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Concentration of adiponectin in relation to insulin resistance in patients with type 2 diabetes

Stężenie adiponektyny a insulinooporność u pacjentów z cukrzycą typu 2

INTRODUCTION

The prevalence and magnitude of type 2 diabetes (T2D) have risen dramatically over the past few decades. T2D is now considered to be one of the major metabolic diseases of 21st century. It has been identified also as a common precursor to the development of cardiovascular disease [11,12,17,28].

The excessive intake of food, sedentary life style and lack of physical activity are responsible for the continually growing obesity epidemic, together with the increasing rate of type 2 diabetes in many parts of the world. Recent estimates suggest that around 80-90% of patients with type 2 diabetes are overweight or obese [11,27,31]. Obesity is recognized as the important element in the pathogenesis of type 2 diabetes, inducing both insulin resistance and pancreatic beta-cell dysfunction [5,6,11,25].

However, during the past few years a lot of attention has been paid to the potential role of adipose tissue, in particular visceral fat in the development of type 2 diabetes [17,24]. Since the discovery of leptin, derived from ob gene in 1994, it has been known that adipose tissue is not only a passive depot storing triglycerides but also an active endocrine organ secreting a variety of hormones and cytokines known as adipocytokines, which may influence the function of many systems [16,24-26,38].

One of these proteins that have attracted a lot of attention recently is adiponectin, the most abundant and adipose-specific adipocytokine with pivotal biological effects [10,27,32,33]. Many clinical studies have suggested that low levels of adiponectin contribute to insulin resistance associated with obesity thus increasing the risk of developing type 2 diabetes [4,8,13,36].

The aim of this study was to compare serum adiponectin concentration between overweight and obese patients with type 2 diabetes and normal-weight subjects with no disturbances in carbohydrate metabolism and to evaluate the relationship between serum adiponectin concentration and metabolic parameters, such as insulin resistance indexes (IRI/G, HOMA-IR) and Quantitative Insulin Sensitivity Check Index in diabetic patients.

MATERIAL AND METHODS

PARTICIPANTS. Forty-five patients with type 2 diabetes, who were hospitalized at the Endocrinology Clinic of the Independent Public Clinical Hospital No. 4 (SPSK 4) in Lublin, were enrolled in this study. The examined group consisted of 22 women and 23 men with mean age 60.1±10.3 years. The average duration of the disease from diagnosis was *10.3*±8.1 years. All patients underwent clinical examination. In the examined group of patients in medical history the following were found: arterial hypertension (80%), coronary artery disease (29%) and myocardial infarction (20%). The mean body mass index (BMI) in diabetic patients was 32.9±5.04 kg/m^2. According to the World Health Organization criteria based on BMI, overweight (BMI 25-29.9 kg/m^2), obesity (BMI 30-39.9 kg/m^2) and morbid obesity (BMI≥ 40 kg/m^2) were present in 33%, 58% and 9% of diabetic patients, respectively. The subjects with underlying disease (cancer, hepatic and renal disorders) were not included in this study. Those with previous history of cerebrovascular accident, myocardial infarction in recent 6 months were also excluded.

The control group comprised 25 healthy subjects matched for age and gender to the study group (14 women and 11 men with the mean age of 57.6±11.9 years), with no disturbances in carbohydrate metabolism and with normal body mass index (21.7±1,9 kg/m^2), undergoing prophylactic examination at the Department of Laboratory Diagnostics of the Independent Public Clinical Hospital No. 1 in Lublin.

Written informed consent was obtained from every participant qualified to enter the study. The study protocol was approved by the local Ethics Committee of Medical University in Lublin.

BLOOD SAMPLING AND MEASUREMENTS. In every subject enrolled to the study concentrations of adiponectin, insulin and glucose were determined in fasting blood samples. Insulin resistance (IR) status was evaluated by calculating IRI/G (Insulin Resistance-Insulin/Glucose) and HOMA-IR (Homeostasis Model Assessment-Insulin Resistance) indexes, which are widely employed in clinical research. To assess insulin sensitivity (IS) status we used QUICKI, Quantitative Insulin Sensitivity Check Index.

