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RESEARCH ARTICLE

Alterations in IL-32, IL-39, and LDH Serum Levels in Iraqi Patients with Acute Leukemia: A Cross-Sectional Comparison of Newly diagnosed and Previously Treated Cohorts

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Abstract

Introduction: Acute leukemia (AL), are marked by abnormal differentiation and uncontrolled proliferation of cancerous hematopoietic stem cells. These malignant cells build up in the bone marrow, inhibiting the development and maturation of healthy blood cells. Acute leukemia is classified into two main types: acute lymphocytic leukemia and acute myeloid leukemia. This study aimed to evaluate IL-32, IL-39, and LDH, along with selected hematological parameters, in Iraqi patients with AL before and after treatment. **Materials and Methods:** In this case-control study, serum levels of IL-32 and IL-39 were measured by enzyme-linked immunosorbent assay (ELISA) in 120 patients from the Hematology and Bone Marrow Transplant Center in Baghdad and 60 healthy controls. The patients group was diagnosed by a hematologist and composed 60 individuals with acute myeloid leukemia (AML) and 60 with acute Lymphocytic Leukemia (ALL). **Results:** Age of patients between 5-85 years. Median age of ALL was 19.5 years while 46.5 years for AML. There was no meaningful statistical variation in gender distribution between study group. Significantly elevated serum levels of IL-39 of ALL and AML pre-treatment (84.89±13.96, 137.25±21.66 pg/ml respectively) when compared to post-treatment (87.58±12.00, 114.02±21.51 pg/mL respectively), while the levels of IL-32 were no statistically significant difference pre and post treatment in ALL (74.82±18.81, 79.688±21.23 pg/mL respectively), but in AML IL-39 was higher in pre-treatment patients (106.11±22.01 pg/mL) compared to post treatment (61.25±11.73 pg/mL). LDH levels were statically significant in ALL and AML pre and post treatment ($P \leq 0.01$). **Conclusions:** Elevated serum levels of IL-32 (particularly in AML) and IL-39 (in both AML and ALL pre-treatment) suggest potential involvement in acute leukemia biology. Changes in IL-39 after chemotherapy in AML warrant further investigation as exploratory markers of treatment response.

Keywords: Acute Leukemia, Interleukin-32 (IL-32), Interleukin-39 (IL-39), Lactate Dehydrogenase (LDH)

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Introduction

Hematologic cancers are the leading cause of death among the top four causes of mortality in children worldwide [1]. Leukemia is a common type of blood cancer marked by an increased number of white blood cells (WBCs) in the blood and bone marrow [2]. It is an oncological condition. In leukemia, a high number of immature white blood cells (leukocytes) are present in the peripheral blood cells that are normally restricted to the bone marrow and lymph nodes [3]. However, in some cases, the total leukocyte count in the peripheral blood doesn't rise; instead, the cells undergo qualitative changes. Its exact causes remain unclear, and currently, there are no known ways to prevent it. This disease can affect individuals of all ages, including children, and adults [4].

Leukemia is categorized according to the type of cell it originates from either myeloid or lymphoid and how quickly it progresses, whether acute or chronic. The four primary subtypes include acute myeloid leukemia (AML), chronic myeloid leukemia (CML), acute lymphoblastic leukemia (ALL), and chronic lymphocytic leukemia (CLL) [5].

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Leukemia is characterized by uncontrolled growth of progenitor cells without proper differentiation. When differentiation is blocked, immature cells build up because they cannot fully mature and eventually die. These rapidly dividing immature cells, which are halted in development and evade the immune system, begin to take over the bone marrow and spread to other organs and tissues, potentially resulting in death [6].

Leukemia is marked by abnormal cytokine signaling, which plays a role in disease progression, blast cell survival, patient diagnosis, and resistance to therapy. Moreover, the immune microenvironment serves as a key regulator and promoter of leukemia and other hematological disorders [7].

Acute leukemia (AL) are aggressive clonal diseases of the blood-forming organs, affecting one or more cell lines within the hematopoietic system. They are characterized by widespread replacement of normal bone marrow with abnormal, immature, and undifferentiated blood cells, leading to decreased levels of red blood cells and platelets in the peripheral blood. Classification of these disorders depends on the origin of the abnormal cells whether they are lymphoid, myeloid, mixed, or undifferentiated [8].

Acute myeloid leukemia (AML) is the most frequently occurring acute leukemia in adults representing 80% of the total cases of this disorder [9]. It is believed to develop due to somatic mutations acquired over time, a process that commonly occurs as part of normal human aging [10]. AML consists of multiple subtypes that differ in their molecular characteristics, response to treatment, and overall prognosis [11]. AML is classified into eight subtypes, which are labeled as M0 through M7 According to (French-American-British) FAB classification [12]. Acute lymphoblastic leukemia (ALL), also known as lymphocytic leukemia, is an aggressive cancer that affects both children and adults [13]. It occurs when lymphoid progenitor cells in the bone marrow, bloodstream, and other tissues undergo malignant transformation and begin to multiply uncontrollably [14]. ALL is the most frequently occurring cancer in children Most ALL cases involve precursor B-cells, while the T-cell form is rare, highly aggressive, and occurs somewhat more frequently in adults than in children [15].

Many genetic risk factors have been linked to leukemia, including conditions like Klinefelter syndrome, Down syndrome, Bloom syndrome, and telomere-related disorders such as Fanconi anemia, dyskeratosis congenita, and Shwachman-Diamond syndrome. Inherited mutations in genes such as RUNX1 and CEBPA are also involved. Moreover, certain viral infections like Epstein-Barr virus and human T-lymphotropic virus as well as exposure to ionizing radiation, previous radiation therapy, environmental benzene, smoking, and chemotherapy drugs (especially alkylating agents and topoisomerase II inhibitors) are all associated with an increased risk of developing acute leukemia [16].

