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The Immediate and Short Term Impact of Successful Percutaneous Transvenous Mitral Commissurotomy on Right Ventricular Function

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Abstract

Objectives: The purpose of this study was to evaluate the immediate and short term follow up impact of Percutaneous Transvenous Mitral Commissurotomy (PTMC) on Right Ventricular (RV) function using two-dimensional and tissue Doppler echocardiographic indices.

Background: In patients with Mitral Stenosis (MS) RV function may be affected due to myocardial and hemodynamic factors. Previous studies using echocardiography have shown discordant results as regards to improvement of RV function immediately after PTMC. Only few studies have evaluated RV function at 6 months follow up.

Methods: A total of 90 patients with severe symptomatic MS, all in sinus rhythm, who got admitted for PTMC to author's institute, were prospectively enrolled. RV function was evaluated by conventional and Tissue Doppler Imaging (TDI) echocardiography before, 48 hours after PTMC, and at 6 months follow up.

Results: All patients underwent successful PTMC. The mitral valve area was significantly increased ($0.80 \pm 0.13 \text{ cm}^2$ versus $1.73 \pm 0.14 \text{ cm}^2$, $p=0.0001$) immediately after PTMC, while the mean transmitral gradient ($21.6 \pm 8.0 \text{ mm Hg}$ versus $5.9 \pm 1.6 \text{ mm Hg}$, $p < 0.0001$) and systolic pulmonary artery pressure ($52.3 \pm 23.4 \text{ mm Hg}$ versus $41.7 \pm 16.9 \text{ mm Hg}$, $p < 0.0001$) were significantly decreased. There was significant improvement in RV function immediately after PTMC (RV outflow tract fractional shortening (RVOTfs): $54.1 \pm 8.7\%$ versus $70.4 \pm 5.0\%$, $p < 0.001$, Tricuspid Annular Plane Systolic Excursion (TAPSE): $16.0 \pm 1.5 \text{ mm}$ versus $18.6 \pm 1.7 \text{ mm}$, $p < 0.0001$, TEI INDEX: 0.51 ± 0.04 to 0.34 ± 0.04 , $p < 0.001$). Myocardial velocities measured at lateral tricuspid annulus were not improved significantly immediately after PTMC but improved significantly at 6 months follow up (Myocardial velocity during isovolumic contraction (IVCv): $10.2 \pm 0.6 \text{ cm/s}$ to $12.0 \pm 1.3 \text{ cm/s}$, $p < 0.001$, systolic myocardial velocity (Sv): $11.7 \pm 0.9 \text{ cm/s}$ to $13.2 \pm 0.9 \text{ cm/s}$, $p < 0.001$, myocardial acceleration during isovolumic contraction (IVA): $3.0 \pm 0.5 \text{ cm/s}^2$ to $3.9 \pm 0.3 \text{ cm/s}^2$, $p < 0.001$).

Conclusion: Immediately after successful PTMC, significant improvement in RV function was observed. TDI myocardial velocities and IVA showed gradual improvement in RV function at 6 months follow up. Prognostic value and clinical significance of this improvement deserve further investigation.

Keywords: Mitral stenosis; Rheumatic heart disease; Doppler tissue imaging

Introduction

Rheumatic heart disease causes significant morbidity and mortality. Mitral stenosis (MS) is the commonest presentation in rheumatic heart disease. Rheumatic MS 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 become established as a safe and effective treatment for rheumatic MS and remains the treatment of choice in patients with a favorable anatomy [4-6].

The Right Ventricular (RV) function is an important determinant of clinical symptoms, exercise capacity, pre-operative survival and postoperative outcome in patients with MS [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 MS, previous studies have shown discordant

results as regards to improvement of Right Ventricular (RV) function immediately after PTMC and only few studies [11-13] have evaluated the RV function during follow up. 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.

Materials and Methods

The study population consisted of 90 patients with symptomatic MS who underwent PTMC in cardiology department of Sri Venkateswara Institute of Medical sciences, Tirupati between August 2011 and April 2012. Patients in all age groups, with evidence of severe MS {Mitral

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Valve Area (MVA) < 1.0 cm² admitted in our institution, in whom PTMC was feasible were included. Those who were fulfilling the PTMC intervention criteria and those who had experienced a successful intervention only were included. All participants gave their informed consent. Institutional Ethical Committee clearance was obtained for conducting the study.

