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Original Article

Pathology Section

Immunophenotypic Aberrancies in Acute Lymphoblastic Leukaemia: A Cross-sectional Study from a Tertiary Care Centre in Rajasthan, India

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ABSTRACT

Introduction: Leukaemic cells display characteristic patterns of surface antigenic expression. Aberrant phenotypes are defined as patterns of antigen expression on neoplastic cells that deviate from the process of normal haematopoietic maturation. Flow cytometric immunophenotyping in acute leukaemia is an important tool for the detection of these aberrancies.

Aim: The present study aimed to access immunophenotypic aberrancies in B and T Acute Lymphoblastic Leukaemia (ALL).

Materials and Methods: This prospective cross-sectional study included 137 newly diagnosed ALL (B and T lineage) patients from June 2023 to November 2024 at Mahatma Gandhi Medical College and Hospital, Jaipur, Rajasthan, India. Haematological parameters, including mean haemoglobin levels, Total Leukocyte Count (TLC), platelet count, and blast percentage were analysed using a cell counter and peripheral blood and bone marrow examination. Flow cytometric analysis was performed using the Beckman Coulter DxFLEX 13-colour flow cytometer. Cytogenetic studies were carried out using conventional G-banding karyotyping. Statistical analysis was conducted using the Statistical Package for Social Sciences (SPSS) statistics software windows version 22.0 released 2013. Statistical tests like Pearson's Chi-square test and Mann-Whitney Test were applied.

Results: Out of 137 cases of ALL; B-ALL comprised 114 (83.2%) cases and T-ALL 23 (16.7%) cases. For B-ALL, the mean age was 23.9±20.05 years whereas T-ALL subjects had a mean age of 25.3±20.88 years. The proportional frequency of aberrant antigen expression in B-ALL and T-ALL was 53 (46.50%) and 7 (30.4%), respectively. The most common aberrant antigen in B-ALL was CD33 and in T-ALL was CD13. No significant association between haematological parameters and cytogenetic abnormalities in cases with conventional and aberrant phenotype.

Conclusion: B-ALL showed greater antigen heterogeneity than T-ALL. Aberrant markers may aid in residual disease monitoring, indicate genetic events, predict prognosis, and serve as therapeutic targets.

Keywords: Aberrant, Blood cancers, Cytogenetic, Flow cytometry, Leukaemia

INTRODUCTION

According to Global Cancer Observatory (GLOBOCAN) 2022, leukaemia is the 8th most common cancer in India with an incidence rate of 3.5% and mortality rate of 4.0% [1]. Acute leukaemia comprises of a diverse group of blood cancers characterised by the clonal expansion of abnormal myeloid or lymphoid cells in the blood and bone marrow [2]. The blast cells in acute leukaemia typically display a specific pattern of surface antigen expression, known as Cluster of Differentiation (CD) antigens, which form the basis for the current classification of blood cancers into various types [3].

However, in some cases, leukaemia blast cells show an abnormal antigen pattern, referred to as aberrant immunophenotype. In ALL, these abnormalities may include: a) expression of myeloid antigens in ALL, B lineage antigens in T-ALL, or T lineage antigens in B-ALL; b) co-expression of both early (immature) and mature antigens; and c) either over expression, under expression, or lack of expression of certain antigens [4,5].

Aberrant phenotype may result from abnormal genetic programming, causing precursor cells to retain characteristics of one lineage that should have been lost during differentiation into another. Flow cytometric immunophenotyping is crucial in determining the lineage of acute leukaemia and detecting aberrant antigens, which aid in treatment monitoring and Minimal Residual Disease (MRD) analysis [6].

Aberrant antigen expression in some leukaemias may suggest an underlying genetic event or have implications for prognosis. For example, CD9 and CD15 abnormalities in B-ALL are associated with KMT2A rearrangement, while Philadelphia chromosome positive ALL (Ph+ALL) often shows aberrant expression of myeloid markers such as CD13 and CD33 [5]. However, the prognostic significance of myeloid antigen expression in ALL remains debated. While some studies have linked myeloid antigen expression with poorer outcomes in ALL patients, others have found no such associations [7-10]. There is a scarcity of data on immunophenotyping and cytogenetics in ALL patients from India. This study was conducted to address this gap.

The aim of the present study was to evaluate the prevalence and patterns of aberrant antigen expression in B-ALL and T-ALL and to identify association between aberrant antigen expression and haematological parameters (mean haemoglobin levels, TLC, platelet count, and percentage of blasts in peripheral blood). Also associate aberrant antigen expression with conventional cytogenetic technique.

