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GECİKMİŞ POSTİNFARKT DÖVRDƏ ÜRƏYİN REMODELLƏŞMƏSİNİN XÜSUSİYYƏTLƏRİ

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Xülasə. *Məqalədə 79 xəstədə kəskin miokard infarktından (MI) sonrakı 12-ci aya qədər ürəkdə baş verən remodelləşmə proseslərinin qiymətləndirilməsi məqsədilə aparılan exokardioqrafiya müayinəsinin nəticələri haqqında məlumatlar təqdim edilir. Xəstələrdən 42 (53,2%) nəfər Q pozitiv (Q+), 37 nəfər (46,8%) Q neqativ (Q-) infarkt keçirmişdir. 12 ay sonra 32 xəstədə xronik ürək çatışmazliği (XÜÇ) aşkar edilmişdir, onlardan 23 (54,8%) nəfər Q (+), 9 (24,3%) nəfərdə isə Q (-) infarkt keçirən xəstə qruplarına aid idi.*

Q (+) MI keçirən və infarktdan sonrakı gec dövrdə XÜÇ olan xəstələrdə sol mədəciyin EDR (mm), ESR (mm), LA (mm), ILS, IS, LVMI (mm. Hg) dəyərləri XÜÇ olmayan və Q (-) MI keçirmiş xəstələrə nisbətən əhəmiyyətli dərəcədə yüksək olmuşdur (p<0,05, p<0,01). Q (-) MI keçirmiş və XÜÇ olmayan xəstələrdə isə EF, ΔS və 2H/D kimi EchoCG parametrlərinin orta dəyəri Q (+) və XÜÇ olan xəstələr qrupuna nisbətən daha yüksək olmuşdur.

Açar sözlər*: kəskin miokard infarktı, exokardioqrafiya, ürəyin gecikmiş postinfarkt remodelləşməsi*

Ключевые слова*: острый инфаркт миокарда, эхокардиография, позднее постинфарктное ремоделирование сердца*

Key words: *acute myocardial infarction, echocardiography, late postinfarction period, cardiac postinfarction remodeling*

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CHARACTERISTICS OF LATE POST-INFARCTION CARDIAC REMODELING

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Summary. *The article presents the results of echocardiography in assessing cardiac remodeling in 79 patients 12 months after acute myocardial infarction (MI). 42 (53.2%) patients had Q-positive (Q+), 37 (46.8%) Q-negative (Q-) myocardial infarction. After 12 months, chronic heart failure (ChHF) was detected in 32 patients, of which 23 (54.8%) as a result of Q(+), and 9 (24.3%) Q(-) infarction.*

In patients who underwent Q (+) MI with ChHF in the late post-infarction period, the values of left ventricular end-diastolic dimension (*EDD),* end-sistolic dimension (*ESD), left atrial dimension (LAD), local contractility index (LCI), sphericity index (SI), left ventricular myocardial mass index (LV MMI,* g/m^2*) were significantly (P<0.05, P<0.01) higher than in patients with Q (-) MI and without ChHF. The average value of such EchoCG parameters as ejection fraction (EF, %), degree of shortening of the anteroposterior dimension of the left ventricle (ΔS, %) and* the ratio of the total wall thickness and LV EDD (*2H/D index) in patients with Q (-) MI was higher than in the group of patients with Q (+) and without ChHF.*

Chronic heart failure (ChHF) is a leading cause of morbidity, hospitalization and mortality in the world [1]. A study conducted by N.R. Jones et al. (2018) showed that in recent decades, the survival rates of patients with CHF have improved and mortality has slightly decreased. In 2010–2019 one-year and five-year survival rates were 89.3% and 59.7%, respectively [2]. Another study conducted by Magnussen C. et al. (2019) showed a five-fold increase in the risk of death in the development of CHF [3].

Ischemic heart disease (IHD) is the major pathophysiological driver of myocardial infarction (MI) and the cause of ChHF [4]. Past myocardial infarction with reduced ejection fraction is a risk factor for chronic heart failure. Expansion of the postinfarction scar and subsequent regional dilatation of the ventricle can cause postinfarction remodeling leading to significant enlargement of the left ventricular (LV) chamber [5, 6].

Adverse postinfarction left ventricular remodeling (LVR) is characterized by an increase in the end-diastolic volume (EDV) >20% or the end-systolic volume (ESV) >15% compared with baseline values [7]. In patients with acute myocardial infarction, mitral insufficiency and diastolic dysfunction are noted in 30-40% of cases, more often due to dilatation of the left ventricular cavity [8].

