



Characterization of the Immunophenotypic and Demographic Landscape of Early T-Cell Precursor Acute Lymphoblastic Leukemia (ETP-ALL): A Comparative Institutional Analysis



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Abstract

Background: Early T-cell precursor acute lymphoblastic leukemia (ETP-ALL) is a biologically distinct malignancy characterized by an immunophenotypic signature reflecting early intrathymic developmental arrest and stem-cell/myeloid plasticity. Precise differentiation of ETP-ALL from conventional non-ETP T-ALL is clinically crucial because of their distinct biological characteristics, therapeutic approaches, and prognostic implications. **Objective:** The purpose of the present study was to determine the prevalence and demographic characteristics of Early T-cell Precursor Acute Lymphoblastic Leukemia (ETP-ALL) among patients with T-cell acute lymphoblastic leukemia (T-ALL) and to evaluate the immunophenotypic features that distinguish ETP-ALL from conventional non-ETP T-ALL. **Methodology:** Patients diagnosed with T-cell acute lymphoblastic leukemia (T-ALL) were included in this study. Cases were subclassified according to predefined immunophenotypic criteria. The ETP-ALL group (n = 19) was defined by cytoplasmic CD3 (cyCD3) and CD7 positivity, absence of CD1a and CD8 expression, and lack of myeloperoxidase (MPO) expression. The non-ETP T-ALL group (n = 51) was characterized by cyCD3 positivity and MPO negativity. Clinical and immunophenotypic parameters, including patient age and the expression levels of 18 flow cytometric markers, together with the progenitor-associated antigen CD10, were quantitatively assessed. Comparisons between groups were performed using Welch's t-test. **Results:** The mean age of the entire cohort was 19.8 years. Patients with ETP-ALL presented at a significantly older age compared to those with non-ETP T-ALL (mean: 25.3 vs. 17.8 years; $p < 0.05$). Overall cyCD3 expression exhibited a left-skewed baseline distribution across the cohort (mean: 75.5%; median: 83.0%). Out of 18 markers, exactly 9 (50.0%) demonstrated statistically significant differences ($p < 0.05$) between the subgroups, with CD8 emerging as the top discriminating maturation parameter ($p < 0.001$). ETP-ALL blasts exhibited a classic immature progenitor and cross-lineage myeloid phenotype, with marked overexpression of CD13 (53.74%), CD33 (42.73%), CD117 (31.13%), CD34 (42.32%), and HLA-DR (39.15%). Furthermore, cohort-wide profiling of CD10 revealed positive expression in 32 out of 70 patients (45.71%), manifesting a wide spectrum of fluorescence intensity from dim to bright (>90% positivity thresholds). **Conclusion:** Our results statistically validate the stringent cross-lineage immunophenotypic characteristics of ETP-ALL. By demonstrating significant expression of myeloid-associated markers alongside heterogeneous CD10 positivity without definitive B-lineage commitment, this study confirms the retention of a primitive and highly plastic progenitor phenotype. Notably, ETP-ALL accounted for 27.5% of all T-ALL cases, a prevalence higher than that reported in many published series, and showed a marked male predominance (M:F = 8.5:1) and older age at presentation, suggesting potential demographic differences within our population. [Bangladesh Journal of Infectious Diseases, June 2026;13(1):71-78]

Keywords: ETP-ALL; T-ALL; Flow-cytometry immunophenotyping; myeloid antigen

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Introduction

T-cell acute lymphoblastic leukemia (T-ALL) is an aggressive hematologic malignancy arising from the malignant transformation and clonal proliferation of immature T-lymphoid progenitor cells. It accounts for approximately 15 to 25.0% of pediatric and 20 to 25.0% of adult acute lymphoblastic leukemia (ALL) cases and is characterized by marked biological and clinical heterogeneity^{1,2}. Advances in immunophenotyping and molecular profiling have identified several distinct subgroups within T-ALL, among which Early T-cell Precursor Acute Lymphoblastic Leukemia (ETP-ALL) represents a unique and high-risk entity². ETP-ALL was first described by Coustan-Smith and colleagues in 2009 as a biologically distinct subtype of T-ALL characterized by an immunophenotypic profile resembling the earliest thymic progenitors³. These leukemic cells originate from thymocytes arrested at the double-negative (DN1) stage of T-cell development, a developmental phase in which cells retain multilineage differentiation potential and exhibit features of both lymphoid and myeloid progenitors^{3,4}.

