ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Structure and function of large arteries in hypertension in relation to oxidative stress markers

Łukasz Klima[1], Kalina Kawecka-Jaszcz[1], Katarzyna Stolarz-Skrzypek[1], Jan Menne[2], Kamil Fijorek[3], Agnieszka Olszanecka[1], Wiktoria Wojciechowska[1], Grzegorz Bilo[4], Danuta Czarnecka[1]

[1]1st Department of Cardiology and Hypertension, Jagiellonian University Medical College, Krakow, Poland
[2]Clinic for Nephrology, Hanover Medical College, Hanover, Germany
[3]Department of Statistics, Krakow University of Economics, Krakow, Poland
[4]Department of Cardiology, The Italian Institute for Auxology, Milan, Italy

Abstract

Background: The relationship between hypertension and oxidative stress remains unexplained. Nevertheless, it is well established that reactive oxygen species have an influence on the arterial wall, endothelial function and therefore determine blood pressure.

Aim: The evaluation of selected oxidative stress markers in hypertensive patients and an assessment of the relationship between oxidative stress markers and the arterial structure and function. We also aimed to investigate whether the influence of oxidative stress on remodelling of arteries, their structural and functional changes is independent of hypertension or is related to hypertension.

Methods: Altogether 217 subjects (114 female, 103 male) were enrolled from hypertensive families. The mean age was 45.5 ± 16 years, and the group included 155 hypertensives. In every subject, the pulse wave velocity and intima–media thickness (IMT) in carotid arteries were measured as well as selected oxidative stress markers such as asymmetric dimethyl-arginin (ADMA), symmetric dimethylarginin (SDMA), advanced oxidation protein products (AOPP) and oxidised low density lipoproteins (ox-LDL).

Results: The results of multivariate analysis show that in hypertensive patients: the ADMA level increased with increasing peripheral pulse pressure (b = 0.003; p < 0.001), and AOPP was related to higher carotid IMT (b = 0.91; p = 0.036). In normotensive subjects, the following associations were found: between ADMA and central pulse pressure (b = 0.006; p = 0.008), between SDMA and the peripheral augmentation index (b = –0.03; p = 0.014), between AOPP and the peripheral augmentation index (b = 0.011; p = 0.04), and between ox-LDL/LDL ratio and the peripheral augmentation index (b = –0.025; p = 0.004). The SDMA/ADMA ratio was associated with estimated glomerular filtration rate in both groups (b = –0.0061; p < 0.0001 and b = –0.005; p < 0.017, respectively). In hypertensives, we observed a relation with peripheral pulse pressure (b = –0.0067; p = 0.0143). Moreover, in normotensives there was an association between the SDMA/ADMA ratio and uric acid (b = 1.3629; p = 0.046).

Conclusions: We found that the influence of oxidative stress on the structure and function of large arteries was independent of hypertension. Therefore oxidative stress may play a significant role in the development of arterial stiffness. Higher oxidative stress is associated with more advanced arterial remodelling in hypertension.

Key words: oxidative stress, arterial stiffness, hypertension

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INTRODUCTION

Hypertension (HT) is one of the commonest cardiovascular risk factors and the leading cause (32%) of death in Poland [1]. Severe complications of HT and its high prevalence have made primary prevention and early diagnosis a priority for healthcare services worldwide.

Recent studies have suggested that oxidative stress plays some role in the functioning and structure of the arteries. Elevated reactive oxygen species (ROS) leads to increased resistance of the arteries due to the reduction of nitric oxide (NO) availability, the peroxidation of membrane lipids, the impairment of relaxation, and the stimulated proliferation of smooth muscle cells. Elevated blood pressure (BP), on the other hand, caused by angiotensin II, xanthine oxidase, mechanical stress increases ROS activation and leads to the impairment of the endothelial function.

The main factor influencing endothelial function is NO. Its production depends on the availability of a cofactor for NO synthase such as tetrahydrobiopterin and endogenous competitive inhibitors of NOS such as asymmetric dimethylarginin (ADMA) or N-monomethylarginin (L-NMMA). The correlation between ADMA and BP has been proven [2]. Weber et al. [3] discovered that the ADMA level in serum is related to the central augmentation index. They evaluated the quantitative relationship between plasma levels of ADMA and arterial wave reflection assessed by pulse wave analysis technique as well as aortic pulse wave velocity (PWV) measured invasively during coronary angiography.

