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Effect of Acute Lymphoblastic Leukemia on Hematological and Biochemical Parameters in Children in Najaf, Iraq

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Abstract

One of the most common childhood cancers is acute lymphoblastic leukemia (ALL). Despite advances in treatment, relapse still occurs in some patients. In ALL, ferritin, lactate dehydrogenase (LDH), and complete blood count (CBC) are essential diagnostic and monitoring markers. Additional liver function tests include serum albumin, SGPT, prothrombin time, and bilirubin.

Aim: In this study, we evaluated the serum levels of prothrombin time, CBC, LDH, ferritin, GH, serum bilirubin, SGPT, serum albumin, and CRP in Iraqi children with acute lymphoblastic leukemia, both before and after achieving clinical remission.

Subjects and Methods: In this study, 90 children were enrolled, including 60 patients (35 boys and 25 girls, aged 10–20 years) with acute lymphoblastic leukemia (ALL) who were attending the National Oncology Teaching Hospital in Najaf, Iraq. Thirty age-matched healthy children (17 boys and 13 girls) served as the control group. The patients were further divided into two subgroups: newly diagnosed (before treatment) and in remission (after treatment). Prothrombin time, serum albumin, ferritin, growth hormone, C-reactive protein (CRP), serum lactate dehydrogenase (LDH), serum bilirubin, and SGPT were measured. The findings revealed that, compared to healthy children, ALL patients had significantly higher ferritin levels (277.3 ± 121.9 ng/ml vs. 67.42 ± 9.33 ng/ml in controls) and LDH levels (694.11 ± 83.3 U/L vs. 183 ± 23.7 U/L in controls) ($p < 0.05$). In addition, growth hormone activity (9.2 ± 1.9 ng/ml vs. 6.2 ± 2.8 ng/ml) was significantly higher ($p \leq 0.05$) in ALL patients prior to treatment compared to those in remission following treatment.

Keywords: Children; Remission; Leukemia; Acute Lymphoblastic Leukemia; Liver Function

1. Introduction

About 41% of all childhood cancers are leukemias, making them the most prevalent form of cancer in children [1,2]. Leukemia is classified into two main categories: acute and chronic. Approximately 97% of all juvenile leukemias are acute, of which 80% are acute lymphoblastic leukemia (ALL) and 20% are acute non-lymphocytic leukemia (ANLL) [3]. Although ALL can occur in both adults and children, it is most common in children aged 2–5 years [4].In Iraq, there are an estimated 1,530 cases of ALL and 500 new cases of pediatric cancer annually [5]. The initial diagnostic procedures for ALL include a detailed medical history, physical examination, complete blood count (CBC), and blood smear analysis. Final confirmation is obtained through a bone marrow biopsy. A spinal tap (lumbar puncture) is often performed to determine central nervous system involvement.

The treatment regimen for ALL includes multimodal chemotherapy, radiation therapy, and bone marrow transplantation. Currently, ALL management follows a risk-based approach. Chemotherapy is administered in phases: induction of remission, consolidation, intermediate maintenance, delayed intensification, and long-term maintenance

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[6,7]. Remission-inducing drugs include methotrexate, cyclophosphamide, vincristine, asparaginase, dexamethasone, and 6-mercaptopurine. Treatment duration ranges from 2 to 3.5 years, with the induction phase typically lasting 28 days. For relapse cases, the standard treatment is allogeneic hematopoietic cell transplantation (HCT) following induction and consolidation [8]. ALL has an excellent prognosis at initial presentation, with up to 98% of cases achieving complete remission after multidrug induction chemotherapy [9]. Five-year event-free survival rates reach approximately 90% [10]. Hepatomegaly (30–40%) is frequently observed in leukemia patients, and liver function tests (LFTs) often reveal biochemical or clinical abnormalities. One cause is leukemic infiltration of the liver. Although hepatic involvement is usually mild and asymptomatic at diagnosis, postmortem examinations have demonstrated liver infiltration in over 95% of ALL cases [11,12]. In rare cases, massive infiltration can lead to fulminant hepatic failure [11-13]. The major factor contributing to liver damage in leukemic patients, however, is chemotherapy. Commonly used drugs—such as asparaginase, vincristine, cytarabine, 6-mercaptopurine, methotrexate, and corticosteroids—vary in their degree of hepatotoxicity. At high doses, these cytotoxic agents increase transaminase levels and significantly elevate bilirubin due to reduced hepatic metabolic activity [14]. LFTs are therefore an essential tool for monitoring the hepatotoxic effects of chemotherapy.

