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EFFECT OF THE COMBINATION OF LERCANIDIPINE AND CARVEDILOL ON THE FUNCTIONAL STATE OF THE HEART IN PATIENTS WITH CHRONIC HEART FAILURE AND ARTERIAL HYPERTENSION

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To study the effect of a combination of lercanidipine and carvedilol on heart failure, 36 patients with arterial hypertension and I–III functional state heart failure according to the New York Heart Association classification were enrolled in the study (Group A). The control group consisted of 32 patients with the same pathology (Group B). The groups were divided into subgroups (3 per group) according to the functional states I, II, and III. Subgroups from Group A received lercanidipine and carvedilol, while subgroups from Group B received only carvedilol for 6 months. After treatment, significant improvements in systolic and diastolic function parameters were observed in all subgroups from Group A ($p<0.05$). The magnitude of improvement was most significant in patients with functional class I and progressively attenuated in functional classes II and III. In all subgroups of Group B, only early diastolic filling deceleration time and left ventricular isovolumetric relaxation time improved reliably ($p<0.05$).

Key words: arterial hypertension, left ventricular hypertrophy. carvedilol, lercanidipine, chronic heart failure.

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ВПЛИВ КОМБІНАЦІЇ ЛЕРКАНІДИПІНУ ТА КАРВЕДИЛОЛУ НА ФУНКЦІОНАЛЬНИЙ СТАН СЕРЦЯ У ПАЦІЄНТІВ З ХРОНІЧНОЮ СЕРЦЕВОЮ НЕДОСТАТНІСТЮ ТА АРТЕРІАЛЬНОЮ ГІПЕРТЕНЗІЄЮ

З метою вивчення ефекту комбінації лерканідипіну та карведилолу на серцеву недостатність у дослідження було включено 36 пацієнтів з артеріальною гіпертензією та серцевою недостатністю I–III функціонального класу за класифікацією Нью-Йоркської кардіологічної асоціації (група А). Контрольна група складалася з 32 пацієнтів з тією ж патологією (група В). Групи були розділені на підгрупи (по 3 підгрупи в кожній) відповідно до функціонального класу I, II, III. Підгрупи з групи А отримували лерканідипін і карведилол, а підгрупи з групи В – тільки карведилол протягом 6 місяців. Після лікування у всіх підгрупах з групи А спостерігалося значне поліпшення параметрів систолічної та діастолічної функції ($p<0,05$). Величина поліпшення була найбільш значною у пацієнтів з функціональним класом I і поступово зменшувалася у пацієнтів з функціональними класами II і III. У всіх підгрупах групи В тільки поліпшення часу уповільнення раннього діастолічного наповнення і часу ізоволюметричної релаксації лівого шлуночка виявилися достовірними ($p<0,05$).

Ключові слова: артеріальна гіпертензія, гіпертрофія лівого шлуночка, карведилол, ерканідипін, хронічна серцева недостатність.

Despite significant advances in the treatment of chronic heart failure (CHF), the prognosis for this group remains unfavorable. More than 50 % of patients die within 4 years. Among the European population, CHF occurs in 0.4–2 %. Of the 900 million people in Europe, 20 million have CHF [9, 15].

With the progression of hypertension, remodeling of the heart and blood vessels occurs as a result of damage to target organs. In recent years, alongside studies of the norm and pathology of the left ventricle in cardiovascular diseases, several studies have focused on changes in its geometric structure. It is necessary to recall that during the cardiac cycle, the left ventricle has an ellipsoidal geometric structure in systole (this mechanism is due to the displacement of a large mass of blood under low stress), and a

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spherical shape in diastole (due to the increase in filling of the left ventricle, it contributes to its early filling) [1, 5, 10].

This is a physiologically forced change in the cardiac cycle. Therefore, at the final stage of left ventricular (LV) remodeling, the spherical shape contributes to the deterioration of hemorrhage. Left ventricular hypertrophy (LVH) in arterial hypertension is an adaptive response that compensates for reduced preload on the myocardium.