Consequently, ETP-ALL occupies a unique biological position between conventional T-lymphoblastic leukemia and stem-cell/myeloid malignancies^{4,5}. The diagnosis of ETP-ALL relies primarily on multiparametric flow cytometry. According to the World Health Organization (WHO) and International Consensus Classification (ICC), ETP-ALL is characterized by expression of cytoplasmic CD3 and CD7, absent or markedly reduced expression of mature T-cell markers such as CD1a, CD8, and CD5, together with aberrant expression of stem-cell and myeloid-associated antigens including CD34, CD117, CD13, CD33, and HLA-DR⁶⁻⁸. This immunophenotypic signature reflects profound developmental arrest and retention of primitive hematopoietic programs. Beyond its distinctive phenotype, ETP-ALL exhibits a unique molecular landscape. Genomic studies have demonstrated frequent alterations involving cytokine receptor signaling pathways, hematopoietic stem-cell regulatory genes, and epigenetic modifiers, distinguishing ETP-ALL from conventional T-ALL^{5,9-10}. Mutations affecting FLT3, DNMT3A, EZH2, RUNX1, JAK-STAT signaling components, and RAS pathway genes are commonly observed, further supporting its stem-cell-like biology^{5,9}.

Clinically, ETP-ALL has historically been associated with high leukocyte counts, resistance to

induction chemotherapy, increased rates of measurable residual disease (MRD), and inferior survival outcomes compared with non-ETP T-ALL¹¹⁻¹⁴. Although contemporary treatment protocols incorporating intensive risk-adapted therapy have improved outcomes, ETP-ALL remains a clinically challenging subtype requiring accurate identification and close therapeutic monitoring^{11-12,15}.

Several studies from Europe, North America, and Asia have reported varying frequencies of ETP-ALL ranging from 10.0% to 30.0% of all T-ALL cases, reflecting differences in diagnostic criteria, population demographics, and referral patterns^{3,11,16}. Reports from South Asia remain relatively limited, and data describing the immunophenotypic characteristics of ETP-ALL in regional populations are scarce¹⁷⁻¹⁸. The present study was undertaken to characterize the demographic and immunophenotypic landscape of ETP-ALL in comparison with conventional non-ETP T-ALL in a cohort of 70 patients diagnosed at our institution. By evaluating the expression patterns of key T-lineage, progenitor, and myeloid-associated markers, as well as the distribution of CD10 expression, we aimed to statistically validate the distinctive biological profile of ETP-ALL and contribute additional data from a South Asian population. Understanding these phenotypic differences may facilitate improved diagnostic accuracy, risk stratification, and future therapeutic decision-making in patients with T-cell acute lymphoblastic leukemia.

Methodology

Study Settings and Population: This retrospective observational study included patients diagnosed with T-cell acute lymphoblastic leukemia (T-ALL) from both inpatient and outpatient services of the Department of Hematology, Bangladesh Medical University (BMU), Dhaka, Bangladesh, during the period from January 2024 to December 2025. Immunophenotypic analysis was performed with technical and laboratory support from the Hematology Laboratory of Ibn Sina Diagnostic & Imaging Centre, Dhanmondi, Dhaka, Bangladesh.

