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Early predictors of heart failure progression in patients after myocardial infarction

Aim	To identify early predictors for progression of chronic heart failure (CHF) in patients with ST-segment elevation myocardial infarction (STEMI).
Material and methods	The study included 113 patients with STEMI aged 52 (95% confidence interval, 36 to 65) years. 24-h ECG monitoring was performed with assessment of ventricular late potentials, QT dispersion, heart rhythm turbulence (HRT), and heart rhythm variability (HRV); XStrain 2D echocardiograpy with determination of volumetric parameters, myocardial strain characteristics and velocities; and measurement of brain natriuretic peptide (BNP) concentrations. The endpoint was CHF progression during 48 weeks of follow-up, which was observed in 26 (23%) patients. Based on the outcome, two groups were isolated, with CHF progression (Prg) (26 (23%)) and with a relatively stable CHF postinfarction course (Stb) (87 (77%)).
Results	At 12 weeks following MI, the Prg group showed increases in left ventricular (LV) end-diastolic dimension (EDD) ($p<0.05$) and end-diastolic and end-systolic volumes (EDV, ESV), ($p<0.01$), and EDV and ESV indexes (EDVi and ESVi, $p<0.01$). In this group, global longitudinal strain (GLS) was decreased at 24 weeks ($p<0.05$) and global radial strain (GRS) was decreased at 48 weeks ($p=0.0003$). In the Prg group, values of strain parameters (GLS, global circular strain (GCS), and GRS) were lower at all times. At 7–9 days, 24 weeks, and 48 weeks, the proportion of patients with pathological HRT was higher in the Prg group (38, 27, and 19% for the Prg group vs 14% ($p=0.006$); 3,4% ($p=0.001$), and 2.3% ($p=0.002$) for the Stb group, respectively). Only in the Stb group, increases in HRV were observed (SDNNi by 13% ($p=0.001$), rMSSD by 24% ($p=0.0002$), TotP by 49% ($p=0.00002$), VLfP by 23% ($p=0.003$), LfP by 22% ($p=0.008$), and HfP by 77% ($p=0.002$). At 7–9 days of MI, the Stb group had greater values of SDANN ($p=0.013$) and HfP ($p=0.01$). CHF progression correlated with abnormal values of turbulence onset (TO), disturbed HRT, increased BNP levels and LV ESD, and low values of GLS, GCS, and GRS. Combined assessment of HRT, LV ESD, and GLS at 7–9 days after STEMI allows identifying patients with high risk for CHF progression in the next 48 weeks.
Conclusion	The markers for CHF progression after STEMI include abnormal TO values, disturbed HRT, increased BNP levels and LV ESD, and low values of GLS, GCS, and GRS. The multifactor logistic regression analysis revealed early predictors of CHF in the postinfarction period, including abnormal TO, increased LV ESD, and reduced GLS.

Keywords	Myocardial infarction; heart failure; heart rhythm turbulence; heart rhythm variability; myocardial strain characteristics
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Introduction

Chronic heart failure (CHF) is a rapidly growing public health problem worldwide. This is mainly due to the increasing life expectancy of patients with cardiac pathology. A key factor in the development of CHF is coronary heart disease (CHD) as well as arterial hypertension [1, 2]. The contribution of the history of myocardial infarction (MI) to the etiology of CHF has increased significantly in the past decade. The use of up-to-date medical technologies in patients with MI has allowed for a reduction in mortality and increased life expectancy. However, the higher survival rate of patients is associated with a higher risk of developing CHF in the post-infarction period [1, 3].

The development and progression of CHF after MI are quite common and have a direct impact on the quality of life (QoL), incapacitate patients, and lead to substantial financial losses [4]. There is a close correlation between the severity of CHF and mortality of patients with a history of MI. According to the FAST-MI registry, 37.5% of patients with ST-elevation MI (STEMI) show signs of heart failure and are at a higher risk of death within the next

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12 months, 26.6% versus 5.2% [5]. Moreover, the direct and indirect costs of treatment increase as CHF progresses from functional class (FC) I to IV [4, 6].

Ventricular arrhythmias are the leading cause of death of patients with CHF [1, 2]. Thus, it is of particular interest to study the correlation of the key mechanisms responsible for cardiac arrhythmias with myocardial contractility, and the major markers of the development and progression of CHF in post-infarction patients.

Objective

To find early predictors of the development and progression of CHF in patients with a history of STEMI, enabling identification of patients at high risk and to take timely prevention and treatment measures aimed at preventing adverse outcomes.

Material and methods

The open-label, prospective, single-center trial included patients with STEMI admitted into the emergency cardiology department of the Penza Regional Clinical Hospital n.a. N.N. Burdenko. The ethics committee of the University of Penza approved the study protocol and informed consent form. The study complies with the ethical principles of the Declaration of Helsinki. The identification number of the clinical trial at https://register.clinicaltrials.gov is NCT02590653.

