Investigating Some Of Blood Parameters In Women With B-Thalassemia

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Abstract. The current study was conducted in the Department of Life Sciences - Faculty of Science - University of Qadisiyah in cooperation with Maternity Hospital and Educational Children and the Center for Hematology in Diwaniyah for the period from 1/10/2017 to 1/4/2018. Fifty (50) samples were taken from women aged 15 to 35 years, and were divided into two groups: 30 women with thalassemia (experimental group) and 20 women without thalassemia (control group). Two (2) milliliter were taken from thalassemia-treated women and 2ml from healthy women for the purpose of conducting blood tests for phytoplankton, blood groups and the level of ferritin in the body. The results of the present study showed a significant decrease (P <0.01) in all blood parameters, hemoglobin, blood corpuscular, corpuscular volume, mean corpuscular hemoglobin in corpuscular and mean corpuscular hemoglobin contraction, compared with control group. Chemo-biological tests like ferritin testing confirmed significant ferritin increase by P<0.01 for the experimental group compared to the control group.

Keywords: Thalassemia Hemoglobin, Ferritin

Introduction

Thalassemia is a genetic disorder that occurs in the gene responsible of the synthesis of globin in red blood cells, resulting in a malfunction of one or more of the chains of the chlorine, and thus leads to failure of manufacturing hemoglobin, which is responsible of transporting oxygen to various parts of the body. There are two types of thalassemia, α and β. β-Thalassemia is the most common genetic blood disease that causes β+ deficiency or the absence of β° for the manufacturing of β-globin chains within the quadruple hemoglobin chains consisting of α and β (1).

The disease spreads in the Middle East, the Arab world and Asia as about 1.5% of people (90-80 million) carrying Beta Thalassemia Major (BTM). The annual cases are estimated to be about 60,000, which were the result of a mating between the carriers or the infected ones. Frequent blood transfusion is a necessary process to sustain the patient's life, but over time it leads to the accumulation of ferritin on the essential organs. The accumulation of ferritin is one of the most important results of recurrent blood transfusions and other complications due to the increased occurrence of Reactive Oxygen Species (ROS) Oxidative Stress (OS), which are working to oxidize fat in cell membranes and organelles, resulting in degradation of cell functions and the destruction of DNA (Deoxyribo Nuclic Acid).
The process of ferritin release from hemoglobin due to thalassemia and associated red blood corpuscle (ROS) or apoptosis resulting in free radicals, increases the ROS and thus increases the effectiveness of oxidative stress. Also, the accumulation of ferritin directly affects the hypothalamic, hypothalamus and female reproductive organs. It also has an indirect effect on the liver and pancreas and works to undermine the metabolic processes of hormones and antioxidants alike. Hence the importance of oxidative stress, which is an important mechanism in the development of some diseases, including the great thalassemia BTM (4).

Methods

The present study has been carried out at the Women's and Children's Hospital and the Hematology Center in Diwaniyah during period extended from 1/10/2017 to 1/4/2018. Thirty samples of women with Thalassemia major and 20 samples of women Slims as control group, the age of states 15-35 years.

Collection of samples

Samples were collected from Thalassemia Center in Diwaniyah as follows:

For the purpose of conducting blood tests and ferritin stock, 2ml of blood from thalassemia-treated patient were taken and the tests were performed directly in the Hematology Department and the Biochemical Department.

As for the control group, 20 healthy cases were sampled from the Faculty of Science - University of Al-Qadisiya from thalassemia free women and in the same way as the patient samples were collected.

Physiological Blood Parameters Tests

For the purpose of estimating MCHC, MCH, MCV, RBC and Hb, periodic tests performed for patients were followed by automated analysis. Abbot ruby was adopted for the purpose of obtaining CBC. The instructions of the manufacturer were adopted for the purpose of obtaining the required results and as follows:

1. Blood samples from women with thalassemia were used in the EDTA tube
2. Blood samples were mixed well by the regurgitation device
3. EDTA tube was put on a special platform at the front of the device
4. start key was pressed
5. The amount of about 2 microliters of blood was taken by very thin tubes where the blood enters special chambers for the purpose of isolating each type of blood cells in a dedicated chamber
6. Special sensors calculated the number of cells passing through the stream
7. The results are printed after they appear on the monitors

Results and Discussion

Physiological Blood Parameters Tests

Table 1.1 and Figures 1-1, 1-2, 1-3, 1-4 and 1-5 refer to some physiological blood parameters that include MCHC, MCH, MCV, RBC, and Hb in the group of patients compared to the control group.

The results of the statistical analysis showed a significant decrease (P <0.01) for all blood parameters studied in patients with major thalassemia type β compared to the control group.
Table 1-1: Levels of MCHC, MCH, MCV, RBC, and Hb in a comparison between the experimental and the control group

| Group       | Hb(g/dl) Mean± SE | RBC(10⁶ cell /ml) Mean± SE | MCV(fl) Mean± SE | MCH (pg) Mean± SE | MCHC(g/dl) Mean± SE |
|-------------|------------------|----------------------------|------------------|------------------|---------------------|
| Control     | 12.9±0.12        | 3.96±0.8                   | 85.08±61         | 29.64±0.22       | 33.42±0.28          |
| Experimental| 7.83±0.25        | 3.20±0.09                  | 7.35±0.71        | 23.88±0.28       | 28.64±0.37          |

