Psychiatr. Pol. 2019; 53(5): 1003–1020
PL ISSN 0033-2674 (PRINT), ISSN 2391-5854 (ONLINE)
www.psychiatriapolska.pl
DOI: https://doi.org/10.12740/PP/108453

Personality traits and hypertension-mediated organ damage

Lech Popiołek[1], Olga Siga[2], Anna Dzieża-Grudnik[2], Iwona Popiołek[3], Małgorzata Moląg[3], Jarosław Królczyk[2], Tomasz Grodzicki[2], Jolanta Walczewska[2], Krzysztof Rutkowski[1]

[1] Jagiellonian University Medical College, Chair of Psychotherapy
[2] Jagiellonian University Medical College, Chair of Internal Medicine and Gerontology
[3] University Hospital in Krakow

Summary

Aim. Arterial hypertension may lead to the development of organ changes. This study compares different personality traits in hypertensive patients with and without left ventricular hypertrophy and arterial stiffness.

Methods. The study group consisted of 93 subjects (47 males and 46 females) with primary hypertension. Left ventricular mass index (LVMI) and pulse wave velocity (PWV) were evaluated and used as markers of cardiac and vascular damage. Personality traits of each patient were assessed using three psychometric tools: NEO-FFI, DS14 and EAS.

Results. Patients with increased PWV scored significantly lower than individuals with normal PWV in the following scales: NEO-FFI Neuroticism (18 vs. 27.5; $p = 0.018$), DS14 Negative affectivity (11.5 vs. 17; $p = 0.035$) and EAS Fear (10 vs. 13; $p = 0.004$). Subjects with left ventricular hypertrophy (increased LVMI values) presented lower levels of openness to experience (measured by the NEO-FFI) than persons with normal LVMI values (23 vs. 26; $p = 0.027$).

Conclusions. These findings suggest that there are significant differences in personality traits between hypertensive patients with and without vascular and cardiac damage.

Key words: personality, arterial hypertension, hypertensive target organ damage

Introduction

Hypertension-mediated organ damage

Arterial hypertension (HT) is a chronic medical condition in which the blood pressure in the arteries is elevated. It is commonly defined as persistent systolic

blood pressure ≥ 140 mmHg and/or persistent diastolic blood pressure ≥ 90 mmHg [1]. Chronically elevated blood pressure may lead to the development of changes in major organs fed by the circulatory system, such as: heart, kidneys, brain, and eyes [2]. These changes are grouped under the term 'target organ damage' (TOD) or 'hypertension-mediated organ damage' (HMOD). ESH/ESC guidelines for the management of arterial hypertension enumerate the following symptoms of HMOD: left ventricular hypertrophy, increased pulse wave velocity, carotid wall thickening or the presence of atherosclerotic plaque, microalbuminuria, decreased estimated glomerular filtration rate, decreased ankle-brachial index and increased pulse pressure in the elderly [1].

The mechanism underlying organ changes in the course of arterial hypertension is complex. Contemporary studies show that at least the following pathophysiological processes are involved in the development of HMOD: endothelial activation, platelet activation and changes in the renin-angiotensin-aldosterone system [2]. To date, many different factors associated with the development of HMOD have been identified (e.g., subjects' age, body fat, blood pressure, smoking status or physical activity level [3–6]). However, to our knowledge, there were no attempts to identify psychological variables that differentiate patients with and without HMOD.

Personality and health

Mutual relations between psychological functioning and somatic health have been studied for many years. Different studies showed that, among others, variables associated with personality may be significantly related to cardiovascular health (e.g., [7–9]). Many studies in this field were based on the big five personality traits theory (i.e., on the five factor model of personality). According to this model, each person (independently of their sex and culture [10]) may be described using five different and independent domains: neuroticism, extraversion, openness to experience, conscientiousness, and agreeableness [11]. Contemporary studies on the associations between five broad dimensions of personality and health revealed that high level of openness to experience may be treated as a protective factor for incident coronary heart disease [7], while high level of conscientiousness may be protective against metabolic syndrome [12].

Other studies on the associations between personality variables and somatic health were based on type D personality theory. People with this type of personality are characterized by the tendency to experience increased negative emotions across time and situations (in other words they have tendency towards negative affectivity). They also tend not to share these emotions with others because of fear of disapproval

(this dimension of type D personality is called social inhibition) [8]. To date, studies revealed that type D personality may be treated as significant predictor of mortality in patients with established coronary heart disease [8] and may be associated with increased prevalence of ventricular arrhythmias in people without coronary heart disease [13]. The prevalence of type D personality among people with arterial hypertension reaches 53% [14].

Some studies in the field of psychosomatics were focused on specific aspects of individual's personality that are regarded to be innate (rather than learned) and that appear during the first year of life. This set of dispositions is collectively called temperament. According to the popular theory created by Buss and Plomin, there are only three basic temperamental variables: emotionality, activity and sociability [15, 16]. One of these variables – activity – has been associated by Finnish researchers with the development of carotid atherosclerosis in young males [17].

The mechanism underlying observed associations between personality traits (or temperamental traits) and cardiovascular health is complex and not well understood. Many independent studies show that some personality traits may be linked to health behaviors, which may be either risky (e.g., smoking, unprotected sex or substance abuse) or protective (e.g., physical activity, weight control or regular dental examinations) [18–20].

