Reversal of abnormal cardiac parameters following mitral valve replacement for severe mitral stenosis in relation to pulmonary artery pressure: A retrospective study of noninvasive parameters – Early and late pattern

USHA T. PARVATHY*, RAJESH RAJAN, ALEXANDER GEORGEVICH FAYBUSHEVICH

MPS Hospital, Department of Cardiac Surgery, Peoples’ Friendship University of Russia, Moscow, Russian Federation

*Corresponding author: Dr. Usha T. Parvathy; MPS Hospital, Department of Cardiac Surgery, Peoples’ Friendship University of Russia, Moscow, Russian Federation; Phone: +91 984 7900278; Fax: +91 471 2306193; E-mail: tushaparvathy@gmail.com

(Received: June 28, 2015; Revised manuscript received: April 11, 2016; Accepted: May 11, 2016)

Abstract: Background and objectives: Although the regression of pulmonary hypertension (PH) in mitral stenosis (MS) has been studied over varying periods postintervention, corresponding studies on the cardiac chamber alterations after surgery are very limited. We sought to determine the degree of reversal of these and the clinical status in connection with that of pulmonary artery pressures (PAPs) in the early and late postoperative periods. Methods: The preoperative, early, and 1-year postoperative data – functional class (FC), cardiothoracic ratio (CTR) in chest X-ray (CXR), and echocardiographically left atrium (LA), right atrium (RA), right ventricle (RV), left ventricle (LV), and pulmonary artery (PA) dimensions, PAP, tricuspid regurgitation (TR) – of 50 patients who had mitral valve replacement (MVR) for MS with PH were retrospectively analyzed for correlations with PAP (Pearson’s), and their change (t-test), in relation to that in PAP. PH group-based [Group (Gp)-I PAP ≤ 60 mmHg, Gp-II PAP > 60 mmHg] analysis highlighted the differences. Results: All parameters significantly correlated with the baseline PAP (p < 0.05), except LA (r = 0.081, p = 0.577). Postoperatively, there was significant reduction in all parameters (p < 0.001) and increase in LV (p < 0.003). The PAP regression was 39.42%; with the decrease in CTR, LA, and RA related to it, the early changes being significant (p < 0.01). The RV and PA showed lesser reduction (8.61% and 9.42%), late reduction being more conspicuous. The changes were greater and significant in Gp-II (especially PAP, RV, and PA). At 1 year, PAP normalized in only 19 (38%). Residual PH and chamber enlargement prevailed more in Gp-II. Conclusions: This study emphasizes the importance of the baseline PAP in MS to which was proportionate the functional disability and the cardiac chamber alterations (except LA). Their postoperative improvement accompanying the PAP regression differed in degree and time frame relative to PAP. The higher pressure group showed greater regression, but greater prevalence of residual abnormalities, suggesting that the pathologic changes in them might take longer to resolve, necessitating further evaluation.

Keywords: mitral valve replacement, mitral stenosis, pulmonary hypertension, cardiac chamber alterations, functional class

Introduction

Pulmonary hypertension (PH), an invariable accompaniment of mitral stenosis (MS), varies in incidence reported in different series [1, 2]. Its negative impact in terms of higher operative mortality and morbidity, the brunt of chronic PH leading to right ventricle (RV) failure, and the prognostic implications are well known. However, PH is documented to regress after surgery, the extent of which varies in individuals and over varying postoperative intervals [2–4]. Though factors determining normalization of PH have been studied, enough attention has not been given to the correlation between PH regression and change in other noninvasive cardiac parameters. This paper is centered on this aspect of degree of pulmonary artery pressures (PAPs) change at discharge, and 1-year postmitral valve replacement (MVR) in relation to clinical disability and cardiac chamber changes, and has

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attempted to emphasize the difference in relation to the PH grades.

**Methods**

*Design: retrospective analysis*  
*Data source – medical case – records and follow-up charts*

Fifty patients, who had MVR for isolated/predominant significant MS, in our institute formed the study group, whose pre- and postsurgical data were analyzed. They were evaluated clinically – symptom duration, functional class (FC), New York Heart Association (NYHA); chest X-ray (CXR) for cardiothoracic ratio (CTR), electrocardiography for rhythm, RV hypertrophy; echocardiography for severity of MS, PAP, tricuspid regurgitation (TR), chamber sizes; and cardiac catheterization to rule out coronary artery disease (CAD) in a few. Patients with ventricular dysfunction, significant mitral regurgitation, multivalvar disease, organic TR, CAD, and with associated procedures were excluded.

Standard transthoracic echocardiography techniques and criteria were used for the cardiac chamber dimensions [1, 5, 6]. The pulmonary artery (PA) dimension was measured in the parasternal short-axis view. Linear RV dimensions included the end-diastolic diameter (RVEDD) measured from the septum to the free RV wall at mid-cavity levels. The TR severity determined by the ratio of TR jet area to the right atrium (RA) area; systolic PAP was measured from TR jet velocity based on Bernoulli’s equation [1, 5, 6].

