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Evaluation of Hepatic, Renal and Hemolytic Markers (AST, ALP, Urea and LDH) in Patients with Beta-Thalassemia

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ABSTRACT

Background: *As a hemolytic disorder, beta-thalassemia major is inherited and undergoes biochemical changes.* ***The Aim:*** *The current study aimed to assess the measure of liver function enzymes, renal function parameters, and selected hematological parameters in beta-thalassemia major patients as compared to healthy controls.* ***Methods:*** *The study consisted of 100 beta-thalassemia major patients and 100 control subjects matched for age and sex. Thalassemia patients show significantly higher AST and ALP levels than controls, suggesting hepatic involvement (as per the increase in liver enzymes). Further, the LDH level was very high due to ongoing hemolysis, a feature of beta-thalassemia.* ***Results:*** *Serum urea levels in patients were lower than healthy controls for renal function. Nonetheless, all values were normal physiologic, indicating that renal function was preserved. A beta-thalassemia patient's hematological evaluation shows a marked reduction in hemoglobin (Hb) and MCV consistent with severe microcytic anemia. A significant difference in liver enzyme activity and hemolysis marker, along with renal biochemical parameters and hematological indices, was observed in beta thalassemia cases.* ***Conclusions:*** *The result indicates that continuous laboratory assessment will improve clinical management and early complication detection in patients with beta-thalassemia major.*

Keywords: **Beta-thalassemia; Liver enzymes; Kidney function; LDH; Hemoglobin; MCV.**

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1. INTRODUCTION

Beta-thalassemia major (β-TM), which leads to chronic hemolytic anemia and regular red-cell transfusions, is a hereditary condition which arises from defective synthesis of β-globin chains (Basu et al, 2023). Although the survival rates have greatly improved due to better transfusion programs and iron-chelating therapy, progressive iron overload continues to be the Achilles' heel of disease management and greatly affects multi-system morbidity and mortality of patients (Pinto & Forni, 2020; Yadav & Singh, 2022).

Accumulation of excess iron in β-thalassemia major (β-TM) occurs in vital organs like liver, heart, endocrine glands, and kidney with manifestations such as oxidative stress, fibrotic changes, and impaired functional activity (Narahari et al., 2024; Basu et al., 2023). The liver, which is the main store of iron and an organ with detoxification, is especially susceptible. Research has observed that individuals with thalassemia have higher

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levels of aspartate aminotransferase (AST) and alkaline phosphatase (ALP) in serum. It is suggested that these elevated levels may indicate early hepatocellular injury, cholestatic stress, and toxicity due to iron being introduced (Almuttrek et al., 2023).

Shared acknowledgment of β-TM related renal involvement has increased. Raised blood urea and creatinine levels, which indicate glomerular and tubular dysfunction, were correlated with serum ferritin and hepatic iron concentration. Consequently, renal damage can be caused by iron-mediated nephrotoxicity and chronic anemic hypoxia (Romadhon et al., 2022; Nashwan & Yassin, 2025). Meanwhile, LDH is an established surrogate marker of haemolysis, indicating ongoing red-cell destruction. As a result, organs such as the kidneys and liver face more strain. (Karim et al., 2016).

The impairment of erythropoiesis and chronic anaemia mainly seen in β-TM, reduces the levels of Hb and thereby tissue oxygenation. It also predisposes to organ dysfunction. As a result, Hb measurement, combined with biochemical markers, can help one better understand disease severity and systemic burden (Oikonomidou & Rivella, 2018).

Considering the interrelationship between iron overload, haemolysis, organ damage and biochemical dysfunction, a study was undertaken at the Thalassaemia Centre of Al-Zahraa Hospital, Al-Najaf, Iraq, to assess hepatic, renal and haemolytic parameters – AST, ALP, Urea, LDH, MCV and Hb in 100 cases of β-thalassemia major in comparison to 100 healthy controls. The aim of this study is to clarify organ-system involvement and to see if iron load is associated with biochemical dysfunction to assist in earlier recognition of organ impairment and to improve management.

