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ORIGINAL ARTICLE

Assessment of interatrial dyssynchrony by Tissue Doppler Imaging in mitral stenosis: Effect of afterload reduction after balloon mitral valvuloplasty

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KEYWORDS

Mitral stenosis;
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Abstract *Background:* The predisposition to atrial fibrillation (AF) in mitral stenosis (MS) has been demonstrated with several electrocardiographic (increased P-wave dispersion) and echocardiographic parameters (atrial electromechanical delay). After percutaneous mitral balloon valvuloplasty (PMBV), the improvement in echocardiographic parameters related to AF risk is unknown. We aimed to assess the interatrial electromechanical coupling by Tissue Doppler Imaging (TDI) echocardiography in MS before and after PBMV.

Patients and methods: This study included 45 patients with moderate to severe MS who underwent successful PMBV without complication at our clinic and 20 healthy volunteers as a control group. We compared the two groups in regard to clinical, electrocardiographic and echocardiographic features. The patients with MS were also evaluated one week after PMBV. Interatrial electromechanical delay (EMD) was measured by TDI before and after PMBV and we compared the results.

Abbreviations: A′ wave, peak late annular velocity by TDI; AF, atrial fibrillation; BSA, body surface area; LA, left atrium; LAA, left atrial area; LAD, left atrial dimension; LAEF, left atrial ejection fraction; LAEI, left atrial expansion index; LAV, left atrial volume; MS, mitral stenosis; MV, mitral valve; MVA, mitral valve area; P–A′, the time interval from the initiation of the P wave on the ECG until the beginning of the late diastolic TDI signal at the lateral border of the annulus; PBMV, percutaneous balloon mitral valvuloplasty; *P*-max, maximum P-wave duration; *P*-min, minimum P-wave duration; PWD, P-wave dispersion; SPAP, systolic pulmonary artery pressure; TDI, Tissue Doppler Imaging

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Results: The interatrial EMD (56.4 ± 15.8 vs. 34.6 ± 7.2 ms, $p < 0.001$) was higher in patients with MS as compared to healthy individuals. The maximum P-wave duration (P-max) and P-wave dispersion (PWD) showed significant positive correlation with the interatrial EMD ($r = 0.37$, $p < 0.05$ and $r = 0.41$, $p < 0.05$ respectively). There was a highly significant decrease in the interatrial EMD (56.4 ± 15.8 vs. 38.3 ± 10.4 ms, $p < 0.001$) one week after PBMV.

Conclusion: The current study showed significant increase in the interatrial EMD in patients with moderate to severe MS. These changes improved significantly after PMBV.

1. Introduction

Atrial electromechanical abnormalities have been reported to promote atrial fibrillation (AF).[1,2] Özer et al.[3] used Tissue Doppler Imaging (TDI) and surface ECG to show interatrial electromechanical delay (EMD) in patients with mitral stenosis (MS). Percutaneous balloon mitral valvuloplasty (PBMV) has become the procedure of choice for isolated, uncomplicated MS with favorable morphology.[4] The initial objectives of the procedure are to increase the cross-sectional valve area and simultaneously to reduce the trans-mitral pressure gradient, left atrial pressure, and mean pulmonary artery pressure.[5,6] Atrial electromechanical coupling time [the time interval from the onset of P wave on surface ECG to the beginning of A′ wave interval with TDI (PA′)] is measured and interatrial EMD can be calculated from PA′ values of selected regions.[3]

1.1. Aim of the work

We aimed to assess the interatrial electromechanical coupling by Tissue Doppler Imaging (TDI) echocardiography in MS before and after PBMV.

