FULL LENGTH ARTICLE

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.**

Keywords: Left atrium, Mitral stenosis, Mitral valvuloplasty, Pulmonary hypertension

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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 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]:

$$Cn(mL/mmHg) = 1270(\text{mitral EOA/E-wave deceleration rate})$$

(3) Percutaneous mitral balloon commissurotomy. PMBC was performed in every patient using the double balloon technique. Balloon size was calculated by dividing the effective balloon dilating area by the body surface area, and combination of two separate balloon catheters was used in all patients [10].

(4) Echocardiography was repeated for every patient on the next day after PMBC, after 1 month and 6 months. In the follow-up echocardiography, we measured MV area by planimetry, SPAP, and Cn, and searched for the presence and severity of mitral regurgitation (MR) for every patient. Immediate successful PMBC was defined as MV area >1.5 cm^2 and MR grade $\leq$2 [11]. Echocardiographic mitral valve restenosis was defined as a MV area $\leq$1.5 cm^2 with loss of initial gain in MV area $\geq$50% [11]. Patients with immediate failure of PMBC (either by failure of dilatation or development of MR grade >2), MV restenosis, and nonadherence to follow-up were excluded from our study.

PPAH was defined as SPAP $\geq$40 mmHg after 6 months in the absence of MV restenosis.

All data were analyzed using SPSS for Windows version 20.0 (IBM, Armonk, NY, USA). Correlations between different variables were investigated by Pearson correlation analysis. The logistic regression analysis was evaluated by the Hosmer–Lemeshow goodness-of-fit test. A p value <0.05 was regarded as being statistically significant.

We repeated the echocardiograms for 20 patients to assess the intraobserver variability. Interobserver and intraobserver variability were calculated by dividing the difference between the two sets of measurements, by the mean of the two observations.

Results

PMBC was successfully performed in 83 patients (94.3%), and we failed to dilate the MV satisfactory in five patients (5.7%) and excluded them from our study. One patient was excluded because of MV restenosis during follow-up and two patients were excluded because of nonadherence to follow-up.

The study group included 80 patients (33 male and 47 female, with a mean age of 39.6 ± 8.3 years).

The receiver operating characteristic curve was made to analyze the cutoff point of Cn for the prediction of PPAH (Fig. 1). For Cn < 4.2 mL/mmHg, area under the receiver operating characteristic curve was 0.875. Accordingly, our patients were divided into two groups. Group I: patients with Cn < 4.2 mL/mmHg. This group included 36 patients, 16 male and 20 female, with a mean age of 41.1 ± 7.85 years. Group II: patients with Cn $\geq$ 4.2 mL/mmHg. This group included 44 patients, 17 male and 27 female, with a mean age of 38.4 ± 7.93 years.

As shown in Table 1, there was no significant difference between the two groups with regard to age, sex, heart rate, New York Heart Association functional class, LA diameter and volume, RA volume, RVEDV, LVEDD, LVESD, FS, EF, peak and mean TMG, EOA, and MV area before, immediately after PMBC, and after 1 month and 6 months.

In patients with Cn < 4.2 mL/mmHg, Cn was significantly lower at baseline, after PMBC, after 1 month and 6 months, and the percentage Cn improvement was significantly higher (p < 0.00001 each), SPAP was significantly higher before PMBC (p = 0.0078), immediately after PMBC, and after 1 month and 6 months (p < 0.00001 each).

The absolute reduction of SPAP and the percent reduction were significantly lower in patients with Cn < 4.2 mL/mmHg (p < 0.00001 each). There were

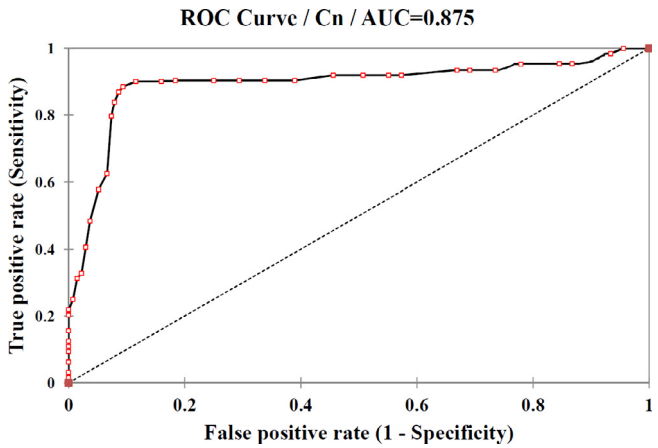

Figure 1. ROC curve for Cn < 4.2 mL/mmHg with persistent pulmonary arterial hypertension. AUC = area under the curve; Cn = net atrioventricular compliance; ROC = receiver operating characteristic.

