Characteristics of changes and clinical and instrumental predictors of the severity of structural remodelling of carotid arteries in hypertensive patients

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Abstract

Background. Mechanisms of activation of the process of vascular wall remodelling in patients affected by arterial hypertension have not been studied in depth and require clarification.

Materials and methods. The study included 381 patients with hypertension — 212 men and 169 women of the average age 53.0 (47; 60) years. The structural-functional vessel status was determined by the method of duplex scanning and colour duplex mapping of blood flow with the Logiq 500 MO apparatus (GE, USA). Statistical analyses were made using Microsoft Excel software kit , Statistica for Windows 6.0.

Results. The patients with hypertension presented some left-right asymmetry of remodelling extracranial carotid arteries. Unlike the impact of remodelling of the right carotid artery, the most essential effect on the left carotid artery was the impact of daytime pulse arterial pressure and variability of the nocturnal systolic arterial pressure (the strength of impact 25.0 and 13.9%, respectively. The processes of remodelling of the right carotid artery are more sensible to the impact of high values of nocturnal diastolic arterial pressure (the strength of impact 16.4%). The beginning of some brain complication is associated with the significant increase in atherosclerotic affection not only of the left, but also of the right carotid artery.

Conclusions. Remarkable remodelling of the right carotid artery is often associated with the severity of the disease and to some extent reflects the severity of the flow of the disease and can be regarded as an additional unfavourable feature.

key words: hypertonic disease, carotid arteries, vessel remodelling, atherosclerosis, predictors

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Introduction

One of the reasons for the increased peripheral resistance in arterial hypertension (AH) is the change in the properties of the vascular wall, the so-called vascular remodelling. It is an important mechanism that is responsible for the process of improvement of vascular reserve, autoregulation of cerebral blood flow and the development of atherosclerosis [1–6]. The vascular lesions in hypertension involve endothelial dysfunction, thickening of the large arteries intima-media, especially the carotid. This results in

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the development and progression of atherosclerosis [2, 5, 7]. The walls of the arteries thicken and become rigid. The lumen of the vessel is changed. Often the arteries elongate and become convoluted. A decrease in organ perfusion, including the brain, is observed [1, 2, 7, 8]. The thickness of the intima-media complex in carotid arteries (CA) is considered one of the independent risk factors of transient ischaemic attacks and stroke. The relationship between type of ischaemic stroke, its localization, extent of the ischaemic damage foci and the intima-media thickness (IMT) were demonstrated [9, 10]. However, it is believed that the importance of IMT as a risk factor of vascular events is not completely defined [11]. Kazmierski *et al.* (2004) confirmed the role of compensatory thickening of IMT to ensure the adequacy of blood flow [12].

The activation mechanisms of the remodelling process in the vascular wall are not reliably found. It is known that vascular remodelling in hypertension is not necessarily accompanied by a significant increase in the number of cells or mass of the medial layer. Changes of the blood vessel lumens can occur as a result of a combination of cell proliferation and apoptosis, activation of the synthesis of connective tissue matrix and/or its degradation [1]. The processes of hypertrophy and proliferation of smooth muscle cells of the blood vessels can be influenced by adrenergic stimulation, platelet growth factor, changes of the matrix metalloproteinase activity, urokinase plasminogen activator and many other factors [1, 7]. Consequently, in the genesis of cerebrovascular and other cardiovascular (CV) complications in essential hypertension (EH) the role of remodelling of blood vessels, which supply blood to the brain, and the activation mechanisms of the remodelling process in the vascular wall in hypertensive patients are not fully understood and require further definition and clarification.

The purpose of the study was to reveal the peculiarities of structural changes of the vascular wall and independent clinical and instrumental predictors of the severity of extracranial carotid arteries structural remodelling in hypertensive patients.

