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Original Research Paper

Cardiology

RV DYSFUNCTION IN PATIENTS OF MITRAL STENOSIS AND ITS IMPROVEMENT AFTER BMV

Mukesh Kumar Sharma	Prof. and Head of Department, Department of Cardiology, R. N. T. Medical college and MB hospital, Udaipur.
Mahesh Narendra Jain*	Senior Resident, Department of Cardiology, R. N. T. Medical college and MB hospital, Udaipur. *Corresponding Author
Deepak Ameta	Assist. Prof. Department of Cardiology, R. N. T. Medical college and MB hospital, Udaipur.
Lekhraj Choudhary	Senior Resident, Department of Cardiology, R. N. T. Medical college and MB hospital, Udaipur.

ABSTRACT

Background: Rheumatic heart disease (RHD) is one of the most common forms of cardiac diseases, particularly in developing countries.1–3 Mitral stenosis (MS) is the most common valve lesion in chronic RHD. RV function plays an important role in development of clinical symptoms and prognosis in patients with MS.5,8 Our study aim was to assess RV function in these subjects by various echocardiographic parameters pre and post-balloon mitral valvuloplasty (BMV).

Methods: Twenty three patients with isolated severe MS in sinus rhythm were assessed for RV function by various echocardiographic and Doppler Tissue Imaging (DTI) parameters before and after BMV (day 1 and 6 months) and also compared with that of thirty two healthy age matched controls.

Results: RVMPI (TEI Index), Estimated PASP and LA dimensions was significantly higher in cases than in controls, where as RVFAC, TAPSE, S' and IVA were significantly lower in cases than in controls. Following BMV, on day 1, the MVA increased from 0.95 ± 0.21 cm2 to 1.60 ± 0.14 cm2 ($P<0.001$). A significant fall in estimated pulmonary artery systolic pressure (PASP) was seen immediately after BMV from 57.30 ± 9.76 mm Hg to 47.22 ± 9.34 mm Hg ($p < 0.001$). A further fall in estimated PASP was observed at 6 months follow-up. Before BMV TAPSE was 17.43 ± 3.42 mm which significantly increased to 18.78 ± 2.91 mm post BMV (Day1) ($p<0.001$), and to 19.91 ± 2.02 mm after 6 months ($p<0.001$). RVMPI(TEI Index), S' (Peak systolic velocity at lateral tricuspid annulus) and IVA (Isovolumic Acceleration) were unchanged immediately after BMV. However there was significant improvement in RVMPI(TEI Index), S' and IVA during follow up period of 6 months. Immediate and long term improvement seen in afterload dependant parameters like RVFAC, PA pressure, TAPSE whereas afterload independent parameters like RVMPI, IVA, S' did not improve immediately but significantly improved after 6 months.

Conclusion: RV systolic and diastolic function is impaired in patients with severe MS and can be assessed by various two-dimensional echocardiographic and DTI parameters before the appearance of clinical signs of systemic venous congestion. Improvement in RV systolic and diastolic function starts immediately after BMV with reduction in RV afterload (PA pressure) and continues to improve over long term period.

KEYWORDS : RHD, MS, RV Dysfunction, BMV.

INTRODUCTION

Rheumatic heart disease (RHD) is one of the most common forms of cardiac diseases, particularly in developing countries, where it remains the second most common cause of cardiovascular morbidity and mortality after atherosclerotic vascular disease.[1–3] Mitral stenosis (MS) is the most common valve lesion in chronic RHD. Several mechanisms have been postulated to explain left ventricle (LV) systolic dysfunction in patients with MS, including chronically reduced preload, resulting in adverse LV remodelling, and the extension of inflammatory process from the mitral valve apparatus into the adjacent myocardium.[4]

Systolic dysfunction of the right ventricle (RV) is well documented in patients with rheumatic MS; RV dysfunction is usually overlooked before the emergence of clinical signs of systemic venous congestion because of difficulties in the quantitative assessment of RV function.[5–7]

RV function plays an important role in development of clinical symptoms and prognosis in patients with MS.[5,8] This is primarily affected by hemodynamic effects on RV due to pulmonary hypertension (PH). RV dysfunction is not detected clinically until the development of clinical signs of systemic venous congestion.

RV functional assessment is difficult and not done routinely because of its complex anatomy and high load dependence. Many indices have been developed for quantifying RV function. As the RV function has a prognostic importance in patients with MS, the present study intended to assess RV function in patients with isolated severe MS, before and after Balloon Mitral Valvotomy (BMV).

