Immediate Impact of Percutaneous Transvenous Mitral Valve Commissurotomy (PTMC) on Right Ventricular Function

SK Kundu, AAS Majumder, D Halder, SK Chakrovortty, MR Khan, B Dutta, KK Karmoker, M Ahmed

Department of Cardiology, NICVD, Dhaka, Bangladesh

Keywords:
Mitral stenosis, Doppler tissue imaging, Right ventricular function, Percutaneous transvenous mitral commissurotomy.

Abstract

Background: *The aim of this study was to evaluate the immediate impact of Percutaneous Transvenous Mitral Commissurotomy (PTMC) on RV function in patients with mitral stenosis (MS).*

Methods: *This study was conducted in the National Institute Cardiovascular Diseases, Dhaka for a period of one year starting from October 2008 to September 2009. A total of 50 consecutive patients (Case group) with mitral stenosis were selected after considering inclusion and exclusion criteria that subsequently undergone PTMC. The control group (n=50) consisted of age and sex matched healthy individual (having no ECG or echocardiographic evidence of structural or functional cardiovascular disease). Healthy control group was taken because there was no data about RV function in our population. Control group used to compare with baseline characteristics of case group.*

Results: *Immediately after PTMC (24 to 48 hours) mitral valve area increased from 0.8± 0.1 to 2.0 ± 0.2 (p <0.001) and RV outflow tract fractional shortening (RVOTfs %) increased from 54.9 ± 4.6 to 74.9 ± 4.8% (*p *<0.001). There was a significant decrease in systolic pulmonary artery pressure from 47.7 ± 7.9mmHg to 28.2 ± 5.9 mmHg (*p *<0.001), in the RV Tei index from 0.5 ± 0.1 to 0.3 ± 0.1 (*p *<0.001), in myocardial acceleration during isovolumic contraction (IVA) at the lateral tricuspid annulus from 0.4 ± 0.1 m/s² to 0.3 ± 0.0 m/s² (*p *<0.001). The RVEF (%) did not exhibit any significant change from pre-PTMC figure (*p *= 0.538).*

Conclusion: *After successful PTMC the parameters of infundibular and global RV function as assessed by RVOTfs and Tei index showed significant improvement and significant decrease in RV contractility as assessed by IVA was observed. Further work using larger numbers of patients is needed to confirm our findings and to assess their utility in patient follow-up and management.*

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Introduction

Rheumatic heart disease causes significant morbidity and mortality among the cardiovascular diseases.Mitral stenosis (MS) is the commonest (54%) of all rheumatic heart disease. [1]A survey was carried out amongst 7062 people of different age groups in Dhaka city and in a village. It was found that 207 (2.92%) persons had some sort of heart disease. Hypertension was present in 83 (1.10%) persons. Rheumatic heart disease, ischaemic heart disease and cardiac arrhythmia were detected in 53 (0.75%), 24 (0.33%) and 16 (0.22%) persons respectively.[2]

A community based study was done on 5923 rural Bangladeshi children aged 5-15 years to determine the prevalence of rheumatic fever (RF) and rheumatic heart disease (RHD). The prevalence was found to be 1.2 (95% confidence interval 0.3-2.1) per 1000 for RF defined by revised Jones criteria and 1.3 (0.4-2.2) per 1000 for Doppler echocardiography confirmed RHD.[3]

Abnormalities of right ventricular function play an important role in the development of clinical symptoms and the over all prognosis of the patients with MS. The right ventricular function is an important determinant of clinical symptoms, exercise capacity, pre-operative survival and post-operative outcome in patients with mitral stenosis.[4]

Address of Correspondence: Dr. Samir Kumar Kundu, Department of Cardiology, National Institute of Cardiovascular Diseases, Dhaka, Bangladesh.

