Abstract---The current study aims to study some biochemical indicators for thalassemia patients in the Najaf governorate, and the study included 25 patients with major beta thalassemia during the period from November 2019 to February 2020 and their ages ranged between (2 - 65) years and 15 healthy people who are not He had genetic blood diseases and their ages ranged between (2-65) years. The results of the study showed that there are in some biochemical indicators, as there was a significant increase in the level of the enzyme of liver function ALT that was the focus in patients 23.74 ± 29.53 U/L and in healthy people it was 4.61 ± 11.67 U/L. Also, the creatinine concentration patients 9.92 ± 26.08 mmol/L and in healthy was, 68.06 ± 51.54 mmol/L. Except for urea, we notice that there was a decrease in its concentration in patients 0.89 ± 3.96 mmol/L and in
healthy was $1.03 \pm 2.36 \text{ mmol/L}$, There was an increase in the concentration of iron in the blood in patients $382.05 \pm 64.37 \text{ Umol/L}$ and in healthy was $9.48 \pm 61.36 \text{ Umol/L}$, as well as a decrease in the concentration of glucose in patients and an increase in healthy people, $18.58 \pm 28.59 \text{ Umol/L}$ 9.10 $\pm 20.44 \text{ Umol/L}$ respectively. We conclude from this study about some changes that occur on some biochemical indicators for patients with thalassemia that can be attributed to liver and heart diseases, as well as to kidney failure arising from the toxicity of high iron concentration, which is a feature of thalassemia.

**Keywords**—β–Thalassemia, Iron overload, urea.

**Introduction**

Thalassemia is a disease of the quantitative hemoglobinopathy that causes disruption in the bioprocessing of peptide chains in hemoglobin, which leads to a disturbance in the forms and functions of red blood cells [1] if this disease is caused by genetic point mutations in the genes controlling the production of alpha or beta chains of peptide. There are two main types of thalassemia. They are alpha α thalassemia and beta thalassemia β-thalassemia, which is a genetic disorder that is caused by point mutations at one or more sites of the B-globin gene site that lead to a decrease in the production of carbene B [2] Recently more than 200 different mutations affecting At levels of expression of the B-globin gene and cause β-thalassemia, these mutations are not uniformly distributed, but have geographical specificity and ethnic origin. This type of thalassemia is characterized by the presence of a small number of joint mutations and variable numbers of those rare [4].

There are a set of clinical symptoms of the disease Beta thalassemia, including fatigue, shortness of breath, loss of appetite and enormous stress Spleen and heart failure and deformation of bones and delayed puberty, and progression of the disease leads to a severe shortage in the level of Alimoklopan in the blood, which periodically requires blood transfusions every 3-4 weeks to maintain a high level of hemoglobin in the blood [3]. Thalassemia can be divided into:

1-Thalassemia Minor
It is a type of beta-thalassemia and is also known as (beta-thalassemia carrier) and has symptoms affecting the patient. (5), and this type of thalassemia varies from the other forms in that the patient has normal hemoclobin in the beta-globin allele. The other is affected by all forms of thalassemia, whether extreme ° β or mild + [6], and by patients suffering from thalassemia. These signs are triggered by regular blood tests, and small blood cells are identified by these tests., Small blood cells are found red in patients with mild thalassemia because there is a potential risk that these symptoms may progress into thalassemia. Major in the case of blood transfusion and the person providing the blood has thalassemia (7).

2-Thalassemia Major
It is the most serious form of beta-thalassemia and has an effect on the patient. In this case, the patient has a mutation in all beta-globin genes, which
contributes to a decrease in the development of beta-globin chains[8] if there are three alleles that are responsible for the phenotype of thalassemia, which is °β °β and °β + β and often + β + \ β [9]. Excessive alpha-globin chains disperse across the body, causing damage to the red blood cell membranes resulting in excess chains. Hemolysis in the blood vessels[10], and In addition, there is substantial damage and degradation to RBC precursors, which causes the development of ineffective RBCs and causes anemia that is hazardous because it prevents the transfer of oxygen to cells[11]; Some patients with thalassemia die from frequent blood transfusions that allow iron to accumulate in their bodies, and there are a variety of clinical symptoms of beta thalassemia, including weakness, shortness of breath, lack of appetite, swollen liver and spleen, heart failure, bone deformity and delayed puberty, and disease progression, leading to a serious hemorrhage deficiency of hemoglobin in the blood [12]. The aim of the study: to evaluate the biochemical and hematological aspects of thalassemia patients in the AL-Najaf community.

