Evaluation of Serum Erythropoietin, Ferritin, Calcium, Magnesium,and Inorganic Phosphorus Levels in β-Thalassemia Patients

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Abstract

Background: Beta-thalassemia is a common inherited chronic blood disorder characterized by defective β-globin synthesis, leading to chronic anemia and complications related to iron overload. Erythropoietin (EPO), ferritin, calcium, magnesium, and inorganic phosphorus are important biomarkers that may reflect disease severity and metabolic alterations in these patients**. Aim**: This study aimed to evaluate and compare serum levels of Erythropoietin (EPO), Ferritin, Calcium, Magnesium, and Inorganic Phosphorus between β-thalassemia patients and healthy controls. **Methodology:** This cross-sectional study included 70 patients diagnosed with beta-thalassemia, recruited from the Oncology and Hematology Center at Tikrit Teaching Hospital and the Thalassemia, Hematology, and Genetic Diseases Center in Sulamaniyah. Patients were classified according to clinical severity into 30 with beta-thalassemia major, 20 with beta-thalassemia intermedia, and 20 with beta-thalassemia minor. In addition, 20 apparently healthy individuals were enrolled as a control group. Blood samples were collected between September 2025 and December 2025. The study population comprised 41 females and 49 males, with ages ranging from 2 to 25 years**. Results:** Erythropoietin levels showed significant elevation in β-thalassemia minor (245.3 ± 48.3 mIU/mL) compared to controls (183.6 ± 4.9 mIU/mL) ($p=0.003$)**.** Ferritin levels exhibited marked elevation in β-thalassemia intermedia (875.3 ± 722.7 ng/mL) and β-thalassemia major (1723.1 ± 585.3 ng/mL) compared to controls (92.7 ± 37.7 ng/mL) ($p<0.001$). Mineral homeostasis analysis showed selective alterations in β-thalassemia patients compared to controls. First, magnesium

levels remained unchanged across all groups (p=0.591). In contrast, inorganic phosphorus levels were significantly elevated in all β-thalassemia groups (6.0 ± 2.0 mg/dL in β-thalassemia minor, 6.4 ± 2.2 mg/dL in β-thalassemia intermedia, and 6.3 ± 1.8 mg/dL in β-thalassemia major) compared to controls (4.1 ± 0.4 mg/dL) (p<0.001). Calcium levels showed no significant changes across all groups (p=0.485). **Conclusions**: β-thalassemia patients exhibit marked increases in EPO, ferritin, and inorganic phosphorus, while calcium and magnesium remain stable. These findings highlight the importance of monitoring hematologic and mineral biomarkers to assess disease impact and guide management strategies**.**

Key word Beta-thalassemia, Erythropoietin , Ferritin, Calcium, Magnesium, Inorganic Phosphorus.

تقييم مستويات الإريثروبويتين، الفيريتين، الكالسيوم، المغنيسيوم، والفوسفور غير العضوي في مصل الدم لدى مرضى بيتا-ثلاسيميا

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الملخص

الخلفية: تُعد الثلاسيميا بيتا اضطرابًا دمويًا وراثيًا مزمنًا شائعًا يتميز بخلل في تصنيع سلاسل بيتا-غلوبين، مما يؤدي إلى فقر دم مزمن ومضاعفات مرتبطة بفرط تحميل الحديد. يُعد كل من الإريثروبويتين(EPO) ، الفيريتين، الكالسيوم، المغنيسيوم، والفوسفور غير العضوي مؤشرات حيوية مهمة قد تعكس شدة المرض والتغيرات الأيضية لدى المرضى. الهدف: هدفت هذه الدراسة إلى تقييم ومقارنة مستويات الإريثروبويتين(EPO) ، الفيريتين، الكالسيوم، المغنيسيوم، والفوسفور غير العضوي في مصل الدم بين مرضى الثلاسيميا بيتا والأفراد الأصحاء. المنهجية: أُجريت هذه الدراسة المقطعية على 70 مريضًا مشخصين بالثلاسيميا بيتا، جرى تجنيدهم من مركز الأورام وأمراض الدم في مستشفى تكريت التعليمي، ومن مركز الثلاسيميا وأمراض الدم والأمراض الوراثية في السليمانية. تم تصنيف المرضى حسب الشدة السريرية إلى 30 حالة ثلاسيميا بيتا كبرى، و20 حالة متوسطة، و20 حالة صغرى. كما تم تضمين 20 فردًا سليمًا ظاهريًا كمجموعة سيطرة. جُمعت عينات الدم خلال الفترة من سبتمبر إلى ديسمبر 2025. وتكوّن مجتمع الدراسة من 41 أنثى

