

Examine the Changes in Several Hematological Parameters in The Blood of Beta Thalassemia Patients.

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Abstract: Measuring serum iron, TIBC, and other hematological markers in patients with mild beta thalassimia was the aim of the current investigation. From April 2024 to July 2025, Khanaqin Hospital (blood bank and blood donation center) in Diyala Governorate, Khanaqin city, and its surrounding territories recorded 140 patients with β thalassemia. Private laboratories in the Iraqi region of Diyala carried out the experiments. The participants in this study were divided according to the following: Group A consists of 55 men with mild thalassemia, and There are 55 women with mild thalassemia (Group B). Group C: 30 healthy people without mild thalassemia (control group). Changes in the respective variables were found to be statistically significant ($P < 0.05$). The patients with thalassemia had significantly ($P < 0.05$) lower hemoglobin concentration, PCV, MCV and MCH percentages when compared to the control group.

Keywords: β -thalassemia minor, Hematological parameters, serum iron, TIBC.

Introduction

There are two α and two β chains in normal hemoglobin A. The beta gene clusters are found on chromosome (1), whereas the globin gene clusters are found on chromosome 16. Autosomal recessive diseases include thalassemias. One globin chain's synthesis may be flawed in thalassemia, either by mutation or deletion, leading to an excess of the other chain's production, which harms the red cell membrane (2). The type and severity of anemia affect the hematological parameters in thalassemic individuals. However, practically all forms of thalassemias are characterized by variable degrees of microcytosis. The genetic disorder known as thalassemia minor (trait) is typically asymptomatic. A high red blood cell (RBC) count and moderate anemia are common blood parameter changes, considerably lower mean corpuscular hemoglobin (MCH) and mean corpuscular volume (MCV). Levels of hemoglobin A₂ (HbA_2) are typically high. (3) The kind and severity of anemia affect blood parameters in thalassemia

patients. Anaemia is usually caused by thalassemia major and can manifest months after birth. Individuals with α -thalassemia major receive monthly blood transfusions on a regular basis. This ensures regular growth and helps avoid severe anemia. (4). However, this approach is not precisely adhered to in a weak socioeconomic situation, which leads to a dismal reality in terms of blood maintenance and pathological indicators for these patients' health. Excessive iron deposition in bodily tissues caused by red blood cell deterioration and transfusion results in a number of pathophysiological situations, including increased plasma volume, decreased glucose tolerance, and cardiac output (5). Patients' blood hematology is impacted by both thalassemia and blood transfusions. The clinical manifestation of β -thalassemia varies greatly depending on the patient's age upon diagnosis and the severity of their illness. Asymptomatic thalassemia characteristics (6) Nevertheless, a laboratory test will reveal fluctuations in hemoglobin levels, which may range from normal to up to 2 g/dL with microcytic

hypochromic erythrocytes. HbF and HbA2 levels will rise, according to hemoglobin analysis (7). The erythrocyte index should be routinely used as a screening tool for thalassemia patients, according to Indonesian thalassemia care guidelines. According to this recommendation, The Indonesian Guideline for Management of Thalassemia states that patients with MCV <80 fL and MCH <27 pg should have further testing. The Indonesian Guidelines for the Management of Thalassemia state (8), a HbA2 test between 3.6 and 4.2% indicates mild β^+ thalassemia characteristics. -thalassemia, while values between 4 and 9% indicate severe β^+ -thalassemia and heterozygote β^0 -thalassemia (9).

Objectives

Serum iron level, TIBC should be checked in patient with β thalassemia minor. Numerous hematological indicators (Hb, PCV, MCV, and MCH) should be estimated. • TIBC and levels of iron in the blood should be assessed in patients with mild B thalassemia.

Materials and Methods

Patients

Between April 2024 and July 2025, Khanaqin Hospital (a blood bank and blood donation center) in the Diyala Governorate, Khanaqin city, and its surrounding territories reported 140 cases of β thalassemia. The experiments were conducted in private labs in the Diyala Governorate of Iraq. Four distinct groups comprised the investigation's participants:

55 men with thalassemia mild patients make up Group A.

