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PULMONARY ARTERIAL CAPACITANCE IN MITRAL STENOSIS AND EFFECT OF PERCUTANEOUS TRANSMITRAL COMMISSUROTOMY ON PULMONARY ARTERIAL CAPACITANCE

Cardiology

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ABSTRACT

BACKGROUND: Inoue balloon technique for mitral commissurotomy is well established and carried out worldwide in the treatment of mitral stenosis. Percutaneous Transmitral Commissurotomy (PTMC) is reported to be a cheaper and effective treatment for mitral stenosis. Although echocardiography has been used to evaluate Pulmonary Vascular Resistance (PVR) and Pulmonary Arterial Capacitance (PAC) in congenital heart disease, it is not routinely performed in patients with mitral stenosis. This study aims to assess the influence of PTMC on Pulmonary Arterial Capacitance in patient with rheumatic mitral stenosis.

METHODS/DESIGN: A prospective, case study was conducted at a tertiary care hospital in India. Participants were selected based on the inclusion criteria. All the patient underwent PTMC and the effect of PTMC procedure on pulmonary arterial capacitance (PAC) was assessed.

RESULTS: PAC was less in patients with mitral stenosis compared with the general population. There was statistically significant difference between PAC measured by echocardiography and by catheterization. No significant increase in PAC was recorded by catheterization immediately following PTMC.

CONCLUSION: The study could not establish any significant relationship between PAC and patients' baseline characteristics including age, functional status, mitral valve area, mitral valve gradient and pulmonary artery pressure.

KEYWORDS

Pulmonary Arterial Capacitance, Pulmonary Vascular Resistance, Percutaneous Transmitral Commissurotomy, mitral stenosis.

INTRODUCTION

Rheumatic mitral valve stenosis is an important sequelae of rheumatic heart disease, one of the common cardiac disorders in developing countries[1-3]. It frequently affects children and young adults in the developing world[4]. Percutaneous transmitral commissurotomy (PTMC) is established as an effective treatment for mitral stenosis and is now the procedure of choice [5-10]. Percutaneous transvenous mitral commissurotomy (PTMC) is a percutaneous procedure designed to open the mitral valve through transvenous access using a transseptal approach to enter the left atrium. The procedure is conventionally performed with a specially designed Inoue balloon or the double balloon technique which has rather become obsolete now. Several modifications of the Inoue balloon like the Accura balloon have also been introduced. Although usually assumed to be a relatively safe procedure but sometimes it too can have a complicated and stormy course [11]. Pulmonary hypertension frequently complicates mitral stenosis and may significantly influence long term prognosis[12,13]. The pulmonary arterial pressure can be out of proportion to the degree of left atrial hypertension, reflecting a major increase in pulmonary vascular resistance. Surgical decompression of the left atrium through mitral commissurotomy or mitral valve replacement has often yielded marked regression of pulmonary hypertension[14]. Pulmonary Vascular Resistance (PVR) is commonly measured as an indicator of pulmonary hemodynamics in patients with congenital heart disease.

Capacitance, the change in volume in relation to the change in pressure, has been associated with right ventricular workload and may be an important factor influencing right ventricular function[15]. Pulmonary Arterial Capacitance (PAC) has necessitated cardiac catheterization to directly measure PA pulse pressure and stroke volume. Although echocardiography has been used to evaluate PVR and PAC in congenital heart disease, it is not routinely performed in patients with mitral stenosis. Hence, this study aims to assess the influence of PTMC on PAC in patient with rheumatic mitral stenosis.

METHODS

Study Design: The study was a prospective, case study assessing the influence of PTMC on PAC in patients with rheumatic mitral stenosis.

Study Site: The study was conducted in the Cardiology Department of PSGIMS .