*Surgical details*

All these patients had undergone MVR with mechanical prosthesis under standard cardiopulmonary bypass (CPB) with cardioplectic arrest and hypothermia. The indications for MVR were based on severe to moderate subvalvar pathology, immobility, calcified valve, left atrium (LA) thrombus (despite anticoagulation), and atrial fibrillation (AF), besides severity of stenosis. (The symptom duration being subjective, and patient-dependent, may not always be exact or reliable.) Eight patients (16%) with severe TR and annular dilatation as evaluated at surgery had tricuspid annuloplasty (TAP) in the same operative setting: DeVeGea-3, modified DeVeGea-3, and ring annuloplasty-2. Patients were assessed clinically (FC, diuretic, and vasodilator requirement) and with CXR and echocardiogram on regular follow-up.

Emphasis was given to the following specific parameters presurgery and 1-year postsurgery: FC, CTR, and echocardiographically all chambers and PA dimensions, PAP, and TR grades. At discharge, echocardiographic parameters were also considered. Their changes were assessed in relation to preoperative PH degree and the change in the PAP.

Patients were classified into groups based on PAP: [Group (Gp)-I <60 mmHg and Gp-II >60 mmHg] for assessing the results. Chamber sizes were graded as normal, mild, moderate, and severe enlargements. Significant TR regression required at least one-grade reduction [5].

**Statistical analysis**

Data analysis used SPSS-PASW-18. Categorical variables were expressed as number and percentages, the continuous variables as mean ± SD in case of normal distribution and as median in the groups. Inferential analysis included: Pearson’s test correlating parameters to PAP, paired t-test for pre- to postoperative comparisons, and Mann–Whitney and χ2 tests for intergroup comparison (demographics, clinical, and hemodynamics). The percentage change in each parameter was calculated and assessed in relation to change in PAP and compared between the groups. A p < 0.05 was considered to be significant.

**Results**

**Baseline characteristics**

**Demographics**

The patients ranged in age from 19 to 53 years (mean 36 ± 8.9), with a female preponderance (M:F, 7:43). The symptom duration varied from 3 months to 20 years (6.31 ± 5.59 years). The FC distribution was 38:12 in III:IV.

**Hemodynamic data**

CTR ranged from a near-normal value of 42.43 to a maximum of 82% (mean 57.75 ± 8.92). Thus, 9 had normal CTR (<50%), 19 mild cardiomegalias (50–59%), 16 moderate (60–70%), and 6 severe cardiomegalias (>70%). PAP-based classification of patients placed 29 (58%) under Gp-I and 21 (42%) under Gp-II. All patients except one had considerable LA enlargement, the size ranging from 3.2 to 8.6 cm (mean 5.8 ± 1.22); 12 had giant LA (>6.5 cm). The RA varied from 2.3 to 6.5 cm (mean 4.02), RV 2.3 to 5.25 (3.27), PA 2.1 to 4.1 (3.04), and LV 3.0 to 5.2 (4.26). RV dilatation (>3.0 cm) was seen in 27 (52%), which was mild (3.1–3.5 cm) – 14, moderate (3.6–4 cm) – 6, and significant/severe (>4) – 7. Normal PA (<2.3 cm) was seen in 2; others (96%) had dilatations: mild (2.3–2.6 cm) – 5, moderate (2.7–3.0 cm) – 19, and severe (>3.0 cm) – 24. TR varied from trivial in 2, mild in 16, moderate in 22 to severe in 10. In these, annular dilatation was frequently found. The mitral valve orifice area quantifying MS severity ranged from 0.5 to 1.4 cm² (0.93 ± 0.21). Twenty-two (44%) had sinus rhythm and 28 (56%) AF. Apart from digitals and oral penicillin, the salient medications included diuretics in the majority, antiarrhythmics in 5, anticoagulants in 11,
and antiarrhythmics and anticoagulants in 6. Comorbid conditions were: diabetes mellitus – 3, hypertension – 2, neurological disorders – 13 (cerebrovascular accident – 4, transient ischemic attacks – 5, and seizures – 4), and mild renal dysfunction – 3.

Table I demonstrates statistically significant relation of PAP with the clinical status and all hemodynamic parameters (p < 0.05) preoperatively except the LA size and postoperatively except the FC and LA size.

Postoperative
The overall assessment by comparison of means (Table II) shows a statistically significant reduction in all parameters (CTR, PAP, and chamber sizes; p < 0.01), an increase in LV dimension and an improvement in FC and TR grade (Table III, Figs 1 and 2) postoperatively. The parameters differ in their degree of regression (seen by percentage change – Table IV), LA and RA showing greater parallelism.

The early changes (assessed at discharge) were significant in all, specifically for PAP, LA, and RA. The late changes were conspicuous for RV, PA, and LV (Tables II and IV).

All patients were in FC III or IV preoperatively and showed improvement to one or two lower class (I, II, and III) (mean 3.24 ± 0.43 to 2.02 ± 0.47, p = 0.000), except three who continued to remain in Class III at 1-year postsurgery (change displayed in Table III, Fig. 1).