Personality may be also linked to health through the situations that people select or evoke [21]. For example, individuals who are high on conscientiousness are more likely to remain consistently married, which has proven to be beneficial for health [22]. What is more, three personality traits that are described in the five-factor model (extraversion, agreeableness and conscientiousness [23]) are related to the quantity and quality of people's social relationships and through this pathway may influence the risk of mortality [24].

Some studies suggest that specific personality traits are associated with the level of pro-inflammatory cytokines. For example, according to Sutin et al. [25] individuals who are high on neuroticism and low on conscientiousness show elevated levels of interleukin 6 (IL-6). At the same time, study by Denollet et al. [26] shows that type D personality is associated with increased tumor necrosis factor alpha (TNF-α) activity in patients with chronic heart failure. Both, IL-6 an TNF-α, were found to be significantly higher in patients with HMOD than in hypertensive individuals without such health complications [27].

All of the abovementioned mechanisms may underlie potential associations between specific personality traits (or temperamental traits) and hypertension-mediated organ damage. Due to the high complexity of topics discussed in this work and due to the fact that relations between personality and hypertensive target organ damage have

not been studied yet, our research hypothesis would be very general. We hypothesize that hypertensive patients with cardiac and vascular damage have different levels of personality traits than individuals with hypertension, but without HMOD.

During the project two databases (MEDLINE and Scopus) have been screened. The following key words have been used: blood pressure, DS14, EAS, end organ damage, HMOD, hypertension, hypertension-mediated organ damage, hypertensive organ damage, left ventricular hypertrophy, left ventricular mass index, LVH, LVMI, markers of TOD, NEO-FFI, NEO-PI-R, organ damage, personality, personality differences, psychology, psychopathology, pulse wave velocity, PWV, target organ damage, TOD, type D personality. No articles on the relationship between hypertension-mediated organ damage and personality have been found.

Material and methods

This study was performed at the Hypertension Outpatient Clinic of the University Hospital in Krakow. It was approved by the Bioethics Committee of the Jagiellonian University (KBET/151/B/2012). Research participants gave informed consent for all procedures performed during the study. This paper is a partial report of the results that were revealed during the study.

Study group

The study population consisted of consecutive patients seen in the Hypertension Outpatient Clinic, who fulfilled the following criteria: age – at least 18 years; ethnicity – Caucasian; confirmed diagnosis of primary arterial hypertension. The exclusion criteria were: documented history of traumatic brain injury; documented history of mental disorders (especially: dementia, schizophrenia, mood disorders, substance dependence); high scores (T-score > 65) in clinical scales of the MMPI-2; current psychiatric medications intake; systolic heart failure; chronic kidney disease; acute or chronic inflammation; neoplasm.

Variables and measurements

All study participants have undergone clinical assessment. All patients have also been offered to participate in the following procedures: echocardiography, pulse wave velocity measurement and psychological testing.

Clinical assessment

Each patient was interviewed to obtain a detailed medical and lifestyle history, specifically including the following data: history of hypertension; current and past chronic diseases; tobacco, drug and alcohol use; physical activity level; medication use and family history of cardiovascular disease. Each interview was supplemented with analysis of medical records submitted by the patient.

Subjects' weight and height were measured and used to calculate body mass index (BMI). Waist and hip measures were taken to assess waist-to-hip ratio (WHR). Patients' blood samples were collected to perform basic biochemical blood tests.

Participants' blood pressure was assessed with the use of ambulatory blood pressure monitoring method (ABPM). 24-hour ABPM was performed using Spacelabs 90207 device (Spacelabs Healthcare, Snoqualmie, WA, USA). The ABPM readings took place on weekdays, and patients were advised to work and behave as usual.

Echocardiography

Echocardiography was performed in 89 out of 93 patients (data from 4 patients were not taken into consideration due to the poor quality of echocardiographic images). Each measurement was performed by an experienced physician using a Toshiba Xario XG device (Toshiba, Tokyo, Japan) equipped with a 2.5–3.5 MHz array transducer probe. Left ventricular mass index (LVMI) was calculated according to the guidelines issued by the American Society of Echocardiography [28].

Women with LVMI values exceeding 95 g/m^2 and men with LVMI values exceeding 115 g/m^2 were included in the group with left ventricular hypertrophy [1].

Pulse wave velocity measurement

Pulse wave velocity (PWV) was assessed using a COMPLIOR device (Colson, Garges les Genosse, France). It was calculated by dividing 80% of the direct carotid-femoral distance by pulse wave transit time [29]. Each measurement was performed by qualified physician following a standardized protocol. The statistical analysis of gathered data was based on the mean of 10 consecutive PWV measurements after 10 minutes of rest in a supine position in a quiet room with stable room temperature [30].

PWV values > 10 m/s in both sexes were classified as increased [1]. Increased PWV was treated as a marker of arterial stiffness.

Psychological testing

Patients enrolled into the study were assessed using the following psychometric methods: NEO-FFI, DS14 and EAS. Each instrument was translated into Polish and standardized by the Psychological Test Laboratory of the Polish Psychological Association [11, 16, 31]. Psychological assessment of each patient was performed by a licensed psychologist trained at the Department of Psychotherapy, Jagiellonian University Medical College.

