

## MITRAL REGURGITATION FOLLOWING FIRST TIME ACUTE MYOCARDIAL INFARCTION: Early and late echocardiographic evaluation

Randhawa MS\*

### ABSTRACT

**Objective:** To compare the prevalence of mitral regurgitation in patients with first time acute myocardial infarction before hospital discharge and after two months follow up and its significance.

**Design:** Prospective analysis of sixty patients presenting with first time acute myocardial infarction with age matched controls.

**Setting:** Department of Medicine and Coronary Care Unit, postgraduate Medical Institute, Services Hospital, Lahore.

**Main Outcome Measures:** Regurgitant flow area and flow velocity, left ventricular dimension, AV displacement, Chamber size.

**Results:** Baseline clinical and Echocardiographic data was not significantly different from age matched controls at presentation, Left ventricular internal dimension. During systole and diastole phases were  $38 \pm 4$  mm and  $52 \pm 2$  to  $36 \pm 3$  mm and  $50 \pm 1$  mm respectively. However at 2 months follow up these dimensions increased to  $40 \pm 2$  mm and  $56 \pm 2$  mm respectively. Mean atrioventricular plane displacement in healthy control and the patients were  $15 \pm 1$  mm and  $11 \pm 2$  mm respectively. Prevalence of Doppler detected mitral regurgitation in healthy subjects was 25% as compared to 65.2% of the patients at 2 months follow up. Regurgitation flow area in these patients was  $1.02 \pm 1.2$  cm<sup>2</sup> from baseline to  $1.13 \pm 1.2$  cm<sup>2</sup> after 2 months follow up occupying  $7.2 \pm 7.3\%$  to  $8.3 \pm 6\%$  of the left atrial area respectively.

**Conclusions:** Prevalence of Doppler detected regurgitation of weak intensity in our study is 25%,: It rose to 65.2% following first time acute myocardial infarction but mild in majority of the patients and remained insignificant after two months follow up. Echocardiographic significant mitral regurgitation was present in only 3 patients(6.52%)

**KEY WORDS:** Acute myocardial infarction, mitral regurgitation, Echocardiography

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\* Dr. Mohammad Shoib Randhawa FCPS  
Associate Professor Medicine,  
Postgraduate Medical Institute,  
Services Hospital, Lahore.

Correspondence:

Dr. Mohammad Shoib Randhawa  
429, Gulshan Block, Allama Iqbal Town,  
Lahore, Pakistan

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### INTRODUCTION

Myocardial Infarction may manifest as mitral regurgitation, which can be, detected non-invasively with the help of Doppler Echocardiography<sup>1,2,3</sup> and has high sensitivity and specificity when compared with angiography.<sup>4</sup>

Prevalence of this regurgitation following acute myocardial infarction shows variability.<sup>1,3,5</sup> Mitral Regurgitation in healthy subjects detected by Doppler technique is mild in severity and therefore called physiological regur-

gitation<sup>6,7</sup>. Prevalence of which also varies<sup>8,9</sup>, 29% reported by Mahbul Alam et al.<sup>10</sup> The purpose of this study was to compare the prevalence of mitral regurgitation in patients with first time acute myocardial infarction before hospital discharge and after two months follow up<sup>11,12</sup> in our set up at Department of Medicine, CCU Services hospital/Postgraduate Medical Institute Lahore.

## PATIENTS AND METHODS

Sixty patients were included in the study with first time acute myocardial infarction and were studied prospectively during year, 2001-2002. Fourteen patients were excluded from the study due to non compliance for follow up, reinfarction, or unsatisfactory Echo images. Patients with bundle branch block were also excluded from the study.

Fortysix (46) patients were finally included in the study, there were 34 male and 12 female patients. Their age ranged from 36 years to 70 years with a mean of  $53.5 \pm 13.06$  SD. All the female patients were between the age of 46 years to 60 years with mean  $51.8 \pm 10.19$  SD.

Following criteria was used to establish the diagnosis of acute myocardial infarction.

1. Typical history with prolonged chest pain of >30 minutes duration.
2. ECG changes of acute myocardial infarction (significant Q- wave, in more than one ECG leads for Q-Wave infarction. Or ST-T wave changes lasting more than 48 hours without Q-wave developing suggestive of non Q-Wave infarction<sup>13</sup>
3. Raised cardiac enzymes.