In this study, we evaluated the serum levels of prothrombin time, CBC, LDH, ferritin, growth hormone (GH), serum bilirubin, SGPT, serum albumin, and C-reactive protein (CRP) in Iraqi children with ALL, both before and after achieving clinical remission. Serum albumin, prothrombin time, serum bilirubin, and SGPT are commonly included in standard LFT panels [15]. A previous study conducted in Japan on 27 children with ALL measured LFTs at baseline and three months later. SGPT levels were three times higher than normal at baseline but normalized following therapy, while prothrombin time, serum albumin, and serum bilirubin remained within normal ranges throughout treatment [16].

According to the Cochrane Review database, liver complications during and immediately after pediatric cancer treatment are common. Moreover, late-onset hepatic adverse effects have been reported in 8–53% of childhood cancer survivors [17]. The present study aims to demonstrate that liver function is significantly impaired in leukemic patients undergoing chemotherapy to induce remission.

2. Materials and methods

2.1. Subjects

The study population was divided into two groups: patients and healthy controls. The patient group consisted of 60 males and females, aged 10–20 years, who had been diagnosed with acute lymphoblastic leukemia. Samples were collected from the National Oncology Teaching Hospital in Najaf City. The control group consisted of 30 healthy males and females, aged 12–19 years.

2.2. Samples Collection and Analysis

2.2.1. Hematological parameters

The hematological parameters were assessed by drawing five milliliters of venous blood from each patient and control. Two milliliters were dispensed into 0.5-milliliter sodium citrate vials for erythrocyte sedimentation rate (ESR) estimation, while three milliliters were collected in EDTA tubes. The EDTA samples were analyzed using the Auto Hematology Analyzer (Hungary, 2011) to measure packed cell volume (PCV) [18], white blood cell count, red blood cell count, platelet count, and lymphocyte count. All tests were performed according to the manufacturer's instructions. The ESR was determined using the traditional Westergren method [19].

2.2.2. Biochemical parameters

Each patient had a venous blood sample collected, which was immediately transferred into a plain, dry tube. After allowing the blood to clot for 10–15 minutes at room temperature, it was centrifuged at 3500 rpm for 10 minutes. The resulting serum was separated and used to measure specific biochemical markers in the Al-Noha and Al-Sadr Teaching Hospital laboratories [20].

3. Methods

3.1. Complete Blood Count (CBC)

Complete blood count (CBC) was determined using a Beckman Coulter LH 750 autoanalyzer (CA, USA). The parameters analyzed included white blood cells (WBC), hemoglobin concentration (Hb), packed cell volume (PCV), red blood cell concentration (RBC), mean cell volume (MCV), mean corpuscular hemoglobin concentration (MCHC), and platelet characteristics. In addition, white blood cell count, neutrophil count and percentage, lymphocyte count and percentage, neutrophil-to-lymphocyte ratio (NLR), platelet count, and platelet-to-lymphocyte ratio (PLR) were assessed.

3.2. Principle of Alanine Aminotransferase (ALT).

To ascertain the activity in the serum, Ton hazy, White, and Umbreit employed a colorimetric approach, which Reitman and Frankel adapted. [6]. R2 Reagent Procedure (1 mL) For five minutes, incubate at 37 °C. Put in 200 milliliters of serum, mix, and incubate at 37 degrees Celsius for precisely one hour. After mixing, leave Reagent R3 (one milliliter) for 20 minutes at room temperature. 10 mL of 0.4 N NaOH is added. After five minutes, compare the quantity of absorption to water at 505 nm. Determine the outcome as the batch-specific enclosed Standard Curves.

3.3. Estimation the activity of lactate dehydrogenase (LDH) level

The method used by the Bay Labo (French) factory kit maker was used to measure serum LDH activity. The following idea underpins how this strategy operates: When NADH is present as a coenzyme, LDH promotes the internal conversion of pyruvate to lactate, as indicated by optical absorption measured at 340 nm. International units (IU\L) are used to quantify enzymatic activity, which is the amount of enzyme needed to catalyze the conversion of one micromole of substrate per minute.

3.4. Estimation the level of ferritin

The test has been conducted using the LIAISON Analyzer series. Sandwich chemiluminescence immunoassay is the basis for this procedure. Ferritin concentrations up to 3000 ng/ml are measured by LIAISON.

