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Assessment of atrial conduction time by tissue Doppler echocardiography and P-wave dispersion in patients with mitral annulus calcification

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Abstract The aim of our study was to investigate atrial conduction time in patients with mitral annulus calcification (MAC) using P-wave dispersion (PWD) and electromechanical coupling measured with the surface electrocardiogram and the tissue Doppler echocardiography. Fifty-nine patients with MAC and 43 control subjects underwent resting the surface electrocardiogram and tissue Doppler echocardiography. The difference between the maximum (Pmax) and minimum P-wave durations was calculated and defined as PWD. Interatrial and intraatrial electromechanical delays were measured with tissue Doppler echocardiography. Both Pmax and PWD were higher in patients with MAC compared with controls (111.4 ± 15.8 vs 97.3 ± 18.8 milliseconds; $P < .0001$ and 46.4 ± 14.6 vs 31.4 ± 13.1 milliseconds; $P < .0001$, respectively). Both interatrial and intraatrial conduction time were also delayed in patients with MAC compared with controls (29.8 ± 13.3 vs 17.6 ± 12.5 milliseconds; $P < .0001$; 9.4 ± 5.1 vs 6.8 ± 4.0 milliseconds; $P < .008$, respectively). Left atrial (LA) diameter was significantly higher in patients with MAC compared with controls (35.4 ± 5.0 mm vs 32.3 ± 4.2 mm; $P < .001$). The LA diameter correlated significantly with both interatrial conduction times and PWD ($r = 0.56$; $P < .0001$ and $r = 0.47$; $P < .0001$, respectively). There is a delay in both intraatrial and interatrial electromechanical coupling intervals in patients with MAC.

Keywords: Mitral annulus calcification; P-wave dispersion; Atrial electromechanical coupling; Tissue Doppler echocardiography

Introduction

It is known that mitral annulus calcification (MAC) is a chronic, noninflammatory, degenerative process of the fibrous support structure of the mitral valve.[1,2] Previous studies have put forward that patients with MAC have a higher incidence of left atrial (LA) enlargement.[3] Furthermore, MAC was proven to be associated with several conduction disorders, such as sinoatrial disease, atrial fibrillation (AF), atrioventricular block, left anterior hemiblock, and interventricular conduction defects, but the true incidence and the exact underlying mechanisms of AF were uncertain.[4,5] The prolongation of intraatrial and interatrial conduction times and the inhomogeneous propagation of sinus impulses are well-known electrophysiologic characteristics of the atrium prone to fibrillate and have been evaluated using tissue Doppler echocardiography and 2 simple electrocardiogram (ECG) markers, maximum P-wave duration (Pmax) and P-wave dispersion (PWD).[6-9] To date, these ECG markers and tissue Doppler echocardiography have not been evaluated in patients with MAC for the detection of atrial conduction abnormalities and electromechanical coupling. The aim of our study was to investigate atrial conduction time noninvasively in patients with MAC and assess the correlation with prognostic factors of atrial arrhythmia.

Materials and methods

Study population consisted of 59 consecutive patients (32 female, 27 male; mean age, 60.8 ± 9.4 years) with recently diagnosed MAC by transthoracic echocardiography and 43 voluntary healthy subjects (22 female, 21 male; mean age, 58.1 ± 7.8 years) who had normal echocardiographic parameters, similar sex and age profiles to the patients