IL-32

Human interleukin-32 (IL-32) is a recently discovered cytokine that has both pro-inflammatory and anti-inflammatory effects. It is crucial for both innate and adaptive immune responses and stimulates the production of several other cytokines, including tumor necrosis factor (TNF)-α, IL-1β, IL-6, and IL-8 [17]. IL-32 is a complex cytokine involved in various diseases and inflammatory disorders. It was first identified in 2005, and prior to that, it was referred to as N-terminal and four kringle domains NK4 [18].

Clinical research has examined IL-32 in a range of conditions, including viral infections, autoimmune disorders, inflammatory diseases, specific cancers, vascular conditions, and lung diseases [19]. IL-32 is a pleiotropic cytokine with multiple isoforms produced through alternative splicing, enabling it to act as a regulator that shifts between pro-inflammatory and anti-inflammatory responses. Subsequent analysis of the IL32 gene identified eight exons that generate four different splice variants: IL-32α, IL-32β, IL-32γ, and IL-32δ [20]. As a result, the range of cells known to produce IL-32 has broadened to include not only immune cells such as NK cells, macrophages/monocytes, and T cells but also non-immune cells like endothelial cells, epithelial cells, and fibroblasts [21].

IL-32 is expressed in various tissues, including the spleen, small intestine, lungs, liver, kidneys, pancreas, colon, and in whole blood [22].

In the peripheral blood of patients with acute leukemia, IL-32 has been closely linked to disease progression. Interestingly, IL-32 exhibits dual roles in cancer development, acting either as a key promoter of cell growth and proliferation or as a tumor suppressor, depending on the cancer type. Elevated IL-32 expression has been associated with increased proliferation and disease advancement in several cancers, including AML, cutaneous T-cell lymphoma (CTCL), gastric B-cell lymphoma (GBCL), multiple myeloma (MM), hepatocellular carcinoma (HCC), as well as breast, lung, colon, pancreatic, gastric, and esophageal cancers. Overall, IL-32's function in cancer can vary, but in many types, higher levels are linked to greater tumor growth and progression [16].

IL-39

Human Interleukin (IL-39), the newest member of the IL-12 cytokine family, is a 54-kDa heterodimer made up of IL-23p19 and Ebi3 subunits. It is mainly secreted by B cells that have been stimulated with lipopolysaccharide (LPS), and its secretion increases with longer stimulation. Its mRNA is also expressed in dendritic cells and macrophages [23].

IL-39 triggers inflammatory responses by binding to the IL-23R/gp130 receptor complex, which leads to the activation of STAT1 and STAT3 signaling pathways. IL-39 supports the development of pancreatic cancer by enhancing the proliferation of cancer cells and preventing their apoptosis [24]. IL-39 causes an increase in serum Alanine Aminotransferase (ALT) and Aspartate Aminotransferase (AST) levels, promotes inflammatory cell infiltration, and leads to hepatocyte damage and necrosis [22].

Overall, IL-12 family acts as an effector cytokine that promotes anti-tumor immunity by stimulating the Th1 response, which is essential for activating cytotoxic T cells and natural killer (NK) cells to eliminate tumors [25].

High Ebi3 expression has been linked to poor prognosis in lung cancer, with patients showing significantly higher serum Ebi3 levels compared to healthy individuals. Moreover, silencing Ebi3 using siRNA reduced cancer cell growth, while introducing external Ebi3 promoted cell proliferation [26].

LDH

Lactate dehydrogenase (LDH) is the final enzyme in the anaerobic glycolysis pathway, catalyzing the conversion of glucose into lactate as the end product [27].

LDH is an enzyme found inside cells that facilitates the final step of glycolysis, involving the reversible transformation of pyruvate into lactate and NADH into NAD^+. It exists in five isoenzyme forms LDH-1 (H4), LDH-2 (H3M), LDH-3 (H2M2), LDH-4 (HM3), and LDH-5 (M4) each with distinct tissue distribution [28]. LDH is mainly active in the cytoplasm, although recent studies suggest it might also be present in the mitochondria [29].

Serum LDH levels are commonly used to aid in the diagnosis and prognosis of hematologic cancers, including AML, myelodysplastic syndromes (MDS), and multiple myeloma. In AML patients, higher LDH levels are generally linked to a poorer prognosis [30].

Elevated serum LDH levels have been associated with a wide range of medical conditions, including anemia, pancreatitis, cancer, muscle injury, trauma, myocardial infarction, liver and kidney diseases, as well as some infectious diseases. LDH is a widely recognized biomarker with important prognostic significance across multiple health disorders [31].

A significantly high LDH level is commonly observed in most patients with ALL, indicating enhanced cell proliferation, increased cell turnover, and elevated white blood cell (WBC) counts during periods of remission or relapse of the disease [32].

Serum LDH is not merely a basic marker of tumor load; rather, it is a multifaceted biomarker linked to the activation of various cancer-related signaling pathways, as well as to the metabolic activity, aggressiveness, and immune-related features of numerous tumors, making it a highly promising target for cancer treatment [33]. Cancer cells utilize LDH to enhance their aerobic metabolism including glycolysis, ATP production, and lactate formation even when oxygen is readily available [34, 35].

The main aim of this study is to investigate the levels of IL-32, IL-39, and LDH in acute leukemia Iraqi patients before and after exposed to chemotherapy, and to investigate their potential diagnostic and prognostic significance. Also to determine whether there is a correlation between all this marker and a set of hematological markers.

Materials and Methods

1.Study design

This was cross-sectional observational study conducted at Hematology and Bone Marrow Transplant Center in Baghdad Medical City during the period from December 2024 to May 2025.