Patient Cohort and Subtype Classification: A total of 70 patients diagnosed with T-ALL were evaluated at our institution. Subtype classification was defined according to rigorous immunophenotypic criteria. The ETP-ALL subgroup (n = 19, 27.5%) was strictly defined by the uniform, mandatory presence of cytoplasmic CD3 (cyCD3; 100.0% positive) and CD7 (100.0%

positive), alongside the complete absence of mature T-lineage markers CD1a (100.0% negative) and CD8 (100.0% negative), and the absolute exclusion of myeloid-specific myeloperoxidase (Anti-MPO; 100.0% negative). The comparison group consisted of typical committed non-ETP T-ALL cases ($n = 51$, 72.5%), which were immunophenotypically anchored by the universal presence of cytoplasmic CD3 (cyCD3; 100.0% positive) and the corresponding absence of myeloperoxidase (Anti-MPO; 100.0% negative). One patient with baseline clinical metrics was excluded from specific marker sub-analyses due to incomplete panel availability.

Immunophenotypic Profiling: Multiparameter flow cytometric immunophenotyping was performed on bone marrow aspirate and/or peripheral blood samples using a BD FACSLyric™ flow cytometer (BD Biosciences, San Jose, CA, USA). Approximately 30,000 events were acquired per tube for each sample according to standard laboratory protocols. The antibody panel for evaluation of T cell acute lymphoblastic leukemia includes CD45, cy MPO, CD34, cy CD3, cyCD79a, CD19, TdT, smCD3, CD1a, CD56, CD5, CD4, CD2, CD7, CD8, CD20, CD10, CD64, CD13, CD33, CD14, HLA-DR, CD117. Fluorochrome conjugated monoclonal antibodies were obtained from BD Bioscience (country) and Fluorochromes used included FITC, PE, perCP Cy5.5, V450, V500c, PE-Cy7, APC and APC-H7. Positive antigen expression was defined as expression in $\geq 20\%$ of gated leukemic blast cells. Negative expression was determined by comparison with appropriate internal negative cell populations and autofluorescence controls. Expression levels were recorded as the percentage of gated leukemic blasts positive for each marker. CD10 expression was additionally evaluated across the entire cohort to assess its frequency and intensity as a potential cross-lineage progenitor-associated marker.

Statistical Analysis: Descriptive statistics were calculated for the entire cohort. Continuous variables, including patient age and specific immunophenotypic marker expression percentages, were compared between the ETP-ALL and non-ETP-ALL subgroups using Welch's t-test. This approach was selected to robustly account for unequal variances and sample sizes between the two groups. Statistical significance was defined a priori as a p -value < 0.05 . All statistical computations were processed using standard analytical software.

Ethical Consideration: All procedures of the present study were carried out in accordance with

the principles for human investigations (i.e., Helsinki Declaration 2013) and also with the ethical guidelines of the Institutional Research Ethics Committee. Formal ethics approval was granted by the local ethics committee. Participants in the study were informed about the procedure and purpose of the study and confidentiality of information provided. All participants consented willingly to be a part of the study during the data collection periods. All data were collected anonymously and were analyzed using the coding system.

Results

Clinical Demographics and Baseline Cohort Characteristics: The mean age of the entire cohort was 19.8 years. When stratified by leukemia subtype, patients in the ETP-ALL subgroup were notably older, exhibiting a mean age of 25.3 years, compared to a mean age of 17.8 years in the non-ETP-ALL subgroup ($p < 0.05$). Across the entire 70-patient cohort, cyCD3 expression demonstrated robust but heterogeneous levels. The overall average cyCD3 expression was 75.5%, with a median expression of 83.0%. This left-skewed distribution indicates high-intensity baseline expression in the majority of the population, although a subset of cases shows lower values. Of the 18 immunophenotypic markers evaluated, exactly half (9) showed statistically significant differences ($p < 0.05$) between the ETP-ALL and non-ETP-ALL cohorts. Among these, CD8 emerged as the strongest discriminating marker, showing the most highly significant down-regulation in the ETP-ALL subgroup ($p < 0.001$) (Table 1).

