

Iron, Ferritin and Hepcidin Levels in Patients with Iron Deficiency Anemia

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ABSTRACT

This study was conducted to identify the role of some blood parameters, levels of some hormones, proteins, and cellular kinetics that have a role in iron transport and storage, in addition to their relationship with each other and with blood and sex parameters for patients with severe and moderate iron deficiency anemia, and to compare the parameters with healthy people. The study period lasted for the period from (December 2020 until March 2021) The presence of iron deficiency anemia was confirmed by examining the blood picture and the criteria of hepcidin and ferritin. The results showed a significant decrease in the concentration of hepcidin, ferritin and iron in patients compared to the control group.

Keywords- iron deficiency anemia, hepcidin, ferritin, iron.

Iron acquisition mechanisms are tightly regulated by hepcidin, a peptide hormone that is primarily synthesized by the liver. It has a role as an acute-phase reactant that modulates fluctuations in plasma iron levels caused by absorbing enterocytes and macrophages in the spleen by binding to and degrading ferroportin, which releases iron from cells (3).

Hepcidin expression increases in response to elevated iron levels while it is inhibited by erythropoiesis, iron deficiency and tissue hypoxia in response to signals originating in bone marrow, liver and possibly muscle tissue and adipocytes (4).

In the event of an iron deficiency, the transcription of hepcidin is inhibited, which facilitates the absorption of iron and the release of iron from the body's stores. In addition to increasing intestinal uptake of iron from the lumen of the alimentary canal (5). Iron is an essential component of many basic biological processes. It is a transition metal that can readily donate and accept electrons to participate in redox reactions that are essential for a number of basic biological processes (6). In humans, iron is incorporated into proteins as a component of heme (for example, hemoglobin, myoglobin) iron-sulfur groups (for example, respiratory complexes I-III, coenzyme Q10, mitochondrial aconitase, DNA primase), and other functional groups (eg, hypoxia inducible factor prolyl hydroxylases) These iron-containing proteins are required for cellular and vital organ functions including oxygen transport, mitochondrial respiration¹², intermediate and exogenous metabolism, DNA replication and repair, and cell signaling (6) The percentage of the body's normal iron content is estimated at 65% in hemoglobin, 3-5% in myoglobin and 0.5% in heme enzymes. The rest of the iron content is 30% in the form of iron stored in (liver, spleen and bone marrow) in the form of Two ferrites and hemosiderin, as the normal ratio of iron stored in the form of two ferrets to that stored in the form of hemosiderin is 1:3(7) Ferritin is the main chemical compound of stored iron. Ferritin is mainly in the cytoplasm of endothelial reticulum cells and hepatocytes, and a few of them are in epiblasts in the bone marrow (8) Ferritin It is a major iron storage protein, essential for iron homeostasis and involved in a wide range of physiological and pathological processes. In clinical medicine, serum ferritin is mostly used as a

I. INTRODUCTION

The human body can conserve iron in a number of ways including recycling iron after red blood cells are broken down and retaining iron in the absence of an excretion mechanism. However, iron absorption is limited to 1 to 2 mg per day and most of the iron required per day is about 25 mg supplied through recycling by macrophages that phagocytose erythrocytes Controlled by hepcidin, a hormone that maintains iron in the entire body Within normal ranges, avoiding both iron deficiency and excess (1).

Iron is essential for the various activities of the human body especially in building hemoglobin. Iron deficiency anemia is a condition in which there is too little iron in the bloodstream. This type of anemia is more common in adolescents and premenopausal women. Prolonged bleeding, internal bleeding from the gut, or donating a large amount of blood can contribute to this disease Low iron level can result, which leads to anemia, for various reasons. Such as pregnancy or childhood growth spurts, long menstrual periods, poor iron absorption, bleeding from the gut, nutritional factors (iron deficiency or a restricted diet), deficiency of certain vitamins (folic acid and vitamin B12) and kidney bleeding, red blood cell problems bone marrow problems (2).

marker for iron stores in the body. In cases of iron deficiency and overload, serum ferritin has a critical role in both diagnosis and management. High serum ferritin is associated with CHD, malignancy, and poor outcome after stem cell transplantation (9).

II. MATERIALS AND WORKING METHODS

Then the patients were divided into groups depending on the type of anemia (severe and moderate) and the group of healthy people, the first group included a control group, which included 10 healthy people, the second group included 30 people with moderate iron deficiency anemia, and the third group included 30

people with anemia Severe iron deficiency. After confirming the patients' conditions, 5 ml of patients' blood were drawn, of which 2 ml were for testing blood parameters, and 3 ml of serum was isolated from them for hormonal and immunological tests for cellular kinetics.

III. STATISTICAL ANALYSIS

Statistical analysis of the current study was carried out using spss program (16 versions) through the T test for variance at the 0.05 probability level.

IV. RESULTS AND DISCUSSION

Table 1: Shows the levels of hepcidin, Ferritin and Iron in the sera of patients with iron deficiency anemia and control.