### Table I

| Parameters | Preoperative | Postoperative at-discharge | p1 | Postoperative 1 year | p2 | p3 |
|------------|--------------|----------------------------|----|----------------------|----|----|
| CTR (%)    | 57.76 ± 8.92 | 48.63 ± 6.29               |    |                      |    |    |
| LA (cm)    | 5.86 ± 1.22  | 4.69 ± 0.91                | 0.000 | 4.53 ± 0.84 | 0.022 | 0.000 |
| RA (cm)    | 4.02 ± 0.84  | 3.68 ± 0.58                | 0.000 | 3.52 ± 0.52 | 0.001 | 0.000 |
| RV (cm)    | 3.27 ± 0.61  | 3.12 ± 0.41                | 0.022 | 2.99 ± 0.40 | 0.001 | 0.000 |
| PA (cm)    | 3.04 ± 0.40  | 2.94 ± 0.41                | 0.090 | 2.76 ± 0.36 | 0.000 | 0.000 |
| LV(IDD) (cm) | 4.26 ± 0.50 | 4.33 ± 0.56                | 0.293 | 4.47 ± 0.42 | 0.044 | 0.003 |
| SPAP (mmHg) | 65.32 ± 24.06 | 42.34 ± 9.46               | 0.000 | 39.57 ± 7.65 | 0.000 | 0.000 |

CTR = cardiothoracic ratio, LA = left atrium, RA = right atrium, RV = right ventricle, PA = pulmonary artery, LV(IDD) = left ventricular internal diameter-diastole, SPAP = systolic pulmonary artery pressure, p1 = early change – pre-op to at-discharge, p2 = late change – at-discharge to 1-year post-op, p3 = overall change – pre-op to one-year post-op

### Table III

| FC | Preoperative | Postoperative 1-year | TR | Postoperative At-discharge | Postoperative 1-year |
|----|--------------|----------------------|----|----------------------------|----------------------|
| I  | 0 (0%)       | 5 (10%)              | Nil | 0 (0%)                     | 1 (2%)               |
| II | 0 (0%)       | 39 (78%)             | Trivial | 2 (4%)                     | 10 (20%)             |
| III| 38 (76%)     | 6 (12%)              | Mild | 16 (32%)                    | 25 (46%)             |
| IV | 12 (24%)     | 0 (0%)               | Moderate | 22 (44%)                   | 13 (26%)             |
|    |              |                      | Severe | 10 (20%)                   | 0 (0%)               |

Table I demonstrates statistically significant relation of PAP with the clinical status and all hemodynamic parameters (p < 0.05) preoperatively except the LA size and postoperatively except the FC and LA size.
CTR reduced in 47 (94%) patients, bringing down the range from 38.31 to 61, with marked reduction in 9 and mild to moderate reduction in the rest. Two had mild increase (2–3%), whereas one had no change. The overall percentage reduction (15.80%) corresponded to only <50% of the change in PAP. Moreover, there was not any difference between the PH groups. The values in the normal, mild, moderate, and severe categories improved from 9, 19, 16, 6 to 30, 15, 5, 0.

Initially, the systolic PAP (noninvasively measured) varied from 33 to 144 mmHg, with significant reduction from 30 to 66 mmHg ($p = 0.000$), then further slow reduction to 30–59 mmHg ($p = 0.000$); an overall mean difference of 25.74 equaled a percentage reduction of 39.42%. Ninety-eight percent of the patients showed regression, the maximum reduction being 70.83%. The change was definitely more in the higher pressure Gp-II. At 1-year postoperative, only 19 (38%) patients had PAP normalized to the range of 30–35 mmHg, another 14 (28%) in the near-normal range (35–40 mmHg), whereas the remaining 17 (34%) had residual PH varying from 41 to 59 mmHg (Gp-I: 3; Gp-II: 14). Many of these had residual RV and PA dilatation.

LA regressed to a mean 4.53 cm (range 3.0–8.0), the early change striking. Forty-nine patients had reduction, with >30% reduction in 14 of them. The reduction was significant that only 2 had giant LA postoperatively compared with 12 preoperatively, but normalization was not complete.

RA size range was reduced to 2.4–5.07 cm (mean 3.52), reduction seen in 78% of patients by 12.87%. RV regressed in 36 patients (72%) that brought down its size range in the series from 2.3–5.25 cm to the postoperative values of 2.2–4.0 cm. The early phase showed just 50% of the total change, rest of the regression seen later. Seven had significant dilatation (>4 cm), six of whom showed regression to an average of 3.39 cm. Twenty-one patients continued to have dilated RV: 16, mild; 4, moderate; and 1, severe.

