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ORIGINAL PAPER

# DISTINCT CLINICAL AND BIOLOGICAL PROFILES OF ACUTE LYMPHOBLASTIC AND ACUTE MYELOID LEUKAEMIAS IN CHILDREN

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## Abstract

**Introduction.** Acute leukaemias in children constitute a heterogeneous group of malignant haematological disorders characterised by the uncontrolled proliferation of immature bone marrow cells. Their clinical and biological features vary according to leukaemia subtype and population context.
**The objective of the study** was to analyse the demographic, clinical, haematological and biological characteristics of paediatric acute leukaemias diagnosed in the Paediatric Oncology Centre of El Hassi (Oran, Algeria), and to assess their associations with leukaemia subtypes.
**Material and methods.** This retrospective study included 163 children aged 2–15 years, who were newly diagnosed with acute leukaemia. Patients were classified as acute lymphoblastic leukaemia (B-ALL and T-ALL) or acute myeloblastic leukaemia (AML). Clinical and biological parameters were compared using appropriate statistical methods.
**Results.** Acute lymphoblastic leukaemia (ALL) accounted for most cases and predominantly affected children under 10 years of age, whereas AML occurred more frequently in older children. Clinical

## Résumé

**Profils cliniques et biologiques distincts des leucémies aiguës lymphoblastiques et myéloïdes chez l'enfant**

**Introduction.** Les leucémies aiguës de l'enfant constituent un groupe hétérogène de maladies hématologiques malignes caractérisées par la prolifération incontrôlée de cellules immatures de la moelle osseuse. Leurs caractéristiques cliniques et biologiques varient selon le sous-type de leucémie et le contexte de la population.
**L'objectif de l'étude** était d'analyser les caractéristiques démographiques, cliniques, hématologiques et biologiques des leucémies aiguës pédiatriques diagnostiquées au Centre d'Oncologie Pédiatrique d'El Hassi (Oran, Algérie), et d'évaluer leurs associations avec les sous-types de leucémie.
**Materiel et méthodes.** Cette étude rétrospective incluait 163 enfants âgés de 2 à 15 ans, nouvellement diagnostiqués avec une leucémie aiguë. Les patients ont été classés en leucémie aiguë lymphoblastique (B-LAL et T-LAL) ou leucémie aiguë myéloblastique (LAM).

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manifestations—including asthenia, thrombocytopenia, lymphadenopathy, splenomegaly and hepatomegaly—were more common in lymphoblastic forms. Biologically, ALL, particularly T-ALL, was characterised by marked hyperleukocytosis, high blast infiltration and elevated lactate dehydrogenase (LDH) levels, reflecting increased proliferative activity.

**Conclusions.** This study highlights that each acute leukaemia subtype is associated with a distinct clinical and biological profile. Early identification of these profiles at the time of diagnosis may improve patient stratification and contribute to more appropriate therapeutic management in children with acute leukaemia.

**Keywords:** hyperleukocytosis, bone marrow blasts, immunophenotyping, lactate dehydrogenase, thrombocytopenia.

**List of abbreviations:**
ALL – acute lymphoblastic leukaemia
AML – acute myeloblastic leukaemia
ANOVA – analysis of variance
B-ALL – B-cell acute lymphoblastic leukaemia
CML – chronic myeloid leukaemia
LDH – lactate dehydrogenase
SPSS – Statistical Package for the Social Sciences
T-ALL – T-cell acute lymphoblastic leukaemia
WHO – World Health Organization

Les paramètres cliniques et biologiques ont été comparés à l'aide de méthodes statistiques appropriées.

**Résultats.** La leucémie aiguë lymphoblastique représentait la majorité des cas et touchait principalement les enfants de moins de 10 ans, tandis que la LAM était plus fréquente chez les enfants plus âgés. Les manifestations cliniques—fatigue, thrombopénie, lymphadénopathie, splénomégalie et hépatomégalie—étaient plus fréquentes dans les formes lymphoblastiques. Biologiquement, la LAL, surtout la T-LAL, se caractérisait par une hyperleucocytose marquée, une infiltration élevée de blastes et des taux élevés de lactate déshydrogénase (LDH), traduisant une activité proliférative accrue.

**Conclusions.** Cette étude démontre que chaque sous-type de leucémie aiguë présente un profil clinique et biologique distinct. L'identification précoce de ces profils dès le diagnostic pourrait améliorer la stratification des patients et contribuer à une prise en charge thérapeutique plus adaptée chez l'enfant.

