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Original Article

Immediate impact of successful percutaneous balloon mitral valvuloplasty on right and left ventricular functions: An echocardiographic study using load independent tissue velocity imaging indices

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ABSTRACT

Background: The impact of successful percutaneous balloon mitral valvuloplasty (PBMV) on left ventricular (LV) function has been a controversial subject. This study aimed to determine the immediate impact of PBMV on biventricular function using recent Tissue Velocity Imaging (TVI) derived load-independent indices.

Methods and results: A total of 30 patients with severe mitral stenosis (MS) who underwent PBMV at a tertiary center of India from August 2012 to December 2013 were included in the study. Thirty age-matched and gender-matched healthy controls were also enrolled.

Out of 30 patients, 27(90%) were female. Mean mitral valve area (MVA) of patients before and after PBMV was 0.78 and 1.82 cm^2 ($p < 0.001$), respectively. All TVI-derived LV and RV basal systolic (IVCV, Sm and the relatively load independent IVA) and diastolic velocities (Em, Em/Am) were significantly decreased in patients with MS compared to controls ($p < 0.001$ for all) which improved significantly after PBMV (6.4 ± 0.7 vs 11 ± 1.6; 5.8 ± 0.7 vs 9.9 ± 1.6; 1.5 ± 0.3 vs 4.2 ± 0.6; 6.4 ± 0.6 vs 13.1 ± 2.1; 0.7 ± 0.1 vs 1.7 ± 0.2 for mitral annulus respectively, $p < 0.001$ for all). Increment in MVA positively correlated with Tricuspid annular plane systolic excursion (TAPSE) and tricuspid annular Sm and isovolumic contraction velocity (IVCV) and inversely with left atrium (LA) size and Pulmonary arterial systolic pressure (PASP) ($p = 0.01$ for LA size; $p < 0.001$ for others) while no such correlation was found with mitral annulus isovolumic acceleration (IVA) ($r = -0.078$; $p = 0.679$).

Conclusion: The improved right ventricular (RV) function appears to be predominantly due to afterload reduction, while that of LV appears to be more due to the acute relief of mechanical restraint.

1. Introduction

Rheumatic fever has been the major engender of mitral stenosis (MS), and though the prevalence of MS has dwindled in developed countries, it still remains one of the most pervasive heart diseases in developing countries.[1] In the developing countries, MS evolves more rapidly, possibly because of severe or repeated streptococcal infections, genetic influences, or economic conditions.[2]

Mitral stenosis instigates deleterious effects on both left and right side of the heart. It leads to rise in left atrial pressure, which passively increases pulmonary venous and arterial pressures. This ultimately results in right ventricular dilatation and dysfunction.[3] MS leads to alteration in interaction between right and left ventricles, myocardial fibrosis and decrease in preload, which probably delineate to be a cause for left ventricular (LV) dysfunction.[4]

While impairment of the right ventricle is quite clinically apparent, the involvement of left ventricle is often subclinical. With conventional parameters of global longitudinal function like LV ejection fraction (LVEF), recognition of LV systolic dysfunction is significantly delayed as these measures are insensitive to the degree of myocardial damage. With advent of newer imaging modalities like tissue velocity imaging (TVI) and speckle tracking echocardiography (STE) derived strain imaging, subclinical LV dysfunction can be recognized before significant myocardial damage.

Isovolumic acceleration (IVA) is a TVI parameter used to assess systolic function of the ventricles. It is calclulated as a ratio of the tissue Doppler derived peak isovolumic contraction velocity (IVCV)

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to the time taken by the IVCV to reach its peak value (isovolumic acceleration time). Isovolumic acceleration (IVA) has been validated in animal[5,6] and clinical[7,8] settings and found to be unaffected to the changes in preload and afterload within physiological range.[5–8] It is sensitive to small changes in contractility and correlated well with invasive and noninvasive measures of LV dP/dT.[6,9]

Though various studies have incorporated TVI and strain imaging parameters in assessing the effect of percutaneous balloon mitral valvuloplasty (PBMV) on LV function, the role of acute changes in the afterload and preload following a successful PBMV on LV function is unclear. Thus, this study aimed to determine the immediate impact of percutaneous balloon mitral valvuloplasty (PBMV) on biventricular function using the TVI derived load independent indices.

2. Methods

2.1. Study design and patient population

This was an observational study in which a total of 30 patients with severe symptomatic MS who underwent PBMV between August 2012 and December 2013 were enrolled at a tertiary centre in India. Thirty age-matched and gender-matched healthy subjects were also enrolled as the control group.