Five main dimensions of subjects' personality were assessed using the NEO-FFI. This tool consists of 5 scales, that measure individuals' neuroticism, extraversion, openness to experience, conscientiousness, and agreeableness [11]. Each scale is comprised of 12 items, which are rated according to a 5-point scale. Raw testing results may be compared to standardized scores separately for men and women of different ages. The psychometric properties of the Polish version of the NEO-FFI are satisfactory with Cronbach's α ranging from 0.68 to 0.82 for all five scales of the inventory [11]. The following Cronbach's α coefficients were calculated in the study group: 'Openness to experience' – 0.66; 'Conscientiousness' – 0.80; 'Extraversion' – 0.77; 'Agreeableness' – 0.78; 'Neuroticism' – 0.86.

Type D personality was assessed using the DS14 scale. This psychometric method consists of two subscales that measure negative affectivity and social inhibition [14]. Each subscale consists of 7 items, which are rated according to a 5-point scale (from 0 – 'false' to 4 – 'true'). People who score high (≥ 10 points) on both negative affectivity and social inhibition are classified as type D [31]. Psychometric properties of the Polish version of the DS14 scale are good with Cronbach's α of 0.86 and 0.84 and test–retest reliability of 0.74 and 0.70 for the 'Negative affectivity' and 'Social inhibition' subscales, respectively [31]. In case of our study group Cronbach's α coefficients were: 0.90 for the 'Negative affectivity' subscale and 0.86 for the 'Social inhibition' subscale.

The EAS Temperament Survey for adults (in Poland: EAS-D) was used to assess temperamental traits. This tool consists of 20 items, which form 5 scales: Emotionality-Distress, Emotionality-Fear, Emotionality-Anger, Activity, Sociability [32]. Each scale consists of 4 statements, which are rated using 5-point scale (from 1 – 'definitely disagree' to 5 – 'definitely agree'). Psychometric properties of the EAS-D are acceptable with Cronbach's α ranging from 0.57 to 0.74 and two-week test–retest reliability ranging from 0.71 to 0.84 for all five scales of the method [16]. Statistical analysis of data obtained during the study revealed the following Cronbach's α coefficient values: 'Emotionality-Distress' – 0.76; 'Emotionality-Fear' – 0.74; 'Emotionality-Anger' – 0.66; 'Activity' – 0.57 and 'Sociability' – 0.62.

Statistical analysis

Statistical analysis of gathered data was performed using STATISTICA 12.0 PL software (StatSoft, Tulsa, OK, USA). The significance level (alpha) was set at 0.05. Statistical distribution of each variable was assessed using the Shapiro-Wilk test. Normally distributed variables were compared using Student's *t*-tests. Mann-Whitney *U* tests and Kruskal-Wallis tests were applied to compare variables that were not normally distributed. Chi-squared tests were used to compare proportions in the study subgroups. Correlation analysis was based on Spearman's rank correlation coefficients.

Results

The study group consisted of 93 adult people (46 women and 47 men) with primary arterial hypertension. Baseline characteristics of the study population are shown in Table 1. Descriptive statistics for psychological variables in the study group are shown in Table 2.

Table 1. **Baseline characteristics of the study group**

Variables	N = 93	Reference range
Age, years, median (IQR)	49 (41–57)	-
Women, n (%)	46 (49.46%)	-
BMI(kg/m^2), median (IQR)	28.09 (25.01–31.25)	18.50–24.99
WHR, median (IQR)	0.93 (0.83–0.99)	< 0.85 (women) or < 0.90 (men)
Blood pressure		
ABPM SBP (mmHg), mean (SD)	127.86 (12.70)	< 130.00
ABPM DBP (mmHg), mean (SD)	79.25 (9.20)	< 80.00
Nocturnal SBP dipping (%), median (IQR)	11.03 (7.77–13.78)	10.00–20.00
Nocturnal DBP dipping (%), mean (SD)	22.65 (8.56)	10.00–20.00
Laboratory measures		
Fasting blood glucose (mmol/L), median (IQR)	5.12 (4.61–5.81)	3.30–5.60
HbA1c (%), median (IQR)	5.60 (5.40–5.80)	4.30–5.90
Total cholesterol (mmol/L), median (IQR)	5.30 (4.60–6.00)	3.20–5.20
LDL (mmol/L), median (IQR)	3.00 (2.40–3.70)	< 3.40
HDL (mmol/L), median (IQR)	1.50 (1.23–1.86)	> 1.00

table continued on the next page

Triglycerides (mmol/L), median (IQR)	1.40 (0.99–1.75)	< 2.26
Serum creatinine (μmol/L), median (IQR)	73.00 (67.00–82.00)	62.00–106.00
Markers of hypertension-mediated organ damage		
PWV (m/s), median (IQR)	12.07 (10.68–14.45)	≤ 10
LVMI (g/m^2), median (IQR)	95.88 (77.66–110.82)*	≤ 95 (women) or ≤ 115 (men)

*N = 89

ABPM – ambulatory blood pressure monitoring; BMI – body mass index; DBP – diastolic blood pressure; HDL – high density lipoproteins; IQR – interquartile range; LDL – low density lipoproteins; LVMI – left ventricular mass index; PWV – pulse wave velocity; SBP – systolic blood pressure; WHR – waist to hip ratio

Table 2. **Descriptive statistics for psychological variables in the study group**

