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Review Article

Biochemical Blood Variations among Thalassemia Patients with Different ABO Blood Groups in Al-Diwaniyah Governorate

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Abstract: Thalassemia is a hematological disorder characterized by a partial or complete deficiency in the biosynthesis of globin polypeptide chains. Consequently, affected adults manifest with chronic anemia, systemic hemosiderosis due to iron overload, and dependency on periodic allogeneic blood transfusions. The experiment was conducted in Diwaniyah Governorate at the Women's and Children's Hospital / Thalassemia Center where data on thalassemia patients was collected between 15 September 2024 and 21 March 2025, including 100 people in total: 50 thalassemia-diagnosed patients and 50 healthy controls. Patients with thalassemia are categorized by gender and blood groups A, B, AB and O. The data collected from the patient and control groups were statistically analyzed using the SPSS statistical program (2013). The results indicated a statistically significant increase in serum urea levels ($P < 0.05$) and significant elevations in serum total bilirubin (TSB), glutamic oxaloacetic transaminase (GOT), and glutamic pyruvic transaminase (GPT) ($P < 0.01$) in thalassemia patients. In contrast, serum creatinine levels showed a significant high decrease ($P < 0.01$) in patients compared to the control group. Alkaline phosphatase (ALP) and creatinine levels were also significantly decreased in the control group ($P < 0.05$), while hemoglobin (Hb) levels were significantly higher ($P < 0.01$) compared to the thalassemia group.

Keywords: Thalassemia, Urea, Creatinine, TSB, Got, GPT, ALK.P, Ferritin, ABO, Hemoglobin.

1-INTRODUCTION

Thalassemia is an inherited hematologic disorder that disrupts the molecular architecture and functional capacity of hemoglobin—the principal oxygen-carrying metalloprotein within erythrocytes. When the synthesis of hemoglobin chains is disrupted, the lifespan of red blood cells is shortened, leading to various forms of anemia. (Shafique *et al*., 2021). Thalassemia is an inherited disorder that results when defective genes are transmitted from one or both parents to their children. These faulty genes affect the body's ability to produce normal hemoglobi (Begum *et al*., 2023). According to the World Health Organization, 68,000 individuals with β-thalassemia are born each year, and the prevalence of symptomatic β-thalassemia in the general population is estimated to be 1 in 100,000 (Jang *et al*., 2021). According to the Iraqi Ministry of Health in 2019, there are about 22,000 persons with thalassemia (Lafta *et al*., 2023).

Thalassemia is etiologically subclassified into alpha (α) and beta (β) variants, contingent upon the specific globin chain biosynthesis that is genetically impaired. Alpha-thalassemia arises from molecular aberrations within the genetic loci responsible for encoding alpha-globin polypeptides, which constitute an integral structural component of the hemoglobin tetramer in erythrocytes. The physiological synthesis of alpha-globin chains necessitates the coordinated expression of four functional alleles—two inherited maternally and two paternally. Any flaw or insufficiency in these genes causes the disease to manifest in different degrees

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(Tripathi, 2022). The most prevalent kind of thalassemia is beta thalassemia. It is caused by a genetic abnormality in the genes that make hemoglobin's beta chains, which impairs the production of red blood cells and causes them to break down quickly. This results in different levels of chronic anemia (Ali *et al*., 2021). The cornerstone of therapeutic intervention for thalassemia, particularly in severe phenotypic manifestations, is the administration of recurrent allogeneic blood transfusions. This modality serves to augment circulating hemoglobin concentrations and replenish the deficient erythrocyte population. Nonetheless, sustained transfusional support is frequently accompanied by the pathological accumulation of iron within somatic tissues, culminating in systemic iron overload. Excess iron builds up in organs like the heart, causing heart failure, or it may lead to cirrhosis, renal failure, hormonal diseases like diabetes, or poor development because the body is unable to eliminate it normally (Saeidnia *et al*., 2023).Ferritin and hemosiderin are the two forms of iron that are stored. Hydroxide of aldehyde or alferritin inside the albuferritin "bag" or sheath. Approximately 20% of the weight of each ferritin is made up of 2000 iron atoms. It is a protein that dissolves in water and is a molecule that stores iron or in groups. The Ferritin test is one of the basic tests for thalassemia patients, where patients, especially those who need frequent blood transfusions, suffer from iron accumulation in the body, which can lead to damage to vital organs such as the heart and liver. This test helps track the level of iron in the body and determine if you need medications to remove excess iron (chelated medications). (Smesam *et al*., 2020). The liver is the only organ in the body that produces the protein ferritin and serves as the main organ for storing iron. Since free iron is very poisonous, iron is often linked to proteins in the liver to lessen its toxicity. Excess unbound iron promotes the production of free radicals, which may harm the kidneys and liver (Rafati Rahimzadeh *et al*.,2023).

