ORIGINAL INVESTIGATION

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Immediate impact of percutaneous transvenous mitral commisurotomy on right ventricle longitudnal strain in patients of mitral stenosis

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Background: Right ventricular (RV) function has prognostic value in terms of survival and symptoms in patients with mitral stenosis (MS). The aim of the study was to assess RV function by strain analysis in the patients of mitral stenosis and the effect of percutaneous transvenous mitral commisurotomy (PTMC) on it.

Methods: Eighty patients of severe mitral stenosis without overt right heart failure and normal sinus rhythm undergoing PTMC were included. Conventional echocardiography and RV function by TDI-derived longitudinal strain and strain rate were assessed prior and 24 hours post PTMC and compared with 40 healthy age-matched controls.

Results: Eighty subjects (mean age 31 + 10 years, 70% females) were included. Patients with MS had significantly lower RV strain of basal and mid-free wall, tricuspid annular plane systolic excursion (TAPSE), and RV fractional area change (FAC) as compared to controls. There was a significant increase in pre- and post-PTMC in TAPSE (19.5 ± 2.7 mm vs 21.4 ± 3.3 mm; $P < 0.001$), RV basal free wall longitudinal strain (−24.4 + 6.1% vs −27.7 + 5.8%; $P < 0.001$), and right ventricle mid-free wall longitudinal strain (−25.6 + 5.5% vs −28.6 + 5.1%; $P < 0.001$), respectively. There was no significant change in RV Tei index (0.43 + 0.06 vs 0.41 + 0.03; $P = 0.06$). There was a significant negative correlation between RV longitudinal strain and right ventricle systolic pressure, left atrium diameter, RV Tei index, and pulmonary capillary wedge pressure, and positive correlation between RV FAC and RV TAPSE.

Conclusion: Patients with severe MS with normal RV systolic function had decreased RV strain, which was significantly increased after a successful PTMC with reduction in afterload.

KEYWORDS

mitral stenosis, myocardial strain, right ventricular function

1 | INTRODUCTION

Systolic dysfunction of the right ventricle (RV) is a well-known prognostic indicator in patients of rheumatic heart disease (RHD) with mitral stenosis (MS).[1,2] RV dysfunction is frequently overlooked before the appearance of clinical features of systemic venous congestion because of difficulties in the quantitative assessment of RV function. The evaluation of global and regional RV function is difficult because of its complex trapezoidal anatomy and thin wall structure.[3,4] As the RV systolic function is a major determinant of prognosis and survival of MS patients, early detection of RV failure is of particular importance.[5-7] Traditionally, global RV systolic function has been assessed by ejection fraction (EF) that has limitations such as being subjective, load dependent, and affected by contractile state.[8] Tissue Doppler Imaging (TDI)- and 2D-derived strain and strain rate analysis are the most widely used noninvasive techniques for the evaluation of RV myocardial function.[9-12]

Strain and strain rate values have been studied in a number of conditions affecting the right heart, including arrhythmogenic RV dysplasia, pulmonary embolism, pulmonary hypertension, systemic right ventricle, and amyloidosis.[13-17] However, there are only a few studies, assessing RV function in patients with mitral stenosis and effect of percutaneous transvenous mitral commisurotomy (PTMC) on RV function assessed by strain.[18]

The aim of the study was to assess RV function by TDI-derived longitudinal strain in patients of severe mitral stenosis and immediate impact of a successful PTMC on these parameters.

2 | METHODS

2.1 | Study population and design

Patients of mitral stenosis presented to our institute from January 2015 to January 2016 were prospectively included in the study. Patients with symptomatic mitral stenosis in NYHA class II–III, normal sinus rhythm, normal right ventricle systolic function and suitable for PTMC were enrolled after taking informed consent. Patients with atrial fibrillation, overt heart failure, left ventricular ejection fraction (LVEF) < 50%, aortic regurgitation greater than a mild degree or aortic stenosis, mitral regurgitation greater than a mild degree, right ventricle failure, hypertension, diabetes mellitus, severe calcification of mitral valve annulus, clinical or laboratory evidence of active rheumatic disease, chronic obstructive or restrictive lung disease, chronic pulmonary thromboembolism, and low-quality echocardiographic image were excluded from the study. Patients who developed more than moderate mitral regurgitation after PTMC were also excluded from the study.

