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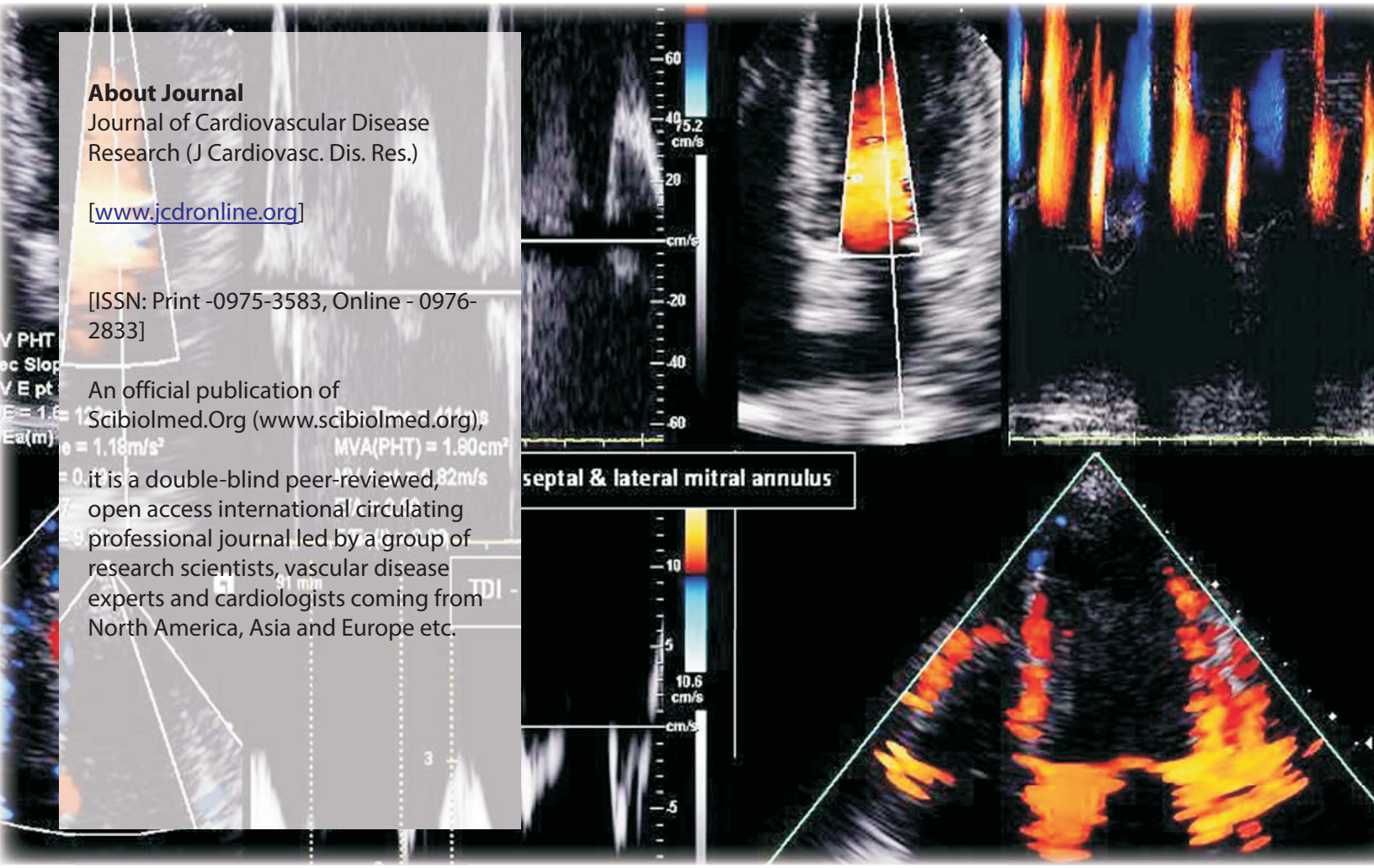

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Original Article

Assessment of RV function following Percutaneous Transvenous Mitral Commissurotomy (PTMC) for rheumatic mitral stenosis