Consecutive symptomatic patients with evidence of severe MS (mitral valve area (MVA) < 1.0 cm^2), in whom PBMV was feasible were included. Those who fulfilled the PBMV intervention criteria and those who had good result, i.e., successful PBMV (MVA > 1.5 cm^2 and less than moderate MR) were included. All participants gave their written informed consent and the study complies with Declaration of Helsinki and study protocol was approved by Institutional Ethics Committee.

Patients were excluded if they had 1) coexistence of moderate or severe mitral regurgitation, 2) concomitant hemodynamically significant valvular disease, 3) past closed or open mitral valvulotomy, past percutaneous mitral commissurotomy, 4) atrial fibrillation or atrioventricular conduction abnormalities or non-sinus rhythm, 5) any disease that could affect myocardial function (e.g. coronary artery disease, chronic lung disease, cardiomyopathies, congestive heart failure, systemic hypertension, diabetes mellitus or pericardial disease), 6) bad quality echocardiographic imaging, 7) PBMV result not meeting the successful PBMV criteria, 8) informed refusal.

2.2. Study procedure

All procedures were performed by the anterograde *trans*-septal approach. The Inoue balloon stepwise technique was used in all patients under fluoroscopy guidance. A successful PBMV was defined as an MVA ≥ 1.5 cm^2 and absence of complications including a mitral regurgitation of >2/4 grade. Results of detailed echocardiographic evaluations performed 1 to 24 h before PBMV and 48–72 h after PBMV were compared with results from 30 age-matched and gender-matched healthy control subjects.

All participants underwent transthoracic echocardiography (TTE) evaluation (iE33, S5-1 transducer, Koninklijke Philips N.V., Amsterdam, Netherlands). The TTE, including 2-dimensional and Doppler echocardiographic studies, were performed in the left lateral decubitus position with conventional views (parasternal long, short axis and apical 4 chamber view) according to the American Society of Echocardiography guidelines.[10,11]

The MVA was calculated both by direct planimetric method on short-axis view during diastole and by pressure half-time method. The mean and peak diastolic transmitral pressure gradients were determined from apical 4-chamber view.

Velocity tissue imaging of the right and left ventricles was performed using standard apical views at a high frame rate (150 ± 10 frames/s) and a sector angle of less than 60°. The spectral velocity signal filters were adjusted to obtain Nyquist limits of + 20 and −20 cm/s, with the lowest wall filter settings and the minimal optimal gain, to eliminate the signals produced by transmitral flow. A 10-mm sampling gate was placed for obtaining velocities from the bright lateral corners of the mitral and tricuspid annulus. The peak annular velocities of systolic excursion in isovolumic contraction and ejection period (IVCV, Sm), early and late diastole (Em, Am), isovolumic contraction and relaxation times (IVCT and IVRT), isovolumic acceleration (IVA = IVCV/IVAT where IVAT: isovolumic acceleration time, is the time taken by the IVCV to reach peak value) and ejection times (ET) were recorded and averaged over 3 heartbeats. The mitral and tricuspid annular velocities were measured by a single observer blinded to the data of the study and the changes in mitral valve geometry and gradients.

2.3. Statistical analysis

All quantitative data were expressed as mean ± SD. Basal parameters for control subjects and patients with mitral stenosis were compared with non-paired Student *t*-test. To compare the differences between pre-procedure and post-procedure parameters among MS patients, paired *t*-test was used where the differences were normally distributed. For the parameters with the differences not normally distributed, Wilcoxon signed rank test was used. Correlation between normally distributed continuous variables was calculated using Pearson's Correlation co-efficient and those not normally distributed using Spearman's Correlation co-efficient (rho). $P < 0.05$ was considered significant. Data was analyzed using open source statistical package R (R Foundation for Statistical Computing; Vienna, Austria), version 3.0.1.

Table 1
Baseline parameters of the patients.

Parameters	Control (N = 30)	MS Patients (N = 30)	p-value
Age (mean ± SD, years)	37.2 ± 6.7	36.8 ± 6.7	0.82
Sex (Male/Female)	3/27	3/27	1.00
BMI (mean ± SD, Kg/m^2)	22.63 ± 2.7	23.03 ± 2.4	0.54
Heart rate (mean ± SD, per min)	70 ± 3	72 ± 4	0.12
NYHA functional class			
Class I, n	30	0	<0.001
Class II, n	0	0	
Class III, n	0	21	
Class IV, n	0	9	
MVA Planimetry (mean ± SD, cm^2)	–	0.78 ± 0.1	–

MS – Mitral Stenosis; BMI – Body Mass Index; NYHA – New York Heart Association; MVA – Mitral Valve Area.

