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COMPARATIVE EVALUATION OF MYOCARDIAL REMODELING IN PATIENTS WITH HYPERTENSIVE DISEASE AND COMORBIDITY WITH CORONARY HEART DISEASE

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231 patients by hypertensive disease with II stages were inspected, 105 men and 126 women, the average ages of 52.3±1.2 years. Duration of arterial hypertension was the average 9.7±0.45 years. At 121 from 231 inspected patient's ischemic heart disease – stable angina of II-III of functional class was diagnosed. Chronic heart failure of II-III FC (NYHA) was present at 90 from the inspected patients: at 39 (35.5 %) from 110 patients in a group with hypertensive disease and at 51 (42.1 %) from 121 – in the group of hypertensive disease in combination with coronary heart disease. Research purpose: estimation of deposit of coronary heart disease in the processes of remodeling and functional states of myocardium for the patients of hypertensive disease II the stages. It was set that tacking of coronary heart disease to hypertensive disease increases the degree of pathological remodeling of myocardium, mainly, due to dilatation of heart, and instrumental in progress of systole and diastolic dysfunction. Combination of hypertensive disease and coronary heart disease is instrumental in the increase of number of patients with violations of cardiac rhythm, including with ventricular extrasystoles of high gradation of Lown. Arterial hypertension and coronary heart disease make worse each another, and their combination results in the increase of cardiovascular risk.

Key words: hypertensive disease, coronary heart disease, structurally-functional indexes of myocardium, remodeling of myocardium, systolic dysfunction, diastolic dysfunction.

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ПОРІВНЯЛЬНА ОЦІНКА РЕМОДЕЛЮВАННЯ МІОКАРДА У ХВОРИХ НА ГІПЕРТОНІЧНУ ХВОРОБУ ТА ПРИ КОМОРБІДНОСТІ З ІШЕМІЧНОЮ ХВОРОБОЮ СЕРЦЯ

Проведене обстеження 231 пацієнта з гіпертонічною хворобою II стадії, 105 чоловіків і 126 жінок, середній вік 52,3±1,2 років. Тривалість артеріальної гіпертонії склала в середньому 9,7±0,45 років. У 121 з 231 обстежених хворих діагностована ішемічна хвороба серця – стабільна стенокардія II-III функціонального класу. Хронічна серцева недостатність II-III ФК (по NYHA) була у 90 з обстежених пацієнтів: у 39 (35,5 %) з 110 хворих в групі з гіпертонічною хворобою і у 51 (42,1 %) з 121 – в групі пацієнтів з гіпертонічною хворобою у поєднанні з ішемічною хворобою серця. Мета дослідження: оцінка внеску ішемічної хвороби серця в процеси ремоделювання і функціональний стан міокарда у хворих на гіпертонічну хворобу II стадії. Встановлено, що приєднання ішемічної хвороби серця до гіпертонічної хвороби збільшує ступінь патологічного ремоделювання міокарда, переважно, за рахунок дилатації серця, і сприяє прогресуванню систолічної і діастолічної дисфункції. Поєднання гіпертонічної хвороби та ішемічної хвороби серця сприяє збільшенню числа хворих з порушеннями серцевого ритму, у тому числі, і з шлуночковою екстрасистолією високої градації за Лауном. Артеріальна гіпертензія та ішемічна хвороба серця мають взаємообтяжливу дію і їх поєднання призводить до підвищення кардіоваскулярного ризику.

Ключові слова: гіпертонічна хвороба, ішемічна хвороба серця, структурно-функціональні параметри міокарда, ремоделювання міокарда, систолічна дисфункція, діастолічна дисфункція.

The work is a fragment of the research project "Cardiovascular remodeling, structural and functional state of the liver and kidneys, and their relationship with cardiometabolic risk factors in patients with cardiac pathology and comorbidities. Possibilities of treatment optimization", state registration No. 0124U002036.