A total of 80 patients of RHD with severe MS (defined as mitral valve area ≤ 1.0 cm^2) with suitable valve morphology for PTMC were enrolled. Indications for PTMC were New York Heart Association (NYHA) class ≥ II, planimetry mitral valve area (MVA) ≤ 1.0 cm^2, mitral regurgitation ≤ 2+, suitable valve morphology, and the absence of concomitant cardiovascular disease requiring surgical correction. All subjects underwent standard 2D transthoracic echocardiography and Tissue Doppler Imaging (TDI)-derived RV strain parameters 24 hours prior to PTMC and 24 hours post-PTMC. These parameters were compared with 40 healthy age matched controls.

2.2 | Echocardiographic measurements

Echocardiographic evaluation was done on GE Vivid 7 ECHO machine (GE healthcare, Waukesha, WI, USA) with 3.5 Hz probe. All the measurements were performed according to guidelines for assessment of right heart set by American society of echocardiography (ASE).[19]

Two-dimensional and pulse wave Doppler echocardiographic studies were performed in the left lateral decubitus position with conventional views (parasternal long and short axis, apical 4-chamber).

All subjects were evaluated by a common observer. Mitral valve area (MVA) was calculated by the planimetry and pressure half-time methods, and the mean value of these 2 measurements was determined as MVA. Maximum and mean diastolic transmitral gradients were calculated by continuous wave Doppler echocardiography.

Right ventricular systolic pressure (RVSP) was estimated by continuous wave Doppler echocardiography using the modified Bernoulli equation (4 × [peak tricuspid regurgitation]^2), adding 10 mm Hg for the assumed right atrial pressure. The Wilkins score was used to judge mitral leaflet mobility, valvular and subvalvular thickening and calcification.

Tricuspid inflow velocity was recorded from the apical 4-chamber view by pulsed wave Doppler sample volume positioned at the tips of the tricuspid leaflets during diastole. Peak early (E) and late (A) tricuspid inflow velocity and deceleration time of E velocity were obtained. The RV outflow velocity was recorded from the parasternal short-axis view with the pulsed wave Doppler sample volume positioned just below the pulmonary valve. RV ejection time (RVET) was measured from the onset to the end of RV outflow. Isovolumetric relaxation time (IRT) was obtained as the time interval from the cessation of RV outflow to the onset of tricuspid valve inflow. Isovolumetric contraction time (ICT) was determined from the cessation of tricuspid inflow to the onset of RV outflow.[20] RV myocardial performance index (RV MPI) was calculated by the formula (ICT + IRT)/RVET.[21]

Tricuspid annular plane systolic excursion (TAPSE) was measured in M-mode, using M-mode cursor in apical 4C view, at the junction of the anterior leaflet of the tricuspid valve with the RV free wall. Maximum displacement during systole was evaluated and averaged over 3 consecutive beats as shown in Figure 1. The RV fractional area change (RV FAC %), defined as (End diastolic area—End systolic area/End diastolic area × 100) was also calculated. RV FAC% is a measure of RV systolic function that has been shown to correlate with RV EF by magnetic resonance imaging (MRI).[22] All the measurements were calculated from 3 consecutive cycles and the average of 3 measurements was recorded.

2.3 | RV TDI strain analysis

Echocardiography images were obtained from the apical 4-chamber view. To calculate strain, high frame rates images were acquired (100 frames/s). Narrow imaging sector focusing on the RV free wall were acquired. Care was taken to align the segment in the center of the sector to avoid errors due to the angle dependence of Doppler. Imaging in color-coded tissue Doppler mode, and ≥3 beats were acquired with suspended respiration. Values for strain were then derived by placing sample volume(s) or regions of interest of varying sizes in the mid-portion of the RV free wall basal and mid-level.[23] Cardiac cycles with extrasystolic beats, postextrasystolic beats, or any rhythm disturbances were excluded. Longitudinal peak systolic strain (PSS) and longitudinal peak strain rate (PSSR) of RV free wall base and mid-segments were analyzed. (Figure 2). Apical segment was excluded from analysis because of increase chance of error due to angle dependency. To avoid the confounding factor of the left ventricle interventricular septum was not assessed. Simsek et al[24] found that the peak systolic strain rate and strain values at the interventricular septum and the lateral left ventricle wall were significantly lower in patients with pure mitral stenosis, who had normal ejection fraction compared to healthy subjects.

