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Adiponectin and uric acid in pre-diabetes and early type 2 diabetes mellitus

Vaidotas Urbanavičius[1, 2],

Agnė Abraitienė[1, 2],

Dalius Vitkus[1, 3],

Regina Borovkienė[3],

Zita Aušrelė Kučinskienė[1, 3]

[1] Faculty of Medicine, Vilnius University, Lithuania

[2] Centre of Endocrinology, Vilnius University Hospital Santariškių Klinikos, Lithuania

[3] Centre of Laboratory Diagnostics, Vilnius University Hospital Santariškių Klinikos, Lithuania

Background. The aim of our study was to evaluate adiponectin and uric acid level in subjects with pre-diabetes and to compare it with the levels in newly diagnosed type 2 diabetes and healthy subjects. We also studied associations of adiponectin level with various anthropometric measurements, indices of glucose metabolism, insulin resistance and uric acid concentration.

Material and methods. The study included 48 individuals with pre-diabetes (IGT), 33 newly diagnosed type 2 diabetes mellitus (DM) subjects, and 68 healthy subjects. Blood samples were analysed for fasting blood glucose, insulin, $HbA1_C$, uric acid and adiponectin. According to BMI control subjects were separated into obese (BMI > = 27) and non-obese (BMI < 27) groups.

Results. Mean serum adiponectin levels were not statistically significantly different in IGT and DM groups (11.23 ± 9.72 vs. 9.23 ± 5.61 μg/ml, p = 0.858). Serum adiponectin was significantly lower in IGT group as compared to obese-control group (11.23 ± 9.72 vs. 15.62 ± 10.38 μg/ml, p = 0.004). Comparison of obese and non-obese control groups showed that serum adiponectin level was lower in obese subjects, but the difference was not statistically significant (15.62 ± 10.38 vs. 19.62 ± 12.20 μg/ml p = 0.181). Uric acid concentration did not differ in IGT and DM groups (359.80 ± 95.69 vs. 340.55 ± 95.39 μmol/l, p = 0.249), whereas in obese and non-obese control subjects uric acid concentration was statistically significantly lower. Adiponectin and uric acid showed inverse correlation with indices of central obesity. Analysis of associations between adiponectin and uric acid in the whole study population revealed negative correlation between these parameters, and this association remained statistically significant after controlling for gender (coefficient of partial correlation r_p = -0.225, p = 0.003).

Conclusions. Adiponectin and uric acid levels was similar in IGT and type 2 diabetes groups. Adiponectin levels were significantly lower whereas uric acid concentrations were statistically significantly higher in subjects with impaired glucose metabolism as compared to the healthy controls.

Key words: adiponectin, uric acid, insulin resistance, prediabetes, type 2 diabetes.
There is no potential conflict of interest.

INTRODUCTION

Obesity is a well-known risk factor for type 2 diabetes, and about 90% of the humans affected by this disease are overweight or obese (1). The pathophysiology linking obesity to type 2 diabetes is not completely understood, but adipokines are thought to be involved (2). Adiponectin is a recently discovered protein that seems to be exclusively secreted by adipocytes and is the most abundant adipose tissue-derived protein (3, 4). In contrast to other adipokines (such as leptin, interleukin-6 and others) that are often elevated in obese subjects, adiponectin is reduced (5–7).

In animal studies, adiponectin has been shown to have insulin-sensitizing properties. Administration of adiponectin reversed insulin resistance in various mouse models of obesity and diabetes (8). Injections of recombinant adiponectin protein into mice acutely lowered plasma fatty acids and glucose, as well as improved insulin sensitivity (9). Chronic administration of the recombinant adiponectin in mice also reduced body weight (10). In animal studies adiponectin increased insulin action via effects on hepatic glucose production and by increasing fat oxidation and lowering circulating free fatty acids (10–12).

