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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

The influence of chosen adipocytokines on blood pressure values in patients with metabolic syndrome

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Abstract

Background: Adipocytes are hormonally active molecules modulating biochemical processes on-going in human organism. Visceral fat tissue is the source of many important substances, such as: adiponectin, TNF-α, resistin and many others. However, their role has not been fully elucidated.

Aim: 1. To measure the concentrations of adiponectin, soluble receptors for TNF-α, and resistin in patients with metabolic syndrome (MetS). 2. To investigate the correlations between chosen adipocytokines and hypertension.

Methods: The study included 85 patients with MetS, diagnosed according to the International Diabetes Federation (IDF) criteria from 2005, and 20 healthy volunteers as control subjects. In both groups, anthropological parameters (body mass index and waist circumference) and concentrations of adiponectin, soluble receptors for TNF-α and resistin were assessed. To measure adiponectin levels, radioimmunological and immunoenzymatic assays (sTNFR1, sTNFR2, and resistin) were employed.

Results: Concentration of adiponectin was markedly lower in patients with MetS compared to the control group (10.892 ± ± 10.440 vs. 19.307 ± 19.370 μg/mL, $p < 0.001$). In patients with MetS, sTNFR1 and sTNFR2 concentrations were significantly elevated and resistin level was significantly higher compared to control subjects (25.865 ± 25.8 vs. 9.60 ± 9.915 ng/mL, $p < 0.001$). Negative correlations between adiponectin and the values of systolic blood pressure (SBP) ($Rp = 0.2898$, $p < 0.001$) and diastolic blood pressure (DBP) ($Rp = 0.2724$, $p < 0.001$) were observed. Positive correlations between sTNFR2 and SBP ($Rp = 0.450$, $p < 0.001$) and DBP ($Rp = 0.4695$, $p < 0.001$) values were found. Resistin levels correlated positively with SBP ($Rp = 0.5154$, $p < 0.001$) and DBP ($Rp = 0.5613$, $p < 0.001$) values. In multiple regression model, the association of SBP with resistin level was observed in 32% of studied patients.

Conclusions: 1. Patients with MetS are characterised by following disturbances: hypo-adiponectinaemia, elevated concentrations of soluble receptors for TNF-α, and hyperresistinaemia. 2. Elevated concentration of resistin may play a role in the development of high blood pressure.

Key words: hypertension, adipocytokines, cardiovascular risk

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INTRODUCTION

Metabolic syndrome (MetS) is a disease entity that continues to raise controversies among researches. MetS has been subject to many clinical and theoretical studies. Recent 2005 European diagnostic criteria for MetS were presented by consensus group of the International Diabetes Federation (IDF) in Berlin [1, 2]. MetS, being a constellation of proatherogenic risk factors, is a topic of special interest due to its proved association with the development of cardiovascular diseases and type 2 diabetes. According to data from WOBASZ study, MetS is diagnosed in more than 20% of men and 17% of women in Polish population aged 20–74 years [3].

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Obesity remains a primary diagnostic criterion for MetS. Visceral adipose tissue is a source of many hormonally active compounds, called adipocytokines, such as adiponectin, tumour necrosis factor alpha (TNF-α), and resistin. The role of these adipocytokines in blood pressure (BP) regulation is still debated.

The control of hypertension (HT) in Poland is still insufficient. Effectiveness of antihypertensive therapy is 10–24%. Research is needed to develop new methods that would enable better and easier HT control. It is of particular importance, because elevated BP is one of the key risk factors for cardiovascular events.

The role of adipocytokines in the development of hypertension

Resistin. Resistin is one of the most important adipocytokines linking HT and obesity [4]. It increases the level of triglycerides and decreases HDL cholesterol, thereby playing an important role in the atherogenesis [5]. Resistin enhances chronic inflammation which is the background of the development of atherosclerosis [6]. Clinical trials assessing the connections between resistin and HT showed increased resistin levels in obese hypertensives compared to normotensive subjects [7]. Resistin is positively correlated with TNF-α and acute phase protein — C-reactive protein. In this mechanism it can be assumed, that resistin indirectly contributes to the development of HT [8]. Resistin is believed to have impact on sympathetic nervous system [9], but its role in the system's activation and, consequently, the elevation of BP has not been fully elucidated. Therefore, further studies are needed [10].