Materials and methods

Overall, 381 patients with EH were examined. There were 212 males and 169 females, aged from 35 to 70 years, the mean age was 53.0 (47; 60). Stage II hypertension was diagnosed in 231 (60.6%) patients, stage III — in 150 (39.4%) patients. There was a combination of hypertension with coronary heart disease (CHD) in 259 (68.0%) patients: stable angina of the II functional class (FC) was found in 56 (14.7%) subjects, stable angina of FC III — in 196 (51.4%). Eighty-eight (23.1%) patients had previous history of myocardial infarction (MI). Forty-five (11.8%) patients suffered from acute cerebrovascular accident (ACVA). The combination of MI and stroke was found in 17 (4.5%) patients. The vast majority of patients had AH of the 1st, 2nd and 3rd degree: 16 (4.2%); 292 (76.6%) and 73 (19.2%) patients, respectively. Chronic heart failure (CHF) of FC I–II [according to the criteria of the New York Heart Association (NYHA)] was diagnosed in 175 (45.9%) patients, III FC — in 163 (42.8%) patients. Duration of hypertension averaged 10.1 ± 0.32 years. Normal geometry was established in 66 patients (17.4%), concentric remodelling — in 90 (23.6%), concentric LVH — in 156 (40.9%) and eccentric LVH — in 69 (18.1%) patients. Diagnosis of EH and CHD was established according to the recommendations of the European Society of Hypertension and European Society of Cardiology ESH/ /ESC (2007) [13]. The study did not include patients with history of MI or stroke less than 3 months prior to the study, subjects with liver or kidney dysfunction, diabetes, heart disease, chronic heart failure of the FC IV, chronic obstructive pulmonary disease and respiratory failure. The control group included 30 healthy subjects of similar age and gender.

Prior to the research, screening, thorough acquisition of complaints and anamnesis had been performed as well as patients' agreement had been obtained to participate in the research according to the ethic norms of the Helsinki Declaration.

Evaluation of structural and functional state of the heart was performed based on echocardiography data (Echo) [14–17]. Daily blood pressure profile was assessed using ambulatory blood pressure monitoring (ABPM) [18]. Structural and functional state of vessels was assessed by duplex scanning and colour Doppler mapping of blood flow [19, 20]. To study the functional state of the endothelium of blood vessels, all patients underwent Doppler ultrasound of the brachial artery [21].

Statistical calculations were performed using the software Microsoft Excel and "Statistica" for Windows 6.0. Average values were expressed as Me (II), where Me — median, II — interquartile interval. Determination of reliability of differences between groups was performed using Mann-Whitney test. A comparative analysis of differences was performed according to Pearson criterion χ^2 [22]. Aggregate indicators of the severity of structural remodelling of the CA were calculated to determine the inde-

pendent predictors of structural remodelling of the left and right CA. Overall, 381 clinical cases of EH of the II–III stages and 132 different clinical and instrumental parameters were used to form basic statistical matrix. Two summary indices (defined as the sum of scores) were analysed as the output parameter: 1) severity of the right CA structural remodelling (SRrca) and 2) severity of the left CA structural remodelling (SRlca). Table I shows the method of summary results calculation. For indicators of SRlca and SRrca, the minimum total score was 0 points and the maximum — 7 points. The average values of SRrca and SRlca were 3.2 ± 0.02 and 3.4 ± 0.03, respectively. Multiple step-by-step regression (module "Multiple Regression" of the package StatSoft "Statistica" V. 5.5) was used to conduct multivariate analysis [22].

Multivariate analysis was presented as the form of linear regression models of type Y = f (X1, X2, ..., Xn), where X1, X2, ..., Xn were the independent variables that characterized clinical and functional state of patients and Y was the output parameter of the severity of carotid arteries structural remodelling. Taking into account the large number of indicators for analysis, we have developed two types of models — clinical and instrumental, that influenced the severity of structural remodelling of the left and right carotid arteries. The clinical and instrumental parameters were considered as independent predictors, if they showed significant ($p < 0.05$) association with the source parameter. The informativeness of complex influence of factors on the output parameter was estimated by multiple regression coefficient calculation (coefficient of determination — R); the adequacy — utilizing Residual analysis for calculation of actual and critical (df) value of Fisher test (F-test, and significance level (p); specificity — using the error calculation of the impact factors (St. error of estimate). The following tables contain obtained models with the minimum and maximum informativeness. Beta-coefficient (β-coefficient) was used for statistical characteristics of the independent predictors that showed the strength of factor influence on the output parameter and the nature of that impact (a positive β-coefficient testified for the direct correlation and a negative β-coefficient — for the feedback). In addition to the β-coefficient, the strength of influence of selected indicators on the degree of impairment of the structural and functional state of the myocardium was determined. For greater clarity, the influence of individual factors on the output parameter was represented as a percentage (counted as the ratio of the β-factor to the sum of all β-coefficients) [22].