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ABSTRACT

Objective: Abnormal right ventricular function plays an important role in development of clinical symptoms and overall prognosis of patients with mitral stenosis. Hemodynamic and radionuclide studies have demonstrated long-term improvement in RV function after PTMC. However, exact quantification by conventional echocardiographic technique is difficult owing to complex 2D anatomy. This study evaluates the immediate and short term follow up impact of successful percutaneous transvenous mitral valve commissurotomy (PTMC) on RV function in patients with mitral stenosis using 2D Echo and Doppler tissue imaging. **Methods:** 219 patients (mean age 26 ± 6yrs) with rheumatic MS, all in sinus rhythm were studied before and 24-48 hrs after PTMC. Parameters of global and longitudinal RV function were assessed by conventional 2D ECHO and Doppler tissue imaging. **Results:** Immediately following PTMC, mitral valve area increased from baseline of 0.71 ± 0.15 cm^2 to 1.84 ± 0.17 cm^2 (P 0.0001), RV outflow tract fractional shortening (RVOT–FS) increased from 33.94 ± 7.55% to 37.33 ± 7.67% (<0.001). There was a significant decrease in systolic pulmonary artery pressure from 48.93 ± 13.08 mmHg to 29.56 ± 7.71 (P<0.0001). RV Tei- index decreased from 0.47 ± 0.12 to 0.32 ± 0.11 (P 0.001). **Conclusion:** Long term evaluation of RV function and benefits using invasive and radionuclide methods post PTMC has shown incongruous results in improvement of right ventricular (RV) function immediately after PTMC. In this study, immediately after successful PTMC significant improvement in parameters of infundibular and global RV function as assessed by RVOT fractional shortening , Tei index and tissue Doppler velocities was observed.

Key words: PTMC, RV function, ECHO indices-2D and DTI, procedural outcome-immediate and short term.

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INTRODUCTION

Rheumatic heart disease causes significant morbidity and mortality among the cardiovascular disease. Mitral stenosis (MS) is the commonest presentation in rheumatic heart disease. Rheumatic mitral stenosis is a frequent cause of valve disease in developing countries.[1] Despite the striking decrease in the prevalence of rheumatic fever in western countries it still accounts for 12% of native valvular heart disease.[2] The treatment option and its timing should be decided on the basis of clinical, morphological, and functional characteristics. Since its introduction in 1984 by Inoue *et al*,[3] percutaneous transvenous mitral commissurotomy (PTMC) has been established as a safe and effective treatment for rheumatic mitral stenosis and remains the treatment of choice in patients with a favourable anatomy.[4,5,6]

The right ventricular(RV) function is an important determinant of clinical symptoms, exercise capacity, pre-operative survival and postoperative outcome in patients with mitral stenosis.[7] In patients with MS, the RV function may be altered due to an increase in the left atrial pressure and/or changes in the pulmonary arteriolar vasculature or may be affected by the rheumatic process directly.[8] The results of previous studies, using either invasive or radionuclide methods, demonstrated long-term improvement in RV function after PTMC.[9,10]

In patients with mitral stenosis (MS), previous studies have shown discordant results as regards to improvement of right ventricular (RV) function immediately after PTMC and none of these studies evaluated RV function. Hence, the purpose of this study was to evaluate the immediate and short term follow up impact of PTMC on RV function using two-dimensional and tissue Doppler echocardiographic indices.

Aim

The aim of this study was to evaluate the immediate impact of successful percutaneous transvenous mitral valve commissurotomy on RV function in patients with mitral stenosis using Two-dimensional echocardiography and Doppler tissue imaging (DTI).

Objectives

1. To assess the RV function in patients with mitral stenosis admitted for PTMC by various echocardiographic measures.
2. To compare RV global systolic function by measuring RVEF, RVFAC, TEI INDEX before and after PTMC.
3. To compare RV function as assessed by using Doppler tissue imaging before and after PTMC.
4. To assess RVOT function by measuring RVOT fractional shortening (fs) before and after PTMC.

MATERIAL AND METHODS

The study population consisted of 219 patients with symptomatic mitral stenosis who underwent percutaneous mitral balloon commissurotomy in cardiology department of Sri Jayadeva Institute of Cardiovascular sciences & Research, Bangalore between January 2011 and July 2014.