و49 ذكرًا، تراوحت أعمارهم بين 2 و25 سنة. النتائج: أظهرت مستويات الإريثروبويتين ارتفاعًا معنويًا في مرضى الثلاسيميا الصغرى (245.3 ± 48.3 ملي وحدة/مل) مقارنةً بمجموعة السيطرة (183.6 ± 4.9 ملي وحدة/مل) .(p=0.003) كما أظهرت مستويات الفيريتين ارتفاعًا ملحوظًا في مرضى الثلاسيميا المتوسطة (875.3 ± 722.7 نانوغرام/مل) والثلاسيميا الكبرى (1723.1 ± 585.3 نانوغرام/مل) مقارنةً بمجموعة السيطرة (92.7 ± 37.7 نانوغرام/مل).(p<0.001) . فيما يتعلق بتوازن المعادن، لم تُظهر مستويات المغنيسيوم فروقًا معنوية بين جميع المجموعات .(p=0.591)في المقابل، ارتفعت مستويات الفوسفور غير العضوي بشكل معنوي في جميع مجموعات الثلاسيميا (6.0 ± 2.0 ملغم/دل في الصغرى، 6.4 ± 2.2 ملغم/دل في المتوسطة، و6.3 ± 1.8 ملغم/دل في الكبرى) مقارنةً بمجموعة السيطرة (4.1 ± 0.4 ملغم/دل) .(p<0.001) أما مستويات الكالسيوم فلم تُظهر فروقًا معنوية بين المجموعات.(p=0.485) . الاستنتاجات: يُظهر مرضى الثلاسيميا بيتا ارتفاعًا واضحًا في مستويات الإريثروبويتين، الفيريتين، والفوسفور غير العضوي، في حين تبقى مستويات الكالسيوم والمغنيسيوم مستقرة نسبيًا. تؤكد هذه النتائج أهمية متابعة المؤشرات الدموية والمعدنية لتقييم تأثير المرض وتحسين استراتيجيات التدبير العلاجي.

الكلمات المفتاحية: الثلاسيميا بيتا، الإريثروبويتين، الفيريتين، الكالسيوم، المغنيسيوم، الفوسفور غير العضوي.

Introduction

Beta-thalassemias represent a heterogeneous group of inherited hemoglobin disorders transmitted in an autosomal recessive manner and characterized by defective β-globin chain synthesis. The condition arises from mutations in the HBB gene located on chromosome 11, leading to partial or complete deficiency in β-globin production. Depending on the zygosity of the mutations, affected individuals may be homozygous or compound heterozygous, resulting in an imbalance between α- and non-α-globin chains. This imbalance disrupts normal erythropoiesis, reduces the formation of functional hemoglobin A, and produces fragile, short-lived red blood cells [1]. Beta-thalassemia is categorized into three clinical forms-beta-thalassemia minor (trait), beta-thalassemia intermedia, and beta-thalassemia major-based on the severity of the underlying genetic defect **[2]**. Beta – thalassemia minor is typically asymptomatic; however, it may be associated with mild anemia and nonspecific symptoms such as fatigue and general weaknes. Beta-thalassemia intermedia is characterized by moderate to severe anemia with variable clinical manifestations, including fatigue, jaundice,