**Mots-clés:** hyperleucocytose, blastes médullaires, immunophénotypage, lactate déshydrogénase, thrombopénie.

## Introduction

Acute leukaemias comprise a heterogeneous group of highly aggressive bone marrow malignancies, characterised by the uncontrolled proliferation and accumulation of immature and undifferentiated haematopoietic precursor cells, termed blasts, resulting in disruption of normal haematopoiesis[1]. Daltveit et al. estimate that leukaemia represents a major global health burden, with marked differences in incidence across age groups and disease subtypes[2]. Childhood leukaemia remains the most frequently diagnosed paediatric malignancy worldwide, with an estimated 58,785 new cases reported in 2021, corresponding to a global incidence rate of 2.92 per 100,000 children[3]. Although recent global analyses indicate that both incidence and mortality rates have declined over the past three decades, leukaemia continues to represent a substantial proportion of the overall burden of paediatric cancers across all regions of the world[3]. Notably, survival outcomes vary markedly according to country income level. In low- and middle-income countries, the five-year overall survival of children with leukaemia is reported to be below 20%, compared with approximately 80% in high-income countries[4].

According to World Health Organization (WHO) estimates based on GLOBOCAN 2022 data, acute lymphoblastic leukaemia (ALL) represents the predominant subtype in the paediatric population, accounting for approximately 70–80% of cases, whereas acute myeloid leukaemia (AML) accounts for about 15–20% of childhood leukaemias[2]. Chronic myeloid leukaemia (CML) is rare in children, constituting less than 5% of paediatric cases and typically presenting in adolescents[5].

Acute leukemias in children are heterogeneous in terms of clinical presentation and biological characteristics. Several studies have assessed clinical, hematological, and biological parameters according to leukemia subtypes (B-ALL, T-ALL, and AML), providing insights into disease severity, prognosis, and therapeutic response[6,7]. However, information remains limited in certain populations, and the relationships between these parameters and leukemia subtypes are not fully described in many regional contexts. In Algeria, for instance, most data on childhood leukaemia come from partial cancer registries or hospital-based studies. The National Childhood Cancer Registry (2020) reports that leukaemias are among the most frequent paediatric cancers, with more than 800 new cases recorded

in 2022, predominantly leukaemias and central nervous system tumours[8,9]. In Oran, the only available registry-based study (1996–2000) reported incidence rates of 2.8 per 100,000 in boys and 1.4 per 100,000 in girls[10]. The lack of recent regional data underscores the need for further epidemiological studies and justifies the conduct of the present study at the El Hassi Paediatric Oncology Centre in Oran.

**The objective of this study** was to investigate the relationship between clinical, haematological, and biological parameters and the different leukaemia subtypes in children diagnosed at the Paediatric Cancer Center of El Hassi, Oran, Algeria.

## Materials and methods

### Study design

This is a retrospective observational study conducted at the Paediatric Oncology Centre of El Hassi, Oran (Algeria).

### Study population

All children aged 2 to 15 years who were newly diagnosed with acute leukaemia and managed at the Paediatric Oncology Centre of El Hassi were included in this study. The patients were classified according to the type and subtype of leukaemia into ALL or AML. ALL was further subdivided into B-lineage ALL and T-lineage ALL.

Initially, 164 cases of leukaemia were identified. One case of CML was excluded from the analysis in order to avoid bias, as the present study focused exclusively on acute leukaemias. Consequently, a total of 163 patients were included, comprising 132 cases of ALL (99 B-ALL and 33 T-ALL) and 31 cases of AML.

Incomplete medical records or records that did not allow reliable classification of leukaemia were excluded from the study.

### Studied parameters

Data were extracted from medical records. Demographic variables included age at diagnosis and sex. Patients were stratified into three age groups (2–5 years, 5–9 years, and 10–15 years) to analyse the distribution of leukaemia according to age.

Clinical parameters collected included asthenia, pallor, anaemia, thrombocytopenia, infections, haemorrhages, lymphadenopathy, splenomegaly, hepatomegaly, and bone involvement.

Biological and haematological variables comprised haemoglobin, white blood cell count, red blood cell count, platelet count, the percentage of neutrophils, lymphocytes, and blasts in peripheral blood smears, the percentage of bone marrow blasts, as well as lactate dehydrogenase (LDH) levels.

The study of the erythroid lineage in bone marrow included the quantification of basophilic, polychromatic, and acidophilic erythroblasts.

### Statistical analysis

Statistical analyses were performed using Statistical Package for the Social Sciences (SPSS). Qualitative variables were expressed as counts and percentages, while quantitative variables were presented as mean±standard deviation. Comparisons between ALL and AML groups were conducted using Pearson's $\chi^2$ test or Fisher's exact test for qualitative variables, and Student's t-test for quantitative variables, depending on data distribution.