Arterial hypertension (AH) is one of the most common diseases of the cardiovascular (CV) system. Numerous studies have convincingly demonstrated the significance of AH as a risk factor for coronary heart disease (CHD), stroke, and chronic heart failure (CHF). The prognosis of the disease worsens and the complexity of therapy increases when AH and CHD are combined, especially in elderly people [1, 7, 10].

The severity and prognosis of AH are determined not only by the level of blood pressure (BP), but also by the degree of damage to target organs. The presence of left ventricular hypertrophy (LVH) is associated with an increased risk of cardiovascular complications (stroke, myocardial infarction) and mortality in patients [12]. It is known that LVH is an independent risk factor that does not depend on other criteria for an unfavorable prognosis [6] and contributes to the development of ventricular arrhythmias, which, in turn, leads to an increased risk of sudden death in patients with hypertension [11]. According to Krumholz H.M. et al., the type of LV remodeling also affects the prognosis for AH [5], with the worst prognosis most often observed in concentric LVH [6]. It has been shown that the presence of systolic and diastolic dysfunction in patients with arterial hypertension leads to the development of signs of chronic heart failure (CHF) [3, 9].

The process of cardiac remodeling involves changes in the geometric characteristics of the ventricles in response to damaging stress or loss of viable myocardium [8]. Initially, cardiac remodeling plays an adaptive role aimed at compensating for hemodynamic changes, but later it leads to the progression of hemodynamic disorders [3, 4]. Although myocardial remodeling processes in patients with hypertension (EH) have been studied in several studies, the effect of concomitant CHD on cardiac remodeling processes and myocardial function remains unclear.

The purpose of the study was to assess the contribution of ischemic heart disease to the processes of remodeling and the functional state of the myocardium in patients with stage II hypertension disease.

Materials and methods. The study included 231 patients with stage II hypertension (105 men and 126 women) aged 35 to 70 years (mean age 52.3±1.2 years). The duration of hypertension in the examined patients ranged from 2 to 20 years, with a mean of 9.7±0.45 years. The control group consisted of 30 healthy individuals matched for gender and age. The diagnosis of hypertension disease (HD) was established after a detailed clinical and instrumental examination and verified in a hospital setting using additional examination methods that allowed symptomatic hypertension to be completely ruled out. The study did not include patients who, at the time of examination, had liver or kidney disease with impaired function, diabetes mellitus, heart defects, stage II-B-III heart failure according to the Strazhesko-Vasilenko classification, chronic obstructive pulmonary disease, or respiratory failure.

Of the 231 patients, 121 were diagnosed with CHD – stable exertional angina pectoris of functional class (FC) II-III. Chronic heart failure (CHF) II-III FC (according to the New York Heart Association (NYHA) classification) was present in 90 patients: including 39 (35.45 %) of 110 patients with EH without clinical and electrocardiographic symptoms of CHD in 51 (42.1 %). Exertional angina was verified using a bicycle ergometer test and Holter ECG monitoring [2]. The average duration of CHD was 4.21±1.6 years. Among clinical signs, ventricular extrasystole was significantly more common in the groups with HD and CHD, which was observed in 34 (28.1 %) patients compared to 9 (8.2 %) patients in the group with HD only (p=0.0001).

To assess the structural and functional state of the myocardium, all patients underwent echocardiography (ECG) using a SIM 7000 Challenge device in M and B modes. Measurements were taken in three consecutive cycles, followed by the averaging of the indicators. The structural and functional parameters of the LV were assessed: end-diastolic and end-systolic dimensions and volumes of the LV (EDD, ESV, EDV, ESV) and their indices. The volumes of the LV cavity and stroke volume (SV) were calculated using the formula of L. Teichholtz et al. The ejection fraction (EF), cardiac index (CI), and stroke index (SI) were calculated using standard methods. The LV myocardial mass (LVMM) and LVMM index were determined according to the recommendations of the ASE (American Society of Echocardiography). A value of more than 115 g/m2 in men and more than 95 g/m2 in women was taken as the criterion for LVH. LV remodeling variants were identified according to the recommendations of A. Ganau et al. based on LVMI and relative wall thickness (RWT). RWT was calculated as the ratio (2× LVW)/EDD. Four types of LV geometry were identified: normal geometry (normal mass and normal relative wall thickness of the LV); concentric remodeling (normal mass and increased relative wall thickness); concentric hypertrophy (increased myocardial mass index and relative LV wall thickness); eccentric hypertrophy (increased mass with normal relative wall thickness).

