Original Article

Study Effect of Periostin Level in Obese Iraqi Females Patients with Type 2 Diabetes Mellitus

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Abstract

Background: Obesity has been connected to a higher risk of acquiring a number of diseases, including cancer, type 2 diabetes mellitus (T2DM), hypertension, and cardiovascular disease. Periostin is a crucial regulator of the growth and maintenance of bones, teeth, and the heart. **Objectives:** The aim of the study was to estimate the level of (periostin, glycated hemoglobin [HbA1c], fasting serum [FBG], total cholesterol [TC], high-density lipoprotein [HDL], low-density lipoprotein [LDL], and triglycerides [TG]) in diabetic Iraqi women obese and nonobese. **Materials and Methods:** Ninety participants were chosen for this study: 30 nonobese female patients with T2DM (nonobese-T2DM), 30 obese female patients with T2DM (obese-T2DM), and 30 healthy participants. Age ranges between 35 and 65 years. **Results:** The result of the present study found a highly significant increase in the (body mass index [BMI], FBG, HbA1c, TC, TG, LDL, and periostin) in obese-T2DM and nonobese-T2DM when compared with the healthy control group, whereas HDL was lower in obese-T2DM and nonobese-T2DM when compared with the healthy control group. **Conclusion:** The current investigation discovered, in summary, that plasma periostin may function as a novel marker for insulin resistance, obesity, and inflammatory responses, which might be used to diagnose T2DM and obesity.

Keywords: Lipid profile, obesity, periostin, type 2 diabetes mellitus (T2DM)

Introduction

Diabetes mellitus (DM) is a metabolic condition characterized by a persistent rise in blood sugar and varied degrees of dysfunction in the metabolism of proteins, lipids, and carbohydrates.[1] Obesity is a chronic condition, according to the World Health Organization, marked by an excessive amount of adiposity, which has detrimental effects on health since fat mass is metabolically active.[2] A body mass index (BMI) of (30 kg/m^2) or more is considered obese, and there are over 108 million obese children and adults worldwide. Although excessive food consumption and a sedentary lifestyle are the main causes of increasing obesity, there hasn't been much development in creating effective therapies for slimming down adiposity and body weight.[3] Obesity persons are more likely to develop conditions including renal insufficiency, cardiovascular diseases, endothelial dysfunction, T2DM, and certain malignancies.[4] Obesity is closely linked to insulin sensitivity and inflammation, which prevents insulin from acting on the body's primary insulin-sensitive tissues, such as the liver, muscle, and adipose tissue. Insulin resistance (IR) causes extravascular stimulation and intra-arterial inflammation, which in turn triggers the release of pro-inflammatory cytokines, which in turn aid in the development of illness.[5] The most frequent metabolic abnormalities seen in clinical practice are caused by adipose tissue endocrinopathies, immunopathies, and lipotoxicity. Elevated blood sugar is one of the adiposopathic effects of obesity, which shows up clinically as T2DM and prediabetes.[6,7] The T2DM risk increases with longer periods of obesity. A risk factor for diabetes is the distribution of body fat, which increases risk independently in women more than males; with

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weight loss, physical exercise can lower the risk of diabetes. At lower levels of obesity, those with a positive family history of diabetes have the same risk as those who are less vulnerable.[8,9] Since its first discovery in 1993, periostin, a matricellular protein, has been the focus of several investigations in scientific studies.[8] Periostin is a (93-kDa) matricellular protein that has been found as a cell adhesion protein. It is also known as osteoblast-specific factor 2.[7,10] This protein has 835 amino acids and is situated on chromosome 13 in the human body.[11] Periostin is created by a number of cytokines and may help to maintain or worsen inflammation.[12] It is spontaneously triggered throughout a number of pathological events to encourage cell migration, proliferation, tissue fibrosis, differentiation, and inflammation. Periostin promotes damage healing in a variety of tissues and serves an important physiological purpose.[13] The aim of the study was to estimate the level of (periostin, glycated hemoglobin [HbA1c], fasting serum [FBG], total cholesterol [TC], high-density lipoprotein [HDL], low-density lipoprotein [LDL], and triglycerides [TG]) in diabetic Iraqi women obese and nonobese.