PA only 9.42% reduction was seen (range from 2.1–4.1 to 2.0–3.5), the main change being in the late postoperative phase, somewhat similar to RV. Thirty-five patients (70%) showed regression, but normal PA size was attained only in seven. Our interesting finding was that 14 (58.33%) of the 24 patients with severe dilatation

| Group | PAP (%) | CTR (%) | LA (%) | RA (%) | RV (%) | PA (%) | LV(IDD) |
|-------|---------|---------|--------|--------|--------|--------|---------|
| Overall | 39.42 | 15.80 | 22.79 | 12.43 | 8.61 | 9.42 | -4.92 |
| Early | 35.18 | 20.87 | 19.21 | 8.35 | 4.59 | 3.22 | -1.90 |
| Late | 6.54 | 20.87 | 3.49 | 4.44 | 4.20 | 6.40 | -3.00 |
| Group-I | 28.06 | 15.88 | 21.90 | 10.40 | 3.43 | 5.08 | -0.96 |
| Early | 23.89 | 15.88 | 20.87 | 5.51 | -0.88 | 1.74 | -0.68 |
| Late | 5.47 | 15.88 | 1.21 | 5.18 | 4.27 | 3.40 | -0.23 |
| Group-II | 48.56 | 15.71 | 23.96 | 14.82 | 4.14 | 14.66 | -10.83 |
| Early | 44.25 | 15.71 | 18.72 | 11.72 | 10.82 | 5.18 | -3.62 |
| Late | 7.73 | 15.71 | 6.43 | 3.51 | 4.11 | 10.19 | -6.95 |

PAP=pulmonary artery pressure, CTR=cardiothoracic ratio, LA=left atrium, RA=right atrium, RV=right ventricle, PA=pulmonary artery, LV(IDD)=left ventricular internal diameter-diastole.
belonged to the higher pressure Gp-II, with more significant reduction ($p < 0.001$) compared with the milder Gp-I ($p = 0.048$). That a good number, 43 (86%), had persistent postsurgery PA dilatation, despite the majority of them (65.12%) attaining normalized/near-normal PAP, is notable and needs emphasis. Only in the remaining 15 (34.88%) did the residual abnormalities keep parallel.

Improvement in TR grade is detailed in Table III and Fig 2. TR decreased (1–2 grades) in 36 (72%) patients (mean from $2.8 \pm 0.81$ to $2.08 \pm 0.78$ ($p = 0.000$) immediately, then to $1.9 \pm 0.93$ ($p = 0.172$), the early reduction being significant. A significant decrease from severe to mild was seen in 3 and moderate to trivial in 5.

Analyzing those with moderate and severe TR separately, eight with severe TR had tricuspid repair (TAP). Six had severe PAH and two moderate (mean 91.5 ± 34.6), the mean chamber and PA sizes larger than others. Their percentage change of parameters was greater compared with overall and those without TAP in the series. The most striking was the RV regression (16.6% – double that of other groups) with considerable early change. TR regression occurred only early with no further change (the corresponding mean TR grades being 4, 2.63, and 2.63, respectively).

The LV size showed 4.92% increase in 32 (64%) (mean 4.26–4.47 cm), mainly a late phase change. Fifteen had a decrease, and three had no change.

Analysis on PH-based groups (Table V) showed that demographically, there was no significant difference between the two; Gp-II had comparatively more patients in Class IV and higher TR grades. CTR, LA, RA, RV, and PA sizes had relatively higher values in Gp-II preoperatively, and postsurgery this relation was maintained (Tables V and VI). The postoperative change was significant in Gp-II for all parameters ($p < 0.01$), whereas in Gp-I, it was significant only for CTR, PAP, LA, and RA ($p < 0.01$) as shown by the comparison in Table VI. The comparative change in FC and TR is shown in Figs 3 and 4. The average FC improved from 3.1 ± 0.31 to 1.89 ± 0.41 ($p = 0.000$) in Gp-I, 3.43 ± 0.51 to 2.19 ± 0.51 ($p = 0.000$) in Gp-II; TR from 2.48 ± 0.74 to 1.59 ± 0.98 ($p = 0.000$) in Gp-I, 3.24 ± 0.70 to 2.33 ± 0.66 ($p = 0.000$) in Gp-II. By percentage change, intergroup difference was not significant for CTR, LA, and RA, whereas RV, PA, and LV showed definitely greater change in Gp-II (Table IV). The percentages (of patients) with residual PH and chamber enlargements were as: Gp-I:Gp-II: PAP > 40 (10.34:66.66), CTR > 50 (3.44:23.81), LA (79.31:66.67), RA (20.69:66.67), RV (20.69:66.67), LA (79.31:66.67), RA (3.44:23.81), RV (42.86:57.14), and PA (86.21:85.71).

In our series, slight deviations from the above findings were noted in a few (Table VII); mild increase in CTR (2–3.5%) in 2 belonging to Gp-I, mild increase in LA (5.13%) in 1 (Gp-I), RA in 8 (GpI – 6, Gp-II – 2), RV in 9 (Gp-I – 8, Gp-II – 1), PA in 11 (Gp-I – 10, Gp-II – 1), and TR in 2 (Gp-I), the reasons being unexplainable.