Group B consists of 55 female patients with thalassemia mild.

Group C consists of thirty healthy individuals who do not have thalassemia minor (the control group group).

Blood Specimen Collecting and Evaluation: Venous blood sample (5 mL) of each patient was taken and divided into an EDTA tube containing 1 mL of blood and a vacutainer plain tube containing 4.0 mL of blood. Scientists disagree on the need for newly drawn blood.

There is disagreement among experts over the necessity of utilizing freshly obtained blood. Therefore, using freshly collected blood is not necessary. Each person was given a 5 mL venous blood sample, which was then divided into a vacutainer plain tube (4.0 mL) and an EDTA tube (1 mL).

After the blood was briefly stored in vacutainer plain tubes to allow for blood coagulation. To create serum samples, the blood was centrifuged for ten minutes at 4000 rpm. The serum was divided into five standard test tubes, sealed, and stored at -20 degrees Celsius until analysis time. After being removed from the freezer and heated to between 4 and 8 degrees Celsius, the serum samples were gently shaken at room temperature to mix.

Complete Blood Counts (CBC)

A 1 mL of blood was taken in an EDTA test tube and analysed on Sysmax device for hematological parameters (RBC, HB, HCT, MCV). Serum iron concentration and total iron-binding capacity (TIBC): human blood contains a protein called transferrin that binds to the iron in the bloodstream to transport it around the body. TIBC can be measured for the detection and monitoring of Transferrin saturation, iron deficiency and chronic inflammatory disorders. The CBC can be used to diagnose a number of diseases and conditions, including leukemia, anemia, and infections. Blood was drawn from patients and tested in a 1 mL EDTA test tube on a Sysmax instrument using hematological tests (RBC, HB, HCT and MCV).

Serum iron content and total iron-binding capacity (TIBC):

Iron deficiency anemia, transferrin saturation, and chronic inflammatory disorders can all be identified and tracked with TIBC measurements. The TIBC test Reagents should be added to the Unbound Iron (A) and TIBC + Unbound Iron (B) wells in the order specified in Table 2. Incubate for the specified duration at 37 °C. TIBC measures can be used to identify and track iron deficient anemia, transferrin saturation, and chronic inflammatory diseases. The Unbound Iron (A) and TIBC + Unbound Iron (B) wells should receive the TIBC test Reagents in the sequence shown in Table 2. At 37 °C, incubate for the specified period of time.

Analysis of Statistics

The Way-One ANOVA statistical program was used to conduct the statistical analysis (11).

Results

Hematological Parameters

As shown in Table 1, individuals with beta thalassaemia

had significantly ($P < 0.05$) reduced hemoglobin concentration (Figure 1), pulmonary capillary wedge pressure (Figure 4.4), mean corpuscular volume (Figure 2), and mean corpuscular hemoglobin concentration (Figure 3). The experimental group had a considerably ($P < 0.05$) lower mean corpuscular volume (Figure 4.4) and a significantly ($P < 0.05$) lower mean corpuscular hemoglobin concentration (Figure 3) compared to the control group (Figure 2).

Table 1 Hematological parameters in the groups under investigation

Groups Parameter	Control (30)		Patients (110)		P-Value
	Male (15)	Female (15)	Male (55)	Female (55)	
Hb (g/dL)	15.82 $\pm$ 1.42	13.92 $\pm$ 1.29	7.51 $\pm$ 0.18*	7.05 $\pm$ 1.31*	0.001
PCV %	44.48 $\pm$ 5.83	40.61 $\pm$ 2.67	29.57 $\pm$ 2.11*	22.95 $\pm$ 1.72*	0.001
MCV (fL)	79.62 $\pm$ 4.92	74.11 $\pm$ 4.84	69.23 $\pm$ 5.53*	63.11 $\pm$ 4.84*	0.003
MCH (Pg)	29.54 $\pm$ 1.28	26.68 $\pm$ 1.17	18.42 $\pm$ 1.07*	15.68 $\pm$ 1.17*	0.002

