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DYNAMICS OF INDICATORS OF STRUCTURAL AND FUNCTIONAL STATE OF THE HEART IN THE POST-INFARCTION PERIOD DEPENDING ON THE PRESENCE OF DECOMPENSATED HEART FAILURE

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Abstract. Echocardiographic indicators in decompensated heart failure demonstrate a high prevalence of structural and hemodynamic abnormalities. In the patients with decompensated heart failure in the early and late post-infarction period, echocardiographic indicators were found to significantly differ from those recorded in the patients without heart failure. Thus, reduced ejection fraction, increased end-diastolic volume and end-diastolic dimension indicate left ventricular systolic dysfunction and left ventricular cavity dilation. Since echocardiography of the heart and the pulmonary artery enables real-time estimation of cardiac filling, signs of systolic dysfunction and pulmonary congestion, it may serve as a predictor of decompensated heart failure development in the early and late post-infarction period.

The objective of the research was to determine the main echocardiographic indicators in terms of rapid monitoring of deterioration in the main parameters of left ventricular overload for early diagnosis of decompensated heart failure, as well as structural and geometric remodeling of left ventricular myocardium in the early and late post-infarction period.

Materials and methods. There were examined 160 patients with acute myocardial infarction. Depending on the development of decompensated heart failure in the early and late post-infarction period, the patients were divided into two subgroups being homogeneous by age and gender.

Results. The results obtained indicated significant hemodynamic changes in the patients with decompensated heart failure in the post-infarction period. They included significantly lower values of ejection fraction indicating left ventricular systolic dysfunction and the signs of left ventricular cavity dilation as evidenced by the increase in left ventricular end-diastolic volume and end-diastolic dimension. The tendency for an increase in left ventricular posterior wall thickness and interventricular septal thickness, as well as left ventricular myocardial mass, left ventricular myocardial mass index and left ventricular radius to wall thickness ratio indicated concentric left ventricular remodeling.

Conclusions. Modern management of patients with decompensated heart failure should be guided by an objective value of left ventricular ejection fraction as it plays a key role in selecting management strategy for this cohort of patients since a significant reduction in this parameter indicates cardiac decompensation. Pulmonary artery pressure and concentric left ventricular hypertrophy play a significant role in cardiac failure development as well.

Keywords: acute myocardial infarction; decompensated heart failure; echocardiographic examination of the heart; left ventricular ejection fraction

Problem statement and analysis of the recent research

Cardiovascular diseases (CVD) are known to account for one-third of all deaths worldwide [3]. According to the American Heart Association, 18% of men and 35% of women with prior acute myocardial infarction (AMI) developed recurrent AMI within 6 years after initial MI; 22% of men and 46% of women were disabled due to the development of chronic heart failure (CHF) [1]. Due to the improvement of the methods for AMI diagnosis and treatment, the mortality rate has significantly decreased. This contributed to the increase in the number of the patients who survived initial and recurrent episodes; thus, the number of the patients with the signs of CHF and its decompensation increased as well [4]. Moreover, as the age of the population increased, treatment of acute coronary syndrome and comorbidities improved, epidemiology of HF changed significantly as well [6]. HF is a global pandemic; its prevalence is constantly increasing worldwide [16]. The patients with acute or decompensated HF represent a heterogeneous group of patients with various causes of the development or exacerbation of this disease and hemodynamic phenotypes [14, 9]. Screening and risk stratification for congestive HF are the most common and urgent problem of modern cardiological society [5]. Echocardiography plays a significant role in diagnosing HF both in practice and multicenter clinical trials [13]. Despite this fact, echocardiographic study being used in phase II and phase III HF clinical trials, is not used in clinical practice. For example, some multicenter clinical trials considered left ventricular (LV) ejection fraction (EF) as a single structural indicator of HF prediction [7]. Therefore, it is unclear whether echocardiography, as an examination method, can serve as a tool for classifying and determining the risk of CHF decompensation in the post-infarction period. Echocardiographic indicators in the patients with acute and decompensated HF cannot be objectively described due to countless causes and pathophysiological mechanisms being involved in the formation of this syndrome; moreover, nowadays there is no strict definition of acute HF [10]. Clinical symptoms and exercise intolerance are limited predictors of LV systolic dysfunction [8]. According to current guidelines on HF management, daily assessment of the signs and symptoms, fluid balance, vital functions, body weight and renal dysfunction should be carried out in patients hospitalized due to HF decompensation to select decongestive therapy, since natriuretic peptide determination turned out to be ineffective for routine treatment of the patients with prior acute HF [15]. The degree of LV remodeling is an important prognostic factor in the patients with decompensated HF and reduced LVEF [11]. Since MI is the most frequent trigger causative for the development of LV remodeling, these patients require continuous and careful clinical and instrumental monitoring to provide adequate treatment and prevent CHF development including its decompensation in the post-infarction period [2]. Cardiac remodeling is the best term to describe the process able to modify the molecules and genes within the cell, as well

as the extracellular matrix that represents the formation of HF syndrome [12].