Impairment of right ventricular (RV) function by post rheumatic mitral stenosis due to passive increase of left atrial pressure and due to the reactive changes of pulmonary arteriolar vasculature, right ventricular after load may increase to values 25 to 30 fold above normal, leading to right ventricular overload and right ventricular failure. It is hypothesized that rheumatic heart disease may directly involve the myocardium, thus directly impairing right ventricular function.[5] Intramyocardial branches of coronary vessels were involved in a form of active rheumatic vasculitis or inactive lesions characterized by medial hypertrophy and replacement fibrosis.[6]

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.[7] The problem is compounded by irregular trabeculations, a separate infundibulum, and variations in right ventricular shape with altered loading conditions. The substernal right ventricle is less accessible than the left ventricle and its dimensions more difficult to standardize.[8]

RV function is closely related to symptoms, functional capacity, need and timing for interventions, perioperative mortality and postoperative results in patients with MS. 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. Thus, RV dysfunction is an important indicator to evaluate the severity of MS. Radionuclide ventriculography, 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. Recently a new tissue Doppler imaging (TDI) derived index of myocardial acceleration during isovolumic contraction (IVA) is a measurement of RV contractile function that is unaffected by preload and afterload change in a physiological range. This novel index (IVA) may be ideally suited to the assessment of acute changes of RV function in clinical studies and could be used in early detection of RV systolic dysfunction in patient with MS, before the signs of systemic venous congestion occur.[9]

Echocardiographic parameters of RV function are RV ejection fraction (RVEF), RV fractional area change (RVFAC), RV outflow tract fractional shortening (RVOTfs), RV total ejection isovolume index (RV Tei index) and tricuspid annular plane systolic excursion (TAPSE).[10]

A combined myocardial performance (RVTei) index may be more effective for analysis of global cardiac dysfunction than systolic and diastolic measure alone. [11]

The importance of the right ventricle as a determinant of exercise capacity and the prognostic value of RV function in heart failure, as well as in cardiac surgery outcome.[12]

Management of the patients with mitral stenosis largely depends upon medical, interventional and surgical management. Medical management is largely symptomatic. Surgical management offers cure which includes close mitral commissurotomy (CMC), open mitral commissurotomy (OMC) and mitral valve replacement (MVR). MVR was the definitive treatment for symptomatic mitral stenosis. But after the introduction of percutaneous transvenous mitral valve commissurotomy (PTMC), it is emerged as a safe and effective procedure for the treatment of symptomatic mitral stenosis.[13]

Dr. Kanzi Inoue introduced PTMC as an alternative to surgery in the treatment of mitral stenosis. The first prototype of Inoue balloon catheter was manufactured in 1980. It was used clinically in 1982 during open heart surgery and initial report on PTMC was published in 1984.[14]

The purpose of this study was to assess the immediate effect of PTMC on RV function using two-dimensional and Doppler echocardiographic indices.

Material and methods: This experimental interventional study was conducted in the National Institute Cardiovascular Diseases, Dhaka for a period of one year starting from October 2008 to

September 2009. A total of 50 consecutive patients with mitral stenosis were selected after considering inclusion and exclusion criteria that subsequently undergone PTMC. The study population was divided into two groups, cases and controls. Case group (n=50): Patients of mitral stenosis, admitted in NICVD fulfilling the inclusion criteria of PTMC.Control group (n=50): The control group consisted of age and sex matched healthy individual (having no ECG or echocardiographic evidence of structural or functional cardiovascular disease).

Inclusion Criteria: Patients admitted in NICVD irrespective of age & sex with the diagnosis of symptomatic mitral stenosis that subsequently undergone PTMC (fulfilling criteria of indications).

Exclusion Criteria

- MR > grade II (truly 3+ to 4+)
- High MGH score or Wilkins score (9 -16)
- Patients with other significant valve lesions (AR > grade II, AS moderate to severe) requiring surgical treatment
- Evidence of left atrial thrombus
- Patients concomitant with PS, ASD
- Patients with documented coronary artery disease requiring surgical revascularization
- Chronic lung diseases
- Cardiomyopathies
- Severe kyphoscoliosis (thoracic / abdominal)
- Patients having contraindication to trans-septal puncture, e.g. very thick IAS, aneurysm of interatrial septum, aortic root dilatation, severe TR, huge right atrium.