MATERIALS AND METHODS

The present cross-sectional study was conducted in the Department of Oncopathology at Mahatma Gandhi Medical College and Hospital, Jaipur, Rjasthan, India. It is a prospective cross-sectional study conducted form June 2023 to November 2024. Ethical clearance was obtained from the Institutional Ethics Committee (No./MGMC&H/IEC/JPR/2024/1906). Diagnosis was established

through Complete Blood Count (CBC), mean haemoglobin levels, TLC, platelet count, Peripheral Blood Smears (PBS), bone marrow examination, and immunophenotyping via flow cytometry.

Inclusion criteria: Newly diagnosed B & T ALL cases.

Exclusion criteria: Already known cases on treatment and biphenotypic leukaemia.

Study Procedure

Peripheral blood or bone marrow aspirate samples, collected in Ethylenediaminetetraacetic Acid (EDTA) or heparin anticoagulants, were utilised for flow cytometry. Immunophenotyping was conducted within 24 hours of sample collection, adhering to the standard stain-lyse-wash protocol. Sample processing and instrument calibration were performed in accordance with EuroFlow guidelines [11]. The antibody panel employed for analysis was CD34, CD2, cCD3, CD4, CD5, CD7, CD8, CD3, CD56, TdT, CD19, CD10, CD20, cCD79a, CD123, CD58, CD86, CD22, CD15, CD13, CD117, MPO, CD13, CD33, HLA-DR, CD64, CD11b, CD14 and CD41, CD61, CD235. A minimum of 50,000 events per sample were acquired using a Beckman Coulter DxFLEX 3 laser, 13-color flow cytometer, and data analysis was carried out using CytExpert software.

After doublet discrimination and debris exclusion, all viable events were plotted against CD 45 and Side Scatter Area (SSC). Blasts were gated by selecting events with low to moderate CD45 expression and low side scatter. The expression of various surface and cytoplasmic markers was evaluated within the target cell population. B lineage was determined by strong CD19 expression with at least one additional strong marker (CD10, CD22, or CD79a) or weak CD19 expression with at least two markers. T lineage requires CD3 (cytoplasmic or surface) expression exceeding 50% intensity of mature T cells in flow cytometry [2].

Based on the presence of aberrant antigens, ALL cases were further divided into Aberrant phenotype (ALL expressing aberrant antigen) and conventional phenotype (ALL without aberrant antigen expression).

For cytogenetic analysis bone marrow aspirate samples collected in heparin anticoagulant. To obtain analysable metaphase cells, pretreatment samples are subjected to unstimulated short-term (24 hour) cultures on Roswell Park Memorial Institute-1640 (RPMI-1640), at hours the end of which the cells are treated with a colcemid that arrests dividing cells in metaphase. Thereafter, microscope slides containing metaphase spreads are made, appropriately aged, stained using G-banding techniques and analysed under microscope. Twenty or more metaphase cells are usually analysed. While in cases with an abnormal karyotype analysis of <20 cells can be acceptable, for a case to be reliably determined as cytogenetically normal, an analysis of at least 20 karyotypes from a BM sample was required [12]. Cytogenetic analysis required the identification of at least >2 cells with the same structural change or chromosomal gain and >3 cells with the same chromosomal loss, in at least 20 metaphases. Karyotypes were documented according to the International System for Human Cytogenomic Nomenclature (ISCN) [13].

These groups (ALL with aberrant and conventional phenotype) were then compared in terms of mean hemoglobin levels, TLC, platelet count, percentage of blasts in peripheral blood and conventional cytogenetic analysis.

STATISTICAL ANALYSIS

The data was analysed through the SPSS statistics software windows version 22.0 released 2013. Armonk, NY: IBM Corp. Appropriate statistical tests like Pearson's Chi-square test and Mann Whitney Test were applied to establish the significant association between different variables.

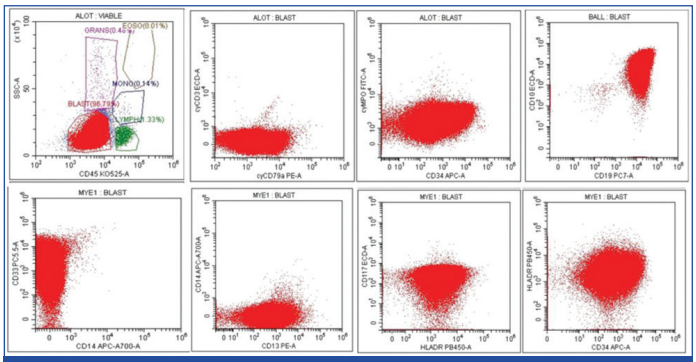
RESULTS

According to the lineage assignment criteria outlined in the World Health Organisation (WHO) 5th edition [2], the immunological classification of the 137 ALL cases revealed that 114 (83.2%) cases were B-ALL, and 23 (16.7%) cases were T-ALL. Out of 114 cases of B-ALL aberrant phenotype was observed in 53 (46.5%) cases and conventional phenotype in 61 (53.5%) cases. While in 23 cases of T-ALL aberrant phenotype was observed in 7 (30.4%) cases and conventional phenopype in 16 (69.6%) cases.

