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Research Article

The Association between Leptin and Asprosin Levels in Female Patients with Type II Diabetes Mellitus

Duha Q.Bakr[1] , ***Ahmed Y.Abed*** [1]

[1]*Department of Chemistry, College of Sciences for Women, University of Baghdad, Baghdad, Iraq.*

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Abstract:

Background: Leptin and asprosin are adipokines secreted by white adipose tissue. The leptin and asprosin molecules have many functions in the central nervous system and other functions of the body: Appetite, glucose metabolism, insulin resistance, and cellular death.

Objectives: The study aims to determine the potential relationship between leptin and asprosin hormones in female patients with type II diabetes mellitus.

Methods: The present study was conducted in Al-Mahmodia Hospital / Baghdad and the laboratories of the College of Science for Girls / University of Baghdad / Iraq, for the period from 1/11/2023 to 1/2/ 2024. This study is a comparative analysis of several essential biomarkers found in the sera of individuals with diabetes via estimating leptin, asprosin, fasting blood glucose, glycated hemoglobin, body mass index, total cholesterol, triglycerides, high-density lipoprotein, low-density lipoprotein, aspartate aminotransferase, alanine aminotransferase and alkaline phosphatase in females with type II diabetes mellitus. The study consisted of 60 participants grouped into: Group I (30 females with diabetes), and group II (30 healthy females). The Biochemical parameters of every participant were ascertained. The quantification of leptin and asprosin in the serum was conducted using the enzyme-linked immunosorbent assay (ELISA).

Results: The levels of leptin and asprosin were markedly high in the diabetic group [(5.1 ± 0.69), and (10.3 ± 1.07)] compared with the control group [(2.0 ± 0.48), and (1.6 ± 0.16)] respectively, with a significant difference.

Conclusion: Despite the high levels of leptin and asprosin in female patients with Type II diabetes mellitus, the relationship between leptin and asprosin was a weak negative one.

Keywords: Asprosin; Fasting Blood Glucose; Glycated hemoglobin; Leptin; Type II diabetes mellitus.

Introduction:

The principal sources of energy for the body are glucose and fatty acids; glucose can be converted to cholesterol and fatty acids. Glucose and lipid metabolism issues give rise to diabetes, cardiovascular disease, and a fatty liver [1]. Diabetes mellitus is a subset of metabolic disorders known as hyperglycemia caused by the poor secretion of insulin from pancreatic [2,3,4]. Type II diabetes mellitus (DMII) is responsible for 90% of all clinical cases of diabetes [5]. Dyslipidemia, hyperglycemia, and insulin resistance (IR) are all conditions associated with type II diabetes mellitus (DMII) [6]. DM impacts lipid, energy, and glucose metabolisms as a result of inadequate glycemic control and resistance to insulin. Furthermore, DM is associated with elevated concentrations of low-density lipoprotein (LDL), triglycerides, and free fatty acids (FFA) in the plasma, while the level of high-density lipoprotein (HDL) decreases. As a result of macroangiopathy and microangiopathy, diabetes-induced long-term hyperglycemia impacts numerous systems, resulting in diabetic nephropathy, retinopathy, neuropathy, and atherosclerosis [7].Adipose tissue functions as a constituent of the endocrine glands, which generate a variety of signaling molecules that govern metabolism, energy expenditure, digestion, production, endocrine function, and immune system functionality [8]. Recent research has devoted a substantial amount of attention to examining the functions of adipokines, particularly asprosin and leptin, in the regulation of metabolism, given their emergence as noteworthy variables of interest [9]. Adipokines are involved in a multitude of physiological processes, encompassing fullness of stomach and appetite regulation, insulin sensitivity, adipogenesis, energy metabolism in insulin-sensitive tissues, endothelial function, maintenance of blood pressure, energy expenditure activity, hemostasis, and fat distribution within pancreatic cells [10]. Leptin is secreted primarily by adipose tissue [11], and it functions as a vital hormone in the regulation of energy balance. Leptin deficiency is associated with weight gain, increased food consumption, and adipose tissue accumulation [12,13]. Leptin's effects on improving glucose homeostasis are mediated either directly

