Mechanism for Differences in Post Operative Left Ventricular Ejection Fraction after Mitral Valve Replacement in Patients with Mitral Stenosis

Chaudhury M Ahmed¹, Sabrina S Husain², SM M Zaman¹, DMM F Osmany³, Farhana Salim⁴, Nilufar Fatema⁵, Jahanara Arzu⁶

¹Professor of Cardiology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh.
²Medical Officer, BSMMU, Dhaka, Bangladesh.
³Registrar, Cardiac surgery, National Institute of Cardiovascular Diseases (NICVD), Dhaka, Bangladesh.
⁴Medical Officer, NICVD, Dhaka, Bangladesh.
⁵Associate Professor of Community Medicine, Shaheed Monsur Ali Medical College, Dhaka, Bangladesh.
⁶Associate Professor of Cardiology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh.

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ABSTRACT

Background: The subvalvular apparatus arrangement can cause ventricular torsion & deformation during cardiac cycle and interruption of papillary annular complex. As a result there was impairment of normal left ventricular strain pattern.⁵ In patients with mitral stenosis, the left ventricle is small. Preservation of subvalvular apparatus thus become important in moderaion of left ventricular volume in long term in patients with mitral stenosis undergoing mitral valve replacement.

Methods: This cross sectional study was performed on the 32 consecutive subjects in department of cardiac surgery and cardiology of BSMMU with rheumatic mitral stenosis undergoing MVR from Jan 2013 to June 2014. Mode of surgery was determined by morphology of subvalvular apparatus which dictated the extent of the preservation. The patients were divided into two groups- Group I- With preservation of subvalvular apparatus &Group II- No preservation- where SVA was completely excised. In 2D and M Mode echocardiographic measurements: Mid-wall circumferential end systolic LV stress was calculated for ellipsoid, LV mass, the mid wall circumferential end systolic LV stress is calculated by mirsky’s formula.⁹,¹⁰ Results: Patients with sub valvular apparatus resection (group I) had deterioration with postoperative ejection fraction in compare to group-II. Left ventricular circumferential wall stress analysis showed increased wall stress in group II after MVR. The wall stress increases further in midterm follow up which may explain the mechanism of long term poor out come in patients with mitral stenosis.

Keywords: Subvalvular apparatus, LV wall stress, MVR, post operative ejection fraction.

INTRODUCTION

After forty years of introduction of mitral sub valvular apparatus, preservation of this sub valvular apparatus during mitral valve replacement remains certain was to be addressed. The subvalvular apparatus contribute to effective left ventricular contraction by their process of connecting mitral annulus with ventricular wall. The annulo-ventricular continuity during systole, the subvalvular apparatus prevents the prolapese of the mitral leaflets into the left atrium. During the isometric phase of the cardiac cycle the papillary muscles contracts and brings the mitral valve down into the left ventricle and thus results into reduction of longitudinal axis of ventricle and eventually increase in short axis.¹¹,¹² This causes increase in ventricular muscle stretch and subsequently generates greater tension, contraction and stroke volume and thus mitral subvalvular apparatus moderates left ventricular wall tension. Also during diastole the papillary muscle and chordae moderates left ventricular distension. Interruption of subvalvular apparatus thus causes impairment of left ventricular stress pattern. The subvalvular apparatus arrangement can cause ventricular torsion & deformation during cardiac cycle and interruption of papillary annular complex. As a result there was impairment of normal left ventricular strain pattern.⁶ The major determinants of ejection fraction are preload, afterload and the contractility. The differences in preload, afterload or contractility may explain the observed difference in ejection fraction between procedures that preserve versus those that transects the subvalvular apparatus.
In patients with mitral stenosis, the left ventricle is small. And excision of subvalvular apparatus do not produce increase afterload and reduced preload situation as seen after mitral valve replacement in patient with mitral regurgitation. But, loss of the annulo ventricular continuity causes progressive ventricular dilation and reduce ventricular function in the long term. Preservation of subvalvular apparatus thus become important in moderation of left ventricular volume in long term in patients with mitral stenosis undergoing mitral valve replacement.