Materials and Methods

The case–control study of Iraqi female patients to estimate the level of periostin in obese T2DM between (September 2022 and November 2022). This study was conducted at the National Diabetes Center/Mustansiriyah University. Ninety participants were chosen for this study: 30 nonobese female patients with T2DM (nonobese-T2DM), 30 obese female patients with T2DM (obese-T2DM), and 30 healthy participants. Age ranges between 35 and 65 years. After an 8–12-h fast, blood samples were taken between 8:30 and 11:30 am using a 10 mL disposable syringe. The blood was collected from each participant and then divided into two tubes. The first tube containing EDTA was used for measurement of the HbA1c test. And the other gel tube to estimate FBG, TC, TG, HDL-C, and periostin. The biochemistry parameters' (HbA1c, FBG, TC, HDL-C, LDL-C, and TG) values estimating was in Cobas c111 instrument, and the kit of periostin was using (ELISA) kits (enzyme-linked immunoassay) (Al-Shkairate establishments Sweilleh, Jordan). The LDL was calculated by (Friedewald formula). By dividing the weight by the square of the height square (m^2), the BMI was determined.

Statistical analysis

In order to conduct the statistical analysis, SPSS (version 26 by IBM, New York) was used. The median (25th and 75th percentiles) was used to analyze the data. A test called (Shapiro–Wilk) was used to look at the data's normal distribution. Numerical variables with irregular distributions were described using the Mann–Whitney and Kruskal–Wallis tests. The (receiver operating characteristic curve) analysis was used to estimate the periostin cutoff value.

Ethical Approval

The National Diabetes Center at Mustansiriyah University's Human Ethics Committee provided approval before the study could be carried out. The Ethical Committee's activities were conducted in accordance with the Helsinki Declaration. All of the participants provided written informed consent prior to being enrolled in the study.

Results

The result of the present study found a highly significant ($P < 0.001$) increase in the median (25th and 75th percentiles) of BMI, FBG, HbA1c, TC, TG, VLDL-C, LDL-C, and periostin in obese-T2DM and nonobese-T2DM when compared with the healthy group, whereas HDL-C was lower in obese-T2DM and nonobese-T2DM when compared with the healthy group.

All groups of patients (obese-T2DM and nonobese-T2DM) and control showed significant differences ($P < 0.05$) among groups themselves in BMI, HDL, and periostin. Both groups of patients) obese-T2DM and nonobese-T2DM) showed a significant difference ($P < 0.05$) in FBG, HbA1c, TC, TG, VLDL-C, LDL-C compared to control groups, whereas no significant difference was found between patient groups themselves as shown in Table 1.

Table 1: The comparison between obese-T2DM, nonobese-T2DM patients, and healthy group

Variables	Obese-T2DM	Nonobese-T2DM	Healthy	*P* value
Age (year)	45.00 (36.75–50.00)	48.00 (39.00–55.00)	44.00 (40.00–50.00)	0.06
BMI (kg/m^2)	34.19 (32.40–35.28)a,c	27.39 (26.75–28.36)b,c	24.27 (23.42–25.0)	0.00
FBG (mg/dL)	264.00 (229.00–329.25)a	216.50 (159.00–282.25)b	87.0 (81.75–89.25)	0.00
HbA1c (%)	9.60 (9.0–10.0250)a	9.30 (8.90–10.30)b	5.0 (4.67–5.02)	0.00
TC (mg/dL)	286.60 (264.5–310.0)a	278.00 (265.0–310.0)b	147.00 (124.5–153.0)	0.00
TG (mg/dL)	205.0 (175.68–260.77)a	209.0 (177.50–266.0)b	87.50 (70.0–90.0)	0.00
HDL-C (mg/dL)	31.00 (28.50–33.60)a,c	33.0 (29.75–40.0)b	47.00 (45.75–49.0)	0.00
VLDL-C (mg/dL)	41.00 (35.08–52.16)a	42.13 (35.65–57.60)b	17.50 (14.00–18.0)	0.00
LDL-C (mg/dL)	210.50 (200.90–327.43)a	203.10 (200.43–338.80)b	80.00 (59.50–88.50)	0.00
Periostin (ng/mL)	1387.75 (1156.38–1579.79)a,c	1085.89 (849.48–1201.72) b,c	444.58 (423.14–487.24)	0.00

The collected data were analyzed by median (25th and 75th percentiles) via the Mann–Whitney test at the 0.05 level, and there was a significant difference between the two independent means