PWV and the distance of reflection points to the heart are the main determinants of the peripheral and central augmentation indices. Wave reflection occurs at sites of changes of arterial impedence along the arterial tree, such as branching points or atherosclerotic plaques. The peripheral and central augmentation indices increase with age and mean arterial pressure and are inversely related to heart rate and body height. Augmentation index predicts clinical events independently of peripheral pressures. As diagnostic thresholds, the following values have been proposed for middle-aged men (i.e. 40 years and above): 90% for the peripheral augmentation index, and 30% for the central augmentation index [4].

Zoccali et al. [5] found that ADMA was an independent predictor of carotid intima–media thickness (IMT) thickening. In the case of another oxidative stress marker (oxidised low density lipoproteins — ox-LDL), it also affects BP because it interacts with the RAA system [6]. Advanced oxidation protein products (AOPP) are considered a risk factor of atherosclerosis and coronary artery disease (CAD) [7]. Lower concentrations of symmetric dimethylarginin (SDMA) that are commonly found in serum, do not inhibit NOS, therefore it was considered an insignificant substance until Kiechl et al. [8] revealed that ADMA is not a better marker than SDMA for cardiovascular risk. Family predisposition to the development of HT may be manifested by exacerbated oxidative stress, and this leads to changes in the arterial wall even before HT develops.

The relationship between oxidative stress and HT remains still unexplained. It is considered to be a vicious circle of reactive oxygen species — that are both cause and result of HT. Thus the aim of this study was to assess selected oxidative stress markers in HT and normotensive subjects, and to analyse their relationship with the parameters of the arterial structure and function while adjusting for confounders. We also aimed to investigate whether the influence of oxidative stress on remodelling of arteries, their structural and functional changes is independent of HT or related to HT.

METHODS

The study was conducted within the framework of The InGenious HyperCare Network of Excellence [9]. Subjects were recruited from the Outpatient Hypertensive Clinic at the 1st Department of Cardiology and Hypertension, Jagiellonian University Medical College. Then their family members were enrolled — at least three people from two generations. We recruited 50 families, with altogether 217 subjects (114 women, 103 men). All participants signed a form which gave their informed consent. The protocol of the study was approved by the Bioethics Committee at the Jagiellonian University in Krakow.

Office BP measurement was performed according to the ESH guidelines [10]. Peripheral pulse pressure was defined as the difference between the mean systolic (SBP) and diastolic (DBP) office blood pressure.

In every subject, 24-h ambulatory blood pressure monitoring (ABPM) was performed with SpaceLabs 90207 (Redmont, WA, USA) with measurements every 15 min during the day (between 6 a.m. and 10 p.m.) and every 20 min at night. We adopted the criteria for incorrect measurements introduced by Staessen et al. [11].

Aortic PWV was evaluated with a Sphygmocor® device. The pulse wave on the carotid and femoral artery was recorded, then on the carotid and radial artery. The distance between the recording points and the sternal notch was measured and PWV was calculated as a fraction of the distance and foot–to–foot time.

For pulse wave analysis after 5 min rest in the supine position, the pulse wave on the radial artery was recorded with a microtip pressure transducer (Model SPT-301, Millar Instruments, Houston, TX, USA) and with Sphygmocor® software (ver. 6.31 AtCor Medical Pty., Ltd., Australia). After recording the radial pulse wave, the central BP wave was obtained and the peripheral and central augmentation indices (AIx p [%] and AIx c [%], respectively) were calculated. Peripheral AIx was defined as the quotient of the second (P2) and first (P1) peak of SBP. The central AIx was defined as the difference between P2 and P1 divided by the central pulse pressure (PP c).

For the evaluation of structural changes in the arterial wall, ultrasonography of the carotid arteries was performed using a two-dimensional projection along a longitudinal axis with 10 MHz probe and a Vivid 7 PRO GE device. The IMT was measured 2 cm proximally from the bulb of the common carotid artery and its bifurcation on far wall with semiautomatic method provided with GE software.