This effect, due to concentric hypertrophy during systole, reduces the stress on the myocardial walls. At the same time, this myocardial adaptation reduces eccentric hypertrophy. Thus, in arterial hypertension, remodeling, along with a compensatory reaction, further contributes to disease progression and becomes an independent prognostic factor [10].

Several epidemiological studies have indicated the negative impact of LVH. It has been shown that in the presence of left ventricular hypertrophy, mortality rates increase 25–fold [11, 12].

According to echocardiography (EchoCG), geometric adaptation and its various types are observed during left ventricular filling: with LV myocardial mass index ≤N and LV relative wall thickness index (RWTI) ≤0.45 – normal LV model (LVNM); with LV myocardial mass index >N and LV relative wall thickness index ≤0.45 – eccentric LV hypertrophy (LVEH); with LV myocardial mass index >N and LV relative wall thickness index ≥0.45 – concentric LV hypertrophy (LVCH); with LV myocardial mass index ≤N and LV relative wall thickness index >0.45 – concentric LV remodeling (LVCR) [5]. Heart remodeling disrupts structural and functional activity. At this time, an increase in myocardial mass due to dilation and geometric changes, along with systolic and diastolic dysfunction, predicts decompensation. In essential arterial hypertension, remodeling is not primary; it is, on the one hand, a response to increased stress; on the other, the initial activation of the neurohumoral system disrupts cellular ion transport [2, 13, 14].

In arterial hypertension, remodeling is more than a compensatory reaction and is an indicator of progression and an independent negative prognostic factor for the disease. The adverse effects of LVH have been demonstrated in several epidemiological studies, as studying the impact of various remodeling variants on prognosis is relevant [2, 4].

It is important to note that ß-blockers are more effective in the case of normal left ventricular geometric structure, while calcium antagonists are more effective in the case of eccentric hypertrophy, and angiotensin-converting enzyme (ACE) inhibitors are more effective in the case of concentric hypertrophy [4, 8]. Lercanidipine, as an ACE inhibitor, best promotes the regression of left ventricular hypertrophy [7, 8].

The purpose of the study was to evaluate the effect of a combination of lercanidipine and carvedilol on heart failure caused by hypertension and to study its structural and functional state.

Materials and methods. The study was conducted at the basis of Azerbaijan Medical University during 2022–2023. 36 patients with an average age of 54.2±4.7 years (37–62 years), with I, II, III FC heart failure according to the New York Heart Association (NYHA) classification, were involved in the study (20 males and 16 females) – Group A. The control group (average age 51.8±5.1 years; 36–59 years) consisted of 32 patients with I, II, or III FC heart failure (21 males and 11 females) – Group B. The groups were divided into subgroups (3 in each) according to FC heart failure classification (NYHA I, NYHA II, NYHA III). Subgroups NYHA IA (n=13), NYHA IIA (n=12) and NYHA IIIA (n=11), which were formed from Group A, received lercanidipine 10 mg (Berlin-Chemie, Germany) once daily and carvedilol (Grindex, Latvia) 12.5–25 mg once daily, while subgroups NYHA IB (n=12), NYHA IIB (n=10) and NYHA IIIB (n=10), which were formed from Group B, received only carvedilol 12.5–25 mg once daily for 6 months.

Inclusion criteria were: age ≥18 years; chronic heart failure, NYHA functional class I–III; stable clinical condition for at least 4 weeks before enrollment; preserved sinus rhythm; ability to undergo Doppler echocardiographic assessment; and written informed consent.

Exclusion criteria were as follows: NYHA functional class IV heart failure; acute decompensated heart failure or acute coronary syndrome within the preceding 3 months; significant valvular heart disease; persistent atrial fibrillation or other clinically relevant arrhythmias; severe arterial hypertension or hypotension; severe renal or hepatic dysfunction; inflammatory, infectious, or systemic diseases affecting myocardial function; contraindications or intolerance to β-blockers or calcium channel blockers; use of investigational drugs within the preceding 3 months.