Variables	Minimum	Q_1	Median (Q_2)	Q_3	Maximum
NEO-FFI					
Neuroticism	5.0	15.0	20.0	29.0	44.0
Extraversion	7.0	22.0	27.0	31.0	43.0
Openness to experience	11.0	21.0	25.0	29.0	37.0
Agreeableness	8.0	27.5	32.0	36.0	42.0
Conscientiousness	18.0	28.5	33.5	38.0	46.0
DS14					
Negative affectivity	0.0	7.5	12.0	17.0	28.0
Social inhibition	0.0	5.0	9.0	14.0	22.0
EAS					
Emotionality-Distress	5.0	8.0	12.0	14.0	20.0
Emotionality-Fear	5.0	8.0	10.5	13.0	20.0
Emotionality-Anger	4.0	10.0	12.0	14.0	20.0
Activity	7.0	11.0	14.0	16.0	20.0
Sociability	6.0	10.0	13.5	15.0	20.0

Q_1 – quartile 1; Q_2 – quartile 2; Q_3 – quartile 3

Study participants were treated with the following antihypertensive medications: diuretics (more than 46% of the study group), calcium channel blockers (almost 40%),

angiotensin-converting-enzyme inhibitors (more than 37%), beta-blockers (about 33%), angiotensin II antagonists (almost 19%), and alpha-blockers (more than 8%). All subjects enrolled into the study were treated according to the recent ESC/ESH guidelines for the management of arterial hypertension. In the case of each study participant treatment scheme was adequate to patients' clinical state.

Correlation analysis

Correlation analysis revealed some significant associations between measured variables. Openness to experience (measured using the NEO-FFI) was negatively correlated with left ventricular mass index ($r = -0.23$; $p = 0.034$). Pulse wave velocity was negatively correlated with activity level assessed using the EAS 'Activity' scale ($r = -0.21$; $p = 0.041$).

Pulse wave velocity was linearly associated with patients' age ($r = 0.49$; $p < 0.001$). Correlation between LVMI and age was not observed. There was no linear correlation between pulse wave velocity and left ventricular mass index.

Comparison of patients with normal and increased values of PWV

The median age of patients with high PWV was significantly higher than that of individuals with normal PWV (50 vs. 45; $p = 0.023$). Patients with high PWV were also more often treated with angiotensin-converting-enzyme inhibitors (ACEI) than individuals with normal PWV (40% vs. 11.11%; $p = 0.021$). In the case of other variables, that are enumerated in Table 3, there were no significant differences between these two groups of patients.

Table 3. **Baseline characteristics of the study group according to normal/high values of PWV and normal/increased LVMI values**

Variables	Normal PWV (n = 18)	Increased PWV (n = 75)	p	Normal LVMI (n = 58)	Increased LVMI (n = 31)	p
Age, years, median (IQR)	45 (34–50)	50 (41–59)	0.023	48.5 (41–57)	49 (41–57)	0.966
Women, n (%)	9 (50.00%)	37 (49.30%)	0.959	32 (55.17%)	13 (41.94%)	0.234
Tobacco smoking, n (%)	8 (44.44%)	39 (52.00%)	0.591	30 (51.72%)	15 (48.39%)	0.744
BMI (kg/m^2), median (IQR)	26.67 (24.15–31.25)	28.65 (25.01–31.49)	0.789	27.86 (24.68–31.20)	29.35 (26.04–32.47)	0.089

table continued on the next page

WHR, median (IQR)	0.92 (0.84–0.98)	0.93 (0.83–0.99)	0.953	0.91 (0.81–0.99)	0.95 (0.88–0.99)	0.466
Blood pressure						
ABPM SBP (mmHg), mean (SD)	127.28 (14.85)	128.00 (12.23)	0.830	126.05 (13.16)	130.90 (11.86)	0.090
ABPM DBP (mmHg), mean (SD)	82.00 (11.54)	78.60 (8.50)	0.160	78.48 (8.36)	80.13 (10.98)	0.431
Nocturnal SBP dipping (mmHg), median (IQR)	11.45 (8.59–14.03)	11.01 (7.12–13.71)	0.558	11.13 (8.54–14.03)	10.30 (5.26–13.50)	0.226
Nocturnal DBP dipping (mmHg), mean (SD)	22.97 (8.27)	22.57 (8.68)	0.860	23.91 (8.41)	21.08 (8.62)	0.053
Laboratory measures						
Fasting blood glucose (mmol/L), median (IQR)	4.83 (4.57–5.23)	5.18 (4.76–5.82)	0.169	5.06 (4.57–5.82)	5.14 (4.76–5.81)	0.458
HbA1c (%), median (IQR)	5.50 (5.40–5.70)	5.60 (5.40–5.90)	0.329	5.55 (5.40–5.80)	5.60 (5.40–5.80)	0.713
Total cholesterol (mmol/L), median (IQR)	5.05 (4.60–5.70)	5.30 (4.70–6.00)	0.636	5.35 (4.70–6.00)	5.25 (4.70–5.90)	0.646
LDL (mmol/L), median (IQR)	2.85 (2.30–3.60)	3.00 (2.40–3.70)	0.910	3.10 (2.40–3.80)	2.90 (2.50–3.70)	0.927
HDL (mmol/L), median (IQR)	1.40 (1.11–1.70)	1.56 (1.25–1.87)	0.347	1.58 (1.26–1.92)	1.34 (1.17–1.68)	0.048
Triglycerides (mmol/L), median (IQR)	1.26 (0.94–1.61)	1.42 (1.00–1.78)	0.541	1.21 (0.89–1.68)	1.59 (1.23–2.11)	0.019
Serum creatinine, (μmol/L), median (IQR)	75.70 (71.0–83.00)	72.00 (67.00–82.00)	0.137	72.50 (67.00–79.00)	73.00 (68.00–84.00)	0.309