skeletal change, hepatosplenomegaly, and gallstones, and may require intermittent blood transfusions. Beta-thalassemia major is characterized by severe chronic hemolytic anemia that manifests in early childhood and necessitates lifelong regular blood transfusions, commonly accompanied by iron chelation therapy to prevent iron overload-related complications affecting the heart, liver and endocrine system **[1]**. β-Thalassemia has historically been most prevalent in the Mediterranean region, the Middle East, and Southeast Asia,but increasing global migration has led to its rising incidence in traditionally non-epidemic regions such as Western Europe and North America, highlighting its growing worldwide public health bueden[1]. Erythropoietin (EPO) is an endocrine hormone and is glycoprotein composed of 166 amino acids, primarily produced in the kidneys during puberty. It functions simultaneously as a peptide hormone and a hematopoietic growth factor, stimulating red blood cell production in the bone marrow. EPO secretion is regulated by an oxygen-sensitive feedback loop, and its biological action is mediated by binding to its homodimeric receptor (EPO-R), promoting cell survival and driving the terminal maturation of BFU-Es and CFU-Es into large numbers of mature red blood cells. During early embryonic development, EPO is produced in the fetal liver, while in adult, the kidneys become its primarly source. Its molecular weight is approximately 30.4 kDa[3]. EPO production is primarily regulated by oxygen levels in the body. When tissues are exposed to hypoxia, the kidneys ability to secrete EPO increases, which stimulates the formation of red blood cells and contributes to improving oxygen delivery to tissue **[4]**. Regular blood transfusions, which represent the cornerstone of management in severe β-thalassemia, inevitably lead to progressive iron overload. Excess iron accumulates in vital organs such as the liver, heart, and endocrine glands, resulting in significant functional impairment [5]. Furthermore, iron excess promotes the generation of reactive oxygen species, contributing to oxidative stress and the development of chronic inflammatory processes [6]. Serum ferritin is commonly used as an indirect marker of total body iron stores, and elevated levels are often associated with increased iron burden and related clinical complications [7]. Despite the significant improvement in survival rates achieved through supportive therapies, patients with β-

thalassemia remain at risk for multiple complications that adversely affect their quality of life, including cardiac, endocrine, hepatic, and renal complications. Renal involvement is primarily associated with the underlying pathophysiology of the disease, such as chronic anemia and renal hypoxia, in addition to iron overload resulting from repeated blood transfusions and the use of iron chelation therapy. The mechanisms contributing to renal dysfunction in β-thalassemia commonly include alterations in vascular resistance, increased renal plasma flow, elevated glomerular filtration, and tubular abnormalities [8]. Calcium is an essential mineral in the human body, playing a critical role in the formation and maintenance of bones and teeth. In addition, it is involved in several vital physiological processes, including muscle contraction, nerve impulse transmission, hormone secretion, intracellular signaling, and blood coagulation [9]. Phosphorus is one of the most abundant minerals in the human body and plays a central role in numerous cellular processes. It is essential for maintaining skeletal integrity and is a key component of phospholipid bilayers in cell membranes. Additionally, phosphorus is involved in cellular signaling pathways, nucleic acid synthesis, and energy metabolism as a fundamental component of adenosine triphosphate (ATP) [10]. Magnesium (Mg) is an essential mineral involved in numerous physiological processes. It acts as a cofactor in over 300 enzymatic reactions and plays a key role in nucleic acid synthesis, energy metabolism, cell membrane stability, and the regulation of bone and calcium homeostasis, as well as supporting nervous and immune system function **[10].** The aim of this study was to evaluate the levels of EPO in patients with Beta-Thalassemia. Additionally, the study aimed to assess the concentrations of Ferritin, Calcium, Magnesium, and Inorganic Phosphorus, and to determine the impact of the disease on these parameters.

Materials and Methods

Study Design and Population

This cross-sectional study was conducted between September 2025 and December 2025 and included a total of 90 participants (41 females and 49

males) aged 2-25 years. The study population was divided into four groups: 30 patients with β-thalassemia major, 20 with β-thalassemia intermedia, 20 with β-thalassemia minor, and 20 healthy controls. Patients were recruited from Tikrit Teaching Hospital and the Thalassemia and Hereditary Blood Disorders Centers in Sulaymaniayah. All patients were previously diahnosed using hemoglobin electrophoresis (Hb electrophoresis) and complete blood count (CBC) by the centers physicians.

Sample Collection and Processing

Five millileters of peripheral venous blood were collected from each participant into gel-containing tubes. The samples were allowed to clot at room temperature, followed by centrifugation at 5000 rpm for 10 minutes. The separated serum was transferred into Eppendrof tubes and syored at -20 C until analysis.

