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RESEARCH ARTICLE

# HEPATORENAL FUNCTION AND GLUCOSE BALANCE AMONG BETA-THALASSEMIA PATIENTS IN MUKALLA CITY, YEMEN

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## Abstract

Thalassemia is a hereditary blood disorder arising from defective synthesis of globin chains, leading to varying degrees of hemolytic anemia. This study aimed to assessing the certain alterations in kidney and liver functions, as well as glucose balance in beta- thalassemia patients. A cross-sectional study at the Sadan Foundation in Mukalla city, Hadhramout Governorate, during the period from December 2021 to March 2022. The study involving 55 participants (35 with beta-thalassemia patients and 20 healthy controls), aged 6 months to 23 years. Blood samples (5 ml) were drawn from volunteers, to measure liver and kidney functions as well as glucose level using the Cobas Integra 400 Plus analyzer, following the diagnostic kits and protocols provided by Roche. The obtained data were analyzed by the statistical program SPSS v24.0. The results indicated significant renal dysfunction, characterized by a significant increase in serum creatinine and uric acid levels, alongside a significant decrease in urea levels ($P < 0.05$). Regarding liver function, a significant elevation in liver enzymes [Alanine Aminotransferase (ALT), Aspartate Aminotransferase (AST), and Alkaline Phosphatase (ALP)] was observed. Furthermore, there was a non-significant increase in albumin levels and a non-significant decrease in total protein levels in patients compared to the control group. As well as, blood glucose level was significantly higher ($P < 0.05$) in patients group. Beta-thalassemia significantly impacts kidney and liver function, necessitating regular monitoring of these functions in patients.

**Keywords:** Beta-Thalassemia; Liver functions; kidney functions; Blood Glucose; Mukalla.

## Introduction

Thalassemia is defined as a hereditary disorder with worldwide prevalence, commonly referred to as Mediterranean anemia, since it was first diagnosed in the Mediterranean region. It is classified as a hemoglobin-related disorder caused by mutations affecting the synthesis of alpha- or beta-globin chains. These mutations lead to structural and functional imbalances in globin chain production, resulting in abnormalities in the morphology and function of red blood cells [1-5]. Thalassemia is typically diagnosed within the first six months of an infant's life. The condition often requires repeated blood transfusions every 3–4 weeks. The definitive treatment for thalassemia is hematopoietic stem cell transplantation (HSCT), which requires donor-recipient blood compatibility, a challenge that often poses significant difficulties. Therefore, the optimal approach to managing thalassemia lies in addressing the underlying causes of the disease [6,7].

Normal hemoglobin consists of four globin chains bound to heme: two alpha-globin chains and two beta-globin chains. As a result of alterations affecting these chains, thalassemia is classified into two main types:

### *1. Alpha-thalassemi*

This form occurs when defects affect the four genes located on chromosome 16. The severity varies according to the number of defective genes; the greater the number of affected genes, the higher the likelihood of developing severe and life-threatening conditions that may lead to fatal hemolytic complications[4,8,9].

### *2. Beta-thalassemia*

Is a hereditary disorder caused by point mutations in one or more loci of the β-globin gene located on chromosome 11, leading to a reduction or deficiency in the production of β-globin chains, which are essential components of hemoglobin structure. This defect results in a decreased amount of functional hemoglobin [10-12].

Beta-thalassemia is characterized by markedly reduced or completely absent production of β-globin protein chains. As a result, the number of red blood cells decreases, and the cells are smaller than normal and contain reduced amounts of hemoglobin, ultimately leading to chronic anemia.Beta-thalassemia is prevalent in various regions worldwide, with its presence reported in more than 60 countries, and with the highest prevalence in the Mediterranean region. According to the World Health Organization (WHO), approximately 150 million people are carriers of thalassemia.The number of newborns affected by thalassemia is estimated at approximately 70,000 infants per year worldwide, his is largely attributed to the high rate of consanguineous marriages, which ranges from 25.6% to 52.9% of all marriages. Thalassemia frequently appears in the Indian subcontinent, Southeast Asia, and West Africa.Based on the number of mutated genes and the severity of symptoms, beta-thalassemia is classified into three main groups, which are as follows: beta-thalassemia minor, beta-thalassemia intermedia, and beta-thalassemia major [11, 13-16].