Method

Serum samples were taken from Thalassemia patients while they were visiting the center at Al-Batoul Hospital And the period of the search period From September 2015 to May 2016 she lived for a year and a half to Ammar It was taken, from 25 to 1.5 years, and they were free of hereditary blood diseases, and the patients were taken And a house cafeteria bacteria; 5 ml of blood was drawn Counterpart biochemical analyzes were performed in the hospital’s blood laboratory, sampling samples from samples This is what they think of the show.

Methods for measuring biochemical indicators

1- Measurement of serum alanine amino transferase (ALT) activity. Serum alanine amino transferase activity was determined using a Randox diagnostic kit The activity of ALT was determined by regulating the concentration of pyruvate hydrazone formed with 2 - 4 - Dinitrophenylhydrazine, which gives a brown color, can be measured by a spectrophotometer at a wavelength of 546 nm if The color intensity is proportional to the amount of enzyme in the serum. (13) .

Determination of the level of creatinine in the blood serum

The level of creatine in the blood serum was measured by using the analysis kit equipped by the German company Roche, and it is basedThis is a Jaffe colorimetric test or colorimetric test. In an alkaline solution, creatine is formed from a compound or compound Reddish-yellow complex, the resulting pigment rate is directly proportional to the concentration of creatine in the sample, as in the following equation. Creatinine + Picric acid ——— ——— Yellow - red complex

- Determination of the level of glucose in the blood serum

The level of glucose in the blood serum was measured by the simple use of the equipped analysis kit from the German company Roche. Enzyme based Hexokinase phosphorylates glucose by ATP and creates Glucose-6-phosphate (G6P). To monitor the reaction, an enzyme Second-Glucose- (G6PDH) 6-phosphate dehydrogenase is used to stimulate the oxidation of glucose-6-phosphate. By NADP to configure NADPH.

D- glucose + ATP → D- glucose -6- phosphate + ADP
D-glucose-6-phosphate + NADP $\rightarrow$ D-6-phosphogluconate + NADPH

The resulting NADPH concentration is directly proportional to the glucose concentration and can be estimated by estimating the increase in absorbance at 340nm (14).

**Measurement of serum urea concentration**

The level of urea in serum was measured by a colorimetric method, using the diagnostic kit equipped with Before the German company Human. Urease hydrolyzes urea and produces ammonia, as shown in the equation below:

Urea + H2O $\rightarrow$ 2 NH3 + CO2

In the basic medium, the ammonium ions resulting from the reaction react with Salicylate and Hypochlorite to give green For endophenols (2,2 dicarboxyl indophenols), this reaction is catalyzed by the addition of Sodium Nitroprusside, as shown in the following equation:

2H 4 + Salicylate + hypochlorite $\rightarrow$ indophenols

The color intensity is directly proportional to the urea concentration in the blood serum

**Determination of serum iron concentration**

The level of iron in the blood serum was measured using the analysis kit equipped by the German company Roche. According to the method, under acidic conditions, the iron released from the lipemic transferrin samples is evident by detergents, ascorbates reduce the iron triple source to the iron source and then react with the resulting ferrosine color complex.

Transferrin – Fe $\rightarrow$ apotransferrin + Fe complex
Ascorbate Fe³ $\rightarrow$ Fe²
Ferrozine + Fe² $\rightarrow$ colored complex

The color rise is directly proportional to the iron concentration, and is estimated by calculating the absorbance at 552 nm.