Adiponectin. Another adipocytokine that participates in the pathogenesis of HT is adiponectin. The deficit of adiponectin may play a role in the complex pathomechanism of HT related with obesity and cardiovascular complications [10, 11]. Available observations suggest protective effect of high levels of adiponectin on vascular endothelium. It has been proved that adiponectin inhibits TNF-α secretion by macrophages and decreases the synthesis of adhesive molecules in endothelial cells, which leads to reduction in monocyte adhesion. Opinions on the impact of adiponectin on BP values are discrepant, therefore further studies are needed [12–14]. Experimental studies in animal models showed that adiponectin reduced insulin resistance and contributed to body mass reduction by decreasing the amount of triglycerides in muscles and the liver, and consequently decreased BP levels in obese mice [15].

TNF-α. The role of TNF-α is also considered in the pathogenesis of obesity-related HT. This cytokine is partly produced and secreted by visceral adipose tissue. After binding of TNF-α to its membrane receptors, extracellular domains of these receptors are released into bloodstream via endoprotease-mediated proteolytic cleavage and soluble receptors for TNF-α — sTNFR1 and sTNFR2 — are formed. Soluble forms of receptors remain in the bloodstream for a long time, which is believed to enable the measurement of biologic effects of TNF-α. Potential mechanisms of TNF-α in the development of HT are related with its indirect impact on the increase of insulin resistance, the levels of leptin, endothelin and angiotensin, as well as the effect on endothelial nitric synthase activity [16–18].

The aim of the study was to assess the impact of chosen adipocytokines, such as adiponectin, sTNFR1, sTNFR2 and resistin, on the development of high BP, which is a significant risk factor for cardiovascular events.

METHODS

The study protocol was approved by a regional bioethics committee of Poznan Medical University (No 1103/08). All patients were informed about the aim and rules of the study. Informed written consent of the patient was required to participate in the study. The participation in the study was voluntary.

The study included 105 subjects aged 25–77 years. The studied population was divided into two groups. The experimental group comprised 85 patients, 42 woman and 43 men (mean age 53.4 ± 12.10 years) in whom MetS was diagnosed according to 2005 IDF criteria. The control group included 20 healthy volunteers matched according to sex and age (10 women and 10 man, mean age 52.4 years). In all patients with MetS, the BP values exceeded 140/90 mm Hg and were classified as I or II stage of HT. Exclusion criteria included: III stage HT, diabetes, history of acute coronary episodes, glomerular filtration rate < 70 mL/min, liver disease, and hormonal therapy. Patients were recommended to withhold smoking and alcohol consumption 3–4 days before the study. In patients taking hypotensive or hypolipaemic drugs, statin therapy was suspended for 5–7 days and hypolipaemic drug therapy: for 3–4 days. Longer suspension of the therapy with these drugs was impossible due to ethical reasons.

Following anthropometric parameters were measured: height, body mass, and waist circumference. Waist circumference > 80 cm in women and > 94 cm in men was considered an indicator of visceral obesity. Body mass index (BMI) was calculated using following formula: BMI = body mass [kg]/(height [m])^2. BP measurements were performed with manual sphygmomanometer according to 2007 guidelines of Polish Society of Hypertension. Three measurement were taken in sitting position, after 10-min rest. Patients were recommended to avoid alcohol, coffee, strong tee and tobacco smoking during 12 h preceding the study. The measurement was performed using the cuff enclosing rubber bladder, 12 cm wide and 35 cm long. In overweight or obese patients, wider cuffs were used, according to the guidelines of Polish Society of Hypertension. Systolic BP (SBP) was determined by the onset of the "tapping" Korotkoff sounds (phase 1) while

