Comparative study between level of iron and liver damage in Iraqi Patients with Thalassemia and Thalassemia Related with DM1

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Abstract

This paper describe the study of the role of iron overload and some biochemical parameters in 30 patients with β-thalassemia major with diabetes type 1 (TD), 30 patients with β-thalassemia major as pathological control group(TM) ,all patients were having chelating therapy (desferrioxamine) , and 30 healthy individuals as control group. The age of all studied groups were range from (12-35)years .And attempted to shed a light on the possible correlation between (serum Iron and ferritin)and some parameters (TP,Alb ,IgM .IgG, IgA, TSB, GPT, GOT and Alk)for TM and TD patients. The results revealed a significant increased in ferritin, iron, immunoglobulins , liver function (TSB,GPT,GOT and ALK) in TM &TD as compared to control ,while a significant decreased in total protein and albumin as compared to control group .

Keywords: Anemia, Ferritin , Immunoglobulins , Liver Functions.

Introduction

Thalassemia is a group of conditions in which there are a reduced rate of synthesis of one or more of the globin chains leading to imbalanced globin chains synthesis, defective hemoglobin production and damage to the red blood cells or their precursors which results from the effects of the globin subunits that are produced in excess. In thalassemia, the imbalance of globin chain synthesis leads to red blood cells damage resulting in destruction of red blood cells in the bone marrow (ineffective erythropoiesis) and peripheral circulation (hemolysis) [(1)]. For many patients with chronic anemia, regular red blood cell transfusions represent life saving therapy. Regular blood transfusions have dramatically extended life expectancy in thalassemia major [(2)].

Blood transfusions contain red cells reach the end of their life due to aging or defects they are broken down, and the hemoglobin molecule broken up and the iron recycled.

Humans are unable to eliminate excess iron and regulate body iron stores by limiting absorption [3]. Too much iron in the body is called iron overload [4]. Iron exists in two stable oxidation states, oxidized ferric (Fe^{+3}) and reduced ferrous (Fe^{+2}) which accounts for its essentiality as a trace element and its crucial role in the oxygen and electron transport reactions of all living cells.

Iron is transported as transferrin to liver or spleen where it is stored as ferretin, which has a high iron storage capacity and prevents iron from participating in the Fenton reaction [5].

Serum ferritin levels are measured in patients as part of the iron studies workup for anemia. The ferritin levels measured have a direct correlation with the total amount of iron stored in the body including cases of anemia of chronic disease. If ferritin is high, there is iron in excess.

Ferritin is also used as a marker for iron overload disorders, such as hemochromatosis, hemosiderosis, and porphyria in which the ferritin level may be abnormally raised[6].

Iron overload has now complicated the course of many patients with this disorder, resulted in increased iron accumulation (ferritin) in the liver and heart worsening of hepatic fibrosis, and development of cardiac fibrosis [7].

Diabetes mellitus (DM) are possible complications in patients with thalassaemia major treated with transfusions. Possible pathogenetic conditions are pancreatic cell destruction with consequent insulin deficiency, liver derangement with consequent insulin resistance, and genetic actors. All these, either singly or together, have been found in our patients with diabetes mellitus. In addition 28% of these patients developed diabetes mellitus shortly after an acute viral hepatitis infection. This suggests that some hepatitis viruses might precipitate diabetes mellitus in thalassaemic patients [8].

Diabetes mellitus (DM) is a heterogeneous disorder of carbohydrate metabolism and characterized by inappropriate hyperglycemia resulting either from defects in insulin secretion, insulin action, or both [9].

Total protein level depends on the balance between their synthesis and their catabolism or loss from body. A total serum protein test is measured total amount of protein in blood serum as well as the

amounts of albumin and globulin which are the main two groups of protein.

Albumin has a single polypeptide chain of 580 amino acids. It is a very stable protein with a high net negative charge at physiologic pH .Its forms the largest portion of the serum proteins. It is produced and degraded by the liver, and it carries many small molecules [10].

