Prognostication of late left ventricular systolic dysfunction in patients with acute coronary syndrome during the acute period

Marija Rūta Babarskienė, Jonė Venclovienė[1], Dalia Lukšienė[1], Birutė Šlapikienė[1], Irena Milvidaitė[1], Rimvydas Šlapikas, Jūratė Janėnaitė[1]

Department of Cardiology, [1]Institute of Cardiology, Kaunas University of Medicine, Lithuania

Key words: *acute coronary syndrome; left ventricular dysfunction; late prognosis.*

Summary. *The aim of the study was to create the model of the combination of clinical and echocardiographic determinants during the acute period of acute coronary syndromes for the prognostication of the risk for left ventricular dysfunction after one year.*

We examined 565 patients with first-time acute coronary syndrome with no recurrence during one-year period. The studied group consisted of 496 patients, and the examined group – of 69 patients. All patients with acute coronary syndrome within the first three days underwent the evaluation of demographic, anamnesis, clinical indicators, risk factors for ischemic heart disease, ECG, and echocardiographic findings for the prognostication of the risk of left ventricular dysfunction after one year. Multiple logistic regression analysis was applied for the identification of independent determinants for the prognostication of left ventricular dysfunction, and three risk groups were identified. The prognostic informative value of the model was verified by comparing the incidence of left ventricular systolic dysfunction in risk groups after one year between the studied and the control groups.

Results. After one year, left ventricular systolic dysfunction (left ventricular ejection fraction <40%) in the presence of acute coronary syndrome remained in more than half (65.3%) of patients and returned to normal (left ventricular ejection fraction ≥40%) in one-third of patients (34.7%). Left ventricular systolic function that was normal during the acute period of coronary syndrome remained such in the majority (80.9%) of patients after one year, whereas one-fifth (19.1%) of patients developed left ventricular systolic dysfunction. The mathematical model for the prognostication of systolic dysfunction after one year was composed of the determinants of acute coronary syndrome: left ventricular ejection fraction <40%, anterior localization of Q-wave myocardial infarction, Killip class 3–4, left ventricular pseudo-normal or restrictive diastolic function, and frequent ventricular extrasystoles. The application of our model in the prognostication of late left ventricular systolic dysfunction during the acute period of coronary syndrome showed that the model was reliable, since after one year, the prognosticated left ventricular systolic dysfunction was determined in the majority (84.3%) of patients.

The designed mathematical model is simple and is based on standard clinical and echocardiographic findings, and the scoring system allows for the prognostication of the risk for late left ventricular systolic dysfunction in any individual patient. The prognostication of the risk for late left ventricular systolic dysfunction during the acute period of coronary syndrome may help in the planning of treatment and outpatient care in patients with acute coronary syndrome.

Introduction

Left ventricular (LV) systolic dysfunction is a common complication of acute coronary syndrome (ACS). In case of ACS, LV systolic dysfunction may occur during the acute period or later. The greatest risk of LV systolic dysfunction arises from myocardial infarction (MI) – especially recurrent – and complex arrhythmias (1).

In the presence of LV systolic dysfunction occurring during the acute period of MI, most patients (70%) experience heart failure (HF), whereas approximately one-third of patients develop no symptoms of HF (2). According to population studies, LV systolic dysfunc-

Correspondence to M. R. Babarskienė, Department of Cardiology, Kaunas University of Medicine, Eivenių 2, 50009 Kaunas, Lithuania. E-mail: ruta@kmu.lt

tion and HF in the presence of MI are detected in 30–40% of patients (3–5). LV systolic dysfunction occurring during the acute period of ACS may disappear or reduce in 20–30% of patients, whereas in more than one-half of patients it may progress or manifest itself through chronic HF (6). According to the findings of the TRACE study, clinical determinants of HF persisted during the later period in 85% of MI patients with severely impaired LV systolic function, while these determinants disappeared in nearly one-half (40%) of patients with slightly impaired LV systolic function (7). During the last decade, the causes, mechanisms, diagnostic options, and outcomes of LV systolic dysfunction have been sufficiently well studied, but the algorithms and models of the clinical course of LV systolic dysfunction are still under development.

The aim of this study was to design a mathematical model of the combination of clinical and echocardiographic (Echo KG) determinants, whose application would allow for the prognostication of the risk for presence and development of LV systolic dysfunction in ACS patients after one year.