Screening for Eligibility: Willingness of the patients to participate in the study was checked by the study physician before starting the study. Eligible participants were patients with moderate-to-severe rheumatic mitral stenosis who were advised to undergo PTMC during the study period,. Exclusion criteria include (1) patients who have failed PTMC either due to failure to carry out the procedure or development of significant mitral regurgitation, more than grade 1 with hemodynamic instability necessitating emergency mitral valve replacement, (2) Presence of significant aortic valve disease or organic tricuspid stenosis, (3) Absence of tricuspid regurgitation jet, (4) Chronic obstructive airway disease and renal failure, and (5) other major medical issues that would not allow the patient to undergo PTMC.

Study Enrollment and Description of Methodology: All patients, prior to the procedure, had a detailed clinical and echocardiographic evaluation (two-dimensional (2D) Doppler and color flow imaging), to assess the severity of mitral stenosis, valve morphology, and mitral regurgitation (MR). The severity of mitral stenosis was assessed by planimetry and Doppler methods. Right ventricular systolic pressure was estimated from TR jet in apical four chamber view or short axis planes by the modified Bernoulli equation ($P = 4V^2$). RA pressure was estimated from JVP. PA systolic pressure was estimated by adding RA pressure to the systolic pressure gradient obtained from TR jet. PA diastolic pressure was derived from PR jet or by using the formula, PA diastolic pressure = 0.49 x PA systolic pressure[15].

The flow velocity of pulmonary valve regurgitation was used to calculate pulmonary artery diastolic pressure using the modified Bernoulli equation**.** Pulmonary artery diastolic pressure was derived by adding end diastolic gradient to the right ventricular diastolic pressure (equal to RA pressure).

$PADP = RVEDP + \Delta P^{pv}$

PADP - Pulmonary Artery Diastolic Pressure
RVEDP - Right Ventricle End Diastolic Pressure
ΔP^{pv} - End diastolic pulmonary regurgitant gradient

Using pulmonary artery diastolic and systolic pressures, mean pulmonary artery pressure (PA mean) can be calculated.

PA mean = (PA systolic + 2 PA diastolic) / 3

RV stroke volume was calculated using the pulmonary valve diameter measured in the parasternal short axis view and the velocity time integral of the RV outflow Doppler.

RV stroke volume (ml) = Cross sectional area x TVI
= $(0.785 \times D^2/4) \times$ TVI

D - Pulmonary valve diameter
TVI - Velocity time integral of the RV outflow Doppler

PAC was estimated as the stroke volume divided by pulmonary artery pulse pressure.

PAC = Stroke volume / Pulmonary artery pulse pressure

Cardiac catheterization: A 9F sheath was placed in the right femoral vein and a 6F sheath was inserted in the right femoral artery. An initial right heart catheterization was done which included measurement of the oxygen saturation in the pulmonary artery and pressure measurements in the pulmonary artery and left atrium. The right femoral artery sheath provided access for the left ventricle and was used to measure transmitral gradient. Oxygen saturation of the pulmonary artery and aorta were used to calculate the cardiac output and pulmonary vascular resistance.

$$PVR = \frac{\text{Mean PA pressure} - \text{LA pressure}}{\text{Pulmonary blood flow}}$$

After the dilatation, hemodynamic parameters were measured and blood sampling for oxymetry was repeated. Immediately after the procedure, a transthoracic echocardiography was done to assess mitral valve area (by planimetry), transmitral gradient, mitral regurgitation, right ventricular systolic pressure, PA pressure and PAC. Evaluation of mitral valve area during the procedure by haemodynamic measurement lacks practicality and may be subject to error because of the instability of patient's condition and inaccuracy of Gorlin's formula in the presence of atrial shunts or in case of mitral regurgitation. So planimetry from two – dimensional echocardiography appears to be the method of choice[16].

Statistical Analysis:

All values were expressed as mean $\pm$ standard deviation. For assessing association between categorical variables, Fischer's exact test were used. Paired t test was used to compare PAC before and after PTMC. p value of < 0.05 was considered statistically significant. t-test and analysis of variance were employed for comparing mean values of PAC obtained by catheterization across functional class, mitral valve area and mitral valve gradient. Linear regression analysis was used to analyze contribution of variables on PAC obtained by catheterization.