2. Patients and methods

The study conducted in Cardiology Department, Faculty of Medicine, Zagazig University from August 2010 to March 2014 included 65 human individuals: 17 males, 48 females who were divided into 2 groups: Group I (Rheumatic Mitral Stenosis group): includes 45 patients; (33 females and 12 males, aged 30.1 ± 8.99 years, with a mean mitral valve area (MVA) of 0.99 ± 0.15 cm^2) who underwent successful PBMV without complications and Group II (Control group): includes 20 healthy volunteers (15 females and 5 males, aged 30.6 ± 7.8 years). The Group II did not have any known cardiovascular disease or cardiovascular risk factors. All patients with MS were New York Heart Association class ⩾ II and were eligible for PBMV. The patients with left ventricular dysfunction, hypertension, diabetes mellitus, left ventricular hypertrophy, coronary artery disease, significant pulmonary disease, or additional moderate or severe valvular heart disease other than secondary tricuspid regurgitation were excluded from the study. All patients were in sinus rhythm during evaluation. The study patients underwent comprehensive echocardiographic examinations one day before and one week after PBMV. Mitral valve anatomy was scored according to the Wilkins echo scoring system.[7] After excluding the contraindications for the procedure with transthoracic and transesophageal echocardiography, PBMV was performed. Also 12-lead ECGs were recorded for each study patient one day before and one week after PBMV. The study protocol was approved by the institutional ethics committee and all patients gave informed consent.

2.1. Echocardiography

All echocardiographic examinations were performed with the VIVID 9 ultrasound system (GE, Horten, Norway) using 2.5–3.5 MHz transducers. In addition to standard M-mode, two-dimensional and Doppler measurements evaluating cardiac chambers and the severity of MS and TDI were used. During echocardiographic evaluation a continuous one-lead ECG recording was provided. Data were recorded from the average of three cardiac cycles. Left ventricle (LV) end-diastolic, end-systolic diameters and left atrial (LA) end-systolic diameter were measured with M-mode in the parasternal long axis view according to American Society of Echocardiography guidelines. MVA was measured using the planimetry method in the parasternal short axis view. From the apical four-chamber view, peak and mean trans-mitral gradients in (mmHg) were measured by using continuous wave Doppler echocardiography according to the modified Bernoulli equitation. Systolic pulmonary artery pressure (SPAP) was calculated from the peak continuous wave Doppler signal of tricuspid regurgitation jet velocity and adding the estimated right atrial pressure to this value. The Sampson's biplane modified method was used to estimate the LA volume. The following LAVs were measured: the maximal volume (V_{max}) during left ventricular end-systole just before mitral valve opening, the minimal volume (V_{min}) just before mitral valve closure on the simultaneously recorded ECG.[8] The LA volume index was calculated by dividing the LA volume by body surface area. LA ejection fraction (LAEF) is defined as $(V_{max} - V_{min})/V_{max} \times 100\%)$.[8–10] LA expansion index (LAEI) is defined as $(V_{max} - V_{min})/V_{min} \times 100\%)$.[8–10]

TDI echocardiography was performed with transducer frequencies of 3.5–4.0 MHz by adjusting the spectral pulsed Doppler signal filters until a Nyquist limit of 15–20 cm/s was reached and using the minimal optimal gain. The monitor sweep speed was set at 100 mm/s. In the apical four-chamber view the pulsed Doppler sample volume was placed on the LV lateral mitral annulus and tricuspid annulus. The time interval from the onset of P wave on the surface electrocardiogram to the beginning of the late diastolic wave, which is referred to as PA′, was obtained from lateral mitral annulus

