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Original Article

Mixed Phenotype Acute Leukemia: Clinical Features and Outcomes in an Observational Cohort

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ABSTRACT

Background: Mixed phenotype acute leukemia (MPAL) is a rare and biologically heterogeneous acute leukemia characterized by the co-expression of myeloid and lymphoid lineage markers. Owing to diagnostic complexity, genetic diversity, and lack of standardized therapeutic strategies, MPAL is associated with poor clinical outcomes. This study aimed to evaluate the clinical presentation, immunophenotypic and molecular characteristics, treatment response, measurable residual disease (MRD) dynamics, and survival outcomes in patients with MPAL.
Methods: This analytical observational study included 80 pediatric and adult patients diagnosed with MPAL according to World Health Organization (WHO) 2016 criteria. Clinical and laboratory data were collected at diagnosis. Immunophenotyping was performed using multicolor flow cytometry, and molecular and cytogenetic analyses assessed recurrent genetic abnormalities, including BCR::ABL1 and KMT2A rearrangements. Patients received myeloid- or lymphoid-directed induction therapy based on institutional protocols, with BCR::ABL1-positive patients additionally receiving imatinib. Treatment response and MRD were evaluated on Days 14, 28, and 42. Survival outcomes were analyzed using Kaplan–Meier methods.
Results: The cohort showed equal representation of children and adults, with a male predominance. B-lymphoid/myeloid MPAL was the most common immunophenotype. Molecular abnormalities were identified in a subset of patients, with BCR::ABL1 fusion being the most frequent. Although more than half of patients achieved early morphologic remission, MRD remained detectable in the majority during induction therapy. Overall survival was poor, with a 12-month survival of approximately 48%. KMT2A rearrangements were associated with adverse outcomes, whereas allogeneic hematopoietic stem cell transplantation was linked to significantly improved survival.
Conclusion: MPAL is an aggressive leukemia with unsatisfactory survival outcomes. Comprehensive immunophenotypic and molecular characterization, early MRD assessment, and timely consideration of transplantation are essential for improving prognosis.

Keywords: *Mixed phenotype acute leukemia; MPAL; immunophenotyping; BCR::ABL1; measurable residual disease; stem cell transplantation.*

INTRODUCTION

Mixed phenotype acute leukemia (MPAL) is a rare, high-risk acute leukemia in which leukemic blasts fulfill lineage-defining criteria for more than one hematopoietic lineage, most commonly myeloid with either B- or T-lymphoid differentiation. MPAL accounts for an estimated 2–5% of all acute leukemias, but its true frequency varies by diagnostic criteria and availability of advanced immunophenotyping and genetics. [1,2]

Accurate diagnosis is challenging and has evolved over time. Earlier scoring systems (e.g., EGIL) were gradually replaced by World Health Organization (WHO) frameworks that emphasize strong lineage-specific markers—particularly myeloperoxidase (MPO) for myeloid lineage and cytoplasmic/surface CD3 for T-lineage—with defined criteria for B-lineage assignment. [3,4] Recent updates in WHO-HEM5 and contemporary reviews further refine the category of acute leukemias of ambiguous lineage, including genetically defined MPAL subsets. [5–7]

Biologically, MPAL is heterogeneous rather than a single entity. Large series have shown aggressive clinical presentation, frequent cytopenias, and substantial marrow blast burden, often accompanied by extramedullary involvement. [2,3] Genomic studies demonstrate that MPAL may arise from early hematopoietic progenitors with lineage plasticity, and that B/myeloid and T/myeloid MPAL can show distinct molecular patterns. [8,9]

From a molecular standpoint, WHO recognizes clinically relevant subgroups such as MPAL with BCR::ABL1 and MPAL with KMT2A rearrangement, alongside MPAL not otherwise specified. [4–6] BCR::ABL1 positivity provides a rationale for adding tyrosine kinase inhibitors, whereas KMT2A rearrangement is consistently linked to adverse biology in acute leukemias and may contribute to poor outcomes in MPAL. [6,10,11]

Therapeutic strategies remain controversial due to limited prospective trials. Retrospective analyses and meta-analyses generally suggest improved outcomes with ALL-like induction approaches for many patients, while allogeneic hematopoietic stem cell transplantation (HSCT) may offer the most durable disease control in high-risk disease or persistent measurable residual disease (MRD). [12–14] MRD has emerged as a key prognostic marker in MPAL, with multiple pediatric and mixed-age studies linking early MRD clearance to superior survival and guiding risk-adapted intensification and transplant decisions. [15–17]