3. Results

Of the 30 patients who were enrolled in the study, mean age of the patients was 36.8 ± 6.7 years. The study population included higher proportion of female (n = 27; 90%). The baseline clinical parameters of patient group and control group are detailed in Table 1.

Conventional echocardiographic parameters like LVEF, left ventricular end-diastolic diameter (LVEDD), mitral annular plane systolic excursion (MAPSE), left atrial diameter (LA size), pulmonary artery systolic pressure (PASP), right atrial pressure (RA pressure) and tricuspid annular plane systolic excursion (TAPSE) (a surrogate of right ventricular systolic function), varied significantly in the patients group when compared to the control group (Table 2).

All the TVI derived left ventricular and right ventricular basal systolic (IVV, IVA, Sm) and diastolic velocities (Em, Am) were decreased significantly in the patients with MS compared to the control group. In patients with MS, IVRT and myocardial performance index (MPI) for both left ventricle and right ventricle were significantly higher than the control group ($p < 0.001$) and the ET was lower than the control group (Table 3).

Various conventional echocardiographic and TVI derived measures of biventricular function were assessed both before (pre PBMV) and after (post PBMV) balloon valvuloplasty. Following successful PBMV, mean MVA significantly increased from 0.78 to 1.82 cm^2 ($p < 0.001$) (Table 4). Significant differences were noted in the TVI indices, before and after PBMV (Table 5). Significant decrease was observed in LA size, mean RA pressure and pulmonary artery systolic pressure (PASP) ($p < 0.001$) after PBMV. The LVEDD increased while LVESD decreased significantly. The M-mode surrogates of left ventricle (MAPSE), right ventricle (TAPSE) and RV dP/dT also significantly improved after PBMV ($p < 0.001$). The TVI derived velocities of both mitral and tricuspid lateral annular area as in systole and diastole (IVCV, Sm, Em, Em/Am), isovolumic and ejection times (IVCT, IVRT, ET) and IVA (isovolumic acceleration) increased significantly after PBMV ($p < 0.001$) (Fig. 1).

All the TVI measures of mitral lateral annulus, acquired before PBMV were analyzed for correlation, with conventional echocardiographic measures like mean gradient, MVA, LA size, LVEF and PASP. No significant correlation was found between any of these measures for the left ventricle (Table 6). On the other hand, MVA showed significantly positive correlation, while LA size and PASP showed significant inverse correlation with tricuspid TVI derived Sm and IVCV ($p < 0.001$) (Table 7).

Further, the increment in MVA following successful procedure was assessed for correlation with the change in the above parameters (Table 8). The increment in MVA showed significant positive correlation with TAPSE ($p < 0.001$), tricuspid annular Sm & IVCV velocities ($p < 0.001$) while it showed significant inverse correlation with LA size ($p = 0.01$) and PASP ($p < 0.001$).

4. Discussion

Tissue velocity imaging is a technique depends on frequency shift information rather than on the reflected signal amplitude required for grey-scale imaging.[12] Therefore, information can be obtained even in the absence of an optimal image quality. Tissue Doppler derived strain and strain rate imaging have been validated in numerous studies to measure myocardial function and contractility, but is limited by its angle dependence (strain analysis is restricted along lines parallel to the ultrasound beam). Speckle tracking echocardiography (STE), on the other hand, by analyzing speckle motion, assesses myocardial tissue velocity, strain and strain rate independently of cardiac translation and beam angle, however, requires further processing and interpretation of data to be done offline. The software requires harmonic and high frame rate imaging. The real power of speckle analysis is the ability to examine several components or planes (i.e. radial, longitudinal and circumferential) in a single data set.

While STE derived strain and strain rate imaging is the current gold standard in assessing the regional myocardial contractility, it is not entirely load independent and the process of indexing the values to end diastolic volumes to make them relatively load independent, is painstaking. While STE is more of research oriented modality and not easily available in most of the echo labs, IVA is easily obtained and available, reproducible and doesn't need offline analysis and more importantly, is load independent.

In this study, TVI derived indices have been applied for assessing LV and RV functions before and after a successful PBMV.

Various studies have evaluated either left[4,13–15] or right ventricular[3,16–18] functions before and after PBMV, through TVI. But the data relating to simultaneous evaluation of both ventricles is scarce. To the best of our knowledge, till date, apart from ours, only one study has utilized TVI derived relatively load independent indices to evaluate biventricular and LA mechanical functions following PBMV.[19]

In this study, 30 symptomatic relatively young patients with severe mitral stenosis were compared with age and gender-matched 30 healthy people. As expected, LA dimension and PASP were higher in the MS group than in the control group. This was due to the decreased forward flow and transmitted back pressure in the pulmonary circulation, leading to reactive and obliterative changes in the pulmonary vascular bed, if not relieved early.