The material for the study was the peripheral blood obtained from the cubital vein. Blood samples were drawn in fasting condition (after an 8-12 h overnight fast between 8:00 and 10:00) into the clot tubes. Blood samples 20-30 minutes after collection were centrifuged for 10 minutes at 2000 rpm and obtained serum was separated to the eppendorf tubes and stored at -20^{o}C until assayed.

Fasting serum concentration of glucose was measured by reference method (the hexokinase/glucose-6-phosphate dehydrogenase method) applied on Cobas 6000 analyzer (Roche, Basel, Switzerland) with dedicated reagents from the same company according to the manufacturer's specification. Fasting insulin concentration in the serum was performed with the use of electrochemiluminescence method on Cobas 6000 analyzer (Roche, Basel, Switzerland) using ready kits provided by Roche. The analytical sensitivity of the test was 0.20 µU/ml and within-run precision expressed as the percentage coefficient of variation (CV%) was 1.9%. Serum adiponectin concentration was determined with the use of Human Adiponectin ELISA Kit (BioVendor Laboratory Medicine, Brno, Czech Republic) according to the manufacturer's protocol. The antibodies were specific for the human adiponectin protein, with an assay sensitivity of 210 ng/mL using a 50 µl

sample (diluted 30-times) size. The intra- and inter-assay precisions expressed as CV(%) were in the ranges 6.4-7.0% and 7.3-8.2%, respectively.

Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. The HOMA-IR was calculated as [fasting insulin (μU/ml) × fasting glucose (mmol/l)]/22,5, as described by Matthews et al. [22], IRI/G index as fasting insulin (μU/ml)/ fasting glucose (mg/dl). The QUICKI index was calculated as 1/[log fasting insulin (μU/ml) + log fasting glucose (mmol/l)], previously described by Katz et al. [14].

STATISTICAL ANALYSIS. The clinical data and values of selected biochemical parameters in all subjects were expressed by using elements of descriptive statistics (mean X, standard deviation SD, median Me, percentile range 25%-75%). Distributions of the analyzed variables were tested using the Shapiro-Wilk test. For comparison of obtained results of investigations in the case of normally distributed variables the Student t-test was used. For variables that did not demonstrate compliance with the normal distribution, non parametric test U Mann-Whitney was applied. Correlations between variables were investigated using Pearson's or Spearman's test. A p value $\leq$ 0.05 was considered as statistically significant in all analyses. For statistical analysis of obtained results, Statistica 7.0 StatSoft was used.

RESULTS

Table 1 shows the results of determinations of glucose, insulin and adiponectin concentrations and values of selected insulin resistance indexes in the patients with type 2 diabetes and healthy participants.

In diabetic patients the mean concentration of adiponectin was 5.1±2.2 μg/ml and was significantly lower ($p<0.0001$) compared to the control subjects (9.9±3.2 μg/ml) (Fig.1). However, in the study ($p<0.01$) and control group ($p<0.05$) the females (6.1±2.2 and 10.7±2.6 μg/ml, respectively) had a significantly higher adiponectin concentration than males (4.2±1.8 and 8.8±3.8 μg/ml, respectively).

Furthermore, in diabetic patients we observed significantly higher concentrations of glucose ($p<0.0001$) and insulin ($p<0.0001$) in comparison with healthy subjects. Among the selected insulin resistance indexes, in the patients with type 2 diabetes the values of HOMA-IR (9.2±9.4) and IRI/G (0.12±0.09) indexes were significantly higher ($p<0.0001$and $p<0.001$, respectively) than in the control group (HOMA-IR 1.05±0.9; IRI/G 0.06±0.04). The mean value of QUICKI index in diabetic patients was 0.3±0.05 and was significantly lower ($p<0.0001$) compared to the control subjects (0,4±0,08).