Inclusion criteria: age 35–65 years, STEMI regardless of location confirmed by the elevated troponin I levels within the diagnostic range, electrocardiogram (ECG), coronary angiography (CA), echocardiogram.

Exclusion criteria: repeated and recurrent MI; presence of the left coronary artery stenosis >30%, stenosis of other coronary arteries, except for infarct-related arteries, > 50% according to CA; CHF FC III-IV; heart rhythm disorders; and severe concomitant diseases.

The mean age of patients was 52 (95% confidence interval [CI] 36–65) years. Most patients (99 [88%]) were male. Patients were examined on days 7–9 of STEMI and at 12, 24, and 48 weeks after STEMI. Patients completed the Minnesota Living with Heart Failure Questionnaire (MLHFQ). They were surveyed and examined using the Symptomatic Hospital and Outpatient Clinical Score (SHOCS, Mareev's modification, 2000). Since week 12 after MI, a 6-minute walk distance test (6MWD) was used to determine CHF FC.

2D-echocardiogram was performed using a MyLab90 ultrasound scanner (Esaote, Italy), and common volumetric parameters were analyzed. The left ventricular ejection fraction (LVEF) was calculated using the modified Simpson method. The left ventricular end-diastolic (LVEDV) and end-systolic (LVESV) volumes were determined by indexing to body surface area. XStrain™ Esaote software was used for speckle-tracking echocardiography. We estimated peak values at the global longitudinal (GLS), global circular (GCS), global radial (GRS) strain segments, and the corresponding rates GLSR, GCSR, GRSR [7]. Since GLS and GCS are negative values, they are presented as scalar values for easier perception.

On day 7–9, at 24 and 48 weeks, blood levels of brain natriuretic peptide (BNP) were determined using an Olympus AU480 device.

12-channel Holter ECG monitoring was carried out on day 7–9, 24 and at 48 weeks using the Holter Analysis–Astrocard complex. We evaluated rhythm and conduction disorders and ischemia events. Based on the Holter ECG monitoring findings, heart rate turbulence (HRT) was analyzed, turbulence onset (TO) and turbulence slope (TS) were estimated. Also ventricular late potentials (VLPs), QT dispersion to the T-wave apex (QTa disp), and to T-wave end (QTe disp), heart rate variability (HRV), and circadian heart rate (HR) dynamics were determined [8].

The endpoints were the progression of CHF, determined by one of the following outcomes [1, 2]: hospitalization for acute decompensated CHF; reduced LVEF versus the levels as of day 7–9 and the transition from the preserve EF (HFpEF) group to the mid-range EF (HFmrEF) group or the reduced EF (HFrEF) group, and from the HFmrEF group to the HFrEF group; 6MWD test results corresponding to FC III or IV.

Patients were treated following the Russian guidelines for the management of patients with STEMI [9]. All patients underwent percutaneous coronary intervention (PCI) with stenting within the first 24 hours after the onset of STEMI, preceded by thrombolysis in 68 (60%) patients. Patients received the following treatment: dual antiplatelet therapy and statins – 100%; angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers – 93 (82%); beta-blockers – 86 (76%); diuretics – 21 (19%); calcium channel blockers – 10 (8.8%); and amiodarone – 5 (4.4%).

The data obtained was analyzed using Statistica 7.0 software. Univariate analysis of variance (ANOVA) with the Newman–Keuls test was used to estimate changes in the nominal values of the parameters. The values of all quantitative signs are presented with 95% CI. When comparing qualitative characteristics, the chi-square test was used for unconjugated samples, and the McNemar test was used for pairwise comparisons. Univariate logistic regression analysis with an odds ratio (OR) and 95% CI was used to determine the effects of the parameters on the endpoint development. When the parameters were included in the multivariate model using the Cox multivariate logistic regression model, the absence of

correlation between them was a strict requirement [10]. The threshold of statistical significance was p<0.05.

Results

The study included 125 patients with STEMI. Two patients died during the observation. One patient died on day 16 from myocardial rupture, and the other one died within 10 months from pulmonary edema. Ten patients dropped out of the trial. Four patients moved to other places, while six patients refused further follow-up for various reasons.

A total of 113 (90.4%) patients completed the trial. Their general characteristics are shown in Figure 1. It should be noted that only 20% of them had angina before the index event. The time from pain onset to stenting was very long in real-life practice, since there is only one center with an angiographic operating room.

The endpoint "the progressive course of CHF within 48 weeks of follow-up" was recorded in 26 (23%) patients. 9 (35%) were hospitalized for decompensated CHF. 2 (7.7%) patients had reduced tolerance as shown by 6MWD to FC III. At the same time, 19 (73%) had decreased LV FV with a transition from HFpEF to HFmrEF in 11 (58%) patients, to HFrEF in 4 (21%) patients, and from HFmrEF to HFrEF in 4 (21%) patients.

