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Evolution of Adipsin and Lipid Profile Among Patients with Beta-Thalassemia

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Abstract

β-thalassemia is an inherited blood disorder characterized by defective β-globin chain synthesis, leading to chronic hemolytic anemia and systemic metabolic alterations. This study evaluated lipid profiles and serum adipsin levels in 60 β-thalassemia patients compared to 26 healthy controls. Results revealed significantly lower total cholesterol and HDL-C levels in patients, alongside elevated triglyceride concentrations, indicating a consistent pattern of dyslipidemia. Additionally, serum adipsin levels were markedly higher in the patient group, suggesting a link between β-thalassemia and adipokine dysregulation. These findings reflect the broader metabolic impact of the disease and highlight the potential of adipsin as a biomarker for disease-associated metabolic disturbances.

Keywords β-thalassemia, dyslipidemia, adipsin, lipid profile, adipokines, metabolic disturbance, chronic anemia, biomarker.

تطور مستويات الأديبسين والدهون لدى مرضى بيتا ثلاسيميا

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الخلاصة : بيتا ثلاسيميا هو اضطراب دم وراثي يتميز بخلل في تخليق سلسلة بيتا غلوبين، مما يؤدي إلى فقر دم انحلالي مزمن واضطرابات أيضية جهازية. قيّمت هذه الدراسة مستويات الدهون ومستويات الأديبسين في المصل لدى 60 مريضًا مصابًا بثالاسيميا بيتا مقارنةً بـ 26 شخصًا سليمًا. كشفت النتائج عن انخفاض ملحوظ في مستويات الكوليسترول الكلي والبروتين الدهني عالي الكثافة لدى المرضى، إلى جانب ارتفاع في تركيزات الدهون الثلاثية، مما يشير إلى نمط ثابت من اضطراب شحميات الدم. بالإضافة إلى ذلك، كانت مستويات الأديبسين في المصل أعلى بشكل ملحوظ لدى مجموعة المرضى، مما يشير إلى وجود صلة بين بيتا ثلاسيميا واضطراب تنظيم الأديبوكين. تعكس هذه النتائج التأثير الأيضي الأوسع للمرض، وتُبرز إمكانات الأديبسين كمؤشر حيوي للاضطرابات الأيضية المرتبطة بالمرض.

كلمات مفتاحية : β-thalassemia, dyslipidemia, adipsin, lipid profile, adipokines, metabolic disturbance, chronic anemia, biomarker

Introduction

Thalassemia comprises a diverse group of inherited anemias caused by mutations that disrupt the synthesis of globin chain subunits in hemoglobin. These genetic defects result in reduced hemoglobin production and the accumulation of unpaired, insoluble globin chains, which impair red blood cell integrity. Consequently, this leads to ineffective erythropoiesis and chronic hemolytic anemia (Benz,2023).

Among the various forms of thalassemia, β-thalassemia is one of the most prevalent and clinically significant subtypes. It arises from mutations in the β-globin gene, which disrupt β-globin chain synthesis through diverse mechanisms, including impaired transcription, defective RNA processing, and translational defects. The imbalance between α- and β-globin chains exacerbates erythroid precursor apoptosis, leading to chronic anemia and necessitating lifelong medical intervention (Shang & Xu, 2017).

Beta-thalassemia syndromes encompass a group of inherited blood disorders marked by diminished or completely absent synthesis of the beta-globin chains (**Galanello & Origa**., 2010).

In severe cases, such as thalassemia major, profound anemia is often evident in the neonatal period. In contrast, thalassemia intermedia typically results in milder anemia, with some individuals remaining asymptomatic (**Haddad** *et al*.,2014). The severity of the disorder is directly linked to the number of gene mutations, with four gene defects in α-thalassemia and two in β-thalassemia (**Modell** *et al.*,2000). In Thailand, approximately 40% of the population carries α- or β-thalassemia genes, yet only 1% of affected individuals require regular transfusions for TDT (Panich *et al.*, 1992).

