e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2023; 15(8); 542-547

Original Research Article

A Hospital Based Assessment of ST Elevation Myocardial Infarction of Inferior Wall and Right Ventricle in Rheumatic Mitral Stenosis Due to Thrombus at Right Coronary Sinus

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Received: 15-06-2023 Revised: 20-07-2023 / Accepted: 24-08-2023
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Conflict of interest: Nil

Abstract

Aim: The aim of the present study was to evaluate ST elevation myocardial infarction of inferior wall and right ventricle in rheumatic mitral stenosis due to thrombus at right coronary sinus.

Methods: The present study was conducted in the Department of Cardiology, Acute MI was diagnosed by the presence of at least 2 of the following criteria: electrocardiographic changes, significant rises in myocardial bound creatine kinase fraction, and typical chest pain. Inferior wall MI was diagnosed by electrocardiography, echocardiography and coronary angiography. In patients with non-ST elevation MI, echocardiography and coronary angiographic findings were used for determination of the diagnosis of inferior wall MI. There were total 200 patients included in the present study.

Results: There were no differences in age, sex and other frequencies of underlying diseases among the 3 groups. There were no differences in the modality of intervention, severity of coronary artery disease. 147 (73.50%) patients had the culprit lesion in the right coronary artery and 53 (26.50%) patients had the culprit lesion in the left circumflex artery. Patients whose culprit lesion in the left circumflex artery had an increased frequency of more severe MR than those with the culprit lesion in the right coronary artery, but this difference did not reach statistical significance. There were no significant differences in annular area within the 3 groups. However, patients with mild or moderate MR had larger tenting area than those without MR.

Conclusion: In the acute phase of inferior wall MI, MR was associated with LV systolic dysfunction with tethering. Therefore, it can be suggested that reduced closing force as a consequence of LV systolic dysfunction in the presence of leaflet tethering would play a more pivotal role in the development of MR in the acute phase of inferior MI, whereas increased tethering forces through a combination of annular dilation and geometric remodeling of the LV would be more important contributor in the chronic phase.

Keywords: Mitral valve, Myocardial infarction, mitral stenosis

Introduction

Acute myocardial infarction (AMI) is very common in clinic, and is often caused by incomplete coronary occlusion or complete occlusion induced by acute plaque rupture. [1] Coronary angiography plays an important role in the etiology diagnosis and treatment of AMI. [2] In previous studies, 1–12% of patients with AMI who underwent coronary angiography did not develop an irregular lumen, or coronary artery stenosis of <50%. [3,4] However, most of the previous studies excluded the patients with MI in the acute phase. Thus, the mechanism of MR during acute phase of inferior MI is unclear. We hypothesized the mechanism of MR during acute phase could be different from that of MR during chronic phase and there could be more contributing factors than local remodeling in patients with inferior MI because of the influence of different chronicity of MI and the diversity of mechanism of MR.

Cardiac magnetic resonance (CMR) allows a complete and accurate assessment of LV status in patients after acute MI and late gadolinium-enhanced CMR visualizes and quantifies infarct size. [5,6] However, few studies investigated the relationship between infarct size by CMR and the severity of MR. Ischemic mitral regurgitation (MR) is MR due to complications of coronary artery disease and not with intrinsic valve disease such as rheumatic or degenerative mitral valvular disease. [7] It is common and clinically important because it increases mortality even when mild, with a graded relationship between severity and reduced survival. [8] Ventricular remodeling with papillary muscle (PM) displacement has been known to be an important mechanism of ischemic MR, especially in patients with inferior wall myocardial Infarction (MI). Therefore, MR occurs at a higher incidence in patients with inferior MI compared with those with anterior MI, despite its less severe left ventricular (LV) remodeling, because of the greater displacement of posterior PM caused by localized inferior basal LV remodeling. [9,10]

The aim of the present study was to evaluate ST elevation myocardial infarction of inferior wall and right ventricle in rheumatic mitral stenosis due to thrombus at right coronary sinus.

Methods

The present study was conducted in the Department of Cardiology, Rajendra Institute of Medicine Sciences (RIMS), Ranchi, Jharkhand, India for two years Acute MI was diagnosed by the presence of at least 2 of the following criteria: electrocardiographic changes, significant rises in myocardial bound creatine kinase fraction, and typical chest pain. Inferior wall MI was diagnosed by electrocardiography, echocardiography and coronary angiography. In patients with non-ST elevation MI, echocardiography and coronary angiographic findings were used for determination of the diagnosis of inferior wall MI. There were total 200 patients included in the present study.