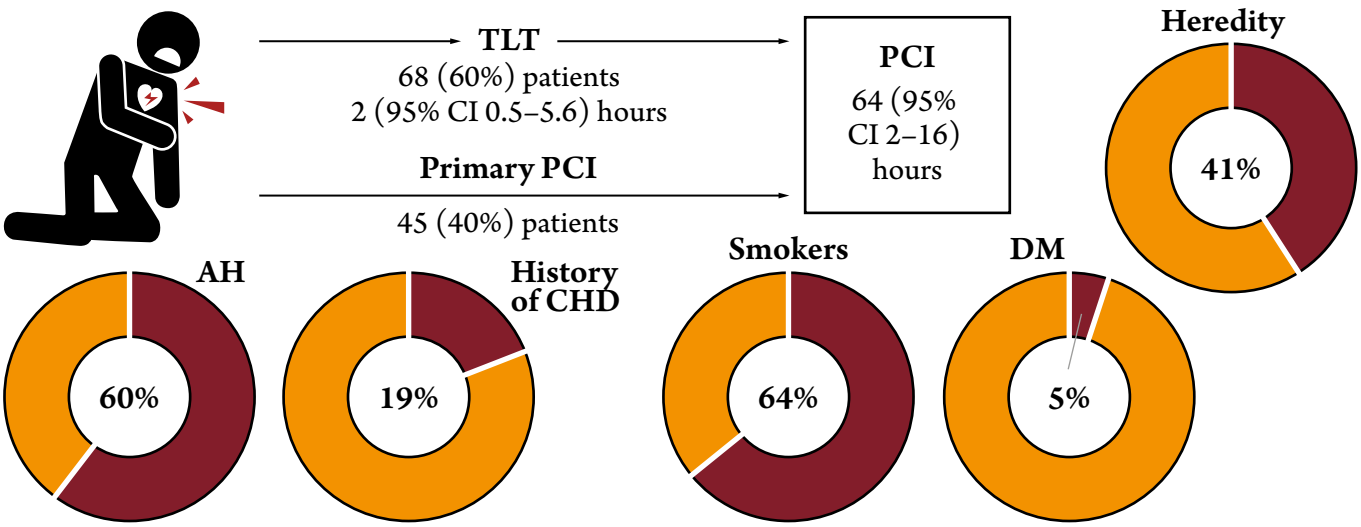
Depending on whether endpoints were achieved or not, two groups were identified: the progressive course of CHF (PC); and the relatively stable course of the post-infarction period (SC). The groups were comparable in several characteristics (Table 1).

In the SC and PC groups, opposite changes of LVEF were observed with a comparable baseline level of on day 7–9: 50% (95% CI 48–52) and 49% (95% CI 47–51), respectively (p=0.313; Figure 2). For example, a decrease in LVEF was observed in the PC group 12 weeks after STEMI (p=0.033). This continued after 24 (p=0.0016) and 48 weeks (p=0.0002). In the SC group, LVEF increased in 12 weeks (p=0.00004), which was the same by the time of completion of follow-up (p=0.0005).

By week 12 after STEMI, most of the volumetric echocardiographic parameters increased: LVEDD from 53.5 (95% CI 51.6–55.4) to 55.7 (95% CI 53.9–57.6; p=0.041) mm; LVEDV from 122 (95% CI 106–133) to 150 (95% CI 128–171; p=0.0006) mL; LVESV from 66 (95% CI 55–78) to 83 (95% CI 68–98) mL; EDVI from 64 (95% CI 55–73) to 76 (95% CI 66–85; p=0.0032) mL/m2; ESVI from 35 (95% CI 28–42) to 46 (95% CI 38–53) mL/m2. These values continued to increase during further follow-up. At the same time, in the SC group, an increase in these parameters versus day 7–9 was established only at 48 weeks: LVEDD from 51.4 (95% CI 50.2–52.6) to 53.2 (95% CI 51.8–54.5; p=0.005) mm, LVEDV from 116 (95% CI 110–123) to 127 (95% CI 119–136; p=0.007) mL and EDVI from 59 (95% CI 56–62) to 64 (95% CI 60–68; p=0.034) mL/m2. Significant intergroup differences of all mentioned LV volumetric characteristics were registered at 12 weeks (p<0.05). It should be noted that the values of these parameters did not differ statistically significantly on day 7–9, except for of LVESD, which was significantly higher in the PC group – 39 (95% CI 34–45) mm compared to the SC group – 33 (95% CI 31–35; p=0.0018) mm.

