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 significantly decreased, and the mitral valve area significantly increased 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 significantly increased, and this increase was maintained up to 1 year in the group without pulmonary hypertension. MPI and late diastolic velocity maintained the 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. (Anadolu Kardiyol Derg 2014; 14(0): 000-000)

Key words: mitral stenosis, percutaneous mitral balloon valvuloplasty, right ventricular functions

Introduction

Several diseases have been acknowledged as pathological causes for 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).

Right ventricle dysfunction, which emerges secondary to chronic pulmonary hypertension, is accepted as an important but undesired result of mitral stenosis. Percutaneous mitral balloon valvuloplasty is the most common type of treatment used in patients with mitral stenosis. The effect of successful percutaneous mitral balloon valvuloplasty (PMBV) on global RV systolic and diastolic functions in patients with rheumatic MS has not been well defined. Conventional echocardiography, Doppler tissue imaging, radionuclide ventriculography and magnetic resonance imaging are the methods that are used to evaluate RV functions. Conventional two-dimensional (2-D) echocardiography and Doppler tissue imaging are methods of measuring systolic and diastolic velocities of annular motions. They are also
potentially non-invasive and appropriate techniques and are less expensive than the others (5).

Previous studies have suggested the investigation of the effect of right ventricle functions in patients with mitral stenosis (6-10). However, few of the 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. A detailed written informed consent was obtained from each patient. Approval of the study was obtained from the local ethics committee. Exclusion criteria were as follows: left ventricular ejection fraction (LVEF) <50%, aortic regurgitation greater than a mild degree or aortic stenosis, mitral regurgitation greater than a mild degree, 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 50 mm/s M-mode sweeping speed was used for M-mode trace recording. Maximum RA volumes were calculated by 2-D apical 2- and 4-chamber views using the area/length method. The tricuspid annular plane systolic excursion (TAPSE) was determined by the difference in the displacement of the RV base during systole and diastole (13).

**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 were 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 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.

Statistical analysis
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 Kolmogorov-Smirnov test. The Friedman test was used to compare consecutive measurement, and the Wilcoxon signed-rank test was used for 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 four (6.1%) patients enrolled in the study. Serious mitral insufficiency due to chorda rupture occurred in three patients 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.

The procedure failed in four (6.1%) patients enrolled in the study. Serious mitral insufficiency due to chorda rupture occurred in three 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 one 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 artery pressure (PAP), and RA maximal volume significantly decreased 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 significantly decreased 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 significantly decreased, and ejection time and E peak significantly increased after successful PMBV (p<0.01). The MPI lost its significance at the end of the first year.

![Figure 2](attachment:image.png)

**Figure 2. Changes in right ventricular myocardial performance index (MPI) and Tricuspid annular plane systolic excursiion (TAPSE) before, after the 3rd month, and after 1 year percutaneous mitral balloon valvuloplasty.**

myocardial velocity during isovolumic contraction (IVV) significantly increased after successful PMBV (p<0.01). However, at one 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 significantly increased after PMBV in the group without pulmonary hypertension and this increase was maintained during their 1 year follow-ups. Late diastolic velocity and MPI significantly decreased after PMBV and this significance continued at 1 year (Table 4).

<table>
<thead>
<tr>
<th>Table 2. Comparison of pre- and post-PMBV (at 48 hours, 3 months, and 1 year) values measured by transthoracic echocardiography</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before PMBV</td>
</tr>
<tr>
<td>LVDD, mm</td>
</tr>
<tr>
<td>LVSD, mm</td>
</tr>
<tr>
<td>Mean GR, mm Hg</td>
</tr>
<tr>
<td>MVA PHT, cm²</td>
</tr>
<tr>
<td>MVA plan, cm²</td>
</tr>
<tr>
<td>PAP, mm Hg</td>
</tr>
<tr>
<td>RA maximum volume, mL</td>
</tr>
<tr>
<td>LA, mm</td>
</tr>
<tr>
<td>RVFAC, %</td>
</tr>
<tr>
<td>TAPSE, mm</td>
</tr>
<tr>
<td>TR jet area/RAA</td>
</tr>
<tr>
<td>Wilkins score</td>
</tr>
<tr>
<td>E peak, cm/s</td>
</tr>
<tr>
<td>A peak, cm/s</td>
</tr>
<tr>
<td>Deceleration time, ms</td>
</tr>
<tr>
<td>Pre-ejection period/ Ejection time</td>
</tr>
<tr>
<td>Myocardial performance index</td>
</tr>
</tbody>
</table>