Table I. The method of summary results calculation

Criteria	Scores
The total index of structural remodelling severity of the left and right carotid arteries (SRlca and SRrca)	
The presence of atherosclerotic plaques	
Plaque absent	0
Plaque(s) present	1
The presence and nature of carotid artery stenosis	
Stenosis absent	0
Stenosis < 50% of vessel lumen	1
Stenosis > 50% of vessel lumen	2
The presence of deformations of the carotid arteries	
Deformation not observed	0
Deformation(s) of carotid arteries present	1
The value of intima-media thickness (IMT)	
IMT ≤ 0.6 mm	0
IMT = 0.7–0.9 mm	2
IMT > 0.9 mm	3

Results and discussion

During the analysis of structural and functional extracranial arteries it was determined that the prevalence of CA atherosclerotic lesions significantly increased in patients with stage II hypertension without coronary artery disease (47.3%) compared with the control group (8.0%). The fact of the asymmetric defeat of CA was discovered. According to the literature review this fact has not received an adequate attention. So, according to our results, a more distinct change of the group with EH without IHD was observed in the left CA: the features of atherosclerotic lesions of the left and right CA were identified in 35 (31.8%) and 17 (15.5%) patients, respectively, $p < 0.05$ [23]. A clear tendency to increase the frequency of defeat of right CA in combination of EH and IHD was observed, compared with EH patients without IHD ($p = 0.08$). Marked thickening of the intima-media (IMT) was observed both in the II and III stage of EH. More significant changes of IMT were found in patients with EH of the III stage. Also, the absolute value of IMT progressively increased with age and duration of hypertension.

The degree of atherosclerotic lesions of the left CA significantly increased from 24.9% to 41.9% ($p < 0.05$) with the increase of the degree of hypertension (from the 2nd to 3rd) in patients with stage II hypertension. The defeat of the right CA remained roughly at the same level as at the II degree of AH. This

indicates that the increase of BP leads to preferential damage of the left CA, which is experiencing greater haemodynamic influence due to anatomical features — as it originates directly from the aortic arch. The increase in the number of atherosclerotic plaques with higher values of BP in patients with stage II hypertension was accompanied by an increase in the prevalence of the left CA stenosis (> 30% of the lumen), $p < 0.05$. The prevalence of the right CA stenosis in this group of patients did not meet the confidence level in relation to patients with stage II hypertension and moderate hypertension [24]. It should be noted that in patients with stage II hypertension and severe hypertension the number of deformations of the right CA [in the form of the curves (angular and accompanying) and gyri] was significantly increased ($p = 0.009$). The increasing prevalence of similar deformities of the left CA during the III degree of AH was on the verge of confidence ($p = 0.06$). Marked thickening of both the left and right CA was found, but without significant differences between groups of patients with different degrees of hypertension. Diameters of both CA were significantly increased in patients with uncomplicated EH, but with severe hypertension, compared with the group of patients with moderate hypertension ($p < 0.05$). A decrease of linear blood flow velocity (LBFV) at the 3rd degree of AH in the left CA was observed ($p < 0.05$). Thus, remodelling of extracranial arteries occurs primarily with the growth of the degree of hypertension in patients with stage II hypertension due to left CA, which is experiencing greater haemodynamic load. Changes in the right CA have the same direction, but do not reach the validity in relation to the group of patients with moderate hypertension. Remodelling of the right CA is mainly due to the increase of the number of different strains.

Further progression of the disease, the appearance of EH complications were accompanied by the progression of the structural and functional restructuring of the CA wall. So, there was a significant increase in the rate of atherosclerotic plaques in extracranial arteries of patients with EH of the III stage and moderate hypertension compared with the group of patients with EH of the II stage and a similar degree of hypertension. It should be noted that a significant increase in the number of plaques was observed not only in the left, but also in the right CA: 50.5% versus 24.9% ($p < 0.0001$) in the left CA and left 34.0 % versus 20.1% ($p = 0.009$) in the right CA, respectively [4, 23]. The revealed changes confirm the influence on the process of atherosclerotic vascular damage not only the magnitude of blood pressure, but also other factors, which increase the severity of the disease. Significant progression of the atherosclerotic process in the right CA in patients with EH of the III stage and moderate hypertension has led to a substantial reduction of LBFV [to 0.76 (0.66–0.84) m/s] compared with the group with stage II hypertension and similar levels of blood pressure.