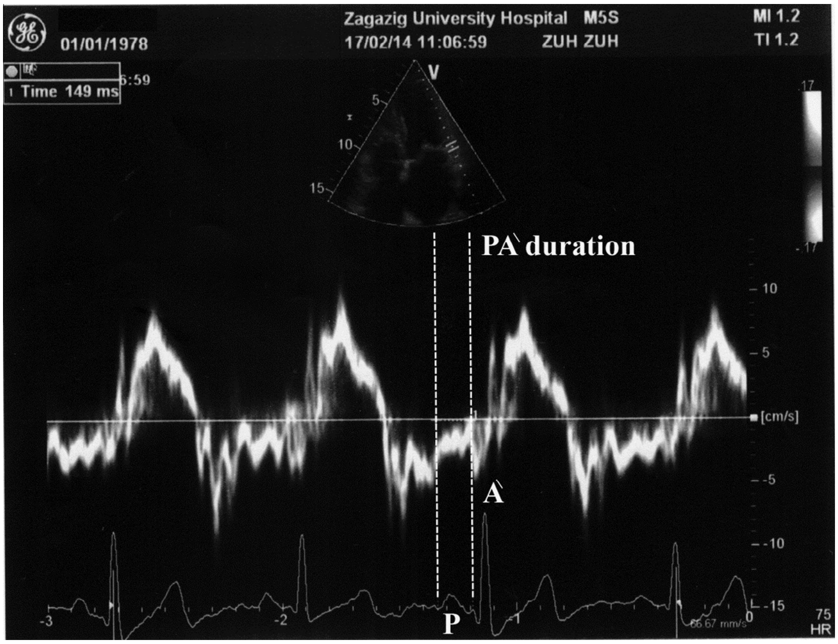

Figure 1 Measurement of the time interval from the onset of P wave on surface ECG to the beginning of A′ wave with tissue Doppler echocardiography (PA′).

Table 1 The clinical, echocardiographic and electrocardiographic parameters of the studied groups.

	Group I (MS) (n = 45)	Group II (Control) (n = 20)	P-value
Age (years)	30.1 ± 8.99	30.6 ± 7.8	0.22
Gender (n%)			
Male	12 (26.7%)	5 (25%)	0.88
Female	33 (73.3%)	15 (75%)	
BSA	1.72 ± 0.1	1.71 ± 0.1	0.37
Systolic BP (mmHg)	115.7 ± 8.4	114.5 ± 6.7	0.5
Diastolic BP (mmHg)	68.8 ± 6.4	71.2 ± 7.4	0.17
HR (beats/min)	79 ± 9.8	76.2 ± 6.5	0.24
LVEDD (mm)	46.1 ± 5.3	41.5 ± 4.4	0.0013
LVESD (mm)	28.8 ± 4.2	26.8 ± 3.3	0.06
IVST (mm)	9.25 ± 0.8	8.8 ± 0.9	0.06
PWT (mm)	8.95 ± 1	8.56 ± 1	0.11
EF (%)	67.2 ± 5.76	65.9 ± 3.8	0.3
LAD (mm)	47 ± 3.7	30.4 ± 3.6	< 0.001
LAV_{max} (ml)	109.7 ± 29.8	41.7 ± 6.9	< 0.001
Ind. LAV_{max} (ml/m^2)	63.8 ± 16.96	24.4 ± 4.5	< 0.001
LAEF (%)	30.9 ± 7.7	49.3 ± 6.2	< 0.001
LAEI (%)	46 ± 19.3	104.1 ± 34.9	< 0.001
Lateral mitral annulus P–A′ (ms)	117.9 ± 16.7	87.9 ± 7.5	< 0.001
Tricuspid annulus P–A′ (ms)	60.8 ± 10	53.3 ± 53	0.08
Interatrial EMD (ms)	56.4 ± 15.8	34.6 ± 7.2	< 0.001
ECG findings			
P_{max} (ms)	137.4 ± 12.1	102.4 ± 6.1	< 0.001
P_{min} (ms)	72 ± 12.9	77.7 ± 4.1	0.056
PWD (ms)	64.4 ± 13.5	24.6 ± 4.7	< 0.001

BP: blood pressure, BSA: body surface area, HR: heart rate, EF: ejection fraction, interatrial EMD: the difference between the P–A′ (lateral) and P–A′ (tricuspid) time intervals, IVST: interventricular septum thickness, LAD: left atrial diameter, LAEF: left atrial ejection fraction, LAEI: left atrial expansion index, LAV: left atrial volume, LVEDD: left ventricle end diastolic diameter, LVESD: left ventricle end systolic diameter, P–A: the time interval from the initiation of the P wave on the ECG until the beginning of the late diastolic TDI signal at the lateral border of the annulus, P_{max}: maximum P-wave duration, P_{min}: minimum P-wave duration, PWD: P-wave dispersion, PWT: posterior wall thickness.

P value > 0.05 = non-significant.