2.4 | Percutaneous transvenous mitral commisurotomy (PTMC)

All patients underwent PTMC by the antegrade transseptal approach using an Accura balloon (Vascular Concepts) and a stepwise dilatation strategy. The nominal balloon diameter was decided as per height of the patient (balloon diameter = height (cm)/10 + 10). The MVA was calculated using the Gorlin equation.[25] The success of the procedure was defined as postprocedural planimetery MVA > 1.5 cm^2 by echocardiography and/or a 50% increase over the preprocedural value and nondevelopment of 3+ or 4+ mitral insufficiency.
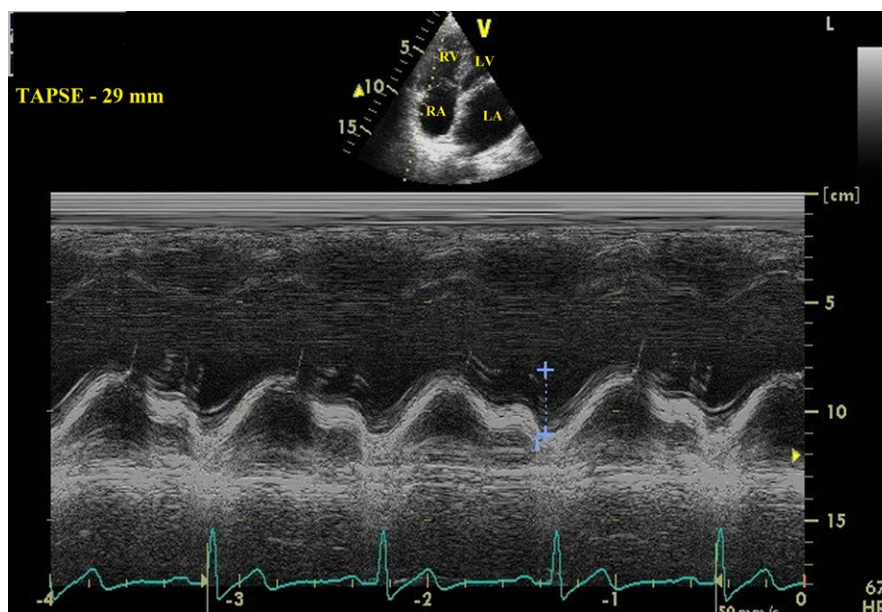

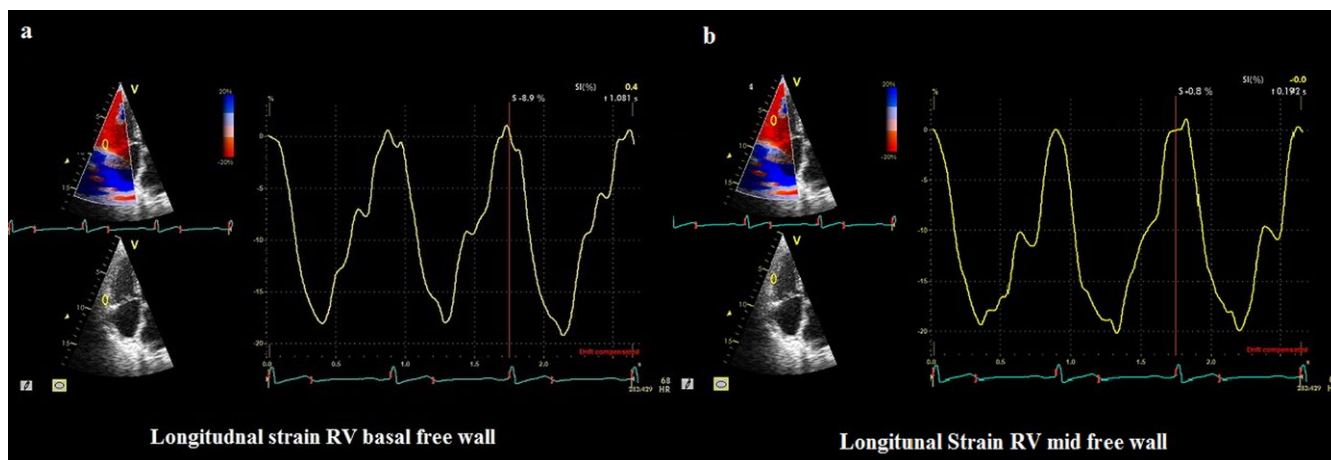**FIGURE 1** Measurement of tricuspid annular plane systolic excursion (TAPSE)

