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Investigate the role of some adipose tissue hormones and IL-1β in a sample of patients with β-thalassemia.

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Abstract :

A cross-sectional study was carried out to evaluate the adipose tissue hormones in the sera of patients with β-thalassemia .The study was conducted on 90 blood samples, (group 1) includes 50 patients' samples with β-thalassemia, while the control group (group 2) includes 40 samples. The ages of both groups range from 20 to 32 years. Patient samples were collected from Baghdad / Center Hematology and Bone Marrow Transplantation/Medical City. Visfatin, omentin, Adiponectin, Hepcidin, IL-1β, superoxide dismutase (SOD), Ferritin, Iron were estimated and evaluated in the serum of both groups (patients and control), The results indicates significant increasing in the level of omentin, hepcidin, IL-1β, SOD, ferritin and iron ($P \leq 0.001$) in patients with β -thalassemia compared with control group, while the level of visfatin was significantly decrease ($P \leq 0.001$) in patients compared with healthy group. From the current study, it was concluded that the infection with β-thalassemia may elevate the adipose tissue hormones and some interleukins, such as IL-1β level in the sera of patients.

Key words: β-Thalassemia, adipose tissue hormone, hepcidin, IL-1β, Visfatin.

التحري عن دور بعض هرمونات النسيج الدهني والانترليوكين IL-1β في عينة من مرضى التلاسيميا المنجلي

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مستخلص:

دراسة مقطعية أُجريت لتقييم هرمونات النسيج الدهني في مصل دم مرضى الثلاسيميا من النوع β. شُملت الدراسة 90 عينة دم، حيث تكون المجموعة 1 من 50 عينة لمصابين بمرض الثلاسيميا β، بينما المجموعة الضابطة (المجموعة 2) من 40 عينة. تتراوح أعمار كلا المجموعتين بين 20 و32 عاماً. جُمعت عينات المرضى من بغداد/ المركز الثقني للدم والتبرع بالنخاع العظمي/ مدينة الطب. تم تقدير وتقييم في الدم المصلوي كل من فيسبتين، أومينتين، أديبونيكتين، الهسبيدين، IL-1β، سوبر أكسيد موزِد (SOD)، الفيريتين، والحديد. وتشير النتائج إلى زيادة معنوية في مستويات الأومنتين، والهسبيدين، وIL-1β، وSOD، والفيريتين، والحديد لدى مرضى الثلاسيميا β مقارنةً بالمجموعة الضابطة ($P \leq 0.001$)، بينما انخفضت مستويات الفيسفين بشكل significati-vo في المرضى مقارنةً بمجموعة الصحة ($P \leq 0.001$). من هذه الدراسة، استنتج أن الإصابة بمرض الثلاسيميا β قد ترفع من مستوى هرمونات النسيج الدهني وبعض الإنترلوكينات، مثل IL-1β، في مصل الدم للمرضى.

الكلمات المفتاحية: β-Thalassemia, adipose tissue hormone, hepcidin, IL-1β, Visfatin.

1. INTRODUCTION

Thalassemia is an autosomal recessive inherited blood disorder that has a lifelong impact on both patients and their families, as people with thalassemia are unable to produce hemoglobin in the body at the normal rate, which leads to their anemia [1]. Thalassemia is widely spread in Iraq, where the number of thalassemia patients is about 15,000. Thalassemia is also a major health problem and has a large burden on health services in Iraq, as pre-marital screening for thalassemia is of utmost importance to educate couples about the danger of transmitting the disease to their offspring and the complications of the disease [2].

The pathogenesis of thalassemia is mainly linked to the formation of inefficient red blood cells, which leads to hemolysis, causing oxygen shortage to the body's tissues. Thus, an imbalance in the α/β-globin chain ratio results in anemia. In chronic hemolytic conditions, red blood cells are malformed, impairing their ability to transport oxygen, and insufficient production of these two chains leads to lifelong anemia starting from childhood [4].

The hormone Omentin-1 is one of the important adipokines. It is known as a major visceral fatty substance. It is a glycoprotein consisting of 295 amino acids and linked to oligosaccharides. Physiological concentrations of Omentin-1 in humans range between 100-800 ng /ml, as it is known to be expressed or produced in many physiological conditions, including obesity and insulin resistance, as well as inflammatory conditions [5, 6].

Adiponectin is one of the hormones secreted from adipose tissue. It is one of the most abundant proteins in the blood circulation. Its concentration in blood plasma is 5-30 mg/mL. It plays an important function in regulating metabolism and preserving energy in the body. It also plays a role in regulating blood sugar and fats, and role in inflammatory response [7, 8].