Table 1. Comparison of studied groups

Parameter	MetS group (n = 85)			Control group (n = 20)			P
	Mean	Median	SD	Mean	Median	SD	
Waist circumference [cm]	113.19	109.00	14.25	84.50	86.00	8.20	< 0.001
Body mass [kg]	100.56	98.00	20.62	72.60	72.00	9.14	< 0.001
Height [cm]	167.60	162.00	8.47	171.30	165.50	8.59	NS
BMI [kg/m^2]	35.52	38.90	5.67	24.67	25.02	1.88	< 0.001
Adiponectin [μg/mL]	10.892	3.978	10.440	19.307	4.623	19.370	< 0.001
Resistin [ng/mL]	25.865	10.031	25.840	9.600	1.351	9.915	< 0.001
sTNFR1 [ng/mL]	4.118	0.749	4.010	2.329	0.628	2.280	< 0.001
sTNFR2 [ng/mL]	6.907	1.400	6.740	2.734	0.761	2.795	< 0.001

Man-Witney U test for BMI, waist circumference, body mass, and adiponectin and resistin levels: differences were considered statically significant at $p < 0.001$; t-Student test for sTNFR1 and sTNFR2: differences were considered statically significant at $p < 0.001$; SD — standard deviation; BMI — body mass index

diastolic BP (DBP) was defined as the fifth Korotkoff sound (phase 5), or the disappearance of Korotkoff sounds. Mean SBP and DBP was calculated as an arithmetic mean from 3 measurements taken on 3 consecutive days preceding the study. Venous blood samples were drawn in the morning, after a 12-h fast, in order to perform following measurements: full blood count, aminotransferases, creatinine, glucose, lipid profile, adiponectin, soluble TNF-α receptors, and resistin.

Adiponectin levels were measured using DRG kits (reference values 5–30 μg/mL) [19]. The levels of soluble TNF-α receptors 1 and 2 were measured using DRG sTNF–RI EASIA KAP1761 kit by BioSource Europe S.A., Belgium.

Statistical analysis

Analysed parameters were presented as means and standard deviations or medians. To check for distribution normality, Shapiro-Wilk test was used. Between-group comparisons (MetS group vs. control group) for normally distributed parameters were performed using t-Student test for independent variables (homogeneity of variance was confirmed with Leven test) or Welch test (in case of heterogeneity). For non-normally distributed parameters non-parametric Mann-Whitney test was used. Pearson's linear correlation coefficient was calculated for normally distributed parameters, whereas for non-normally distributed parameters Spearman's non-parametric correlation coefficient was calculated. All statistical analyses were performed with STATISTICA v. 8 and Analyse-it for Microsoft Excel v. 2.20 software packages. Standard forward stepwise regression analysis was carried out with adiponectin, sTNFR1, sTNFR2 and resistin levels as dependent variables.

RESULTS

Patients with MetS were characterised by hyperadiponectinaemia and increased levels of soluble receptors sTNFR1 and sTNFR2. In MetS patients, resistin levels were significantly higher compared with the control group (Table 1). Negative correlation between adiponectin levels and SBP ($R = –0.2898$; $p < 0.01$) as well as negative Spearman's rang order correlation between adiponectin levels and DBP ($R = –0.2724$; $p < 0.001$) were observed (Fig. 1). The level of sTNFR2 was significantly associated with SBP ($R = 0.4504$; $p < 0.001$) and DBP ($R = 0.4695$; $p < 0.001$) values (Fig. 2).

