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Research Article

# HEART REMODELING, STATE OF HEMODYNAMICS AND STRUCTURAL AND FUNCTIONAL STATE OF MYOCARDIA IN PATIENTS WITH POST-INFARCTION HEART FAILURE

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## ABSTRACT

In patients with left ventricular (LV) systolic dysfunction, changes occurring in cardiomyocytes and the extracellular matrix after myocardial injury (eg, myocardial infarction or myocarditis) lead to pathological remodeling of the ventricle with dilatation, changes in geometry (the LV becomes more spherical), and impaired contractility. Next, there is a systemic response to a decrease in LV systolic function. In patients, there is an increase in the activity of pressor systems: sympathoadrenal system (SAS), renin - angiotensin - aldosterone system (RAAS), endothelin, vasopressin and cytokines systems. Activation of the renin-angiotensin-aldosterone and sympathetic nervous systems is of key importance. These neurohumoral factors not only cause peripheral vasoconstriction, sodium and fluid retention, but, consequently, an increase in the hemodynamic load on the left ventricle, but also have a direct toxic effect on the myocardium, stimulating fibrosis and apoptosis, which leads to further remodeling of the heart and disruption of its function. Clinically, all these changes are associated with the development and progression of symptoms of CHF and lead to a deterioration in the quality of life, a decrease in the physical activity of patients, decompensation of heart failure requiring hospitalization, and death as a result of both "pumping" heart failure and the appearance of life-threatening ventricular arrhythmias. The severity of clinical manifestations of diastolic CHF and the prognosis of patients is primarily determined by the severity of diastolic dysfunction [19-22] which leads to further remodeling of the heart and disruption of its function.

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KEYWORDS

sympathoadrenal system (SAS), renin - angiotensin - aldosterone system (RAAS), endothelin, vasopressin and cytokines systems.

## INTRODUCTION

Clinically, all these changes are associated with the development and progression of symptoms of CHF and lead to a deterioration in the quality of life, a decrease in the physical activity of patients, decompensation of heart failure requiring hospitalization, and death as a result of both "pumping" heart failure and the appearance of life-threatening ventricular arrhythmias. The severity of clinical manifestations of diastolic CHF and the prognosis of patients is primarily determined by the severity of diastolic dysfunction [19-22] which leads to further remodeling of the heart and disruption of its function.

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**Purpose of the study:** to conduct a comparative analysis of the main clinical, laboratory and instrumental parameters of 112 patients with chronic heart failure who had myocardial infarction with diastolic and mixed LV dysfunction

## MATERIAL AND METHODS OF RESEARCH

a comparative analysis of the main clinical, laboratory and instrumental parameters of 112 patients with chronic heart failure who had myocardial infarction with diastolic and mixed (diastolic and systolic) LV dysfunction was carried out on the basis of the cardiology department of the Bukhara branch of the RRCEM.

## RESULTS AND DISCUSSIONS

Analysis of complaints and physical examination data showed the following: the main complaints (shortness

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of breath, weakness, palpitations) were present in patients of both groups of remodeling, but they were significantly pronounced in patients with mixed LV dysfunction. In this group, edema of the lower extremities was noted much more often than in CHF patients with LV diastolic dysfunction (p<0.05).

During physical examination in patients with mixed LV dysfunction, such signs of circulatory insufficiency as small bubbling rales in the lungs, swollen jugular veins, cardiomegaly, palpitations, hepatomegaly (p<0.05) were significantly more often detected (Figure 1).