RESULTS

Patient Characteristics

A total of 25 patients underwent PTMC during the study period out of which 22 were females and 3 were males with a mean age of 33.84 (range 8-59) years. This is shown in figure 1.

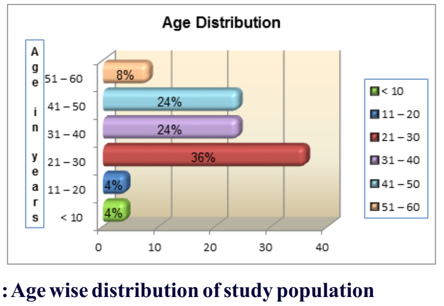

Figure 1: Age wise distribution of study population

The functional status of patients prior to PTMC was class II in 17 patients, class III in 6 patients and class I in 2 patients. There were10 patients with a history of prior mitral valve procedure, among whom 6 had undergone Closed Mitral Commissurotomy and 4 had undergone PTMC. 20 of the subjects (80%) were found to be in sinus rhythm while 5 (20%) were in atrial fibrillation. All patients were subjected to a detailed echocardiography and right heart catheterization prior to PTMC. Pulmonary artery capacitance, as calculated by echocardiography was $\leq 3 mL^3$/mmHg in 80% of the population and > 3 mL^3/mmHg in the remaining 20% of the population.

Pulmonary artery capacitance by echocardiography and its relation to baseline characteristics

Pre-PTMC PAC was correlated with patients' baseline characteristics. For analysis, patients were grouped, according to baseline PAC into PAC $\leq$ 3 mL^3/mmHg (Group I) and PAC > 3 mL^3/mmHg (Group II). No significant difference in PAC was noted between patients with age $\leq$ 40 years and more than 40 years (p = 1.0000). Among the 20 patients in Group I, 15 were in NYHA functional class $\leq$ II and 5 were in NYHA functional class > II, whereas in Group II, 4 patients were in functional class $\leq$ II and 1 patient was in functional class > II. Although, there was a trend towards better functional class in patients in Group 1,it was not statistically significant (p value = 1.0000). This is shown in Table 1.

Table 1: Comparison of PAC by Echocardiography with various parameters

PARAMETERS	Group I PAC < 3 (mL3/mm Hg)	Group II PAC >3 (mL3/mmHg)	P value
Age (no. of patients) ≤40 yrs > 40 yrs	14 6	3 2	1.0000
NYHA Class (N) ≤II > II	15 5	4 1	1.0000
Rhythm (N) Sinus rhythm Atrial fibrillation	15 5	5 0	0.5440
Mean MV gradient (mmHg)[a]	12.47 + 7.20	9.74 + 3.04	0.6870
Mitral valve area (cm^2)[a]	0.871 + 0.27	0.812 + 0.26	0.6570
Mean PA pressure (mmHg)[a]	35 + 11.90	36.6 + 4.72	0.7509
Absolute increase in MVA (cm^2)[a]	0.823 + 0.26	0.88 + 0.11	0.5492
Absolute decrease in mean MV gradient (mmHg)[a]	8.56 + 6.66	5.5 + 2.63	0.0920
Absolute decrease in mean PA pressure (mmHg)[a]	9.69 + 8.18	11.2 + 4.66	0.4106
Successful PTMC (N)	15	3	1.0000
Unsuccessful PTMC (N)	6	1	

[a] - mean $\pm$ SD; N - Frequency

Mean transmitral gradient was 12.47 $\pm$ 7.20 mmHg in Group I compared to 9.74 $\pm$ 3.04 mmHg in Group II. Among 10 patients with pre-PTMC transvalvular gradient between 5-10 mmHg, 80% had PAC $\leq$ 3 mL^3/mmHg and among 15 patients with gradient > 10mmHg, 80 % (12 patients) had PAC $\leq$ 3 mL^3/mmHg. The difference was not significant (p = 0.687), implying that transmitral gradient does not influence pre-PTMC PAC.

