

Net atrioventricular compliance can predict persistent pulmonary artery hypertension after percutaneous mitral balloon commissurotomy

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Background: Pulmonary hypertension is a common complication of rheumatic mitral stenosis (MS). Patients with similar mitral valve (MV) areas may have different pulmonary artery pressures. Net atrioventricular compliance (Cn) was found to play an important role in the development of pulmonary hypertension.

Aim: To test the value of Cn in predicting persistent pulmonary artery hypertension (PPAH) after percutaneous mitral balloon commissurotomy (PMBC).

Patients and Methods: Eighty patients with severe MS, suitable for PMBC were included in the study. We excluded patients with contraindication to PMBC, atrial fibrillation, failure of PMBC, and restenosis. All patients had undergone electrocardiography, echocardiography with measurement of MV area, systolic pulmonary artery pressure (SPAP), and Cn, PMBC, and follow-up echocardiography.

Results: Patients were divided into two groups: Group I: Cn < 4.2 mL/mmHg (36 patients), Group II: Cn ≥ 4.2 mL/mmHg (44 patients). Group I patients had significantly higher SPAP, and significantly lower SPAP reduction. Sensitivity of Cn < 4.2 mL/mmHg in prediction of PPAH was 88.9%, specificity was 88.6%, and accuracy was 88.8%. Independent predictors for PPAH were baseline Cn ($p = 0.0027$), and Cn improvement after PMBC ($p = 0.0085$). There was a significant negative correlation between Cn and baseline SPAP ($r = -0.349$, $p = 0.0015$), and a significant positive correlation between Cn and percent SPAP reduction ($r = 0.617$, $p < 0.00001$).

Conclusion: Measuring Cn can predict PPAH in MS patients after PMBC. It also may add value in evaluating MS patients undergoing PMBC and may help in predicting their prognosis.

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Abbreviations

MS	mitral stenosis
MV	mitral valve
Cn	net atrioventricular compliance
PMBC	percutaneous mitral balloon commissurotomy
PPAH	persistent pulmonary artery hypertension
LA	left atrial
LVEDD	left ventricular end-diastolic dimension
LVESD	left ventricular end-systolic dimension
FS	fraction of shortening
EF	ejection fraction
RA	right atrial
RVEDV	right ventricular end diastolic volume
TMG	trans-mitral gradient
SPAP	systolic pulmonary artery pressure
EOA	effective orifice area
VTI	velocity time integral
MR	mitral regurgitation
ROC	receiver operating characteristic
AUC	area under the curve
CI	confidence interval

Introduction

Pulmonary hypertension is a common complication of rheumatic mitral stenosis (MS), which tends to have a bad effect on functional state and prognosis in these patients [1]. The severity of MS is an important factor in the development of pulmonary hypertension. However, patients with similar mitral valve (MV) areas may have different pulmonary artery pressure (PAP) [2].

Net atrioventricular compliance (Cn) plays an important role in the development of pulmonary hypertension and is responsible, at least in part, for the presence and severity of symptoms in MS patients [3].

Percutaneous mitral balloon commissurotomy (PMBC) is associated with reduction of PAP and other hemodynamic parameters [4]. Previous studies have shown excellent short-term results following PMBC in patients with different degrees of pulmonary hypertension [5]. Other studies have shown that PAP may fail to drop in many cases despite successful dilatation of MV and significant increase in MV area [6].

The aim of our study was to test the value of Cn in prediction of persistent pulmonary artery hypertension (PPAH) after PMBC.

Patients and methods

This prospective study was carried out in the Cardiology Department, Zagazig University Hospitals. We started our study with 88 rheumatic MS patients (36 male and 52 female, with a mean age

of 40.2 ± 8.5 years). All patients were diagnosed as pure, isolated, severe MS with $MV \leq 1.5 \text{ cm}^2$, and were suitable for performing PMBC according to the American Heart Association/American College of Cardiology guidelines for the management of patients with valvular heart disease [7].

Patients were excluded from the study if they had one or more of the following: (1) any contraindication to PMBC [7]; (2) atrial fibrillation or flutter; (3) more than mild aortic or pulmonary stenosis, mitral or aortic regurgitation grade >2 , or severe tricuspid regurgitation; (4) significant congenital heart disease that may affect pulmonary artery pressure; or (5) history of hypertension or coronary artery disease.

The study protocol was approved by the Institutional Review Board of the Faculty of Medicine, Zagazig University. After giving informed written consent, all patients underwent the following.

- (1) Complete 12-lead electrocardiography.
- (2) Echocardiography. Echocardiographic and Doppler studies were performed for all patients by two expert operators unaware of each other's results or other data. Echocardiograms were performed using a GE VIVID E9 machine (General Electric Healthcare, USA) with 2.5-MHz transducers. All views and measures were obtained at rest with the patient in the left lateral position. From the two-dimensional guided M mode of the left parasternal long-axis view, we measured left atrial (LA) diameter, left ventricular end-diastolic dimension (LVEDD), left ventricular end-systolic dimension (LVESD), fractional shortening (FS), and ejection fraction (EF), and we measured LA volume, right atrial (RA) volume, and right ventricular end diastolic volume (RVEDV) from the apical two-chamber and four-chamber views [8]. MV areas were measured by planimetry from two-dimensional images in the parasternal short axis view. Peak and mean transmitral gradients (TMGs) were measured.

As all the patients in our study had mild or moderate tricuspid regurgitation, systolic PAP (SPAP) was calculated from the peak continuous wave Doppler signal of tricuspid regurgitant jet velocity and adding a constant value for RA pressure to it (10 mmHg) [9].

As none of our patients had more than mild mitral or aortic regurgitation, MV effective orifice area (EOA) was determined by the continuity equation using the LV outflow tract area multiplied by its velocity time integral (VTI) and divided by the VTI of the MV flow during diastole [10].

Cn was calculated by dividing EOA over deceleration rate (dV/dt) of the mitral velocity profile (E-wave downslope) and multiplying the result by 1270, according to following formula [3]:

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