Immunoglobulins are glycoprotein molecules that are produced by plasma cells in response to an immunogen and which function as antibodies. The immunoglobulins derive their name from the finding that they migrate with globular proteins when antibody-containing serum is placed in an electrical field [11].

Beta-thalassemia major patients suffer from too many problems rather than severe anemia including increased susceptibility to bacterial infections which plays a major role in the patient's morbidity and mortality[12-13].

The possible changes of immune system in thalassemic patients, considering the humeral and cellular immune systems; but no consistent defect in white cells or immune function had been documented yet [14,15].

Factors such as splenectomy, iron overload, repeated exposure to foreign antigens at the time of blood transfusion and the use of chelating agents, have been suggested to induce profound deleterious effects on the immune system.

Degradation of heme after 120 days in the circulation is to produce green pigment biliverdin after two steps of oxidation reactions. Biliverdin is reduced forming bilirubin, the iron is either used to make new hemoglobin molecule in the red bone marrow or stored in the liver as an iron – protein complex by protein ferritin.

Bilirubin binding to albumin and enters a hepatocyte, in hepatocyte the solubility of Bilirubin is increased by the addition two molecules of glucorinc acid and excretion of bilirubin into bile .Its can be measured as direct or conjugated. Sometimes the total amount of bilirubin in the blood measured [16].

AlanineTransaminasese(ALT) also called serum glutamate - pyruvate transaminase (SGPT). ALT is present in high concentration in liver lesser extent in skeletal muscle, kidney, and heart [17], measurement of ALT activity in serum is used an indicator of hepatoceller damage [17.18] .

Aspartate Transaminase (AST) this enzyme is also called glutamate oxaloacetate transaminase (SGOT), present in high concentration in the following organs heart, liver, skeletal muscle, kidney and erythrocytes. Damage to any of these tissues may result in increased plasma AST levels [(18)].

Alkaline phosphatase is an enzyme found in all living tissues, and is concerned with the removal of the phosphate from protein and other molecules. It is found in a large amount in the liver and bones [(19)]. This enzyme is also found in small amounts inside the bile duct, lining of the intestine, kidney and placenta.

The aims of this work were to estimate the levels of (TP, Alb, IgM .IgG, IgA, TSB,GPT,GOT and Alk.) and relationships between them and (Fe ,ferritin) in patients with β-thalassemia major with diabetes type 1 (TD), patients with β-thalassemia major as pathological control group(TM), [all patients were having chelating therapy (desferrioxamine)], and compared with healthy individuals as control group.

Materials and Methods

1-Selection of subjects and blood sampling:-

Three (3) mLs of venous blood samples were obtained from 30 patients with β-thalassemia major with diabetes type 1 (DM1), 30 patients with β-thalassemia major as pathological control group, all patients were having chelating therapy (desferrioxamine) , and 30 healthy individuals as control group. The age of all studied groups were range from (12-35) years. The samples collected from patients treated in Ibn-Al-Baladi hospital. The samples were transferred into plain tube without anticoagulant, after about 15 minutes centrifuged at 3500 rpm for(10 minutes).The serum was separated and frozen at (-20 ˚c) till used.

2-Determination of serum iron concentration:-

Iron concentration were measured by Colorimetric methods according to Garcic A.et.al ,Human kit,Ref.10229,Germany.

3-Determination of serum ferritin levels:-

Ferritin levels were measured by Enzyme Linked Florescent Assay (ELFA) according to Vernet ,M.,et.al ,bioMerieux sa kit, Ref. 30411, France .

4-Determination of serum Total Protein &Albumin Concentration:-

Total Protein were measured by using ALAGO S PR-N protein o-129/dl and Albumin were measured by using colorimetric methods according to Doumas BT.,et.al ,BioSystems S.A. kit. Ref. 11573 , Spain.

