The Association between Adiponectin, Adiponectin Receptor, and Obesity for Diabetic Female Type II

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ABSTRACT

Aims Adiponectin is associated with obesity and unregulated diabetes, although it may be useful considering the consequences of glycemic control and obesity on Adiponectin. This study investigated the association between Adiponectin, Adiponectin Receptor, and Obesity for Diabetic Female Type II.

Material & Methods In this research, 54 female patients with type 2 diabetes and 36 non-diabetics participated, individuals from two groups, obese and non-obese, compared with apparently healthy control. The Adiponectin and Adiponectin receptor were measured by ELISA sandwich and Colorimetric methods were used for calculating fasting blood glucose. Blood samples were collected and drawn (5 ml) for each individual. For statistical analysis, SPSS 26.0 by ANOVA and Pearson's correlation coefficients were used.

Findings Adiponectin and the Adiponectin receptor were measured, and the values of both were decreased in patients with type 2 diabetes who have the highest FBG, and also decreased for obese females, which have the highest BMI, where the positive significant values were observed when correlated body mass index (r=-0.57, p=0.001), and fasting blood glucose (r=0.291, p=0.0.06), while a negative correlated observed between Adiponectin receptor (r=-0.59, p=0.203) and Adiponectin (r=-0.201, p=0.158).

Conclusion The adipo levels, which consider a pro-inflammatory marker for obesity enhance complications such as metabolic syndrome, insulin resistance, ischemic heart disease, and diabetic renal disease for women obese diabetes patients compared with non-obese diabetes patients.

CITATION LINKS

[1] Adipose-tissue plasticity in health and disease [2] Adiponectin suppresses tumor growth of nasopharyngeal carcinoma through activating AMPK signaling pathway [3] Cancer related anemia: an integrated multitarget approach and lifestyle interventions [4] Adiponectin activates adenosine monophosphate-activated protein kinase and decreases luteinizing hormone secretion in LβT2 gonadotropes [5] Adiponectin and its mimics on skeletal muscle: insulin sensitizers, fat burners, exercise mimickers, muscling pills ... or everything together? [6] Physiological and pathophysiological roles of adiponectin and adiponectin receptors in the integrated regulation of metabolic and cardiovascular diseases [7] Type 2 diabetes–an autoinflammatory disease driven by metabolic stress [8] Clinical implications of adipocytokines and newly emerging metabolic factors with relation to insulin resistance and cardiovascular health [9] More than a simple storage organ: adipose tissue as a source of adipokines involved in cardiovascular disease [10] Relation of serum adiponectin levels to number of traditional atherosclerotic risk factors and all-cause mortality and major adverse cardiovascular events (from the Copenhagen City Heart Study) [11] The role of adiponectin in obesity, diabetes, and cardiovascular disease [12] The Association between Lipocalin 2 and obesity for diabetic Female Type II [13] The association between known risk factors for type 2 diabetes, and the body mass index of diabetic adults [14] Relationship of adiponectin to body fat distribution, insulin sensitivity and plasma lipoproteins: evidence for independent roles of age and sex [15] Relationship between serum adiponectin and leptin concentrations and body fat distribution [16] The metabolically obese, normal-weight individual revisited [17] Associations of adiponectin levels with incident impaired glucose metabolism and type 2 diabetes in older men and women: the hoorn study

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Introduction

Metabolic syndrome is thought to be caused by obesity. Where obesity in adulthood is characterized by the occurrence of hypertrophy of adipocytes. In the regulation of energy homeostasis, adipose tissue is involved as an important endocrine organ and secretes a number of "bioactive lipoproteins [1].

Adiponectin and adiponectin receptors (AdipoRs) have been found to have important roles in chronic diseases and are a pathogen associated with obesity [2]. The functional and genetic studies confirm that adiponectin is a therapeutic target adipokine. Several interactions and roles with some other biomolecules (MCP-1) have been clearly identified to form a sinister adipokine network that is a cause of obesity-related insulin resistance and metabolic syndrome; PPARγ regulates HMW adiponectin and PPARα (Peroxisome proliferator-activated receptor α) regulates AdipoRs; dietary osmotin may act as a natural adiponectin receptor agonist [3].

Under starvation conditions, MMW (Low-molecular-weight adiponectin) activates AMPK (AMP-activated protein kinase). in the hypothalamus, food intake enhances, and at the same time, HMW adiponectin activates AMPK in peripheral tissues, such as skeletal muscle, and stimulates the combustion of fatty acids [4, 5].

Pathophysiological conditions, such as diabetes and obesity, have only decreased HMW adiponectin; Therefore, strategies to increase HMW adiponectin only lead to a logical approach that may provide a new treatment modality for obesity-related diseases, such as insulin resistance, type 2 diabetes, and atherosclerosis. It is hoped that this data will be useful in developing treatments to counteract the devastating, painful, and costly effects of obesity [6].