Positive correlations between SBP values and resistin levels ($R = 0.5154$; $p < 0.001$) (Fig. 3) as well as positive Spearman's rang order correlation between resistin levels and DBP values ($R = 0.5613$; $p < 0.001$) were also found. Relationships of SBP and DBP values with the levels of adipocytokines were determined in MetS patients. Stepwise multiple regression model was calculated for chosen adipocytokines. Thus, sTNFR1 associations with sTNFR2 ($p < 0.005$) and resistin ($p < 0.004$) were determined. Furthermore, stepwise multiple regression analysis showed sTNFR2 relationships with

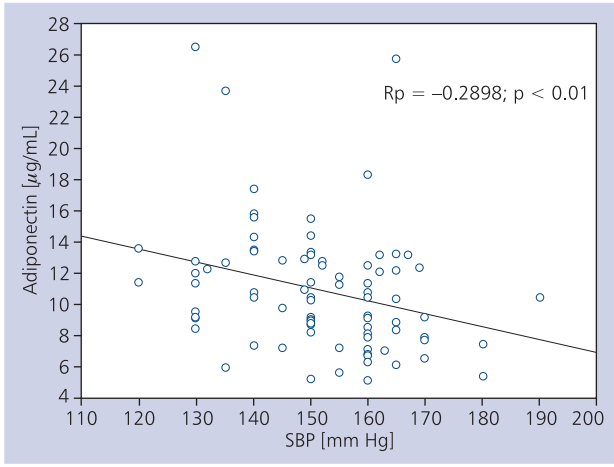

Figure 1. Relationship between systolic blood pressure (SBP) and adiponectin level; Spearman's rang order correlation between adiponectin level and diastolic blood pressure was shown, $R = –0.2724$, $p < 0.001$

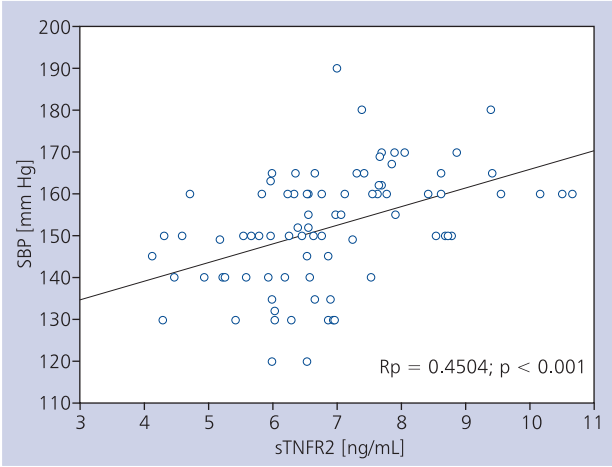

Figure 2. Relationship between systolic blood pressure (SBP) and the level of soluble TNF-α receptor 2 (sTNFR2); the relationship between diastolic blood pressure and sTNFR2 was shown; Rp = 0.4695, p < 0.001

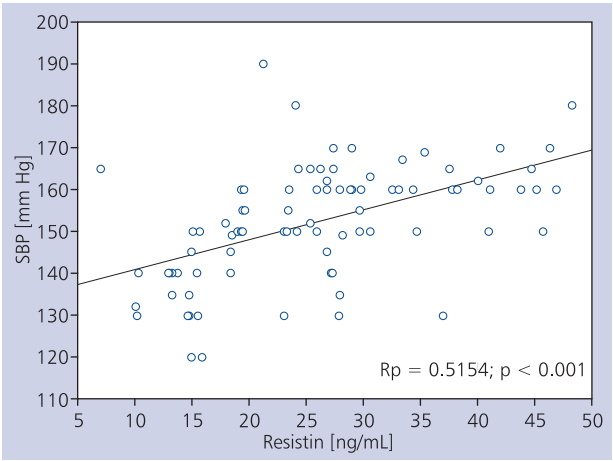

Figure 3. Relationship between systolic blood pressure (SBP) and resistin level; Spearman's rang order correlation between diastolic blood pressure and resistin level was shown; R = 0.5613, p < 0.001

resistin (p < 0.001) and sTNFR1 (p < 0.009). Adjusted R^2 of 0.640 indicated that 64% of sTNFR variability could be explained by above-mentioned regression model. In multiple regression analysis, association between SBP and resistin levels was found (p < 0.0008). Adjusted R^2 equalled 0.324 suggesting that 32% of SBP variability could be attributed to resistin. The association between DBP and resistin was found (p < 0.0223). Adjusted R^2 equalled 0.3022 indicating that 30% of DBP variability could be attributed to resistin.