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involved in the study. All the patients were in sinus rhythm during the study period. Patients with a mitral valve area less than 2.5 cm^2 or a mean transvalvular gradient more than 2 mm Hg, left bundle or right bundle branch block, prior pacemaker implantation, congenital heart disease, pericarditis, pulmonary emboli, heart failure, rheumatic valvular disease, prosthetic valves, malignancy, active inflammatory or infective disease, and hematologic disorder were excluded. We also excluded patients receiving digitalis, β-blockers, or any other antiarrhythmic drugs. To reach age-matching, patients older than 70 years were excluded from the control group before recruitment. Diabetes was defined as hyperglycemia requiring previous or ongoing pharmacologic therapy; hypertension was defined as either systolic or diastolic elevation of blood pressure (140/90 mm Hg) or ongoing antihypertensive pharmacologic therapy; hypercholesterolemia was defined as a total cholesterol level of 200 mg/dL or ongoing antilipidemic pharmacologic therapy; significant smoking history was defined as 10 cigarettes/d or more for at least 1 year during the last 10 years of cigarette use. Before ECG recording, if the patients had any medications, they were stopped within 1 week. The study was carried out according to the principles of the Declaration of Helsinki and approved by Inonu University, School of Medicine (Malatya, Turkey), investigational review board.

Echocardiographic analysis

All echocardiographic examinations were performed in all patients and control subjects with ATL HDI-5000 (Philips Company, Bothell, WA) while resting at the left lateral decubitis position. An average of 3 beats was analyzed. All the measurements were obtained by a single observer who was blinded to the clinical status of the patients. During echocardiography, 1-lead ECG was recorded continuously. The LA dimension, left ventricular (LV) diameters, and LV ejection fraction were measured. The 2-dimensional echocardiographic criteria for MAC included the thickness of the intense echo-producing structure, greater than 3 mm in width, located at the junction of the atrioventricular groove and posterior mitral valve leaflet on the parasternal long-axis, apical 4-chamber, or parasternal short-axis views by 2-dimensional echocardiograpy.[10]

Doppler tissue echocardiography was performed by the same echocardiograph machine, adjusting the spectral pulsed Doppler signal filters until a Nyquist limit of 15 to 20 cm/s was reached and using the minimal optimal gain. The monitor sweep speed was set at 50 to 100 mm/s to optimize the spectral display of myocardial velocities. In an apical 4-chamber view, the pulsed Doppler sample volume was placed at the level of LV lateral mitral annulus, septal mitral annulus, and right ventricular (RV) tricuspid annulus. The time interval from the onset of the P wave on surface ECG to the beginning of the late diastolic wave (A wave), which is called PA, was obtained from the lateral mitral annulus (lateral PA), septal mitral annulus (septal PA), and RV tricuspid annulus (tricuspid PA), respectively (Fig. 1). The difference between lateral PA and tricuspid PA (lateral PA − tricuspid PA) was defined as interatrial electromechanical delay, and the difference between septal PA and tricuspid PA (septal PA − tricuspid PA) was defined as intraatrial electromechanical delay.[6]

Reproducibility of electromechanical parameters was assessed by coefficients of variation (SD of differences between the repeated measurements divided by the mean value and expressed as percentage) between measurements. Intraobserver variability was calculated from 40 subjects selected randomly from the study participants (20 patients with MAC and 20 control subjects) by repeating the measurements under the same basal conditions. Intraobserver variability was 5.3% for PA lateral, 5.8% for PA septal, and %5.3 for PA tricuspid; respectively. Interobserver variability was 5.0% for PA lateral, 5.2% for PA septal, and 5.7% for PA tricuspid, respectively.

Electrocardiographic analysis

All subjects underwent a 12-lead ECG recording after a 20-minute resting period in supine position at a paper speed of 50 mm/s and 2 mV/cm. The P-wave duration was measured manually in all simultaneously recorded 12 leads of the surface ECG by 2 of the investigators unaware of the study hypothesis. In each lead, the mean values for the 3 complexes were calculated. The onset of the P wave was defined as the point of first visible upward departure from baseline for positive waveforms and as the point of first downward departure from the baseline for negative waveforms. The return to the baseline was considered to be the end of the P wave. The Pmax measured in any of the 12 leads of the surface ECG was used as the longest atrial conduction time. The difference between Pmax and the minimum P-wave duration (Pmin) was calculated and defined as PWD (PWD = Pmax − Pmin).