Figure 2 shows the values distribution of insulin concentrations and HOMA-IR index in diabetic patients and healthy participants.

Table 1. Serum concentrations of adiponectin, glucose and insulin and values of selected insulin resistance indexes in the study and control group.

Parameters	Study group (n=45)			Control group (n=25)		
	X±SD	Me	25-75%	X±SD	Me	25-75%
Fasting glucose (mg/dl)	168.3±61.6‡	156.0	132.0-192.0	85.2±9.4	87.0	79.0-92.0
Fasting insulin (µU/ml)	19.9±17.6‡	12.8	8.6-22.5	4.8±4.0	3.9	1.3-5.5
IRI/G index	0.12±0.09†	0.08	0.05-0.14	0.06±0.04	0.05	0.02-0.06
HOMA-IR index	9.2±9.4‡	4.7	2.8-12.8	1.05±0.9	0.8	0.3-1.3
QUICKI index	0.3±0.05‡	0.30	0.27-0.33	0.4±0.08	0.4	0.4-0.5
Adiponectin (µg/ml)	5.1±2.2‡	5.0	3.6-6.3	9.9±3.2	9.0	7.7-11.6

† p<0.001; ‡ p<0.0001

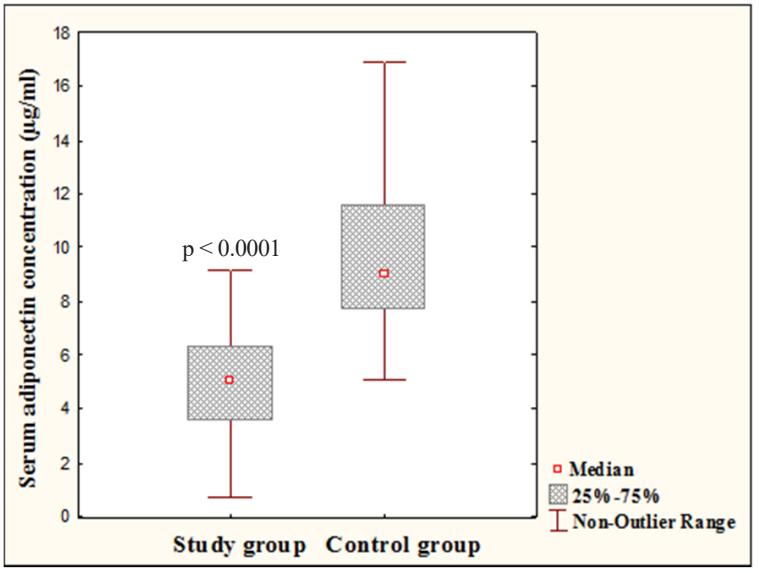

Figure 1. Serum adiponectin concentration in the study and control group.

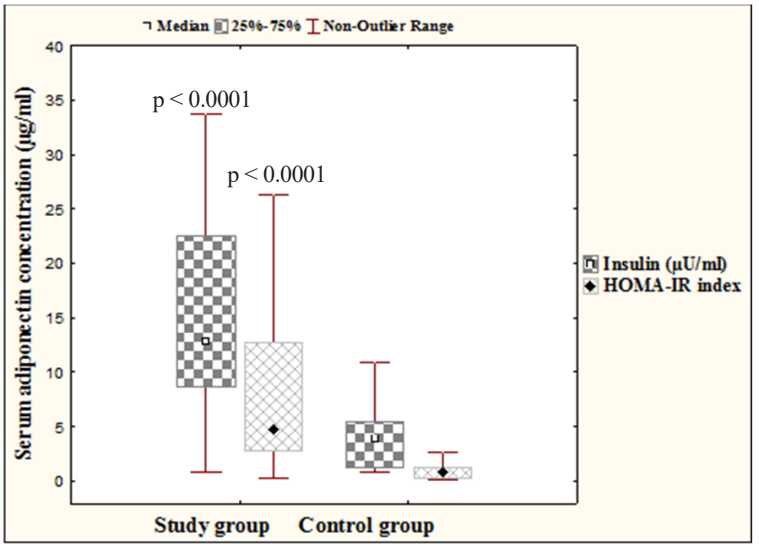

Figure 2. Values distribution of insulin concentration and HOMA-IR index in diabetic patients and healthy participants.