FIGURE 2 TDI-derived longitudinal strain at basal (A) (yellow arrow) and mid-segment of right ventricular free wall (B) (red arrow)

2.5 | Statistical analysis

Data are expressed as mean ± standard deviation for continuous variables, and number and percentage of subjects for categorical ones. The chi-square test with Yates' correction was used to assess the significance of differences between categorical variables. Continuous variables were compared by Student t test or the Mann–Whitney rank sum test. Correlations between different parameters were determined using Pearson product moment correlation coefficient. Statistical analyses were performed using SPSS software package (SPSS Inc., Chicago, IL, USA) version 16.0 for Windows and $P < 0.05$ was considered statistically significant.

3 | RESULTS

Baseline characteristic of patients are shown in Table 1. The study screened total 81 patients of RHD with severe MS, out of which 1 patient developed severe Mitral regurgitation (MR) post-PTMC, hence only 80 patients (98%) were analyzed pre- and post-PTMC. Mean age of patients were 31 ± 10 years. Majority of them were female (70%). New York heart association (NYHA) functional class III was the predominant class of presentation (68%), none was in class IV. Mean duration of symptom was 36 ± 14 months with all patients in sinus rhythm. Severe mitral regurgitation occurred due to chordae

TABLE 1 Baseline demographic characteristics of the patients with severe mitral stenosis (cases)

Parameters	N = 80
Mean age (y)	31 ± 10
Female sex	56 (70%)
NYHA	
II	16 (32%)
III	34 (68%)
IV	0 (0%)
Normal sinus rhythm	80 (100%)
Mean heart rate (beats/min)	72 ± 11
Mean duration of symptoms (mo)	36
Mean MVA (cm^2)	0.8 ± 0.2
Mitral regurgitation	
No	28 (57%)
Mild	22 (43%)
Tricuspid regurgitation	
Mild	30 (60%)
Moderate	17 (34%)
Severe	3 (6%)
Mean Wilkin's score	8 ± 2
Mean RVSP (mm Hg)	50 ± 11

MVA = mitral valve area; NYHA = New York Heart Association; RVSP = right ventricular systolic pressure.

rupture in 1 patient and mitral valve replacement was performed in the same hospital admission.

3.1 | Comparison with control group

Echocardiographic parameters were compared between 80 mitral stenosis patient and 40 healthy controls (Table 2). Age was similar in both the groups. Left atrial (LA) anteroposterior dimension in parasternal long-axis (PLAX) view measured by M-mode echocardiography was greater in MS group (32.4 ± 4.1 mm vs 48.2 ± 4.6 mm; $P < 0.001$). Patients with mitral stenosis had higher estimated RVSP (50 ± 11 mm Hg vs 25 ± 4.7 mm Hg; $P < 0.001$), RV Tei index (0.43 ± 0.06 vs 0.34 ± 0.03; $P < 0.001$), lower TAPSE (19.5 ± 2.7 mm vs 23.5 ± 2.8 mm; $P = 0.001$), and lower FAC (43.8 ± 8.5% vs 52.0 ± 5.7%; $P < 0.001$) compared to controls. Patients with severe MS had significantly lower longitudinal PSS RV basal free wall (−24.4 ± 6.1% vs −29.7 ± 3.1%; $P < 0.001$) and mid-RV free wall (−25.6 ± 5.5% vs −28.4 ± 2.2%; $P = 0.001$). PSSR?