Beta-thalassemia minor: It is characterized by the absence of clinical symptoms in affected individuals, as the mutation occurs in only one of the β-globin genes[17].

Beta-thalassemia intermedia: This type involves mutations in both β-globin genes, leading to a moderate reduction in the production of β-globin chains and hemoglobin. Individuals with this form typically do not require regular blood transfusions[19, 20].

Beta-thalassemia major: This is the most severe and clinically significant form of beta-thalassemia. It is characterized by the presence of ineffective red blood cells and severe anemia It is known as hemolytic anemia, It is associated with serious complications in affected patients, such as iron accumulation in the body, because the treatment usually requires long-term blood transfusions on a periodic basis, typically starting between 6 and 24 months of age.".[17,19,20]. Since thalassemia causes chronic anemia, the body responds by increasing the production of erythropoietin, a hormone that stimulates the formation of red blood cells to compensate for the deficiency. This continuous stimulation can lead to complications such as bone marrow expansion and the formation of extramedullary hematopoietic cells. Additionally, excessive iron accumulation may result in growth retardation and progressive deterioration of liver and kidney functions. Clinical manifestations of thalassemia in affected patients may include fatigue, severe weakness, shortness of breath, loss of appetite, hepatomegaly, splenomegaly, heart failure, and bone deformities[21, 22].

Patients with beta-thalassemia require regular treatment with iron chelators due to frequent blood transfusions, which lead to elevated iron levels and its accumulation in the body. This iron overload results in serious multi-organ dysfunctions, including cardiopulmonary disorders, endocrine insufficiencies, liver impairment, and renal failure. Renal failure is considered the fourth most common complication associated with beta-thalassemia major, primarily resulting from iron overload and chronic anemia[23].

This study was conducted to identify and evaluate certain alterations in kidney function, liver enzymes, and selected biochemical parameters that may occur in patients with beta-thalassemia, and to compare these findings with a healthy control group.

## Materials and Methods:

### *Study Design, Period and Setting:*

A cross-sectional study was conducted to assessment of liver and kidney functions, along with glucose balance among patients with beta-thalassemia. The study was carried out at the Sadan Foundation for Thalassemia and Genetic Blood Disorders in Mukalla- Hadhramout, Yemen, over a four-month period from December 2021 to March 2022.

### *Study Populations*

The study populations comprised a total of 55 participants, divided into two groups:

* Patient Group: 35 individuals diagnosed with beta-thalassemia major, all of whom were receiving regular blood transfusion therapy at the Sadan Foundation.
* Control Group: 20 age- and gender-matched healthy individuals with no history of chronic diseases or blood disorders.

The age range for both groups was established between 6 months and 23 years.

### *Ethical consideration*

Ethical approval was obtained through official correspondence from the Faculty of Science at Hadhramaut University to the administration of Sadan Foundation for Thalassemia and Genetic Blood Disorders. Furthermore, informed consent was obtained from all participants prior to their inclusion in the study, ensuring full awareness of the research objectives and procedures.

### *Blood Collection and Laboratory Analysis*

Venous blood samples (5 mL) were collected from each patient with beta-thalassemia as well as from the healthy controls using 4 mL sterile syringes. The puncture site was disinfected with ethyl alcohol (70%) prior to blood collection. The blood samples were then placed in clean,

dry, and anticoagulant-free tubes and left to stand for 10 minutes at 37°C to allow clotting.

Subsequently, the serum was separated by centrifugation at a speed of 3000–4000 rpm for 5 minutes; to measure the following parameters:

* Renal Function Parameters: Creatinine, Urea, and Uric acid.
* Liver Function Parameters: AST, ALT, Alkaline Phosphatase ALP, Albumin, and Total Protein.
* Metabolic Marker: Fasting Blood Glucose levels.