2-METHODS & TOOLS

2-1 The Design of the Study

This investigation was undertaken in Al-Diwaniyah Governorate, targeting individuals diagnosed with thalassemia who routinely seek therapeutic intervention at the Women and Children's Hospital, subsequent to definitive clinical confirmation of their condition. This study included 100 participants: 50 patients diagnosed with thalassemia and 50 healthy individuals.

2.2 Collection and Blood Sample

Venous blood specimens were procured at the Women and Children's Hospital in Diwaniyah Province. Following informed consent, 5 milliliters of intravenous blood were aseptically drawn from both thalassemia patients and control subjects within the healthy cohort. Blood sampling in the patient group was conducted prior to their scheduled transfusion sessions. Each specimen was approximately bisected into two equal aliquots of 2.5 ml and allocated into distinct collection tubes: one containing EDTA as an anticoagulant to preserve cellular components, and the other a clot-activator gel tube intended for serum extraction. The latter was allowed to stand at ambient temperature for 30 minutes to facilitate complete coagulation, followed by centrifugation at 4000 revolutions per minute for a duration of 5 minutes. Serum was subsequently separated under strict conditions to prevent hemolysis and was transferred into sterile Eppendorf microtubes. The obtained serum was employed in a battery of biochemical analyses, while the EDTA-preserved blood was utilized for comprehensive hematological profiling. The procedural workflow is schematically illustrated in Figure (1).

2-3 Hematologic Profiling

Hematological parameters were quantified utilizing an automated biochemical hematology analyzer (Sysmex XS-1000i), an advanced diagnostic apparatus engineered to compute comprehensive blood indices—including hemoglobin concentration (Hb) and ABO/Rh blood group typing—through high-precision volumetric and optical assessments.

2.4 Biochemical Analysis

Quantitative assessment of biochemical analytes—including alkaline phosphatase (ALP.k), total serum bilirubin (TSB), urea, creatinine, serum glutamic oxaloacetic transaminase (GOT), and serum glutamic pyruvic transaminase (GPT)—was performed via fully automated spectrophotometric analysis using the COBAS® c 111 clinical chemistry analyzer. Ferritin concentrations were concurrently determined through automated immunoassay techniques employing the VIDAS analytical platform.

2.5 Statistics Analysis

All empirical data obtained in the present investigation were subjected to statistical evaluation and presented as arithmetic means accompanied by their corresponding standard deviations (mean ± SD) for individuals diagnosed with β-thalassemia major. Inferential statistical analysis was employed to determine the presence of significant disparities between the patient cohort and the control group, utilizing the Statistical Package for the Social Sciences (SPSS), version 2013.