Study Procedure

Relevant history was taken; clinical examination was done and recorded in a pre-designed data collection form.

Preprocedural Evaluation

(a) Routine blood tests were done for each patient, i.e. complete blood count, blood urea, serum creatinine, random blood sugar, blood grouping, bleeding time, clotting time, VDRL, HBsAg, Anti HCV, and Anti HIV.

(b) A 12 lead surface ECG

(c) X-ray chest (P/A) view

(d) Echocardiography

- All studies were obtained using a SIEMENS ACUSON X 500 ultrasonographic machine equipped with a 3.5 MHz transducer.
- Mitral valve area was calculated by planimetry method in short axis view and also by pressure half time (PHT) method.
- Mitral valve morphology was studied extensively by several methods.
- Mitral valve echo score (Wilkins score) was done and detailed information about leaflet mobility, valvular thickening, calcification and subvalvular thickening were also obtained.

2-dimensional and M-mode study: Left atrial diameter was calculated and LV study was done.

Mean and peak pressure gradient across the mitral valve were calculated by using continuous wave Doppler.

Pulmonary artery systolic pressure (PASP) was measured considering the tricuspid regurgitation by Continuous wave Doppler.

Colour flow imaging was done.

Echocardiographic Parameters of Right Ventricular Function

RVOT fs (%) (Right ventricular outflow tract fractional shortening)

RV Tei (Right ventricular total ejection isovolume) index

RVEF (%) (Right ventricular ejection fraction)

RV IVA (Right ventricular myocardial acceleration during isovolumic contraction.[10]

RVOTfs (%): From the parasternal short axis view at the level of the aortic root, the RV outflow tract diameters at end-diastole and end-systole calculated and RVOTfs (%) calculated by (RVEDD - RVESD / RVEDD) X 100 .[9,10]

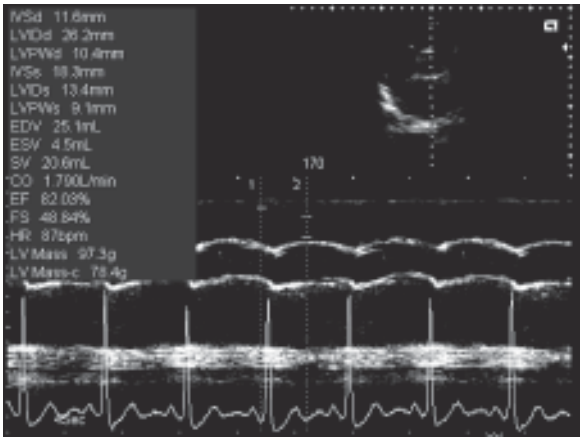

Fig.-1: *Measurement of RVOTfs (%).*

RVEF(%): In Apical 4 chamber view, calculated by using Simpson's (single plane) Rule (tracing around the endocardial border going from one side of the tricuspid valve annulus to the other and joining the two ends with a straight line both in systole and diastole.[10]

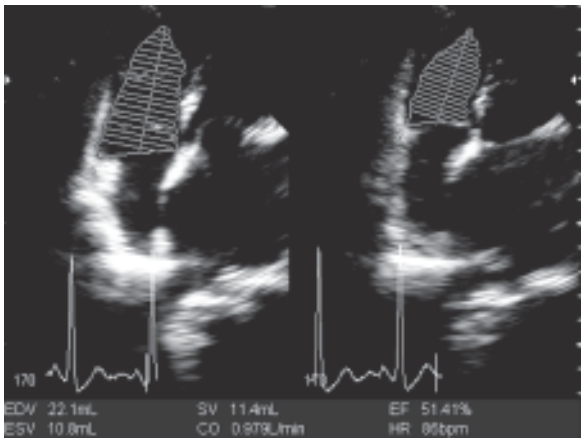

Fig.-2: *Measurement of RVEF (%).*

RV Tei Index: In apical 4 chamber view a sample volume (Pulsed wave Doppler) placed at the tricuspid valve. RVTei Index calculated as the sum of isovolumic contraction time (IVCT) and isovolumic relaxation time (IVRT) divided by ejection time (ET).[9]

