ABSTRACT

Objective: To evaluate the short- and mid-term effects of percutaneous mitral balloon valvuloplasty (PMBV) on right ventricular functions in mitral stenosis.

Methods: A prospective study was conducted in 61 patients who had mitral stenosis in normal sinus rhythm (68% female, age: 42±11-16 years). Right ventricular functions were measured before, immediately after, and at 3 months and 1 year after PMBV by conventional and tissue Doppler echocardiography imaging methods. Additionally, the patients were evaluated in two groups (PAP≥40 mm Hg, n: 46; PAP<40 mm Hg, n: 15) according to the systolic pulmonary artery that was measured by echocardiography prior to PMBV.

Results: Post-PMBV mean gradient, pulmonary artery pressure (PAP), and left atrial size decreased significantly, and the mitral valve area increased significantly in both patient groups. This significance in pulmonary artery pressure was lost at 1 year. The significant post-PMBV increase in tricuspid annular point systolic excursion (TAPSE), systolic velocity, early diastolic velocity, and peak myocardial velocity during isovolumic contraction (IVV), indicating right ventricular functions, disappeared at 1 year. The significant post-PMBV decrease in myocardial performance index (MPI) and late diastolic velocity lost its significance at 1 year. No significant change was observed in myocardial acceleration during isovolumic contraction (IVA). The group with pulmonary hypertension demonstrated significance similar to the results of the overall group. Post-PMBV TAPSE, systolic velocity, early diastolic velocity, IVV, and IVA increased significantly, and this increase was maintained up to 1 year in the group without pulmonary hypertension. MPI and late diastolic velocity maintained their significantly decreased values up to 1 year.

Conclusion: The positive effect of PMBV on right ventricular function in the acute period decreases and even disappears in the mid-term in patients developing pulmonary hypertension. Intervention in the patients prior to the development of hypertension is very important for the improvement in right ventricular functions. (Anatol J Cardiol 2015; 15: 289-96)

Keywords: mitral stenosis, percutaneous mitral balloon valvuloplasty, right ventricular functions

Introduction

Several diseases have been acknowledged as pathological causes of mitral valve stenosis (MS), of which rheumatic heart disease is the most prevalent. Rheumatic heart disease is a chronic manifestation of rheumatic carditis, which occurs in 60% to 90% of cases of rheumatic fever (1).

Reduced exercise capacity and fatigue are common symptoms in patients with MS; increased pulmonary venous pressure and left atrium (LA) are not the solely responsible factors for these symptoms (2). Right ventricular (RV) function plays an important role in the development of clinical symptoms, exercise capacity, prognosis, and survival in MS (3, 4).

Effect of percutaneous mitral balloon valvuloplasty on right ventricular functions in mitral stenosis: Short- and mid-term results

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potentially non-invasive and appropriate techniques and are less expensive than the others (5).

Previous studies have investigated the effect of right ventricle functions in patients with mitral stenosis (6-10). However, few studies have examined the effect of mitral valvuloplasty on the echocardiographic markers of RV systolic and diastolic functions in the short term and mid-term (4, 11, 12). The purpose of this study was to assess the impact of PMBV on RV function in the short-term and mid-term using two-dimensional and Doppler echocardiographic indices.

Methods

Subjects
This study was performed in our clinics between April 2008 and June 2010. A prospective study was conducted in 61 patients (68% female, age: 42.7±11.6 years) with isolated rheumatic mitral valve stenosis who underwent PMBV. Indications for PMBV were New York Heart Association class ≥II, ≤IV, planimetric mitral valve area (MVA), ≤1.5 cm², mitral regurgitation ≤2+, suitable valve morphology, and the absence of concomitant cardiovascular disease requiring surgical correction. All patients had sinus rhythm. Additionally, the patients were evaluated in two groups (PAP≥40 mm Hg, n: 46; PAP<40 mm Hg, n: 15) according to the systolic pulmonary artery pressure (PAP) that was measured by echocardiography prior to PMBV. Detailed written informed consent was obtained from each patient. Approval of the study was obtained from the local ethics committee. The exclusion criteria were as follows: left ventricular ejection fraction (LVEF) <50%; aortic regurgitation greater than mild or aortic stenosis; mitral regurgitation greater than mild; clinical, echocardiographic, or angiographic evidence of coronary artery disease; hypertension; diabetes mellitus; severe calcification of mitral valve annulus; clinical or laboratory evidence of active rheumatic disease; chronic obstructive or restrictive lung disease; chronic pulmonary thromboembolism; and low-quality echocardiographic image for tissue Doppler imaging (TDI).

