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Age-related changes in myocardial deformation and arterial stiffness in hypertensive males

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Key words: *Hypertension, Vascular Stiffness, Left Ventricular Hypertrophy, Transthoracic Echocardiography.*

In terms of the general aging of population the role of senescence in the vascular and cardiac remodeling development remains controversial. The ways the ventricular and arterial stiffening affects the myocardial functioning are still unclear.

Objective. The aim of this study was to evaluate the impact of aging on myocardial deformation and arterial stiffness in hypertensive men using aortic pulse wave velocity (aoPWV) and speckle tracking echocardiography.

Materials and Methods. 32 men with arterial hypertension stage II and 12 healthy males from 45 to 72 years were included into this study. Aortic stiffness was evaluated with the use of BPLab Vasotens System. Transthoracic echocardiography was performed using My Lab 50 equipment (Esaote, Italy). The patients with hypertension and healthy individuals from the control group were divided into 2 groups according their age.

Results. ASI significantly correlated with age (r=0.26, p<0.05), PP (r=0.22, p< 0.05), aoPWV (r=0.16, p<0.05), AIx (r=0.16, p=0.002). PPA appeared related to BP (r=0.27, p<0.05 for SBP with PPA). Global longitudinal strain (GLS) becomes significantly reduced in hypertensive patients compared to the control group. It was markedly diminished in both groups over 55 years. GLS correlated with aoPWV (r=0.26, p<0.05). Circumferential and radial strain at the basal and the apical LV segments did not show significant difference in groups divided by age.

Conclusions. The present study demonstrates the additional impact of aging on the development of both cardiac and vascular remodeling, leading to myocardial longitudinal strain disorders and an enhanced arterial stiffness. At the basis of revealed correlations aoPWV and global longitudinal strain may be considered also as the marker of vascular aging in hypertensive men and in general population.

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Вікові особливості деформації міокарда та жорсткості артерій у чоловіків із гіпертонічною хворобою

В. А. Візір, А. С. Садомов, О. В. Насоненко

З огляду на прогресування старіння популяції в цілому вивчення ролі вікових змін у розвитку судинного та кардіального ремоделювання залишається актуальним.

Мета роботи – оцінити вплив старіння на деформацію міокарда та артеріальну жорсткість у чоловіків із гіпертонічною хворобою з використанням визначення швидкості пульсової хвилі в аорті (аоШПХ) і параметрів, що одержані за допомогою спекл-трекінг ехокардіографії.

Матеріали та методи. 32 пацієнти чоловічої статі з гіпертонічною хворобою II стадії та 12 практично здорових чоловіків віком від 45 до 72 років включили в дослідження. Жорсткість артерій оцінювали з використанням системи BPLab Vasotens. Трансторакальну ехокардіографію здійснили з використанням обладнання My Lab 50 (Esaote, Італія). Хворих із ГХ і досліджуваних із контрольної групи поділили на 2 групи за віком.

Результати. ASI вірогідно корелював із віком (r=0,26, p<0,05), пульсовим АТ (r=0,22, p<0,05), аоШПХ (r=0,16 , p<0,05), AIx (r=0,16, p=0,002). Виявлено взаємозв'язок РРА з АТ (r=0,27, p<0,05 для САТ із РРА). Глобальна поздовжня деформація (GLS) у хворих на артеріальну гіпертензію була значно нижчою порівняно з контрольною групою. Зниження GLS відзначали в обох групах старше за 55 років. GLS корелювала з аоШПХ (r=0,26, p<0,05). Циркулярна та радіальна деформації в базальних, апікальних сегментах ЛШ не показали істотних відмінностей у групах, котрі поділені за віком.

Висновки. Дослідження демонструє додатковий вплив старіння на розвиток як кардіального, так і васкулярного ремоделювання, що призводить до порушення поздовжньої деформації міокарда та підвищеної жорсткості артерій. На підставі виявлених кореляцій аоШПХ і глобальна поздовжня деформація можуть розглядатися також як маркер судинного старіння в чоловіків із ГХ і в популяції в цілому.

