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HEART AND VESSEL REMODELING IN DIFFERENT AGE GROUPS OF PATIENTS WITH ARTERIAL HYPERTENSION

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SUMMARY

Actuality. It is known that each of the factors (arterial hypertension (AH) and age) has a negative effect on the remodeling of both the heart and vessels, while their combined effect has not been sufficiently studied.

The aim. Assessment of manifestations of cardiac and vascular remodeling (by the example of common carotid arteries (CCA) and vasomotor endothelium function (right brachial artery)) in patients with AH degree I-III in different age groups.

Material and methods. The study involved 124 (86 men and 38 women) patients at the age from 27 to 81 years (average age of patients 55.7 ± 1.01 years) with AH degree I-III. The presence and variant of myocardial remodeling with calculation of criteria of left ventricular hypertrophy (LVH), myocardial mass (MM) and MM index (MMI) of LV by indexation to the patient's body surface area were studied in all patients by means of echocardiographic investigation (EchoCG) using the Vivid S5 3Sc-RS echocardiograph with a multifrequency sensor 2.0-3.5 MHz. Normal values of MM were considered to be 67-162 grams (g) for women and 88-224 for men, and MMI – not more than 95 and 115 g/m², respectively. Types of LVH were determined according to recommendations of Pugliese N.R. et al. (2017), being subdivided into concentric (C) and eccentric (E) LVH. Ultrasound scanning was used to investigate CCA on both sides. The results of reactive hyperemia test

on the right brachial artery and blood lipid profile were studied with the calculation of atherogenicity coefficient (AC). The data analysis was carried out with the help of Statistica 12 application package with determination of mean values (M), minimum, maximum and mean error (m), median and interquartile range for the CCA intima-media thickness (IMT). The Spearman rank correlation coefficient (r) was used to estimate the relationship between these variables. The significance level of all statistical tests was accepted at $p < 0.05$.

The results of the study confirmed the presence of cardiac and vascular remodeling in patients with AH of different degrees with the tendency to increase in older age groups. The CCA remodeling parameter values were also significantly higher in the groups of middle-aged and elderly persons, some of which also had atherosclerotic plaques (AP). The number of patients with disturbed endothelial vasomotor function also somewhat increased with age.

Conclusion. Thus, the age of the examined patients with AH had a significant influence on aggravation of the heart and vessel remodeling processes and increasing frequency of endothelial dysfunction development.

Keywords: arterial hypertension, age, intima-media thickness, interadventitial diameter of carotid arteries, left ventricular hypertrophy.

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INTRODUCTION

In view of modern conceptions, heart and vessel remodeling nearly always to some or other extent accompanies the course of AH being not only its complication but also the factor for further progression [4; 16; 22]. The main manifestations of heart remodeling include increased LV MM, LVH of different degree and types (predominant localization of the hypertrophied myocardial region) [17]. The vessel remodeling is understood as vascular function and morphology modification under the influence of hemodynamic (increased

BP level and circulating blood volume) and non-hemodynamic factors (activation of the renin-angiotensin-aldosterone and sympathoadrenal systems, endothelial dysfunction with disturbed nitrogen oxide metabolism, dyslipidemia and others), the intensity of which is caused also to a different extent by patient's individual genetic features [20]. The manifestations of vessel remodeling include increased IMT and interadventitial diameter (IAD) of magistral arteries, worsened functional condition of vascular endothelium, general microcirculation condition and others [26].

At the same time, the characteristic features of heart and vessel remodeling in patients with AH taking into account their age have been still studied insufficiently.

In the context of the above, the aim of the present study was to assess structural and functional changes in the heart, CCA and vasomotor endothelium function in patients with AH degree I-III of different age groups.

MATERIALS AND METHODS

We examined 124 patients with AH degree I-III aged from 31 to 62 years (average age: 55.7±1.01 years), 56 men (average age: 54.7±1.2 years) и 65 women (average age: 57.8±1.7 years). AH and its degree were diagnosed according to WHO criteria [27].