The study was conducted in accordance with the principles of the Declaration of Helsinki and the International Conference on Harmonisation–Good Clinical Practice (ICH-GCP) guidelines.

All participants provided written informed consent before enrollment. They were fully informed about the study objectives, procedures, potential benefits, and possible risks, and were free to withdraw at any time without consequences for their medical care. The study did not include vulnerable populations as defined by ICH-GCP, such as children, cognitively impaired individuals, prisoners, pregnant or lactating

women, economically disadvantaged individuals, refugees, or ethnic minorities with restricted autonomy. Only adult patients with stable chronic heart failure (NYHA I–III) who were able to give informed consent were enrolled. All clinical and echocardiographic assessments were performed according to standard procedures. Patient data were anonymized, and privacy was maintained in compliance with applicable national regulations on personal data protection.

Echocardiography was performed at the beginning and end (6 months) of treatment. Using the echocardiograph Vivid-4 (GE, USA) were determined the end-diastolic volume of the left ventricle (LVEDV), end-systolic volume of the left ventricle (LVESV), stroke volume (SV), ejection fraction (EF %), interventricular septum thickness (IVST), posterior wall thickness (PWT), left ventricular myocardial mass index (LVMMI). LV myocardial mass (LVMM) was determined by the formula of R. Devereux. The LVMMI was calculated as the LVMM divided by body surface area. Left ventricular diastolic properties were investigated by studying the transmitral diastolic flow spectrum.

The following were determined: maximum transmitral diastolic flow velocity during early LV filling (E, m/s), maximum transmitral diastolic flow velocity during late LV filling (A, m/s), the ratio of early and late diastolic filling velocities E/A, early diastolic filling deceleration time (DT, ms), LV isovolumetric relaxation time (IVRT, ms).

Statistical data processing was performed using Statistica 6.0 for Windows. Standard statistical methods, including the unpaired Student's t-test, were used to assess group differences. Normality of continuous variables was assessed using the Shapiro-Wilk test. Results are presented as M±m, where M is the mean and m is the standard error of the mean. Overall comparisons were performed using one-way ANOVA. Post hoc pairwise comparisons were conducted using Bonferroni correction. Differences were considered statistically significant at $p<0.05$.

Results of the study and their discussion. At the first stage, hemodynamic parameters of patients in both groups were assessed to obtain an idea of the initial changes before therapy. The results obtained corresponded to those expected for the different NYHA classes of systolic and diastolic function. In both groups (and subgroups), at the beginning of treatment for left ventricular dilation, indicators of left ventricular remodeling were observed (Table 1).

Table 1

Characteristics of groups of patients with CHF against the background of arterial hypertension

Indicator	Group A			pA
	NYHA IA (n=13)	NYHA IIA (n=12)	NYHA IIIA (n=11)	
EF, %	44±2	39±2	34±3	<0.001
LVEDV, ml	170±8	185±10	200±12	0.002
LVESV, ml	100±8	125±10	150±12	<0.001
SV, ml	70±5	60±6	50±7	0.003
IVST, cm	1.25±0.05	1.34±0.05	1.42±0.06	0.001
PWT, cm	1.00±0.05	1.09±0.06	1.18±0.07	0.002
LVMMI, g/m²	145±10	155±10	165±12	0.004
E, m/s	0.85±0.05	0.76±0.05	0.65±0.06	<0.001
A, m/s	0.60±0.03	0.69±0.04	0.78±0.05	<0.001
E/A	1.42±0.07	1.21±0.08	0.83±0.06	<0.001
IVRT, ms	110±4	115±4	125±5	0.001
DT, ms	120±7	133±8	145±9	0.002
	Group B			
Indicator	NYHA IB (n=12)	NYHA IIB (n=10)	NYHA IIIB (n=10)	pB
EF, %	43±0.8	38±0.7	33±0.9	<0.001
LVEDV, ml	175±3.0	190±3.5	205±4.0	<0.01
LVESV, ml	105±4.0	130±4.5	155±5.0	<0.001
SV, ml	72±1.8	60±1.9	50±2.1	<0.01
IVST, cm	1.25±0.02	1.32±0.02	1.40±0.02	<0.05
PWT, cm	1.02±0.02	1.10±0.02	1.18±0.02	<0.05
LVMMI, g/m²	148±3.5	158±3.8	168±4.2	<0.05
E, m/s	0.82±0.02	0.73±0.02	0.62±0.02	<0.001
A, m/s	0.58±0.02	0.66±0.02	0.75±0.02	<0.001
E/A	1.41±0.03	1.19±0.03	0.83±0.03	<0.001
IVRT, ms	108±1.2	112±1.3	122±1.6	<0.05
DT, ms	125±2.5	139±2.8	150±3.2	<0.01