Ключові слова: *гіпертонічна хвороба, жорсткість артерій.*

Запорізький медичний журнал. – 2016. – №6 (99). – С. 21–24

Возрастные особенности деформации миокарда и жёсткости артерий у мужчин с гипертонической болезнью

В. А. Визир, А. С. Садомов, А. В. Насоненко

Учитывая прогрессирующее старение популяции в целом, изучение роли возрастных изменений в развитии сосудистого и кардиального ремоделирования остаётся актуальным.

Цель работы – оценить влияние старения на деформацию миокарда и артериальную жёсткость у мужчин с гипертонической болезнью с использованием определения скорости пульсовой волны в аорте (аоСПВ) и параметров, полученных при помощи спекл-трекинг эхокардиографии.

Материалы и методы. 32 пациента с гипертонической болезнью (ГБ) II стадии и 12 практически здоровых лиц в возрасте от 45 до 72 лет были включены в исследование. Жёсткость артерий оценивали с использованием системы BPLab Vasotens (осциллометрическим методом). Трансторакальная эхокардиография проводилась с использованием оборудования My Lab 50 (Esaote, Италия). Больные с ГБ и испытуемые из контрольной группы были разделены на 2 группы по возрасту.

Результаты. ASI достоверно коррелирует с возрастом (r=0,26, p<0,05), пульсовым АД (r=0,22, p<0,05), скоростью распространения пульсовой волны в аорте (аоСПВ) (r=0,16, p<0,05), AIx (r=0,16, p=0,002). Выявлена взаимосвязь РРА с АД (r=0,27, p<0,05 для САД с РРА). Глобальная продольная деформация (GLS) у больных артериальной гипертензией была значительно ниже по сравнению с контрольной группой. Это снижение отмечалось в обеих группах старше 55 лет. GLS коррелировала с аоСПВ (r=0,26, p<0,05). Циркулярная и радиальная деформации в базальных и апикальных сегментах ЛЖ не показали существенных различий в группах, распределённых по возрасту.

Выводы. Настоящее исследование демонстрирует дополнительное влияние старения на развитие как кардиального, так и васкулярного ремоделирования, приводящего к нарушению продольной деформации миокарда и повышенной жёсткости артерий. На основании выявленных корреляций скорость распространения пульсовой волны и глобальная продольная деформация могут рассматриваться также в качестве маркера сосудистого старения у мужчин с ГБ и у популяции в целом.

Ключевые слова: *гипертоническая болезнь, жёсткость артерий.*

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In view of continuing aging of the population as a whole and an increasing rate of cardiovascular morbidity the issue of aging influences on the structural and functional properties of arterial wall and myocardial deformation remains relevant. Recent studies pay substantial attention to the measurement of global myocardial strain as a sensitive and strong marker of subclinical myocardial dysfunction [4].

Myocardial strain is a principle for quantification of left ventricular (LV) function which is now available to assess with speckle-tracking echocardiography (STE) [3]. The assessment of functional parameters of LV by STE has represented a significant advance as a method of non-invasive evaluation of LV morphology and function. Left ventricular hypertrophy (LVH) is a common consequence of hypertension. It is considered as an independent risk factor for cardiovascular morbidity and mortality. Hypertension induces both cardiac and vascular remodeling. Progressive cardiac remodeling supposed to integrate an increased size of the cardiomyocytes and development of fibrosis in the extracellular matrix [2]. LV deformation assessed by longitudinal, radial and circumferential strain is expected to be linked with the myofiber architecture.

Vascular remodeling is more accelerated to the natural aging processes compared to the heart. It is influenced by systemic hypertension as well. Coupled ventricular-arterial stiffening can be shown as the contributor of systolic and diastolic reserve limitation, blood pressure variability, coronary and peripheral blood flow regulation [1].

The pathophysiological ways of chronic ventricular-arterial stiffness affect on the myocardial functioning are still unclear.

Objective

The aim of this study was to evaluate the impact of aging on myocardial deformation and arterial stiffness in hypertensive men using aortic pulse wave velocity (aoPWV) and speckle tracking echocardiography.

Materials and Methods

A total of 32 nonobese (BMI=24.2 [23.0; 26.8] kg/m^2) men with arterial hypertension stage II aged from 45 to 72 years were included into this study. Written informed consent was obtained from all participants. Patients with prior history of major cardiovascular (CV) events, chronic kidney disease, diabetes mellitus, significant valvular abnormalities, atrial fibrillation or flutter, congenital heart disease or inadequate echocardiographic acoustic windows were excluded from this study. Patients with any evidence of secondary hypertension were also not included. Demographic data, including an assessment of risk factor status and history of CV disease were recorded. Twelve individuals of male sex without prior history of any CV diseases were recruited into the control group. Exclusion criteria for these participants were the same as for the patients from the hypertension sample. The main features of the study population are demonstrated at the *Table 1*.

