Anna Niemirska, Łukasz Obrycki, Mieczysław Litwin

ORIGINAL PAPER

Department of Nephrology, Kidney Transplantation and Arterial Hypertension, The Children's Memorial Health Institute, Warsaw, Poland

Utility of pulse wave velocity and pulse wave analysis in assessment of hypertensive target organ damage in children with primary hypertension

Przydatność pomiarów prędkości fali tętna oraz analizy fali tętna w ocenie powikłań narządowych u dzieci z nadciśnieniem tętniczym pierwotnym

Streszczenie

Wstęp Sztywność ścian naczyń, określana pomiarem prędkości fali tętna (PWV), jest uważana za marker uszkodzenia narządowego u dorosłych z nadciśnieniem tętniczym. Analiza fali tętna (PWA) umożliwia ocenę centralnego ciśnienia tętniczego, wstecznej fali tętna, rzutu serca oraz całkowitego oporu obwodowego. Brak jednak danych dotyczących przydatności wyżej wymienionych pomiarów w ocenie ryzyka powikłań narządowych u dzieci z nadciśnieniem tętniczym.

Celem badania było określenie związku między PWV i PWA a ustalonymi markerami uszkodzenia narządowego (wskaźnikiem masy lewej komory serca — LVMI, grubością kompleksu błona środkowa–błona wewnętrzna tętnicy szyjnej wspólnej — cIMT, polem przekroju tętnicy szyjnej — WCSA) u dzieci z nieleczonym nadciśnieniem tętniczym pierwotnym.

Materiał i metody W badaniu wzięło udział 95 pacjentów (14,9 ± 2,5 roku; 23 dziewczynki), skierowanych z podejrzeniem nadciśnienia tętniczego pierwotnego, u których wykluczono wtórne postaci nadciśnienia tętniczego. Prędkość fali tętna i analiza fali tętna były mierzone przy użyciu funkcji oscylometrycznej aparatu Vicorder. PWV i cIMT były wyrażane w wartościach absolutnych oraz w formie odchylenia standardowego (SDS) — uwzględniając niedawno opublikowane wartości referencyjne.

Wyniki Spośród 95 pacjentów skierowanych z podwyższonym ciśnieniem tętniczym i poddanych pełnemu procesowi diagnostycznemu u 32 stwierdzono prawidłowe wartości ciśnienia tętniczego, u 12 stan przednadciśnieniowy, u 7 ambulatoryjne nadciśnienie tętnicze, u 44 ciężkie ambulatoryjne nadciśnienie tętnicze. Pacjenci z przerostem lewej komory serca (LVH) mieli znacząco większe skurczowe ciśnienie tętnicze w ciągu doby, jak również ciśnienie tętna, cIMT i WCSA w porównaniu z pacjentami bez LVH. Nie obserwowano różnic w PWV, wskaźniku augmentacji (AugIndex), ciśnieniu augmentacji (AugPress) oraz CBP między pacjentami z LVH i/lub z cIMT > 2SDS, a pacjentami z cIMT < 2SDS i prawidłowym LVMI. Porównanie między grupami, określonymi na podstawie wartości ciśnienia tętniczego, wykazało znacząco większe AugPree i AugIndex w grupie z ciężkim ambulatoryjnym nadciśnieniem tętniczym w porównaniu z pozostałymi trzema grupami. Grupy te nie różniły się pod względem PWV. Wartość prędkości fali tętna była skorelowana ze stężeniem kwasu moczowego ($p = 0{,}001$; $r = 0{,}357$) oraz stężeniem cholesterolu LDL ($p = 0{,}01$; $r = 0{,}242$), jednak nie z TOD. AugPress i AugIndex korelowały ze stężeniem trójglicerydów (odpowiednio $p = 0{,}003$; $r = 0{,}307$ i $p = 0{,}002$; $r = 0{,}319$) i LVMI (odpowiednio $p = 0{,}03$; $r = 0{,}206$ i $p = 0{,}03$; $r = 0{,}242$. Na podstawie regresji krokowej wykazano, że jedynymi predyktorami LVMI są wartość wskaźnika augmentacji oraz ciśnienie skurczowe w ciągu doby ($R^2 = 0{,}095$; $B = 0{,}308$; $p = 0{,}006$ i $R^2 = 0{,}142$; AugIndex: $B = 0{,}280$; $p = 0{,}011$; ABPM SBP: $B = 0{,}218$; $p = 0{,}047$).