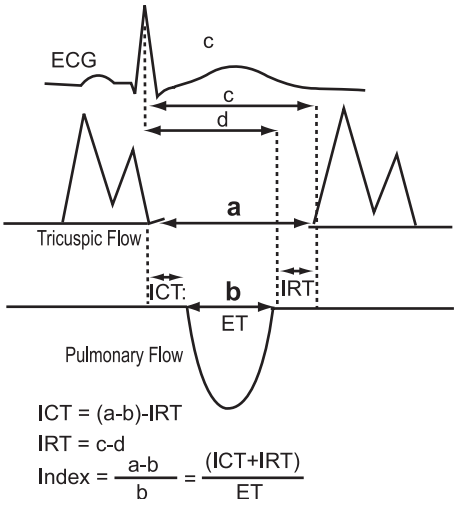

Fig.-3: *Measurement of RVTei index.*

Doppler Tissue Imaging (DTI) derived myocardial acceleration during isovolumic contraction (IVA) of tricuspid lateral annulus

RV IVA: in apical 4 chamber view, a sample volume placed at the tricuspid lateral annulus. IVA measured by dividing myocardial velocity during isovolumic contraction (IVV) by the time interval from onset of the myocardial velocity during isovolumetric contraction to the time at peak velocity of this wave (AT).[10] The ratio of IVV (myocardial velocity during isovolumic contraction) divided by the acceleration time (AT). IVA = IVV / AT.[9]

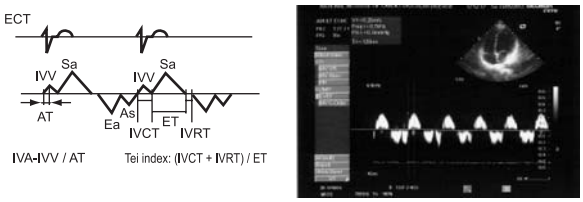

Fig.-4: *Measurement of RV IVA (m / s^2)*

Statistical Analysis

Procedural outcome analysis was done by statistical analysis of all data including clinical and echocardiographic variables. Both computer programmed based SPSS (statistical programme for social sciences) version 11.5 and manual technology were applied for statistical analysis. Data were expressed as mean ± SD. Analysis employed the Student's *t*-test for paired data to determine the significance of differences before and after PTMC. The differences between patients with MS and healthy subjects were identified using an unpaired Student's *t*-test. A p value < 0.05 as considered statistically significant. p value (Probability value) <0.05 was considered as (*) significant.

Results:

The findings of the study obtained from data analysis are presented below:

Table-I

Comparison of Age between Case and Control Groups

Age (yrs)	Group		p-value
	Case (n = 50)	Control (n = 50)	
≤20	8 (16.0)	10 (20.0)	
20 – 30	20 (40.0)	20 (40.0)	
> 30	22 (44.0)	20 (40.0)	
Mean ± SD	28.2 ± 6.7	27.8 ± 6.5	0.728

Mean data were analyzed using unpaired *t*-test; Figures in the parenthesis denote corresponding %.

Table I shows that 44% of patients in the case group was >30 years followed by 40% between 20 – 30 years and the rest 16% was 20 or below 20 years old. In the control group, 40% was over 30 years; another 40% between 20 – 30 years and remaining 20% was 20 or less than 20 years old. The mean

age was almost identical between group (28.2 ± 6.7 vs. 27.8 ± 6.5, $p = 0.728$).