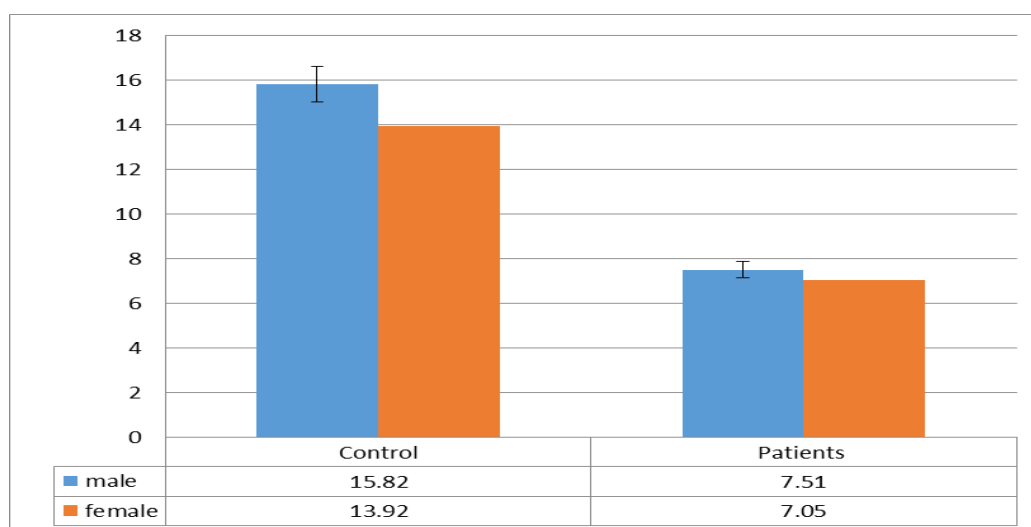


Figure 1 Participants' hemoglobin levels

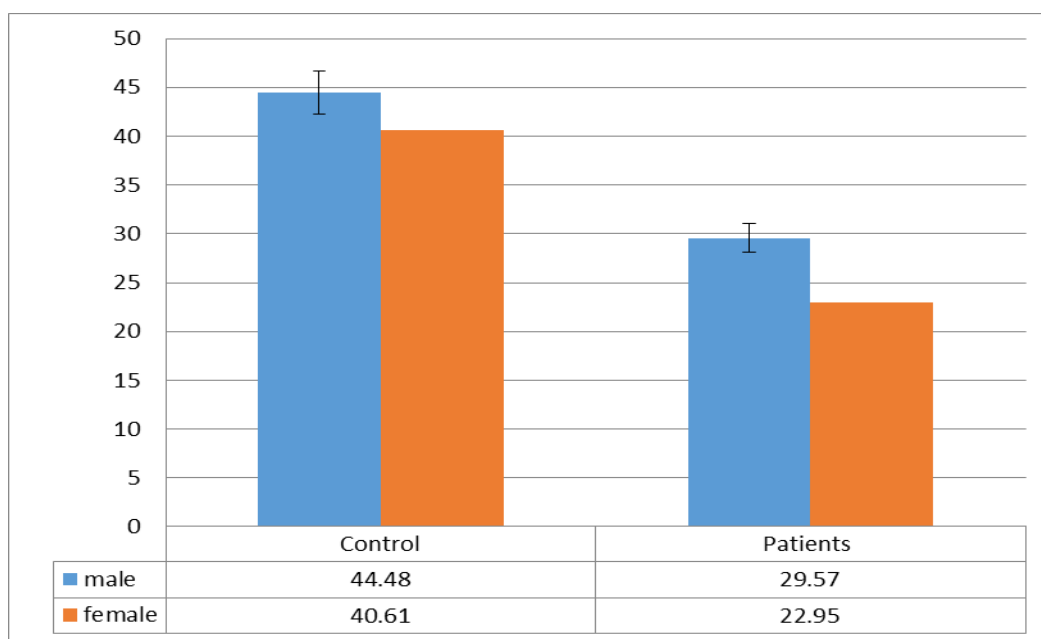


Figure 2 Average percentage of PCV in populations under investigation

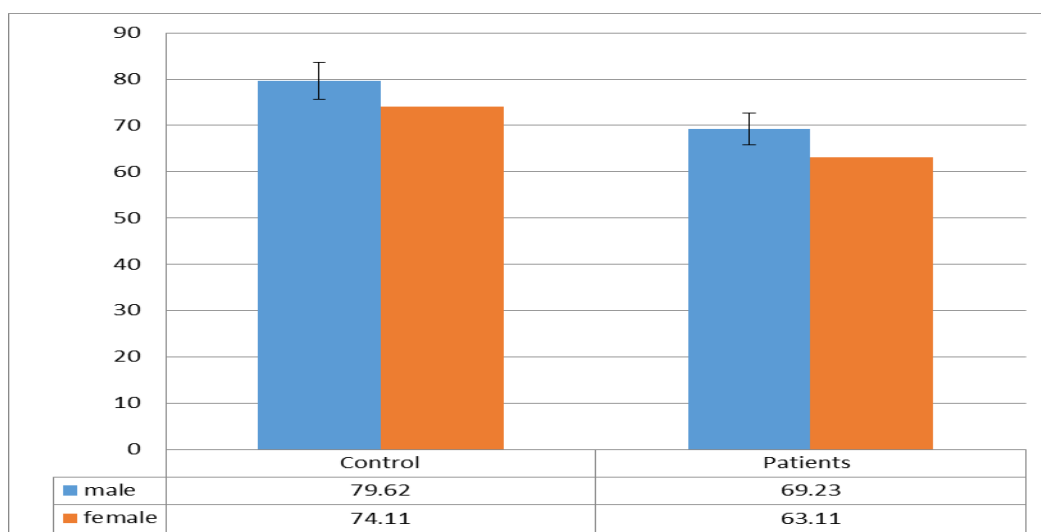


Figure 3 MCV % for each group

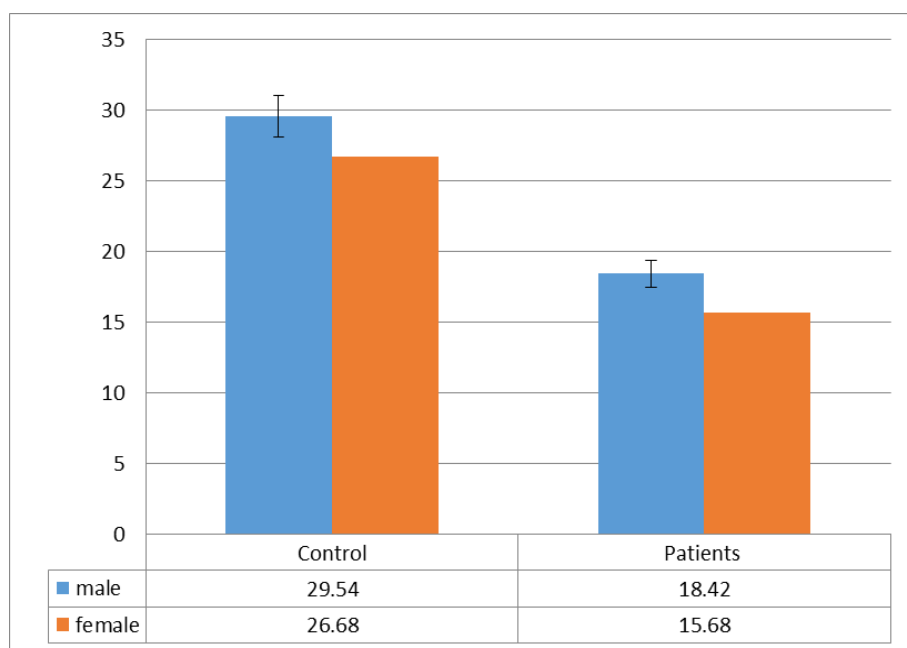


Figure 4 MCH prevalence in research groups

Iron and TIBC

Patients with beta thalassimia have considerably ($P > 0.05$) higher iron concentrations than the control

group, as shown in Figure 5 and the related data in Table 2. The TIBC was considerably ($P > 0.05$) greater in the sick group than in the control group, as seen in Figure 4.8.