Discussion

Investigators have commented on the regression pattern of PH following the relief of MS at varying postoperative periods; hours to days, months, and years [2, 3, 7, 8]; some stating that decrease of PAP and resistance occurs during the first few hours postprocedure with no further decline [2, 9], others showing a progressive decline throughout the subsequent weeks or months [4, 6, 8]. Few authors in the recent past have concentrated on factors, such as age, PAP, LA pressure (LAP), and severity of MS as determining the PAP reduction, with their experience in balloon mitral valvotomy (BMV) [1, 10, 11].

Cardiac chamber changes in MS are known: the major effect being on the LA, which often dilates, and later on hypertrophies. With chronicity and increased PAP, RV takes the burden followed by the tricuspid valve and RA. A reversal of these is expected postintervention; the extent of resolution/nonresolution may vary. The postoperative assessment of MS has been more difficult than the presurgical, as there appears in the literature some perplexity relating to the existing radiologic signs. Even Seningen’s extensive roentgenologic studies found that those changes most useful in evaluating preoperative severity were not that useful for postoperative hemodynamic assessment [12]. Hence, it would be interesting to assess their changes and their reversal postsurgery in connection with that of PAP, a less attended aspect in the surgical setting. The present analysis conducted in this context has shown certain important findings.

That all parameters bear relation to PAP in our correlation table, while not the LA size supports the concept that LA is often disproportionate to PAP. The variations in the degree and time frame of their postsurgical improvement related to PAP catches attention.

The preoperative relations implicate the influence of PH on the clinical status, but despite significant postsurgical improvement as in many studies, the postoperative disproportionalism stood unclear. Mild, moderate residual, PH and TR accounted for nonimprovement in two.

The roentgenological cardiac enlargement in MS, often accompanying PH, may even denote its height. Though its postoperative reduction attributed to the reduction in the LA and RV sizes, while not of PA, has been noted in the earlier studies of Kaul et al. [8], its exact correlation with PH reduction is not well documented. We did not measure chamber sizes on CXR but attempted correlating with that on echo. The roentgenographic reduction, much less than the PAP change, appeared mainly by LA-size reduction, while that of RV and PA were not corresponding significantly. With LA
Table V

Comparison of demographics and hemodynamic parameters between PH-based groups [categorical-variables: no (percentage); continuous-variables: median, with mean-ranking below]

| Parameters                  | Group-I PAP <60 mmHg n = 29 | Group-II PAP >60 mmHg n = 21 | Mann–Whitney U | p-value |
|-----------------------------|------------------------------|-----------------------------|----------------|---------|
| 1. Clinical                 |                              |                             |                |         |
| Age (years)                 | 37                           | 39                          | 278.0          | 0.600   |
| 24.59                       | 26.76                        |                             |                |         |
| Symptom-duration (years)    | 7                            | 5                           | 291.5          | 0.797   |
| 25.05                       | 26.12                        |                             |                |         |
| Sex (M/F)                   | 5:24                         | 2:19                        | NS             |         |
| Comorbid conditions         | 10 (34.48%)                  | 10 (47.62%)                 | 0.349          |         |
| Sinus rhythm/atrial fibrillation | 14/15                      | 8/13                        | 0.474          |         |
| Medications                 |                              |                             | 0.128          |         |
| Diuretics                   | 26 (89.65%)                  | 21 (100%)                   |                |         |
| Antiarrhythmics             | 2                            | 3                           |                |         |
| Anticoagulants              | 5                            | 6                           |                |         |
| Antiarrhythmics + anticoagulants | 3                        | 3                           |                |         |
| FC I                        | 0 (0%)                       | 0 (0%)                      |                |         |
| FC II                       | 0 (0%)                       | 0 (0%)                      |                |         |
| FC III                      | 26 (89.66%)                  | 12 (57.14%)                 |                |         |
| FC IV                       | 3 (10.34%)                   | 9 (42.86%)                  |                |         |
| 2. Hemodynamic              |                              |                             |                |         |
| CTR (%)                     | 55                           | 60                          | 177.50         | 0.012   |
| 21.12                       | 31.55                        |                             |                |         |
| Echocardiogram              |                              |                             |                |         |
| PAP (mmHg)                  | 50                           | 80                          | 0.000          | 0.000   |
| 15.00                       | 40.00                        |                             |                |         |
| LA (cm)                     | 5.6                          | 5.85                        | 294.50         | 0.844   |
| 25.16                       | 25.98                        |                             |                |         |
| RA (cm)                     | 3.6                          | 4.5                         | 160.50         | 0.005   |
| 20.53                       | 32.36                        |                             |                |         |
| RV (cm)                     | 3                            | 3.5                         | 122.50         | 0.000   |
| 19.22                       | 34.17                        |                             |                |         |
| PA (cm)                     | 2.8                          | 3.2                         | 117.50         | 0.000   |
| 19.05                       | 34.40                        |                             |                |         |
| LV(IDD) (cm)                | 4.4                          | 4.16                        | 189.00         | 0.023   |
| 29.48                       | 20.00                        |                             |                |         |
| Mitral valve orifice area (sqcm) | 0.93                     | 0.90                        | 220.00         | 0.093   |
| 28.41                       | 21.48                        |                             |                |         |
| TR                          |                              |                             |                |         |
| Nil                         | 0 (0%)                       | 0 (0%)                      |                |         |
| Trivial                     | 2 (6.896%)                   | 0 (0%)                      |                |         |
| Mild                        | 13 (44.83%)                  | 3 (14.29%)                  |                |         |
| Moderate                    | 12 (41.38%)                  | 10 (47.62%)                 |                |         |
| Severe                      | 2 (6.896%)                   | 8 (38.10%)                  |                |         |