Biomarker Analysis

Serum levels of Erythropoietin (EPO) was assessed using commercially available enzyme-linked immunosorbent assay (ELISA) kits (Sunlong Biotech, China), following the manufacturers protocols. Serum ferritin levels were measured using the Cobas e411 analyzer (Roche, Germany), while inorganic phosphorus (P), magnesium (Mg), and calcium (Ca) were assessed using the Biolis 30i automated analyzer (Japan). All assays were performed according to the manufacturers' protocols under standardized laboratory conditions to ensure accuracy, reproducibility, and reliability of the results.

Ethical Apoproval

The study protocol was reviewed and approved by the Ethics Committee of the University of Tikrit. All laboratory procedures were conducted in the central laboratories of the University of Tikrit. Informed consent was obtained from all participants or their legal guardians before sample collection.

Statistical Analysis

The Shapiro-Wilk test ($\alpha = 0.05$) was used to test data normality. Continuous variables were reported as mean ± SD or median (IQR), accordingly. Differences among groups were assessed using ANOVA or Kruskal-Wallis tests, and post-hoc analyses were performed where necessary. The level of statistical significance was $p < 0.05$. Python (version 3.13.5), together with the NumPy, pandas, SciPy, and Matplotlib libraries, was used to perform all analyses and visualizations.

Results

This study involved 90 participants, including 41 females and 49 males between the ages of 2 and 25 years. The population under study was divided into four groups including; β-thalassemia major (n = 30), β -thalassemia intermedia (n = 20), β -thalassemia minor (n = 20) and healthy controls (n = 20).

A total of 90 participants were enrolled including 41 females and 49 males, with ages ranging from 2 to 25 years. No significant differences were observed among the study groups regarding age ($p = 0.381$) or sex distribution ($p = 0.416$), indicating comparable baseline characteristics between patients and control. AS shown in Table (1).

Table 1: The demographic characteristics of the study population.

Parameter	**Group**	**N**	**Mean± SD/n(%)**	**Median (IQR)**	**Mean diff Vs Control**	**p-value vs control**	**Overall p-value**

Age (years)	**Control**	20	16.85 ± 6.45	17.50 (8.25)	Reference	Reference	0.381
	β-thalassemia minor	20	15.45 ± 6.82	15.00 (9.75)	-1.40	0.509	
	β-thalassemia intermedia	20	15.55 ± 7.00	16.50 (11.00)	-1.30	0.545	
	β-thalassemia major	30	13.47 ± 7.15	13.50 (10.50)	-3.38	0.095	
Sex (M/F)	**Control**	20	12 (60)/ 8 (40)	—	—	—	0.416
	β-thalassemia minor	20	13 (65)/ 7 (35)	—	—	—	
	β-thalassemia intermedia	20	8 (40)/ 12 (60)	—	—	—	
	β-thalassemia major	30	16 (53.3)/ 14 (46.7)	—	—	—	

Serum Levels of Erythropoietin (EPO), Ferritin, Calcium, Magnesium, and Inorganic Phosphorus in β-Thalassemia Patients and Healthy Controls

Table 2. Serum levels of Erythropoietin (EPO), Ferritin, Calcium, Magnesium, and Inorganic Phosphorus among β-Thalassemia patients and healthy controls

Parameter	Group	N	Mean ± SD/n (%)	Median (IQR)	Mean diff vs Control	*p*-value vs Control	Overall *p*-value
Erythropoietin (mIU/mL)	**Control**	20	183.58 ± 4.86	183.17 (7.62)	*Reference*	*Reference*	**0.003**
	β-thalassemia minor	20	245.28 ± 48.32	232.95 (73.06)	61.70	**<0.001**	
	β-thalassemia intermedia	20	298.77 ± 246.99	220.02 (160.56)	115.19	0.076	
	β-thalassemia major	30	312.03 ± 373.92	189.41 (122.20)	128.45	0.804	
Ferritin (ng/mL)	**Control**	20	92.70 ± 37.72	86.34 (54.78)	*Reference*	*Reference*	**<0.001**
	β-thalassem	2	106.60 ±	81.54	13.90	0.892	