Finally, although MPAL is uncommon, it contributes to the overall leukemia burden, which remains substantial worldwide. Contemporary global estimates highlight hundreds of thousands of leukemia incident cases and deaths annually and underscore persistent disparities across regions. [18–21]

In this context, the present study describes the clinical features, immunophenotypic patterns, molecular/cytogenetic abnormalities, treatment approaches, MRD dynamics, and survival outcomes of MPAL, with particular attention to genotype-defined subsets and prognostic factors influencing overall survival. [22]

MATERIALS AND METHODS

This analytical observational study included **80 patients** diagnosed with mixed phenotype acute leukemia (MPAL). Patients were classified according to the **World Health Organization (WHO) 2016 criteria** for acute leukemias of ambiguous lineage. Both pediatric and adult patients were included. Clinical data were collected at presentation, including demographic details, clinical features, and laboratory parameters. Hematological evaluation comprised complete blood counts, peripheral blood smear examination, and bone marrow aspiration with assessment of cellularity and blast percentage.

Immunophenotypic characterization was performed on bone marrow and/or peripheral blood samples using **multicolor flow cytometry**. A comprehensive antibody panel was employed to identify myeloid markers (MPO, CD13, CD33, CD117), B-cell markers (CD19, CD10, CD22), T-cell markers (cytoplasmic CD3, CD7, CD2), and stem cell markers such as CD34. Cases were categorized as B-lymphoid/myeloid or T-lymphoid/myeloid MPAL.

Molecular analysis included screening for **BCR::ABL1 fusion**, **KMT2A rearrangements**, and selected mutations. Conventional cytogenetic analysis was performed where feasible, and karyotypes were interpreted using **ISCN guidelines**. Patients received myeloid- or lymphoid-directed induction therapy according to institutional protocols; BCR::ABL1-positive patients additionally received **imatinib**. Treatment response and **minimal residual disease (MRD)** were assessed on Days 14, 28, and 42. Survival outcomes were analyzed using **Kaplan–Meier methods**, with statistical analysis performed using SPSS and significance set at $p < 0.05$.

RESULTS

A total of 80 patients diagnosed with mixed phenotype acute leukemia were included in the analysis. The results summarize the baseline demographic, clinical, immunophenotypic, molecular, and cytogenetic characteristics of the cohort, followed by treatment modalities, early response assessment, minimal residual disease dynamics, and survival outcomes. Comparative analyses were performed to evaluate the impact of key molecular abnormalities, particularly BCR::ABL1 fusion and KMT2A rearrangements, as well as therapeutic interventions such as bone marrow transplantation, on treatment response and overall survival.

Table 1. Baseline Demographic, Clinical, and Laboratory Characteristics of the Study Cohort (N = 80)

Parameter	Value
Age (years), median (range)	20 (1–63)

Children (≤18 years), n (%)	40 (50.0)
Adults (>18 years), n (%)	40 (50.0)
Sex, n (%)	
Male	60 (75.0)
Female	20 (25.0)
Clinical manifestations, n (%)	
Fever	42 (52.5)
Hepatomegaly	33 (41.3)
Splenomegaly	32 (40.0)
Lymphadenopathy	35 (43.8)
CNS infiltration	10 (12.5)
Bone marrow failure signs	39 (48.8)
Laboratory characteristics	
TLC ($\times 10^3/\mu L$), median (range)	28.5 (1.2–880)
Hemoglobin (g/dL), mean ± SD	8.1 ± 2.3
Platelet count ($\times 10^3/\mu L$), median (range)	50 (6–390)
Peripheral blood blasts (%), median (range)	26 (0–98)
Bone marrow blasts (%), median (range)	80 (22–99)
Bone marrow cellularity, n (%)	
Normocellular	20 (25.0)
Hypercellular	60 (75.0)

The study cohort comprised 80 patients with mixed phenotype acute leukemia, with an equal distribution of children and adults (50% each) and a clear male predominance (75%). The median age was 20 years, reflecting inclusion of both pediatric and adult populations. Common clinical manifestations included fever (52.5%), lymphadenopathy (43.8%), hepatomegaly (41.3%), and splenomegaly (40%), indicating a high burden of systemic disease at presentation. Nearly half of the patients exhibited signs of bone marrow failure. Laboratory evaluation showed markedly abnormal hematological parameters, with a median total leukocyte count of 28.5 $\times 10^3/\mu L$, severe anemia (mean hemoglobin 8.1 g/dL), and thrombocytopenia (median platelet count 50 $\times 10^3/\mu L$). A high disease burden was evident from the elevated blast percentages in both peripheral blood and bone marrow. Bone marrow examination revealed predominantly hypercellular marrow in three-quarters of patients, consistent with aggressive leukemic infiltration.