Hepcidin is the main regulator of iron balance in the body within the normal range, as it controls the entry of iron into the blood circulation through its effect on the absorption process by intestinal cells. The hormone hepcidin directly inhibits the iron source protein

ferroportin, which is the protein that transports iron out of cells; however, a hepatic bactericidal protein is rich in cysteines, and it has a role in serum as a diagnostic tool for measuring iron overload in patients with thalassemia [9, 10].

Interleukin-1 beta is a pro-inflammatory cytokine, it is a major regulator of inflammation by controlling inflammation and it is a substance known to be released by cells of the immune system [11]. Interleukin-1 beta serves as a pivotal mediator in numerous inflammatory reactions during the acute phase. Its functions encompass the activation of inflammatory cells, stimulation of lymphocytes, and induction of hepatocytes to produce acute-phase proteins. Typically elevated under normal circumstances, Interleukin-1 beta levels in blood serum remain undetectable during mild inflammation. However, its escalation is implicated in various diseases, particularly in the elderly population, including certain lymphomas, Alzheimer's, and thalassemia [12].

Ferritin serves a crucial role in regulating the absorption of dietary iron, acting as a preventive measure against iron overload. This function is particularly beneficial in reducing infection risks among thalassemia patients and mitigating oxidative stress [13]. Furthermore, ferritin serves as a diagnostic tool for identifying iron deficiency anemia, while primarily preserving iron in a soluble, non-toxic state. Functioning as an antioxidant, ferritin plays a vital role in sequestering potentially harmful iron, especially when endogenous antioxidants fail to alleviate oxidative stress, as observed in conditions like thalassemia [14]. In such cases, exogenous antioxidants can bolster the body's antioxidant defense system. Additionally, the relationship between adipose tissue hormones and blood disorders, including beta thalassemia, underscores the interconnectedness of various physiological processes.

The present study seeks to assess specific adipose tissue hormones and biochemical variables among individuals diagnosed with thalassemia.

2. Materials and Methods

Experiment design

The study was structured by procuring 90 blood samples, comprising both males and females, divided into two distinct cohorts. The initial group comprised 40 blood samples obtained from healthy individuals, while the second group comprised 50 blood samples sourced from patients diagnosed with beta thalassemia. The age range of the samples spanned from 20 to 35 years and was collected from the patients at the Baghdad Center for Hematology and Bone Marrow Transplantation, Medical City, during the period between December 15, 2023, and February 20, 2024. Following sample collection, the levels of physiological and immunological variables were assessed.

Methods

The present study includes the determination of:

- The levels of Visfatin, adiponectin, Omentin, Hepcidin, and IL-1β in the blood serum were determined through the ELISA method, employing dedicated measuring kits for each hormone. These kits were sourced from the manufacturer Monobind Inc, USA, and utilized as per the manufacturer's instructions.
- Ferritin levels in the blood of both thalassemia patients and healthy individuals were quantified using the enzyme-linked immunosorbent assay (ELISA) method. This approach followed the protocols and instructions outlined by the Chinese company HCUSABIO [15].
- Superoxide dismutase enzyme activity was determined utilizing the photochemical reaction method, following the specific procedure outlined by the researcher [16].

Statistical analysis

The obtained results underwent statistical analysis utilizing a T-test within a statistical analysis program with significance set at a probability level of ($P \leq 0.001$).

3. RESULTS

Table 1 and Figure 1 below illustrate the assessment of physiological variables, including Omentin, Visfatin, adiponectin, Hepcidin, IL-1β, SOD, Ferritin, and Iron, in the blood sera of both patients and healthy individuals.

Table 1: Comparison of physiological variables between patients and healthy people

Groups Parameters	Mean ± SD	
	Control (n=40)	Patients (n=50)
Omentin-1 (pg/ml)	25.12±4.2	70.32±5.54
Visfatin (ng/ml)	1.13±0.14	0.79±0.2
Hepcidin (ng/ml)	360.19±218.01	995.03±505.12
(ng/ml) Adiponectin	33.09±19.11	59.13±18.9
IL-β1 (pg/ml)	6.12±0.9	23.32±4.71
SOD IU/ml	17.32±0.07	41.52±0.68
(ng/ml)Ferritin	177.31±117.91	376.01±135.21
Iron (µmol/L)	16.14±4.85	35.61±9.22
$P \leq 0.001$		

The current study reveals a noteworthy increase in levels of Omentin-1, adiponectin, hepcidin, IL-1β, SOD, ferritin, and iron among patients compared to healthy individuals. Conversely, there is a marked reduction observed in Visfatin hormone levels in patients compared to their healthy counterparts, with statistical significance at the level of probability $P \leq 0.001$, as depicted in Figure 1.

DISCUSSION

Beta thalassemia stands out as a prevalent and perilous hereditary condition, distinguished by irregular hemoglobin. Typically, it manifests alongside symptoms of hemolytic anemia and carries a substantial global mortality rate of 15% [17]. There exists a correlation between adipose tissue hormones and thalassemia, with several endocrine glands, including adiponectin and omentin, among others, being linked to patients with thalassemia [18].