Inclusion criteria

- All patients undergoing PTMC during the study period
- Presence of sinus rhythm

Exclusion Criteria

- Suboptimal results of PTMC
- Presence of severe AR/severe AS or organic tricuspid valve disease
- >Grade 2 MR
- Atrial fibrillation
- Coronary artery disease
- COPD
- LV dysfunction

Study Protocol

After informed consent all patients underwent a detailed clinical evaluation as per proforma.

I. History, Physical examination & Cardiovascular examination.

II. Electrocardiogram.

III. Chest roentgenogram.

IV. Baseline blood investigations & blood grouping, typing

V. Echocardiographic measurements

Two-dimensional (2D) echocardiography and doppler tissue imaging studies were performed before PTMC and 24-48 hrs after PTMC.

Mitral valve area (MVA) was determined by planimetry in every patient. The peak and mean transmitral pressure gradients were measured using the Bernoulli principle from continuous wave Doppler recordings through the centre of mitral inflow.

The Wilkins score was used to assess mitral leaflet mobility, valvular and subvalvular thickening, and calcification. Twenty four to 48 h after mitral balloon dilatation, MVA was again determined by planimetry. Systolic pulmonary artery pressure (SPAP) was derived from the tricuspid regurgitant jet peak velocity using the modified Bernoulli equation (peak gradient = $4V^2$+mean Right atrial pressure, where V is the maximal velocity of the tricuspid regurgitant jet).

All echocardiographic parameters of RV function were measured according to guidelines for echocardiographic assessment of the right heart in adults.[11] From the parasternal short-axis view at the level of the aortic root, the RV outflow tract diameters at end-diastole and end-systole were measured. RV outflow tract fractional shortening (RVOTfs) was calculated using the formula:

$$RVOTfs = [RVOTd-RVOTs]/RVOTd$$

Where RVOTd and RVOTs represent end-diastolic and end-systolic dimensions of RVOT.

The tricuspid annular plane systolic excursion (TAPSE) was determined by the difference in the displacement of the RV base during systole and diastole.

RV end-diastolic and end-systolic areas were measured from the apical four chamber view to calculate RV fractional area change (RVFAC). With the same views, the RV ejection fraction was calculated using Simpson's rule.

Pulsed wave DTI

The Tei index of RV myocardial performance was calculated as the time between tricuspid valve closure to tricuspid valve opening, divided by the RV ejection time, determined by pulsed Doppler. A 3.5 mm sample volume was placed at the septal and lateral side of the tricuspid annulus. Peak myocardial velocities during systole, early and late diastole were measured at a sweep speed of 100 mm/s.

Myocardial acceleration during isovolumic contraction (IVA) was measured by dividing [myocardial velocity during isovolumic contraction] by [the time interval from onset of the myocardial velocity during isovolumic contraction to the time at peak velocity of this wave].[12] The final values of all parameters were obtained after averaging over three cardiac cycles. All measurements were recorded at pre-PTMC and post-PTMC.

Statistical Analysis

Parameters as defined in table 3 and 4 were recorded before PTMC and 24-48 hours after PTMC. The data was collected and statistical analysis was performed with SPSS version 20.0.

Data was presented as mean ± SD for continuous variables and as percentages for categorical variables.

To compare the means before and after in specified population paired t test was used.

Null hypothesis was stated as, difference between the means is zero, whereas alternate hypothesis was stated as, difference between the means is not equal to zero.

Standard deviation of the differences was computed from matched pairs as

$$sd = sqrt [(\Sigma(di-d)2/(n-1)]n$$

Where di is the difference for pair i, d is the sample mean of the differences, and n is the number of paired values. A p value less than 0.05 was considered to indicate statistical significance.

RESULTS

Demographic characteristics of the patients:

In this study, Mean age of the patients was 26 ± 6yrs and ranged from 13-55 years. (Table 1). Majority of patients (61.2%) were in the age group of 21-30 years (Table 1). Out of 219 patients 132 (60.3%) were female and 87 (39.7%) were male (Table 2).

