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KEY WORDS: Coronary Artery Disease, Ischemic Mitral Regurgitation, Left Ventricle Dysfunction, Papillary Muscles

ECHOCARDIOGRAPHIC ANALYSIS OF ISCHEMIC MITRAL REGURGITATION IN PATIENTS WITH RECENT MYOCARDIAL INFARCTION

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ABSTRACT

Introduction: The incidence of coronary artery disease is on the rise in both urban and rural sections in India. Ischemic Mitral Regurgitation is one of the complications of coronary artery disease and is an independent prognostic factor in chest pain. We aimed to study various factors contributing to the mechanism of Ischemic Mitral Regurgitation (IMR) using 2D echocardiography among patients with recent myocardial infarction.

Methodology: A comparative cross sectional study was carried out among 30 patients with recent myocardial infarction visiting our hospital for their follow up. Echocardiographic. and tissue Doppler echocardiography was done for all patients. The study participants were divided into two groups Group A had recent anterior wall myocardial infarction and Group B had recent inferior wall myocardial infarction. Group B participants were further subdivided into 2 groups based on their left ventricular spherecity into B1 and B2.

Results: The average left ventricular ejection fraction is higher in group B2 compared to group B1 and A ($p<0.01$). The incidence of moderate to severe mitral regurgitation is high in group B1 and A compared to B2 ($p<0.01$). The LV dysfunction of group B1 Vs B2 and all cases in mild and mod-severe MR were statistically significant ($p<0.05$).

Conclusion: There is a central role of leaflet tethering in the mechanism responsible for ischemic MR. Therapeutic approaches to relieve ischemic MR need to be targeted to reduce tethering by LV remodeling. Surgical approaches can be targeted at relieving tethering by aneurysm application and repositioning of Papillary muscles

INTRODUCTION

Ischemic mitral regurgitation (IMR) is defined as mitral regurgitation due to coronary artery disease with structurally normal mitral valve leaflet and chordate [1]. The incidence of coronary artery diseases in rural and urban population in India is reported to be between 14.8 per thousand to 65.4 per thousand [2]. IMR is one of the complications of coronary artery diseases occuring in approximately 20% of patients after myocardial infarction and 56% of patients with heart failure. IMR can occur in coronary artery disease both during acute phase and chronic phase. It is more common in inferior wall myocardial infarction [3]. IMR is an independent prognostic factor in patients with chest pain even without myocardial infarction [4]. It is a common complication of ischemic heart disease and it adversely affects the prognosis [5], [6]. There are three theories proposed to the mechanism of IMR namely Mitral Annular Dilatation, Papillary Muscle Displacement and Left Ventricle geometry.

Mitral annular dilatation is usually the result of vlentricular enlargement. When the dilatation is large enough to overcome the normal area of redundant leaflet overlap, IMR develops. The role of Mitral Annular Dilatation in IMR was put forth in 1956 by Dr. Friedberg but the first clinical study came from Dr. Boltwood et al in 1983 which was supported by Dr. Shengqin He et al in 1997. However, this was contradicted by Dr. Yataka Otsuji et al in 2002. [7], [8], [9]

The role of global and regional ventricular dysfunction in Papillary Muscle (PM) displacement has been recently explored. When the Papillary muscles are displaced towards the LV apex, it causes tenting of the leaflet, resulting in less leaflet overlap and development of MR. This leaflet tethering is measured as leaflet tethering distance.

Several studies suggest that global change in the geometry of the Left Ventricle (LV) results in IMR. Several authors have also suggested that impairment of LV systolic function results in a decrease in the generation of force needed to close the leaflets, thereby compounding the issue. [12-14] Three-dimensional echocardiography demonstrated an increased tethering distance which correlated with LV sphericity [15]. However, the same amount of LV dysfunction is associated with varying degrees of MR [16], [17]. Kono et al demonstrated that changes of LV geometry due to the regional hyperkinesias of the LV segment. [1] Thus apical displacement of the PM as a result of changes in the regional LV dysfunction is sufficient to cause IMR.