Thirty patients had anterior and sixteen had inferior myocardial infarction. Thirtyeight (38) patients (82.6%) developed Q-wave and 8(17.35%) developed non Q- wave myocardial infarction. Amongst the risk factors twenty (43.47%) patients had diabetes mellitus, fourteen (30.43%) had history of systemic hypertension. Ten (21.73%) patients had both diabetes and hypertension. Thirty patients received thrombolytic therapy with streptokinase. Ten

patients could not afford and six patients had contraindications to antithrombotic therapy like active peptic ulcer disease(n=3), bleeding hemorrhoids(n=2) and history of cerebrovascular accident(n=1). 2D, M-mode Echocardiography, Color flow imaging and Doppler echocardiography was done after clinical stabilization of patients and two months after discharge from hospital during follow up. Twelve (n=12) aged matched healthy volunteers ware used as controls. SSH.140 A Toshiba made machine was used for the purpose. The patients and subjects were examined in left lateral decubitus position, left parasternal long axis, apical four chamber and two chamber views as described in previous studies.<sup>14,15,16</sup>

The left ventricular global functions was measured as mean of atrioventricular plane displacement from all the sites.

Mitral valve prolapse was diagnosed on 2-D Echo in accordance with Alpert et al<sup>17</sup>. Mitral valve calcification was diagnosed as described elsewhere<sup>18</sup>.

Pulse wave and continuous wave Doppler methods were used for the detection of mitral regurgitation. Color flow was used to mark the direction, maximum area of regurgitant signals and percentage of left atrial area filled with regurgitant flow.

Pulse wave Doppler with a sample volume placed in the left atrium just beneath the mitral valve and carefully searching the regurgitation in systole as described by Berger et al<sup>8</sup>.

Velocity signals that occupied 50% or more of the systole and velocity of  $\geq 1.5$  m/s with audio output typical for regurgitation defined the presence of mitral regurgitation. Continuous wave Doppler echocardiography by positioning the cursor along the direction of the regurgitant flow from the color flow Doppler was performed to record the velocity, wave form and the peak systolic velocity of the regurgitant jet.

## RESULTS

Table-I shows the comparison of age groups of patients and controls. Table-II shows that

controls and the patients had a baseline clinical and echocardiographic data not much different regarding the Left Ventricular dimensions and left arterial dimensions. Left ventricular internal dimensions during systole and diastolic phases  $38\pm4$  mm and  $52\pm2$  mm to  $36\pm3$  mm and  $50\pm1$  mm respectively. However

after two months follow up of patients with infarction these dimensions increased to  $40\pm2$  mm and  $56\pm2$  mm during systolic and diastolic phases of cardiac cycles respectively. Left atrial dimensions were also increased from  $40\pm4$  mm to  $43\pm4$  mm after two months follow up. Mean atrioventricular plane displacement

Table-I: Age group of patients and controls

Age group Years	No. of Patients			Controls Data		
	Female	Male	Total	Female	Male	Total
36-45	--	6	6	--	1	1
46-50	8	14	22	2	4	6
51-60	4	12	16	1	3	4
61-70	--	2	2	--	1	1
Total	12	34	46	3	9	12

Table-II: Echocardiographic data in patient in early and late (2months) phases of myocardial infarction