The criteria for inclusion in the study were patients with AH degree I-III. The AH duration was on average 8.36±0.37 years in the range from 1 to 21 years; it was 8.42±0.42 years in the range from 2 to 21 years in male subgroup and 8.21±0.74 years in the range from 1 to 18 years in the female subgroup.

The presence of diabetes mellitus type 2 and metabolic syndrome, symptomatic AH forms, acute myocardial infarction, cardiac rhythm and conduction disorders, blood diseases, cancer diseases, history of cerebral circulation disorders, heart defects, respiratory, renal, hepatic and heart failure was ruled out in all subjects.

The total number of examined patients was subdivided during the study in the respective age subgroups according to appropriate WHO age criteria [28]), AH degree, absence and presence of sings and type of heart and vessel remodeling (as per the suggested classification [21]). The distribution of examined patients included in the above compared subgroups is presented in following Table 1.

Some patients took hypolipidemic metabolic drugs: 28 patients took Atorvastatin (Atoris) in the daily dose of 40 mg (out of these, 7 (5.65%) patients used the drug on regular basis, 21 (16.94%) patients used it occasionally) and 19 (15.32%) patients received Thiotriazoline in the daily dose of 0.1 g (out of these, 6 (4.84%) patients used the drug on regular basis and 13 (10.48%) patients took it irregularly).

All examined patients underwent EchoCG using the Vivid S5 3ScRS echocardiograph (USA) with a sensor 2.0-3.5 MHz with the pulse-wave mode and color doppler.

The standard EchoCG approaches were used (parasternal approach along the long and short axes, apical and subcostal approaches) with assessment of the end-diastolic (EDD) and end-systolic (ESD) dimensions of the left ventricle (LF), LV end-

diastolic (EDV) and end-systolic (ESV) volumes, thickness of the interventricular septum (IVS) and LV posterior wall (LVPW), anteroposterior dimension of the left atrium (LA), right ventricle (RV) and right atrium (RA).

The cardiac contractile function and central hemodynamics were calculated using the biplane method of Simpson et al. (1972). The analysis of EchoCG parameters included calculation of the parameter vales of central and cardiac hemodynamics: systolic output (SO), ejection fraction (EF) using the generally recognized procedures. LV MM was calculated using formula [11] basing on the findings of EchoCG M-mode.

The peak transmitral blood flow velocities were measured in the pulse mode with calculation of E/A coefficient (ratio of maximum blood flow velocities in the period of early and late LV filling) in order to assess the cardiac diastolic function.

Ultrasonic scanning of the right and left CCA in the B-mode in combination with Doppler sonography and color mapping of intravascular blood flow in the arteries was performed using the Semi-yeni apparatus (manufactured in Hong Kong) with the help of a linear sensor 7 MHz. When assessing CCA remodeling bilaterally in three planes, we oriented at changes in rectilinearity in the artery course, revealing deformations in different regions, changes in the wall structure (calcinosis, calcifications), detection of increased IMT, changes in the interadventitial diameter (IAD), presence of intravascular AP. The pulsatility index (PI) (according to Gosling) and resistance index (according to Pourselot) (RI) were determined.

The intima-media complex structure was investigated as per recommendations of the international consensus [25]. IMT was assessed quantitatively at the distal section of CCA at a distance of 1-1.5 cm from bifurcation, outside a zone of AP on its posterior wall with averaging three maximum measurements. The cursor is placed at the border "artery lumen - vessel intima" and at the border "media-adventitia". The image was synchronized with diastole. The presence of structural changes in CCA was documented basing on detection of initial atherosclerotic changes in the form of IMT increase by more than 0.9 mm [25]. The literature data was taken as standard values for IAD and ratio IMT/IAD (ore than 8.3 mm for IAD, less than 0.13 for IMT/IAD [3].