Exclusion criteria

1. Atrial fibrillation
2. Systemic hypertension
3. Diabetes mellitus
4. More than mild mitral or aortic regurgitation and/or aortic stenosis, organic pulmonary and tricuspid valve disease
5. New York Heart Association functional class IV
6. Patients with lung disease
7. Pregnancy.

Echocardiographic measurements

All patients underwent Two-dimensional (2D) echocardiography and Doppler Tissue Imaging (DTI) studies before PTMC, 48 hours after PTMC and at 6 months follow up. All studies were obtained using a Philips IE-33 ultrasonographic machine equipped with a 3.5 MHz transducer.

Evaluation of MS severity and general parameters

Mitral Valve Area (MVA) was determined by planimetry in every patient. The peak and mean mitral valve transmitral pressure gradients were measured using the Bernoulli principle from continuous wave Doppler recordings through the center of mitral inflow [5].

The Wilkins score was used to judge mitral leaflet mobility, valvular and subvalvular thickening, and calcification. Forty eight hours after mitral balloon dilatation and at 6 months follow up; 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 $4V^2 + \text{mean right atrial pressure}$, where V is the maximal velocity of the tricuspid regurgitant jet) (Right atrial pressure is calculated by estimating the inferior vena caval size and its variation with respiration) [14].

Right ventricular function assessment

All echocardiographic parameters of RV function were measured according to guidelines for echocardiographic assessment of the right heart in adults [14]. 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 [14].

$RVOTfs = (RVOTd - RVOTs) / RVOTd$ 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 [15] (Figure 1). RV end-diastolic and end-systolic areas were measured from the apical four chamber view to calculate RV Fractional Area Change (RVFAC) [16,17]. With the same views, the

RV ejection fraction was calculated using Simpson's rule [14].

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 [18] (Figure 2). 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 [18,19] (Figure 3).

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 [20]. The final values of all parameters were obtained after averaging over

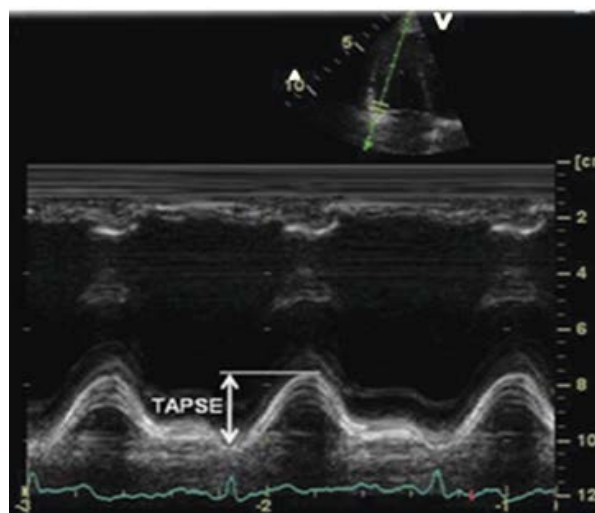


Figure 1: Measurement of tricuspid annular plane systolic excursion (TAPSE).

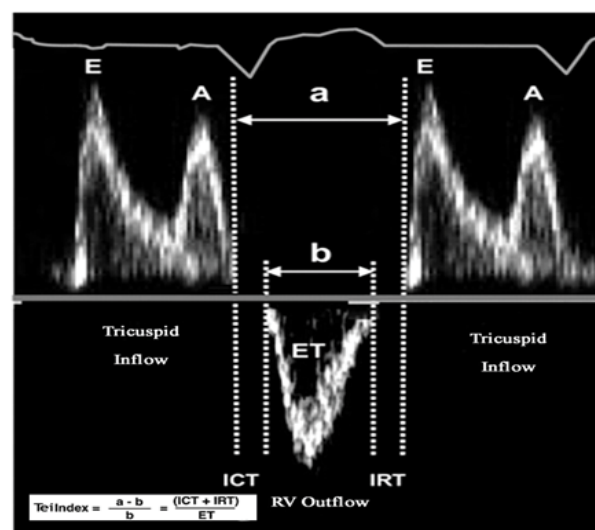


Figure 2: Measurement of right ventricular Ejection Time (ET), Isovolumic Contraction Time (ICT), Isovolumic Relaxation Time (IRT), and calculation of Tei Index.