Hypertrophic adipocytes that have been associated with obesity with increased MCP-1, and decreased adiponectin action constitute a sinister adipokine network to causes insulin resistance, obesity-related diabetes, and metabolic syndrome. Strategies for activating the adiponectin/AdipoRs pathway may provide a new treatment modality for insulin resistance, metabolic syndrome, and atherosclerosis [7].

there are Multiple mechanisms linking obesity with CVDs [2, 8]. Many adipokines mediate the cross-talk between vasculature, heart, and adipose tissues in the "adipo-cardiovascular axis"; the altered release of adipokines promotes a prothrombotic state contributing to atherosclerosis disease [9]. Some studies indicate that adiponectin has a beneficial role in CVDs and atherosclerosis. Low serum adiponectin levels are predictors of myocardial infarction atherosclerosis [10].

The relationship between obesity and type 2 cardiovascular disease. It has now become a new concern. Adiponectin is a collagen plasma protein that is secreted by adipocytes that are responsible for the development of insulin resistance and hematological diseases. Studies have found that low protein leads to insulinomas, diabetes, atherosclerosis, and coronary artery disease. Up-regulation of adiponectin and its receptors is partly related to insulin sensitivity to antidiabetic drugs. We discuss the anti-hormone antagonism of adiponectin, its association with insulin resistance and obesity, and the use of adiponectin and its receptors as a therapeutic measure [11, 12].

High BMI is one of the risk factors associated with T2DM And the unhealthy diet followed gives an inactive body Low socioeconomic status are factors that contribute to both obesity and T2DM where obesity was diagnosed when BMI measurements were used alone. People who are obese suffer T2DM and this leads to poor control of blood sugar when exacerbated by diabetes [13].

This study investigated the association between Adiponectin, Adiponectin Receptor, and Obesity for diabetic female Type II.

Material and Methods

Individuals were divided into two groups. The effect, the Tuck group was also divided into two parts. The research was conducted for a case-control study in the endocrinology center of Al-Murjan Al-Talafi Hospital in Al-Hilla, Babylon, Iraq. The following lists were of patients and Healthy people: age 26 to 70 years with type 2 DM who visited the diabetes study center there were interrogators and laws with both individuals and patients. The search was done using parameters to collect; pathological sex and. Weights and background. Body mass index (BMI) was measured as an average weight of 23.11-26.69kg/m² or obesity >30kg/m² according to the classification of the World Health Organization.

Blood samples for patients and observers were collected and drawn (5 ml) for each individual. And blood samples are allowed to coagulate and then use a centrifuge where it was centrifuged after 10 minutes at 4500rpm. The walk was separated and stored deeply Frozen until analysis.

For statistical analysis, statistics from the SPSS version (V.26.0) were used. The results were used as means±SD, ANOVA to find out the difference between groups two average semesters were compared with students' ratings. As for the relationships between variables, they were compared using Pearson's correlation coefficients. P-values two-tailed were scored considered significant at 0.05.

Findings

The research was including ninety women classified into two groups, 36 female monitors (18 obese and 18 non-obese) and 54 diabetic women (27 obesity, 27 non-obesity) patients. In Tables 1 and 2, the studied contributors were faced with biochemical

and clinical variables. In the patient group, Adipo was significant compared with controls (p<0.05). AdipoR1 was not significant compared with controls (p>0.05). FBS was significant in patients relative to controls (p<0.05), and BMI was non-significant compared with controls (p>0.05).

Table 1) Clinical properties for DM2 patients and controls

Parameters	Patients	Control	p-value
Age (years)	48.80±13.13	52.48±13.41	0.129
BMI (kg/m²)	30.00 ±5.40	27.82±2.69	0.06
Duration of disease (years)	8.15±7.32	-	-
Duration of medication (years)	6.02±6.06	-	-
FBG (mg/dL)	191.52±71.95	81.90±12.80	0.001

Table 2) The comparison of patient and control groups for Adipo (ng/mL)

Groups	Mean± SD	CI 95%		Compared groups		p-value
		Lower	Upper			
Po	16.27±2.59	14.9733	17.5574	Po	Pnon	0.052
					Co	0.668
					Cnon	0.001
Pnon	32.50±11.99	26.5454	38.4732	Pnon	Co	0.198
					Cnon	<0.001
Co	15.45±3.575	14.0391	16.8682	Co	Cnon	<0.001
Cnon	18.85±1.78	18.1495	19.5537			

Po: Patients obese, Pnon: patients non-obese, Co: controls obese, Cnon: controls non-obese The mean difference is significant at P≤0.05.

Adipo level comparison between different groups in Table 2. Adipo was significantly decreased in the patients obese compared to patients with non-obese (p=0.052) and controls (p<0.001); while Adipo levels were not significantly elevated in the Pnon compared to controls (p=0.001).

