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RV FUNCTION ASSESSMENT PRE AND POST PTMC IN PATIENTS WITH SEVERE MITRAL STENOSIS

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ABSTRACT

The purpose of this research paper is to assess the immediate and short term effect of PTMC on RV function using two dimensional and doppler echocardiographic indices and also to assess the RV function in patients who undergo PTMC for isolated severe mitral stenosis before, after and at 6 months cohort follow up after PTMC. Cohort prospective study included adult patients who are > 18 years of age with isolated severe MS, in sinus rhythm, and cases were admitted for the BMV. Exclusion criteria ; patients with atrial fibrillation, in accordance with NYHA class I or IV symptoms, overt right heart failure, pregnancy, chronic obstructive pulmonary disease (COPD), other valvular lesions or undergoing emergency BMV were excluded from the study. Present study consisted of 50 patients with isolated severe MS in sinus rhythm, who met the inclusion criteria. The baseline characteristics of the study population is summarized in Table 1 Mean age of the population studied was 38±9 yrs. 62% were females and 38% were males. RV Free wall thickness decreased from 0.51±0.11cm to 0.44±0.07 which was statistically no t significant. RV dimensions decreased from baseline in basal, mid and apex to base dimensions which was statistically not significant. RV systolic area decreased from 29.30±5.28cm^2 to 25.18±4.40 cm^2 at 6 months which was statistically not significant. RV diastolic area increased from 46.30±9.20 cm^2 to 51.82±9.87cm^2 at 6 months which was statistically significant ($p<0.05$). This study showed RV function parameters in patients with severe mitral stenosis did not improve immediately after PTMC. However these parameters showed improvement compared to baseline at 6 months of follow up.

KEYWORDS: *RV Function, PTMC, Stenosis, Follow Up*

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INTRODUCTION

A right ventricular function plays an important role in the development of clinical symptoms and the overall prognosis of patients with mitral stenosis [1–3]. The RV function is predominantly affected through hemodynamic changes due to pulmonary vascular alterations. The right ventricular function is an important determinant of exercise capacity, survival and postoperative outcome in patients with mitral stenosis [3]. RV functional assessment is difficult and not done routinely because of its complex anatomy and high load dependence. Right ventricular functions cannot reliably be evaluated by conventional echocardiography techniques because of asymmetrical shape (crescentic), narrow acoustic window, irregular endocardial surface, complex contraction mechanism[5]. The problem is compounded by irregular trabeculations, a

separate infundibulum, and variations in right ventricular shape with altered loading conditions[5]. The substernal right ventricle is less accessible than the left ventricle and its dimensions more difficult to standardize. RV function is closely related to symptoms, functional capacity, need and timing for interventions, perioperative mortality and postoperative results in patients with MS[6]. In mild degree of MS, secondary pulmonary hypertension occurs due to reactive changes in pulmonary vascular resistance. Although, it is reversible in mild MS, long standing severe MS is associated with fixed pulmonary arteriolar constriction and obliterative changes in vascular bed, giving rise to significant RV afterload and RV dysfunction[9]. Thus, RV dysfunction is an important indicator to evaluate the severity of MS. Radio nuclide ventriculo graphy, cardiac catheterization; cardiac magnetic resonance imaging (MRI) and 3-dimensional echocardiography could be used for the assessment of RV function. However, these methods are time consuming, costly and not widely available[5]. The purpose of this research paper is to assess the immediate and short term effect of PTMC on RV function using two dimensional and doppler echocardiographic indices and also to assess the RV function in patients who undergo PTMC for isolated severe mitral stenosis before, after and at 6 months cohort follow up after PTMC.

