Medical Laboratory Journal
Online ISSN: 2538-4449
Golestan University of Medical Sciences

Evaluation of serum adiponectin and leptin levels in type 2 diabetic patients: The potential role in predicting metabolic syndrome

Sharabeh Hezarkhani [1], Khosro Ghojoghi [2], Sara Hosseinzadeh [2], Naser Behnampour [3], Farshid Fathabadi [2], Zahra Hesari [1*], Hamid Reza Joshaghani [2*]

1. Metabolic Disorders Research Center, Golestan University of Medical Sciences, Gorgan, Iran
2. Laboratory Sciences Research Center, Golestan University of Medical Sciences, Gorgan, Iran
3. Biostatistics Department, Faculty of Health, Golestan University of Medical Sciences, Gorgan, Iran

* Correspondence: Zahra Hesari. Metabolic Disorders Research Center, Golestan University of Medical Sciences, Gorgan, Iran.; Email: hesari7@yahoo.com and Hamid Reza Joshaghani. Laboratory Sciences Research Center, Golestan University of Medical Sciences, Gorgan, Iran. Tel: +981732456105; Email: joshaghani@goums.ac.ir

Abstract

Background: Leptin and adiponectin, two members of the adipokine family, play roles in increasing lipid metabolism and inhibiting lipogenesis. Reduced levels of these cytokines are associated with obesity and insulin resistance. This study aimed to determine the serum levels of leptin and adiponectin in type-2 diabetic patients with and without metabolic syndrome compared to a control group.

Methods: Three groups of individuals participated in this study: 47 type-2 diabetic patients with metabolic syndrome (DM+MetS), 25 type-2 diabetic patients without metabolic syndrome (DM-MetS), and 40 individuals with no history of diabetes or metabolic syndrome (Control group). Fasting blood samples were collected, and serum levels of fasting blood sugar, cholesterol, triglycerides, HDL-cholesterol, and LDL-cholesterol were measured using the enzymatic method. Blood pressure, height, and weight were recorded using stadiometers, while leptin and adiponectin levels were determined via enzyme-linked immunosorbent assay.

Results: A significant difference was observed between the DM+MetS group and the DM-MetS group in serum leptin ($p = 0.004$) and adiponectin ($p < 0.001$) levels. In patients with type-2 diabetes and metabolic syndrome, serum leptin ($p = 0.530$) and adiponectin ($p < 0.001$) levels were lower compared to the control group.

Conclusion: A decrease in the serum levels of key adipokines, such as leptin and adiponectin, in type-2 diabetic patients may serve as a predictor of metabolic syndrome.

Article History

Received: 8 August 2024
Received in revised form: 9 October 2024
Accepted: 12 November 2024
Published online: 24 December 2024
DOI: 10.29252/mlj.18.6.28

Keywords

Leptin
Adiponectin
Diabetes Mellitus
Metabolic Syndrome

Article Type: Original Article

Introduction

Metabolic syndrome is a multifactorial metabolic disease associated with an elevated risk of heart disorders and diabetes. The etiology of the disease is not yet completely understood, but the factors implicated in its development can be categorized into three main groups: insulin resistance, obesity, disorders of adipose tissue, and a collection of other independent factors (1,2).

Most patients with metabolic syndrome exhibit no symptoms of diabetes until they develop classical diabetes. On the other hand, non-diabetics with metabolic syndrome are at a higher risk of developing diabetes and experiencing milder metabolic disorders in the future. Therefore, identifying methods and factors to predict metabolic syndrome is highly valuable.

Recently, adipose tissue has been identified as the primary endocrine organ in the body. This organ secretes numerous biologically active molecules, known as adipokines, which regulate whole-body metabolism and immunity (3-5).

Adiponectin, a member of the adipokine family, is abundant in systemic circulation and serves as a protective biomarker for metabolic syndrome. Its concentrations are inversely proportional to obesity and insulin resistance. Unlike other adipokines that increase in obese individuals, adiponectin levels decrease in those with excess body fat, as well as in patients with diabetes and metabolic syndrome (6-10). In addition, weight loss and the use of insulin-sensitizing medications lead to an increase in adiponectin levels (11).