Thalassemia leads to anemia of varying severity, ranging from mild to life-threatening forms. Individuals of Mediterranean, Middle Eastern, African, and Southeast Asian ancestry are at an increased risk of being carriers of thalassemia-related genes (**Weatherall**, 1997).

Thalassemia constitutes a considerable global health burden, with its prevalence varying significantly across regions and populations. Historically concentrated around the Mediterranean, the disorder has now emerged as a worldwide health concern, affecting millions globally (**Kattamis *et al.*,** 2020). A thorough understanding of thalassemia epidemiology is essential for effective disease management and the strategic allocation of resources. The prevalence of thalassemia is notably higher in the Middle East, Asia, and Mediterranean regions compared to Europe and North America (**Musallam *et al*.,2020)**.

Thalassemia is an inherited blood disorder transmitted in an autosomal recessive pattern, requiring the inheritance of two defective gene copies—one from each

parent—for the disease to manifest (Angastiniotis & Lobitz, 2019). Individuals with only one abnormal gene copy (heterozygotes) are typically asymptomatic but can transmit the mutation to their offspring, thereby increasing the risk of thalassemia in subsequent generations.

This group of inherited anemias is primarily caused by mutations affecting the globin genes, particularly the β-globin gene, leading to diminished or absent synthesis of β-globin chains (Sundaresan *et al*.,2023). The clinical consequences of thalassemia can be profound, with severe forms necessitating lifelong blood transfusions and iron chelation therapy (Yousuf *et al*.,2022). Beyond the direct impact on patients, the disease imposes significant emotional, social, financial, and physical challenges on family members and caregivers, underscoring the far-reaching implications of this condition (Iheanacho & Okeke, 2023).

The pathophysiology of thalassemia is characterized by a disruption in the synthesis of these globin chains, resulting in an imbalance between alpha- and beta-globin production. This imbalance adversely affects the formation and functionality of red blood cells, ultimately leading to the clinical manifestations of the disease (Gluba-Brzózka *et al*.,2021).

Lipids and lipoproteins play a significant role in the pathogenesis of various clinical manifestations associated with thalassemia. These include disruptions in endocrine function, heightened susceptibility to infections, and vascular complications such as thromboembolism. Notably, the impact of these lipid alterations differs between patients with β-thalassemia major (β-TM) and β-thalassemia intermedia (β-TI) (Haghpanah *et al*.,2010).

Abnormal lipid profiles in thalassemia patients have been linked to increased carotid artery intima-media thickness and an elevated risk of premature atherosclerosis (Sherief *et al*.,2017). Various liver diseases, including hepatitis C virus (HCV) infection and iron overload (IOL), also significantly affect lipid synthesis and metabolism in β-thalassemia major (β-TM) patients. The use of iron chelators such as deferasirox (DFX) and deferiprone (DFP) further influences lipid metabolism (Brittenham,2011).

Adipokines, a group of bioactive molecules secreted by adipose tissue, play pivotal roles in various physiological and pathological processes. These molecules are integral to regulating metabolism, inflammation, immunity, cardiovascular homeostasis, and cancer pathogenesis. However, dysregulation of adipokine secretion and function is strongly associated with the development of obesity-related disorders, underscoring their clinical significance (Fasshauer & Blüher, 2015). Adipocytes are broadly categorized into two primary types: white

