Research Article

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The Role of Adiponectin, Leptin and Female Hormones in Iraqi Type 2 Diabetic Patients with and without Obesity

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Abstract

Background: Leptin and adiponectin are two major adipokines involved in the regulation of energy balance, glucose metabolism, and insulin sensitivity. These adipokines are also expected to have an important role in the development of type 2 diabetes mellitus (T2DM).

Objective: To measure the levels of leptin, adiponectin, progesterone, luteinizing hormone (LH), and follicle-stimulating hormone (FSH) in Iraqi patients with type 2 diabetes who are obese or non-obese.

Methods: The 120 participants in this study were divided into three groups: Group 2 (G2) included 40 obese patients with type 2 diabetes, Group 3 (G3) included 40 non-obese patients with type 2 diabetes, and Group 1 (G1) was an age- and sex-matched healthy control group.

Results: Leptin concentrations were significantly elevated in the T2DM and T2DM-obese groups compared with the control group. Adiponectin levels were markedly reduced in the T2DM groups. LH was lowered in the T2DM non-obese group only. FSH and progesterone levels were downregulated only in the T2DM obese group.

Conclusion: According to patient weight status, leptin and adiponectin may have important diagnostic value during the course of type 2 diabetes mellitus.

Keywords: DM2, leptin, adiponectin, progesterone, FSH, LH

1. INTRODUCTION

Type 2 diabetes mellitus (T2DM) is characterized by insufficient tissue responsiveness to insulin and reduced insulin release by pancreatic β-cells [1, 2, 3, 4]. These activities are tightly regulated because glucose homeostasis depends on the molecular mechanisms underlying insulin production, release, and sensing. Defects in any of the pathways that support these activities may cause metabolic imbalance and disease [5, 6].

Obesity is a serious and expanding public health concern. Excess body fat is an established risk factor for type 2 diabetes, and the likelihood of developing the condition increases directly with body mass index (BMI). As a result, the global rise in obesity has coincided with an increase in type 2 diabetes incidence. Complex biochemical and physiological factors, including dysregulation of adipose tissue, altered β-cell activity, and systemic insulin resistance, underlie this association. Notably, weight loss has been shown to improve, and in some cases restore, these metabolic processes [7, 8].

Leptin is an adipokine protein that is now recognized as a hormone. It has a molecular weight of approximately 16 kDa and consists of 167 amino acids. It is primarily produced by adipocytes, and its levels are closely related to the amount of fat stored in the body. Leptin has been shown to reduce food intake and body weight, making it a potential therapeutic target in obesity. However, obese individuals frequently have elevated circulating leptin levels but are resistant to exogenous leptin therapy. The inability of leptin to generate its anorexigenic effects in obese individuals is known as leptin resistance, which limits its therapeutic utility in obesity. Effective use of leptin in obesity treatment requires an understanding of the molecular processes behind leptin resistance. One important factor is that leptin must pass through the blood-brain barrier to enter the hypothalamus and initiate its anorexigenic effects. Several unclear processes are involved, and new methods for restoring leptin response in obese individuals have recently emerged [9, 10, 11].

Adiponectin is an adipokine released by adipocytes and is a well-known homeostatic factor that controls insulin sensitivity, lipid metabolism, and glucose levels through antioxidant, anti-inflammatory, and anti-fibrotic properties. These metabolic activities are regulated by two adiponectin receptors, AdipoR1 and AdipoR2. Adiponectin is also one of the most abundant hormones in plasma. Its levels rise with weight loss or calorie restriction, indicating improved insulin sensitivity. Therefore, the adiponectin signaling pathway may play an important role in the development of treatments for type 2 diabetes and other obesity-related conditions characterized by insulin resistance, such as cardiovascular disease and cancer. Adiponectin appears to improve insulin sensitivity by enhancing glucose and lipid metabolism [12, 13].

Progesterone is a naturally occurring steroid hormone produced mainly by the gonads and adrenal cortex. The ovarian corpus luteum secretes it during the first ten weeks of pregnancy; after this period, the placenta gradually takes over progesterone production. This transition usually occurs around week 10. Derived from cholesterol, progesterone plays multiple roles in the body, especially in the reproductive system, and is essential for maintaining normal uterine function during pregnancy [14].