Comparisons among ALL subtypes (B-ALL, T-ALL) and AML were performed using Fisher's exact test for qualitative variables and analysis of variance (ANOVA) for quantitative variables. A p-value $< 0.05$ was considered statistically significant.

### Ethical considerations

This study was conducted in accordance with the principles of the Declaration of Helsinki. Data were collected anonymously from medical records. The study was approved by the Ethics Committee of the Paediatric Oncology Centre of El Hassi Oran (approval no. 28, January 7, 2026).

## Results

### Demographic characteristics and age distribution of the patients

The study population was predominantly composed of patients with ALL (n = 132) compared with AML (n = 31). A male predominance was observed in both groups, with no statistically significant difference (p = 0.284). In contrast, the age distribution differed significantly between the two leukaemia types (p=0.014). ALL predominantly affected children under 10 years of age, with a peak incidence between 2 and 9 years, whereas AML was more frequent in older children aged 10–15 years (Table 1).

### Age distribution according to leukaemia subtype

A highly significant difference in age distribution across leukaemia subtypes was observed ($p < 0.001$). B-ALL predominantly affected children aged 2–9 years, whereas T-ALL and AML were more frequent in children aged 10–15 years (Table 2).

### Clinical and visceral manifestations

Several clinical features differed significantly between ALL and AML. Patients with ALL more frequently presented with asthenia ($p < 0.001$),

**Table 1.** Demographic and age distribution of patients by leukaemia type

| *Parameters* | *ALL (n=132) n(%)* | *AML (n=31) n(%)* | *p-value* |
|---|---|---|---|
| Male | 90 (68.2) | 18 (58.1) | 0.284 |
| Female | 42 (31.8) | 13 (41.9) | |
| **Age group (years)** | | | |
| 2–<5 | 54 (40.9) | 11 (35.5) | |
| 5–9 | 57 (43.2) | 8 (25.8) | **0.014** |
| 10–15 | 21 (15.9) | 12 (38.7) | |

Data are expressed as n (%). A p value < 0.05 was considered statistically significant. **ALL**, acute lymphoblastic leukaemia; **AML**, acute myeloblastic leukaemia.

**Table 2.** Age distribution according to leukaemia subtype

| *Age group* | *B-ALL* | *T-ALL* | *AML* | *p-value* |
|---|---|---|---|---|
| 2–<5 years | 53 (53.5) | 1 (3.0) | 11 (35.5) | |
| 5–9 years | 40 (40.4) | 17 (51.5) | 8 (25.8) | **<0.001** |
| 10–15 years | 6 (6.1) | 15 (45.5) | 12 (38.7) | |

Data are expressed as n (%). A p value < 0.05 was considered statistically significant. **ALL**, acute lymphoblastic leukaemia; **AML**, acute myeloblastic leukaemia.

**Table 3.** Clinical and visceral manifestations

| *Parameters* | *ALL (n=132) n (%)* | *AML (n=31) n (%)* | *p-value* |
|---|---|---|---|
| Asthenia | 40 (30.3) | 0 (0.0) | **<0.001** |
| Pallor | 114 (86.4) | 28 (90.3) | 0.554 |
| Anaemia | 84 (63.6) | 14 (45.2) | 0.059 |
| Thrombocytopenia | 103 (78.0) | 20 (64.5) | **<0.001** |
| Haemorrhage | 40 (30.3) | 7 (22.6) | 0.393 |
| Infection | 6 (4.5) | 1 (3.2) | 0.744 |
| Lymphadenopathy | 67 (50.8) | 8 (25.8) | **0.012** |
| Splenomegaly | 66 (50.0) | 5 (16.1) | **<0.001** |
| Hepatomegaly | 60 (45.5) | 4 (12.9) | **<0.001** |
| Bone involvement | 68 (51.5) | 13 (41.9) | 0.337 |
| Other site | 3 (2.3) | 0 (0.0) | 0.397 |

Data are expressed as n (%). A p value < 0.05 was considered statistically significant. ALL, acute lymphoblastic leukaemia; AML, acute myeloblastic leukaemia.

thrombocytopenia (p < 0.001), lymphadenopathy (p = 0.012), splenomegaly (p < 0.001) and hepatomegaly (p < 0.001). In contrast, no statistically significant differences were observed for the other clinical signs between the two groups (Table 3).