Laboratory tests
We collected fasting blood samples for measurements of the following parameters: glucose, cholesterol, low density lipoprotein cholesterol (LDL-C), high density lipoprotein

Table 1. Characteristics of the study population

	Hypertensives N = 155 (71.4%) Men/women: 74/81	Normotensives N = 62 (28.6%) Men/women: 29/33	P
Age [years]	48.95 ± 15.19	36.98 ± 14.73	< 0.001
Height [cm]	168.80 ± 9.28	169.23 ± 9.04	0.7563
Weight [kg]	81.29 ± 14.69	70.28 ± 14.43	< 0.001
Body mass index [kg/m²]	28.47 ± 4.26	24.43 ± 3.99	< 0.001
Waist to hip ratio	0.89 ± 0.08	0.85 ± 0.09	< 0.001
SBP [mm Hg]	144.15 ± 22.11	127.64 ± 11.89	< 0.001
DBP [mm Hg]	85.87 ± 11.69	78.86 ± 8.49	< 0.001
HR [bpm]	72.82 ± 11.62	74.31 ± 9.30	0.3235
Glucose [mmol/L]	5.42 ± 1.30	4.94 ± 0.66	< 0.001
LDL-C [mmol/l]	2.99 ± 0.90	2.82 ± 0.97	0.97
Cholesterol [mmol/L]	5.17 ± 1.05	4.87 ± 1.19	0.0867
HDL-C [mmol/L]	1.43 ± 0.37	1.61 ± 0.39	0.0021
Triglycerides [mmol/L]	1.70 ± 0.93	1.12 ± 0.60	< 0.001
eGFR [mL/min/1.73 m²]	100.41 ± 21.82	116.07 ± 26.20	< 0.001
24 h DBP [mm Hg]	77.10 ± 9.05	72.92 ± 6.71	< 0.001
24 h SBP [mm Hg]	126.77 ± 11.38	119.85 ± 8.43	< 0.001
24 h HR [bpm]	71.17 ± 9.69	76.11 ± 8.24	< 0.001

Data is shown as mean ± SD; 24h DBP, SBP, HR — 24h ABPM parameters respectively systolic, diastolic blood pressure and mean heart rate; eGFR — estimated glomerular filtration rate; HDL — high density lipoprotein cholesterol; LDL — low density lipoprotein cholesterol

cholesterol (HDL-C), triglycerides, creatinine, and uric acid. Repeated morning urine samples were collected to measure albumin and creatinine in urine. Selected oxidative stress markers were assessed: ADMA, SDMA and ox-LDL with ELISA tests and AOPP with spectroscopic analysis.

Statistical analysis

All statistical analyses were performed with STATISTICA 9.1 software and R 2.13. Continuous variables were presented as means with standard deviation. Categorical variables were presented as numbers and percentages. The Shapiro-Wilk test was used to assess the normality of continuous variables. The AOPP and ox-LDL/LDL ratio were logarithmically (log10) transformed. To examine differences between two independent groups, the Student's t-test was used. The Fischer exact or χ^2 tests were used to compare categorical variables. The unadjusted associations between continuous variables were examined by Pearson's or Spearman's correlation coefficient as appropriate. The association between variables adjusted for confounders was examined by stepwise multivariate regression. Two sided p-values < 0.05 were considered statistically significant.

RESULTS
Group characteristics

In our group of 50 families, the most prevalent family structure consisted of a subject and his/her parents and one sib-

ling (20 families). The second most common structure was a subject, one parent and two siblings (six families). The study population characteristics are summarised in Table 1. Normotensives (n = 62) compared to hypertensives (n = 155) were younger, had lower body mass index (BMI) and waist to hip ratio (WHR), lower glucose and triglyceride level, and higher HDL-C. Hypertensives had a lower estimated glomerular filtration rate (eGFR). Night-time DBP decrease was significantly more pronounced in normotensives.

Selected arterial parameters which differ between the groups of hypertensive and normotensive subjects are presented in Table 2.