The central role of adiponectin in addressing risk factors for metabolic syndrome, along with its anti-inflammatory effects, has been noted (8,10).

Leptin is another member of the adipokine family that inhibits food intake and induces energy expenditure, thereby modulating adipose tissue mass and body weight (12-14).

The serum level of this adipokine depends on the amount of adipose tissue, as well as the patient's age and sex (8). Unlike adiponectin, there is a positive correlation between the serum level of leptin and obesity (15,16). It has been shown that a reduction in the level of this adipokine leads to obesity and insulin resistance (10).

It is believed that adiponectin and leptin are involved in the development of metabolic diseases in opposite ways (10). Therefore, this study aimed to measure the serum levels of adiponectin and leptin in patients with type 2 diabetes and metabolic syndrome compared to a control group.

Methods

Three groups of people participated in the study. Group (DM+MetS) included 47 type 2 diabetic patients (12 males and 35 females) with a mean age of 53.3 ± 10.4 years (Range 30-74), who had recently been diagnosed and were recruited from central clinics. They were diagnosed with metabolic syndrome according to the National Cholesterol Education Program's Adult Treatment Panel III (ATP III) criteria: fasting blood sugar (FBS) >100 mg/dl, triglycerides (TG) >150 mg/dl, blood pressure (BP) >130/80 mmHg, HDL-cholesterol ≤50 mg/dl in women and ≤40 mg/dl in men, and waist circumference ≥88 cm in women and ≥102 cm in men (17). Group (DM-MetS) consisted of 25 recently diagnosed type 2 diabetic patients without metabolic syndrome according to the ATP III criteria (11 males and 14 females), with a mean age of 53.7 ± 14.0 years (Range 24-86). The control group included 40 non-diabetic, healthy individuals without metabolic syndrome (8 males and 32 females), with a mean age of 50.0 ± 11.7 years (Range 29-78). Informed consent was obtained from all participants after explaining the purpose and objectives of the study prior to inclusion. This study was approved by the Ethics Committee of the Golestan University of Medical Sciences (10059004135).

After measuring height and waist circumference using a meter, weight with a standard scale, and BP with a manometer, 5 ml blood samples were collected from subjects diagnosed with type 2 diabetes who were referred to the laboratory in a fasting state. Serum separation was performed, and the sera were stored at -70 °C until use. The serum levels of FBS, cholesterol, TG, HDL-C, and LDL-C were measured using the enzymatic method (Pars Azmun, Iran). Adiponectin and leptin concentrations in the serum samples were determined using enzyme-linked immunosorbent assay kits (Biovendor, Czech Republic) according to the manufacturer's instructions.

SPSS software version 18 was used for data analysis. One-way ANOVA and multiple comparison tests were applied to identify differences between the three groups. Results were expressed as mean ± standard deviation (S.D.), with a p-value of ≤0.05 considered statistically significant. All assays were performed at least three times. For further data analysis, Duncan's test was used in cases of non-homogeneous variances, while Dunnett's T3 test was applied for homogeneous variances.

Results

The anthropometric characteristics of the subjects, grouped according to metabolic syndrome status, included a total of 112 serum samples (72.3% female and 27.7% male). The average ages of the groups were 53.31 ± 10.40, 53.76 ± 14.03, and 50.05 ± 11.77 years, respectively, for the (DM+MetS), (DM-MetS), and control groups (P-value = 0.348). In addition, the body mass index (BMI, kg/m²) in these groups was 29.96 ± 4.53, 26.40 ± 9.69, and 26.79 ± 2.74, respectively ($p < 0.001$). Body weight (kg) was reported as 79.46 ± 14.82, 67.98 ± 21.72, and 71.37 ± 9.52, respectively ($p < 0.001$).