In the present study, we also examined the relationship between serum adiponectin concentration and selected demographic, anthropometric and biochemical parameters and insulin resistance indexes in the group of diabetic subjects (Table 2).

Based on statistical analysis we observed significant negative correlations between adiponectin concentration and body mass (r=-0.435; p=0.003), BMI (r=-0.387; p=0.009), glucose (r=-0.319; p=0.032) and insulin (r=-0.384; p=0.009) level and values of HOMA-IR (r=-0.382, p=0.000) and IRI/G (r=-0.326, p=0.029)indexes. Furthermore, significant positive correlations were found between adiponectin concentration and age (r=0.359, p=0.015) and values of QUICKI index (r=0.382, p=0.000). However, no significant relationships were observed between duration of the disease and adiponectin concentration in diabetic patients.

Table 2. Correlation coefficients of the studied variables with adiponectin concentration in diabetic patients.

PARAMETERS		STUDY GROUP (n=45)	
		Correlation coefficient r	Level p
ADIPONECTIN (µg/ml)	Age (years)	0.359	0.015
	Duration of disease (years)	0.121	0.509
	Body mass (kg)	-0.435	0.003
	BMI (kg/m^2)	-0.387	0.009
	Fasting glucose (mg/dl)	-0.319	0.032
	Fasting insulin (µU/ml)	-0.384	0.009
	IRI/G index	-0.326	0.029
	HOMA-IR index	-0.382	0.000
	QUICKI index	0.382	0.000

p - level of statistical significance (p≤0.05)

DISSCUSION

The relationship between obesity and the development of type 2 diabetes has long been recognized, but the mechanisms for this relationship are still unclear [25]. It is now suggested that adipose tissue plays an important role in insulin resistance through the dysregulated production and secretion of adipose-derived proteins [10,18]. Most authors and our research confirm that a low adiponectin levels are associated with insulin resistance and may play a crucial role in the pathogenesis of type 2 diabetes associated with obesity [3,4,8].

In the present study we demonstrated that concentration of adiponectin in overweight and obese patients with type 2 diabetes, which was statistically significantly lower in comparison with lean healthy participants, was negatively correlated with anthropometric indices (body mass and BMI) and positively with age. It is consistent with the results of scientific studies in the world literature [2,35,36].

Numerous studies highlight the importance of adiponectin as a physiological regulator of glucose homeostasis and insulin sensitivity [20,24,26,34]. The majority of data for animal studies suggest that adiponectin acts as an insulin-sensitizing hormone by decreasing hepatic glucose output and thereby contributing to the regulation of whole-body glucose homeostasis [27,33]. These data are supported by clinical studies in humans [30,35]. Therefore, it is not surprising that hypoadiponectinaemia is associated with insulin resistance in humans [15,36] and that a relationship between low adiponectin level and insulin resistance has been established in type 2 diabetes [8,13].

Although not entirely known, the cellular and molecular mechanisms linking adiponectin to improved insulin sensitivity are likely multifactorial [27]. It is suggested that adiponectin binding with the membrane receptor, occurring mainly in the skeletal muscles and liver, exerts beneficial metabolic action in pathogenesis of insulin resistance increasing oxidation of free fatty acids in muscles and inhibiting lipogenesis and gluconeogenesis in the liver, which causes an increase in insulin sensitivity in these tissues [7,27,32,32].