Data are median values compared using Mann-Whitney U test, mean vales compared using Student's t-test or proportions (%) compared using Chi-squared test

ABPM – ambulatory blood pressure monitoring; ACEI – angiotensin-converting-enzyme inhibitors; ARB – angiotensin II receptor blockers; ASA – acetylsalicylic acid; BMI – body mass index; CCB

– calcium channel blockers; DBP – diastolic blood pressure; HDL – high-density lipoproteins; IQR – interquartile range; LDL – low-density lipoproteins; LVMI – left ventricular mass index; NS – not significant; PWV – pulse wave velocity; SBP – systolic blood pressure; SD – standard deviation; WHR – waist to hip ratio

Both SBP and DBP were measured using ABPM.

Patients with normal PWV had significantly higher levels of negative affectivity (DS14), temperamental fear (EAS) and neuroticism (NEO-FFI) than subjects with increased PWV (Table 4). In the case of other psychological variables, no significant differences between these two groups of people were found.

Table 4. **Differences in psychological variables according to normal/high values of PWV and normal/increased LVMI values**

Variables	Normal PWV (n = 18)	Increased PWV (n = 75)	p	Normal LVMI (n = 58)	Increased LVMI (n = 31)	p
NEO–FFI						
Neuroticism, median (IQR)	27.5 (20.0–34.0)	18.0 (15.0–28.0)	0.018	20.0 (15.0–28.0)	20.0 (15.0–29.0)	0.578
Extraversion, median (IQR)	27.0 (22.0–31.0)	27.5 (22.0–31.0)	0.824	27.0 (22.0–31.0)	29.0 (23.0–31.0)	0.543
Openness to experience, median (IQR)	26.0 (25.0–30.0)	24.0 (20.0–29.0)	0.154	26.0 (22.0–30.0)	23.0 (19.0–27.0)	0.027
Agreeableness, median (IQR)	32.0 (28.0–35.0)	32.0 (27.0–36.0)	0.733	33.0 (30.0–37.0)	31.0 (26.0–35.0)	0.053
Conscientiousness, median (IQR)	30.5 (29.0–36.0)	34.0 (28.0–38.0)	0.388	34.0 (30.0–38.0)	33.0 (27.0–37.0)	0.506
DS14						
Negative affectivity, median (IQR)	17.0 (9.0–22.0)	11.5 (7.0–16.0)	0.035	12.0 (8.0–17.0)	11.5 (7.0–17.0)	0.728
Social inhibition, median (IQR)	11.0 (6.0–14.0)	8.5 (5.0–13.0)	0.265	8.5 (5.0–13.0)	9.5 (6.0–14.0)	0.259
EAS						
Emotionality-Distress, median (IQR)	13.5 (10.0–14.0)	11.0 (8.0–14.0)	0.153	12.0 (8.0–14.0)	11.5 (8.0–15.0)	0.733
Emotionality-Fear, median (IQR)	13.0 (10.0–16.0)	10.0 (8.0–13.0)	0.004	10.0 (8.0–13.0)	11.0 (9.0–12.0)	0.856
Emotionality-Anger, median (IQR)	13.0 (10.0–14.0)	11.0 (10.0–14.0)	0.162	12.0 (10.0–14.0)	12.0 (10.0–15.0)	0.700

table continued on the next page

Activity, median (IQR)	15.0 (13.0–16.0)	13.0 (11.0–15.0)	0.117	14.0 (11.0–16.0)	13.5 (12.0–15.0)	0.901
Sociability, median (IQR)	14.0 (12.0–15.0)	13.0 (10.0–15.0)	0.703	13.5 (11.0–15.0)	12.0 (10.0–15.0)	0.568

Data are median values compared using Mann-Whitney U test.

IQR – interquartile range; LVMI – left ventricular mass index; NS – not significant; PWV – pulse wave velocity

Comparison of patients with normal and increased values of LVMI

Comparative analysis showed no significant differences between patients with increased LVMI and subjects with normal LVMI in the distribution of majority of variables listed in Table 3. People with high left ventricular hypertrophy had significantly higher average levels of triglycerides (medians: 1.59 vs. 1.21 mmol/L; $p = 0.020$) and significantly lower average levels of HDL (medians: 1.34 vs. 1.58 mmol/L; $p = 0.048$) than patients with normal LVMI. The proportion of subjects treated with diuretics and the proportion of individuals treated with ACEI were significantly higher in the case of patients with increased LVMI in comparison to patients with normal LVMI (61.29% vs. 29.31%; $p = 0.003$ and 48.39% vs. 25.86%; $p = 0.032$).