For B-ALL, the mean age was 23.9±20.05 years whereas T-ALL subjects had a mean age of 25.3±20.88 years. The most common age group in B and T ALL 1-10 years and 11-20 years, respectively. In B-ALL cases, 65 (57%) were male and 49 (43%) were female, showing a balanced distribution. However, T-ALL displayed a significant gender disparity with 20 (87%) male and 3 (13%) female. The most common aberrant antigen in B ALL [Table/Fig-1] was CD 33, found in 19 (35.8%) of cases. Following this, paired aberrant expression of CD13 and CD 33 [Table/Fig-2] was present in 14 (26.4%) cases.

Type of aberrancy	Aberrant type in B lineage ALL (n=53) n (%)	Aberrant type in T lineage ALL (n=7) n (%)
CD 33	19 (35.80%)	2 (28.60%)
CD13, CD 33	14 (26.40%)	1 (14.30%)
CD 13	6 (11.30%)	3 (42.90%)
CD 7	3 (5.70%)	0
CD 2	2 (3.80%)	0
CD 15, CD 33	2 (3.80%)	0
CD 5	1 (1.90%)	0
CD 15	1 (1.90%)	0
CD 56	1 (1.90%)	0
CD 2, CD 33	1 (1.90%)	0
CD 13, CD 33 and CD 2	1 (1.90%)	0
CD 13, CD 33 and CD 7	1 (1.90%)	0
CD 13, CD 33 and CD 15	1 (1.90%)	0
CD11B and CD33	0	1 (14.30%)

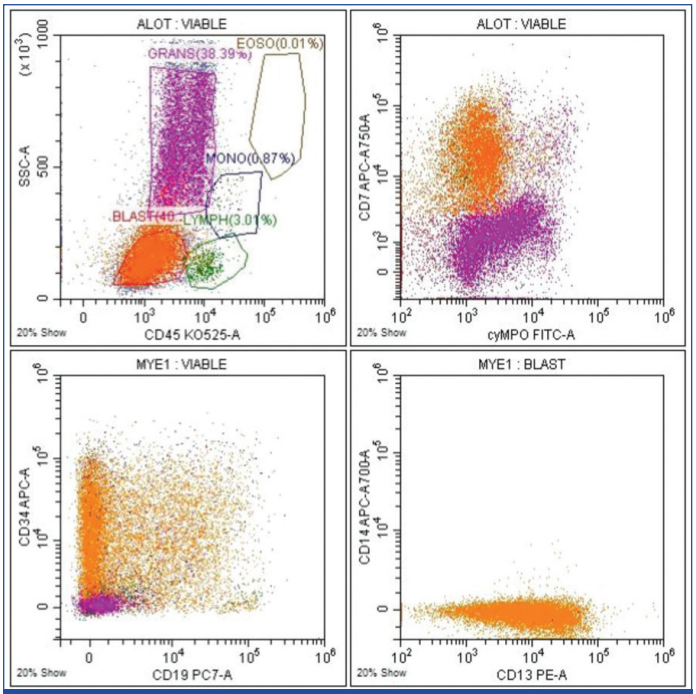
[Table/Fig-1]: Distribution of type of aberrancy in B-ALL and T-ALL cases.



[Table/Fig-2]: Flow cytometry of B-ALL with aberrant CD33 and CD13: Blasts were gated by selecting events with low to moderate CD45 expression and low side scatter using CD45 vs SSC plot. Blasts expresses strong CD19, moderate cyCd79a, CD13, CD33 and CD34 and lacks expression of cy CD3, cyMPO CD34 and HLADR.

In T-ALL, CD13 (40%) was the most common aberrant antigen seen in 3 (42.9%) [Table/Fig-1] followed by CD33 seen in 2 (30%) cases [Table/Fig-3].