3.2 | Pre- and Post-PTMC echocardiography

Comparison between pre- and post-PTMC echocardiographic parameters are shown in Table 3. After PTMC, there was a significant increase in MVA (0.8 ± 0.2 cm^2 vs 1.6 ± 0.2 cm^2; $P < 0.001$) and significant decrease in peak transmitral gradient (24 ± 7 vs 12 ± 3 mm Hg; $P < 0.001$) and the mean transmitral gradient (13 ± 4 vs 6 ± 2 mm Hg; $P < 0.001$). There was significant fall in RVSP (50 ± 11 vs 32 ± 7 mm Hg; $P < 0.001$) and left atrium diameter (48.2 ± 4.6 mm vs 42.1 ± 3.2 mm; $P < 0.001$) after PTMC. RV FAC

TABLE 2 Comparison of echocardiographic parameters in patients with severe mitral stenosis and controls

Variable	Control (n = 40) mean ± SD	Cases (n = 80) mean ± SD	P value
Mean age (y)	31.8 ± 5.25	31 ± 10	0.79
Left atrium size (mm)	32.4 ± 4.1	48.2 ± 4.6	<0.001*
RVSP (mm Hg)	25 ± 4.7	50 ± 11	<0.001*
RV FAC (%)	52.0 ± 5.7	35.8 ± 8.5	<0.001*
TAPSE (mm)	23.5 ± 2.8	19.5 ± 2.7	0.001*
RV MPI	0.34 ± 0.03	0.43 ± 0.06	<0.001*
PSS RV basal free wall	−29.7 ± 3.1%	−24.4 ± 6.1%	<0.001*
PSS RV mid-free wall	−28.4 ± 2.2%	−25.6 ± 5.5%	0.001*
PSSR RV basal free wall (s^{-1})	−1.98 ± 0.42	−2.05 ± 0.30	0.43
PSSR RV mid-free wall (s^{-1})	1.88 ± 0.56	−1.97 ± 0.35	0.32

PSS = peak systolic strain; PSSR = peak systolic strain rate; RV FAC = right ventricle fractional area change; RV MPI = right ventricle myocardial performance index; RVSP = right ventricular systolic pressure; TAPSE = tricuspid annular plane systolic excursion. *P value significant.

(43.8 ± 8.5% vs 49.7 ± 10.5%; P < 0.001) and TAPSE (19.5 ± 2.7 mm vs 21.4 ± 3.3 mm; P < 0.001) S′ wave were increased significantly immediately post-PTMC. There was nonsignificant fall in RV MPI immediately after PTMC (0.43 ± 0.06 vs 0.41 ± 0.03; P = 0.06). There was a significant fall in the invasive PA systolic pressure and mean PCWP.

There was a significant increase in longitudinal PSS RV basal free wall (−24.4 ± 6.1% vs −27.7 ± 5.8%; P < 0.001) and longitudinal PSS RV mid-free wall (−25.6 ± 5.5% and −28.5 ± 5.1%; P < 0.001) after PTMC (Figure 3). There was no significant difference between longitudinal PSSR RV basal free wall (−2.05 ± 0.30 s^{-1} vs −1.97 ± 0.31 s^{-1}; P = 0.15) and longitudinal PSSR RV mid-free wall (−1.97 ± 0.35 s^{-1} vs −2.09 ± 0.34 s^{-1}; P = 0.2) after PTMC.