Ventricular remodeling already occurs within the first hours after cardiomyocyte necrosis and proceeds for several months. This process is characterized by a change in the left ventricular shape and size, and its dysfunction. Early remodeling develops within three months after acute MI; mid-term and late remodeling develop within six and twelve months, respectively [9].

The aim of the study was to determine the quantitative parameters of cardiac remodeling in the late post-infarction period using echocardiography.

Material and methods. A retrospective analysis of the results of echocardiography (EchoCG) was carried out in 79 patients who had a myocardial infarction of the left ventricle 12 months ago. Among those surveyed, 42 had a Q-positive infarction and 37 had a Q-negative one. According to the results of clinical and instrumental studies among patients with Q (+) myocardial infarction, chronic heart failure (ChHF) was observed in 23 (54.8±7.7%) patients, and with Q (-) myocardial infarction in 9 (24 .3±7.1%) of patients ($P<0.01$). The age of patients varied within 35-69 years, averaging 46±5 years for patients with Q (+) and 54±7 years for patients with Q (-) infarction. Among the examined men there were 47 (59.5%), women 32 (40.5%).

EchoCG in B, M and Doppler modes was performed using a Philips HD 11 device. The linear dimensions of the heart chambers were determined, global and local contractility of the left ventricle (LV), diastolic transmitral and transpulmonary blood flow were assessed.

To assess the differences in quantitative indicators between the compared groups, Student's t-test was used. Differences were considered significant at $p < 0.05$.

Results and discussion. We compared EchoCG parameters in patients with Q (+) and Q (-) myocardial infarction at 3-4 weeks (baseline) and 12 months after its onset. In M and B modes, the following EchoCG parameters were determined: LV end-diastolic dimension (EDD, mm), LV end-systolic dimension (ESD, mm), left atrial dimension (LAD, mm), ejection fraction (EF, %), left ventricular anteroposterior dimension shortening degree (ΔS, %), local contractility index (LCI), sphericity index (SI - the ratio of transverse and longitudinal dimensions of the left ventricle), 2H/D index (the ratio of the total wall thickness and LV EDD), left ventricular myocardial mass index (LV MMI g/m^2) (Table 1).

As can be seen from the table 1, in patients who underwent Q (+) myocardial infarction in the late postinfarction period were recorded an increase in EDD, ESD, LAD, LCI, SI, LV MMI, and a decrease in EF, ΔS, 2H/D. This was due to dilatation of the left heart and indicated a decrease in global and local LV contractility. At the same time, a significant ($P<0.05$) difference between the indicators of the early and late postinfarction period was noted only in the value of ΔS (26.5%±1.1 versus 23.1±1.1%). Such dynamics of most EchoCG parameters was noted in patients with Q (-) MI, but with a minimum value. With Q (+) MI, the negative dynamics of EchoCG parameters in the late postinfarction period compared with the initial data was more significant than with Q (-) MI. This difference was also reflected in clinical symptoms, as CHF was observed more often in the group with Q (+) MI.

Table 1. **Dynamics of EchoCG parameters of LV intracardiac hemodynamics in the early and late postinfarction period, taking into account the nature of the lesion**

EchoCG parameters	Q (+) MI		Q (-) MI	
	Outcome	After 12 mouths	Outcome	After 12 mouths
LV EDD, mm	57.4±2.4	60.9±2.6	55.2±2.4	55.8±2.5
LV ESD, mm	42,2±2,0	46,8±2,5	39.5±1.9	39,9±2.1
LAD, mm	38.7±2.2	41.6±2.4	36.5±2.3	37.9±2.2
LV EF, %	50,6±2,1	48,2±2,2	52.4±2.0	50.5±2.1
LV ΔS, %	26.6±1.1	28.4±1.5	23.2±1.1	28.8±1.2
LV LCI	1.31±0.06	1.32±0.06	1.19±0.05	1.20±0.05
LV SI	0.59±0.03	0.60±0.03	0.56±0.04	0.57±0.04
LV 2H/D	0.33±0.02	0.34±0.03	0.32±0.02	0.34±0.03
LV MMI g/m^2	127.4±11.9	144.7±11,3	121.8±10.6	134.6±11.4

Note: LV EDD – left ventricular end-diastolic dimension, LV ESD – left ventricular end-systolic dimension, LAD – left atrial dimension, EF – ejection fraction, ΔS – left ventricular anteroposterior dimension shortening degree, LCI – local contractility index, SI – sphericity index (the ratio of transverse and longitudinal dimensions of the left ventricle), 2H/D index – the ratio of the total wall thickness, LV MMI – left ventricular myocardial mass index.