Daily blood pressure monitoring (DBP) was performed using an ambulatory blood pressure monitoring system. An ABPM-04 recorder (Meditech, Hungary) was used. BP was recorded every 15 minutes during the active period (from 6 a.m. to midnight) and every 30 minutes during the passive period (from midnight to 6 a.m.). The mean values of SBP, DBP, and HR were calculated for 24 hours, periods of wakefulness (from 6 to 24 hours), and sleep (from 24 to 6 hours). The frequency of BP elevation during monitoring (“pressure load”) was assessed using the time index (TI), defined as

the percentage of BP measurements during which BP exceeded the threshold values: 140/90 mmHg for the waking period and 120/80 mmHg for the nighttime period. As an indicator of BP variability, the standard deviation of SBP and DBP was calculated for 24 hours and separately for daytime and nighttime. The daily profile was assessed by the day-night BP difference and the degree of nighttime BP reduction (diurnary index – DI), which is defined as the ratio of the difference between the average BP values during wakefulness and sleep to the average daytime BP values, expressed as a percentage. A normal DI was considered to be greater than 10 % and less than 20 %. The morning rise in BP (MR) from 5 to 10 a.m. was calculated using the formula: MR BP = BP max – BP min.

Statistical calculations were performed using Microsoft Excel and Statistica for Windows 12.0 software packages, applying the t-test for normal distribution of the studied parameters and the Mann-Whitney test for non-parametric distribution. Correlation analysis was performed using the Pearson and Spearman methods, and the linear contrast method was used to identify differences in specific parameters in individual groups.

Results of the study and their discussions. Analysis of changes in structural and functional parameters of the myocardium in stage II HD revealed an increase in the mass of the LV myocardium, due to both an increase in LV wall thickness and an increase in the size of the LV cavity and its volumes.

A comparison of structural and functional parameters in patients with stage II HD and healthy individuals showed that patients with HD had increased mean values of EDV and ESV, wall thickness and LV myocardial mass index, an increase in the diameter of the left atrium (LA), a moderate decrease in ejection fraction (EF), and a decrease in the Ve/Va ratio, which evidenced not only myocardial hypertrophy but also impaired diastolic and, at least, systolic heart function.

Analysis of the frequency of LV remodeling types (according to A. Ganau et al. (1992)) showed that concentric LVH was significantly more common in patients with stage II HD ($p<0.05$). An increase in the degree of AH in patients with stage II HD had little effect on the frequency of formation of different types of LV geometry – the vast majority of patients had concentric LV geometry: concentric LV remodeling or concentric LVH (23.8 % and 45.0 % with stage II AH, respectively, and 22.6 % and 48.4 % with stage III AH, respectively, $p>0.05$).

Analysis of echocardiography parameters characterizing differences in the structural and functional features of the LV showed that in the group of patients with HD and CHD, compared with patients with HD alone, there was a significant increase in LA diameter to 40.0 (37.0; 43.0) mm ($p<0.01$), an 11.3 % increase in LV ESV ($p<0.05$), and a decrease in LV EF to 51.2 (45.2; 56.5) % ($p<0.01$). A direct correlation was found between LV EDS, LV EDD, and the presence of CHD ($r=0.39$, $p<0.01$ and $r=0.31$, $p<0.01$, respectively). A significant increase in the size of the right ventricle to 31.0 (29.2; 35.0) mm ($p<0.01$) was also determined. Changes in the remaining indicators were identical. The LV myocardial mass index did not differ significantly and was equal in patients with both HD and HD+CHD: 118.0 (100.2; 139.5) g/m² and 120.5 (102.3; 143.6) g/m², respectively ($p=0.56$). However, positive correlations were found between LVMM and the presence of CHD ($r=0.33$, $p<0.01$).