3.3 | Correlation of RV PSS

Regression analysis showed a significant inverse correlation between pre-PTMC, RVSP and longitudinal PSS basal RV free wall (r = −0.42, P = 0.03) and longitudinal PSS mid-RV free wall (r = −0.23, P = 0.05) which maintained in post-PTMC (Table 4). There was also inverse relation between pre-PTMC RV Tei index and basal RV free wall strain (r = −0.35; P = 0.02) and mid-RV free wall strain (r = −0.46; P = 0.05) which was lost after post-PTMC. LA diameter also showed a strong

TABLE 3 Comparison of echocardiographic parameters before and after PTMC in patients with severe mitral stenosis

Variable	Pre-PTMC (n = 80)	Post-PTMC (n = 80)	P value
Mean MV peak gradient (mm Hg)	24 ± 7	12 ± 3	<0.001*
Mean MV mean gradient (mm Hg)	13 ± 4	6 ± 2	<0.001*
Mean MV area (cm^2)	0.8 ± 0.2	1.6 ± 0.2	<0.001*
Mean RV systolic pressure (mm Hg)	50 ± 11	32 ± 7	<0.001*
RV FAC (%)	35.8 ± 8.5	44.7 ± 10.5	<0.001*
LA diameter (mm)	48.2 ± 4.6	42.1 ± 3.2	<0.001*
TAPSE (mm)	19.5 ± 2.7	21.4 ± 3.3	<0.001*
RV MPI	0.43 ± 0.06	0.41 ± 0.03	0.06
PSS RV basal free wall	−24.4 ± 6.1%	−27.7 ± 5.8%	<0.001*
PSS RV mid-free wall	−25.6 ± 5.5%	−28.5 ± 5.1%	<0.001*
PSSR RV basal free wall (s^{-1})	−2.05 ± 0.30	−1.97 ± 0.31	0.15
PSSR RV mid-free wall (s^{-1})	−1.97 ± 0.35	−2.09 ± 0.34	0.12
Cath PAP (mm Hg)	49.4 ± 13.5	30.8 ± 8.3	<0.001*
Cath PCWP (mm Hg)	26.7 ± 5.3	15.5 ± 3.9	<0.001*

FAC = fractional area change; LA = left atrium; MV = mitral valve; PAP = pulmonary artery pressure; PCWP = pulmonary capillary wedge pressure; PSS = peak systolic strain; PSSR = peak systolic strain rate; RV MPI = right ventricle myocardial performance index; RV= right ventricle; TAPSE = tricuspid annular plane systolic excursion. *P value significant.
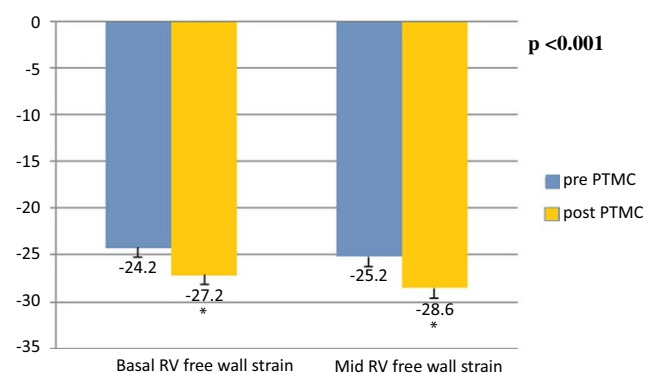
negative correlation with RV longitudinal strain at both segment before PTMC, which were lost after PTMC. RV basal and mid-free wall longitudinal PSS was positively correlated with RV TAPSE and RV FAC both pre-and post-PTMC. There was no significant correlation between MVA and right ventricle longitudinal strain both pre- and post-PTMC, except that it showed positive correlation with RV mid-free wall strain after PTMC (r = 0.2; P = 0.03). Similarly the MV peak and mean gradient did not show any significant correlation to RV strain before and after PTMC.