**Statistical analysis**

Statistical analysis was carried out using the statistical package for social sciences program in determining the mean and standard deviation in addition to other variables. The differences in the case of the probability p ≥ 0.05 were considered significant.
Results and Discussion

Table (1)
Comparison of some biochemical indicators between healthy people and patients with thalassemia in relation to the mutation IVS.I.5 (G-C) and for all districts in Najaf Governorate

<table>
<thead>
<tr>
<th>Parameters</th>
<th>N=65 IVS-I-5 (G-C)</th>
<th>P value (T test)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>ALT U/L Patients</td>
<td>30.03</td>
<td>24.81</td>
</tr>
<tr>
<td>Control</td>
<td>10.719</td>
<td>2.57</td>
</tr>
<tr>
<td>Creatinine mmol/L Patients</td>
<td>26.56</td>
<td>10.04</td>
</tr>
<tr>
<td>Control</td>
<td>40.49</td>
<td>5.65</td>
</tr>
<tr>
<td>UNB mmol/L Patients</td>
<td>4.00</td>
<td>.880</td>
</tr>
<tr>
<td>Control</td>
<td>3.59</td>
<td>6.71</td>
</tr>
<tr>
<td>Glucose mmol/L Patients</td>
<td>5.92</td>
<td>1.61</td>
</tr>
<tr>
<td>Control</td>
<td>4.52</td>
<td>1.36</td>
</tr>
<tr>
<td>Control</td>
<td>60.07</td>
<td>10.36</td>
</tr>
<tr>
<td>Fe umol/L Patients</td>
<td>35.48</td>
<td>11.10</td>
</tr>
<tr>
<td>Control</td>
<td>24.16</td>
<td>13.36</td>
</tr>
</tbody>
</table>

The results of the current study, as shown in Table (1), show a comparison of some biochemical indicators between healthy and infected patients with thalassemia in relation to the IVS.I.5 (G-C) mutation, the presence of this mutation increased among patients with thalassemia by 90% It is the highest rate among other mutations, and its spread was observed in all districts of Diyala governorate. If we notice an increase in enzyme levels ALT in patients compared to healthy subjects, if this increase in the enzyme ALT is caused by determining whether the liver has been exposed to damage as liver disease is associated with chronic blood transfusions in thalassemia patients, which causes chronic hepatitis And that the role of iron excess in pregnancy causes liver impairment in patients suffering from thalassemia, and an elevation of this enzyme has been observed In thalassemia patients, knowing the concentration of this enzyme is evidence of Diagnosis of liver function in patients with beta thalassemia (16).

Iron Fe levels increased in patients, and the reason for the increase in iron in the blood is due to its accumulation in various body systems (the heart). And liver and kidneys (and that repeated blood transfusions lead to abnormalities in the red blood cells, so this extra iron is required to be eliminated In the body by removing this mineral and that these patients suffer from thalassemia, heart disease and high blood pressure The pulmonary process is to remove this mineral and it takes a long time (17).

The results showed an increase in glucose levels, as the cause of an increase in the concentration of glucose in the blood among patients with: Thalassemia may reach beyond the normal range and cause harmful health effects in some types of cells such as muscle cells Fatty acids need insulin in order to absorb glucose, and blood glucose levels will rise when these cells fail in
responding appropriately to the proliferation of insulin, it helps the liver regulate glucose levels by reducing secretion Glucose in the presence of insulin, and this natural reduction in glucose production in the liver may not occur in people with resistance Of insulin and that insulin resistance in muscle and fat cells reduces glucose absorption (18).

The reason for increasing the concentration of urea in the blood is due to protein intake and in some cases associated with protein catabolism, such as diabetes Untreated, hyperthyroidism and in thalassemia diseases, as the high concentration of urea in patients with beta thalassemia is due to Deposition of iron in excess and short life of red blood cells, and in the case of renal insufficiency, relatively affect the level of urea in the blood And in thalassemia patients who suffer from an enlarged liver It consists of ammonia that the liver fails to convert into urea(19). The reason for the decrease in the concentration of creatinine in the blood is due to the significant decrease in the concentration of creatine in the blood serum, and it may be related A link in low BMI due to developmental delays and reduced body muscle mass and in these patients Of thalassemia, and that creatine is the most commonly used metric Knowing the functions of the kidney and this leads to a process dysfunction Filtration carried out by the kidneys, and that the low concentration of creatine in the blood is due to insufficiency in the functions of the kidneys that reduce the process Glomerular filtration and that the glomerular filtration rate is clinically important because it is a measure of kidney function (20).

Reference


