Shvec D. A.[1], Povetkin S. V.[2]

[1] «Orel clinical regional hospital», Orel, Russia

[2] Kursk State Medical University, Kursk, Russia

The Role of Residual Stenosis of the Coronary Arteries in the Dynamics Systolicdiastolic Left Ventricular Function after Acute Coronary Syndrome

Objective	The aim of the research under consideration was to study the dynamics of a local systolic-diastolic function of patients with various ischemic heart disease (IHD) progressions after survival of an acute coronary syndrome (ACS) provided there are residual stenoses of coronary arteries.
Materials and Methods	There were 112 patients suffering from ACS who took part in the research. The diagnosis was verified (acute myocardial infarction or unstable angina) in accordance with the recommendations of European Society of Cardiology (ESC). All patients were divided into two groups depending on the occurrence of major acute cardiac events (MACE): 59 patients with aggravated IHD progression and 152 patients with non-aggravated course. Echo-cardiography was performed on a scanner Philips iE33 (the Netherlands) with a consideration to systolic and diastolic functions parameters of a left ventricle. Quantitative analysis of the left ventricle was executed in the mode of Tissue Doppler Imaging (TDI) and according to the method of tracing the patches of the ultra-sound image gray scale (ST).
Results	In the course of the aggravated IHD a decrease in systolic-diastolic function of left ventricle has been discovered. It has been found out that the amount of the systolic peak of longitudinal strain of the left ventricle anteroseptal wall less than 12% is associated with a greater extent of coronary atherosclerosis and aggravated progression of IHD. TDI and ST methods have enabled to reveal that in the course of non-aggravated IHD the contractility and the diastolic function of the left ventricle anteroseptal wall improve in combination with the increase in the contractility of the left ventricle inferolateral wall. During an aggravated IHD progression the contractility and diastolic function of the left ventricle anteresoptal wall decreases without an increase in contractility and diastolic function of the inferior and inferolateral walls of the left ventricle. The reason for such results might be a progressing myocardial ischemia of the left ventricle anteroseptal wall despite the sufficient anterior interventricular artery stenting. The presence of the relevant residual stenoses of the circumflex and right coronary arteries increases the possibility of the aggravated IHD progressing especially by the end of the fourth year of observation.
Conclusion	The disbalance of the local contractility of anterior, inferior and inferolateral left ventricle walls in the course of aggravated IHD is connected with the CA residual stenoses presence and forwards the decrease in global systolic-diastolic function of the left ventricle. The final results can serve as a foundation for optimization of recommendations for performing PCI on the patients with multivessel disease of CA.

Keywords	Acute coronary syndrome, left ventricle dysfunction
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Corresponding author Shvec Denis. E-mail: denpost-card@mail.ru

High cardiovascular mortality prompts researchers to seek the causes and mechanisms of cardiovascular events (CVEs) in patients with coronary artery disease (CAD). The prognosis is particularly poor in patients with acute coronary syndrome (ACS). Numerous international registers, such as GRACE, TIMI, CRUSAD, and EHS-ACS, were used to develop diagnosis and treatment algorithms for ACS [1, 2]. Myocardial revascularization in early-stage CAD is the most requested and fastest-growing field of interventional cardiology. The prognostic role of stenting in ACS is distinct – it is to save myocardium, preserve its contractile function, and prevent remodeling of the LV. The questions of time frames and methods of myocardial revascularization in patients with ACS and multivessel CAD are still open. The COMPLETE, PRAMI, and CvL PRIT studies showed that stenting of an infarction-unrelated artery following the revascularization of the infarction-related CA is feasible. According to clinical

guidelines, delayed stenting of infarction-unrelated CAs is required before discharge. This approach reduced the risk of myocardial infarction (MI), cardiac death, and refractory angina [3–6]. The revascularization of asymptomatic stenoses with uncertain hemodynamic significance was not shown to be feasible [7].