The mean haemoglobin, TLC and platelet counts were higher in B-ALL cases expressing aberrant antigens compared to those with conventional phenotype. Similarly, in T-ALL, the mean haemoglobin and platelet count were higher in the aberrant phenotype group. However, statistical analysis revealed no significant association between mean haemoglobin levels, TLC, platelet count, or peripheral blood blast percentage between aberrant and conventional phenotypes in both B-cell and T-cell ALL [Table/Fig-4].



[Table/Fig-3]: Flow cytometry of T-ALL with aberrant CD13: Blasts (Orange colour) were gated by selecting events with low to moderate CD45 expression and low side scatter using CD45 vs SSC plot. Blasts expresses strong CD7, cyCD3 (not shown) moderate CD13 and CD34 and lacks expression of CD19, cyMPO CD14 and HLADR.

Out of 23 T-ALL cases, karyotyping was performed in six cases. Both conventional and aberrant phenotype groups had a normal karyotype, with no cytogenetic abnormalities detected.

In B-ALL, the t (9;22) translocation, involving the BCR-ABL1 fusion gene, was observed in seven cases with aberrant antigen expression, in contrast to only two case with a conventional B-ALL phenotype. BCR-ABL1 translocation in B-ALL was most commonly associated with aberrant CD33 [Table/Fig-6].

DISCUSSION

In the present study, 137 cases of acute leukaemia were diagnosed using flow cytometry. Among these, 114 cases (82.3%) were classified as B-ALL, while 23 cases (16.7%) were T-ALL. These findings align with previous research like Sivakumar M et al., reported 65 (86.6.6%) cases of B-cell lineage and 10 (13.3%) cases of T-cell lineage [14]. Similarly, Jalal SD et al., found 241 cases (85.5%) of B-ALL and 41 cases (14.5%) of T-ALL, Gupta et al., reported 273 cases (81.7%) of B-ALL and 61 cases (18.3%) of T-ALL [15,16]. Likewise, Gupta M et al., found 38 (88.4%) B-ALL cases and 5 (11.6%) T-ALL cases (11.6%) [17].

In the present study, the proportional frequency of B-ALL Aberrancies was 53 (46.5%), which aligned with the findings of Gupta M et al., Lopes TC et al., and Ahuja S and Malviya A [17-19]. The proportional frequency of T-ALL aberrancy was 7 (30.4%), which was consistent with the results of Khurram MM et al., Sivakumar M et al., and Suggs JL et al., [3,14,20]. As depicted in [Table/Fig-7]

Haematological parameters	B-ALL			T-ALL		
	Aberrant phenotype B-ALL (n=53)	Conventional phenotype B-ALL (n=61)	p-value	Aberrant phenotype T-ALL (n=7)	Conventional phenotype T-ALL (n=16)	p-value
Mean haemoglobin (g/dL)±SD	7.50±2.47	6.97±2.14	0.26	8.954±2.79	7.80±2.36	0.39
Mean TLC (/cu mm)±SD	81910±95040	53920±66120	0.35	34180±27160	86730±68520	0.06
Mean platelet count (/cu mm)±SD	71620±96130	46570±38780	0.71	130000±112960	60940±39490	0.08
Percentage of blasts in PB±SD	57.62±20.68	58.70±23.35	0.76	62.57±18.83	62.81±21.54	0.95

[Table/Fig-4]: Comparison of mean values of haematological parameters between conventional and aberrant phenotypes in B-ALL and T-ALL cases.
PB: Peripheral blood; SD: Standard deviation

Cytogenetic studies were performed on 65 samples from patients with ALL. Fourteen samples could not be analysed due to lack of cell growth {Mitotic Index (MI) zero}. Cytogenetic analysis was not conducted on the remaining 72 cases due to financial constraints and loss to follow-up.

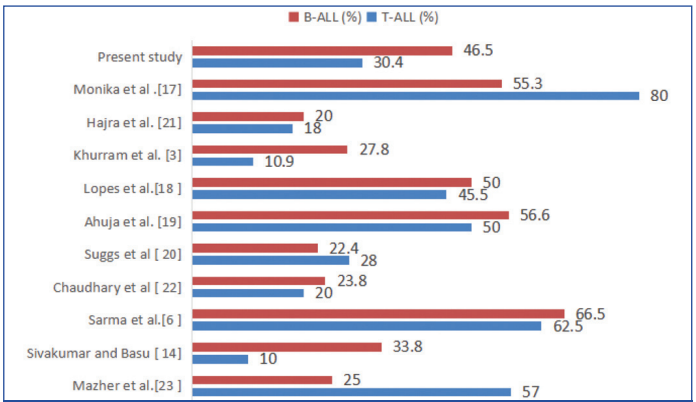
The study compared the presence of cytogenetic abnormalities between cases with aberrant antigen expression and those with a conventional phenotype in B-ALL [Table/Fig-5]. Among conventional phenotype subjects, 10 (45.5%) had an abnormal karyotype, while 12 (54.5%) had a normal karyotype. For those with an aberrant phenotype, 9 (39.1%) had an abnormal karyotype, and 14 (60.9%) had a normal karyotype. There was no significant difference in karyotype distribution between conventional and aberrant phenotypes (p=0.25), suggesting phenotype type did not significantly impact karyotype status.