### Haematological and biological parameters

Patients with ALL exhibited higher leukocyte counts (p = 0.017), elevated lymphocyte levels (p = 0.035), a greater proportion of blasts in peripheral blood smears (p = 0.002) and in bone marrow (p < 0.001), as well as significantly increased LDH levels (p < 0.001). In contrast, patients with AML had higher red blood cell counts (p = 0.027) and a greater proportion of neutrophils (p = 0.010).

Furthermore, analysis of the erythroid lineage revealed significant differences favouring AML, with higher proportions of basophilic, acidophilic, and polychromatic erythroblasts (p < 0.05) (Table 4).

### Clinical manifestations according to leukaemia subtype at diagnosis

Table 5A highlights significant differences between B-cell ALL, T-cell ALL, and AML with respect to clinical manifestations. Asthenia was absent in AML patients but more frequent in T-cell ALL (p < 0.001). Splenomegaly and hepatomegaly were significantly more common in ALL, particularly B-cell ALL (p = 0.002), while lymphadenopathy was more frequently observed in lymphoblastic than in myeloblastic forms (p = 0.043) (Table 5A).

### Haematological and biological parameters according to leukaemia subtype

The analysis of haematological and biological parameters presented in Table 5B revealed highly significant differences between the studied subtypes. T-cell acute lymphoblastic leukaemia was characterised by the highest degree of hyperleukocytosis (p < 0.001)

**Table 4.** Haematological and biological parameters

| *Parameters* | *ALL (n=132)* | *AML (n=31)* | *p-value* |
|---|---|---|---|
| Haemoglobin (g/dL) | 9.24 ± 2.62 | 9.44 ± 2.68 | 0.701 |
| White blood cells ($\times 10^9$/L) | 34.41 ± 89.19 | 13.94 ± 19.17 | **0.017** |
| Red blood cells ($\times 10^{12}$/L) | 3.28 ± 0.75 | 3.60 ± 0.40 | **0.027** |
| Platelets ($\times 10^9$/L) | 118.0 ± 200.4 | 295.0 ± 804.6 | 0.233 |
| Neutrophils (%) | 8.58 ± 11.49 | 15.99 ± 14.22 | **0.010** |
| Lymphocytes (%) | 23.26 ± 26.99 | 16.58 ± 11.49 | **0.035** |
| Basophilic erythroblasts (%) | 9.87 ± 3.61 | 11.50 ± 4.31 | **0.031** |
| Acidophilic erythroblasts (%) | 8.47 ± 3.09 | 9.87 ± 3.69 | **0.030** |
| Polychromatic erythroblasts (%) | 9.87 ± 3.61 | 11.50 ± 4.31 | **0.031** |
| Blasts in peripheral blood (%) | 61.67 ± 29.41 | 43.07 ± 29.56 | **0.002** |
| Blasts in bone marrow (%) | 75.45 ± 15.40 | 49.29 ± 18.54 | **<0.001** |
| LDH (U/L) | 1277.61 ± 804.07 | 722.58 ± 130.07 | **<0.001** |

Data are expressed as mean ± standard deviation. A p value < 0.05 was considered statistically significant. ALL, acute lymphoblastic leukaemia; AML, acute myeloblastic leukaemia; LDH, lactate dehydrogenase.

**Table 5 A.** Clinical manifestations according to leukaemia subtype at diagnosis

| *Parameters* | *B-ALL (n=99)* n (%) | *T-ALL (n=33)* n (%) | *AML (n=31)* n (%) | *p-value* |
|---|---|---|---|---|
| Asthenia | 26 (26.3) | 14 (42.4) | 0 (0.0) | **<0.001** |
| Anaemia | 65 (65.7) | 19 (57.6) | 14 (45.2) | 0.120 |
| Thrombocytopenia | 77 (77.8) | 26 (78.8) | 20 (64.5) | 0.288 |
| Haemorrhage | 29 (29.3) | 11 (33.3) | 7 (22.6) | 0.629 |
| Infection | 5 (5.1) | 1 (3.0) | 1 (3.2) | 0.839 |
| Splenomegaly | 51 (51.5) | 15 (45.5) | 5 (16.1) | **0.002** |
| Hepatomegaly | 48 (48.5) | 12 (36.4) | 4 (12.9) | **0.002** |
| Lymphadenopathy | 50 (50.5) | 17 (51.5) | 8 (25.8) | **0.043** |
| Bone involvement | 56 (56.6) | 12 (36.4) | 13 (41.9) | 0.084 |
| Other sites | 3 (3.0) | 0 (0.0) | 0 (0.0) | 0.372 |

Data are expressed as n (%). A p value < 0.05 was considered statistically significant. ALL, acute lymphoblastic leukaemia; AML, acute myeloid leukaemia.