Although within normal range, the LVEDD and LVEF were significantly lower compared to control group. These parameters along with MAPSE significantly increased following the PBMV while the LVESD decreased. This seemingly suggests increased left ventricular filling and contractility. But these parameters are not load independent. This was contrary to the studies by Sengupta et al,[20] Drighil et al[16] and Mahfouz et al,[18] which showed no difference in these parameters after the procedure. This may be due to the stringent inclusion (pre PBMV MVA < 1.0 cm^2 and post

Table 2
Conventional echocardiographic parameters between control and mitral stenosis patients' groups.

Parameters	Control (N = 30)	MS Patients (N = 30)	p-value
LV EDD (mean ± SD, cm)	4.6 ± 0.3	4.1 ± 0.3	<0.001
LV ESD (mean ± SD, cm)	2.8 ± 0.2	2.9 ± 0.1	0.02
LV EF (mean ± SD, %)	63 ± 4	55 ± 5	<0.001
MAPSE (mean ± SD, mm)	18 ± 2	8 ± 1	<0.001
LA size (mean ± SD, cm)	2.9 ± 0.3	4.8 ± 0.6	<0.001
PASP (mean ± SD, mmHg)	20.7 ± 1.8	68.4 ± 14	<0.001
RA pressure (mean ± SD, mmHg)	3.0 ± 2.0	6.9 ± 4	<0.001
TAPSE (mean ± SD, mm)	23.2 ± 1.9	11.7 ± 2.2	<0.001

LVEDD – left ventricular end-diastolic diameter; LVESD – left ventricular end-systolic diameter, LVEF – left ventricular ejection fraction; MAPSE – mitral annular plane systolic excursion; LA size – left atrial diameter; PASP – pulmonary artery systolic pressure; RA pressure – right atrial pressure; TAPSE – tricuspid annular plane systolic excursion.

Table 3
Tissue velocity derived indices comparing mitral stenosis patients with control group.

Parameters	Control (N = 30)	MS Patients (N = 30)	p-value
Mitral Sm (mean ± SD, cm/s)	12.1 ± 1.5	5.8 ± 0.7	<0.001
Em (mean ± SD, cm/s)	15.6 ± 1	6.4 ± 0.3	0.02
Em/Am (mean ± SD)	1.5 ± 0.2	0.7 ± 0.1	<0.001
IVCV (mean ± SD, cm/s)	14.1 ± 1.5	6.4 ± 0.7	<0.001
IVA (mean ± SD, m/s^2)	4.4 ± 0.4	1.5 ± 0.3	<0.001
ET (mean ± SD, ms)	359 ± 16	250 ± 9	<0.001
IVRT (mean ± SD, ms)	68.8 ± 2.9	97.5 ± 10.6	<0.001
MPI (mean ± SD)	0.31 ± 0.03	0.68 ± 0.1	<0.001
Tricuspid Sm (mean ± SD, cm/s)	16 ± 2.3	7 ± 1.3	<0.001
Em (mean ± SD, cm/s)	14.7 ± 2.3	6 ± 0.7	<0.001
Em/Am (mean ± SD)	1.3 ± 0.3	0.6 ± 0.2	<0.001
IVCV (mean ± SD, cm/s)	17.5 ± 2.4	7.5 ± 1.3	<0.001
IVA (mean ± SD, m/s^2)	4.4 ± 0.4	1.7 ± 0.2	<0.001
ET (mean ± SD, ms)	349 ± 16	220 ± 9	<0.001
IVRT (mean ± SD, ms)	50.1 ± 4.2	79.9 ± 6.9	<0.001
MPI (mean ± SD)	0.41 ± 0.03	0.66 ± 0.09	<0.001

Sm – peak myocardial velocity during systole; Em – peak myocardial velocity during early diastole; Am – peak myocardial velocity during late diastole; IVCV – peak myocardial velocity during isovolumic contraction; IVA – myocardial acceleration during isovolumic contraction; ET – ejection time; IVRT – isovolumic relaxation time; MPI – myocardial performance index.

Table 4
Mitral valve area and transmitral gradients before and after percutaneous balloon mitral valvuloplasty.

Parameters	Pre PBMV (n = 30)	Post PBMV (n = 30)	p-value
MVA (mean ± SD, cm^2)	0.78 ± 0.1	1.82 ± 0.3	<0.001
Peak gradient (mean ± SD, mmHg)	23.4 ± 3.2	10.8 ± 1.6	<0.001
Mean gradient (mean ± SD, mmHg)	13.7 ± 2.9	4.1 ± 0.6	<0.001
PHT (mean ± SD, ms)	307 ± 44.3	119 ± 14.3	<0.001

MVA – mitral valve area; PHT – pressure half time.