4 | DISCUSSION

Our study showed significantly lower longitudinal PSS of right ventricle in patients with MS as compared to the controls which was significantly increased after a successful PTMC. Correlation analysis revealed a significant negative correlation between RVSP, LA diameter, RV MPI, invasive PAP, and PCWP with longitudinal PSS. There was significant positive correlation between RV TAPSE and RV FAC with longitudinal PSS. It correlates with earlier hemodynamic and clinical studies, which showed impaired RV function in MS patients assessed by various echocardiographic parameters.[2,5,18,26–28]

The increase in RV afterload is mainly contributed to RV dysfunction in these patients. Left atrial hypertension due to mitral stenosis leads to chronic pulmonary venous congestion, which ultimately leads to pulmonary hypertension. However some authors had suggested that the direct rheumatic involvement of the RV with resultant myocyte necrosis, replacement fibrosis, and calcification is the explanation of such depressed myocardial function.[29–31]

Our study found out that peak longitudinal segmental strain at basal RV free wall and mid-RV free wall was significantly lower, in patient with RHD with severe MS, as compared to controls. These results are not in concordance with the findings by Ozdemir et al[27] who showed lower global RV and interventricular septum strain with normal RV free wall strain in patients with MS as compare to control subjects, but they had included only patient with mild to moderate MS. MS. However, Yildirimturk et al[32] found significantly reduced strain and strain rate at the RV free wall segments in patients with

FIGURE 3 Difference in longitudinal strain at basal and mid-segment of right ventricle free wall pre- and immediate post-PTMC

moderate–severe MS compared to control group. The impaired RV free all strain values were attributed to increased RV afterload and higher wall stress to ventricular dilatation. There were significant rise in peak systolic segmental RV strain at basal and mid-free wall post-PTMC. This supports the observation by Weidemann et al,[33] that strain can be affected by loading conditions.

Immediate after PTMC, there occurred significant fall in RVSP (50 ± 11 mm Hg vs 32 ± 7 mm Hg; P < 0.001) along with significant rise in RV FAC (43.8 ± 8.5% vs 49.7 ± 10.5%; P < 0.001) and TAPSE (19.5 ± 2.7 mm vs 21.4 ± 3.3 mm; P < 0.001), which indicates improvement in RV function. These observations are same as by Burger et al[34] who showed immediate improvement in RV ejection fraction post-PTMC. This rapid improvement of RV function can be explained only by reduction in afterload by relieving left atrial hypertension and resultant decrease in RVSP. It has been reported that pulmonary pressure usually normalizes within 6 months, but may stay elevated for more than 2 years in some patients.[2]

The PSS RV free wall was significantly negative correlated with RVSP (..) which suggest strain values depends on afterload. This was concordant with the study by Drighil et al[35] which included 21 patients who demonstrated a significant decrease in RVSP post-PTMC and a positive correlation between Tie index and RVSP before PTMC. We also observed a negative correlation of RV longitudinal strain both pre- and post-PTMC, with invasive PA pressure and invasive PCWP; however this was stronger before the PTMC. There was no significant correlation found between RV longitudinal strain and mitral valve area. The peak and mean mitral inflow gradient showed some nonsignificant negative correlation with RV free wall segmental strain at both basal and mid-level, post-PTMC (r = −0.14; P = 0.1 and r = −0.13; P = 0.1, respectively, for mean mitral gradient), this is in concordance with a previous study done by Yildirimturk et al,[32]

they found a significant inverse correlation between RV strain and mean transmitral gradient (r = −0.358, P = 0.027).

There was significant immediate reduction in LA anteroposterior dimension post-PTMC (48.2 ± 4.6 mm vs 42.1 ± 3.2 mm; P < 0.001) and the LA diameter was found to have a negative correlation with RV longitudinal strain pre-PTMC (r = −0.43; P = 0.01 with basal RV strain and r = −0.36; P = 0.04 with mid-RV strain) but not after the PTMC. This finding is concordant with a recently published study by Adavane et al,[36] who showed immediate decrease in LA volume after BMV in patients in sinus rhythm. The most probable explanation of this immediate reduction in LA size is decompression of LA and better emptying by releasing the mitral valve obstruction by the BMV.