Results are shown as rank (min-max) value
LVDD - left ventricular diastolic diameter; LVSD - left ventricular systolic diameter; Mean GR - mitral mean gradient; MVA - plan metric mitral valve area; PAP - pulmonary artery pressure; PHT - pressure half time mitral valve area; PMBV - percutaneous balloon mitral valvuloplasty; RA - right atrium; RV - right ventricle; RVFAC - RV fractional area change; TAPSE - tricuspid annular plane systolic excursion; TR jet area/RAA-tricuspid valve regurgitation area/right atrial area
*P<0.05 comparison with baseline-after PMBV, at 3 months and 1 year
**P<0.01 comparison with baseline-after PMBV, at 3 months and 1 year

Before PMBV | After PMBV | 3rd month | 1st year
LVDD, mm | 47.44 (41-52) | 47.32 (42-52) | 47.15 (42-52) | 48.33 (43-54) |
LVSD, mm | 31.30 (25-37) | 31.15 (25-39) | 32.22 (27-41) | 32.36 (27-42) |
Mean GR, mm Hg | 11.3 (17-8) | 4.3 (2-6)** | 4.3 (2-6)** | 4.4 (2-6)** |
MVA PHT, cm² | 1.1 (0.9-1.6) | 2.2 (1.8-2.8)** | 2.2 (1.7-2.9)** | 2.1 (1.8-2.7)** |
MVA plan, cm² | 1.1 (1-1.6) | 2.2 (1.9-2.7)** | 2.2 (1.9-2.7)** | 2.2 (1.9-2.7)** |
PAP, mm Hg | 47.20 (25-75) | 39.49 (25-65)** | 39.26 (25-70)** | 47.29 (25-75) |
RA maximum volume, mL | 35.13 (30-42) | 33.22 (25-39)* | 33.86 (27-39)* | 35.16 (28-41) |
LA, mm | 48 (42-57) | 46 (39-55)* | 44 (38-52)** | 43 (38-52)** |
RVFAC, % | 37.71 (35-41) | 42.26 (38-45)** | 40.59 (35-45)* | 37.63 (33-42) |
TAPSE, mm | 17.24 (15-20) | 18.68 (17-22)** | 18.06 (16-21)* | 17.56 (15-20) |
TR jet area/RAA | 0.23 (0.07-0.43) | 0.17 (0.05-0.32)** | 0.18 (0.05-0.35)* | 0.21 (0.06-0.41) |
Wilkins score | 8 (6-10) | 6 (4-8) | 6 (4-8) | 6 (4-8) |
E peak, cm/s | 41.98 (35-55) | 50.03 (42-56)** | 44.40 (35-56)* | 43.49 (35-55)* |
A peak, cm/s | 45.41 (40-50) | 39.61 (35-48)** | 43.89 (37-49)* | 44.48 (34-50) |
Deceleration time, ms | 247.47 (220-282) | 214.16 (160-260)** | 238.44 (216-280)* | 245.32 (216-285)* |
Pre-ejection period/ Ejection time | 0.62 (0.52-0.68) | 0.39 (0.35-0.47)** | 0.55 (0.42-0.64)* | 0.58 (0.45-0.55)* |
Myocardial performance index | 0.48 (0.43-0.55) | 0.42 (0.36-0.46)** | 0.44 (0.40-0.50)* | 0.48 (0.43-0.55) |
Discussion

This study evaluated the acute-and mid-term effects of PMBV on RV functions by echocardiographic tissue Doppler technique with the intent to investigate whether this acute improvement is a progressive process or an acute response to changes in cardiopulmonary systems. 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 shown to sustain at 1 year, also.

The quantitative echocardiographic assessment of RV function is difficult because of the ventricle’s complex trapezoidal anatomy. A wide variety of techniques have been proposed, but none is currently considered the gold standard. 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 cannot be performed in a short time. In practice, clinicians largely rely on two modalities: two-dimensional echocardiography and TDI echocardiography.