In patients with LV dysfunction, Otsuji Yia et al used quantitative echocardiography to determine the relationship between the degree of functional mitral deformation and local ventricular remodeling [17]. They found that the major determinant of Effective Regurgitant Orifice (ERO) of IMR was the degree of systolic mitral tenting, which was directly related to local remodeling that caused apical and posterior displacement of both the PM [18]. However, another hypothesis suggests that PM ischemia may in fact result in a decrease in MR [19], [20], as demonstrated by Messas et al [24]

OBJECTIVE

To study various factors contributing to the mechanism of IMR using 2D echocardiography among patients with recent myocardial infarction

METHODOLOGY

Study Design:

A comparative cross sectional study was carried out.

Study area:

All the out patients visiting the cardiac care centre of our tertiary care hospital, situated in Chennai.

Study population:

Patients with recent myocardial infarction associated with mitral regurgitation who were following up with us at our cardiac care center.

Inclusion criteria:

1. Presence of recent myocardial infarction
2. Presence of Mitral regurgitation
3. Age >30 years.

Exclusion criteria:

1. Acute myocardial infarction(less than 1 week)
2. Multiple MI

3. Mitral regurgitation due to other causes (RHD, IE and MVPS etc.)
4. Dilated cardiomyopathy
5. Congenital heart disease.
6. Pericardial diseases.

Study duration:
The study was carried out for two years between 2012 and 2014.

Sample size and sampling:
A total of 30 patients participated in this study. The sampling technique used was consecutive sampling, based on the inclusion and exclusion criteria.

Data collection tools:
Echocardiographic evaluation of all patients was done using VIVID 7E GE machine. Standard M-Mode, Two Dimensional, Color flow and tissue Doppler echocardiography was done for all patients. LV end-diastolic and end-systolic cavity areas were traced in those views, and the LV end-diastolic volume (EDV) and ejection fractions (EF) were calculated by the method of discs.

Ethical committee approval and informed consent:
Approval from the Institutional Ethics Committee was obtained prior to data collection. Informed consent was obtained from each participant prior to the commencement of data collection.

Pre- testing:
Pre testing was done on 3 participants to assess the feasibility of the study protocol. The data of pilot study were not included in the analysis.

Data collection:
The study population comprised of 30 patients with recent myocardial infarction associated with Mitral regurgitation followed up at our cardiac care centre. The study participants were divided into two groups according to location of Myocardial Infarction. Group A had recent anterior wall myocardial infarction and Group B had recent inferior wall myocardial infarction. Group B participants were further subdivided into 2 groups based on their left ventricular spherecity. Participants with increased left ventricular spherecity belonged to Group B1 and those with normal left ventricular spherecity belonged to Group B2.

Operational definitions:
A. Recent Myocardial infarction was defined as
1. History of myocardial infarction
2. ECG evidence of recent MI
3. RWMA in echo
4. Elevation of cardiac enzymes at the time of Acute Myocardial Infarction

B. Left Ventricular Sphericity
Left Ventricular Sphericity was defined in 2- Dimensional Echocardiography as ratio of short axis to long axis dimension of left ventricle. This was measured in apical two-chamber view during mid-systole. Normal value of sphericity by 2-Dimensional echo method is less than 0.60. Based on the degree of basal infero-posterior LV bulging, Group B the patients were further subdivided into two namely groups B1 with significant LV bulging with a D/L > 0.60 and group B2 without significant LV bulging with D/L < 0.6.

C. Mitral Annular Area (MAA)
Annular dimension of mitral valve was measured in apical 4 chamber and apical two chamber view during mid systole as shown in figure 1 and mitral annular area was calculated using the formula;

MAA = d1× d2 × Π/4.

D. Leaflet Tethering Distance:

Mitral Leaflet Tethering Distance of AML and PML were measured separately. AML tethering distance is the distance between tip of AML to the contra lateral posterior mitral annulus. Both the tethering distance was measured in apical 4C and 2C views during mid-systole.

E. Mitral Regurgitation:
Mitral Regurgitation was diagnosed and assessed by color flow and CW Doppler echocardiography. In our study Mitral regurgitation severity was assessed by the following methods

1. Jet Area
2. Density of CW Doppler.
3. Vena Contracta
4. Mitral Regurgitation Volume.

F. Evaluation Of Papillary Muscle Function

In our study, three methods were used to evaluate PM function
1. M Mode Echocardiography
2. 2 Dimension Echocardiography
3. Tissue Doppler Imaging.

RESULTS
The Demographic characteristics of patients are detailed in table 1. The mean age of the study participants was 53.50+12 years. The distribution of coronary risk factors was equal in all the groups .The average time since Acute MI (AMI) in all groups was 5 weeks.