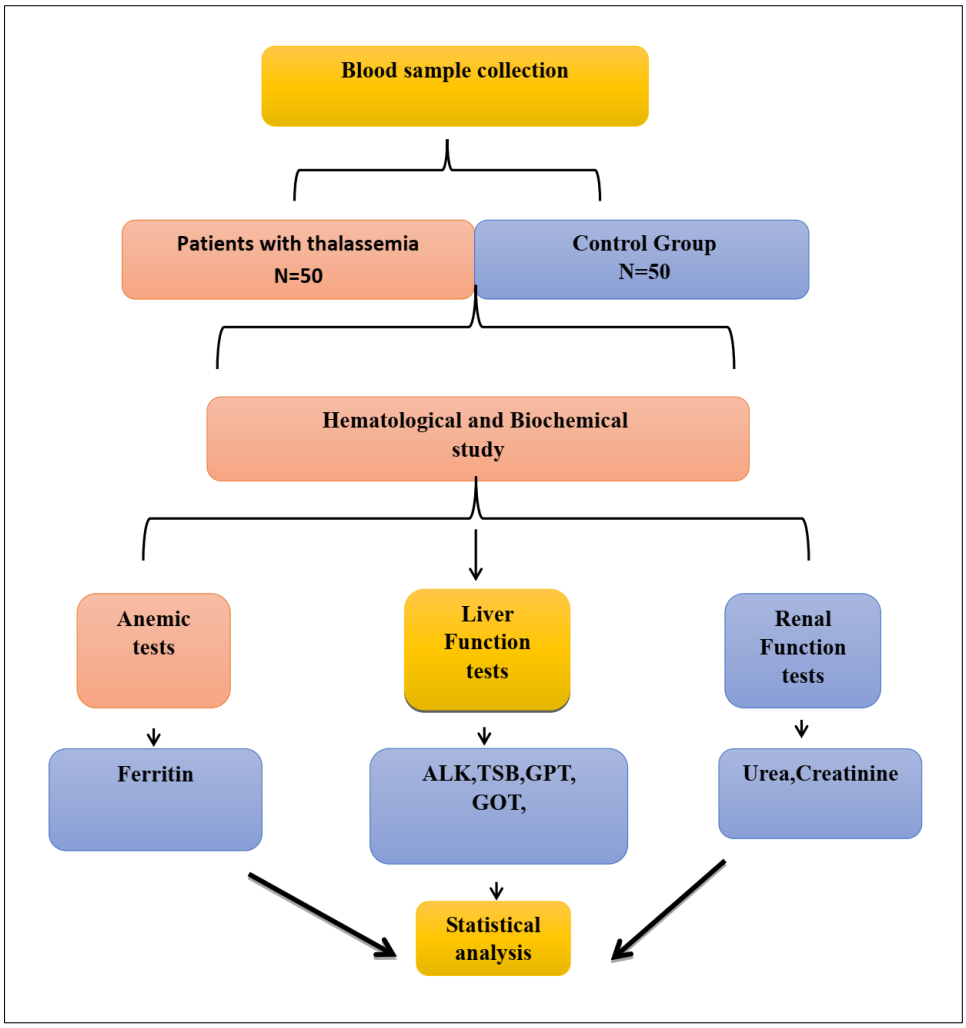

Figure 1: General design of the research

3-DISCUSSION & RESULTS

3-1Serum Urea, Creatinine, TSB, Got, GPT, ALK.P, Ferritin, and Hb Levels in Thalassemia Patients and Control Groups

Table (3-1) show Patients have significant effect ($p<0.05$) in urea trait, and high significant effect ($p<0.01$) in TSB, Got, GPT trait and ferritin, Urea levels increased significantly in patients. This result is in agreement with studies done by (Sen *et al.* ,2015), (Mahmoud., *et al.,* 2021) The higher blood urea level in thalassemia patients may reflect compromised renal function, which could be exacerbated by iron overload and warrants further investigation. (Alzubaidi *et al.,*2025). There were highly significant ($p<0.01$) decreases in creatinine levels. The finding of this study was concordant with the studies done by (Al-Ghanimi *et al.,* 2019) and contradicted with studies done by (Mohammed *et al.,* 2015) Empirical findings reported by Mohammed *et al*., (2015) demonstrated elevated serum creatinine levels in approximately 38% of individuals diagnosed with thalassemia. This observation was corroborated by Bekhit *et al*., (2017), who identified a similar increase in 40% of patients. The pathophysiological basis for renal impairment in thalassemia is hypothesized to stem primarily from systemic iron overload; however, ancillary contributors such as the nephrotoxic impact of iron chelation regimens and oxidative stress–induced cellular damage may also be implicated (Bhowad *et al*., 2022). Hemoglobin (Hb) concentrations were consistently lower among thalassemia patients in comparison to healthy controls, a trend congruent with the findings of De *et al*., (2019) and Ayyash & Sirdah (2018). This hypochromic microcytic anemia is attributed to impaired globin chain synthesis and heightened erythrocyte turnover, particularly via extravascular hemolysis in the spleen, culminating in ineffective erythropoiesis (Kumar, 2019). Total serum bilirubin (TSB) levels were markedly elevated ($P < 0.01$) in the thalassemia group relative to controls, in alignment with the outcomes reported by Gharehdaghi & Yasuj (2023). This elevation is presumably linked to excessive erythrocyte destruction and accelerated heme catabolism (Al-Ghanimi *et al*., 2019). Furthermore, substantial elevations in serum levels of hepatic transaminases—glutamic oxaloacetic transaminase (GOT) and glutamic pyruvic transaminase (GPT)—were documented in thalassemia patients compared to the control group, mirroring prior investigations (Jwaid & Gata, 2020). Hepatic dysfunction is recognized as a principal morbidity and