**Corresponding Author: doha.bakr2305m@csw.uobaghdad.edu.iq.*

or indirectly by several target tissues, such as skeletal muscle, liver, central nervous system, and pancreas. Research indicated that subjects with diabetes have higher leptin levels than controls. Additionally, insulin levels, insulin sensitivity, age, sex, body mass index, central adiposity, and anti-diabetes therapy are all variables that influence leptin levels [14]. Asprosin, a new adipokine, is expressed and secreted primarily by white adipose tissue [15]. Asprosin is transported to the liver after being liberated from white adipose tissue. Hepatic gluconeogenesis is induced by asprosin, leading to elevated levels of circulating insulin and glucose. By traversing the blood-brain barrier, plasma asprosin directly stimulates orexigenic neurons via a cAMP-dependent signaling pathway, thereby inducing weight gain and stimulating appetite [16]. Numerous effects of asprosin have been documented on peripheral tissues and organs, as well as the central nervous system. Diabetes has been linked to elevated levels of serum asprosin, according to recent studies. Asprosin levels in the serum are significantly elevated in DMII patients compared to controls. It has also been shown that circulating asprosin is associated with fasting blood glucose and glycosylated hemoglobin [16]. This study aims to explore the relationship between leptin and asprosin in female patients with type II diabetes mellitus (DM) and in control subjects. Ultimately, we are trying to ascertain whether leptin and asprosin can serve as indicators for DMII. This finding may contribute to the development of innovative strategies to combat DMII and improve the lives of individuals affected by this prevalent metabolic disorder.

Participants and Methods

Thirty blood samples were collected from female patients with type II diabetes, and 30 blood samples were collected from female controls, in Al-Mahmodia Hospital / Baghdad and the laboratories of the College of Science for Girls / University of Baghdad / Iraq, for the period from 1/11/2023 to 1/2/ 2024. The ages of the patients ranged from 30 to 60 years. The controls were volunteers from the College of Science and Al-Mahmodia hospital workers. They were age-matched with the cases. Data was collected through face-to-face interviews with the patients and controls using a questionnaire designed for the study. Five ml of venous blood was drawn from the subjects after a period of 10 to 12 hours fasting using a 5 ml syringe. One milliliter of the blood was placed in a tube containing Ethylene diamine tetra-acetic acid (EDTA) to conduct the HbA1c test, and 4 ml were placed in a gel tube not containing an anticoagulant. The blood components were then separated in a centrifuge at a speed of 3000 rpm for 15 minutes for the purpose of conducting biochemical tests. A fasting blood glucose level and lipid profile assessment were conducted on 2 ml of the serum, while an evaluation of liver function (ALT, AST, and ALP) was conducted on 1ml of the serum. The remaining serum was transferred to Ependorf tubes and frozen at -20°C to quantify leptin and asprosin later by using (ELISA) technique.

Inclusion Criteria

1. Diabetes medical history: Each of the individuals was already diagnosed with type II diabetes mellitus, based on criteria of the Expert Committee on Diabetes Mellitus of the World Health Organization.
2. Patients between 30 and 60 years old.

Exclusion Criteria: Patients who are obese.

Results

Table (1) shows the mean ± SD values of leptin, asprosin, and body mass index (BMI) for the two study groups. A significant difference (p=0.0001) was found in leptin, and asprosin levels between patients and control groups [(5.1 ± 0.69), (2.0 ± 0.48), and (10.3 ± 1.07), (1.6 ± 0.16)] respectively. The mean BMI values did not differ significantly (p=0.811) between patients and controls.