In typical pulse wave Doppler imaging obtained with Doppler echocardiography, it is possible to measure the duration of the systolic and diastolic waves.

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

<table>
<thead>
<tr>
<th></th>
<th>Before PMBV</th>
<th>After PMBV</th>
<th>3rd month</th>
<th>1st year</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic velocity, cm/s</td>
<td>12.29 (9-17)</td>
<td>15.03 (11-17)**</td>
<td>13.08 (10-16)*</td>
<td>12.50 (10-15)</td>
</tr>
<tr>
<td>Early diastolic velocity, cm/s</td>
<td>10.14 (8-14)</td>
<td>13.08 (10-16)**</td>
<td>11.14 (8-16)</td>
<td>11.06 (8-15)</td>
</tr>
<tr>
<td>Late diastolic velocity, cm/s</td>
<td>12.88 (8-16)</td>
<td>11.14 (8-16)**</td>
<td>11.06 (8-15)**</td>
<td>11.45 (8-15)*</td>
</tr>
<tr>
<td>Right ventricular IVV, cm/sec</td>
<td>0.11 (0.06-0.16)</td>
<td>0.14 (0.08-0.19)**</td>
<td>0.13 (0.07-0.18)*</td>
<td>0.12 (0.07-0.16)</td>
</tr>
<tr>
<td>Right ventricular IVA, m/sec²</td>
<td>2.21 (1.60-2.90)</td>
<td>2.19 (1.80-2.80)</td>
<td>2.20 (1.70-2.90)</td>
<td>2.22 (1.60-2.90)</td>
</tr>
</tbody>
</table>

IAV - myocardial acceleration during isovolumic contraction; IVV - peak myocardial velocity during isovolumic contraction

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

**P<0.01 comparison with baseline-after PMBV, 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

<table>
<thead>
<tr>
<th></th>
<th>Pulmonary hypertension (+) (n:46)</th>
<th>Pulmonary hypertension (-) (n:15)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before PMBV</td>
<td>After PMBV</td>
</tr>
<tr>
<td>MPI</td>
<td>0.49 (0.42-0.58)</td>
<td>0.42 (0.36-0.54)**</td>
</tr>
<tr>
<td>PAP, mmHg</td>
<td>55.6 (40-75)</td>
<td>43.73 (30-65)**</td>
</tr>
</tbody>
</table>

Tricuspid annulus

<table>
<thead>
<tr>
<th></th>
<th>SV, cm/s</th>
<th>EV, cm/s</th>
<th>AV, cm/s</th>
<th>IVV, cm/sec</th>
<th>IVA, m/sec²</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>12.15 (9-17)</td>
<td>15.15 (11-19)**</td>
<td>13.08 (10-16)*</td>
<td>0.11 (0.06-0.16)</td>
<td>2.19 (1.6-2.9)</td>
</tr>
<tr>
<td></td>
<td>10.02 (8-14)</td>
<td>13.08 (11-17)**</td>
<td>11.15 (8-16)*</td>
<td>0.12 (0.07-0.17)*</td>
<td>2.16 (1.6-2.8)</td>
</tr>
<tr>
<td></td>
<td>12.8 (10-16)</td>
<td>11.15 (8-16)**</td>
<td>12.02 (9-17)*</td>
<td>0.12 (0.07-0.16)**</td>
<td>2.18 (1.6-2.9)</td>
</tr>
<tr>
<td></td>
<td>0.11 (0.06-0.16)</td>
<td>0.12 (0.07-0.16)*</td>
<td>0.11 (0.06-0.16)</td>
<td>0.11 (0.06-0.15)</td>
<td>2.20 (1.6-2.9)</td>
</tr>
<tr>
<td></td>
<td>2.28 (1.9-2.6)</td>
<td>2.28 (1.9-2.6)</td>
<td>2.34 (1.9-2.6)</td>
<td>0.13 (0.07-0.17)**</td>
<td>2.38 (2-2.7)*</td>
</tr>
</tbody>
</table>

