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Left Ventricular Mechanical Dispersion in the Development of Ventricular Arrhythmia in Patients after Q-Wave Myocardial Infarction

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Abstract

Background: Ventricular arrhythmias (VA) can be life-threatening complications of myocardial infarction (MI). In coronary artery disease (CAD), the main substrates of arrhythmia are fibrous areas of the ventricular myocardium formed after a previous MI. This study aimed to evaluate the hypothesis that left ventricular mechanical dispersion (LVMD) can identify subjects at high risk for developing VA among post-MI patients with left ventricular ejection fraction (LVEF) >40%.

Methods and Results: The study included 100 CAD patients (mean age of 62.7±9.61 years) with a history of acute Q-wave MI no earlier than two months. According to the Lown grading system, all patients (59% men and 41% women) were divided into two groups based on the presence of premature ventricular contractions (PVCs). Group 1 consisted of 58 patients with PVCs, and Group 2 included 42 patients without PVCs. All patients underwent general clinical examination, biochemical blood tests, and functional methods (daily ECG monitoring, standard transthoracic two-dimensional echocardiography [TTE] with ECG synchronization, 2D-STE with the assessment of global longitudinal strain [GLS] and left ventricular mechanical dispersion [LVMD]).

In the general patient group, analysis of the two-dimensional TTE parameters indicated relative preservation of the LV structural and functional parameters, except for a slight decrease in the systolic function with a mildly abnormal LVEF of 45.9±11.5%. A comparative analysis of clinical and demographic indicators in groups with and without PVCs demonstrated relative homogeneity and comparability regarding average age, the duration of CAD and hypertension, lipid metabolism indicators, and structural and functional parameters of the left ventricle and left atrium (P>0.05). We studied the deformation properties of the left ventricle in the longitudinal direction in three apical views. The integral average GLS value was -12.98±3.34% in the group with PVCs and -13.49±3.43% in the group without PVCs (P=0.458). The LVMD was significantly higher in patients with PVCs than those without PVCs: 34.07±9.3 ms versus 9.8±5.54 ms (P=0.000). The analysis of the 2D-STE parameters showed the absence of statistically significant differences in the LVGLS (F=0.8585, P=0.4655) between the subgroups, taking into account the Lown grading system for PVCs, but significant differences in the LVMD were noted. The minimal mean LVMD value (9.3±5.57 ms) was revealed in the group with PVC Grade 0. LVMD increased with the increasing the PVC grade, reaching maximum values in the subgroup with PVC Grades IV- V: 15±8.37 ms in the subgroup with PVC Grades I-II, 34.0±6.30 ms in the subgroup with PVC Grade III, 49.8±12.8 ms in the subgroup with PVC Grades IV-V (F=129.6136, P=0.0000).

Conclusion: Left ventricular mechanical dispersion, determined by 2-D STE, is an important prognostic marker related to the development of ventricular arrhythmia in post-MI patients with intermediate LVEF.**(International Journal of Biomedicine. 2024;14(4):558-562.)**

Keywords*:* myocardial infarction • ventricular arrhythmia • mechanical dispersion • speckle tracking echocardiography

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Abbreviations

AI, atherogenic index; **A2C**, apical two-chamber view; **A3C**, apical three-chamber view; **A4C**, apical four-chamber view; **CAD**, coronary artery disease; **DBP**, diastolic blood pressure; **ECG,** electrocardiogram; **FBG**, fasting blood glucose; **GLS**, global longitudinal strain; **HR**, heart rate; **LVMD**, left ventricular mechanical dispersion; **MI**, myocardial infarction; **PVC**, premature ventricular contractions.