Table 2: Periostin (AUC and validity) in distinguishing in the study groups

Variables	Obese-T2DM and control	Nonobese-T2DM and control	Obese-T2DM with nonobese-T2DM
AUC	0.97	0.996	0.822
Cutoff value	>768	>610	>1119
Sensitivity	96.7	100.00	93.33
Specificity	100	93.33	60.00
Accuracy	0.9667	0.9333	0.5333
NPV	96.8	100.0	90.0
PPV	100.0	93.7	70.0
P-value	0.001	0.0001	0.0001

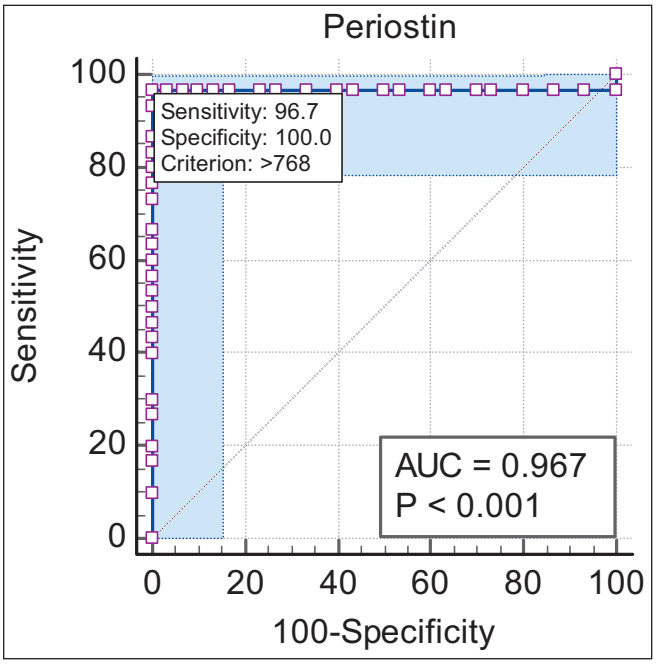

Figure 1: The ROC curve analysis to investigate the predictive value of periostin serum levels in obese-T2DM versus control

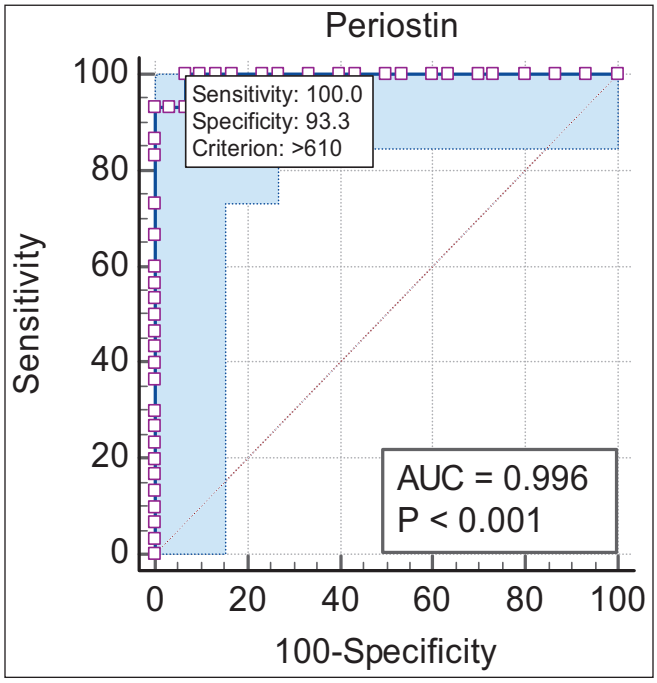

Figure 2: The ROC curve analysis to investigate the predictive value of periostin serum levels in nonobese-T2DM versus control

Periostin showed an excellent ability (since AUC is between 0.9 and 0.99) to identify (obese-T2DM and control) and (nonobese-T2DM and control). Periostin showed a good ability (since AUC is between 0.8 and 0.89) to identify (obese-T2DM with nonobese-T2DM). Periostin showed an excellent ability to (obese-T2DM and control) and (nonobese-T2DM and control); it had sensitivity ([96.7], [100]) and very high specificity ([100], [93.33]), and accuracy ([0.9667], [0.9333]). In terms of the posterior probability, both the PPV is very high ([100.0], [93.7]) and the NPV was very high ([96.8], [100.0]), as illustrated in Table 2 and Figures 1–3, respectively.

