



Original Research Article

ASSESSMENT OF RIGHT VENTRICULAR LONGITUDINAL STRAIN AND LEFT ATRIUM STRAIN IN PATIENTS WITH ISOLATED SEVERE RHEUMATIC MITRAL STENOSIS BEFORE AND AFTER BALLOON MITRAL VALVOTOMY

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ABSTRACT

Background: Rheumatic mitral stenosis (MS) continues to be a major cause of valvular heart disease in developing countries. Chronic left atrial (LA) pressure overload leads to progressive atrial fibrosis, remodeling, and right ventricular (RV) dysfunction. Myocardial strain imaging offers a sensitive tool for detecting these functional impairments and evaluating improvement after balloon mitral valvotomy (BMV). **Objective-** To assess the difference in right ventricular longitudinal strain and left atrial global strain at baseline and after BMV in patients with rheumatic severe mitral stenosis, and to compare these parameters along with conventional echocardiographic indices between cases and healthy controls.

Materials and Methods: This observational analytic study was conducted in the Department of Cardiology, Sawai Man Singh Medical College, Jaipur. A total of 44 patients with isolated rheumatic severe MS undergoing BMV and 44 age-matched healthy controls were enrolled. Standard echocardiographic parameters and strain imaging were obtained before and 24–48 hours after BMV. Statistical analysis was performed using Student's t-test and chi-square test, with $p \leq 0.05$ considered significant.

Results: The mean mitral valve area increased significantly from 0.87 ± 0.20 cm² to 1.52 ± 0.17 cm² ($p < 0.001$), while the transmitral gradient decreased from 13.20 ± 1.68 mmHg to 4.50 ± 0.85 mmHg ($p < 0.001$). Both LA strain and RV strain improved significantly after BMV. Pulmonary artery systolic pressure decreased markedly, while TAPSE and RVS' showed modest changes. Compared with controls, cases had reduced strain values and larger LA dimensions.

Conclusion: BMV leads to significant improvement in LA and RV strain and reduces pulmonary pressures in severe rheumatic MS, highlighting the role of strain imaging in peri-procedural evaluation and therapeutic monitoring.

Keywords: Balloon Mitral Valvotomy, Mitral Stenosis, Strain Imaging, Valvular Heart Disease

INTRODUCTION

Rheumatic mitral stenosis (MS) remains a major cause of cardiovascular morbidity in low- and middle-income regions. Progressive commissural fusion and subvalvular disease elevate left atrial (LA) pressure, transmit load to the pulmonary

circulation, and chronically increase right-ventricular (RV) afterload, setting the stage for RV remodeling and dysfunction even when left-ventricular ejection fraction is preserved. Contemporary practice guidelines and rheumatic heart disease (RHD) statements emphasize accurate echocardiographic characterization (valve area,

gradients, LA size) and timely intervention in anatomically suitable patients.^[1,2] Percutaneous balloon mitral valvotomy (PBMV) is widely recommended as the treatment of choice for symptomatic severe rheumatic MS with favorable anatomy because it reduces the transmitral gradient, lowers LA and pulmonary pressures, and increases mitral valve area (MVA).^[2,3]

Beyond conventional measurements, myocardial deformation imaging by speckle-tracking has refined our understanding of atrial and ventricular mechanics in MS. Multiple studies show that LA reservoir, conduit, and contractile strain are markedly reduced in severe rheumatic MS compared with healthy subjects, and these indices correlate with stenosis severity (MVA, gradients) and markers of hemodynamic burden.^[4,5] LA strain has also been linked with pro-thrombotic milieu and adverse rhythm outcomes, supporting its potential role in risk stratification for thromboembolism and atrial fibrillation.^[6] From an educational standpoint, LA strain functions as a sensitive barometer of chronic pressure and volume loading of the atrium—changes that may not be fully captured by LA diameter or volume alone.^[4-6]