METHODS

Cohort prospective study included adult patients who are > 18 years of age with isolated severe MS, in sinus rhythm, and cases were admitted for the BMV. Exclusion criteria ; patients with atrial fibrillation, in accordance with NYHA class I or IV symptoms, overt right heart failure, pregnancy, chronic obstructive pulmonary disease (COPD), other valvular lesions or undergoing emergency BMV were excluded from the study. Patients who developed more than mild mitral regurgitation after BMV were also excluded from the study group. A total 50 cases with isolated severe mitral stenosis in sinus rhythm with valve morphology suitable for PTMC were selected based on the regularity criteria of NYHA. A brief history, clinical examination and relevant investigations were done at hospital.Echocardiographic measurements were done,the 2D echo was performed before PTMC, after PTMC (within 48 hours) and at 6 months after PTMC using 3.5Hz probe in Philips HD7 XR machine followed by 2 D Echo parameters were assessed simultaneously:All the measurements were performed according to standard guidelines for assessment of right heart set by American society of echocardiography (ASE) [9]. For Mitral valve orifice area, parasternal short axis view at the level of commissures was taken. MVOA was determined using planimetry techniques. Mitral valve gradient was estimated in apical four chamber view using continuous wave doppler across mitral valve. Mitral regurgitation was assessed in multiple views- apical four- chamber, apical-two chamber and parasternal long axis views. Tricuspid regurgitation was determined in apical four- chamber view using continuous wave doppler across tricuspid valve. The pulmonary artery systolic pressure was measured by adding RA pressure to TR jet and also LV- EF was measured using modified Simpson's disc summation method. RV free wall thickness was estimated in sub costal four- chamber view using M- mode across tricuspid chordal level. RV dimensions were demonstrated at three levels in end diastole. Further, the RV basal diameter is measured at the level of tricuspid annulus. RV mid diameter was measured at the level of mid cavity. RV apex to base diameter was taken from RV apex to tricuspid annulus. Fractional area change (FAC) is measured by tracing the RV endocardial borders at end diastole and end systole, taking care to include RV apex and lateral wall. Trabeculations, leaflets and chords are included in the chamber. FAC is calculated based on the following formula FAC = $\frac{\text{End diastolic area-End systolic area}}{\text{End diastolic area}} \times 100$

Tricuspid annular motion (TAM) or Tricuspid annular plane systolic excursion (TAPSE) measures the longitudinal shortening of RV based on the distance the tricuspid annulus moves towards the RV apex from end diastole to

end systole. It is measured from M -mode intersecting the lateral tricuspid annulus from an RV optimized apical 4four-chamber view. Myocardial performance index (MPI or Tei index) is the ratio of isovolumetric contraction (IVC) plus isovolumetric relaxation (IVR) is divided by ejection time (ET). The MPI has been calculated based on tricuspid valve closure time (TCO), which includes RV ejection time plus isovolumetric contraction and relaxation time. It is measured as total duration of tricuspid regurgitation or as the interval between the end of tissue Doppler A' velocity and onset of next E' velocity. Ejection time is measured as the tissue Doppler systolic velocity duration.MPI = (TCO-ET)/ET. Colour tissue Doppler Imaging was used to measure systolic velocities in annular, basal, mid and apical regions. Cardiac catheterization was done during PTMC and LA pressure and PA systolic pressure was measures before and after PTMC. Coronary angiography was done in male patients over 40 years and females over 50 years or in presence of risk factors. Collected data was analysed by R-open source statistical software,multivariate analysis was done for testing the hypothesis

RESULTS

Study group consisted of 50 patients with isolated severe MS in sinus rhythm, who met the inclusion criteria. The baseline characteristics of the study population is summarized in Table 1 Mean age of the population studied was 38±9 yrs. 62% were females and 38% were males. All patients had dyspnea on exertion. 56% were in NYHA Class II, 44% were in NYHA Class III. Mean duration of symptoms was 11.9±3.6 months. Mean pulse rate was 77.9±8.7 bpm. Evidence of LA enlargement on ECG was present in 74% of patients.

Table 1: Baseline Characteristics

Variables	No (%)Mean±SD	P-Value
Age	38.04±9.79	P≦0.001
Sex	M-19(38.00%) F-31(62.00%)	P≦0.001
NHYA Class	II-28(56%) III-22(44%)	P≦0.001 P≦0.001
Duration of symptoms (in months)	11.93±3.63	P≦0.001
Pulse rate(bpm)	77.96±8.72	P≦0.001
LAE on ECG	37(74.0%)	P≦0.001

Table 2: Over View of Echocardiographic Parameters that were Assessed

Parameters	Pre PTMC	Post PTMC	At 6 Months	Pre PTMC V/S Post PTMC P-Value	Pre PTMC V/S 6 Months P-Value	Post PTMC V/S6 Months P-Value
MVOA(cm2)	0.81±0.16	1.76±0.12	1.65±0.12	≦0.001	≦0.001	≦0.001
MV Gradient Peak(mmHg)	22.60±4.20	10.96±3.32	12.46±3.79	≦0.001	≦0.001	≦0.001
MV Gradient Mean(mmHg)	14.30±2.94	4.76±1.06	5.73±1.1	≦0.001	≦0.001	≦0.001
TR Jet Gradient (mmHg)	60.80±23.95	39.32±14.44	37.48±14.31	≦0.001	≦0.001	≦0.001
PASP(mmHg)	68.86±25.92	43.32±15.12	38.96±13.82	≦0.001	≦0.001	≦0.001
LV EF%	59.76±0.59	59.60±0.76	59.80±0.57	≧0.001	≧0.001	≧0.001