Intraobserver and interobserver coefficients of variation were 3.0% and 3.4% for maximum P-wave duration, and 3.5% and 3.8% for PWD, respectively.

Statistical analysis

Statistical analysis was performed using SPSS software package (version 17.0; SPSS Inc, Chicago, IL). Continuous variables were expressed as means ± SD. Categorical variables were expressed as counts and percentages. Student *t* test was used for the comparison of continuous variables. Comparison of categorical variables was made by Pearson χ^2 test. Correlations were examined by Pearson correlation. *P* values of less than .05 were considered to be significant.

Results

The clinical and echocardiographic data are presented in Table 1. There was no significant difference between the MAC patients and controls with respect to age, sex, resting heart rate, smoking, diabetes mellitus, documented coronary heart disease, and the history of coronary heart disease. Hypertension and hypercholesterolemia in patients with MAC were higher than controls (40 [67.8%] vs 19 [42.2]; $P < .05$ and 34 [57.6%] vs 10 [23.3%]; $P < .001$, respectively). The LV end-diastolic and end-systolic

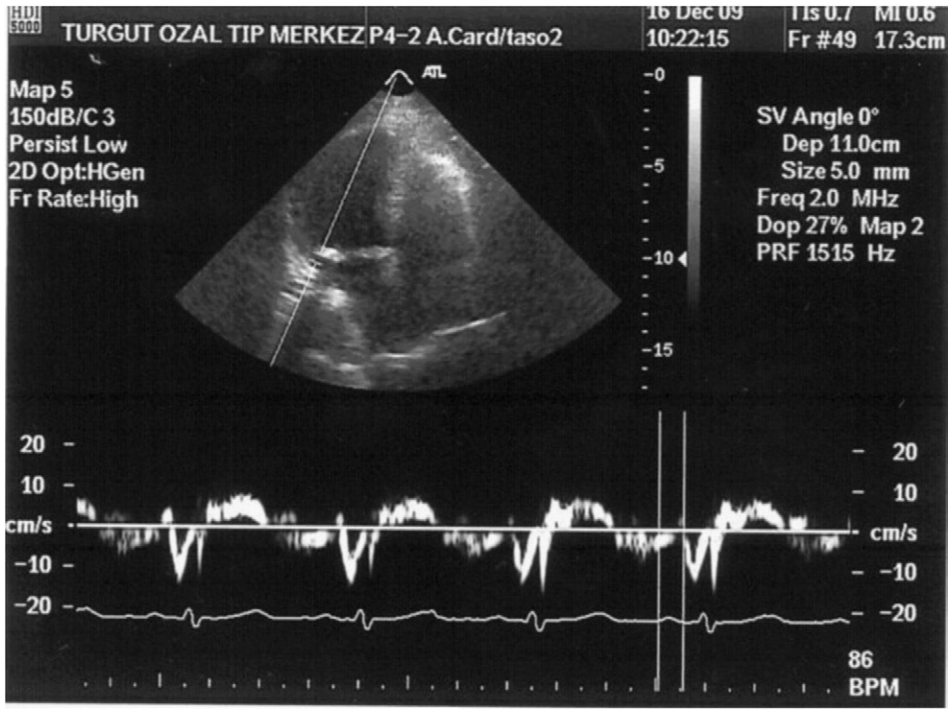

Fig. 1. Measurement of the time interval from onset of the P wave on surface ECG to beginning of the A wave (PA) with tissue Doppler echocardiography.

diameters and LV ejection fraction were also similar between the groups. In patients group, 6 patients had 1° to 2°, and 17 patients had minimal to 1° mitral regurgitation. In control group, 9 of them had minimal to 1° mitral regurgitation. Seven patients had degenerative aortic sclerosis. The LA diameter was significantly higher in patients with MAC compared with controls (35.4 ± 5.0 mm vs 32.3 ± 4.2 mm; $P < .001$). A strong positive correlation was detected between LA diameter and interatrial conduction times ($r = 0.56$; $P < .0001$), also LA diameter correlated significantly with both Pmax and PWD ($r = 0.43$; $P = .001$ and $r = 0.47$; $P < .0001$, respectively).