Hemodynamically significant residual coronary stenoses increase the risk of cardiovascular complications (CVCs). Myocardial ischemia of the LV associated with such stenoses results in the progression of systolic dysfunction. Most studies aiming to assess the severity of post-ACS disorders of LV have used global echocardiographic systolic and diastolic parameters. Few studies explain the mechanisms of CAD complications in relation to the dynamics of local myocardial contractility [8–10]. When tissue Doppler imaging (TDI) and speckle tracking imaging (STI) became available, researchers for the first time had access to a noninvasive, inexpensive tool for the quantitative assessment of LV systolic function [11–15]. The quantitative parameters of local myocardial contractility and relaxation make it possible to discover the causes of global systolic/diastolic dysfunction of the LV, which will help detect the most unfavorable ratios of residual coronary stenoses and optimize the management of patients with post-ACS CAD.

The objective was to study the dynamics of local systolic/diastolic function in patients with different courses of post-ACS CAD in terms of residual coronary stenoses.

Materials and Methods

The study included 211 patients with ACS treated in the emergency cardiology department from 2011 to 2016. The study was implemented under Good Clinical Practices and the principles of the Declaration of Helsinki. The ethics committee of Kursk State Medical University approved the study protocol. Before inclusion in the study, all subjects signed written informed consent. The only inclusion criterion was nosological: all patients had ACS at admission. Diagnosis (acute myocardial infarction or unstable angina) was verified as recommended by the European Society of Cardiology [16]. Patients with poor-quality ultrasound images of the LV segments, atrial fibrillation, complete bundle branch block, and endocardial pacing were excluded.

Patients included in the study were divided into two groups depending on the course of CAD. The grouping criterion was the onset of one of the following complications, more specifically major adverse cardiac events (MACEs): death due to cardiac causes, recurrent ACS (unstable angina, MI). [17] Group 1 included 59 patients with complicated CAD, and Group 2 included 152 patients with uncomplicated CAD. MACEs were tracked prospectively; the follow-up median was 62 [36.0; 71.0] months.

Echocardiography was performed using the Philips iE33 (Netherlands) scanner and the S5–1 sensor (1.7–3.5 MHz). The main parameters were scanned and measured from the apical view of the LV. Following the recommendations of the American Society of Echocardiography and the European Association of Cardiovascular Imaging [18], the following parameters were determined: left atrial (LA) volume index from the four-chamber view of LV (LAVI, mL/m^2); LV end-systolic volume index (LVESVI, mL/m^2). All volume parameters were normalized to body surface area calculated using the R. D. Mosteller formula (1987). Left ventricular ejection fraction (LVEF) was assessed by Simpson's method. When mitral valve (MV) and aortic flows were recorded simultaneously, isovolumic relaxation time (IVRT, msec), isovolumetric contraction time (IVCT, msec), and the duration of systolic flow (St, msec) of the LV were determined. The systolic dysfunction index was calculated (Tei, U) – (IVRT + IVST)/St.

Data were registered in TDI mode using the standard method with one of two options: mitral annular velocity (the root of the anterior cusp on the 2nd and 3rd views of the LV chamber) and assessment of myocardial strain. In the TDI mode, a mitral annular motion curve was obtained in the low-velocity (tissue) range, which was used to measure the velocity of the systolic and early diastolic annular motion (S, e', cm/sec). When data were registered in TDI mode to analyze myocardial strain, a two-dimensional echocardiographic image of good quality was used. The resulting images were stored on a CD. The QLAB 7.1 software package was used to process the data obtained. All images of poor quality were rejected. Data postprocessing was carried out using the SQ and TMQA modules in QLAB. In this mode, a curve was obtained, which was used to measure a peak rate of longitudinal systolic strain (strain long).

The STI examination was implemented from the apical LV views. Peak systolic longitudinal strain, time to peak of longitudinal strain, and late systolic strain rate of the LV segment examined were measured in the STI mode. Systolic and diastolic functions of the LV anterior wall were determined by the TDI and STI parameters of the middle anteroseptal. Systolic and diastolic function of the lower LV wall according to the parameters of the basal segment of the lower LV wall. and inferior walls of the LV by the parameters of the basal inferior wall of LV.

To estimate the dynamics of the parameters examined, the percentage difference was calculated: (final value – initial value)/initial value × 100% (%).

Selective coronary angiography was carried out using the standard procedure (M. P. Judkins, 1967) on the Philips angiographic machine. In Group 1 and Group 2, 52 (88.1%)

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and 118 (77.6%) exams were performed, respectively. Stenoses were considered hemodynamically significant if the inner diameter of a coronary artery according to coronary angiography had narrowed to 70% or less.