adipocytes and brown adipocytes (Mancuso.2016). White adipocytes primarily function as energy reservoirs, storing surplus energy in the form of triglycerides. Additionally, they secrete a diverse array of cytokines, including leptin, adipsin, adiponectin, omentin, tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), monocyte chemoattractant protein-1 (MCP-1), plasminogen activator inhibitor-1 (PAI-1), resistin, visfatin, and retinol-binding protein 4 (RBP4) (**Mattu & Randeva,2013)**. Adipokines play a pivotal role in modulating immune responses by regulating the production of pro- and anti-inflammatory cytokines, thereby influencing inflammatory pathways. Furthermore, they impact cardiovascular function through their effects on blood pressure, vascular tone, endothelial function, lipid metabolism, and thrombosis. Additionally, adipokines significantly contribute to the progression of atherosclerosis by modulating plaque stability, promoting smooth muscle cell proliferation, facilitating macrophage infiltration, and contributing to foam cell formation (Farkhondeh *et al.*, 2020). Adipokines also play a significant role in mental health disorders, including depression, anxiety, schizophrenia, bipolar disorder, and eating disorders. They influence brain function either by crossing the blood–brain barrier or by activating specific receptors on neurons and glial cells. Additionally, adipokines modulate mood, cognitive function, stress responses, reward pathways, and eating behavior by interacting with key neurotransmitters such as serotonin, dopamine, norepinephrine, and glutamate (Carvalho *et al.*,2014). Adipsin is a prominent adipokine that constitutes one of the primary proteins expressed by adipocytes (Cook *et al.*,1987). Initially identified in 1987, adipsin was later recognized as complement factor D, a critical component of the complement system (Xu *et al.*,2001). This protein plays a key role in amplifying the alternative complement pathway (Ansorge & Täger, 2014), where it participates in an enzymatic cascade that leads to the formation of the C5–C9 membrane attack complex and the release of anaphylatoxins, such as C3a and C5a (Lo et al., 2014). Primarily synthesized by adipose tissue cells (White *et al.*, 1992), adipsin has been shown to correlate with markers of obesity and glucose metabolism (Pomeroy et al., 1997). Notably, adipsin catalyzes the production of complement factor C3a, which has been demonstrated to stimulate insulin secretion in pancreatic β-cells. Reduced serum levels of adipsin are observed in patients with type 2 diabetes (T2D) and β-cell dysfunction (Lo *et al.*,2014). Additionally, adipsin enhances glucose uptake and promotes triglyceride synthesis in adipocytes (Litvinova *et al.*,2014).

Material and methods

The experiment was designed to examine specific biomarkers, including lipid profile parameters, and assess certain adipokinetic factors, such as Adipsin, in patients diagnosed with β-thalassemia. The study included 60 confirmed cases of

β-thalassemia, regardless of gender, recruited from the Thalassemia Center at Al-Karama Teaching Hospital in Baghdad, who formed the patient group. The control group consisted of 26 healthy individuals. The age range for both groups was between 20 and 40 years, with the study conducted from February to April 2025. Samples were collected in the morning from the study participants using disposable 10 mL syringes. The blood samples were then transferred into gel tubes specifically designed for serum separation. The tubes were left at room temperature or placed in a water bath maintained at 37°C until complete coagulation of the blood sample was achieved. Following coagulation, the tubes were centrifuged at 4000 rpm for 10 minutes to separate and obtain the serum.

The ELISA (Enzyme-Linked Immunosorbent Assay) technique is widely recognized in scientific research and medicine for detecting and quantifying specific compounds in biological samples. This method relies on the interaction between antibodies and antigens through an enzyme-linked immunosorbent assay (ELISA) process (British Society for Immunology, 2024).

The Human Adipsin ELISA Kit (SL3326Hu) utilizes a sandwich ELISA methodology for the quantitative detection of adipsin levels in human biological samples such as serum, plasma, cell culture supernatants, body fluids, and tissue homogenates. In this assay, the microplate wells are pre-coated with antibodies specific to adipsin. Standards or samples are added to the wells, allowing adipsin to bind to the immobilized antibodies. A secondary antibody conjugated with horseradish peroxidase (HRP) is then introduced, forming an antigen-antibody complex. After unbound substances are removed through washing, a chromogenic substrate (TMB) is added, producing a color change. The color intensity is measured as optical density (OD) at 450 nm, which is directly proportional to the adipsin concentration. A standard curve is used to calculate the sample concentrations (SunLong,2015).

