



Distinctive features of the connection of diastolic function of the heart with electrical instability myocardium in patients with q wave myocardial infarction

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Abstract

Purpose of the research. Studying the role of arrhythmogenic mechanisms of the formation of ventricular cardiac arrhythmias in patients with coronary heart disease, depending on the degree of left ventricular dysfunction.

Keywords: diastolic dysfunction, myocardial infarction, heart rate variability

Introduction

The main problem in patients with coronary heart disease, especially after myocardial infarction and the development of remodeling of the left ventricle, is the alarming of not only repeated myocardial infarction, chronic heart failure, but also heart rhythm disturbances ^[1, 2]. The relationship of proarrhythmic indicators with indicators of systolic function is quite clearly defined, but the role of diastolic dysfunction in the formation of electrical myocardial instability remains unclear ^[3].

Purpose of the research

To study the specific role of arrhythmogenic mechanisms in the formation of cardiovascular arrhythmias in patients with coronary heart disease with left ventricular dysfunction ^[4].

Material and Methods

131 male patients with primary Q-wave myocardial infarction aged 30 to 69 years (51.9 ± 9.13 years) were examined. At the stationary stage of severe myocardial infarction, the treatment was carried out in accordance with the recommendations for the management of patients with myocardial infarction with ST segment elevation and included thrombolytic therapy, if indicated, early administration of beta-blockers, antiplatelet agents, anticoagulants, nitrates, lipid-lowering drugs, ACE inhibitors, loop diuretics.

While the therapy was ongoing on 10-14 days of severe myocardial infarction, all patients underwent a clinical examination, which includes general examination, medical history taking, ECG in 12 standard leads, EchoCG, and HMECG.

Echocardiography and dopplerographic studies were performed at "Sonoline Versa Pro" apparatus by standard methods using the American Echocardiographic Society ^[5] recommendations. The following indicators were measured and calculated: thickness of the interventricular septum (TIVS); thickness of the left ventricular posterior wall (TLVPW); end diastolic size of the left ventricle (FDSL); end systolic size (FSS) of the left ventricle; left ventricular ejection fraction (LVEF); end diastolic volume of the left ventricle (EDVLV); end systolic volume of the left ventricle (ESVLV).

LV myocardial mass (LVMM) was calculated by the formula of R. Devereux *et al.*

$MMLV = 1,04 * ((FDSL + IVSD * + LVPWD^{**})^3 - EDVLV) - 13,6 \text{ gr.}$

*IVSD - interventricular septum in diastole

**LVPWD - left ventricular posterior wall in diastole

For further analysis, indexed metrics of EDVLV, ESVLV, LVMM were used in total body area.

As an indicator that most reflects the process of heart remodeling, the relative wall thickness (RWT) was calculated by the formula $(TIVS + TLVPW)/EDVLV$. For an increase in the relative wall thickness 0.45 or greater values were taken.

Doppler echocardiography was used to evaluate diastolic function. To characterize diastolic dysfunction of the left ventricle, the following indicators were evaluated: the maximum blood flow velocity during the fast filling phase (peak E, cm/s), the maximum blood flow velocity in the atrial systole (peak A, cm/s), and their ratio (E/A). A sign of impaired diastolic function was considered to be a decrease in the E/A ratio to less than 1.0.

To characterize ventricular extrasystoles (VE), the gradational classification of B. Lown (1971) and the prognostic classification of J. Bigger (1982) were used. Hourly qualitative and quantitative assessment of VE was carried out in accordance with Lown-Wolf gradations: 0-VE is absent, 1 - rare ventricular extrasystole; 2 - frequent ventricular extrasystole; 3 - polymorphic ventricular extrasystoles; 4A-pair ventricular extrasystoles; 4B - group ventricular extrasystoles; 5 - early ventricular extrasystoles. According to the classification of J. Bigger after myocardial infarction, prognostically unfavorable ventricular arrhythmias (PNA) are those that ventricular extrasystole > 10 per hour, paired ventricular extrasystoles and group ventricular extrasystoles.

The data obtained were processed using computer softwares such as Microsoft Excel, STATISTICA 6 and Biostat. The odds ratio (OR) and the 95% confidence interval (95% CI) were calculated using logistic regression. The significance of differences in the indicators was evaluated using nonparametric criterion ^[2] (Pearson test). Quantitative data are presented as $M \pm SD$. Correlation relationships were investigated using regression analysis and Spearman's rank

correlation coefficient. Differences between groups were considered statistically significant at $P < 0.05$. The observation period was 24 months.