This study showed that RV Tie index did not changed significantly 24 hours after PTMC (0.43 ± 4.6 vs 0.41 ± 0.03; P < 0.001). This is similar to a study done by Mohan et al[2] who studied 25 consecutive patients with isolated rheumatic MS before, immediately after (mean, 40 ± 12 hours) and a mean follow-up of 11.5 months after PTMC. They found that The RV Tie index was not affected immediately after a successful PTMC, however, at follow-up of about 1 year, the Tie index showed a significant decrease. However in a study done by Drighil et al,[35] there was a decrease in RV Tei index post-PTMC.

Right ventricular Tei index is a relatively load independent parameter of the global RV function, which may not change immediately after PTMC. There is change in afterload immediately post-PTMC, which results in improvement in most of the load dependent parameters. We also observed that RV Tei index is negatively correlated with RV strain pre-PTMC but not post-PTMC. The drop in RV Tie index and RVSP together immediately post-PTMC suggest that RV systolic function improved as a result of

TABLE 4 Correlation between basal and mid-free wall right ventricle strain with other echocardiographic parameters

| Variable | PSS RV basal free wall | | | | PSS RV mid-free wall | | | |
| | Pre-PTMC | | Post-PTMC | | Pre-PTMC | | Post-PTMC | |
	r	P	r	P	r	P	r	P
MVA	0.02	0.4	0.03	0.3	0.11	0.1	0.2	0.03*
MVPG	0.12	0.1	−0.10	0.2	0.16	0.1	−0.10	0.3
MVMG	0.15	0.1	−0.14	0.1	0.06	0.2	−0.13	0.1
LA diameter	−0.43	0.01*	0.02	0.8	−0.36	0.04*	0.08	0.7
RVSP	−0.42	0.03*	−0.56	0.02*	−0.23	0.05*	−0.35	0.04*
RV TAPSE	0.53	0.04*	0.45	0.02*	0.39	0.03*	0.51	0.01*
RV FAC (%)	0.44	0.01*	0.34	0.05*	0.39	0.05*	0.40	0.03*
RV MPI	−0.35	0.02*	0.03	0.6	−0.46	0.05*	0.02	0.3
Cath PAP (mean)	−0.39	0.02*	−0.09	0.3	−0.54	0.01*	−0.03	0.5
Cath PCWP (mean)	−0.46	0.04*	−0.12	0.08	−0.49	0.03*	−0.08	0.06

FAC = fractional area change; LA = left atrium; MVA = mitral valve area; MVMG = mitral valve mean gradient; MVPG = mitral valve peak gradient; PAP = pulmonary artery pressure; PCWP = pulmonary capillary wedge pressure; r = Pearson's correlation coefficient; RV MPI = right ventricle myocardial performance index; RVSP = right ventricle systolic pressure; TAPSE = tricuspid annular plane systolic excursion.
*P value significant.

an acute decrease in RV afterload. This is concordant with the study by Borges et al[37] who demonstrated an improvement in Tie index after vasodilator therapy in patients with chronic pulmonary hypertension.

Thus, in the patients with severe MS, RV performance is decreased predominantly due to increase in RV afterload, which improves after PTMC, reflected by improved RV segmental strain at basal, mid-free wall. Alternatively there is a possibility that this immediate improvement in RV function is due to improved hemodynamics, better LV filling and RV emptying after PTMC as only the load dependent parameters were improved. Since there is no immediate change in load independent parameters like RV Tei index in previous studies as well as in the current study, this suggest RV myocardial structural changes secondary to long-standing increased pressure overload. Finally, significant improvement in these parameters in long-term studies supports the gradual remodeling of the RV after PTMC.

4.1 | Study limitations

The sample size of the study is small and larger sample size study is required to confirm the findings. Secondly RV strain was analyzed by TDI-derived method which is angle-dependent, speckle tracking 2D strain would have given a better results. Thirdly, only immediate post-PTMC values are available and no long-term follow-up is done. Fourthly, LA dimension was measured only in anteroposterior diameter in parasternal long-axis view. This single dimension may not represent actual LA size (particularly in dilated atria) and LA volume calculation would have been more accurate.

5 | CONCLUSION

RV strain can detect early RV systolic dysfunction before the appearance of overt right ventricle failure. RV strain is afterload-dependent and significantly improves after PTMC with reduction in right ventricle afterload.

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