Table 1
Clinical characteristics of patients and controls

	MAC, n (59)	Controls, n (43)	*P*
Age (y)	60.8 ± 9.4	58.1 ± 7.8	NS
Female, n (%)	32 (54.2)	22 (51.2)	NS
Resting heart rate, beats/min	76.6 ± 11.4	78.5 ± 9.5	NS
Diabetes mellitus, n (%)	17 (28.8)	6 (14.0)	NS
Hypertension, n (%)	40 (67.8)	19 (42.2)	<.05
Hypercholesterolemia, n (%)	34 (57.6)	10 (23.3)	<.001
Smokers, n (%)	12 (20.3)	7 (16.3)	NS
Coronary artery disease, n (%)	14 (23.7)	8 (18.6)	NS
Family history for coronary artery disease, n (%)	9 (15.3)	4 (9.3)	NS
LV ejection fraction (%)	60.5 ± 7.2	60.6 ± 8.3	NS
Left atrial diameter (mm)	35.4 ± 5.0	32.3 ± 4.2	<.001
LVEDD (mm)	47.7 ± 3.6	46.4 ± 3.7	NS
LVESD (mm)	32.0 ± 4.8	31.3 ± 4.8	NS
Septal thickness (mm)	10.4 ± 0.9	10.1 ± 1.0	NS
PW thickness (mm)	10.3 ± 0.9	10.1 ± 0.9	NS

LVEDD indicates left ventricular end-diastolic diameter; LVESD, left ventricular end-systolic diameter; PW, posterior wall; NS, not significant.

Pmax was significantly higher in patients with MAC compared with controls (111.4 ± 15.8 vs 97.3 ± 18.8 milliseconds; $P < .0001$). Pmin did not differ significantly between the groups (65.0 ± 14.7 vs 65.9 ± 11.9 milliseconds; P = not significant). P-wave dispersion was significantly higher in the patients with MAC compared with controls (46.4 ± 14.6 vs 31.4 ± 13.1 milliseconds; $P < .0001$) (Fig. 2; Table 2).

Table 3 shows the results of tissue Doppler measurements. The time intervals from the onset of the P wave on the

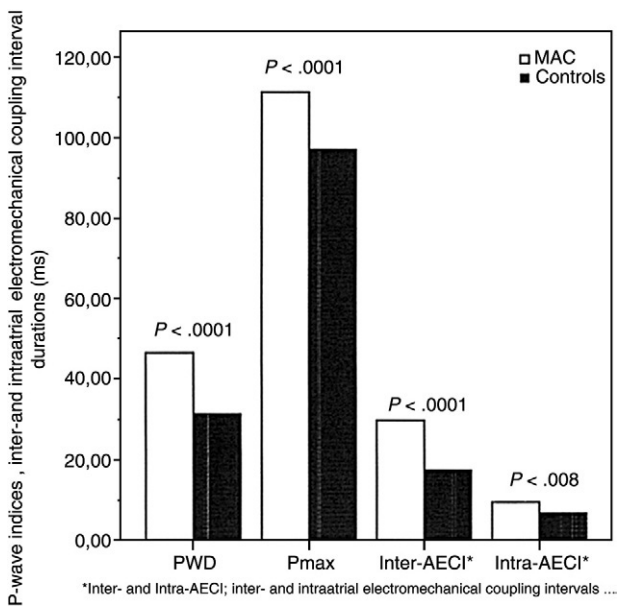

Fig. 2. Comparison of P wave indices, interatrial and intraatrial electromechanical coupling intervals between the patients with MAC and the control subjects.