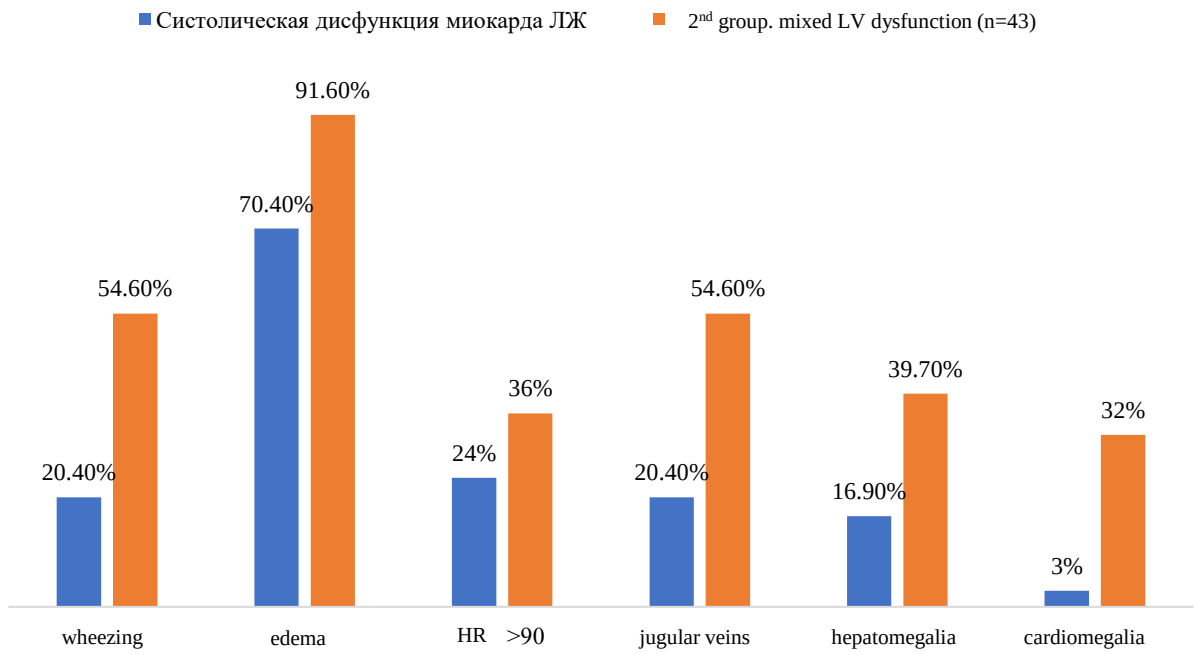


**Figure 1. Indicators of a comparative analysis of data from a physical examination of patients with CHF who had a myocardial infarction with diastolic and mixed LV dysfunction**

Table 1 and Figure 2 present the assessment of the clinical condition of patients (according to the test with a 6-minute walk.

**Table 1.**

**Evaluation of the clinical condition of patients with postinfarction heart failure depending on the characteristics of LV myocardial dysfunction according to the test with a 6-minute walk (M±m)**

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| Groups / Index | I. Group Diastolic dysfunction n=69 (56.3%) | Group II. Mixed dysfunction n=43 (43.7%) |
|---|---|---|
| 6-minute walk test, m | 306.9 ± 98.7 | 179 ± 71.8* |

**Note: * - significant difference (p<0.05) from that of group I**

Patients with mixed LV dysfunction had a significantly lower tolerance (179 ± 71.8 m) to physical activity, in contrast to patients with diastolic dysfunction (306.9 ± 98.7 m), according to the test with a 6-minute walk. It should also be noted that patients with mixed LV dysfunction had a significantly higher score for SHOKS, which indicates the severity of clinical manifestations of CHF in this group of patients.