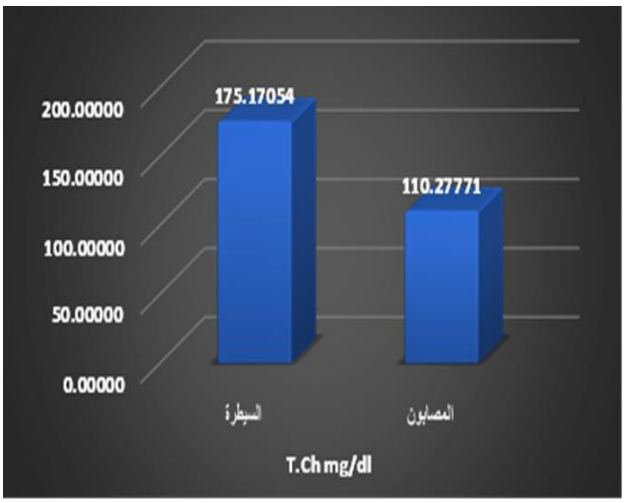
The CHOD-PAP (Cholesterol Oxidase-Peroxidase-Aminophenazone) method is an enzymatic assay for the quantitative determination of total cholesterol in human serum or plasma. The GPO-PAP (Glycerol-3-Phosphate Oxidase-Peroxidase-Aminophenazone) method is an enzymatic assay for the quantitative determination of triglycerides in human serum or plasma. Principle of HDL Cholesterol Very low-density lipoproteins (VLDL), chylomicrons, and LDL are precipitated using phosphotungstic acid (PTA) and magnesium chloride (Biolabo SAS,2022).

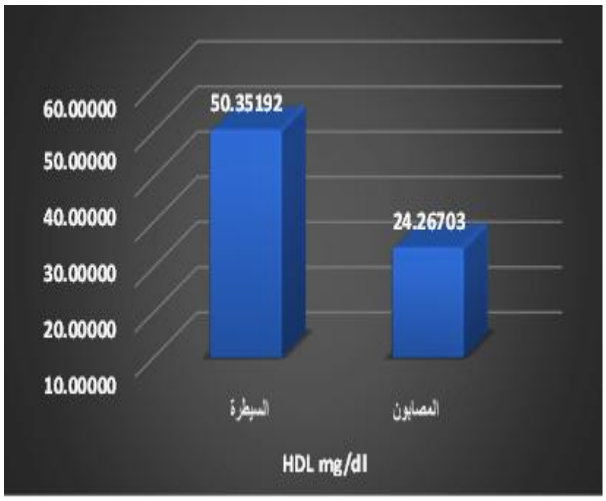
Results and Discussion

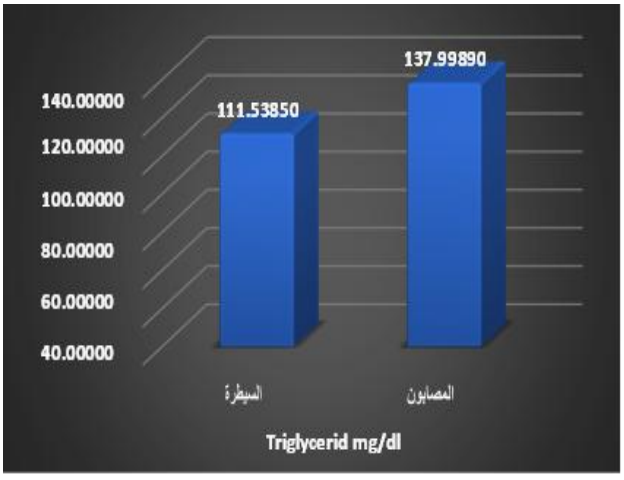
The mean total cholesterol concentration in the β-thalassemia patient group was significantly reduced at 110.28 ± 5.19 SD mg/dL compared to the control group (healthy individuals), which exhibited a mean of 175.17 ± 6.22 SD mg/dL. This