Figure 1-1: The Impact of Major Thalassemia Infection on Hb

Figure 1-2: The Impact of Major Thalassemia Infection on RBC

Figure 1-3: The Impact of Major Thalassemia Infection on MCV
Noteworthy, (5) and colleagues (2014) have shown that RBC in thalassemia patients has a severe defect in hemoglobin chains, either to be free of hemoglobin B or containing beta chain, but deficient in β+ formation. This is usually in the formation stage of Erythropoiesis, which is the pathway that produces RBC from the Hematopoiesis stem cell in the bone marrow and it includes several stages, most notably of which is differentiation. In both cases $^0\beta$ or $\beta+$, the sovereignty will be for $\alpha$-type chains, i.e., productivity will be in favor of alpha $\alpha$ compared to $\beta$. This surplus of alpha chains will no doubt accumulate in red blood cells, creating an unequal red blood cells that are unable to carry large amounts of oxygen all over the body.

One of the most important results of the study of (6) (2005) is that the rapid death of RBC occurs due to imbalance in the chains of hemoglobin, as red blood cells lose life in 120 days less than the normal duration of death of red blood cells, leading to bone marrow inability to compensate the missing Of RBC, i.e., the broken cyst of red blood cells is higher than that of newly formed cells. As a result, we would have deformed bones due to increased Erythropoiesis processes and osteoporosis due to the depletion of the components of the spinal cord in the double-reactivity of the pellets (7).

Also, (8) explained that the cause of increased early death of pellets in the polychromatophil stage in cellular differentiation in the bone marrow is partly due to the accumulation of alpha chains at this stage (9). Additionally, (10) indicates that the accumulation of alpha chains with ROS increases leads
to changes in the cell membrane and its stability. This leads to damage to the cell in general, and thus the breakdown of peripheral blood cells.

There is some evidence that oxidation of membranes by ROS plays an important role in the regression of the physiological condition of thalassemia patients. That the increase of ferritin and its stock works to generate ROS that breaks proteins, fats and amino acids into the cell membrane and the organs. ROS activation of programmed cell death leads to accelerated cell death in Erythropoiesis stages in the bone marrow (11).

The amount of ferritin generated by the large number of blood transfusions or RBC, which will spread the contents of the cell from the ferritin to the bloodstream, will lead to a series of exaggerated self-oxidative reactions that drain potential to the rest of the body's various cells and tissues (12).

Results of (13) showed that the abnormal condition of thalassemia patients is closely related to the reduction of Hb, which is associated with decreased red blood cell count and low MCV, MCH and MCHC, leading to the suffering of thalassemia patients from chronic acute anemia due to low oxygen content the blood.

**Ferritin Level**

The results of the current study of ferritin levels showed a significant increase of p <0.01 in women with thalassemia compared to the control group as shown in Table 1-2 and Figure 1-6.

| Group      | Ferritin Mean ng/ml ± SE |
|------------|--------------------------|
| Control    | 122.9 ±12.52             |
| Experimental | 5415.3± 3424.46         |

**Figure 1-6:** The Impact of Major Thalassemia on Ferritin Level

The increase of ferritin in patients with major thalassemia was attributed to the accumulation of ferritin as a result of frequent blood transfusions on patients. Ferritin is the iron stock that is excess of
the body's need, which is generated by the continuity of transfusions of patients, may be every 10 days or less.

We can imagine the amount of iron that is excess of the body's need, which iron excretions, taken as treatments for patients, may not be removed from the body.

Many blood transfusions come in order to maintain the level of hemoglobin at an appropriate level for the purpose of surviving almost normal, as the level of hemoglobin remains lower than 8g/dl, which is the result of this study and the previous studies as well. This causes the patient to suffer from fatigue and dizziness, which makes some patients visit, even before their time, health centers for the tests and then conduct blood transfusion, and return every ten days then the consequent would be surplus iron stock in the form of Ferritin. The lack of regularity in receiving appropriate treatments or even not taking treatments completely are of devastating consequences (15).

Excess iron can stimulate fat oxidation and generate high-impact oxidants on blood cells, thereby reducing the antioxidant properties of catalase in patients (16).

Also (16) shows that the increase in iron is due to the large number of transfusions of patients due to the urgent need for this, as excess iron works to increase the accumulation of iron that urges oxidative stress, which plays a role in the depletion of most antioxidants in the cells, up to the level of oxidants that damages the membranes and organelles of the body (17).

Additionally, (17) indicates that increased iron may increase the possibility of oxidation of membranes of red blood cells, and that enzymatic antioxidants in particular protect Erythrocyte from oxidative stress (OS), and even considered enzymatic antioxidants the first line of defense against this condition.

There is a significant increase in the absorption of iron in the intestines, whether in food or from frequent transfusions of patients. Excessive elevation of ferritin is a sign of thalassemia injury without iron deficiency anemia (19).

Moreover, (20) notices that the blood transfusion provides about 1mg of iron for every 1ml of blood. This will weaken the mechanism of iron removal from the body, and there is a high surplus in ferritin due to the increased absorption of the small intestine for iron, with the need to confirm the study of (13) who said that what the body actually needs is 1-2 milligrams of iron every day.

The study of (4) shows that the inefficient treatment of iron and iron accumulations can be the beginning of other diseases as a result of the breakdown of oxidants of the membranes of red blood cells, which are in the stages of formation (such diseases are called secondary diseases).

Increased iron accumulation is one of the most common complications affecting thalassemia patients because it works to increase free stems, and this is accompanied by tissue breakdown and depletion of internal antioxidants (21).

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