Our results showed a significant difference between the (DM+MetS) group and the control group in systolic BP ($p < 0.001$), fasting blood sugar ($p = 0.000$), TG ($p < 0.001$), HDL-Cholesterol ($p = 0.023$), and adiponectin ($p < 0.001$). However, this difference was not statistically significant for diastolic BP ($p = 0.084$), cholesterol ($p = 0.322$), LDL-Cholesterol ($p = 0.562$), and leptin ($p = 0.596$). Meanwhile, a significant difference was found between the (DM-MetS) group and the control group in leptin ($p < 0.001$), adiponectin ($p < 0.001$), fasting blood sugar ($p < 0.001$), and LDL-Cholesterol ($p = 0.019$) (Figure 1 and Table 1).

There was also a significant difference between the (DM+MetS) and (DM-MetS) groups in systolic BP ($p < 0.001$), diastolic BP ($p < 0.001$), TG ($p < 0.001$), cholesterol ($p = 0.025$), leptin ($p = 0.008$), adiponectin ($p = 0.017$), and BMI ($p < 0.001$). However, no significant difference was observed for LDL-Cholesterol ($p = 0.057$) or HDL-Cholesterol ($p = 0.061$) (Figure 1 and Table 2).

The results of our study also revealed a significant positive correlation between adiponectin and HDL-Cholesterol ($p = 0.004$), as well as a significant negative correlation between adiponectin and TG ($p < 0.001$) and FBS ($p < 0.001$). However, the negative correlation between leptin and FBS was not significant (Table 2).

Table 1. Serum levels of leptin and adiponectin in subjects

Variable (P-value)	Groups (Mean ± SD)		P-value
Leptin (ng/ml) (0.004)	**(DM+MetS)** (24.94±17.57)	**(DM-MetS)**	0.008
		Control group	0.596
	(DM-MetS) (11.27±7.31)	**Control group** (27.38±17.16)	< 0.001
Adiponectin (ng/ml) (< 0.001)	**(DM+MetS)** (0.65±0.61)	**(DM-MetS)**	0.017
		Control group	< 0.001
	(DM-MetS) (0.83±0.71)	**Control group** (1.12±0.42)	< 0.001

Data are presented as mean ± SD. (DM+MetS): Diabetic patients with metabolic syndrome, (DM-MetS): Diabetic patients without metabolic syndrome, Control group: Patients without diabetes and metabolic syndrome. A p-value ⩽ 0.05 was considered significant (* p-value ⩽ 0.05; ** p-value ⩽ 0.01; *** p-value ⩽ 0.001).

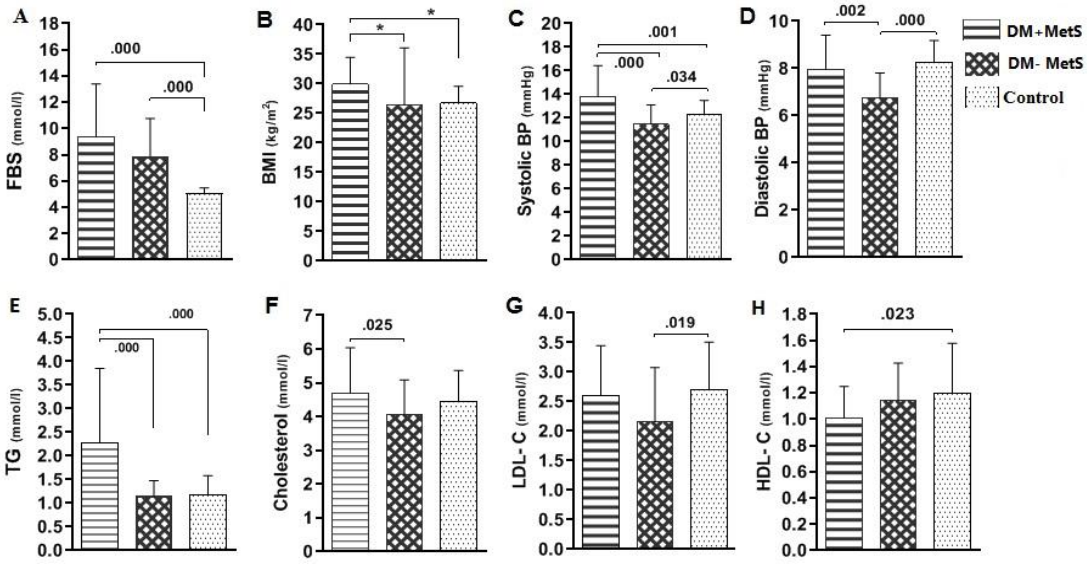

Figure 1. Biochemical parameters of subjects. (DM+MetS): Diabetic patients with metabolic syndrome; (DM-MetS): Diabetic patients without metabolic syndrome; Control group: Patients without diabetes and metabolic syndrome. TG: triglyceride; LDL-C: low-density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol; FBS: fasting blood sugar. *p ⩽ 0.05; **p ⩽ 0.01; ***p ⩽ 0.001.