In the PC group, GLS and GRS decreased significantly at 24 weeks (p<0.05) and at 48 weeks (p=0.0003), respectively (Table 2). Changes were favorable in the comparison group, GLSR increased significantly at 48 weeks (p=0.005). At baseline and later, lower values of all strain parameters were

Figure 1. Characteristics of patients included in the trial



TLT, thrombolytic therapy; PCI, percutaneous coronary intervention; CI, confidence interval; AH, arterial hypertension; CHD, coronary heart disease; DM, diabetes mellitus.

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Table 1. Comparative characteristics of the SC and PC groups

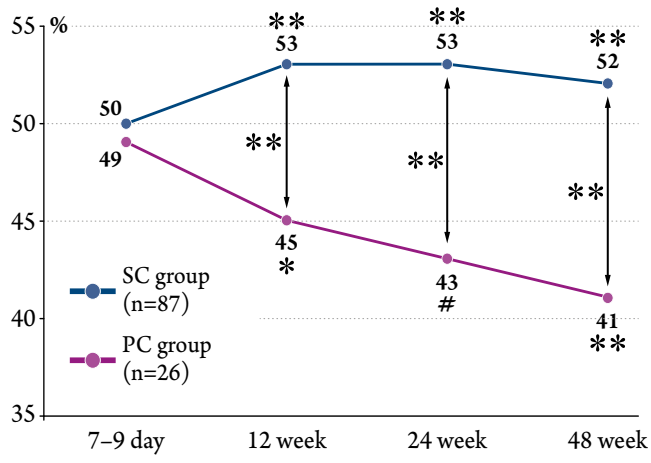
Parameter	SC group (n=87)	PC group (n=26)	p
Age, mean, years	51 (от 49 до 53)	51 (от 48 до 55)	0.982
Male/female, n (%)	77 (89)/10 (11)	22 (85)/4 (15)	0.598
BMI, kg/m^2	28 (от 27 до 29)	28 (от 26 до 29)	0.839
History of CHD, n (%)	18 (21)	3 (12)	0.293
AH, n (%)	55 (63)	13 (50)	0.228
Burdened family history, n (%)	34 (39)	12 (46)	0.520
Smoking, n (%)	57 (66)	15 (58)	0.467
DM type 2, n (%)	5 (5.7)	1 (4)	0.705
Pain-TLT time, h	3.8 (от 2.6 до 5)	2.6 (от 0.6 до 4.6)	0.298
Pain-PCI time, h	8.9 (от 4.5 до 13.3)	6.9 (от 3.7 до 11.2)	0.413
MI of left ventricular anterior/posterior wall, n (%)	47 (54)/40 (46)	17 (65)/9 (35)	0.306
Drug therapy, n (%)			
Dual anti-platelet	87 (100)	26 (100)	1.000
Statins	87 (100)	26 (100)	1.000
ACE inhibitors/ARBs	73 (84)	20 (77)	0.413
Beta-blockers	67 (77)	19 (73)	0.680
Diuretics	17 (20)	4 (15)	0.633
Calcium channel blockers	8 (9.2)	2 (7.7)	0.813
Amiodarone	4 (5.7)	1 (4)	0.871

Values are presented with a 95% confidence interval unless otherwise specified. SC, stable course; PC, progressive course; BMI, body mass index; CHD, coronary heart disease; AH, arterial hypertension; DM, diabetes mellitus; TLT, thrombolytic therapy; PCI, percutaneous coronary intervention; MI, myocardial infarction; ACE, angiotensin-converting enzyme; BRAs, angiotensin II receptor blockers.

registered in the PC group versus the SC group: GLS, GCS, GRS, lower GLSR at 12 weeks (p=0.002), GCSR in 12 (p=0.015), 24 (p=0.002), and at 48 weeks (p=0.009).

The cumulative QoL scores on day 7–9 of STEMI were comparable: 12 (95% CI 9–14) in the SC group and 13 (95% CI 8–20) in the PC group (p=0.253). At 12 weeks, the total scores increased in both groups: 17 (95% CI 14–20; p=0.0008) and 24 (95% CI 18–31; p=0.034), respectively, and at 48 weeks to 16 (95% CI 13–20; p=0.005) in the SC group and 24 (95% CI 17–31; p=0.023) in the PC group. Despite the similar course, after week 12 of the post-infarction period, the PC group had higher scores (p<0.05), which corresponded to decreased QoL in those patients. A similar trend was observed for the SHOCS scores of the subjective assessment by patients of their clinical status in the SC and PC groups. With comparable baseline levels in both groups, the 12-week scores increased and were significantly higher in the PC group (p<0.05). The trend continued, and the scores increased from day 7–9 to week 48 from 1 (95% CI 0.8–1.1) to 1.3 (95% CI 1–1.5; p=0.021) in the SC group and from 1.1 (95% CI 0.6–1.6) to 2 (95% CI 1.4–2.7; p=0.006) in the PC group. This orresponded to the progression of CHF symptoms in the latter group.