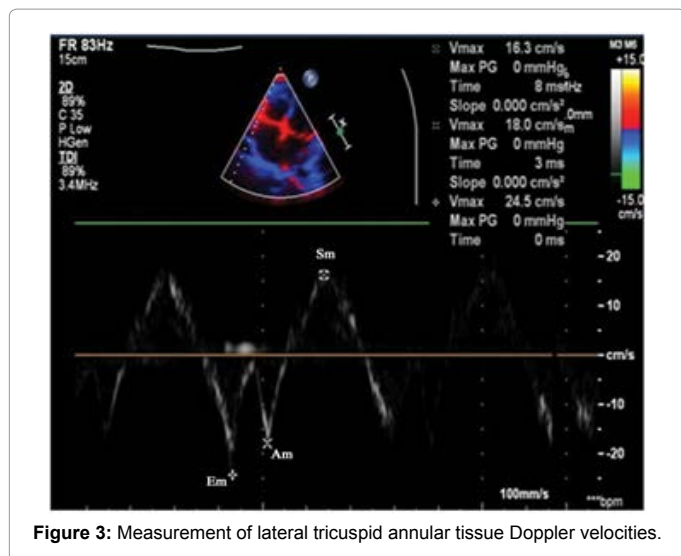


Figure 3: Measurement of lateral tricuspid annular tissue Doppler velocities.

three cardiac cycles. All measurements were recorded at pre-PTMC, post-PTMC and at 6 month follow up.

Percutaneous transvenous mitral commissurotomy

The PTMC was performed via an antegrade transvenous approach using an Inoue balloon and stepwise dilatation strategy [3]. The nominal balloon diameter was decided according to the height of the patient (i.e. height (cm)/10+10 = balloon diameter). Successful PTMC was defined as either post commissurotomy mitral valve area (MVA) >1.5 cm² or a MVA of more than twice the pre-procedural value, together with no worsening of mitral regurgitation >grade 2+.

Statistical analysis

Statistical analysis was performed with SPSS version 20.0. Data was presented as mean ± SD for continuous variables and as percentages for categorical variables. Kolmogorov-smirnov test of normality was performed to check the normal distribution. Comparisons of values before and after PTMC were performed using paired sample t-test. A p value less than 0.05 was considered to indicate statistical significance.

Results

Demographic characteristics of the patients

Ninety patients with MS in sinus rhythm were enrolled prospectively into the study. All patients had severe MS and were symptomatic, while

10 patients had a history of previous commissurotomy. The clinical and 2D echocardiographic characteristics of patients are summarized in Tables 1 and 2, respectively.

2D echocardiographic parameters before and after PTMC

Following PTMC, the 2D MVA increased from 0.80 ± 0.13 cm² to 1.73 ± 0.14 cm² (p=0.0001). Peak transmitral gradients and mean transmitral gradients before PTMC were 33.0 ± 10.2 mm Hg and 21.6 ± 8.0 mm Hg, and significantly decreased after PTMC to 11.0 ± 2.5 mm Hg and 5.9 ± 1.6 mm Hg (p < 0.0001 and < 0.0001) respectively.

A significant fall in systolic pulmonary artery pressure (SPAP) was seen immediately after PTMC from 52.3 ± 23.4 mm Hg to 41.7 ± 16.9 mm Hg (p < 0.0001). A further fall in SPAP was observed at 6 months follow-up (38.1 ± 16.7). LA diameter was 44.9 ± 5.9 mm prior to PTMC, significantly decreased to 42.4 ± 6.0 mm (p=0.006) after PTMC. LV Ejection Fraction (LVEF), LV End-Diastolic Diameter (LVEDD) and LV End-Systolic Diameter (LVESD) measurements were unchanged after PTMC.

2D echocardiographic assessment of RV function

Mean RV Ejection Fraction (RVEF) of patients before PTMC was 50.0 ± 6.9%, did not change significantly immediately after PTMC. But there was a significant improvement in RVEF seen at 6 months follow-

Characteristics	
Age 33.6 (±9.8) yrs	Age 33.6 (± 9.8) yrs.
Sex	
Male	62 (69%)
Female	28 (31%)
Functional class (NYHA)	
Class I	0
Class II	36 (40%)
Class III	54 (60%)
Class IV	0
Previous PTMC	10 (11%)
Previous CMV	2 (2.2%)
CVA History	0
Atrial fibrillation	0
Height	155.7 ± 9.3 cm
LA size	44.9 ± 5.9mm
Left ventricular dysfunction	3 (3.3%)
Left ventricular ejection fraction	60.01 ± 5.8%
Wilkins score	6.8 ± 1.0

CVA: Cerebro Vascular Accident; CMV: Closed Mitral Valvotomy; NYHA: New York Heart Association; LA: Left Atrium; PTMC: Percutaneous Transvenous Mitral Commissurotomy.