**Table 5B.** Haematological and biological parameters according to leukaemia subtype

| **Parameters** | **B-ALL (n=99)** | **T-ALL (n=33)** | **AML (n=31)** | **p-value** |
|---|---|---|---|---|
| Platelets ($\times 10^9$/L) | 125.4 ± 224.6 | 95.7 ± 96.4 | 295.0 ± 804.6 | 0.076 |
| White blood cells ($\times 10^9$/L) | 20.35 ± 36.58 | 76.14 ± 160.82 | 13.94 ± 19.17 | **<0.001** |
| Red blood cells ($\times 10^{12}$/L) | 3.24 ± 0.44 | 3.43 ± 1.30 | 3.60 ± 0.40 | **0.035** |
| Haemoglobin (g/dL) | 9.15 ± 2.34 | 9.49 ± 3.37 | 9.44 ± 2.68 | 0.754 |
| Myeloblasts (%) | 67.60 ± 31.23 | 71.86 ± 32.53 | 88.52 ± 126.40 | 0.260 |
| Neutrophils (%) | 8.70 ± 11.78 | 8.21 ± 10.74 | 15.99 ± 14.22 | **0.010** |
| Lymphocytes (%) | 25.04 ± 27.67 | 17.97 ± 24.50 | 16.58 ± 11.49 | 0.150 |
| Basophilic erythroblasts (%) | 9.92 ± 3.69 | 9.71 ± 3.40 | 11.50 ± 4.31 | 0.094 |
| Acidophilic erythroblasts (%) | 8.51 ± 3.16 | 8.33 ± 2.90 | 9.87 ± 3.69 | 0.091 |
| Polychromatophilic erythroblasts (%) | 9.92 ± 3.69 | 9.71 ± 3.40 | 11.50 ± 4.31 | 0.094 |
| Peripheral blood blasts (%) | 59.91 ± 29.53 | 66.97 ± 28.85 | 43.07 ± 29.56 | **0.004** |
| Bone marrow blasts (%) | 74.89 ± 16.31 | 77.12 ± 12.34 | 49.29 ± 18.54 | **<0.001** |
| LDH (IU/L) | 1131 ± 452 | 1716 ± 1326 | 723 ± 130 | **<0.001** |

Data are expressed as mean ± standard deviation. A p value < 0.05 was considered statistically significant. ALL, acute lymphoblastic leukaemia; AML, acute myeloid leukaemia; neutrophils; LDH, lactate dehydrogenase.

and the highest LDH levels ($p < 0.001$). AML showed higher neutrophil counts ($p = 0.010$) and lower bone marrow blast percentages compared with ALL ($p < 0.001$). B-cell ALL occupied an intermediate position, with substantial blast infiltration but less marked biological activity than T-ALL. In addition, significant differences were observed in red blood cell counts, which were higher in AML than in lymphoblastic forms ($p = 0.035$). Furthermore, the proportion of blasts on peripheral blood smears was significantly higher in lymphoblastic leukaemias, particularly T-ALL, compared with AML ($p = 0.004$) (Table 5B).

## Discussion

Our study addressed the challenge of characterizing clinical and biological profiles of paediatric acute leukaemias in a North African single-centre cohort, where epidemiological data remain scarce[11,12]. Key findings confirm that ALL predominates (80.9%) in younger children (peak 2–9 years), contrasting with AML in older children (10–15 years; p=0.014). These align with international (75–85% ALL)[13,14,15] and regional data from Algeria and Morocco.

In children, B-cell ALL predominantly affects younger age groups, whereas T-cell ALL and AML are more frequently observed in older children. This age-related distribution may reflect differences in the developmental dynamics of haematopoietic stem and progenitor cells, with B-cell precursors being particularly susceptible to leukaemogenic events, which may occur as early as foetal life. These observations suggest a window of vulnerability during early immune development, during which environmental exposures or endogenous cellular stressors may interact with pre-existing genetic alterations to initiate leukaemogenesis. Moreover, age-dependent changes in the bone marrow microenvironment, including niche availability, cytokine signalling and stromal support, may influence the proliferation and survival of malignant precursors, thereby contributing to the observed differences in onset between B-cell ALL, T-cell ALL and AML. Finally, the distinct maturation trajectories of lymphoid versus myeloid lineages provide a mechanistic explanation for why B-cell ALL manifests earlier, whereas T-cell ALL and AML tend to present later, reflecting the delayed expansion or transformation of T-cell and myeloid progenitors[13].