Immunophenotypic Profiles of ETP-ALL versus Non-ETP-ALL: The comparative immunophenotypic signature of ETP-ALL ($n = 19$) versus non-ETP-ALL ($n = 51$) cases revealed stark lineage divergences. ETP-ALL cases demonstrated a classic myeloid/stem-cell phenotype, showing marked over-expression of the myeloid markers CD13 (53.74% vs. 12.04%, $p < 0.001$), CD33 (42.73% vs. 7.02%, $p < 0.001$), and CD117 (31.13% vs. 3.16%, $p = 0.010$). Immature and progenitor-associated markers were also significantly elevated in the ETP-ALL subgroup, including CD34 (42.32% vs. 12.57%, $p < 0.001$) and HLA-DR (39.15% vs. 11.56%, $p = 0.007$). Conversely, ETP-ALL patients exhibited a profound absence or down-regulation of mature T-cell differentiation markers. Beyond the strict categorical baseline exclusions of CD8 and CD1a, surface CD3 expression (smCD3: 3.68% vs. 22.20%, $p < 0.001$) and CD5 expression (8.39% vs.

34.01%, $p < 0.001$) were significantly depressed. No statistically significant differences were observed between the two groups for pan-T-cell markers CD2 or CD7 ($p \geq 0.069$). Similarly, expression levels of CD19, Anti-TdT, CD79a, CD45, and CD56 did not differ significantly between the ETP-ALL and non-ETP-ALL cohorts ($p \geq 0.066$).

CD10 Expression Matrix: Analysis of the B-lymphoid associated marker CD10 demonstrated broad heterogeneity across the study population. Out of the 70 total evaluable T-ALL cases, CD10

positivity was verified in 32 patients, yielding an overall cohort positivity rate of 45.71%. Positive cases displayed a highly variable spectrum of fluorescence intensity, spanning from dim to moderate and bright signals. Markedly prominent, bright expressions exceeding 90.0% positivity thresholds (e.g., individual tracking showing 90.6%, 91.06%, 97.0%, and 98.0% blast coverage) were observed in a distinct subset of the cohort, reinforcing the presence of active primitive-lymphoid surface programs.

Table 2: Comparison of Anti-GAD and Proinsulin Concentrations between Diabetic Patients and Control Group According to Gender

Parameter / Marker	Total Cohort (N = 70)	ETP-ALL Mean (n = 19)	Non-ETP-ALL Mean (n = 51)	Absolute Difference	p-value ¹	Significance Status
Demographics						
Mean Age (years)	19.8	25.3	17.8	+7.50 yrs	< 0.05	Significant
T-Lineage Anchors						
cyCD3	100% Pos / 75.5% ²	100% Positive	100% Positive	—	—	Mandatory/Diagnostic
CD7	—	100% Positive	93.86%	+6.14%	0.088	Diagnostic Exclusion
CD1a	—	100% Negative	29.59%	-29.59%	< 0.001	Significant
CD8	—	100% Negative	50.96%	-50.96%	< 0.001	Significant (Top)
smCD3	—	3.68%	22.20%	-18.53%	< 0.001	Significant
CD5	—	8.39%	34.01%	-25.62%	< 0.001	Significant
CD2	—	53.67%	73.81%	-20.14%	0.069	Not Significant
Lineage Exclusions						
Anti-MPO	100% Negative	100% Negative	100% Negative	0.00%	0.864	Lineage Exclusion
Cross-Lineage / Progenitor						
CD10 (Overall Pos: 45.71%)	32/70 Positive	Evaluated	Evaluated	—	—	Cross-Lineage Marker
CD13	—	53.74%	12.04%	+41.70%	< 0.001	Significant
CD33	—	42.73%	7.02%	+35.71%	< 0.001	Significant
CD34	—	42.32%	12.57%	+29.75%	< 0.001	Significant
CD117	—	31.13%	3.16%	+27.96%	0.010	Significant
HLA-DR	—	39.15%	11.56%	+27.60%	0.007	Significant
Other Markers						
CD19	—	11.69%	1.55%	+10.15%	0.066	Not Significant
Anti-TdT	—	56.16%	63.83%	-7.66%	0.278	Not Significant
CD79a	—	12.26%	6.39%	+5.86%	0.412	Not Significant
CD45	—	90.79%	92.86%	-2.07%	0.552	Not Significant
CD56	—	38.24%	31.81%	+6.44%	0.575	Not Significant

¹ Calculated via Welch's t-test. Markers are grouped logically by lineage and diagnostic hierarchy.

