Diastolic Heart Function and Myocardial Electrical Instability in Patients with Q-wave Myocardial Infarction

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Abstract

The study included 131 male patients between the ages of 30 and 69 (51.9 ± 9.13 years) with primary Q-wave myocardial infarction (Q-MI). All patients underwent clinical examination, including a physical examination, medical history, ECG in 12 conventional leads, echocardiography, and 24-hour ECG monitoring from the 10th to the 14th day of MI. The progression of left ventricular diastolic dysfunction in Q-MI patients is associated with a longer history of coronary heart disease and arterial hypertension. With worsening diastolic dysfunction, a marked decrease in LV systolic function is revealed. The severe diastolic dysfunction in Q-MI patients is closely associated with myocardial electrical instability. (*Int J Biomed.* 2015;5(4):192-194.)

Key words: diastolic heart function; Q-wave myocardial infarction; 24-hour ECG monitoring; myocardial electrical instability.

Abbreviations

LVEDD, left ventricular end-diastolic dimension; **LVESD**, left ventricular end-systolic dimension; **LVEDV**, left ventricular end-diastolic volume; **LVESV**, left ventricular end-systolic volume; **LAESV**, left atrium end-systolic volume; **IVST**, interventricular septal thickness; **LVPWT**, left ventricular posterior wall thickness; **LVM**, left ventricular mass; **EF**, ejection fraction; **PVCs**, premature ventricular contractions; **VA**, ventricular arrhythmias; **PE**, peak early filling velocity; **PA**, peak atrial filling velocity; **EPIA**, early post-infarction angina.

Introduction

The main problem of patients with coronary heart disease (CHD), especially after myocardial infarction (MI) and development of left ventricular remodeling, is preventing reinfarction, congestive heart failure, and cardiac arrhythmias [1,2].

The relationship between pro-arrhythmic indicators and systolic dysfunction is well defined, but the role of diastolic dysfunction (DD) in the formation of myocardial electrical instability remains unclear [3]. It is necessary to clarify the role played by formation of the arrhythmogenic mechanisms of ventricular arrhythmias in CHD patients, depending on the degree of left ventricular dysfunction [4].

Materials and Methods

The study included 131 male patients between the ages of 30 and 69 (51.9 ± 9.13 years) with primary Q-MI. The study was approved by the Republican Specialized Center of Cardiology Ethics Committee. Written informed consent was obtained from each patient.

The treatment of acute MI was carried out in accordance with recommendations for the Management of Patients with ST-Elevation Myocardial Infarction and included thrombolytic therapy, early administration of beta-blockers, antiplatelet agents, anticoagulants, nitrates, statins, ACE inhibitors, and loop diuretics. All patients underwent clinical examination, including a physical examination, medical history, ECG in 12 conventional leads, echocardiography, and 24-hour ECG monitoring on the 10th through the 14th days of MI.

Echocardiography and Doppler sonography study carried out on the unit «Sonoline Versa Pro» by standard methods using the recommendations of the American Society

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of echocardiography [5]. The following parameters were measured and calculated: IVST, LVPWT, LVEDD, LVESD, LVEF, LVEVD, LVESV, and LVM (LVM was calculated using the formula R.Devereux [6]). Further analysis was indexed to body surface area indicators: LVEDVI, LVESVI, IVSI, LA ESVI, and LVMI.

As an indication, to the greatest extent reflecting the process of cardiac remodeling, RWT was calculated by the formula $(IVS+PW)/LVEDD$. A value of ≥ 0.45 showed an increase in RWT. The diastolic function was evaluated by Doppler echocardiography. A decrease in PE/PA ratio less than 1.0 was considered as a sign of DD.

To characterize the PVCs, the B. Lown and M. Wolf classification (1971) and the prognostic classification of J.Bigger (1984) were used. Hourly qualitative and quantitative assessment of PVCs was performed in accordance with the Lown-Wolf gradation: Class 0 – absence of PVCs, Class I – rare monomorphic PVCs; Class II – frequent single PVCs; Class III – polymorphic (polytopic) PVCs; Class IVA – paired PVCs; Class IVB – group PVCs; Class V – early PVCs, R/T phenomena. After MI, according to the J.Bigger classification, PVCs >10 per hour, pair and group PVCs are potentially hazardous ventricular arrhythmias. Follow-up was 24 months.

The obtained data were processed using computer software Microsoft Excel, STATISTICA 6 and Biostat. The mean (M) and standard deviation (SD) were calculated. The odds ratio (OR) and 95% confidence interval (95% CI) were calculated using logistic regression. Group comparisons with respect to categorical variables are performed using chi-square tests or, alternatively, Fisher's exact test when expected cell counts were less than 5. Correlations were examined using regression analysis and Spearman rank correlation coefficient. A probability value of $P < 0.05$ was considered statistically significant.

Results and Discussion

To assess the relationship between LV systolic dysfunction and LV diastolic dysfunction, patients were divided into 2 groups (Table 1): a group with preserved LV systolic function ($EF \geq 50\%$) and a group with a reduced EF ($EF < 50\%$). Analysis of the prevalence of different types of LV diastolic dysfunction showed that severe diastolic dysfunction (pseudonormal and restrictive types) was significantly more frequent in patients with reduced LV systolic function [7,8].

Table 1.

Prevalence of LV diastolic dysfunction in patients with different LV contractility

Diastolic dysfunction	LV EF $\geq 50\%$	LV EF $< 50\%$	P
Impaired relaxation	36/80(45%)	44/80(55%)	0.27
Pseudonormal filling	15/42(35.7%)	27/42 (64.3%)	0.016
Restrictive filling	4/25(16%)	21/25(84%)	0.00001

All patients were divided into 3 groups, depending on the severity of LV diastolic dysfunction: Group 1 (n=80) included patients with impaired relaxation of LV (type 1),

Group 2 (n=42) included patients with pseudonormal filling (type 2), and Group 3 (n=25) included patients with restrictive filling (type 3).