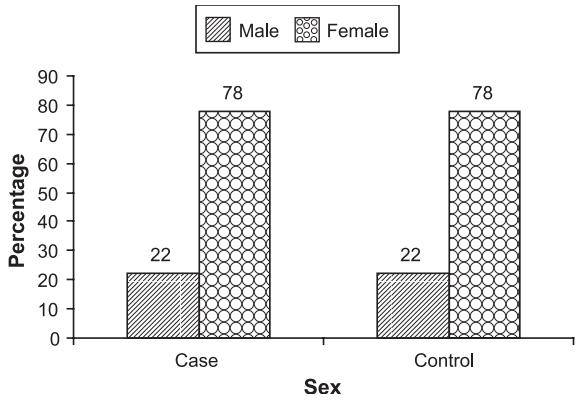

Fig.-5: *Comparison of sex between groups (n = 100)*

Figure 1 shows the sex distribution of the patients. A female preponderance was observed in both case and control groups (78% of the subjects was female).

Table-II

Comparison of BMI between groups

BMI	Group		*p*-value
	Case (*n* =50)	Control (*n*= 50)	
Underweight (<18.9 kg/m^2)	23 (46.0)	8 (16.0)	
Normal (18.9 – 24.9 kg/m^2)	27 (54.0)	42 (84.0)	
Mean ± SD	19.6 ± 2.4	20.9 ± 1.7	0.001

n = total number of patients
# Mean data were analyzed using unpaired *t*-test; Figures in the parenthesis denote corresponding %.

Body mass index of the patients depicts that 46% of the patients in the case group was underweight, and 54% was of normal BMI as opposed to 16% and 84% respectively in the control group.

Table-III

Distribution of clinical parameters of the study subjects (n = 50)

Clinical parameters	Frequency	Percentage
Shortness of breath	40	80.0
Palpitation	32	64.0
Cough	15	30.0
Chest pain	12	24.0
Hemoptysis	03	6.0
History suggestive of rheumatic fever	24	48.0
Rheumatic fever prophylaxis	18	36.0

Total will not correspond to 100% because of multiple responses.

Table III demonstrates the mode of clinical presentation. Majority (80%) of the patients complained shortness of breath, followed by 64% palpitation, 30% cough, 24% chest pain and 6% hemoptysis. About half (48%) of the patients had past history suggestive of rheumatic fever, while 36% patients was on prophylactic treatment of rheumatic fever.

Table-IV

Distribution of the patients according to New York Heart Association (NYHA) class before and after PTMC (n=50)

NYHA functional class	Pre-PTMC		Post-PTMC	
	Number	%	Number	%
I	0	0.0	15	30.0
II	18	36.0	25	50.0
III	26	52.0	10	20.0
IV	06	12.0	0	0.0

More than half of the patients were in NYHA functional Class III 26 (52.0%) followed by class II 18 (36.0%) and only 06 (12.0%) were in class IV. After procedure, 15 (30.0%) of the show symptoms of class I, 25 (50.0%) in class II and 10 (20.0%) in class III and no patient in class IV.

Table V

Percentage distribution of patients by pattern of complications (n=50)

Pattern of complications	Number	%
Severe MR (grade III)	1	2.0
MR grade II	5	10.0
MR grade I	7	14.0
Local vascular complications	6	12.0

Pattern of in hospital complications indicated that 1 patient (2.0%) developed severe MR grade III, 5 patients (10%) MR grade II, 7 patients (14%) MR grade I. Local vascular complications like hemorrhage, hematoma developed in 6 patients (12%).