Table 1. Clinical and echocardiographic data

	Cn < 4 mL/mmHg (n = 36)	Cn ≥ 4 mL/mmHg (n = 44)	p
Age (y)	41.1 ± 7.85	38.4 ± 7.93	0.132
Sex			
Male	16 (44.4%)	17 (38.6%)	0.599
Female	20 (55.6%)	27 (61.4%)	
NYHA class			
II	7 (19.4%)	16 (36.4%)	0.153
III	28 (77.8%)	28 (63.6%)	
IV	1 (2.8%)	0	
Heart rate (beat/min)	75.3 ± 10.4	72.5 ± 9.6	0.219
LA diameter (mm)	50.1 ± 5.42	48.9 ± 4.82	0.304
LA volume (mL)	126.4 ± 23.8	120.2 ± 28.3	0.29
RA volume (mL)	72.3 ± 16.5	66.8 ± 17.1	0.149
RVEDV (mL)	121.1 ± 23.6	112.4 ± 22.8	0.101
LVEDD (mm)	46.3 ± 6.43	47.1 ± 5.61	0.559
LVESD (mm)	30.1 ± 3.88	30.7 ± 4.33	0.516
FS (%)	35.4 ± 3.47	34.8 ± 3.86	0.587
EF (%)	65.3 ± 4.45	64.4 ± 4.29	0.535
Peak TMG (mmHg)	25.4 ± 11.6	23.9 ± 10.5	0.572
Mean TMG (mmHg)	13.2 ± 6.12	12.1 ± 4.82	0.331
EOA (cm^2)	0.87 ± 0.285	0.92 ± 0.279	0.715
Cn (mL/mmHg)			
Baseline	3.13 ± 0.47	4.81 ± 0.51	< 0.00001
After PMBC	3.14 ± 0.51	5.23 ± 0.62	< 0.00001
After 1 month	3.22 ± 0.57	5.51 ± 0.68	< 0.00001
After 6 months	3.45 ± 0.61	6.21 ± 0.65	< 0.00001
Improvement (%)	13.9 ± 7.52	26.5 ± 9.86	< 0.00001
MV area (cm^2)			
Baseline	0.99 ± 0.213	1.03 ± 0.198	0.318
After PMBC	1.98 ± 0.288	2.09 ± 0.333	0.104
After 1 month	1.92 ± 0.321	2.01 ± 0.384	0.232
After 6 months	1.89 ± 0.283	1.96 ± 0.374	0.301
SPAP (mmHg)			
Baseline	58.3 ± 14.6	49.9 ± 12.5	0.0078
After PMBC	44.4 ± 13.7	28.6 ± 8.78	< 0.00001
After 1 month	44.5 ± 14.9	27.2 ± 9.44	< 0.00001
After 6 months	47.3 ± 15.12	25.5 ± 9.18	< 0.00001
Reduction	13.9 ± 7.54	21.2 ± 8.25	< 0.00001
Reduction (%)	23.7 ± 12.67	42.7 ± 13.88	< 0.00001
PPAH	31 (86.1%)	6 (13.6%)	< 0.00001

Data are expressed as mean ± standard deviation or n (%). Cn = net atrioventricular compliance; EF = ejection fraction; EOA = effective orifice area; FS = fractional shortening; LA = left atrial; LVEDD = left ventricular end-diastolic dimension; LVESD = left ventricular end-systolic dimension; MV = mitral valve; NYHA = New York Heart Association; PMBC = percutaneous mitral balloon commissurotomy; PPAH = persistent pulmonary artery hypertension; RA = right atrial; SPAP = systolic pulmonary artery pressure; TMG = transmitral gradient.

Table 2. Validity Cn < 4.2 mL/mmHg in predicting PPAH

	Cn < 4.2 mL/mmHg		Cn ≥ 4.2 mL/mmHg		Total
PPAH	32		5		37
No PPAH	4		39		43
Total	36		44		80
Sensitivity	Specificity	PPV	NPV	Overall accuracy	κ	p
88.9%	88.6%	86.5%	90.7%	88.8%	0.814	0.0001

Cn = net atrioventricular compliance; NPV = negative predictive value; PPAH = persistent pulmonary artery hypertension; PPV = positive predictive value.

