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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 $\geqslant$ 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