MATERIALS AND METHODS

This cross sectional study was performed on the 32 consecutive subjects who presented to department of cardiac surgery and cardiology of BSMMU with rheumatic mitral stenosis undergoing MVR from Jan 2013 to June 2014. Patients with RHD properly selected for MVR with St. Judesmechanical bileaflet valve. Valve lesions were judged as rheumatic in origin on the basis of echocardiographic findings: commissural fusion with leaflets deformity for mitral stenosis.[3] Consecutive patient was taken and mode of surgery was determined by morphology of subvalvular apparatus which dictated the extent of the preservation.

Exclusion criteria

Non Rheumatic mitral valve disease, acute rheumatic carditis, severe LV systolic dysfunction, Re do surgery, previous stroke, emergency surgery, renal failure, pre operative hepatic dysfunction, concomitant procedure like AVR, CABG.

Study Procedure

Initial evaluation of the patients by history, demographic data (age, sex, occupation), clinical examination and relevant investigations and was recorded.

Surgical procedure- The mitral valve was carefully inspected. The potential for repair was thoroughly assessed, as it was always first option. If MVR is indicated, notes as regard technique, types and size of prosthesis and the cardiopulmonary bypass protocol was recorded.

The patients were divided into two groups

1) With preservation:
   a. Complete- where entire chordo-papillary apparatus was preserved.
   b. Partial - posterior leaflet was preserved.
2) No preservation- where SVA was completely excised.

Surgical technique was different according to patient’s surgical requirement and one of the either technique as adopted by Fuster et al or by Miki et al.[4,5]

Echocardiographic measurement- Preoperative, postoperative and 6 months follow up echocardiographic data of the different modalities was followed as per the Douglas PS 6, Rudski LG 7 and Lang RM 8 and the following measurements was taken.

2D and M Mode measurements

- Mid-wall circumferential end systolic LV stress as calculated for ellipsoid.
- LV mass (1.04 (ID+2h3-D3 D3)3.14).
- The mid wall circumferential end systolic LV stress (ESS) is calculated by minksy’s formula 9,10 as follows:

ESS= Pb1/h [1- (h/2b)-(b2/2a2)] x 1.332 kdyne/cm2

Where p = 0.98 x mean arterial pressure (cuff) + 11 mmHg.

h = End systolic wall thickness

b = End systolic semi minor axis (D+h/2)
a = End systolic semi major axis (L+h/2)

Statistical analysis

Data was entered and analyzed with SPSS. Descriptive data was calculated for continuous and categorical variables. Comparison of continuous variable between the groups was analyzed by unpaired t test. A paired t test was done to compare pre and post operative measures. A p value of less than 0.05 was considered as significant.

RESULTS

The study was conducted in the department of Cardiac surgery, BSMMU from January 2013 to June 2014.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group I n=22</th>
<th>Group II n=22</th>
<th>P value 1</th>
<th>P value 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEDV</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre operative</td>
<td>74±6.2</td>
<td>76±6.4</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Post operative</td>
<td>72±6.1</td>
<td>82±6.8</td>
<td>&lt;0.01</td>
<td></td>
</tr>
<tr>
<td>6 months follow up</td>
<td>67±5.2</td>
<td>84±6.9</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>P value 1</td>
<td>NS</td>
<td>&lt;0.01</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P value 2</td>
<td>&lt;0.01</td>
<td>&lt;0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVESV</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre operative</td>
<td>50±4.2</td>
<td>52±4.6</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Post operative</td>
<td>49±4.1</td>
<td>55±4.8</td>
<td>&lt;0.01</td>
<td></td>
</tr>
<tr>
<td>6 months after surgery</td>
<td>47±3.9</td>
<td>56.5.1</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>P value 1</td>
<td>NS</td>
<td>&lt;0.01</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P value 2</td>
<td>&lt;0.01</td>
<td>&lt;0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ejection Fraction</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre operative</td>
<td>58±5.2</td>
<td>56±5.4</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Post operative</td>
<td>58±5.6</td>
<td>53±4.9</td>
<td>&lt;0.01</td>
<td></td>
</tr>
<tr>
<td>6 months follow up</td>
<td>63±6.4</td>
<td>50±4.6</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>P value 1</td>
<td>NS</td>
<td>&lt;0.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P value 2</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Group I: Sub valvular apparatus preserved. Group II: Sub valvular apparatus was not preserved
n= Number of subjects. NS= Not significant
P value Data were analyzed using unpaired t-test. Presented with mean±SD between the groups
p value1 Data were analyzed using paired t-test (within the groups before & after MVR)
p value2 Data were analyzed using paired t-test (within the groups before MVR & at 6 months follow up)
The mean age of the patients were 32±8 years. In group I, patients age was 29±7 years and in group II 36±9 years. There was no significant difference of age distribution between the groups. In both the groups female number was dominant.