The pituitary gland produces and releases follicle-stimulating hormone (FSH), which is essential for controlling sexual development and reproductive processes. It affects ovarian function. FSH and luteinizing hormone (LH) help regulate the menstrual cycle. FSH contributes to ovulation, the process by which the ovaries produce an egg each month. FSH levels change during the menstrual cycle and reach their maximum level immediately before ovulation [15].

Luteinizing hormone (LH) is a glycoprotein hormone released together with FSH by gonadotrophin cells in the adenohypophysis. In this pathway, gonadotropin-releasing hormone (GnRH) stimulates LH secretion, whereas estrogen inhibits it in females [16].

2. MATERIALS AND METHODS

Blood was drawn from 80 female diabetes patients between the ages of 30 and 50 years. Forty of these patients had type 2 diabetes without obesity, whereas the other 40 had type 2 diabetes with obesity. Forty healthy females in the same age range were included as the control group.

From January to March 2025, all patients were enrolled at the City of Medicine Hospital and Al-Wasti Teaching Hospital in Baghdad. Adiponectin and leptin A were measured using ELISA kits (E-EL-H1581 ELABSCIENCE and E-EL-0135 ELABSCIENCE, USA), and progesterone, LH, and FSH were estimated using the colorimetric technique.

2.1. EXCLUDED GROUP

Patients with type 2 diabetes who had any of the following conditions were excluded from the study:

1. kidney failure;
2. liver disease;
3. cardiovascular disease;
4. rheumatoid arthritis;

5. osteoporosis;

6. diabetic retinopathy.

2.2. STATISTICAL ANALYSIS

Microsoft Excel 2013 and the Statistical Package for Social Sciences (SPSS version 26.0) were used for the analysis. The t-test results for the current study were displayed as mean ± SD. A t-test was also used to compare the patient and control groups. Furthermore, p-values less than 0.05 and 0.001 were considered significant. The area under the receiver operating characteristic (ROC) curve, sensitivity, and specificity were also investigated.

The t-test data The mean ± SD for the current study was shown. Additionally, the t-test was used to compare the patient and control groups. Furthermore, p values were considered significant if they were less than 0.05 and 0.001.Moreover, the area under the receiver operating characteristic (ROC) curve, sensitivity, and specificity [17, 18].

3. RESULTS AND DISCUSSION

Levels of leptin, adiponectin, progesterone, LH, and FSH were detected in women with type 2 diabetes, with and without obesity, and in healthy control women by ELISA method. The results are summarized in Tables 1 and 2.

Table 1. *Comparison of selected biomarkers among type 2 diabetes patients with obesity, type 2 diabetes patients without obesity, and controls (mean ± SD).*

Parameters	Mean ± SD			
	Control No.40	DM2 with Obesity No.40	DM2 Without Obesity No.40	P-Value
Leptin(pg/ml)	222.54±59.88	724.88 ± 161.50	468.26 ± 97.24	0.001*
Adiponectin(ug/ml)	5.17±0.54	0.26± 0.094	0.73 ± 0.10	0.001*
Progesterone(ng/ml)	9.88±0.47	5.02 ± 0.69	6.28 ± 0.44	0.001*
LH(mlU/mL)	8.65± 1.52	7.31 ± 0.95	5.38 ± 0.94	0.001*
FSH(mlU/mL)	9.46±0.75	5.08±1.20	7.06±0.87	0.001*

*Significant at $p = 0.001$.

Table 2. *Comparison of selected biomarkers between type 2 diabetes patients with obesity and type 2 diabetes patients without obesity (mean ± SD).*

Parameters	Mean ± SD		
	DM2 with Obesity No.40	DM2 Without Obesity No.40	P-Value
Leptin(pg/ml)	724.88 ± 161.50	468.26 ± 97.24	0.001*
Adiponectin(ng/ml)	0.26± 0.094	0.73 ± 0.10	0.001*
Progesterone(ng/ml)	5.02 ± 0.69	6.28 ± 0.44	0.001*
LH(mlU/mL)	7.31 ± 0.95	5.38 ± 0.94	0.001*
FSH(mlU/mL)	5.08±1.20	7.06±0.87	0.001*

*Significant at $p = 0.001$.