Table-VI

Echocardiographic data of cases before PTMC and control group

Echocardiographic data	Group		*p*-value
	Pre-PTMC cases ($n = 50$)	Control ($n = 50$)	
LA antero-posterior diameter (mm)	48.3 ± 2.4	33.0 ± 2.4	<0.001
LVEDD (mm)	46.0 ± 2.7	46.3 ± 1.4	0.317
LVESD (mm)	30.0 ± 3.3	29.0 ± 1.8	0.062
LVEF (%)	68.9 ± 5.1	70.0 ± 5.5	0.365
MVA by Planimetry (cm^2)	0.8 ± 0.1	4.4 ± 0.3	<0.001
MVA by pressure half time (cm^2)	0.9 ± 0.1	-	-
Peak transmitral pressure gradient (mm of Hg)	26.0 ± 3.9	-	-
Mean transmitral pressure gradient (mm of Hg)	14.9 ± 2.5	-	-
PASP (mm of Hg)	47.7 ± 7.9	20.0 ± 1.4	<0.001

Table-VII

Echocardiographic measurements between Pre and post PTMC

Echocardiographic data	Case group ($n = 50$)		*p*-value
	Pre-PTMC	Post-PTMC	
LA antero-posterior diameter (mm)	48.3 ± 2.4	39.9 ± 2.5	<0.001
LVEDD (mm)	46.0 ± 2.7	45.8 ± 1.3	0.224
LVESD (mm)	30.0 ± 3.3	34.9 ± 4.3	<0.001
LVEF (%)	68.9 ± 5.1	67.5 ± 2.4	0.080
MVA by Planimetry (cm^2)	0.8 ± 0.1	2.0 ± 0.2	<0.001
MVA by pressure half time (cm^2)	0.9 ± 0.1	1.8 ± 0.1	<0.001
Peak transmitral pressure gradient (mm of Hg)	26.0 ± 3.8	8.0 ± 1.5	<0.001
Mean transmitral pressure gradient (mm of Hg)	14.9 ± 2.5	4.5 ± 1.0	<0.001
PASP (mm of Hg)	47.7 ± 7.9	28.2 ± 5.9	<0.001

Table-VIII

Echocardiographic data of right ventricular function between cases before PTMC and controls

Echocardiographic data	Group		*p*-value
	Pre-PTMC (case group)($n = 50$)	Control($n = 50$)	
RVEF (%)	51.1 ± 4.6	66.6 ± 4.9	<0.001
RVOTfs (%)	54.9 ± 4.6	79.3 ± 4.5	<0.001
RV Tei Index	0.5 ± 0.1	0.2 ± 0.04	<0.001
RV IVA (m/s^2)	0.4 ± 0.1	0.5 ± 0.1	<0.001

Data were analyzed using unpaired *t*-test and level of significance was < 0.05.

Table-IX

Echocardiographic parameters of RV function between pre and post PTMC (n = 50)

Echocardiographic data	Group		*p*-value
	Pre PTMC	Post PTMC	
RV EF (%)	51.1 ± 4.6	51.5 ± 2.1	0.538
RVOTfs (%)	54.9 ± 4.6	74.9 ± 4.8	<0.001
RV Tei Index	0.5 ± 0.1	0.3 ± 0.1	<0.001
RV IVA (m/s²)	0.4 ± 0.1	0.3 ± 0.1	<0.001

Data were analyzed using Paired *t*-test and level of significance was < 0.05.

Table VI. Presents the comparison of baseline echocardiographic data between case and control groups before PTMC. The mean LA antero-posterior diameter (mm) and PASP (mm of Hg) were significantly higher in case group prior to PTMC compared to healthy control group (48.3 ± 2.4 vs. 33.0 ± 2.4, $p < 0.001$ and 47.7 ± 7.9 vs. 20.0 ± 1.4, $p < 0.001$ respectively). The mean MVA (cm²) by planimetry were higher in control group than those in case group prior to PTMC (4.4 ± 0.3, 0.8 ± 0.1 $p < 0.001$ respectively). The LVEDD, LVESD and LVEF (%) were almost identical between the groups ($p = 0.317$, $p = 0.062$ and $p = 0.365$ respectively). The MVA by pressure half time, mean peak transmitral pressure gradient and mean transmitral pressure gradient of the case group were 0.9 ± 0.1 cm^2, 26.0 ± 3.9 mm of Hg and 14.9 ± 2.5 mm of Hg respectively.