The echocardiographic characteristics of the patient are listed in table 2. The incidence of moderate to severe mitral regurgitation was high in group B1. The average systolic thickening of PPM in B1 and B2 in 2D echo were 34.62% & 32.83% respectively.

The results of Doppler echocardiography analysis are reported in table 3. The peak systolic velocity of posterior papillary muscle in group B1 and B2 are 9.02 and 6.54 m/s where in group A is 7.10 m/s.

The determinants of IMR were analyzed in table 5. The LV dysfunction of group B1 Vs B2 and all cases in mild and mod-severe MR were statistically significant ($p < 0.05$). The average leaflet tethering distance is significant statistically in group A and B1.

The PM function of systolic velocity mode was significant in group B. The PM function of systolic velocity ECHO was statistically significant in group B1 ($p<0.05$). Systolic thickening of PPM and APM were compared, group B1 was statistically significant.

The relationship between leaflet tethering distance and severity of ischemic mitral regurgitation is given in figure 2. In all groups of patients when there is increased leaflet tethering distance there is severe MR.

The relationship between MAA with ischemic mitral regurgitation is given in figure 3. There is proportionate increase in the MAA with the severity of ischemic mitral regurgitation in all groups of patients.

The relationship between papillary muscle function with ischemic mitral regurgitation is given in figure 4. In group B1 papillary muscle systolic peak velocity has linear correlation with ischemic mitral regurgitation.

The relationship of LV sphericity with severity of ischemic mitral regurgitation is given in figure 5. The LV sphericity has good correlation with severity of MR in group B1.

DISCUSSION
IMR is a common complication of both acute and chronic ischemic heart disease and adversely affects the prognosis. [5],[6]. Since its initial recognition by Burch and De Pasquale [21], IMR has been attributed to dysfunction of papillary muscle impairment. Explanations are as follows:

(1) Ischemic distortion of left ventricular geometry.[9-16]
(2) Decreased left ventricular (LV) force acting to close the leaflets[15], resulting in incomplete mitral leaflet closure (IMLC).
(3) Increased tethering and diminished LV contraction

In our study the mitral annular area was found to be significant

among all the groups, highest values were seen in group A. Also, the average LV sphericity is high in group B1. The average leaflet tethering distance is significant statistically in group A and B1. Our study correlated well with the result of Dr. Robert W Godley, MD, et al. [6], [23] Mitral leaflet tethering distance was proposed by Dr. Fang Zhu, MD et al [22] as the primary mechanism of persistent MR even after mitral Annuloplasty.

Papillary muscle dysfuncton was first described by DR GE BURCH et al [19] in 1968 and contributed to the pathogenesis of IMR. In our study the PM function of systolic velocity mode was significant in group B (PPM) when compared between mild and mod-severe MR. The PM function of systolic velocity was statistically significant in group B1.

The results of the study suggest a central role of leaflet tethering [25-29], in the mechanism responsible for IMR. Therapeutic approaches need to be targeted to reduce tethering by LV remodeling. Revascularization of the viable adjacent LV wall is expected to relieve IMR. The results also support the surgical approaches targeted at relieving tethering.

Conclusion and recommendations

1. Mitral leaflet tethering distance is consistently directly proportional to severity of IMR.
2. Papillary muscle dysfunction is not an independent determinant of IMR.
3. Papillary muscle dysfunction attenuates ischemic MR in patients with recent inferior wall MI.

Limitations

The present study found an inverse relationship between PM dysfunction IMR. Such an inverse relationship may not be relevant in patients with different pathophysiology. Because PM contraction varies according to the spatial direction, it is necessary to establish a standard angle used for echocardiographic evaluation.

Tables & Figures

TABLE – 1: Demorgaphic characteristics of study participants:

S.No	Characteristic	Group A	Group B1	Group B2
1	Age (years)	53.50 ± 12.44	56.20 ± 7.2	56.30 ± 7.10
2	Male (%)	70	60	60
3	Female (%)	30	40	40
3	Body Mass Index (kg/m2)	26.29 + 2.59	25.98 ± 3.10	26+ 2.39
4	Time since AMI (months)	1.10	1.11	1.10
5	Hypertension (%)	50	60	50
6	Type 2 Diabetes Mellitus (%)	60	60	60
7	Smoking (%)	30	30	30
8	Dyslipidemia (%)	50	60	50