Both diabetes groups had considerably higher serum leptin concentrations than the lean control group, with the DM2 with obesity group (G2) showing the largest increase (Table 1). Similarly, serum adiponectin concentrations were highly significantly decreased in G2 and G3 compared with lean controls from G1 ($p < 0.01$). The mean serum adiponectin concentration in the lean control group, 5.17 ± 0.54 μg/mL, was within the normal range for adult women.

Diabetes mellitus and obesity are growing global public health problems. Leptin is an adipocyte hormone that is crucial for controlling several metabolic functions, including insulin sensitivity, energy expenditure, and food intake [19, 20, 21].

Adiponectin is an adipokine secreted by adipocytes and is involved in regulating glucose and lipid metabolism. Normal serum adiponectin levels in healthy adults range from 5 to 30 μg/mL. However, patients with type 2 diabetes and obesity have lower serum adiponectin levels than normal [21, 22].

Serum progesterone, LH, and FSH levels were affected. In the DM2 with obesity group (G2), serum progesterone and FSH levels were highly significantly reduced, whereas serum LH levels were significantly decreased in both diabetic groups compared with controls ($p = 0.001$).

Table 2 shows that leptin concentration was significantly elevated in obese diabetics (G2) compared with non-obese diabetics (G3). Adiponectin and progesterone concentrations were significantly lower in obese diabetics than in non-obese diabetics, with significant differences in LH and FSH concentrations between the diabetic subgroups ($p = 0.001$).

Correlations between adipokines (leptin and adiponectin) and sex hormones (progesterone, LH, and FSH) are shown in Tables 3 and 4.

In both diabetes groups (G2 and G3), there was a substantial positive association between leptin and these hormones as well as between adiponectin and progesterone, LH, and FSH. Strong correlations were found between adipokines and

reproductive hormones in women with type 2 diabetes mellitus.

Table 3. *Correlation of adiponectin and leptin with biochemical parameters in G2.*

Parameters	progesteron	LH	FSH
Adiponectin	R= 0.988*	R= 0.962*	R= 0.952*
	P= 4.81368E-08	P= 1.49531E-08	P= 4.62784E-06
Leptin	R= 0.964*	R= 0.971*	R= 0.974*
	P= 8.5117E-07	P= 8.77767E-07	P= 8.4993E-07

*Significant at $p = 0.001$.

Table 4. *Correlation of adiponectin and leptin with biochemical parameters in G3.*

Parameters	progesteron	LH	FSH
Adiponectin	R= 0.968*	R= 0.998*	R= 0.982*
	P= 2.87556E-10	P= 6.95188E-07	P= 1.78298E-08
Leptin	R= 0969*	R= 0.955*	R= 0.961*
	P= 4.91064E-06	P= 4.81396E-06	P= 4.97403E-06

*Significant at $p = 0.001$.

Tables 5 and 6 show the sensitivity, specificity, and AUC of leptin and adiponectin in distinguishing diabetic patients from healthy controls.

Table 5. *Sensitivity and Specificity and AUC of Studied Groups (G1,G2)*

Parameter	Sensitivity%	Specificity%	AUC%
Leptin	100%	96%	98 (95-100)
Adeponectin	92%	94%	93 (90-100)

Table 6. *Sensitivity and Specificity and AUC of Studied Groups (G1,G3)*

Parameter	Sensitivity%	Specificity%	AUC%
Leptin	100%	98%	98 (95-100)
Adeponectin	97%	95%	96 (95-100)

Leptin and adiponectin showed very good diagnostic values, with high sensitivity and specificity and AUC values close to 1.0. This indicates that these biomarkers have very good discriminatory ability for type 2 diabetes with and without obesity compared with healthy controls. The ROC curves are presented in Figures 1–4.