There were no significant differences between the groups in the indicators characterizing diastolic heart function Ve, Va, Ve/Va ratio, and DT. There was an increase in the frequency of systolic myocardial dysfunction (EF<45 %) in patients with a combination of HD and CHD to 9.9 % compared to patients with HD alone, where it was 3.6 % ($p<0.05$). A direct correlation was found between the presence of CHD and systolic dysfunction ($r=0.51$, $p<0.01$).

The distribution of trans-mitral flow (TMF) types reflected the general patterns observed in patients with HD. The rigid type of TMF was more common (60.0 % vs. 49.6 %, $p>0.05$) in patients with stage II HD without CHD, while the occurrence of pseudonormal and restrictive types of TMF was slightly, but more frequently, in patients with a combination of HD and CHD ($p>0.05$).

When comparing the indicators in groups of patients with HD without CHD and HD with CHD (Table 1), significant differences were found in the increase in LV EDD and LV ESD ($p=0.048$), a decrease in EF ($p=0.0002$), as well as an increase in the size of the left atrium ($p=0.0006$) and right ventricle ($p=0.004$). At the same time, no significant differences were found in the indicators characterizing the severity of LVM.

A comparison of the mean daily, mean daytime, and mean nighttime BP values, the time index characterizing “pressure load”, and the degree of nighttime BP reduction with the morphofunctional indicators of the myocardium in the group of patients with HD without CHD revealed a fairly close correlation between iMLS and SBP24 ($r=0.57$ DBP ($r=0.41$ and $r=0.36$, $p<0.05$), the value of nighttime SBP ($r=0.51$, $p<0.01$) and the degree of nighttime BP reduction (for SBP $r=0.58$ and for DBP $r=0.53$, $p<0.01$).

Table 1

Structural and functional indicators of the myocardium in patients with stage II hypertension

Parameter	Control group (n=30)	HD without CHD (n=110)	HD with CHD (n=121)
LVMI, g/m^2	78.6 (65.4; 93.2)	118.0 (100.2; 139.5) *	120.5 (102.3; 143.6) *
IVSD, mm	9.3 (8.9; 10.4)	12.0 (11.0;13.0) *	12.0 (11.0; 13.5) *
PWD, mm	8.9 (8.2; 9.6)	12.0 (11.0;13.0) *	12.0 (11.0; 13.5) *
RWT	0.37 (0.35; 0.39)	0.48 (0.44; 0.50) *	0,47 (0.44; 0.53) *
EF, %	60.7 (57.3; 63.4)	55.7 (51.5; 60.0)	51.2 (45.2; 56.5) *^
ESV, ml	45.3 (37.8; 51.5)	71.0 (62.4; 85.0) *	82.4 (65.5; 98.5) *^
EDV, ml	115.2 (108.5; 121.4)	160.2 (140.7; 193.2) *	168.9 (140.7; 200.6) *
LA, mm	34.5 (32.3; 36.9)	38.0 (34,1; 40,0) *	40.0 (37,0; 43,0) *^
RV, mm	25.24 (24.4; 27.6)	29.0 (27.0; 33.0)	31.0 (29.2; 35.0)*^
RA, mm	24.4 (23.2; 25.8)	31.2 (28.0; 34.0)*	31.0 (28.0; 34.0)*
Ve, m/sec	0.78 (0.72; 0.82)	0.53 (0.42; 0.61) *	0.53 (0.47; 0.62) *
Va, m/sec	0.51 (0.49; 0.55)	0.37 (0.33; 0.45) *	0.38 (0.34; 0.44) *
Ve/Va	1.52 (1.47; 1.58)	1.53 (0.93; 1.79) *	1.47 (1.01; 1.66) *