5-Determination of serum IgM, IgA and IgG:-

Immunoglobulins (IgM, IgA and IgG) were determined by radial immunodiffusion plate according to Mancini,et.al.,LTA S.r.I kit, Ref.RK01000 for IgM,RK 00800 for IgA, RK00900 for IgG,Milano 15/F.

6-Determination of serum liver function:-

Bilirubin concentration(TSB) ,Transaminases (COT&GPT)activity and Phospatase alkaline activity were measured by colorimetric methods according Jendrassik and Grof ,Randox kit,Ref.BR411,CE.for Bilirubin ,and according to Reitman and Frankle .al,BioMerieux® sa .kit.Ref 61 691/61 692 ,France for Transaminases ,and according to Kind P.R.N., et.al , BioMerieux® sa .kit.Ref 61 511,France

7-Statistical Analysis:-

Statistical analyses were done using Microsoft office (SPSS version 10.01) which includes the following: Mean ± standard deviation, Student t-test, ANOVA test, Correlation coefficient, P value of less than 0.05 was considered significant.

Results

Table (1) showed the all different parameters in the study which expressed as mean ±SD in 30 patients with major thalassemia with diabetes (TD), 30 patients with major thalassemia (TM) and 30 control group (Cot). Table (2) showed Pearson's correlation coefficient between some parameters in TM&TD.

Table (1):Mean±SD values of different parameters in TM,TD and Cot.

Parameters	Mean±SD			p-value of ANOVA	p-value of t-test		
	Cot(I)	TM(II)	TD (III)		Ivs II	I vs III	II vs III
Iron µmol/L	15.74±4.70	34.09±8.79	38.68±3.78	P<0.05	0.061	0.058	0.006*
Ferritin ng/dL	162.9±97.82	3300±2093	2657±2973.4	P<0.05	0.00*	0.00*	0.104
Total protein g/L	7.74±0.64	7.27±0.56	7.19±0.95	P<0.05	0.637	0.09	0.034*
Albumin g/L	33.52±5.63	30.64±5.64	30.71±9.67	P>0.05	0.096	0.09	0.09
IgM mg/dL	216.86±94.7	295.9±66.2	296.02±69.9	P<0.05	0.148	0.147	0.893
IgA mg/dL	301.5±158	468.1±166	595.9±207.6	P<0.05	0.671	0.179	0.324
IgG mg/dL	1236.9±237	1736.2±469	1968±588	P<0.05	0.005*	0.00*	0.204
TSB mm/L	11.38±2.18	31.56±14.8	35.39±21.1	P<0.05	0.00*	0.00*	0.445
GPT U/L	9.866±3.00	9.866±3.00	38.12±19.5	P<0.05	0.00*	0.00*	0.063
GOT U/L	13.8±3.37	33.45±25.3	46.37±1 9.5	P<0.05	0.00*	0.00*	0.133
Alkaline U/L	39.24±9.95	90.59±46.6	131.62±78.5	P<0.05	0.00*	0.00*	0.228

*

The t-test for two independent means is significant at the P value 0.05 levels or less.

Table (2):Correlation coefficient between different parameters in TM and TD.

		TM		TD	
		Fe	**Ferritin**	**Fe**	**Ferritin**
TP	**r**	-0.138	0.162	0.014	-0.231
	p-value	N.S*	N.S*	N.S*	N.S*
Alb	**r**	0.064	-0.122	-0.102	٠,٠٢٧
	p-value	N.S*	N.S*	N.S*	N.S*
IgM	**r**	0.0.13	٠,٢٣٧	-0.135	0.229
	p-value	N.S*	N.S*	N.S*	N.S*
IgG	**r**	-0.292	-0.176	-0.196	-0.084
	p-value	N.S*	N.S*	N.S*	N.S*
IgA	**r**	-0.091	٠,٠٠٩	-0.130	-0.258
	p-value	N.S*	N.S*	N.S*	N.S*
TSB	**r**	-0.062	٠,٠٨٢	0.102	-0.002
	p-value	N.S*	N.S*	N.S*	N.S*
GPT	**r**	0.037	-0.204	-0.18	-0.335
	p-value	N.S*	N.S*	N.S*	N.S*
GOT	**r**	-0.151	-0.004	٠,٠٥٩	٠,١٣٩-
	p-value	N.S*	N.S*	N.S*	N.S*
Alk	**r**	٠,٢٤٣	-0.030	٠,١١٩	٠,٠٤٩
	p-value	N.S*	N.S*	N.S*	N.S*

