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Original Article

Cardiovascular Magnetic Resonance in Predicting the Reduction in Pulmonary Artery Pressure in Patients With Mitral Stenosis After Surgical or Interventional Treatment

Hamidreza Sanati [1], MD; Reza Rezaei Tabrizi [2], MD; Hamid Reza Pouraliakbar [2*], MD; Ali Zahedmehr [1], MD; Ata Firouzi [1], MD; Farshad Shakerian [1], MD; Reza Kiani [1], MD; Nasim Naderi [2], MD

ABSTRACT

Background: Pulmonary hypertension (PH) is a common consequence of mitral stenosis (MS). After treatment, PH reverses depending on the chronicity and severity of MS. The characteristic changes in the pulmonary artery (PA) secondary to an elevated pulmonary artery pressure (PAP) can be evaluated via cardiovascular magnetic resonance imaging (CMR). In this study, we aimed to evaluate if there was any correlation between PAP and hemodynamic findings measured by CMR and whether these findings could be useful in predicting the PAP response after MS relief.

Methods: Thirty-three patients with a diagnosis of severe MS, who were candidated for percutaneously transvenous mitral commissurotomy (PTMC) or mitral valve replacement (MVR), were included. CMR was performed in all of them before the procedure and PA distensibility, PA peak velocity, PA forward volume, and PA forward flow were measured. Transthoracic echocardiography was performed at baseline, immediately after the procedure, and 3 months after MS relief for the assessment systolic PAP.

Results: Thirty-three patients with a diagnosis of MS+PH (15 PTMC and 18 MVR) were enrolled in this study. The mean PAP at baseline catheterization ranged from 25 to 70 mm Hg. There was a significant drop in systolic PAP immediately after the procedure and 3 months after MS relief. There was no relationship between the PA distensibility index and systolic PAP changes after MS relief. PA peak velocity was significantly higher in the patients with > 50% drops in their systolic PAP 3 months after the treatment. The multivariable analysis showed that none of the CMR findings was an independent predictor of a more systolic PAP decline.

Conclusions: Although we found no significant relationship between CMR findings and systolic PAP changes after MS treatment, the result of this study can be used for further investigations in this regard. ***(Iranian Heart Journal 2017; 18(1):30-36)***

Keywords : Pulmonary artery pressure, Cardiovascular magnetic resonance imaging, Mitral stenosis

[1] Cardiovascular Intervention Research Center, Rajaie Cardiovascular , Medical , and Research Center, Iran University of Medical Sciences, Tehran, I.R. Iran.
[2] Rajaie Cardiovascular , Medical , and Research Center, Iran University of Medical Sciences, Tehran, I.R. Iran.

***Corresponding Author:** Hamid Reza Pouraliakbar, MD; Rajaie Cardiovascular , Medical , and Research Center, Iran University of Medical Sciences, Tehran, I.R. Iran.
E-mail: hamidpou@yahoo.com **Tel:** 09122775591

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Pulmonary hypertension (PH) is a common consequence of left-heart diseases, including mitral stenosis (MS). [1-3] Pulmonary artery pressure (PAP) rises chronically in these patients as a result of chronic pathological changes in pulmonary arteries and veins secondary to an increase in left ventricular filling pressures. After treatment, PH reverses depending on the chronicity and severity of MS and the underlying pathophysiological remodeling. [2, 3] Generally, in MS, a rapid decline in PAP is observed after relieving the stenosis. However, PAP remains high or decline more slowly in some patients. [2, 3] The prediction of the PAP response to the treatment is very difficult. Consequently, an investigation of the predictive factors is of great importance. Cardiovascular magnetic resonance imaging (CMR) has been used in the evaluation of PH for the past 25 years. [4-7] The characteristic changes of the right heart and the pulmonary artery (PA) secondary to an elevated PAP can be evaluated via CMR examination, [4-12]
In this study, we aimed to evaluate whether there was any correlation between PAP and hemodynamic findings measured by CMR and whether these findings could be useful in predicting the PAP response after MS relief.