Notes:

1. The results are presented as Med (kvart_1; kvart_3), where Med is the median and kvart_1; kvart_3 are the 1st and 3rd quartiles, respectively; the comparison ofresults was performed using the nonparametric median method.
2. The normality of distribution was assessed using the Shapiro-Wilk and Liljefors methods.
3. * – significance of differences compared to the control group of healthy individuals.
4. ^ – significance ofintergroup differences.

A significant correlation was found when comparing pulse BP (PBP_{24}) with LVMI and interventricular septal thickness (r=0.65 and r=0.59, respectively, p<0.01), which indicates the role of this factor in the development of myocardial hypertrophy. The correlation was more pronounced in patients with AH without CHD (Table 2). This may indicate that the degree of increase in BP, especially systolic and pulse BP, and the degree of decrease in nighttime BP play a leading role in the development of LVH. In patients with EH without CHD, a fairly close relationship was also found between the mean daily SBP and DBP and the Ve/Va index, which characterizes the diastolic function of the left ventricle (r=0.53 and r=0.39, respectively, p<0.05). A weaker correlation was observed between ABPM indicators and LV size and volume.

Table 2

Correlation between SBP and structural and functional parameters of the myocardium in patients with stage II hypertension (r at p<0.05)

Indicators DM BP	Structural and functional parameters ofthe myocardium									
	HD without CHD (n=110)					HD with CHD (n=121)				
	LVMI	IVS	Ve/Va	SDF	DDF	LVMI	IVS	Ve/Va	SDF	DDF
SBP_{24}	0.64	0.43	0.53	ns	0.46	0.53	0.45	0.42	ns	0.41
SBPd	0.41	0.32	0.57	ns	0.53	0.38	0.27	0.34	ns	0.29
DBPd	0.36	0.29	0.39	ns	0.42	0.27	0.23	0.28	ns	0.26
SBPn	0.51	0.39	0.33	0.29	0.54	0.42	0.37	0.26	0.27	0.32
PBP_{24}	0.65	0.59	0.54	0.34	0.42	0.47	0.35	0.28	0.24	0.35
HI	0.54	0.38	0.42	0.23	0.45	0.41	0.33	0.25	ns	0.24
DI SBP	-0.58	-0.51	0.45	0.30	-0.49	-0.50	-0.44	0.37	0.25	0.27
DI DBP	-0.53	-0.42	0.31	ns	-0.28	-0.38	-0.26	0.25	0.24	0.25

Notes:

1. The reliability of the relationship was established at a p<0.05 correlation coefficient.
2. ns – the relationship is unreliable: for a correlation coefficient p>0.05.
3. SDF – frequency of systolic dysfunction.
4. DDF – frequency of diastolic dysfunction.

In the group of patients with HD and CHD, the degree of correlation between the indicators characterizing both LVH and cavity dilatation and BP values was lower, which could indirectly indicate the role of other factors in the development of LV remodeling, apart from BP values. When comparing structural and functional changes at different FCs of angina pectoris, an increase in LVEDD and LVEDV (p=0.027) and an increase in the severity of diastolic dysfunction (DDF) – an increase in the Ve/Va ratio with an increase in the severity of angina pectoris (p=0.034) – were found. Systolic dysfunction was significantly more common in patients with FC III angina. The deterioration of intracardiac hemodynamics, systolic and diastolic function as FC angina increases in patients with AH indicates the role of myocardial ischemia in LV remodeling processes.