The contingent and the methods of the study

The sample consisted of 565 patients with first-time ACS and with no recurrence of the condition within the period of one year. The patients' age ranged from 22 to 86 years (mean age 59.8±9.5 years). The studied group consisted of 496 patients (96 patients hospitalized for high risk of unstable angina pectoris (UAP) and 400 patients with MI). The examined group in which the prognostic models of the risk of LV systolic dysfunction were tested consisted of 69 patients (10 patients with high risk of UAP and 59 patients with MI). There were no significant differences between the groups concerning patients' age, gender, risk factors, damage to coronary arteries (CA), class of acute HF, or treatment tactics applied. The patients underwent inpatient treatment with platelet antiaggregants, anticoagulants, nitrates, β-blockers, ACE inhibitors, and statins. CA angiography was performed in 376 patients of the studied group and in 60 patients of the examined group. Two hundred eight patients underwent percutaneous transluminal angioplasty, and 130 – coronary artery bypass surgery. The characteristics of the patients in the studied and the examined groups are presented in Table 1.

Demographic factors, anamnesis, clinical findings, risk factors, ECG, and Echo KG findings in all patients were evaluated during the first 3–5 days of ACS and after 1 year.

Myocardial infarction was diagnosed based on the World Health Organization (WHO) recommendations: substernal (angina) pain and its equivalents, indicators of ischemic damage on ECG (changes in Q wave, ST seg-

Table 1. **Characteristics of the groups**

Determinant	Contingent*			
	the studied group (n=496)		the examined group(n=69)	
	n	%	n	%
Age >60 years	141	28.4	23	33.3
Males	383	77.2	52	75.4
Arterial hypertension	253	51.0	39	56.5
Diabetes mellitus	31	6.2	5	7.2
Performed CA angiography	376	75.8	60	86.9
CA stenosis ≥70 %				
one	184	48.9	27	45.0
two	97	25.6	19	31.6
three	53	14.1	11	18.3
Q-wave MI	266	53.6	42	60.9
Non-Q-wave MI	134	27.0	17	24.6
Unstable angina pectoris	96	19.3	10	14.5
Q-wave anterior MI	173	34.9	20	29.0
Killip class II-IV	235	47.3	36	52.2
Interventional treatment	295	59.7	43	62.3

CA – coronary artery, MI – myocardial infarction.
*No significant difference was found.

ment, and T wave), and increased levels of cardio-specific enzymes. The diagnosis of unstable angina pectoris was confirmed after the detection of the angina syndrome, the emergence of ischemic changes in the ECG without any increase in the blood levels of cardio-specific enzymes, and in the presence of CA damage detected angiographically. More than two-thirds (75.8%) of patients underwent CA angiography according to the Judkins technique. Significant CA stenosis was evaluated when CA constriction was 70% or more. Risk factors were indicated in the following cases: diabetes mellitus – if indicated in the anamnesis, hypoglycemic medications were used, or glucose concentration in blood plasma exceeded 7.0 mmol/L; arterial hypertension, when arterial blood pressure was 140/90 mmHg or higher, or the patient received antihypertensive medications; overweight, when the body mass index was 25 kg/m^2 or greater, frequent extrasystoles (more than 10 per min).

Echocardiographic data were gathered using commercially available second harmonic imaging systems by experienced ultrasonographers, and it was repeated by the same investigator. All examinations were performed according to the criteria presented by the American Society of Echocardiography. All measurements were obtained after calculating the mean value of three consecutive measurements. All subjects underwent the evaluation of LV end-diastolic size, the level of remodeling, LV ejection fraction (EF), LV wall motion index (LV WMI), and the degree of mitral regurgitation. LV EF of <40% was considered to be decreased, and LV EF of ≥40% – normal.

LV diastolic dysfunction was evaluated using pulse Doppler from apical four chamber view. The evaluation of the degree of diastolic dysfunction (DD) was the following: the 1st degree – impaired relaxation (E/A<1, deceleration time >240 ms); the 2nd degree – pseudo-norm (E/A <2 – >1, the duration of the reverse "a" wave of pulmonary veins exceed that of the "A" wave); the 3rd degree – restriction (E/A>2, deceleration time <140 ms). Pseudo-normal and restrictive LV diastolic dysfunction was seen as severe LV diastolic dysfunction.

Statistical data analysis

Data collection and processing was performed using the standard software packages – "Microsoft Excel 2000" and "Statistika." Quantitative indicators were evaluated using the following statistical characteristics: mean value and standard deviation. The difference was considered to be statistically significant when P<0.05.

The prognostication of the risk of individual determinants for LV systolic dysfunction after one year was evaluated using odds ratio (OR) and confidence interval (CI). The determinants, significant in univariate analysis at a P level of 0.10, were entered in the multiple regression models, and their maximal capability of prognosticating LV dysfunction after 1 year was evaluated. The suitability of the models in patient selection was evaluated using c statistics, where the function was equivalent to the area located under the ROC curve. The model was considered to have good discriminatory power when the c index exceeded 0.8. Based on the value of e^{β} (standardized OR) in the model with the highest discriminatory power, each determinant or its level was evaluated in points, and the total score was calculated from the arithmetical sum of these points. The determinants of the selected model were used to evaluate the total score during the acute period. According to the score, all patients were differentiated into three risk groups for the prognostication of LV systolic dysfunction after 1 year. The risk groups were composed so that the difference in the incidence of LV dysfunction would be the greatest, and the P value of the χ^2 – the lowest. After one year, we calculated the incidence of LV systolic dysfunction in each risk group.