Table 2 Levels of iron and TIBC in the groups being studied

Groups Parameter	Control (30)		Patients (110)		P-Value
	Male (15)	Female (15)	Male (55)	Female (55)	
S. iron (mcg/dL)	68.21±13.8	73.15± 9.41	118.5± 21.3*	109.41± 16.6*	0.039
TIBC (mcg/dL)	163.2 ± 24.4	151.7± 19.4	283.5 ± 41.9*	274.1± 35.6*	0.026

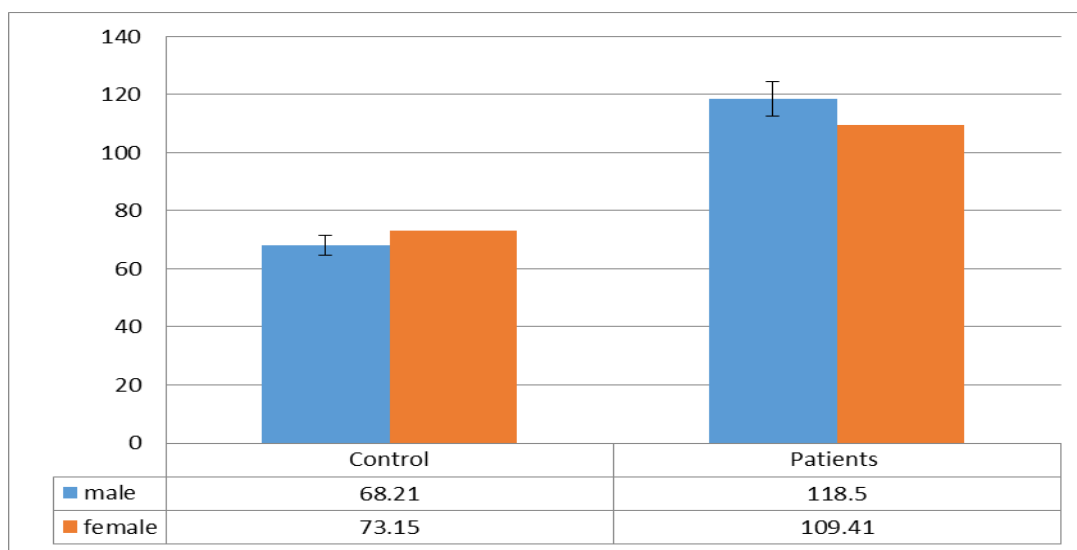


Figure 5 Group-to-group variation in S. iron concentration

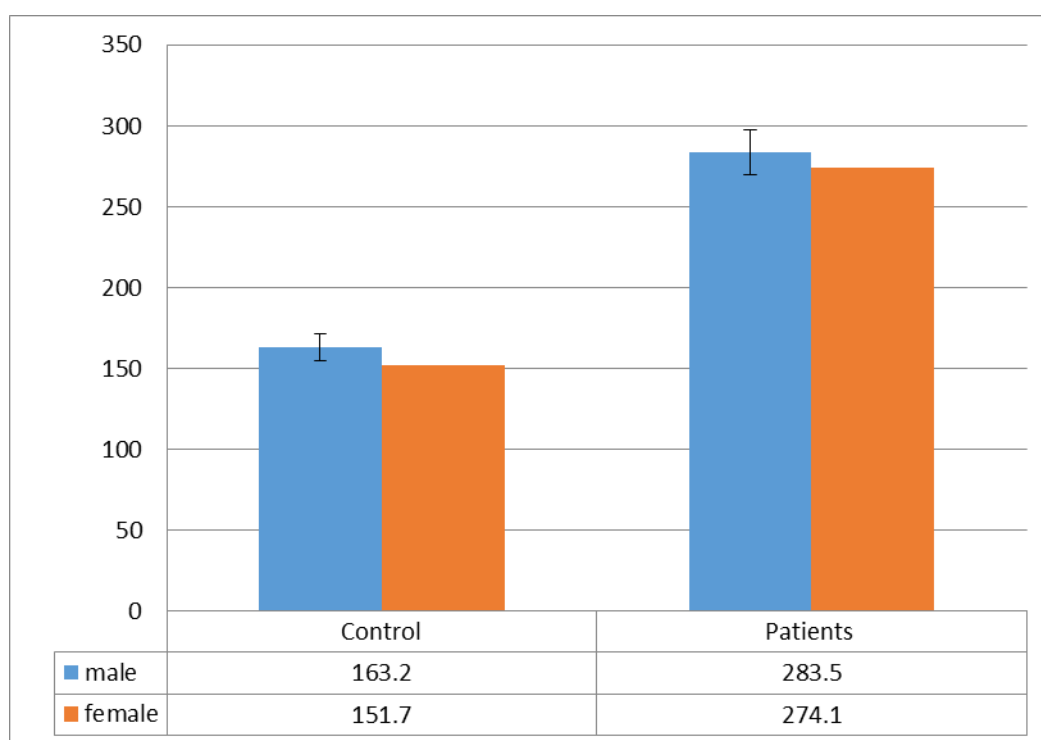


Figure 6 Total intracellular cadherin concentrations in study participants

Discussion

When compared to the control group, thalassemia patients' Hb concentration, HCT %, average cell volume, and quantity of red blood cells all significantly decreased ($p < 0.05$), as shown in Table 1. Because red blood cells have excess unbound globin protein in their cell membranes and are vulnerable to phagocytic cells in the bone marrow when their hemoglobin molecules undergo structural changes, this is caused by a decrease in beta globin chains in hemoglobin molecules. (12) claims that these cells have the ability to identify and damage aberrant cells, which in turn triggers erythropoiesis and increases the destruction of red blood cells.. These findings are in line with earlier research by (13) and (14), which shown that the spleen's macrophages function to eliminate aberrant and senescent cells, resulting in aberrant red blood cells having a limited lifespan and a propensity to self-degrade. The β globin gene is situated in a cluster with other β -like genes on the short arm of chromosome 11, is primarily influenced by the size and quantity of red blood cells, hemodilution, and hemoconcentration, which explains the low percentage of HCT. Over 200 mutations known to cause beta thalassaemias.4 Only a few deletions (15) have been found in the vicinity of the gene or in the gene itself to cause a β thalassemia, whereas Point mutations occurring in The genetic code