The endothelium vasomotor function was investigated by the example of the right brachial artery using the method [10] in modification [7]. The diameter increment of the right brachial artery of more than 10% from the initial value was considered as positive

Table 1. The distribution of patients in respective subgroups depending on the above mentioned signs

Age			
Patient grades	18-44 years (young)	49-59 years (middle)	60-74 years (elderly)
Men	18 (14.52%)	38 (30.65%)	30 (24.19%)
Women	2 (1.61%)	19 (15.32%)	16 (12.9%)
Total	20 (16.13%)	57 (45.97%)	46 (37.1%)
AH degrees			
Patient grades	Degree I	Degree II	Degree III
Men	11 (8.87%)	66 (53.23%)	9 (7.26%)
Women	6 (4.84%)	22 (17.74%)	10 (8.06%)
Total	17 (13.71%)	88 (70.97%)	19 (15.32%)
Heart remodeling (considering the type)			
	Without remodeling	Eccentric type	Concentric type
Men	6 (4.84%)	34 (27.42%)	46 (37.1%)
Women	12 (9.68%)	14 (11.29%)	12 (9.68%)
Total	18 (14.52%)	48 (38.71%)	58 (46.77%)

Table 2. Means ($M \pm m$) of some parameters of structural-functional myocardium condition and intracardial hemodynamics in patients of different age

Parameters	18-44 years (n = 20)	45-59 years (n = 57)	60-74 years (n = 47)
IVST	1.18 + 0.04 (0.7 – 1.42)	1.21 + 0.02 (0.85 – 1.5)	1.23 + 0.02 (0.9 – 1.5)
LV PWT	1.09 + 0.03 (0.7 – 1.33)	1.14 + 0.02 (0.9 – 1.5)	1.14 + 0.02 (0.8 – 1.5)
LV EDD	5.19 + 0.1 (4.4 – 6.0)	5.05 + 0.06 (3.8 – 5.9)	5.07 + 0.07 (4.2 – 6.1)
LV ESD	3.72 + 0.12 (3.0 – 4.8)	3.56 + 0.06 (2.5 – 4.5)	3.59 + 0.08 (2.6 – 4.9)
LV EDV	116.6 + 3.89 (87 – 145)	112.6 + 2.75 (62 – 161)	116.2 + 3.3 (78 – 168)
LV ESV	48.65 + 2.42 (32 – 67)	49.37 + 1.72 (22 – 79)	52.78 + 2.57 (28 – 103)
LV SV	67.59 + 2.54* (51.0 – 93.0)	63.09 + 1.8* (34.0 – 97.0)	63.41 + 1.72 (37.0 – 93.0)
LV EF	58.44 + 1.29 (48.06 – 71.17)	56.29 + 1.0 (36.28 – 69.0)	55.24 + 1.19 (36.03 – 67.65)
$\Delta S\%$	28.65 + 1.21 (17.86 – 36.54)	29.67 + 0.56 (18.18 – 39.13)	29.4 + 0.86 (18.64 – 39.53)
LV MM	237.0 + 13.42 (118.7 – 344.5)	239.7 + 6.35 (145.9 – 367.8)	243.3 + 8.01 (140.5 – 386.1)
LV MMI	108.4 + 5.18 (65.17 – 152.7)	115.5 + 3.13 (71.23 – 186.9)	118.1 + 3.7 (72.96 – 182.3)

result, and the diameter increment of less than 10% was considered as negative result; if the diameter decreased as compared to the initial value this was assessed as paradoxical reaction [7].

The laboratory tests included measurement of blood plasma glucose, total cholesterol (TC), triglycerides (TG), high density lipoprotein (HDL) cholesterol levels using “Biocon” kits (Germany). LDL content was calculated using formula of Friedwald W. [13].