Subjects with normal LVMI scored significantly higher than study participants with left ventricular hypertrophy in the NEO-FFI ‘Openness to experience’ scale. There were no significant differences between these two groups of patients in scales: NEO-FFI ‘Neuroticism’, NEO-FFI ‘Extraversion’, NEO-FFI ‘Conscientiousness’, NEO-FFI ‘Agreeableness’, and in all DS14 and EAS scales (Table 4).

The prevalence of type D personality in the study group

37 patients (39.78% of the study group) were initially diagnosed with type D personality using the DS14 scale (it is important to notice that final diagnosis must be based on thorough interview, and not on the basis of screening test). No significant differences in the prevalence of type D personality were found between subjects with normal and high PWV and between individuals with normal and increased LVMI.

Discussion

The following relationships were found during the study:

(1) High PWV values were associated with low EAS ‘Activity’ scale scores.

(2) High LVMI values were associated with low levels of ‘Openness to experience’ measured by the NEO-FFI.
(3) Hypertensive patients with normal PWV values scored significantly higher than subjects with hypertension and arterial stiffness in the following scales: DS14 ‘Negative affectivity’, EAS ‘Emotionality-Fear’ and NEO-FFI ‘Neuroticism’.
(4) Individuals with normal LVMI values scored significantly higher than subjects with left ventricular hypertrophy in the NEO-FFI ‘Openness to experience’ scale.
(5) There were no statistically significant differences in the prevalence of type D personality between hypertensive patients with normal and high PWV values.
(6) The prevalence of type D personality in the study group was not significantly different between patients with normal LVMI values and subjects with left ventricular hypertrophy.

The first result should be interpreted carefully due to the low reliability of the EAS ‘Activity’ scale in our study group. The observed relationship between pulse wave velocity and the EAS ‘Activity’ scale scores may be at least partially mediated by physical activity level. Some studies show that subjects with higher temperamental activity level prefer pastimes that involve physical activity [33]. They exercise more than other people and they are less likely to develop arterial stiffness. This line of thought is supported by results of many different studies, which show that physical exercise may improve arterial stiffness [34].

In our opinion, the link between openness to experience and LVMI values may be understood by a reference to a study by Williams et al. [35], according to whom individuals with high levels of this personality trait have lower blood pressure reactivity than other people. At the same time, there is some evidence that blood pressure hyper-reactivity may be linked to left ventricular hypertrophy [36]. These findings combined suggest that lower blood pressure reactivity (associated with higher openness to experience) may be linked to lower tendency towards left ventricular hypertrophy. It is important to note that the abovementioned result of this study should be interpreted carefully due to the low value of Cronbach’s alpha in the case of the NEO-FFI ‘Openness to experience’ scale in the study group.

The third finding is surprising because there is a large body of evidence that high levels of negative affectivity, fear (as one of temperamental dimensions) and neuroticism may contribute to the development of different somatic diseases [e.g., 12, 13, 37–39]. However, there are also studies which show that higher levels of neuroticism may be associated with some health benefits. In 2013, American researchers revealed that higher levels of neuroticism (accompanied by higher levels of conscientiousness)

may be related to lower blood levels of pro-inflammatory interleukin 6 [40]. Nine years earlier, Canadian researchers showed that higher neuroticism may be associated with lower heart rate and lower levels of serum norepinephrine during an aversive challenge [41]. Unfortunately, during our research we were not able to find convincing studies focused on health benefits related to high levels of negative affectivity and high levels of temperamental fear. However, since neuroticism, negative affectivity and fear as the temperamental trait are closely related constructs, we may assume that increased negative affectivity and increased temperamental fear may also have some positive impact on patients' health. In our opinion the mechanism behind observed differences in negative affectivity, temperamental fear and neuroticism in the study group is very simple. Patients who score high in these three scales are more concerned about their health than other people. That is why they quickly seek for medical advice when they detect any health problems. Consequently, they may be diagnosed with HT at early stage of disease, they may start treatment early and they may develop less advanced hypertension-mediated organ damage than other people.

The fourth result stays in line with the second finding. In our opinion, observed differences in openness to experience level between subjects with and without left ventricular hypertrophy should be attributed to blood pressure reactivity.

This fifth finding and sixth result should be interpreted carefully due to a small sample size (e.g., there were only 8 people suspected of type D personality and normal values of PWV). It is also important to remember that in statistical methods used in this study the absence of evidence for differences between the analyzed groups is not evidence of their absence [42].

Psychological testing using the DS14 scale suggested that type D personality may be found in 37 participants (39.87% of the study population). This result is not surprising because different studies show that the prevalence of type D personality among hypertensive patients ranges between 30.7% [43] and 53% [14].

This study has some limitations. First, it was conducted among patients from a single outpatient clinic, so the results cannot be easily generalized to the population of patients with arterial hypertension. Second – it was designed as cross-sectional study, so we were not able to assess the direction of causality between measured variables. Third – some findings of this study should be interpreted carefully due to the low reliability of four scales (NEO-FFI 'Openness to experience', EAS 'Emotionality-Anger', EAS 'Activity' and EAS 'Sociability') in our study group. And last, but not least – because of the small sample size we were not able to perform methodologically correct subgroup analysis according to patients' age, sex and treatment scheme.

The findings of our study suggest that there are significant differences in personality traits between hypertensive patients with and without hypertension-mediated organ

damage. However, it is important to notice that all of the abovementioned outcomes should be treated as preliminary and should be confirmed in further studies.