The prognostic value of the designed model was verified by comparing the incidence of LV systolic dysfunction in the risk groups after one year between the studied and the examined groups.

Results

The incidence of LV systolic dysfunction among patients with first-time ACS and after one year

During the acute period, LV systolic dysfunction (EF<40%) was detected in 30.3% of patients who experienced ACS for the first time and survived for one year without ACS.

In patients with detected LV systolic dysfunction during the acute period, LV systolic dysfunction after one year persisted in more than one-half (65.3%) and returned to normal in about one-third of patients (34.7%) (Fig. 1).

In patients with normal LV systolic function during the acute period, after a year it remained such in the majority of patients (80.9%), and one-fifth of patients (19.1%) developed LV systolic dysfunction.

Thus, after one year following ACS, the incidence of LV systolic dysfunction increased by 3.2% as compared to the acute period.

The informative value of clinical and echocardiographic determinants of coronary syndrome during the acute period for the prognostication of LV systolic dysfunction after one year

We compared clinical, angiographic, and Echo KG determinants of ACS during the acute period in patients

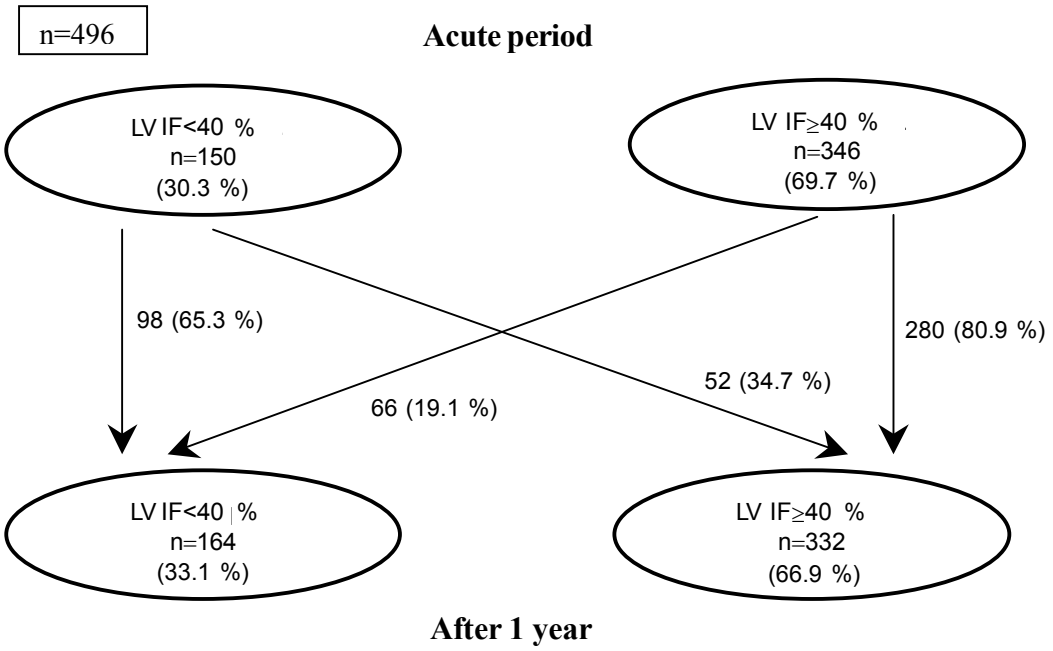

Fig. 1. **The incidence of LV systolic dysfunction among patients with first acute coronary syndrome during the acute period and after one year**

LV - left ventricle; ACS - acute coronary syndrome; EF - ejection fraction.

who after one year developed LV systolic dysfunction with the respective determinants of patients in whom LV systolic function after one year remained normal; subsequently, we applied linear logistic regression analysis to determine the odds ratio (OR) of these determinants for the prognostication of LV systolic dysfunction after one year (Table 2).

The following determinants were informative: Killip class III–IV (OR=7.02), Q-wave MI (OR=16.85), anterior Q-wave MI (OR=3.57), ST segment elevation more than 3 mm (OR=5.44), paroxysmal atrial fibrillation (OR=4.17), frequent ventricular extrasystoles (OR=3.54), and Echo KG findings – LV EF less than 40% (OR=11.6), LV WMI >1.5 (OR=7.35), LV end-diastolic diameter (EDD) >56 mm (OR=3.11), severe diastolic dysfunction (OR=9.1–27.0), and degree 3–4 mitral regurgitation (OR=27.0) (P<0.05).