Lower plasma levels of adiponectin relative to the normal controls were documented in human subjects with obesity (4), insulin resistance (5, 13) and type 2 diabetes (6) in several cross-sectional studies. Results from a few prospective studies suggest that low adiponectin level is predictive of insulin resistance or diabetes (14–17). Although the association of low adiponectin concentration and type 2 diabetes is rather well studied, only few studies investigated adiponectin level in pre-diabetes (e.g. impaired glucose tolerance and impaired fasting glucose) (18, 19). The results of these studies are rather conflicting, one group showing similar levels of adiponectin in pre-diabetes and type 2 diabetes (19), while the other one reporting significantly higher levels of adiponectin in pre-diabetes as compared to type 2 diabetes (18). It seems that association of adiponectin with

Correspondence to: Vaidotas Urbanavičius, Centre of Endocrinology, Vilnius University Hospital Santariškių Klinikos, Santariškių 2, LT-08661 Vilnius, Lithuania. E-mail: vaidotas.urbanavicius@santa.lt

insulin resistance occurs early in obesity development; however it remains unclear if this association is of further importance in prediabetes and early stages of type 2 diabetes.

Several epidemiologic studies have shown elevated uric acid levels to predict increased risk of cardiovascular events (20–22) and the development of type 2 diabetes (23, 24). Subjects with type 2 diabetes and pre-diabetes were shown to have increased levels of uric acid in cross-sectional studies (25), and serum uric acid was positively associated with indices of insulin resistance (26, 27). Some evidences suggest that uric acid may exert a negative effect on insulin resistance and atherogenesis by stimulating inflammation.

The objective of our study was to evaluate adiponectin and uric acid levels in subjects with pre-diabetes and to compare it with the levels in newly diagnosed type 2 diabetes and healthy (normal glucose tolerance) subjects. We also studied associations of adiponectin and uric acid levels with insulin resistance indices. Furthermore, we examined the relationship between serum adiponectin level and uric acid concentration.

MATERIALS AND METHODS

Research design and methods

The present study included 48 individuals with pre-diabetes (impaired glucose tolerance or impaired fasting glucose (IGT)), 33 newly diagnosed type 2 diabetes mellitus subjects (DM), and 68 healthy subjects.

The study protocol was approved by the Lithuanian Bioethics Committee, and all the subjects signed an informed consent form after being informed on the purpose and procedures of the study.

The subjects were evaluated at Vilnius University Faculty of Medicine, the Centre of Endocrinology and the Centre of Laboratory Diagnostics of Vilnius University Hospital Santariškių Klinikos from September 2005 until May 2007. The evaluation involved a full medical history and physical examination, including anthropometric measurements (weight, height, waist and hip circumferences, total body fat mass and percentage measured by bioelectrical impedance (OMRON BF 302 body fat monitor), arterial blood pressure and pulse rate). Subjects were excluded if they had a known history of cardiovascular disease, stroke or transient ischemic attack, uncontrolled hypertension, or any other serious chronic disease requiring active treatment.

Metabolic studies

Venous blood samples were taken in the morning after 12 hours of fasting. With the subject in the sitting position, an intravenous needle was inserted into a forearm vein. Blood samples were drawn for fasting blood glucose, insulin, uric acid and adiponectin. Standard OGTT was performed for all the study subjects: 75 grams of oral glucose load over a 2-minute period was given, and blood samples were obtained again 2 hours after for plasma glucose measurement. Based on the fasting serum glucose and OGTT, categories of glucose tolerance status were defined by WHO 1998 recommendations (28), and study subjects were divided into impaired glucose tolerance or impaired fasting glucose (IGT), type 2 diabetes mellitus (DM) and control (C) groups. According to BMI control subjects were separated into obese (BMI > = 27) (Obese-C) and non-obese (BMI < 27) (Non-obese-C) groups.

Glucose, insulin and uric acid were determined using standard laboratory methods. Serum adiponectin was measured with the radioimmunoassay (RIA) kits following the manufacturer's protocols (Linco Research Inc., St. Louis, MO).

Calculations and Statistical analyses

The body mass index (BMI) was calculated as weight (kilograms) divided by height squared (meters). The homeostasis model assessment of insulin resistance (HOMA-IR), an index of insulin resistance, was calculated using an equation as described (29).

The data were summarized using standard procedures. Descriptive statistics are presented as mean ± SD, unless stated otherwise. The Student's t-test was used to analyse data with normal distribution, whereas Mann-Whitney U test was applied to compare non-parametrically distributed parameters. Spearman rank correlation coefficient was calculated to explore the correlation between adiponectin and other variables. SPSS 15.0 for Windows software (SPSS, Chicago, IL) was used for statistical analysis. A p value <0.05 was considered statistically significant.