As per the research findings, the mean MVOA was 0.81±0.16 cm^2 which increased to 1.76±0.12 cm^2 post PTMC and was 1.65±0.12cm^2 at 6 months which was statistically significant ($p<0.05$). Peak MV gradient start down from

22.60±4.20mm Hg to 10.96±3.32mm Hg after PTMC which was 12.46±3.79 mm Hg at 6 months which was statistically significant ($p<0.05$). Mean MV gradient showed down from 14.30±2.94 mm Hg to 4.76±1.06mm Hg after PTMC which was 5.73±1.1mmHg at 6 months which was statistically significant ($p<0.05$). TR Jet Gradient attained 60.80±23.95mm Hg to 39.32±14.44mm Hg after PTMC which was 37.48±14.31mmHg at 6 months which was statistically significant ($p<0.05$). The mean PA Systolic pressure was 68.86±25.92mm Hg to 43.32±15.12mm Hg after PTMC which was 38.96±13.82 mm Hg at 6 months which was statistically significant ($p<0.01$). LV EF was 59.76±0.59% before PTMC, 59.60±0.76% after PTMC and increased to 59.80±0.57% at 6 months follow up period ($p>0.01$) (table 1.2)

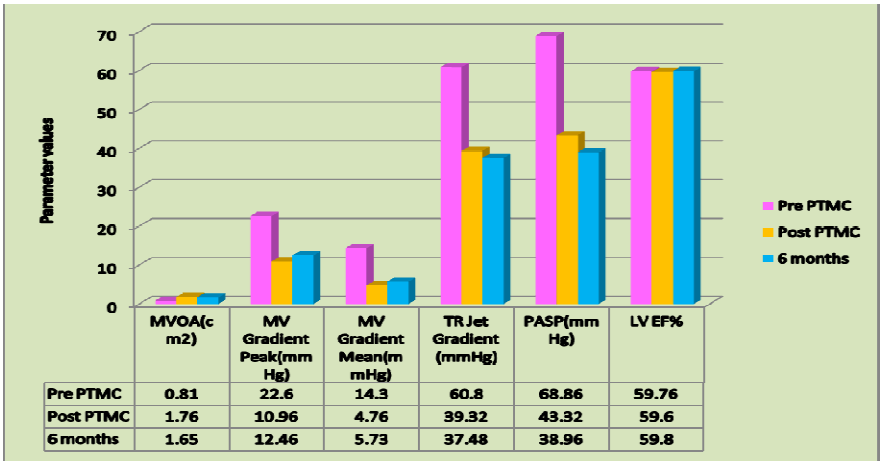
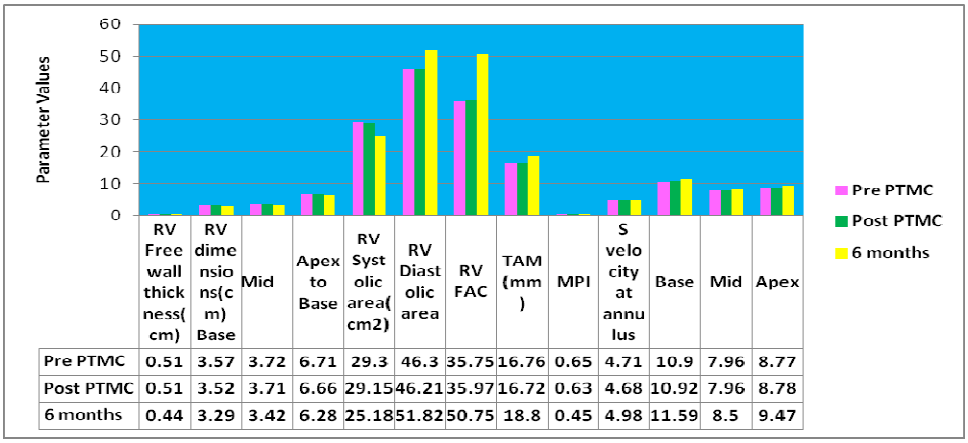


Figure 1: Mean Values of Pre and Post PTMC w Fith Different Time Period.