METHODS

In this case series, a total of 33 patients with severe rheumatismal MS who had PH (mean PAP ≥ 25 mm Hg in right-heart catheterization) and were candidated for percutaneous transvenous mitral commissurotomy (PTMC) or mitral valve replacement (MVR) in Rajaie Cardiovascular, Medical, and Research Center were included.
The diagnosis of severe MS (mitral valve area [MVA] < 1.5 cm^2) was made in accordance with the European guideline for the management of valvular heart disease. [1] The presence of PH was confirmed in the catheterization laboratory using the European guideline for the diagnosis and management of PH. [2]
The exclusion criteria were comprised of the presence of other valvular heart diseases with more than moderate severity needing intervention, mitral regurgitation, significant left ventricular (LV) systolic dysfunction (left ventricular ejection fraction [LVEF] ≤ 40%), and inability to attend the CMR field (claustrophobia).

Echocardiographic examination

Comprehensive transthoracic echocardiography was performed by an echocardiography specialist using a Vivid 7 ultrasound system (GE Medical System, Horton, Norway) and a 1.7/3.4 MHz transducer. Images were acquired with the subjects at rest, lying in the left lateral supine position at the end of expiration. 2D ECG was superimposed on the images, and end-diastole was considered at the peak R-wave of the ECG. LV global systolic function was evaluated using a modified biplane Simpson method for calculating LVEF. In all the patients, the MVA was assessed using the 2D planimetry method in the parasternal short-axis view at valve tips. PAP was evaluated considering the guidelines on the assessment of the right heart before the procedure, the 1st time after the procedure during index hospitalization and subsequently 3 months after discharge.

Cardiovascular magnetic resonance examination

CMR with 1.5-T clinical magnet (Magnetom Avento; Siemens Medical Solutions, Erlangen, Germany) with a gadolinium-based contrast agent (Magnevist) was performed, and the following data were obtained from a thorough CMR examination: PA diameter in systole and diastole, distensibility index (the difference between the maximum and minimum PA diameter divided by the maximum diameter), PA peak velocity, PA forward volume, PA forward flow.
CMR study was performed with the patients in the supine position by using a phased-array

surface coil as a receiver and retrospective ECG gating. Images were obtained during end-expiratory breath holds preceded by brief hyperventilation. After obtaining standard localizer views, 2 double-oblique views oriented along the main axis of the pulmonary trunk and the ascending aorta were acquired with a standard steady-state free precession cine MR sequence. The 1st one was in a double-oblique section perpendicular to the direction of the ascending aorta at the level of the pulmonary bifurcation, and the 2nd was in a double-oblique section perpendicular to the main PA, 10 mm above the pulmonary valves. Only 1 set of velocity measurements was acquired for each patient, perpendicular to the main PA.

Both cine loops were used as the reference to prescribe a plane truly perpendicular to the main PA for the acquisition of phase-contrast MR images and to ensure that the imaging plane remained between the pulmonary valve and the PA bifurcation throughout the whole cardiac cycle.

The imaging plane was selected on an oblique sagittal localizing image that showed the ascending aorta. We preferred an imaging plane at the level of the pulmonary bifurcation since it was easy to reproduce. This imaging plane also had a sufficient distance from the aortic valve so that the effect of mild valvular disease did not disturb the flow measurements.

Phase-contrast MR images were acquired with a segmented fast gradient-echo MR sequence , with velocity encoding perpendicular to the imaging plane and a predefined upper velocity limit of 100 cm/sec. If aliasing was noted, the velocity was progressively raised in 50-cm/sec steps until the aliasing disappeared. Imaging parameters comprised the following: 7.5/3.1; flip angle, 15°; section thickness, 6 mm; field of view, 320–380 × 240–300 mm; matrix, 256 × 128 (typical in-plane resolution, 2.7 × 1.4 mm); number of signals acquired, 1; number of segments, 5 to 7; temporal resolution, 75–105 msec; number of reconstructed cardiac phases, 20; and bandwidth, 260 Hz/pixel. The typical breath-hold time ranged from 15 to 25 seconds. The patients were encouraged to hold their breath during the whole acquisition. Supplemental oxygen was administered as clinically indicated or if the patients experienced difficulties completing the period of apnea. If obvious breathing artifacts were noted, the acquisition was repeated.