Table 1: Baseline characteristics of the population.

Variable	Pre PTMC (A)	Post PTMC (B)	p value (A Vs B)	follow-up (C)	p value (A Vs C)
LA, mm	44.9 ± 5.9	42.4 ± 6.0	0.006	40.3 ± 5.9	< 0.001
LVEF, %	60.0 ± 5.8	60.2 ± 5.7	NS	59.6 ± 8.2	NS
LV diastolic diameter, mm	41.1 ± 5.1	41.4 ± 5.1	NS	42.2 ± 5.8	NS
LV systolic diameter, mm	27.2 ± 4.2	27.3 ± 4.5	NS	27.8 ± 5.1	NS
Max Gradient, mm Hg	33.0 ± 10.2	11.0 ± 2.5	< 0.001	9.8 ± 2.7	< 0.001
Mean Gradient, mm Hg	21.6 ± 8.0	5.9 ± 1.6	< 0.001	5.6 ± 1.5	< 0.001
MVA PHT, cm2	0.84 ± 0.13	1.87 ± 0.14	< 0.001	1.84 ± 0.21	< 0.001
MVA planimetry, cm2	0.80 ± 0.13	1.73 ± 0.14	< 0.001	1.73 ± 0.14	< 0.001
PASP, mm Hg	52.3 ± 23.4	41.7 ± 16.9	< 0.001	38.1 ± 16.7	< 0.001

LVEF: Left ventricular ejection fraction; LA: left atrial diameter; LV: left ventricular; Max Gr, transmitral Maximum gradient; Mean Gr: transmitral mean gradient; MVA PHT: pressure half-time mitral valve area; MVA plan: planimetric mitral valve area; NS: statistically not significant; PASP: pulmonary artery Systolic pressure; (A Vs B), Pre PTMC Values compared with immediate post PTMC values; (A Vs C), Pre PTMC values compared with 6 months follow-up values.

Table 2: Comparison of transthoracic echocardiographic variables before and after PTMC.

up (52.9 ± 6.0%, p=0.003). RV Fractional Area Change (RVFAC) before PTMC was 34.4 ± 4.3%, did not change significantly immediately after PTMC, but significant improvement was noted at 6 months follow up visit (36.0 ± 4.7%, p < 0.001), shown in (Table 3).

RV TEI index decreased significantly from 0.51 ± 0.04, to 0.34 ± 0.04 after PTMC, no further change was noted at 6 months follow up. Both RVOT Fractional Shortening (RVOTfs) and Tricuspid annular plane systolic excursion (TAPSE) were 54.1 ± 8.7% and 16.0 ± 1.5 mm, respectively prior to PTMC. Both these parameters increased significantly immediately after PTMC to 70.4 ± 5.0% and 18.6 ± 1.7 mm. At 6 month follow up both these variables further improved to 74.1 ± 5.5% and 19.9 ± 2 mm (Table 3).

Tissue doppler imaging variables of RV function

Both myocardial acceleration during Isovolumic Contraction (IVA) and myocardial velocity during Isovolumic Contraction (IVCV) measured at lateral tricuspid annulus did not change significantly immediately after PTMC (p = 0.1 and 0.09 respectively), but increased significantly when measured at 6 months after PTMC (Table 4). Systolic myocardial velocities (Sv) measured at lateral tricuspid annulus did not change immediately after PTMC but increased significantly at 6 month follow up period (Table 4).

Early and late diastolic myocardial velocities measured at lateral tricuspid annulus did not change after PTMC, both immediately and at 6 months follow up.

Correlation between MVA by planimetry and other parameters before PTMC

Table 5 showed significant correlation between MVA by planimetry with (MVA by PHT, mean pressure and SPAP) and no relation with the other parameters. Comparison of pre- and post-PTMC (at 48 hours and 6 months) echocardiographic variables in patients with or without baseline pulmonary hypertension (Table 6).