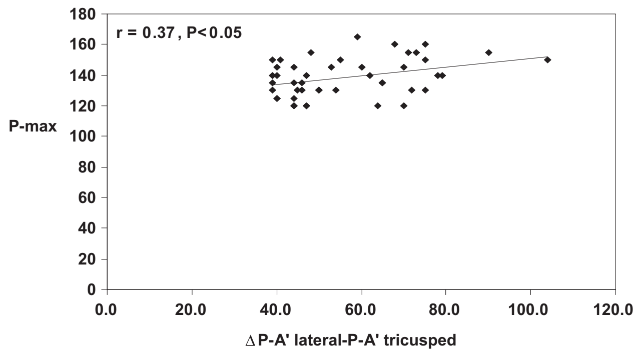

Figure 2 Pearson's correlation between interatrial EMD and *P*-max in MS group. Abbreviation: Δ Lateral P–A′ – Tricuspid P–A′ (ms): The difference between the P–A′ (Lateral mitral) and P–A′ (Tricuspid) time intervals.

(lateral PA′) and tricuspid annulus (tricuspid PA′) Fig. 1. The difference between both lateral mitral PA′ and tricuspid PA′ was defined as interatrial electromechanical delay (EMD).[11]

2.2. Twelve-lead ECG

Twelve-lead ECGs were recorded at a rate of 25 mm/s in a supine position. P wave duration was measured from the onset to the offset of P wave. The measurements were made using a digital caliper and magnifying lens in order to improve accuracy. Maximum and minimum P-wave durations were determined from 12-lead ECGs. Subsequently, P-wave dispersion (PWD) was defined as the difference between maximum P-wave and minimum P-wave durations.[11]

2.3. Percutaneous balloon mitral valvuloplasty

Indications for PBMV were as follows: symptomatic patients with moderate or severe MS with favorable valve morphology and asymptomatic patients with moderate or severe MS and pulmonary hypertension (SPAP $\geqslant$ 50 mmHg at rest or 60 mmHg with exercise) with favorable valve morphology.[11]

Before PBMV, all patients were evaluated by transthoracic and trans-esophageal echocardiography in order to exclude the contraindications of the procedure such as moderate or severe mitral regurgitation, severe or bi-commissural calcification and presence of LA thrombus.[11]

An experienced cardiologist performed all PBMV procedures via a double balloon, multi-track technique in a fasting state utilizing percutaneous trans-femoral approach. Standard hemodynamic measurements of right and left heart pressures were evaluated during the procedure.[12]

2.4. Statistical analysis

Data checked, entered and analyzed by SPSS version 20. Data were expressed as mean ± SD for quantitative values, numbers and percentage for categorical variables. ANOVA (*f* test), paired-*t* test, Chi-square (x^2) and Pearson's correlation *r* were used when appropriate. We considered results statistically significant when *p* value $\leqslant 0.05$.

3. Results

The baseline demographic, clinical, echocardiographic and electrocardiographic parameters of Group I and Group II are shown in Table 1. There were no significant differences between Group I and Group II as regards age, gender, body surface area, blood pressure, LV end systolic diameter (LVESD) and LV function ($p > 0.05$). The LA diameter, LA volume, LA volume index and lateral mitral PA′ values were significantly higher in Group I ($p < 0.001$), but LAEF and LAEI were highly significant decrease in Group I ($p < 0.05$) as compared with Group II. Interatrial EMD was also highly significant longer in Group I as compared with Group II ($p < 0.001$). *P*-max duration and PWD were found highly significant increase in Group I as compared with Group II ($p < 0.001$). However, there was no statistical difference

Table 2 The effects of PMBV on echocardiographic and electrocardiographic parameters.

