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P-wave dispersion in patients with stable coronary artery disease and its relationship with severity of the disease

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Abstract

Aim: P-wave dispersion (PD) has been reported to be associated with inhomogeneous and discontinuous propagation of sinus impulses. The purpose of this study was to investigate the PD in patients with stable coronary artery disease (CAD) and to determine its relationship with severity of the disease.

Methods: We prospectively analyzed 66 subjects with normal coronary angiogram (group 1) and 68 patients with significant ($\geq$50%) coronary stenosis; 25 had 1-vessel disease (group 2), 27 had 2-vessel disease (group 3), and 16 had 3-vessel disease (group 4). The maximum and minimum P-wave duration (Pmax and Pmin) and PD were measured from the 12-lead surface electrocardiogram. Echocardiographic examination was also performed. Angiographic vessel score and Gensini score were used to evaluate the severity of CAD.

Results: Pmax was longer in groups 3 and 4 compared with group 1 (P = .001 for both comparison). PD was greater in groups 2, 3, and 4 compared with group 1 (P < .001 for all comparison), and also in group 4 compared with group 2 (P = .001). However, there was no statistically significant difference among the groups regarding Pmin. In bivariate correlation, increased PD was correlated with presence of hypertension (r = 0.278, P = .013), left ventricular ejection fraction (r = −0.231, P = .044), left atrial diameter (r = 0.223, P = .032), presence of moderate to severe mitral regurgitation (r = 0.284, P = .017), vessel score (r = 0.465, P < .001), and Gensini score (r = 0.338, P = .005). Multiple linear regression analysis showed that only vessel score was independently associated with PD (β = .471, P = .01).

Conclusions: PD was found to be greater in patients with stable CAD than in controls and to be associated with severity of the disease.

Keywords: P-wave duration; P-wave dispersion; Coronary artery disease

1. Introduction

Maximum P-wave duration (Pmax) and P-wave dispersion (PD), which is defined as Pmax minus minimum P-wave duration (Pmin), on standard electrocardiogram (ECG) are noninvasive markers of inhomogeneous and discontinuous propagation of sinus impulses through the atrial wall, which are believed to be the main electrophysiological cause of atrial fibrillation (AF) [1,2]. Previously, P-wave parameters have been studied in some cardiac conditions such as hypertension, paroxysmal AF, mitral stenosis, aortic stenosis, dilated cardiomyopathy, and acute myocardial infarction and during spontaneous angina pectoris and during coronary angioplasty [3-11]. Although it has been demonstrated that PD is increased during abovementioned acute ischemic conditions, the effect of stable coronary artery disease (CAD) on the P-wave durations and PD, and relation between P-wave parameters and severity of CAD have not yet been investigated. Therefore, the purpose of our investigation was to evaluate whether Pmax and PD are increased in patients with stable CAD and related to severity of the disease.

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2. Materials and methods

2.1. Subjects

The study sample consisted of 134 consecutive subjects who underwent elective coronary angiography in our center and were found to have either normal coronary angiogram (group 1, as a control group; n = 66) or significant obstruction (≥50%) at least in 1 major epicardial coronary artery (n = 68). From the patients group, 25 had 1-vessel disease (group 2), 27 had 2-vessel disease (group 3), and 16 had 3-vessel disease (group 4). Controls were included on basis of a normal coronary angiogram, no suspicious history of cardiovascular disease, and a normal ECG. Forty-six of 68 patients with CAD had stable angina pectoris (Canadian Cardiovascular Society classes I-III), and 22 of them had positive exercise stress test, which was performed because of their high-risk profile for CAD. Angina had started within the last 2- to 12-month time interval in all patients with stable angina. Patients with acute coronary syndromes or angina pectoris at rest (Canadian Cardiovascular Society classes IV) were excluded. Moreover, patients were excluded for any of the following reasons: other rhythm than stable sinus rhythm, paroxysmal AF, atrioventricular or intraventricular conduction disturbances, valvular heart disease, abnormal thyroid function, cardiomyopathies, congenital heart diseases, lung diseases and pulmonary hypertension, abnormal serum electrolyte values, receiving any antiarrhythmic drugs, and history of myocardial infarction, collagen disease, chronic renal failure, and percutaneous coronary intervention, or cardiac surgery. ECG recordings and echocardiographic examinations were performed before coronary angiography.