In [Table I] patients with sub valvular apparatus resection (group I) had deterioration with postoperative ejection fraction in compare to group-II (Preserved mitral valve apparatus).

**Table 2: Comparison of left ventricular wall stress between the groups in total patients and in patients comprising mitral stenosis alone.**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group I n=16</th>
<th>Group II n = 16</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left ventricular systolic wall stress in mitral stenosis (K dynes)</td>
<td>Pre operative: 107±22</td>
<td>108±18</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>Post operative: 96±17</td>
<td>109±21</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td></td>
<td>6 months total: 98±19</td>
<td>116±26</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>P value 1</td>
<td>&lt;0.05</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>P value 2</td>
<td>&lt;0.05</td>
<td>&lt;0.01</td>
<td></td>
</tr>
</tbody>
</table>

Group I: Sub valvular apparatus preserved. Group II: Sub valvular apparatus was not preserved
n= Number of subjects. NS= Not significant.
P value Data were analyzed using unpaired t-test. Presented with mean±SD between the groups
p value1 Data were analyzed using paired t-test (within the groups before & after MVR).
p value2 Data were analyzed using paired t-test (within the groups before MVR & at 6 months follow up)

**DISCUSSION**

None of the studies in literature search showed left ventricular wall stress measurement in patients with mitral stenosis undergoing MVR with or without subvalvular apparatus preservation. Previous reports which showed increased wall stress after MVR in non-preserved group included patients with mitral regurgitation. In patients with mitral regurgitation, following mitral valve replacement there is increase of left ventricular afterload due to loss of low resistance forward volume to left atrium and reduction of preload caused by elimination of regurgitation. All these changes contribute to increase stress in patients with mitral regurgitation after chordal transection. The similar changes do not occur in patients with mitral stenosis. In our study patients with mitral stenosis showed increased wall stress after transection of sub valvular apparatus during MVR. This early change is probably attributed to lack of subvalvular apparatus that helps in restoration of normal early systolic spherical shape of the left ventricle after MVR and thus contributes to increase preload in the circumferential wall and eventually reduce the stress. [3,11,12] Subsequently in long run there is increase in left ventricular dimensions in the non-preserved group that contributes further to the increase in wall stress in this group.

The decrease in wall stress after MVR in preserved group could be attributed to increase in preload due to elimination of left ventricular inflow obstruction and unchanged ventricular dimension. The preserved sub valvular apparatus may also help in reducing stress in this group by maintaining left ventricular spherical shape during early systole. Probably this can also explain the increment of left ventricular ejection fraction in some studies after MVR with preserved subvalvular structure [3,13,14]

Data of the present study from sub group analysis of patients with mitral stenosis are the first to demonstrate increase of left ventricular wall stress after MVR with chordal transection in patient with mitral stenosis which may be the probable mechanism of worsening of left ventricular function in these patients.

**CONCLUSION**

The increased left ventricular wall stress is responsible for poor outcome in non-preserved group after MVR. The wall stress increases further in midterm follow up which may explain the mechanism of long term poor out come in patients with mitral stenosis.

**REFERENCES**

9. Yun KL, Sintek CF, Miller DC, Plofker TA, Kochamba GS, Khonsari S et al. Randomized trial comparing partial versus complete chordal-sparing mitral valve replacement: effects on


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