Table 3: RV Function Parameters Assessed By 2D Echocardiography

Parameters	Pre PTMC	Post PTMC	At 6 Months	Pre PTMC V/S Post PTMC P-Value	Pre PTMC V/S 6 Months P-Value	Post PTMC V/S6 Months P-Value
RV Free wallthickness(cm)	0.51±0.11	0.51±0.11	0.44±0.07	0.34	0.38	0.44
RV dimensions(cm) Base	3.57±0.98	3.52±0.92	3.29±0.88	0.52	0.55	0.62
Mid	3.72±0.93	3.71±0.93	3.42±0.84	0.28	0.26	0.33
Apex to Base	6.71±0.97	6.66±0.94	6.28±0.95	0.38	0.36	0.34
RV Systolic area(cm2)	29.30±5.28	29.15±5.28	25.18±4.40	0.44	0.48	0.51
RV Diastolic area	46.30±9.20	46.21±9.20	51.82±9.87	0.11	0.18	0.00*
RV FAC%	35.75±9.96	35.97±9.85	50.75±7.02	0.36	0.32	0.00*
TAM(mm)	16.76±3.15	16.72±3.04	18.80±2.91	0.28	0.26	0.05*
MPI	0.65±0.08	0.63±0.07	0.45±0.07	0.18	0.20	0.02*
S velocity at annulus	4.71±0.71	4.68±0.62	4.98±0.65	0.33	0.34	0.38
Base	10.90±1.50	10.92±1.51	11.59±1.50	0.21	0.23	0.28
Mid	7.96±1.46	7.96±0.46	8.50±0.52	0.44	0.43	0.16
Apex	8.77±0.59	8.78±0.52	9.47±0.62	0.27	0.29	0.12
*significant ‘p’ value						

RV Free wall thickness decreased from 0.51±0.11cm to 0.44±0.07 which was statistically not significant. RV dimensions decreased from baseline in basal, mid and apex to base dimensions which was statistically not significant. RV systolic area decreased from 29.30±5.28cm^2 to 25.18±4.40 cm^2 at 6 months which was statistically not significant. RV diastolic area increased from 46.30±9.20 cm^2 to 51.82±9.87cm^2 at 6 months which was statistically significant ($p<0.05$). RV FAC increased from 35.75±9.96% at baseline to 50.75±7.02% at 6 months which was statistically significant ($p<0.05$). TAM increased from 16.76±3.15mm at baseline to 18.80±2.91mm at 6 months which was statistically significant ($p=0.05$). MPI decreased from 0.65±0.08 at baseline to 0.45±0.07 at 6 months which was statistically significant ($p=0.02$). Systolic velocity measured by tissue Doppler at annulus, base, mid and apex increased from baseline to 6 months which was not statistically significant.



	RV Free wall thickness(cm)	RV dimensions(cm) Base	Mid	Apex to Base	RV Systolic area(cm2)	RV Diastolic area	RV FAC	TAM (mm)	MPI	S velocity at annulus	Base	Mid	Apex
Pre PTMC	0.51	3.57	3.72	6.71	29.3	46.3	35.75	16.76	0.65	4.71	10.9	7.96	8.77
Post PTMC	0.51	3.52	3.71	6.66	29.15	46.21	35.97	16.72	0.63	4.68	10.92	7.96	8.78
6 months	0.44	3.29	3.42	6.28	25.18	51.82	50.75	18.8	0.45	4.98	11.59	8.5	9.47

Figure 2: PTMC Associated Parameters Mean Values S.

Table 3: Cardiac Catheterization Data

Cardiac Catheterization Data	Pre PTMC	Post PTMC	P-Value
Mean LA pressure (mmHg)	24.60±6.49	12.20±2.84	≦0.0001*
PA systolic pressure (mmHg)	73.14±30.85	45.30±16.49	≦0.0001*

Mean LA pressure reduced from 24mmHg to 12mmHg and mean PASP reduced from 73mmHg to 45mmHg after PTMC, which was statistically significant ($p<0.0001$)