The objective of the research was to determine the main echocardiographic indicators in terms of rapid monitoring of deterioration in the main parameters of LV overload for early diagnosis of decompensated HF, as well as structural and geometric remodeling of LV myocardium in the early and late post-infarction period.

Materials and methods

The study was carried out in the infraction department No 2 of the Ivano-Frankivsk Regional Clinical Cardiology Dispensary and the Department of Internal Medicine No 2 and Nursing of the Ivano-Frankivsk National Medical University. There were examined 160 patients with prior MI; the average age was (56.67±5.72) years. All the patients were divided into 2 groups depending on the presence of signs of stage II A-B decompensated chronic HF according to the classification by Vasylenko V.Kh. and Strazhesko M.D., the New York Heart Association (NYHA) functional class (FC) III-IV. Group I (the main group) included 120 patients with prior Q-QS wave MI or non-Q wave MI; Group II (the control group) comprised 40 patients with prior MI without any signs of decompensated HF. Inclusion criterion was prior Q-QS wave MI or non-Q wave MI occurred within 28 days before the research started. Study groups were homogenous by age, gender, disease severity, duration of the post-infarction period, clinical signs of decompensation. Clinical diagnosis was made in accordance with the European Society of Cardiology Guidelines, on the basis of anamnestic data, physical examination, laboratory and instrumental findings, namely urine and blood tests, electrocardiography, echocardiography, chest X-ray.

In all the patients, the indicators of remodeling (architectonic changes and changes in LV function with connective tissue expansion in neurohumoral activation due to post-infarction cardiosclerosis, stunned myocardium, hibernating myocardium, transient ischemia), LV end-diastolic dimension (EDD), LV end-diastolic volume (EDV), LV interventricular septal thickness (IVST) in systole and diastole, LV posterior wall thickness (PVT) in systole and diastole, LV myocardial mass (MM), LV myocardial mass index (MMI), LV radius to wall thickness ratio were assessed. LVEF was also assessed by Simpson's rule, since according to the recommendations of the American College of Cardiology and the American Heart Association, EF findings after AMI are determined as Class I, level of evidence B. Thus, LVEF in the range of 40 - 49% was considered as borderline LV dysfunction; LVEF >50% was defined as preserved LV function; LVEF <40% was considered as reduced LV systolic function

The results obtained were statistically processed by means of an advanced analytics software package STATISTICA-7 and a statistical software package "Microsoft-Excel" using the statistical variation analysis. There were calculated the mean M, the mean squared error r, the standard error of the mean m, the sample size (n). P-value was considered significant at $p<0.05$.

Results and discussion

Echocardiographic indicators indicated significant changes in metric and volumetric indicators of LV and its contractility in the patients with decompensated HF (Table 1). Both LVEDV and end-systolic volume (ESV) were significantly higher as compared to healthy individuals without decompensated HF. This was probably due to the highest indicators of the EDD and end-systolic dimension (ESD) in this group. In the patients with decompensated HF, the EDD was (6.78±0.24) cm, while in the patients without decompensated HF, it was (5.39±0.35) cm ($p_2<0.05$) (normal ranges for LVEDD are (4.54±0.32) cm ($p_1<0.05$)) that resulted in an increase in the EDV to (216.00±9.89) ml. In

Table 1. Echocardiographic indicators in the patients with prior MI depending on the presence of decompensated HF

Indicator	Healthy individuals (n=40)	Patients with prior MI without decompensated HF, (n=40)	Patients with prior MI and decompensated HF, (n=120)
EDV, ml	128.9±10.17	138.9±9.95 p_1? 0.05	216.00±9.89 p_1? 0.5 p_2?0.05
ESV, ml	50.55±9.68	65.87±10.93 p_1? 0.05	122.09±3.76 p_1?0.05 p_2?0.05
EDD, cm	4.54±0.32	5.39±0.35 p_1? 0.05	6.78±0.2 p_1?0.05 p_2?0.05
ESD, cm	3.25±0.34	3.79±0.32 p_1? 0.05	5.09±0.1 p_1?0.05 p_2?0.05
IVSTs, cm	0.85±0.11	1.11±0.17 p_1? 0.05	1.34±0.12 p_1?0.05 p_2?0.05
IVSTd, cm	0.89±0.12	1.13±0.11 p_1? 0.05	1.28±0.1 p_1?0.05 p_2?0.05
LVPVTs, cm	0.92±0.11	1.17±0.13 p_1? 0.05	1.48±0.12 p_1?0.05 p_2?0.05
LVPVTd, cm	0.94±0.12	1.15±0.11 p_1? 0.05	1.36±0.12 p_1?0.05 p_2?0.05
EF, %	60.18±4.21	53.18±5.99 p_1? 0.05	40.57±2.22 p_1?0.05 p_2?0.05
Systolic output, ml	74.75±6.9	68.82±5.72 p_1? 0.05	88.14±4.2 p_1?0.05 p_2?0.05
Pulmonary artery pressure, mm Hg	19.06±3.83	28.1±4.3 p_1? 0.05	37.88±4.04 p_1?0.05 p_2?0.05