Variable	Pre PTMC (A)	Post PTMC (B)	p value (A Vs B)	follow-up (C)	p value (A Vs C)
RV EF, %	50.0 ± 6.9	50.9 ± 6.0	NS	52.9 ± 6.0	= 0.003
RVFAC, %	34.4 ± 4.3	35.1 ± 4.7	NS	36.0 ± 4.7	< 0.001
RVOTfs, %	54.1 ± 8.7	70.4 ± 5.0	< 0.001	74.1 ± 5.5	< 0.001
TAPSE, mm	16.0 ± 1.5	18.6 ± 1.7	< 0.001	19.9 ± 2.6	< 0.001
TEI INDEX	0.51 ± 0.04	0.34 ± 0.04	< 0.001	0.32 ± 0.04	< 0.001

RVEF: RV Ejection Fraction; RVFAC: RV fractional area change; RVOTfs: RV outflow tract fractional Shortening; TAPSE: Tricuspid Annular Plane Systolic Excursion; (A Vs B), Pre PTMC Values compared with immediate post PTMC values; (A Vs C), Pre PTMC values compared with 6 months follow-up values.

Table 3: Comparison of 2D echocardiographic parameters of RV function before and after PTMC.

Variable	Pre PTMC (A)	Post PTMC (B)	p value (A Vs B)	follow-up (C)	p value (A Vs C)
Lateral EV CM/S	13.7 ± 0.8	14.1 ± 1.8	0.22	14.4 ± 1.3	0.08
Lateral AV CM/S	13.3 ± 0.6	13.8 ± 1.6	0.34	14.2 ± 0.8	0.1
Lateral SV CM/S	11.7 ± 0.9	11.9 ± 1.3	0.08	13.2 ± 0.9	< 0.001
Lateral IVC V CM/S	10.2 ± 0.6	10.5 ± 0.9	0.09	12.0 ± 1.3	< 0.001
Lateral IVA CM/S ²	3.0 ± 0.5	3.1 ± 0.5	0.1	3.9 ± 0.3	< 0.001

RV: Right Ventricle; TDI: Tissue Doppler Imaging; PTMC: Percutaneous Transvenous Mitral Commissurotomy; IVCv: Myocardial velocity during isovolumic contraction; IVA: Isovolumetric Velocity Acceleration; Ev: E wave velocity; Av: A wave velocity; Sv: Systolic myocardial velocity.

Table 4: RV TDI myocardial velocities of lateral tricuspid annulus.

MVA by planimetry		
	r- value	p-value
MVA PHT	0.436	< 0.0001
Max Gr	-0.201	0.057
Mean Gr	-0.430	< 0.0001
PASP	-0.229	0.030
RVOTfs	-0.104	0.330
RVFAC	0.047	0.660
RV TEI INDEX	-0.115	0.280
TAPSE	0.107	0.316
RV EF	0.050	0.639
Lateral E'	0.053	0.623
Lateral A'	0.060	0.576
Lateral Sv	0.005	0.962
Lateral IVCv	0.005	0.965
Lateral IVA	0.024	0.825
WILKINS Score	-0.101	0.346

MVA: Mitral Valve Area; PTMC: Percutaneous Transvenous Mitral Commissurotomy; MVA PHT: Pressure Half-Time Mitral Valve Area; Max Gr: Transmitral Maximum Gradient; Mean Gr: Transmitral Mean Gradient; PASP: Pulmonary Artery Systolic Pressure; RVOTfs: RV Outflow Tract fractional shortening, RVFAC: RV Fractional Area Change; TAPSE: Tricuspid Annular Plane Systolic Excursion; RVEF: RV Ejection Fraction, Ev: E wave velocity; Av: A wave velocity; Sv: Systolic myocardial velocity; IVCv: Myocardial velocity during isovolumic contraction; IVA: Isovolumetric Velocity Acceleration.

Table 5: Correlation between MVA by planimetry and other parameters before PTMC.

There was significant fall in SPAP in both pulmonary hypertensive and non-pulmonary hypertensive group immediately after PTMC and after 6 months. Various two-dimensional echocardiographic parameters of RV function and DTI findings were represented in Table 6.

Discussion

In patients with MS, 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 MS immediately after successful PTMC and at 6 months follow-up period.

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 [21]. In RV function assessment DTI is not significantly affected by volume loading and demonstrates a good reproducibility [22].