Regarding sex distribution, a male predominance was observed in both groups (ALL: 68.2%; AML: 58.1%; $p = 0.284$). Although this difference was not statistically significant, it is consistent with epidemiological data reported in the paediatric literature[16]. This pattern may be related to sex-dependent biological factors, including X chromosome–linked genetic mechanisms, hormonal influences on haematopoietic stem cell proliferation and differentiation, and differences in susceptibility to oncogenic mutations[17].

From a pathophysiological perspective, acute leukaemias result from the uncontrolled proliferation of haematopoietic progenitors arrested at an early stage of differentiation, leading to blast accumulation and bone marrow failure, manifested by anaemia, thrombocytopenia and neutropenia. In ALL, initiating genetic events often occur prenatally, followed by postnatal "second hits" that promote clonal expansion[13]. In AML, genetic alterations predominantly affect myeloid differentiation and progenitor self-renewal, which may explain its later onset[17]. Moreover, early-life exposure to low doses of ionising radiation or maternal pesticide exposure has been associated with an increased risk of paediatric leukaemia[18].

Clinical manifestations differed according to leukaemia subtype. Asthenia was more frequent in ALL (≈30%) and was almost absent in AML ($p < 0.001$), reflecting a higher tumour burden and an increased production of pro-inflammatory cytokines[19]. Fever and infections were also common at admission, reflecting bone marrow failure and the increased vulnerability to bacterial and viral infections. Regarding thrombocytopenia, this was significantly more frequent in ALL ($p < 0.001$). The reduction in platelet counts can be explained by bone marrow infiltration by blasts, which disrupts the normal production of the megakaryocytic lineage[20].

Moreover, involvement of lymphoid organs and the reticuloendothelial system, manifested by lymphadenopathy (51%), splenomegaly (50%) and hepatomegaly (46%), is a hallmark of ALL. This predilection reflects the affinity of lymphoblasts for secondary lymphoid organs, which is mediated by the expression of specific adhesion molecules and chemokines, promoting homing, migration and accumulation of malignant cells within these tissues[13]. The resulting organ infiltration contributes to both local and systemic manifestations, including abdominal discomfort, organomegaly related cytopenias, and altered immune function. Additionally, subtype-specific patterns were observed: asthenia predominated in T-cell ALL, likely reflecting higher tumour burden and metabolic activity, whereas splenomegaly and hepatomegaly were more pronounced in B-cell ALL, consistent with the propensity of B-cell precursors to infiltrate reticuloendothelial organs[21]. These differences may have implications for clinical monitoring, risk stratification, and therapeutic planning, as organ involvement can influence both the presentation and the early complications of the disease.

With regard to biological and clinical parameters, patients with ALL, particularly T-cell ALL, frequently present with marked hyperleukocytosis, a high proportion of circulating and bone marrow

blasts, and significantly elevated LDH levels, reflecting intense blast proliferation, rapid cellular turnover, and high metabolic activity, in line with the Warburg effect and associated with a biologically aggressive phenotype[16,19,22,23]. This metabolic reprogramming, as evidenced by documented cases of lactic acidosis in childhood leukaemias consistent with the Warburg phenotype, further underscores the clinical relevance of altered glucose metabolism in these malignancies[24]. These features account for the increased risk of asthenia, leucostasis, tumour lysis syndrome, and early metabolic complications such as hyperuricaemia and electrolyte disturbances[25,26].

In contrast, B-cell ALL exhibits moderate metabolic activity and a generally more favourable prognosis, with lower risk of early metabolic complications and a predominance of splenomegaly and hepatomegaly, likely reflecting the pattern of organ infiltration rather than overwhelming blast burden[27,28]. AML, on the other hand, is characterised by lower peripheral blast counts, heterogeneous bone marrow infiltration, and relative preservation of normal haematopoietic lineages, which partly explains the less acute systemic symptomatology compared with ALL. Nevertheless, AML often carries a higher risk of early relapse and poorer overall survival, particularly in patients with high-risk cytogenetic or molecular abnormalities, underscoring the need for risk-adapted therapy and careful metabolic monitoring[29,30].

Overall, these distinct biological and metabolic profiles between ALL subtypes and AML highlight the importance of integrating laboratory parameters, blast kinetics, and metabolic markers into prognostic assessment and treatment planning, to anticipate complications and optimise patient outcomes.