2. METHODS AND MATERILAS

Study Design and Participants

This study was conducted as a comparative cross-sectional study at Al-Zahraa Hospital/ Thalassemia Center, Al-Najaf, Iraq, with the Clinical Chemistry Laboratory of the same hospital. The study period was from September 1, 2025, and November 1, 2025, about 200 people were enrolled in this study and were distributed into two groups:

• Group I (patients): it includes 100 diagnosed cases of beta-thalassemia major who were regularly attending for follow-up at the Thalassemia Center.

• Group II, constituting controls, will include 100 individuals having no history of hematological or chronic systemic disease, apparently healthy, matched for age and gender.

Sample Collection and Preparation

Five milliliters (5 mL) of venous blood was collected from each subject using sterile disposable syringe. The samples were collected in plain tubes and allowed to clot at room temperature for centrifugation at 3000 rpm for 10 minutes to obtain serum. The serum samples were analyzed to check the liver, renal and hemolytic marker biochemically. Besides, a small amount of blood was collected in EDTA tubes for hemoglobin (Hb) estimation and MCV.

Biochemical Analysis

Studies on biochemical parameters:

• Liver function markers; Aspartate aminotransferase (AST), Alkaline phosphatase (ALP).

• Urea - Renal function marker.

•Hemolytic markers: Lactate Dehydrogenase (LDH), MCV and Hemoglobin (Hb).

ollowing the manufacturer instructions, liver and renal function tests were performed using a UV–Visible Spectrophotometer (Spectrophotometric Method). The automated hematology analyzer helped to measure hemoglobin.

Reagents and test kits used were of analytical grade and supplied by standard commercial diagnostic companies.

Statistical Analysis

All data obtained were tabulated and analyzed using the Statistical Package for the Social Sciences (SPSS) version 26. Results were expressed as mean ± standard deviation (SD). The Independent Student's t-test was applied to compare biochemical parameters between thalassemia patients and healthy controls. A p-value less than 0.05 was considered statistically significant.

3. RESULTS

This study involved 200 subjects in total. The statistical analysis was carried out by applying Independent Samples t-test to assess the biochemical and hematological parameters of both patients and healthy controls. As shown in Table 1, all data measured were displayed in the table. The total sample size included 100 patients with β-thalassemia and 100 healthy patients as the control group.

The significant differences between patients and controls for all parameters ($p \leq 0.01$) were highly significant. The AST, ALP and LDH levels were statistically significant ($p < 0.001$) higher in β-thalassemic patients than in control subjects. According to the serum urea levels, patients exhibited a vast metabolic and biochemical profile alterations.

There were also marked abnormalities in the hematological indices. Compared to healthy individuals, the hemoglobin and MCV values of the β-thalassemia patients were significantly lower, which is suggestive of the disease's characteristic of microcytic hypochromic anemia. The biochemical as well as hematological parameters assessed in the current study differed significantly between β-thalassemia patients and healthy subjects as shown in Table 1.

Table 1: Comparison of Haematological and biochemical parameters between Beta-Thalassemia Patients and Healthy Controls

Parameters	Beta-thalassemia	Control	p. value
	Mean±SD	Mean ±SD	N
Age (years)	20.486±11.389	21.184±11.574	NS
Hb(g/dl)	8.328±1.114	13.846±1.082	0.00>
MCV(μm^3)	69.400±7.850	88.620±4.930	0.00>
AST(IU/L)	46.314± 17.900	27.846± 9.732	0.00>
ALP(IU/L)	204.806 ±42.800	96.427±22.583	0.00>
Urea(mg/dL)	26.908±7.570	31.284±5.913	0.05>
LDH(IU/L)	269.718±78.600	184.392±41.763	0.00>

All values were considered statistically significant when $p \leq 0.05$. AST:Aspartate aminotransferase; ALT: Alanine aminotransferase; ALP: Alkaline phosphatase; LDH: Lactate dehydrogenase; Hb: Hemoglobin; MCV: Mean corpuscular volume; Urea: Blood urea