Variables	Patients(n=46)		Control(n=12)	
	Initial	After 2 months follow up	Initial	After 2 months follow up
<b>Blood pressure</b>				
Systolic	$126\pm12$	$120\pm10$	$124\pm10$	$124\pm10$
Diastolic	$80\pm6$	$78\pm4$	$80\pm4$	$80\pm4$
LVID(distole)	$52\pm2$	$56\pm2$	$50\pm1$	$50\pm1$
LVID (systole)	$38\pm4$	$40\pm2$	$36\pm3$	$36\pm3$
AV Displacement mean (mm)	$11\pm2$	$11.2\pm2$	$15\pm1$	$15\pm1$
LA (mm)	$40\pm4$	$43\pm4$	$38\pm2$	$38\pm2$
<b>Mitral Regurgitation</b>				
No.of Patients (%)	30(65.2%)	36(78.2%)	3(25%)	3(25%)
<b>Color Flow</b>				
Regurgitant flow area(cm <sup>2</sup> )	$1.02\pm1.2$	$1.13\pm1.2$	$0.3\pm0.2$	$0.3\pm0.2$
Flow occupying LA area(%)	$7.2\pm7.3$	$8.3\pm6$	$2\pm1.2$	$2.1\pm1$
<b>Continous Wave Doppler</b>				
Pan systolic	n=29(63%)	n=33(71.74%)	n=2(16.66%)	---
Early to mid systolic	n=1(2.17%)	n=3(6.52%)	n=1(8.33%)	---
Peak flow Velocity(m/sec)	2.2—3.5(n=28)	3.6-6(n=8)	1.8 to 3m/sec	---
<b>LV aneurysm</b>				
	n=2(4.34%)	n= 2(4.34%)	---	---
<b>Mural Thrombus</b>				
	4(8.7%)	2(4.34%)	---	---
<b>Pericardial Effusion</b>				
	6(13%)	NIL(zero)	---	---

Abbreviations: LVID: Left ventricle internal dimension, LA: Left atrium, LV: Left ventricle, AV: atrioventricular plane displacement

in healthy subjects was significantly more compared to the patients  $15\pm1$  mm compared to  $11\pm2$  mm. Echocardiographic signs of mitral valve prolapse were present in two patients and three patients showing mitral annulus calcification.

Pulse wave, continuous wave and color flow Doppler Echocardiography detected the mitral regurgitation with equal frequency. Prevalence of Doppler detected mitral regurgitation in healthy subjects was 25% (three out of twelve). one of them had mitral valve prolapse. Regurgitation flow area in the normal subjects was  $0.3\pm0.2$  cm<sup>2</sup> occupying  $2\pm1.2\%$  of the left atrial area. All these subjects had spectral envelope which was poorly defined and of low intensity and velocity, 1.8 m/sec to 3.0 m/sec. 65.2% (n=30) patients on the other hand showed Doppler detected mitral regurgitation after two months follow up. Six new patients developed mitral regurgitation (Total n=36). Regurgitant flow area in these patients was  $1.02\pm1.2$  cm<sup>2</sup> from baseline to  $1.13\pm1.2$  cm<sup>2</sup> after two months follow up occupying  $7.2\pm7.3\%$  (range 2-42%) to  $8.3\pm6\%$  (range 2-47%) of the left atrial area from the baseline to two months follow up respectively and the peak flow velocity in these patients were 2.2 m/sec to 3.5 m/sec (n=28, 77.7%) to 3.6 m/sec to 6 m/sec (n=8, 22.22%). Except for three patients all had regurgitant flow of <4 cm<sup>2</sup> and <20% of the left atrial area. Separate analysis for anterior and inferior myocardial infarction groups showed no significant difference for frequency of mitral regurgitation (65.2% vs 63% respectively).

Auscultative finding of systolic murmur suggestive of mitral regurgitation was present in 21.74% of the patients (n=10). 4.34% (n=2) had LV aneurysm. 8.7% (n=4) had mural thrombus that disappeared with anticoagulation therapy after two months. Mild pericardial effusion was noted in 13% (n=6) which disappeared after two months follow up.

## DISCUSSION

Coordinated function of the mitral valve annulus leaflets, cordae, papillary muscles and

left ventricular myocardium contribute to the competence of the mitral valve (19,20). Doppler Echocardiography is sensitive and specific method established for detecting mitral regurgitation<sup>4</sup>.