Table 3. Logistic regression analysis for independent predictors of PPAH

Variables	Odds ratio	95% CI	*p*
Baseline Cn	6.19	4.59–7.83	0.0027
Cn improvement	5.41	4.23–6.59	0.0085
Baseline SPAP	2.99	1.65–4.33	0.0771
LA volume	2.45	1.04–3.86	0.103
Baseline MV area	1.58	0.83–2.32	0.314

CI = confidence interval; Cn = net atrioventricular compliance; LA = left atrial; MV = mitral valve; PPAH = persistent pulmonary artery hypertension; SPAP = systolic pulmonary artery pressure.

also significantly more patients with PPAH among patients with Cn < 4.2 mL/mmHg ($p < 0.00001$).

The validity of Cn < 4.2 mL/mmHg in prediction of PPAH is shown in Table 2. Sensitivity was 88.9%, specificity was 88.6%, positive predictive value was 86.5%, negative predictive value was 90.7%, overall accuracy was 88.8%, and κ was 0.814, $p = 0.0001$.

Regression analysis of the relation of different parameters to PPAH is shown in Table 3. The independent predictors for PPAH after successful PMBC were Cn (odds ratio = 6.19, 95% confidence interval = 4.59–7.83, $p = 0.0027$) and Cn improvement (odds ratio = 5.41, 95% confidence interval = 4.23–6.59, $p = 0.0085$). Other echocardiographic and clinical parameters were nonsignificant predictors for PPAH, including: baseline SPAP, LA volume, baseline MV area, mean TMG, RA volume, peak TMG, EOA, RVEDV, age, heart rate, sex, LV diameter, LVEDD, FS, EF, and LVESD.

Fig. 2 shows the significant negative correlation between Cn and baseline SPAP ($r = -0.349$, $p = 0.0015$). Fig. 3 shows the significant positive correlation between Cn and the percent SPAP reduction ($r = 0.617$, $p < 0.00001$).

Inter- and intraobserver variability for different echocardiographic parameters ranged from 2.2% to 8.3%. For Cn, inter- and intraobserver variability was 6.5 ± 3.2% and 7.9 ± 3.3%, respectively. For MV area, inter- and intraobserver variability was 5.8 ± 3.1% and 6.6 ± 2.8%, respectively. For SPAP, inter- and intraobserver variability was 2.4 ± 1.1% and 3.1 ± 1.8%, respectively.

Discussion

Our results have shown a strong relationship between SPAP reduction after PMBC and Cn. Cn ≥ 4.2 mL/mmHg was able to predict SPAP reduction after PMBC by > 35% of baseline, with good accuracy.

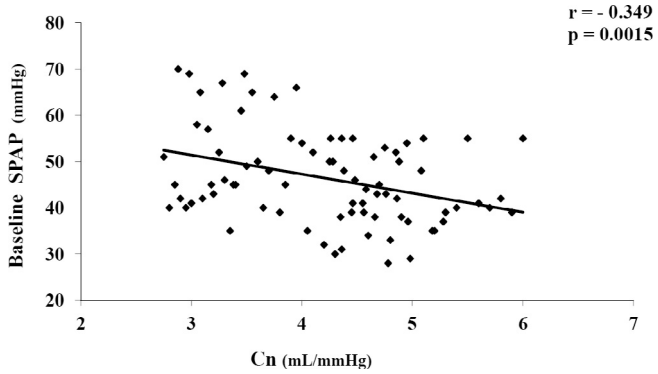

Figure 2. Correlation between Cn and baseline SPAP. Cn = net atrioventricular compliance; SPAP = systolic pulmonary artery pressure.

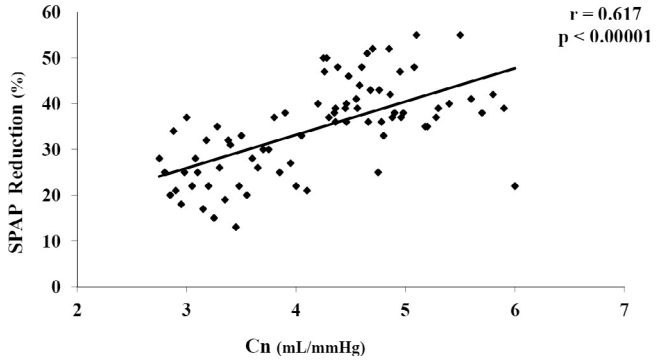

Figure 3. Correlation between Cn and SPAP reduction. Cn = net atrioventricular compliance; SPAP = systolic pulmonary artery pressure.