Table 1: Mean ± SD levels of leptin and asprosin hormone in DMII patients and controls

Parameters	DMII Group (N=30)	Control Group (N=30)	P-value
Leptin (ng/ml)	5.1 ± 0.69	2.0 ± 0.48	0.001
Asprosin (ng/ml)	10.3 ± 1.07	1.6 ± 0.16	0.001
BMI (Kg/m^2)	28.1 ± 2.69	26.9 ± 2.79	0.811

Table (2) shows that females with DMII had significantly different FBG, HbA1c, TG, and HDL levels than the healthy females (p-value ≤0.05), while the mean levels of total cholesterol, and LDL showed no significant difference (P-value ≥0.05) between patients and controls.

Table 2: Mean ± SD of FBG, HbA1c, and lipid profile of the study groups

Parameters	(DMII) Group (N=30)	Control Group (N=30)	P-value
	189.4 ± 54.59		0.001
			0.001
FBG (mg/dl)	8.3 ± 1.65	93.1 ± 7.75	0.055
HbA1c %	208.6 ± 39.54	5.0 ± 0.51	0.002
Cholesterol (mg/dl)		178.5 ± 24.38	0.024
Triglycerides (mg/dl)	185.5 ± 50.06	112.7 ± 23.03	0.618
HDL (mg/dl)		44.6 ± 8.61	
LDL (mg/dl)	31.7 ± 6.43	109.5 ± 20.03	
	119.9 ± 16.77		

The mean ± SD values of Alkaline Phosphatase (ALP) showed a significant difference at (p<0.05) when comparing patients and controls. There was no significant differences between mean Aspartate Aminotransferase (AST) and Alanine Aminotransferase (ALT) levels between the study groups (P > 0.05), as shown in Table (3).

Table 3: Mean ± SD levels of liver enzymes in the DMII and control groups

Parameters	(DMII) Group (N=30)	Control Group (N=30)	P-value
AST (U/L)	19.7 ± 6.08	18.0 ± 5.67	0.688
ALT (U/L)	14.5 ± 4.78	12.7 ± 3.56	0.169

ALP (U/L)	77.4 ± 19.66	72.1 ± 9.95	0.004

As shown in Figures 1 and 2, a weak negative non-significant correlation was found between leptin and asprosin in both study groups (r = -0.095, p - value = 0.616) and (r = -0.023, p -value = 0.904) respectively.