In patients with MS, previous studies have shown discordant results as regards to improvement of RV function immediately after BMV and only few studies[8-10] 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 BMV on RV function using two-dimensional and tissue Doppler echocardiographic indices.

METHODS

Study Population:

This was a prospective observational study which included all patients referred for elective BMV in the Cardiology Department, R. N. T. Medical college in 2018-19. The study included 23 patients and 32 healthy age- and sex-matched controls.

Inclusion Criteria:

The inclusion criteria were as follows: Patients undergoing BMV,

(i) patients' age from 16 to 40 years. (ii) Symptomatic moderate-to-severe MS. (iii)Asymptomatic moderate-to-severe MS with PH (PASP more than 50 mmHg at rest).

Exclusion Criteria:

The exclusion criteria were as follows:

(i) Patients with mild MS (MVA > 1.5 cm2). (ii)Patients who are not candidates for BMV (due to either Wilkins score ≥10, commissural calcification or LA thrombus). (iii) Moderate-to-severe valvular disease other than MS. (iv) Congenital MS. (v) Patients with organic tricuspid valve disease. (vi) Evidence of rheumatic activity during the preceding 6 months. (vii) Patients with atrial fibrillation or other atrial arrhythmias. (viii) Patients with atrioventricular conduction abnormalities. (ix) Patients with hypertension, diabetes, or ischemic heart disease. (x) Patients with chronic obstructive pulmonary disease.

Standard trans-thoracic echocardiographic study:

All patients underwent Two-dimensional (2D) echocardiography and Doppler Tissue Imaging (DTI) studies before PTMC, 24 hours after BMV and at 6 months follow up. All patients were studied in the left lateral decubitus position using an ultrasound system (GE vivid 7). Standard 2D and M-mode echocardiograms were obtained according to the American Society of Echocardiography (ASE) guidelines. Basic measurements included LVEF by M mode, left atrial (LA) anterior–posterior dimensions. The conventional indices for assessment of the severity of MS; MVA by planimetry and pressure half-time and the peak and mean mitral valve pressure gradients and PASP were measured as recommended.[11]

RV fractional area change (FAC), Tei index or RV myocardial performance index (MPI) and tricuspid annular plane systolic excursion (TAPSE) were also calculated as per ASE guidelines.[12] Peak systolic velocity at the lateral tricuspid annulus (S') was obtained from pulsed TDI and highest systolic velocity was recorded without averaging the Doppler envelope (Fig. 1). Isovolumic contraction time (IVCT) was measured from the duration of isovolumic velocity (IVV) measured by pulse wave TDI at the lateral tricuspid annulus. Isovolumic relaxation time (IVRT) was measured from the end of S' to the onset of early diastolic lateral tricuspid annular velocity (E'). RV MPI was calculated by dividing isovolumic time (IVCT + IVRT) by ejection time (ET). Isovolumic acceleration (IVA) was defined as the peak IVV divided by activation time i.e. time to peak velocity.

PASP was estimated from peak tricuspid regurgitation jet velocity (V) using modified Bernoulli equation (PASP = 4 V^2 + estimated RA pressure). RA pressure was estimated from inferior vena cava (IVC) diameter and extent of inspiratory collapse. IVC diameter was measured at end of expiration in subcostal view; just proximal to the site of hepatic vein drainage. The change in diameter of the IVC with quite respiration and with sniff was

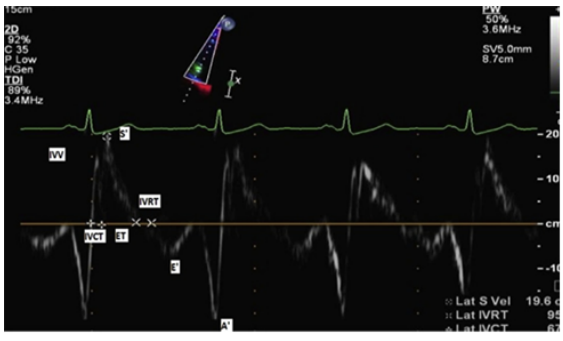

Figure 1: Tissue Doppler Imaging At The Lateral Tricuspid Annulus.

measured. If IVC diameter was <2.1 cm and collapse with a sniff was >50%, RA pressure was taken as 3 mm Hg, whereas if the IVC diameter was >2.1 cm with <50% collapse with a sniff, RA pressure of 15 mm Hg was taken. In cases with IVC diameter <2.1 cm, but collapse with sniff <50% or IVC diameter >2.1 cm and collapse >50%, intermediate value of 8 mmHg was taken.

