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Mechanisms and predictors of ischemic mitral regurgitation at rest and on exertion in patients at early stage of myocardial infarction

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Aim. Determination of the mechanisms and predictors of ischemic mitral regurgitation (IMR) at rest and on exertion in patients at early stage of myocardial infarction (MI).

Material and methods. Seventy-seven patients with inferoposterior MI and 79 patients with anteroseptal apical MI were examined on the 7th day at rest and after exertion. We determined the degree of IMR (according to the PISA method), posteromedial and anterolateral papillary muscle (PM) displacement, closure height of the mitral valve (MV), systolic and diastolic mitral valve orifice area, volume of the left ventricle (LV), LV contractility index, deformation of the infarction regions, general LV deformation, deformation and systolic dyssinchrony of the PM.

Results. IMR was more common in inferior MI (42% vs 28%). LV volumes in cases with anteroseptal apical MI and IMR were greater and LV deformation was less than in patients without IMR. In inferoposterior MI and IMR, differences were observed in the index of local contractility and function of the posteromedial PM. The differences in MI of both localizations and IMR compared with MI without IMR were the areas of the mitral orifice and dyssinchrony of the PM. The degree of IMR after exertion did not depend on the degree of IMR at rest. Predictors of IMR at rest in MI of both localizations were the apical displacement of MV closure and the area of the mitral orifice. In inferoposterior, posteromedial PM displacement, deformation of the infarcted areas, PM dyssinchrony were also predictors. In anteroseptal apical MI, the area of the mitral orifice was the predictor of IMR. Predictors of anteroseptal apical MI after physical exertion after inferior MI were mitral orifice areas, contractility index, displacement and deformation of the posteromedial PM. In anteroseptal apical MI, the IMR predictors were MV closure height and systolic area of mitral orifice.

Conclusion. The study confirms the significance of changing the spatial orientation of the MV structures in MI of both localizations, impaired regional contractility in inferoposterior MI and LV volume in anteroseptal apical MI at early stage of the disease.

Key words: ischemic mitral regurgitation; longitudinal deformation; papillary muscle dyssinchrony.

Relationships and Activities: not.

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Ischemic mitral regurgitation (IMR) is a complication of myocardial infarction (MI) and an independent predictor of morbidity and mortality [1, 2]. IMR develops in patients with normal mitral valve (MV) leaflets as a result of left ventricular (LV) dysfunction and remodeling, mitral annulus dilatation, and papillary muscle (PM) displacement [2, 3]. These processes depend both on the MI size and localization [4]. IMR is the result of a change in the geometric relationship of the LV and the MV apparatus, and it can change both in time and depending on the state of rest or physical activity [5].

The aim was to study the mechanisms of IMR and its changes at rest and during exertion in the early stage of MI with various localization.

Material and methods

The study included 77 patients with primary inferior-posterior myocardial infarction and 79 patients with acute anteroseptal-apical myocardial infarction aged 57±5 years, who were hospitalized within 12 hours from the MI onset.

The control group consisted of 50 healthy individuals of the same age, gender and weight.

There were following exclusion criteria: history of mitral insufficiency, structural changes of MV, aortic valve disease, arrhythmia, diabetes and kidney failure.

Patients underwent stenting of culprit artery.

Patients with MI of each localization were divided into two subgroups depending on the severity of mitral regurgitation: grade 0-I or grade I-IV.

Left ventricular ejection fraction (LVEF), left atrial systolic volume index, left ventricular mass index (LVMI), and LV global and regional contractility indices were calculated according to the American Society of Echocardiography guidelines using GE Vivid 7 ultrasound system [6].

The LV sphericity index was calculated as the ratio of LV end-systolic volume (x 100%) to hypothetical sphere volume (4/3) π(d/2)3, where 'd' is the diameter of LV long axis.

The MV coaptation height was assessed at end-diastole in a 3-chamber view. The surface area of apical mitral leaflet displacement, formed by the mitral annulus and leaflets, was measured at middle systole in a 3-chamber position.

Apical displacement of PM was measured for anterolateral and posteromedial PM, respectively (Figure 1, 2). The posterior and lateral displacement of PM was measured in the parasternal long axis view at the PM level [7].