On the right heart, conventional parameters—TAPSE, RV fractional area change (FAC), and tissue-Doppler S'₁—are integral but can miss early, regional dysfunction. Speckle-tracking-derived RV global longitudinal strain (RV-GLS) detects subclinical impairment in MS and relates to disease severity.^[7,8] This aligns with the pathophysiology: chronic LA hypertension and pulmonary vascular remodeling raise pulmonary artery pressure and RV afterload, precipitating longitudinal fiber dysfunction before global systolic indices decline.^[1,7,8] PBMV favorably modifies upstream loading, and classic hemodynamic series consistently demonstrate immediate reductions in pulmonary pressures and transmitral gradients with concomitant increases in MVA.^[9-11] Emerging deformation studies further suggest that RV longitudinal mechanics can improve soon after successful PBMV, indicating load-dependence and potential for early reverse remodeling.^[12]

By jointly tracking LA deformation (a marker of atrial remodeling and reservoir function) and RV-GLS (a sensitive index of right-sided systolic performance) across the peri-PBMV course, we can characterize the dynamic myocardial response to relief of mitral inflow obstruction and better delineate baseline differences versus health. Our study was designed to determine the difference in right ventricular longitudinal strain and left atrial global (reservoir) strain at baseline and after balloon mitral valvotomy in patients with severe rheumatic mitral stenosis, and to compare right ventricular longitudinal strain, left atrial strain, and conventional echocardiographic right ventricular parameters (fractional area change, TAPSE, and S'₁) between patients with severe rheumatic mitral stenosis and healthy controls.

MATERIALS AND METHODS

This observational analytic study was conducted in the Department of Cardiology, Sawai Man Singh Medical College, Jaipur, after approval from the Professor and Head of the Department and the Institutional Ethics Committee. Written informed consent was obtained from all participants prior to enrolment. The study population consisted of adult patients above 18 years of age with isolated rheumatic severe mitral stenosis, defined as a mitral valve area less than 1.5 cm² by two-dimensional planimetry, who were in sinus rhythm and admitted for balloon mitral valvotomy. Age-matched healthy volunteers were recruited as controls. A minimum of 36 patients was required at a 95% confidence interval and 80% power to detect the expected difference in strain parameters before and after balloon mitral valvotomy, and an equal number of controls was included for comparison. Thus, a total of 44 patients of mitral stenosis, and 44 controls were enrolled in the study. Patients with atrial fibrillation, New York Heart Association class I symptoms, pregnancy, chronic obstructive pulmonary disease, other valvular lesions, systemic hypertension, renal failure, poor echocardiographic windows, or those who developed more than mild mitral regurgitation after the procedure were excluded. Patients with a previous history of balloon mitral valvotomy or those who did not provide consent were also excluded.

Eligible patients fulfilling the inclusion criteria were enrolled consecutively until the required sample size was achieved. Data were collected using a structured proforma after explaining the study details and obtaining written consent. All participants underwent echocardiographic evaluation before the procedure and again at 24–48 hours after balloon mitral valvotomy. Echocardiography was performed using a GE Vivid S6 system with a 3.5 MHz probe, and all measurements were obtained according to the guidelines of the American Society of Echocardiography. Right ventricular longitudinal strain was assessed from the apical four-chamber RV-focused view. The right ventricular myocardium was divided into basal, mid, and apical segments of the interventricular septum and free wall, and both segmental and global peak systolic strain values were calculated. Conventional right ventricular functional parameters including fractional area change, tricuspid annular plane systolic excursion, and tissue Doppler systolic velocity at the tricuspid annulus were measured. The myocardial performance index was calculated as the sum of isovolumic contraction and relaxation times divided by ejection time. Pulmonary artery systolic pressure was estimated from tricuspid regurgitation jet velocity using the modified Bernoulli equation, with right atrial pressure determined from inferior vena cava diameter and its collapsibility with inspiration.

Left atrial strain was measured from apical four-chamber and two-chamber views by manual endocardial tracing and automated epicardial border detection to generate a region of interest across the atrial wall thickness. Global left atrial strain was calculated as the average peak atrial longitudinal strain across twelve atrial segments during the reservoir phase, using the onset of the QRS complex as the reference point. Left atrial volume and dimension were also recorded. All echocardiographic parameters were measured for five cardiac cycles, and the mean of three cardiac cycles was used for analysis. Strain analysis was performed using QLAB software of the GE Vivid S6 system, and frame rates between 40 and 80 frames per second were used for strain imaging.