The role of myocardial ischemia in LV remodeling may be evidenced by an analysis of data obtained by comparing the indicators of patients with HD without CHD and HD with CHD at the same degree of CHF severity (FC III). In patients with hypertension with CHD and CHF FC III, a large increase in ESV and EDV (p=0.011) and a decrease in LVEF (p=0.0001) were observed. At the same time, an increase in the size of the right ventricle (p=0.046) was observed. In this group, systolic LV dysfunction (p=0.039) and mitral regurgitation (p=0.022) were observed significantly more often, and the frequency of ventricular extrasystoles (p=0.0002) increased with an increase in their severity according to the classification of B. Lown and M. Wolf.

The heterogeneity of CC remodeling processes is due to the mosaic effect of hemodynamic parameters of AH and risk factors for CC complications. There is no doubt that the degree of myocardial hypertrophy depends on the level of BP and the duration of hypertension. It is known that daily ambulatory blood pressure monitoring (ABPM) readings correlate more closely with target organ damage in hypertension and disease prognosis than clinical BP [6, 7]. These data are confirmed by the results obtained in our study on a close direct correlation between the values of LVMI and thickness of the IVSD with the values of SBP_{24}, nocturnal SBP, the degree of nocturnal decrease in SBP and DBP, and, especially, PBP_{24}.

It can be assumed that an increase in the average values of daily, daytime, and nighttime BP, the pressure rise time index, and a decrease in the degree of nighttime BP reduction largely determine the development of LVH due to a constant increase in "pressure load" and may contribute to more frequent and earlier development of CHD, which coincides with the results of other researchers [3, 4].

The results obtained in our study on a significant increase in ESV, the size of the left atrium and right ventricle, and a decrease in EF in patients with CHD combined with HD coincide with the opinion of some authors that a decrease in the coronary blood flow reserve of the myocardium with the development of ischemic myocardial dysfunction is itself characterized by an increase in ESV, EDV, and LV myocardial mass, regardless of the absence of obvious pathomorphological signs of myocardial destruction [4, 8].

Remodeling is the main trigger factor in the formation of systolic and diastolic myocardial dysfunction, the basis for the occurrence and progression of CHF. The results of epidemiological studies indicate that LVH is an independent risk factor for death from cardiovascular diseases, which does not depend on the level of blood pressure, age and other factors, and mortality from cardiovascular pathology and overall mortality are 2-4 times higher than in patients with normal LV mass [6]. The data obtained in our study confirm the results of other researchers, namely, that with the progression of LVH, LV diastolic dysfunction and the likelihood of developing CHF increase, and the risk of developing atrial fibrillation and ventricular arrhythmias progresses [3, 9, 11, 12].

Thus, the addition of CHD to AH increases the degree of pathological remodeling, mainly due to cardiac dilatation, which contributes to the progression of systolic and diastolic dysfunction and increased arrhythmic activity.

Coronary circulation disorders associated with the addition of CHD lead to a deterioration in myocardial energy supply, progression of pathological remodeling of the left ventricle, and a decrease in its contractile capacity and diastolic function. AH and CHD are mutually aggravating factors, and their combination leads to an increase in cardiovascular risk with the onset of cardiovascular complications.

Conclusions

1. When CHD is added to stage II hypertension, no significant differences were found between the indicators of the LVH severity, however, there was a significant increase in LV ESD and LV ESV, LA and RV dimensions, and a significant decrease in LV EF, which increases the degree of pathological myocardial remodeling, mainly due to cardiac dilatation, and contributes to the progression of systolic and diastolic dysfunction.

2. The combination of HD and CHD contributes to an increase in the number of patients with cardiac arrhythmias, including Lown's high-class ventricular extrasystoles.

Further research in this area, studying the structural and functional state of the myocardium in patients with AH associated with various manifestations of CHD, and assessment of the relationship between myocardial remodeling processes and indicators of vegetative and neurohumoral regulation, will reveal additional mechanisms that influence the development and progression of cardiac geometry abnormalities and cardiovascular complications, which is extremely important for practical healthcare.

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