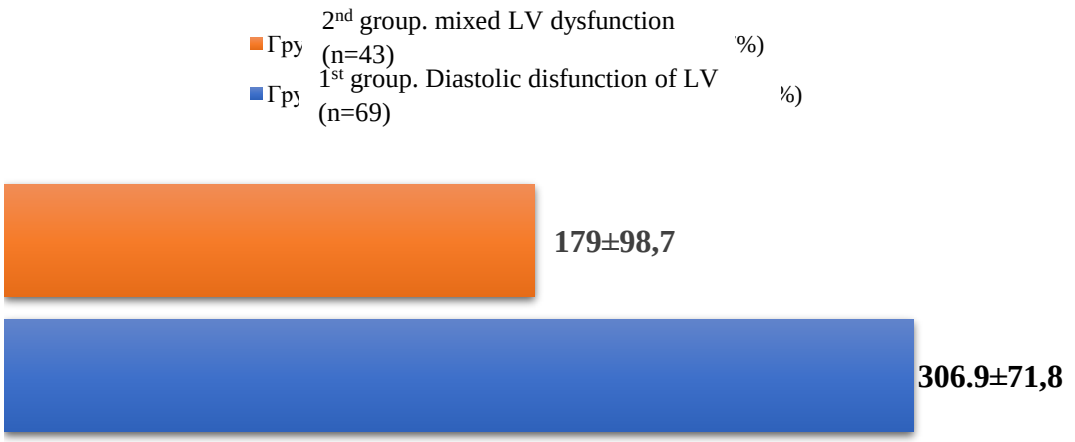


**Figure 2. Evaluation of the clinical condition of patients with postinfarction heart failure, depending on the characteristics of LV myocardial dysfunction according to the test with a 6-minute walk**

In patients with mixed LV dysfunction, III-IV FC CHF was significantly more often observed, compared with patients with diastolic LV dysfunction, in whom FC I-II CHF was more often noted.

Table 2 and Figure 3 show data on the structural and functional state of the myocardium in patients with postinfarction heart failure - in the group of patients

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with LV diastolic dysfunction and in the group of patients with a mixed form of LV dysfunction.

**Table 2.**

**Parameters of structural and functional parameters of the myocardium in patients with postinfarction heart failure, depending on the characteristics of LV myocardial dysfunction (M±m)**

| Groups / Index | Group ILV diastolic dysfunction (n=69) | Group IIMixed LV dysfunction(n=43) |
|---|---|---|
| EDD, mm | 56.6 ± 6.55 | 71.6 ± 5.26* |
| ESD, mm | 40.5 ± 6.81 | 58.4 ± 9.23* |
| BWW, ml | 157.4 ± 48.7 | 278.7 ± 71.2* |
| CSR, ml | 80.1 ± 23.4 | 179.1 ± 46.2* |
| EF, % | 52.9 ± 7.26 | 35.1 ± 5.34* |
| MM, gr | 238.1 ± 56.5 | 287.4 ± 76.1* |
| OTS LV | 0.46 ± 0.2 | 0.36 ± 0.11 |
| MS, dyne/cm2 | 229.5 ± 63.8 | 288.3 ± 67.5* |
| PV/MS | 0.24 ± 0.03 | 0.13 ± 0.04* * |

**Note: * - significant difference (p<0.05) from that of group I**

As can be seen from the data presented in Table 2, patients of group II with mixed LV dysfunction were characterized by a more pronounced violation of systolic and diastolic function of the heart (mixed dysfunction) and they had a higher CHF FC.

In particular, with mixed LV diastolic dysfunction: indicators of EDR 71.6 ± 5.26 mm, ECR 58.4 ± 9.23 mm, ECD 278.7 ± 71.2 ml, ESR 179.1 ± 46.2 ml, which exceeds these indicator of the group of patients who have LV diastolic dysfunction: CDR 56.6 ± 6.55 mm, CSR 40.5 ± 6.81 mm, CSR 157.4 ± 48.7 ml, CSD 80.1 ± 23.4 ml, respectively (figure 3)

Between groups of patients depending on the characteristics of LV myocardial dysfunction there are statistically significant differences in the remodeling of

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the heart, respectively, and the state of systemic hemodynamics.

Against the background of the development of further dilatation of the LV cavity and an increase in myocardial mass, an increase in myocardial systolic stress occurred in both groups (Figure 3).