All parameters were measured using the Cobas Integra 400 Plus analyzer, following the diagnostic kits and protocols provided by Roche.

### *Statistical Analysis*

Data were processed and analyzed using the Statistical Package for the Social Sciences (SPSS v 24.0). Descriptive statistics were used to summarize the data, and independent t-tests were applied to compare the mean values between the patient and control groups. A p-value of less than 0.05 ($P < 0.05$) was considered statistically significant.

## Results

### *Biochemical Variables*

### *Blood Glucose*

Table (1) shows a significant increase in the average blood glucose levels in beta-thalassemia patients compared to the control group. However, this increase remained within the normal range and did not have any significant effect on the beta-thalassemia patients.

**Table (1):** Blood Glucose Levels in the Study Groups

| Parameters | Mean ± Standard Deviation | | P-value |
|---|---|---|---|
| | Patient Group(n=35) | Control Group(n=20) | |
| **Glucose (mg/dL)** | 99.0 ± 14.10 | 82.30 ± 16.92 | 0.000 |

### *Kidney Function Results*

Table (2) a significant decrease in the mean values of urea in the beta-thalassemia group compared to the healthy control group. Additionally, an increase in the mean values of uric acid was observed in the beta-thalassemia group compared to the healthy control group, although no significant differences were found. These values were within the normal range for the tests. Furthermore, a significant increase in the mean values of creatinine was found in the beta-thalassemia group compared to the healthy control group, with values also within the normal range for the tests.

**Table (2):** Kidney Function Levels in the Study Groups

| Parameters | Mean ± Standard Deviation | | P-value |
|---|---|---|---|
| | Patient Group(n=35) | Control Group (n=20) | |
| **Urea (mg/dl)** | 16.89 ± 4.61 | 23.15 ± 8.00 | 0.016 |
| **Creatinine (mg/dl)** | 0.35 ± 0.12 | 0.23 ± 0.12 | 0.011 |
| **Uric Acid (mg/dl)** | 4.54 ± 1.89 | 3.78 ± 0.79 | 0.252 |

### *Liver Enzyme*

Table(2) The results of the statistical analysis of the study indicated a significant increase beyond the normal reference range for the liver enzyme levels (Aspartate Aminotransferase (AST), Alanine Aminotransferase (ALT), and Alkaline Phosphatase (ALP)) in the beta-thalassemia group compared to the healthy control group. Additionally, no significant changes were observed in the total protein and albumin levels when compared to the control group. These values were also within the normal reference range for the tests in the beta-thalassemia group.

**Table (3):** Liver Function Tests (Total Protein - Albumin - ALP - ALT - AST) in the Study Groups

| Parameters | P-value | Mean ± Standard Deviation | |
|---|---|---|---|
| | | Patient Group(n=35) | Control Group(n=20) |
| **AST (Aspartate Aminotransferase) IU/L** | 0.041 | 57.58 ± 17.05 | 24.95 ± 8.78 |
| **ALT (Alanine Aminotransferase) IU/L** | 0.000 | 47.84 ± 14.89 | 14.33 ± 5.88 |
| **ALP (Alkaline Phosphatase) IU/L** | 0.000 | 198.35 ± 38.08 | 124.23 ± 12.25 |
| **Total Protein g/dL** | 0.123 | 7.09 ± 1.39 | 6.38 ± 1.81 |
| **Albumin g/dL** | 0.630 | 3.78 ± 1.19 | 3.81 ± 1.26 |

## Discussion:

Thalassemia is defined as an inherited disorder characterized by a defective synthesis of globin chains, which in turn leads to abnormal hemoglobin production. It is characterized by a range of metabolic disturbances, iron overload, chronic hypoxia, and cellular damage, which collectively lead to the production of abnormal red blood cells. It is a disorder that leads to hemolytic anemia of varying severity, which can range from mild to life-threatening [24, 21]. Thalassemia is considered one of the most severe inherited disorders worldwide, as patients with severe forms often require lifelong regular blood transfusions. They are also susceptible to numerous serious complications related to iron overload, as well as hepatic and cardiac diseases, which are the leading causes of mortality in most cases [25].