Address for correspondence: prof. Mieczysław Litwin
The Children's Memorial Health Institute, Department of Nephrology, Kidney Transplantation and Arterial Hypertension
Al. Dzieci Polskich 20, 04–730 Warsaw, Poland
tel.: +48 22 815 15 40; fax: +48 22 815 15 41
e-mail: m.litwin@czd.pl

Wnioski U dzieci z nadciśnieniem tętniczym pierwotnym wykazano zależność między kategorią nadciśnienia tętniczego, centralnym ciśnieniem tętniczym, wskaźnikiem augmentacji oraz ciśnieniem augmentacji a przerostem lewej komory serca. Ustalono, iż prędkość fali tętna nie koreluje z LVH, ma natomiast związek z biochemicznymi czynnikami ryzyka uszkodzenia narządowego. Brak bezpośredniego związku między wartościami PWV a uszkodzeniem narządowym może być spowodowany stosunkowo niewielkim zwiększeniem wartości ciśnienia tętniczego oraz krótkim czas jego trwania u dzieci z nadciśnieniem tętniczym pierwotnym.
słowa kluczowe: sztywność naczyń, prędkość fali tętna, centralne ciśnienie tętnicze, uszkodzenie narządowe
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Background

Arterial stiffness and its hemodynamic consequences are established to be the main predictors of adverse cardiovascular outcomes. During aging and progression of cardiovascular disease, central elastic arteries become stiffer, diastolic pressure decreases, and central systolic blood pressure (sCBP) and pulse pressures are augmented due to increased pulse wave velocity (PWV). Recently, several parameters of arterial stiffness have been employed and have given some valuable information about arterial wall properties. Among these, PWV and central blood pressure (CBP) are the parameters which have gained the most attention, and its predictive accuracy has been demonstrated in a number of studies [1–8]. They are positively associated with arterial hypertension, coronary artery disease, stroke, heart failure and survival in dialysed patients [9–12]. Moreover, an increased PWV was also added to the list of markers of subclinical target organ damage and prognostic factors in the European Guidelines for management of arterial hypertension in adults [13, 14]. However, there are only few reports on PWV and CBP in hypertensive children. Thus, the aim of our study was to evaluate arterial stiffness markers in the early stages of cardiovascular disease in children with newly diagnosed primary hypertension (PH).

Material and methods

The study was performed according to the Declaration of Helsinki and with the approval of the Children's Memorial Health Institute Ethics Committee. All patients (pts) and parents gave consent to participate in the study.

Ninety-five patients (age; 15.3 years, range; 13.3–17.3 years; 27 girls) with newly diagnosed and untreated primary hypertension, who underwent full diagnostic approach to exclude secondary hypertension, were included to the study. The exclusion criteria were the following: the presence of any significant chronic disease (except for PH) including diabetes mellitus and chronic kidney disease, any acute illness including infections in the 6 weeks preceding enrolment, and incomplete data.

PH was diagnosed according to The Fourth Task Force Report and European Society of Hypertension guidelines and confirmed by 24-hour ambulatory BP monitoring (ABPM) [15, 16]. Blood pressure (BP) status was defined according to the ambulatory blood pressure measurement classification [17].

ABPM measurements

All ABPM measurements were assessed oscillometrically using SpaceLabs Monitor 90207, and the most appropriate cuff was applied on the non-dominant arm. Readings were taken every 20 minutes during daytime and every 30 minutes at night. Recordings lasting ≥ 20 hours with ≥ 80% of readings were considered as valid and were included in the analysis. We used a recently published classification system based on ABPM to classify patients as having normal BP, ambulatory hypertension, and severe ambulatory hypertension [17, 18].

Measurement of carotid to femoral PWV

The Vicorder system provides a simple and quick non-invasive oscillometric method of obtaining the Pulse Wave Velocity for an arterial segment. Measurement was performed in the supine position after 5 minutes of rest using the Vicorder device according to the actual guidelines [19]. A 100-mm wide BP cuff was placed around the right upper thigh to measure the femoral pulse wave and a 30-mm plethysmographic partial inflatable sensor was placed over the carotid region, able to pick up the carotid pulse wave. Both cuffs are automatically inflated to 65mmHg and the corresponding oscillometric signal from each cuff is digitally analysed using the latest patented technique to accurately extract, in real time, the pulse time delay and the consequent Pulse Wave Velocity.