Statistical analysis of data was carried out using the STATISTICA 13 software. The parametric and nonparametric methods were used for statistical processing of the data obtained. In the case of normal distribution (the Kolmogorov-Smirnov test was used), the significance of differences between mean values was estimated using the Student's t-test with Bonferroni correction for multiple comparisons. Otherwise, the comparisons we performed using the Mann-Whitney U-test. The tables provide the mean and standard deviations (M ± SD) or the median and interquartile range [25th percentile; 75th percentile]. The chi-square test and Fisher's coefficient with Yates correction were used to compare the rates. The Kaplan-Meier survival curves were built to estimate the effect of the parameters examined on the survival of patients without MACEs. The Gehan-Wilcoxon test was calculated to assess the differences between empirical functions of survival in the study groups. The differences were considered significant at $p<0.05$.

Results

Nosological characteristics of the patients with CAD are shown in Table 1. The groups of patients with CAD have comparable nosology (Table 1).

The clinical profile of the included patients is provided in Table 2.

The history of patients with complicated CAD most commonly included previous MI, extracardiac atherosclerosis, and diabetes mellitus (Table 2). We failed to establish differences in the risk factors of CAD. Patients with complicated CAD had a higher functional class of chronic heart failure and were more commonly administered drugs of the main classes. As for the laboratory data, reduced levels of WBCs and increased levels of uric acid are noticeable in Group 1.

Progressive coronary atherosclerosis and myocardial ischemia result in myocardial contractile dysfunction

Table 1. Nosological characteristics of patients with CAD

Parameter		Complicated CAD (n=59)	Uncomplicated CAD (n=152)	p
Unstable angina		21 (35.6 %)	45 (29.6 %)	>0.05
Anterior MI	Non-Q	9 (15.2 %)	27 (17.8 %)	>0.05
	Q	9 (15.2 %)	22 (14.5 %)	>0.05
Inferior MI	Non-Q	12 (20.3 %)	31 (20.3 %)	>0.05
	Q	8 (13.7 %)	27 (17.8 %)	>0.05

CAD, coronary artery disease; MI, myocardial infarction.

Table 2. Clinical characteristics of patients with CAD

Parameter		Complicated CAD (n=59)	Uncomplicated CAD (n=152)	p
Age, years		59.1 ± 9.6	56.9 ± 10.4	>0.05
Sex	Male	44 (74.6)	112 (73.7)	>0.05
	Female	15 (25.4)	40 (26.3)	>0.05
BMI, kg/m^2		28.4 ± 4.8	28.5 ± 4.7	>0.05
SBP, mmHg		145 [125; 170]	140 [130; 160]	>0.05
DBP, mmHg		90 [80; 100]	80 [80; 90]	>0.05
CAD risk factors	Hereditary	25 (42.4)	58 (38.1)	>0.05
	Hypertension	49 (83)	114 (75)	>0.05
	Dyslipidemia	11 (18.7)	31 (20.4)	>0.05
	Smoking	25 (42.4)	74 (48.7)	>0.05
	DM	15 (25.4)	17 (11.2)	0.005
History	MI	15 (25.4)	19 (12.5)	0.02
	Revascularization	5 (8.5)	7 (4.6)	>0.05
	CVA	7 (11.8)	12 (7.9)	>0.05
Extracardiac atherosclerosis		6 (10.2)	5 (3.2)	0.03
Previous angina		36 (61)	80 (52.6)	>0.05
Previous therapy	Antiplatelet drugs	24 (40.7)	35 (23)	0.002
	ACE inhibitors/ ARB	26 (44.1)	45 (29.6)	0.02
	Beta-blockers	17 (28.8)	38 (25)	>0.05
	Statins	13 (22)	16 (10.5)	0.02
CHF	FC I	14 (23.7)	46 (30.3)	>0.05
	FC II	23 (38.9)	77 (50.6)	>0.05
	FC III	22 (37.4)	29 (19.1)	0.007
TC, mmol/L		5.5 [4.4; 6.9]	5.5 [4.3; 6.5]	>0.05
HDL, mmol/L		1.12 [0.96; 1.23]	1.13 [1.0; 1.28]	>0.05
LDL, mmol/L		3.9 [2.4; 4.9]	3.8 [2.4; 4.6]	>0.05
RBCs, 1012/L		4.4 ± 0.48	4.5 ± 0.43	>0.05
Hemoglobin, g/L		145 [133; 155]	148 [138; 157]	>0.05
WBCs, 109/L		8.5 [7.3; 10.4]	9.6 [7.2; 12.6]	0.02
ESR, mm/h		16.2 ± 9.5	15.6 ± 10.7	> 0.05
Creatinine, mmol/L		88.0 [75.5; 101.0]	84 [72.0; 96.0]	> 0.05
Uric acid, mmol/L		392 [299; 460]	334 [275; 441]	< 0.03
Glucose, mmol/L		6.4 [5.8; 8.6]	6.3 [5.7; 7.6]	> 0.05
Positive troponins		29 (49.1)	82 (53.9)	> 0.05
HR, bpm		70 [64; 80]	72 [63; 84]	>0.05
QRS, msec		96 [90; 106]	96 [88; 102]	>0.05
Q-Tc, msec		421.3 ± 27.4	419.9 ± 24.5	>0.05