2. Study Populations

The study includes 180 individuals recruited two independent cohorts:

1. Group 1: Patients group

-newly diagnosed, untreated acute leukemia patients (n=30 AML, n=30 ALL)

-A separate group of acute leukemia patients previously exposed to chemotherapy (n=30 AML, n=30 ALL).

The treated patient cohort received heterogeneous chemotherapy regimens, determined by institutional protocols and the stage of disease. In the ALL group (n = 30), one-third of patients (n = 10, 33.3%) received intensive chemotherapy, while the remaining two-thirds (n = 20, 66.7%) were treated with non-intensive regimens. Similarly, in the AML group, 30% of patients (n = 9) underwent intensive chemotherapy, whereas the majority (n = 21, 70%) received non-intensive therapy.

2. Group 2: 60 healthy individuals as control group.

This was described in detail in the diagram (1) below:

3. Inclusion and Exclusion Criteria:

Inclusion Criteria: Patients age between 5-80 years, newly diagnosed patients and patients exposed only to chemotherapy.

Exclusion Criteria: Patients with other cancers or any hematological disorders, patients age under 5 years, patients with secondary acute leukemia, patients treated with immunotherapy, Patients who underwent bone marrow transplant and patients who refused to participate.

4. Sample collection:

Five ml of blood were collected from the median cubital vein using 5 ml disposable syringes. The blood was placed in a gel tube and left to clot for 10 minutes at room temperature, after the serum was separated by centrifugation at 4000 rpm for 10 minutes and preserved in deep freezer at -20 C before analysis.

5. Measurements:

Cytokine levels of IL-32 and IL-39 was collected from 120 patients and 60 control samples. Serum IL-32 levels were measured using human IL-32 ELIZA kit (Cat NO. EH1938, Fine Test, China), The assay rang is 15.625-1000 pg/ml. Specifically recognize IL32, no obvious cross reaction with other analogues has been observed. With Intra-assay CV: 5.1% and Inter-assay CV: 5.03%. Assay linearity for serum sample when diluted 1:2 is 90-100%, 1-4 is 82-99% and 1:8 is 84-95%.

Serum IL-39 levels were measured using human IL-39 ELIZA kit (Cat NO. EH5153, Fine Test, China). The assay

range is 15.625-1000 pg/ml. Specifically recognize IL-39, no obvious cross reaction with other analogues. With Intra-assay CV: 4.98% and Inter-assay CV: 6.2%. Assay linearity for serum sample when diluted 1:2 is 92-102%, 1-4 is 87-98% and 1:8 is 80-93%.

Serum LDH levels were measured using LDH-LSL kit (Lot NO. 24-0523, ELITech, France). The normal range is 125-220 U/L, according to the instructions provided by the manufacturer. Hematological indices, including total leukocyte count, neutrophils, lymphocytes, monocytes, eosinophils, basophils, red blood cells (RBCs), and hemoglobin (Hb), were assessed using a complete blood count (CBC) analysis.

6. Statistical analysis

Analysis of data was carried out using the available statistical package of IBM SPSS-29 (IBM Statistical Packages for Social Sciences- version 29, Chicago, IL, USA). The Data were analyzed by Chi-sequare test to compare between percentages (qualitative data). One-way ANOVA with post hoc analysis was used to investigate differences between the studied groups (more than 2 groups). The Pearson's correlation coefficients (r) assess the relationship between ILs and some hematological parameters to determine their correlation another variable. Receiver operating characteristic (ROC) curves were utilized to assess the diagnostic accuracy of the evaluated biomarkers. The cut-off values of the levels yield optimal sensitivity and specificity based on the data was compiled and analyzed by using on the area under the curve (AUC). $P < 0.05$ was considered statistically significant. For all hypothesis tests.

Results

Acute leukemia cases were classified into AML and ALL, with further subdivision into their respective subtypes to support statistical analysis. Diagnosis was based on several approaches, with bone marrow biopsy serving as the definitive test to confirm blast cell presence and percentage as indicators of leukemia. The sample collection was evenly distributed between the two main leukemia types, though variations were noted among their subtypes.

The findings revealed that ALL was categorized into three subtypes, with B-ALL being the most common (65%), followed by T-ALL (33.4%) and MPALL (1.7%). In contrast, AML was classified into seven subtypes, where M3 showed the highest frequency (31.7%), followed by M5 (23.3%), M4 (20%), M1 (10%), M2 (6.7%), M0 (5%), and M7 (3.3%). The distribution of acute leukemia among the patients studied is presented in (Table 1).

The age of patients ranged from 5-90 years, whereas the control group ranged from 8-70 years. The highest prevalence of ALL was observed in the 15–24years age group, accounting for 33 out of 60 cases (55%). In contrast, AML was more frequent in patients aged above 54 years, with 22 out of 60 cases (36.7%), while the lowest occurrence was noted in the 5–14year age group. The age differences between patients and controls were statistically significant ($P \leq 0.01$) (Figure 1).

With respect to sex, no significant association was found between leukemia incidence and gender ($P = 0.13$). Among ALL cases, males were more frequently affected (38 cases; 63.3%) compared to females (22 cases; 36.7%). Conversely, AML cases were slightly more common in females (32 cases; 53.3%) than in males (28 cases; 46.7%) (Figure 2).

To assess changes in biomarker levels before and after treatment, each patient group was compared separately with the control group for each type. In patients with ALL, the mean serum level of IL-32 was elevated both before and after treatment (74.82 ± 18.8 pg/mL and 79.68 ± 21.23 pg/mL, respectively) when compared with the control group (56.26 ± 6.09 pg/mL). Similarly, IL-39 levels were markedly higher in ALL patients before and after treatment (84.89 ± 13.96 pg/mL and 87.58 ± 12.00 pg/mL, respectively) than in controls (69.05 ± 1.83 pg/mL), with a highly significant difference observed in newly diagnosed ($P = 0.02$). In contrast, LDH levels, though reduced following treatment (743.78 ± 117.68 U/L) compared with pretreatment values (883.06 ± 115.80 U/L), remained substantially elevated relative to the control group (190.88 ± 3.23 U/L), showing a difference that approached statistical significance. As shown in (Table 2) and (Figure 3).

