +11, +21) and t(9;22), suggesting a varied but less frequent pattern of abnormalities compared to males.

Table 6: Age-wise Pattern of Chromosomal Abnormalities
Among 20 patients exhibiting chromosomal abnormalities, the majority (n=15; 75%) were aged ≤10 years, while only five were older than 10 years. Translocations were the most frequently identified aberration, seen in 9 cases—6 of which occurred in the younger age group. Hyperploidy (n=6) and rare anomalies like aneuploidy, p-arm additions (add), and q-arm deletions (del) were predominantly observed in children under 10. Additionally, combinations of translocations with deletions were present in two younger patients, indicating that complex chromosomal alterations tend to be more prevalent in pediatric AML. No cases showed overlapping combinations of translocation, hyperploidy, and deletion together.

Table 1: Distribution of Acute Myeloid Leukemia in Different Age Groups

Age Group	No. of Cases (n = 34)	Percentage (%)
Up to 10 years	25	73.53%
More than 10 years	9	26.47%

Table 2: Gender-wise Distribution of Cases with Chromosomal Abnormalities

Sex	Number of Cases	Karyogram Obtained	Normal Karyogram	Abnormal Karyogram
Male	22	12	4	8
Female	12	10	6	4
Total	34	22	10	12

Table 3: Different Types of Structural Chromosomal Abnormalities

Structural Chromosomal Abnormalities	Number of Patients	Percentage (%) (N = 22)
t(9;11)	2	9.09%
t(8;21)	2	9.09%
t(9;22)	2	9.09%

t(15;17)	1	4.54%
t(3;9)	1	4.54%
t(4;7)	1	4.54%
add(19p+)	1	4.54%
del(16q-)	1	4.54%

Table 4: Prevalence of Numerical Chromosomal Abnormalities in Overall Studied Karyograms / Total Cases with Abnormal Karyograms

Numerical Chromosomal Abnormalities	Number of Patients	Percentage (%) (N = 22)
+21	3	13.63%
+8	2	9.09%
+11	1	4.54%
-8	1	4.54%
Total	7	31.8%

Table 5: Age & Gender-wise Distribution of Chromosomal Abnormalities

S.N.	Karyogram	Age	Sex
1	47XY, +21	8 years	Male
2	46XX, t(9;22)	38 years	Female
3	46XY, t(9;11)	36 years	Male
4	46XY, t(8;21)	45 years	Male
5	47XX, +8	26 years	Female
6	45XY, -8, t(9;22), add(19p)	10 years	Male
7	46XY, t(3;9), t(4;7), t(15;17), del(16q)	10 years	Male
8	46XY, t(8;21)	12 years	Male
9	46XY, t(9;11)	5 years	Male
10	47XY, +21	22 years	Male
11	47XX, +8	6 years	Female
12	48XX, +11, +21	2 years	Female

Table 6: Age-wise Distribution of Cases with Different Types of Chromosomal Abnormalities

Chromosomal Aberrations	Total Cases	Age ≤ 10 years	Age > 10 years
Translocation	9	6	3
Hyperploidy	6	4	2
Aneuploidy	1	1	0
p-Arm Abnormality (add)	1	1	0
q-Arm Abnormality (del)	1	1	0
Translocation + Hyperploidy	0	0	0
Translocation + Deletions	2	2	0
Hyperploidy + Deletions	0	0	0
Translocation + Hyperploidy + Deletions	0	0	0
Total	20	15	5

DISCUSSION

The present study was conducted in the Department of Anatomy, King George's Medical University (KGMU), Uttar Pradesh, Lucknow. Blood and bone marrow samples were collected from the Department of Pathology, while patients were screened in the Departments of Pediatrics and Medicine between April 2012 and June 2013. A total of 34 cases (22 males and 12 females) were analyzed.

Cytogenetic abnormalities were detected in 54.54% of cases, a figure comparable to previous large-scale studies from different geographic regions, which report abnormalities ranging from 52% to 80%.[7,8] These findings closely align with Enjeti et al. (2004),[9] who reported 61% cytogenetic abnormalities among AML patients in Southeast Asia (Singapore). Similarly, population-based data from Sweden showed no significant gender-based difference in chromosomal abnormalities (Mauritzson et al., 1999),[10] which is consistent with the absence of gender differences observed in our study.

In our analysis, t(9;11) was found in two cases (9.09%), in concordance with Ten et al. (1992)[11], who documented this translocation in three Malaysian patients aged 5, 31, and 2 years, respectively. Although not the most common translocation, its presence has been noted in diverse populations. Enjeti et al. (2004),[9] evaluated 454 patients over 15 years of age in Singapore and found abnormal cytogenetics in 275 (61%) of them.

Translocation t(15;17), classically associated with acute promyelocytic leukemia (APL), was reported by Park et al. (2008)[12] as the most frequent chromosomal abnormality along with trisomy 8, occurring in 11% and 7.3% of cases, respectively. In the present study, t(15;17) was observed in 1 case (4.54%) and trisomy 8 in 2 cases (9.09%) in the North Indian population.