Echocardiographic study
A Vingmed System Five Doppler echocardiographic unit (GE Vingmed Ultrasound, Horten, Norway) with a 2.5-MHz FPA probe was used. Two-dimensional and pulse-wave Doppler echocardiographic studies were performed in the left lateral decubitus position with conventional views (parasternal long- and short-axis, apical four-chamber) and the in the supine position for the subxiphoid approach. An electrocardiogram was recorded simultaneously with the M-mode and Doppler tracings on the same monitor, and a 50-mm/s M-mode sweeping speed was used for the M-mode trace recording. Maximum RA volumes were calculated by 2-D apical 2- and 4-chamber views using the area/length method. Tricuspid annular plane systolic excursion (TAPSE) was determined by the difference in the displacement of the RV base during systole and diastole (13). RV end-diastolic and end-systolic areas were measured from the apical 4-chamber view to calculate RV fractional area change (RVFAC) (14). Tricuspid inflow velocity was recorded from the apical 4-chamber view by pulsed-wave Doppler sample volume, positioned at the tips of the tricuspid leaflets during diastole. Peak early (E) and late (A) tricuspid inflow velocity and deceleration time of E velocity were obtained. The RV outflow velocity was recorded from the parasternal short-axis view with the pulsed-wave Doppler sample volume positioned just below the pulmonary valve. Pre-ejection period (PEP) was measured from the onset of the QRS wave to the onset of RV ejection flow. RV ejection time (RVET) was measured from the onset to the end of RV outflow. Isovolumetric contraction time (ICT) was determined from the cessation of tricuspid inflow to the onset of RV outflow (15). Myocardial performance index (MPI) was calculated from the formula (ICT+IRT)/RVET (Fig. 1) (15, 16). Tissue Doppler imaging (TDI) was applied in the pulse-Doppler mode of the tricuspid annulus velocity at its lateral corners with the same echocardiographic unit, and systolic (S), early diastolic (E), and late diastolic (A) velocity; peak myocardial velocity during isovolumic contraction (IVV, cm/sec); and myocardial acceleration during isovolumic contraction (IVA, m/sec²) defined as the ratio of IVV divided by the acceleration time) were measured. All measurements were calculated from three consecutive cycles, and the average of the three measurements was recorded.

Percutaneous balloon valvuloplasty
MS patients underwent PMBV, which was performed by three investigators. The mitral valve area was calculated using the Gorlin equation (17). Balloon dilation of the mitral valve was performed using a single-balloon dilating technique. Pre-
procedural and post-procedural mitral insufficiency was evaluated based on Sellers classification on left ventriculography (18). The success of the procedure was defined as post-procedural planimetered mitral valve area (MVA) >1.5 cm² echocardiographically and/or a 50% increase over the pre-procedural value and non-development of 3+ or 4+ mitral insufficiency.

Follow-up
Clinical and echocardiographic evaluations were performed before, 24-48 hours after, 3 months after, and 1 year after percutaneous mitral balloon valvuloplasty. Recurrent stenosis was defined as >50% loss of planimetered MVA calculated after the procedure and/or a valve area of 1.5 cm². A major cardiovascular event was defined as death, repeat of balloon valvuloplasty, and the need for mitral valve replacement during the follow-up period.

Statistical analysis
The statistical evaluation was performed using SPSS 15.0 (Statistical package for the social sciences, Chicago, IL, USA). Categorical variables were presented as frequencies and percentages and were compared with the \( \chi^2 \) test. Continuous variables were expressed as means and SD. The normal distribution of continuous variables was tested with the Kolmogorov-Smirnov test. The Friedman test was used to compare consecutive measurements, and the Wilcoxon signed-rank test was used for the post-hoc analysis. A value of p<0.05 was considered significant.