Conclusions

1. Patients with hypertension-mediated vascular damage had lower levels of neuroticism, negative affectivity and temperamental fear than hypertensive subjects without vascular damage.
2. Study participants with hypertension-mediated cardiac damage (left ventricular hypertrophy) had lower levels of openness to experience than hypertensive persons without cardiac damage.

References

1. Mancia G, Fagard R, Narkiewicz K, Redón J, Zanchetti A, Böhm M et al. *2013 ESH/ESC Guidelines for the management of arterial hypertension: The Task Force for the management of arterial hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC)*. Eur. Heart J. 2013; 34(28): 2159–2219.
2. Nadar SK, Tayebjee MH, Messerli F, Lip GY. *Target organ damage in hypertension: Pathophysiology and implications for drug therapy*. Curr. Pharm. Des. 2006; 12(13): 1581–1592.
3. Blacher J, Asmar R, Djane S, London GM, Safar ME. *Aortic pulse wave velocity as a marker of cardiovascular risk in hypertensive patients*. Hypertension 1999; 33(5): 1111–1117.
4. Levy D, Anderson KM, Savage DD, Kannel WB, Christiansen JC, Castelli WP. *Echocardiographically detected left ventricular hypertrophy: Prevalence and risk factors. The Framingham Heart Study*. Ann. Intern. Med. 1988; 108(1): 7–13.
5. Park W, Park H-Y, Lim K, Park J. *The role of habitual physical activity on arterial stiffness in elderly individuals: A systematic review and meta-analysis*. J. Exerc. Nutr. Biochem. 2017; 21(4): 16–21.
6. Elewa U, Fernandez-Fernandez B, Alegre R, Sanchez-Niño MD, Mahillo-Fernández I, Perez-Gomez MV et al. *Modifiable Risk Factors for Increased Arterial Stiffness in Outpatient Nephrology*. Aguilera AI, editor. PLoS One 2015; 10(4): e0123903.
7. Lee HB, Offidani E, Ziegelstein RC, Bienvenu OJ, Samuels J, Eaton WW et al. *Five-factor model personality traits as predictors of incident coronary heart disease in the community: A 10.5-year cohort study based on the Baltimore epidemiologic catchment area follow-up study*. Psychosomatics 2014; 55(4): 352–361.
8. Denollet J, Sys SU, Stroobant N, Rombouts H, Gillebert TC, Brutsaert DL. *Personality as independent predictor of long-term mortality in patients with coronary heart disease*. Lancet 1996; 347(8999): 417–421.
9. Atroszko PA, Kowalczyk J, Kowalczyk W. *Emotion-related personality traits in hypertensive patients – Pilot study*. Arter. Hypertens. 2013; 17(1): 30–37.

10. Schacter DL, Gilbert DT, Wegner DM. *Psychology*. 2nd ed. New York, NY: Worth Publishers; 2011.

11. Zawadzki B. *Inwentarz osobowości NEO-FFI Paula T. Costy Jr i Roberta R. McCrae: adaptacja polska: podręcznik*. Warsaw: Psychological Test Laboratory of the Polish Psychological Association; 2010.

12. Sutin AR, Costa PT, Uda M, Ferrucci L, Schlessinger D, Terracciano A. *Personality and metabolic syndrome*. Age (Dordr.) 2010; 32(4): 513–519.

13. Einvik G, Dammen T, Namtvedt SK, Hrubos-Strøm H, Randby A, Kristiansen HA et al. *Type D personality is associated with increased prevalence of ventricular arrhythmias in community-residing persons without coronary heart disease*. Eur. J. Prev. Cardiol. 2014; 21(5): 592–600.

14. Denollet J. *DS14: Standard assessment of negative affectivity, social inhibition, and Type D personality*. Psychosom. Med. 2005; 67(1): 89–97.

15. Saudino KJ. *Behavioral genetics and child temperament*. J. Dev. Behav. Pediatr. 2005; 26(3): 214–223.

16. Oniszczenko W. *Kwestionariusz temperamentu EAS Arnolda H. Bussa i Roberta Plomina: wersje dla dorosłych i dla dzieci: adaptacja polska: podręcznik*. Warsaw: Psychological Test Laboratory of the Polish Psychological Association; 1997.

17. Pulkki-Råback L, Puttonen S, Elovainio M, Raitakari OT, Juonala M, Keltikangas-Jarvinen L. *Adulthood EAS-temperament and carotid artery intima-media thickness: The Cardiovascular Risk in Young Finns study*. Psychol. Health 2011; 26(1): 61–75.

18. Ginting H, Ven van de M, Becker ES, Näring G. *Type D personality is associated with health behaviors and perceived social support in individuals with coronary heart disease*. J. Health Psychol. 2016; 21(5): 727–737.

19. Vollrath ME, Torgersen S, Torgersen L. *Associations of children's Big Five personality with eating behaviors*. BMC Res. Notes. 2018; 11(1): article number 654 (2019). https://bmcresnotes.biomedcentral.com/articles/10.1186/s13104-018-3768-9 (retrieved: 20.02.2019).

20. Song M, Corwyn RF, Bradley RH, Lumeng JC. *Temperament and physical activity in childhood*. J. Phys. Act. Health 2017; 14(11): 837–844.

21. Kern ML, Friedman HS. *Personality and pathways of influence on physical health*. Soc. Personal. Psychol. Compass. 2011; 5(1): 76–87.