Measurement of central blood pressure (pulse wave analysis)

The Vicorder device analyses the waveform of radial artery pulse obtained oscillometrically and then using transfer function calculates the aortic waveform. Prior

to the measurement it was necessary to enter the individual features and the value of blood pressure measured at the brachial artery. System after the measurement calculates and presents approximated waveform of the aortic valve, which we can acquire a number of parameters describing the characteristics of the arterial system including aortic (central) blood pressure (CBP), augmentation pressure (Aug Press), augmentation index (AugIndex), aortic pulse pressure (AoPP), cardiac output (CO) and total peripheral resistance index (TPRI) [19–21].

Echocardiography

All echocardiography examinations were performed by 1 examiner who knew the clinical diagnosis, but was not aware of the severity of hypertension and the effectiveness of treatment. Echocardiography measurements were performed according to American Society of Echocardiography guidelines [22]. To standardize the left ventricular mass to height, left ventricular mass index (LVMI) was calculated according to the de Simone formula [23]. Left ventricular hypertrophy (LVH) was defined as an LVMI value above the 95th percentile for age- and sex-based reference data [24].

Carotid-intima media thickness (cIMT) and wall cross sectional area of carotid arteries (WCSA) measurements

cIMT was evaluated by ultrasound, and SD of normal values for cIMT was obtained according to the methodology described previously [25, 26]. Mean WCSA was calculated from the equation: WCSA = π (dD/2+IMT)2–π(dD/2)2, where dD is the mean diastolic diameter [25].

Laboratory investigations

The following metabolic cardiovascular risk factors were assessed at diagnosis: plasma glucose level, lipid profile and serum uric acid. Blood samples were taken after 12 hours of fasting. The plasma glucose level was measured by a Dimension analyser.

Definition of obesity and metabolic syndrome (MS)

Obesity was diagnosed according to the International Obesity Task Force recommendations [27]. MS was defined according to the International Diabetes Federation Consensus on MS in children [28]. Waist circumference (WC) was referred to Polish reference values [29].

Statistical analysis

The anthropometrical indices, IMT, WCSA, LVMI and PWV values were expressed as absolute values and SDS values according to the referential normative values published recently [26, 30]. The homogeneity of variance was checked with the Shapiro-Wilk test. Continuous variables with a normal distribution were compared using the Student t test for independent variables. Continuous values with abnormal distribution were compared using the Wilcoxon test. Variables with normal distribution were presented as mean and SD values, whereas variables with abnormal distribution were presented as median and range values between the 5th and 95th percentiles. The correlation analysis was performed using Spearman test for abnormal distribution. Variables with significant correlation including changes in anthropometrical parameters and changes in BP and metabolic parameters were then included in the step-wise multiple regression analysis. P values < 0.05 were considered statistically significant, and values between 0.05 and 0.1 were considered as demonstrating trend toward significance.

Results

Of 95 patients (pts) referred with elevated BP who underwent full diagnostic process, normotension (white coat hypertension) was diagnosed in 32, prehypertension in 12 , ambulatory hypertension in 7 and severe ambulatory hypertension in 44 pts. MS was diagnosed in 9 pts (9%) (table I).

The comparison of patients divided into four groups according to BP status revealed that only patients with severe ambulatory hypertension had significantly greater CBP and AugPress compared to other groups. However, all groups did not differ regarding PWV (table II).

PWV correlated with uric acid (p = 0.001; r = 0.357) and LDL-cholesterol (p = 0.01; r = 0.242), but not with markers of target organ damage (fig. 1, fig. 2).

AugPress and AugIndex correlated with triglycerides concentrations (p = 0.003; r = 0.307; p = 0.002; r = 0.319) (fig. 3, fig. 4).

AugPress and AugIndex correlated with body mass index SDS values (BMI-SDS) (p = 0.014; r = 0.274 and p = 0.007; r = 0.303 respectively) and PWV-SDS correlated not only with BMI-SDS (p = 0.001; r = 0.357) but also with WC-SDS (p = 0.004; r = 0.350).