Karyotypes	Conventional phenotype		Aberrant phenotype		p-value
	n	%	n	%	
t (1; 19)(q23; p13.3)	1	4.50%	0	0.00%	0.25
t (4; 11)(q21; q23)	1	4.50%	1	4.30%	
t (9; 22)(q34; q11.2)	2	9.10%	7	30.40%	
t (12; 21)(p13; q22)	1	4.50%	0	0.00%	
t (9; 12)(q22; q24)	1	4.50%	0	0.00%	
Hyperdiploidy	2	9.10%	1	4.30%	
Complex karyotypes	2	9.10%	0	0.00%	
Normal karyotype	12	54.50%	14	60.90%	

[Table/Fig-5]: Comparison of cytogenetic findings between conventional and aberrant phenotype in B-ALL.

Variables	t (9;22)	t (4;11)	Hyperdiploidy	Normal karyotype
CD33	3	1	1	6
CD13, CD33	2	0	0	4
CD13, CD33, CD7	1	0	0	0
CD2	0	0	0	2
CD33+CD15	0	0	0	1
CD15	0	0	0	1
CD13	1	0	0	0

[Table/Fig-6]: Distribution of cytogenetic abnormalities in B-ALL cases with aberrant antigen expression.



[Table/Fig-7]: Comparison of proportional frequency of aberrant B & T ALL cases in different studies [3,6,14,17-23].

[3,6,14,17-23], the prevalence rate of aberrant antigens according to various studies ranged from 10 to 65% [17-24]. This wide

variation in prevalence could be attributed to several factors, such as the use of different reagents against the CD surface antigens, the differences in phenotypic characteristics of blast cells between children and adult patients, ethnic differences, and the variations in flow cytometer instruments, reagents, and cut-off values for the various antibodies used.

In the present study, it was found CD33 was the common aberrant marker in B-ALL. This result was similar to findings by Bhushan B et al., Sivakumar M et al., and Momani A et al., [9,14,24]. CD13 emerged as the most frequent aberrancy in T-ALL in the current study. This finding was consistent with the results observed in previous studies by Sarma A et al., Mazher N et al., Momani A et al., and others [6,23,24].

In the present study and the studies by Bhushan B et al., and Laxminarayana K et al., no significant association was observed in haematological parameters (mean haemoglobin levels, TLC, platelet count, and percentage of blasts in peripheral blood) between conventional and aberrant phenotypes of B and T ALL [9,25]. However, in the studies by Sharma M et al., and Ahuja S and Malviya A aberrant phenotypes were associated with a lower mean TLC and a reduced number of blasts in peripheral blood compared to conventional B-ALL cases [4,19].

The study revealed that abnormal expression of CD33 and CD13 in B-ALL was most commonly seen with t (9; 22)(q34; q11.2). This observation was supported by multiple studies, including those by Vitale A et al., Swerdlow S et al., Seegmiller AC et al., Khalidi HS et al., Gupta, and Jaso J et al., [7,26-29].

The study also investigated the association between aberrant antigen expression in B-ALL and cytogenetic abnormalities. The findings of this study revealed no significant association. This lack of association was consistent with the findings of studies by Vitale A et al., and Shivakumar M et al., [7,14].

Limitation(s)

Cytogenetic study was available in few cases only, so the association with immunophenotypic parameters was performed solely for those cases.

CONCLUSION(S)

The present study demonstrated that aberrant antigen expression is more common in B-ALL as compared to T-ALL. Differences in cytogenetic abnormalities or hematological parameters, including haemoglobin levels, TLC, platelet count and blast percentage were found between aberrant and conventional cases, but were not statistically significant. Identifying these aberrant phenotypes aids in monitoring MRD and guiding alternative treatment strategies. Some aberrant antigens may serve as therapeutic targets, like anti-CD33 monoclonal antibodies. However long-term follow-up is necessary to identify prognostic implications of aberrancies and chromosomal abnormalities.

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