Mitral valve orifice area (MVOA) at the systole and diastole was calculated using the ellipse equation π*r1*r2/4, where 'r1' and 'r2' are the anteroposterior and intercomissary diameters of the MV, respectively. The mitral annular fractional shortening was calculated by the equation 100% x (diastolic MVOA — systolic MVOA)/diastolic MVOA.

The grade of mitral regurgitation (MR) was assessed by the PISA method [8] with the determination of regurgitant orifice area (ROA) and regurgitant volume (RV). There were following grades of MR: I — RV <20 ml or ROA <0,20 cm^2; II — RV= 20-39 ml or ROA =0,20-0,29 cm^2; III — RV =40-59 ml or ROA =0,30-0,39 cm^2; IV — RV >60 ml or ROA >0,40 cm^2. The change in the IMR grade was determined by the ROA change ⩾0,1 cm^2.

The PM function was studied using the speckle tracking echocardiography in 4- and 3-chamber views to determine the longitudinal strain of both PM (Figure 3) [9]. The strain peaks and the time from QRS onset to the strain peak of both PM were measured. The difference of this value specifies the PM dissynchrony [10].

Segment and global LV strain were measured by particle tracking method (Figure 4). The strain of LV infarcted walls was determined as the ratio of the sum of the infarcted walls' segment strain and the number of analyzed segments.

Treadmill stress echocardiography was performed until symptom onset or heart rate reached 120 bpm. Images obtained during the first minute of rest. Contractility improvement was evaluated by increasing the contractility index ⩾1.

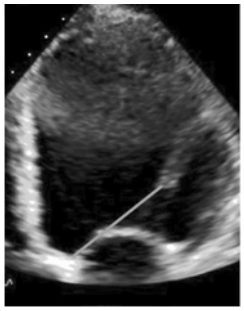

Figure 1. Determination of the apical displacement of anterior PM.

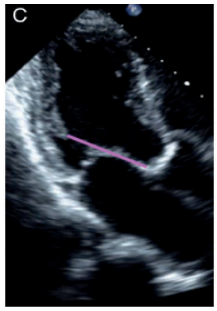

Figure 2. Determination of the apical displacement of posteromedial PM.

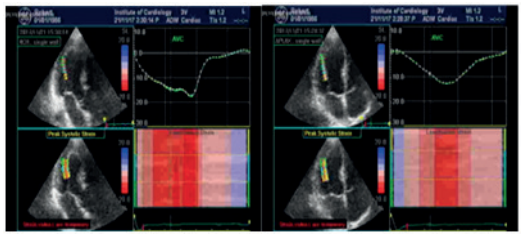

Figure 3. Measurement of PM strain and dyssynchrony.

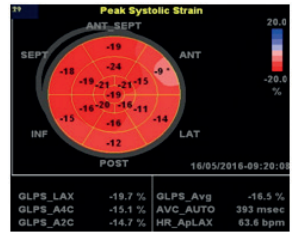

Figure 4. Determination of strain of LV and infarcted segments.

Statistical analysis was performed using the software package SPSS 21.0. Values are presented as m±SD. Continuous parameters were evaluated by Student's t-test and Mann-Whitney U-test. Differences were considered significant at $p<0,05$.

Variables were studied to determine the normal distribution and equal deviations using the Kolmogorov-Smirnov test.

Correlation analysis was performed to assess linear dependencies. Correlation between parameters were considered reliable at $R \geqslant 0,4$.

Binary regression analysis was used to identify independent predictors of IMR.

This study was performed in accordance with the Helsinki declaration and Good Clinical Practice standards. The medical ethics committees of all participating centers approved this study. All patients signed informed consent.

Results

The number of patients with inferior-posterior MI and IMR at rest was significantly higher than patients with anteroseptal apical MI and IMR. The data are shown in Table 1. In patients with MI of the same localization, the parameters differed depend-

Table 1

Clinical characteristics of patients with inferior-posterior and anteroseptal apical MI and IMR at rest