In comparison, B-cell ALL exhibited a more moderate metabolic activity and a generally more favourable prognosis, which is reflected in higher long-term remission and survival rates compared with AML in paediatric patients, likely due to differences in molecular drivers and treatment responsiveness[27,28]. This relatively favourable outcome has been attributed in part to the greater chemosensitivity of lymphoid blasts and the availability of targeted therapies such as asparaginase and tyrosine kinase inhibitors in specific genetic subgroups, which improve treatment efficacy and survival. By contrast, AML is characterised by heterogeneous genetic abnormalities, clonal complexity and a poorer overall prognosis, with responses to standard chemotherapeutic regimens varying widely among cytogenetic risk groups. Some recurrent genetic lesions in paediatric AML, such as FLT3-ITD and other high-risk mutations, are associated with worse outcomes and increased relapse rates[29]. Clinically, AML often presents with lower blast counts in peripheral blood but more heterogeneous bone marrow infiltration and a relative preservation of normal haematopoietic lineages, which partly explains the differences in symptomatology between the two entities. For instance, organ infiltration and systemic symptoms may be less prominent in AML at presentation than in ALL, but the risk of early treatment-related complications and relapse remains high[29,30]. These biological and clinical distinctions have important implications for risk stratification and therapeutic planning in paediatric acute leukaemias.

This study has several limitations, notably its single-centre design and the small sample size, particularly for AML, which limits the generalisability of the findings. The lack of cytogenetic and molecular data also precluded the exploration of associations between clinical profiles and genetic abnormalities, which are recognised as major prognostic factors. Nevertheless, the consistency of our results with the existing literature lends them valuable descriptive and comparative relevance.

Overall, the analysis of clinical and biological data indicates that each leukaemic subtype is characterised by a distinct profile, reflected in both the variable severity of clinical manifestations and differences in metabolic and blast activity. These findings highlight the need for tailored management strategies to optimise treatment response and reduce the risk of complications.

## Conclusions

This study confirms the predominance of ALL in children, predominantly affecting younger age groups, whereas AML occurs more frequently in older children. The clinical and biological differences observed between leukaemia subtypes—particularly in terms of blast burden, hyperleukocytosis and LDH levels—reflect distinct pathophysiological mechanisms. Early identification of these profiles at the time of diagnosis is essential to improve patient stratification and to optimize therapeutic management.

### Authors contributions

*"N.R.: data curation, investigation, project administration, writing – review & editing. M.R.Z.A.K.: conceptualization, methodology, data interpretation, writing – review & editing. S.O.: formal analysis, statistical analysis. A.B.: data curation, investigation. All authors have read and approved the final version of the manuscript and agree to be accountable for all aspects of the work."*

### Compliance with Ethics Requirements:

*"The authors declare no conflict of interest regarding this article"*

*"The authors declare that all the procedures and experiments of this study respect the ethical standards in the Helsinki Declaration of 1975, as revised in 2008(5), as well as the national law. Informed consent was obtained from all the patients included in the study"*