mortality factor in individuals with β-thalassemia, particularly in its major and intermediate phenotypes. The hepatopathy observed may manifest as hepatocellular toxicity, viral hepatitis (types B and C), or cirrhosis, predominantly driven by chronic iron deposition secondary to repeated transfusions, hemolytic episodes, and dysregulated gastrointestinal iron absorption exacerbated by persistent erythropoietin stimulation (Jwaid & Gata, 2020). Elevated serum ferritin concentrations were also observed, consistent with the findings of De *et al*., (2019) and Şen *et al*., (2015), reflecting the systemic iron burden characteristic of transfusion-dependent thalassemia. The excessive accumulation of serum iron in these patients is primarily attributable to transfusional siderosis. Moreover, gastrointestinal iron absorption in β-thalassemia patients is estimated to be three to four times higher than in individuals without the disorder, potentially resulting in an annual iron accumulation of 2–5 grams (Suman *et al*., 2016). Interestingly, serum alkaline phosphatase (ALP) levels were significantly decreased ($P < 0.05$), which stands in contrast to the findings of Al-Ghanimi *et al*., (2019), who reported significantly elevated ALP activity in β-thalassemia patients. This divergence may point to underlying abnormalities in hepatic or muscular physiology, suggesting complex organ-specific biochemical alterations (Nafady *et al*., 2017).

Table 3-1: Effect of treatment groups on different characters

Characters	Treatment	Mean	Std. Error	95% Confidence Interval	
				Level of	L.S.D
Urea	Patient	24.843 a	0.925	0.05	2.99
	Control	21.955 b	0.992		
Creat	1.00	0.358 b	0.019	0.01	0.064
	2.00	0.915 a	0.020		
TSB	1.00	2.172 a	0.133	0.01	0.455
	2.00	0.902 b	0.143		
GOT	1.00	35.866 a	2.633		9.289
	2.00	12.982 b	2.825	0.01	
GPT	1.00	132.827 a	6.659	0.01	22.841
	2.00	13.446 b	7.146		
ALK.P	1.00	45.548 a	5.211	0.05	13.447
	2.00	55.025 a	5.591		
FERRITIN	1.00	2793.133 a	236.671	0.01	810.94
	2.00	194.759 b	253.962		
Hb	1.00	7.912 b	0.134	0.01	0.528
	2.00	**14.301 a**	**0.144**		

3-2 The Influence of ABO and Rh Hemotypic Classifications on Hematologic Indices and Biochemical Biomarkers among Individuals with Thalassemia versus Normotypic Controls.

The empirical outcomes of the present investigation elucidated the distributional patterns of ABO and Rh blood group phenotypes among individuals afflicted with thalassemia, juxtaposed with normotypic control cohorts. Data presented in Tables (2–3) delineate the modulatory impact of hemotypic classifications on a spectrum of biochemical and hematological indices. Notably, Table (2–4)a reveals a statistically significant diminution ($P < 0.05$) in serum urea concentrations among individuals bearing blood group B relative to the control population, corroborating findings reported by Aziz *et al*., (2019). This biochemical aberration may be pathophysiologically attributable to compromised renal function, plausibly induced by nephrotoxic accumulation of metabolic byproducts and xenobiotic compounds, which impair glomerular filtration and tubular excretory capacity (Cappellini *et al*., 2014).

Furthermore, stratified analyses across all major blood groups (O, A, B, AB) indicated statistically significant decrements in serum creatinine concentrations ($p < 0.05$) within the thalassemic cohort, with respective mean values of 0.377 ± 0.029, 0.392 ± 0.032, 0.316 ± 0.034, and 0.347 ± 0.052, as illustrated in Table (2–4)b. Deviations in creatinine levels, whether hypo- or hypercreatininemia, are indicative of renal insufficiency. Hypercreatininemia may arise from acute nephritic syndromes or protein-rich diets, whereas hypocreatininemia can result from senescence, malnourishment, nephroblastoma, or pharmacological agents (Al-Shawi & Al-Hameedawi, 2022).