Data are presented as the absolute value (%), M ± SD, or Mean (5^{th} percentile; 75^{th} percentile). CAD, coronary artery disease; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; DM, diabetes mellitus; MI, myocardial infarction; CVA, cerebrovascular accident; ACE, angiotensin-converting enzyme; ARB, angiotensin II receptor blockers; CHF, chronic heart failure; FC, functional class; TC, total cholesterol; HDL, high-density lipoprotein cholesterol; LDL, low-density lipoprotein cholesterol; ESR, erythrocyte sedimentation rate; HR, heart rate; QRS, duration of the QRS complex on the electrocardiogram; Q-Tc, duration of the corrected Q-T interval on electrocardiograph.

and abnormal LV diastolic filling. Table 3 summarizes the dynamics of the main echocardiographic parameters of the patients studied.

According to these data, complicated course CAD is associated with LV and LA dilation, which is a result of increased end-diastolic pressure. The global systolic/diastolic function of LV is significantly depressed. LVEF was reduced only in complicated CAD. Median delta% of LVEF in complicated CAD was –8.6 [– 21; 1.9]; p=0.004.

Tissue Doppler details the changes detected in global parameters via assessment of the function of each LV wall.

Table 4 summarizes the dynamics of the main parameters of mitral annular velocity describing the global systolic and diastolic function of LV.

Reduced systolic and diastolic functions of the LV anteroseptal wall is typical for patients with complicated CAD (Table 4). The early mitral annular velocity of the LV anteroseptal wall, in particular, was considerably reduced. Median delta% of e' in complicated CAD was –10.6 [– 23.5; 5.0]; p=0.004. There were no intergroup differences in the dynamics of parameters or the final values.

Table 5 shows the functional parameters of the anterior wall of the LV in patients with CAD obtained by STI examination of the middle anteroseptal wall of the LV.

These data show that, in uncomplicated CAD, contractility and diastolic function of the anterior wall of LV improves. Thus, all parameters were significantly different between the groups at the second examination. Comparison of the initial and remeasured systolic and diastolic parameters of the inferior wall of the LV did not identify any significant differences between the groups.

Table 3. Dynamics of the main echocardiographic parameters in patients with CAD

Parameter		Complicated CAD (n=59)	Uncomplicated CAD (n=152)	p
LVESVI, mL/m²	Initial	20 [13.9; 26.5]	17.4 [12.3; 24]	>0.05
	Control	25.7 [17; 39.2]***	17.0 [13.9; 23..6]	0.0001
LVEF, %	Initial	63 [54; 70]	65 [57; 70]	>0.05
	Control	59 [47; 65]***	64 [56; 69]	0.0001
ILCD, U	Initial	1.25 [1.0; 1.5]	1.1 [1.0; 1.37]	0.02
	Control	1.25 [1.0; 1.75]	1.1 [1.0; 1.37]	0.009
LAVI, mL/m²	Initial	35 [28; 41.8]	31.3 [26; 38.1]	0.04
	Control	37.4 [30.5; 45.3]	31.4 [25.2; 39.8]	0.0009
Tei, U	Initial	0.46 [0.4; 0.53]	0.42 [0.35; 0.49]	0.03
	Control	0.51 [0.44; 0.57]*	0.43 [0.36; 0.53]	0.0002

Differences from the first visit data are significant (*p<0.05; **p < 0.01; ***p<0.001). CAD, coronary artery disease; LVESVI, left ventricular end-systolic volume index; LVEF, left ventricular ejection fraction; LAVI, left atrial volume index; ILCD, index of local contractility disturbances; Tei, index of left ventricular systolic/diastolic dysfunction.