Table 1: Age distribution of patients studied

Age in years	No. of patients	%
11-20	33	15.1
21-30	134	61.2
31-40	52	23.7
Total	219	100.0

Mean ± SD: 26.58 ± 6.55.

Table 2: Gender distribution of patients studied

Gender	No. of patients	%
Female	132	60.3
Male	87	39.7
Total	219	100.0

Table 3: Evaluation of Tricuspid Regurgitation grade pre PTMC and post PTMC

TR Grade	Pre	Post	% change
Nil	28 (12.8%)	30 (13.7%)	0.9
Trivial	22 (10%)	82 (37.4%)	27.4
1	116 (53%)	103 (47%)	-5.9
2	42 (19.2%)	4 (1.8%)	-17.4
3	11 (5%)	0 (0%)	-5.0
Total	219 (100%)	219 (100%)	-

Table 4 : Evaluation of study parameters pre PTMC and post PTMC

	Pre	Post	difference	t value	P value
LVIDD	4.80 ± 5.10	4.07 ± 0.45	0.733	2.115	0.036*
LVIDS	2.73 ± 0.36	2.7 0 ± 0.37	0.032	2.227	0.027*
RVDD	2.14 ± 0.37	1.62 ± 0.35	0.521	20.921	<0.001**
MVA	0.71 ± 0.15	1.84 ± 0.17	1.129	69.309	<0.001**
MVG	17.42 ± 3.69	5.54 ± 1.09	11.881	49.750	<0.001**
PASP	48.93 ± 13.08	29.56 ± 7.71	19.370	31.261	<0.001**
HR	77.84 ± 12.25	70.71 ± 8.82	7.128	11.289	<0.001**
LA	4.47 ± 0.41	3.70 ± 0.45	0.768	37.250	<0.001**
EF	59.31 ± 2.23	58.68 ± 1.63	0.630	5.004	<0.001**
TAM	16.99 ± 3.03	17.55 ± 2.62	0.551	4.273	<0.001**
RVMPI	0.4 7± 0.12	0.32 ± 0.11	0.152	24.428	<0.001**
FAC	33.9 4±7.55	37.33 ± 7.67	3.385	9.745	<0.001**
Sv	9.71 ± 1.88	10.26 ± 2.06	0.553	7.577	<0.001**

Presenting symptom was breathlessness in all patients in this study. History of paroxysmal nocturnal dyspnoea was present in 18 (8.2%) patients. History of haemoptysis was present in 6 (2.7%) patients. 18 patients (8.2%) underwent prior PTMC and two patients (0.91%) underwent prior closed mitral valvotomy. Majority of patients were in NYHA functional class III (60%) and 40% were in NYHA class II.

LA size ranged from 33-62 mm. Mean LA size was 44.9 ± 5.9 mm. Majority of patients (97%) had adequate LV function (EF >50%). Mean Ejection Fraction was 60.0 ± 5.8 %. Three patients had mild LV systolic dysfunction. Wilkins score of patients ranged from 5-9. Mean score was 6.8 ± 1.0.

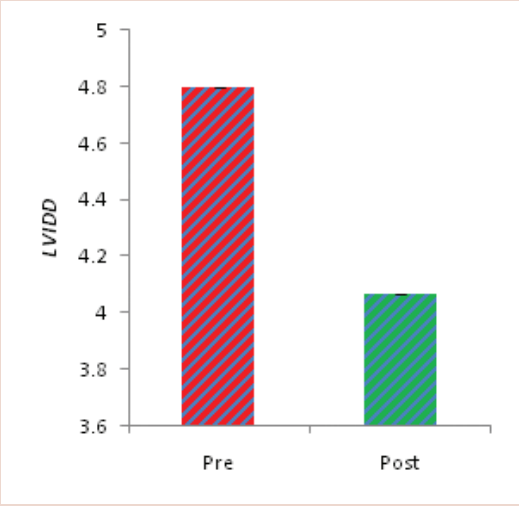

Figure 1: LVIDD (left ventricular internal dimension in diastole) Parameters Pre PTMC and Post PTMC (p=0.036)