Systolic myocardial velocity (Sv)

In our study tricuspid annular (lateral) systolic velocities did not show significant change immediately after PTMC and is comparable to previous studies. Bensaid et al observed a non-significant increase

	Pulmonary hypertension (-) (n:24)			Pulmonary hypertension (+) (n:66)		
	Pre PTMC	After PTMC	After 6 months	Pre PTMC	After PTMC	After 6 months
PSAP	30 ± 5.04	26.34 ± 3.92*	24.34 ± 3.43*	60.44 ± 22.23	47.31 ± 16.42*	35.78 ± 11.02*
TAPSE	16.08 ± 1.74	18.21 ± 1.47*	19.79 ± 4.01*	16.07 ± 1.43	18.75 ± 1.82*	18.18 ± 1.72*
RV EF	48.08 ± 6.79	50.18 ± 6.12	53.87 ± 6.02*	47.40 ± 7.07	49.48 ± 6.05	52.56 ± 6.02*
RVOTfs	53.87 ± 7.9	69.58 ± 5.29*	73.25 ± 5.91*	54.18 ± 9.12	70.77 ± 4.92*	74.5 ± 5.45*
RVFAC	35.00 ± 4.24	37.21 ± 4.61	39.12 ± 4.54*	33.25 ± 4.45	34.09 ± 4.84	36.0 ± 4.82*
RV TEI Index	0.44 ± 0.06	0.35 ± 0.06*	0.34 ± 0.06*	0.45 ± 0.05	0.32 ± 0.04*	0.31 ± 0.05*
Tricuspid annulus						
Lateral E'	13.54 ± 0.72	14.28 ± 1.02*	18.34 ± 0.78*	13.82 ± 0.82	14.10 ± 0.92	15.66 ± 1.44*
Lateral A'	13.19 ± 0.66	13.67 ± 0.98*	17.01 ± 0.88*	13.39 ± 0.63	13.60 ± 0.73	14.10 ± 0.93*
Lateral Sv	11.80 ± 0.99	12.52 ± 0.91*	13.39 ± 1.79*	11.79 ± 0.99	12.06 ± 0.96	13.10 ± 1.26*
Lateral IVCv	10.07 ± 0.65	10.70 ± 0.98*	12.29 ± 0.79*	10.31 ± 0.87	10.58 ± 0.79	11.94 ± 1.49*
Lateral IVA	3.02 ± 0.65	3.42 ± 0.52*	3.90 ± 0.34*	3.13 ± 0.54	3.16 ± 0.52	3.74 ± 0.31*

*P < 0.05 comparison with baseline-after PMBV and 6 months

PTMC: Percutaneous transvenous mitral Commissurotomy; PASP: Pulmonary Artery Systolic Pressure; TAPSE: Tricuspid Annular Plane Systolic Excursion; RVEF: RV Ejection Fraction; RVOTfs: RV outflow Tract Fractional Shortening; RVFAC: RV Fractional Area Change; Ev: E wave velocity; Av: A wave velocity; Sv: Systolic Myocardial Velocity; IVCv: Myocardial velocity during isovolumic contraction; IVA: Isovolumetric Velocity Acceleration

Table 6: Comparison of pre- and post-PTMC (at 48 hours and 6 months) echocardiographic variables in patients with or without baseline pulmonary hypertension.

in tricuspid annular Sv [23]. Drighil et al. found that Sv did not change immediately after PTMC [24]. In study done by Wang et al. tricuspid Sv was the best predictor of RV ejection fraction among several echocardiographic parameters [25]. Saxena et al. [26] noticed a strong correlation between tricuspid Sv and RV fractional area change, regardless of pulmonary artery pressures. On the other hand Ragab A et al. [27] noticed significant increase in tricuspid Sv after PTMC and also concluded that tricuspid Sv and TAPSE may precociously recognize patients with poor prognosis especially after PTMC. The absence of change in Sv immediately after PTMC may be due to less load dependence of this parameter.

On follow up at 6 months after PTMC we noticed a significant improvement in the tricuspid Sv suggesting that improvement in RV contractility occurs during follow up period after PTMC. This may be the result of positive RV remodeling after successful PTMC.

On subgroup analysis, tricuspid annular Sv improved significantly immediately after PTMC and continued to improve at 6 months in patients without PAH. In patients with PAH, Sv did not improved significantly immediately after PTMC and improved significantly at 6 months due to the positive RV remodeling after successful PTMC.

Isovolumetric velocity acceleration (IVA)

In our study we noticed no significant change in IVA measured at lateral tricuspid annulus immediately after PTMC comparable to other studies. At 6 months follow up there was significant improvement indicating improvement of RV function unrelated to after load reduction. On subgroup analysis, IVA improved significantly immediately after PTMC and continued to improve at 6 months in patients without PAH. In patients with PAH, IVA did not improved significantly immediately after PTMC but improved significantly at 6 months.