The frequency of CA atherosclerotic lesions was significantly increased in patients with EH of the stage III compared with EH of the stage II. More expressive changes were determined in the left CA throughout the group of patients with EH of the stage III: 82 (54.7%) versus 48 (32.0%) in the right CA ($p < 0.0001$). The frequency of stenosis and deformation of CA significantly increased in patients with EH of the stage III. When evaluating the frequency of atherosclerotic lesions of CA, depending on the nature of the complications, the predominant lesion by the atherosclerotic process was observed in the left CA compared with right one in patients without cerebral catastrophes in history: 15 (44.1%) in the left CA versus 7 (20.6%) in the right CA in the group of patients without myocardial infarction (MI) and CHF IIA, respectively ($p < 0.05$), and 38 (53.5%) versus 17 (23.9%), respectively, in patients with cardiosclerosis after MI ($p < 0.0003$) [25]. In the analysis of structural and functional parameters of CA that were obtained from the patients with EH of the stage III, who had history of acute cerebrovascular disease (ACVD), it was determined that in patients with cerebral stroke (MI) there was a gradual increase in the frequency of atherosclerotic injury of the right CA compared with patients who did not have history of brain accidents: 42.9% in the group with ACVD compared with 23.9% in patients with IM ($p < 0.05$), and 20.6% in patients without MI and MI ($p < 0.05$), respectively. Prevalence of the right CA damage in patients with a history of a brain catastrophe significantly increased and almost equalled to the prevalence of the left CA lesions. Significant difference between the number of atherosclerotic plaques in the left and right CA was not determined in patients with a history of cerebral stroke [50.0% versus 42.9%, respectively ($p = 0.59$)] and in patients with the history of MI and stroke [88.2% vs 70.6%, respectively ($p = 1.62$)]. So, the atherosclerotic lesions were almost identical in the left and right CA. The increase in the number of atherosclerotic plaques, mostly in the right CA, was accompanied by the process of stenosis, that led to a significant reduction of the lumen of the vessel and has gained confidence in comparison with MI group ($p = 0.011$) and group without MI and stroke ($p = 0.013$), respectively. In parallel with the progression

of the atherosclerotic process and the development of haemodynamically significant stenosis, statistically significant increase in the number of different strains in the right CA occurred in patients with ACVD compared with patients with EH who had not had brain complications. The highest prevalences of atherosclerotic plaques in the right CA in parallel with the increase of atherosclerotic plaques in the left CA were registered in patients with a double disaster: MI and stroke. Thus, the appearance of cerebral complications was associated with a significant increase of atherosclerotic lesions not only in the left, but also in the right CA. Significant remodelling of the right CA was often associated with the severity of the disease. Moderate and direct correlation between the presence of plaques in right CA and systolic dysfunction of the left ventricle was identified ($r = 0.27$, $p = 0.002$). Also, it was a correlation between stenosis of the right CA and systolic dysfunction of the left ventricle ($r = 0.23$, $p = 0.01$) [25].

Thus, increase of the right CA remodelling reflects the severity of the disease and can be regarded as an additional adverse prognostic sign. So, in the study of the extracranial vessels remodelling process and factors associated with it, we revealed noticeable differences in the degree of changes and in the time of their occurrence in the left and right common CA. It can be assumed that this is due to different influence of clinical and instrumental parameters on the structural remodelling of extracranial vessels, which was confirmed by the results of a multivariate regression analysis.

On the right CA remodelling the most pronounced influence provided such clinical factors as patient's age, stage of hypertension and waist circumference (impact force is equal to 55.7%; 30.3%; 14.0%, respectively). The explanations are given in Tables II and III.

From the parameters identified by instrumental methods, the greatest influence was exerted by the presence of diastolic and systolic LV dysfunction (DT and EF, respectively, as its value); impairment of vasodilative vascular endothelial function evidenced by a reduced response of the brachial artery to decompression test, as well as a small degree of night decrease of NDP in %. The power of influence of these factors was equal to 29.8%; 31.7%; 22.1%; 16.4%, respectively (Tables IV and V).

Analysis of the impact of studied clinical parameters on remodelling of the left CA showed common factors with the right carotid artery. They are the following: patient's age, waist circumference, stage of EH (the strength of the influence was equal to 48.0%; 20.3%; 18.7%, respectively). However, the strength of the influence of atrial fibrillation significantly increased (13.0%), which was not observed in the analysis of the remodelling process of the right CA (Tables VI and VII).