RESULTS

Demographic and clinical characteristics of all the study groups are presented in Table 1. As shown in Table 1, the subjects from IGT and DM groups were older, more often suffered from arterial hypertension whereas more controls, particularly non-obese ones, were smoking.

Table 1. **Baseline demographic and clinical characteristics of the study groups**

Parameters	IGT	DM	Obese-C	Non-obese-C
n	48	33	44	24
Age (years)	53.67 ± 9.28	55.82 ± 8.45	48.14 ± 9.32	47.87 ± 9.06
Sex (F/M)	20/13	30/18	31/13	16/8
Hypertension (n)	41 (85.4%)	30 (90.9%)	28 (63.6%)	9 (37.5%)
Smoking (n)	9 (18.8%)	8 (24.2%)	12 (27.3%)	7 (29.2%)
Systolic BP (mmHg)	146.34 ± 20.96	159.35 ± 24.69	142.32 ± 23.63	134.00 ± 21.93
Diastolic BP (mmHg)	90.94 ± 12.45	95.77 ± 12.11	89.59 ± 12.92	86.13 ± 13.03

Table 2. **Anthropometric and biochemical variables of IGT, DM and Obese-C groups**

Parameters	IGT	DM	p[1]	Obese-C	p[2]
BMI (kg/m^2)	33.31 ± 5.38	35.28 ± 6.71	0.150	32.34 ± 4.62	0.360
Waist (cm)	105.31 ± 12.08	110.38 ± 12.83	0.077	100.74 ± 11.72	0.071
Hip (cm)	113.74 ± 10.00	117.38 ± 12.91	0.236	113.95 ± 9.03	0.857
W/H	0.93 ± 0.06	0.94 ± 0.08	0.498	0.88 ± 0.07	*0.003*
Fat mass (kg)	34.68 ± 10.02	35.41 ± 9.53	0.755	33.81 ± 9.57	0.678
Fat mass (%)	36.61 ± 7.15	37.13 ± 7.56	0.764	36.2 ± 7.10	0.789
Systolic BP (mmHg)	146.34 ± 20.96	159.35 ± 24.69	0.013	142.32 ± 23.63	0.311
Diastolic BP (mmHg)	90.94 ± 12.45	95.77 ± 12.11	0.038	89.59 ± 12.92	0.419
Heart rate (beats/min)	69.94 ± 8.04	76.35 ± 8.12	0.001	69.89 ± 7.92	0.879
Fasting blood glucose (mmol/l)	6.26 ± 0.43	9.89 ± 4.07	<0.0001	5.33 ± 0.38	<0.0001
Insulin (μU/ml)	14.20 ± 8.09	16.94 ± 9.58	0.226	13.03 ± 6.82	0.525
HOMA-IR	4.00 ± 2.30	6.78 ± 3.71	<0.0001	3.10 ± 1.61	0.093
Uric acid (μmol/l)	359.80 ± 95.69	340.55 ± 95.39	0.249	299.38 ± 84.42	*0.001*
Adiponectin (μg/ml)	11.23 ± 9.72	9.23 ± 5.61	0.858	15.62 ± 10.38	0.004

p[1] for pairwise comparisons, IGT versus DM.

p[2] for pairwise comparisons, IGT versus Obese-C.

Table 3. **Anthropometric and biochemical characteristics of Obese-Control and Non-obese-Control groups**

Parameters	Obese-Control	Non-obese-Control	p
BMI (kg/m^2)	32.34 ± 4.62	23.55 ± 1.93	<0.0001
Waist (cm)	100.74 ± 11.72	83.17 ± 7.85	<0.0001
Hip (cm)	113.95 ± 9.03	98.90 ± 3.90	<0.0001
W/H	0.88 ± 0.07	0.84 ± 0.06	0.004
Fat mass (kg)	33.81 ± 9.57	18.26 ± 5.30	<0.0001
Fat mass (%)	36.2 ± 7.10	26.64 ± 7.44	<0.0001
Systolic BP (mmHg)	142.32 ± 23.63	134.00± 21.93	0.210
Diastolic BP (mmHg)	89.59 ± 12.92	86.13 ± 13.03	0.406
Fasting blood glucose (mmol/l)	5.33 ± 0.38	5.24 ± 0.43	0.333
Insulin (μU/ml)	13.03 ± 6.82	7.42 ± 3.88	<0.0001
HOMA-IR	3.10 ± 1.61	1.74 ± 0.91	<0.0001
Uric acid (μmol/l)	299.38 ± 84.42	283.75 ± 90.20	0.445
Adiponectin (μg/ml)	15.62 ± 10.38	19.62 ± 12.20	0.181