Aortic stiffness was evaluated with the use of BPLab Vasotens System (cuff-based oscillometry method). Pulse wave analysis was performed with the non-invasive measurement of aortic pulse wave velocity (aoPWV), augmentation index (AIx) adjusted to the 75 beats per minute of heart rate, arterial stiffness index (ASI), pulse pressure amplification (PPA) and subendocardial viability ratio (SEVR).

Table 1

Main characteristics of the study population

Parameters	Hypertensive male patients (n=27)	Control group (n=12)
Age, years	56 [49.0; 61.5]	54 [47.2; 60.6]
Hypertension experience, years	8 [4.4; 13.2]	-
Smoking habits (%)	36	32
24 h SBP, mm Hg	132.7 [122.4; 151.6] *	121.4 [118.6; 136.6]
24 h DBP, mm Hg	84.5 [80.0; 92.7]	80.5 [76.2; 84.5]
24 h PP, mm Hg	61.4 [58.6; 67.2]*	41.7 [40.4; 47.1]
Aortic pulse wave velocity (aoPWV), m/s	10.4 [9.8; 11.2]*	9.4 [8.7; 10.6]
Arterial stiffness index (ASI), mm Hg	181.54 [162.7; 195.8]*	173.15 [158.7; 186.6]
Pulse pressure amplification (PPA), %	129.82 [121.3; 156.4]	141.7 [121.5; 166.2]
Subendocardial viability ratio (SEVR), %	77.6 [70.2; 86.5]*	92.2 [76.3; 98.1]
Interventricular septum thickness (IVST), mm	119 [117.0; 119.0]*	110.5 [110.0; 117.0]
LV mass index (LVMI), g/m^2	146.4 [138.6; 147.6]*	122.8 [121.8; 124.5]
LV ejection fraction (LVEF), %	59.2 [52.8; 63.2]	63.2 [55.1; 63.0]
Global longitudinal strain (GLS), %	-18.29 [-20.0; -17.14]*	-21.01 [-21.7; -18.2]
Circumferential strain (CS), %	-22.0 [-27.2; -21.0]	-25.2 [-28.1; -21.8]
Radial strain (RS), %	24.1 [22.6; 24.8]	24.6 [22.8; 25.6]

Note: * – $p<0.05$ compared to the control group.

Transthoracic echocardiography was performed using My Lab 50 echocardiographic equipment (Esaote, Italy). All participants were examined using standard methods. Interventricular septum thickness (IVST), posterior wall thickness (PWT), LV mass index (LVMI) and LV ejection fraction (LVEF) were measured. LVH was detected by LV mass above the reference values for LVM 88–224 g in male individuals. Measurements of segmental evaluation of longitudinal, circumferencial and radial strain were obtained from basal, apical parasternal short axis and apical four-chamber, three-chamber and two-chamber views.

The patients with hypertension and healthy individuals from the control group were divided into 2 groups by mediana of age (less than 55 years; more than 55 years). The main and control groups were comparable by age.

Statistical analysis was performed using Statistica 6.0 software. Variables were tested for normality by the Kolmogorov-Smirnov and Lilliefors tests. Differences between groups were analyzed using chi-square test for categorical data, Student's test for continuous normally distributed data and Wilcoxon test for continuous non-normally distributed data. Correlations were assessed for normal and non-normal variables using Pearson's and Spearman's coefficients respectively. Univariate and multivariate analyses were used to evaluate the relationship between age and such variables as arterial stiffness, general conventional echocardiographic and speckle tracking parameters. *p*-value of ≤0.05 to 95 % confidence interval was taken as the criterion for statistical significance. The results are expressed as Me [Q1; Q2].

Results

Table 2 shows the data obtained by using two-dimensional echocardiography and arterial stiffness evaluation of all groups.

Regarding risk factors of the study population, there were no significant differences between the studied groups. The main groups and control sample were comparable by age.