Usually atrial fibrillation (AF) occurs in patients with severe LVH, a long course of EH that may be accompanied by changes in the myocardium and vascular wall. This is the evidence of the presence of correlation between LVH and IMT in patients with severe EH. It was noted that remodelling of the left CA occurred earlier and was more pronounced than of the right one. This may be explained by more pronounced haemodynamic stress due to anatomical features of left CA — it originates from the aortic

Table II. Clinical models of the severity of the right CA structural remodelling in patients with EH

Step 1 SRrca = –2.19 + 0.10 Age R = 0.62, p < 0.0001; St. error of estimate = 1.97
Step 2 SRrca = –4.57 + 0.10 Age + 0.95 EHst R = 0.73, p < 0.0001; St. error of estimate = 0.85
Step 3 SRrca = –7.00 + 0.11 Age + 0.90 EHst + 0.023 WC R = 0.75, p < 0.0001; St. error of estimate = 0.83

Notes: 1. Age — patient's age in years; 2. EHst — binary measure coded in points, characterizing clinical stage of EH (2 points mean EH of the stage II, 3 points — EH of the stage III); 3. WC — waist circumference in cm

Table III. The analysis of clinical predictors of the severity of the right CA structural remodelling

The factor	β-coefficient	R	Strength of the influence (%)
Age	0.68	0.000003	55.7
EHst	0.37	0.0001	30.3
WC	0.17	0.008	14.0

Table IV. Instrumental models of the severity of the right CA structural remodelling in patients with EH

Step 1 SRrca = 1.04 + 0.013 DT R = 0.40, p = 0.013; St. error of estimate = 1.15
Step 2 SRrca = 3.95 + 0.014 DT – 0.05 EF R = 0.53, p = 0.003; St. error of estimate = 1.08
Step 3 SRrca = 3.78 + 0.012 DT – 0.05 EF + 0.62 RVBAd R = 0.58, p = 0.002; St. error of estimate = 1.04
Step 4 SRrca = 4.28 + 0.011 DT – 0.05 EF + 0.57 RVBAd – 0.02 DI DBP R = 0.61, p = 0.002; St. error of estimate = 1.01

Notes: 1. DT — deceleration time of early diastolic left ventricular filling in ms (Doppler Echocardiography); 2. EF — ejection fraction of the left ventricle in %; 3. RVBAd — reduced vasodilatation of the brachial artery on decompression (reactive hyperaemia test) is a binary indicator (0 points — response is normal and 1 point — reduced); 4. DI DBP — the value of the daily index of DBP in %

Table V. Analysis of instrumental predictors of the right CA structural remodelling severity in patients with EH

The factor	β-coefficient	R	Strength of the factor influence (%)
DT	0.31	0.003	29.8
EF	–0.33	0.002	31.7
RVBAd	0.23	0.011	22.1
DI DBP	–0.17	0.023	16.4

Table VI. Clinical models of the severity of left CA structural remodelling in patients with EH

Step 1 SRlca = –1.69 + 0.093 Age R = 0.54, p < 0.0001; St. error of estimate = 1.10
Step 2 SRrca = –6.72 + 0.11 Age + 0.05 WC R = 0.61, p < 0.0001; St. error of estimate = 1.04
Step 3 SRrca = –7.68 + 0.11 Age + 0.04 WC + 0.60 EHst R = 0.65, p < 0.0001; St. error of estimate = 1.01
Step 4 SRrca = –7.00 + 0.10 Age + 0.037 WC + 0.60 EHst + 0.73 AF R = 0.69, p < 0.0001; St. error of estimate = 0.98

Notes: 1. Age — patient's age in years; 2. WC — waist circumference in cm; 3. EHst — stage of EH in points (2 and 3 points mean the stage II of EH); 4. AF — a binary feature which characterizes the presence of AF episodes (0 points — no history of AF; 1 point — prior history of AF episodes)

Table VII. The analysis of clinical predictors of the severity of the left CA structural remodelling in patients with EH

The factor	β-coefficient	R	Strength of the factor influence (%)
Age	0.59	0.000001	48.0
WC	0.25	0.003	20.3
EHst	0.23	0.003	18.7
AF	0.16	0.014	13.0

Table VIII. Instrumental models of the severity of left CA structural remodelling in patients with EH

Step 1 SRlca = 7.11 — 0.065 EF R = 0.39, p = 0.014; St. error of estimate = 1.24
Step 2 SRlca = 7.97 — 0.075 EF — 0.04 %Δd br. art. R = 0.50, p = 0.006; St. error of estimate = 1.19
Step 3 SRlca = 5.63 — 0.086 EF — 0.039 %Δd br. art. + 0.047 DPAP R = 0.57, p = 0.003; St. error of estimate = 1.14
Step 4 SRlca = 6.50 — 0.092 EF — 0.030 %Δd br. art. + 0.053 DPAP — 0.64 V_E/V_A R = 0.63, p = 0.001; St. error of estimate = 1.09
Step 5 SRlca = 4.25 — 0.086 EF — 0.030 %Δd br. art. + 0.064 DPAP — 0.55 V_E/V_A + 0.082 NV CBP R = 0.67, p = 0.0009; St. error of estimate = 1.05

