

Research Article

Assessment Serum Liver Enzymes, Bilirubin and Ferritin in β -Thalassemia Major and Intermedia Compared with Healthy Controls at Al-Zahra Teaching Hospital, Najaf

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Abstract

Background: To assess serum liver enzymes, bilirubin and ferritin in Iraqi patients with β -thalassemia major and β -thalassemia intermedia compared with healthy controls. **Methodology:** In this study, 100 patients (57 females and 43 males) attending Al-Zahra Teaching Hospital in Najaf City were enrolled. They comprised 53 patients with β -thalassemia major and 47 patients with β -thalassemia intermedia. **Results:** The mean age of the patients was 14.28 years. In addition, the study included 30 healthy controls (16 males and 14 females) with an average age of 15.25 years. Both groups were matched for age, gender, socioeconomic status and residence. Serum GOT, GPT, ALP, total bilirubin and ferritin were significantly higher in both patient groups than in controls ($P < 0.001$), with no significant difference in age, gender, WBC count, PCV or ferritin between the β -thalassemia major and intermedia groups. **Conclusion:** Pre-marital detection and medical analyses are crucial to avoiding thalassemia. However, because these tests are often neglected owing to unregistered (out-of-court) marriages and early marriage, the incidence of thalassemia has recently increased in Iraq.

1. Introduction

Thalassemia is a hereditary blood circumstance that is characterized by reduced production of hemoglobin, the main component of red blood cells responsible for oxygen transport. The disorder is caused by a genetic defect inherited from parents to their children and is due to a gene concerned within the creation of hemoglobin. The symptoms of thalassemia rely upon the type and severity of the circumstance; Some newborns show signs and symptoms at birth, whereas others remain asymptomatic until they are several years old. For instance, it is transmitted in beta shape. Individuals who carry only one affected gene are usually asymptomatic or have mild symptoms [1]. More than 200 mutations have been documented in the β -globin gene on chromosome 11. Many of the mutations related to β -thalassemia are commonplace. These mutations alter the nucleotide sequence of the gene. According to De Sanctis et al. [2], abnormalities of the β -globin gene can take diverse forms. Genetic assessments can understand these changes in people and facilitate disease prevention at an early degree. Al-Allawi et al. [3] reported that this technique can also be used for prenatal diagnosis of the disease.

Etiology

Thalassemia is caused by a decreased production of hemoglobin due to the reduced synthesis of one of the polypeptides that comprise the molecule of hemoglobin, namely beta, alpha, gamma, and delta. The disease is inherited in an autosomal recessive manner. Beta thalassemia is caused by a decreased production of beta peptide chains. Individuals that are heterozygous for the condition have various symptoms and are considered to have β -thalassemia trait (minor), which is usually associated with mild anemia. Homozygous individuals have a severe form of anemia called beta thalassemia major or Cooley anemia, this condition is characterized by increased activity in the bone marrow and a severe form of anemia. Alpha thalassemia is caused by a decreased production of alpha globin chains. When a person lacks a gene, a variant that is silent about the alpha gene is present. When the mutation involves of the 4 genes, the person has the characteristic of α -thalassemia trait, which results in mild anemia. Defects that affect three of the 4 genes have a extra impact at the synthesis of alpha chains, this outcomes within the formation of excess β -chain tetramers (HbH) or γ -chain tetramers (Hb Bart's) at some stage in childhood. All four genetic abnormalities are fatal if they may be not handled with intrauterine blood transfusions during pregnancy, this is because hemoglobin can not transport oxygen to the baby's cells without the alpha chain. The second form of thalassemia is associated with hemoglobin E (HbE). HbE is common and is caused by a missense mutation at codon 26 of the β -globin gene. This mutation reduces the production charge of β -globin and alters its structure. As a result, the typical phenotype associated with HbE/ β -thalassemia is β -thalassemia intermedia. The frequency of HbE is particularly high in India, Bangladesh and Southeast Asia [4]. Double heterozygotes for HbE and β -thalassemia commonly require blood transfusion, whereas HbE carriers usually have only mild anemia [5]. HbE/ β -thalassemia is the most common genotype, accounting for approximately half of all cases of severe β -thalassemia worldwide [4]. In addition, patients with HbE/ β -thalassemia are more likely to develop vitamin D deficiency or pulmonary hypertension [6].

Signs and Symptoms

The symptoms of thalassemia are dependent on the seriousness of the genetic flaw. The more severe the genetic flaw, the less hemoglobin is created and the more severe the anemia that results. Thalassemia minor is typically silent and has no symptoms. When this occurs, the most common symptoms are mild anemia and fatigue. Thalassemia major is typically accompanied by a severe case of hemolytic anemia [2].

2. Materials and methods

We collected blood samples from 100 patients (57 females and 43 males) who attended the Thalassemia Center of Al-Zahra Teaching Hospital over a two -month period, from April to May 2025. The patients were divided into two groups: 53 with β -thalassemia major and 47 with β -thalassemia intermedia. The mean age of the patients was 14.28 years. For assessment, the control group of this study comprised 30 healthy individuals (16 males and 14 females), with a median age of 15.25 years.