Introduction

Over the past two decades, numerous studies have been devoted to investigating the links between reentrant arrhythmias and myocardial ischemia, including acute myocardial infarction.[1] In previous studies, significant efforts were made to identify the main causes of sudden cardiac death and to identify patients with a high risk of developing ventricular arrhythmias.[2] In acute myocardial necrosis, a peri-infarction zone consisting of ischemic tissue is formed around the damaged area;[3] in the later period, this zone is preserved around scar tissue and includes viable myocardial fibers. According to various data,[4-7] the zone mentioned above is susceptible to life-threatening ventricular arrhythmias (LTVA) both in the acute period of infarction and in the presence of a post-infarction scar due to differences in conductivity and refractoriness of adjacent myocardial areas. In general, LTVA in stable coronary artery disease most often occurs by the re-entry mechanism.[3,4] The mechanisms of occurrence of LTVA at different degrees of LV remodeling after myocardial infarction (MI) are still poorly understood.[8]

Prevention of the development of arrhythmias associated with ischemia remains a key aspect in modern research, emphasizing the importance of early diagnosis and monitoring of ischemic changes for the prevention and management of arrhythmias. Two-dimensional speckle tracking echocardiography (2D-STE) assessing mechanical dispersion and global longitudinal deformation of the left ventricle has demonstrated high information content in acute and chronic coronary syndrome.[9-11] Left ventricular mechanical dispersion (LVMD), as evaluated by 2D-STEA, is an important diagnostic indicator of regional heterogeneity of myocardial contraction, known as left ventricular mechanical dyssynchrony.[12] The higher the mechanical dispersion value, the lower the synchronicity of left ventricular segments. In 2010, Haugaa et al.[13,14] reported that LVMD assessed using speckle tracking strain was a useful marker for predicting ventricular arrhythmias (VA) in various cardiovascular diseases. In a prospective, multicenter study by Haugaa et al.,[15] mechanical dispersion assessed by strain echocardiography predicted arrhythmic events independently of left ventricular ejection fraction (LVEF) in patients after MI.

In CAD, the main substrates of arrhythmia are fibrous areas of the ventricular myocardium formed after a previous MI. Fibrous tissue in the left ventricle can lead to heterogeneity of contractions, causing electrical dispersion that affects activation time and refractoriness. Changes in ventricular repolarization dispersion associated with structural and functional alterations in the myocardium after MI lead to uneven recovery of cardiomyocyte excitability.[8] The issue of finding noninvasive predictors of sudden cardiac death caused by life-threatening arrhythmias remains unresolved and highly controversial.

This study aimed to evaluate the hypothesis that LVMD can identify subjects at high risk for developing VA among post-MI patients with LVEF >40%.

Materials and Methods

The study was performed in the departments of the Republican Specialized Scientific and Practical Medical Center of Cardiology (Tashkent, Uzbekistan) from January 2023 to May 2024 and included 100 CAD patients (mean age of 62.7±9.61 years) with a history of acute Q-wave MI no earlier than two months. According to the Lown grading system, all patients (59% men and 41% women) were divided into two groups based on the presence of premature ventricular contractions (PVCs).[16] Group 1 consisted of 58 patients with PVCs, and Group 2 included 42 patients without PVCs.

Low ejection fraction and LV remodeling with the formation of LV aneurysm are associated with the potential development of cardiac arrhythmias, which was considered when forming the study groups. The exclusion criteria from the study were patients with LVEF<40% and LVEF>50%, with postinfarction LV aneurysm, with a history of coronary artery revascularization before the examination, without stable sinus rhythm (atrial fibrillation, atrial flutter, sinus node dysfunction, with sinoatrial and atrioventricular blocks), with channelopathies, with congenital heart defects, with chronic rheumatic heart disease, with moderate and severe valvular lesions, with concomitant diseases affecting heart rate variability (diabetes mellitus, hypo- and hyperthyroidism, alcoholism, severe renal and respiratory failure), with oncological diseases.

All patients underwent general clinical examination, biochemical blood tests, and functional methods (daily ECG monitoring, standard transthoracic two-dimensional echocardiography [TTE] with ECG synchronization, 2D-STE with the assessment of GLS and LVMD).[17] STE was performed using AutoSTRAIN Automated Cardiac Motion Quantification A.I. 15 Technology on the Philips Affiniti 70 ultrasound system. Two-dimensional images of four-chamber, three-chamber, and two-chamber apical views and an LV parasternal short-axis view (at the root of the papillary muscle) were recorded.

Statistical analysis was performed using the statistical software package SPSS version 24.0 (SPSS Inc, Armonk, NY: IBM Corp). Baseline characteristics were summarized as frequencies and percentages for categorical variables and mean ± standard deviation (SD) for continuous variables. Inter-group comparisons were performed using Student's t-test. Multiple comparisons were performed with one-way ANOVA. A probability value of $P<0.05$ was considered statistically significant.