The models of clinical and echocardiographic determinants of coronary syndrome during the acute period for the prognostication of LV systolic dysfunction after one year

Linear regression analysis was used to select 14 significant determinants during the acute period of ACS, and multiple logistic regression analysis was used to evaluate the informative value of these determinants. The findings showed that seven determinants – Q-wave MI, anterior localization of Q-wave MI, Killip class II–IV, LV EF <40%, severe diastolic dysfunction (pseudo-normal or restrictive), LV WMI >1.5, and frequent ventricular extrasystoles – independently increased the probability of the persistence or development of LV systolic dysfunction after one year. Using these determinants, three models were designed for the prognostication of LV systolic dysfunction after one year (Table 3). Since the models contained different combinations of the determinants, their OR differed: for LV EF <40% OR was 5.13–2.57; for anterior Q-wave MI, 4.26–1.96; the OR for Killip class increased (by 1.9–1.4 times) with increasing class; and LV pseudo-normal or restrictive dysfunction increased the probability of the persistence or development of LV systolic dysfunction by 2.4–2.1 times.

The evaluation of the sensitivity and specificity of the models using ROC curve showed that the models were both sensitive and specific (had good discriminatory power) for the prognostication of LV systolic dysfunction after one year. The first model had the best discriminatory power (c=0.855): anterior Q-wave MI, LV EF <40%, severe LV diastolic dysfunction, Killip class III–IV, and frequent ventricular extrasystoles. The discriminatory powers of the second and the third models, compared to that of the first one, were lower but were not statistically significantly different (c=0.852 and 0.837 *versus* 0.855).

The risk groups of subjects for the prognostication of LV systolic dysfunction after one year

Table 2. **The informative value of clinical and electro-physiological determinants during the acute period of ACS for the prognostication of LV systolic dysfunction after one year (n=496)**

Determinants during the acute period	EF<40 % After 1 year (n=164)		EF≥40 % After 1 year (n=332)		Odds ratio*	Confidence interval
	n	proc.	n	proc.		
Age (years)	58.9±9.6		58.3±9.6			
Age >65 years	50	30.9	91	27.4	1.08	0.71–1.64
Males	125	76.2	258	77.7	0.91	0.74–1.85
Arterial hypertension	84	51.2	131	52.6	1.02	0.69–1.49
Diabetes mellitus	18	11.0	13	3.9	1.7	0.88–3.31
Performed CA angiography	119	72.6	257	77.4		
CA stenosis ≥70 % one	61	51.3	123	47.9	1.9	0.79–4.59
two	31	26.1	66	25.7	1.88	0.74–4.78
three	20	16.8	33	12.8	2.47	0.91–6.69
Q-wave MI	133	81.1	133	40.1	16.85	6.63–42.9
Non-Q-wave MI	25	15.2	109	32.8	3.67	1.34–10.04
Unstable angina pectoris	6	3.7	90	27.1	1	
Q-wave anterior MI	103	62.8	70	21.1	3.57	1.5–6.1
Killip class I	45	27.4	214	64.5	1	
II	102	62.2	109	32.8	3.16	2.0–58
III–IV	17	10.4	9	2.7	7.02	5.1–12.4
HR >90 bpm.	30	18.3	32	9.6	2.43	1.4–5.1
Frequent ventricular extrasystoles	79	48.2	91	27.4	3.54	1.62–7.75
Paroxysmal atrial fibrillation	18	11.0	11	3.3	4.17	8.0–8.4
Non-fatal ventricular fibrillation	12	7.3	11	3.3	2.41	1.04–5.59
ST elevation >3 mm	74	45.1	41	12.3	5.44	4.7–7.3
LV EDD >56 mm	13	7.9	7	2.1	3.11	2.1–5.8
LV geometry unchanged, or concentric remodeling present	79	49.4	205	61.7	1.0	
LV concentric hypertrophy	66	40.9	117	33.4	1.73	1.123–2.66
LV eccentric hypertrophy	15	9.8	10	3.0	2.27	1.24–4.16
Left atrial enlargement >60 mm	39	23.8	37	11.1	2.1	1.2–3.2
LV ejection fraction <40%	98	59.8	52.7	15.7	11.6	6.9–22.2
LV wall motion index >1.5	119	72.6	72	21.7	7.35	5.4–9.8
Normal LV diastolic function	4	2.4	56	16.9	1	
Impaired LV relaxation	105	64.0	210	63.3	9.09	2.78–29.7
Restrictive/pseudo-normal LV diastolic dysfunction	46	28.4	28	8.4	27.0	7.63–95.8
Degree 3-4 mitral regurgitation	18	13.3	21	7.1	1.97	1.014–3.834
Inpatient myocardial revascularization PTCA	75	45.7	104	31.3	1.3	0.76–2.3
CABG	36	22.0	80	24.1	1.14	0.68–1.93

ACS – acute coronary syndrome; MI – myocardial infarction, CA – coronary artery, HR – heart rate, LV – left ventricle, EDD – end-diastolic diameter, PTCA – percutaneous transluminal coronary angioplasty, CABG – coronary artery bypass surgery.