Echocardiographic Measurements

LV end-diastolic and end-systolic cavity was traced in the apical 4-chamber and 2-chamber views, and LV volume was obtained by using the modified biplane Simpson method. LV ejection fraction was calculated from the LV end-diastolic and end-systolic volumes. Regional wall motion was assessed by assigning a segmental score to each of the 17 LV segments. [11] All segment scores were added and divided by the number of segments analyzed to obtain the regional wall motion score index (RWSI). [12] LV sphericity was assessed by using the LV short-axis/long-axis dimension ratio in the end systolic apical 4-chamber view. [13] Left atrial (LA) volume was calculated using the prolate ellipsoid model. [14] From mitral Doppler tracings with the sample volume at the mitral leaflet tips, the following variables were measured: peak velocity of early rapid filling wave (E), peak flow velocity at atrial contraction (A), E/A ratio, and deceleration time of early filling. A restrictive LV filling pattern was defined as an E/A ratio >2, with a deceleration time of <150 ms.14 Early and late diastolic tissue Doppler velocities (E' and A') were measured at the medial mitral annulus using a tissue Doppler image. MI systolic mitral annular dimension was measured in the apical 4- and 2-chamber views, and its area was calculated by using an ellipsoid assumption (annular area = $d1 \times d2 \times \pi/4$). The mitral leaflet-tenting area between the leaflets and the line connecting the annular hinge points in the apical 4-chamber view was traced at MI systole to estimate the apical displacement of the mitral leaflets.

The leaflet-tethering distance between the PM tips and the contralateral anterior mitral annulus was also measured in the apical 4- and 2-chamber views (Figure 1, L1 and L2) to estimate PM displacement. The severity of MR was determined by the ratio of color Doppler jet area to LA area at MI systole. MR grade was estimated as mild, moderate, or severe on the basis of ratios of greater than 10% to 20%, greater than 20% to 40%, and greater than 40%, respectively.

CMR Measurements

CMR was performed within 3.9±1.7 days (range 1–7) after acute MI using a 1.5 Tesla (T) imaging unit (Gyroscan Intera, Philips Medical Systems, Netherlands) equipped with a dedicated cardiac software package and a dedicated cardiac phased-array surface coil. Delayed enhancement images was performed using a segmented inversion recovery radiofrequency spoiled gradient echo (T1-TFE) sequence (typical TR/TE =5.3/1.6 ms, flip angle =15°, slice thickness =10 mm, field of view =360 mm, matrix size = 512 × 512, number of signal average =2) 10 min after the intravenous injection of gadolinium-diethylene-triamine-pentaacetic acid at a dose of 0.2 mmol/kg body weight. The inversion time was determined by a dedicated TI-determining sequence (Look- Locker) and ranged from 220 to 300 ms. Contiguous end-diastolic short-axis slices of the LV were acquired from base to apex without gaps (8–10 slices in number) to cover the whole LV. For quantitative analysis, we used the scanner's workstation using commercially available software (View Forum, version 4.1, Philips Medical Systems). Epicardial and endocardial contours of the entire LV were manually traced for determining the LV mass. Each slice was divided into 12 circumferential segments on up to 6 short axis views. The region of hyper-enhancement area (infract area) was semi-automatically defined by the software program using automatic thresholding technique. Manual adjustments of the infarct region of interest were made where the computer algorithm failed to correctly delineate the infarct area. The same density (1.05 g/cm3) was assumed for both hyperenhanced and non-hyperenhanced myocardium. LV mass and infarct mass were indexed to body surface area. Infarct size was defined as the total amount of hyper-enhancement area in all short axis slices and expressed as a percentage of LV mass. The extent of hyperenhanced area within each segment (referred to as the infarct transmurality) was defined as percent of the hyperenhanced myocardium to total area of the involved segment of the myocardium. Infarct transmurality was defined as percent of the hyper-enhanced myocardium to total area of the involved myocardium and transmurality index was graded as 1, 2, 3, or 4 based on its occupation of 1–25%, 26–50%, 51–75%, or 76–100% of the myocardium, respectively. The mean transmurality index for each patient was calculated as the average of all segments with of a grade >1.

Statistical Analysis

All continuous variables were presented as mean ± standard deviation and compared by analysis of variance. Discrete variables were compared using $\chi 2$ analysis or the Fisher exact test, as appropriate. Independent correlates of the severity of MR were identified by forward stepwise multivariable regression (forward method, with $P<0.05$ for entrance into the model and $P>0.10$ for removal from the model). The variables entered in the multivariable model were LV ejection fraction, tenting area, LV end-systolic volume index and infarct size, which were significant variables in the univariate analysis. A value of P less than 0.05 was considered significant.