Moreover, the current data indicate statistically significant elevations ($P < 0.05$) in bilirubin, aspartate transaminase (GOT), and alanine transaminase (GPT) levels across all hemotypic categories in the patient group, relative to controls. These perturbations likely reflect hepatocellular compromise, transaminasemia, and hepatic metabolite efflux into systemic circulation (Jafari *et al*., 2015). Hyperbilirubinemia in transfusion-dependent thalassemia may be mechanistically associated with hemolytic activity and oxidative hepatic insult due to diminished cytochrome c oxidase activity, thereby impairing mitochondrial bioenergetics

(Sadeghian *et al*., 2009). Additionally, hepatic dysfunction may exacerbate systemic iron dysregulation and overload (Obeidi *et al*., 2011).

Data in Table (3–2)f demonstrate a statistically significant increase ($P < 0.05$) in serum alkaline phosphatase (ALP) activity among thalassemic patients relative to B blood group controls, suggestive of cholestatic liver injury or osteopathic pathophysiology. Furthermore, Table (3–2)g indicates hyperferritinemia across all blood groups within the thalassemic population ($P < 0.05$), underscoring a systemic iron burden. In β-thalassemia, the absence of β-globin synthesis culminates in the aggregation of unbound α-globin chains, precipitating cellular oxidative stress and cytotoxic iron deposition. Elevated ferritin concentrations serve as biomarkers of labile plasma iron accumulation, catalyzing a secondary cascade of oxidative erythrocytic membrane damage—referred to as the "second disease" (Rakib *et al*., 2023).

Clinical hematological profiling revealed that reductions in hemoglobin (Hb) concentrations were accompanied by proportional declines in erythrocyte count and red cell indices (MCV, MCH, HCT), as shown in Table (2–4)h. The marked variability in these parameters across different ABO groups suggests a lack of statistically significant association between hemotypic classification and hematobiochemical perturbations in thalassemia. This dissociation may be attributable to genomic incongruence, as the ABO locus resides on chromosome 9, whereas the β-thalassemia gene is situated on chromosome 11 (Rund & Rachmilewitz, 2005).

Table 3-2: Hematological and biochemical characteristics of patients and control groups according to blood groups

(3-2)a: urea levels in patients and control groups according to blood groups:

Characters		Blood groups	Mean	Std. Error	95% Confidence Interval	
					Level of	L.S.D
Urea	Patient	O	24.689 bac	1.433	0.05	5.217
		A	26.149 ba	1.584		
		B	20.986 c	1.673		
		AB	27.549 a	2.566		
	Control	O	21.759 bc	1.440	0.05	5.217
		A	22.127 bac	1.755		
		B	20.939 c	2.010		
		AB	22.996 bac	2.364		

(3-2)b: creat levels in patients and control groups according to blood groups:

Characters		Blood groups	Mean	Std. Error	95% Confidence Interval	
					Level of	L.S.D
creat	Patient	O	0.377 b	0.029	0.05	0.105
		A	0.392 b	0.032		
		B	0.316 b	0.034		
		AB	0.347 b	0.052		
	Control	O	0.913 a	0.029	0.05	0.105
		A	0.920 a	0.036		
		B	0.915 a	0.041		
		AB	0.910 a	0.048		

(3-2)c: T.S.B levels in patients and control groups according to blood groups:

Characters		Blood groups	Mean	Std. Error	95% Confidence Interval	
					Level of	L.S.D
T.S.B	Patient	O	2.892 a	0.206	0.05	0.747
		A	1.919 bc	0.228		
		B	1.259 dc	0.241		
		AB	2.620 dc	0.369		
	Control	O	0.911 d	0.207	0.05	0.747
		A	0.905 d	0.252		
		B	0.904 d	0.289		
		AB	0.889 d	0.340		

(3-2)d: GOT levels in patients and control groups according to blood groups:

Characters		Blood groups	Mean	Std. Error	95% Confidence Interval	
					Level of	L.S.D
GOT	Patient	O	44.874 a	4.079	0.05	15.264
		A	34.907 ba	4.509		
		B	28.200 b	4.762		
		AB	35.483 ba	7.302		
	Control	O	12.457 c	4.099	0.05	15.264
		A	13.047 c	4.994		
		B	12.668 c	5.722		
		AB	13.757 c	6.727		

(3-2)e: GPT levels in patients and control groups according to blood groups:

Dependent Variable		Blood groups	Mean	Std. Error	95% Confidence Interval	
					Level of	L.S.D
GPT	Patient	O	101.237 c	10.317	0.05	37.533
		A	115.473 bc	11.407		
		B	140.626ba	12.047		
		AB	173.973 a	18.472		
	Control	O	14.097 d	10.368	0.05	37.533
		A	12.703 d	12.632		
		B	13.372 d	14.475		
		AB	13.611 d	17.017		