*N.S=non-significant

Discussion

This study showed that:

1. The levels of iron (Fe) in serum were significantly lower in TD patients& TM pateints compared to Cot group, while ferritin levels were significantly higher in TD patients & TM patients compared to Cot group. And iron concentration was significantly higher in TD patients compared to TM patient's .While ferritin levels were significantly lower in TD patients compared to TM patients. .This study showed that mean firritin &iron was significant increase in TM and TD patients compared to control .

Some researches demonstrated the relationship between iron and glucose metabolism, because iron modulates insulin action in the human.

Iron is an essential element for many important metabolic functions (oxygen transportation and utilization; DNA synthesis, electron transport and many other biological processes.

Iron is part of hemoglobin; iron is also part of myoglobin, which helps muscle cell store oxygen, dietary iron available as either heme or non heme

The most common treatment for all major forms of thalassemia is blood transfusions. These transfusions are necessary to provide the patient with hemoglobin capable of carrying the oxygen that the patient's body

needs. Most patients with a major form thalassemia receive red blood cell transfusions every two to three weeks about 250 milligram of iron.

The principal goal of chelating therapy is to decrease tissue iron to concentration where iron mediated toxicity can not occur. The chelator should also have a high specificity of iron (III) [20].

Iron loaded patients with β-thalassemia are attributed to high oxidative stress in these patients which is secondary to iron derived free radicals to the patient's diminished antioxidant.

Antioxidant reserve productions of super oxide radical in some diseases leads to release of iron from ferritin, further more ferritin iron can be released either by a reductive process. These free iron catalyst subsequent reaction lead to production of hydroxyl radicals $OH^{\cdot}$ (Fenton reaction) .

$$Fe\ (II) + H_2O_2 \rightarrow OH^{\cdot} + OH^{\cdot} + Fe\ (III)$$

Moreover, free ion catalyses the reaction $\cdot O^{\cdot}{}_2^{-}$ and H_2O_2 which also produce hydroxyl radicals.

$$O^{\cdot}{}_2^{-} + H_2O_2 \xrightarrow{Fe^{++}} OH^{\cdot} + OH^{\cdot} + O_2$$ (Haber -Wiss reaction)

Iron has six electrochemical coordination sites that should be tightly bound to block the ability of the iron ions to catalyze redox reaction redistribution.

Iron chelators should reduce tissue iron levels, prevent excessive organ iron accumulation and neutralize toxic labile iron pools based on the number of the coordination [20,21].

Iron accumulators at different rates in various organs and these organs show a different susceptibility to the damage induced by reactive iron species such as non trans ferring bound iron (NTBI) and the intracellular labile iron pools.

Iron is catalyst of free radicals stress, and it has been suggested that free radicals and lipid peroxidation play a part in the etiology of diabetes. The cells that produce insulin are extraordinarily sensitive to damage from oxidation [22].

The iron is stored in the liver as ferritin . Ferritin is normally found mainly inside the cells, will only a small amount in the blood when there is damage to organs that contain ferritin (especially the liver, spleen and bone marrow [23, 24].