3.5. Estimation the level of growth hormone (GH)

The test was conducted using the LIAISON Analyzer series. This procedure is founded on the sandwich chemiluminescence immunoassay concept. LIAISON GH is used to quantify concentrations (ng/ml) up to 80 ng/ml or 240 μIU/m.

3.6. Estimation the level of C-Reactive Protein (CRP)

Using a test made by Bio-Ditch Med Inc. in the UK, the amounts of C-Reactive Protein in serum were determined. Fluorescence immunoassay technology serves as the test's fundamental component, and C-Reactive Protein is expressed in milligrams per liter.

3.7. Estimation the level LFT

S-albumin was measured with a Shimadzu spectrophotometer or colorimeter configured to measure at 630 nm and standard lab equipment. To quantify serum bilirubin, the TBIL Flex reagent cartridge (Cat. No. DF 67 A) was utilized. Prothrombin time was measured using NEOPLASTNE CL PLUS (5) Kits. All biochemical measurements were carried out by the chief technician of the laboratory at the Alnohba and Al-Sadr Teaching Hospital. A second sample was taken once the acute lymphoblastic leukemia treatment's induction remission period was over.

3.8. Statistical Analysis

The findings are shown as mean± standard deviation. An expression for correlation analysis is the Pearson's correlation coefficient. When the difference between a group of patients and the control is less than 0.05, the P value is deemed significant. The standard error and mean were also used to characterize the values of the biochemical parameters.

4. Results

The hematological parameter data from 60 male and female patients with acute lymphoblastic leukemia and 30 controls are shown in Table 1.A comparison is made between the hematological parameters (Mean±SD) of acute lymphoblastic leukemia and controls. According to the findings, the studied population's mean white blood cell counts, packed cell volumes, platelets, lymphocytes, and red blood cell counts were all considerably lower than those of their control group ($p < 0.05$).

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Table 1 Hematological parameters of Acute Lymphoblastic Leukemia (ALL) children's patients and control subjects

Tests	ALL PATIENTS	CONTROLS
ESR mm/hr	18.379±5.214*	7.610±1.150
Hb (gm/dl)	7.94± 1.77*	12.7± 2.94
WBC $X10^3$ /µL	6.607±1.342*	5.321±0.745
RBC $x10^6$ /µL	4.459±0.521*	3.177±0.603
Packed cell volume (%)	33.732±1.814*	44.488±3.210
Platelets $X10^3$ /µL	192.33±22.31*	205.61±7.88
Lymphocytes (%)	42.199±3.219*	54.783±6.032

*Significant differences at P≤0.05

The biochemical parameter (Means) in children with Acute Lymphoblastic Leukemia and controls is contrasted in table; 2. Ferritin and C-Reactive Protein (CRP) were greater than control, while the mean levels of growth hormone and lactate dehydrogenase were higher than control, according to the findings. Only 13.1% of the children had raised S-GPT during treatment, according to LFT, but all of the children had low S-bilirubin. On the other hand, 47% of the youngsters had a decreased prothrombin time and 39.9% had a decreased albumin.

Table 2 Comparison of biochemical markers in the children before chemotherapy and after induction of remission of chemotherapy

Tests	ALL PATIENTS	CONTROLS
LDH (U/L)	694.11±83.3	183±23.7
Ferritin (ng/ml)	277.3±121.9	67.42±9.33
GH (ng/ml)	9.2 ±1.9	6.2±2.8
CRP (mg/L)	10.1±6.11	7.44± 4.7
S-bilirubin (mg/dl)	0.8 ± 0.2	1.2 ± 0.6
S-GPT (IU/L)	33.0 ± 2.0	95.4 ± 17.9
Prothrombin-time (sec)	12.9 ± 0.2	12.7 ± 0.9
S-albumin (g/dl)	4.2 ± 0.02	3.6 ± 0.02

*The Significant differences at P≤0.05

5. Discussion

This study showed that children are diagnosed with ALL has a significant increase in some blood cells such as ESR, WBC and Red blood cells (RBC). Increased erythrocytes, and ESR can be associated anemia associated with ALL cancer, in general, including Acute Lymphoblastic Leukemia. By inhibiting RBC, bone marrow transduction can be connected to ALL. A variety of cancers that cause hemolysis in red blood cells (anemia) and blood cells may be linked to infection [21]. All red cell indices of cases were lower than the healthy (control) group in a 2006 study that assessed the prevalence of anemia in cancer patients, but Compared to the healthy group, cancer patients' mean RDW (the coefficient of variation of red blood cell anisocytosis) was larger [22]. And a significant of ALL decrease in other some blood parameters such as hemoglobin, PCV, Platelets and Lymphocytes. Several clinical investigations worldwide have verified that interactions between tumor cells and WBCs and platelets can result in quantifiable alterations in blood images. Furthermore, research has identified potential diagnostic values for the ALL-blood test parameter indicator [23-27].