*Odds ratio >2.0 (significant informative value).

Table 3. **The models of the determinants of acute coronary syndromes for the prognostication of LV systolic dysfunction after one year**

Models	Determinants during the acute period	Coefficient β	Waldo chii square (χ^2)	Standardized risk for LV dysfunction	Confidence interval	p	c index
I	LV ejection fraction <40%	1.619	24.581	5.13	2.67–10.00	0.000	0.855
	Anterior Q-wave MI	1.450	25.843	4.264	2.44–7.46	0.000	
	Severe LV diastolic dysfunction	0.891	6.905	2.438	1.25–4.74	0.000	
	Frequent ventricular extrasystoles	0.649	5.304	1.913	1.1–3.32	0.000	
	Killip class III-IV	0.469	4.708	1.598	1.05–2.44	0.000	
II	LV ejection fraction <40%	0.927	5.432	4.96	2.44–9.8	0.000	0.851
	LV wall motion index >1.5	1.155	10.154	3.174	1.560–6.457	0.000	
	Anterior Q-wave MI	1.137	14.674	3.119	1.713–5.581	0.000	
	Severe LV diastolic dysfunction	0.773	5.125	2.166	1.109–4.231	0.009	
	Killip class III-IV	0.469	4.876	1.611	1.059–2.460	0.000	
III	Female gender + LV EF <40%	2.164	4.085	8.709	1.068–71.043	0.043	0.837
	LV wall motion index >1.3	1.238	14.334	3.448	1.817–6.544	0.000	
	LV ejection fraction <40%	0.946	10.375	2.576	1.448–4.580	0.001	
	Q-wave MI	0.662	3.909	1.938	1.006–3.736	0.048	
	Anterior Q-wave MI	0.567	3.447	1.762	0.969–3.205	0.068	
	Killip class III-IV	0.359	3.373	1.432	0.976–2.100	0.066	

MI – myocardial infarction, LV – left ventricle, EF – ejecion fraction.

Table 4. **Evaluation of the determinants in points**

Determinant	Points
LV ejection fraction <40%	3
Anterior Q-wave myocardial infarction	3
Killip class II	1
Killip class III	2
Killip class IV	3
Frequent ventricular extrasystoles	1
Pseudo-normal or restrictive LV diastolic dysfunction LV	2
Total score	12

LV - left ventricle.

The informative value of the independent determinants of the first model, calculated in risk score index, is presented in Table 4.

The total score index (SI) for each patient was calculated according to the following formula:

SI=3 × (EF<40%) + 3 × (anterior Q-wave MI) + (Killip class – 1) + (frequent ventricular extrasystoles) + 2 × (LV pseudo-normal or restrictive diastolic dysfunction).

The total score index for the prognostication of late LV systolic dysfunction varied between 0 and 12 (Table 4). The median score was 3.2 points. In the majority of patients (72.1%), the total score was less than 6 points. The following determinants had the highest informative value in the models: LV EF <40% (3 points, *i.e.* 25% of the total score), anterior localization of Q-wave MI (3 points), and Killip class III (3 points).

The obtained total score was used as the basis for the composition of three risk groups of patients with ACS for the prognostication of LV systolic dysfunction after one year. Low risk was determined when the total score was 3 points or less, medium risk – when the total score was 4–5 points, and high risk – when the total score was 6–12 points. More than one-half of patients (58.2%) during the acute period were assigned to the low risk group, one-tenth (13.9%) of patients – to the medium risk group, and nearly one-third of patients (27.9%) – to the high-risk group for LV systolic dysfunction (Fig. 2).

LV systolic dysfunction, prognosticated during the acute period of ACS, after one year was determined in the majority of patients in the high-risk group, in one-third of patients in the medium risk group, and in less than one-fifth of patients in the low risk group (P<0.000).