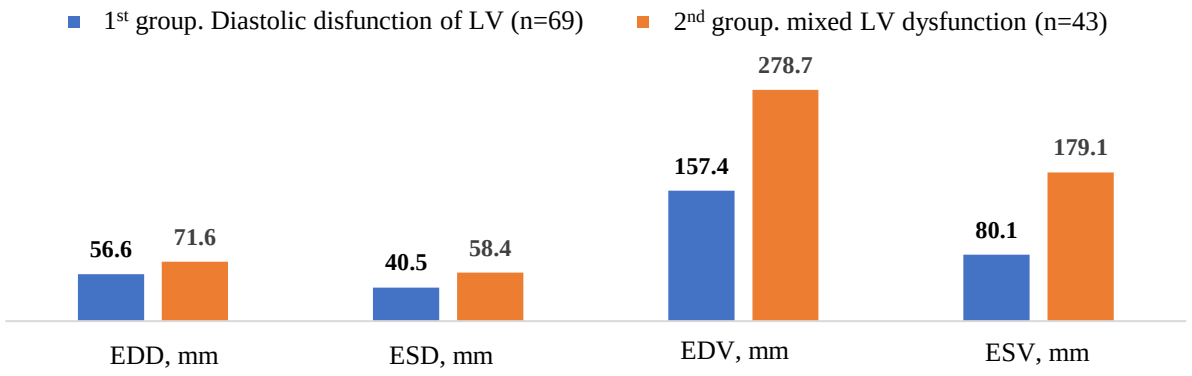


**Figure 3. Structural and functional state of the myocardium (end-diastolic anteroposterior LV size, end-systolic anteroposterior LV dimension, end-diastolic volume, end-systolic volume) in patients with postinfarction CHF, depending on the characteristics of LV myocardial dysfunction.**

Thus, in patients of the first group with LV diastolic dysfunction, this indicator exceeded the normal values, and with the development of mixed LV dysfunction as a result of remodeling, this indicator naturally increased.

In patients with mixed LV dysfunction against the background of worsening clinical symptoms of CHF, there was a decrease in LV EF, and averaged 35.1 ± 5.34%. In patients with LV diastolic dysfunction, the decrease in LV EF was insignificant (52.9 ± 7.26%), and in most patients of this group it was within normal limits, despite the presence of clinical symptoms of CHF (Figure 4).

Also, with mixed LV diastolic dysfunction, myocardial mass (MM) with mixed dysfunction is 287.4 g, and with systolic dysfunction, the indicator is better and less than 238.1 g, and the wall thickness of the left ventricle with mixed dysfunction (UTS) is - 0, 36 mm and with systolic dysfunction - 0.46 mm, respectively (Figure 4).

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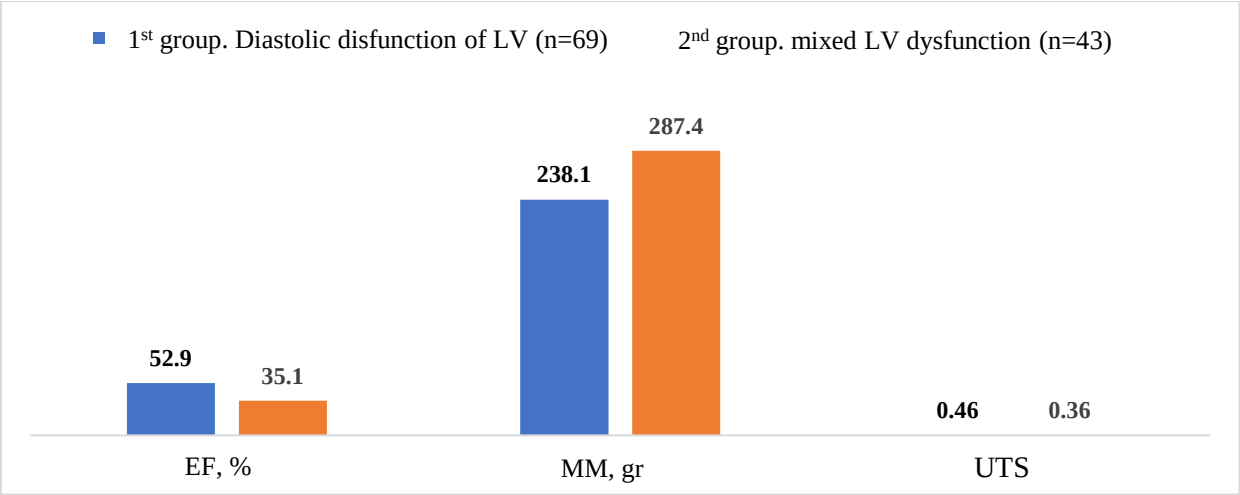


**Figure 4. Structural and functional state of the myocardium (ejection fraction, myocardial mass, left ventricular wall thickness) in patients with postinfarction CHF, depending on the characteristics of LV myocardial dysfunction**

Thus, it can be noted that as LV dysfunction develops, as a result of its remodeling, we noted a tendency to an increase in myocardial mass. Also, in patients with mixed dysfunction, there was a decrease in the relative LV wall thickness index, which indicates the development of eccentric left ventricular hypertrophy and the progression of remodeling with the development of mixed LV dysfunction, the severity of which increases as the functional class of CHF decreases.