² Overall cohort mean cyCD3 expression was 75.5%; median expression was 83.0%.

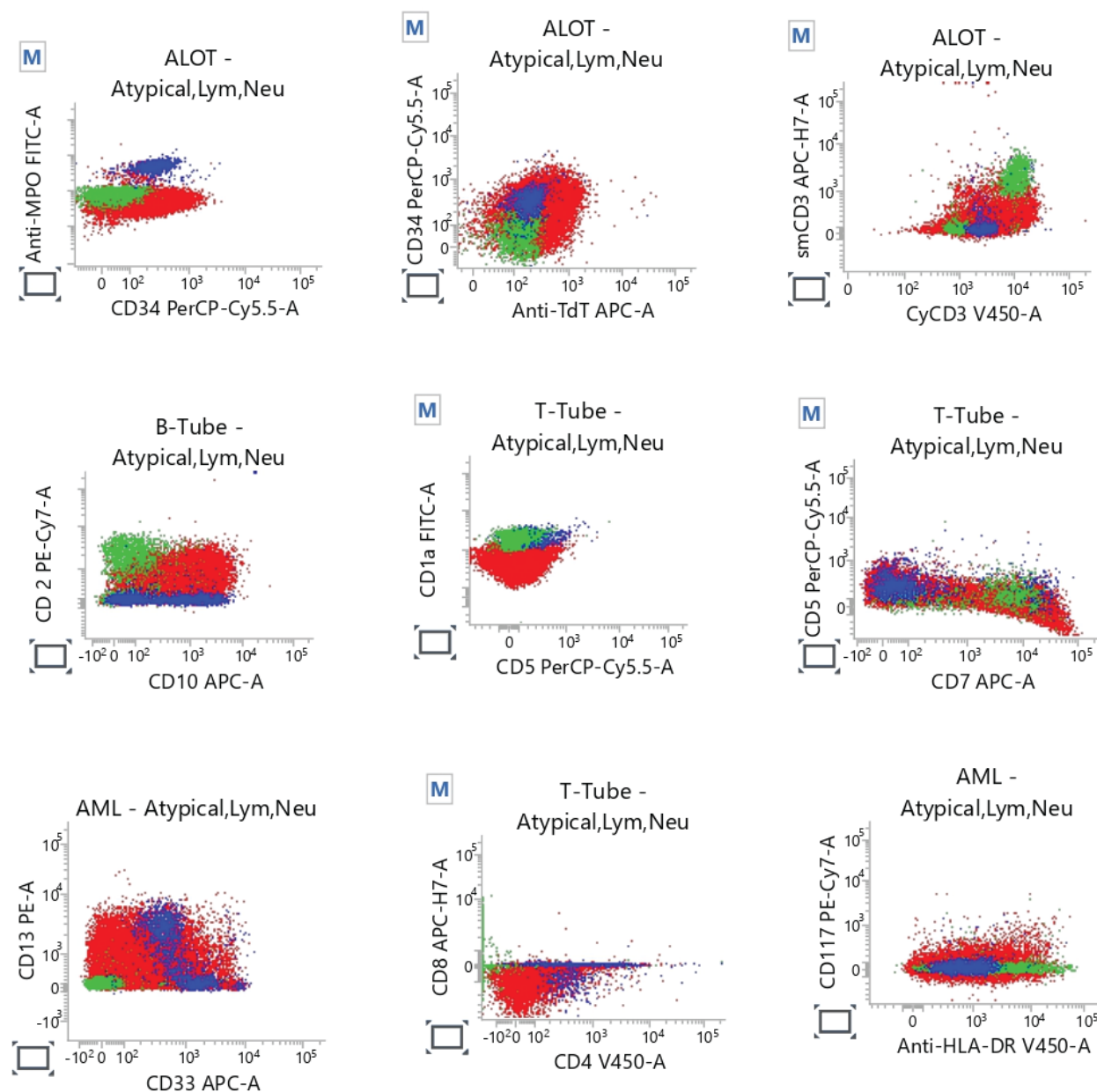


Figure I: Flow cytometric immunophenotypic characterization of an Early T-cell Precursor Acute Lymphoblastic Leukemia (ETP-ALL) case.

The neoplastic blast population (red dots) exhibits a characteristic ETP-ALL immunophenotype, including expression of cytoplasmic CD3 (cyCD3), CD2, CD7, CD10, CD13, CD33, CD34, HLA-DR, and TdT. The blasts are negative for myeloperoxidase (MPO), surface CD3 (smCD3), CD1a, CD4, CD5, CD8, and CD117. The combination of absent mature T-cell markers together with expression of progenitor and myeloid-associated antigens supports the diagnosis of ETP-ALL and reflects arrest at an early stage of T-cell differentiation.

Discussion

Early T-cell precursor acute lymphoblastic leukemia (ETP-ALL) is a distinct subtype of T-ALL characterized by an immature immunophenotype and the retention of multilineage differentiation potential^{2,3}. In the present study, the immunophenotypic profile of our ETP-ALL cohort closely aligned with the diagnostic criteria proposed by the World Health Organization (WHO) and the International Consensus Classification (ICC)⁵⁻⁷. The uniform expression of cytoplasmic CD3 and CD7, together

with the absence of CD1a, CD8, and myeloperoxidase (MPO), supports the classification of these cases as ETP-ALL and reflects arrest at a very early stage of T-cell differentiation^{2,3,5-7}.

A defining biological feature of ETP-ALL is its overlap with primitive hematopoietic progenitors^{2,4}. This characteristic was reflected in our cohort by the frequent expression of myeloid-associated antigens, particularly CD13, CD33, and CD117. The presence of these markers supports previous observations that ETP-ALL retains stem cell-like and myeloid differentiation programs despite maintaining a T-lineage identity^{4,8-9}. Such immunophenotypic plasticity has been associated with treatment resistance, increased risk of induction failure, and poorer clinical outcomes compared with conventional T-ALL¹⁰⁻¹³.