Percutaneous transvenous mitral commissurotomy (PTMC/BMV):

The BMV was performed via an antegrade transvenous approach using an Inoue balloon and stepwise dilatation strategy.[13] No atrial or atrial appendage thrombus was detected in all patients before the procedure by using TEE. Reassessment of mitral valve score and evaluation of MR was also done by TEE. The nominal balloon diameter was decided according to the height of the patient (i.e. height (cm)/10+10 = balloon diameter). Successful BMV 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:

All the variables were expressed as mean ± standard deviation (SD). For paired data, student's t-test was used to determine the significance of differences in RV function parameters pre- and post-BMV after testing for normal distribution.

RESULTS

Study group consisted of 23 patients with isolated severe MS in sinus rhythm, who met the inclusion criteria and 32 healthy age and sex matched controls. Baseline variables are summarized in **Table 1**.

Mean age of the cases was 30.83±5.99 years and that of controls was 33±6.22 years. There was no difference in age between cases and controls.

In cases MVA was 0.95±0.21 by 2 D before BMV. Peak transmitral gradients and mean transmitral gradients before BMV were 23.57±3.58 mm Hg and 12.52±2.39 mm Hg respectively in cases.

Table 1 : Baseline Variables Of Cases And Controls.

VARIABLE	CASES (n= 23)	CONTROLS (n= 32)	P Value
	Mean ± SD	Mean ± SD	
AGE(Years)	30.83 ± 5.99	33.00 ± 6.22	0.207
RVMPI	0.45 ± 0.11	0.26 ± 0.03	<0.001
RVFAC(%)	38.74 ± 10.90	59.75 ± 1.39	<0.001
TAPSE(mm)	17.43 ± 3.42	26.75 ± 1.13	<0.001
ESTIMATED PASP(mmHg)	57.30 ± 9.76	19.13 ± 2.90	<0.001
S'(cm/s)	11.52 ± 1.97	16.31 ± 1.20	<0.001
IVA(m/s^2)	2.79 ± 0.44	3.43 ± 0.50	<0.001
LA DIMENSION(cm)	5.01 ± 0.60	2.95 ± 0.18	<0.001

RVMPI- Right Ventricular Myocardial Performance Index; RVFAC- Right ventricular Fractional Area Change; TAPSE- Tricuspid Annular Plane Systolic Excursion; PASP- Pulmonary Artey Systolic Pressure; S'- Peak systolic velocity at lateral tricuspid annulus; IVA- Isovolumic Acceleration; LA- Left Atrium; NS- Non Significant; S- Significant; SD- Standard Deviation.

RVMPI (TEI Index) was 0.45±0.11 in cases and 0.26±0.03 in controls; RVFAC in cases was 38.74±10.90 % and that in controls was 59.75±1.39 %; TAPSE of cases and controls was 17.43±3.42 mm and 26.75±1.13 mm respectively. Estimated PASP of cases was 57.30±9.76 mmHg and that of controls was 19.13±2.90 mmHg; similarly S' was 11.52±1.97 cm/s in cases and 16.31±1.20 cm/s in controls. IVA in cases was 2.79±0.44 m/s^2 and that in controls was 3.43±0.50 m/s^2; LA dimension in cases was 5.01±0.60 cm and in controls was 2.95±0.18 cm. LVEF in cases was 60.43±7.52 % and in controls was 67.19±2.56 %.

There was significant difference in RVMPI (TEI Index), RVFAC, TAPSE, PASP, S', IVA, LA Dimension, and LVEF between cases and controls (P< 0.001). RVMPI (TEI Index), Estimated PASP and LA dimensions was significantly higher in cases than in controls, where as RVFAC, TAPSE, S' and IVA were significantly lower in cases than in controls.