or its surrounding regions are responsible for the great majority of β thalassaemias.. The pathophysiologic features of the thalassaemia syndromes are created by the degree of globin chain imbalance. Globin chain imbalance is the greatest in β^0 thalassaemias, and the lowest in quiet β thalassaemias (16). The variability of the heterozygous state of β thalassaemias is enormous. In severe β^+ or normal β^0 thalassaemia thalassaemia, the alleles show reduced amounts of Hb, MCV, and MCH. However, in certain carriers, the severity of the β thalassaemias is such that anemia or haematological abnormalities are absent (17).

Iron overload can cause severe and irreversible biological damage, including cirrhosis, liver fibrosis, heart disease, and endocrine problems. Patients with β -thalassemic syndrome have significant morbidity and death due to the severe toxicity of excess iron on all bodily tissues. and other iron overload diseases. (18). Even while the majority of transfusion-dependent β TM patients die from cardiomyopathy, liver damage brought on by severe iron overload and/or persistent viral infections is becoming a more significant cause of death (19).However, new developments in chelation therapy methods, monotherapy, or combined therapy, along with suitable monitoring and ongoing health education campaigns, may improve thalassemia patients' quality of life and survival by preventing or

reducing these symptoms.[20] The majority of studies have concentrated on how iron overload negatively affects liver function as measured by liver the activity of enzymes. When a patient with transfusion-dependent β -thalassemia has a serum ferritin level greater than 1000 ng/ml and more than 30 transfusions, they begin to have liver enzyme disruption. [21,22]

Conclusions

In the Diyala Governorate population, hematological data can be utilized as a screening technique to predict β and thalassemia minor in the relatives of thalassemia patients.

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