Statistical Analysis: All continuous variables were expressed as mean and standard deviation. For paired data, Student's t-test was applied to assess the significance of differences in right ventricular functional parameters and left atrial parameters before and after balloon mitral valvotomy, after checking for normal distribution. Differences in mean values of the parameters between patients with mitral stenosis and healthy controls were analysed using the unpaired Student's t-test for normally

distributed variables, after verifying the assumption of homogeneity of variance, and the Mann-Whitney U test for non-normally distributed variables. A p-value of ≤ 0.05 was considered statistically significant. All statistical analyses were performed using SPSS software.

RESULTS

In the present study, the mean left atrial dimension among cases was 42.00 ± 2.17 mm. The mean Wilkins score, assessed out of 16, was 8.00 ± 0.41 . [Table 1] In the present study, among cases, the majority belonged to the 35–44 years age group (45.5%), followed by 25–34 years (22.7%), 45–54 years (20.5%), and 55–64 years (11.4%). In the control group, the most common age group was 35–44 years (36.4%), followed by 25–34 years (31.8%), 55–64 years (25.0%), and 45–54 years (6.8%). Females constituted the majority among cases (68.2%) compared to controls (52.3%). Males accounted for 31.8% of cases and 47.7% of controls. Both groups were comparable in terms of Age and Sex distribution ($p > 0.05$).

Table 1: Age and Sex wise distribution of Cases and Control

Variable	Case	Control	Test of significance
Age group (Years)	25-34	10(22.7)	X ² = 6.361, Df=3; P = 0.125
	35-44	20(45.5)	
	45-54	9(20.5)	
	55-64	5(11.4)	
Sex	Female	30(68.2)	X ² = 1.708 ,Df=1, P = 0.191
	Male	14(31.8)	

[Table 2] In the present study, the mean mitral valve area (MVA) significantly increased from 0.87 ± 0.20 before BMV to 1.52 ± 0.17 after BMV ($p < 0.001$). The mean transmitral gradient decreased from 13.20 ± 1.68 to 4.50 ± 0.85 , and the difference was statistically significant ($p < 0.001$). The mean longitudinal strain improved significantly from 13.61 ± 0.63 to 18.31 ± 0.99 following BMV ($p < 0.001$). Similarly, global RV strain increased from 13.34 ± 1.20 to 17.41 ± 1.82 , showing a statistically significant improvement ($p < 0.001$). The mean RVS Prime improved modestly from

11.27 ± 0.69 to 11.82 ± 0.58 , with a significant difference ($p = 0.001$). The pulmonary artery systolic pressure (PASP) decreased significantly after BMV, from 48.78 ± 4.27 to 30.11 ± 2.85 ($p < 0.001$). The RV MPI reduced from 0.58 ± 0.03 to 0.55 ± 0.03 , which was also statistically significant ($p < 0.001$). The mean TAPSE showed a slight but significant increase from 20.73 ± 1.35 to 21.25 ± 1.31 ($p = 0.018$). The fractional area change (FAC) also improved significantly after BMV, from 37.05 ± 1.28 to 42.82 ± 2.03 ($p < 0.001$).

Table 2: Comparison of echocardiographic parameters before and after balloon mitral valvotomy in cases

Variable	Pre		Post		p value
	Mean	SD	Mean	SD	
MVA	0.87	0.20	1.52	0.17	<0.001
Mean Gradient	13.20	1.68	4.50	0.85	<0.001
Longitudinal Strain	13.61	0.63	18.31	0.99	<0.001
Global RV Strain	13.34	1.20	17.41	1.82	<0.001
RVS Prime	11.27	0.69	11.82	0.58	0.001
PASP	48.78	4.27	30.11	2.85	<0.001
RV MPI	0.58	0.03	0.55	0.03	<0.001
TAPSE	20.73	1.35	21.25	1.31	0.018
FAC BMV	37.05	1.28	42.82	2.03	<0.001

[Table 3 & Figure 1] In the present study, the mean peak longitudinal strain among cases was

13.61 ± 0.63 , while in controls it was 33.77 ± 4.24 , and this difference was statistically significant

($p < 0.001$). The mean global RV strain in cases was 13.34 ± 1.20 , compared to 20.07 ± 2.25 in controls, and this difference was also statistically significant ($p < 0.001$). For RVS Prime, the mean value was 11.27 ± 0.69 in cases and 11.03 ± 0.59 in controls. The difference between the two groups was not statistically significant ($p = 0.087$). Similarly, the

mean TAPSE was 20.73 ± 1.35 in cases and 21.34 ± 1.85 in controls, with no statistically significant difference ($p = 0.080$). The mean left atrial dimension was significantly higher in cases (42.45 ± 2.20 mm) compared to controls (31.39 ± 2.30 mm), and this difference was statistically significant ($p < 0.001$).