Table 4. **Spearman rank correlation of adiponectin with metabolic and anthropometric variables among study participants; all subjects combined and study groups**

Parameter	All subjects	IGT	DM	Obese-C	Non-obese-C
BMI (kg/m^2)	–.279*	–.108	–.359	–.120	–.342
Waist (cm)	–.447**	–.366*	–.508**	–.274	–.649**
Hip (cm)	–.205*	–.069	–.202	–.062	–.374
W/H	–.509**	–.487**	–.345	–.371*	–.642**
Fat mass (kg)	–.251**	–.143	–.434*	–.102	–.034
Fat mass (%)	–.033	–.017	.029	.038	.155
Systolic BP (mmHg)	–.148	–.048	.268	–.233	.033
Diastolic BP (mmHg)	–.110	.116	.020	–.207	–.109
Fasting blood glucose (mmol/l)	–.221**	.226	–.150	.113	–.082
Insulin (μU/ml)	–.316**	–.247	–.253	–.336*	–.156
HOMA-IR	–.340**	–.375**	–.285	–.340*	–.191

* Correlation is significant at the 0.05 level (2-tailed).

** Correlation is significant at the 0.01 level (2-tailed).

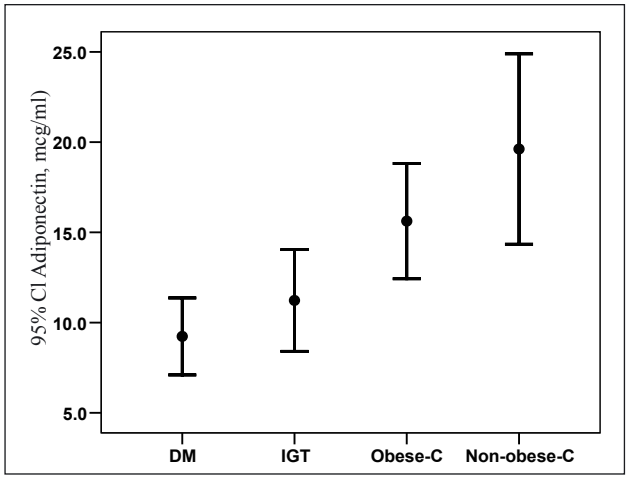

Fig. 1. Serum adiponectin in IGT, DM, Obese-Control and Non-obese-Control subjects
Note. Error bars show 95.0% CI of mean; dots show means.

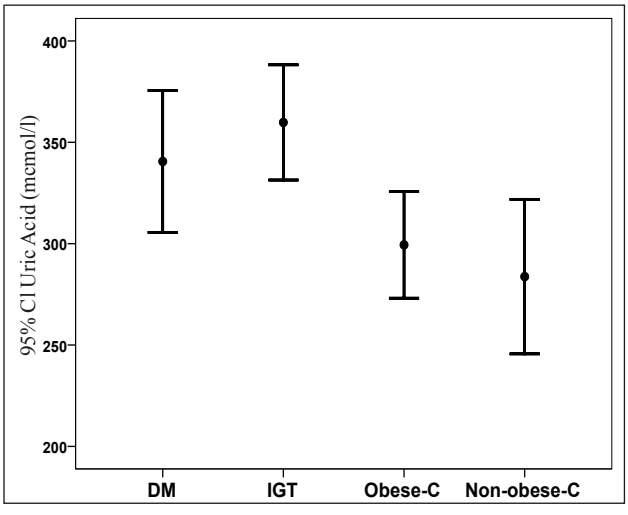

Fig. 2. Serum uric acid in IGT, DM, obese-control and non-obese-control subjects
Note. Error bars show 95.0% CI of mean; dots show means