Table 4. Mitral annular velocity in patients with CAD

Parameter			Complicated CAD (n=59)	Uncomplicated CAD (n = 152)	p
Antero-septal wall of LV	S, cm/sec	Initial	5.4 ± 1.0	5.6 ± 1.0	>0.05
		Control	5.1 ± 1.3*	5.7 ± 1.1	0.03
	e', cm/sec	Initial	4.9 ± 1.2*	5.6 ± 1.5	0.03
		Control	4.4 ± 1.4**	5.5 ± 1.5	0.003
Infero-lateral wall of LV	S, cm/sec	Initial	6.2 [5.4; 7.8]	7.0 [6.1; 8.1]	0.01
		Control	6.4 [5.3; 7.7]	7.6 [6.5; 9.2]**	0.005
	e', cm/sec	Initial	6.4 ± 2.2	7.8 ± 2.8	0.001
		Control	6.2 ± 2.6	7.9 ± 2.8	0.0003
Inferior wall of LV	S, cm/sec	Initial	6.4 ± 1.4	6.8 ± 1.3	>0.05
		Control	6.2 ± 1.5	7.1 ± 1.4*	0.002
	e', cm/sec	Initial	5.1 [3.9; 6.1]	5.7 [4.5; 7.7]	0.006
			4.9 [4.0; 6.5]	6.2 [4.8; 7.8]	0.002

Differences from the first visit data are significant (*p <0.05; **p< 0.01). CAD, coronary artery disease; LV, left ventricle.

The values of the longitudinal systolic strain of the middle anteroseptal wall of the LV were compared according to the severity of coronary atherosclerosis (Figure 1). TDI chart: if there is no significant atherosclerosis (1), longitudinal strain is 16.9%; if there is significant atherosclerosis (2), longitudinal strain is 10.8%. ST chart: 52.4% and –10.9%, respectively.

Decreased contractility of the middle anteroseptal wall of the LV less than –12% is associated with more severe coronary atherosclerosis (Figure 1). When delta% of the longitudinal strain of the middle anteroseptal wall of the LV was compared, a two different trends was observed: a

Table 5. STI parameters of systolic and diastolic functions of the middle anteroseptal wall of LV

Parameter		Complicated CAD (n=28)	Uncomplicated CAD (n=91)	p
Longi-tudinal strain, %	Initial	–14,4 [–17.1;–10.7]	–14.9 [–19.2; –9.2]	>0.05
	Control	–12,7 [–15.8; –8.3]	–16.3 [–19.2; –12.0]*	0.03
Time to peak strain, msec	Initial	325 [301; 346]	332 [292; 356]	>0.05
	Control	321 [295; 349]	336 [208; 439]	>0.05
Peak strain rate a, s^{-1}	Initial	1.16±0.37	1.16±0.41	>0.05
	Control	1.1±0.4	1.35±0.5*	0.03

Differences from the first visit data are significant (*p <0.05). LV, left ventricle; CAD, coronary artery disease; peak strain rate a, rate of late diastolic peak strain of the LV segment examined.

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Figure 1. Longitudinal systolic strain of the middle anteroseptal wall of LV depending on severity of coronary atherosclerosis of left anterior descending artery (inner diameter of less than 70% or more than 70%)