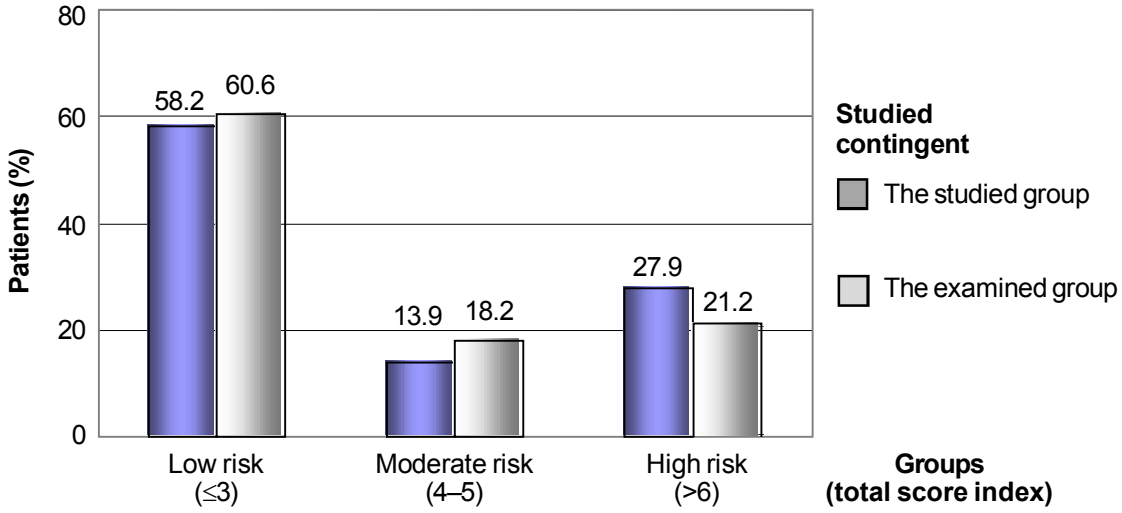

Fig. 2. **The distribution of patients in the studied and the examined groups during the acute period of ACS according to the risk of LV systolic dysfunction after one year**

LV – left ventricle, ACS – acute coronary syndrome.

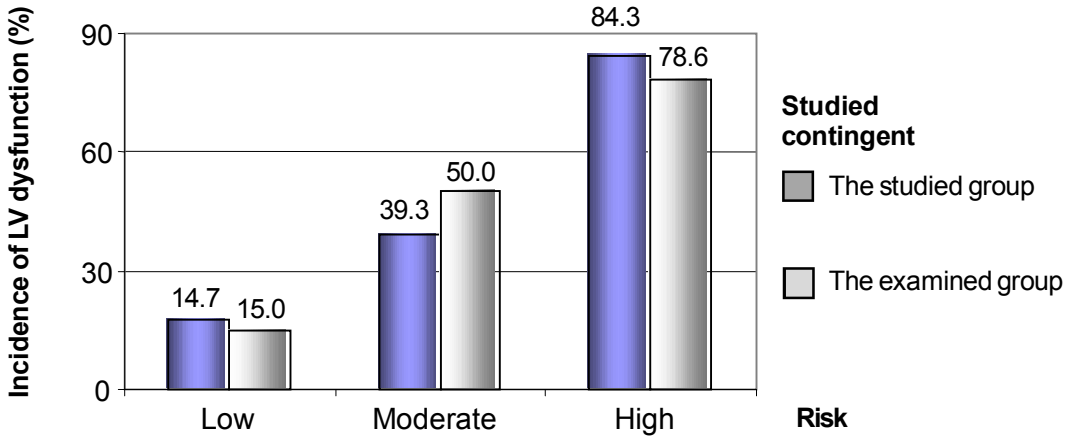

Fig. 3. **The incidence of LV systolic dysfunction after one year in the studied and the examined groups according to the risk prognosticated during the acute period of ACS**

LV – left ventricle, ACS – acute coronary syndrome.

The comparison of the incidence of LV systolic dysfunction after one year in different risk groups of patients of the studied group with the incidence of LV systolic dysfunction after one year in the examined group did not yield any statistically significant difference (in the high risk group – 84.3 and 78.6%; in the medium risk group – 39.3 and 50.0%; and in the low risk group – 17.4 and 15.0%, P>0.05) (Fig. 3).

Discussion

Most frequently LV systolic dysfunction is caused by ACS. The rate of the progression of LV systolic dysfunction and its clinical manifestation – heart failure – is determined by the degree and duration of myocardial ischemia, stunning myocardium, the damaged area of the myocardium, the localization of the damage, the degree of infarct-related coronary artery lesion, and changes in the myocardium prior to ACS (8–11). In the presence of myocardial changes, the course of LV systolic dysfunction is related to the myocardial remodeling process and its consequences, dilatation of cardiac chambers, mitral regurgitation, and diastolic dysfunction. LV systolic dysfunction is significantly influenced by changes in the myocardium (hypertensive or diabetic cardiomyopathy) caused by risk factors – arterial hypertension and diabetes mellitus - before ACS. Increased activity of neurohumoral factors (renin-angiotensin system, genome expression, and cell mediators – endothelin and growth factor), as well as age, gender, and lifestyle are equally important for the development

of LV systolic dysfunction and its sequelae (12, 13).