Figure 2. Echocardiographic changes in left ventricular ejection fraction in the SC and PC groups



*, p<0.05; #, p<0.01; **, p<0.001 – for the differences from the baseline values. SC, stable course; PC, progressive course.

At 12 weeks after STEMI, 6MWD was significantly less in the PC group: 459 m (95% CI 417–502) versus 502 m (95% CI 481–522; p=0.041). Exercise tolerance increased in the SC group at 24 and 48 weeks: 6MWD was 528 (95% CI 507–549; p=0.004) and 531 (95% CI 513–549; p=0.0035) m, respectively. Exercise tolerance did not change statistically significantly in the PC group: 6MWD was 492 m (95% CI 448–536; p=0.256) at the end of follow-up.

In the SC group, BNP decreased from 126 (95% CI 64–188) pg/mL on days 7–9 of STEMI to 64 (95% CI 26–118) pg/mL at 24 weeks (p=0.007) and to 56 (95% CI 35–77) pg/mL at 48 weeks (p=0.0009). In the PC group, BNP did not change statistically significantly: 192 (95% CI 95 to 289), 145 (95% CI 24 to 266) and 138 (95% CI 36 to 241) pg/mL

Table 2. Comparative characteristic of myocardial strain parameters in the SC and PC groups

Parameter	Group	Day 7–9	Week 12	Week 24	Week 48	p_{1-2}	p_{1-3}	p_{1-4}
		1	2	3	4			
GLS, %	SC	17.4 (от 16.5 до 18.2)*	17 (от 16.1 до 17.8)**	17.2 (от 16.3 до 18.2)#	17.3 (от 16.4 до 18.1)**	0.523	0.905	0.777
	PC	15 (от 13.5 до 16.6)*	13.8 (от 12.2 до 15.3)**	13.8 (от 12.4 до 15.1)#	13.8 (от 12.5 до 15)**	0.140	0.035	0.041
GLSR, %	SC	1.5 (от 1.4 до 1.6)	1.37 (от 1.3 до 1.44)#	1.4 (от 1.35 до 1.48)	2.3 (от 1.5 до 3.2)	0.896	0.769	0.005
	PC	1.31 (от 1.2 до 1.5)	1.2 (от 1.1 до 1.25)#	1.3 (от 1.2 до 1.4)	1.7 (от 0.6 до 2.8)	0.900	0.965	0.294
GCS, %	SC	19.1 (от 17.8 до 20.5)#	19.3 (от 17.8 до 20.8)#	19.3 (от 17.9 до 20.6)#	18.3 (от 17 до 19.6)#	0.829	0.973	0.176
	PC	15.3 (от 13.4 до 17.2)#	15.1 (от 13.4 до 16.9)#	14.9 (от 13.3 до 16.6)#	14.3 (от 12.4 до 16.2)#	0.866	0.930	0.736
GCSR, s^{-1}	SC	1.9 (от 1.6 до 2.3)	1.8 (от 1.6 до 1.9)*	1.7 (от 1.6 до 1.8)#	1.7 (от 1.6 до 1.8)#	0.220	0.338	0.284
	PC	1.5 (от 1.3 до 1.6)	1.4 (от 1.2 до 1.6)*	1.4 (от 1.3 до 1.6)#	1.4 (от 1.2 до 1.6)#	0.983	0.981	0.977
GRS, s^{-1}	SC	32 (от 30 до 34)#	30 (от 28 до 32)#	31 (от 29 до 32)#	30 (от 28 до 32)**	0.271	0.224	0.240
	PC	26 (от 23 до 29)#	24 (от 22 до 27)#	24 (от 22 до 27)#	20 (от 18 до 23)**	0.224	0.163	0.0003
GRSR, s^{-1}	SC	2.8 (от 2.7 до 3)	2.9 (от 2.5 до 3.3)	3.4 (от 2.7 до 4.1)	3.3 (от 2.6 до 4)	0.832	0.366	0.334
	PC	2.6 (от 2.4 до 2.9)	2.6 (от 2.4 до 2.9)	2.7 (от 2.5 до 3)	2.5 (от 2.3 до 2.8)	0.931	0.617	0.803

Values are presented with 95% confidence interval. *, p<0.05; #, p<0.01; **, p<0.001 – for intergroup differences of the parameters.
GLS, global longitudinal strain; GLSR, global longitudinal strain rate; GCS, global circular strain; GCSR, global circular strain rate; GRS, global radial strain; GRSR, global radial strain rate.

at the corresponding follow-up time point. Furthermore, the BNP levels in the PC group was clearly higher than those in the comparison group both at baseline (p=0.028) and later (p=0.038 and p=0.008).