Elevated iron stores, reflected in elevated plasma ferritin levels, may increase baseline glucose levels and induce other metabolic abnormalities that ultimately result in diabetes. Alternatively, elevated plasma ferritin

may be just one of several metabolic abnormalities related to the underlying process that ultimately results in diabetes, rather than a causal factor for diabetes development. Further more, while plasma ferritin levels reflect iron stores, ferritin levels are also determined by many other genetic and environmental factors. It is thus possible that the association between ferritin and diabetes may not reflect changes in iron stores [25.26]**.** While an elevated plasma ferritin level may be a causal factor for diabetes, it is also possible that the moderately increased ferritin levels of diabetes patients are just a marker for the metabolic alterations that ultimately result in diabetes, without a causal role in diabetes development. In fact, metabolic abnormalities may lead to increased ferritin levels through a variety of mechanisms. Insulin stimulates the expression of ferritin mRNA, an effect that is probably mediated through insulin-like growth factor receptors. In certain insulin-sensitive cells, such as adipocytes, receptors for transferrin, glucose, and insulin-like growth factor II colocalize in the cell membrane, and the presence of insulin results in the simultaneous translocation of all three proteins [27]. Therefore, it has been hypothesized that insulin mediated glucose transport may lead to increased transferring receptors on the cell surface, resulting in increased uptake of extracellular iron.

2. The levels of total protein (Tp), albumin (Alb) and immunoglobulin (IgM and IgA) in serum were significantly lower in both patients groups compared to Cot group and Alb, IgM, IgG and IgA in TD patients as compared to TM patients, while the levels of IgG in serum were significantly higher in both patients groups compared to Cot group and serum total protein levels in TD patients as compared to TM patients. A significant decrease in TP in TM and TD patients compared to Cot and non significant Alb in TM and TD patients compared to Cot .This study showed that mean IgG,IgM and IgA was significant increase in TM and TD patients compared to Cot .In TD patients ferritin was negatively correlated to TP and IgA and positively correlated to IgM and non correlated to Alb and IgG Iron was negatively correlated to Alb IgM,IgA and IgG and non correlated to TP . In TM patients Ferritin was positively correlated to Tp and IgM and negatively correlated to Alb and IgG and non correlated to IgA while Iron was non correlated to Alb ,IgA and IgM and negatively correlated to TP and IgG .

Total protein is the most abundant compounds in serum. The protein makeup of the individual is important diagnostic significance because of

proteins involvement in enzymes, hormones and antibodies as well as osmotic pressure balance.

Protein measurements can reflect nutritional state, kidney disease, liver disease, if total protein is abnormal, further tests must be performed to identify which protein function, and then which specific protein is abnormal.

The possible cause of decreased serum total protein secondarily decreased synthesis of protein by the liver .The main aspect in management of severe β- thalassemia is to keep the patient on maintenance blood transfusion. The hyper transfusion regimen is the best, because the stimulus to unlimited bone marrow expansion, which causes much of the pathology, is reduced but the most serious side effect of life long transfusion therapy is iron overload. Albumin is the major constituent of serum protein (usually over 50%). It is manufactured by the liver from the amino acids taken through the diet. It helps in osmotic pressure regulation, nutrient transport and waste removal [28].

Albumin is the protein of the highest concentration in plasma. Albumin transports many small molecules in the blood for example bilirubin, calcium, progesterone and drugs [28]. It is also of prime importance in maintaining the osmotic pressure of the blood (that is, keeping the fluid from leaking out into the tissues), because albumin is synthesized by the liver, decreased serum albumin may result from liver disease .In thalassemic patients need repeated blood transfusions lead to buildup of iron in the body damaging the liver and other organs [29].Levels may be misleading and may be normal in the face of quite mark changes in the constituent proteins ,only low albumin levels are of clinical importance[30].

Globulins is a collective term ,(made up of different proteins),used to refer to the proteins other than albumin .With the exception of the immunoglobulins and some complement proteins which is formed by immune system ,most of the globulins are produced in the liver [30].

These proteins are important in preventing and fighting infections. Other globulins transport or bind metals [31].

Many studies have been carried out to evaluate the possible changes of the immune system in thalassemic patients, considering the humeral and cellular immune system ;but no consistent defect in white cells or immune function had been documented.

The immunoglobulins, which are antibodies, are a heterogeneous group of plasma proteins produced by B lymphocytes .The (IgA) is a major class of antibodies in secretions, including tears, saliva and secretion of the respiratory system. Increased levels are seen in infections and β-thalassemia [32].