Table 2. Immunophenotypic, Molecular, and Cytogenetic Characteristics (N = 80)

Parameter	**n (%)**
Immunophenotype	
B-lymphoid/Myeloid	64 (80.0)
T-lymphoid/Myeloid	16 (20.0)
Myeloid markers	
MPO positive	79 (98.8)
CD33 positive	75 (93.8)
CD13 positive	74 (92.5)
B-cell markers	
CD19 positive	61 (76.3)
CD10 positive	62 (77.5)
CD22 positive	52 (65.0)
T-cell markers	
CD7 positive	23 (28.8)
CD2 positive	17 (21.3)
CD3 positive	16 (20.0)
CD34 positive	66 (82.5)
Molecular abnormalities	
BCR::ABL1 fusion	11 (13.8)
KMT2A rearrangement	3 (3.8)
t(12;21)	2 (2.5)
JAK2 V617F	1 (1.3)
FLT3-ITD	1 (1.3)
Cytogenetics available	40 (50.0)
Abnormal karyotype	36/40 (90.0)
Chromosome 21 abnormality	16/40 (40.0)

Immunophenotypic analysis demonstrated that the majority of patients (80%) had a B-lymphoid/myeloid phenotype, while T-lymphoid/myeloid leukemia accounted for 20% of cases. Myeloid lineage commitment was confirmed by near-universal MPO expression, with high positivity for CD33 and CD13. B-cell marker expression, particularly CD19 and CD10, was frequently observed, whereas T-cell markers were present in a smaller subset, reflecting the heterogeneous lineage ambiguity characteristic of MPAL. CD34 expression was detected in over 80% of cases, supporting the stem/progenitor cell origin of the disease. Molecular abnormalities were identified in approximately one-quarter of patients, with BCR::ABL1 fusion being the most frequent genetic alteration (13.8%), followed by KMT2A rearrangements. Cytogenetic data, available in half of the cohort, revealed a high rate of abnormal karyotypes, and chromosome 21 abnormalities were observed in a substantial proportion, underscoring the genetic complexity of MPAL.

Table 3. Treatment Modalities and Early Response Assessment

Parameter	**n (%)**
Initial treatment protocol	
Myeloid-directed	62 (77.5)
Lymphoid-directed	18 (22.5)
Imatinib therapy	11 (13.8)
Bone marrow transplantation	6 (7.5)
Day 14 response	
Complete remission	46 (57.5)
Partial remission	6 (7.5)
Refractory disease	28 (35.0)
Day 14 MRD	
< 0.1%	6 (7.5)
≥ 0.1%	74 (92.5)
Day 28 MRD	
< 0.1%	13 (16.3)
≥ 0.1%	67 (83.7)
Day 42 MRD	
< 0.1%	18 (22.5)
≥ 0.1%	62 (77.5)

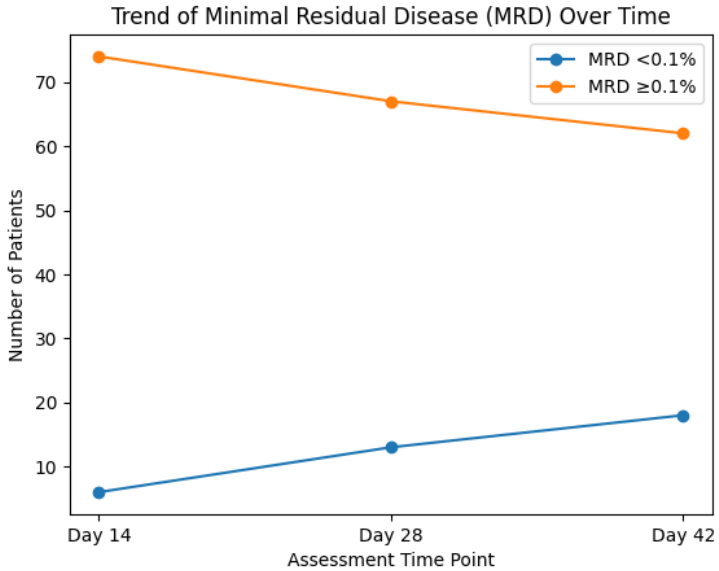

Figure 1. Trend of minimal residual disease (MRD) status during induction therapy in patients with mixed phenotype acute leukemia.