Table 3 showed the changes in biomarker levels in AML patients before and after treatment compared with controls. IL-32 and IL-39 levels were significantly elevated in patients newly diagnosed (106.11 ± 22.01, 137.25 ± 21.66 pg/ml, respectively) and decreased after treatment (61.25 ± 11.73 a,114.02 ± 21.51 pg/ml), approaching control levels (56.26 ± 6.09, 69.05 ± 1.83 pg/ml). In contrast, LDH levels remained markedly elevated both before (659.40 ± 57.02 pg/ml) and after treatment (686.66 ± 58.41 pg/ml) compared with controls (190.88 ± 3.23 pg/ml) as showed in (Figure 4). Table (4)

Table 1. Distribution of Subtypes Collected During the Study

ALL	T-ALL	B-ALL	T+B ALL				
N	19	39	1				
%	33.4	65	1.7				
AML	M0	M1	M2	M3	M4	M5	M7
N	3	6	4	19	12	14	2
%	5	10	6.7	31.7	20	23.3	3.3

Table 2. Comparative Levels of Parameters Levels in ALL before and after Chemotherapy Induction

	Groups (M±SE)		
Variable	ALL-newly diagnosed	Control	P-value
IL-32 (pg/ml)	74.82±18.81	56.26±6.09	0.35
IL-39 (pg/ml)	84.89±13.96	69.05±1.83	0.002
LDH (U/L)	883.06±115.80	190.88±3.23	≤0.01
	Groups (M±SE)		
Test	ALL-after treatment	Control	P-value
IL-32 (pg/ml)	79.688±21.23	56.26±6.09	0.29
IL-39 (pg/ml)	87.58±12.00	69.05±1.83	0.13
LDH (U/L)	743.78±117.68	190.88±3.23	≤0.01

Table 3. Comparative Levels of Parameters Levels in AML before and after Chemotherapy Induction

	Groups (M±SE)		
Variable	AML-newly diagnosed	Control	P-value
IL-32 (pg/ml)	106.11±22.01	56.26±6.09	0.03
IL-39 (pg/ml)	137.25±21.66	69.05±1.83	0.004
LDH (U/L)	659.40±57.02	190.88±3.23	≤0.01
	Groups (M±SE)		
Test	AML- after treatment	Control	P-value
IL-32 (pg/ml)	61.25±11.73	56.26±6.09	0.7
IL-39 (pg/ml)	114.02±21.51	69.05±1.83	0.04
LDH (U/L)	686.66±58.41	190.88±3.23	0.009

showed the analysis of the complete blood count of acute leukemia patients before and after treatment.

Diagnostic accuracy of IL-32 and IL-39

The receiver operating characteristic (ROC) analysis was done to assess the diagnostic accuracy of IL-32 and IL-39 by assessing its diagnostic sensitivity and specificity, among acute leukemia patients and control group.

The ROC analysis for ALL and AML patients revealed the diagnostic performance of IL-32 and IL-39. In ALL patients, IL-32 showed an AUC of 0.485 with a cut-off value of 50.52 pg/mL, yielding a sensitivity of 35% and specificity of 93%, although the difference was not statistically significant (p = 0.773). IL-39 in ALL patients had an AUC of 0.462 and a cut-off value of 83.32 pg/mL, with a sensitivity of 30% and specificity of 92.3% (p = 0.469). For AML patients, IL-32 exhibited an AUC of 0.480 with a cut-off value of 50,02 pg/mL, showing a sensitivity of 40% and specificity of 93% (p = 0.700). IL-39 in AML patients demonstrated an AUC of 0.593 and a cut-off value of 83,57 pg/mL, with a sensitivity of 53% and specificity of 93.2%, showing a trend toward significance (p = 0.079). The results of the ROC analysis of ILs as shown in Table (5, 6) and Figures (5, 6, 7, and 8).

To assess if there is association between hematological markers and Interleukins among both type of acute leukemia, a Pearson's correlation coefficients (r) was performed as shown in (Table 7 and 8).

Table 7 showed significant correlation were observed between IL-32 and platelets, basophils, and monocytes, while IL-39 was significantly correlated with platelets and basophils. No significant associations were observed with other hematological parameters among AML.

In ALL IL-32 showed no significant correlations with

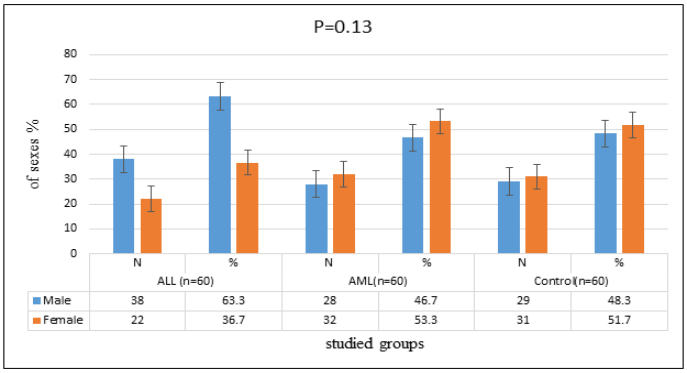

Figure 1. Distribution of Acute Leukemia Groups According to Gender with (P=0.13).