### *Biochemical Variables Studied*

#### *Glucose*

The results of the present study demonstrated a statistically significant elevation in blood glucose levels; however, these values remained within the normal reference range in patients with β-thalassemia when compared with the healthy control group. This finding is consistent with the results reported by [26] in a study conducted in Tunisia. Although blood glucose levels in the present study remained within the normal physiological range, the significant increase observed compared with the control group suggests a potential future risk of pancreatic dysfunction. Therefore, regular monitoring of metabolic status is warranted, as emphasized in previous studies evaluating the overall physiological condition of patients with thalassemia[27]. The observed increase in blood glucose levels among patients with β-thalassemia may be attributed to the impaired responsiveness of certain muscle and adipose tissue cells to insulin, whose primary physiological role is to facilitate glucose uptake. Alternatively, it may result from the failure to achieve the normal suppression of hepatic glucose production in individuals with insulin resistance. Consequently, insulin resistance in muscle and adipose tissues leads to reduced glucose uptake, as also reported by [28] in their study conducted in Diyala Province, Iraq.

A study by [20] indicated that patients with thalassemia may develop atypical diabetes or overt diabetes after a prolonged period of β-cell desensitization and dysfunction. It was also found that non-diabetic patients with impaired glucose tolerance are more likely to experience hypoglycemia than hyperglycemia, since routine screening often detects cases at an early stage. Furthermore, a hematopoietically active spleen may influence immune-mediated death of pancreatic islet (Langerhans) cells, making splenomegaly a potential risk factor for the development of diabetes.

#### *Kidney Function*

The results of the statistical analysis in the present study revealed a significant increase in serum creatinine levels and a non-significant change in serum uric acid levels in patients with β-thalassemia compared with healthy controls. These findings are consistent with the study conducted [28] in Al-Hoor City, Pakistan. Similarly, a study conducted in Yemen [29], as well as study[31], reported elevated serum creatinine levels in patients compared with healthy individuals, suggesting the presence of early-stage renal impairment or subclinical nephropathy resulting from iron deposition in the renal tubules. Moreover, the elevated uric acid levels reported in study[31] support the hypothesis that increased red blood cell turnover and heightened bone marrow activity in these patients lead to enhanced production of purine metabolism byproducts, a mechanism that has been confirmed and supported by recent studies in this field..

Furthermore, the results of the current study demonstrated a significant decrease in serum urea levels in patients with β-thalassemia compared with the control group. These findings are in agreement with the study by [32] conducted in Iran, as well as the study by [33] carried out in Koya City, Iraq. In addition, [34] reported decreased urea levels and increased creatinine levels in patients with β-thalassemia compared with healthy subjects in a study conducted in Najaf, Iraq. kidney complications are among the most significant comorbidities associated with thalassemia, occurring primarily as a result of excessive iron accumulation in vital body tissues. Consequently, patients require regular treatment with iron chelation therapy to reduce iron overload, which may expose the kidneys to potential nephrotoxicity and lead to drug-induced acute kidney injury (AKI). Such effects can result in renal tubular dysfunction in patients with β-thalassemia major [3, 35]. Recent evidence indicates that renal tubular injury can be detected at early stages in children with thalassemia through the use of specific biomarkers[27]. Renal impairment in patients with β-thalassemia is often associated with increased frequency of blood transfusions and hypercalciuria [36]. Furthermore, reduced kidney function may manifest as decreased serum creatinine and reduced body mass, which can be attributed to impaired muscle growth and lower muscle mass [37, 38]. Some studies have indicated that the severity of kidney damage correlates with the degree of anemia, regardless of creatinine levels [39]. Iron is thought to be the primary factor responsible for the physiological damage observed in the kidneys of patients with thalassemia. The elevated serum creatinine levels in patients with β-thalassemia may be attributed to increased immune complex deposition in the kidneys [40]. Uric acid levels are also elevated in β-thalassemia patients due to heightened bone marrow activity and DNA turnover. The increase in uric acid may serve as a protective mechanism against iron-induced oxidative damage, as uric acid is a major antioxidant in plasma [41]. The reduced serum urea levels observed in patients with β-thalassemia can be explained by impaired kidney function resulting from the accumulation of toxins and metabolic wastes in the kidneys, which consequently diminishes renal filtration and fluid excretion [24].