Table 2. Correlation coefficient between biochemical parameters of subjects

Spearman's rho	Variables	FBS	TG	Cholesterol [a]	HDL	LDL [a]
Leptin	Correlation Coefficient	- 0.188	0.138	0.169	0.036	0.120
	Sig. (2-tailed)	0.083	0.205	0.120	0.740	0.270
Adiponectin	Correlation Coefficient	- 0.422**	- 0.300**	0.080	0.273**	0.186
	Sig. (2-tailed)	0.000	0.001	0.401	0.004	0.063
Leptin/Adiponectin	Correlation Coefficient	0.034	0.255*	0.069	- 0.105	- 0.024
	Sig. (2-tailed)	0.758	0.018	0.531	0.335	0.825

**. Correlation is significant at the 0.01 level (2-tailed).
*. Correlation is significant at the 0.05 level (2-tailed).
a. Spearman correlation coefficient

Discussion

Evidence suggests that adiponectin and leptin are closely associated with metabolic syndrome in opposite directions and may serve as useful diagnostic markers for the disease (10).

Sanjari et al. investigated the correlation between serum adiponectin levels and metabolic syndrome. In this study, subjects with metabolic syndrome had lower serum adiponectin levels than healthy individuals. They concluded that the serum level of this adipokine is negatively correlated with metabolic syndrome (18).

Zhuo et al. examined the association between adiponectin, leptin, and the leptin-to-adiponectin ratio with metabolic syndrome. Subjects with metabolic syndrome showed higher leptin, TG, FBS, and BMI levels, while adiponectin and HDL were lower than in the control group (10).

In another study by Henneman et al., postmenopausal women with low levels of adiponectin showed the highest risk for developing metabolic syndrome (19).

Inconsistent with these findings, our results revealed that in the (DM+MetS) and (DM-MetS) groups, the serum adiponectin levels were significantly lower than in the control group. In addition, in our study, the (DM+MetS) group had higher levels of serum FBS, TG, cholesterol, and BMI than the control group, while their serum HDL-C level was decreased.

An inverse correlation has been discovered between obesity and adiponectin, which identifies this adipokine as a negative marker for metabolic syndrome. Today, it is well-known that this adipokine plays an important role in type 2 diabetes and dyslipidemias. Its main role is through its insulin-sensitizing effect. Diabetic patients have lower levels of serum adiponectin compared to normal individuals, and the risk of developing type 2 diabetes decreases with an elevation in plasma adiponectin levels (20). This adipokine has anti-diabetic, anti-atherogenic, and anti-inflammatory activities and holds distinct potential as a therapeutic agent for obesity-related diseases (21,22).

In a study by Abdelgadir et al., type 2 diabetic patients were studied with respect to the adipokine effects on glucose metabolism. They discovered that the serum levels of adiponectin were significantly decreased in type 2 diabetic patients (23).

Trevaskis et al. showed that there is a correlation between severe insulin resistance and decreased serum adiponectin in genetically modified mouse models receiving a high-fat diet (24).

Matyjaszek-Matuszek et al. designed a study to explore the correlation between this adipokine and biochemical risk factors for atherosclerosis. They measured adiponectin concentration in blood samples from women with gestational diabetes mellitus. This research did not show a significant difference in adiponectin levels between the groups during gestation. Regarding the lipid profile, there was a notable difference between the gestational diabetes mellitus group during gestation and the control group. The concentrations of TG and LDL-C were elevated, while HDL-C was significantly decreased in comparison to the healthy group (25).