Most patients were treated with myeloid-directed induction protocols (77.5%), while a smaller proportion received lymphoid-directed therapy. Targeted therapy with imatinib was administered exclusively to BCR::ABL1-positive patients, and a limited subset underwent bone marrow transplantation. Early treatment response assessment demonstrated that more than half of the patients achieved complete remission by Day 14; however, a significant proportion remained refractory. Minimal residual disease (MRD) analysis revealed persistently high MRD levels in the majority of patients at early time points. Although the proportion of patients achieving MRD <0.1% increased progressively from Day 14 to Day 42, most patients continued to have detectable disease. This trend, illustrated in Figure 1, highlights the difficulty in achieving deep molecular remission during induction therapy and explains the high relapse risk associated with MPAL.

Table 4. Comparison of Clinical Characteristics and Treatment Response by BCR::ABL1 Status

Parameter	BCR::ABL1 Positive (n=11)	BCR::ABL1 Negative (n=69)	p-value
Adults, n (%)	9 (81.8)	31 (44.9)	**0.04**
Male sex, n (%)	9 (81.8)	51 (73.9)	0.61
B/Myeloid phenotype, n (%)	10 (90.9)	54 (78.3)	0.33
Complete remission, n (%)	9 (81.8)	52 (75.4)	0.72
MRD ≥0.1% on Day 42, n (%)	11 (100)	51 (73.9)	0.09

Comparison between BCR::ABL1-positive and BCR::ABL1-negative patients showed that the fusion gene was significantly more common in adults than in children. Other baseline characteristics, including sex distribution, immunophenotype, and complete remission rates, were comparable between the two groups. Although all BCR::ABL1-positive patients demonstrated persistent MRD ≥0.1% at Day 42, this association did not reach statistical significance. These findings suggest that while BCR::ABL1 represents a distinct molecular subtype of MPAL, its presence alone does not predict early treatment response or remission status, emphasizing the multifactorial nature of treatment resistance in this disease.

Table 5. Overall Survival According to Prognostic Factors (Kaplan–Meier Analysis)

Variable	n		Deaths	12-month OS (%)	Median OS (months)	p-value
Overall cohort	80		49	48.0	10.2	—
Children	40		24	50.5	12.4	0.94
Adults	40		25	45.6	10.0	
Male	60		35	53.8	13.6	0.26
Female	20		14	30.0	6.5	
BCR::ABL1 positive	11		4	58.0	NR	0.41
KMT2A rearranged	3		3	0	2.2	**0.001**
Bone marrow transplant	6		0	100	NR	**0.03**
No transplant	74		49	44.5	9.8	

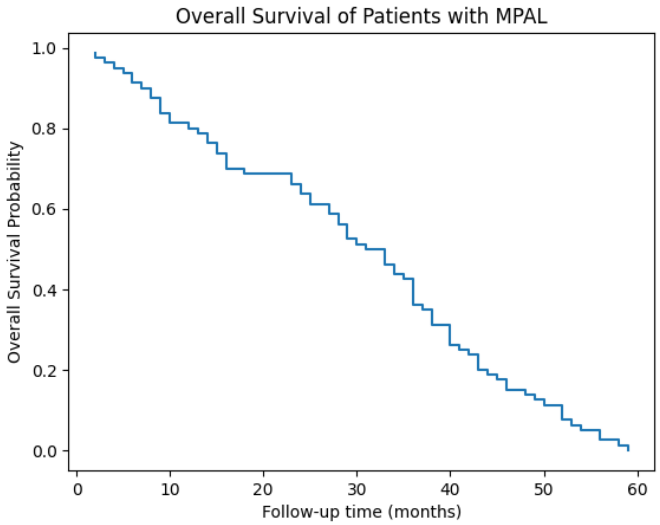

Figure 2. Overall survival curve of patients with mixed phenotype acute leukemia (MPAL). The Kaplan–Meier analysis demonstrates a median overall survival of approximately 10 months, with a cumulative survival of about 48% at 12 months, reflecting the aggressive nature and poor prognosis of MPAL despite contemporary treatment strategies.