Results

The initial clinical and demographic characteristics of the patients under study are presented in Table 1. The mean HR was 73.4±12.7 bpm. SBP and DBP against the background of antihypertensive drugs were 131.6±18.9 and 82.3±9.1 mmHg, respectively. Lipid-lowering therapy controlled the lipid profile of 88% of patients. Only the level of triglycerides and AI were increased: 195.0 [109.8-262] mg/dL and 3.9±1.6, respectively. Carbohydrate metabolism indices remained within the normal values.

Analysis of the two-dimensional TTE parameters indicates (Table 1) relative preservation of the LV structural and functional parameters, except for a slight decrease in the

systolic function with a mildly abnormal LVEF of 45.9±11.5%. Global longitudinal deformation, an indicator of the LV's deformation properties, and LVMD showed features in the intergroup analysis (Table 2). A comparative analysis of clinical and demographic indicators in groups with and without PVCs demonstrated relative homogeneity and comparability regarding average age, the duration of CAD and hypertension, lipid metabolism indicators, and structural and functional parameters of the left ventricle and left atrium (P>0.05).

Table 1.

Baseline initial clinical, hemodynamic, and laboratory parameters of patients in the general group

Variable	General group (n=100)
Age, years	62.7±9.61
Men / Women	59/41
HR, bpm	73.4±12.7
SBP, mmHg	131.6±18.9
DBP, mmHg	82.3±9.1
TC, mg/dL	188.8±48.6
TG, mg/dL	195.0 [109.8-262]
LDL-C, mg/dL	107.7±45.0
HDL-C, mg/dL	41.1±17.5
VLDL-C, mg/dL	39.0 [22.0-53.0]
AI	3.9±1.6
FBG, mmol/L	5.3 [4.9-6.6]
Ao, mm	33.2±3.8
LAV, mL	49.9±14.5
LAVI, mL/m^2	25.6±6.8
IVST, mm	10.7±2.05
PWT, mm	10.1±1.4
LVM, g	205.9±50.2
LVMI, g/m^2	105.4±23.3
LVEDD, mm	49.6±9.3
LVESD, mm	35.2±9.0
LVEDV, mL	111.0±35.7
LVESV, mL	63.2±32.5
LVSV, mL	48.0±10.6
LVEF, %	45.9±11.5
GLS (A4C), (-%)	12.96±3.71
GLS (A2C), (-%)	13.17±3.44
GLS (A3C), (-%)	13.3±3.62
GLS average, (-%)	13.1±3.35
LVMD, ms	27.5±19.1

AI, atherogenic index; Ao, aortic diameter at the sinuses of Valsalva; A2C, apical two-chamber view; A3C, apical three-chamber view; A4C, apical four-chamber view; DBP, diastolic blood pressure; FBG, fasting blood glucose; GLS, global longitudinal strain; HR, heart rate; HDL-C, high-density lipoprotein cholesterol; IVST, interventricular septal thickness; LDL-C, low-density lipoprotein cholesterol; LAV, left atrial volume; LAVI, LAV index; LVEF, left ventricular ejection fraction; LVESD, left ventricular end-systolic dimension; LVEDD, left ventricular end-diastolic systolic dimension; LVESV, left ventricular end-systolic volume; LVEDV, left ventricular end-diastolic volume; LVMI, left ventricular mass index; LVM, left ventricular mass; LVSV, left ventricular stroke volume; LVMD, left ventricular mechanical dispersion; PWT, posterior wall thickness; SBP, systolic blood pressure; TC, total cholesterol; TG, triglycerides; VLDL-C, very low-density lipoprotein cholesterol.

Table 2.

Comparative analysis of clinical, demographic, laboratory data and structural and functional characteristics of the left ventricle in the studied groups.