Note: pA–overall p-value (NYHA I–IIIA), the coefficient of significance. pB–overall–p-value (NYHA I–IIIB), the coefficient of significance.

Progressive worsening of systolic and diastolic function parameters was observed in both groups with increasing NYHA functional class, with significant reductions in EF, SV, and E/A ratio and concomitant increases in LV volumes, IVRT, and DT (overall p<0.05–0.001). After 6 months of treatment, results improved in both groups (Table 2).

Table 2

Dynamics of indicators after 6 months of treatment

Indicator	NYHA IA (n=13)			NYHA IB (n=12)		
	Before	After	pA-value	Before	After	pB-value
EF, %	44±2	49±2	0.001	43±0.8	44.0±0.8	0.28
LVEDV, ml	170±8	155±7	0.002	175±3.0	172±3.0	0.22
LVESV, ml	100±8	102±6	>0.05	105±4.0	103±4.0	0.25
SV, ml	70±5	71±3	>0.05	72±1.8	73±1.8	0.30
IVST, cm	1.25±0.05	1.21±0.03	>0.05	1.25±0.02	1.24±0.02	0.34
PWT, cm	1.00±0.05	1.02±0.02	>0.05	1.02±0.02	1.01±0.02	0.36
LVMMI, g/m²	145±10	135±9	0.004	148±3.5	146±3.4	0.19
E, m/s	0.85±0.05	0.95±0.05	0.001	0.82±0.02	0.83±0.02	0.30
A, m/s	0.60±0.03	0.55±0.03	0.010	0.58±0.02	0.57±0.02	0.27
E/A	1.42±0.07	1.72±0.08	<0.001	1.41±0.03	1.45±0.03	0.21
IVRT, ms	110±4	95±4	0.001	108±1.2	100±1.1	0.004
DT, ms	120±7	105±6	0.002	125±2.5	112±2.3	0.003
	NYHA IIA (n=12)			NYHA IIB (n=10)		
Indicator	Before	After	pA-value	Before	After	pB-value
EF, %	39±2	43±2	0.003	38.0±0.7	38.6±0.7	0.31
LVEDV, ml	185±10	172±9	0.004	190±3.5	187±3.4	0.25
LVESV, ml	125±10	124±8	>0.05	130±4.5	128±4.4	0.28
SV, ml	60±6	62±4	>0.05	60±1.9	61±1.9	0.33
IVST, cm	1.34±0.05	1.29±0.03	>0.05	1.32±0.02	1.31±0.02	0.35
PWT, cm	1.09±0.06	1.01±0.04	>0.05	1.10±0.02	1.09±0.02	0.37
LVMMI, g/m²	155±10	145±10	0.006	158±3.8	156±3.7	0.23
E, m/s	0.76±0.05	0.82±0.05	0.004	0.73±0.02	0.74±0.02	0.33
A, m/s	0.69±0.04	0.63±0.04	0.012	0.66±0.02	0.65±0.02	0.29
E/A	1.21±0.08	1.30±0.08	0.008	1.19±0.03	1.21±0.03	0.24
IVRT, ms	115±4	105±4	0.005	112±1.3	104±1.2	0.006
DT, ms	133±8	120±7	0.004	139±2.8	124±2.6	0.005
	NYHA IIIA (n=11)			NYHA IIIB (n=10)		
Indicator	Before	After	pA-value	Before	After	pB-value
EF, %	34±3	37±3	0.020	33.0±0.9	33.8±0.9	0.34
LVEDV, ml	200±12	190±11	0.030	205±4.0	202±3.9	0.29
LVESV, ml	150±12	151±10	>0.05	155±5.0	152±4.8	0.31
SV, ml	50±7	50±8	>0.05	50±2.1	51±2.1	0.35
IVST, cm	1.42±0.06	1.40±0.02	>0.05	1.40±0.02	1.39±0.02	0.38
PWT, cm	1.18±0.07	1.11±0.05	>0.05	1.18±0.02	1.17±0.02	0.40
LVMMI, g/m²	165±12	155±11	0.040	168±4.2	166±4.1	0.27
E, m/s	0.65±0.06	0.70±0.06	0.030	0.62±0.02	0.63±0.02	0.35
A, m/s	0.78±0.05	0.72±0.05	0.040	0.75±0.02	0.74±0.02	0.32
E/A	0.83±0.06	0.97±0.07	0.020	0.83±0.03	0.85±0.03	0.26
IVRT, ms	125±5	118±5	0.040	122±1.6	114±1.5	0.020
DT, ms	145±9	135±8	0.030	150±3.2	138±3.0	0.018