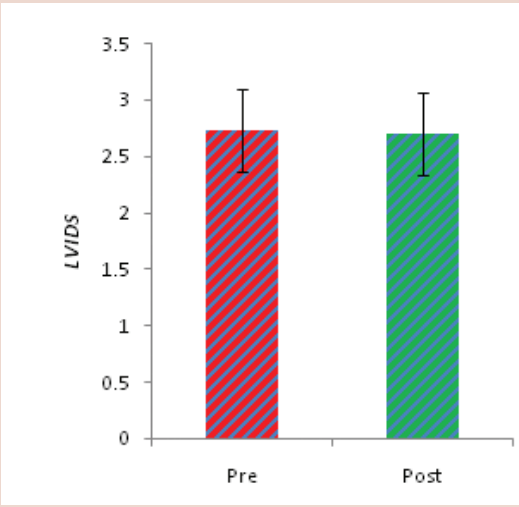

Figure 2: LVIDS (left ventricular internal dimension in systole) Parameters Pre PTMC and Post PTMC (p=0.027)

DISCUSSION

In patients with mitral stenosis, RV function is closely related to symptoms, functional capacity, need and timing for interventions, perioperative mortality, and postoperative results.[7] Evaluation of RV function by conventional transthoracic echocardiography is difficult due to its asymmetrical shape, narrow acoustic window, and geometrical assumptions for calculation of volumes. Quantitative echocardiographic assessment of the RV is difficult, a wide variety of techniques have been proposed but none of the echocardiographic indices is considered gold standard at present. We studied various two-dimensional echocardiographic and doppler tissue imaging (DTI) parameters to assess the RV function in patients with mitral stenosis immediately after successful PTMC.

DTI of lateral tricuspid annulus:

DTI echocardiography permits assessment of longitudinal RV function by measuring systolic myocardial velocities and measuring the velocities during the isovolumetric contraction period, and appears to provide additional information beyond two dimensional measurements.[13] In RV function assessment DTI is not significantly affected by volume loading and demonstrated a good reproducibility.[14]

Systolic myocardial velocity (S_v)

In our study tricuspid annular (lateral) systolic velocities showed significant change immediately after PTMC, contrary to previous studies. Bensaid *et al.*[15]observed a non-significant increase in tricuspid annular systolic myocardial velocity. Drighil *et al.* found that systolic myocardial velocity did not change immediately after PTMC.[16] In study done by Wang *et al.*[17] tricuspid systolic myocardial velocity was the best predictor of RV ejection fraction among several echocardiographic parameters.

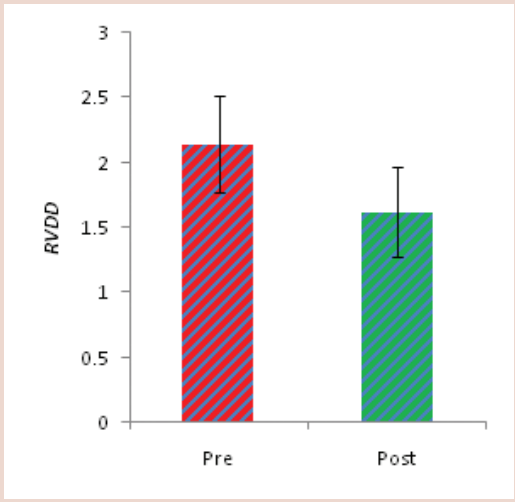

Figure 3: RVDD (right ventricular dimension in diastole) Parameters Pre PTMC and Post PTMC (p<0.001)

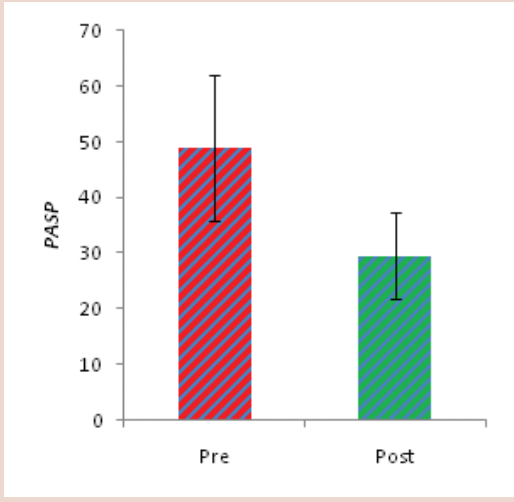

Figure 6: PASP (pulmonary artery systolic pressure) Parameters Pre PTMC and Post PTMC (p<0.001)