Parameters	Inferior-posterior MI	Anteroseptal apical MI	Control	P		
	A	B	C	A vs B	A vs C	B vs C
Demographic and clinical data						
Number of patients (n/%)	32/42	22/28	50	0,03	<0,001	<0,001
Age (years)	54±5	54±5	54±5	НД	НД	НД
Gender, women (%)	43,2	38,3	34,6	НД	НД	НД
BMI, kg/m^2	26,4±3,2	25,9±3,0	25,5±3,1	НД	НД	НД
Hypertension (%)	56,7	57,1	0	НД	НД	<0,001
Dyslipidemia (%)	72	76	32	НД	НД	<0,001
Drugs						
Beta blockers (%)	94	95	0	НД	НД	<0,001
ACE inhibitors (%)	92	97	0	НД	НД	<0,001
Statins (%)	83	85	0	НД	НД	<0,001
Spironolactone (%)	39	41	0	НД	НД	<0,001
Standard echocardiography						
ESVI (ml/m^2)	27,5±9,1	36,4±10,2	21,3±5,9	0,02	НД	0,01
EDVI (ml/m^2)	52,4±9,8	63,7±11,3	52,3±9,8	0,02	НД	0,01
SI (%)	16±5	16±9	15±7	НД	НД	НД
EF (%)	52,3±6,2	45,4±4,1	58,7±4,3	0,01	0,03	0,01
LVMI (g/m^2)	110±19,8	114±21,4	73,6±18,4	НД	0,04	0,04
LAVI (ml/m^2)	29,5±5,1	29,2±5,3	27,8±4,7	НД	НД	НД

Contractility (RCI, GCI)						
Inferior wall	5,8±1,5	4,3±1,1	3,0±0,0			
Inferior-septal wall	5,9±1,4	3,0±0,0	3,0±0,0	0,008	0,008	НД
Posterior wall	5,7±1,6	3,0±0,0	3,0±0,0	0,009	0,009	НД
Lateral wall	3,1±0,3	3,2±0,6	3,0±0,0	НД	НД	НД
Anterior wall	3,0±0,0	7,3±1,8	3,0±0,0	0,003	НД	0,003
Anterior-septal wall	3,0±0,0	6,9±1,9	3,0±0,0	0,004	НД	0,004
GCI	1,4±0,3	1,6±038	1,0±0,0	0,02		0,002
Echocardiography of mitral valve apparatus						
MV coaptation height (cm)	0,97±0,23	0,98±0,21	0,86±0,19	НД	0,03	0,03
Systolic mitral valve prolapse area (mm^2)	163,4±63,4	166,3±68,2	121,6±37,4	НД	0,01	0,01
Systolic MVOA (mm^2)	564,6±114,2	486,7±117,6	342,6±114,2	0,01	НД	НД
Diastolic MVOA (mm^2)	715,4±128,3	681,2±119,4	594,6±117,2	0,01	НД	НД
MAFS (%)	18±3	24±5	35±7	0,02	0,008	НД
Apical displacement of ALPM (cm)	3,28±0,86	3,64±0,97	3,12±0,81	НД	НД	НД
Apical displacement of PMPM (cm)	3,79±0,91	3,37±0,90	3,21±0,78	НД	НД	НД
PMPMpost (mm)	4,5±1,9	3,1±1,8	2,4±1,3	0,02	0,01	НД
PMPMlat (mm)	2,6±1,5	2,2±1,1	1,9±1,1	0,04	0,02	НД
ALPMpost (mm)	2,4±1,2	2,6±1,5	2,1±1,2	НД	НД	НД
ALPMlat (mm)	2,3±1,4	2,5±1,4	2,0±1,0	НД	НД	НД
Speckle tracking echocardiography						
LVLS (%)	-16,8±1,9	-14,6±1,8	-20,3±2,1	0,04	НД	0,01
ISLS (%)	-9,2±6	-9,7±3	—	НД	—	—
LS ALPM (%)	-13,9±1,2	-13,9±1,1	-15,8±1,7	НД	0,03	0,03
LS PMPM (%)	-13,8±1,3	-15,1±1,2	-15,6±1,7	0,02	0,01	НД
PMSD (ms)	38±11	42±14	16±9	НД	0,02	0,01

Abbreviations: ALPM — antero-lateral papillary muscle, ALPMlat/post — direction of anterolateral papillary muscle displacement, EDVI — end-diastolic volume index, EF — ejection fraction, ESVI — end-systolic volume index, GCI — global contractility index, IMR — ischemic mitral regurgitation, ISLS — longitudinal strain of infarcted segments, LAVI — left atrial volume index, LS — longitudinal strain, LVLS — left ventricular longitudinal strain, LVMI — left ventricular mass index, MAFS — mitral annular fractional shortening, MV — mitral valve, MVOA — mitral valve orifice area, PMPM — posteromedial papillary muscle, PMSD — papillary muscle systolic dyssynchrony, PMPMlat/post — direction of posteromedial papillary muscle displacement, RCI — regional contractility index, SI — sphericity index.