The study materials were subject to statistical processing using methods for parametric and non-parametric analysis. Accumulation, correction, systematization of the baseline information and visualization of obtained results were made in electronic tables Microsoft Office Excel 2016. The statistical analysis was performed using STATISTICA 12.1 software (developed by StatSoft, Inc). The quantitative values were assessed for compliance with the normal distribution, for which purpose we used Shapiro-Wilk test (if subject number was less than 50) or Kolmogorov-Smirnov test (if subject number was more than 50) and also parameters of asymmetry and excess; two independent groups with normal distribution of quantitative signs were compared using Student's t-test. Median (Me) and interquartile ranges were calculated for statistical analysis of the data with non-normal distribution. Correlation analysis was performed using Pearson (r) coefficient. The critical significance level (p) was accepted as 0.05.

RESULTS

Means of the test parameters of the heart and vessel remodeling in different age subgroups of patients are presented in Tables 2 and 3.

As the data presented in Table 2 show that the parameter values reflecting the structural and functional myocardium condition and remodeling were characterized by increased LV SV in the young age group (as compared to middle age group reaching significance) and also by insignificant trend to decrease of LV EF and vice versa to increasing IVST, LVPVT, LV EDV, LV MM and LV IMM in older age groups.

The analysis of the data characterizing the presence and type of cardiac LV remodeling, structural and functional CCA condition and vasomotor endothelium function in the brachial artery (Table 3) showed that absence of LVH was observed in all compared groups with approximately equal frequency while the cases of revealing both asymmetric (AsH) and concentric (CH) hypertrophy prevailed insignificantly in older age groups of patients.

CCA IMT (right and/or left) exceeded 0.9 mm in 27 (10.9%) patients from young age subgroup, 91 (36.7%) patients from middle age subgroup and 81 (32.7%) from elderly age subgroup. Both IMT median for right and left CCA and the values of the lower and upper quartile increased with the age of examined patients.

IAD values and, consequently, IMT/IAD ratio in right and left CCA increased significantly in older age groups (in patients of middle age and to even greater extent in elderly patients). And AP in the CCA system were revealed only in patients of the middle and elderly age. PI in left CCA was significantly higher among the subjects of the elderly age as compared to patients of the young age.

Besides that, cases of pathologic IMT thickening and also deviations of I values and IMT/IAD ratio from the normal values were observed significantly more often in patients of the middle age as compared to young age subgroup. Patients of the elderly age had the same trends not reaching the statistical significance as compared to young age subgroup and they were similar to those in middle age group. The correlations between the age and IMT in the right ($r = 0.21198$) and left ($r = 0.1836384$) CCA were weak.

The number of patients with the negative and paradoxical vascular reaction during the reactive hyperemia test also increased with the age what was evidence of increasing number of cases of endothelium vasomotor function disturbance.

The analysis of the main blood lipid profile parameter values (Table 4) demonstrated absence of significant differences

Table 4. Means ($M \pm m$) of the blood lipid profile parameter values in patients of different age

Parameters	18-44 years (n = 20)	45-59 years (n = 57)	60-74 years (n = 47)
TC (mmol/l)	5.72 + 0.22 (4.6 – 8.3)	5.7 + 0.18 (3.0 – 11.2)	5.43 + 0.16 (3.1 – 8.2)
HDL (mmol/l)	1.11 + 0.05 (0.8 – 1.73)	1.11 + 0.03* (0.75 – 1.8)	1.01 + 0.03* (0.7 – 1.6)
LDL (mmol/l)	3.78 + 0.17 (2.66 – 5.33)	3.75 + 0.14 (1.83 – 7.49)	3.61 + 0.15 (1.72 – 6.06)
VLDL (mmol/l)	0.84 + 0.07 (0.46 – 1.73)	0.83 + 0.05 (0.36 – 2.0)	(0.81 + 0.04) (0.36 – 1.86)
TG (mmol/l)	1.84 + 0.14 (1.0 – 3.8)	1.83 + 0.1 (0.8 – 4.4)	1.79 + 0.09 (0.8 – 4.1)
AC	4.24 + 0.17 (3.17 – 5.56)	4.25 + 0.15 (2.0 – 7.2)	4.55 + 0.22 (1.62 – 8.57)