(3-2)f: ALK.P levels in patients and control groups according to blood groups:

Dependent Variable		Blood groups	Mean	Std. Error	95% Confidence Interval	
					Level of	L.S.D
ALK.P	Patient	O	58.656 a	8.073	0.05	29.25
		A	57.876 a	8.925		
		B	19.505 b	9.426		
		AB	46.156 ba	14.453		
	Control	O	52.081 a	8.113	0.05	29.25
		A	53.148 a	9.884		
		B	52.009 a	11.326		
		AB	62.864 a	13.315		

(3-2)g: Ferritin levels in patients and control groups according to blood groups:

Dependent Variable		Blood groups	Mean	Std. Error	95% Confidence Interval	
					Level of	L.S.D
Ferritin	Patient	O	2450.865 a	366.682	0.05	1332.5
		A	2864.860 a	405.409		
		B	2821.454 a	428.160		
		AB	3035.351 a	656.487		
	Control	O	235.070 b	368.493	0.05	1332.5
		A	181.896 b	448.964		
		B	207.577 b	514.439		
		AB	154.492 b	604.792		

(3-2)h: Hb levels in patients and control groups according to blood groups:

Dependent Variable		Blood groups	Mean	Std. Error	95% Confidence Interval	
					Level of	L.S.D
Hb	Patient	O	7.466c	0.208	0.05	0.868
		A	7.876c	0.230		
		B	8.108c	0.243		
		AB	8.199c	0.372		
	Control	O	13.949b	0.209	0.05	0.868
		A	14.582a	0.255		
		B	14.267 ba	0.292		
		AB	14.406 ba	0.343		

The different letters mean that differences among means of different traits is significant at 0.05 level of significant and similar letters mean there are not significant among means.

3.3 Effect of Interaction of Treatment Groups with Genus on Hematological and Biochemical Characteristics

Table (3-4) delineates the interactive influence of clinical grouping (thalassemia versus control) and biological sex on an array of hematological and biochemical biomarkers. In particular, Table (3-4a) reveals that male subjects afflicted with thalassemia manifested a statistically significant elevation in serum urea concentrations relative to their healthy male counterparts, whereas analogous comparisons among other gender-based subgroups failed to reach statistical significance. The observed hyperuremia in thalassemic males may be pathophysiologically attributed to renal parenchymal deposition of excess iron, reduced erythrocyte lifespan, and systemic hemosiderosis, all of which contribute to multi-organ dysfunction (Mansi *et al*., 2013).

Evaluation of renal excretory competence, as indicated by serum creatinine levels, is a cornerstone in nephrological assessment (Table 3-4b). The measured values resided within normative reference intervals (0.4–1.2 mg/dL), implying an absence of overt deviation in glomerular filtration among β-thalassemia patients (Waqas *et al*., 2024).

Moreover, total serum bilirubin (T.S.B.) levels were markedly elevated in both male and female thalassemic cohorts when compared to their corresponding control groups (Table 3-4c). This hyperbilirubinemia is mechanistically consistent with excessive intravascular hemolysis and accelerated turnover of erythrocytes. Comparable findings were reported by Sultana *et al*., (2011), affirming this correlation.

Tables (3-4d) and (3-4e) further exhibit elevated hepatic aminotransferase levels—GPT (ALT) and GOT (AST)—in β-thalassemia patients. These elevations are likely a consequence of hepatic iron overload. When concomitant with severe anemia and splenomegaly, transaminitis is often prognostic of advanced disease severity and diminished therapeutic outlook (Gira *et al*., 2013; Abdulla, 2018).

With respect to alkaline phosphatase (ALP) activity, data in Table (3-4f) suggest negligible significance among thalassemic patients of both sexes, while controls demonstrated highly significant enzymatic activity. This observation contradicts the findings of Kanbour *et al*., (2018), who posited that escalating ferritin levels induce a proportional rise in hepatic ALP activity.

Furthermore, Table (3-4g) confirms the presence of significantly elevated ferritin levels in both male and female thalassemia patients compared to their healthy counterparts. This hyperferritinemia is a well-recognized sequela of chronic erythrocyte transfusion regimens, culminating in systemic iron overload. These findings align with those of prior investigations (Surchi *et al*., 2018; Eghbali *et al*., 2014).