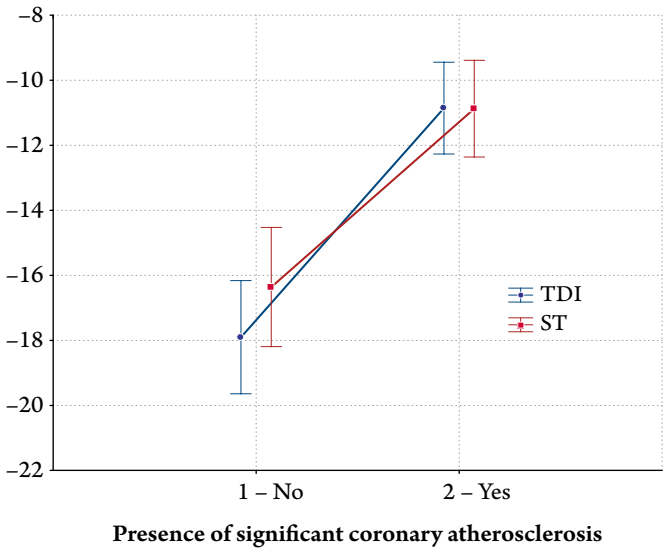
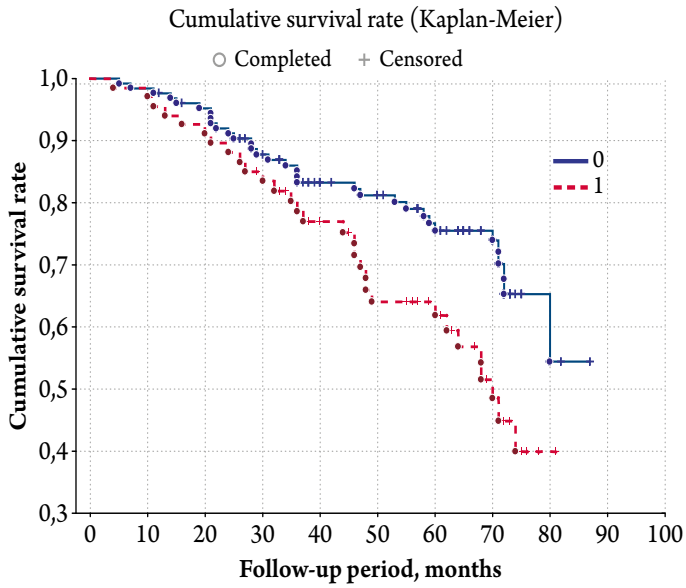


Figure 2. Kaplan-Meier survival curves for patients with CAD without MACEs according to the presence of hemodynamically significant stenoses of LCX and RCA



0—there are no hemodynamically significant residual stenoses of LCX and RCA. 1—hemodynamically significant residual stenoses of LCX and RCA were identified (chi-square =5.2; p=0.02). The number of patients without residual stenoses was 123, of whom 36 patients (29.3%) had complicated CAD. The number of patients with residual stenoses of LCX and RCA was 49, of whom 24 (48.9%) had complicated CAD. CAD, coronary artery disease; MACEs, major adverse cardiac events; LCX, left circumflex artery; RCA, right coronary artery.

reduction in complicated CAD (– 13.9 [– 38.3, 5.5]) and an increase in uncomplicated CAD (– 12.7 [– 6.9; 44.3]); p=0.003.

Complications of CAD are caused by the progression of coronary atherosclerosis. Table 6 shows the results of coronary angiography of patients studied in terms of significant and residual coronary stenoses.

The prevalence of hemodynamically significant stenoses was identified only in the left anterior descending artery (LAD). Revascularization was most common in this CA in Group 1 (the same number of residual stenoses). In the left circumflex artery (LCX) and the right coronary artery (RCA), significant residual stenoses requiring revascularization were predominant. In the group of complicated CAD, there were significantly fewer patients with single-vessel CAD (48.9% vs. 69.8%; p=0.003) and more patients with three-vessel CAD (21.3% vs. 7.6%, p=0.003). The differences in single- and multivessel CAD between the groups were significant (chi-square =5.2; p=0.02).

Despite revascularization of the myocardium, hemodynamically significant coronary stenoses remain in the case of multivessel disease. The dependence of the presence of hemodynamically significant residual coronary stenoses and the probability of a complicated course of CAD were studied. Residual stenoses of LAD do not affect the probability of MACEs. Figure 2 shows survival curves without MACEs according to the presence of residual stenoses of the LCX and RCA.

If there are hemodynamically significant stenoses of the LCX and RCA, the complicated course of CAD is more likely

Table 6. Number of patients studied with CAD who have significant (initial) and residual (post-PCI) coronary stenoses, n (%)

Parameter		Complicated CAD (coronary angiography, n=52)	Uncomplicated CAD (coronary angiography, n=118)	p
LAD	Significant stenoses	35 (67.3)	63 (53.3)	0.03
	Residual stenoses	15 (42.8)	22 (34.9)	>0.05
LCX	Significant stenoses	18 (34.6)	35 (29.6)	>0.05
	Residual stenoses	15 (83.3)	22 (62.8)	0.007
RCA	Significant stenoses	29 (55.8)	49 (41.5)	0.02
	Residual stenoses	17 (58.6)	17 (34.6)	0.006

Residual stenoses, number and percentage of significant nonstented stenoses of the total number of significant stenoses. CAD, coronary artery disease; PCI, percutaneous coronary intervention.