Table 4. Comparison between the Hematological Parameters in the Studied Groups

	Groups (M±SD)					
Parameter	ALL-pre treatment	ALL-after treatment	AML-pre treatment	AML-after treatment	Control	P-value
WBC (10^9/L)	53.25±13.58	6.91±1.56	47.86±10.67	14.57±3.91	7.24±0.17	≤0.01
NEW (10^9/L)	12.52±3.14	4.54±1.03	11.84±3.33	6.77±2.37	4.59±0.12	0.005
EOS (10^9/L)	0.13±0.04	0.12±0.06	0.03±0.01	1.04±0.89	0.15±0.01	0.265
MONO (10^9/L)	6.73±2.29	0.45±0.10	17.60±6.72	2.46±1.17	0.41±0.02	≤0.01
BASO (10^9/L)	0.57±0.21	0.03±0.01	0.20±0.09	0.26±0.15	0.02±0.002	0.002
LYMP (10^9/L)	43.65±14.78	3.90±3.01	16.57±4.28	4.08±1.18	2.57±0.43	≤0.01
Hb (g/dl)	9.71±0.48	9.06±0.94	7.62±0.30	9.07±0.44	14.01±0.25	≤0.01
RBC (10^{12}/L)	4.36±0.91	4.89±0.63	2.45±0.10	3.94±0.49	4.82±0.06	0.002
PLT (10^9/L)	76.56±13.76	207.46±40.39	45.03±4.50	109.33±15.81	229.41±6.79	≤0.01

Table 5. ROC Analysis for IL-32 and IL-39 between ALL Patient and Controls

Variable	AUC	Cutoff	SE	P-value	C.I95%		Sensitivity	Specificity
					L.B	U.B		
IL-32 (pg/ml)	0.485	50.52	0.057	0.773	0.373	0.596	35	93
IL-39 (pg/ml)	0.462	83.32	0.057	0.469	0.351	0.573	30	92.3

L.B: Lower Bound, U.B: Upper Bound, SE: Standard Error, AUC: Area Under the Curve.

Table 6. ROC Analysis for IL-32 and IL-39 between AML Patient and Controls

Variable	AUC	Cutoff	SE	P-value	C.I95%		Sensitivity	Specificity
					Lower Bound	Upper Bound		
IL-32 (pg/ml)	0.48	50.02	0.059	0.7	0.365	0.594	40	93
IL-39 (pg/ml)	0.593	83.57	0.06	0.079	0.475	0.711	53	93.2

L.B: Lower Bound, U.B: Upper Bound, SE: Standard Error, AUC: Area Under the Curve.

hematological markers, whereas IL-39 was positively correlated with WBCs and Lymphocytes only as showed in Table 8.

Discussion

Leukemia represents about 8% of all cancer cases worldwide. Among its types, acute myelogenous leukemia (AML) is the most frequent in both adults and children, while acute lymphocytic leukemia (ALL) is recognized as the most common form in children [36]. In our study, the most frequent subtype in our study of acute myeloblastic leukemia is AML-M3 (n=19;31.7%) of patients, followed by M5 and M4, this result was consistent with the findings of Shwan et al [37] who found that M3 subtype was the most common subtype of AML. In contrast of this study Shurooq and Ismail [38] reported that AML-M5 subtype was the most common followed by M4. In cause of ALL in our investigation, B-ALL was the most frequent subtype (n=39;65%) of patients, and the remainder T-ALL (19;33.4%) with one patient of MPALL, this result agreed with Sana et al. [39] who found that B-ALL was the most common subtype with (85.5%), and the reminder T-ALL.

The present study revealed that patient ages ranged between 5 and 90 years. ALL was most frequently diagnosed in the 15–24year age group. while AML occurred more commonly in patients over 54 years. The overall median age of ALL participants was 19.5 years, these results differ from the findings of Dyna et al. [40] who found that the median age of ALL in Iraqi patients was 24 years. whereas the median age for AML cases was 46.5 years, with 36.7% of these patients being older than 54, this result shown to be consistent with results of a study conducted in Iraq in Baghdad that reported the median age of AML patients was 46.40 years [41] and in Sulaimaniyah was 43 years [40]. From a global standpoint, the American Cancer Society reports that the average age at diagnosis for AML is approximately 68 years [42].

Data previously reported by the United States Statistics and Leukemia Treatment Institution demonstrate that

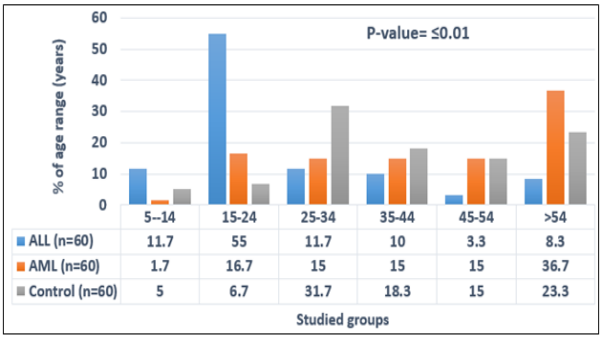

Figure 2. Distribution of Studied Groups According to Age groups with (P=≤0.01)

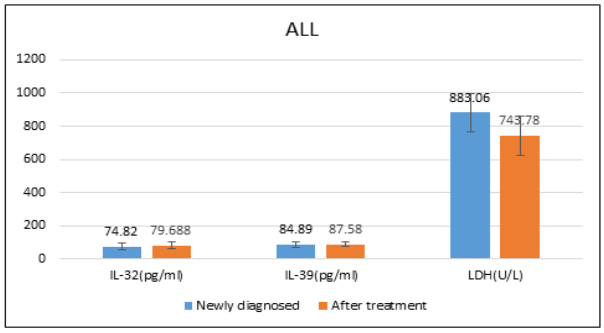

Figure 3. Changes in IL-32, IL-39 and LDH Levels in Newly Diagnosed and after Treatment Patients among ALL