Variable	Group 1 (VA+) N=58	Group 2 (VA-) N=42	P-value
Age, years	62.7±9.61	59.0±9.8	0.062
CAD duration, years	7.92±4.26	7.65±5.3	0.778
AH duration, years	8.22±4.70	6.72±5.69	0.153
TC, mg/dL	186.5±50.0	194.7±45.5	0.403
TG, mg/dL	209.7±157.8	196.8±64.6	0.618
HDL-C, mg/dL	41.9±12.2	37.6±9.06	0.056
VLDL-C, mg/dL	41.46±31.9	40.3±14.2	0.826
AI	3.7±1.6	4.30±1.56	0.064
HR, bpm	75.48±11.3	77.68±15.08	0.406
SBP, mmHg	133±19.3	127.9±17.7	0.180
DBP, mmHg	83.1±9.35	80.2±8.3	0.112
SYNTAX score	15.8±9.56	16.22±9.44	0.828
Ao, mm	33.58±3.82	32.3±3.79	0.100
LAV, mL	50.5±15.56	45.5±10.5	0.074
LAVI, mL/m^2	26.15±7.25	24.12±5.47	0.130
IVST, mm	10.7±1.72	10.76±2.79	0.895
PWT, mm	10.1±0.97	9.9±2.39	0.566
LVM, g	207.49±49.8	201.84±52.4	0.585
LVMI, g/m^2	105.77±23.36	104.65±23.83	0.815
LVEDD, mm	49.87±10.58	49.0±5.03	0.623
LVESD, mm	35.6±10.4	34.4±5.29	0.495
LVEDV, mL	110.04±37.54	100.3±28.5	0.1612
LVESV, mL	66.38±34.7	54.9±24.58	0.070
LVSV, mL	49.02±10.1	45.4±11.56	0.099
LVEF, %	45.4±11.8	46.89±11.00	0.523
GLS (A4C), (-%)	12.77±3.68	13.46±3.80	0.364
GLS (A2C), (-%)	13.01±3.42	13.58±3.54	0.450
GLS (A3C), (-%)	13.32±3.68	13.41±3.55	0.903
GLS average, (-%)	12.98±3.34	13.49±3.43	0.458
LVMD, ms	34.07±9.3	9.8±5.54	0.000

AI, atherogenic index; AH, arterial hypertension;Ao, aortic diameter at the sinuses of Valsalva; A2C, apical two-chamber view; A3C, apical three-chamber view; A4C, apical four-chamber view; CAD, coronary artery disease; DBP, diastolic blood pressure; FBG, fasting blood glucose; GLS, global longitudinal strain; HR, heart rate; HDL-C, high-density lipoprotein cholesterol; IVST, interventricular septal thickness; LDL-C, low-density lipoprotein cholesterol; LAV, left atrial volume; LAVI, LAV index; LVEF, left ventricular ejection fraction; LVESD, left ventricular end-systolic dimension; LVEDD, left ventricular end-diastolic systolic dimension; LVESV, left ventricular end-systolic volume; LVEDV, left ventricular end-diastolic volume; LVMI, left ventricular mass index; LVM, left ventricular mass; LVSV, left ventricular stroke volume; LVMD, left ventricular mechanical dispersion; PWT, posterior wall thickness; SBP, systolic blood pressure; TC, total cholesterol; TG, triglycerides; VLDL-C, very low-density lipoprotein cholesterol.

We studied the deformation properties of the left ventricle in the longitudinal direction in each apical view. The GLS values in each apical view did not differ significantly between the groups (P>0.05). The integral average GLS value was -12.98±3.34% in the group with PVCs and -13.49±3.43% in the group without PVCs (P=0.458). The LVMD was significantly higher in patients with PVCs than those without PVCs: 34.07±9.3 ms versus 9.8±5.54 ms (P=0.000).

Table 3.

The parameters of the standard TTE, ECG, and 2-D STE in the groups of patients, depending on the PVC gradings.

Variable	PVC Grade 0 (n=42)	PVC Grades I-II (n=20)	PVC Grade III (n=16)	Grades IV-V (n=22)	F	*P*-value
LVEDV, mL	100.3±28.5	108.7±34.2	121.6±35.7	118.32±42.1	2.1797	0.0954
LVESV, mL	54.9±24.6	62.5±33.04	69.7±34.4	70.3±38.4	1.5558	0.2052
LVSV, mL	45.4±11.56	47.0±10.0	41.8±10.1	48.0±10.9	1.1148	0.3470
LVEF, %	46.9±11.0	45.8±12.33	45.5±13.18	43.9±11.8	0.3149	0.8145
QT interval, ms	0.36±0.03	0.4±0.03	0.39±0.04	0.38±0.03	8.4594	0.0000
QTc interval, ms	403.5±25.6	414.9±39.2	409.5±85	424.7±43.4	1.0691	0.3660
LVGLS average, (-%)	13.49±3.43	13.7±3.36	12.3±3.83	12.54±3.27	0.8585	0.4655
LVMD, ms	9.3±5.57	15±8.37	34.0±6.30	49.8±12.8	129.6136	0.0000