LVH was found in 23 pts (24%) and subclinical arterial injury assessed as cIMT > 2 SDS in 32 pts (33%). WCSA > 2 SDS was noticed in 30 pts (31%).

24h ABPM systolic (24h SBP), diastolic (24h DBP) and mean (24h MAP) blood pressure, Au-

Table I. Characteristic of patient group (n = 95, girls — 23)
Tabela I. Charakterystyka grupy pacjentów (n = 95, 23 dziewczynki)

Age (years)	15.3 ± 2.0
Height [cm]	170.3 ± 11.1
Weight [kg]	73.3 ± 15.5
BMI [kg/m^2]	25.1 ± 3.8
BMI-SDS	1.24 ± 0.80
WC [cm]	81.7 ± 10.3
WC-SDS	1.20 ± 0.87
Classification of hypertension based on ABPM	Normal blood pressure — 32
	Prehypertension — 12
	Ambulatory blood pressure — 7
	Severe ambulatory blood pressure — 44 pts
ABPM 24h SBP [mm Hg]	129 ± 9
ABPM 24h DBP [mm Hg]	73 ± 7
ABPM 24h MAP [mm Hg]	92 ± 6
ABPM heart rate	77 ± 11
cIMT [mm]	0.47 ± 0.04
cIMT-SDS	1.66 ± 0.92
cIMT > 2 SDS	32 pts (33%)
LVMI [g/m$^{2.7}$]	35 ± 8
LVH	23 pts (24%)
PWV [m/s]	5.6 ± 0.6
PWV-SDS	1.43 ± 1.40
AugPress [mm Hg]	4 ± 3
AugIndex [mm Hg]	8 ± 5
CO [l/min]	6.01 ± 1.72
CI [l/min/m^2]	3.29 ± 1.05
Cholesterol [mg/dl]	160 ± 32
Triglycerides [mg/dl]	101 ± 42
HDL-cholesterol [mg/dl]	50 ± 10
LDL-cholesterol [mg/dl]	90 ± 27
Uric acid [mg/dl]	5.7 ± 1.4
Fasting glucose [mg/dl]	89 ± 6
Metabolic syndrome	9 pts (9%)

BMI — body mass index; WC — waist circumference; ABPM — ambulatory blood pressure monitoring; 24h — 24 hours; SBP — systolic blood pressure; DBP — diastolic blood pressure; MAP — mean arterial pressure; HR — heart ratio; cIMT — carotid intima-media thickness; LVMI — left ventricular mass index; LVH — left ventricular hypertrophy; PWV — pulse wave velocity; AugPress — augmentation pressure; AugIndex — augmentation index; CO — cardiac output; CI — cardiac index; LDL — low density lipoprotein; HDL — high density lipoprotein

gIndex, AugPress and CBP correlated with LVMI (table III).

Patients with LVH had significantly greater 24h SBP, 24h DBP, 24h MAP and tendency to the greater AugPress than pts without LVH (table IV). There were no differences in PWV, and CBP between patients with LVH or without LVH.

There were no differences in PWV and arterial stiffness markers between patients with arterial injury and those with normal cIMT and WCSA.

The stepwise regression analysis revealed that only AugIndex and 24h SBP predicted LVMI (R2 = 0.095; B = 0.308; p = 0.006 and R2 = 0,142; AugIndex : B = 0.280; p = 0.011; ABPM 24h SBP: B = 0.218; p = 0.047 respectively).

Discussion

Although the majority of studies related to arterial stiffness focused on PWV, which has been considered the most accurate non-invasive assessment of arterial stiffness and predictor of cardiovascular outcomes, in our study we did not find any differences in PWV between patients with different BP status. In comparison, in adults PWV gradually increases as a function of BP classification. Furthermore, some reports have shown that subjects having elevated BP in childhood are at higher risk of increased arterial stiffness in adulthood [31]. The lack of changes in PWV in our children with different BP status may be partially explained by the young age and early phase of hypertensive disease. Although an increasing number of data have focused on PWV in adults, only a few studies have specifically concerning the relationship between PWV and BP level in children [32, 33]. Moreover, recently published data evaluated PWV in healthy children and adolescents, indicated on gender, racial and age related differences in PWV, but findings regarding BP were inconclusive [32, 34–38]. Thus, some authors suggest, that although PWV might be a more sensitive marker of arterial alteration and cardiovascular risk in older individuals, CBP and AugIndex may be better predictors in younger individuals [39, 40].