Table 5
Tissue velocity derived Indices before and after percutaneous balloon mitral valvuloplasty.

Parameters	Pre PBMV (n = 30)	Post PBMV (n = 30)	p-value
LA size (mean ± SD, cm)	4.8 ± 0.6	3.9 ± 0.7	<0.001
LV EDD (mean ± SD, cm)	4.1 ± 0.4	4.6 ± 0.3	<0.001
LV ESD (mean ± SD, cm)	2.9 ± 0.2	2.6 ± 0.2	<0.001
LV EF (mean ± SD, %)	54.5 ± 4.5	67.4 ± 3.1	<0.001
MAPSE (mean ± SD, mm)	8.3 ± 1.2	14.7 ± 0.6	<0.001
RA pressure (mean ± SD, mmHg)	6.9 ± 3.9	3.0 ± 1.8	<0.001
RV dP/dT (mean ± SD, mmHg/s)	322.9 ± 46.8	1051.5 ± 251.8	<0.001
TAPSE (mean ± SD, mm)	11.7 ± 2.2	22.6 ± 2.2	<0.001
PASP (mean ± SD, mmHg)	68.4 ± 14	29.4 ± 5.7	<0.001
Mitral Sm (mean ± SD, cm/s)	5.8 ± 0.7	9.9 ± 1.6	<0.001
Em (mean ± SD, cm/s)	6.4 ± 0.6	13.1 ± 2.1	<0.001
Em/Am (mean ± SD)	0.7 ± 0.1	1.7 ± 0.2	<0.001
IVCV (mean ± SD, cm/s)	6.4 ± 0.7	11 ± 1.6	<0.001
IVA (mean ± SD, m/s^2)	1.5 ± 0.3	4.2 ± 0.6	<0.001
IVRT (mean ± SD, ms)	97.5 ± 10.6	67.8 ± 6.1	<0.001
MPI (mean ± SD)	0.68 ± 0.1	0.39 ± 0.03	<0.001
Tricuspid Sm (mean ± SD, cm/s)	7.0 ± 1.3	16.8 ± 1.6	<0.001
Em (mean ± SD, cm/s)	6.0 ± 1.3	16.0 ± 2.6	<0.001
Em/Am (mean ± SD)	0.7 ± 0.2	1.6 ± 0.2	<0.001
IVCV (mean ± SD, cm/s)	7.5 ± 1.3	17.2 ± 1.5	<0.001
IVA (mean ± SD, m/s^2)	1.7 ± 0.2	4.4 ± 0.7	<0.001
IVRT (mean ± SD, ms)	79.9 ± 6.8	54.6 ± 6.7	<0.001
MPI (mean ± SD)	0.66 ± 0.1	0.37 ± 0.1	<0.001

LVEDD – left ventricular end-diastolic diameter; LVESD – left ventricular end-systolic diameter; LVEF – left ventricular ejection fraction; MAPSE – mitral annular plane systolic excursion; LA size – left atrial diameter; PASP – pulmonary artery systolic pressure; RA pressure – right atrial pressure; TAPSE – tricuspid annular plane systolic excursion; Sm – peak myocardial velocity during systole; Em – peak myocardial velocity during early diastole; Am – peak myocardial velocity during late diastole; IVCV – peak myocardial velocity during isovolumic contraction; IVA – myocardial acceleration during isovolumic contraction; ET – ejection time; IVRT – isovolumic relaxation time; MPI – myocardial performance index.