	ia minor	0	75.08	(35.97)			
	β-thalassemia intermedia	20	875.29 ± 722.75	673.50 (1146.50)	782.59	**<0.001**	
	β-thalassemia major	30	1723.07 ± 585.30	1726.00 (957.75)	1630.37	**<0.001**	
Calcium (mg/dL)	**Control**	20	9.39 ± 0.57	9.47 (0.83)	*Reference*	*Reference*	0.485
	β-thalassemia minor	20	9.35 ± 1.44	9.72 (0.74)	-0.04	0.273	
	β-thalassemia intermedia	20	9.39 ± 0.49	9.54 (0.67)	-0.00	0.925	
	β-thalassemia major	30	9.37 ± 0.61	9.38 (0.59)	-0.02	0.866	
Magnesium (mmol/L)	**Control**	20	0.84 ± 0.11	0.85 (0.16)	*Reference*	*Reference*	0.591
	β-thalassemia minor	20	1.00 ± 0.39	0.87 (0.15)	0.16	0.189	
	β-thalassem	2	0.88 ±	0.86	0.03	0.579	

	ia intermedia	0	0.12	(0.10)			
	β-thalassemia major	30	0.89 ± 0.24	0.86 (0.17)	0.05	0.656	
Inorganic phosphorus (mg/dL)	**Control**	19	4.14 ± 0.41	4.15 (0.65)	*Reference*	*Reference*	**<0.001**
	β-thalassemia minor	20	6.05 ± 2.00	5.70 (1.38)	1.91	**<0.001**	
	β-thalassemia intermedia	20	6.42 ± 2.21	5.78 (1.74)	2.28	**<0.001**	
	β-thalassemia major	30	6.33 ± 1.80	5.92 (1.39)	2.19	**<0.001**	

Serum Erythropoietin (EPO) levels differed significantly among the studied groups. The median (IQR) EPO concentrations were 232.95 (73.06) pg/mL in β-thalassemia minor, 220.02 (160.56) pg/mL in β-thalassemia intermedia, and 189.41 (122.20) pg/mL in β-thalassemia major compared to controls 193.17(7.62) pg/mL.These differences were statistically significant (p<0.003). Ferritin levels exhibited marked elevation in β-thalassemia intermedia (875.3 ± 722.7 ng/mL) and β-thalassemia major (1723.1 ± 585.3 ng/mL) compared to controls (92.7 ± 37.7 ng/mL) (p<0.001). Mineral homeostasis analysis showed selective alterations in β-thalassemia patients compared to controls (Table 1, Figure 2). First, magnesium levels remained

unchanged across all groups (p=0.591). In contrast, inorganic phosphorus levels were significantly elevated in all β-thalassemia groups (6.0 ± 2.0 mg/dL in β-thalassemia minor, 6.4 ± 2.2 mg/dL in β-thalassemia intermedia, and 6.3 ± 1.8 mg/dL in β-thalassemia major) compared to controls (4.1 ± 0.4 mg/dL) (p<0.001). Calcium levels showed no significant changes across all groups (p=0.485).