The findings of the current study align with [19], who reported an elevation in omentin hormone levels in the sera of patients with β-thalassemia. This observation underscores the sig-

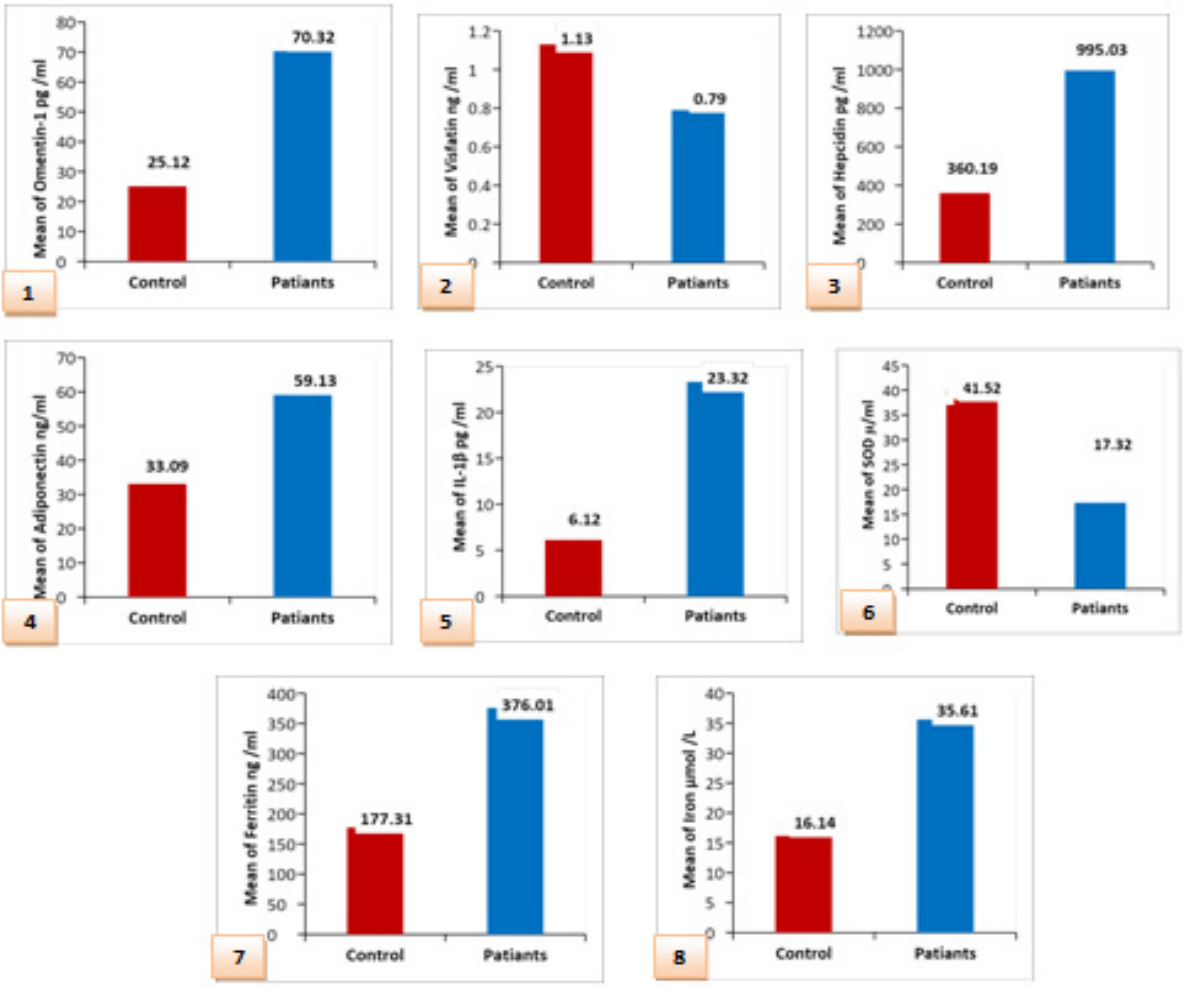

Figure 1:
1- Concentration of Omentin-1 in both groups; 2- Concentration of Visfatin in both groups3- ; Concentration of Hepcidin in both groups; 4- Concentration of Adiponectin in both groups; 5- Concentration of IL-1β in both groups; 6- Concentration of SOD in both groups; 7- Concentration of Ferritin in both groups; 8. Concentration of Iron in bothgroups.

nificant role of omentin in the pathophysiology of iron, as it serves as a receptor for lactoferrin, facilitating iron absorption in the intestines [20]. Consequently, it appears to contribute to the regulation of iron levels in patients, with elevated levels being associated with iron overload in individuals with thalassemia [21].