Echocardiographic parameters before and after BMV (Table 2):
Following BMV, on day 1, the 2D MVA increased from 0.95 ± 0.21 cm^2 to 1.60 ± 0.14 cm^2 (P<0.001). Peak transmitral gradients and mean transmitral gradients before BMV were 23.57 ± 3.58 mm Hg and 12.52 ± 2.39 mm Hg, and significantly decreased after BMV to 8.91 ± 1.44 mm Hg and 4.43 ± 0.90 mm Hg (p < 0.001 and < 0.001) respectively, on day 1. A significant fall in estimated pulmonary artery systolic pressure (PASP) was seen immediately after BMV from 57.30 ± 9.76 mm Hg to 47.22 ± 9.34 mm Hg (p < 0.001). A further fall in estimated PASP was observed at 6 months follow-up (35.78 ± 7.01; p < 0.001). LA diameter was 5.01 ± 0.60 cm prior to BMV, decreased to 4.98 ± 0.61 cm (p=0.11) after BMV which was nonsignificant. At 6 months LA diameter was significantly decreased to 4.6 ± 0.50 cm (p<0.001). LV Ejection Fraction (LVEF), was unchanged immediately after BMV but there was significant increase at 6 months (60.43 ± 7.52 % before BMV Vs 63.48 ± 5.10 % after 6 months; p < 0.001).

RVMPI (TEI Index) was 0.45 ± 0.11 and 0.45 ± 0.10 before and after BMV (Day1) respectively, which was nonsignificant (p= 0.213). A significant change in RVMPI was observed during follow up period of 6 months. RVMPI was 0.38 ± 0.06 at 6 months follow up which was improved significantly (p< 0.001) as compared to pre BMV.

RVFAC was 38.74 ± 10.90 % before BMV which significantly increased to 43.09 ± 10.42 % post BMV (Day1) (p<0.001); and to 48 ± 7.62 % after 6 months (p<0.001).

Before BMV TAPSE was 17.43 ± 3.42 mm which significantly increased to 18.78 ± 2.91 mm post BMV (Day1) (p<0.001), and to 19.91 ± 2.02 mm after 6 months (p<0.001).

Table 2 : Comparison Of Echocardiographic Parameters Before And After BMV (day 1 And 6 Months) In Patients With Severe Ms. (n= 23)

VARIABLE	Pre BMV	Post BMV (Day 1)	Post BMV (6 Months)	P Value		
	Mean ± SD	Mean ± SD	Mean ± SD	Pre BMV vs Post BMV (Day1)	Pre BMV vs Post BMV (6 Months)	Post BMV (Day1) vs Post BMV (6 Months)
MVA (cm^2)	0.95 ± 0.21	1.60 ± 0.14	1.61± 0.15	<0.001	<0.001	0.103
PG(Across MV) (mm Hg)	23.57 ± 3.58	8.91 ± 1.44	8.52 ± 1.16	<0.001	<0.001	0.016
MG(Across MV) (mm Hg)	12.52 ± 2.39	4.43 ± 0.90	3.91 ± 0.73	<0.001	<0.001	0.001
LA Dimension(cm)	5.01 ± 0.60	4.98 ± 0.61	4.6 ± 0.50	0.110	<0.001	<0.001
RVMPI	0.45 ± 0.11	0.45 ± 0.10	0.38 ± 0.06	0.213	<0.001	<0.001
RVFAC (%)	38.74 ± 10.90	43.09 ± 10.42	48 ± 7.62	<0.001	<0.001	<0.001
TAPSE (mm)	17.43 ± 3.42	18.78 ± 2.91	19.91 ± 2.02	<0.001	<0.001	<0.001
Estimated PASP (mm Hg)	57.30 ± 9.76	47.22 ± 9.34	35.78 ± 7.01	<0.001	<0.001	<0.001
S'(cm/s)	11.52 ± 1.97	11.70 ± 1.84	12.65 ± 1.37	0.100	<0.001	0.001
IVA (m/s^2)	2.79 ± 0.44	2.81 ± 0.42	3.10 ± 0.27	0.083	<0.001	<0.001
LVEF (%)	60.43 ± 7.52	60 ± 7.69	63.48 ± 5.10	0.162	<0.001	0.001

RVMPI- Right Ventricular Myocardial Performance Index; RVFAC- Right ventricular Fractional Area Change; TAPSE- Tricuspid Annular Plane Systolic Excursion; PASP- Pulmonary Artey Systolic Pressure; S'- Peak systolic velocity at lateral tricuspid annulus; IVA- Isovolumic Acceleration; LA- Left Atrium; LVEF- Left Ventricular Ejection Fraction; NS- Non Significant; S- Significant; PG(Across MV)- Peak gradient across mitral valve; MG(Across MV)- Mean gradient across mitral valve; SD- Standard Deviation.