Results are shown as rank (min-max) values

Ar - late diastolic velocity; Ev - early diastolic velocity; IVA - myocardial acceleration during isovolumic contraction; IVV - peak myocardial velocity during isovolumic contraction; MPI - myocardial performance index; SV - systolic velocity; TAPSE - tricuspid annular plane systolic excursion

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

**P<0.01 comparison with baseline-after PMBV, at 3 months and 1 year
function, isovolumetric contraction time increases, whereas ejection time decreases. However, in the case of diastolic dysfunction, in which flexibility decreases, isovolumetric relaxation time increases. MPI (Tei index), which is calculated using these three indices of time, is a reliable parameter evaluating both systolic and diastolic functions (22). The Tei index is not greatly influenced by changes in blood pressure, afterload, heart rate, preload, RV pressure, and dilatation, or tricuspid regurgitation in the clinical setting (11). Several studies have been published regarding the use of the Tei index and pulsed TDI to identify patients with impaired systolic and diastolic function (23-27).

Recently, a new TDI-derived index of myocardial acceleration during isovolumic contraction (IVA) has been shown to be a reliable and relatively load-independent measure of RV systolic function (28). Despite certain limitations, TAPSE, which can be performed using echocardiography (14, 29), can be readily used in daily practice. The comparative studies on the right ventricle have shown that TAPSE is correlated with magnetic resonance imaging and radionuclide ventriculography (30).

RV function is an important determinant of clinical symptoms, exercise capacity, and survival in patients with MS. MS has a physiopathological process, which results in RV failure. Increased pulmonary wedge pressure is associated with a boost in pulmonary artery pressure, increased afterload, and consequently, failure in RV ejection fraction. RV functions are afterload dependent, which may be physiologically defined as systolic wall stress (31). RV is sensitive to changes in afterload because of smaller mass and higher wall stress (32). In this study, the recovery of right ventricle function (a decrease in RV, Tei index, and pulmonary arterial pressure, and an increase in TAPSE, Sa and IVV) right after PMBV can be explained by the recovery of the RV outflow tract systolic functions due to the acute decrease in the RV afterload (12). However, no significant change was observed in IVA in both the acute period and mid-term follow-ups. This is an expected outcome since the IVA is not specifically affected by hemodynamic changes. Borgers et al. (33) showed recovery in the Tei index after vasodilator therapy in patients who had chronic pulmonary hypertension. Vogel et al. (34) demonstrated that IVA was an accurate parameter to assess RV systolic dysfunction and was able to measure the force-frequency relation. Previous studies also showed that RV function decreased in MS due to increased RV afterload (6, 8, 9) and demonstrated the positive effect of PMBV on RV functions in the acute period (11, 12, 35). However, to our knowledge, there are no studies that demonstrate whether this effect is carried into the mid-term.

It was surprising to the researchers that the 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 that the re-elevation of PAP at the 1-year follow-ups, especially in the pulmonary hypertension group, the non-observation of clinical conditions that might cause this during the follow-up such as restenosis, development of paroxysmal AF, 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 the pulmonary artery stiffness was significantly lower in the patients who had permanent improvement in right ventricular functions and regression in 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 RV, 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 occurred in the acute period in the group without pulmonary hypertension were maintained at 1 year. This is explained by myocardial dysfunction secondary to the rheumatic process, directly affecting RV myocardium or high wall stress due to ventricular dilatation (37). Malhotra et al. (38) in a histo-morphological study of cases of rheumatic heart disease, found 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. They speculated that these changes might affect myocardial function. The study of Mohan et al. (39) showed that pulmonary artery pressure decreased immediately after the balloon valvuloplasty and that the right ventricular functions, as assessed by the Tei index, returned to normal values within one year in 65% of such patients. It was shown that there was right ventricular fractional shortening and improvement for a given time due to the decreased post-PMBV afterload. The study by Arat et al. (11) evaluated the 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 the RV functions significantly increased in the early period in the group without pulmonary HT, and maintained its high level.
in the mid-term. Mahfouz et al. (35) determined a significant decrease in the 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 is not clear data showing whether 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 is single-center and not randomized, and the study population is relatively small. The parameters 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 the right cardiac function are 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 significantly improved 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.


**References**