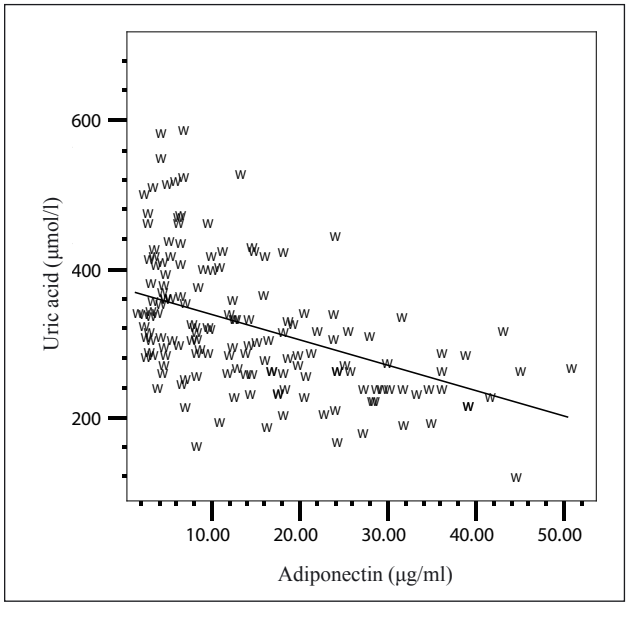

Fig. 3. Correlation between adiponectin and uric acid in the whole study population ($r = -.417$, $p < 0.0001$)

Effects of glucose metabolism on serum adiponectin levels

In our study we have compared anthropometric and biochemical parameters in IGT and DM groups. As shown in Table 2, anthropometric parameters (BMI, waist, hip, waist to hip ratio (W/H), body fat mass and fat percentage) did not statistically significantly differ in IGT and DM groups, whereas systolic and diastolic blood pressure and heart rate were significantly higher in DM subjects. Despite the fact that fasting blood glucose and HOMA-IR were significantly higher in DM group, adiponectin (11.23 ± 9.72 vs. 9.23 ± 5.61 μg/ml, $p = 0.858$). and uric acid (359.80 ± 95.69 vs. 340.55 ± 95.39 μmol/l, $p = 0.249$) levels did not statistically significantly differ (Figs 1, 2).

To further assess the influence of glucose metabolism status on adiponectin and uric acid levels, we compared IGT and Obese-C groups, which had similar anthropometric and clinical characteristics (Table 2). IGT and Obese-C groups were comparable with regard to anthropometric measures (BMI, waist, hip, fat mass and percentage); they also had similar blood pressure and heart rate. Fasting blood glucose was significantly higher in IGT group, but plasma insulin and HOMA-IR did not significantly differ. The analysis showed that in IGT group adiponectin (11.23 ± 9.72 vs. 15.62 ± 10.38 μg/ml, $p = 0.004$) and uric acid (359.80 ± 95.69 vs. 299.38 ± 84.42 μmol/l, $p = 0.001$) levels were significantly higher as compared to Obese-C group (Figs. 1, 2).

Effects of obesity on adiponectin and uric acid levels in subjects with normal glucose tolerance

To evaluate the influence of obesity on adiponectin concentration we compared Obese-C and Non-obese-C subjects. As shown in Table 3, Obese-C subjects had significantly higher BMI, waist and hip circumferences, larger amount of body fat mass and percentage; they also had higher systolic and diastolic blood pressure. Comparison of the metabolic parameters showed that fasting blood glucose was quite similar in these study groups, whereas insulin and HOMA-IR, level were significantly higher in Obese-C subjects. Serum adiponectin concentration was lower in Obese-C than in Non-obese-C group (15.62 ± 10.38 vs. 19.62 ± 12.20 μg/ml,), but the difference was not statistically significant ($p = 0.181$), whereas the levels of uric acid were similar in obese and non-obese control subjects (299.38 ± 84.42 vs. 283.75 ± 90.20 μmol/l, $p = 0.445$) (Figs. 1, 2).

Relationship of adiponectin levels with metabolic and obesity parameters

Among all the study subjects, adiponectin showed strongest inverse correlations with waist circumference, W/H, and HOMA-IR, whereas more modest but statistically significant negative correlations were observed between adiponectin and BMI, hip circumference, body fat mass, fasting glucose and insulin (Table 4).

Correlation analysis in the study groups (Table 4) showed that adiponectin did not significantly correlate with indices of overall obesity (BMI and body fat mass or percentage body fat), but negative associations with indices of visceral obesity (waist circumference and W/H) were rather strong in all the study groups. Adiponectin significantly correlated with W/H ($r = -.487$, $p < 0.0001$) in IGT group and with waist circumference ($r = -.508$,

p = 0.004) in DM subjects. Inverse correlation was observed between adiponectin and HOMA-IR in all the subjects combined, although individual variations probably had some influence on final data in different study groups. In Obese-C group adiponectin showed the inverse correlations with HOMA-IR, insulin and W/H. In Non-obese-C subjects adiponectin significantly inversely correlated with waist ($r = -.649$, $p < 0.0001$) and W/H ($r = -.642$, $p < 0.0001$).