	Group I before PBMV	Group I after PBMV	*P*-value
MVA (cm^2)	0.99 ± 0.15	1.88 ± 0.2	< 0.001
Mean transmitral gradient (mmHg)	15.4 ± 3.55	5 ± 1.5	< 0.001
SPAP (mmHg)	56.7 ± 12.5	35.9 ± 5.2	< 0.001
MVA pHt. (cm^2)	1.05 ± 0.16	1.89 ± 0.2	< 0.001
LAD (mm)	47 ± 3.7	41.5 ± 3.02	< 0.001
LAV_{max} (ml)	109.7 ± 29.8	86.4 ± 26.2	< 0.001
Ind. LAV_{max} (ml/m^2)	63.8 ± 16.96	49.9 ± 14.8	< 0.001
LAEF (%)	30.9 ± 7.7	43.4 ± 5.4	< 0.001
LAEI (%)	46 ± 19.3	78.6 ± 17.4	< 0.001
Lateral mitral annulus P–A′ (ms)	117.9 ± 16.7	95.9 ± 12.7	< 0.001
Tricuspid annulus P–A′ (ms)	60.8 ± 10	57.2 ± 10.9	0.16
Interatrial EMD (ms)	56.4 ± 15.8	38.3 ± 10.4	< 0.001
ECG findings			
P_{max} (ms)	137.4 ± 12.1	115.6 ± 13	< 0.001
P_{min} (ms)	72 ± 12.9	71.7 ± 12.2	0.1
PWD (ms)	64.4 ± 13.5	47.4 ± 12.6	< 0.001

MVA: mitral valve area, SPAP: systolic pulmonary artery pressure.
P value > 0.05 = non-significant.

between the two groups regarding minimum P-wave duration (*P*-min) ($p > 0.05$).

A comparison of changes in electrocardiographic and echocardiographic parameters in Group I before and after PMBV is shown in Table 2. All patients underwent successful PMBV and statistically highly significant improvement occurred in MVA, trans-mitral mean gradient, pulmonary artery pressure, LA diameter, both LA volume and volume index, LAEF and LAEI. Accompanying these hemodynamic effects of the procedure, the ECG parameters of *P*-max duration and PWD showed a highly significant decrease one week after PMBV ($p < 0.001$). However, the procedure had no significant effect on minimum P-wave duration ($p > 0.05$). Lateral mitral PA′ values were found to be highly significantly decreased after PMBV ($p < 0.001$) but tricuspid PA′ interval was statistically not affected by the procedure (p > 0.05). In addition, interatrial EMD was highly significantly improved as compared to previous values ($p < 0.001$).

In correlation analysis, a significant positive correlation was detected between interatrial EMD and *P*-max duration ($r = 0.37$, $p < 0.05$) and PWD ($r = 0.41$, $p < 0.05$) Figs. 2 and 3 respectively.

The LA diameter, LAV_{max} and Ind. LAV_{max} had a positive association with interatrial EMD ($r = 0.31, p < 0.05; r = 0.33, p < 0.05$ and $r = 0.33, p < 0.05$, respectively) Table 3.

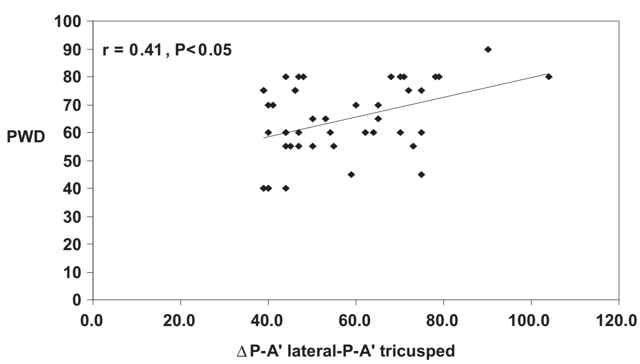

Figure 3 Pearson's correlation between interatrial EMD and PWD in MS group. Abbreviation: Δ Lateral P–A′ – Tricuspid P–A′ (ms): The difference between the P–A′ (Lateral mitral) and P–A′ (Tricuspid) time intervals.

Table 3 Correlations between interatrial EMD and other ECG and echocardiographic parameters in Group I.