(Figure 2). The curves become particularly significantly separated in 4 years of follow-up.

If there are residual stenoses of the LCX and RCA, contractility of the inferior wall of the LV decreases (Figure 3).

Residual stenoses of the LCX and RCA result in a significant decrease in contractility of the inferior wall of the LV (peak systolic longitudinal strain less than –11%).

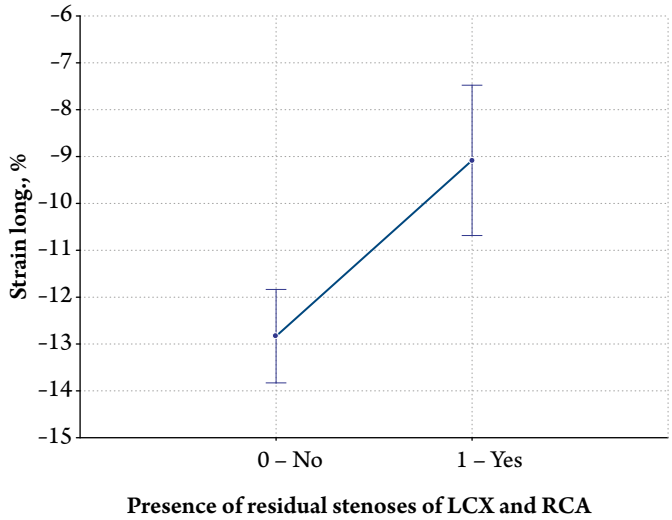
Discussion

The risk of severe CVCs increases with the severity of coronary atherosclerosis. Conservative lipid-lowering therapy and surgical methods of myocardial revascularization (coronary stenting and bypass) are currently common. If indicated, the symptom-dependent CA is stented in patients with ACS in the first 24 and 72 hours, which reduces ischemic dysfunction of the LV and thus improves the survival of patients with CAD. As for stenting of residual coronary stenoses, which are not directly associated with the current acute ischemic process, opinions differ [6]. It was proven necessary to restore blood flow using a method that allows complete myocardial revascularization (grade of recommendations and level of evidence 11A) [7]. The method of revascularization was chosen individually, depending on the specific clinical situation. LVEF was one of the main criteria. There is no consensus on the time of delayed stenting of hemodynamically significant infarction unrelated arteries. Obviously, there is no need to perform complete coronary stenting in the acute MI period (even in cardiogenic shock). However, in the case of hemodynamically significant residual stenoses, revascularization should be repeated within a short time [5]. Complicated CAD was often associated with multivessel CAD. When the number of residual stenoses was estimated, RCA and LCX stenoses were found to be most common in complicated CAD. This can be explained by the prevalence of patients with anterior MI in Group 1; that is, LAD stenting was more common. Delayed stenting of the LCX and RCA in Group 1 patients was not performed adequately, which could cause an increase in the number of CVCs.

Reduced systolic and diastolic function of LV is the marker that can be used to identify patients with poor prognosis. The advent of major CVCs, as such, can be both a cause and a consequence of systolic dysfunction of the LV. The study showed that, in the group of complicated CAD, LVESVI, LAVI, and Tei indices are significantly increased. These parameters exceeded the references in patients of Group 1 [18]. In real-world clinical practice, LVEF is most often used as a global systolic index, and mitral annular velocity e' is used as a noninvasive marker of the level of LV filling pressure.