Pre-PTMC mitral valve area was compared between the 2 groups. Mean mitral valve area in Group I was 0.871$\pm$0.27 cm^2 and 0.82$\pm$0.26 cm^2 in Group II . Among 19 patients with mitral valve area $\leq$ 1 cm^2, 78.9% had PAC $\leq$ 3mL^3/mmHg and among 6 patients with mitral valve area > 1 cm^2, 83.3 % had PAC $\leq$ 3 mL^3/mmHg. Again, it was not statistically significant (p = 0.657), indicating that the severity of mitral stenosis, or high transmitral gradient does not influence pre-PTMC PAC.

The pre-PTMC pulmonary artery mean pressure was 35 $\pm$ 11.90 mm Hg in Group I patients and 36.6 $\pm$ 4.72 mm Hg in Group II patients.Among 25 patients, 16 patients had pre - PTMC pulmonary artery systolic pressure > 40mm Hg. Among them, 87.5 % had PAC $\leq$ 3 mL^3/mmHg compared to 66.7 % in patients with pre - PTMC pulmonary artery systolic pressure $\leq$ 40 mmHg, which was not statistically significant (p = 0.230). After successful PTMC, all patients underwent right heart catheterization, immediately after which a detailed echocardiogram was performed.

Absolute change in PAC by echocardiography and its relation to baseline characteristics

Patients with absolute increase in PAC (ΔPAC) from baseline were categorized into 2 groups, Group A with ΔPAC ≤ 2 mL^3/mmHg (n=14) and Group B with ΔPAC > 2 mL^3/mmHg (n=11).

Influence of age on absolute increase in PAC was analyzed. Group A had 10 patients and Group B had 7 patients with age ≤ 40 yrs (p value = 1.0000). Group A had 11 patients and group B had 8 patients with NYHA functional class ≤ II (p value = 1.0000), implying that absolute increase in PAC is not influenced by patient's age or baseline functional class.

Pre-PTMC mean transmitral gradient, mitral valve area and mean pulmonary artery pressure were 12.5 ± 6.63 mm Hg , 0.84 ± 0.27 cm^2 , 37 ± 9.18 mm Hg in Group A and 12.27 ±7.71 mm Hg , 0.89 ± 0.26 cm^2, 33.18 ± 12.66 mm Hg in Group B respectively and the difference was not significant (p value = 0.6560, 0.9967 and 0.3108 respectively). Pre-PTMC mean transmitral gradient, mitral valve area and mean pulmonary artery pressure do not therefore influence absolute increase in PAC. This is shown in Table 2.

Table 2: Comparison of ΔPAC by Echocardiography with various parameters

Parameters	**Group A ΔPAC < 2 (mL3/ mmHg)**	**Group B ΔPAC > 2 (mL3/ mmHg)**	**P value**
Age (N) ≤40 yrs > 40 yrs	 10 4	 7 4	1.0000
NYHA Class (N) ≤II >II	 11 3	 8 3	1.0000
Rhythm (N) Sinus rhythm Atrial fibrillation	 10 4	 10 1	0.3406
Mean MV gradient (mmHg)[a]	12.5 + 6.63	12.27 + 7.71	0.6560
Mitral valve area (cm^2)[a]	0.84 + 0.27	0.89 + 0.26	0.9967
Mean PA pressure (mmHg)[a]	37 + 9.18	33.18 + 12.66	0.3108
Absolute increase in MVA (cm^2)[a]	0.85 + 0.44	0.81 + 0.45	0.4910
Absolute decrease in mean MV gradient (mmHg)[a]	7.77 + 5.18	8.1 + 7.36	0.6819
Absolute decrease in mean PA pressure (mmHg)[a]	7.46 + 4.55	14.29 + 9.05	0.3135
Successful PTMC (N)	11	7	0.6564
Unsuccessful PTMC (N)	3	4	