Results
The baseline clinical and demographic properties of all study subjects are presented in Table 1. Forty-two of the 61 patients that were enrolled in the study were female (68%). The average age of the patients was 42±11-16 years. NYHA functional capacity was class 3 in 43 cases, class 2 in 14 cases, and class 4 in 4 cases before PMBV. Of the patients, 19 developed right heart failure, and 28 patients were receiving diuretics. The number of patients receiving diuretics decreased to 22 during the follow-ups.

The procedure failed in 4 (6.1%) patients enrolled in the study. Serious mitral insufficiency due to chorda rupture occurred in 3 patients after the procedure, and valve replacement was performed. Cardiac tamponade due to myocardial rupture occurred in another patient in whom the procedure failed. This patient underwent pericardiocentesis in the catheter laboratory and had no problems during the follow-up in the intensive care unit. The patient was then referred to the surgery department under elective conditions. Clinically insignificant pericardial effusion was found after the procedure in 1 patient who had a successful valvuloplasty. Other major cardiovascular events (death, repeated balloon valvuloplasty) and restenosis were not observed throughout the follow-up.

A comparison of pre- and post-PMBV (at 48 hours, 3 months, and 1 year) values measured by transthoracic echocardiography is presented in Table 2. The mitral valve area that was measured increased significantly after successful PMBV and in the follow-up period (p<0.01). The value of the mean gradient, pulmonary

### Table 1. Clinical characteristics of study patients

|                      | Mean     |
|----------------------|----------|
| Age, year            | 42.11±11.16 |
| Female               | 42 (68%) |
| Systolic blood pressure, mm Hg | 109.8±7.8 |
| Diastolic blood pressure, mm Hg | 71.6±4.3 |
| Heart rate, bpm      | 82.9±5.27 |
| NYHA functional class|          |
| Class 2              | 14 (23%) |
| Class 3              | 43 (70%) |
| Class 4              | 4 (7%)   |
| LVDD, mm             | 47.4±3.2 |
| LVSD, mm             | 31.3±3.8 |
| RA maximal volume, mL| 35.1±3.03 |
| RVFAC, %             | 37.7±1.8 |
| PAP, mm Hg           | 47.2±15.4 |
| MVA PHT, cm²         | 1.2±0.14 |
| Mean gradient, mm Hg | 11.3±2.5 |
| Right heart failure, %| 19 (31%) |

Data expressed as mean±SD or percentage. Variables were recorded before the procedure.
LVDD - left ventricular diastolic diameter; LVSD - left ventricular systolic diameter; MVA PHT - pressure half-time mitral valve area; PAP - pulmonary artery pressure; RA - right atrium; RVFAC - RV fractional area change; RV - right ventricle diameter
artery pressure (PAP), and RA maximal volume decreased significantly after successful PMBV. The maximal PAP and RA volumes began to increase again in the third-month and first-year follow-up measurements and lost significance at the end of the first year. The Wilkins score decreased significantly after successful PMBV and in the follow-up period (p<0.01). TAPSE and RVFAC increased significantly after PMBV but reached their basal levels in the follow-up measurements and lost significance at the end of the first year. Deceleration time, pre-ejection period, A peak, and myocardial performance index decreased significantly, and ejection time and E peak increased significantly after successful PMBV (p<0.01). The MPI lost its significance at the end of the first year. The changes in TAPSE and MPI in the follow-up measurements are presented in Figure 2. Systolic velocity and early diastolic velocity increased significantly, and late diastolic velocity decreased significantly after successful PMBV (p<0.01). However, at 1 year, the statistical significance disappeared. A comparison of pre- and post-PMBV (at 48 hours, 3 months, and 1 year) did not differ significantly with respect to right ventricular myocardial acceleration during isovolumic contraction (IVA) (p=0.23, 0.37, 0.14), respectively, whereas peak myocardial velocity during isovolumic contraction (IVV) increased significantly after successful PMBV (p<0.01). However, at 1 year, the statistical significance disappeared (Table 3). The subgroup analysis that was conducted showed similar results in the pulmonary hypertension group compared to the overall group. TAPSE, systolic velocity, early diastolic velocity, IVV, and IVA increased significantly after PMBV in the group without pulmonary hypertension, and this increase was maintained during their 1-year follow-ups. Late diastolic velocity and MPI decreased significantly after PMBV, and this significance continued at 1 year (Table 4).