The differences in SBP, DBP and PP with age became the levels in appliance with population data. After the age of 55 SBP increases, whereas DBP remains relatively stable.

ASI was significantly correlated with age (r=0.26, p< 0.05), PP (r=0.22, p<0.05), aoPWV (r=0.16, p<0.05), cAIx (r=0.16, p=0.002), but correlation with LVMI becomes non-significant (r=0.03, p=0.49).

PPA appeared in relation to BP: than higher the BP, than lower the PPA is (r=0.27, p<0.05 for SBP and PPA). PPA was widely variable within and between study subjects. It supposed to be influenced by a range of factors, such as heart rate and natural aging processes occurring in the arterial wall.

GLS becomes significantly reduced in hypertensive patients compared to the control group. It was markedly diminished in both groups over 55 years. GLS correlated with aoPWV (r=0.26, p<0.05). Circumferential and radial strain at the basal and the apical LV segments did not show significant difference in groups divided by age.

The multiple regression analysis with the use of age as an independent variable and aoPWV and LGS, CS, RS as dependent variables has shown the relationships between experienced age and enhanced arterial stiffness as well as parameters of myocardial deformation in both study groups divided by age. The relationships between age and aoPWV and LGS were representative whereas the link between age, aoPWV, CS and RS was not significant.

Discussion

The study showed that PWV correlated with markers of regional myocardial function, including global longitudinal strain.

Table 2

Parameters of study groups obtained by using two-dimensional echocardiography and arterial stiffness evaluation

Parameter	Hypertensive male patients under 55 years (n=12)	Hypertensive male patients over 55 years (n=15)	Participants from control group under 55 years (n=6)	Participants from control group over 55 years (n=6)
aoPWV, m/s	10.0 [9.2; 11.0]*	10.7 [9.9; 11.8]	9.1 [8.4; 10.2]**	9.6 [8.8; 10.4]
ASI, mm Hg	178.6 [160.7; 188.9]*	184.4 [166.8; 198.1]	170.1 [158.7; 186.7]	176.2 [159.9; 189.3]
PPA, %	138.2 [122.2; 162.0]	121.4 [119.2; 146.1]	148.4 [129.4; 169.1]**	135.0 [129.6; 164.4]
SEVR, %	80.4 * [71.4; 89.4]	74.7 [70.0.2; 85.1]	95.7 ** [79.4; 97.3]	88.7 [78.3; 90.8]
IVST, mm	118 [116.0; 121.0]*	120 [117.0; 121.0]	110 [110.0; 117.0]	111 [110.0; 116.0]
LVMI, g/m^2	144.4 [136.2; 148.4]*	148.4 [140.6; 149.5]	121.1 [121.8; 124.5]	124.6 [122.6; 126.5]
LVEF, %	59.4 [53.0; 64.4]	58.9 [52.1; 63.2]	63.4 [55.2; 63.6]	63.1 [54.0; 63.7]
GLS, %	-18.72 * [-21.1; -17.25]*	-17.24 [-20.0; -17.14]	-21.92 ** [-22.9; -17.9]	-20.11 [-21.7; -18.2]
CS, %	-22.2 [-27.1; -21.1]	-21.8 [-27.0; -20.7]	-25.2 [-28.0; -22.2]	-25.1 [-28.1; -21.8]
RS, %	24.0 [22.2; 24.6]	24.2 [22.6; 25.0]	24.6 [22.4; 25.2]	24.5 [22.9; 25.8]

Note: * – p<0.05 compared to the more aged sample of the hypertension group; ** – p<0.05 compared to the more aged sample of the control group.

Our findings are in agreement with these previous findings. The differences of LVEF between study groups were non-significant. On the other hand, GLS demonstrated gradual decline among all hypertensive patients (up to –17 %). This progressive decreasing was more expressed with age.

Limitations of the study. Measures of aortic stiffness parameters were non-invasively and automatically calculated from peripheral pressure waveforms. The sample population size is not enough to generalize the findings.

Conclusions

The present study demonstrates the additional impact of aging on both the cardiac and vascular remodeling development, leading to myocardial longitudinal strain disorders and an enhanced arterial stiffness. At the basis of revealed correlations aoPWV and global longitudinal strain may be considered also as the marker of vascular aging in hypertensive men and in general population.

Conflicts of interest: authors have no conflict of interest to declare.

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