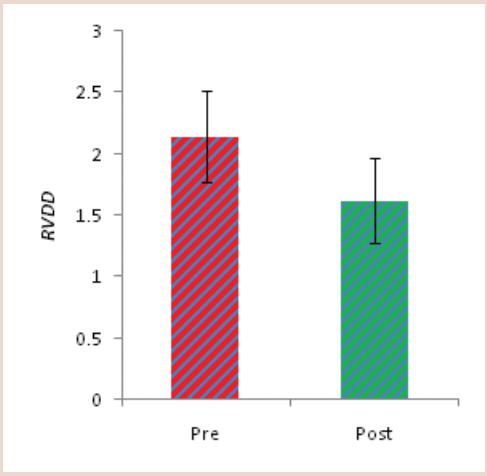

Figure 4: MVA (mitral valve area) Parameters Pre PTMC and Post PTMC (p<0.001)

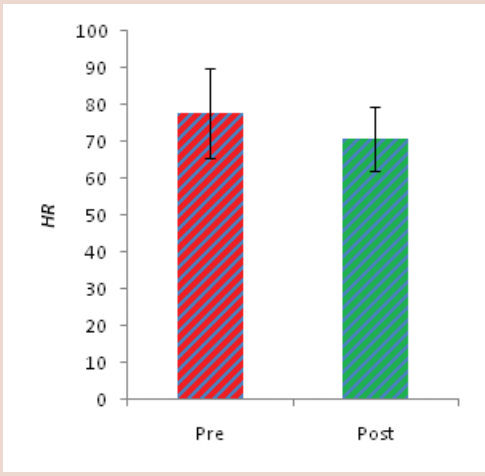

Figure 7: HR (heart rate) Parameters Pre PTMC and Post PTMC (p<0.001)

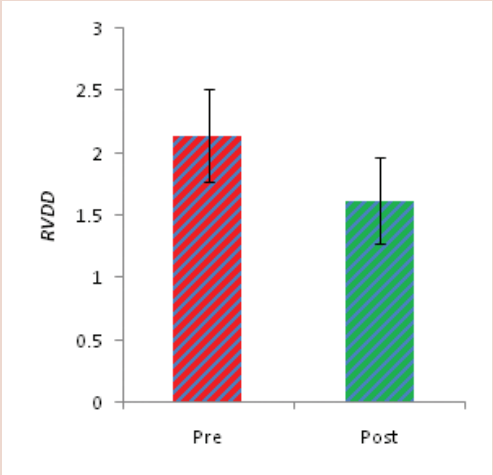

Figure 5: MVG (mitral valve gradient) Parameters Pre PTMC and Post PTMC (p<0.001)

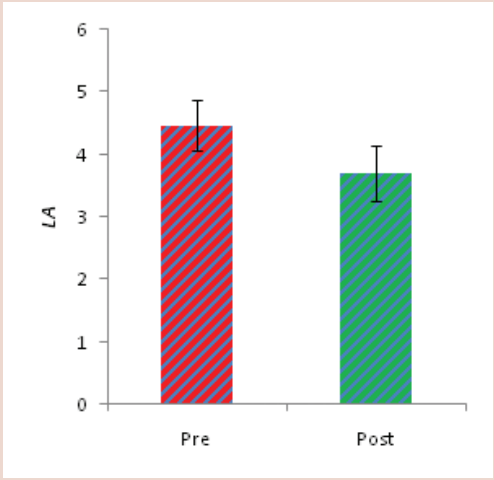

Figure 8: LA (left atrium) Parameters Pre PTMC and Post PTMC(p<0.001)