Clinical symptoms of CHF are not directly related to left ventricular dysfunction, and activation of compensatory mechanisms leads to activation of the sympathetic nervous system, renin-angiotensin-aldosterone system, as well as increased synthesis of pro-inflammatory cytokines.

In our study, we noted a weak correlation between LV EF and clinical signs of CHF, so we calculated the EF/MS index, which reflects the degree of adequacy of the global systolic function of the heart to the test load with a given LV geometry. A significant decrease in this indicator was revealed as CHF developed and progressed (Table 2, Figure 5).

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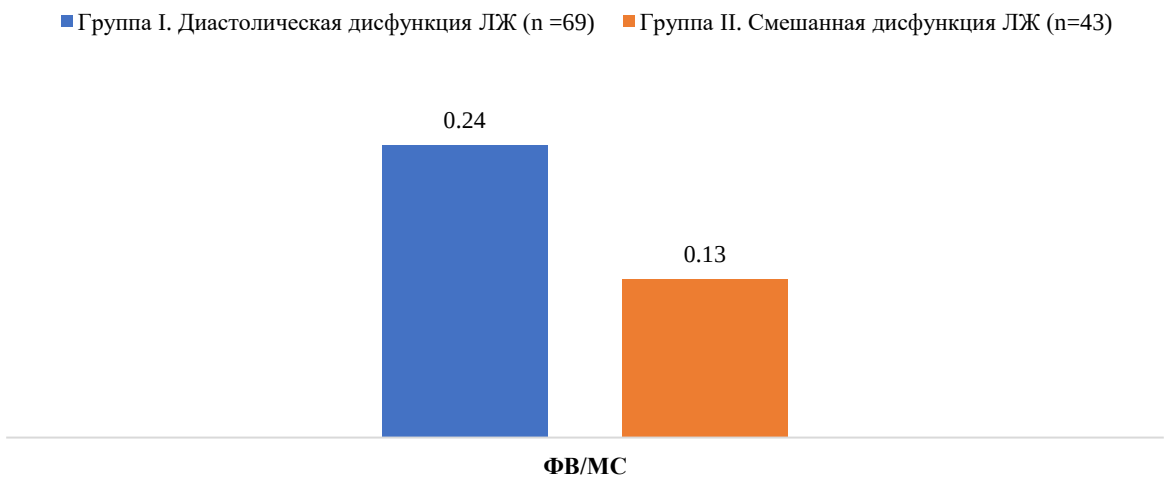


**Figure 5. Dynamics of the EF/MS indicator as CHF develops and progresses.**

Thus, the identified relationships characterize the commonality of the processes of remodeling and the development of LV myocardial dysfunction, occurring against the background of an increase in the level of proinflammatory cytokines in the blood serum and LV remodeling in patients with postinfarction heart failure.

## CONCLUSIONS

As a result of this study, the following conclusion can be drawn: the process of LV remodeling in patients with postinfarction heart failure is directly related to immune activation. We have noted that the development of mixed systolic-diastolic (mixed) myocardial dysfunction as a result of LV remodeling is accompanied by a sharp rise in the level of pro-inflammatory cytokines and highly sensitive serum C-reactive protein.

Therefore, when developing a modified comprehensive program for the treatment of patients with postinfarction heart failure, we paid special attention to its complex multicomponent pathogenesis, functional and immuno-inflammatory bases for the development and progression of myocardial remodeling and the development of myocardial dysfunction in order to stop the mechanisms of the above effects on the progression of LV myocardial dysfunction, patients who have had AMI.

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