The levels of oxidative stress markers did not differ between men and women with the exception of AOPPs which were higher in men (3.69 ± 0.96 vs. 3.4 ± 0.9 μmol/L; p = 0.027).

In the hypertensive group, angiotensin converting enzyme (ACE) inhibitors were administered in 71 patients and angiotensin receptor blockers (ARB) in 25. Thus, 62% of hypertensive subjects were treated with drugs with a strong influence on oxidative stress [12]. Moreover beta-blockers were administered in 61, calcium channel blockers in 46 and diuretics in 74 patients, whereas statins were administered in 28 patients.

Multivariate analysis

We did not observe any significant differences in ADMA, SDMA, AOPP or ox-LDL between normotensives and hyper-

Table 2. Arterial stiffness parameters

	Hypertensives N = 155 (71.4%)	Normotensives N = 62 (28.6%)	P
PWV (c-r) [cm/s]	841.51 ± 139.58	785.48 ± 119.18	0.004
PWV (c-f) [cm/s]	881.19 ± 217.89	745.13 ± 158.27	< 0.001
IMT [mm]	0.63 ± 0.16	0.53 ± 0.13	< 0.001
AIx p [%]	79.11 ± 24.22	60.23 ± 20.54	< 0.001
AIx c [%]	23.38 ± 16.32	10.93 ± 16.23	< 0.001
PP p [mm Hg]	57.58 ± 13.37	52.34 ± 11.44	0.0046
PP c [mm Hg]	44.45 ± 12.64	35.36 ± 9.63	< 0.001

Data is shown as mean ± SD; PWC c-r — carotid radial pulse wave velocity; PWV c-f — carotid femoral pulse wave velocity; IMT — intima-media thickness; AIx p — peripheral augmentation index; AIx c — central augmentation index; PP p — peripheral pulse pressure; PP c — central pulse pressure

Table 3. Oxidative stress markers

	Hypertensives N = 155 (71.4%)	Normotensives N = 62 (28.6%)	P
ADMA [μmol/L]	0.56 ± 0.16	0.58 ± 0.17	0.4989
SDMA [μmol/L]	0.74 ± 0.20	0.71 ± 0.17	0.4287
Log (AOPP) [μmol/L]	3.54 ± 0.96	3.54 ± 0.90	0.9983
Log (ox-LDL/LDL) [ng/mL/mmol/L]	4.18 ± 1.25	4.19 ± 1.25	0.9425
SDMA/ADMA	1.40 ± 0.53	1.31 ± 0.45	0.223

Data is shown as mean ± SD; ADMA — asymmetric dimethylarginin; SDMA — symmetric dimethylarginin; ox-LDL — oxidised low density lipoproteins; AOPP — advanced oxidation protein products

Table 4. Determinants of oxidative stress markers in hypertensive patients: multivariate analysis

	ADMA		SDMA		Log (AOPP)		Log (ox-LDL/LDL)		SDMA/ADMA	
	b	p	b	p	b	p	b	p	b	p
Intercept	0.2818	0.0002	1.0000	0.0000	2.4406	0.0000	6.2664	0.0000	2.3534	< 0.001
Hypertension	−0.0199	0.4516	−0.0047	0.8841	−0.2059	0.1740	0.1604	0.4272	0.0542	0.5147
BMI	NS	NS	-0.0064	0.0489	NS	NS	NS	NS	NS	NS
Uric acid	NS	NS	0.4495	0.0089	2.6258	0.0018	NS	NS	NS	NS
eGFR	0.0012	0.0171	−0.0023	0.0001	NS	NS	NS	NS	-0.006	< 0.001
Ex-smoker	NS	NS	NS	NS	−0.4052	0.0104	NS	NS	NS	NS
IMT	NS	NS	NS	NS	0.9108	0.0368	NS	NS	NS	NS
PP p	0.0031	0.0005	NS	NS	NS	NS	NS	NS	−0.0067	0.0143
DBP	NS	NS	NS	NS	NS	NS	−0.026	0.001	NS	NS

Additional variables included in multivariate analysis: age, waist to hip ratio and gender were non-significant; ADMA — asymmetric dimethylarginin; SDMA — symmetric dimethylarginin; ox-LDL — oxidised low density lipoproteins; AOPP — advanced oxidation protein products; IMT — intima-media thickness; b — regression coefficient; PP p — peripheral pulse pressure; PP c — central pulse pressure; BMI — body mass index; eGFR — estimated glomerular filtration rate; DBP — diastolic blood pressure; NS — non-significant

tensives after adjusting for potential confounders, i.e. age, BMI, WHR, uric acid, eGFR, sex, smoking, PWV, IMT, AIx, office BP in standard measurements and ABPM. The details are presented in Table 3. Correlations are related to office BP.