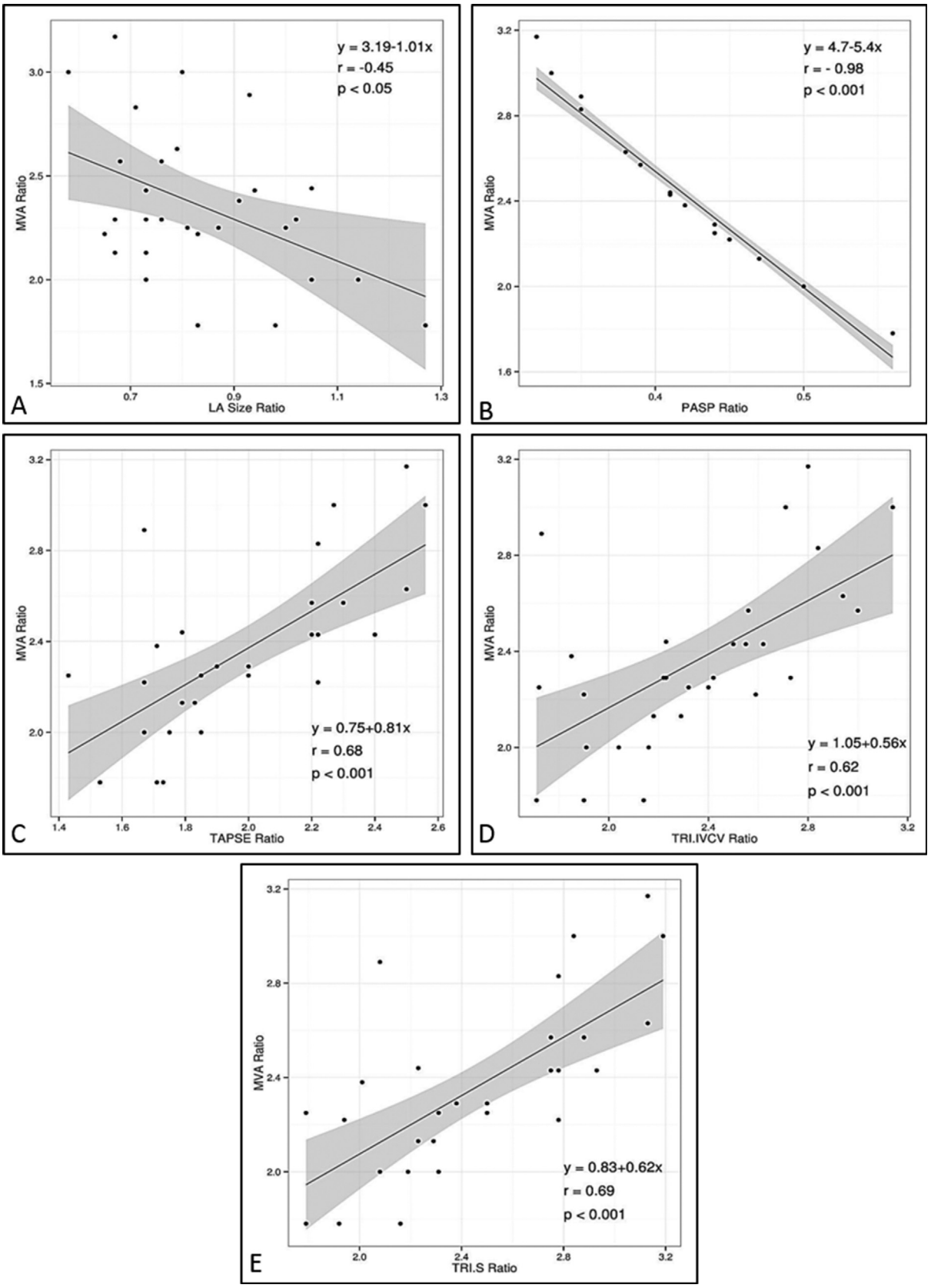

Fig. 1. Plot showing the correlation between increment in mitral valve area (ratio of mitral valve area after percutaneous balloon mitral valvuloplasty to before percutaneous balloon mitral valvuloplasty) and left atrial size (A), pulmonary artery systolic pressure (PASP) (B), tricuspid annular plane systolic excursion (TAPSE) (C), Tricuspid isovolumic contraction velocity (IVCV) (D) and Tricuspid myocardial velocity during systole (Sm) (E).

Table 6
Correlation between tissue velocity imaging derived measures of mitral lateral annulus and conventional echocardiographic parameters before percutaneous balloon mitral valvuloplasty.

Parameters		Sm	Em	Am	Em/Am	IVCV	IVA	IVRT	MPI
Mean Gradient	r-value	0.21	−0.18	−0.08	−0.007	0.120	0.249	−0.247	0.059
	p-value	0.26	0.34	0.68	0.743	0.528	0.183	0.189	0.757
MVA	r-value	0.750	0.051	0.132	−0.214	0.134	0.414	−0.147	0.289
	p-value	0.694	0.789	0.458	0.256	0.480	0.222	0.438	0.121
LA size	r-value	−0.175	−0.121	−0.205	0.134	−0.041	0.316	0.056	0.418
	p-value	0.355	0.523	0.278	0.481	0.829	0.088	0.769	0.214
EF	r-value	0.129	−0.008	−0.110	0.168	0.122	−0.007	0.144	0.306
	p-value	0.497	0.963	0.563	0.375	0.520	0.972	0.448	0.100
PASP	r-value	−0.225	−0.155	−0.133	−0.021	−0.119	−0.455	0.088	0.410
	p-value	0.233	0.413	0.483	0.913	0.532	0.116	0.644	0.245

EF – ejection fraction; LA size – left atrial diameter; PASP – pulmonary artery systolic pressure; Sm – peak myocardial velocity during systole; Em – peak myocardial velocity during early diastole; Am – peak myocardial velocity during late diastole; IVCV – peak myocardial velocity during isovolumic contraction; IVA – myocardial acceleration during isovolumic contraction; IVRT – isovolumic relaxation time; MPI – myocardial performance index.