Table 2 compared EchoCG parameters of patients depending on the presence of ChHF between the group of patients with Q (+) and Q (-) MI only in the late postinfarction period. As can be seen from the table 2, in both groups with and without ChHF, such parameters as EDD, ESD, LAD, LCI, SI, LV MMI were higher among patients with Q (+) of MI than with Q (-) of MI, and EF, ΔS, 2H/D, on the contrary, with Q (-) of MI were greater than among patients with Q (+) of MI. The highest EDD value was recorded among patients who underwent Q (+) MI with ChHF and amounted to 64.9±2.7 mm, which is significantly (P<0.05 and P<0.001) more than in patients without ChHF, both with Q wave (56.8±2.4 mm), and without a Q wave (52.5±2.4 mm), respectively. EF in patients with ChHF and Q (+) MI was significantly lower (P<0.01) than in patients without ChHF and averaged 43.4±2.1%. EchoCG parameter ΔS among patients with Q (+) MI most significantly (P<0.001) differed from the corresponding parameter in patients with Q (-) MI.

Table 2. **Dynamics of EchoCG parameters of LV hemodynamics in the late post-infarction period, taking into account the presence of ChHF and the nature of the lesion**

EchoCG parametrs	ChHF (+) n = 32		ChHF (-) n = 47	
	Q (+), n = 23	Q (-), n = 9	Q (+), n = 19	Q (-), n = 28
LV EDD, mm	*64.9±2.7* *P2<0,05* *P3<0,001*	59.1±2.6	*56.8±2.4*	52,5±2.4
LV ESD, mm	49.7±2.8 *P3<0,001*	43,5±2.3	43.9±2.4	36.2±1.9
LAD, mm	*45.1±2.6* *P2<0,05* *P3<0,05*	39.6±2.1	*38.1±2.2*	36.1±2.3
LV EF, %	43.4±2.1 *P2<0,01* *P3<0,01*	47.1±2.1	53.1±2.3	53.9±2.4
LV ΔS, %	23.5±1.2 *P3<0,001*	26.4±1.3	22.9±0.9 P1<0,001	31.2±1.6
LV LCI	1.43±0.07 P1<0,05 *P2<0,01* *P3<0,01*	1.26±0.05	1.19±0.05	1.14±0.04
LV SI	0.63±0.05	0.60±0.03	0.56±0.04	0.52±0.06
LV 2H/D	0.30±0.02	0.32±0.03	0.34±0.03	0.36±0.05
LV MMI g/m ^{2}	161.9±12.4 *P2<0,05* *P3<0,05*	145.7±12.3	126.8±10.3	123,5±10.4

Note: P1 is the difference between subgroups of patients with Q (+) and Q (-) within the ChHF group; P2 - difference between groups with ChHF and without ChHF within subgroups with Q wave; P3 - difference between subgroups of patients with Q (+) and Q (-) taking into account the presence of ChHF.

Table 3. **Dynamics of dopplerometric parameters of LV hemodynamics in the late post-infarction period, taking into account the presence of ChHF and the nature of the lesion**

The Doopler parameters	ChHF (+) n = 32		ChHF (-) n = 47	
	Q (+), n = 23	Q (-), n = 9	Q (+), n = 19	Q (-), n = 28
	1	2	3	4
Mitral regurgation (n = 28)	21 (91.3±5.9%) P 1-2<0,01 P 1-3<0,001	4 (44.4±16.6%)	3 (15.8±8.4%)	-
Hypertrophic type of mitral diastolic flow (n = 18)	11 (47.8±10.4%) P 1-3<0,05	5 (55.6±16.6%) P 2-3<0,001	2 (10.5±7.0%)	-
The mitral diastolic flow by pseudo-normalization type (n = 13)	12 (52.2±10.4%) P 1-2<0,01	1 (11.1±10.5%)	-	-
PASP mmHg	41.4±4.7 P 1-3<0,001 P 1-4<0,001	31.6±3.5 P 2-3<0,05 P 2-4<0,01	19.3±3.2	16.8±3.1

