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The Impact of Ribulose-5-Phosphate Isomerase Enzyme and Zinc on Leukemia

Maha Fadel Mohammed, Asmaa Hashim Shaker
Tikrit University, College of Education for Women, Department of Chemistry, Tikrit, Iraq

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Annotation: Leukemia is a malignancy that affects blood cells and the tissues responsible for their formation, such as bone marrow and the lymphatic system. It results from mutations in hematopoietic stem cells, leading to abnormal proliferation and accumulation of cells, which impairs normal hematopoiesis. Types of leukemia include acute myeloid leukemia (AML), acute lymphoblastic leukemia (ALL), chronic myeloid leukemia (CML), and chronic lymphocytic leukemia (CLL). Common symptoms include anemia, bleeding, recurrent infections, and hepatosplenomegaly. Diagnosis is based on blood tests, bone marrow biopsy, genetic analysis, and imaging studies. Treatment options include chemotherapy (mainstay for AML, though less effective in the elderly) and stem cell transplantation, especially in younger patients. This study investigates the activity of the enzyme ribulose-5-phosphate isomerase (RPI) and evaluates the role of zinc in the metabolic and biochemical status of patients with leukemia. Understanding these factors may provide insights into the disease's pathophysiology and potential therapeutic targets.

Keywords: Ribulose-5-phosphate isomerase; Leukemia; Zinc.

Introduction

Leukemia, a malignancy of the hematopoietic system, represents a critical challenge in oncology due to its aggressive nature and complex pathophysiology. It involves the uncontrolled proliferation of abnormal white blood cells, impairing normal blood formation and immune function. Among its major forms acute and chronic variants of lymphocytic and myeloid leukemia the disease presents with symptoms ranging from anemia and infection to systemic organ involvement. Modern research increasingly recognizes the importance of metabolic and biochemical regulators in cancer development, notably enzymes and trace elements critical for cellular homeostasis.

A particularly significant enzyme in this context is **ribulose-5-phosphate isomerase (RPI),** a key player in the pentose phosphate pathway (PPP), which governs nucleotide synthesis and oxidative stress regulation. Meanwhile, **zinc**, an essential micronutrient, is involved in DNA repair, apoptosis, immune modulation, and enzymatic reactions. Disruption of either component may influence leukemogenesis or disease progression. However, the mechanistic link between RPI activity, zinc levels, and leukemia remains poorly characterized, especially in clinical patient cohorts.

Existing literature highlights RPI's role in supporting rapid cell proliferation and redox balance, suggesting its suppression could impair DNA synthesis and antioxidant capacity conditions conducive to malignant transformation. While several studies have explored zinc deficiency and immune compromise in cancer, few have specifically quantified serum zinc in leukemia patients in conjunction with enzymatic markers. This knowledge gap calls for a focused investigation into the dual influence of RPI and zinc as metabolic and biochemical indicators in leukemia.

This study adopts an empirical approach, using enzyme-linked immunosorbent assay (ELISA) to quantify RPI concentration and spectrophotometric methods to assess serum zinc levels in diagnosed leukemia patients versus healthy controls. The objective is to evaluate whether statistically significant differences in these biomarkers exist, and if so, how they may correlate with known leukemia pathophysiology. By combining clinical sampling with biochemical assays, the research aims to bridge the gap between metabolic function and disease diagnosis or progression.

Preliminary findings reveal a significant decrease in RPI levels among leukemia patients, implying impaired metabolic adaptation and potential vulnerability to oxidative damage. In contrast, zinc levels do not exhibit statistically significant differences, though a mild downward trend was observed. These results suggest that RPI may serve as a more sensitive biomarker for leukemia-associated metabolic disruption, while zinc’s subtle variations warrant further exploration. The implications of this work extend toward the development of diagnostic tools and therapeutic strategies targeting metabolic vulnerabilities in leukemia

Materials and Methods

1. Determination of Ribose-5-phosphate Isomerase (RPI) in Blood Serum

The method employed is an enzyme-linked immunosorbent assay (ELISA). The assay plate is pre-coated with antibodies specific to human RRM1 protein. Upon adding the serum sample, the RRM1 protein binds to the immobilized antibody. Then, a biotin-labeled anti-RRM1 antibody is added, forming a complex. Streptavidin conjugated to horseradish peroxidase (HRP) binds to the biotin-labeled antibody. Following incubation, unbound substances are washed off, and a substrate solution is added. The intensity of the resulting color is proportional to the concentration of human RRM1. Absorbance is measured at 450 nm.

2. Zinc Concentration Estimation in Blood Serum

Zinc reacts with a chromogen in the reagent to form a colored complex, where the intensity of color is proportional to the zinc concentration. The reagents include borate buffer (pH 8.2),

Salicyladoxime, and NITRO-PAPS. Absorbance is measured at 578 nm after a 5-minute reaction at room temperature.

Results and Discussion

The concentration of ribulose-5-phosphate isomerase in the blood serum of leukemia patients and healthy controls was measured. The control group had a mean ± standard deviation of 368.4 ± 48.3 ng/L, while the leukemia patients had a significantly lower level of 259.9 ± 40.8 ng/L. The p-value ($p < 0.001$) indicates a statistically significant decrease in enzyme levels among leukemia patients, suggesting impaired metabolic regulation in cancerous cells.

RPI plays a vital role in nucleotide synthesis and oxidative stress resistance via the pentose phosphate pathway. Its suppression in leukemia may reflect altered cellular demands or metabolic shifts. The enzyme's involvement in rapid cell division and NADPH generation underlines its potential as a therapeutic target. In contrast, the serum zinc levels showed no statistically significant difference between patients and controls ($p > 0.05$), with mean values of 149.8 ± 12.2 mg/dL in patients and 154.0 ± 21.4 mg/dL in controls. Although not significant, the slight reduction in zinc levels may still carry biological importance, as zinc is critical for immune function, genome stability, and enzymatic activities.

Research suggests that even marginal zinc deficiency could impair anti-tumor immunity, particularly by affecting T cell maturation, IFN-γ secretion, and NK cell cytotoxicity. Zinc's influence on Notch signaling and inflammatory modulation through NF-κB and STAT3 pathways further supports its relevance in cancer biology.

Conclusions

The observed significant reduction in RPI levels in leukemia patients suggests metabolic dysregulation, which may represent a potential therapeutic target. Meanwhile, zinc levels, though not statistically different, indicate a trend that warrants further investigation due to zinc's immunological and genomic roles. These findings underline the need for continued research into the biochemical underpinnings of leukemia.

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