Note: pA–overall p-value (NYHA IA–IIIA) before and after treatment, the coefficient of significance. pB–overall p-value (NYHA IB–IIIB) before and after treatment, the coefficient of significance.

After treatment, significant improvements in systolic and diastolic function parameters were observed in all NYHA subgroups. In Group A, EF increased, while LVEDV and LV myocardial mass index decreased significantly (p<0.05). Diastolic filling improved, as reflected by increased E velocity and E/A ratio, along with reduced A velocity, IVRT, and DT. The magnitude of improvement was most significant in patients with NYHA functional class I and progressively attenuated in NYHA functional classes II and III.

In Group B, the hemodynamic parameters EF %, LVEDV, LVMMI, E, A, E/A were not reliable (P>0.05) when compared, but IVRT, DT were reliable (P<0.05).

The present study demonstrated a clear difference in therapeutic response between Group A (combined lercanidipine and carvedilol therapy) and Group B (carvedilol monotherapy).

In Group A, significant improvements were observed not only in diastolic indices (IVRT and DT), but also in systolic function (EF), left ventricular remodeling parameters (LVEDV, LVMMI), and transmitral flow characteristics (E, A, E/A), particularly in patients with NYHA functional class I.

In contrast, Group B showed a more limited response: while IVRT and DT significantly decreased across all NYHA classes, no reliable changes were detected in EF, LV volumes, myocardial mass index, or transmitral flow parameters. Significantly, in both treatment arms, the magnitude of improvement progressively declined from NYHA class I to NYHA class III, suggesting reduced myocardial reversibility at more advanced stages of heart failure.

Overall, these findings indicate that combined therapy exerts a broader and more pronounced effect on both systolic and diastolic dysfunction, whereas monotherapy predominantly influences diastolic relaxation, especially in patients with milder functional impairment.

The hemodynamic effect of ß-blockers and Ca antagonists complements each other well. ß-blockers, by prolonging diastole and reducing oxygen demand, improve diastolic filling of the left ventricle with blood [8]. Lercanidipine, in turn, weakens myofibrils and increases calcium deposition in the sarcoplasmic reticulum, slowing the heart rate and increasing the diastolic filling time of the left ventricle. Lercanidipine, by causing arterial vasodilation, reduces cardiac afterload. The combination of lercanidipine and carvedilol had a significant effect on myocardial remodeling, promoting regression of LVH and exerting cardioprotective effects [5].