Notes: 1. EF — ejection fraction of the left ventricle in %; 2. %Δd br. art. — the value that characterizes the % increase in brachial artery diameter for 90 sec after decompression (reactive hyperaemia test); 3. DPAP — daily pulse AP in mm Hg; 4. Ve/Va is the ratio of the velocity of early to late diastolic left ventricular filling; 2. NV CBP — nocturnal variability of CBP in mm Hg

Table IX. Analysis of instrumental predictors of the severity of the left CA structural remodelling in patients with EH

The factor	β-coefficient	R	Strength of the factor influence (%)
EF	–0.51	0.0004	31.5
%Dd br. art.	–0.24	0.009	14.8
DPAP	0.40	0.007	25.0
Ve/Va	–0.24	0.018	14.8
NV CBP	0.23	0.021	13.9

arch. However, the progression of the disease significantly activated remodelling of the right CA.

Differences in instrumental parameters which influence the processes of the left CA remodelling were more pronounced compared with those of the right one (Tables VIII and IX). Such factors as the decline of EF, impairment of the vasoactive function of endothelium (reduction of increase of the brachial artery diameter in the test with decompression), diastolic dysfunction of the left ventricle (the force of the impact was equal to 31.5%; 14.8% and 14.8%, respectively) continued to be important for remodelling of the left and the right CA. In contrast to the right CA remodelling the daily PBP and variability of the night CBP was significant for the left CA (the power of influence was equal to 25.0% and 13.9%, respectively). It was supposed that increasing values of BP were more conductive to remodelling of the left CA than of the right one.

Recent studies have convincingly demonstrated the adverse role of peripheral PBP in the development of CHD, CHF, chronic renal failure and increased cardiovascular risk. It allowed the experts of ESH to attribute PBP to the risk factors of adverse prognosis in patients with hypertension in older age groups [21, 25]. It was found that the magnitude of PBP effected on the appearance of structural changes in the aorta and large vessels, as well as their degree of compliance.

Thus, the remodelling processes of the extracranial vessels were deeply connected with structural and functional changes that occur in the myocardium. However, structural changes in the left and right CA

did not occur synchronously. Most of the identified factors affected the remodelling of both CA. However, some differences in clinical and instrumental parameters were revealed. So, the remodelling of the left CA was more influenced by high values of DPA and variability of the CBP. The remodelling of the right CA was influenced by high values of DAP.

Conclusions

1. Certain asymmetry of extracranial vessels' remodelling was revealed in patients with EH. In patients with uncomplicated EH most of the changes were defined in the left CA, but not in the right one (31.8% versus 15.5%, $p < 0.05$). The increase in the degree of hypertension led to the progression of atherosclerotic lesions prevalence mainly in the left CA. The combination of IHD and EH was associated with an increase of the right CA damage. Changes in the right CA were more extensive in patients with severe EH. They were associated with the severity of the disease rather than depended on the degree of hypertension.
2. The largest structural changes in the left and right CA (88.2% and 70.6%, respectively) were determined in patients with a history of a "double catastrophe" — MI and CS. In the presence of MI, atherosclerotic lesions were observed mainly in the left CA (53.5% versus 23.9%, $p < 0.05$). The appearance of cerebrovascular complications was associated with a significant increase of atherosclerotic lesions not only in the left, but also in the right CA (50.0% and 42.9%, respectively; $p > 0.05$).
3. Predictors of structural and functional changes in the left and right CA are the stages of EH (the strength of influence is 30.3% and 18.7%, respectively), patients' age (the strength of influence is 55.7% and 48.0%, respectively) and increased waist circumference (the strength of influence is 14.0% and 20.3%, respectively). The remodelling of the left CA was more influenced by high values of DPAP, variability of the CBP (the strength of influence is 25.0% and 13.9%, respectively). The remodelling of the right CA was influenced by high values of DAP (the strength of the influence is 16.4%). The remodelling processes of both CA were closely associated with the presence of endothelial dysfunction, structural and functional changes in the myocardium.

Continued research in this direction will allow us to study more deeply the pathogenesis of disorders of structural and functional state of arteries. It will give the possibility to determine the degree of cardiovascular risk in patients with essential hypertension and the presence of carotid arteries remodelling more accurately and to develop preventive and therapeutic measures, which are very important and relevant to modern cardiology.

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