DISCUSSION

Currently, there is insufficient evidence from large clinical trials to demonstrate the relationship between adipocytokines and BP values in obese hypertensives. However many researchers have described the role of particular molecules produced by adipose tissue in normotensive and hypertensive subjects with normal body mass. In a study performed on 967 non-obese Japanese subjects without metabolic disturbances aged 30––65 years, Yamamoto et al. [20] showed a relationships between adiponectin levels and BMI, SBP, DBP, and lipid profile. Hyperadiponectinaemia and negative correlation of adiponectin with SBP and DBP were also observed in hypertensive patients. These observations were confirmed by my own study. Significantly lower adiponectin levels were found in obese patients with HT compared to healthy control subjects (normotensives with normal body mass).

The effect of adiponectin on BP values indicates that it is negatively correlated with SBP and DBP. It should be however highlighted that the role of adiponectin in the modification of HT values is still controversial.

Adamczak et al. [12] showed that adiponectin levels are lower in HT patients. Conversely, Mallamaci et al. [13] observed that adiponectin levels are higher in hypertensives than in normotensive subjects. However, a study on adiponectin gene polymorphism performed by Leu et al. [14] in 2011 did not confirm the latter observation. Recent reports from 2011 indicate the role of adiponectin as a BP regulating hormone via mechanism of stimulation of endovascular nitric oxide synthase (eNOS) [21].

Another adipocytokine that may affect BP values is TNF-α. In the population of HT patients, significantly higher levels of soluble receptors for TNF-α were noticed. Also my own study revealed significant association between plasma sTNFR2 levels and BP values. Although the role of TNF-α in the induction of insulin resistance and the development of HT and atherosclerosis has not been fully understood, it still remains an attractive target of pharmacologic interventions [22].

Resistin is also considered as a factor involved in the development of high BP. Degawa-Yamauchi et al. [23] showed higher resistin levels in obese patients compared to subjects with normal body mass. They also found a positive correlation between BMI and the level of resistin. Results of my own study confirm the presence of hyperresitinaemia in obese patients with MetS. The role of resistin in human population has not been definitively confirmed. However Chinese study performed on 1,102 diabetic patients showed that resistin gene polymorphism (3'UTR + 629>A) is independently associated with SBP and DBP values [24]. The relationship between resistin level and BP values was also confirmed by results of my own study, where in stepwise multiple regression model, SBP and DBP values correlated with resistin concentration. This may result from the fact that resistin induces insulin resistance which, in turn, leads to high BP. Above observations were further confirmed by Zhang at al. [25]. Literature review indicates that resistin is also involved in the inflam-

matory process. Resistin remains an interesting subject for future research, because is considered a pro-inflammatory cytokine that, like TNF-α, might play a significant role in the development of cardiovascular complications [26].

CONCLUSIONS

1. MetS is a cluster of interrelated metabolic disturbances including hypo-adiponectinaemia, elevated concentrations of soluble receptors for TNF-α, and hyperresistinaemia.
2. Elevated concentration of resistin may play a role in the development of high BP.

This paper contains parts of the author's PhD thesis titled "Assessment of chosen adipocytokines and markers of inflammation in patients with metabolic syndrome".