Although they are simple and commonly used for assessment of MS patients, the hemodynamic effects of conventional measures like MV area and TMG are markedly variable. Izgi et al. [12] have found that mitral valve resistance is superior to MV area and mean TMG in predicting pulmonary hypertension.

Pulmonary hypertension tends to improve after PMBC. However, SPAP remains high in many patients even after successful PMBC [6].

Previous studies have found a lack of association between the increase in MV area and the decrease in SPAP [13–15]. This discrepancy between MV gain and SPAP decrease has raised attention to other factors that might have an effect on pulmonary hemodynamics.

In an attempt to study the effect of different factors on the improvement of the hemodynamics of pulmonary vasculature, Gamra et al. [16] found that pulmonary vascular resistance failed to normalize after successful PMBC in 43% of patients. They found that older age, higher Wilkin's echocardiographic score, smaller MV area, and higher mean PAP at baseline predicted failure of pulmonary vascular resistance normalization [16].

Also, Nair et al. [6] found that persistence of pulmonary hypertension after successful treatment was related to age, smaller MV area, advanced rheumatic heart disease, higher Wilkin's echocardiographic score, higher mean and systolic blood pressure at baseline, and presence of atrial fibrillation. However, in our study, baseline Cn and Cn improvement were the only independent predictors for PPAH. This might be explained by the smaller number of patients in our study.

The mechanism of pulmonary hypertension in MS patients is complex. The increased severity of MS, with the resultant increase in LA pressure, plays an important role [17]. However, other factors have important roles in the development of pulmonary hypertension; for example, vasoactive substances such as endothelin [18] and adrenomedullin [19].

LA compliance is a major contributing factor determining PAP in MS patients [2]. It has been shown that Cn derived by Doppler echocardiography has the ability to represent LA compliance when there is no additional cardiovascular condition that impairs LV diastolic function, such as in MS [20].

The above data supports our results, as we found that SPAP at baseline, after PMBC, and after 1 month and 6 months, was significantly higher and the SPAP reduction after PMBC was significantly lower in patients with low Cn; a representative of LA compliance.

Many investigators have previously studied the effect of Cn on different clinical and hemodynamic parameters in MS patients. Nunes et al. [21] found that Cn was a powerful predictor of adverse outcome, and the need for intervention even in asymptomatic patients and in those with a wide spectrum of MS severity. These results are concordant with our results, as we found that Cn was a significant predictor of PPAH, which is associated with poor outcome [6].

Salem Omar et al. [22] have found that Cn can even affect the measurement of MV area when measured by the method of pressure half time and this effect was obvious in patients with Cn < 4 mL/mmHg.

Choi et al. [23] found that Cn was a good predictor of exercise capacity in MS patients. In our study, there was a tendency toward higher New York Heart Association class in patients with low Cn. However, this tendency was not significant; probably because of the small number of patients in our study.

Kim et al. [24] found that Cn was useful for prediction of the future occurrence of MV replacement or PMBC, with particular utility in patients with moderate MS.

Li et al. [25] found that Cn was a major independent determinant of LA pressure and PAP measured by catheterization. These results are similar to ours, as we found that Cn was a good determinant of SPAP as measured by echocardiography, and its decrease after PMBC.

Schwammenthal et al. [3] found a strong negative correlation between Cn and increase in SPAP with exercise. They also found that patients with lower Cn were more symptomatic.

In concordance with the results of the previous studies, we found that patients with low Cn had significantly higher SPAP before and after PMBC, and after 1 month and 6 months.

To the best of our knowledge, there is no previous study on the relation between Cn and PPAH after PMBC. Patients with PPAH after PMBC usually have poor prognosis, and require intense and more frequent follow-up [6]. So, it may be of value to search for predictors of this condition.

Our study had some limitations. First, there was a small number of patients. However, this was because of the small number of patients with pure MS suitable for PMBC. Second, the study was conducted in a single center.

In conclusion, Cn can predict PPAH in MS patients after PMBC with good predictive value. Measuring Cn, which is a simple, feasible, and noninvasive marker of LA compliance, may add value to evaluating patients with MS undergoing PMBC and may help to predict their prognosis.

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