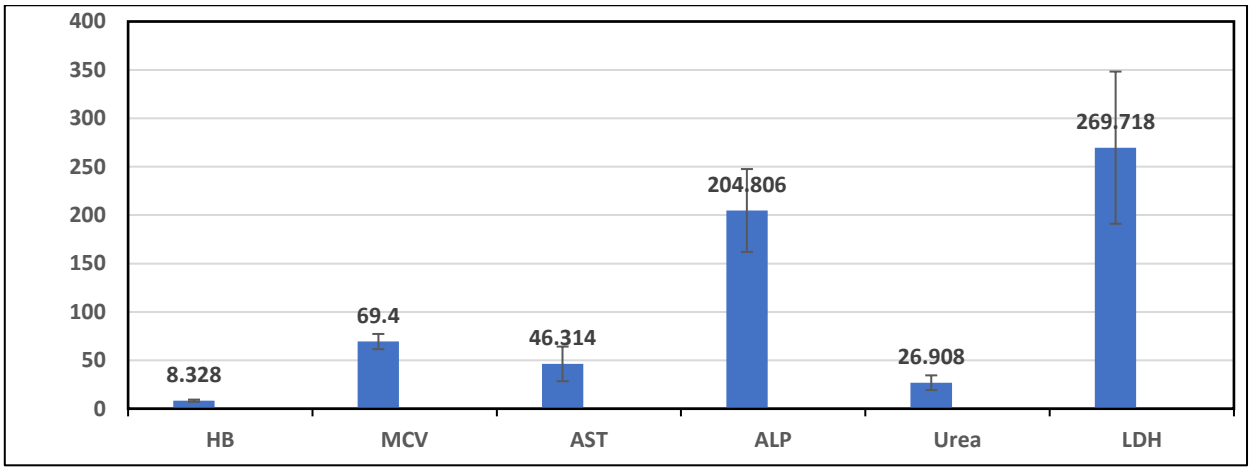

Figure 1. HB, MCV, AST, ALP, Urea and LDH, mean values of Beta-Thalassemia patients.

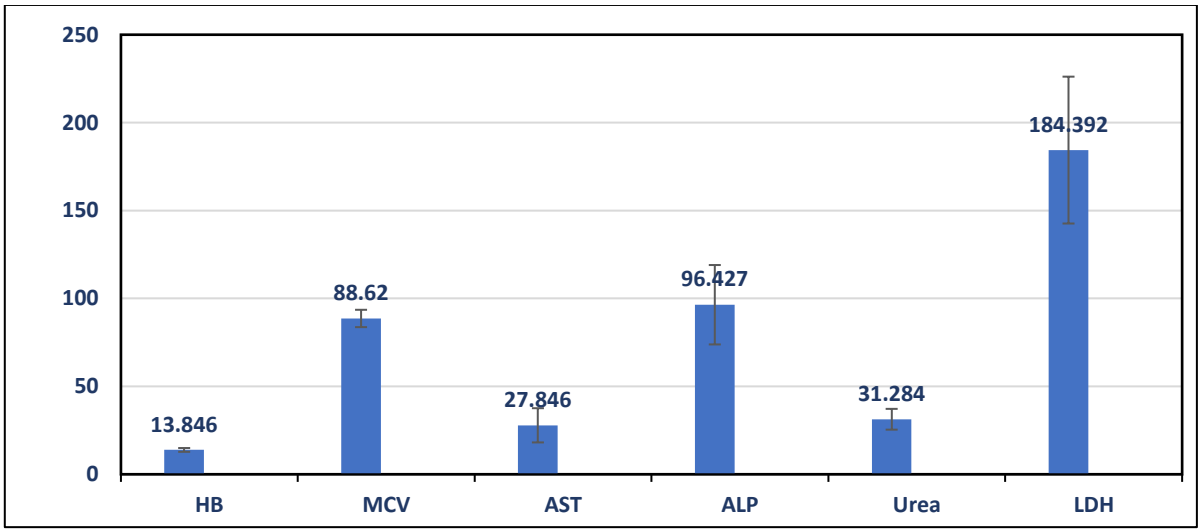

Figure 2. HB, MCV, AST, ALP, Urea and LDH mean values of control.