Moreover, the presence of increased PWV in young population, may indicate on the premature arterial stiffness as a consequence of an early vascular ageing [41].

Because CBP tends to be more closely associated with intermediate markers of cardiovascular events than brachial BP, classification of cardiovascular risk on the basis of CBP would appear to be important. In our study we divided patients according to BP strata and we found that only patients with severe ambulatory hypertension had significantly greater CBP and AugPress compared to other groups.

CBP is typically affected by both a forward pulse wave, generated during left ventricular contraction and a reflected wave raised from arterial crotch. In healthy people the reflected wave reaches ascending

Table II. Anthropometrical and hemodynamic data compared with the category of hypertension
Tabela II. Dane antropometryczne i hemodynamiczne w grupach pacjentów określonych na podstawie wartości ciśnienia tętniczego

Variable	Normal blood pressure/White coat hypertension N = 32 pts	Prehypertension N = 12 pts	Ambulatory blood pressure N = 7 pts	Severe ambulatory blood pressure N = 44 pts	p
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	P
PWV [m/s]	5.7 ± 0.71	5.5 ± 0.6	5.6 ± 0.4	5.6 ± 0.65	n.s.
PWV-SDS	1.4 ± 1.4	1.36 ± 1.13	1.32 ± 0.7	1.45 ± 1.5	n.s.
CBP [mm Hg]	112 ± 7.1*	112 ± 10.2	114 ± 9.3	118 ± 10.1*	0.04
AoPP [mm Hg]	46 ± 6	46 ± 9	49 ± 9	51 ± 9	n.s.
AugPress [mm Hg]	3.4 ± 1.7*	3.0 ± 2.1**	3.8 ± 2.6	5.0 ± 3.2*. **	*0.04 **0.08
AugIndex (%)	7.2 ± 3.1	6.5 ± 4.3	7.5 ± 4.4	9.5 ± 5.3	n.s.

PWV — pulse wave velocity; CBP — central blood pressure; AoPP — aortic pulse pressure; AugPress — augmentation pressure; AugIndex — augmentation index

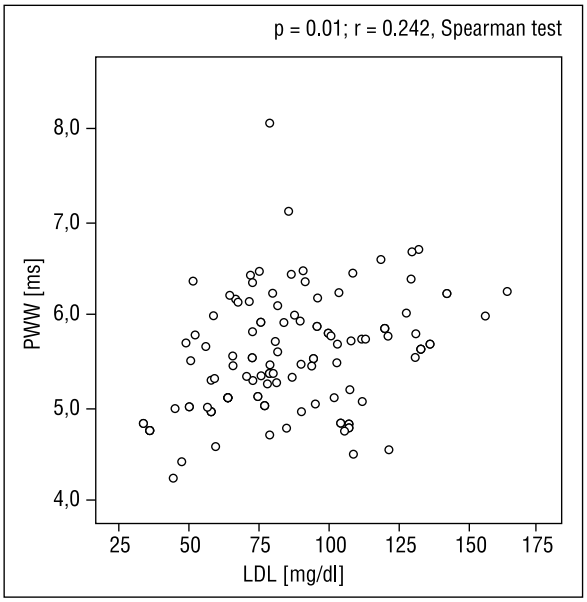

Figure 1. The correlation between pulse wave velocity (PWV) and LDL-cholesterol concentrations

Rycina 1. Korelacja między prędkością fali tętna (PWV) a stężeniem cholesterolu LDL we krwi