Table 2

Parameters with significant differences in patients with anteroseptal apical MI depending on the presence of IMR at rest

Parameters	No IMR (A)	IMR (B)	Control (C)	P		
				A vs B	A vs B	A vs B
ESVI (ml/m^2)	32,8±8,4	36,4±10,2	21,3±5,9	<0,03	<0,01	<0,01
EDVI (ml/m^2)	57,8±10,3	63,7±11,3	52,3±9,8	<0,02	<0,01	<0,01
Systolic MVOA (mm^2)	418,4±113,6	486,7±117,6	342,6±114,2	<0,05	<0,05	<0,02
Diastolic MVOA (mm^2)	656,3±121,3	681,2±119,4	594,6±117,2	<0,05	<0,05	<0,02
MAFS (%)	29±7	24±5	35±7	<0,05	<0,05	<0,01
LVLS (%)	-15,2±1,5	-14,6±1,8	-20,3±2,1	<0,03	<0,05	<0,01
PMSD (ms)	23±10	42±14	16±9	<0,01	<0,05	<0,01

Abbreviations: EDVI — end-diastolic volume index, ESVI — end-systolic volume index, IMR — ischemic mitral regurgitation, LVLS — left ventricular longitudinal strain, MAFS — mitral annular fractional shortening, MVOA — mitral valve orifice area, PMSD — papillary muscle systolic dyssynchrony.

Table 3

Parameters with significant differences in patients with inferior-posterior MI depending on the presence of IMR at rest

Параметры	No IMR (A)	IMR (B)	Control (C)	A vs B	A vs B	A vs B
Systolic MVOA (mm^2)	467,3±117,8	564,6±114,2	342,6±114,2	<0,02	<0,03	<0,01
Diastolic MVOA (mm^2)	697,2±124,2	715,4±128,3	594,6±117,2	<0,02	<0,03	<0,01
MAFS (%)	28±5	18±3	35±7	<0,01	<0,02	<0,003
PMSD (ms)	21±7	38±11	16±9	<0,001	<0,001	<0,001
RCI (inferior wall)	5,2±1,3	5,8±1,5	3,0±0,0	<0,05	<0,02	<0,01
RCI (inferior-septal wall)	4,6±1,1	5,9±1,4	3,0±0,0	<0,05	<0,02	<0,01
RCI (posterior wall)	4,8±1,2	5,7±1,6	3,0±0,0	<0,05	<0,03	<0,01
Apical displacement of PMPM (cm)	3,35±0,86	3,79±0,91	3,21±0,78	<0,03	НД	<0,03
PMPMpost (mm)	3,2±0,9	4,5±1,9	2,4±1,3	<0,02	<0,02	<0,01
PMPMlat (mm)	2,1±1,2	2,6±1,5	1,9±1,1	<0,05	<0,03	0,02
ISLS (%)	-12,7±4	-9,2±6	—	<0,03	—	—
LS PMPM (%)	-11,3±1,4	-13,8±1,3	-15,6±1,7	<0,02	<0,01	<0,02

Abbreviations: IMR — ischemic mitral regurgitation, ISLS — longitudinal strain of infarcted segments, MAFS — mitral annular fractional shortening, MVOA — mitral valve orifice area, LS — longitudinal strain, PMPM — posteromedial papillary muscle, PMPMlat/post — direction of posteromedial papillary muscle displacement, PMSD — papillary muscle systolic dyssynchrony, RCI — regional contractility index.