Prevalence of Mitral regurgitation in healthy subjects in our study was 25%. It is mild in severity and called physiological regurgitation<sup>6,7</sup>. The reported prevalence has shown marked variability<sup>8,9</sup> and has been occurring early in systole<sup>6</sup>. In our study 16.6% (n=2) showed pan systolic and 8.3% (n=1) showed early to mid systolic mitral regurgitation. All three normal healthy subjects had Doppler signals of weak intensity with a poorly defined spectral envelope and low velocity (Table-II). The mechanism and clinical significance of physiological regurgitation is uncertain. 65.2% of the patients (n=30) with first time acute myocardial infarction showed Doppler detected mitral regurgitation. After two months six new patient developed mitral regurgitation. The prevalence is significantly higher than in healthy subjects. Pattern of regurgitation and spectral envelope resembles that of healthy subjects. It is possible that some of the patients already had physiological regurgitation and myocardial infarction in these patients might have increased the prevalence in this group. Moreover according to Morita et al<sup>7</sup> hypertension causes a greater prevalence of mitral regurgitation. 30.43% (n=14) of our patients with myocardial infarction had hypertension and 21.73% (n=10) had both hypertension and diabetes mellitus.

Severity of regurgitation has been assessed by quantification of regurgitant jet<sup>7,21</sup>. A regurgitant flow of <4 cm<sup>2</sup> that occupies <20% of the left atrium has been claimed to be of mild intensity when compared with angiography<sup>21</sup>. In our study all but three patients fulfilled such criteria at baseline indicating benign nature of regurgitation. Left ventricular dimension was slightly increased after two months follow up with unchanged global functions.

Based on this study it is probably unlikely that ventricular dimension as a cause of mitral regurgitation would contribute to increased fre-

quency after two months. This was reflected by relatively unchanged prevalence of mitral regurgitation after two months. In patients with healed myocardial infarction mitral regurgitation usually is caused by dyskinesia of the left ventricular myocardium at the base of the papillary muscles<sup>22</sup>. In addition left ventricular and annular dilatation are probably also a mechanism of mitral regurgitation<sup>23</sup>.

The increased intensity of regurgitation and appearance of regurgitation in few(n=6) after two months may be due to a more pronounced decrease in left ventricular functions than in the rest of the patients. Three of our six patients were diabetic and two were both diabetic and hypertensive.

Loperfido et al<sup>1</sup> had reported higher prevalence of mitral regurgitation in anterior than in inferior myocardial infarction. In the present study we could not appreciate significant difference. Some observation was made by Lehmann et al<sup>24</sup>, and they considered it to be due to regional dysfunction but not due to early ventricular dilatation. However, they considered it as an important predictor of cardiovascular mortality.

Alam et al<sup>25</sup> seem to have findings similar to ours. In their study the prevalence of MR in controls was 29% and in patients with acute MI it was 74%. The prevalence of MR was about the same in anterior and inferior MI (75% and 72% respectively).

In the opinion of Neskovie et al<sup>26</sup>, early Mitral Regurgitation (MR) after acute MI does not contribute significantly to the subsequent ventricular remodeling in the first year after MI.

A Japanese study by Ma HH et al<sup>27</sup> indicates that MR was present in 21% patients at onset of MI. They did not observe any correlation between presence or course of MR and site of infarction. Successful recanalization after AMI reduced the incidence of MR. They suggested that it can prevent LV remodeling which results in secondary improvement of MR.

Atrioventricular plane displacement of 10mm or more has a high sensitivity and specificity in predicting a normal ejection fraction<sup>28</sup>.

Present study showed that atrioventricular plane displacement of less than 10mm had a higher prevalence of mitral regurgitation than those having 10mm or more; however results being not statistically significant due to small number of patients in this group.

## CONCLUSION

Prevalence of Doppler detected physiological mitral regurgitation in healthy subjects in our study is 25%. All had Doppler signal of weak intensity and poorly defined spectral envelope.

Echocardiographically significant mitral regurgitation was present in only 3 patients, all other showed benign nature of regurgitation ie, flow less than 4cm<sup>2</sup> occupying <20% of the left atrial area<sup>7,21</sup>. Prevalence of Doppler detected mitral regurgitation following first time acute myocardial infarction is high (65.2%) but mild and insignificant in severity in majority of patients and changes remain insignificant after two months follow up.

No significant difference was noted in regurgitation in anterior or inferior myocardial infarction. Atrioventricular plane displacement less than 10mm may have a higher prevalence of mitral regurgitation. Regurgitation may not be detected clinically on auscultation due to trivial nature of regurgitant jet. Diabetes, hypertension and poor LV function may contribute to the development of mitral regurgitation.

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