It is obvious that the prognostication of the clinical course of LV systolic dysfunction, related to the aforementioned factors (some precipitating and some suppressing its progression), is complicated, as is the relationship of LV systolic dysfunction with heart failure. Martinez-Salles *et al.* indicate that LV systolic dysfunction following previous MI is a significant prognostic factor for the evaluation of heart failure and mortality (14). However, in patients with ACS in whom LV systolic dysfunction was detected during the acute period, the prognosis and the relationship of the condition with the development of heart failure (HF) cannot be determined based solely on the decreased LV EF. LV systolic dysfunction may develop within the first hours or within the first several days from the occurrence of ACS, may pass or persist, may be asymptomatic, and may manifest itself through acute HF or – later on – through chronic HF (15, 16). It has been indicated that systolic dysfunction (LV EF <40%) is detected in 40% of patients with MI and later on in 1.3–8.6% cases per year (17–20).

Although LV systolic dysfunction resulting in HF is a common complication of ACS, data on the prognostication of its later course are scarce. Most scientific publications focus on LV remodeling and HF at the same time evaluating LV systolic dysfunction.

LV systolic dysfunction during the acute period of the first ACS was detected in less than one-third (30.3%) of our studied patients. The lower incidence of LV systolic dysfunction, compared to that indicated by other authors, may be due to the difference in the studied contingents – our patients had unstable angina pectoris (UAP) and MI for the first time and survived for one year without any recurrence of ACS.

We found that the risk of the persistence and development of LV systolic dysfunction following ACS was not uniform. LV systolic dysfunction that developed during the acute period of ACS persisted after one year in the majority of the studied patients (65.3%) and returned to normal in one-third of patients (34.7%), whereas 19.1% of patients with previously normal LV systolic function developed LV systolic dysfunction; thus, the incidence of LV systolic dysfunction increased by 3.2%. The instability of LV systolic dysfunction that develops during the acute period of ACS is another reason for the prognostication of LV systolic dysfunction that may develop later on.

The recovery in LV EF after one year in one-third of patients may have been conditioned by the impairment of LV function during the first days of ACS that occurred as a result of damage to certain segments of the myocardium, whose function subsequently improved after the normalization of blood flow in coronary arteries. In part of patients with remaining large-scale myocardial damage, failed or delayed normalization of blood circulation in the CA resulted in the myocardial function remaining low or decreasing further due to LV remodeling processes. Gaudron and Gianuzi *et al.* indicated that progressing late LV remodeling that results in LV systolic dysfunction develops in one-fifth of patients who had MI (21, 22). According to Zhang *et al.*, late remodeling of the myocardium occurs if MI involves more than 15% of the myocardium in case of anterior MI and more than 20% of the myocardium in case of inferior MI (23).

The most significant independent determinants of the acute period of ACS for the prognostication of late LV systolic dysfunction were used in our designed model: decreased LV systolic function (EF <40%), anterior Q-wave MI, Killip class III–IV, frequent ventricular extrasystoles, pseudo-normal/restrictive LV dysfunction, and LV WMI>1.5. The determinants of CA stenoses and mitral regurgitation II–III° that influenced LV systolic dysfunction were strongly correlated with the aforementioned determinants, but their informative value was lower and did not increase the accuracy of the model. The determinants of the acute period of ACS are indicated by numerous researchers as determinants also having a prognostic value for the prognostication of the unfavorable course of the disease (LV remodeling, the development of chronic HF, and death) and used in the development of models for the prognostication of such events (24–30).

The determinants of the acute period of ACS, reflecting an unfavorable course of the disease, were evaluated in points, and a mathematical model was designed allowing for the prognostication of the risk for late LV systolic dysfunction. Our model for the prognostication of late LV systolic dysfunction during the acute period of ACS is simple and is based on standard – clinical and Echo KG – findings; the scoring system prognosticates individual risk for late LV systolic dysfunction, the model has good sensitivity and specificity, and correct prognosis is made in the majority of cases. The selection of high-risk patients (in whom LV systolic dysfunction may either persist or develop during the later period) during the acute period of ACS may help in planning the treatment and close observation of such patients. This would decrease the risk of chronic HF and death.

Conclusions

1. Left ventricular systolic dysfunction that developed during the first acute coronary syndrome after one year persisted in more than one-half (65.3%) of patients and returned to normal in one-third (34.7%) of patients.

2. In the majority of patients with normal systolic left ventricular function during the acute period of acute coronary syndrome, this function remained normal after one year, while one-fifth (19.1%) of such patients developed left ventricular systolic dysfunction.