Table 3: Comparison of echocardiographic parameters between cases and controls

Variable	Cases		Control		t value	p value
	Mean	SD	Mean	SD		
Peak Longitudinal Strain	13.61	0.63	33.77	4.24	31.208	<0.001
Global RV Strain	13.34	1.20	20.07	2.25	17.171	<0.001
RVS Prime	11.27	0.69	11.03	0.59	1.732	0.087
TAPSE	20.73	1.35	21.34	1.85	1.773	0.080
Left Atrium (mm)	42.45	2.20	31.39	2.30	23.021	<0.001

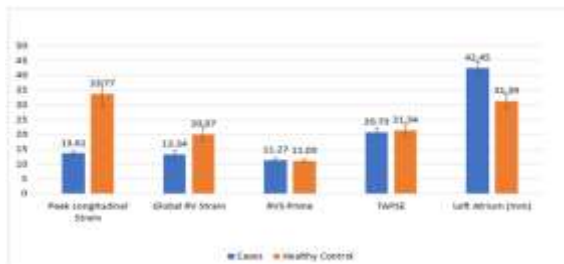


Figure 1: Echocardiographic parameters of cases and controls

DISCUSSION

Rheumatic mitral stenosis is characterized by progressive left atrial fibrosis, atrial remodeling, and right ventricular dysfunction secondary to chronic pressure overload. These pathophysiological changes lead to impaired atrial compliance, reduced reservoir function, and early right ventricular dysfunction, even before overt clinical deterioration. Myocardial strain imaging, which quantifies deformation as percentage change, provides a sensitive tool to detect these subclinical alterations and to assess improvement following balloon mitral valvotomy.

Demographic Profile: In our cohort, most patients with rheumatic severe MS were in the 35–44 year age band, and groups were age-comparable. This age profile aligns with contemporary PBMV series from endemic regions, where patients typically present in the third to fourth decade, often younger than Western degenerative valve cohorts. The similarity in age structure between cases and controls limits confounding by age on strain and conventional RV indices.

There was a female predominance among cases (68.2%), consistent with the well-described excess of rheumatic MS in women across registries and PBMV cohorts.^[13,14] This concordance reflects the usual epidemiology of rheumatic MS, supporting generalizability of the echocardiographic comparisons.

Pre- vs Post-BMV changes in cases: MVA increased and mean gradient fell markedly after BMV, reflecting commissural splitting and relief of

inflow obstruction. Similar immediate hemodynamic gains are consistently reported in PBMV studies and reviews.^[15,16] Mechanistically, the larger orifice reduces left-atrial (LA) pressure and pulmonary venous congestion, setting up downstream improvements in RV afterload.

The significant rise in longitudinal strain (your table's "Longitudinal Strain") after BMV is in line with evidence that LA global longitudinal strain (PALS/GLS) improves immediately and continues to improve at 6–12 months once the pressure load is relieved.^[17] Concordantly, prior work shows LA volume/dimension falls within 24–48 h and further regresses over weeks, tracking the drop in gradient and pulmonary venous pressures.^[16] Our short-term improvement therefore likely reflects prompt LA decompression; the degree of longer-term recovery depends on baseline fibrosis/remodeling.

Global RV strain improved significantly post-BMV, with modest gains in TAPSE and RVS' and a small decrease in RV MPI. This pattern mirrors studies showing that load-dependent indices (RV strain, TAPSE) respond early to afterload reduction, while load-independent or composite indices (e.g., MPI) may change less or later.^[15,18] Importantly, several series demonstrate immediate enhancement of RV strain after PBMV, attributable to reduced pulmonary pressures and improved RV–PA coupling.^[18–20] However, not all parameters evolve uniformly in the very early window; discordant immediate changes (e.g., IVA vs Tei) have been reported, underscoring metric-specific load sensitivity.^[15] Our findings therefore fit with an acute afterload-driven improvement in RV mechanics, with the possibility of further remodeling on follow-up.