Our findings are consistent with the seminal work of Coustan-Smith et al², who first defined ETP-ALL as a subgroup lacking CD1a and CD8 expression while demonstrating reduced expression of mature T-cell markers. Similarly, our ETP-ALL cases exhibited absent CD1a and CD8 expression and markedly lower CD5 expression compared with non-ETP T-ALL, supporting the concept that these leukemic cells originate from an early thymic precursor stage before definitive T-cell commitment^{2,4}.

The prevalence of ETP-ALL in our study was higher than that reported in several regional and international series. While studies from Western India have reported ETP-ALL frequencies of approximately 10–15% among T-ALL cases¹⁵, ETP-ALL accounted for 27.5% of our cohort. This difference may reflect referral bias associated with our tertiary-care center, population-specific biological variation, or the relatively higher proportion of adult patients included in our study population. Similar observations have been reported in studies from the Indian subcontinent, where adult T-ALL cohorts frequently demonstrate a greater representation of immature immunophenotypic subtypes^{12,15-16}.

An important observation in our study was the significantly older age at presentation among ETP-ALL patients compared with those with non-ETP T-ALL. The mean age of patients with ETP-ALL was 25.3 years, compared with 17.8 years in the non-ETP group. Furthermore, ETP-ALL demonstrated a striking male predominance (17 males and 2 females; M:F = 8.5:1). This finding is consistent with previous reports suggesting that ETP-ALL is relatively more

common in adolescents and adults than in younger pediatric populations^{12-13,18}. The predominance of older patients may partially contribute to the historically poorer outcomes observed in ETP-ALL, as adult patients often tolerate intensive pediatric-inspired treatment protocols less effectively¹⁰⁻¹³.