In an animal model, Vogel et al. [28] showed that IVA as a measure of RV contractility was not affected by preload within physiological ranges. IVA measures rate change of contractile force during isovolumic contraction; hence it was suggested as a strong index of RV contractility. Tayyareci et al. showed the reliability of RV IVA in the early detection of RV dysfunction in MS patients [29].

Myocardial velocity during isovolumic contraction (IVCv)

Our study showed that IVCv of lateral tricuspid annulus not changed significantly immediately after PTMC, but significant increase was noted at 6 months follow up comparable to previous studies [24,30]. Myocardial velocities of lateral tricuspid annulus and RV IVA were not changed immediately after PTMC because they are not affected by acute change in afterload after PTMC. But there was significant increase in these parameters at 6 months follow up study, suggesting that there is a true improvement in RV contractility over time after PTMC. On subgroup analysis, IVCv improved significantly immediately after PTMC and continued to improve at 6 months in patients without PAH. In patients with PAH, IVCv did not improved significantly immediately after PTMC but improved significantly at 6 months.

Two-Dimensional echocardiographic assessment of RV function

a) RV TEI INDEX and RVOTfs

Immediately after PTMC RV TEI index decreased significantly in both PAH and non PAH groups, comparable to previous studies. Drighil et al. noticed a significant improvement in RV TEI index from 0.5 ± 0.2 to 0.3 ± 0.2 (p < 0.0001) and Bensaid et al. observed a non-significant improvement in TEI index after PTMC from 0.33 ± 0.1 to 0.36 ± 0.12 (p=0.2) [30,23]. 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 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. RVOTfs increased significantly from $57 \pm 15\%$ to $72 \pm 12\%$, $p < 0.0001$ [30]. When we studied at 6 months there was further improvement in both RV TEI index and RVOTfs may be due to further fall in RV afterload in both PAH and non PAH groups.

b) RVEF and FAC

Both RVEF and RVFAC did not change immediately after PTMC but improved significantly 6 months after PTMC in both PAH and non PAH groups. In the study by Drighil et al. there was a positive trend in the data of these parameters ($p=0.27$ and $p=0.24$) [30]. Lack of significant improvement immediately after PTMC may reflect that these are measures of RV inflow and are afterload independent. In the study by Burger et al., he noticed that improvement in RVEF after PTMC depends directly on gain in stroke volume and inversely on RVEF before PTMC [9].

c) TAPSE

In our study TAPSE increased significantly from 16.0 ± 1.5 mm to 18.6 ± 1.7 mm, $p < 0.0001$. This is comparable to previous study by Ragab et al. [27]. 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 [23,30]. At 6 months follow up TAPSE improved further to 19.9 ± 2.6 mm ($p < 0.0001$, compared to both pre PTMC and immediate post PTMC values), suggesting there was gradual improvement in RV longitudinal function during follow up period after PTMC. On subgroup analysis TAPSE was significantly higher in both PAH and non PAH groups.

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 LVEDD and LVESD was seen after PTMC implying that overall LV function was unchanged.

Correlation between MVA and other parameters before PTMC

We did not find any correlation between MVA by planimetry and DTI findings similar to the previous study [12].

Limitations

Patients with atrial fibrillation were excluded in the study; hence results cannot be generalized to all patients with MS. As controls were not included, magnitude of right ventricular dysfunction in patients with MS 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.

Conclusions

In patients with MS who underwent successful PTMC, there was immediate improvement in infundibular and global RV function as assessed by RVOTfs, TAPSE and RVTEI index. During follow-up period after PTMC there was gradual improvement in RV contractility as assessed by DTI. The myocardial velocities measured at lateral

tricuspid annulus are afterload independent measures of RV systolic function, along with isovolumic velocity acceleration are useful for assessment of RV contractility in MS particularly in the early stages.

Clinical implications of the present study

The myocardial velocities measured at the lateral tricuspid annulus are afterload independent measures of RV systolic function, along with isovolumic velocity acceleration are useful for assessment of RV contractility in MS particularly in the early stages.

As improvement in RV contractility assessed by DTI less in PAH group when compared with non PAH group, MS patients should be intervened at an earlier stage before the development of PAH.

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