IgM, is the first antibody that appears in response of antigen stimulation .Increased IgM concentration is found in toxoplasmosis, herpes, syphilis, various bacterial and fungal diseases and β-thalassemia.

Approximately (75%) of plasma immunoglobulins are IgG. Increased levels are seen in infectious diseases, liver diseases, myelomas, and β-thalassemia [32].

This observation can be attributed to many factors. For instance repeated blood transfusion in β-thalassemia patients will result in a continuous exposure to various antigens and will lead to increased levels of serum immunoglobulins. It is recommended that thalassemia patients blood matched for Rh and kill antigens and presto rage leukodepleted RBC_s [33].

Thalassemia patients are prone to many bacterial and viral infections. Repeated infections also stimulate the immune system and may result in increased immunoglobulin levels .Iron overload was suggested by some investigators as an important contributing factor in altering the immune parameters in thalassemia patient. It has been suggested that iron overload results in increased migration of T helper cells to the gut and lymph nodes and this causes an increase in serum immunoglobulin levels in thalassemia patients [34].The observed immune disorder represents mostly a secondary immune system defect rather than a primary problem. These changes can not fully explain the increased susceptibility to infection among patients, and it seems that, β-thalassemia major patients have rather a normal antibody response to bacterial and viral infections with normal levels of complement factors .Changes of complement factors seem to follow an increase in immunoglobulin levels ;but ,the possibility of deficient complement factors synthesis can not be ruled out [35].

3- The levels of total serum bilirubin (TSB), Transaminases (GPT, GOT) and Alkaline phosphatase (Alk) in serum were significantly increased in both patients groups compared to Cot group and non significant in TD patients as compared to TM patients. This study showed that mean of TSB, GPT, GOT and Alk were significant increased in TM and TD patients compared to Cot. In TD patients ferritin level was positively

correlated to GOT and non correlated to TSB and Alk and negatively correlated with GPT while Iron level was non correlated to GOT and positively correlated to TSB and Alk and negatively correlated to GPT . In TM patients Ferritin level was negatively correlated to GPT and non correlated to TSB,GOT and Alk while iron was positively correlated to Alk and non correlated to TSB and GPT and negatively correlated to GOT.

A yellow pigment that is the end result of hemoglobin breakdown. This pigment is metabolized in the liver and excreted from the body through the bile. Bloodstream levels are normally low; however, extensive red cell destruction leads to excessive bilirubin formation and jaundice[36,37].

Thalassemia patients will have an increased amount of bilirubin in the blood. This is due to the increased destruction of red blood cells (hemolysis) by the spleen. This is the main cause of hyperbilirubinemia. In the other hand liver cells are damaged as a side effect of iron overload, bilirubin can escape into blood stream [38.39]. The liver has the capacity to conjugate and excrete over 3000 mg of bilirubin per day; where as the normal production with covers pounding increase in conjugation and secretion of bilirubin diglucuonide . However, massive lysis of red blood cells (for example in patients with thalassemia, sickle cell anemia, pyruvate kinase, malaria) may produce bilirubin is excreted into the bile, the amount of urobilinogen entering the enterohepatic circulation is increased, and urinary urobilinogen is increase. Unconjugated bilirubin levels become elevated in the blood causing jaundice [40].

ALT is an enzyme found primarily in the liver but also in the heart and other tissues, it is more useful in diagnosing liver function than SGOT [41].

The mean ALT activity in thalassemic patients was reported to be increase and this increase in ALT was generally transient and occurred more commonly in patients with hepatitis C [42].

ALT activity was elevated in all thalassemic patients, which is due to the symptoms of liver damage [43]. Transfusional iron overload occurs with severe conditions that fulfill this criteria include thalassemia major.

β-thalassemia major is associated with varying degree of liver damage which causes the elevated plasma transaminase activities in those patients .