2.2. Electrocardiographic measurements

A 12-lead surface ECG was obtained from all subjects in the supine position by using Hewlett-Packard Electrocardiograph Sanborn Series (Chine) machine. All patients were breathing freely but not allowed to speak during the ECG recordings. The ECGs were recorded at a paper speed of 50 mm/s. Three leads were recorded simultaneously. Two investigators without knowledge of patients' clinical status measured the P-wave durations manually. To improve accuracy, measurements were performed with calipers and magnifying lens for defining the ECG deflection [12-14]. Higher calibration was not needed to determine the onset and offset of P waves. The onset of P wave was defined as the junction between the isoelectric line and the start of P-wave deflection, and the offset of the P waves as the junction between the end of the P-wave deflection and the isoelectric line [15]. Pmax in any of the 12-lead surface ECG was calculated and used as a marker of prolonged atrial conduction time. PD, defined as the difference between Pmax and Pmin, was calculated from the 12-lead ECG. Intraobserver and interobserver variabilities of P-wave duration were 4% ± 4% and 3% ± 5%, and those of PD were 4% ± 5% and 3% ± 4% (absolute difference divided by mean value of measurements).

2.3. Echocardiographic examination

Echocardiographic examination was performed in all study subjects by using a commercially available system (Aloka Prosound SSD 5000 machine with a 3-MHz transducer). Measurements were made during normal breathing at end-expiration. M-mode echocardiographic measurements were obtained on the basis of the standards of the American Society of Echocardiography [16]. Left atrial diameter, left ventricular end-systolic and end-diastolic diameters, end-diastolic interventricular septal thickness, and end-diastolic left ventricular posterior wall thickness were measured. Left ventricular ejection fraction was determined by using the Simpson biplane formula according to the suggestions of the American Society of Echocardiography [17]. All echocardiographic measurements were calculated from an average of 3 consecutive cardiac cycles. Echocardiographic left ventricular mass was determined by using the corrected formula proposed by Devereux et al [18], and indexed by body surface area (grams per square meter). Mitral regurgitation was assessed and graded according to the method of Helmcke et al [19].

2.4. Coronary angiographic scoring

Coronary angiography was performed by the femoral approach with 7-Fr diagnostic catheters. Images were recorded in multiple projections for left and right coronary arteries on a digital system. The interpretation of the coronary angiograms was made by 2 cardiologists who were blinded for the characteristics of the patients during the interpretation. P-wave measurements of all ECGs were performed in a period. The interpretation of all angiograms was made in another period. Investigators were blind to the results of the angiograms during the measurements of the P-wave durations. Moreover, during the interpretation of the angiograms, they were unaware of the values of the P-wave durations. The CAD was evaluated by "vessel score" [20]. Vessel score was the number of vessels with a significant stenosis (≥50% reduction in lumen diameter). Scores ranged from 0 to 3, depending on the number of vessels involved. Left main artery stenosis was scored as 1-vessel disease. The intraobserver and interobserver correlations in this scoring system both were >0.95.

In addition to the vessel score, the severity of CAD was assessed by using the Gensini score [21], which grades narrowing of the lumens of the coronary arteries as 1 for 1% to 25% narrowing, 2 for 26% to 50% narrowing, 4 for 51% to 75% narrowing, 8 for 76% to 90% narrowing, 16 for 91% to 99% narrowing, and 32 for total occlusion. This score is then multiplied by a factor that takes into account the importance of the lesion's position in the coronary arterial tree, for example, 5 for the left main coronary artery; 2.5 for the proximal left anterior descending coronary artery and proximal left circumflex