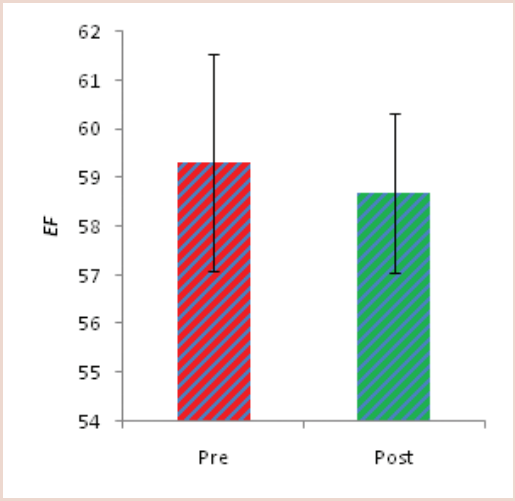

Figure 9: RVEF (right ventricular ejection fraction) Parameters Pre PTMC and Post PTMC (p<0.001)

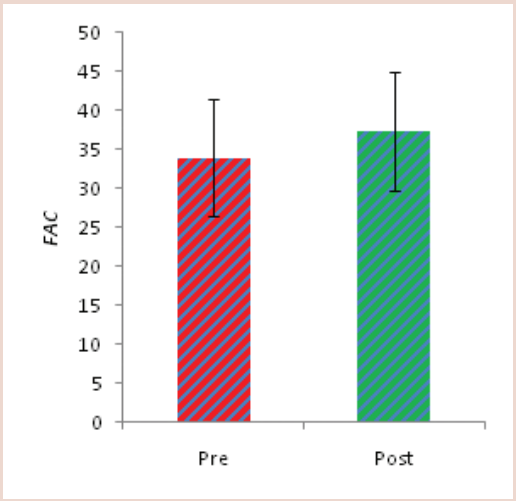

Figure 12: FAC (fractional area change) Parameters Pre PTMC and Post PTMC (p<0.001)

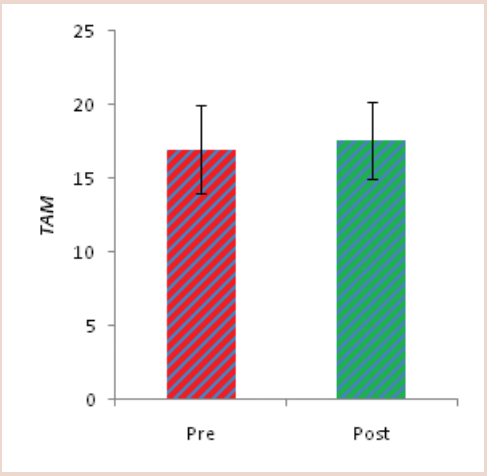

Figure 10: TAM (tricuspid annular motion) Parameters Pre PTMC and Post PTMC (p<0.001)

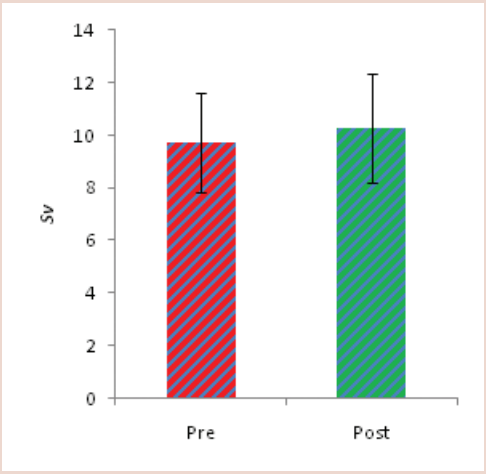

Figure 13: Sv (systolic myocardial velocities) Parameters Pre PTMC and Post PTMC (p<0.001)