It was shown that, in comparison to patients with benign proliferative acute lymphoblastic leukemia, patients with ALL had considerably lower neutrophils for lymphocyte levels. According to multiple studies, patients with the greatest 5-year death rates had both the platelet-lymphocyte ratio (PLP) and the neutrophil-to-lymphocyte ratio (NPL), both of which concur with the current investigation [28, 29]. Because the study's blood's hemoglobin and red blood cell (RBC) contents significantly increased [30, 31], blood parameters were also examined in accordance with the findings that cancer induces myeloid suppression and anemia. The adoption of mitochondria for di hydro rotate dehydrogenase, a crucial enzyme in de novo pyrimidine biosynthesis, is one of the most significant processes suggested by mitochondrial malfunction in alterations in nucleotide pools [32].

Lactate dehydrogenase (LDH) activity was significantly higher ($p \leq 0.005$) in ALL patients (694.11±83.3.3U/L) than in the healthy group (183±23.7U/L), as seen in Table 2 [33]. This could be because people with acute lymphoblastic leukemia require more glucose breakdown, which raises the enzyme's activity [34], and LDH is progressively elevated in cell damage [35]. Consequently, LDH may be regarded as an indication tumor marker for patients with acute lymphoblastic leukemia. Anaerobic glucose metabolism may be the cause of tumor breakdown syndrome in leukemia patients [35]. Ferritin levels in ALL patients were significantly higher ($p \leq 0.0001$) at (277.3±121.9ng/ml) than in the healthy group (67.42±9.33 ng/ml), according to table 2 data [36]. This could be the result of cell injury, which would raise the amount of ferritin released into the blood. Additionally, elevated serum ferritin levels are caused by enhanced transfer receptors on leukemia cells that reproduce malignantly, as well as an increase in the rate at which cells are destroyed, which exposes them to ferritin transfer and raises serum ferritin levels [37]. In contrast, leukemia patients' growth hormone levels (9.2 ±1.9 ng/ml) were non-significantly higher ($p \leq 0.005$) than those of the healthy group (6.2±2.8 ng/ml). The findings in Table 2 also revealed that the C-reactive protein levels of patients with acute lymphoblastic leukemia (10.1±6.11 mg/L) were non-significantly lower ($p=0.146$) than those of healthy individuals (7.44± 4.7 mg/L). These findings contradict [38], presumably as a result of the male-to-female ratio and the different sample size under investigation. With the exception of a brief rise in serum SGPT during remission induction, the current investigation showed that ant leukemic treatment administered for remission induction did not cause hepatotoxicity. A liver test is deemed clinically relevant if the SGPT level is more than three times the upper limit of normal values and the total billirubin concentration is more than twice the upper limit. Increases in albumin concentration, prothrombin time, and bilirubin levels are indicators of overall liver function, while elevated blood enzyme levels are believed to be an indication of liver injury [39]. The SGPT levels of only three patients in this study were more than three times the upper limit of a normal range. S-albumin, S-billirubin, and prothrombin-time levels did not alter substantially. Pediatricians treating ALL cases are particularly concerned about the fact that, in spite of improvements in the care of children with acute lymphoblastic leukemia, Studies reveal that the liver is still negatively impacted by the present chemotherapy. There is no question that the true prevalence of subclinical hepatic disease is greater. This is due to the rarity of faulty liver function tests detecting subclinical cirrhosis. Liver biopsies or liver scans are rarely performed after treatment, and transaminasemia may not show any signs [40].

Figure.1 show how the factors of chemotherapy for patients with ALL are related to hemoglobin (Hb) and red blood cells (RBC). According to the results, chemotherapy courses significantly reduced the levels of Hb and raised RBC, which is in line with [30]. One possible explanation for this could be that hemoglobin is released when patients with acute lymphocytic leukemia experience cell crashes.