Using multivariate analysis, we found that in hypertensive patients (Table 4), ADMA was independently related to eGFR and the peripheral pulse pressure (b = 0.003; p < 0.001).

SDMA showed a positive correlation to uric acid and a negative correlation to eGFR. AOPPs were significantly correlated to IMT (b = 0.91; p = 0.036) and uric acid. Ox-LDL/LDL ratio had a negative relation to DBP.

In normotensives, an association between ADMA and eGFR and the central pulse pressure (b = 0.006; p = 0.008) was observed. SDMA was related to the peripheral AIx

Table 5. Family hypertension burden and oxidative stress

	1 relative	≥ 2 relatives	P
ADMA [μmol/L]	0.57 ± 0.16	0.58 ± 0.17	0.7362
SDMA [μmol/L]	0.71 ± 0.17	0.72 ± 0.18	0.8270
Log (AOPP) [μmol/L]	3.33 ± 1.07	3.62 ± 0.84	0.3748
Log (ox-LDL/LDL) [ng/mL/mmol/L]	3.88 ± 1.38	4.31 ± 1.21	0.3024
SDMA/ADMA	1.32 ± 046	1.32 ± 0.45	0.9862

Data is shown as mean ± SD; abbreviations as in Table 3

(b = –0.03; p = 0.014). For AOPP we observed a relationship with peripheral AIx (b = 0.011; p = 0.04) and sex. For the ox-LDL/LDL ratio, there were significant relations with BMI, central pulse pressure (b = 0.053; p = 0.008) and peripheral AIx (b = –0.025; p = 0.004) (Table 5). Moreover, there was a significant relationship between the SDMA/ADMA ratio and uric acid and eGFR in both hypertensives and normotensives (b = –0.0061; p < 0.0001 and b = –0.005; p < 0.017, respectively). Additionally, in hypertensives there was a relationship with peripheral pulse pressure (b = –0.0067; p = 0.0143), and in normotensives SDMA/ADMA ratio was associated with uric acid (b = 1.3629; p = 0.046).

DISCUSSION

Methylated arginines, ox-LDL or advanced oxidation protein products represent the set of markers recently widely investigated as potential measures of oxidative stress [13].

In our study we did not observe differences in ADMA, SDMA, ox-LDL or AOPP plasma levels between hypertensive and normotensive participants. Studies on oxidative stress markers in HT have provided varying results. Schlaich et al. [14] had similar results to ours. On the other hand, Surdacki et al. [15] observed an elevated ADMA concentration in newly diagnosed, not yet treated, hypertension. Perticone et al. [16] showed that the ADMA level is higher in hypertensives compared to a normotensive control group. Most studies by other groups have observed ADMA concentrations of approximately 2.6 μmol/L or more in hypertensives, such as would be found in end-stage renal disease or heart failure [17]. In our study, the mean ADMA concentration was 0.58 μmol/L in normotensives, and 0.56 μmol/L in hypertensives, much lower than in other studies. Schlaich et al. [14] also had lower concentrations of ADMA and SDMA in serum (for ADMA 1.11 μmol/L in normotensives without a family history of HT, 0.87 μmol/L in normotensives with a family history of HT and 1.61 μmol/L; for SDMA 0.37 μmol/L in normotensives without a family history of HT, 0.55 μmol/L in normotensives with a family history of HT and 0.59 μmol/L in hypertensives). Our findings may be the result of the treatment of hypertensive patients with RAAS antagonists such as ACE inhibitors or ARB as these drugs have a proven effect on oxidative stress reduction.