Table 2
Electrocardiographic variables

	MAC, n (59)	Controls, n (43)	P
Heart rate (beat/min)	79.6 ± 14.3	81.4 ± 15.2	NS
Pmax (ms)	111.4 ± 15.8	97.3 ± 18.8	<.0001
Pmin (ms)	65.0 ± 14.7	65.9 ± 11.9	NS
PWD (ms)	46.4 ± 14.6	31.4 ± 13.1	<.0001

NS indicates not significant.

surface ECG to the beginning of the late diastolic wave (A wave) obtained by tissue Doppler echocardiography (PA) were significantly delayed at the lateral PA (78.5 ± 10.8 vs 65.4 ± 11.1 milliseconds; $P < .0001$) and the septal PA (58.0 ± 7.4 vs 54.5 ± 7.5 milliseconds; $P < .02$), but there was no delay at the tricuspid PA (48.8 ± 7.3 vs 47.7 ± 6.5 milliseconds; P = not significant) in patients with MAC. Both interatrial conduction time (lateral PA − tricuspid PA) and intraatrial conduction time (septal PA − tricuspid PA) were also delayed in patients with MAC compared with controls (29.8 ± 13.3 vs 17.6 ± 12.5 milliseconds; $P < .0001$; 9.4 ± 5.1 vs 6.8 ± 4.0 milliseconds; $P < .008$, respectively). A significant positive correlation was detected among interatrial electromechanical delay in both Pmax and PWD ($r = 0.36$; $P = .006$ and $r = 0.37$; $P = .004$, respectively). Also, Pmax and PWD both significantly correlated with intraatrial electromechanical delay ($r = 0.32$; $P = .013$ and $r = 0.39$; $P = .002$, respectively).

Discussion

In the present study, atrial conduction and electromechanical coupling in patients with MAC were evaluated, using 2 simple ECG markers, Pmax and PWD, and tissue Doppler echocardiography. P-wave dispersion is defined as the difference between the maximum and minimum P-wave duration, and it has been reported to be associated with inhomogeneous and discontinuous atrial conductions of the sinus impulses. Increased values of PWD and Pmax have been recognized as markers of an increased risk of AF.[11-15] We have shown that patients with MAC had higher Pmax and PWD values and delayed interatrial and intraatrial electromechanical coupling intervals compared with controls (Fig. 2).

Mitral annulus calcification is a chronic degenerative noninflammatory process of fibrous structure of mitral valve, which is more frequent in females and increases with aging.[16] It is frequently asymptomatic and does not affect the functions of mitral valve. In rare cases, it can cause severe mitral regurgitation and/or stenosis.[16] None of the participants of our study had severe enough MAC to affect the functions of the valve.

Pathologic studies have shown that the deposits of calcium occur predominantly in the posterior portion of the annulus but may extend into the LA and LV behind the mitral valve leaflets.[1,17] The deposits of calcium may be extensive, 1 to 3 cm in diameter, and may encircle all or most of the mitral orifice. The calcium may extend as far as 3 to 4 cm into the adjacent myocardium. Calcific deposits may also extend into the membranous portion of the intraventricular septum in the vicinity of the bundle of His and its branches.[18]

Several studies have shown that patients with MAC have a higher incidence of LA enlargement and AF.[1,5,19,20] In addition, MAC was proven to be associated with several conduction disorders, but the true incidence of them was uncertain.[2,4,5] This association is apparently due to damage caused by calcium deposits in the conduction system, probably in consequence of the close association of the atrioventricular node and bundle of His to the cardiac fibrous skeleton.[21,22] Previously, a lot of studies have suggested that the degenerative process within the mitral annulus may be associated with or accompanied by a "sclerodegenerative process" within the conduction system in these elderly patients.[23,24] Although the exact mechanism underlying AF is not certain, it has been suggested that the diffuse conduction system disease may interrupt interatrial and intraatrial conduction.[2]