Associations of uric acid levels with obesity parameters and adiponectin

In the whole study population the strongest positive associations were observed between the uric acid levels and indices of central obesity: waist circumference ($r = -.44$, $p < 0.01$) and W/H ($r = -.45$, $p < 0.01$), whereas associations with parameters of overall obesity were more modest (Table 5). Positive statistically significant correlations between indices of insulin resistance and fasting glycaemia and levels of uric acid were also noted. Analysis of associations between adiponectin and uric acid revealed negative statistically significant correlation between these variables ($r = -.42$, $p < 0.01$) (Fig. 3). Adiponectin serum concentration correlated negatively with uric acid consistently in all study groups (Table 4). After accounting for the influence of gender, negative correlation between adiponectin and uric acid concentrations in the whole study population remained statistically significant (coefficient of partial correlation $r_p = -.225$, $p = 0.003$).

Table 5. **Spearman rank correlation of uric acid with metabolic and anthropometric variables among study participants**

Parameter	All subjects
BMI (kg/m^2)	.31*
Waist (cm)	.44**
Hip (cm)	.26*
W/H	.45**
Fat mass (kg)	.27**
Fat mass (%)	.11
Systolic BP (mmHg)	.34**
Diastolic BP (mmHg)	.31**
Fasting blood glucose (mmol/l)	.26**
Insulin (μU/ml)	.39**
HOMA-IR	.38**
Adiponectin (μg/ml)	–.42**

* Correlation is significant at the 0.05 level (2-tailed).

** Correlation is significant at the 0.01 level (2-tailed).

DISCUSSION

In this study we have demonstrated that adiponectin level was similar in IGT and type 2 diabetes groups and in both these groups it was significantly lower when compared with the obese control group. On the other hand, adiponectin was not statistically significantly different in obese control when compared with non-obese control subjects.

Previous studies have found that plasma adiponectin levels in IGT were both lower (18) and similar (19) when compared with type 2 diabetes subjects. Osei et al. (18) studied first degree relatives of African-American patients and showed that adiponectin levels were significantly lower in the type 2 diabetes group, whereas in IGT and normal glucose tolerance subjects adiponectin levels were significantly higher. Although it was not the primary endpoint of the study, Bluher et al. (19) reported similar levels of total plasma adiponectin in IGT and type 2 diabetes Caucasian subjects, and in both groups adiponectin levels were lower than in subjects with normal glucose tolerance. Possibly due to the same racial group studied and similar study groups characteristics the results of our study confirmed the findings of Bluher et al. (19). The results of our study suggest that adiponectin secretion is already altered in pre-diabetic conditions, and as the majority of subjects with IGT eventually will develop type 2 diabetes, based on the results of our study we speculate that transition from pre-diabetes to type 2 diabetes is not associated with further alterations in adiponectin metabolism. Our data also suggest that metabolic alterations seen in IGT state are strong enough to further lead to diabetes development.

Adiponectin level was found to be decreased in obese subjects in several previous studies (4,30). In our investigation obese subjects with normal glucose tolerance had lower adiponectin levels than non-obese ones, but the difference did not reach statistical significance, probably due to a rather small number of non-obese control subjects studied.

The results of our study confirm that adiponectin levels are strongly associated with visceral obesity and insulin resistance (5, 13, 30). Indeed, we demonstrated that adiponectin significantly inversely correlated with waist circumference and waist-to-hip ratio, but not with indices of overall obesity: BMI and body fat mass.

We also found that uric acid levels in subjects with pre-diabetes and early type 2 diabetes are significantly higher than in healthy controls. This is in consort with the report of another scientific research (25). Based on these results we conclude that subjects with impaired glucose metabolism have increased levels of uric acid, and this increment might accelerate the progression of atherosclerosis in these patients. We also confirmed negative associations between levels of adiponectin and uric acid reported by other scientific group (30, 31) and for the first time demonstrated that this association remained statistically significant after controlling for the influence of gender.