3. Results and Discussion

Investigations include 47 patients with thalassemia who are intermediate in degree and 53 patients with thalassemia, which are the main and treatment in Al-Zahra Teaching Hospital, Najaf Governorate. Patients with thalassemia major and thalassemia intermediate had comparable demographic and clinical characteristics, as shown in Table 1. There was no significant difference between age, gender, WBC or PCV between patients with BTM and BTI. Serum ferritin did not differ significantly between BTM and BTI (3215 ± 1693 vs. 2720 ± 1425 ng/mL, respectively; $P > 0.05$). There was no significant difference in sex, age, WBC calculation, PCV value or serum ferritin level between patients with BTM and BTI, though the iron content of both groups was significantly higher than the control group. Serum ferritin is used to monitor iron overload and the effectiveness of iron-chelation therapy; patients without a marked rise in iron stores have a better prognosis. There are causes of elevated iron content in patients, including increased intestinal iron absorption due to ineffective erythropoiesis and frequent blood transfusions. Some research has shown the same result [7].

Table 2 also demonstrates that the concentrations of GOT and GPT had been substantially more than those of the control group ($P < 0.001$). Blood ALP and overall bilirubin ranges were significantly higher in patients with thalassemia than in controls ($P < 0.001$), whereas no significant differences were found between the BTM and BTI groups. Other investigations have demonstrated that GOT, GPT, ALP, iron and ferritin levels are elevated in BTM as compared to controls [8].

Table 1: Demographic and hematological characteristics of patients with β -thalassemia major (BTM) and β -thalassemia intermedia (BTI). Data are presented as mean $\pm$ SD

	BTM (n=53)	BTI (n=47)	P- value
Age (year)	12.32 $\pm$ 7.42	16.58 $\pm$ 13.75	Ns*
Sex (M/F)	28/25	29/18	Ns*
W.B.C count (cell/c.mm)	7.93 $\pm$ 3.55	7.21 $\pm$ 2.83	Ns*
P.C.V (%)	29.39 $\pm$ 4.59	28.82 $\pm$ 3.81	Ns*
Ferritin (ng/mL)	3215 $\pm$ 1693	2720 $\pm$ 1425	Ns*
NS*: Not significant			

Table 2: Mean $\pm$ SD of each parameter (GOT, GPT, ALP, bilirubin, and ferritin) in BTM, BTI patients, and controls:

	BTM n=53	BTI n=47	Controls n=30	P-value ANOVA	BTM vs BTI	BTM vs Controls	BTI vs Controls
GOT	44.707 $\pm$ 24.09	41.53 $\pm$ 16.68	20.70 $\pm$ 8.80	P<0.001	0.441 (NS)	<0.001*	<0.001*
GPT	54.83 $\pm$ 20.12	50.31 $\pm$ 11.21	15.08 $\pm$ 3.34	P<0.001	0.163 (NS)	<0.001*	<0.001*
ALP	157.53 $\pm$ 90.31	146.37 $\pm$ 88.21	75.09 $\pm$ 25.3	P<0.001	0.93	<0.001*	<0.001*
Bilirubin	24.31 $\pm$ 11.31	29.61 $\pm$ 15.31	9.11 $\pm$ 3.12	P<0.001	0.09	<0.001*	<0.001*
Ferritin (ng/mL)	3215 $\pm$ 1693	2720 $\pm$ 1425	113.54 $\pm$ 93.23	P<0.001	0.086	<0.001*	<0.001*

Note: GOT, GPT, ALP in U/L; bilirubin in μ mol/L; ferritin in ng/mL. *Significant at P<0.05; NS: not significant.

Article Information

Ethical Approval: The study was approved by the Scientific Research Ethics Committee of the College of Medical Sciences, Jabir Ibn Hayyan University for Medical and Pharmaceutical Sciences, and the Najaf Health Directorate, Iraqi Ministry of Health (Approval No. 3; dated January 3, 2025). The study was conducted in accordance with the principles of the Declaration of Helsinki.

Informed Consent: Written informed consent was obtained from all participants before their inclusion in the study; for participants younger than 18 years, consent was obtained from their parents or legal guardians.

Consent for Publication: Not applicable. The manuscript does not contain any individual person's identifiable data.

Data Availability: The datasets generated and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Clinical Trial Registration: Not applicable. This is an observational case–control study, not a clinical trial.

Conflict of Interest: The authors declare that they have no conflict of interest.

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Author Contributions: A.H.A. - Conceptualization, Investigation, Data Curation, Formal Analysis, Writing – Original Draft, Project Administration; H.H.A. - Conceptualization, Methodology, Formal Analysis, Resources, Writing – Review & Editing, Supervision.

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Competing Interests: Authors have declared that no competing interests exist.

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