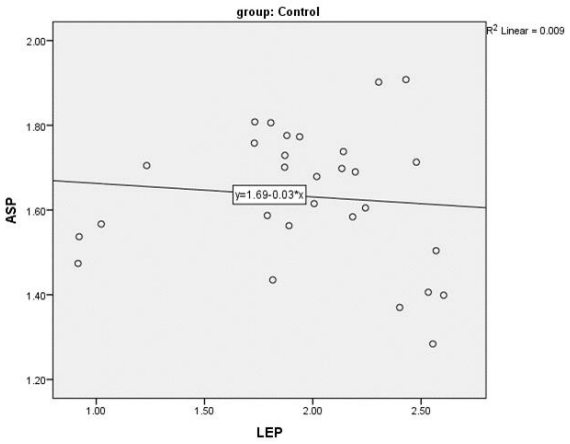

Figure 1: The correlation between leptin and asprosin in the control group

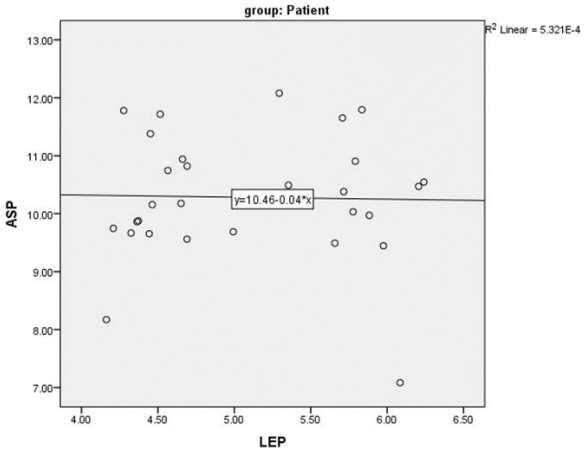

Figure 2: The correlation between leptin and asprosin in the DMII group

Discussion

The higher levels of leptin and asprosin in the DMII than the control group are in agreement with the findings of Peng et al, Katsiki et al, and Hameed et al [12,13,17], but are in disagreement with the findings of Onyemelukwe, et al [14]. The results of the current study and those of Peng, et al, suggest that the high levels of leptin may be related to its action of reducing glucose levels. Leptin can reduce blood glucose levels autonomously, specifically in hyperglycemic models of insulin deficiency [12]. The regulatory functions of leptin regarding energy expenditure and food intake via central signaling pathways are well-defined. Leptin transmits signals to the hypothalamus and hindbrain, which are central to inducing a reduction in food intake and an increase in energy expenditure [13,17,18]. Furthermore, central signaling pathways may be utilized by leptin to exert significant glucoregulatory and insulin-sensitizing effects [12,18]. The direct influence of leptin on neurons that express pro-opiomelanocortin (POMC) has been shown to contribute to the glucoregulatory effects of leptin signaling, thereby expanding upon these findings. Leptin receptor selective re-expression in POMC-expressing central neurons reduces circulating glucagon concentrations and ameliorates dyslipidemia, hepatic insulin resistance, and blood glucose. Moreover, by influencing lipid metabolism, leptin may potentially contribute to enhancements in insulin sensitivity [18]. The current study has also shown that the levels of asprosin hormone were also significantly higher in the DMII than in the control group. This agrees with the findings of Farrag et al and Naiemian et al [19,20]. Research has demonstrated that asprosin inhibits β-cell autophagy via the adenosine monophosphate-activated protein kinase (AMPK) and mammalian target of rapamycin (mTOR) signaling pathways, thereby inducing β-cell apoptosis [21]. Asprosin may cause β-cell dysfunction and impaired glucose tolerance in patients with DMII, according to a Wang et al [21]. Romere et al revealed that asprosin elevates the concentration of glucose in the circulation by promoting hepatic gluconeogenesis [22]. Since obese DMII cases were excluded from the study groups, the mean BMI was not significantly different between the DMII female patients and their controls. The current study showed that the serum levels of Cholesterol, TG, and, LDL were higher in the DMII group than the control group, while serum HDL level was lower in patients compared to controls. This is in agreement with another study that showed that patients with DMII had elevated levels of all lipid markers except HDL [23]. It had been suggested that the elevation in triglyceride levels could be attributed to an insufficiency of insulin, leading to impaired glucose utilization, hyperglycemia, and fatty acid mobilization from adipose tissues. Blood glucose is not utilized by tissues in individuals with diabetes, leading to hyperglycemia. The fatty acids are mobilized from the adipose tissue for energy production and surplus fatty acids are accumulated in the liver, and are converted to triglyceride [23]. Insulin stimulates the production of LDL receptors; therefore, chronic insulin deficiency may be linked to a reduction in LDL receptor levels. This results in the elevation of LDL particles and a subsequent rise in LDL-cholesterol levels in individuals with diabetes mellitus. Liver enzymes (ALT and AST) levels were not significantly different when comparing DMII patients and their controls. However, a significantly higher ALP level was found in fasting serum samples from diabetic patients when compared to the control group. 4. Few prior studies have documented that a considerable number of diabetics may also manifest elevated levels of ALP [24], which may be due to the direct hepatotoxic impact of fatty acid on the liver when it is excessively produced. Potential mechanisms underlying this phenomenon encompass high-concentration cell membrane disruption, mitochondrial dysfunction, toxin formation, as well as activation and inhibition of crucial

metabolic regulation steps [24]. Accordingly, individuals diagnosed with diabetes exhibit metabolic abnormalities that cause raising leptin and asprosin levels.