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## References

1. Raina R, Gondhi NK, Chaahat, Singh D, Kaur M, Lee HN. A systematic review on acute leukemia detection using deep learning techniques. *Arch Comput Methods Eng.* 2023;30(1):251–270.
2. Daltveit DS, Morgan E, Colombet M, Steliarova-Foucher E, Bendahhou K, Marcos-Gragera R, et al. Global patterns of leukemia by subtype, age, and sex in 185 countries in 2022. *Leukemia.* 2025;39(2):412–419.
3. Liu W, Fang J, Zhu M, Zhou J, Yuan C. Global, regional, and national burden of childhood leukemia from 1990 to 2021. *BMC Pediatr.* 2025;25(1):571.
4. Likiliwike A, Gwanika Y, Joas H, Shayo A, Kissila L, Majaliwa E. Predictors of survival and relapse among children diagnosed with acute leukemia in Northern Tanzania. *Open J Pediatr.* 2025;15(1):52–65.
5. Boychenko EG. Chronic myeloid leukemia in children: a rare and unique entity. *Pediatr Hematol Oncol Immunopathol.* 2022;21(1):156–172.
6. Khalid A, Ahmed M, Hasnain S. Biochemical and hematologic profiles in B-cell acute lymphoblastic leukemia children. *J Pediatr Hematol Oncol.* 2023;45(7):e867–e872.
7. Carvalho MPSS, Almeida FV, Ferreira DW, Cavalcante FH, Oliveira MV. Systemic immunological profile of children with B-cell acute lymphoblastic leukemia. *Front Oncol.* 2023;13:1290505.
8. Tahri H, Abdelaziz S. Childhood cancer in Algeria: an analytical study of the registry of children and adolescents for 2020. *Migration Lett.* 2024;21(8):700–711.
9. Esseha. Cancers pédiatriques en Algérie: 837 nouveaux cas en 2022 et des défis persistants. *Esseha.* 2025. Available from: https://www.esseha.dz
10. Midoun N, Mokhtari L, Ahmed Fouatih Z, et al. Étude de l'incidence des leucémies et lymphomes chez l'enfant à Oran (Algérie). *Rev Epidemiol Sante Publique.* 2002;Suppl:P104.
11. Bachir Bouiadjra C, Seddiki OK, Diaf MB. Hematologic malignancies in children: epidemiological aspects in the pediatric oncology department of Oran Anti-Cancerous Center, Algeria (2009–2013). *J Drug Deliv Ther.* 2020;10(4):168–174.
12. Chebihi ZT, Belkhayat A, Chadli E, et al. Cytogenetic profile of Moroccan pediatric acute lymphoblastic leukemia: analysis of 155 cases with a review of the literature. *Clin Lymphoma Myeloma Leuk.* 2018;18(6):e241–e248.
13. Inaba H, Mullighan CG. Pediatric acute lymphoblastic leukemia. *Haematologica.* 2020;105(11):2524–2539.
14. Lustosa de Sousa DW, de Almeida Ferreira FV, Cavalcante Félix FH, de Oliveira Lopes MV. Acute lymphoblastic leukemia in children and adolescents: prognostic factors and analysis of survival. *Rev Bras Hematol Hemoter.* 2015;37(4):223–229.
15. Hayashi H, Makimoto A, Yuza Y. Treatment of pediatric acute lymphoblastic leukemia: a historical perspective. *Cancers.* 2024;16(4):723.
16. Siegel RL, Miller KD, Wagle NS, Jemal A. Cancer statistics, 2023. *CA Cancer J Clin.* 2023;73(1):17–48.
17. Malard F, Mohty M. Acute lymphoblastic leukaemia. *Lancet.* 2020;395(10230):1146–1162.
18. Onyije FM, Olsson A, Baaken D, et al. Environmental risk factors for childhood acute lymphoblastic leukemia: an umbrella review. *Cancers.* 2022;14(2):382.
19. Terwilliger T, Abdul-Hay M. Acute lymphoblastic leukemia: a comprehensive review and 2017 update. *Blood Cancer J.* 2017;7(6):e577.
20. Zhang L, Zhou Y, Zhang Y, Wang J. Platelet–acute leukemia interactions. Life Sci. 2022;305:120747.
21. Mitura-Lesiuk MM, Dubaj M, Dembowska A, Bigosiński K, Raniewicz M. Hyperleukocytosis in pediatric patients with acute lymphoblastic leukemia: demographic and clinical characteristics. *J Clin Med.* 2024;13(17):5185.
22. Bewersdorf JP, Zeidan AM. Hyperleukocytosis and leukostasis in acute myeloid leukemia: can a better understanding of the underlying molecular pathophysiology lead to novel treatments? *Cells.* 2020;9(10):2310.
23. Polak R, Kowalska A, Nowak J, Zielinska M. Warburg effect in leukemia: mechanisms and therapeutic opportunities. *Front Oncol.* 2022;12:845647.
24. Permtawee K, Tengsujaritkul M, Choed-Amphai C, et al. Warburg effect mimicking inborn errors of metabolism in childhood hematologic malignancies: a case-based systematic review. *World J Clin Pediatr.* 2023;12(5):350–358.
25. Raetz EA, Teachey DT. T-cell acute lymphoblastic leukemia. *Hematology Am Soc Hematol Educ Program.* 2016;2016(1):580–588.
26. Gibson BES, Sauer MG, Ramanathan S, Amrolia PJ. Acute myeloid leukaemia in children. In: Sureda A, Corbacioglu S, Greco R, Kröger N, Carreras E, editors. The EBMT Handbook: Hematopoietic Cell Transplantation and Cellular Therapies. 8th ed. Cham: Springer; 2024. p. Chapter 71.
27. Aasebø E, Brenner AK, Hernandez-Valladares M, et al. Patient heterogeneity in acute myeloid leukemia: leukemic cell communication by release of soluble mediators and its effects on mesenchymal stem cells. *Diseases.* 2021;9(4):74.
28. Urs AP, Goda C, Kulkarni R. Remodeling of the bone marrow microenvironment during acute myeloid leukemia progression. *Ann Transl Med.* 2024;12(4):63.
29. Debnath A, Nath S. Prognosis and treatment in acute myeloid leukemia: a comprehensive review. *Egypt J Med Hum Genet.* 2024;25(1):23–42.
30. Cook JL, Roberts K, Patel A. Current perspectives on paediatric acute myeloid leukaemia: biology, prognosis, and therapeutic challenges. *Oncol Rev.* 2022;16(3):341–356.