Analysis of clinical and anamnestic indicators (Table 2) showed that patients with severe LV diastolic dysfunction were more likely to have anterior localization of MI (72% vs. 38% and 66.6%, respectively, Groups 1 and 2). Arterial hypertension (AH) history with the same frequency was found in all comparisons, but it should be noted that the duration of AH was significantly higher in Group 3 with LV diastolic dysfunction type 3 (9.5 versus 4.9 and 6.5 years in Group 1 and 2, respectively, $P < 0.05$). Diabetes mellitus (DM) with a significant frequency prevailed in Group 3 patients (20% vs. 7.5%, 7.1% in Group 1 and 2, respectively). A similar trend can be seen in relation to CHD duration before the MI onset: 10.8 years in Group 3 with severe LV diastolic dysfunction compared with 4.1 and 6.5 years in Groups 1 and 2, respectively ($P < 0.05$). Significant differences were also identified in relation to body mass index (BMI). BMI was 30.5 kg/m² in Group 3 vs. 27.1 kg/m² and 27.7 kg/m² in Groups 1 and 2, respectively.

Table 2.

Comparative characteristics of groups with different types of LV diastolic dysfunction

Variable	Group 1	Group 2	Group 3
Anterior MI	31/38%	28/66.6%*	18/72%^
Posterior MI	49/61.2%	14/33.4%*	7/28%^
AH	72/90%	32/76.2%	20/80%
DM	6/7.5%	3/7.1%	3/20%^Δ
Aneurism	24/30%	11/26.2%	13/52%
Thrombolysis	11/13.7%	5/12%	3/12%
EPIA	30/37.5%	17/40.1%	10/40%
BMI, kg/m ²	27.1±3.8	27.7±3.7	30.5±4.9^Δ
RWT	0.40±0.10	0.34±0.09	0.35±0.10
LVMI, g/m ²	133.4±37.7	132.3±34.5	141.6±38.4
IVSI _{diast} , cm/m ²	1.11±0.29	0.97±0.27	1.0472±0.37
LV EDVI, ml/m ²	74.4±19.9	89.9±28.7*	93.8±36.3 ^Δ
LV ESVI, ml/m ²	38.5±16.4	48.97±20.38	50.9±23.47 ^Δ
LA ESVI, ml/m ²	38.7±1.9	44.8±2.6	52.0±2.4^Δ
LVEF, %	49.2±11.6	46.08±9.33	38.4±12.06^Δ

* - $P < 0.05$ between Groups 1 and 2; ^ - $P < 0.05$ between Groups 2 and 3; Δ - $P < 0.05$ between Groups 1 and 3

Analysis of the geometry and contractility of the left chambers of the heart showed that the LV volume indicators (the indexed EDV and ESV) in patients of Groups 2 and 3 were significantly higher compared with Group 1 patients. In particular, the indexed EDV and ESV were higher by 20% and 27% in Group 2 vs. Group 1, and by 26% and 32% in Group 3 vs. Group 1, respectively ($P < 0.05$) [9-11].

Group 3 patients experienced an increase in dilatation of the left chambers and LVM, a decrease in LV contractility, and significant differences in the index of contractile function of the left atrium. The indexed ESV of the left atrium was

significantly different compared to this parameter in Groups 1 and 2 ($P < 0.05$).

According to 24-hour ECG monitoring, VAs, including the potentially malignant forms, were detected in 81/57% and 54/36.7% patients, respectively, on the 12th through the 14th days of MI. In Q-MI patients, the comparative analysis of the structure of the ectopic activity, depending on the type of LV diastolic dysfunction, identified a high grade of VA, according to the J.Bigger classification, in 55.6%, 81.2% and 78.5% of cases in Groups 1, 2, and 3, respectively. A similar trend was observed in relation to PVCs, according to the Lown-Wolf classification (Table 3). In particular, Class I was detected in 11/50%, 5/26.3% and 1/7.7% patients in Groups 1, 2, and 3, respectively; Class II in 4/18.2%, 2/21.1%, and 2/15.4% patients in Groups 1, 2, and 3, respectively; Class III in 6/27.3%, 7/36.8%, and 4/30.8% patients in Groups 1, 2, and 3, respectively. Class IV+V was detected in 1/4.5%, 3/15.8%, and 6/46.2% patients in Groups 1, 2, and 3, respectively. We did not observe significant differences in the registration of Class II and Class III PVCs between the groups of patients with type 2 and type 3 LV diastolic dysfunction, but complex forms of PBCs were often identified in Group 3 patients with the restrictive type of diastolic dysfunction vs. Group 2 patients with a pseudonormal type of diastolic dysfunction ($P < 0.05$).

Table 3.

PVCs in patients with different types of LV diastolic dysfunction

PVCs	Group 1	Group 2	Group 3
Number of patients with PVCs	22	19	13
Class I, n/%	11/50%	5/26.3%	1/7.7%*
Class II, n/%	4/18.2%	4/21.1%	2/15.4%
Class III, n/%	6/27.3%	7/36.8%	4/30.8%
Class IV+V, n/%	1/4.5%	3/15.8%	6/46.2%*^

*- $P < 0.05$ between Groups 1 and 3; ^- $P < 0.05$ between Groups 2 and 3.

In conclusion: The progression of LV diastolic dysfunction in Q-MI patients is associated with a longer history of CHD and hypertension. With worsening diastolic dysfunction, a marked decrease in LV systolic function is revealed. The severe diastolic dysfunction in Q-MI patients is closely associated with myocardial electrical instability.

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