To identify the mechanisms of global systolic/diastolic dysfunction, parameters characterizing the dysfunction of individual LV walls and segments were used. Tissue Doppler

Figure 3. Peak systolic longitudinal strain of basal inferior wall of LV according to the presence of residual stenoses of LCX and RCA in patients with CAD



If there are no residual stenoses of LCX and RCA (0), the longitudinal strain is –12.9%, otherwise (1) it is –9.0%. Strain tang,%, peak systolic longitudinal strain of the basal inferior wall of LV ($p<0.001$). LV, left ventricle; CAD, coronary artery disease; LCX, left circumflex artery; RCA, right coronary artery.

imaging is the most affordable, easy-to-learn, and user-friendly method of quantitative assessment of segmental function. Systolic and diastolic mitral annular velocity parameters from different LV views can be used to obtain real-time information about the systolic/diastolic function of the anterior, inferior, and lateral walls of the LV. The values of mitral annular velocity were lower than those cited in the literature, due to the concomitant hypertrophy and ischemic dysfunction of the LV [15]. STI allows performing more specific identification of LV segmental dysfunction. The control examination revealed significant differences between the groups in all parameters analyzed. However, examination of the lateral wall is often challenging. That is why TDI and STI are used to complement each other.

These trends in the studied parameters were detected: Contractility and diastolic function of the LV anteroseptal wall improve in uncomplicated CAD, and the contractility of the inferolateral wall increases. In complicated CAD, contractility and diastolic function of the LV anterior wall decrease without an increase in contractility and diastolic function of the inferior and inferolateral walls of the LV. This imbalance is likely to cause a decrease in EF in Group 1. The reason is that in the complicated course of coronary artery disease, the number of patients with anterior myocardial infarction and hemodynamically significant stenosis of the permanent myocardial infarction prevails. Although the number of residual stenoses in the groups is similar, a diffuse atherosclerotic process in the LAD is likely to be more pronounced. It could be the reason of the

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decrease in contractility of the anterior wall in the group with complicated CAD and corresponding higher rate of LAD revascularization in this group. The risk of MACEs after LAD stenting can be assumed to decrease to a lesser extent than after revascularization of LCX and RCA. The revascularization technique also matters. The recovery of LAD circulation using coronary bypass surgery is known to improve survival, and restoration of RCA and LCX circulation eliminates the symptoms [19]. Given the role of residual stenoses of the LCX and RCA in prognosis, the factor of insufficient increase in contractility of the inferior and inferolateral walls of LV is highly significant. It can be assumed that the decrease in global contractility of the LV is a result of the simultaneous reduction of the anterior wall contractility and insufficient increase or decrease in contractility of the inferolateral wall of the LV. Increased contractility of the inferior and inferolateral walls does not occur, due to the progression of ischemia in the myocardial regions supplied by the LCX and RCA. Similar dynamics in the diastolic parameters can be observed. In uncomplicated CAD, an increase in contractility of the inferior and basolateral walls of the LV was detected, despite the prevalence of inferior MI. Over time, increase in contractility is a result of optimal revascularization and less severe atherosclerosis of the LCX and RCA. These results, showing the rise in the number of MACEs in patients with residual stenoses of the LCX and RCA, are consistent with the results of the COMPLETE, PRAMI, and CvL PRIT studies [5]. Moreover, we obtained data evidencing not only the increase in the number of deaths and MI but also the progression of systolic/diastolic dysfunction. LAD stenting in multivessel CAD was shown to be less effective in the prevention of MACEs than stenting of the LCX and RCA.

The problem of repeat revascularization lies in the organizational aspects of providing medical care to patients with CAD in the Russian Federation. Repeated delayed stenting within one hospital stay is difficult due to the progressively decreasing period of hospitalization of patients with ACS and restrictions on refunding the repeated cases of high-tech medical care via the compulsory medical insurance fund. Thus, despite instructions in the discharge summary that repeated stenting is required, for various reasons, some patients do not undergo repeat revascularization. Furthermore, in most cases, it is not possible to assess the hemodynamic significance of borderline coronary stenoses. It is suggested that, during stenting of an infarction-related artery, it is reasonable to perform simultaneous estimation of the hemodynamic significance of residual coronary stenoses. After stenting of the LAD, if there are residual stenoses of the LCX and/or RCA, the identified longitudinal strain of the anterior wall of the LV less than –12% and the inferior wall less than –11% can be used as the basis for early revascularization.

Conclusion

Local contractile imbalance in the anterior, inferior, and inferolateral walls of the left ventricle in complicated coronary heart disease is associated with the presence of residual stenoses of the coronary arteries. It contributes to the decrease in left ventricular systolic function. These findings can be used to improve the recommendations for percutaneous coronary interventions in patients with multivessel disease of the coronary arteries.

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