The study was approved by institutional research and ethics committee, and informed consent was obtained from all the study participants.

Statistical Analysis

All the analyses were conducted using SPSS software, version 22 (SPSS Inc, Chicago, IL, USA). All the data initially were analyzed using the Kolmogorov–Smirnov test to assess for normality. The categorical variables are presented as numbers (percentages) and analyzed using the χ^2 test. The normally-distributed quantitative variables are presented as means ± standard deviations (SDs) and those with non-Gaussian distributions are presented as medians (interquartile ranges [IQRs]). For the analysis of the quantitative variables, the Student *t*-test was drawn upon. The relationships were assessed using the Spearman rank correlation coefficient (ρ) or the Pearson correlation test as appropriate. Binary logistic regression analysis was applied to assess the independent CMR factors associated with a higher drop in systolic PAP (> 50% decline in systolic PAP or systolic PAP ≤ 35 mm Hg immediately after the procedure or 3 months after the procedure). All the *P* values were 2-tailed, and a *P* value < 0.05 was considered statistically significant.

RESULTS

Thirty-three patients with a diagnosis of MS+PH (15 patients scheduled for PTMC and

18 for MVR) were enrolled in this study. About 70 % of the patients were female and the mean (SD) of age was 51.3 (12) years (between 32 and 74 y).The most common chief complaint was dyspnea on exertion, which was obvious in all the patients.

The median (IQR) of the MVA was 0.82 (0.5–1.3) cm^2, and the mean (SD) of systolic PAP was 62 (19) mm Hg in baseline echocardiography. The mean PAP at baseline catheterization ranged from 25 to 70 mm Hg in our study population (mean [SD] = 42 [12.1]).

Table 1 shows the demographic, echocardiographic, and hemodynamic findings of the study population. There was a significant drop in systolic PAP immediately after the procedure and 3 months after MS relief compared to before the treatment in our study population.

The mean (SD) of systolic PAP was 62 (19) mm Hg, which dropped to 44.7 (13.7) and 36.7 (9.7) mm Hg immediately after the procedure and 3 months afterward, respectively (P<0.001 for both measurements).

Table 1. Demographic, clinical, and echocardiographic findings of the study population (N=33)

Characteristics	Mitral Stenosis Treatment Group (N=33)		*P*
	MVR (n=18)	PTMC (n=15)	
Age (y) mean (SD)	48.6(11.5)	53.5(12.2)	0.2
Gender, F/M, count (%)	11(47.8)/4(40)	12(52.2)/6(60%)	0.5
NYHA class, count (%)			
I-II,II	7(46.6)	8(44.4)	0.1
II-III,III	6(40)	7(38.9)	
IV	2(13.3)	3(16.7)	
Orthopnea, count (%)	4(27)	8(44.4)	0.04
Palpitation, count (%)	6(40)	7(38.9)	0.2
Chest pain, count (%)	2(13.3)	3(16.7)	0.6
Concomitant CAD, count (%)	1(6.7)	1(5.6)	0.3
LVEF, %, mean (SD)	49.6(7.2)	47.7(7.1)	0.3
MVA,cm^2, median (IQR)	0.7(0.6-1.1)	0.9(0.7-1)	0.5
Baseline PASP	65.7(25)	59(12)	0.3
PASP after the procedure	46(13)	43.6(13.8)	0.6
PASP 3 months after the procedure	38.3(11.6)	35.3(7.8)	0.4

NYHA, New York Heart Association; LVEF, Left ventricular ejection fraction; MVA, Mitral valve area; PASP, Pulmonary artery systolic pressure

Cardiac magnetic resonance findings

Table 2 shows the CMR findings in our study population. There was no relationship between the PA distensibility index and systolic PAP at baseline, immediately after the procedure, or 3 months after the treatment. There was also no difference between the patients who had a greater drop in their systolic PAP (systolic PAP < 35 mm Hg or > 50% decrease in systolic PAP) and those who did not regarding the PA distensibility (P = 0.3).