[a] - mean ± SD; N - Frequency

Absolute increase in PAC was correlated with absolute increase in mitral valve area and absolute decrease in mean gradient and mean pulmonary artery pressure. Group A had an absolute increase in mitral valve area of 0.85 ± 0.44 cm^2 and Group B had 0.81 ± 0.45 cm^2 and the correlation was not statistically significant (p value = 0.4910). Absolute decrease in mean mitral valve gradient and pulmonary artery mean pressure was 7.77 ± 5.18 mm Hg and 7.46 ± 4.55 mm Hg in Group A compared to 8.1± 7.36 mm Hg and 14.29 ± 9.05 mm Hg in Group B. Although the absolute decrease in mitral valve gradient and mean pulmonary artery pressure appeared to be associated with absolute increase in PAC, there was no statistical significance. (p value 0.6819 and 0.3135 respectively).

Successful PTMC was defined as

1) Final valve area larger than 1.5 cm^2 and an increase in valve area of at least 25% and
2) Final valve area larger than 1.5 cm^2 without worsening of mitral regurgitation to greater than 2/4[17].

Successful PTMC was correlated with baseline PAC and absolute increase in PAC. Among 25 patients, the procedure was successful in 18 patients and unsuccessful in 7 patients. Out of 18 patients in the successful PTMC group, 15 patients had baseline PAC ≤ 3 mL^3/mmHg as against 6 patients in the unsuccessful group which was not statistically significant (p value = 1.0000), suggesting that baseline PAC did not influence the success of the procedure. Absolute increase in PAC was > 2 mL^3/mmHg in 7 patients in the successful group and in 4 patients in the unsuccessful group and again was not statistically significant (p value = 0.6564), implying that ΔPAC also did not influence the success of the procedure.

Comparison of PAC by Catheterization with baseline parameters

All 25 patients underwent right heart catheterization before and after PTMC and PAC was calculated. Mean PAC before PTMC was 1.7 ± 0.96 mL^3/mmHg and it was compared with the expected mean PAC in the general population (taking normal pulmonary artery pressure and RV stroke volume, PAC = 3.5). This was statistically significant (p value < 0.001), establishing that PAC is less in patients with mitral stenosis compared with the general population. Pre-PTMC PAC was compared with baseline NYHA functional class. 2 patients were in functional class I, 17 in class II and 6 in class III with mean PAC 1.91 ± 1.53 mL^3/ mmHg, 1.78 ± 0.97 mL^3/mmHg and 1.69 ± 0.977 mL^3/mmHg respectively . The difference was not statistically significant (p value = 0.959). Pre-PTMC PAC was compared with baseline mitral valve area and mitral valve mean gradient. Mean PAC in patients with mitral valve area ≤ 1 cm^2 (n=16) was 1.8236 ± 1.14 mL^3/mmHg and with mitral valve area 1-1.5 cm^2 (n=9) was 1.68 ± 0.549 mL^3/mmHg and it was not statistically significant (p = 0.739). This is shown in Table 3.

Table 3: Comparison of PAC by Catheterization with baseline parameters

Baseline Parameters	**Number of Patients**	**PAC (mL^3/mmHg)[a]**	**P value**
NYHA Class I II III	 2 17 6	1.91 + 1.53 1.78 + 0.97 1.77 + 0.96	0.959
Mean MV gradient 5-10mmHg >10mmHg	10 15	1.69 + 0.76 1.82 + 1.09	0.747
Mitral valve area(cm2) ≤1 >1	16 9	1.82 + 1.14 1.68 + 0.54	0.739

[a] - mean ± SD

Patients with mitral valve mean gradient of 5-10 mmHg (n=10) had a mean PAC of 1.69 ± 0.76 mL^3/mmHg and with gradient > 10 mmHg had a mean PAC of 1.82 ± 1.09 mL^3/mmHg which was not statistically significant (p value = 0.747), implying that baseline mitral valve area or mitral valve mean gradient did not influence pre-PTMC PAC. Linear regression analysis was used to analyze the influence of age, sex, NYHA functional class, mitral valve area and mitral valve mean gradient on PAC. There was no correlation with these variables and the total variation explained by these variables was only 18.5% (R square = 0.185).