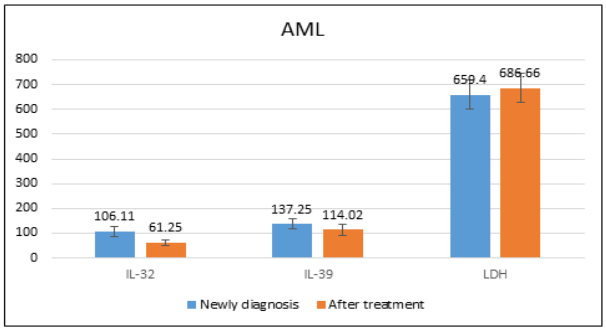

Figure 4. Changes in IL-32, IL-39 and LDH Levels in Newly Diagnosed and after Treatment Patients among AML

the incidence of leukemia is not equally distributed between males and females. An earlier epidemiological investigation in Iraq indicated that myeloid malignancies were more prevalent in males than in females [43]. Unlike the study conducted by Shatha et al. [41], the current results showed a higher proportion of females than males were observed among AML, which may reflect underlying biological, environmental, or sampling-related factors. While its consistent with Yasmine and Mona [32] who found that the incidence of AML was found to be higher in females compared to males.

The present study observed a higher incidence of ALL in males compared to females, which aligns with the results reported by AL-Saidi [36], who found that The prevalence of the disease was higher in males for ALL.

The immune microenvironment plays a pivotal role in regulating and promoting the progression of leukemia and other hematological disorders [43]. In the present study, we compared AML and ALL patients before and after chemotherapy with the control group.

The results showed that serum IL-32 level were markedly elevated in AML compared to ALL, relative to the controls. In study of 82 patients of acute leukemia and 30 healthy controls, Li-Gang et al. reported that was higher level of serum IL-32 in both acute lymphocytic leukemia and acute myeloid leukemia [44]. One of the aims of this study was to determine whether interleukins levels are more elevated before or after treatment. To address this, we conducted two separate comparisons: newly diagnosed patients versus the control group, and post-treatment patients versus the control group. This method enabled us to track how interleukins levels change with treatment and to gain insight into their possible role in disease progression and treatment response. Our findings demonstrated that in ALL patients, IL-32 levels remained relatively stable, showed no huge differences between before and after treatment values. Moreover, when both groups were compared with the control group, the differences did not reach statistical significance. This observation is inconsistent with finding of previous study, which reported high significant alterations in IL-32 levels in newly diagnosed ALL patients than those in control group [44]. This discrepancy may reflect differences in sample size, patient population characteristics, or disease burden at sampling. In contrast to ALL, patients with AML showed a significant elevation of IL-32 levels in newly diagnosed versus control group. After exposure to treatment, this difference was not statistically significant, suggesting that treatment may have redound to normalizing interleukin level toward control levels. These results corroborate the finding of Li-Gang et al. who found that serum level of IL-32 in newly diagnosed AML was higher than control group.

One of the earliest studies to emphasize the critical role of IL-32 in cancer was reported by Marcondes et al. in 2008 [45]. Monitoring changes in IL-32 levels could provide valuable information regarding disease severity, and in combination with complementary molecular and cytogenetic analyses, maybe we can support the evaluation of this biomarker role in assessing therapeutic response and diagnosis in AL patients [44].

In the current study, serum levels of IL-39 were significantly higher in newly diagnosed patients when compared to control, whereas no significant differences were reported between treated patients and control in ALL, while in AML patients the level of IL-39 remained significantly higher in both newly diagnosed patients and treated patients when each group was compared separately with control group. To the best of our knowledge, no studies

Table 7. Correlation Analysis between Hematological Markers among AML Groups Patients (n=60)

Variable		WBC	Lymphocyte	Plateletes	Eosinophile	Monocyte	Basophile
	R	0.246	0.131	-0.272*	-0.025	0.268*	0.293*
IL32	P-value	0.058	0.318	0.035	0.852	0.039	0.023
	N	60	60	60	60	60	60
	R	0.164	0.143	-0.368**	-0.047	0.144	0.338**
IL39	P-value	0.21	0.277	0.004	0.723	0.271	0.008
	N	60	60	60	60	60	60

** Correlation is significant at the 0.01 level (2-tailed). *Correlation is significant at the 0.05 level (2-tailed).

Table 8. Correlation Analysis between Hematological Markers among ALL Groups Patients (n=60).

		WBCs	Lymphocyte	Platelets	Eosinophil	Monocyte	Basophile
IL32	R	-0.101	-0.101	-0.087	-0.092	-0.086	-0.1
	P-value	0.444	0.447	0.509	0.489	0.516	0.449
	N	60	60	60	60	60	60
IL39	R	0.255*	0.388**	-0.032	-0.005	0.12	-0.034
	P-value	0.049	0.002	0.808	0.971	0.361	0.799
	N	60	60	60	60	60	60

**. Correlation is significant at the 0.01 level (2-tailed). *. Correlation is significant at the 0.05 level (2-tailed).

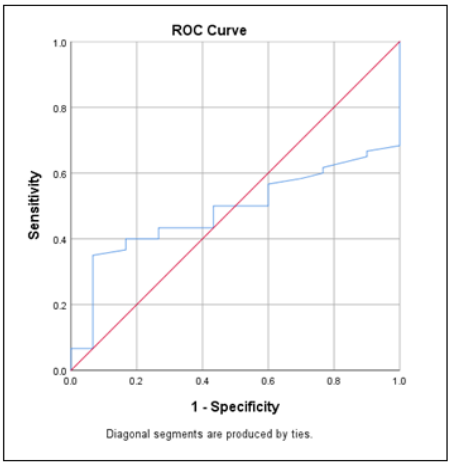

Figure 5. ROC Curve of IL-32 in ALL

were found that directly comparable with the finding of the current study. Specifically, no published research has investigated the role of IL-39 in acute leukemia. Nevertheless, its association with other cancers has been documented, which may provide indirect evidence of its potential role in hematological cancer biology. While IL-39 enhances proliferation in pancreatic and bladder cancer in cell culture models [46, 47], the applicability of these findings to acute leukemia pathogenesis is uncertain. Acute leukemia arise from hematopoietic progenitors and may have distinct IL-39 signaling dependencies compared to solid tumors. Direct investigation of IL-39 effects on acute leukemia blasts is warranted, targeting IL-39 effects on tumor cells may represent a promising therapeutic approach. When Amal et al. examined the levels of IL-39 in 90 breast cancer patients and 40 healthy participants, they reported significantly lower IL-39 levels in patients versus controls, and suggesting that serum IL-39 may serve as a potential early diagnostic biomarker [48]. Although its specific involvement appears to vary between cancer types and warrants further investigation.