At a median follow-up of approximately 10 months, nearly two-thirds of patients had died, reflecting the aggressive clinical course of MPAL. The cumulative overall survival at 12 months was 48%, with a median overall survival of just over 10 months. Survival outcomes did not differ significantly by age group or sex. Patients with BCR::ABL1 fusion showed a numerically higher survival, although this was not statistically significant. In contrast, patients with KMT2A rearrangements had an extremely poor prognosis, with no survivors beyond early follow-up. Bone marrow transplantation emerged as the most important favorable prognostic factor, with transplanted patients demonstrating significantly superior survival compared to those managed with chemotherapy alone. The Kaplan–Meier survival curve (Figure 2) clearly illustrates the poor overall survival of MPAL and underscores the potential survival benefit associated with transplantation in selected patients.

DISCUSSION

Mixed phenotype acute leukemia (MPAL) remains one of the most challenging categories of acute leukemia because of its lineage ambiguity, biological heterogeneity, and lack of standardized treatment protocols. Successive refinements in the World Health Organization (WHO) classification, culminating in WHO-HEM5, have emphasized the use of stringent lineage-defining markers and the recognition of genetically defined MPAL subgroups, improving diagnostic clarity and prognostic stratification (Arber et al.; Choi et al.) [4–7]. The present cohort highlights the aggressive nature of MPAL and reinforces the importance of integrated immunophenotypic, molecular, and response-based assessment in routine clinical practice.

The demographic and clinical profile of patients in this study, including male predominance, frequent systemic symptoms, cytopenias, and high marrow blast burden, is consistent with findings reported by Matutes et al. and Béné and Porwit, who described MPAL as presenting with advanced marrow involvement and early bone marrow failure [2,3]. The predominance of hypercellular marrow further supports the highly proliferative behavior of this leukemia subtype.

Immunophenotypic evaluation demonstrated that B-lymphoid/myeloid MPAL was the most common subtype, in agreement with large series reported by Matutes et al. and Kandeel et al. [2,10]. The frequent expression of stem cell marker CD34 and near-universal MPO positivity support the concept proposed by Alexander et al. that MPAL originates from early hematopoietic progenitors with retained lineage plasticity [8]. Molecular profiling identified BCR::ABL1 as the most frequent genetic abnormality, consistent with WHO-recognized MPAL subgroups and prior institutional cohorts (Kandeel et al.; Khan et al.) [10,12]. Although KMT2A rearrangements were less common, their presence is clinically relevant, as these alterations have been repeatedly associated with aggressive biology and poor outcomes in both MPAL and other acute leukemias (Guarnera et al.) [11].

Early treatment response analysis revealed that while morphologic remission was achieved in a substantial proportion of patients, deep molecular remission was uncommon during early induction. Persistent measurable residual disease (MRD) was observed in most cases, highlighting the limitations of conventional induction strategies. Several studies, including those by Orgel et al. and Oberley et al., have demonstrated that MRD status is a powerful predictor of survival in MPAL and should guide treatment intensification and transplant decisions [15,16].

Patients with BCR::ABL1-positive disease were predominantly adults, a finding consistent with previous observations by Kandeel et al. and Khan et al. [10,12]. However, remission rates and early response parameters were comparable between BCR::ABL1-positive and negative patients, suggesting that molecular subtype alone does not fully predict treatment response.

Overall survival remained poor, in line with population-based analyses by Shi and Munker and global outcome data reported in recent reviews [13,18–20]. Importantly, patients who underwent allogeneic hematopoietic stem cell transplantation demonstrated markedly superior survival, supporting conclusions from the EBMT study by Munker et al. that transplantation offers the best chance of durable disease control in high-risk MPAL [14]. Collectively, these findings emphasize the need for early risk stratification, MRD-guided therapy, and timely consideration of transplantation to improve outcomes in this rare but aggressive leukemia.

CONCLUSION

Mixed phenotype acute leukemia represents a rare but highly aggressive form of acute leukemia characterized by marked clinical, immunophenotypic, and molecular heterogeneity. In this study, MPAL commonly presented with advanced disease features, high marrow blast burden, and frequent cytopenias, reflecting its aggressive biological behavior. The B-lymphoid/myeloid phenotype was the predominant subtype, and BCR::ABL1 fusion emerged as the most frequent molecular abnormality, while KMT2A rearrangements were associated with particularly poor outcomes. Although early morphologic remission was achieved in a substantial proportion of patients, persistent measurable residual disease remained common, underscoring the limitations of conventional induction therapy. Overall survival was unsatisfactory, emphasizing the high-risk nature of this entity. Importantly, allogeneic hematopoietic stem cell transplantation was associated with significantly improved survival, highlighting its critical role in the management of selected high-risk patients. These findings reinforce the importance of accurate WHO-based classification, comprehensive molecular profiling, and MRD-guided, risk-adapted treatment strategies to improve outcomes in MPAL.

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