Comparison between PAC by Echocardiography and Catheterization

Pre- and post- PTMC PAC by echocardiography was compared with PAC by catheterization. Pre-PTMC PAC by echocardiography was 2.925 ± 1.51 mL^3/mmHg and that by catheterization was 1.774 ± 0.96 mL^3/mmHg (p value = 0.0046). Post-PTMC PAC by echocardiography was 4.887 ± 2.84 mL^3/mmHg and that by catheterization was 2.080 ± 0.82 mL^3/mmHg (p value < 0.0001). There was a statistically significant difference between PAC measured by echocardiography and catheterization. This is shown in Table 4.

Table 4 : Comparison between PAC by Echocardiography and Catheterization

PAC (mL^3/mmHg)	**Echocardiography**	**Catheterization**	**P value**
Pre-PTMC PAC[a]	2.925 + 1.51	1.774 + 0.96	0.0046
Post-PTMC PAC[a]	4.887 + 2.84	2.080 + 0.82	<0.0001

[a] - mean ± SD

Comparison of PAC before and after PTMC

Pre-PTMC PAC by echocardiography was 2.925 ± 1.51mL^3/mmHg and it increased to 4.887 ± 2.84 mL^3/mmHg after PTMC which was statistically significant (p value < 0.0001). However, values measured by catheterization were 1.774 ± 0.96 mL^3/mmHg and 2.080 ± 0.82 mL^3/mmHg which was not statistically significant (p = 0.2234), shown in Table 5.

Table 5: Comparison of PAC before and after PTMC

Method	Pre-PTMC PAC (mL^3/mmHg)[a]	Post-PTMC PAC (mL^3/mmHg)[a]	P value
Echocardiography	2.925 + 1.51	4.887 + 2.84	<0.0001
Catheterization	1.774 + 0.96	2.080 + 0.82	0.2234

[a] - mean ± SD

DISCUSSION

The progressive improvement in pulmonary vascular resistance as well as the underlying mechanisms of regression in pulmonary vascular resistance and pulmonary arterial pressure following PTMC have been studied before[18]. It has been demonstrated that pulmonary vascular resistance continues to decline in the weeks after a successful balloon mitral valvuloplasty resulting in progressive improvement of pulmonary arterial pressure in these patients. Pulmonary arterial capacitance, the change in volume in relation to change in pressure, is associated with right ventricular workload[19]. Pulmonary arterial capacitance reflects the ability of the pulmonary vessel to dilate during systole and recoil during diastole. By storing blood during systole, a high capacitance blood vessel dampens pulmonary artery systolic pressure[20]. The primary factor encountered by the right ventricle during ejection is the distensibility of the large and medium sized pulmonary arteries which can be assessed with PAC.

Twenty five patients fulfilling the inclusion criteria were selected as subjects of this study. There were more females as compared to males, as rheumatic mitral valve disease is known to be more common in females. Baseline pulmonary artery capacitance estimated before PTMC was not significantly influenced by patients' age, baseline functional status, rhythm, mitral valve area, mean gradient across mitral valve, pulmonary artery systolic pressure or the mean pulmonary artery pressure.

Along with the improvement in the mitral valve area after successful PTMC, the increase in PAC was not dependent upon patient's age, baseline functional class, baseline mitral valve gradient, mitral valve area, pulmonary artery systolic pressure or pulmonary artery mean pressure. Non invasive assessment of pulmonary hemodynamics by echocardiography has previously been studied with regard to pulmonary artery pressures and PVR in patients with idiopathic pulmonary artery hypertension[20], but this is the first instance of a study attempting to assess PAC in rheumatic mitral stenosis.