It was of particular interest to estimate changes in vegetative regulation of heart rate on a comparative basis in the study groups based on the Holter ECG data. Both on baseline day 7–9 and in repeated 24- and 48-week examinations, the percentage of patients with abnormal HRT was greater in the PC group (38, 27, and 19%) than in the SC group: 14% (p=0.006), 3.4% (p=0.001); and 2.3% (p=0.002), respectively. These results are explained by the fact that the percentage of patients with an inadequate rapid response to premature ventricular beats (TO) decreased when compared to the baseline levels only in the SC group: from 9.2% to 4.3% at 24 weeks (p=0.012) and 2.3% at 48 weeks (p=0.008). The percentage of patients did not change statistically significantly in the PC group (38, 27 and 19% at the corresponding time points) and remained higher than in the SC group (p<0.001). The number of patients with inadequate TS did not change significantly throughout the follow-up period.

Favorable transformation of the HRV parameters was registered at 24 weeks after STEMI in both patient groups, which reflected the recovery of vegetative regulation of sinus rhythm: increased SDNN (p<0.001), SDANN (p<0.05); TINN (p<0.05), pNN50 (p<0.05), ULfP (p<0.05); and decreased L/H (p<0.01). However, other indicators also increased only in the SC group: SDNi by 13% (p=0.001); rMSSD by 24% (p=0.0002); TotP by 49% (p=0.00002); VLfP by 23% (p=0.003); LfP by 22% (p=0.008); and HfP by 77% (p=0.002).

Differences in HRV between the groups on day 7–9 of STEMI were observed for SDANN and HfP. In the SC group, the values were higher: 98 (95% CI 92–105) ms and 794 (95% CI 639–949) ms2 versus 88 (95% CI 75–101; p=0.013) ms and 288 (95% CI 94–670; p=0.009) ms2 in the PC group, respectively.

Despite statistically significant differences in the status and changes in HRV, the groups did not differ in baseline mean 24-hour HR according to Holter ECG monitoring: 71 (95% CI 69–73) bpm in the SC group and 72 (95% CI 69–75) bpm in the PC group (p=0.462). Later, with the use of beta-blockers and as sympathetic effects were neutralized when compared to the acute period of STEMI in both groups, HR decreased at 24 weeks to 68 (95% CI 67–70) and 68 (95% CI 64–71) bpm, respectively (p<0.05).

Favorable transformation of the markers of electrical myocardial instability, VLPs and QT dispersion, were registered only in the SC group. The duration of low-amplitude potentials at the end of QRS (HFLA) decreased from 28 (95% CI 27–30) ms on day 7–9 to 25 (95% CI 23–27) ms at 48 weeks (p=0.0023), root-mean-square (RMS) voltage of the last 40 ms of QRS from 48 (95% CI 41–54) to 62 (95% CI 52–71) μV (p=0.0014). However, there were no statistically significant changes in the percentages of patients with VLPs within the groups or intergroup differences. In the SC group, the parameters of QT dispersion, QTe disp

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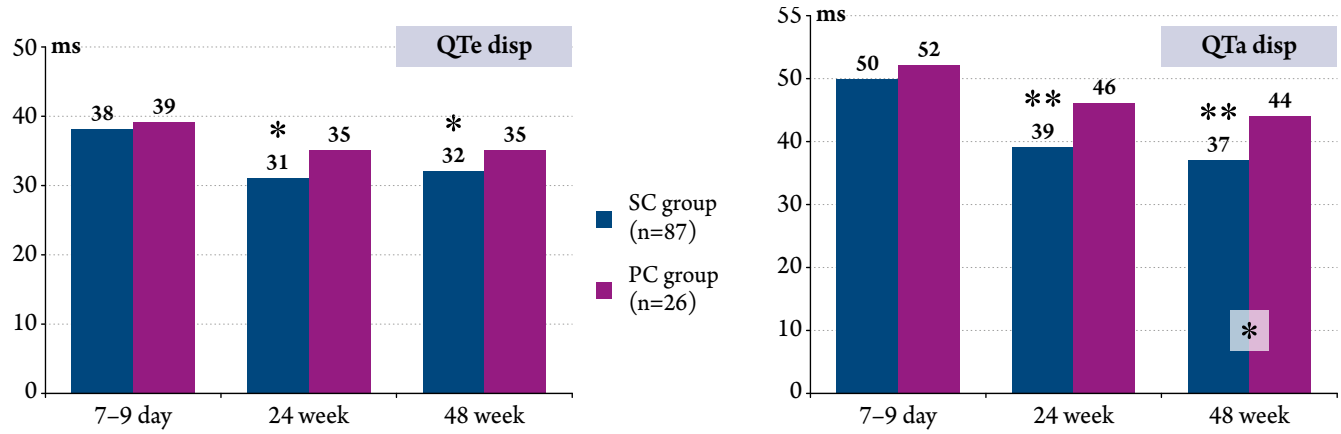
Figure 3. Changes of QT dispersion in the SC and PC groups



* – p<0.05, ** – p<0.001 – for the differences between parameters. QTa disp, QT dispersion to T-wave apex; QTe disp, QT dispersion to T-wave end.

and QTa disp, regressed by week 24 (Figure 3). QTa disp was higher in the PC group than in the comparison group at 48 weeks (p=0.042).