In addition, lercanidipine differs from earlier generations of dihydropyridine calcium channel blockers (DHP–CCBs) by not provoking sympathetic hyperactivation. Unlike first- and second-generation agents, it induces vasodilation of both afferent and efferent glomerular arterioles, thereby maintaining stable intraglomerular pressure. Clinical data indicate that it can reverse structural alterations in the microvasculature of patients with arterial hypertension. The medicine is generally well tolerated, showing a considerably lower incidence of peripheral edema – especially ankle swelling -compared with amlodipine and nifedipine [11].

Similar studies were conducted by the other authors [3, 6, 7]. Derosa G et al. (2015), to compare the impact of lercanidipine and barnidipine on echocardiographic indices in patients with left ventricular hypertrophy, enrolled 144 individuals. They were randomly allocated to receive either lercanidipine (20 mg/day) or barnidipine (20 mg/day), in combination with losartan (100 mg/day), for 6 months. Echocardiographic examinations were conducted both at baseline and after the intervention period. A more pronounced decrease in LVMMI was observed in the barnidipine + losartan group. IVST during diastole also showed a significant decline with barnidipine therapy, whereas PWT was reduced under both regimens, although barnidipine + losartan achieved a superior effect. The ratio of early diastolic filling velocity to atrial contraction velocity increased notably in patients treated with barnidipine + losartan, but remained unchanged in those receiving lercanidipine + losartan. Additionally, barnidipine in combination with losartan significantly shortened the isovolumetric relaxation time and reduced the left atrial volume index, whereas no such changes were observed with lercanidipine + losartan. Thus, the authors reported a more negligible effect of lercanidipine than in our study, but it should be noted that they studied patients with type 2 diabetes mellitus. Probably, this factor can impact on the results [3].

Some studies supported the effectiveness of CCBs, with no differences in antihypertensive efficacy. One such work was conducted by Fici F, et al (2017). The purpose of their study was to investigate the effect of follow-up monitoring on blood pressure regulation in patients enrolled in the HYT (HYperTension survey), who were receiving combined therapy with various dihydropyridine calcium channel blockers and inhibitors of the renin-angiotensin-aldosterone system (RAAS), yet had uncontrolled blood pressure at baseline the evaluation included determining the proportion of patients who achieved blood pressure control after 3 and 6 months of follow-up, and comparing both the control rates and mean blood pressure levels among subgroups treated with different DHP-CCB regimens. The results indicate that regular follow-up of hypertensive patients undergoing treatment increases the likelihood of achieving blood pressure control, and no significant differences in antihypertensive efficacy were observed across the different CCB regimens [6]. However, in this study, the impact on cardiac function (systolic and diastolic) was not evaluated.

Conclusions

1. After treatment, significant improvements in systolic and diastolic function parameters were observed in all NYHA subgroups. In Group A, EF increased, while LVEDV and LV myocardial mass index decreased significantly ($p<0.05$). Diastolic filling improved, as reflected by increased E velocity and E/A ratio, along with reduced A velocity, IVRT, and DT. The magnitude of improvement was most significant in patients with NYHA functional class I and progressively attenuated in NYHA functional classes II and III.

2. In all subgroups of Group B, the changes of hemodynamic parameters EF %, LVEDV, LVMMI, E, A, E/A were not reliable ($P>0.05$) when compared before and after treatment, but IVRT, DT improvement were reliable ($P<0.05$).

The study results showed that heart failure developed due to arterial hypertension, treated with a combination of lercanidipine and carvedilol, along with regression of remodeling, improves heart function. Combining lercanidipine and carvedilol in patients with CHF and hypertension improves left ventricular diastolic function. Future studies will examine the impact of this combination on hospitalization time and mortality.

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