Data from literature have shown that circulating adiponectin levels are positively correlated with insulin sensitivity evaluated by using different insulin sensitivity techniques [13,14]. The euglycemic-hyperinsulinemic clamp (clamp-IR), the gold standard technique for estimation of insulin resistance, is accurate but difficult to perform and time consuming and can be used only in studies with limited number of type 2 diabetic patients or populations at risk for insulin resistance. Many investigators have studied simple surrogate indices of insulin resistance in comparison with the index assessed by clamp-IR for example, fasting plasma insulin, HOMA-IR, and the fasting glucose-to-insulin ratio. It has been established that HOMA-IR is a useful surrogate index of insulin resistance in diabetic and nondiabetic subjects [19,22,23,37]. Therefore, we determined fasting serum glucose and insulin levels and evaluated the relationship between serum adiponectin concentration and insulin resistance by calculating IRI/G and HOMA-IR and also insulin sensitivity by calculating QUICKI in diabetic patients.

Maeda et al [21] demonstrate that adiponectin-knockout mice develop insulin resistance either independently of diet or only after high-fat and high-sucrose diet, and treating these mice with adiponectin ameliorates their insulin resistance. Furthermore, Hotta et al [9] in a longitudinal study assessed insulin resistance in obese rhesus monkeys. They found negative correlation between adiponectin concentration and insulin resistance, and the obese monkeys with higher plasma levels of adiponectin had less severe insulin resistance.

However, our results and the studies of numerous authors show that serum adiponectin concentration is negatively correlated with fasting glucose, insulin, insulin resistance and positively with insulin sensitivity in diabetic patients [4,20,29,34-37]. A retrospective study of Pima Indians, a unique population with high propensity of obesity and type 2 diabetes, confirmed these results [20]. Low concentrations of plasma adiponectin correlated strongly with reduced insulin sensitivity, and individuals with high concentrations of adiponectin were less likely to develop type 2 diabetes than those with low concentrations. These findings are in agreement with other human clinical studies conducted in Japanese people [4], and Europeans [29]. Moreover, these research studies have suggested that low adiponectin level is a predictor of insulin resistance and type 2 diabetes, independently of adiposity parameters. Interestingly, a study conducted by Abbasi et al [1] also have shown that adiponectin levels are low in insulin-resistant subjects regardless of whether they are obese, suggesting that concentration of plasma adiponectin independently predicts the progression of diabetes. These clinical studies provide an indication that adiponectin may contribute to the development of insulin resistance and type 2 diabetes.

CONCLUSION

Taking into account the available literature reports as well as the results obtained during this study, it can be concluded that decreased adiponectin concentration in overweight and obese patients with type 2 diabetes in comparison to healthy subjects and the observed correlations of adiponectin with selected indexes of insulin resistance (IRI/G, HOMA-IR, QUICKI) may suggest a participation of this protein in the pathogenesis of insulin resistance in diabetic patients.

In particular, further clinical and experimental investigations should illuminate the patho-physiological significance of adiponectin, a potentially promising target for the prevention and treatment of the type 2 diabetes and other diseases. Thus, adiponectin has the potential to become a clinically relevant parameter to be measured routinely in the diabetes clinic of the future.