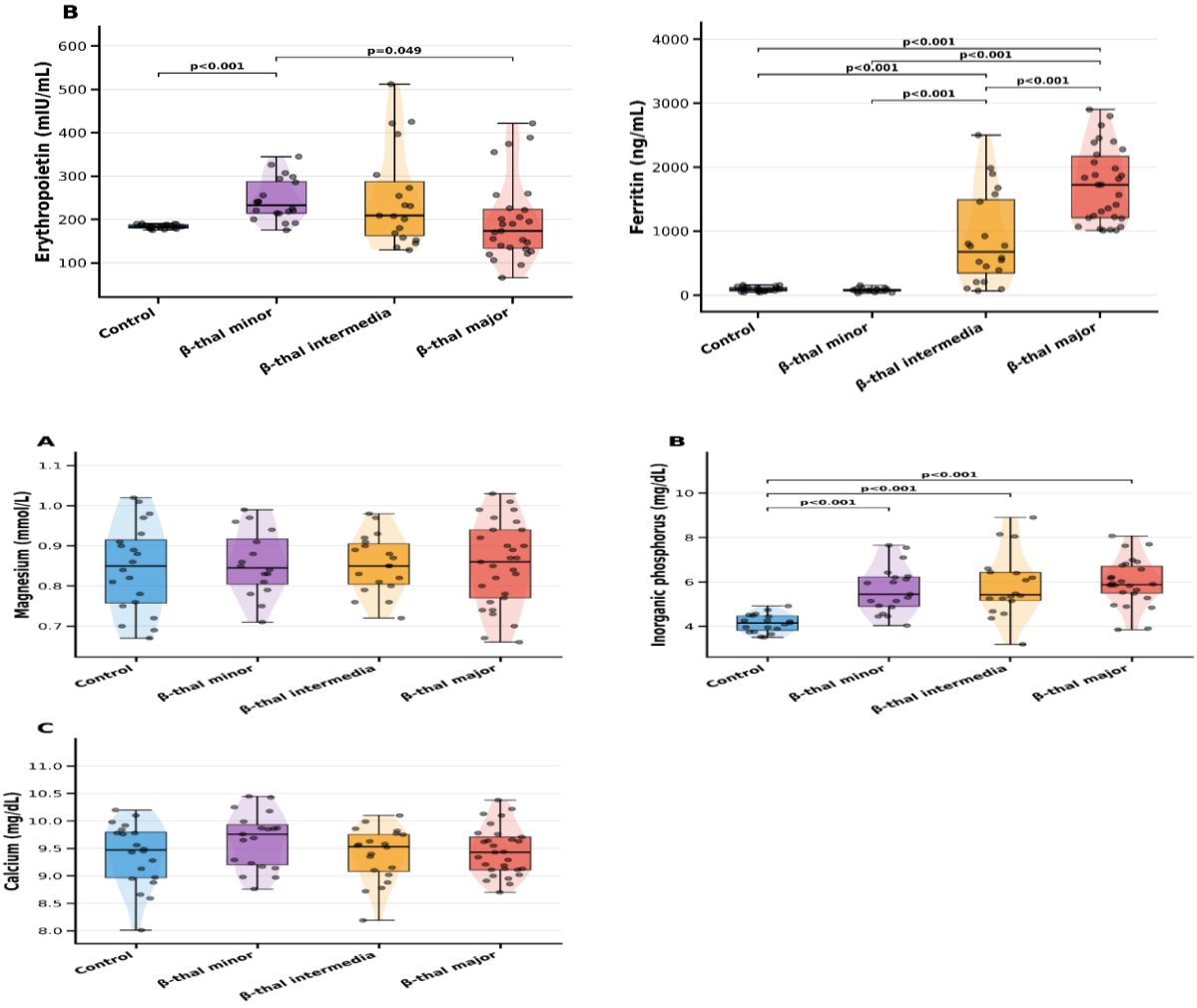

Figure1. Comparison of serum Magnesium (mmol/L), Inorganic Phosphorus (mg/dL), Calcium (mg/dL), and Erythropoietin (EPO, pg/mL) across study groups (Control, β-thalassemia minor, β-thalassemia intermedia, and β-thalassemia major). Data are presented as violin plots showing the probability density distribution, with box plots displaying the median

(horizontal line), interquartile range (box), and whiskers extending to 1.5× IQR. Individual data points are shown as jittered scatter points overlaid on each distribution. Statistical comparisons were performed using appropriate parametric (ANOVA, independent t-test) or non-parametric (Kruskal-Wallis) tests based on data normality assessed by the Shapiro-Wilk test. Only significant pairwise comparisons ($p < 0.05$) are indicated by brackets with p-values**.**

Discussion

Erythropoietin (EPO) is one of the most important hormones that regulate the production of red blood cells in response to hypoxia [11]. Chronic anemia stimulates EPO secretion continuously in β-thalassemia. The results of the current research revealed marked differences in the EPO levels among the patient groups, with elevated levels in all subtypes, the highest level in β-thalassemia minor, and lower levels in the intermedia and major types, as well as very high variability. These findings are in line with other studies, such as [12,13], because they all concur that EPO is a compensatory mechanism in response to chronic anemia and can be used as a marker of the severity of the illness.

Besides EPO, the current study also found that the serum ferritin levels of the 32 patients with β-thalassemia were significantly high as compared to the healthy controls, indicating iron overload, which is one of the symptoms of the disease. These are consistent with [14], who found high levels of ferritin among patients with β-thalassemia major and also emphasized the correlation with other complications like endocrine disorders. The same was found by [15], who explained high levels of ferritin by repeated blood transfusions and related issues, such as cardiac, hepatic, and renal dysfunction. Moreover, the findings correspond to those of [16] who showed that ferritin levels were significantly higher in β-thalassemia intermedia. Contradictory findings, however, were reported by [17], who reported high ferritin in β-thalassemia minor. Conversely, [18] did not observe any significant difference between minor patients and healthy people, and this

indicates that it varies according to the characteristics of the disease and the management of the clinical condition.