The course of changes in pulmonary vascular resistance and pulmonary artery hypertension and mechanisms of their regression after PTMC have also been studied by Vishwa Dev et al[18]. Their data pertained to 53 patients (19 men and 34 women ranging in age from 11 to 35 years). In 24 of these patients, repeat cardiac catheterization was performed at 1 week. Nineteen patients were restudied at 3 to 12 months after the procedure. Immediately after the procedure, PA pressure fell from 45 ± 13.4 to 30.1 ± 12.9 mm Hg (p < 0.001) and PA wedge pressure decreased from 29.0 ± 5.4 to 12.4 ± 4.1 mm Hg (p < 0.001). Pulmonary vascular resistance did not change significantly (7.3 ± 7.1 Wood U before and 5.8 ± 5.0 Wood U after PTMC, the difference was not significant).

In this study, all 25 patients underwent immediate post procedure cardiac catheterization. Pre-PTMC PA mean pressure was 35.32 ± 10.78 mm Hg which was reduced to 29.20 ± 10.108 mm Hg and was not statistically significant. Pre-PTMC PVR was 3.69 ± 3.03 Wood U, after PTMC it was 3.656 ± 1.864 Wood U which was not statistically significant. Pre- PTMC PAC calculated by catheterization was 1.7739 ± 0.963 mL^3/mmHg and post-PTMC, it increased to 2.0794 ± 0.820 mL^3/mmHg which was not statistically significant (p value = 0.2234). In our study there was no significant decrease in PVR immediately after PTMC and there was a trend towards increase in PAC after PTMC which was not statistically significant. Mark K Friedberg and Jeffery A studied PAC in 31 children with pulmonary hypertension using echocardiography and compared the results with those obtained at catheterization[15]. PAC derived from echocardiography was similar to that derived from catheterization. (Mean ± SD 1.16 ± 1.0 vs 1.10 ± 0.95 ml^3/ mm Hg, p = not significant) and the two correlated well (r = 0.74, p < 0.0001). There was a highly significant polynominal relationship between PAC and RV anterior wall thickness indexed to body surface area (p < 0.0001) but not between pulmonary vascular resistance and RV wall thickness. In our series, pre- and post-PTMC PAC derived by echocardiography was 2.925 ± 1.51mL^3/mmHg and 4.88 ± 2.8 mL^3/mmHg and by catheterization was1.774± 0.963 and 2.080 ± 0.820 mL^3/mmHg and there was no correlation between the two.

In this study, there was no significant relationship between PAC or absolute change in PAC after PTMC with patients' age, functional status, mitral valve area, mitral valve gradient and pulmonary artery pressure.

LIMITATIONS OF THE STUDY

Our study recruited only 25 patients, which is small and the results cannot be generalized to the whole population. Moreover, 20% of the patients were having atrial fibrillation. To avoid erroneous interpretation, 5 readings were carried out at different cycles and the average of values obtained was taken for analysis. Technically, the pulmonary artery and pulmonary valve need to be clearly visualized for precise measurements. Generally, the largest dimension was used as it most likely represented the true diameter while the smaller measurements may have represented tangential cuts through the circular outflow tract. In addition, there must be an adequate jet of tricuspid regurgitation present to determine pulmonary artery systolic pressure. This was resolved by measuring PAC by cardiac catheterization pre- and post- PTMC and it was taken for final analysis.

CONCLUSION

The study could not establish any significant relationship between PAC and patients' baseline characteristics including age, functional status, mitral valve area, mitral valve gradient and pulmonary artery pressure. There was no significant relationship between the absolute change in PAC after PTMC with patient's age, functional status, mitral valve area, mitral valve gradient and pulmonary artery pressure. PAC is less in patients with mitral stenosis compared with the general population. There was a statistically significant difference between PAC measured by echocardiography and by catheterization. No significant increase in PAC was recorded by catheterization immediately following PTMC.

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