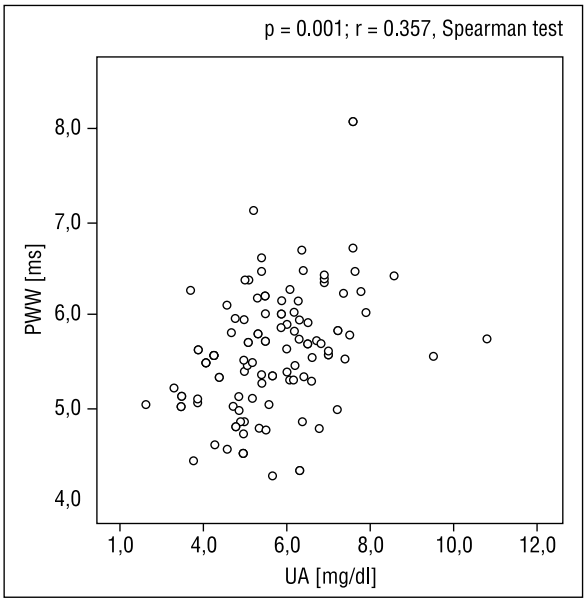

Figure 2. The correlation between pulse wave velocity (PWV) and uric acid (UA) concentrations

Rycina 2. Korelacja między prędkością fali tętna (PWV) a stężeniem kwasu moczowego we krwi

aorta during diastole and therefore does not significantly augment central systolic BP. However, when arteries become stiffened the reflected wave returns earlier. This causes an augmentation of the forward wave and an increase in the systolic pressure [35, 39]. In this manner an increased AugPress and CBP in patients who had higher values of BP may indicate on the beginning of vascular stiffness.

In many studies the early markers of arterial stiffness, such as AugIndex, AugPress and elevated CBP have been shown as the main predictors of adverse cardiovascular events and target organ damage [42–50]. Also in hypertensive adolescents and young adults increased arterial stiffness is associated with LVH independently of traditional cardiovascular risk factors [51].

We found that AugIndex, AugPress and CBP, but also ABPM values of BP correlated with LVH, but not with arterial injury markers. Patients with LVH had significantly higher 24h systolic, diastolic and

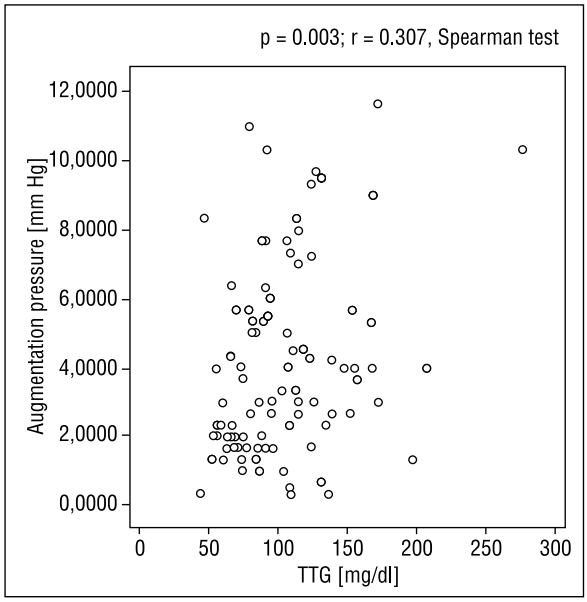

Figure 3. The correlation between augmentation pressure and triglycerides (TTG) concentrations

Rycina 3. Korelacja między ciśnieniem augmentacji (AugPress) a stężeniem trójglicerydów we krwi

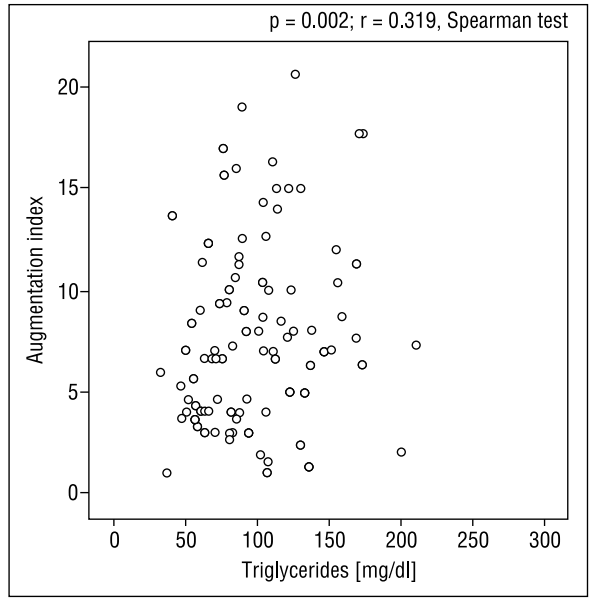

Figure 4. The correlation between augmentation index (AugIndex) and triglycerides (TTG) concentrations