The clinical utility of IL-32 and IL-39 as biomarkers cannot be assessed without correlation with genetic risk categories and outcome date, our findings are exploratory and hypothesis-generating; validation will require future studies integrating molecular profiling, clinical outcome data, and formal prognostic modeling.

In this study, in both types of acute leukemia, LDH levels were significantly elevated compared to the control group, both before and after treatment. These results are in line with multiple studies that have reported similar observations who found that LDH levels were significantly higher in acute leukemia patients compared to the control group. Increased LDH expression represents an important prognostic marker, particularly in supporting treatment planning and risk stratification in leukemia [49, 50]. Unexpectedly, LDH levels increased in AML following chemotherapy, which may reflect marrow recovery with rebound RBC production, persistence of disease, or measurement variability. Stratiflcation by morphologic response (CR vs. resldual disease) was not performed and is needed to interpret LDH kinetics.

Although significant differences in LDH levels were observed in both AML and ALL cases when compared with the control group, the mean LDH concentration was notably higher in ALL than in AML. This observation is in agreement with previous studies that have reported that LDH level was statistically significant increase in acute lymphoblastic leukemia than in acute myeloid leukemia [50].

In the present study, significant alterations in hematological parameters were observed among acute leukemia patients compared with healthy controls. In both ALL and AML patients, the mean WBC count was markedly elevated in newly diagnosed and returned to the normal range following therapy. This finding is consistent with Moussavi et al., who reported leukocytosis in 39% of patients [51]. Neutrophil counts were also significantly higher in newly diagnosed and declined toward control values after treatment, in agreement with Jabbour et al., who observed normalization of differential counts during remission induction [52]. However, these results differ from those of Emmanuel et al., who noted a decrease in

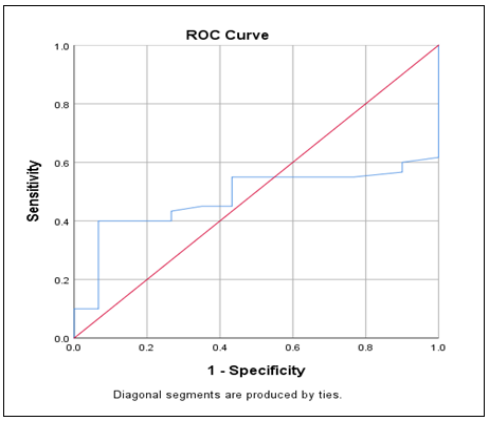

Figure 6. ROC Curve of IL-32 in AML

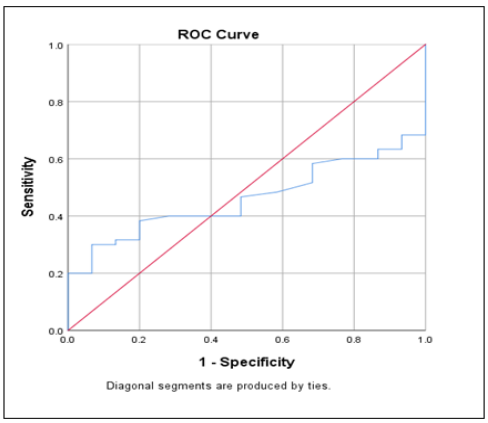

Figure 7. ROC Curve of IL-39 in ALL

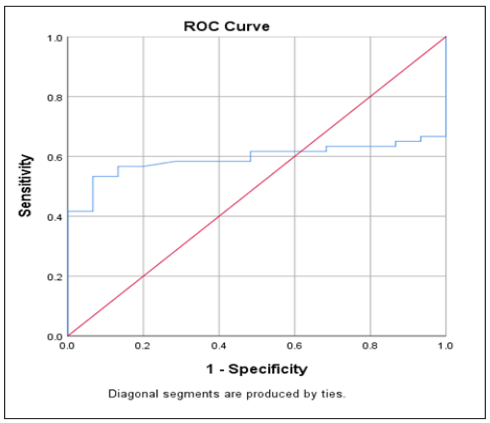

Figure 8. ROC Curve of IL-39 in AML

neutrophil levels [53]. Monocyte counts were significantly elevated compared to controls in newly diagnosed. This aligns with Jianhua et al., who reported that an increased absolute monocyte count (AMC) at diagnosis is associated with inferior overall survival and may serve as an independent prognostic marker in hematological malignancies [54]. Conversely, eosinophils did not appear to play a major role in leukemic progression in our study, consistent with Hemraj et al., who noted that elevated eosinophil counts are more commonly associated with myeloproliferative neoplasms, while lower counts may indicate acute leukemia [55].

Basophil and lymphocyte counts were significantly higher in patients compared to controls, a finding that contrasts with Nadia et al., who reported a decrease in absolute lymphocyte counts and percentages [56]. Our results, however, are in agreement with Lutfi et al., who found basophilia in 38.3% of patients [57]. Additionally, a significant reduction in hemoglobin concentration and red blood cell count was observed in our patient group compared with healthy controls, consistent with O González Llano, who found that four out of five patients with acute leukemia had anemia [58]. Platelet counts were also significantly lower in patients compared to controls, in line with Munir and Khan, who reported low platelet counts in ALL, AML, and CLL cases, but elevated counts in CML [59].