The PA peak velocity was significantly higher in the patients who had > 50% drops in their systolic PAP 3 months after the treatment compared to the baseline (mean [SD] PA peak velocity [cm/sec] = 76.2 [20] vs 60 [16.7]) (P = 0.04).

The PA forward volume and the PA forward flow showed no association with baseline systolic PAP or systolic PAP after the treatment. However, in the subgroup analysis, the PA forward flow showed a significant negative correlation with systolic PAP 3 months after the procedure in the PTMC group (P = -0.6 and P = 0.01).

The multivariable analysis was performed using binary logistic regression with the low likelihood backward elimination method to assess whether any CMR findings might predict > 50% drops in systolic PAP 3 months after MS relief independently. This analysis showed that none of the above-mentioned CMR findings was an independent predictor of a more systolic PAP decline. The same result was observed when we considered a systolic PAP < 35 mm Hg 3 months after MS relief as the end point in the multivariable analysis.

Table 2. Cardiac magnetic resonance findings in the study population (N=33)

Characteristics	Mitral Stenosis Treatment Group (N=33)		*P*
	PTMC N=15	MVR N=18	
Distensibility, %,mean (SD)	21(8)	21(9)	0.9
PA peak velocity, cm/sec, mean (SD)	65.7(21.5)	59.8(17.4)	0.3
PA flow per minute, ,mean (SD)	6(1.7)	5.8(1.3)	0.7
PA forward Volume mean (SD)	80.6(33)	75.5(19.2)	0.5

PA, Pulmonary artery

DISCUSSION

In the present study, we could not find any relationship between PA hemodynamic measures in CMR and systolic PAP in our severe MS patients with PH in the multivariate analysis and none of the CMR hemodynamic measures was independently correlated with systolic PAP. It is, therefore, not useful for predicting PH reversal after MS relief.

Recently, many investigators have focused on CMR findings in PH in order to better manage approaches and follow-ups. [5, 7, 8, 10-19] Several studies in idiopathic pulmonary arterial hypertension (IPAH) have shown a significant relationship between PA peak velocity, distensibility, and PA forward flow and hemodynamic findings including vasoreactivity. [10, 11, 13, 14, 16] We aimed to assess such correlations in patients with MS and PH as another category of PH. However, despite a good correlation between PAP and PA distensibility as well as PA velocity in CMR among the patients with IPAH in some studies, systolic PAP at baseline as well as post procedure was not associated with these variables in our study population.

The only positive finding of the present study was a higher PA peak velocity in the patients who had > 50% drops in their systolic PAP 3 months after MS relief, which showed a higher reversibility tendency in this group of patients. Laffon et al [15, 16] studied IPAH patients and showed that those with a higher PA velocity were more probable to be vasoreactive to pulmonary vasodilators in the vasoreactive challenge test. A similar mechanism can be considered for our study population. However, the multivariable study demonstrated that none of these factors, including PA peak velocity, was independently correlated with a higher drop in systolic PAP. The different pathophysiologic mechanism for PH in MS setting (post capillary PH) might be responsible for this finding.

On the other hand, our sample size was small and we considered systolic PAP measured by echocardiography. Our results would have been augmented had we been able to consider catheterization data, including mean PAP, pulmonary vascular resistance, and trans-pulmonary gradient.

CONCLUSIONS

Considering the limitation of this study, we would recommend that future studies investigate the association between catheterization data as gold standard and CMR findings in different categories of PH. These studies may shed some light on the pathophysiologic mechanisms in PH and enhance the management and follow-up of this group of patients.

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