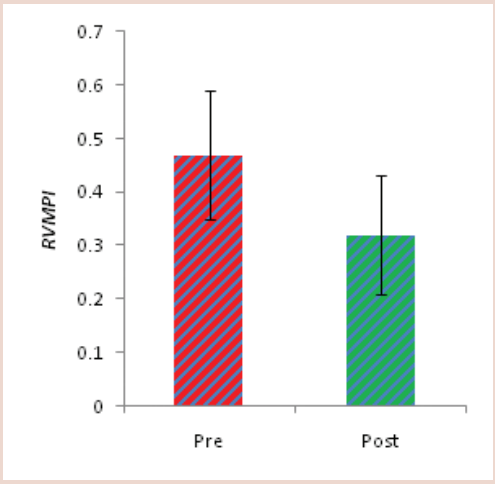

Figure 11: RVMPI (RV myocardial performance indices) Parameters Pre PTMC and Post PTMC (p<0.001)

Saxena *et al.*[18] noticed a strong correlation between tricuspid systolic myocardial velocity and RV fractional area change, regardless of pulmonary artery pressures.

On the other hand Ragab A *et al.*[19] noticed significant increase in tricuspid systolic myocardial velocity after PTMC and also concluded that tricuspid systolic myocardial velocity and TAPSE may precociously recognize patients with poor prognosis specially after PTMC. The absence of change in systolic myocardial velocity immediately after PTMC may be due to less load dependence of this parameter.

RV Tei INDEX and RVOT Fractional shortening (fs)

Immediately after PTMC RV Tei index decreased significantly from 0.47 ± 0.12 to 0.32 ± 0.11, comparable to previous studies (p<0.001). Drighil *et al.*[16] noticed a significant improvement in RV Tei index from 0.5 ± 0.2 to 0.3 ± 0.2 (p<0.001) and Bensaid *et al.*[15] observed a non-significant improvement in Tei index after PTMC from 0.33 ± 0.1 to 0.36 ± 0.12 (p=0.2). There was good correlation between Tei index and PASP before PTMC. These findings may mean an improvement in RV contractility but may also reflect the acute fall in afterload.

The improvement in RV Tei index along with improvement in RVOTfs immediately after PTMC (from 54.1 ± 8.7% to 70.4 ± 5.0%, p<0.001) suggest an improvement in RV outflow tract systolic function as a result of acute decrease in RV afterload and not necessarily to improvement in RV contractility. In the study by Drighil *et al.*[20] RVOTfs increased significantly from 57 ± 15% to 72 ± 12% , p<0.001.

TAPSE

In our study TAPSE increased significantly from 16.0 ± 1.5 mm to 18.6 ± 1.7 mm, p<0.001. This is comparable to previous study by Ragab *et al.*[19] TAPSE increased significantly after PTMC from 17.1 ± 2.1 to 19.1 ± 2.5, p<0.05. On the other hand Bensaid *et al.* and Drighil *et al.* noticed a non significant change in TAPSE after PTMC.[15,20] TAPSE improved further to 19.9 ± 2.6 mm (p<0.001), compared to both pre PTMC and immediate post PTMC values, suggesting there was gradual improvement in RV longitudinal function following PTMC.

There was no significant change in left ventricle EF after PTMC from 60.0 ± 5.8 to 60.2 ± 5.7 (p=0.2). Similarly no change in LVIDD and LVIDS was seen after PTMC implying that overall LV function was unchanged.

LIMITATIONS

Patients with atrial fibrillation were excluded in the study, hence results cannot be generalised to all patients with mitral stenosis.

As controls were not included, magnitude of right ventricular dysfunction in patients with mitral stenosis admitted for PTMC could not be compared to age and sex matched controls.

In order to know the prognostic value of echocardiographic parameters of RV function after PTMC long term follow up with clinical variables is needed.

CONCLUSION

Long term evaluation of RV function and benefits using invasive and radionuclide methods post PTMC has shown incongruous results in improvement of right ventricular (RV) function immediately after PTMC. In this study, immediately after successful PTMC significant improvement in parameters of infundibular and global RV function as assessed by RVOT fractional shortening , Tei index and tissue Doppler velocities was observed.

ABBREVIATION USED

PTMC: Percutaneous transvenous mitral valve commissurotomy, **RVF:** Right Ventricular Function, **2D Echo:** 2 Dimentional Echocardiography, **DTI:** Doppler Tissue Imaging, **MS:** Mitral stenosis, **RVFAC:** Right Ventricular fractional area change.

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