The favorable changes in the markers of electrical myocardial instability in the SC group were combined with a small percentage of patients with life-threatening short runs of ventricular tachycardia: 5% on day 7–9, and 1% at 48 weeks of follow-up (p=0.17). In contrast, the percentage of patients in the PC group increased from 0 to 15% (p=0.038), which was higher than that in the SC group (p=0.01).

In order to determine predictors of the progression of CHF after STEMI, a univariate logistical regression analysis of variables with statistically significant intergroup differences on day 7–9 of STEMI was performed (Table 3). It was established that abnormal TO, disturbed HRT, BNP, LVESD, GLS, GCS, and GRS in the early period, on day 7–9 of STEMI, were CHF progression factors in the post-infarction period.

The correlations were found between TO and HRT, BNP and GLS, GCS and LVESD, GLS. Based on the univariate analysis results, a multivariate logistical regression model of the progression of CHF in patients with STEMI was developed by means of a stepwise selection of correlated variables, including abnormal HRT, LVESD, and GLS estimated on day 7–9 of STEMI (Table 4).

As shown in Table 4, abnormal HRT on day 7–9 of STEMI was associated with a 3.9-fold increase in the risk of CHF progression in the post-infarction period. A 1 mm increase in LVESD improves the likelihood of CHF after STEMI by 4.4%, while a 1.1% increase in GLS is associated with a 10% decrease in the risk of CHF progression.

Discussion

Implementing the vascular disease management program [11] lead to improved management of patients with STEMI. However, PCI is still insufficiently available in real-life clinical practice. Due to geographical remoteness and the lack of angiographic devices in the Russian rural areas, delays in endovascular interventions pose a serious problem. Delayed hospitalizations of patients due to late seeking treatment should also be noted[12]. For example, the pain-to-stent period was 6.4 (95% CI 2–16) hours in this trial, which is significantly higher than reported in some countries [13].

Delayed revascularization in STEMI increases exposure to acute ischemia and, thus, an extension of the necrosis area, which is subsequently associated with LV systolic dysfunction and CHF progression. According to this data, the progressive course of CHF was observed in 23% of patients.This is evidenced by a decrease in LVEF after week 12 of follow-up with increases in all volumetric characteristics (LVEDD, LVEDV, LVESD, LVESV, EDVI, ESVI) at 24 weeks. In the group which demonstrated a relatively favorable course of the disease, a moderate increase in specific parameters (LVEDD, LVEDV, EDVI) was registered only at 48 weeks.

Since the negative changes of LVEF in the post-infarction period were quite naturally used in this work as a criterion for the progressive course of CHF, LVEF values were not included in the multivariate logistic regression analysis.

Patients with a history of MI are at higher risk of developing fatal arrhythmias.This is why implantable cardioverter-defibrillator (ICD) implantation can be indicated to prevent sudden cardiac death (SCD) [14]. However, if the risk of SCD can not be accurately stratified in patients with CHF, ICDs are not always used for those patients who are in the most urgent need [15].

Haugaa et al. [16] showed that a combination of impaired cardiac biomechanics and global strain characteristics could improve the selection of patients with MI for ICD implantation, especially those with LVEF >35%.

An approach combining the assessment of LV volumetric and strain characteristics and the determination of markers of electrical myocardial instability and vegetative rhythm

Table 3. Parameters correlating with the progression of CHF in patients with a history of STEMI

Variable	Regression coefficient (B)	Chi-square test	p	OR (95% CI)
Abnormal TO	1.01	5.62	0.018	2.75 (от 1.191 до 6.326)
Abnormal HRT	0.97	5.5	0.019	2.64 (от 1.17 до 5.92)
SDANN	–0.011	2.49	0.109	0.98 (от 0.97 до 1.0)
HfP	0.00028	1.49	0.221	1.0 (от 0.99 до 1.01)
BNP	0.0011	5.18	0.023	1.001 (от 1.0001 до 1.0002)
SHOCS, score	0.13	0.39	0.534	1.14 (от 0.75 до 1.74)
LVESD	0.039	9.41	0.0022	1.04 (от 1.01 до 1.07)
GLS	–0.11	5.69	0.017	0.89 (от 0.82 до 0.98)
GCS	–0.085	6.6	0.011	0.92 (от 0.86 до 0.98)
GRS	–0.049	6.38	0.012	0.95 (от 0.92 до 0.99)

CHF, chronic heart failure; STEMI, ST-segment elevation myocardial infarction; OR, odds ratio; CI, confidence interval; TO, turbulence onset; HRT, heart rate turbulence; SDANN, standard deviation of 5-minute average of normal R–R intervals; HfP, high-frequency power; BNP, brain natriuretic peptide; SHOCS, Symptomatic Hospital and Outpatient Clinical Score; LVESD, left ventricular end-systolic dimension; GLS, global longitudinal strain; GCS, global circular strain; GRS, global radial strain.