Table 3) The Comparison of Patient and Control Groups for AdipoR1 (ng/mL)

Groups	Mean±SD	CI 95%		Compared groups		P-value
		Lower	Upper			
Po	19.90±3.02	18.40	21.40	Po	Pnon	0.007
					Co	0.048
					Cnon	0.001
Pnon	29.25±7.98	25.28	33.22	Pnon	Co	0.692
					Cno	0.001
Co	16.85±3.91	15.31	18.40	Co	Cnon	0.001
Cnon	20.43±2.34	19.51	21.36			

AdiporR1 level comparison between different groups in Table 3. AdipoR1 was significantly decreased in the patients obese compared to patients non-obese, (p=0.007). Similarly, the patients were obese compared to controls non-obese (p=0.048). while AdipoR1 levels were not significantly elevated in the Pnon compared to control obes, (p=0.692). and for Pnon compared with Cnon (p=0. 001).
FBS levels and BMI were compared between different groups of patients and controls as illustrated in Table 4. Obese patients have low FBS (80.59±14.90) with BMI (33.16±2.81) compared to non-obese patients with FBS (83.11±10.76) with BMI (22.47±2.57), according to criteria compared to obese control FBS (177.68±64.74) with BMI

(33.64±2.86) compared another non-obese control FBS (207.02±71.50) with BMI (25.39±2.62).
Adipo in Table 5 shows a positive correlation with diabetes duration FBG, BMI Duration of medication, Duration of medication, Age, and Adipo R1.
Adipo and FBS in Table 6 show, weak associated with Duration of medication and positive correlation for parameter.

Table 4) The FBS Levels (mg/dL) and BMI (kg/m²) for Patient Groups Compared to Control Groups 2

Groups	Mean±SD	CI 95%		Compared groups		p-value
		Lower	Upper			
FBG						
Po	80.59±14.90	73.1835	88.0120	Po	Pnon	0.026
					Co	0.526
					Cnon	0.001
Pnon	83.11±10.76	77.7599	88.4690	Pnon	Co	0.001
					Cnon	0.07
Co	177.68±64.74	152.0703	203.2956	Co	Cnon	0.001
Cnon	207.02±71.50	178.7320	235.3080			
BMI						
Po	33.16±2.81	31.7626	34.55	Po	Pnon	0.48
					Co	0.001
					Cnon	0.001
Pnon	22.47±2.57	21.1959	23.75	Pnon	Co	0.001
					Cnon	0.001
Co	33.64±2.86	32.5178	34.78	Co	Cnon	0.865
Cnon	25.39±2.62	24.3573	26.43			

Table 5) The correlation between Adipo and variables for obese diabetic patients

parameters	r	p
Age (years)	0.086	0.670
BMI (kg/m²)	0.066	0.743
Duration of disease (years)	0.187	0.349
Duration of medication (years)	0.281	0.115
FBG (mg/dL)	0.044	0.827
Adipo R1	0.778	0.111

Table 6) The correlation between Adipo and variables for non-obese diabetic patients

Parameters	r	p
Age (years)	0.120	0.552
BMI (kg/m²)	0.277	0.162
Duration of disease (years)	0.191	0.341
Duration of medication (years)	0.018	0.928
FBG (mg/dL)	0.066	0.742
Adipo R1	0.127	0.529

Discussion

Kawano and Arora, are found a negative correlation was found between plasma adiponectin levels and body mass index (BMI) in men and women. Plasma adiponectin concentration was negatively correlated with percentage body fat and waist-high ratio, while also having an inverse relationship with fasting plasma insulin concentration. The study helped to confirm a link between obesity and type 2 diabetes in association with low plasma adiponectin concentration [11].
The result of Cnop et al also shows that plasma adiponectin concentration is more closely related to insulin sensitivity and fasting insulinemia than to glycemia and adiposity. The results suggested that type 2 diabetes and obesity in patients with low

adiponectin levels were in large part attributable to insulin resistance and/or hyperinsulinemia [14, 15].

Researchers have found that the adiposity associated with low circulating levels of adiponectin contains the expansion of visceral and abdominal adipose tissue rather than total body adiposity [7, 8] The importance of this is related to the fact abdominal fat expansion has a greater effect on insulin resistance than total obesity [16].

The study associated a lower adiponectin level with a higher risk of type 2 diabetes The relationship was found through fasting and post-load glucose levels. Increased adiponectin levels were also associated with a lower risk of impaired glucose metabolism in women. The results are shown in this study propose that levels of adiponectin are in some way complicated in the pathophysiology linking adiposity and type 2 diabetes [17].

Conclusion

The conclusion of our study suggested that adipo levels, which consider a pro-inflammatory marker for obesity enhance complications such as metabolic syndrome, insulin resistance, ischemic heart disease, and diabetic renal disease for women obese diabetes patients compared with nonobese diabetes patients.

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