The expression of CD10 in a substantial proportion of cases deserves particular consideration. Although CD10 is traditionally regarded as a marker of early B-cell differentiation, its expression in ETP-ALL does not necessarily indicate true B-lineage commitment. Instead, CD10 expression likely reflects the primitive developmental status of early hematopoietic progenitors before complete lineage restriction. The coexistence of CD10 with myeloid-associated markers further supports the concept that ETP-ALL arises from an immature precursor population with preserved multilineage transcriptional potential^{3,4,9}.

The present study highlights the value of comprehensive multiparametric flow cytometry in accurately distinguishing ETP-ALL from conventional T-ALL. Precise immunophenotypic classification is clinically important because ETP-ALL is associated with distinct biological behavior, therapeutic responses, and prognostic implications^{2,10-14}. Early identification of this high-risk subgroup may facilitate improved risk stratification and support the development of more individualized treatment approaches. Recent studies have further demonstrated the prognostic value of measurable residual disease (MRD) assessment in T-ALL and ETP-ALL, emphasizing the importance of accurate baseline immunophenotypic characterization^{14,17}.

The main limitations of this study include its single-center design and the absence of molecular, cytogenetic, and measurable residual disease (MRD) data, which could have provided additional prognostic insights. Future multicenter studies integrating immunophenotypic, genomic, and clinical outcome data are warranted to further characterize the biological heterogeneity and clinical significance of ETP-ALL in South Asian populations^{4,8-9}.

Our findings confirm that ETP-ALL exhibits a characteristic immature immunophenotypic profile marked by loss of mature T-cell markers and frequent expression of myeloid-associated antigens. ETP-ALL constituted 27.5% of all T-ALL cases in our cohort, a prevalence higher than that reported in many regional and

international studies, and demonstrated a marked male predominance (M:F = 8.5:1) and older age at presentation^{12,15-16}. These observations emphasize the importance of accurate flow cytometric identification of this biologically distinct and clinically relevant subtype of T-ALL.

Conclusion

In conclusion, our findings demonstrate significant immunophenotypic and demographic differences between ETP-ALL and non-ETP T-ALL. ETP-ALL was characterized by an immature T-cell phenotype, retention of stem-cell and myeloid-associated markers, and an older age at presentation, highlighting its biological distinctiveness. Furthermore, ETP-ALL constituted T-ALL cases in our cohort, with a prevalence higher than that reported in most regional and international studies, and exhibited a striking male predominance. These findings emphasize the importance of comprehensive flow cytometric evaluation for accurate identification of ETP-ALL and support its recognition as a distinct high-risk subtype requiring appropriate risk stratification and clinical management.

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Conflict of interest:

The authors declare that they have no conflicts of interest related to this study. The authors alone are responsible for the content and writing of this manuscript.

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Authors' contributions

Conception and design: Rahman F, Haque SA; Acquisition, analysis, and interpretation of data: Rahaman MFU, Akter N; Data collection, consultation, and surgical team: Rahaman MFU, Akter N, Kabir AL; Manuscript drafting and revising it critically: Rahman F, Haque SA; All authors read and approved the final manuscript.

Data Availability

The datasets generated and analyzed during this study are not publicly available due to patient confidentiality and institutional data protection policies. However, anonymized data may be made available from the corresponding author upon reasonable request, subject to approval by the institutional ethics committee.

Ethics Approval and Consent to Participate

Ethical approval for the study was obtained from the Institutional Review Board. As this was a prospective study the written informed consent was obtained from all study participants. All methods were performed in accordance with the relevant guidelines and regulations. The ethical approval was obtained from July 2024

to December 2024.

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