PAP = pulmonary artery pressure, FC = functional class, CTR = cardiothoracic ratio, LA = left atrium, RA = right atrium, RV = right ventricle, PA = pulmonary artery, LV(IDD) = left ventricular internal diameter-diastole, TR = tricuspid regurgitation
Measurement units as Table II
| Parameter | Group-I (preoperative-PAP < 60mmHg) (n = 29) | Group-II (preoperative-PAP > 60mmHg) (n = 21) |
|-----------|--------------------------------|---------------------------------|
|           | Pre-op | At-discharge | 1 year | p-value | Pre-op | At-discharge | 1 year | p-value |
| CTR       | 55.11 ± 7.95 | 46.36 ± 5.44 | 0.000 | 61.41 ± 9.07 | 51.77 ± 6.13 | 0.000 |
| LA        | 5.76 ± 1.17 | 4.55 ± 0.72 ††† | 4.49 ± 0.70 | 0.000 | 6.01 ± 1.30 | 4.88 ± 1.11 ††† | 4.57 ± 1.01 ‡ | 0.000 |
| RA        | 3.75 ± 0.79 | 3.54 ± 0.59 | 3.36 ± 0.48 ‡‡ | 0.002 | 4.39 ± 0.79 | 3.87 ± 0.52 ††† | 3.74 ± 0.51 | 0.000 |
| RV        | 3.00 ± 0.40 | 3.03 ± 0.37 | 2.89 ± 0.37 ‡‡ | 0.086 | 3.65 ± 0.65 | 3.25 ± 0.42 † | 3.12 ± 0.42 ‡ | 0.000 |
| PA        | 2.87 ± 0.38 | 2.82 ± 0.33 | 2.72 ± 0.35 † | 0.048 | 3.28 ± 0.30 | 3.11 ± 0.45 | 2.80 ± 0.36 ‡‡‡ | 0.000 |
| LV(IDD)   | 4.40 ± 0.47 | 4.43 ± 0.47 | 4.44 ± 0.39 | 0.571 | 4.06 ± 0.49 | 4.21 ± 0.65 | 4.50 ± 0.47 ‡ | 0.000 |
| PAP       | 50.21 ± 6.95 | 38.21 ± 6.67 ††† | 36.12 ± 5.70 ‡‡ | 0.000 | 48.05 ± 9.91 ††† | 44.33 ± 7.54 ‡‡‡ | 0.000 |
| FC        |           |               |        |        |           |               |        |        |
| I         | 0 (0%) | 4 (13.79%) |        | 0 (0%) | 1 (47.6%) |        |        |
| II        | 0 (0%) | 24 (82.76%) |        | 0 (0%) | 15 (71.43%) |        |        |
| III       | 26 (89.66%) | 1 (3.45%) |        | 12 (57.14%) | 5 (23.81%) |        |        |
| IV        | 3 (10.34%) | 0 (0%) |        | 9 (42.86%) | 0 (0%) |        |        |
| TR        |           |               |        |        |           |               |        |        |
| Nil       | 0 (0%) | 1 (3.45%) |        | 0 (0%) | 0 (0%) |        |        |
| Trivial   | 2 (6.896%) | 9 (31.03%) |        | 0 (0%) | 1 (4.76%) |        | 2 (9.52%) |        |
| Mild      | 13 (44.83%) | 15 (51.72%) |        | 3 (14.29%) | 8 (38.09%) |        | 10 (47.62%) |        |
| Moderate  | 12 (41.38%) | 4 (13.79%) |        | 10 (47.62%) | 12 (57.14%) |        | 9 (42.85%) |        |
| Severe    | 2 (6.896%) | 0 (0%) |        | 8 (38.10%) | 0 (0%) |        | 0 (0%) |        |

PAP = pulmonary artery pressure, CTR = cardiothoracic ratio, LA = left atrium, RA = right atrium, RV = right ventricle, PA = pulmonary artery, LV(IDD) = left ventricular internal diameter-diastole, FC = functional class, TR = tricuspid regurgitation

Measurement-units as Table II

Each intragroup comparisons
† p < 0.05, †† p < 0.01, ††† p < 0.001 – for early change pre-op vs at-discharge
‡ p < 0.05, ‡‡ p < 0.01 – for late change at-discharge vs 1-year post-op
p-value – for overall change pre-op vs 1-year post-op
size not relating to PAP, and its percentage reduction showing no intergroup difference, the above finding explains the lack of difference in CTR reduction between PH groups. The reduction rate varies as seen in different series [8, 12, 13]; the influence of PAP on the degree of CTR reduction is rather unpredictable.