The large fall in PASP after BMV reproduces the classic separation of a "passive" component (falls immediately with LA pressure and gradient) and a "reactive/fixated" arteriolar component (regresses over weeks, or may persist if longstanding). Patients with advanced pulmonary vascular remodeling can show attenuated or incomplete PASP regression despite adequate MVA, which has prognostic implications.^[21] Our early reduction is therefore

expected and mechanically coherent with the strain improvements.

In severe MS, chronic LA hypertension and pulmonary venous congestion increase RV afterload. BMV promptly unloads the LA, lowers transmitral gradient, and reduces RV afterload—improving load-sensitive indices (RV GLS, TAPSE, PALS) within 24–48 h. Composite or relatively load-independent indices (e.g., RV MPI) may lag, especially when myocardial remodeling/fibrosis is established.^[15,18,21] Immediate post-BMV gains in MVA and gradient; early reduction in PASP; improvement in RV strain and LA strain; and early but modest changes in TAPSE.

Cases vs Controls (between-group comparison)

Cases had markedly lower peak longitudinal strain than controls and substantially larger LA dimensions. This reproduces prior observations that LA reservoir strain is depressed in rheumatic MS and improves after BMV, while LA size is chronically enlarged from pressure overload.^[17,22] The magnitude of LA strain impairment in our cases is compatible with reported values for severe MS and underscores the sensitivity of LA strain to chronic LA load and fibrosis.

Global RV strain was significantly worse in cases than controls, aligning with studies showing subclinical RV systolic dysfunction in MS that is detectable by 2D-speckle tracking even when conventional indices are only mildly abnormal.^[18] In contrast, between-group differences in TAPSE and RVS' were small and not statistically significant in our data—also described in the literature, where strain often reveals early RV impairment before TAPSE/S' diverge from normal.^[23,24] Differences across studies likely reflect variation in PH severity, timing of measurements, and load-dependence of the indices used.

Overall, our results are similar to previous studies demonstrating immediate post-BMV gains in MVA and gradient, early PASP reduction, and improvement in RV and LA strain, along with modest changes in TAPSE.^[15-18] However, unlike some series that reported significant decrements in TAPSE and RVS' at baseline, our cohort showed preserved longitudinal annular motion. This may be explained by relatively less advanced disease and heterogeneity in pulmonary vascular involvement.^[18]

The strength of the present study lies in the use of both strain imaging and conventional echocardiographic parameters, with paired assessment before and after balloon mitral valvotomy, along with inclusion of an age-matched control group for comparison. However, the study was limited by its relatively small sample size, single-center design, short-term follow-up, and exclusion of patients with atrial fibrillation. It is recommended that larger multicenter studies with longer follow-up be conducted to validate these findings and to assess the long-term impact of

balloon mitral valvotomy on atrial and ventricular function.

CONCLUSION

In the present study, patients with rheumatic severe mitral stenosis undergoing balloon mitral valvotomy (BMV) demonstrated significant improvement in both conventional and advanced echocardiographic parameters. The mitral valve area increased and the transmitral gradient reduced significantly after BMV, indicating effective relief of obstruction. Right ventricular function, assessed by global strain, TAPSE, RVS', RV MPI, and fractional area change, showed measurable improvement following intervention. Left atrial global strain also improved, highlighting the beneficial impact of BMV on atrial compliance and reservoir function. When compared with healthy controls, patients with mitral stenosis had significantly impaired right ventricular strain and enlarged left atrial dimensions, underscoring the burden of functional impairment associated with the disease.

From a procedural and therapeutic point of view, balloon mitral valvotomy should be encouraged as an effective and less invasive treatment option for patients with rheumatic mitral stenosis, especially in younger individuals and in settings with limited resources. The regular use of strain imaging during the procedure can help in better selection of patients, deciding the right time for intervention, and monitoring treatment response, which may guide further management.

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