Following PTMC the mean LA antero-posterior diameter (48.3 ± 2.4 mm to 39.9 ± 2.5mm), mean peak transmitral pressure gradient (26.0 ± 3.8 mm of Hg to 8.0 ± 1.5 mm of Hg), mean transmitral pressure gradient (14.9 ± 2.5 mm of Hg to 4.5 ± 1.0 mm of Hg) and mean PASP (47.7 ± 7.9 mm of Hg to 28.2 ± 5.9 mm of Hg) decreased significantly from their baseline figures ($p < 0.001$), while MVA by planimetry (0.8 ± 0.1 cm² to 2.0 ± 0.2 cm²) and MVA by pressure half time (0.9 ± 0.1 cm² to 1.8 ± 0.1 cm²) increased significantly from their baseline figures ($p < 0.001$). LVEDD, and LVEF (%) did not experience any significant change from their baseline figures ($p = 0.224$ and $p = 0.080$ respectively) and LVESD did significantly increased from their baseline figure ($p = 0.001$).

The right ventricular ejection fraction (RVEF %), right ventricular outflow tract fractional shortening (RVOTfs %) and right ventricular myocardial acceleration during isovolumic contraction (RV IVA m/s²) were significantly lower in patients before PTMC than those in normal subjects (51.1 ± 4.6% vs. 66.6 ± 4.9%, $p < 0.001$; 54.9 ± 4.6% vs. 79.3 ± 4.5%, $p < 0.001$; 0.4 ± 0.1 m/s^2 vs. 0.5 ± 0.1 m/s^2, $p < 0.001$ respectively). In contrast, RV Tei index was much higher in the case group before PTMC than that in the control group (0.5 ± 0.1 vs. 0.2 ± 0.04, $p < 0.001$).

Table IX. Shows that RVOTfs (%) significantly increased after PTMC (54.9 ± 4.6% vs. 74.9 ± 4.8%, $p < 0.001$ respectively). The mean right ventricular Tei index and mean RV IVA significantly decreased after PTMC (0.5 ± 0.1 vs. 0.3 ± 0.1, $p < 0.001$ and 0.4 ± 0.1 m/s² vs. 0.3 ± 0.1 m/s², $p < 0.001$ respectively). The RVEF (%) did not exhibit any significant change from pre-PTMC figure ($p = 0.538$).

Discussion:

The quantitative echocardiographic assessment of RV function is difficult because of the ventricle's complex trapezoidal anatomy. A wide variety of techniques have been proposed, but none is currently considered the gold standard. From the first modality ejection fraction (EF) reflect global systolic function, [15-17] the- Tei index reflects both RV systolic and diastolic function[17] and RVOTfs reflects RV infundibular function.[18]

In this study RVOTfs (%) increased after PTMC from 54.9 ± 4.6 % to 74.9 ± 4.8% (p= < 0.001) this is comparable to study done by Drighil et al[10] Which showed RVOTfs (%) increased from 57 ± 15% to 72 ± 12%. Significant lower RVOTfs (%) 54.9 ± 4.6% in patients group than control group RVOTfs (%) 79.3 ± 4.5% which is consistent with Tayyareci et al [9] and also Drighil et al.[10]

We observed RV Tei index decreased after PTMC from 0.5 ± 0.1 to 0.3 ± 0.1 (p= < 0.001). This is

comparable to study done by Drighil et al[10] where they found RV Tei index decreased from 0.44 ± 0.25 to 0.29 ± 0.17. Patients with MS before PTMC significantly higher RV Tei index than control groups (0.5 ± 0.1 vs.0.2 ± 0.04). Tayyareci et al [9] showed RV Tei index higher in the patients with mitral stenosis 0.69 ± 0.2 than in the control group 0.28 ± 0.06 our study is consistent with this result.