PH regression in our series was significant (p < 0.001); the substantial early phase drop with atrial sizes displaying a similar trend corroborates the venous element equivalent. The greater change in the higher pressure Gp-II (50% reduction in 8) is consistent with the earlier reports of Foltz et al. [3], but their study was confined to early postoperative period. In our case, further regression (6.5%) occurred over subsequent months probably due to the slow resolution of its reactive component. Similarity was also seen with the 18 months postvalvotomy rates of Noor et al. (29%) [1], and Ghaffari et al. (44.80%) [6], but much less than the rates of Oraby and Mahfouz [5] and Kaul et al. (57.2%) [8]. The differing rates despite similar demographics in these and ours reflect the variability in the disease severity and pulmonary reactivity. With the factor of residual mitral gradient eliminated, the nonresolution of PH in 17 (34%) at 1 year may be due to organic changes in the pulmonary vasculature (part of pulmonary vascular disease) also reflected in the nonresolution of PA and RV. We did not see that significant late reduction noted by Oraby and Mahfouz [5] for the same probability of advanced pathology in some patients.

LA size gains importance as it reflects the disease history (chronicity and severity). Studies analyzing its predictive value on PH regression noted that its decrease post-BMV, though significant [1], did not have impact on PH [1, 14]. But postsurgical correlation lacks detailed study. We noted that LA size though, disproportionate with PAP pre- and postoperatively, showed significant early reduction with a modest parallelism with PH regression, reasoned by the congestive component. However, there was no significant intergroup difference. Despite its percentage reduction (greatest of all changes), contributing to major part of CTR reduction, values at 1 year revealed that it did not completely normalize as 37 patients continued to have LA sizes above the normal range. The percentage regression of LA size and PAP in this series is similar to Ahmed et al.’s report (22.85 and 44.80, respectively) [6], but much less than the reversion rate achieved by Darr et al. [4]. Probably, this may be due to stiff noncompliant LA as most of our patients had advanced disease needing valve replacement. Herein, one should consider measuring atrial/atrioventricular compliance, as LAP and PAP relate more to LA compliance than the size [14, 15]. However, some involution over the ensuing years is expected to occur.

The pre- and postoperative correlations and the significant regression of RA with PAP reflect its functional nature. Prolonged distension, AF, and minor tricuspid lesion (with/without repair) possibly prevent its normal
involvement [13]. This is seen in our striking finding in six patients with persistent large RA > 4.4 cm; all had severe PH preoperatively, with mild to moderate residual PH (37–59 mmHg), and TR persisting.

Besides controversies regarding RV function and changes in MS, and the sophisticated/complex RV assessments, linear RV dimensions (midcavity-RVEDD) on 2D-echo gained importance in assessing the regression pattern [1, 5, 6, 11]. Cherepenin et al. [16] and Ramakrishnan et al. [17] noted that RV size increases with PAP, which was also the case in our pre- and postoperative correlation assessments. Seventy-two percent showed reasonable reduction, fairly close to Darr et al.’s [4] and Ahmed et al.’s series [6]. However, the percentage reduction (8.06), not closely relating to that of PAP, and much less than the rate, 25% shown by Oraby and Mahfouz, [5] and Ghaffari et al. [11], did not reach their level of parallelism or that shown by Ahmed et al. [6]. It is interesting to note that all patients with significant RV dilatation belonged to GP-II, with only incomplete regression.

The PA dilatation, a likely function of severity of pulmonary arteriolar resistance, has shown fairly good correlation with PAP in various imaging modalities [18–20]. Its postsurgical decrease is to be inline with the PAP regression; Gotsman et al.’s [13] CXR studies noted fall in both main PA and branch sizes. Seningen et al. [12] found PA and LA sizes to be useful and sensitive indicators of postoperative hemodynamic change. However, its echocardiographic correlation has not so far been elucidated. Our echo evaluation revealed varying degrees of PA dilatation relating significantly with PAP preoperatively, but not postsurgery; the percentage reduction not corresponding to that of PAP. The lack of parallelism was also noted in Kaul et al.’s [8] X-ray-based assessment and earlier by Thomas and Trenckmann [21], who found PA size reduction in only 13% of their patients.

Our findings point that regression/normalization of PAP does not seem to have a direct bearing to the regression of RV and PA sizes, the probability being, while the congestive and vasoconstrictive element of PH may reduce, the structural changes of remodeling in PA and RV prevent their complete involution or slow down their resolution. The phase-related pattern confirms this and needs emphasis. Also, variability in the response of the pulmonary bed is to be considered. This raises the need to study RA compliance, which will be more reliable in this context. The vascular changes though predominant in the small muscular and distal vessels; their effect is also reflected in the elastic proximal vessels wherein distensibility and compliance get altered. Although PA size regresses with the pressure drop, it may take longer for their wall resilience to become normal, the surgical implication/significance being extension of the postoperative RV dysfunction and morbidity.