Conflict of interest: *none declared*

References

1. Alberti KG, Zimmet P, Shaw J; IDF Epidemiology Task Force Consensus new worldwide definition. Lancet, 2005; 366: 1059––1062.
2. www.idf.org/metabolic_syndrome, website of the International Diabetes Federation.
3. Biela U, Pająk A, Kaczmarczyk-Chałas K et al. Częstość występowania nadwagi i otyłości u kobiet i mężczyzn w wieku 20–74 lat. Wyniki programu WOBASZ. Kardiol Pol, 2005; 63 (suppl. 4): 632–635.
4. Steppan M, Bailey T, Bhat S et al. The hormone resistin links obesity to diabetes. Nature, 2001; 409: 307–312.
5. Lozano-Nuevo JJ, Estrada-Garcia T, Vargas-Robles H et al. Correlation between circulating adhesion molecules and resistin levels in hypertensive type-2 diabetic patients. Inflamm Allergy Drug Targets, 2011; 10: 27–31.
6. Reilly P, Lehrke M, Wolfe L et al. Resistin is an inflammatory marker of atherosclerosis in humans. Circulation, 2005; 111: 932–939.
7. Papadopolous D, Makris T, Krespi P et al. Adiponectin and resistin level in healthy individuals with prehypertension. J Clin Hypertens, 2005; 7: 12: 729–733.
8. Zdrojewski T, Chwojnicki K, Bandosz P. Distribution of C-reactive protein and its relation to arterial hypertension in a country representing a high-risk region for cardiovascular diseases. Blood Press, 2006; 15: 20–26.
9. Bo S, Cavallo-Perin P. Hypertension: shall we focus on adipose tissue? J Am Soc Nephrol, 2010; 21: 1067–1068.
10. Szulińska M, Pupek-Musialik D, Bogdański P. Udział produktów adipocytów w rozwoju insulinooporności u pacjentów z otyłością i nadciśnieniem tętniczym. Nadciśnienie Tętnicze, 2004; 8: 33–40.
11. Liu JT, Song E, Xu A et al. Lipocalin-2 deficiency prevents endothalial dysfunction associated with dietary obesity: role of cytochrome P450 2C inhibition. Br J Pharmacol, 2012; 165: 520–531.
12. Adamczak M, Wiecek A, Funahashi T et al. Decreased plasma adiponectin concentration in patients with essential hypertension. Am J Hypertens, 2003; 16: 72–75.
13. Mallamaci F, Zoccali C, Cuzzola F et al. Adiponectin in essential hypertension. J Nephrol, 2002; 15: 507–511.
14. Leu HB, Chung CM, Lin SJ et al. Adiponectin gene polymorphism is selectively associated with the concomitant presence of metabolic syndrome and essential hypertension. PLoS One, 2011; 6: 27.
15. Yamauchi T. Kamon J, Waki H et al. The fat- derived hormone adiponectin reverses insulin resistance associated with both lipoatrophy and obesity. Nat Med, 2001; 7: 941–946.
16. Pausova Z, Deslauriers B, Gaudet D et al. Role of tumor necrosis factor-alpha gene locus in obesity and obesity-associated hypertension in French Canadians. Hypertension, 2000; 36: 14–19.
17. Fernandez-Real J, Broch M, Ricart W et al. Plasma levels of the soluble fraction of tumor necrosis factor receptor 2 and insulin resistance. Diabetes, 1998; 47: 1757–1762.
18. Cottone S, Vadala A, Vella MC et al. Comparison of tumor necrosis factor and endothelin-1 between essential and renal hypertensive patients. J Hum Hypertens, 1998; 12: 351–354.
19. Linh AS, Torben O., Birgit N. Adiponectin mRNA expression in subcutaneous adipose tissue is reduced in first degree relatives of type 2 diabetic patients. Am J Physiol Endocrinol Metab, 2002; 284: E443–E448.
20. Yamamoto Y, Hirose H, Saito I et al. Correlation of the adipocite-derived protein adiponectin resistance index and serum high density lipoprotein cholesterol, independent of body mass index, in the Japanese population. Clin Sci, 2002; 103: 137–142.
21. Boden G. Obesity, insulin resistance and free fatty acids. Curr Opin Endocrinol Diabetes Obes, 2011; 18: 139–143.
22. Takai S, Jin D, Miyazaki M. Irbesartan prevents metabolic syndrome in rats via activation of peroxisome proliferator-activated receptor gamma. J Pharmacol Sci, 2011; 116: 309–315.
23. Degawa-Yamauchi M, Considine E, Tataranni P. Serum resistin (FIZZ3) protein is increased in obese humans. J Clin Endocrinol Metab, 2003; 88: 5452–5455.
24. Tan M, Chang S, Chang D et al. Association of resistin gene 3'-untranslated region + 629>A polymorhism with type 2 diabetes and hypertension in a Chinese population. J Clin Endocrinol Metabol, 2003; 88: 1258–1263.
25. Zhang MH, Na B, Schiller NB, Whooley MA. Association of resistin with heart failure and mortality in patients with stable coronary heart disease: data from the heart and soul study. J Card Fail, 2011; 17: 24–30.
26. Díez J, Iglesias P, Fernández-Reyes M et al. Serum concentrations of leptin, adiponectin and resistin, and their relationship with cardiovascular disease in patients with end-stage renal disease. Clin Endocrinol, 2005; 62: 242–249.