Rycina 4. Korelacja między wskaźnikiem augmentacji (AugIndex) a stężeniem trójglicerydów we krwi

Table III. Correlations between LVMI and independent variables

Tabela III. Korelacje między wskaźnikiem masy lewej komory serca (LVMI) a innymi parametrami hemodynamicznymi (ujęto wyłącznie korelacje istotne statystycznie)

	LVMI
AugPress	P = 0.02; r = 0.259
AugIndex	P = 0.03; r = 0.242
CBP	P = 0,03; r = 0.233
24h SBP	P = 0.01; r = 0.262
24h DBP	P = 0.02; r = 0.291
24h MAP	P = 0.02; r = 0.295

AugPress — augmentation pressure; AugIndex — augmentation index; CBP — central blood pressure; 24h — 24 hours; SBP — systolic blood pressure; DBP — diastolic blood pressure; MAP — mean arterial pressure

Table IV. The comparison of patients with and without LVH

Tabela IV. Porównanie grup pacjentów z przerostem lewej komory serca (LVH) i bez przerostu

	LVMI > 95cc N = 23 pts	LVMI < 95cc N = 72 pts	
	Mean ± SD	Mean ± SD	p
ABPM 24h SBP [mm Hg]	135 ± 10	128 ± 9	0.01
ABPM 24h DBP [mm Hg]	77 ± 7	73 ± 7	0.026
ABPM 24h MAP [m m Hg]	95 ± 6	91 ± 6	0.018
ABPM 24h PP [mm Hg]	60 ± 14	55 ± 7	0.1
PWV [m/s]	5.58 ± 0.59	5.61 ± 0.62	n.s.
PWV-SDS	1.47 ± 1.43	1.35 ± 1.25	n.s.
CBP [mm Hg]	117 ± 11	114 ± 8	n.s.
AoPP [mm Hg]	51 ± 10	48 ± 8	n.s.
AugPress [mm Hg]	4 ± 3	3 ± 2	0.07
AugIndex (%)	9.33 ± 5.02	7.53 ± 4.39	n.s.

ABPM — ambulatory blood pressure monitoring; 24h — 24 hours; SBP — systolic blood pressure; DBP — diastolic blood pressure; MAP — mean arterial pressure; PP — pulse pressure; PWV — pulse wave velocity; CBP — central blood pressure; AoPP — aortic pulse pressure; AugPress — augmentation pressure; AugIndex — augmentation index; LVMI — left ventricular mass index

mean BP and tendency to higher AugPress than patients without LVH. However, there were no differences in PWV and CBP between patients with LVH or without LVH. It confirms that some abnormalities in arterial wall properties can be identified at the early stages of hypertension and influence TOD, but advanced arterial stiffness expressed as increased PWV develop on more advanced stages of hypertensive disease.

Recently, it has been shown that adults and children with obesity have increased aortic stiffness, independent of BP level, ethnicity, and age. An increased aortic stiffness has been shown to be more related to body fat deposition and visceral adiposity than to increased BMI [52–54]. On the other hand, some data indicated that total trunk fat has been found to be associated adversely with PWV [33, 55]. In our study AugPress and AugIndex correlated with BMI-SDS, and PWV-SDS correla-

ted positively with BMI-SDS and with WC-SDS. Several factors have been proposed to explain the increased stiffness observed in individuals with visceral obesity. Impaired mechanisms of endothelium-dependent flow dilation and nitrite oxide disorders are suggested as well as sympathetic system activation. Moreover, the reduced arterial elasticity may be the consequence of metabolic abnormalities with hyperglycaemia and insulin disorders leading to the development of advanced glycation end products. Non-enzymatic glycosylation of the matrix proteins of arterial vessels may amplified the production of collagen fibres, which in turn are responsible for increased arterial stiffness and hypertension [56, 57]. There is some evidence that also children with worse cardiometabolic profile and prediabetes are characterized by increased arterial stiffness [58, 59]. Metabolic syndrome in childhood predicted the increased arterial stiffness in adulthood, and recovery from childhood metabolic abnormalities was associated with decreased arterial PWV in adulthood [60]. In our study we did not find any associations between arterial stiffness markers and insulin sensitivity/resistance markers, but we found positive correlations with other metabolic risk factors. There was a positive correlation of PWV with uric acid and LDL-cholesterol and between AugPress and AugIndex with triglycerides concentrations. It may suggest the important role of metabolic abnormalities at the early stages of cardiovascular disease.