Table 4. Results of the multivariate logistic regression analysis

Variable	Regression coefficient (B)	Chi-square test	p	OR (95% CI)
Abnormal HRT	1.37	9.75	0.0018	3.92 (от 1.66 до 9.25)
LVESD	0.043	8.97	0.0027	1.044 (от 1.015 до 1.07)
GLS	–0.11	4.82	0.028	0.9 (от 0.815 до 0.98)

OR, odds ratio; CI, confidence interval; HRT, heart rate turbulence; LVESD, left ventricular end-systolic dimension; GLS, global longitudinal strain.

regulation in patients with STEMI is likely to improve the accuracy of predicting CHF development and SCD risk. Reduced contractility combined with the arrhythmogenic substrate constitute a critical risk of SCD.

Reduced GLS and LVEF are well-known indicators of the unfavorable course of CHF [17, 18], and are associated with an increase in CHF FC and LV volume [19–21]. Our findings show that lower values of all types of strain were registered in the group with CHF events on day 7–9 of STEMI. Most importantly, the regression of GLS and GRS was observed later in the post-infarction period.

BNP is one of the most commonly used indicators of CHF [2, 22]. We found opposite changes in the groups: BNP decreased by week 24 in a relatively favorable course of the disease, and the marker remained at significantly higher levels in patients with symptoms of CHF progression.

Several trials showed that low HRV is a reliable predictor of the high risk of overall mortality and progression of CHF [23, 24], which was confirmed in this trial. If CHF progressed, post-infarction patients showed a sluggish pattern of growth of specific HRV parameters, with the positive evolution of vegetative effects on the sinus rhythm in the stable course group.

HRT is an up-and-coming method of preventing the risk of developing CHF after MI [23]. Cygankiewicz [25] showed HRT, LVEF, and CHF to be correlated. According to our findings, the percentage of patients with impaired baroreflex sensitivity of patients with clinically relevant progression of CHF remained consistently high, when compared to the comparison group. This indicated an imbalance in the vegetative regulation of the sinus rhythm.

Thus, we have proposed a multivariate model for predicting the development and progression of CHF in patients with a history of STEMI. This includes the LV volumetric and strain characteristics and markers of myocardial electrical instability and vegetative regulation of heart rate. Comprehensive diagnosis on day 7–9 of STEMI and the estimation of the independent variables HRT, LVESD, and GLS allow CHF progression to be predicted in the post-infarction period. Reliable stratification of the risk of CHF progression after STEMI is required, in order to identify patients who may need preventive measures against adverse outcomes: proactive antiremodeling therapy, ICD implantation, if indicated, and/or heart surgery.

Conclusions

1. In real-life clinical practice, 23% of patients with a history of ST-segment elevation myocardial infarction who underwent invasive or pharmacoinvasive revascularization experienced the progression of chronic heart failure within 12 months.
2. It has been established that the markers of chronic heart failure progression after ST-segment elevation myocardial infarction are: abnormal onset of turbulence; impaired heart rate turbulence; high levels of brain natriuretic peptide; increased left ventricular end-systolic

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СЕРДЕЧНАЯ НЕДОСТАТОЧНОСТЬ РАЗБИВАЕТ ТЫСЯЧИ ЖИЗНЕЙ

Эта болезнь может коснуться каждой семьи.

Каждую минуту в России погибает один пациент с ХСН, и смертность от нее примерно в 10 раз выше смертности от инфаркта миокарда[1,2].

Сегодня наиболее эффективный подход, позволяющий взять под контроль ХСН, состоит в сочетании медицинского лечения и активного участия самого пациента в изменении своего образа жизни[3].

Помогите своим пациентам и их членам семьи узнать больше о заболевании. Это поможет снизить проявление симптомов сердечной недостаточности и замедлить прогрессирование заболевания.

МОЛОДЫСЕРДЦЕМ.РФ

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1. Клинические рекомендации ОССН – РКО – РНМОТ. Сердечная недостаточность: хроническая (ХСН) и острая декомпенсированная (ОДСН). Диагностика, профилактика и лечение. Кардиология. 2018;58(S6). DOI: 10.18087 / cardio. 2475
2. http://med-info.ru/content/view/6032
3. https://www.heartfailurematters.org/ru_RU/Что-можете-сделать-вы%3F/RU-What-can-you-do

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dimension; decreased global longitudinal, circular, and radial strains.

3. The multivariate logistical regression analysis identified the early predictors of chronic heart failure development in the post-infarction period: abnormal heart rate turbulence; increased left ventricular end-systolic dimension, and decreased longitudinal strain.

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