Mizayaki et al. [2] found a positive correlation between the concentration of ADMA and mean BP in 118 subjects (40 with HT) without concomitant cardiovascular diseases. Moreover, Weber et al. [3] showed a negative correlation of ADMA with DBP on the brachial artery and in the aorta. In multivariate analysis, we did not however observe a relationship between ADMA and BP. In the group of normotensives, an independent relationship between ADMA and the central BP was observed. On the other hand, in multivariate analysis, as in the study by Weber et al. [3], we observed a negative correlation between DBP and the oxidative stress marker.

A recently published meta-analysis [18] showed that carotid–femoral PWV is an independent risk factor for death and cardiovascular death. Weber et al. [3] observed that ADMA levels in serum are associated with arterial stiffness estimated with central augmentation index (c AIx) as well as carotid–femoral PVW. The authors showed that in the whole cohort ADMA correlates with AIx and augmentation pressure [3], and that this correlation was significant for adults below as well as above the age of 60. In our study, in multivariate analysis, the peripheral AIx correlated with AOPP in normotensives and an association between ADMA and PP p was present. In the Delles et al. study [19] comparing arterial stiffness in CAD patients to a control group, there was no difference in AIx (%) between the two groups. There was a difference in terms of PWV and aortic compliance. Ox-LDLs were lower in the study group but the ratio ox-LDL/LDL was higher. In our study there was no difference between groups for ox-LDL/LDL but Delles et al. [19] investigated patients with CAD. Our patients had HT without CAD. Additionally, we did not observe a correlation between the oxidative stress markers and the superoxide anion production in the vascular wall [19]. However, in the study by Delles et al. [19], patients with CAD were treated with ACE inhibitors, statins. Delles et al. [19] showed a correlation between O_2 production in the vascular wall and aortic compliance which persisted even after adjusting for classical risk factors. In multivariate analysis, we observed a positive correlation between AIx p and AOPP and a negative correlation for SDMA and ox-LDL/LDL ratio in normotensives. There was a positive correlation between ADMA and central pulse pressure in this group. The relation-

ship between ox-LDL/LDL and arterial stiffness was shown in a group of young, middle-aged subjects and in the elderly (n = 2295) [20]. Brinkley et al. [20] observed that higher ox-LDLs are related to higher LDL and triglyceride, and that smoking, age, and drugs were not different in terciles of ox-LDL. The same investigators compared PWV in the same age category with high and low ox-LDL values and described higher arterial stiffness in the group with higher ox-LDL values.

Our study did not confirm the hypothesis that ox-LDL are markers of oxidative stress and are independent of other cardiovascular risk factors influencing arterial stiffness.

Zoccali et al. [5] observed that elevated ADMA were independent predictors of carotid IMT. Furuki et al. [21] showed that ADMA correlates with carotid IMT independently of BP, age and sex. In our observations in multivariate analysis the relationship between AOPP and IMT stayed significant in hypertensive patients. ADMA was also shown to be connected with IMT in healthy subjects, which suggests it is an important marker of atherosclerosis [2].

We suspected that antihypertensive pharmacotherapy may be one of the reasons why we did not observe any differences in oxidative stress markers between normotensives and hypertensives. Most of our patients were on drugs which inhibited the RAA system. These drugs have a proven effect on oxidative stress markers [22–24]. Additionally, several individuals were treated with statins or beta-blockers which also interfere with oxidative stress [25].

The novel findings of our study may throw some light on understanding the relation between organ damage and oxidative stress in HT and on understanding the role of methylated arginines or advanced oxidation protein products which may be significant in the search for new antihypertensive drugs.

CONCLUSIONS

We found that the influence of oxidative stress on the structure and function of large arteries is independent of HT. Therefore, oxidative stress may play a significant role in the development of arterial stiffness. Higher oxidative stress is associated with more advanced arterial remodelling in HT.