Furthermore, the findings of the current study contradict those of [22], who reported an elevation in visfatin levels

among patients with β-thalassemia in their study. Visfatin, a pro-inflammatory lipid predominantly expressed in visceral adipose tissue, has been noted to correlate with platelet count, implying a potential association with the progression of thalassemia [23]. Conversely, it was observed that adiponectin levels increase in patients with beta thalassemia, consistent with the results reported by [24], which documented an elevation in adiponectin concentration. This suggests that adiponectin may contribute to various vascular disorders and subsequent inflammation [24]. Thus, the inflammation associated with adipose tissue could potentially be linked to the development of beta thalassemia major (ßTM) [23].

The current study also revealed an increase in IL-1β concentration, aligning with previous findings cited [25]. Those researchers observed elevated IL-1β levels in beta thalassemia patients, suggesting that during anemia, the upregulation of inflammatory cytokine receptors on cell membranes could impact red blood cells, potentially contributing to thalassemia development.

Concerning hepcidin hormone levels, our study observed a notable increase in thalassemia patients compared to healthy controls, aligning with the findings of [26]. Hepcidin plays a pivotal role in regulating iron balance within the body, participating in various metabolic pathways of iron metabolism. By inhibiting ferroportin, hepcidin impedes the release of iron from intestinal absorption cells into the hepatic portal system, consequently diminishing iron absorption [27]. Its regulation involves administering iron supplements to replenish the body's iron stores, thereby aiding in infection and inflammation prevention, in addition to considering factors such as hypoxia, anemia, and iron deficiency. Furthermore, genetic alterations could potentially influence hepcidin synthesis in individuals with thalassemia, possibly instigated by inflammatory cytokines, prompting heightened synthesis [28].

Regarding ferritin levels, our current research revealed a significant increase in thalassemia patients compared to the healthy group, consistent with the findings of [29]. This elevation

may stem from the excessive accumulation of iron in the body, particularly within the bone marrow. Additionally, ferritin serves as a protein marker for iron in thalassemia patients who regularly undergo blood transfusions. The concentration of ferritin rises with the frequency of blood transfusions and non-adherence to iron chelation medications. The notable elevation in ferritin concentration in serum is primarily attributed to the multiple blood transfusions undergone by thalassemia patients, which are considered life-saving treatments. However, this also leads to increased iron levels and its deposition in tissue organs such as the liver, inducing toxicity [30].

The SOD enzyme exhibits a notable increase in the serum of thalassemia patients compared to the healthy group, in accordance with the findings of [31], which reported elevated levels of superoxide dismutase enzyme in their studies. This elevation in SOD levels may be attributed to its active role in scavenging superoxide radicals, thereby forming hydrogen peroxide and protecting cell membranes from damage. Additionally, increased activity of red blood cells (RBCs) and a higher percentage of RBCs in infected thalassemia patients could contribute to this rise [32]. Thalassemia patients are particularly vulnerable to oxidative stress due to their elevated iron levels. Thus, maintaining antioxidant systems could be beneficial in shielding them from severe complications of the disease, such as anemia. Oxidative damage to red cell progenitors in thalassemia patients can accelerate cell death and render the blood formation process inefficient. Oxidative stress appears to be the primary mechanism driving pathological changes in thalassemia patients [33].

Iron overload in thalassemia primarily arises from increased intestinal iron absorption, either due to ineffective blood transfusions or regular blood transfusions, as the human body lacks physiological mechanisms to eliminate the iron overload resulting from transfusions [34].

On the other hand, low iron levels align with the findings of Al-Hakeim et al., 2020 [35], who observed elevated total iron levels in thalassemia patients compared to healthy individuals. This

rise could be attributed to repeated blood transfusions and excessive iron absorption. Similarly, regarding serum iron levels, our study corroborates with Widyastiti et al., 2018 [36], who reported elevated serum iron levels following blood transfusions in thalassemia patients. A significant increase in blood iron levels also signifies severe anemia due to ineffective erythropoiesis, the primary cause of iron overload. Furthermore, iron overload resulting from low hepcidin levels, leading to increased iron absorption, as indicated by Neufeld (2010 [37], can be toxic to various tissues, including the liver, potentially resulting in fatalities.

CONCLUSIONS

Thalassemia, an autosomal recessive inherited blood disorder, exerts a lifelong impact on both patients and their families. Individuals affected by thalassemia are unable to produce hemoglobin at the normal rate, resulting in anemia. There exists a connection between thalassemia and adipose tissue hormones, where an increase in hormone levels has been observed. This association suggests that adipose tissue hormones could serve as crucial physiological indicators for detecting thalassemia.

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CONFLICT OF INTEREST

No conflict of interest.

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