CONCLUSION

Our study has demonstrated that adiponectin levels are reduced in pre-diabetic conditions to the same extend as in early type 2 diabetes, therefore we suggest that adiponectin secretion is more closely related to insulin resistance than to simply transition from pre-diabetes to diabetes which was not associated with further alteration in adiponectin secretion. We conclude that insulin sensitivity and visceral obesity rather than obesity *per se* appear to be the major determinants of serum adiponectin levels. We also demonstrated that serum adiponectin concentration is negatively associated with the level of uric acid and that this association is gender-independent.

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Vaidotas Urbanavičius, Agnė Abraitienė, Dalius Vitkus, Regina Borovkienė, Zita Aušrelė Kučinskienė

ADIPONEKTINAS IR ŠLAPIMO RŪGŠTYS ESANT PREDIABETUI IR ANKSTYVOSIOMS II TIPO CUKRINIO DIABETO FORMOMS

S a n t r a u k a

Įvadas. Šio tyrimo tiklas buvo įvertinti adiponektino ir šlapimo rūgšties koncentracijas kraujyje asmenims su prediabetinėmis būklėmis ir sergantiems pradinėmis 2 tipo cukrinio diabeto stadijomis, bei nustatyti adiponektino ryšius su antropometriniais išmatavimais, gliukozės apykaitos ir rezistentiškumo insulinui rodikliais bei šlapimo rūgšties koncentracija kraujyje.

Tiriamieji asmenys ir metodai. Ištirti 48 asmenys, kuriems buvo diagnozuotos prediabetinės būklės (sutrikusi tolerancija gliukozei ir / arba sutrikusi alkio glikemija), 33 asmenys, kuriems buvo naujai diagnozuotas 2 tipo cukrinis diabetas, ir 68 sveiki tiriamieji. Priklausomai nuo kūno masės indekso (KMI) sveikų asmenų grupė buvo padalinta į nutukusių (KMI > = 27) ir nenutukusių (KMI < 27) pogrupius.

Rezultatai. Adiponektino koncentracijos prediabeto ir diabeto grupėse statistiškai nesiskyrė (11,2 ± 9,72 vs. 9,23 ± 5,61 μg/ml, p = 0,858). Nepaisant antropometrinių ir kitų klinikinių panašumų, minėta koncentracija buvo statistiškai patikimai mažesnė asmenų su sutrikusia gliukozės tolerancija, palyginus su sveikų nutukusių asmenų grupe (11,23 ± 9,72 vs. 15,62 ± 10,38 μg/ml, p = 0,004). Nutukusių sveikų asmenų adiponektino koncentracija buvo mažesnė negu nenutukusių sveikų asmenų, tačiau šis skirtumas nebuvo statistiškai patikimas (15,62 ± 10,38 vs. 19,62 ± 12,20 μg/ml, p = 0,181). Sergančiųjų 2 tipo cukriniu diabetu šlapimo rūgšties kiekis kraujyje nesiskyrė nuo asmenų su prediabetinėmis būklėmis (359.80 ± 95.69 ir 340.55 ± 95.39 μmol/l, p = 0.249), tuo tarpu sveikų nutukusių ir nenutukusių kontrolinių tiriamųjų grupėse šlapimo rūgšties koncentracija kraujyje buvo statistiškai patikimai mažesnė. Adiponektino ir šlapimo rūgšties koncentracijos kraujyje neigiamai koreliavo su centrinio nutukimo rodikliais. Koreliacinė analizė parodė, kad visoje tyrimo populiacijoje adiponektino kiekis kraujyje neigiamai koreliavo su šlapimo rūgšties koncentracija; ryšys tarp šių rodiklių išliko statistiškai patikimas atmetus lyties įtaką (dalinės koreliacijos koeficientas r_d = –.225, p = 0.003).

Išvados. Prediabeto ir 2 tipo cukrinio diabeto grupių tiriamųjų adiponektino ir šlapimo rūgšties koncentracijos buvo panašios, tuo tarpu sveikų nutukusių ir nenutukusių asmenų grupėse adiponektino kiekis kraujyje buvo statistiškai patikimai mažesnis, o šlapimo rūgšties – didesnis nei asmenų su pakitusia angliavandenių apykaita.

Raktažodžiai: adiponektinas, šlapimo rūgštis, atsparumas insulinui, prediabetas, II tipo cukrinis diabetas