Notes to Table 4: * significance of differences between the groups “45-59 years” and “60-74 years”: Student's t-test – 1.12, number of degrees of freedom $f = 101$ ($p = 0.264924$)

Table 3. Means ($M \pm m$) of some parameters of the structural and functional heart and CCA condition (remodeling), rate of cases of their pathologic change and worsened vasomotor endothelium function in patients of different age

Parameters	18-44 years (n = 20)	45-59 years (n = 57)	60-74 years (n = 47)
Rate of LV hypertrophy cases			
Asymmetric type	10 (8,1%)* ¹	23 (18,5%)* ¹	15 (12,1%)
Concentric type	6 (4,8%)* ² ; ^{^1}	27 (21,8%)* ²	25 (20,2%)* ^{^1}
Without LVH	4 (3,2%)	7 (5,6%)	6 (4,8%)
Parameters of the structural and functional condition of the right (r) and left (l) CCA			
		CCA IMT (r)	
Median	1,0	1,2	1,19
Lower quartile	0,88	1,0	1,09
Upper quartile	1,26	1,36	1,37
		CCA IMT (l)	
Median	1,1	1,18	1,26
Lower quartile	0,98	1,0	1,09
Upper quartile	1,3	1,36	1,35
CCA IAD (r)	7,39 + 0,18 ^{^4} (6,07 – 9,21)	7,55 + 0,11 ^{#1} (5,45 – 9,02)	8,2 + 0,16 ^{^4} ; ^{#1} (6,4 – 11,1)
CCA IAD (l)	7,35 + 0,16 ^{^5} (5,52 – 8,46)	7,57 + 0,1 (5,99 – 9,8)	7,97 + 0,18 ^{^5} (6,17 – 12,18)
CCA IMT/ IAD (r)	0,138 + 0,006* ⁴ (0,096 – 0,180)	0,159 + 0,004* ⁴ (0,091 – 0,270)	0,151 + 0,006 (0,081 – 0,257)
CCA IMT/ IAD (l)	0,150 + 0,007 (0,108 – 0,221)	0,153 + 0,004 (0,106 – 0,235)	0,156 + 0,005 (0,100 – 0,254)
CCA PI (r)	1,11 + 0,04 (0,69 – 1,33)	1,18 + 0,05 (0,78 – 3,8)	1,12 + 0,03 (0,85 – 1,5)
CCA PI (l)	1,16 + 0,05 ^{^6} (0,62 – 1,54)	1,16 + 0,16 (3,7 – 0,72)	1,78 + 0,03 ^{^6} (0,67 – 1,54)
CCA RI (r)	0,71 + 0,02 (0,51 – 0,81)	0,74 + 0,01 (0,56 – 1,0)	0,73 + 0,01 (0,59 – 0,98)
CCA RI (l)	0,74 + 0,02 (0,44 – 0,87)	0,73 + 0,01 (0,53 – 0,9)	0,75 + 0,01 (0,53 – 0,89)
Number of pathologically changed parameter values (in the right and left CCA, n = 248)			
AP	0 (0,0%)	7 (2,82%)	11 (4,44%)
Pathologic IMT	27 (10,89%)* ¹ ; ^{^1}	91 (36,69%)* ¹	81 (32,66%)* ^{^1}
Pathologic IAD	36 (14,52%)* ²	93 (37,5%)* ²	59 (23,79%)
Pathologic IT/IAD	26 (10,48%)* ³ ; ^{^2}	85 (34,27%)* ³	69 (27,82%)* ^{^2}
Variants of endothelium response (in the right brachial artery, n = 124)			
Positive	36 (14,52%)* ⁵ ; ^{^7}	7 (2,82%)* ⁵	7 (5,65%)* ^{^7}
Negative	5 (4,03%)	15 (12,1%)	11 (8,87%)
Paradoxic	8 (6,45%)* ⁶ ; ^{^8}	24 (19,35%)* ⁶	22 (17,74%)* ^{^8}