P-wave abnormalities have been reported to be associated with LA enlargement and LA hypertension.[25,26] In our study, the increased LA diameter in patients with MAC was also remarkable, and there was significant correlation among the LA diameters in both Pmax and PWD. We also found that hypertension and hyperlipidemia were more often in patients with MAC. The responsible etiologic factors of the increased LA enlargement may be caused by the accompanying comorbid diseases of MAC such as hypertension; besides, MAC itself may be the cause of LA enlargement without an accompanying disease. Furthermore, MAC is a sclerodegenerative disease that can cause inflammation, growth, and increased stiffness in LA.[2]

To the best of our knowledge, there is no study that PWD and tissue Doppler echocardiography have been evaluated in patients with MAC for the detection of atrial conduction abnormalities and electromechanical coupling. Increases in the P-wave duration and PWD from standard ECGs with subsequent development of AF have been identified in patients with a wide range of cardiovascular disorders and in patients undergoing aortocoronary bypass grafting or hemodialysis.[11-15] The patients in our study group were asymptomatic and did not have a history of AF. Thus, increased PWD and Pmax in patients with MAC probably indicate the conduction system involvement and subsequent prolongation of intraatrial and interatrial conduction times and the inhomogeneous propagation of sinus impulses. Also, in our study, there was a correlation among interatrial and intraatrial electromechanical delay in both Pmax and PWD.

Recent developments in tissue velocity imaging allow precise analysis of atrial motion from different regions of the

Table 3
Tissue Doppler finding

	MAC, n (59)	Controls, n (43)	P
Lateral PA, ms	78.5 ± 10.8	65.4 ± 11.1	<.0001
Septal PA, ms	58.0 ± 7.4	54.5 ± 7.5	<.02
Tricuspid PA, ms	48.8 ± 7.3	47.7 ± 6.5	NS
Lateral PA − tricuspid PA, ms	29.8 ± 13.3	17.6 ± 12.5	<.0001
Septal PA − tricuspid PA, ms	9.4 ± 5.1	6.8 ± 4.0	<.008

NS indicates not significant.

RV and LV with high temporal resolution. Recently, it was showed that atrial electromechanical delay measured by tissue Doppler echocardiography was significantly longer in patients with paroxysmal AF and mitral stenosis than in the control groups.[6,27] In previous studies, a positive correlation was found between LA diameter and interatrial electromechanical delay,[6,27] and based on this literature, increased atrial electromechanical delay seems to be related with AF. In our study, we demonstrated that LA diameter was higher in patients with MAC compared with controls and interatrial conduction time significantly correlated with LA diameters. We also showed the delayed electromechanical coupling in both left and right atria and the delay in interatrial contraction in patients with MAC by using tissue Doppler echocardiography. This is possibly due to the evident prolongation measured in septal PA, despite a lack of detectable change in tricuspid PA. An increase in LA diameter and conduction delay may also affect RA because of the common septum. Therefore, the prolongation in intraatrial coupling time shows a conduction delay in RA as well as LA.

The most important limitation of our study is the cross-sectional design of the study, in which we could not follow up the patients prospectively for future arrhythmic events. Therefore, we do not know whether prolongation of PWD and atrial electromechanical delay predict atrial arrhythmias in patients with MAC. The second limitation of this study is the lack of studying a parameter such as brain natriuretic peptide, reflecting LA overload. Finally, we calculated Pmax and Pmin manually using magnifying lens instead of a more reliable computer-assisted P-wave calculating system.[28]

In conclusion, our study confirmed that there is delay in interatrial and intraatrial electromechanical coupling, which is correlated with PWD, in patients with MAC. Thus, we speculated that prolongation of atrial electromechanical conductions and PWD in patients with MAC probably indicate the involvement of conduction system. This might contribute to development of adverse functional and electrophysiologic atrial characteristics in these patients. We believe these findings should be evaluated with electrophysiologic investigation of the interatrial and intraatrial conduction times and long-term follow-up studies to identify patients developing AF and the predictive accuracy of PWD in patients with MAC.

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