22. Perelli-Harris B, Hoherz S, Addo F, Lappegård T, Evans A, Sassler S et al. *Do marriage and cohabitation provide benefits to health in mid-life? The role of childhood selection mechanisms and partnership characteristics across countries*. Popul. Res. Policy Rev. 2018; 37(5): 703–728.

23. Asendorpf JB, Wilpers S. *Personality effects on social relationships*. J. Pers. Soc. Psychol. 1998; 74(6): 1531–1544.

24. Holt-Lunstad J, Smith TB, Layton JB. *Social relationships and mortality risk: A meta-analytic review*. Brayne C, editor. PLoS Med. 2010; 7(7): e1000316.

25. Sutin AR, Terracciano A, Deiana B, Naitza S, Ferrucci L, Uda M et al. *High neuroticism and low conscientiousness are associated with interleukin-6*. Psychol. Med. 2010; 40(9): 1485–1493.

26. Denollet J, Vrints CJ, Conraads VM. *Comparing Type D personality and older age as correlates of tumor necrosis factor-alpha dysregulation in chronic heart failure*. Brain Behav. Immun. 2008; 22(5): 736–743.

27. Morillas P, de Andrade H, Castillo J, Quiles J, Bertomeu-González V, Cordero A et al. *Inflammation and apoptosis in hypertension. Relevance of the extent of target organ damage*. Rev. Esp. Cardiol. (Engl. Ed.) 2012; 65(9): 819–825.

28. Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pellikka PA et al. *Recommendations for chamber quantification*. Eur. J. Echocardiogr. 2006; 7(2): 79–108.

29. Van Bortel LM, Laurent S, Boutouyrie P, Chowienczyk P, Cruickshank JK, De Backer T et al. *Expert consensus document on the measurement of aortic stiffness in daily practice using carotid-femoral pulse wave velocity*. J. Hypertens. 2012; 30(3): 445–448.

30. Safar M. *Arteries in clinical hypertension*. Philadelphia: Lippinott-Raven; 1996.

31. Juczyński Z, Ogińska-Bulik N, Polish Psychological Association, Psychological Test Laboratory. *Narzędzia pomiaru stresu i radzenia sobie ze stresem*. Warszawa: Psychological Test Laboratory of the Polish Psychological Association; 2012.

32. Pisula E, Kawa R, Danielewicz D, Pisula W. *The Relationship between temperament and autistic traits in a non-clinical students sample*. Pavlova MA, editor. PLoS One 2015; 10(4): e0124364.

33. Janssen JA, Kolacz J, Shanahan L, Gangel MJ, Calkins SD, Keane SP et al. *Childhood temperament predictors of adolescent physical activity*. BMC Public Health 2017; 17(1): 8.

34. Ashor AW, Lara J, Siervo M, Celis-Morales C, Mathers JC. *Effects of exercise modalities on arterial stiffness and wave reflection: A systematic review and meta-analysis of randomized controlled trials*. PloS One 2014; 9(10): e110034.

35. Williams PG, Rau HK, Cribbet MR, Gunn HE. *Openness to experience and stress regulation*. J. Res. Personal. 2009; 43(5): 777–784.

36. Kaneda R, Kario K, Hoshide S, Umeda Y, Hoshide Y, Shimada K. *Morning blood pressure hyperreactivity is an independent predictor for hypertensive cardiac hypertrophy in a community-dwelling population*. Am. J. Hypertens. 2005; 18(12): 1528–1533.

37. Warmuz-Stangierska I, Baszko-Błaszyk D, Sowiński J. *Emotions and features of temperament in patients with Addison's disease*. Endokrynol. Pol. 2010; 61(1): 90–92.

38. Brickman AL, Yount SE, Blaney NT, Rothberg ST, De-Nour AK. *Personality traits and long-term health status. The influence of neuroticism and conscientiousness on renal deterioration in type-1 diabetes*. Psychosomatics 1996; 37(5): 459–468.

39. Drossman DA, Leserman J, Li Z, Keefe F, Hu YJ, Toomey TC. *Effects of coping on health outcome among women with gastrointestinal disorders*. Psychosom. Med. 2000; 62(3): 309–317.

40. Turiano NA, Mroczek DK, Moynihan J, Chapman BP. *Big 5 personality traits and interleukin-6: Evidence for "healthy Neuroticism" in a US population sample*. Brain Behav. Immun. 2013; 28: 83–89.

41. LeBlanc J, Ducharme MB, Thompson M. *Study on the correlation of the autonomic nervous system responses to a stressor of high discomfort with personality traits*. Physiol. Behav. 2004; 82(4): 647–652.

42. Altman DG, Bland JM. *Statistics notes: Absence of evidence is not evidence of absence*. BMJ 1995; 311(7003): 485–485.

43. Oliva F, Versino E, Gammino L, Colombi N, Ostacoli L, Carletto S et al. *Type D personality and essential hypertension in primary care: A cross-sectional observational study within a cohort of patients visiting general practitioners*. J. Nerv. Ment. Dis. 2016; 204(1): 43–48.

Address: Lech Popiołek
Chair of Psychotherapy
Jagiellonian University Medical College
31-138 Kraków, Lenartowicza Street 14
e-mail: nhlech@gmail.com