Table 4

ROA change after exertion in patients with inferior-posterior MI

Parameters	ROA change after exertion		P	Correlation with ROA	
	Decrease, n=12 (37,5%)	Increase, n=20 (62,5%)		R	P
Heart rate (bpm)	32±15	35±14	НД	0,11	0,61
SBP (mm Hg)	28±15	29±10	НД	0,19	0,34
ESV (ml)	-12±11	-13±12	НД	0,12	0,27
EDV (ml)	-5,3±11	0,8±19	НД	0,14	0,35
SI (%)	-3±4	-1±3	НД	0,12	0,39
EF (%)	7,3±6,4	7,9±6,8	НД	0,21	0,18
LAV (ml)	-1,3±2,3	-1,4±2,1	0,06	0,42	0,08
MV coaptation height (cm)	-0,21±0,18	0,07±0,11	<0,002	0,65	0,01
Systolic mitral valve prolapse area (mm^2)	-1,2±1,9	0,6±2,1	<0,002	0,74	<0,002
Systolic MVOA (mm^2)	-51,3±72,4	62,2±52,1	0,01	0,52	0,02
Diastolic MVOA (mm^2)	-42,4±68,3	51,6±48,3	0,01	0,48	0,02
MAFS (%)	2,3±11,4	-1,2±9,7	0,03	0,46	0,03
Apical displacement of ALPM (cm)	-3,4±2,1	-3,2±1,9	НД	0,31	0,17
Apical displacement of PMPM (cm)	-2,0±1,7	0,6±2,1	<0,03	0,57	<0,03
PMPMpost (mm)	-2,5±1,7	1,1±1,5	<0,002	0,67	<0,005
PMPMlat (mm)	-1,7±1,7	-0,5±1,5	<0,005	0,51	<0,005
ALPMpost (mm)	-2,1±1,5	0,7±1,6	<0,002	0,60	<0,002
ALPMlat (mm)	-1,3±1,0	0,9±1,1	НД	0,34	0,07
RCI (inferior wall)	-0,7±0,15	-0,3±0,12	<0,002	0,68	<0,002
RCI (inferior-septal wall)	-0,6±0,13	-0,2±0,14	<0,002	0,65	<0,002
RCI (posterior wall)	-0,5±0,12	-0,3±0,11	<0,05	0,47	<0,05
RCI (lateral wall)	-0,2±0,18	-0,1±0,11	НД	0,31	0.09
GCI	-0,6±0,14	-0,24±0,13	<0,002	0,64	<0,002

LS ALPM (%)	-1,3±0,7	-1,4±0,7	НД	0,28	0,12
ISLS (%)	3,7±5	-1,8±4	<0,003	0,68	0,004
LS PMPM (%)	-1,9±1,2	1,8±1,2	<0,001	0,78	<0,001
LVLS (%)	1,1±1,3	-0,8±1,8	НД	0,38	<0,07
PMSD (ms)	2,1±1,5	6,9±3,2	<0,01	0,53	<0,01

Abbreviations: ALPM — antero-lateral papillary muscle, ALPMlat/post — direction of anterolateral papillary muscle displacement, EDVI — end-diastolic volume index, EF — ejection fraction, ESV — end-systolic volume, EDV — end-diastolic volume, GCI — global contractility index, IMR — ischemic mitral regurgitation, ISLS — longitudinal strain of infarcted segments, LAV — left atrial volume, LAVI — left atrial volume index, LS — longitudinal strain, LVLS — left ventricular longitudinal strain, LVMI — left ventricular mass index, MAFS — mitral annular fractional shortening, MV — mitral valve, MVOA — mitral valve orifice area, PMPM — posteromedial papillary muscle, PMSD — papillary muscle systolic dyssynchrony, PMPMlat/post — direction of posteromedial papillary muscle displacement, RCI — regional contractility index, SBP — systolic blood pressure, SI — sphericity index.