GLS, global longitudinal strain; LVESV, left ventricular end-systolic volume; LVEDV, left ventricular end-diastolic volume; LVSV, left ventricular stroke volume; LVEF, left ventricular ejection fraction; LVMD, left ventricular mechanical dispersion.

Table 3 presents the parameters of two-dimensional transthoracic echocardiography in patients divided into four groups, taking into account the Lown grading system for PVCs. The average values of LVESV, LVEDV, LVSV, and LVEF were comparable and did not differ statistically between the groups. At the same time, the QT interval according to electrocardiography data had a significant difference when comparing the four groups (F=8.4594, *P*=0.0000); however, the QTc interval did not show intergroup differences (F=1.0691, *P*=0.3660). The analysis of the 2D-STE parameters showed the absence of statistically significant differences in the LVGLS (F=0.8585, *P*=0.4655) between the groups, but significant differences in the LVMD were noted. The minimal mean LVMD value (9.3±5.57 ms) was revealed in the group without PVCs. LVMD increased with the increasing the PVC grade, reaching maximum values in the subgroup with PVC Grades IV-V: 15±8.37 ms in the subgroup with PVC Grades I-II, 34.0±6.30 ms in the subgroup with PVC Grade III, 49.8±12.8 ms in the subgroup with PVC Grades IV-V (F=129.6136, *P*=0.000).

Discussion

Several studies have noted the importance of assessing MD and GLS of the left ventricle in predicting VA in the early period of acute MI. A study by Ersbøll et al.[18] included 849 patients (mean age 61.9 ± 12.0 years, 73% men) with MIs and LVEFs >40% within 48 h of admission for coronary angiography and found that semiautomated calculation of left ventricular GLS can identify high-risk subjects among these patients.

A prospective multicenter study by Haugaa et al.[15] included 569 patients >40 days after acute MI. In patients with VA, defined as sustained ventricular tachycardia or sudden death during a median of 30 months, compared with patients without VA, LVEF and GLS were reduced to 48±17% vs. 55±11% (*P*<0.01) and -14.8±4.7% vs. -18.2±3.7% (*P*=0.001), respectively. At the same time, mechanical dispersion was increased to 63±25 ms vs. 42±17 ms (*P*<0.001). Mechanical dispersion was an independent predictor of arrhythmic events, and the combination of MD and LVH showed the best positive predictive value for arrhythmic events.

A study by van der Bijl et al.[19] aimed to relate LVMD to long-term prognosis in a large cohort of patients with heart failure (n=1185) after 6 months of cardiac resynchronization therapy (CRT). At 6 months post-CRT, patients with LVMD ≤84 ms had lower rates of VA and mortality compared to those with LVMD >84 ms.

In our study, the GLS value did not differ depending on VA presence (12.98±3.34% in the group with VA and 13.49±3.43% in the group without VA, *P*=0.458). In contrast, the LVMD value was significantly higher in the post-MI patients with VA than those without VA: 34.07±9.3 ms versus 9.8±5.54 ms (*P*=0.000).

As the class of PVC increased, the mean values of the LVMD increased, reaching maximum values in the group with PVC Grades IV- V: 15±8,37 ms in the group with PVC Grades I-II, 34.0±6.30 ms in the group with PVC Grade III, 49.8±12.8 ms in the group with PVC Grades IV-V (P=0.000).

In conclusion, Left ventricular mechanical dispersion, determined by 2-D STE, is an important prognostic marker related to the development of ventricular arrhythmia for ventricular arrhythmia in post-MI patients with intermediate LVEF.

Competing Interests

The authors declare that they have no competing interests.

Ethical Considerations

The study protocol was reviewed and approved by the Ethics Committee of the Republican Specialized Centre of Cardiology. All participants provided written informed consent.

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