Table 7
Correlation between tissue velocity imaging derived measures of tricuspid lateral annulus and conventional echocardiographic parameters before percutaneous balloon mitral valvuloplasty.

Parameters		Sm	Em	Am	Em/Am	IVCV	IVA	IVRT	MPI
Mean Gradient	r-value	−0.223	−0.195	−0.178	0.007	−0.178	−0.066	0.135	0.089
	p-value	0.237	0.303	0.346	0.969	0.346	0.729	0.475	0.639
MVA	r-value	0.716*	0.080	−0.087	0.110	0.690*	0.110	−0.166	−0.093
	p-value	<0.001*	0.673	0.645	0.562	<0.001*	0.565	0.381	0.628
LA size	r-value	−0.896*	0.046	−0.061	−0.011	−0.870*	0.296	0.065	−0.275
	p-value	<0.001*	0.247	0.325	0.951	<0.001*	0.112	0.730	0.142
EF	r-value	−0.091	0.105	−0.037	0.005	−0.064	0.320	0.234	0.306
	p-value	0.630	0.556	0.198	0.976	0.735	0.085	0.195	0.101
PASP	r-value	−0.924*	0.107	−0.134	0.083	−0.910*	0.246	0.129	−0.195
	p-value	<0.001*	0.566	0.711	0.664	<0.001*	0.190	0.498	0.302

EF – ejection fraction; LA size – left atrial diameter; PASP – pulmonary artery systolic pressure; Sm – peak myocardial velocity during systole; Em – peak myocardial velocity during early diastole; Am – peak myocardial velocity during late diastole; IVCV – peak myocardial velocity during isovolumic contraction; IVA – myocardial acceleration during isovolumic contraction; IVRT – isovolumic relaxation time; MPI – myocardial performance index.

Table 8
Correlation between increments in planimetered mitral valve area with improvement in tissue velocity imaging derived measures of mitral and tricuspid lateral annulus and conventional echocardiographic parameters after percutaneous balloon mitral valvuloplasty.

Parameters	MVA (Post PBMV/Pre PBMV)	
	r-value	p-value
Mean gradient	−0.277	0.138
LA size	−0.457	0.011*
LV EF	0.080	0.672
MAPSE	0.024	0.897
RV dP/dT	0.285	0.126
TAPSE	0.687	<0.001*
PASP	−0.982	<0.001*
Mitral Sm	−0.008	0.962
Em	0.279	0.134
IVCV	−0.007	0.972
IVA	−0.078	0.679
IVRT	−0.307	0.098
MPI	0.230	0.222
Tricuspid Sm	0.697	<0.001*
IVCV	0.620	<0.001*
IVA	0.345	0.061
MPI	−0.051	0.786

LVEF – left ventricular ejection fraction; MAPSE – mitral annular plane systolic excursion; LA size – left atrial diameter; PASP – pulmonary artery systolic pressure; TAPSE – tricuspid annular plane systolic excursion; Sm – peak myocardial velocity during systole; Em – peak myocardial velocity during early diastole; IVCV – peak myocardial velocity during isovolumic contraction; IVA – myocardial acceleration during isovolumic contraction; IVRT – isovolumic relaxation. *TAPSE ($p < 0.001$), tricuspid annular Sm & IVCV velocities ($p < 0.001$), LA size ($p = 0.01$) and PASP ($p < 0.001$).

PBMV MVA > 1.5 cm^2) criteria of this study and both increased filling and improved LV function would account for these results.

The IVCV and the IVA, that is found to be relatively load independent in previous studies by Vogel et al[5,6] and Drighil et al,[16] were found to be significantly low in this study when compared with healthy controls. Thus, all measures of LV systolic function (Sm, IVCV, IVA, ET) and diastolic function (Em, Am, Em/Am) derived by TVI were significantly lower in patients with MS than those in controls (MPI & IVRT were higher in MS patients than in the controls) and no correlation was found between any of these parameters and MVA, mean transmitral gradient, LA size, LVEF and PASP.