Hepatitis C virus (HCV) infection is a major cause of chronic liver disease.

Among patients with chronic infection 5% - 20% have been reported to develop cirrhosis, thalassemia major, other liver diseases, clinically identified cirrhosis, contraindication for liver biopsy. The elevation of serum alanine aminotransferase (ALT) levels in prediction of severity of liver injury in patients with chronic hepatitis C is debated [43]

AST is an enzyme found primarily in the liver, heart, kidney, pancreas, and muscles, elevated in tissue damage especially heart and liver, decreased levels can be found in vitamin B deficiency and pregnancy.

Liver fibrosis and cirrhosis are well known complications of thalassemia .Transaminases are expressed as multiplied by the upper level of the normal range to identify the role of iron overload in the natural history of liver fibrosis [44].

Alkaline phosphatase is an enzyme found primarily in bone and liver .Abnormalities can reflect increased activity of the obstruction to bile flow in the liver. Among the liver diseases, hepatitis, cirrhosis, cholestasis, cholecystitis, cholangitis, fatty liver and liver tumor can cause elevated liver enzymes and alkaline phosphatase along with liver diseases.

Elevation of serum alkaline phosphatase concentration in patients with diabetes mellitus has been observed for several years, but the source and reasons are unknown [45]

The most common treatment for all major forms of thalassemia is blood transfustions. Blood transfusion therapy, although it is lifesaving treatment, is expensive has many risks', for example carries a risk of transmission of viral and bacterial diseases .It also leads to excess iron in blood (iron overload) which can damage, liver, pancreas , heart and other parts of the body. To prevent damage, iron chelation therapy (desferrioxamine). [46].

The iron is stored in the liver as ferritin, elevated iron stores, reflected in elevated plasma ferritin levels. The patients in spite of taked (desferrioxamine) showed this elevation in iron and ferritin levels[47,48), and that's lead to damage for pancreas and liver.

Conclusion: This study showed that iron must be kept from high values in patients with β-thalassemia major in order not to destroy pancreatic cells and result diabetes type 1.The patients with β-thalassemia major were having chelating therapy (desferrioxamine), but in spite of this, serum iron levels were high, therefore we suggest using another chelating therapy more active and useful since (desferrioxamine) not helpful.

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دراسة مقارنة بين مستويات الحديد واضرار الكبد في مرضى فقر دم البحر الابيض المتوسط ومرضى فقر دم البحر الابيض المتوسط المصابين بداء السكري النوع الاول في العراق

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الخلاصة:-

هذا البحث يصف دور الحديد المتراكم وبعض الدوال الكيموحيوية في مصول الدم لمرضى فقر دم البحر الابيض المتوسط ومرضى فقر دم البحر الابيض المتوسط المصابين بداء السكري النوع الاول مقارنة بالاصحاء.

اجريت هذه الدراسة على ٣٠ شخصا" مصابين بمرض فقر دم البحر الابيض المتوسط مع داء السكري النوع الاول و٣٠ شخصا " مصابين بمرض فقر دم البحر الابيض المتوسط كمجموعة سيطرة مرضية و ٣٠ شخصا" من الاصحاء كمجموعة سيطرة طبيعية ،تتراوح اعمار المجاميع المدروسة بين (١٢-٣٥) سنة وجميع المرضى يتناولون العلاج الكلابي (الديسفرال).

وتم القاء القليل من الضوء على علاقة الترابط بين الحديد والفرتين وبعض الدوال الاخرى (الكلوبيولينات المناعية ،دوال وظائف الكبد ،الالبومين والبروتين الكلي) في مجاميع المرضى.

وقد اشارت النتائج الى وجود زيادة معنوية في نسبة الحديد والفرتين ووظائف الكبد والكلوبيولينات المناعية في مجاميع المرضى مقارنة مع الاصحاء ،بينما وجد نقصان معنوي في نسبة الالبومين والبروتين الكلي في مجاميع المرضى مقارنة مع الاصحاء.