Discussion

Obesity poses a major public health risk and contributes significantly to the prevalence of noncommunicable diseases around the globe, including cardiovascular disease, T2DM, some cancers, and hypertension. Obesity is linked to early death.[14] The adipokines and exosomes, which are produced and secreted by adipose tissue, play a role in the control of vital physiological processes like insulin action, reproductive function, and hunger.[15] The results of the study found an increase in (TC, TG, LDL-C, and VLDL-C). This result agrees with other studies achieved by Mirjalili *et al.*[16] and Ahmed *et al.*[17] Increased fat accumulation in the adipose tissues caused by obesity eventually limits their ability to store extra calories. Adipose cells now produce free fatty acids (FFAs) through increased lipolysis, which stay in the bloodstream. It causes elevated FFA levels throughout the body, which in turn encourages hepatic and muscular insulin resistance (IR) as well as decreased ß-cell insulin release in the pancreas.[18] Increased liver VLDL-C production or decreased removal of VLDL-C and LDL-C from circulation are the two causes of the rise in VLDL-C and LDL-C. Due to a reduction in clearance from circulation, serum triglyceride content also rises.[5,19] Serum HDL-C levels drop as a result of excessive metabolism, and there is a bad correlation between HDL-C concentration and LDL concentration.[20] A matricellular protein called

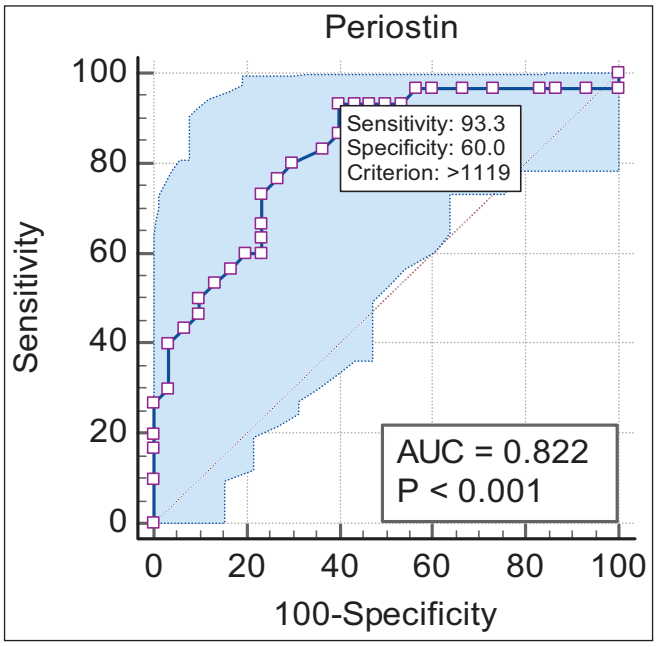

Figure 3: The ROC curve analysis for examine the predictive value of periostin serum levels in obese-T2DM versus nonobese-T2DM

periostin actively adds to inflammation, atherosclerosis, fibrosis, and tissue damage. Wu *et al.*[21] found that rodents given a high-fat diet had significantly higher levels of periostin expression in their livers. This characteristic was also seen in obese mice and (nonalcoholic fatty liver disease [NAFLD]) patients, demonstrating that periostin overproduction in the liver is a conserved phenotype in both obese animals and obese people. It's interesting to note that elevated glucose levels caused the expression of hepatic periostin and that overexpressing periostin livers led to hepatic (TG) accumulation and higher liver weight. In addition to glucose, hypoxia and cytokines can also cause periostin to be produced.[22] It is uncertain whether periostin is involved in the transition from liver steatosis to steatohepatitis despite the fact that it actively adds to the development of numerous inflammatory diseases and fibrosis in many tissues.[23] In addition, Luo *et al.*[24] found that serum periostin significantly increased T2DM and obesity in Chinese patients. And that insulin resistance, chronic inflammation, and TG metabolism are all highly correlated with periostin.[25] It is well known that the liver can control lipid and glucose metabolism, both of which are essential for sustaining energy balance during fasting transitions.[26] Additionally, excessive liver and intestinal lipoprotein synthesis can result in hypertriglyceridemia and raise levels of (free fatty acids), both of which can lead to insulin resistance by lowering glucose-6-phosphate intracellular levels.[27-29]

Conclusion

The level of periostin can be impacted by T2DM. In the diagnosis of obesity with T2DM, periostin is the most sensitive and specific marker. Periostin may function as a novel marker for insulin resistance, obesity, and inflammatory responses, which might be used to diagnose type 2 diabetes (T2DM) and obesity.

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Conflicts of interest

There are no conflicts of interest.

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