Notes: statistically significant difference in the indicators:
p_1- as compared to healthy individuals;
p_2 - as compared to the patients without decompensated HF

the patients without decompensated HF, the EDV was (138.9±9.95) ml ($p_2<0.05$), while in healthy individuals, it was (128.9±10.17) ml, respectively ($p_1<0.05$).

Changes in the indicators of LVPVT and IVST were ambiguous. In the patients with decompensated HF, LVPVTd was (1.36±0.12) cm ($p_1<0.05$); in the patients without decompensated HF, it was (1.15±0.11) cm ($p_2<0.05$); in healthy individuals, LVPVTd was (0.94±0.12) cm, respectively ($p_1<0.05$). In the patients with decompensated HF, IVSTd was (1.28±0.1) cm ($p_1<0.05$); in the patients without decompensated HF, it was (1.13±0.11) cm ($p_2<0.05$); in healthy individuals, IVSTd was (0.89±0.12) cm, respectively ($p_1<0.05$). In the patients with decompensated HF, EF was (40.57±2.22) %, that was significantly lower ($p_1<0.05$) as compared to the patients with prior MI without HF (53.18±5.99) % ($p_2<0.05$) and healthy individuals (60.18±4.21) % ($p_1<0.05$). The reduction in EF was combined with the increase in S-wave amplitude and pulmonary artery pressure - (88.14±4.2) mm Hg ($p_1<0.05$) and (37.88±4.04) mm Hg ($p_1<0.05$).

In the patients with decompensated HF, LVMMI was (234.76±7.14) g/m^2 ($p_2<0.05$); in the patients without

Table 2. Indicators of LVMMI, LVMM and LV radius to wall thickness ratio in the patients with prior MI depending on the presence of decompensated HF

Indicator	Healthy individuals (n=40)	Patients with prior MI without decompensated HF, (n=40)	Patients with prior MI and decompensated HF, (n=120)
LVMMI, g/m^2	76.41±10.88	128.98±8.4 p_1?0.05	234.76±7.14 p_1?0.05 p_2?0.05
LVMM, g	144.76±9.97	248.63±6.63 p_1?0.05	447.62±4.87 p_1?0..05 p_2?0.05
Radius to wall thickness ratio, c.u.	0.39±0.04	0.42±0.05 p_1?0.05	0.47±0.04 p_1?0.05 p_2?0.05

Notes: statistically significant difference in the indicators:
p_1- as compared to healthy individuals;
p_2 - as compared to the patients without decompensated HF

decompensated HF, it was (128.98±8.4) g/m^2 (p_1<0.05); normal ranges for LVMMI are (76.41±10.88) g/m^2 (p_1<0.05) (Table 2). In the patients with decompensated HF, LVMM was (447.62±4.87) g that was significantly higher (p_2<0.05) as compared to the patients with prior MI without CF (248.63±6.63) g (p_1<0.05) and healthy individuals (144.76±9.97) g (p_1<0.05). According to the indicators obtained, the radius to wall thickness ratio was significantly higher (p_2<0.05) in the patients with decompensated HF - (0.47±0.04) c.u. as compared to the patients without CF - (0.42±0.05) c.u. (p_1<0.05) and healthy individuals - (0.39±0.04) c.u. (p_1<0.05).

Among the hemodynamic indicators in the patients with decompensated HF, a significant reduction in EF and EDV, a tendency towards increase in IVST and LVPVT, as indicated by LV systolic dysfunction, as well as myocardial hypertrophy and remodeling, significant pulmonary hypertension were observed.

Conclusions

1. The indicator of EF plays a key role in selecting management strategy for the patients with CF, since a significant reduction in this parameter indicates cardiac decompensation.

2. Modern management of patients with CHF should be guided by an objective value of LF EF, the increase in which should be regarded as a predictor of CHF decompensation in the post-infarction period, even without any clinical signs.

3. The determination of pulmonary artery pressure plays a significant role as well since most patients with decompensated HF present with mixed or passive pulmonary hypertension due to vasoreactive response to passive increase in the pressure.

Prospects for further research

Since the main hemodynamic parameters reflecting the development of HF decompensation in patients with prior AMI have been determined, we plan to analyze their changes in the course of decongestive therapy.

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