Table 2: Echocardiographic characteristics of the patients:

S. No	Characteristics	Group A	Group B1(>60%)	Group B2(<60%)
1	**LVFF (%)**	41.00±6	44.00 ± 5	52.00 ± 6
	MR (Mild %)	50	30	80
	MR (Mod-severe %)	50	70	20
2	**LV sphericity (%)**	58.70% ± 1.2	64.20% ± 1.99	50.30% ± 4.16
3	**MAA (cm2)**	4.95 cm2± .15	4.41 cm2± .42	3.79 cm2± .04
4	**Leaflet tethering distance**			
	AML tethering distance (mm)	20.76 ± 2.86	16.61 ± .33	17.5 ± .30
	PML tethering distance (mm)	16.61± 0.20	23.24± 3.82	19.27 ± 2.19
5	**M mode**			
	APM (%)	30.51 ± 0.33	38.31 ± 0.22	37.31 ±0.41
	PPM (%)	41.32 ± 0.23	37.36 ± 3.98	31.51 ±1.15
6	**2D ECHO**			
	APM (%)	27.60± 0.19	34.62 ± 1.92	32.83 ±0.86
	PPM (%)	31.38± 0.56	34.62 ± 1.57	32.83±0.86
7	**Systolic peak velocity in TDI**			
	APM (m/s)	6.52 ±0.26	7.28 ±0.19	7.58 ±0.32
	PPM (m/s)	7.41 ±0.17	7.44 ±0.69	6.39± 0.41

[LVEF – left ventricular ejection fraction; MR- mitral regurgitation; MAA-Mitral annular area; AML-Anterior mitral leaflet; PML-Posterior mitral leaflet; APM-Anterio lateral papillary muscle; PPM-Posterio medial papillary muscle; TDI- Tissue Doppler imaging]

Table 3: Tissue Doppler Imaging among study participants

S. No	Characteristics	B1	B2	A
1	**APM**			
	Sm peak m/s	6.17 + 0.59	9.31±0.25	9.47 +0.25
	Em peak m/s	9.31 +0.30	9.46 +0.22	9.63 ± 0.29
	Am peak m/s	9.86 +0.41	10.39 +0.24	10.44 +0.17
2	**PPM**			
	Sm peak m/s	9.02 +0.57	6.54 + 0.26	7.10 +0.53
	Em peak m/s	9.61 + 0.20	10. 27 +0.26	9.55 ± 0.30
	Am peak m/s	9.96 +0.53	10.19+0.36	9.86± 0.45
3	**Anterior septum**			
	Sm peak m/s	5.37+ 0.34	8.75+0.23	9.74+0.39
	Em peak m/s	8.68+0.18	10.27+0.23	9.37± 0.41
	Am peak m/s	9.80+0.45	10.14+0.68	10.39+0.23
4	**Inferior wall**			
	Sm peak m/s	8.58+0.22	5.64+0.55	5.56 ±.55
	Em peak m/s	9.36+0.40	9.55+.27	9.57 +0.28
	Am peak m/s	10.29 +0.14	10.32 +0.32	10.0+0.16

[APM-Anterior papillary muscle; PPM-Posterior papillary muscle; TDI- Tissue Doppler imaging; Sm-systolic peak velocity, Em-early diastolic peak velocity Am-atrial peak velocity]

TABLE 5: Comparison of Determinants of Ischemic MR:

S. No	Characteristic	Mild MR	Mod –severe MR	P value
1.	**LV Dysfunction (%)**			
	B1	49.6 ±0.04	41 ±0.03	< 0.05
	B2	54.6 ±0.02	41± 0.01	< 0.05
	A	44 ±0.04	38 ± 0.06	Ns
	All cases	50 ± 0.06	39.9 ± 0.45	< 0.05
2.	**LV spherecity (%)**			
	B1	62.33 ±0.57	65 ±1.82	< 0.05
	B2	48.62 ±2.44	57± 1.41	< 0.05
	A	58.01 ±1.20	59.40 ± 1.81	Ns
	All cases	54.13 ± 6.15	61.85± 3.78	< 0.05
3	**Mitral Annular Area**			
	B1	3.81 ± 0.06	4.66 ± 0.10	< 0.05
	B2	3.78 ± 0.04	3.82 ± 0.01	< 0.05
	A	4.82 ± 0.03	5.07 ± 0.10	< 0.05
	All cases	3.81 ± 0.06	4.66 ± 0.10	< 0.05
4	**Leaflet tethering Distance (mm)**			
	B1	17.73 ± 0.41	25.60± 0.42	<0.01
	B2	18.25± 0.47	23.35± 0.21	Ns
	A	18.07± 0.34	23.43± 0.57	<0.05
	All cases	17.66± 0.80	22.04±4.30	0.01
5	**PM function (m/s)**			
	Systolic Velocity MODE			
	B1(PPM)	31.63± 0.15	39.81 ± 0.57	<0.05
	B2(PPM)	30.98± 0.37	33.60 ± 0.28	<0.05
	A(APM)	30.36± 0.38	30.66 ± 0.19	Ns
	PM function			
	Systolic Velocity ECHO			