S' (Peak systolic velocity at lateral tricuspid annulus) and IVA (Isovolumic Acceleration) were unchanged immediately after BMV. S' was 11.52 ± 1.97 cm/s and 11.70 ± 1.84 cm/s, before and after BMV (Day1), respectively (p=0.10). IVA was 2.79 ± 0.44 m/s^2 before BMV and 2.81 ± 0.42 m/s^2 after BMV (Day1) (p=0.083). However there was significant improvement was observed in S' and IVA during follow up period of 6 months. S' and IVA were 12.65 ± 1.37 cm/s and 3.10 ± 0.27 m/s^2 respectively at 6 months (p<0.001 for both).

All parameters including RVMPI, RVFAC, TAPSE, Estimated PASP, S', IVA, LA Dimension, LVEF showed improvement at 6 months as compared to day 1 post BMV.(p<0.001)

Table 3 : Number Of Patients Having RV Dysfunction By Various Criteria After BMV(n=23)

VARIABLE	Pre BMV	Post BMV Day1	Post BMV 6 Months	P Value		
				Pre BMV vs Post BMV (Day1)	Pre BMV vs Post BMV (6 Months)	Post BMV (Day1) vs Post BMV (6 Months)
RVMPI/S'/IVA	8	7	1	0.753	0.031	0.019
RVFAC (%)	12	5	0	0.032	<0.001	0.017
TAPSE (mm)	13	6	1	0.036	<0.001	0.040

RVMPI>0.55; S'<10; IVA<2.2; RVFAC<35; TAPSE<16.

8 of the cases had abnormal RVMPI, S' or IVA values by tissue doppler, 12 had abnormal RVFAC and 13 had abnormal TAPSE values before BMV.

Out of 8 patients who have had abnormal RVMPI, S' or IVA values before BMV 1 patient improved at day 1, while 7 more patients improved at 6 months. 1 patients had abnormal RVMPI, S' or IVA values at 6 months.

Out of 12 patients who have had abnormal RVFAC values before BMV 7 patients improved at day 1, while 5 more patients improved at 6 months. None had abnormal RVFAC values at 6 months.

Out of 13 patients who have had abnormal TAPSE value

before BMV 7 patients improved at day 1, while 5 more patients improved at 6 months. 1 patient had abnormal TAPSE value at 6 months.

DISCUSSION

In patients with MS, RV function is closely related to symptoms, functional capacity, need and timing for interventions, perioperative mortality, and postoperative results.[8] 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.

Our study showed that the RV systolic and diastolic function is impaired in patients with severe MS as assessed by various two-dimensional echocardiographic and Doppler Tissue Imaging (DTI) parameters. RVMPI (TEI Index), Estimated PASP and LA dimensions was significantly higher in cases than in controls, where as RVFAC, TAPSE, S', IVA and LVEF were significantly lower in cases than in controls. It correlates with earlier hemodynamic and clinical studies, which showed impaired RV function in MS patients. [8,14-16]

In accordance with results, Drighil et al.[17] suggested that patients with MS have depressed global and regional RV function compared with normal subjects; findings go hand in hand with previous radionuclide and haemodynamic studies. Study by Mahfouz et al.[18] revealed a significant decrease in TAPSE in MS denoting that the RV function is usually impaired (even subclinical) in those patients despite of mild pulmonary hypertension.

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 PAH. 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.[19,20]

In addition in our study, a significant rise in FAC and TAPSE and decrease in estimated PASP was noted post BMV, which indicates improved RV function. This finding is concordant with the observation by Burger et al.[16] who showed immediate improvement in RV ejection fraction post BMV. Such a rapid improvement of RV function can be explained only by a reduction in afterload by relieving left atrial hypertension and resultant decrease in PASP. Also in our study the FAC, TAPSE and estimated PASP continued to improve in long term, as denoted by further rise in FAC and TAPSE as well as decrease in estimated PASP at 6 months.

In study by Santosh Kumar C et al.[21], both RVEF and RVFAC did not change immediately after PTMC but improved significantly at 6 months after PTMC in both PAH and non PAH groups. In the study by Drighil et al.[17] there was a positive trend in the data of these parameters (p=0.27 and p=0.24).