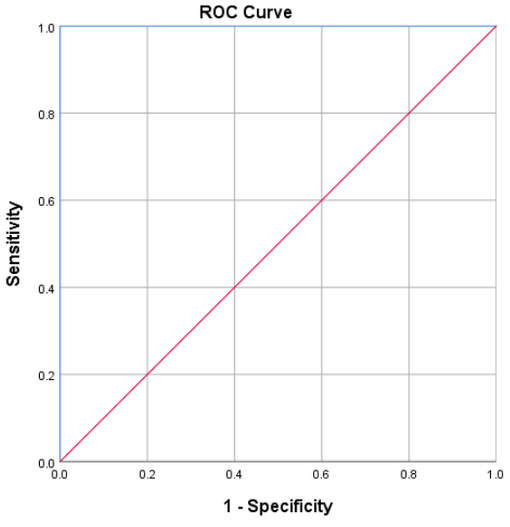

Figure 1. *ROC curve for leptin in type 2 diabetes with obesity.*

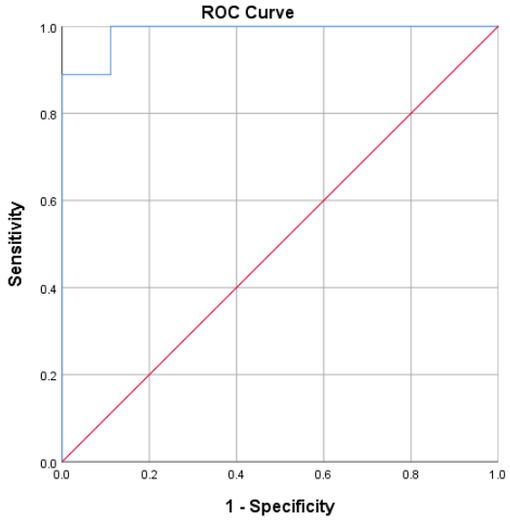

Figure 2. *ROC curve for leptin in type 2 diabetes without obesity.*

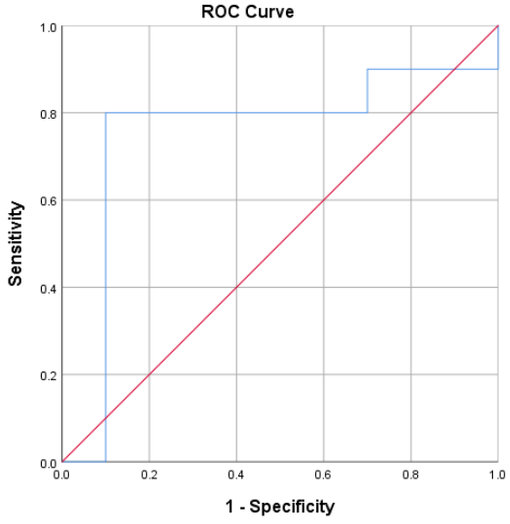

Figure 3. *ROC curve for adiponectin in type 2 diabetes with obesity.*

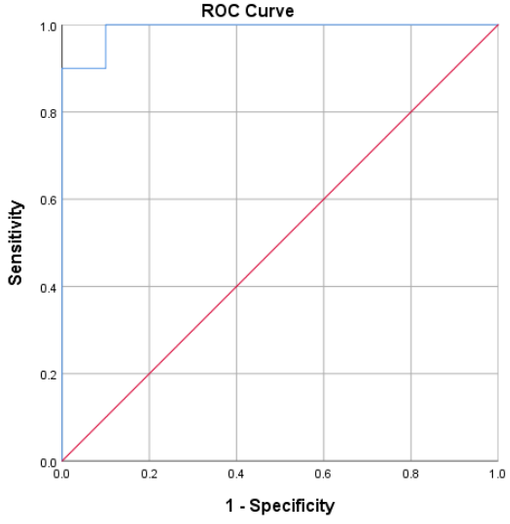

Figure 4. *ROC curve for adiponectin in type 2 diabetes without obesity.*

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