Immune variant	Control group number (30)	Medium number of iron deficiency anemia patients' group (30)	Severe iron deficiency anemia group (30)	Statistical significance (p value)
	Mean ± SD	Mean ± SD	Mean ± SD	
Hepcidin	5.031± 1.050	3.418±1.946	1.4227±0.686	≤ 0.01**
Ferritin	126.89±9.306	36.416 ± 7.629	7.465±2.418	≤ 0.01**
Iron	15.901±2.550	9.8900±1.011	4.3593±1.35	≤ 0.01**

There are significant differences according to the value of (P<0.05)

Hepcidin levels:

The results of our study indicated that there was a significant decrease in the level of hepcidin in patients with anemia. Moderate and severe iron deficiency, and the results of our study agreed with (10) by recording it. Significant significant decrease in the value of hepcidin in the group of patients with iron deficiency anemia Where it recorded 6.01 ± 2.83 ng/ml compared with the control group 24.96 ± 9.09 ng/ml The researcher attributed the decrease in hepcidin to the fact that hepcidin is a major regulator of iron balance. Systemic, where expression is stimulated by inflammatory stimuli and suppressed in iron deficiency, where hepcidin levels were positively correlated with IL-6 levels in patients with pure ACD, proving the role of IL-6 in stimulating hepcidin production, and hepcidin levels were correlated with positively correlated with serum ferritin concentrations and negatively correlated with STfR in both patients pure ACD and IDA. Also, hepcidin levels were negatively correlated with TIBC in both Patients with pure ACD and IDA, serum hepcidin levels were assessed as a diagnostic test for ID and IDA in patients.

The results of our study agreed with (11) in his study on mothers suffering from poverty. Iron deficiency blood with non-infected patients. Low levels of hepcidin are noted in the deficiency group. Iron and anemia group recorded 76.6 ± 22.7 µg/l, while the group of mothers was not the female patients recorded 92.4 ± 10.5 µg/L, indicating that hepcidin is the regulatory hormone the

key to iron balance, preventing iron absorption or reuse by binding to ferroportin, the main source of iron, which leads to its absorption and breakdown, thus reducing. Export of iron to plasma, where the most important regulating factor of serum hepcidin is anemia and weakness. Iron, the normal serum hepcidin concentration in healthy women ranges from 12 ng/ Ml or mg/L to 746 ng/ml or mg/L. hepcidin is also regulated by hepcidin gene expression via cytokines. Inflammatory iron and iron are expressed through the JAK/STAT and BMP/SMAD pathways. hepcidin through activation of the transcription factor signal transducer (STAT3) by cytokine. Inflammatory action of interleukin (IL-6) (12) in which IL-6 and TNF-A can facilitating the expression and regulation of hepcidin to mediate homeostasis of iron metabolism by regulating the pathway. STAT3 hepcidin signaling and leads to increased expression of TfR224 (13). IL-10 can also downregulate hepcidin expression via inhibition of inflammatory factors IL-6 and TNF-A. Thus suppressing STAT3 expression and improving the balance of iron metabolism (14).

Ferritin levels:

The results of our current study indicated a significant decrease in ferritin concentration in patients with severe and moderate iron anemia when compared to the control group, and here our results agreed with the researcher (15) A significant decrease in iron deficiency anemia was recorded in women with iron deficiency anemia of 138.9 ng/ml, while the non-affected women

recorded 200.7 ng/ml, it was well established that serum ferritin is an indicator of the level of iron deficiency in the body. Indicates low iron stores in newborns in addition to it. The results of our study agreed with (16) by recording a significant decrease in the group of people with iron deficiency anemia (IDA 6.5 ng/ml compared with the control group 70.8 ng/ml, ferritin and sTfR complement each other, and indicated that ferritin reflects stored iron status, while sTfR corresponds to tissue iron supply. The soluble transferrin receptor (sTfR) is a marker of iron status in humans that is proportional to elevated sTfR with tissue iron deficiency, reflecting the body's demand for iron for erythropoiesis. To assess iron status in cases where serum ferritin was not recorded. An abrupt rise in sTfR level usually coincides with iron storage depletion and decreased serum ferritin, but elevations of sTfR may occur for other reasons, such as erythrocytic hyperplasia (due to hemolytic anemia or megaloblastic thalassemia), hypoxia, malignancy, and physiological stress (such as in pregnancy, if the sTfR remains low in the face of iron deficiency, chronic renal failure or a hypoproliferative state (eg, aplastic anemia) is likely.

Iron levels:

The results of the current study showed a decrease in iron concentration levels in patients with moderate and severe iron deficiency anemia compared to the control group, and the study agreed with (16). It recorded a decrease in iron levels in the blood serum of people with iron deficiency anemia IDA and recorded 15 µg/dL, as well as a decrease in levels in patients with chronic iron deficiency anemia ACD 36 µg/dL compared to the control group 111 µg/dL. The iron deficiency anemia ADI is due to iron deficiency. Without enough iron, the body cannot produce enough of a substance in red blood cells that enables it to carry oxygen (hemoglobin) and thus there is fatigue and shortness of breath, and one of the main results is in chronic ACD anemia, iron uptake and retention within certain cells is increased, resulting in decreased amounts of functional iron available for hemoglobin production. The study agreed with (10). It recorded lower levels of iron in IDA patients (35.37 ± 10.17 µg/dl) compared to the control group (92.22 ± 10.02 µg/dl).

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