Results and Discussion

Undoubtedly, with the progression of coronary heart disease, the nature of diastole parameters and its functions undergo complex changes associated with both exacerbation of diastolic disorders and the development of hemodynamic adaptive reactions acting through an increase in pressure in the left atrium and/or the final diastolic pressure of the left ventricle and leading to the formation of various types of diastolic dysfunction: abnormal relaxation, pseudo-normal and restrictive [6, 7]. To assess the relationship between disorders of the contractile function of the left ventricle and diastolic dysfunction of the left ventricle, the patients were divided into 2 groups (Table 1): a group with preserved systolic function of the left ventricle (ejection fraction $\geq 50\%$) and a group with a reduced ejection fraction ($< 50\%$). A prevalence analysis of various types of left ventricular

diastolic dysfunction showed that severe disorders of the left ventricular diastolic function — pseudo-normal and restrictive types — were significantly more likely to occur in the group of patients with reduced left ventricular systolic function.

Table 1: The prevalence of diastolic dysfunction of the left ventricle in groups of patients with different contractility of the left ventricle

Diastolic dysfunction	LVEF $\geq 50\%$	LVEF $< 50\%$	P
Relaxation disorder	36/80(45%)	44/80(55%)	0,27
Pseudo-normal	15/42(35,7%)	27/42(64,3%)	0,016
Restrictive	4/25(16%)	21/25(84%)	0.00001

All patients, depending on the severity of diastolic dysfunction of the left ventricle, were divided into 3 groups: I (n = 80) - with relaxation disorder; II (n = 42) - with a pseudo-normal type; III (n = 25) - with a restrictive type of filling.

Table 2: Comparative characteristics of groups with various types of left ventricular diastolic dysfunction

Metrics	I group (n = 80)	II group (n=42)	III group (n = 25)
Front	31/38%	28/66,6%*	18/72%**
Rare	49/61,2%	14/33,4%*	7/28%**
Hypertonic disease	72/90%	32/76,2%	20/80%
Diabetes	6/7,5%	3/7,1%	
Aneurysm	24/30%	11/26,2%	13/52%
Thrombolysis	11/13,7%	5/12%	3/12%
early post-infarction angina	30/37,5%	17/40.1%	10/40%
BMI, kg/m ²	27,05 + 3,84	27,7 + 3,74	
Relative thickness of LV wall	0,40 + 0,10	0,34 + 0,09	0,35 + 0,10
LVMM index	133,35 + 37,69	132,3 + 34,52	141,58 + 38,39
IVSD index	1,11+0,29	0,97 + 0,27	1,0472 + 0,37
EDV index	74,36 + 19,94	89,94 + 28,7*	93,79 + 36,35***
ESV index	38,53 + 16,41	48,97 + 20,38	50,92 + 23,47**
ESVLA, ml/m ²	38,7±1,9	44,8±2,6	52.0±2,4***
EF,%	49.21596 + 11,57727	46,07571+9,322993	38.41823+12.06217**

Note: *- $p < 0.05$ between groups I and II; ** - $p < 0.05$ between groups II and III; *** - $p < 0.05$ between groups I and III.

Analysis of clinical and anamnestic indicators showed that patients with severe diastolic left ventricular dysfunction more often had anterior localization of myocardial infarction (72% versus 38 and 66.6, respectively for groups I and II). A history of hypertension with an almost identical frequency occurred in all compared groups, but it should be noted that its prescription was significantly higher in groups with type III left ventricular diastolic dysfunction (9.5 versus 4.9 and 6.5 years, respectively (I and group II, $p < 0.05$). Diabetes mellitus prevailed with a significant frequency in the group of patients with the restrictive type of left ventricular diastolic dysfunction (7.5, 7.1 versus 20%). A similar trend is observed in the limitation of coronary heart disease to shifted for myocardial infarction, for example, in the group with severe diastolic dysfunction (DD) of the left ventricle, it was 10.8 years versus 4.1 and 6.5 years in groups I and II, respectively ($p < 0.05$). Significant differences were also identified with respect to body mass index (BMI). So, in the group with restrictive filling type, it amounted to 30.5 kg/m² against 27.05 kg/m² in I and 27.7 kg/m² in II groups, respectively.