As to mineral metabolism, the current paper revealed that there were no statistically significant differences in serum calcium levels among β-thalassemia patients, which proves that calcium homeostasis is preserved in all patients despite the severity of the disease. This is contrary to the report of [19], who found a lower level of calcium in patients with β-thalassemia major receiving regular transfusions, which could be attributed to hypoparathyroidism. The present findings are in line with the findings by [20], who did not observe any significant differences in the level of calcium in patients and controls. This could also be attributed to the nature of β-thalassemia minor, in which the patients are not usually dependent on regular transfusion and are less likely to be affected by iron overload and endocrine complications [21]. Also, dietary calcium and supplementation could have an effect on serum calcium levels, and the absence of dietary evaluation in the current study could have been a cause of the observed normal levels [22].

On the same note, serum magnesium levels did not exhibit any significant differences between the patients and the controls, and they were within normal ranges. This implies that magnesium homeostasis can be maintained in β-thalassemia patients, which might be because of proper clinical care, iron chelation, or proper nutrition. However, previous literature has reported conflicting findings. A study [23] noted high magnesium levels, which they explained by chronic hemolysis and the extracellular release of intracellular magnesium. Conversely, [24] found reduced levels of magnesium, which might be due to oxidative stress, hemolysis, malnutrition, and endocrine disruptions. In addition, magnesium deficiency was associated with decreased PTH secretion, which is caused by iron deposition in the parathyroid gland [25]. However, the current results align with the ones presented by [26], who did not identify any significant differences in magnesium levels between β-thalassemia patients and normal participants.

Unlike calcium and magnesium, the current research showed a significant rise in serum inorganic phosphorus in all the groups of β-thalassemia as compared to controls, with an increase ranging from 45% to 55% across different levels of severity of the disease. These results are in line with those of [19,27], all of whom reported high levels of phosphate in patients with β-thalassemia, especially those receiving frequent blood transfusions. The fact that the phosphorus levels have been on the rise can be explained by a number of factors, such as chronic hemolysis, recurrent transfusions, as well as potential hypoparathyroidism. Moreover, iron overload can have an impact on the regulation of fibroblast growth factor-23 (FGF-23), which is a key factor in the process of phosphate metabolism and thus has a central role in the change of serum phosphorus levels and its renal processing [28].

Altogether, the results of the current research point to the complicated connection between hematological and biochemical parameters in β-thalassemia. While calcium and magnesium levels are stable, EPO and ferritin levels indicate the underlying pathophysiology of chronic anemia and iron overload, and this indicates the apparent control of physiological parameters in some clinical conditions. Conversely, the increase in the level of phosphorus suggests a possible disruption of mineral metabolism and may be connected with endocrine and iron-dependent processes. These findings highlight that comprehensive clinical management that involves monitoring iron status and mineral balance is essential in patients with β-thalassemia.

Limitations

This paper has a number of limitations. To start with, the sample size used is rather small and the study is cross-sectional, which can reduce the applicability of the results and make it impossible to establish any causality between Erythropoietin (EPO) and other analyzed biomarkers. Second, the lack of assessment of some molecular and clinical determinants of inflammation, hypoxia, and iron metabolism might affect the obtained outcomes. Lastly, dietary habits, supplementation and other lifestyle factors that might influence mineral levels were not considered. These findings

require future large-scale, prospective studies to confirm and elaborate on them.

Conclusions

The current report established that serum Erythropoietin (EPO) and ferritin levels are increased in patients with β-thalassemia, which reflects compensatory erythropoiesis and iron overload as a result of frequent transfusions. The serum calcium and magnesium levels were normal, and this showed that under sufficient medical care and chelation therapy, mineral homeostasis was maintained. Conversely, the levels of inorganic phosphorus in serum were elevated, probably due to chronic hemolysis, repeated transfusions, and abnormal regulation of FGF-23. These results indicate the significance of monitoring iron and mineral status to avoid complications and optimize patient care.

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