Conclusions

Serum levels of leptin and asprosin are higher in patients with DMII. A weak negative correlation exists between leptin and asprosin in those patients. Leptin and asprosin may influence glucose levels; thus, they may play a significant role in the diagnostic and therapeutic management of diabetes.

Authors' Declaration

We confirm that all the Figures and Tables in the manuscript belong to the current study. Authors sign an ethical consideration's approval-Ethical Clearance: The project was approved by the local ethical committee in (The project was approved by the local ethical committee in College of Science for Women/University of Baghdad/Iraq) according to the code number (67621) on (8/ 11/2023).

Conflicts of Interest

The authors declare no conflict of interest.

Funding/Non.

Authors' Contributions

Duha Q.Bakr and Ahmed Y.Abed contributed to the design and implementation of the research, to the analysis of the results, and the writing of the manuscript.

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العلاقة بين مستويات اللبتين والاسبروسين لدى الإناث المصابات بداء السكري من النوع الثاني

ضحى قحطان بكر[1]*
احمد يونس عبد[1]
[1]قسم الكيمياء، كلية العلوم للبنات، جامعة بغداد، بغداد، العراق.

الخلاصة:
خلفية البحث: اللبتين والأسبروسين هي عبارة عن أديبوكينات تفرزها الأنسجة الدهنية. تلعب جزيئات اللبتين والأسبروسين العديد من الأدوار في الجهاز العصبي المركزي ووظائف الجسم الأخرى: الجوع وإستقلاب الجلوكوز ومقاومة الإنسولين (IR) وموت الخلايا.
الأهداف: معرفة العلاقة المحتملة بين هرمون اللبتين وهرمون الأسبروسين لدى الإناث المصابات بداء السكري من النوع الثاني DMII.
المنهجية: أجريت الدراسة الحالية في مستشفى المحمودية \ بغداد ومختبرات كلية العلوم للبنات \ جامعة بغداد \ العراق خلال الفترة الزمنية من 1\11\ 2023 إلى 1\2\2024. صممت هذه الدراسة لمقارنة بعض المؤشرات الحيوية في أمصال مرضى السكري من خلال تقدير هرمون اللبتين والأسبروسين ونسبة الجلوكوز في الدم الصائم والهيموجلوبين السكري ومؤشر كتلة الجسم والكوليسترول والدهون الثلاثية والبروتين الدهني عالي الكثافة والبروتين الدهني منخفض الكثافة وناقلة أمين الأسيارتات وناقلة أمين الألانين والفوسفاتيز القلوي لدى الإناث المصابات بداء السكري من النوع الثاني. تكونت مجموعة الدراسة من 60 عينة تم تقسيمها إلى مجموعتين: المجموعة الأولى (30 أنثى مصابة بداء السكري) والمجموعة الثانية (30 أنثى سليمة). تم تحديد المتغيرات الكيميائية الحيوية في جميع المشاركات. تم قياس تراكيز مصل اللبتين والأسبروسين باستخدام مقياس الإمتزازالمناعي المرتبط بالإنزيم.
النتائج: كانت مستويات هرموني اللبتين والأسبروسين مرتفعة بشكل ملحوظ لدى مجموعة المرضى (0.69 ± 5.1) و (1.07 ± 10.3) مقارنة بالمجموعة الضابطة (0.48 ± 2.0) و (0.16 ± 1.6)، على التوالي مع وجود فرق معنوي
الإستنتاجات: على الرغم من إرتفاع مستويات هرمون اللبتين والأسبروسين في الإناث المصابات بداء السكري من النوع الثاني إلا أن العلاقة بين اللبتين والأسبروسين كانت سلبية ضعيفة.
مفتاح الكلمات: داء السكري من النوع الثاني، اللبتين، الأسبروسين، جلوك وز الدم الصائم، الهيموجلوبين السكري.