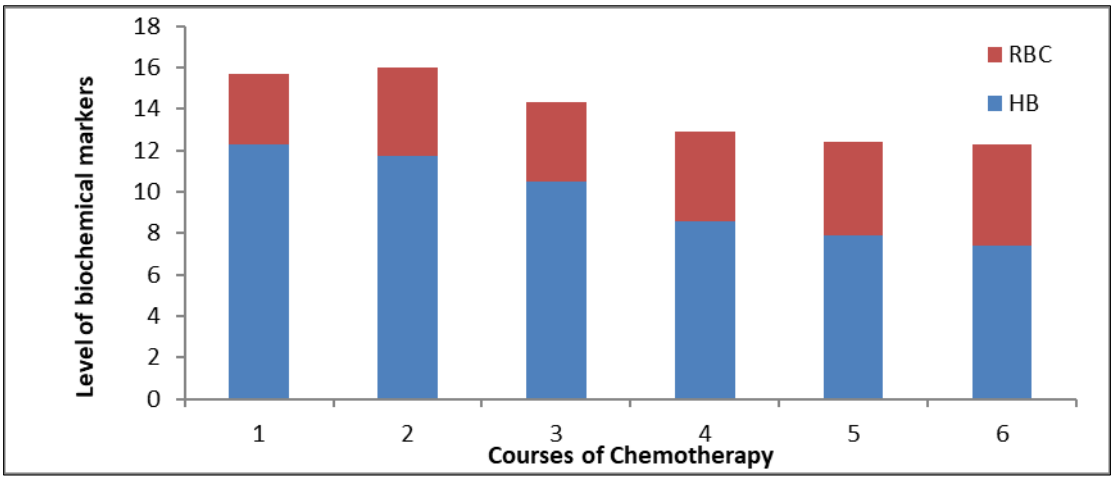

Figure 1 Relationship between serum Hb and RBCs levels variation in cancer patients across five rounds of chemotherapy

Figure.2 show how the investigated variables of chemotherapy for ALL patients relate to serum albumin. According to the data, the amount of serum albumin increased significantly during the treatment course, which is in line with [14]. This could be because patients with acute lymphocytic leukemia experience cell crashes, which cause serum albumin to be released.

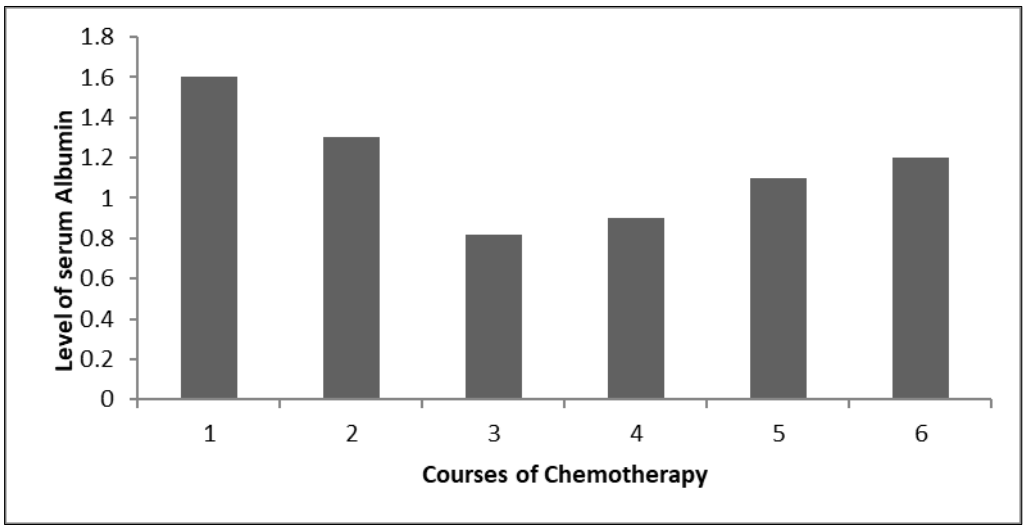

Figure 2 Relationship between serum Albumin and courses of chemotherapy in acute lymphocytic leukemia

6. Conclusion

Hematological parameters: Increase levels of ESR, hemoglobin (Hb), WBC and Red blood cells (RBC) but decrease levels packed cell volume (PCV), Platelets and Lymphocytes diagnostic power and can discriminate patients with ALL from patients without cancer, adding increase in levels some biochemical parameters such as Lactate dehydrogenase (LDH), Ferritin, GH, CRP, Prothrombin-time and S-albumin but decrease level of S-bilirubin and S-GPT in patients become Screening Markers the gold standard in acute lymphocytic leukemia screening and thereby increase the early detection of cancer.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare that they have no conflict of interest.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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