Gene knockout mice have been used to investigate the role of adiponectin in insulin resistance. Despite having normal insulin levels, these mice could not modulate blood glucose levels. This finding highlighted the role of adiponectin in glucose tolerance (21).

In rodents, the administration of adiponectin led to a reduction in resting blood glucose levels (9,26), inhibited insulin resistance caused by diet-induced obesity (27), and protected ob/ob mice from the development of diabetes (26).

Based on our results and the data mentioned above, it may be concluded that adiponectin plays a role in metabolic syndrome and diabetes development by regulating FBS, TG, and HDL-Cholesterol levels.

Leptin and its association with diabetes and metabolic syndrome have also been studied previously.

Mirza et al. demonstrated a strong association between diabetes and elevated leptin levels. Furthermore, data analysis of the lipid profiles showed that diabetic patients had higher levels of TG, while HDL levels were noticeably decreased (28).

Buyukbese et al. conducted a study to investigate leptin levels in obese female patients with type 2 diabetes. They reported that leptin levels were lower in obese women with diabetes than in those without diabetes. TG levels were higher, while HDL-C levels were lower compared to the control group. They also identified a negative correlation between leptin and TG (29).

Garcia-Jimenez et al. investigated the correlation between the circulating levels of this adipokine and metabolic syndrome. Their results indicated that leptin levels were significantly higher in patients with metabolic syndrome than in those without metabolic syndrome. This investigation confirmed the association between leptin levels and metabolic syndrome, particularly in relation to obesity and insulin resistance. Furthermore, they found a positive association between excess visceral fat and the lipid profile (TG, LDL, and cholesterol), while this association was inverse for HDL (30).

Balasoiu et al. examined 80 participants with metabolic syndrome and demonstrated that the levels of proatherogenic adipocytokines, particularly leptin, were elevated in patients with metabolic syndrome. Leptin resistance demonstrated the pathogenic link to obesity (31).

Mohammadzadeh et al. showed that patients with type 2 diabetes had significantly lower levels of leptin than the control group (32).

Therefore, the available reports regarding the role of leptin and its correlation with the lipid profile (Especially TG) in diabetes are inconsistent. Our data revealed that in type 2 diabetic patients, with (Not significantly) and without metabolic syndrome (Significantly), the serum leptin levels were lower than those in the control group. Numerous variables, including insulin sensitivity, insulin level, adiposity, BMI, and gender, affect serum leptin levels. Variations in these factors among different studies may explain the conflicting reports. In addition, in our investigation, the reduced leptin levels in diabetic patients may be attributed to pancreatic dysfunction.

Conclusion

According to the results of the present study, both adiponectin and leptin levels were lower in type 2 diabetic patients with metabolic syndrome compared to healthy subjects. This finding suggests that the reduced levels of these adipokines, especially adiponectin, could be used as predictive markers of type 2 diabetes. Identifying the disease at early stages allows for effective control, reducing morbidity and mortality.

Acknowledgement

Not applicable.

Funding sources

This study was a research project supported by the Golestan University of Medical Sciences (Grant No. 9003170123).

Ethical statement

Ethical approval for this research study was granted by the Golestan University of Medical Sciences Ethics Committee (10059004135). All procedures were performed in accordance with the guidelines for studies involving human participants, considering the ethical standards of the institutional and/or national research committee, as well as the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. During data collection, the researchers obtained informed consent from the participants after explaining the purpose and objectives of the study.

Conflicts of interest

The authors declare that they have no conflict of interest.

Author contributions

All authors have accepted responsibility for the entire content of this submitted manuscript and approved the final version. HRJ and ShH conceptualized the study, provided the project design, and interpreted the data. NB analyzed the data. KhGh was a major contributor in collecting the serum and data. ZH and FF interpreted the data, drafted, and wrote the manuscript. N.H collected the data and contributed to writing the manuscript.

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How to Cite:

Hezarkhani Sh, Ghojoghi Kh, Hosseinzadeh S, Behnampour N, Fathabadi F, Hesari Z, Joshaghani HR. Evaluation of serum adiponectin and leptin levels in type 2 diabetic patients: The potential role in predicting metabolic syndrome. *Med Lab J.* 2024;18(6):28-31.