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Original research article

A HOSPITAL BASED OBSERVATIONAL EVALUATION OF EVALUATE 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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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, Burdwan medical college and hospital, West Bengal, India. 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 was total 100 patients included in the present study.

Results: Echocardiography was performed within 1.7±1.4 days (range 0–5) after acute MI. 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. Fifty-five (73.34%) patients had the culprit lesion in the right coronary artery and 20 (26.6%) 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 (P=0.420).

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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

Perioperative myocardial infarction (MI) during mitral valve surgeries, namely mitral valve repair and mitral valve replacement (MVR) is more common within the first year (incidence around 0.5%–1%).[1] Acute myocardial infarction (AMI) is a major cause of death and disability worldwide. When spontaneous AMI occurs, there is a >90% chance that the underlying aetiology is primarily due to coronary events such as plaque rupture, erosion, or dissection referred to as myocardial infarction (MI) type 1. MI can also occur secondary to an ischemic insult in the absence of overt coronary artery disease (CAD), by an imbalance between myocardial oxygen supply and demand termed type 2 MI.[2] Coronary artery embolism (CE) in which a thrombus arising from sources other than the coronary vasculature propagates into the coronary arteries causing AMI falls in this second category.

Mitral Stenosis (MS) is the most frequent cause of systemic emboli, where the presence of atrial fibrillation (AF) may increase the risk of embolic events. A few data were examined on the incidence of coronary embolism in MS patients with or without AF. Coronary angiography plays an important role in the etiology diagnosis and treatment of AMI.[3] 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%.[4,5] In this type of AMI, causes of infarct-related artery occlusion vary from person to person, such as increased platelet or coagulation activity (hypercoagulability) [6-8], coronary artery spasm[9], coronary artery embolism[10,11], as well as coronary myocardial bridge, coronary artery ectasia and coronary artery dissection.[12]

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.[13] It is common and clinically important because it increases mortality even when mild, with a graded relationship between severity and reduced survival.[14]

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.[15,16]

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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.

MATERIALS AND METHODS

The present study was conducted in the Department of Cardiology, Burdwan Medical College and Hospital, West Bengal, India for six months

Methodology

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 was total 100 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. All segment scores were added and divided by the number of segments analyzed to obtain the regional wall motion score index (RWSI).10 LV sphericity was assessed by using the LV short-axis/long-axis dimension ratio in the end systolic apical 4-chamber view. Left atrial (LA) volume was calculated using the prolate ellipsoid model. 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. Mid 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 mid 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

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at mid 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 were 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 scan ner'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.

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RESULTS

Table 1: Baseline Clinical Characteristics

MR	**None (n=75)**	**Mild (n=15)**	**Moderate (n=10)**	**P value**
Age (years)	58.9±12.1	58.9±9.9	62.4±11.3	0.450
Male	60 (80%)	12 (80%)	6 (60%)	0.230
BSA (m2)	1.79±0.18	1.77±0.15	1.65±0.13	0.025
Body mass index (kg/m2)	25.0±3.3	23.9±2.5	23.4±2.6	0.170
STEMI	30 (40%)	9 (60%)	3 (30%)	0.460
Hypertension	27 (33.34%)	8 (53.34%)	4 (40%)	0.620
Diabetes mellitus	18 (24%)	2 (13.34%)	2 (20%)	0.550
Dyslipidemia	30 (40%)	6 (40%)	3 (30%)	0.542
Severity of coronary artery disease				
1-vessel	28 (37.34%)	5 (33.33%)	2 (20%)	
2-vessel	26 (34.66%)	5 (33.34%)	4 (40%)	
3-vessel	21 (28%)	5 (33.33%)	4 (40%)	
Culprit lesion				
Right coronary artery	55 (73.34%)	10 (66.66%)	7 (70%)	0.420
Left circumflex artery	20 (26.66%)	5 (33.34%)	3(30%)	
Lipid profile				
Total cholesterol	179.3±42.7	180.9±33.5	165.3±48.5	0.540
LDL cholesterol	116.7±39.0	109.3±26.3	111.0±43.7	0.760
HDL cholesterol	37.8±8.0	41.3±11.2	38.5±6.0	0.350

Echocardiography was performed within 1.7±1.4 days (range 0–5) after acute MI. 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. Fifty-five (73.34%) patients had the culprit lesion in the right coronary artery and 20 (26.6%) 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 (P=0.420).