Discussion

This study evaluated the acute and mid-term effects of PMBV on RV functions by echocardiographic tissue Doppler imaging.
technique with the intent to investigate whether this acute improvement is a progressive process or an acute response to changes in the cardiopulmonary system. In this study, the improved right ventricular functions in mitral stenosis patients with pulmonary hypertension in the post-PMBV acute period were shown to decrease in the following period and disappeared at the end of 1 year. The improvement in the group without pulmonary hypertension in the acute period was also shown to be sustained at 1 year.

The trapezoidal anatomy of the ventricle makes the quantitative echocardiographic evaluation extremely difficult. Up to date many different assessment methods has been investigated but none of them appears to be the gold standard at this era. New methods have been evaluated in recent years. Cardiac catheterization, MR, radionuclide ventriculography, and 3D-echocardiography have shown that right ventricle functions can be used reliably (19-21). On the other hand, these methods are not readily accessible and can not be performed in a short time. In practice, clinicians largely rely on two modalities: two-dimensional echocardiography and TDI echocardiography. Conventional M-mode, Doppler echocardiography evaluation, and TDI echocardiography, which is used to evaluate right and left ventricle functions, are preferred, because they are less affected by physiologic changes in flow velocities and indicate subclinical functional effects. In typical pulse-wave Doppler imaging obtained with Doppler echocardiography, it is possible to measure the duration of the systolic and diastolic waves. In the case of systolic dysfunction, isovolumetric contraction time increases, whereas ejection time decreases. However, in the case of diastolic dysfunction, in which flexibility decreases, isovolumet-

Table 3. Comparison of pre- and post-PMBV (at 48 hours, 3 months, and 1 year) pulsed tissue Doppler

| Parameter                      | Before PMBV | After PMBV | 3rd month | 1st year |
|-------------------------------|-------------|------------|-----------|----------|
| Systolic velocity, cm/s       | 12.29 (9-17)| 15.03 (11-17)** | 13.08 (10-16)* | 12.50 (10-15) |
| Early diastolic velocity, cm/s| 10.14 (8-14)| 13.08 (10-16)** | 11.14 (8-16) | 11.06 (8-15) |
| Late diastolic velocity, cm/s | 12.88 (8-16)| 11.14 (8-16)** | 11.06 (8-15)** | 11.45 (8-15)* |
| Right ventricular IVV, cm/sec  | 0.11 (0.06-0.16) | 0.14 (0.08-0.19)** | 0.13 (0.07-0.18)* | 0.12 (0.07-0.16) |
| Right ventricular IVA, m/sec^2 | 2.21 (1.60-2.90) | 2.19 (1.80-2.80) | 2.20 (1.70-2.90) | 2.22 (1.60-2.90) |

IVA - myocardial acceleration during isovolumic contraction; IVV - peak myocardial velocity during isovolumic contraction
*P<0.05 comparison with baseline after PMBV and at 3 months and 1 year.
**P<0.01 comparison with baseline after PMBV and at 3 months and 1 year.