The identification and analysis of LV DD variants is of great clinical importance, since they indicate the severity of diastolic disorders that contribute to the formation of

chronic heart failure [8, 9, 10]. Therefore, we consider it necessary to provide our own data on the analysis of the main indicators of myocardial contractility and the geometry of the left heart chambers in the observed patients, depending on the type of diastolic dysfunction. Analysis of the contractility and geometry of the left chambers of the heart showed that the volumetric indices of the left ventricle — the indexed end-diastolic volume and the indexed end-systolic volume in the group with pseudo-normal and restrictive type were significantly higher compared to the group of patients with relaxation disorder. So, the metrics indexed end-diastolic volume in groups II and III exceeded the same indicator in group I by 20 and 26%, and the indexed end-systolic volume by 27 and 32%, respectively ($p < 0.05$).

In the group with the restrictive type of diastolic dysfunction, there was an increase in dilatation of the cavities of the left chambers, a decrease in contractile function, an increase in the mass of the left ventricle, significant differences in the contractile function of the left atrium. There was a significant difference in the indexed final systolic volume of the left atrium ($p < 0.05$) in patients with type III diastolic disorders compared with similar parameters in other variants of left ventricular diastolic

dysfunction.

As you know, the main cause of sudden cardiac death (SCD) in patients with coronary artery disease is ventricular tachyarrhythmias [L. Bakera. *et al.*, 1998]. Identification of patients with a high risk of developing malignant ventricular arrhythmia is one of the main methods to reduce the incidence of SCD [Stern S, Tzivoni D., 1974; Wit A.L.

JanseM. J "1992]. The mechanism for increasing the frequency of detection of arrhythmias in patients with coronary artery disease complicated by heart failure, compared with patients with uncomplicated coronary artery disease, is still not clear. Various factors, including ischemia or structural changes, such as fibrosis or cardiosclerosis, may be leading in arrhythmogenesis at heart failure.

Table 3: Ventricular arrhythmias in patients with various types of left ventricular diastolic dysfunction

Metrics	Options for violation of diastolic function of the left ventricle		
	I group	II group	III group
Number of patients, n	80	42	25
Number of patients with heart rhythm disorder, n	22	19	13
Ventricular extrasystole analysis			
I gradation, n/%	11/50%	5/25%	1/77%**
II gradation, n/%	4/18,2%	4/20%	2/15,4%
III gradation, n/%	6/27,3%	7/39%	4/30,8%
IV-V gradation, n/%	1/4%	3/16%	6/46,1 %**

Note: ** - $p < 0.05$ between groups I and III.

According to Holter ECG monitoring, on 12-14 days of myocardial infarction, ventricular arrhythmias were detected in 81 (57%) patients, while in 54 (36.7%) patients it was assessed as potentially dangerous ventricular arrhythmias. A comparative analysis of the structure of ectopic activity in patients with Q-wave myocardial infarction depending on the type of diastolic dysfunction of the left ventricle showed that ventricular arrhythmia of high gradations according to J Bigger in group I was observed in 55.6%, in groups II and III in 81.2 and 78.5% of cases, respectively. A similar trend was observed with respect to ventricular arrhythmias, according to the Lown-Wolf classification. So, ventricular arrhythmia of class I was detected in 11 (50%), 5 (25%) and 1 (7.7%) patients, class II in 4 (18.2%), 2 (20%) and 2 (15, 4%) of patients of class III in 6 (27.3%), 7 (39%) and 4 (30.8%) patients respectively in groups I, II and III. Class IV and V ventricular arrhythmias were detected in 1 (4.5%), 3 (16%), and 6 (46.1%) patients respectively in groups I, II, and III. We did not note a significant difference in the registration of ventricular extrasystole of the II and III gradations between groups of patients with diastolic dysfunction of the left ventricle of type II and III, however, complex forms of ventricular extrasystole were more often detected precisely with the restrictive type of diastolic disorders (46.1%) compared with the pseudo-normal type (16.0%) ($X^2 = 6.43$; $p = 0.01$).

Conclusion

The development of diastolic dysfunction of the left ventricle in patients with myocardial infarction with Q wave is associated with the severity, course and duration of coronary heart disease and arterial hypertension. With the progression of diastolic dysfunction, a marked decrease in the ejection fraction of the left ventricle is noted. The severity of diastolic dysfunction in patients with Q-induced myocardial infarction is closely related to the manifestation and course of coronary heart disease.

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