The RV IVA decreased after PTMC from 0.4 ± 0.1 to 0.3 ± 0.1 m/s² (p= < 0.001) this is comparable to study done by Drighil et al [10] showed RV IVA decreased from 0.36 ± 0.11to 0.25 ± 0.07. Significant lower RV IVA 0.4 ± 0.1 in patients group than control group RV IVA 0.5 ± 0.1 which is consistent with Drighil et al [10] and also Tayyareci et al .[9]

We found RVEF (%) shows no significant change after PTMC from 51.1 ± 4.6 to 51.5 ± 2.1 % (p=0.538). This is comparable to study done by Drighil et al,[10] which also showed no significant change from 50 ± 11 to 55.5 ± 12 %. But significantly lower RVEF (%) 51.1 ± 4.6% in patients group than control group 66.6 ± 4.9% which consistent with Drighil et al.[10]

This study was done to find out the improvement of right ventricular function immediately after PTMC. Our results suggest that patients with MS have depressed global and regional function compared with normal subjects. The reasons for this impaired function in MS due to passive increase of left atrial pressure and the reactive changes of pulmonary arteriolar vasculature, right ventricular after load may increase to values 25 to 30 fold above normal, leading to right ventricular overload and right ventricular failure. The rheumatic heart disease may directly involve the myocardium, thus impairing right ventricular function Burger et al.[5] Intramyocardial branches of coronary vessels were involved in a form of active rheumatic vasculitis or inactive lesions characterized by medial hypertrophy and replacement fibrosis to cause dysfunction Malhotra et al.[6]

In this study shows immediately after PTMC, the decrease in RV Tei index and PASP together with the increase in RVOTfs (%) suggests that RV outflow tract systolic function improved as a result of an acute decrease in RV afterload. This is consistent with the study by of Drighil et al [10] in 12 patients. The RV Tei index decreased significantly which indicates improvement of global right ventricular function. RV IVA significantly decreased which actually reflect the acute decrease in RV afterload and decrease in RV contractility immediately after PTMC that is consistent with Drighil et al.[10] RVEF (%) shows no significant change after PTMC may reflect this is a measure of change in the RV inflow, not the outflow or may be afterload independent reported by Drighil et al.[10]

IVA is likely the most trustworthy parameter of RV function. However, in the absence of a gold standard, we decided to utilize multiple parameters to study RV functional changes before and after PTMC. To our knowledge, this is the first study in Bangladesh to use tissue Doppler echocardiography to assess RV functional changes in patients with MS acutely after PTMC.

The decrease in RV Tei index and peak pulmonary artery systolic pressure together with the increase in RVOTfs immediately post-PTMC suggest that RV outflow tract systolic function improved as a result of an acute decrease in RV afterload.

Our finding of a decrease in RV contractility, as assessed by IVA, may be clinically useful in prompting further diagnostic evaluation for patients with MS. After PTMC the LV ejection fraction showed no significant change implying that the overall LV function was unchanged.

Conclusion:

In addition to conventional parameters for assessment of immediate outcome of PTMC, right ventricular functions can be assessed by two-dimensional and Doppler echocardiographic indices before and after PTMC may be a useful tool. Myocardial acceleration during isovolumic contraction on the lateral side of the tricuspid annulus also decreased after PTMC. This decrease immediately after PTMC may actually reflect the acute decrease in RV afterload with its consequence on contractile function of the RV.

Study limitations:

Despite exercise of utmost caution throughout the study, it has got some important limitations:

- The sample size was small in number
- Inoue balloon had been reused it may have influence upon PTMC.
- Only in hospital observation was done no long term outcome was observed.

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