Notes to Table 3: * – significance of differences between groups “18-44 years” and “45-59 years”; ^ – significance of differences between groups “18-44 years” and “60-74 years”; # – significance of differences between groups “45-59 years” and “60-74 years”; ** – differences between groups “18-44 years” and “45-59 years” reached significance by the chi-square test, chi-square test with Yates correction and with likelihood ratio but they were not significant by two-sided Fischer’s exact test; ^^ – differences between groups “18-44 years” and “60-74 years” reached significance by chi-square test, chi-square test with Yates correction and with likelihood ratio, but they were not significant by two-sided Fischer’s exact test

*¹ – Fisher’s exact test (two-sided) = 0.01151 ($p < 0.05$); *² – Fisher’s exact test (two-sided) = 0.00016 ($p < 0.05$);

*³ – $p = 0.004697$; Student’s t-test – 2.92, (number of degrees of freedom $f = 75$); *⁴ – $p = 0.004742$; Student’s t-test – 2.91;

*⁵ – Fisher’s exact test (two-sided) = 0.0000001 ($p < 0.05$); *⁶ – Fisher’s exact test (two-sided) = 0.00155 ($p < 0.05$)

**¹ – chi-square test = 56.270 ($p < 0.001$); chi-square test with Yates correction = 54.525 ($p < 0.001$); chi-square test with likelihood ratio = 56.638 ($p < 0.001$); **² – chi-square test = 43.337 ($p < 0.001$); chi-square test with Yates correction = 41.830 ($p < 0.001$); chi-square test with likelihood ratio = 44.548 ($p < 0.001$); **³ – chi-square test = 49.030 ($p < 0.001$); chi-square test with Yates correction = 47.382 ($p < 0.001$); chi-square test with likelihood ratio = 50.979 ($p < 0.001$)

^{^1} – Fisher’s exact test (two-sided) = 0.000017 ($p < 0.05$); ^{^2} – Student’s t-test – 3.26 (number of degrees of freedom $f = 64$);

$p = 0.001807$; ^{^3} – Student’s t-test – 2.06; $p = 0.043735$; ^{^4} – Student’s t-test – 3.36; $p = 0.001314$; ^{^5} – Student’s t-test – 2.57;

$p = 0.012404$; ^{^6} – Student’s t-test – 10.63; $p = 0.000000000001$; ^{^7} – Fisher’s exact test (two-sided) = 0.0000001 ($p < 0.05$); ^{^8} – Fisher’s exact test (two-sided) = 0.00401 ($p < 0.05$)

^{^^1} – chi-square test = 45.225 ($p < 0.001$); chi-square test with Yates correction = 43.566 ($p < 0.001$); chi-square test with likelihood ratio = 46.843 ($p < 0.001$); ^{^^2} – chi-square test = 30.151 ($p < 0.001$); chi-square test with Yates correction = 28.765 ($p < 0.001$); chi-square test with likelihood ratio = 30.990 ($p < 0.001$)

^{#1} – Student’s t-test – 3.35, (number of degrees of freedom $f = 101$); $p = 0.001150$

between the patient groups being compared, only the plasma HDL level was significantly lower in patient group of the elderly age as compared to patients of the middle age (the differences from young age group did not reach statistical significance).

DISCUSSION

In view of modern conceptions, the heart remodeling is a sequence of interrelated changes at the molecular and cell level resulting in progressing physiologic and anatomic transformations of the cardiovascular system on the whole. The heart remodeling can include, first of all, cardiac cavity dilation, wall hypertrophy which, finally, cause changes of the cardiac geometry from the ellipsoid shape to more round spherical shape [8]. The heart remodeling may develop for up to several years without any symptoms manifesting clinically only when rather serious organ changes occurred.

All patients examined in the present study had AH of different degree (I-III) and, according to our findings, the greater degree of both the heart and vessel remodeling was observed as patient's age increased. The effect of AH on the development of heart remodeling was reported also in other studies [9].