TAPSE is one of the parameters which reflect systolic function of RV and showed an excellent correlation with RV ejection fraction calculated using echocardiography[22] or radionuclide angiography,[23] and its measurement has been proven to be highly reproducible and easy to obtain.[24]

In the our study TAPSE showed significant increase (between before and after 24 hours; before and after 6 months; after 24 hours and after 6 months). This was concordant with the study by Mahfouz et al.[18] which included 147 patients in rheumatic MS. Similar results were also found by Adavane et al.[25] in a study on 33 patients with MS. They compared echocardiographic parameters of RV function before BMV, 24–48 h after BMV and 1 month after BMV and showed a significant increase of TAPSE (P= 0.01 immediately after BMV; P <0.001 at 1 month) and a significant correlation with the decrease in PVR, RVRA pressure gradient, the immediate decrease in MV Gradient, and the increase in MVA at one month. In patients with MVA> 1.5 cm2 after BMV, successful BMV results in a significant improvement of RV systolic function assessed by TAPSE.

In the current study RV MPI (TEI Index), S' or IVA showed no significant change immediately after BMV. This lack of immediate improvement in RV MPI, S' or IVA may be due to effects of sustained pressure overload on the RV for prolonged periods. These have shown improvement over 6 months follow up. In our study RV MPI significantly decreased at 6 months and S' as well as IVA significantly increased at 6 months. This again is concordant with the observation by Mohan et al.,[8] who showed that RV MPI shows no immediate change after BMV, but decrease in long term follow up. However in a study done by Drighil et al,[17] there was a decrease in RV MPI post-BMV. In the same study IVA was decreased after BMV, whereas in our study IVA showed no change immediately after BMV but significantly increased after 6 months follow up. IVA and 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.

In our study tricuspid annular (lateral) systolic velocity (S') did not show significant change immediately after BMV and is comparable to previous studies. Bensaid et al,[26] observed a non-significant increase in tricuspid annular S'. Drighil et al,[27] found that S' did not change immediately after PTMC. In study done by Wang et al.,[28] tricuspid S' was the best predictor of RV ejection fraction among several echocardiographic parameters. Saxena et al.,[29] noticed a strong correlation between tricuspid S' and RV fractional area change, regardless of pulmonary artery pressures. On the other hand Ragab A et al.,[30] noticed significant increase in tricuspid S' after PTMC and also concluded that tricuspid S' and TAPSE may precociously recognize patients with poor prognosis especially after PTMC. The absence of change in S' 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 S' 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.

Similar results were found by Santosh Kumar C et al.,[21] They also showed that, tricuspid annular S' improved significantly immediately after PTMC and continued to improve at 6 months in patients without PAH. In patients with PAH, S' did not improved significantly immediately after PTMC and improved significantly at 6 months due to the positive RV remodeling after successful PTMC.

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. Similar results were

found by Santosh Kumar C et al.,[21] They also showed that, 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 improve significantly immediately after PTMC but improved significantly at 6 months.

In an animal model, Vogel et al.,[31] 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.[32]

Myocardial velocity 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.

Thus, in the patients with severe MS, RV performance is decreased predominantly due to increase in RV afterload, which improves after BMV. Alternatively there is a possibility that this immediate improvement in RVfunction is due to improved hemodynamics, better LV filling and RV emptying after BMV as only the load dependent parameters are improved. Since there is no immediate change in load independent parameters like RV MPI in previous studies[8] and S', IVA as well as RV MPI in the current study, they suggest RV myocardial structural changes secondary to long standing increased pressure overload. Finally, significant improvement in these parameters in long term in our study as well as other studies[33] supports the gradual remodeling of the RV after BMV and the need for strict medical and secondary rheumatic chemoprophylaxis.

There was insignificant immediate and significant long term reduction in LA anteroposterior dimension. A study by Adavane et al,[34] showed immediate decrease in LA volume after BMV in patients in sinus rhythm. The most probable explanation of this immediate reduction in LA size is decompression of LA and better emptying by releasing the mitral valve obstruction by the BMV.

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.[35] The exact reason for this immediate improvement is unclear, but improvement in the atrial contribution to LV filling,[36] improved myocardial contractility[37] may be the possible explanations and probably better RV structural changes might also lead to improved LV function over time.

CONCLUSION

RV systolic and diastolic function is impaired in patients with severe MS and can be assessed by various two-dimensional echocardiographic and Doppler Tissue Imaging (DTI) parameters before the appearance of clinical signs of systemic venous congestion. Improvement in RV systolic and diastolic function starts immediately after BMV with reduction in RV afterload (PA pressure) and continues to improve over long term period.

Study Limitations

The number of patients was small, which is the main limitation of this study. To confirm these findings a study with larger sample size is required.

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