Conflict of interest: *none declared*

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Wskaźniki stresu oksydacyjnego a struktura i funkcja tętnic w nadciśnieniu tętniczym

Łukasz Klima[1], Kalina Kawecka-Jaszcz[1], Katarzyna Stolarz-Skrzypek[1], Jan Menne[2], Kamil Fijorek[3], Agnieszka Olszanecka[1], Wiktoria Wojciechowska[1], Grzegorz Bilo[4], Danuta Czarnecka[1]

[1]I Klinika Kardiologii i Nadciśnienia Tętniczego, *Collegium Medicum*, Uniwersytet Jagielloński, Kraków
[2]Klinika Nefrologii, Medyczna Szkoła Wyższa w Hanowerze, Hanower, Niemcy
[3]Katedra Statystyki, Uniwersytet Ekonomiczny, Kraków
[4]Klinika Kardiologii, Włoski Instytut Auksologii, Mediolan, Włochy

Streszczenie

Wstęp: Zależność między nadciśnieniem tętniczym a stresem oksydacyjnym nadal jest przedmiotem badań. Udowodniono, że reaktywne formy tlenu wpływają na ścianę tętnic oraz funkcję śródbłonka i w ten sposób mogą determinować wartość ciśnienia tętniczego.

Cel: Celem badania była ocena wybranych markerów stresu oksydacyjnego u pacjentów z nadciśnieniem tętniczym oraz ocena zależności między tymi markerami a wskaźnikami struktury i funkcji naczyń tętniczych.

Metody: Do badania włączono 217 osób (114 kobiet, 103 mężczyzn), pochodzących z rodzin, w których występowało nadciśnienie tętnicze. Średni wiek badanych wynosił 45,5 ± 16 lat, u 155 osób rozpoznano nadciśnienie tętnicze. U każdego z uczestników wykonano pomiar prędkości fali tętna oraz ultrasonograficzną ocenę grubości kompleksu błony środkowej i wewnętrznej (IMT) tętnic szyjnych wspólnych. Jako wskaźniki stresu oksydacyjnego oznaczono asymetryczną dimetyloargininę (ADMA), symetryczną dimetyloargininę (SDMA), produkty zaawansowanej oksydacji białek (AOPP) i utlenowane lipoproteiny o niskiej gęstości (ox-LDL).

Wyniki: W analizie wieloczynnikowej wykazano, że u pacjentów z nadciśnieniem stężenie ADMA zwiększało się wraz ze wzrostem obwodowego ciśnienia tętna (b = 0,003; p < 0,001), a AOPP wiązało się z wyższym wskaźnikiem IMT w tętnicach szyjnych (b = 0,91; p = 0,036). Wśród osób z prawidłowymi wartościami ciśnienia zaobserwowano zależności między ADMA i centralnym ciśnieniem tętna (b = 0,006; p = 0,008), SDMA i obwodowym wskaźnikiem wzmocnienia (b = –0,03; p = 0,014), AOPP i obwodowym wskaźnikiem wzmocnienia (b = 0,011; p = 0,04), wskaźnikiem ox-LDL/LDL a obwodowym wskaźnikiem wzmocnienia (b = –0,025; p = 0,004). Wskaźnik SDMA/ADMA wiązał się z estymowaną filtracją kłębuszkową w obydwu grupach (odpowiednio: b = –0,0061; p < 0,0001 i b = –0,005; p < 0,017). U pacjentów z nadciśnieniem tętniczym zaobserwowano związek powyższego parametru z obwodowym ciśnienia tętna (b = –0,0067; p = 0,0143). Ponadto u osób z prawidłowymi wartościami ciśnienia zanotowano zależność między wskaźnikiem SDMA/ADMA a stężeniem kwasu moczowego (b = 1,3629; p = 0,046).

Wnioski: Stres oksydacyjny wpływa na strukturę i funkcję naczyń tętniczych niezależnie od nadciśnienia tętniczego i może odgrywać istotną rolę w rozwoju sztywności tętnic.

Słowa kluczowa: stres oksydacyjny, sztywność tętnic, nadciśnienie tętnicze

Adres do korespondencji:
dr n. med. Łukasz Klima, I Klinika Kardiologii i Nadciśnienia Tętniczego, *Collegium Medicum*, Uniwersytet Jagielloński, ul. Kopernika 17, 31–501 Kraków, tel: +48 12 424 73 00, e-mail: lklima@s.u.krakow.pl