Table 5

ROA change during exercise in patients with anteroseptal apical MI

Parameters	ROA change after exertion		P	Correlation with ROA	
	Decrease, n=10 (45,5%)	Increase, n=12 (54,5%)		R	P
Heart rate (bpm)	34±12	36±15	НД	0,14	0,68
SBP (mm Hg)	30±17	28±14	НД	0,21	0,42
ESV (ml)	-14±9	-12±8	НД	0,18	0,24
EDV (ml)	-5,7±10	1,1±17	НД	0,24	0,28
SI (%)	-4±4	-2±4	НД	0,27	0,21
EF (%)	6,5±6,1	7,4±6,3	НД	0,31	0,11
LAV (ml)	-1,2±2,4	1,1±2,0	0,07	0,39	0,09
MV coaptation height (cm)	-0,24±0,14	0,13±0,14	<0,002	0,64	0,002
Systolic mitral valve prolapse area (mm^2)	-1,3±2,1	0,8±2,0	<0,002	0,74	<0,001
Systolic MVOA (mm^2)	-46,3±68,7	31,6±53,1	0,04	0,47	0,03
Diastolic MVOA (mm^2)	-38,1±39,4	37,4±42,6	0,03	0,43	0,04
MAFS (%)	1,1±9,3	-0,9±8,2	0,04	0,42	0,04
Apical displacement of ALPM (cm)	-3,1±1,9	1,2±1,7	<0,003	0,58	0,002
Apical displacement of PMPM (cm)	-2,2±2,1	-0,6±1,2	<0,05	0,51	<0,05
PMPMpost (mm)	-1,5±1,2	1,4±1,6	<0,004	0,63	0,03
PMPMlat (mm)	-1,9±1,7	-1,5±1,2	НД	0,29	0,23
ALPMpost (mm)	-1,7±1,3	0,9±1,4	<0,002	0,63	0,03
ALPMlat(mm)	-1,4±1,1	0,9±1,1	НД	0,54	0,06
RCI (lateral wall)	-0,1±0,19	-0,2±0,13	НД	0,31	0,09
RCI (anterior wall)	-0,5±0,11	-0,3±0,10	НД	0,23	0,08
RCI (anterior-septal wall)	-0,4±0,16	-0,2±0,15	НД	0,34	0,09
GCI	-0,28±0,12	-0,21±0,15	НД	0,21	0,18
LS ALPM (%)	-1,3±0,7	-1,4±0,7	НД	0,28	0,09
LS PMPM (%)	-2,2±1,1	-1,9±1,3	НД	0,22	0,14
ISLS (%)	1,3±0,9	0,7±0,8	НД	0,32	0,09
LVLS (%)	6,8±1,7	1,3±1,4	<0,003	0,65	<0,002
PMSD (ms)	2,3±1,3	2,5±1,6	НД	0,21	0,14

Abbreviations: ALPM — antero-lateral papillary muscle, ALPMlat/post — direction of anterolateral papillary muscle displacement, EDVI — end-diastolic volume index, EF — ejection fraction, ESV — end-systolic volume, EDV — end-diastolic volume, ESVI — end-systolic volume index, GCI — global contractility index, IMR — ischemic mitral regurgitation, ISLS — longitudinal strain of infarcted segments, LAV — left atrial volume, LAVI — left atrial volume index, LS — longitudinal strain, LVLS — left ventricular longitudinal strain, MAFS — mitral annular fractional shortening, MV — mitral valve, MVOA — mitral valve orifice area, PMPM — posteromedial papillary muscle, PMSD — papillary muscle systolic dyssynchrony, PMPMlat/post — direction of posteromedial papillary muscle displacement, RCI — regional contractility index, SBP — systolic blood pressure, SI — sphericity index.

Table 6

Predictors of IMR (ROA) at rest on the 7th day of MI

Parameters	Inferior-posterior MI	Anteroseptal apical MI
MV coaptation height	0,03	0,01
Systolic mitral valve prolapse area	0,01	0,02
Systolic MVOA	0,01	0,04
Diastolic MVOA	0,03	0,03
MAFS	0,04	0,04
Apical displacement of PMPM	0,001	0,09
ISLS	0,001	0,12
PMSD	0,01	0,05
EDVI	0,23	0.002
ESVI	0,31	0.01
R^2	0,68	0,65

Abbreviations: EDVI — end-diastolic volume index, ESVI — end-systolic volume index, IMR — ischemic mitral regurgitation, ISLS — longitudinal strain of infarcted segments, MAFS — mitral annular fractional shortening, MV — mitral valve, MVOA — mitral valve orifice area, PMPM — posteromedial papillary muscle, PMSD — papillary muscle systolic dyssynchrony.