Furthermore, Sm, Em/Am, IVCV, IVA, IVRT and MPI from TVI of mitral lateral annulus showed significant improvement after PBMV but the improvement in none of these parameters correlated with the increment in MVA, suggesting this improvement was not due to altered loading conditions alone. This proves that the functional changes in LV myocardium are partly reversible and show immediate improvement after PBMV. While chamber atrophy because of myocardial fibrosis in mitral stenosis is unlikely to show immediate improvement after PBMV, internal myocardial restriction caused by increased myocardial stiffness because of immobility of the mitral leaflets and subvalvular apparatus can be reversed immediately after PBMV. Mobilization of the mitral apparatus can lead to rapid reversal of the increased myocardial stiffness and to improved motion and function of the subvalvular structures and myocardial segments, which explains the immediate improvement in mitral annular velocities seen in our patients. Significant increase in load independent parameter, i.e., IVA, was observed in our patients after PBMV. Thus, indicating that LV myocardial

contractile function was improved due to PBMV and not due to the increased LV filling alone. The improvement in annular velocities after PBMV, thus, most probably reflects the acute changes in motion and compliance of the mitral valve apparatus and underlying tethered myocardial segments apart from improved LV filling.

Impaired RV contractile reserve portends high risk for a poor outcome after PBMV. Hence, the use of load independent measures of RV function is crucial for preoperative RV assessment and determining eventual prognosis.[21] Kjaergaard et al[22] showed that RV IVA was unaffected by at least moderate levels of augmented volume and pressure loading. Therefore, it appears that IVA is likely the most trustworthy parameter of RV function.

In this study, the LA size and PASP were significantly higher and conventional surrogate markers of right ventricular systolic function like TAPSE and RV dP/dT were significantly lower than in the control groups. Consistent with other studies by Mahfouz et al[18] and Tayyeraci et al,[23] this study showed significantly decreased TVI derived RV parameters (Sm, Em, Em/Am, IVCV, IVA) compared to control subjects while the IVRT and MPI were significantly higher. Also, the peak tricuspid annular velocities Sm and IVCV showed strong positive correlation with MVA and strong inverse correlation with LA size and PASP before PBMV, suggesting the depressed longitudinal function of right ventricle is mainly due to increased pulmonary artery pressure (afterload) and that, lack of correlation between RV IVA and MVA suggests RV IVA to be relatively load independent. Moreover, the increment in MVA after PBMV strongly correlated with the decrement in LA size and PASP and increment in TAPSE, peak tricuspid annular velocities Sm and IVCV, but not with improvements in IVA. This was contrary to a study by Drighil et al,[16] who found no significant difference in Sm, IVCV and IVA in MS patients after PBMV, explained probably by the inclusion of patients with more severe disease and better outcomes in this study.

The significantly depressed function of both the ventricles as assessed by the regional and global TVI derived parameters, in symptomatic severe MS patients compared to healthy controls, reflects the significance of timely intervention of the stenotic mitral valve. The Sm and IVCV of tricuspid annulus correlated with the MVA before PBMV, suggesting their load dependency especially in the presence of RV dysfunction as showed by Sade et al,[21] and the lack of correlation between MVA and IVA may suggest the latter's relative load independency. On the other hand, the lack of correlation between mitral annular TVI parameters (Sm, Em, Em/Am, IVCV, IVA, IVRT and MPI) with MVA before PBMV and lack of correlation between the increment in MVA after PBMV and these indices suggest the depression of the LV longitudinal function is more due to internal myocardial restriction, myocardial stiffness, decreased compliance of the mitral valve apparatus and possibly the underlying tethered myocardial segments, rather than change in the loading conditions alone (because the load independent parameter i.e., IVA, had significantly increased after PBMV).

Thus, the fear of inducing more left ventricular dysfunction, by performing PBMV in patients with severe MS, due to sudden increase in preload, in a chronically underfilled and unprepared LV, is unwarranted in majority of patients, as PBMV not only improves hemodynamic factor but also relieves the myocardial restraint and improves LV mechanical function.

4.1. Limitations of the study

The current study has some limitations. First, a small number of patients included in this study. Second, systolic function parameters were not compared with the parameters obtained from cardiac catheterization and magnetic resonance imaging or other new modalities such as three-dimensional echocardiography and Strain imaging. Follow-up study of the patients to reassess the biventricular function was not done.

4.2. Conclusions

Both, load dependent peak annular velocities Sm, IVCV and the relatively load independent, isovolumic acceleration are significantly depressed in patients with severe MS and these parameters significantly improve after successful PBMV.

While the improved RV function appears to be predominantly due to afterload reduction (reduction in pulmonary vascular resistance and pulmonary artery pressure), improvement in LV function appears to be more due to acute changes in motion and compliance of the mitral valve apparatus, reduction in myocardial stiffness and release of the underlying tethered myocardial segments, rather than change in the loading conditions alone.

Conflict of interest

None of the authors have any conflict of interest.

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