3. The determinants of the acute period of acute coronary syndrome, which were included into the mathematical model for the prognostication of late left ventricular systolic dysfunction after one year, were the following: LV EF <40%, anterior localization of Q-wave MI, Killip class III–IV, pseudo-normal or restrictive left ventricular diastolic dysfunction, and frequent ventricular extrasystoles.

4. When applying the designed mathematical model, late left ventricular systolic dysfunction was correctly prognosticated in 84.3% of high-risk patients in the studied group, and in 78.6% of patients in the examined group.

Kairiojo skilvelio vėlyvosios sistolinės disfunkcijos prognozavimas sergantiesiems ūminiu išeminiu sindromu ūminiu laikotarpiu

Marija Rūta Babarskienė, Jonė Venclovienė[1], Dalia Lukšienė[1], Birutė Šlapikienė[1], Irena Milvidaitė[1], Rimvydas Šlapikas, Jūratė Janėnaitė[1]

Kauno medicinos universiteto Kardiologijos klinika, [1]Kardiologijos institutas

Raktažodžiai: ūminis išeminis sindromas, kairiojo skilvelio disfunkcija, vėlyvoji prognozė.

Santrauka. Tyrimo tikslas. Sudaryti klinikinių ir echokardiografinių žymenų derinio modelį ūminių išeminių sindromų ūminiu laikotarpiu prognozuojantį kairiojo skilvelio disfunkcijos riziką po vienerių metų.

Ištyrėme 565 ligonius, pirmą kartą susirgusius ūminiu išeminiu sindromu, kuriems vienerius metus sindromas nepasikartojo. Tiriamąją grupę sudarė 496 ligoniai, egzaminuojamąją – 69 ligoniai. Visiems ligoniams, susirgusiems ūminiu išeminiu sindromu, per pirmąsias tris paras įvertinta demografinių, anamnezės, klinikinių žymenų, išeminės širdies ligos rizikos veiksnių, EKG, echokardiografijos rodmenų rizika kairiojo skilvelio disfunkcijai numatyti po vienerių metų. Daugiamatės logistinės regresijos metodu nustatyti nepriklausomi žymenys kairiojo skilvelio sistolinei disfunkcijai prognozuoti, išskirtos trys rizikos grupės. Modelio prognozės informatyvumas patikrintas palyginus tiriamųjų ir egzaminuojamųjų grupių kairiojo skilvelio sistolinės disfunkcijos dažnumą rizikos grupėse po vienerių metų.

Rezultatai. Kairiojo skilvelio sistolinė disfunkcija (kairiojo skilvelio išstūmimo frakcija mažiau nei 40 proc.), atsiradusi ištikus ūminiam išeminiam sindromui, po vienerių metų išliko daugiau negu pusei (65,3 proc.) ligonių, o trečdaliui (34,7 proc.) normalizavosi (kairiojo skilvelio išstūmimo frakcija ≥40 proc.). Kairiojo skilvelio normali sistolinė funkcija, buvusi ūminio išeminio sindromo ūminiu laikotarpiu, daugumai (80,9 proc.) ligonių ir po vienerių metų išliko normali, o penktadaliui (19,1 proc.) atsirado kairiojo skilvelio sistolinė disfunkcija. Matematinį modelį kairiojo skilvelio sistolinei disfunkcijai prognozuoti po vienerių metų sudarė ūminio išeminio sindromo žymenys ūminiu laikotarpiu: kairiojo skilvelio išstūmimo frakcija mažiau nei 40 proc., priekinė Q bangos miokardo infarkto lokalizacija, III–IV Kilipo klasė, kairiojo skilvelio pseudonormali arba restrikcinė diastolinė funkcija, dažnos skilvelinės ekstrasistolės. Taikant mūsų sudarytą modelį, vėlyvoji kairiojo skilvelio sistolinė disfunkcija, prognozuota ūminio išeminio sindromo ūminiu laikotarpiu, po vienerių metų išliko daugumai (84,3 proc.) ligonių.

Sudarytas matematinis modelis nesudėtingas, pagrįstas standartiniais klinikiniais ir echokardiografiniais rodmenimis, remtasi balų sistema, galima prognozuoti kiekvienam ligoniui vėlyvąją kairiojo skilvelio sistolinės disfunkcijos riziką. Kairiojo skilvelio vėlyvosios sistolinės disfunkcijos rizikos numatymas ūminio išeminio sindromo ūminiu laikotarpiu gali padėti numatyti ligonių, susirgusių ūminiu išeminiu sindromu, gydymo ir ambulatorinės priežiūros taktiką.

Adresas susirašinėti: M. R. Babarskienė, KMU Kardiologijos klinika, Eivenių 2, 50009 Kaunas. El. paštas: ruta@kmu.lt

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