Children with Down syndrome are known to have a 10–20-fold increased risk of developing acute leukemia.[13,14,15] Mandal et al. (2013),[16] reported trisomy 21 in a case from West Bengal involving a 1-year-8-month-old child. In our study, trisomy 21

was noted in 3 cases (13.63%) in the North Indian population. One of these also had associated trisomy 11 (4.54%). However, we did not find the t(21;21) translocation noted by Bakshi et al. (2003),[17] in a 3-year-old female child with Down syndrome.

The t(8;21) translocation, commonly seen in AML-M2 (FAB classification), was reported in various populations by Ghosh et al. (2005), Movafagh et al. (2011), Moorman et al. (2002), and Othman et al. (2012)[18,19,20,21] Ghosh et al. reported two rare cases, one with biphenotypic leukemia and del(8;21). Movafagh et al. found it in 35 out of 127 Iranian adults (27.5%). Moorman et al. (2002),[20] found t(8;21) in 32 out of 600 patients (5.33%). In our study, it was observed in 2 patients (9.09%), a rate close to that in Singapore (14.5%) and lower than that reported in Japan and Taiwan (33.1% and 34%, respectively),[22,8,9] It is also lower compared to 22% in North America and 15.3% in Australia,[23,24]

Trisomy 8 is widely reported across various neoplasms and is considered to contribute to tumor progression. In our series, trisomy 8 was detected in 2 cases (9.09%). Its prognostic significance in lymphoid malignancies remains inconclusive. The role of trisomy 8 in leukemogenesis may be related to uniparental disomy and gene imprinting effects[26] . Moorman et al. (2002),[20] found trisomy 8 in 32 patients (5.33%), with similar frequencies reported across Europe and North America (6–9%),[25,22,23]

The overall frequency of t(15;17) in our study was 4.54%, which is lower than that in Taiwan (15%), Japan (11%), Southeast Asia (11%), and North America/Europe (3–10%),[25,8,9] Movafagh et al. (2009),[27] reported it in 22 Iranian cases (33.8%) and 12 Indian patients (19.3%), highlighting regional variation. We also observed t(9;22) in 2 cases (9.09%), which is lower than the 16.9% in Iran and 11.3% in Indian cases described by Movafagh et al.

We did not observe inv(16) or 11q23 abnormalities, both of which have been reported previously. Toogeh et al. (2003) analyzed 104 Iranian AML patients aged 12–60 years and obtained karyograms in 39 cases, with 29 abnormal and 10 normal. They observed high frequencies of t(9;22), trisomy 11, and trisomy 8. Trisomy 11 and t(9;22) were seen in 4 cases (10.3%) each, while trisomy 8 was present in 3 cases (7.7%). They reported only one case each of t(8;21) and trisomy 21 (2.6%).

To the best of our knowledge, no prior study has reported t(3;9), although t(4;7) was observed by Achkar et al. (2013),[29] in a 65-year-old female, similar to one case (4.54%) in our study. Movafagh et al. (2009),[27] also noted numerical aberrations in chromosomes 5, 7, and 8 in 4 Iranian (6%) and 5 Indian patients (8%). We identified monosomy 8 in one case and trisomy 8 in two cases (total 9.09%). Most autosomal trisomies arise from maternal nondisjunction events during meiosis, with some due to postzygotic mitotic errors.

Trisomy 11, which is considered to have a poor prognosis when found as an isolated abnormality[28] , was noted in our study along with trisomy 21 in one case (4.54%). Deletion 16(q) was detected in one case (4.54%) in our series, while Enjeti et al. (2004),[9] reported inv(16) and t(16;16) as rare. Movafagh et al. (2009),[27] found inv(16) in 3.2% of Indian and 3% of Iranian patients. Notably, our study identified add(19p) in one case (4.54%), a finding not previously described in the literature.

Ayesh et al. (2012),[30] evaluated 35 Jordanian AML patients and found cytogenetic abnormalities in 20 of 31 successful karyograms (65%). Trisomy 8 was the most common anomaly (13%) in his cohort, which corresponds to our finding of 9.09%. However, he did not report any t(8;21) cases.

Population-based studies on AML remain limited,[31,10] Ethnic and geographic variation may contribute to the observed differences in chromosomal abnormalities across studies. Larger epidemiological investigations across multiple regions are necessary to fully elucidate the interplay of environmental and genetic factors in AML.

CONCLUSION

This study highlights the significant prevalence of chromosomal abnormalities in acute myeloid leukemia (AML), particularly among pediatric patients. Translocations were the most frequently observed structural abnormalities, while trisomy 21 was the most common numerical anomaly. The findings emphasize the importance of cytogenetic analysis in AML for early diagnosis, classification, and guiding prognosis. Age and gender-specific patterns of aberrations further support the need for individualized cytogenetic profiling in AML management.

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