Table 2: Echocardiographic Characteristics

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MR	None (n=75)	Mild (n=15)	Moderate (n=10)	P value
Mitral deformation				
Tenting area/BSA (cm2/m2)	0.61±0.21	0.79±0.23	0.80±0.21	0.003
Annular area/BSA (cm2/m2)	4.19±1.31	4.23±1.27	4.02±1.34	0.9
LV global and local systolic function				
LV ejection fraction (%)	52.1±10.6	48.0±9.6	41.7±11.6	0.002
Total RWSI	1.41±0.29	1.54±0.37	1.66±0.23	0.025
Inferior RWSI	1.87±0.65	2.08±0.64	2.15±0.57	0.22
Posterior RWSI	1.45±0.53	1.64±0.78	1.88±0.64	0.05
LV diastolic function				
E (cm/s)	73.3±18.7	76.4±18.1	68.1±19.1	0.505
A (cm/s)	68.5±20.6	66.2±23.1	77.3±32.0	0.38
E/A	1.20±0.64	1.30±0.53	0.95±0.28	0.28

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Deceleration time (ms)	178.1±34.6	172.8±37.9	169.3±31.3	0.66
Restrictive LV filling	3 (4%)	1 (6.66%)	1 (10%)	0.82
E' (cm/s)	6.0±1.7	5.3±1.5	5.2±1.9	0.21
A' (cm/s)	7.8±1.6	7.1±2.3	6.8±2.2	0.21
E/E' (cm/s)	13.1±5.3	14.7±5.5	15.8±7.9	0.34
LV global remodeling				
Sphericity	0.47±0.09	0.46±0.08	0.44±0.11	0.68
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	0.18
Annular to PPM/BSA (mm/m2)	25.0±3.5	25.9±3.3	26.2±4.5	0.45
LA remodeling				
LAVI (ml/m2)	26.1±6.4	30.3±5.4	30.5±7.4	0.012

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Mitral Deformation

There were no significant differences in annular area within the 3 groups (P=0.900). However, patients with mild or moderate MR had larger tenting area than those without MR (0.79 or 0.80 vs. 0.61, P=0 .004 and 0.007).

LV Systolic and Diastolic Function

Regarding LV systolic function, there was a graded relationship between the severity of MR and LV ejection fraction (P=0.003). Patients with moderate MR had more decreased LV systolic function than those without MR (41.7% vs. 52.1%, P=0.001). Patients with moderate MR had significantly more increased total RWSI than those without MR (1.66 vs. 1.41, P=0.005) 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 (inferior RWSI 2.15 vs. 1.87, P=0.239 and posterior RWSI 1.88 vs. 1.45, P=0.050.

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 (P=0.820).

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 (P=0.016), although in the case of LV end-diastolic volume it was not significant statistically (P=0.075). 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 (annular to anterior PM, P=0.180 and annular to posterior PM, P=0.450), 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.[17] Mitral stenosis presenting for the first time as acute STEMI is rare. 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%)4. 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

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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.[18]

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.[15]

Our results are also consistent with previous study that LV dysfunction without dilatation fails to produce significant MR.[19] 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.[14,20] 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.[21,22]

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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REFERENCES

Habibian M, Batra R, Mengel C, Sweeny A, Walters D. Cardiologists Beware: An unusual cause of myocardial infarction after aortic valve replacement. Heart, Lung and Circulation. 2016 Aug 1;25:S35.

Kolodgie FD, Virmani R, Finn AV, Romero ME. Embolic myocardial infarction as a consequence of atrial fibrillation: a prevailing disease of the future. Circulation. 2015 Jul 28;132(4):223-6.

Gensini GG. Coronary arteriography: role in myocardial revascularization. Postgraduate Medicine. 1978 Jan 1;63(1):121-38.

Alpert JS. Myocardial infarction with angiographically normal coronary arteries. Archives of internal medicine. 1994 Feb 14;154(3):265-9.

Larsen AI, Galbraith PD, Ghali WA, Norris CM, Graham MM, Knudtson ML, APPROACH Investigators. Characteristics and outcomes of patients with acute myocardial infarction and angiographically normal coronary arteries. The American journal of cardiology. 2005 Jan 15;95(2):261-3.

Fuster VA, Chesebro JH, Frye RL, Elveback LR. Platelet survival and the development of coronary artery disease in the young adult: effects of cigarette smoking, strong family history and medical therapy. Circulation. 1981 Mar;63(3):546-51.

Ross R, Kay R, Ambrose J, Herman MV. Coronary thrombosis in the absence of angiographically-evident obstructive coronary disease. Chest. 1983 Dec 1;84(6):768-70.