#### *Liver Function*

results of the present study demonstrated a significant increase in liver enzyme levels (AST, ALT, ALP) This is consistent with the findings of [21], as well as with the study conducted by Mekkey et al., who indicated that elevated hepatic enzyme levels are a prominent feature in patients with beta-thalassemia. This elevation is primarily attributed to iron overload resulting from repeated blood transfusions, which in turn leads to

increased oxidative stress and subsequent hepatocellular damage[27], as well as a non-significant increase in serum albumin levels in patients with β-thalassemia compared with healthy controls. These findings are consistent with the studies conducted by [29] in Al-Hoor City, Pakistan, and [26] in Tunisia. ALT is considered more specific than AST, although both enzymes are present in various tissues, including the heart, liver, skeletal muscles, and kidneys, with the liver containing particularly high levels of ALT [42]. The elevation of AST and ALT levels in β-thalassemia patients is likely due to increased serum iron levels, which accumulate in multiple organs, including the liver, leading to lipid breakdown in some of their cells [43, 44]. This elevation also reflects abnormal liver function [45]. The increase in ALP levels is attributed to the fact that this enzyme originates from both liver and bone tissues, and patients with thalassemia often experience damage to these tissues, resulting in the leakage of ALP into the bloodstream, which consequently enhances its activity [46, 47], The elevation of ALP enzyme levels in patients with β-thalassemia is attributed to liver diseases; it is also considered another marker of tissue injury and an indicator of hepatic dysfunction and leakage of liver metabolites [45]. This confirms that abnormal liver function in β-thalassemia patients is associated with increased ferritin levels and frequent blood transfusions. The progression of iron-induced liver disease is further aggravated by viral infections, and conditions such as hepatic hemosiderosis, portal fibrosis, and liver cirrhosis may develop despite iron-chelation therapy [48]. Albumin is the principal protein responsible for binding fatty acids in plasma, typically accounting for more than 50% of total plasma protein. It possesses approximately seven fatty acid–binding sites with moderate to high affinity. Albumin is synthesized by the liver, and its physiological functions include the regulation of oncotic pressure and the transport of fatty acids, bilirubin, cholesterol, drugs, and metabolic waste products. In addition, albumin plays an important role in the antioxidant capacity of blood plasma against free radicals [49, 50]. The elevated albumin levels observed in patients with β-thalassemia may be attributed to dehydration, burns, and certain acute inflammatory conditions, or to the presence of cardiovascular disorders such as myocardial infarction [51].

The results of the present study demonstrated a non-significant decrease in total protein levels in patients with β-thalassemia compared with healthy controls. These findings are consistent with the studies conducted by [33] and [50].in Iraq. Total protein is known to be one of the most abundant components in blood serum, encompassing enzymes, hormones, antibodies, in addition to proteins involved in the regulation of osmotic pressure balance, The decrease in total serum protein levels may be secondarily attributed to reduced protein synthesis by the liver [49, 50]. Recent studies have shown that different iron chelation therapy regimens may significantly affect liver enzyme levels in patients with thalassemia[26].

## Study Limitation:

This study is primarily limited by its small sample size and specific geographical focus in Mukalla, which may affect the generalizability of the findings. Its cross-sectional design provides a snapshot of the patients health but does not track the long-term progression of orgen dysfunction over time. Additionally, the history of splenectomy or serum ferritin levels, both of which are critical factors that could further explain the variations in liver and kidney function among beta- thalassemia patients.