	R	*P*
P_{max}	0.37	$\leq$0.05
PWD	0.41	$\leq$0.05
LAD	0.31	< 0.05
LAV_{max}	0.33	< 0.05
Ind.LAV_{max}	0.33	< 0.05

Ind. LAV_{max}: maximal left atrial volume indexed to BSA; LAD: left atrial dimension; *P*-max: maximum P-wave duration; PWD: P-wave dispersion.
P value > 0.05 = non-significant.

4. Discussion

Atrial conduction disorders are frequent for patients with mitral valve disease. The resultant electrophysiological and electromechanical abnormalities caused by atrial conduction abnormalities are associated with a higher risk of AF.[13] Both the hemodynamic changes that result in increased LA pressure and volume and the fibrotic process impairing atrial conduction are reported to be related to the higher risk of AF in these patients.[14]

TDI is a technology that allows direct and non-invasive measurement of myocardial velocities.[15] Atrial electromechanical delay is evaluated by TDI, a relatively novel noninvasive method for assessing atrial conduction time.[16]

This study aimed to assess the interatrial electromechanical coupling by TDI in mitral stenosis before and after PBMV. We included 45 patients with MS with mean MVA $0.99 \pm 0.15\,cm^2$, who underwent PBMV with improved MVA ($1.89 \pm 0.2\,cm^2$) and 20 healthy individuals as control group. All were examined by TTE with TDI to measure the electromechanical coupling.

We found significant increase in both *P*-max and PWD in MS group compared to control group ($p < 0.001$).

Our findings are in agreement with Krishnamoorthy and colleagues [17], who demonstrated that *P*-max and PWD increased in patients with MS and attributed this to LA hypertension that caused LA dilatation, fibrosis, and electrical inhomogeneity.

Concerning the echocardiographic parameters, our results showed the significant increase in LAD and both maximal and minimal left atrial area in MS group compared to control group ($p < 0.001$).

These findings are in agreement with the results of Özer et al.[3] who stated that LA size and area increase for patients in MS group. As LA size increases, the pathway of the cardiac impulse will also increase. Accordingly, interatrial EMD will increase.

Krasuski et al.[18] correlated the development of AF to the severity of MS, LA pressure and also to the degree of LA enlargement. In a dilated LA, the time interval for impulse conduction will be longer. In addition to LA enlargement, loss of atrial myocytes and ongoing interstitial fibrosis results in electrical remodeling characterized by inhomogeneous electrical properties and abnormalities in conduction velocity.[19]

Our results regarding left atrial indices, showed significant decrease in LAEF and LAEI in MS group compared to control group ($p < 0.001$). These may lead to impairment of LA reservoir function in MS patients by using both previous indices. These indices are analogous to the LA diastolic function. The LA reservoir dysfunction, which reflects "diastolic" dysfunction of LA, is likely to occur before LA booster dysfunction, which represents "systolic" dysfunction of the LA.[20] LA dysfunction may induce thrombogenesis, intra-atrial stasis with dense spontaneous contrast in LA, and occurrence of AF.[21]

We calculated LAEF and LAEI from LA volumes as these are more predictive of future AF and other cardiovascular events than LA dimension. These results come in agreement with Tsang et al.[22,23] who suggesting that LA volume may be a more sensitive index of LA remodeling than LA dimension.

One of the main findings in this study is prolongation of the interatrial EMD in MS group compared with control group ($p < 0.001$).

These findings come in agreement with the results of Özer et al.[3] who stated that interatrial EMD gets longer in MS patients group compared with control healthy subjects. Also, Demirkan et al.[11] found that patients with moderate to severe MS had longer interatrial conduction time as compared with control subjects.

PBMV, which is the procedure of choice in the treatment of rheumatic MS, has excellent immediate and long-term results in patients with favorable mitral valve morphology but those with less favorable anatomy may still have reasonably good hemodynamic and symptomatic relief.[24] Although there are no data from comparative studies, PBMV is estimated to reduce the incidence of AF.[25]

Our findings after one week from the valvuloplasty reflect mitral valve afterload reduction by the highly significant increase in mitral valve area and LAEF, while there was highly significant decrease in mean and peak mitral valve gradients, LAD and pulmonary systolic pressure ($p < 0.001$).