Table 4. Comparison of pre- and post-PMBV (at 48 hours, 3 months, and 1 year) echocardiographic variables in patients with or without baseline pulmonary hypertension

| Parameter                      | Pulmonary hypertension (+) (n:46) | Pulmonary hypertension (-) (n:15) |
|-------------------------------|-----------------------------------|-----------------------------------|
| TAPSE, mm                     | Before PMBV 17.19 (13-24)         | Before PMBV 17.4 (13-25)          |
|                               | After PMBV 18.6 (15-24)**         | After PMBV 18.8 (15-27)**         |
|                               | 3rd months 18 (14-24)**           | 3rd months 18.5 (15-27)**         |
|                               | 1st year 17.21 (13-24)            | 1st year 18.3 (15-26)**           |
| MPI                           | Before PMBV 0.49 (0.42-0.58)      | Before PMBV 0.48 (0.42-0.59)      |
|                               | After PMBV 0.42 (0.36-0.54)**     | After PMBV 0.41 (0.34-0.56)**     |
|                               | 3rd months 0.45 (0.39-0.56)**     | 3rd months 0.42 (0.34-0.56)**     |
|                               | 1st year 0.49 (0.42-0.59)         | 1st year 0.42 (0.35-0.56)**       |
| PAP, mm Hg                    | Before PMBV 55.6 (40-75)          | Before PMBV 29.53 (25-39)         |
|                               | After PMBV 43.73 (30-65)**        | After PMBV 26.46 (25-32)          |
|                               | 3rd months 43.36 (30-70)**        | 3rd months 26.66 (25-35)          |
|                               | 1st year 54.13 (40-75)            | 1st year 27.4 (25-42)             |

Tricuspid annulus

| Parameter                      | SV, cm/s               | Ev, cm/s               | AV, cm/s               | IVV, cm/sec | IVA, m/sec^2 |
|-------------------------------|------------------------|------------------------|------------------------|-------------|--------------|
| Sv, cm/s                      | 12.15 (9-17)           | 15.15 (11-19)**        | 13.08 (10-16)*         | 0.01 (0.06-0.16) | 2.19 (1.6-2.9) |
| Early diastolic velocity, cm/s| 10.02 (8-14)           | 13.08 (11-17)**        | 11.15 (8-16)*          | 0.12 (0.07-0.17)* | 2.16 (1.6-2.8) |
| Late diastolic velocity, cm/s | 12.8 (10-16)           | 11.15 (8-16)**         | 12.02 (9-17)*          | 0.12 (0.06-0.16) | 2.16 (1.7-2.9) |
| Right ventricular IVV, cm/sec | 12.8 (10-16)           | 11.15 (8-16)**         | 12.02 (9-17)*          | 0.11 (0.06-0.15) | 2.18 (1.6-2.8) |
| Right ventricular IVA, m/sec^2| 12.7 (9-16)            | 11.15 (8-16)**         | 12.02 (9-17)*          | 0.11 (0.06-0.15) | 2.20 (1.7-2.9) |
| PAP, mm Hg                    | 14.6 (10-16)           | 11.15 (8-16)**         | 12.02 (9-17)*          | 0.12 (0.07-0.16)* | 2.28 (1.9-2.6) |
| MPI                           | 14.06 (10-17)*         | 11.15 (8-16)**         | 12.02 (9-17)*          | 0.13 (0.07-0.17)** | 2.34 (1.9-2.6) |
| Tricuspid annulus             | 13.67 (10-17)*         | 11.15 (8-16)**         | 12.02 (9-17)*          | 0.13 (0.08-0.18)** | 2.38 (2-2.7)* |

*Results are shown as rank (min-max) values.

Av - late diastolic velocity; Ev - early diastolic velocity; IVA - myocardial acceleration during isovolumic contraction; IVV - peak myocardial velocity during isovolumic contraction.
**P<0.01 comparison with baseline after PMBV, at 3 months and 1 year.

*P<0.05 comparison with baseline after PMBV, at 3 months and 1 year.