Note: PASP – pulmonary artery systolic pressure

Table 3 shows the frequency of mitral regurgitation and diastolic dysfunction, as well as the value of systolic pressure in the pulmonary artery (PA SP, mmHg) depending on the presence of ChHF and the type of myocardial damage in the late postinfarction period. Mitral regurgitation among patients with ChHF and Q wave was noted in 21 (91.3±5.9%) cases, in the subgroup with ChHF and without Q wave - in 4 (44.4±16.6%), in the subgroup without ChHF, but with Q wave - in 3 (15.8±8.4%) cases, respectively (P<0.01 and P<0.001). Diastolic dysfunction of the left ventricle in 18 cases was recorded as a hypertrophic type and in 13 cases as a pseudo-normalization. Diastolic dysfunction of the left ventricle according to the type of pseudo-normalization of mitral diastolic flow was significantly (P<0.01) more often recorded in patients with ChHF and Q (+) MI. PASP was also the highest among patients with Q (+) MI and ChHF (41.4±4.7 mmHg), which was significantly (P<0.001) higher than in the group of patients without ChHF (Fig. 1-4).

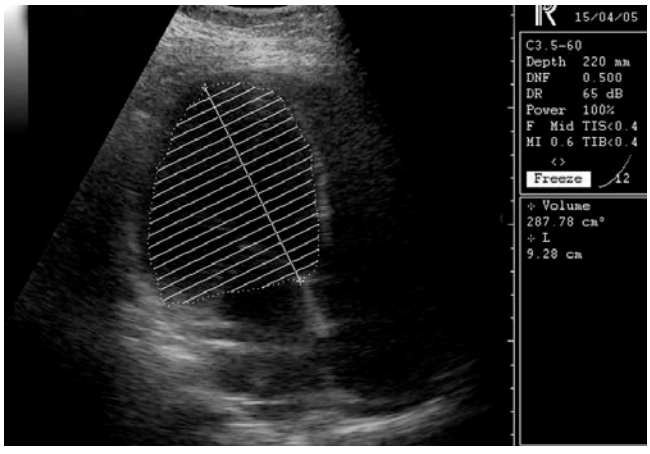

Fig. 1. 4-chamber apical view of the heart in a patient one year after suffering Q (+) anterior myocardial infarction. Significant dilatation of the cavity of the left ventricle. The sphericity index of the left ventricle is 0.81.

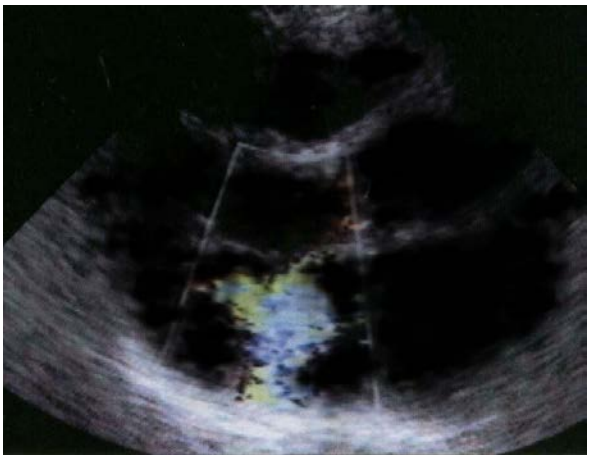

Fig. 2. Parasternal long axis view of the left ventricle in a patient one year after Q (+) posterior myocardial infarction. Mitral regurgitation is recorded - in the cavity of the left atrium, a blue-colored flow is determined at the time of left ventricular systole.

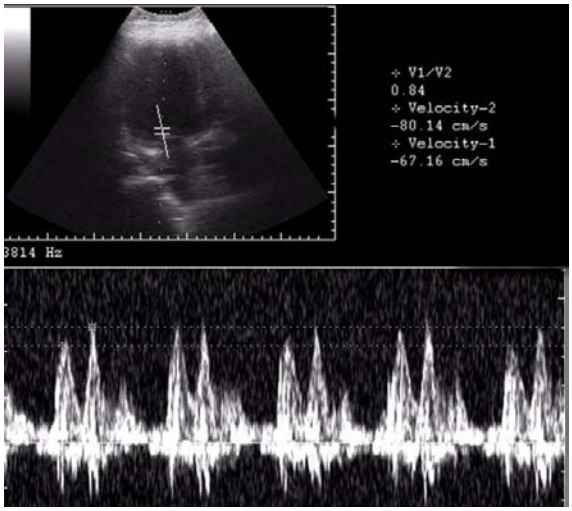

Fig. 3. 4-chamber apical view of the heart in a patient one year after Q (+) myocardial infarction of anterior apical localization. Registration of diastolic mitral flow (E/A - 0.84) by hypertrophic type.