Limitations and strengths of the study

The limitations of the study include a relatively narrow age range of participants, which made it difficult to determine whether parameters of arterial stiffness changed with age. Also, due to cross-sectional study it was unfeasible to observe any changes in arterial stiffness over time.

Strength of our study was the large number of young participants at the early stage of disease, detailed biochemical and hemodynamic investigations and assessment of intermediate phenotype. Moreover, our study was able to compare commonly used peripheral BP measurements to non-invasive CBP pressure and arterial stiffness markers as predictors of TOD.

Conclusions

CBP and AugPress assessed non-invasively with oscillometric method correlated with markers of hypertensive TOD. Because the role of CBP and 24h ABPM BP as predictors of TOD was similar, non-invasive assessment of arterial stiffness offers a useful tool in the clinical evaluation of blood pressure.

Summary

Background Arterial stiffness measured as pulse wave velocity is regarded as one of the markers of target organ damage in hypertensive adults. Pulse wave analysis adds the information about central blood pressure, backward pulse wave, cardiac output and total peripheral resistance. However, there are no data regarding the usefulness of measurements of pulse wave velocity, central blood pressure, augmentation pressure and augmentation index in assessment of risk of target organ damage in hypertensive children. The aim of the study was to estimate the relationship of pulse wave velocity and pulse wave analysis with established markers of target organ damage (left ventricular mass index, carotid intima-media thickness, carotid wall cross sectional area) in children with untreated primary hypertension.

Material and methods Ninety-five patients (14.9 ± 2.5 years; 23 girls) referred as newly diagnosed primary hypertension were included in the study. Pulse wave velocity and pulse wave analysis were measured by oscillometric method using Vicorder device. Pulse wave velocity and carotid intima-media thickness were expressed as absolute and SDS values regarding recently published normative values.

Results In 95 patients referred with elevated blood pressure and who underwent full diagnostic process, normotension was diagnosed in 32, prehypertension in 12, ambulatory hypertension in 7 and severe ambulatory hypertension in 44 patients. Patients with left ventricular hypertrophy had significantly greater 24h systolic, diastolic and mean blood pressure and tendency to the greater augmentation pressure than patients without left ventricular hypertrophy. There were no differences in pulse wave velocity and central blood pressure between patients with left ventricular hypertrophy and/or carotid intima-media thickness > 2SDS and patients without target organ damage. The comparison of patients divided into four groups according to blood pressure status revealed that only patients with severe ambulatory hypertension had significantly greater augmentation pressure and central blood pressure compared with other groups. However, groups did not differ regarding pulse wave velocity. Pulse wave velocity correlated with

uric acid (p = 0.001; r = 0.357) and LDL-cholesterol (p = 0.01; r = 0.242), but not with target organ damage. Augmentation pressure and augmentation index correlated with triglycerides (p = 0.003; r = 0.307; p = 0.002; r = 0.319). The stepwise regression analysis revealed that only augmentation index and 24h systolic blood pressure predicted left ventricular mass index (R^2 = 0.095; B = 0.308; p = 0.006 and R^2 = 0,142; Augmentation Index: B = 0.280; p = 0.011; ABPM SBP: B = 0.218; p = 0.047 respectively).

Conclusions In children with primary hypertension, blood pressure status, central blood pressure, augmentation pressure and augmentation index, but not pulse wave velocity, correlate with left ventricular hypertrophy. However, pulse wave velocity correlates with biochemical risk factors of target organ damage. The lack of direct association between pulse wave velocity and target organ damage may be due relatively modest elevation of blood pressure and short duration of hypertensive disease in children with primary hypertension.

key words: primary hypertension, metabolic syndrome, arterial stiffness, pulse wave velocity, central blood pressure, target organ damage

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