marked difference (P = 0.05) underscores the impact of β-thalassemia on lipid metabolism, as depicted in Figure (1), highlighting the disparity between the two groups, according to a systematic review by Mohamed et al. (2024), which included 21 studies. Patients with beta-thalassemia exhibited significant reductions in total serum cholesterol levels. Beta-thalassemia major and intermedia patients demonstrated marked decreases in total cholesterol, LDL-C, and HDL-C levels. In contrast, beta-thalassemia minor patients experienced less pronounced but still notable reductions in these lipid parameters. Additionally, triglyceride levels were elevated in beta-thalassemia major patients. These lipid profile abnormalities were consistently observed in beta-thalassemia intermedia patients, emphasizing the significant metabolic impact of the disease. The patient group demonstrated a lower mean HDL cholesterol level (24.27 ± 7.83 SD mg/dL) compared to the control group, which exhibited a higher mean of 50.35 ± 18.01 SD mg/dL. This disparity is illustrated in Figure (2). In the study by Doshi and Sutay (2016), serum HDL-C levels were significantly lower in β-thalassemia major-affected individuals compared to healthy controls ($P < 0.05$). This reduction highlights a key lipid abnormality associated with β-thalassemia major. The mean triglyceride level was higher in the patient group (137.99 ± 45.91 mg/dL, SD) compared to the control group (111.54 ± 46.53 mg/dL, SD). The results were statistically significant ($P = 0.05$), reflecting an increase in triglycerides among patients. This finding is illustrated in Figure 3. The findings of our study align with those reported by Doshi and Sutay (2016), who also observed significant alterations in lipid profiles among β-thalassemia major patients. Both studies highlighted elevated triglyceride levels in patients compared to healthy controls, underscoring a consistent pattern of dyslipidemia associated with β-thalassemia. The increase in triglycerides observed in both investigations reflects a common metabolic disturbance linked to the disease, reinforcing the robustness of these findings across different populations and study designs. The mean adipsin level was significantly higher in the patient group (252.63 ± 34.62 ng/mL, SD) compared to the control group (130.49 ± 31.77 ng/mL, SD). This difference was statistically significant ($P = 0.05$), emphasizing the association between elevated adipsin levels and the disease. This finding is illustrated in Figure 4 . Studies have highlighted the diverse roles of adipsin (Factor D) in metabolic regulation and disease biomarkers. Corvillo et al. (2021)

reported significant variations in plasma adipsin levels among patient groups compared to healthy controls, with levels either elevated or reduced depending on the cohort. Their protein analysis confirmed these differences, suggesting adipsin's potential as a diagnostic biomarker in specific conditions, as indicated by ROC curve analysis. They also observed a positive correlation between adipsin levels and age across several groups, with no significant sex-based differences.







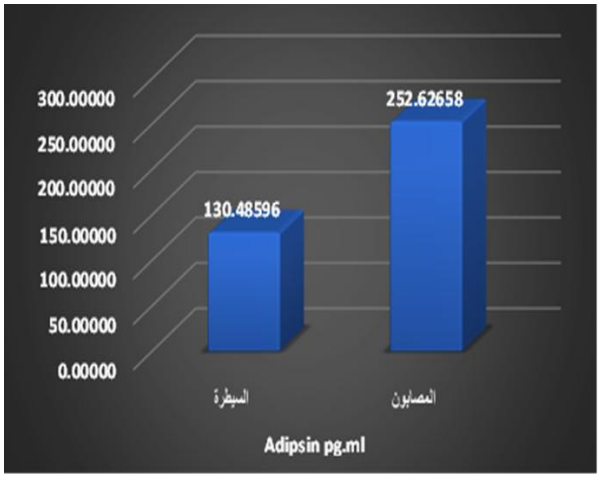


Figure (1) shows the mean values for both the control and patient groups for the variable (T. Ch).

Figure (2) shows the mean values for both the control and patient groups for the variable (HDL).

Figure (3) shows the mean values for both the control and patient groups for the variable (TG).

Figure (4) shows the mean values for both the control and patient groups for the variable Adipsin.

Conclusion

β-thalassemia is associated with significant lipid abnormalities and elevated adipsin levels, indicating underlying metabolic disturbances. These findings suggest that adipsin may serve as a potential biomarker for monitoring metabolic changes in β-thalassemia patients.

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