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It was surprising to the researchers that pulmonary artery pressure decreased after PMBV, and this decrease reached the basal level at the 1-year follow-up, although it was maintained during the 3-month follow-ups. The subgroup evaluation revealed that there was a similar case in the group with pulmonary hypertension; however, the decrease that occurred in the acute period in the group without pulmonary hypertension continued during the 1-year follow-up, as well. Given the re-elevation of PAP at the 1-year follow-ups, especially in the pulmonary hypertension group, and the non-observation of clinical conditions that might cause this during the follow-up, such as restenosis, development of paroxysmal AF, and mitral regurgitation, it is difficult to explain the underlying mechanism. It may be that the irreversible changes in the pulmonary vascular bed in the group with pulmonary hypertension presented a pseudo-improvement for a given time due to the decreased post-PMBV afterload. The study by Mahfouz et al. (36) analyzed the long-term effect of pulmonary artery stiffness on right ventricular functions and tricuspid regurgitation. Based on the evaluations before, immediately after, and at 6 months and 12 months after the procedure, the investigator demonstrated that pulmonary artery stiffness was significantly lower in patients who had permanent improvement in right ventricular functions and regression of tricuspid regurgitation. The investigator argued that the tricuspid regurgitation and the continued right ventricular dysfunction in some patients, even though a sufficient mitral valve area opening could be ensured after PMBV, may be the increased pulmonary artery stiffness in this patient group and highlighted the importance of early intervention.

In the present study, the recovery of RV functions decreased in the mid-term and disappeared at the end of the first year (an increase in the Tei index, and pulmonary arterial pressure and a reduction in TAPSE). Although similar results were achieved in the group with pulmonary hypertension, the improved right ventricular functions that occurred in the acute period in the group without pulmonary hypertension were maintained at 1 year. High wall stress due to ventricular dilatation that affects RV myocardium or rheumatic conditions that occur as a secondary result of myocardial dysfunction can elaborate this situation (37). In a previous study by Malhotra et al. (38), it is shown that intra myocardial branches of myocardial vessels were also involved in a form of active rheumatic vasculitis or inactive lesions characterized by medial hypertrophy and replacement fibrosis in patients with rheumatic heart disease affecting. The study of Mohan et al. (39) showed that pulmonary artery pressure decreased immediately after balloon valvuloplasty and that right ventricular functions, as assessed by the Tei index, returned to normal values within 1 year in 65% of such patients. It was shown that there was right ventricular fractional shortening and improvement in systolic functions, as assessed by the Tei index, after balloon valvuloplasty in patients with mitral stenosis, whereas a decrease was observed in right ventricular contraction, as assessed by IVA (12). The lack of observed improvement in right ventricular function despite the improved hemodynamic status suggested irreversible myocardial damage due to rheumatic pathology or long-lasting hemodynamic burden in these patients. Arat et al. (11) evaluated RV functions in the early (first 48 hours) and mid (3rd month)-term after PMBV and did not observe a significant difference in the Tei index. The authors determined that RV functions increased significantly in the early period in the group without pulmonary HT and maintained their high level in the mid-term. Mahfouz et al. (35) determined a significant decrease in pulmonary arterial pressure and a significant increase in TAPSE in the post-PMBV evaluation. As demonstrated in these literature findings, during the acute phase and
short term, there are no clear data showing the effect of PMBV on RV functions, and follow-up studies with larger numbers of patients are needed to assess whether this finding has any prognostic implications. Although echocardiography is a non-invasive and reproducible method to evaluate cardiac functions, it should be kept in mind that RV function parameters are also not fully independent parameters.

**Study limitations**

One of the limitations of this study is that it was a single-center study and not randomized, and the study population was relatively small. The patients used to predict RV dysfunction were not independent parameters. In the current study, invasive measurements were not made, and RV ejection fraction was not measured. Additionally, strain imaging has a lower temporal resolution compared to tissue Doppler-derived deformation indices, resulting in less reliable estimates. Because of the complex geometry of the RV, real-time 3-D echocardiography is estimated to accurately evaluate the morphology and function of this chamber. However, none of the methods used to evaluate right cardiac function is free of limitations (40, 41). Tissue Doppler imaging is a method with proven efficacy and relative reliability. Also, these limitations were minimized because of this being a follow-up study.

**Conclusion**

The data of the present study revealed that right ventricle functions improved significantly immediately after PMBV, but the observed recovery decreased and even disappeared in patients with pulmonary hypertension in the early- and mid-term follow-ups. This condition may indicate the importance of intervention in MS patients prior to the development of latent right ventricle myocardial dysfunction.

**Conflict of interest:** None declared.

**Peer-review:** Externally peer-reviewed.

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