Table 7

Предикторы ИМР (ПРО) после нагрузки

Parameters	Inferior-posterior MI	Anteroseptal apical MI
MV coaptation height	0,005	0,001
Systolic mitral valve prolapse area	0,0001	0,002
Systolic MVOA	0,001	0,01
PMPMpost	0,0001	0,04
Apical displacement of PMPM	0,02	0,07
LS PMPM	0,0001	0,09
ISLS	0,004	0,08
R^2	0,73	0,71

Abbreviations: IMR — ischemic mitral regurgitation, ISLS — longitudinal strain of infarcted segments, MV — mitral valve, MVOA — mitral valve orifice area, PMPM — posteromedial papillary muscle, PMPMlat/post — direction of posteromedial papillary muscle displacement.

ing on the presence of IMR (Tables 2, 3). Changes of IMR grade after exertion in patients with MI of both localizations did not depend on the IMR grade at rest. These changes after exertion are shown in Tables 4, 5.

Predictors of IMR at rest and after exertion differed in patients with MI of both locations (Tables 6, 7).

Discussion

The study is devoted to identifying the mechanisms of early IMR at rest and during exercise in patients with MI of various localization. Studies of IMR both in the early period of MI, and depending on the MI localization, are few. The prevalence of IMR in patients with MI is 50%; 38% is characterized by moderate severity, and 12% — moderate-severe and severe [2]. IMR is diagnosed between 7 and 30 days after onset of MI [4].

We performed the study on the 7th day of MI for early detection of changes that contribute to IMR development. According to our data, IMR is present in the early stages of MI and more often in patients with inferior-posterior MI. To study LV contractility and PM function, the speckle tracking echocardiography with strain assessment was used. According to the study, the deformation of infarcted segments was a predictor of IMR for inferior-posterior MI, and the PM dyssynchrony — IMR predictor for MI of both localizations. Little research has been done on PM strain and dyssynchrony [9, 10], and there are no studies on the early period of MI. According to our data, PM dyssynchrony >30 ms is a predictor of IMR.

The aim of the study was also to determine the mechanisms and predictors of IMR during exertion.

The contractility change during exertion alters the impact on the MV coaptation. During exertion, changes in LV geometry can alter the orientation of the MV structures and IMR severity. On the other hand, Increased contractility of PM can aggravate MV leaflets displacement and exacerbate the IMR. IMR at rest depends on ROA, the systolic pressure gradient, and the duration of systole [5]. During exercise, the systolic pressure gradient increases, the systole duration decreases, and the RV becomes dependent mainly on the ROA.

According to our data, the IMR severity during exertion in patients with MI does not depend on the IMR severity at rest.

In patients of both subgroups, with a IMR severity change, the same increase in heart rate and systolic blood pressure was observed. Thus, systolic shortening does not affect the IMR severity. The initial LV sizes, which play a role in the IMR onset at rest in anteroseptal apical MI, did not affect the IMR severity and were not predictors of IMR changes during exertion.

In fact, IMR severity change during exertion does not depend on the LV size and function, but depends on the MV geometry.

Indeed, MV coaptation height and the systolic area under MV leaflets during exertion correlated with the ROA and were predictors of IMR severity. The systolic mitral valve orifice area was a predictor of IMR severity changes in during exertion in patients with MI of both localizations.

An aggravation in the apical displacement of anterior PM during exertion was associated with an IMR severity increase in patients with anteroseptal apical MI and correlated with the ROA. However, this parameter did not affect the IMR severity at rest in these patients.

The direction of the regurgitant jet was different in patients of both groups. Patients with inferior-posterior MI had eccentric regurgitation; in patients with anteroseptal apical MI, the jet direction was more central. In these patients, the apical displacement of both PM leads to a more symmetrical displacement, which provides a central direction of IMR. Contractility impairment of LV segments in patients with inferior-posterior MI can lead to a greater displacement of the posteromedial PM, providing asymmetric displacement of MV leaflets and eccentric IMR.

IMR severity decrease during exertion was more often observed in patients with inferior-posterior MI due to improved contractility of the infarcted zone, which led to MVOA decrease, an improvement of the mitral annular function, and orientation of the posteromedial PM.

In patients with inferior-posterior MI, posteromedial PM strain augment during exertion increased the IMR severity due to the greater posterior displacement of MV leaflets. In patients with anteroseptal apical MI, a change in the strain of both PM did not affect the IMR severity during exertion, and a change in LV deformation led to alteration in IMR severity.

Thus, in patients with MI of early stage, IMR is dynamic in nature, and its intensity varies at rest and during exercise. The parameters that correlate with the presence and severity of IMR at rest and during exertion, like the IMR predictors, are different.

Relationships and Activities: not.

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