DISCUSSIONS

This was a prospective study which studied RV function in patients with severe mitral stenosis before, after and 6 months after PTMC. Our study showed that the RV systolic function is impaired in patients with severe MS which correlates with earlier hemodynamic and clinical studies.[3,9,10.] The cause of RV dysfunction is attributed to the increased RV afterload in these patients. Left atrial hypertension in these patients leads to chronic pulmonary venous congestion, which ultimately leads to PH. This is thought to be responsible for increased RV after load and subsequent RV dysfunction in these patients. 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 [11,12].The right ventricular function plays an important role as a determinant of symptoms, exercise capacity and survival in patients with valvular disease of the left heart[1]. An increase in pulmonary wedge pressure as a result of MS is associated with a rise in mean pulmonary artery pressure, increased afterload and, consequently, a drop in right ventricular ejection fraction. The mean age of the study group was 38.04±9.79 yrs which is

comparable to other studies by S.N. Murthy Jayanthi Sriram et al [6]. and Vipin Kumar et al[][5]. 62% of the study population was female which is comparable to Inci et al[7]. NHYA Class II symptoms were seen in 56% of patients and NYHA Class III was seen in 44% of patients which was comparable to Ahmad Noor et al study [8]. MVOA was 0.81±0.16cm2 pre PTMC which increased to 1.76±0.12cm2 post PTMC, MV gradient came down from 22/14 to 11/5mm Hg which was comparable to study by A. Drighil et al [16].Our study indicates that despite a rapid fall in pulmonary artery pressures concomitant with a significant increase in mitral valve area following PTMC, right ventricular global function does not show significant improvement in the immediate post-procedure period. RV FAC, TAM and MPI all showed statistically significant improvement in RV function after 6 months of PTMC. This is attributable to slow reduction in PA pressure and decrease in RV afterload. Pulmonary artery pressure decreases immediately after PTMC[8,18]. Pulmonary pressure usually normalises within six months, but may stay elevated for more than two years in some patients [8]. This can be explained by the structural changes of the pulmonary vessels, which may return only slowly after operation or may be irreversible. A normalisation of right ventricular ejection fraction, concurrent with a normalisation of pulmonary artery pressure, has been reported in most patients after PTMC [2,3,5]. However, in some cases, the right ventricular ejection fraction remains depressed. This is explained by the occurrence of myocardial dysfunction due to rheumatic pathology[19,20] or high wall stress secondary to ventricular dilatation [17]. In the latter study, the right ventricular ejection fraction normalised in only 19% of patients within three months following mitral valve replacement[17]. The extent of right ventricular dilatation and the duration of chronic pressure overload beyond which complete normalization of right ventricular function cannot be achieved is, however, not known. MPI (Tei index), which is calculated using three indices of time, is a reliable parameter evaluating both systolic and diastolic functions [21]. The Tei index is not greatly influenced by changes in blood pressure, afterload, heart rate, preload, RV pressure, dilatation, or tricuspid regurgitation in the clinical setting. The study of Mohan et al [9].showed that pulmonary artery pressure decreased immediately after balloon valvuloplasty and that right ventricular function, as assessed by the Tei index, returned to normal values within 1 year in 65% of such patients. It was shown that there was right ventricular fractional shortening and improvement in systolic functions, as assessed by the Tei index, after balloon valvuloplasty in patients with mitral stenosis.In addition, significant rise in FAC and TAPSE and decrease in PASP was noted post BMV 6 months, which indicates improved RV function. In the current study RV MPI, S' showed no significant change after BMV. This lack of immediate improvement in RV MPI, S' may be due to effects of sustained pressure overload on the RV for prolonged periods and these might have shown improvement over longer follow up [5]. This again is concordant with the observation by Mohan et al [9]who showed that RV MPI shows no immediate change after BMV. RV MPI is a relatively load independent parameter of the global RV function, which may not change immediately after BMV. There is change in afterload immediately post BMV, which results in improvement in most of the load dependent parameters.We also found that there was minor improvement in LV ejection fraction. This correlated with the findings in an earlier study by Mohan et al [13].The exact reason for this immediate improvement is unclear, but improvement in the atrial contribution to LV filling, improved myocardial contractilityn may be the possible explanations[14–15].

CONCLUSIONS

This study showed RV function parameters in patients with severe mitral stenosis did not improve immediately after PTMC. However these parameters showed improvement compared to baseline at 6 months of follow up. The study bearing with certain limitations this study had a small sample size of 50 patients. Follow up was limited to 6 months. Hence some parameters of RV function might have showed improvement in long term follow up. Inadequate image quality in some patients can be responsible for errors of measurement as RV is difficult to image.

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