	B1(PPM)	32.53± 0.23	35.51 ± 0.57	<0.05
	B2(PPM)	32.47± 0.37	34.25± 0.91	Ns
	A(APM)	27.60± 0.20	27.66 ± 0.19	Ns
	Systolic thickening			
	B1(PPM)	6.50 ± 0.30	7.84± 0.22	<0.05
	B2(PPM)	6.35 ± 0.45	6.55 ± 0.07	Ns
	A(APM)	6.54± 0.29	6.50± 0.25	Ns

[MAA-Mitral annular area; AML-Anterior mitral leaflet; PML-Posterior mitral leaflet; APM-Anteriolateral papillary muscle; PPM-Posteriomedial papillary muscle; TDI- Tissue Doppler imaging]

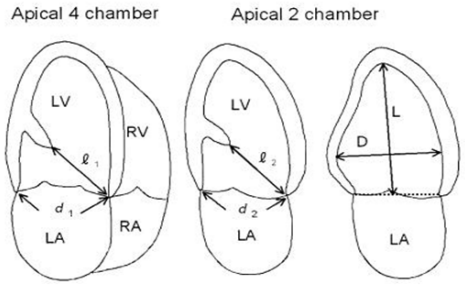

FIGURE – 1: Mitral Annular Area

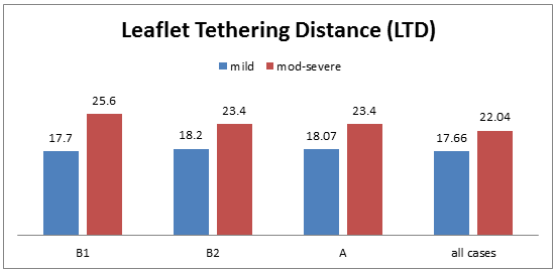

FIGURE 2: Leaflet tethering distance

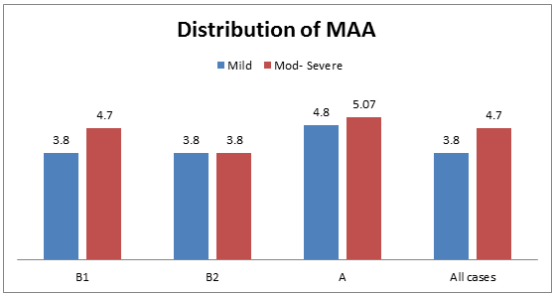

FIGURE 3: Distribution of MAA among the study participants

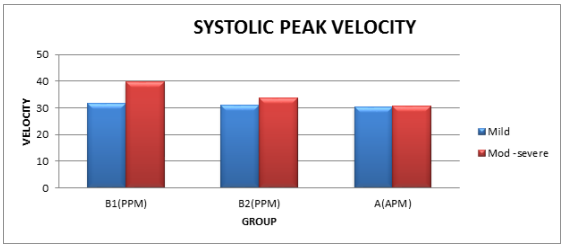

FIGURE 4: Systolic peak velocity among the study participants

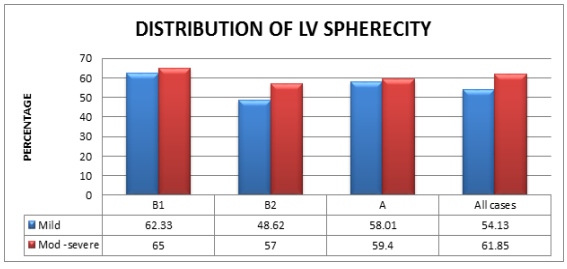

FIGURE 5: Distribution of LV spherecity among the study participants

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