Results

Table 1: Baseline Clinical Characteristics

MR	None (n=150)	Mild (n=30)	Moderate (n=20)	P value
Age (years)	59.3±13.4	60.8±8.82	63.7±10.5	0.472
Male	123 (82%)	25 (83.34%)	6 (60%)	0.222
BSA (m2)	1.78±0.12	1.76±0.14	1.68±0.12	0.032
Body mass index (kg/m2)	24.0±3.7	23.8±2.7	23.2±2.8	0.165
STEMI	63 (42%)	20 (66.66%)	7 (35%)	0.4675
Hypertension	54 (36%)	18 (60%)	8 (40%)	0.612
Diabetes mellitus	39 (26%)	5 (16.66%)	5 (25%)	0.575
Dyslipidemia	63 (42%)	12 (40%)	6 (30%)	0.560
Severity of coronary artery disease				
1-vessel	54 (36%)	10 (33.33%)	4 (20%)	
2-vessel	51 (34%)	10 (33.34%)	8 (40%)	
3-vessel	45 (30%)	10 (33.33%)	8 (40%)	
Culprit lesion				
Right coronary artery	114 (76%)	18 (60%)	15 (75%)	0.442
Left circumflex artery	36 (24%)	12 (40%)	5 (25%)	
Lipid profile				
Total cholesterol	178.2±41.9	181.9±32.6	166.4±48.2	0.555
LDL cholesterol	115.5±38.0	108.2±27.3	112.0±44.6	0.789
HDL cholesterol	36.4±8.0	42.4±11.3	39.6±7.0	0.309

There were no differences in age, sex and other frequencies of underlying diseases among the 3 groups. There were no differences in the modality of intervention, severity of coronary artery disease. 147 (73.50%) patients had the culprit lesion in the right coronary artery and 53 (26.50%) patients had the culprit lesion in the left circumflex artery. Patients whose culprit lesion in the left circumflex artery had an increased frequency of more severe MR than those with the culprit lesion in the right coronary artery, but this difference did not reach statistical significance.

Table 2: Echocardiographic Characteristics

MR	None (n=150)	Mild (n=30)	Moderate (n=20)	P value
Mitral deformation				
Tenting area/BSA (cm2/m2)	0.62±0.22	0.78±0.22	0.82±0.22	0.004
Annular area/BSA (cm2/m2)	4.18±1.32	4.24±1.26	4.04±1.36	0.912
LV global and local systolic function				
LV ejection fraction (%)	52.3±10.5	46.0±9.7	42.8±12.8	0.003
Total RWSI	1.42±0.28	1.56±0.34	1.68±0.22	0.027
Inferior RWSI	1.88±0.62	2.06±0.66	2.16±0.54	0.228
Posterior RWSI	1.46±0.54	1.65±0.75	1.86±0.64	0.055
LV diastolic function				
E (cm/s)	73.5±18.4	76.7±18.2	68.2±19.3	0.510
A (cm/s)	69.6±20.5	67.3±23.3	78.2±33.0	0.392
E/A	1.20±0.64	1.30±0.53	0.95±0.28	0.280
Deceleration time (ms)	178.2±34.7	173.7±36.8	168.2±32.4	0.660
Restrictive LV filling	6 (4%)	2 (6.66%)	2 (10%)	0.820
E' (cm/s)	6.0±1.8	5.4±1.6	5.3±1.8	0.210
A' (cm/s)	7.8±1.6	7.1±2.3	6.8±2.2	0.210
E/E' (cm/s)	14.2±5.4	14.6±5.6	15.6±7.8	0.340
LV global remodeling				
Sphericity	0.48±0.08	0.48±0.07	0.46±0.11	0.680
LVEDVI (ml/m2)	52.9±16.1	60.6±16.2	66.4±35.8	0.075
LVESVI (ml/m2)	25.3±8.8	29.7±10.4	34.7±16.8	0.016
LV local remodeling				
Annular to APM/BSA (mm/m2)	26.2±4.1	28.0±2.9	27.9±5.3	0.180
Annular to PPM/BSA (mm/m2)	25.0±3.5	25.9±3.3	26.2±4.5	0.450
LA remodeling				
LAVI (ml/m2)	26.1±6.4	30.3±5.4	30.5±7.4	0.012

Mitral Deformation

There were no significant differences in annular area within the 3 groups. However, patients with mild or moderate MR had larger tenting area than those without MR.

LV Systolic and Diastolic Function

Regarding LV systolic function, there was a graded relationship between the severity of MR and LV ejection fraction. Patients with moderate MR had more decreased LV systolic function than those without MR. Patients with moderate MR had significantly more increased total RWSI than those without MR and patients with moderate MR had decreased wall motion at inferior/posterior wall than those without MR but this did not reach a statistical significance in the inferior wall.

With regard to LV diastolic function, there were no significant differences in E, A, E/A, deceleration time of E velocity, E', A' and E/E' between the 3 groups. There were also no significant differences in the frequency of restrictive LV filling.