Our results are in agreement with Vieira et al.[26] who stated that the main acute impacts on the LA were a decrease in the mitral valve afterload, as reflected by an increase in the mitral valve area, a decrease in the mean and peak mitral valve gradients, a decrease in pulmonary systolic pressure, and a decrease in the LA anteroposterior diameter.

Also, these results come in agreement with Morttada et al.[27] who assess the impact of successful PBMV with a significant increase in the mitral valve area, decrease in the pressure gradient across the mitral valve and decrease in SPAP leading to a decrease in left atrial pressure, pulmonary venous congestion and pulmonary artery pressure.

One week after PBMV, we documented a highly significant improvement in all LA indices (LAA_{max}, LAV_{max}, Ind. LAV_{max} and Ind. LAV_{min}). We documented a highly significant reduction in the Ind. LAV_{max} by 21.8% ($p < 0.001$).

These findings come in agreement with Antonini et al.[28] who stated that LA reverse remodeling after PBMV, considered to have occurred when the Ind. LAV_{max} decreased by >15%, as previously described for LA analysis after mitral valve surgical repair.

Also, Adavane et al.[29] found that the Ind. LAV_{max} reduced immediately after PBMV by 14.2% and reduced by 23.2% 30 days after PBMV. In another study, the Ind. LAV_{max} reduced by 16% at 72 h and by 21.5% 12 months after PBMV.

We found highly significant increase in LAEF and LAEI one week after PBMV ($p < 0.001$), indicating improvement in the LA reservoir function, and these results are not agreed with Pritchett et al.[30] who stated that assessing atrial mechanical function by measuring volumes is time-consuming and depends on age, gender, and body surface area.

We documented a highly significant decrease in *P*-max and PWD ($p < 0.001$) one week after PBMV.

These findings are in agreement with Turhan et al.[31] who demonstrated decrease in *P*-max and PWD, which were regarded as ECG signs of the susceptibility to AF in both the short and long terms after PBMV.

One week after PBMV, regarding the interatrial EMD, there was a highly significant decrease in interatrial EMD using TDI ($p < 0.001$).

This result is in agreement with that of Demirkan et al.[11] who studied the interatrial EMD before and 72 h after valvuloplasty, and they explained this by the immediate decline in LA stretch (as a result of the immediate drop in LA pressure), also in their study both LA diameter and LA volume were decreased significantly after PBMV.

Finally, we found that interatrial EMD significantly correlated positively with LAD and showed positive association with LAA, and these results are in agreement with Özer et al.[3] who showed that LA size and area are correlated positively with interatrial EMD measured with noninvasive method for patients with MS.

In our study, we found significant positive correlation between echocardiographic interatrial EMD and both electrocardiographic AF markers (*P*-max and PWD) ($p < 0.05$).

Our results are in agreement with Özer et al.[3] who used this TDI method for investigating interatrial EMD in patients with MS, and they detected a positive correlation between interatrial EMD and LA size, as well as between interatrial EMD and electrocardiographic AF markers (*P*-max and PWD).

5. Study limitations

In addition to the small sample size, there are some limitations of our study. We did not have long-term follow-up data. Therefore, the relationship between these parameters and the development of AF is not clearly known. Moreover, our patients were relatively young and had somewhat lower Wilkins scores. Therefore, our results might not be applicable to all patients with MS who have more severe disease with higher Wilkins scores, patients of relatively advanced age or patients who have more inflammatory response. Further prospective studies should be carried out to clarify the relationship between these parameters and the incidence of AF.

6. Conclusions

This study shows that interatrial EMD gets longer in patients with moderate to severe MS compared to control subjects and is positively correlated with PWD, which is a marker of AF for such patients. Also, percutaneous balloon dilatation of the mitral valve had a favorable effect on atrial conduction properties evaluated with both TDI echocardiography and ECG.

These parameters, obtained by TDI, are easily measured in the clinical setting, and are useful in atrial function evaluation and hence in the prediction of AF.

Conflict of interest

The authors declare that there are no conflicts of interest.

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