Lastly, Table (3-4h) reveals that hemoglobin (Hb) concentrations among healthy male and female individuals were significantly greater than those observed in the thalassemic population. This disparity is emblematic of the hypochromic, microcytic anemia intrinsic to thalassemia pathology and corroborates the observations of Karim *et al*., (2016).

Table 3-4: Effect of interaction of treatment groups with gender on different characters.

(3-4)a Urea levels in patients and control groups according to gender:

Characters		Gender	Mean	Std. Error	95% Confidence Interval	
					Level of	L.S.D
Urea	Patient	Male Female	25.739 a 23.947 ba	1.233 1.320	0.05	3.551
	Control	Male Female	21.882 b 22.028 ba	1.146 1.569	0.05	3.551

(3-4)b: creat levels in patients and control groups according to gender:

Characters		Gender	Mean	Std. Error	95% Confidence Interval	
					Level of	L.S.D
creat	Patient	Male Female	0.348 b 0.368 b	0.025 0.027	0.05	0.070
	Control	Male Female	0.914 a 0.915 a	0.023 0.032	0.05	0.070

(3-4)c: T.S.B levels in patients and control groups according to gender:

Characters		Gender	Mean	Std. Error	95% Confidence Interval	
					Level of	L.S.D
T.S.B	Patient	Male	2.232 a	0.177	0.05	0.561
		Female	2.113 a	0.190		
	Control	Male	0.900 b	0.165	0.05	0.561
		Female	0.904 b	0.226		

(3-4)d: G.O.T levels in patients and control groups according to gender:

Characters		Gender	Mean	Std. Error	95% Confidence Interval	
					Level of	L.S.D
GOT	Patient	Male	42.266 a	3.509	0.05	10.094
		Female	29.466 b	3.757		
	Control	Male	12.987 c	3.262	0.05	10.094
		Female	12.978 c	4.465		

(3-4)e: GPT levels in patients and control groups according to gender:

Characters		Gender	Mean	Std. Error	95% Confidence Interval	
					Level of	L.S.D
GPT	Patient	Male	138.745 a	8.877	0.05	24.436
		Female	126.910 a	9.504		
	Control	Male	13.466 b	8.251	0.05	24.436
		Female	13.426 b	11.294		

(3-4)f: ALK.P levels in patients and control groups according to gender:

Characters		Gender	Mean	Std. Error	95% Confidence Interval	
					Level of	L.S.D
ALK.P	Patient	Male	47.016 a	6.946	0.05	20.507
		Female	44.081 a	7.436		
	Control	Male	54.479 a	6.456	0.05	20.507
		Female	55.572 a	8.837		

(3-4)g: Ferritin levels in patients and control groups according to gender:

Characters		Gender	Mean	Std. Error	95% Confidence Interval	
					Level of	L.S.D
Ferritin	Patient	Male	2609.071 a	315.509	0.05	879.43
		Female	2977.194a	337.775		
	Control	Male	184.315 b	293.237	0.05	879.43
		Female	205.203 b	401.397		

(3-4)h: Hb levels in patients and control groups according to gender:

Characters		Gender	Mean	Std. Error	95% Confidence Interval	
					Level of	L.S.D
Hb	Patient	Male	8.058 c	0.179	0.05	0.520
		Female	7.766 c	0.192		
	Control	Male	15.058 a	0.166	0.05	0.520
		Female	13.545 b	0.228		

The different letters mean that differences among means of different traits is significant at 0.05 level of significant and similar letters mean there are not significant among means.

CONCLUSIONS

1. The study results indicate statistically significant changes in several biochemical and hematological indicators in thalassemia patients compared to healthy individuals, reflecting the physiological and pathological effects associated with the disease.
2. The results showed a significant increase in the levels of urea, bilirubin (TSB), and GOT and GPT enzymes in thalassemia patients, which may indicate the presence of liver and kidney effects resulting from the disease or related to repeated blood transfusions.
3. The study showed a significant decrease in creatinine, hemoglobin, and alkaline phosphatase levels in patients, indicating impaired renal function and chronic anemia.

4. The results revealed that ferritin levels were significantly elevated in thalassemia patients of all blood types, confirming the presence of iron overload common in these patients.
5. The study showed an interaction effect between gender and health status (patient/control), with male thalassemia patients showing higher urea levels than healthy males, with differences in other indicators depending on gender.
6. The results also showed variations in biochemical markers across blood types, especially B, suggesting a possible relationship between blood type and some biomarkers in thalassemia patients.

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