The negative relation of LV size to PAP may be explained by the fact of PH severity increasing in relation to severity and chronicity of MS. With increased flow established across the mitral valve, LV size improved in 64%, with insignificant changes in the rest.

A mild early increase in the chamber sizes in some (more with RV and RA), probably reflecting postsurgery dysfunction, recalls that CPB-induced alterations in the circulatory dynamics influences the immediate postsurgical changes, its reversion being variable in time, extent, and individuals.

Although earlier reports suggested that TR regresses with relief of mitral obstruction [22], conflicting results later [23–25] have raised controversies. Age and AF were considered associated with advanced/irreversible pathology responsible for persistent PH and TR [5, 26]. Recently, Oraby and Mahfouz, [5] and Zoroufian et al. [27] have highlighted the parallelism of reduction of the two parameters. Similarly, surgical series showed that patients without significant annular dilatation improve after mitral surgery [28]. In this study, tricuspid repair was done for significant TR with significant annular dilatation. The post-1-year situation was reasonable TR reduction with PH decrease; quite comparable to other series [4, 5]. The TR regression pattern in the repair and nonrepair group infers that TR due to annular dilatation regresses with repair, the remaining accounted by the residual PH. The greater parameter change in the TAP group was striking only proviso RA and RV as by the described concepts. Their significant RV regression supports and calls for concomitant TAP in these, but the numbers are small to make further comparisons and inferences.

Our patients had a wide PH range: mild to moderate to severe, few even suprasystemic pressures. However, variations in the definition of severe PH in the literature have raised controversies on PH regression [2, 9]. Our PH-based stratification gave better analysis reflecting hemodynamically more severe disease in GP-II [higher average FC (p = 0.007), TR grades (p = 0.001), and chamber enlargements inline with the PAP], despite comparable/similar demographics. The improvement with PH regression nevertheless was in effect more in GP-II for RV, PA, TR, and LV. The pattern in these somewhat conforms to the degree of changes in the pulmonary system with the pressures. Despite their distinctly significant PH regression well in agreement with some of the early reports on hemodynamics [3, 29], the greater degree of residual PH reflects the fixed structural component.

That the regression rate of many of the parameters appears less than the quoted literature can be explained by the probable fact that reports [1, 5, 11], except a very few, on some chamber alterations belong to post-BMV, where the patients form a different set with early disease, unlike the subset considered by us where most had advanced/severe disease.
The contrary/reverse finding as increase in CTR/chamber sizes, or no change in some, did not bear direct/definite relation to degree of PH/other parameters or their changes.

One would stress that despite statistically significant reductions in all studied major parameters, even after 1 year, residual abnormalities were persisting in all. The striking greater prevalence of residua in Gp–II stands with their advanced disease. A similar observation by Ahmed et al. [6] in a small subset neither brought out the exact figures nor their relation to PAP. However, our patients differed from these other referred series by having only predominant MS, without other valvar disease. Thus, we corroborate with few earlier reports [6, 8, 13] that these chamber and vascular abnormalities regress, immediately and thereafter to varying extent, and is reversible, although not to the extent of normalization. A structural correlation indicating the degree of damage will solve more of these issues. Also, the rheumatic disease can have a relatively prolonged effect on these. The clinical significance lies on the functional incapacity, or exercise intolerance, and continued need for medications, as in many of our patients. These patients need serial follow-up evaluations. Despite successful surgery, these abnormal indices sometimes reflect significant chamber stiffness and subclinical myocardopathy that may contribute to cardiac events/arrrhythmias. Thus studying these may help to precociously recognize patients likely to have long-term morbidity.

Study Limitations and Advantages

Although its small numbers and use of only echocardiographic evaluation may limit this retrospective study, it is restricted to a fairly uniform subsector with predominant MS, analyzing changes in each chamber and overall cardiac size. Additionally, noninvasive measures both pre- and postoperatively maintains uniformity. In today’s practice, mitral disease mostly diagnosed and intervened based on echocardiography, this applies to the postoperative and follow-up evaluation. Only linear dimensions have been considered. Further evaluation of thickness, compliance, and functional assessment including 3D would give more insight into the hemodynamic aspects of the disease.

Summary and Conclusions

Noninvasive evaluations in MS showed that functional disability and cardiac chamber alterations correlated with PAP to varying extent pre- and postoperatively (LA size being disproportionate), although not all had direct proportionate postoperative improvement with PAP. This study emphasizes the importance of higher baseline PAP, with greater chamber enlargements, greater postoperative regression, but higher prevalence of residual-abnormalities, suggesting that pathologic changes in them might take longer to resolve, urging/advocating intervention before PH consequences are established.

Funding sources: None declared.

Authors’ contribution: UTP participated in the data acquisition, data analysis, and preparation and drafting of the manuscript. RR participated in data analysis and the manuscript preparation. AGF participated in data acquisition, data analysis, and the manuscript preparation. All authors had access to data and take responsibility for the integrity of data and the accuracy of data analysis. All authors have read and approved the manuscript.

Conflict of interest: None declared.

Ethics: An informed consent was taken from all patients for any kind of publications in the medical journal.

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