ISSN: 0975-3583, 0976-2833 VOL 13, ISSUE 08, 2022

Steele P, Rainwater J, Vogel R. Abnormal platelet survival time in men with myocardial infarctionand normal coronary arteriogram. The American Journal of Cardiology. 1978 Jan 1;41(1):60-2.

Gersh BJ, Bassendine MF, Forman R, Walls RS, Beck W. Coronary artery spasm and myocardial infarction in the absence of angiographically demonstrable obstructive coronary disease. InMayo Clinic Proceedings 1981 Nov 1 (Vol. 56, No. 11, pp. 700-708).

Glazier JJ, Mcginnity JG, Spears JR. Coronary embolism complicating aortic valve endocarditis: treatment with placement of an intracoronary stent. Clinical cardiology. 1997 Oct;20(10):885-8.

Granger EK, Rankin J, Larbalestier RI, Hockings BE. Obstruction of the right coronary artery ostium by an aortic valve papillary fibroelastoma. Heart, Lung and Circulation. 2005 Dec1;14(4):266-8.

Rigatelli G. Normal angiogram in patients with acute coronary syndrome: searching for unusual substrates of myocardial ischemia. International Journal of Cardiology. 2005 Mar 10;99(1):25-7.

Bursi F, Enriquez-Sarano M, Jacobsen SJ, Roger VL. Mitral regurgitation after myocardial infarction: a review. The American journal of medicine. 2006 Feb 1;119(2):103-12.

Grigioni F, Enriquez-Sarano M, Zehr KJ, Bailey KR, Tajik AJ. Ischemic mitral regurgitation: long-term outcome and prognostic implications with quantitative Doppler assessment. Circulation. 2001 Apr 3;103(13):1759-64.

Kumanohoso T, Otsuji Y, Yoshifuku S, Matsukida K, Koriyama C, Kisanuki A, Minagoe S, Levine RA, Tei C. Mechanism of higher incidence of ischemic mitral regurgitation in patients with inferior myocardial infarction: quantitative analysis of left ventricular and mitral valve geometry in 103

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patients with prior myocardial infarction. The Journal of thoracic and cardiovascular surgery. 2003 Jan 1;125(1):135-43.

Arikawa K, Otsuji Y, Zhang H, Yu B, Uemura T, Hamasaki S, Biro S, Kisanuki A, Minagoe S, Tei C. Left ventricular remodeling is less while left atrial remodeling is greater in inferior compared to anterior myocardial infarction: importance of ischemic mitral regurgitation. Journal of Echocardiography. 2004;2(2):43-8.

Thomas, D J, Bonow, O R. Mitral Valve Disease. In: Zipes DP, Libby P, Bonow RO, Mann DL, Tomaselli GF, Braunwald E. Eds. Braunwald's heart disease: A textbook of cardiovascular medicine. Philadelphia, PA: Elsevier. 2019: 1415–44.

PRIZEL KR, HUTCHINS GM, BULKLEY BH. Coronary artery embolism and myocardial infarction: a clinicopathologic study of 55 patients. Annals of internal medicine. 1978 Feb 1;88(2):155-61.

Otsuji Y, Handschumacher MD, Schwammenthal E, Jiang L, Song JK, Guerrero JL, Vlahakes GJ, Levine RA. Insights from three-dimensional echocardiography into the mechanism of functional mitral regurgitation: direct in vivo demonstration of altered leaflet tethering geometry. Circulation. 1997 Sep 16;96(6):1999-2008.

Lamas GA, Mitchell GF, Flaker GC, Smith Jr SC, Gersh BJ, Basta L, Moyé L, Braunwald E, Pfeffer MA. Clinical significance of mitral regurgitation after acute myocardial infarction. Circulation. 1997 Aug 5;96(3):827-33.

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Otsuji Y, Handschumacher MD, Liel-Cohen N, Tanabe H, Jiang L, Schwammenthal E, GuerreroJL, Nicholls LA, Vlahakes GJ, Levine RA. Mechanism of ischemic mitral regurgitation with segmental left ventricular dysfunction: three-dimensional echocardiographic studies in models of acute and chronic progressive regurgitation. Journal of the American College of Cardiology. 2001 Feb;37(2):641-8.

Perez de Isla L, Zamorano J, Quezada M, Almeria C, Rodrigo JL, Serra V, et al. Functional mitral regurgitation after a first non-ST segment elevation acute coronary syndrome: Contribution to congestive heart failure. Eur Heart J 2007; 28: 2866 – 2872.