LV Global and Local Remodeling

With regard to LV global remodeling, there were no significant differences in sphericity of LV. However, there was a graded relationship between the severity of MR and LV dimension. Patients with moderate MR had larger end-systolic volume than those with mild MR and without MR, although in the case of LV end-diastolic volume it was not significant statistically. In contrast to LV global remodeling, there was no relationship between the severity of MR and tethering distance between the both PM tips and the contralateral anterior mitral annulus within the 3 groups suggesting no relationship between ischemic MR and local remodeling of LV.

Discussion

There are three possibilities of chest pain in a patient with mitral stenosis, severe pulmonary hypertension secondary to the pulmonary vascular disease concomitant coronary atherosclerosis coronary obstruction caused by coronary embolization. [15] Mitral stenosis presenting for the first time as acute STEMI is rare.

There were no differences in age, sex and other frequencies of underlying diseases among the 3 groups. There were no differences in the modality of intervention, severity of coronary artery disease. 147 (73.50%) patients had the culprit lesion in the right coronary artery and 53 (26.50%) patients had the culprit lesion in the left circumflex artery. Patients whose culprit lesion in the left circumflex artery had an increased frequency of more severe MR than those with the culprit lesion in the right coronary artery, but this difference did not reach statistical significance. In the study by Prizel et al, coronary artery embolic infarcts comprised 13% of the autopsy-studied infarcts. Underlying diseases predisposing to coronary emboli included valvular heart disease (40%), cardiomyopathy (29%), coronary atherosclerosis (16%), and chronic atrial fibrillation (24%). Mural thrombi were present in 18 (33%).[16] Myocardial infraction, clinically diagnosed in 15 (27%) patients, caused death in 11 (20%). Most emboli involved the left coronary artery and lodged distally, causing infarcts that were usually transmural. Because of their distal location and recanalization, coronary emboli may be a cause of infarcts with angiographically normal coronaries. Thus, coronary emboli are not rare, may produce signs and symptoms indistinguishable from atherosclerotic coronary disease, and by lodging distally in coronary arteries that are usually previously normal, they most often cause small but transmural myocardial infarction. [16]

There were no significant differences in annular area within the 3 groups. However, patients with mild or moderate MR had larger tenting area than those without MR. Regarding LV systolic function, there was a graded relationship between the severity of MR and LV ejection fraction. Patients with moderate MR had more decreased LV systolic function than those without MR. Patients with moderate MR had significantly more increased total RWSI than those without MR and patients with moderate MR had decreased wall motion at inferior/posterior wall than those without MR but this did not reach a statistical significance in the inferior wall. With regard to LV diastolic function, there were no significant differences in E, A, E/A, deceleration time of E velocity, E’, A’ and E/E’ between the 3 groups. There were also no significant differences in the frequency of restrictive LV filling. With regard to LV global remodeling, there were no significant differences in sphericity of LV. However, there was a graded relationship between the severity of MR and LV dimension. Patients with moderate MR had larger end-systolic volume than those with mild MR and without MR, although in the case of LV end-diastolic volume it was not significant statistically. In contrast to LV global remodeling, there was no relationship between the severity of MR and tethering distance between the both PM tips and the contralateral anterior mitral annulus within the 3 groups suggesting no relationship between ischemic MR and local remodeling of LV.

Previously, a number of groups proved PM displacement tethered the mitral leaflets into the LV and restricted their ability to coapt effectively at the level of mitral annulus. It has been known that MR occurs in higher incidence for patients with inferior MI compared with those with anterior MI in spite of less LV remodeling, because localized inferior basal LV remodeling in patients with inferior MI can potentially cause greater geometric changes in the mitral valve apparatus with displacement of posterior PM, despite lesser global LV remodeling and dysfunction than that seen in patients with anterior MI. [9]

Our results are also consistent with previous study that LV dysfunction without dilatation fails to produce significant MR. [17] Although LV systolic dysfunction was the most important factor as a determinant of the severity of MR, increased tenting area was also significant in multivariable regression analysis. MR in the acute phase of MI conveys adverse prognosis similarly in the chronic phase by doubling mortality after MI and the development of heart failure. [8,18] Until now, it is certain that ischemic MR is an independent predictor of outcome, but the mechanism linking MR and the outcome is not well understood. Probably, the development of heart failure secondary to the development of ischemic MR is one of the main links between both entities. Thus, the development of ischemic MR leads to the development of heart failure and cardiac death. [19,20]

Conclusion

Although rear cause of chest pain in mitral stenosis is myocardial infraction, we should keep in mind to evaluate the cause of chest pain in mitral stenosis patient. In the acute phase of inferior wall MI, MR was associated with LV systolic dysfunction with tethering. Therefore, it can be suggested that reduced closing force as a consequence of LV systolic dysfunction in the presence of leaflet tethering would play a more pivotal role in the development of MR in the acute phase of inferior MI, whereas increased tethering forces through a combination of annular dilation and geometric remodeling of the LV would be more important contributor in the chronic phase.

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