There are some limitation in the current study that should be acknowledged. First, the relatively small sample size may have affected the statistical power and generalizability of the results. Second, the study was performed solely within Iraq, which limits the applicability of the finding to rest of regions due to possible geographical differences and variations in patient ethnicity. Third, a key limitation is that pre- and post-treatment measurements were not obtained from the same individuals. The treated group represents a different cohort, which introduce potential confounding by unmeasured factors (treatment intensity, time since treatment, response status) that may differ between newly diagnosed and treated patients, and the protocols of chemotherapy and their stages were not detailed. Forth, long-term follow-up of patients was not performed, and samples collected after treatment were not from the same individuals as those before treatment, partly due to time constrains. This limitation may influence the predictive and diagnostic claims regarding the evaluated markers. Finally, an important limitation is the absence of molecular and cytogenetic risk stratification data (FLT3-TD, NPMI, TP53 mutations, cytogenetic categories). These data are essential for positioning our findings within contemporary risk-based treatment paradigms. Future studies should investigate whether IL-32 and IL-39 correlate with established genetic risk factors or provide independent prognostic information.

In conclusion, our study revealed distinct hematological and cytokines profiles in patients with acute leukemia. IL-32 levels were notably elevated in AML at diagnosis, while ALL exhibited no significant changes. Serum IL-39 levels were higher in newly diagnosed patients, especially in AML. LDH levels were significantly elevated in both AML and ALL compared to controls. These findings are consistent with existing literature documenting LDH as a prognostic marker in acute leukemia. These results suggest that IL-39 may reflect disease burden or treatment response in acute leukemia; however, establishing clinical utility requires prospective studies correlating biomarker changes with morphologic response assessment survival outcomes, and formal comparison with established prognostic factors. Functional investigation of 1L-39 in leukemia biology is also needed to clarify whether, biomarker elevations reflect pathogenic processes or

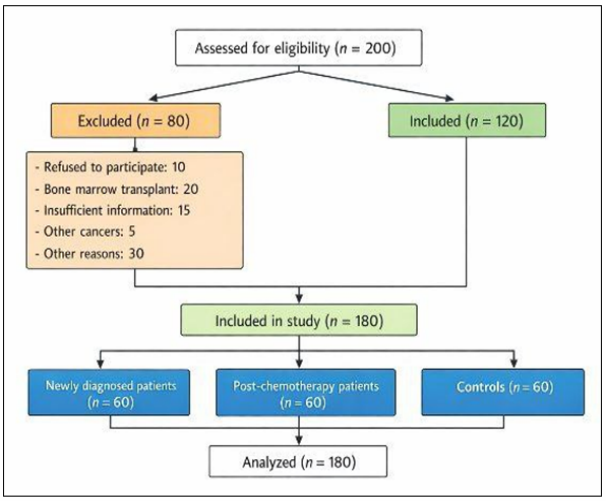

Figure 9. STROBE Flow Diagram of Study Participants

reactive phenomena.

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Conflicts of interest

The authors confirm the absence of any financial conflicts of interest concerning the material presented in this manuscript.

Ethical approval

The research adhered to the ethical standards of the Helsinki Declaration. Prior to sample collection, verbal and informed consent was secured from all adult participants. For pediatric participants (age<18), informed consent was obtained from parents. The study protocol, participant information sheet, and consent form were reviewed and approved by the local ethics committee under approval number (40322, dated November 12, 2024).

Consent to participate

Written informed consent was obtained from all participants, and the trial was conducted in accordance with the declaration of Helsinki.

Consent for publication

Written informed consent was obtained from all participants, and the trial was conducted in accordance with the Declaration of Helsinki.

Availability of data and material

The data sets used and analyzed during the current study are available from the corresponding authors per reasonable request.

Clinical Trial registration

Not applicable.

Authors contribution

Study conception& design: (Rasha Majid Abdul Amir Alhumairi). Literature search:(Noor Saad Mansour). Data acquisition: (Noor saad Mansour). Data analysis& interpretation:(Rasha Majid Abdul Amir Alhumairi and Noor Saad Mansour). Manuscript preparation: (Noor Saad Mansour). Manuscript editing & review: (Rasha Majid Abdul Amir Alhumairi and Noor Saad Mansour). All authors read and approved the final version of the manuscript.

Originality Declaration for Figures

All figures included in this manuscript are original and have been created by the authors specifically for the purposes of this study. No previously published or copyrighted images have been used. The authors confirm that all graphical elements, illustrations, and visual materials were generated from the data obtained in the course of this research or designed uniquely for this manuscript.

Declaration on AI use

We hereby declare that we did not used artificial intelligence extensively in the preparation of this manuscript. Some AI tools were used minimally to paraphrase some sentence and to ensure grammatical accuracy. In addition, an AI tool was used to generate Figure (9).

Recommendations

The findings are promising but our findings are exploratory and hypothesis-generating. We recommend the following future directions: (1) Prospective studies in larger, more diverse international cohorts to assess generalizability:(2) Integration of molecular and cytogenetic data (FLT3-ITD, NPM1, TP53 mutations, cytogenetic risk categories) to evaluate whether Il-32 and IL-39 provide independent prognostic information beyond established risk factors; (3) Longitudinal follow-up studies correlating biomarker levels with clinical outcomes (complete remission, event-free survival, overall survival, minimal residual disease status) to formally establish prognostic utility:(4) Mechanistic investigations to elucidate whether elevated IL-32 and IL-39 in acute leukemia reflect tumor-intrinsic signaling, reactive immune activation, or other pathogenic processes. Only after such validation would these biomarkers be candidates for personalized, clinically Predictive applications.

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