As it was mentioned, LV hypertrophy and remodeling observed often in patients with essential hypertension result from the complex interaction between several hemodynamic and non-hemodynamic mechanisms. The structural changes in LV are influenced by such factors as ethnicity, gender, salt consumption, obesity and diabetes mellitus and also neurohumoral and genetic factors. The LV remodeling in hypertension has the adaptive nature and develops as a response to increased stress for its walls while when the adaptation resources are exhausted, the cardiac chambers are dilated and heart failure develops [16]. LV hypertrophy itself is not directly associated with the increased cardiovascular morbidity and mortality. But recently mainly experimental data appeared which is evidence of the fact that LV hypertrophy can be not obligatorily considered as an adaptive response to systemic hypertension, but it is a more complex phenotype [19].

LV SV values were significantly higher in young age subgroup as compared to middle age subgroup. The insignificant trend to LV EF decrease and vice versa increase of IVST, LV PWT, LV EDV, LV MM and LV NNI with the age.

The age being the risk factor for development of heart failure causes the higher prevalence of myocardial infarction in elderly patients, who can develop more likely heart failure after myocardial infarction. As it was mentioned above, the increasing action of stresses and shifts in the signal paths with the age change biology of cardiomyocytes in which metabolism by products and damaged organelles are accumulated gradually, the intracellular autophagia recirculation process are blocked and cell predisposition to apoptosis is increased. Besides that, the lowered ability to cardiomyocyte regeneration in elderly people because of decreased rates and extent of cell division and disturbed function of stem cells results in further heart dysfunction and non-adaptive response to diseases or stress [23]. This can partially explain the increase of the signs of LV remodeling in patients of the older age revealed by us.

Our findings are in agreement with other studies which reported the increased LV MMI, LV PWT values and also LV fraction shortening and dimensions of the left atrium in patients of the elderly age as compared to the middle age what was explained by the authors with several basic factors described above [18].

According to our data, the frequency of both AsH and CH was increased in patients of the middle and elderly age.

The similar results were obtained also in other studies which

showed that both men and women more often had LV remodeling of concentric type as the age increased. The authors revealed the direct correlation between LV mass and systolic BP level and also body weight index and the negative correlation with the level of BP decrease during the treatment of AH and the high level of high-density lipoprotein cholesterol [12].

Thus, although the age is one of the most powerful risk factors for development of cardiovascular diseases, the mechanisms mediating the cardiovascular morbidity and mortality with ageing still have been studied insufficiently.

According to our data, the parameter values reflecting the right and left CCA remodeling (IMT, IAD and IMT/IAD ratio) increased significantly in older age groups. AP in CCA were revealed only in patients of the middle and elderly age. PI in left CCA was significantly higher among subjects of the elderly age.

The causes for age-related differences in the context of vascular remodeling are not finally known today, though other studies also revealed the correlation between ageing and considerable changes in the structural and functional vessel characteristics [14]. The diameter, tortuosity and bifurcation angle are increased in CCA with ageing what was explained by the authors with degradation and fragmentation of intramural elastin [15]. There were evidences of the fact that the age being an independent predictor of artery remodeling, can cause the lowered capability for adaptation vessel remodeling during the organism ageing [29].

The PI increase in elderly subjects revealed by us is in agreement with the data of other researchers [6] and an evidence the remodeling in the microcirculation system on the whole. The more often revealing of vasomotor endothelium dysfunction in older age groups of patients is also in accordance with the literature data [5]. The lowered HDL content at elderly age revealed by us was observed also by other researchers [1; 2]. Although the study performed by Bulgakova et al. revealed moderate correlations between the biologic age and TC, LDL and AC which were not detected in our study.

Thus, according to our findings, the age of the examined patients with AH had a significant influence on aggravation of the heart and vessel remodeling processes and increasing frequency of endothelial dysfunction development and blood lipid profile disorders, mainly, due to lowering of antiatherogenic HDL fraction.

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