4. DISCUSSION

Beta-thalassemia is a genetic blood disorder that reduces the synthesis of the beta chain of hemoglobin and causes anemia. It also causes pigmented but not iron overload (Dordevic et al., 2025, Prahasanti et al., 2026). The liver, kidneys, and bone marrow can all be affected by these disruptions (Dallal Bashi&fathi 2022; Sadeghi et al., 2021; Demosthenous et al., 2018).

The study evaluated liver and kidney biochemical markers (AST, ALP, LDH, Urea), hemoglobin and MCV on 100 patients of β-thalassemia with 100 healthy individuals. Results showed a significant change in most of the parameters indicating the pathophysiological effect of chronic hemolysis, ineffective erythropoiesis and iron utilization. The levels of serum AST and ALP were notably higher in thalassemia patients in comparison to controls. The recent discoveries are similar to the older findings including hepatic stress and hepatocellular injury in β-thalassemia which happens due to the deposition of iron and oxidative stress as well as chronic hemolysis (Hamza et al., 2025; Hosen et al., 2015). Serum LDH levels were noted to be significantly higher in β-thalassemia patients than in healthy controls.

These reports are similar to some previous studies which stated that high levels of LDH are the biochemical features of Thalassemia (Toren et al., 1996; Gunawan et al., 2021). The commonly recognized marker for hemolysis is LDH, which elevation reflects the process, generally in thalassemic patients, in which abnormal red blood cells undergo accelerated destruction (Abdullah et al., 2024).

In patients with β-thalassemia, ineffective erythropoiesis and ongoing peripheral hemolysis release intracellular enzymes into the circulation, resulting in markedly elevated LDH levels. A lower count of red blood corpuscles and an increase in lactate dehydrogenase (LDH) indicate high turnover of erythrocytes and breakdown of fragile RBC in disease. Thus, the increased LDH concentration in our study may be due to the above reason. Additionally, the occurrence of oxidative stress and inflammatory responses due to chronic hemolysis may also lead to elevated levels of LDH, released from damaged erythrocytes. The findings indicate that LDH is a sensitive marker of the hemolytic activity in βthalassemia and may serve as a useful biochemical tool to assess the severity of the disease It is also possible to measure hemolytic processes to evaluate the situation of patients. In this study, we see a significantly raised level of LDH which is in accordance with the previously published data and indicates that there is a continuous burden of hemolysis in beta-thalassemia patients. Evaluating the levels of LDH can aid clinicians in both measuring the severity of hemolysis and tracking the overall progression of the disease.

The β-thalassemia patients had a significantly different urea level from healthy controls in terms of serum urea levels. Though the urea values in the β-thalassemia patients were physiologically normal. (24 words): This study's urea levels from the sera of thalassemic patients are lower in comparison to many studies, which reported higher levels (Shaalan et al., 2022). The variation in nutritional status, the loss of muscle mass, the worsening of the disease, or the preservation of renal function may be possible culprits. Therefore, the reduced serum urea levels may not indicate impairment of the kidneys. Oxygen deficiency is due to metabolic and physiological adaptations of β-thalassemia patients.

A hematological examination revealed a reduction in hemoglobin (Hb) and mean corpuscular volume (MCV), which are the characteristic features of microcytic hypochromic anemia of beta-thalassemia pacific. According to Lazarte et al (2015), thalassemia carriers exhibit lower levels of hemoglobin (Hb) and mean corpuscular volume (MCV). According by Goretti et al., (2022) lower MCV is a hallmark of the disease, the results seem to be in accord to this as well. A continuous decline in MCV indicates RBC production impairment, resulting in progressively smaller cell size.

CONCLUSIONS

The result indicates that continuous laboratory assessment will improve clinical management and early complication detection in patients with beta-thalassemia major.

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