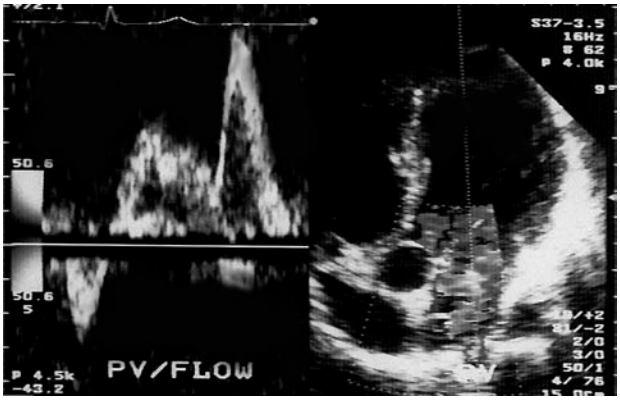

Fig. 4. Registration of blood flow in the pulmonary veins in a patient after Q (+) myocardial infarction 12 months ago. The systolic velocity is less than the early diastolic velocity, and an increased reverse blood flow is also recorded. This is a sign of pseudo-normalization of the diastolic mitral flow, which looks "normal".

Discussion The development of CHF post-MI is driven by the complex pathophysiological mechanisms underlying cardiac remodeling: an inflammatory reaction in the area of myocardial necrosis, isolation of intracellular signaling proteins, activation of neurohumoral systems, followed by the development of hypertrophy and cardiac dilatation, and the formation of a connective tissue scar. The structural and functional remodeling of LV was followed by a decrease in its contractile function leading to impairment of hemodynamics in organs and tissues [10]. The adverse cardiac remodeling post-MI leads to CHF development, associated with increased re-hospitalization rate, disability, and mortality of patients [11].

The functioning of the cardiovascular system as a whole is determined by the adequacy of the interaction between the heart and the arterial system during the ejection of blood from the LV. The predictive significance of diastolic mitral flow has been demonstrated in a number of studies. In particular, in patients with ischemic cardiomyopathy, the ratio Ea/Ees < 1.47 was characterized by better survival rate compared to those whose indicator exceeded the specified threshold value [12, 13].

In the postinfarction period, left ventricular (LV) remodeling is based on the replacement of a significant part of dead cardiomyocytes with fibrous tissue and hypertrophy of intact myocytes with a change in their relative position, which leads to dilatation of the LV cavity and its geometric shape. These postinfarction changes in the LV myocardium contribute to the development of chronic heart failure. Ventricular remodeling includes dilatation of the ventricle, the formation of scar, and geometrical changes in the overall left ventricle shape. i.e., ellipsoid to more spherical [14].

Over time, the expansion of the LV cavity causes an increase in wall tension, which leads to an increase in end-systolic and end-diastolic volumes of the left ventricle, increase myocardial oxygen demand, and ultimately may increase the risk of ischemia. Progressive dilatation leads to further hemodynamic consequences, including the formation of both ischemic and functional mitral regurgitation, which were discussed earlier. As LV preload increases without the subsequent ability to generate sufficient myocardial contractility, LV end-systolic volume increases and ejection fraction decreases [15].

Our studies have shown that diastolic dysfunction of the left ventricle, especially mitral flow by the type of pseudo-normalization, is significantly more often observed in patients with Q-positive infarction and is accompanied by the development of chronic heart failure.

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ОСОБЕННОСТИ ПОЗДНЕГО ПОСТИНФАРКТНОГО РЕМОДЕЛИРОВАНИЯ СЕРДЦА

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Резюме. В статье представлены результаты эхокардиографии, проведенного с целью оценить ремоделирования сердца у 79 больных через 12 мес. после острого инфаркта миокарда (ИМ). 42 (53,2%) больных перенес Q-положительный (Q+), 37 (46,8%) Q-отрицательный (Q-) инфаркт миокарда. Через 12 мес. хроническая сердечная недостаточность (ХСН) выявлена у 32 больных, из них у 23 (54,8%) в результате Q (+), а у 9 (24,3%) Q (-) инфаркта миокарда.

У пациентов перенесших Q (+) ИМ с наличием ХСН в позднем постинфарктном периоде величина КДР, КСР, ЛП, ИЛС, ИС, ИММлж была достоверно ($p<0,05$, $p<0,01$) выше, чем у пациентов с Q (-) ИМ и без ХСН. редняя величина таких ЭхоКГ параметров, как ФВ, ΔS и 2H/D у пациентов с Q (-) ИМ были выше, чем в группе пациентов с Q (+) и без ХСН.

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