Wpływ wybranych adipocytokin na wartości ciśnienia tętniczego u chorych z zespołem metabolicznym

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Streszczenie

Wstęp: Adipocyty stanowią źródło wielu hormonalnie czynnych związków, modulujących procesy biochemiczne zachodzące w ludzkim organizmie. Tkanka tłuszczowa stanowi źródło wielu ważnych substancji, takich jak: adiponektyna, TNFα czy rezystyna. Rola adipocytokin nie została w pełni poznana.

Cel: Celem pracy było określenie stężenia adiponektyny, rozpuszczalnych receptorów dla TNFα i rezystyny u chorych z zespołem metabolicznym. Poszukiwano korelacji między wybranymi adipocytokinami a nadciśnieniem tętniczym.

Metody: Badaniami objęto 85 chorych z zespołem metabolicznym, rozpoznanym na podstawie kryteriów *International Diabetes Federation* (IDF) z 2005 r. Grupę kontrolną stanowiło 20 osób zdrowych. W obu badanych populacjach oceniano parametry antropometryczne (wskaźnik masy ciała, obwód talii) i stężenie wybranych adipocytokin: adiponektyny, rozpuszczalnych receptorów sTNFR1 i sTNFR2 oraz rezystyny. Stężenia powyższych adipocytokin oszacowano metodami: radioimmunometryczną (adiponektyna) i immunoenzymatycznymi (sTNFR1, sTNFR2 i rezystyna).

Wyniki: W grupie chorych z zespołem metabolicznym wykazano istotnie niższe stężenia adiponektyny niż w grupie kontrolnej (10,892 ± 10,440 *vs*. 19,307 ± 19,370 μg/ml; $p < 0,001$). Stężenia sTNFR1 i sTNFR2 były istotnie wyższe w populacji z zespołem metabolicznym ($p < 0,001$), także stężenia rezystyny w badanej grupie były wyższe niż w grupie kontrolnej (25,865 ± 25,8 *vs*. 9,60 ± 9,915 ng/ml; $p < 0,001$). Wykazano ujemną zależność między adiponektyną a ciśnieniem skurczowym (SBP) ($Rp = 0,2898$; $p < 0,001$) i ciśnieniem rozkurczowym (DBP) ($Rp = 0,2724$; $p < 0,001$). Zaobserwowano dodatnią korelację między sTNFR2 a SBP ($Rp = 0,450$; $p < 0,001$) i DBP ($Rp = 0,4695$; $p < 0,001$). Wykazano dodatnią korelację między stężeniami rezystyny a SBP ($Rp = 0,5154$; $p < 0,001$) i DBP ($Rp = 0,5613$; $p < 0,001$). Wszystkie korelacje oszacowano w grupie chorych z zespołem metabolicznym. W modelu regresji wielokrotnej uzyskano zależność SBP od rezystyny, skorygowane $R2 = 0,3241$, co tłumaczy, że 32% zmienności SBP zależy od rezystyny.

Wnioski: 1. U osób z zespołem metabolicznym występują takie zaburzenia, jak hipoadiponektynemia, podwyższone stężenie rozpuszczalnych receptorów dla TNFα i hiperrezystynemia. 2. Hiperrezystynemia może być jednym z czynników wpływających na wzrost wartości ciśnienia tętniczego.

Słowa kluczowe: nadciśnienie tętnicze, adipocytokiny, powikłania sercowo-naczyniowe

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