## Conclusions and Recommendation

Based on the findings of this study, it can be concluded that:

* Thalassemia is a hereditary disease transmitted through affected parents or carriers, and its severity increases with a higher degree of consanguinity.
* Regular and repeated blood transfusions lead to iron overload in several vital organs, such as the heart, liver, kidneys, and endocrine glands, resulting in organ damage, functional impairment, organ failure, and eventually death.
* Beta-thalassemia affects renal and hepatic functions, leading to elevations or reductions in the associated biochemical markers in blood serum.
* Beta-thalassemia contributes to elevated blood glucose levels.

This study recommends the following:

premarital screening is recommended, along with regular follow-up with a specialist physician and adherence to medical instructions, including routine laboratory investigations such as assessments of renal function, liver enzymes, and blood glucose levels, to minimize complications and maintain health stability. Regular blood transfusions and strict compliance with iron chelation therapy are emphasized to prevent iron accumulation in vital organs. The study also recommends conducting further research to evaluate physiological and biochemical parameters in patients with beta-thalassemia before and after splenectomy, and urges the Ministry of Health to establish more advanced healthcare centers for the management of thalassemia and hereditary blood disorders.

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مقالة بحثية

# وظائف الكبد والكلى ومستوى الجلوكوز لدى مرضى بيتا ثلاسيميا في مدينة المكلا، اليمن

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## المُلخّص

يعد الثلاسيميا اضطراب دموي وراثي ينشأ عن خلل في تصنيع سلاسل الجلوبين، مما يؤدي إلى درجات متفاوتة من فقر الدم الانحلالي. هدفت الدراسة إلى تقييم بعض التغيرات في وظائف الكلى والكبد، بالإضافة إلى توازن الجلوكوز لدى مرضى بيتا ثلاسيميا. أجريت دراسة مقطعية في مؤسسة سدن بمدينة المكلا، محافظة حضرموت، خلال الفترة من ديسمبر 2021 إلى مارس 2022. شملت الدراسة 55 مشاركًا (35 مريضًا بالبيتا ثلاسيميا و20 شخصًا سليمًا كمجموعة ضابطة)، تراوحت أعمارهم بين 6 أشهر و23 عامًا. تم سحب عينات دم (5 مل) من المشاركين لقياس وظائف الكبد والكلى ومستوى الجلوكوز باستخدام جهاز تحليل Cobas Integra 400 Plus، وفقًا لمجموعات التشخيص والبروتوكولات المقدمة من شركة روش. تم تحليل البيانات باستخدام برنامج SPSS الإحصائي الإصدار 24.0. أشارت النتائج إلى وجود خلل كلوي، تميز بارتفاع معنوي في مستويات الكرياتينين وحمض اليوريك في الدم، إلى جانب انخفاض معنوي في مستويات اليوريا. ($P < 0.05$) أما بالنسبة لوظائف الكبد، فقد لوحظ ارتفاع معنوي في إنزيمات الكبد [ألانين أمينوترانسفيراز (ALT)، وأسبارتات أمينوترانسفيراز (AST)، والفوسفاتاز القلوي (ALP)] علاوة على ذلك، لوحظ ارتفاع غير معنوي في مستويات الألبومين وانخفاض غير معنوي في مستويات البروتين الكلي لدى المرضى مقارنةً بالمجموعة الضابطة. كما كان مستوى سكر الدم أعلى بشكل معنوي ($P < 0.05$) في مجموعة المرضى. يؤثر مرض بيتا ثلاسيميا بشكل كبير على وظائف الكلى والكبد، مما يستدعي عمل فحوصات دورية للمرضى لتقييم تلك الوظائف.

**الكلمات المفتاحية:** بيتا ثلاسيميا؛ وظائف الكبد؛ وظائف الكلى؛ سكر الدم؛ المكلا.

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