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SUMMARY

During the past few years a lot of attention has been paid to the potential role of adipose tissue in the development of type 2 diabetes Recent research studies have shown that adipose tissue is not only a passive depot storing triglyceride but also an active endocrine organ, which plays a key role in the integration of endocrine, metabolic, and inflammatory signals for the control of energy homeostasis secreting a variety of hormones and cytokines known as adipocytokines. One of these proteins that have attracted a lot of attention recently is adiponectin. Many experimental and clinical studies have suggested that low levels of adiponectin contribute to insulin resistance associated with obesity thus increasing the risk of developing type 2 diabetes. The aim of this study was to compare serum adiponectin concentration between overweight and obese patients with type 2 diabetes and normal-weight subjects with no disturbances in carbohydrate metabolism and to evaluate the relationship between serum adiponectin concentration and metabolic parameters, such as insulin resistance indexes (IRI/G, HOMA-IR) and Quantitative Insulin Sensitivity Check Index in diabetic patients. Forty-five patients with type 2 diabetes were enrolled in this study (22 women and 23 men with mean age 60.1±10.3 years). The control group comprised 25 healthy subjects matched for age and gender to the study group. In diabetic patients the concentration of adiponectin was significantly lower compared to the control subjects. We observed significant negative correlations between adiponectin concentration and body mass, BMI, glucose and insulin level and values of HOMA-IR and IRI/G indexes. Furthermore, significant positive correlations were found between adiponectin concentration and age and values of QUICKI index. Taking into account the available literature reports as well as the results obtained during this study, it can be concluded that decreased adiponectin concentration in overweight and obese patients with type 2 diabetes in comparison to healthy subjects and the observed correlations of adiponectin with selected indexes of insulin resistance (IRI/G, HOMA-IR, QUICKI) may suggest a participation of this protein in the pathogenesis of insulin resistance in diabetic patients.

Keywords: diabetes, obesity, insulin resistance, adiponectin

STRESZCZENIE

Badania prowadzone w ciągu ostatnich lat wykazały, że istotną rolę w patomechanizmie wielu zaburzeń metabolicznych, w tym cukrzycy typu 2 odgrywa tkanka tłuszczowa. To nie tylko magazyn zbędnej energii, ale również aktywny narząd wydzielania wewnętrznego. Adipocyty biorą udział w regulacji przemian metabolicznych ustroju, reakcjach zapalnych i immunologicznych. Jest to możliwe dzięki substancjom białkowym o działaniu dokrewnym, produkowanych przez tą tkankę i wydzielanych do krwiobiegu, zwanych adipocytokinami. Spośród wielu adipocytokin szczególnym zainteresowaniem cieszy się adiponektyna. Liczne badania przeprowadzone zarówno na modelach zwierzęcych jak i populacji ludzkiej wskazują na istotny związek niskich stężeń adiponektyny z rozwojem otyłości, insulinooporności oraz cukrzycy typu 2. Celem niniejszej pracy była ocena stężenia adiponektyny u pacjentów z cukrzycą typu 2 z towarzyszącą nadwagą i otyłością oraz osób zdrowych bez zaburzeń gospodarki węglowodanowej i z prawidłową masą ciała. Szczególną uwagę poświęcono poszukiwaniu i ocenie zależności pomiędzy stężeniem adiponektyny a wybranymi wskaźnikami oceniającymi insulinooporność (IRI/G, HOMA-IR, QUICKI) w grupie pacjentów z cukrzycą typu 2. Badania przeprowadzono u 45 chorych, u których rozpoznano cukrzycę typu 2 (22 kobiet i 23 mężczyzn w wieku 60,1±10,3 lat). Grupę kontrolną stanowiło 25 osób zdrowych. Stężenie adiponektyny w surowicy krwi w grupie badanej było istotnie statystycznie niższe niż u osób zdrowych. Zaobserwowano istotne ujemne zależności pomiędzy stężeniem adiponektyny a masą ciała, BMI, wartościami glukozy, insuliny oraz wartościami wskaźników insulinooporności IRI/G i HOMA-IR w grupie badanej. Stężenie adiponektyny znamiennie dodatnio korelowało z wiekiem oraz wartościami wskaźnika QUICKI. Obniżone stężenie adiponektyny u chorych z cukrzycą typu 2 z towarzyszącą nadwagą i otyłością w porównaniu do osób zdrowych oraz obserwowane zależności pomiędzy stężeniem adiponektyny a wybranymi wskaźnikami (IRI/G, HOMA-IR, QUICKI) mogą sugerować udział adiponektyny w rozwoju insulinooporności w tej grupie chorych.

Słowa kluczowe: cukrzyca, otyłość, insulinooporność, adiponektyna