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Original article

Clinical factors associated with the development of atrial fibrillation in the year following STEMI treated by primary PCI

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ABSTRACT

Background: Advanced age, poor left ventricular function, and congestive heart failure are known predictors of atrial fibrillation (AF) in acute myocardial infarction (AMI) patients. Recent advances in AMI treatment may have changed the occurrence of new-onset AF. Thus, we investigated the factors associated with the development of new-onset AF in ST elevation myocardial infarction (STEMI) patients.
Methods: This study included 527 STEMI patients [mean age, 60.6 ± 12.8 years; 102 (19.4%) women] who underwent primary percutaneous coronary intervention (PCI) in the previous 7 years. New-onset AF was evaluated following STEMI treated by primary PCI. Patients who developed AF during this follow-up period were compared with those who did not develop AF to identify factors that were associated with the development of AF.
Results: New-onset AF was documented in 81 patients (15.4%) at 1 year after STEMI. Patients with new-onset AF ($n = 81$) tended to be older ($p < 0.001$); were more often female ($p = 0.009$); had more congestive heart failure ($p = 0.015$); had less use of beta-blockers ($p = 0.001$); had more often used antiarrhythmic drugs ($p < 0.001$); experienced cardiogenic shock more frequently ($p = 0.038$); had lower left ventricular ejection fraction ($p = 0.024$); and had higher E velocity ($p < 0.001$), E/e′ ($p = 0.011$), and left atrial volume index (LAVI; $p = 0.029$) than the 446 patients with no AF. Multivariate regression analysis revealed that cardiogenic shock, LAVI, and age were predictors of new-onset AF in STEMI patients (OR 2.823, 1.254, and 1.124; $p = 0.005$, <0.001, and 0.028, respectively).
Conclusion: Cardiogenic shock was a new predictor of new-onset AF in STEMI patients.

Introduction

Atrial fibrillation (AF) is one of the most common supraventricular arrhythmias in acute myocardial infarction (AMI), with an incidence of 5–18% in patients with AMI [1,2]. Its occurrence in AMI patients is important because AF increases the risk of cardiovascular events [3]. Notably, structural heart disease, hypertension, diabetes, and advanced age induce progressive structural remodeling in the atria, which leads to the development of AF [4]. Dysfunctions of the sinoatrial (SA) and atrioventricular (AV) nodes also contribute to the development of AF. In AMI patients, acute ischemic insults to the atria and ventricles can induce AF [5–9]. Moreover, the location of the culprit vessel also affects the occurrence of AF in AMI [8]. Many predictors of AF after AMI such as advanced age, female sex, left ventricular hypertrophy, and congestive heart failure (CHF) have been identified [10], but predictors of AF in patients with ST elevation myocardial infarction (STEMI) have yet to be clearly defined. Our aim was to identify factors that predict the development of AF in STEMI patients.

Methods

STEMI patients who underwent primary percutaneous coronary intervention (PCI) between January 2009 and December 2015 at Dong-A University Hospital (Busan, Korea) were screened. Of the 545 patients, we excluded 5 patients with a previous history of AF, 10 patients who died during admission, and 3 patients who did not complete the 1-year follow-up. Thus, a total of 527 patients with STEMI were enrolled in the study. The diagnosis of STEMI was based on typical chest pain symptoms, electrocardiographic (ECG) changes, and serial elevation of serum cardiac enzymes. The ECG

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criterion for the diagnosis of STEMI was an ST-segment elevation of ≥0.2 mV in ≥2 contiguous leads or new left bundle branch block. AF was defined as the absence of P waves with irregular RR intervals. Cardiogenic shock was defined as a state of systolic blood pressure less than 80 mmHg due to STEMI in the absence of hypovolemia that was associated with clinical signs of hypoperfusion or CHF [11]. CHF was diagnosed based on a known history of CHF and/or Killip class ≥2 at any time during hospitalization with the need for diuretics [12]. Dyslipidemia was defined by either documentation of the diagnosis in the patient's history, current use of lipid-modifying drugs, a fasting total cholesterol level ≥200 mg/dl, or a low-density lipoprotein cholesterol level of ≥130 mg/dl. Hypertension and diabetes were defined based on documentation of the clinical diagnosis. All patients received standard pharmacological therapies [13] and revascularization, had continuous ECG monitoring in the coronary care unit, and had 12-lead ECG performed daily during their hospital admission. After hospital discharge, all patients were followed up with ECG check up within 1 month and every 2 or 3 months after. New-onset AF patients were divided into two groups. Early AF was defined as AF documented during admission, and late AF was defined as new-onset AF within 1 year after discharge. This retrospective study was approved by our institutional review board.

Echocardiography

All patients underwent echocardiography within 48 h after hospital admission. Echocardiography was performed using the iE33 ultrasound system with 2.5-MHz transducer (Philips, Amsterdam, The Netherlands). Standard 2D and Doppler echocardiography were performed according to the recommendations of the American Society of Echocardiography [14,15]. Left ventricular end-diastolic dimension (LVEDD) and left ventricular end-systolic dimension (LVESD) were measured using 2D echocardiography. Left ventricular ejection fraction (LVEF) was assessed semi-quantitatively with visual estimation and the wall-motion score index. Left atrial (LA) volume was measured using the biplane Simpson method at end-systole from apical 4- and 2-chamber views. LA volume index (LAVI) was calculated as the LA volume divided by the body surface area. LV diastolic function was assessed by conventional Doppler (mitral E, mitral A, and the E/A ratio) and by tissue Doppler imaging (TDI). Peak systolic (s′), early diastolic (e′), and late diastolic velocities (a′) were obtained at the lateral mitral annulus from the apical 4-chamber view. LV systolic and diastolic dysfunction were defined as LVEF <40% and E/e′ ratio >15, respectively.

Coronary angiography

Coronary angiography was performed in all patients. The culprit vessel was defined as a vessel with complete closure or the most significant stenosis corresponding to ECG changes. Patients were further classified by the site of occlusion: left anterior descending artery (LAD), left circumflex artery (LCX), right coronary artery (RCA), and left main coronary artery (LMCA).

Statistical analysis

Baseline clinical, echocardiographic, and angiographic data were collected and analyzed. The data are presented as mean values with standard deviations for normally distributed continuous variables and as numbers and percentages for categorical variables. The differences between the two groups were assessed with the unpaired 2-tailed *t*-test for normally distributed continuous values and with the chi-square test for categorical variables. Logistic regression analysis was used to identify independent risk factors for the development of AF in patients with STEMI. Variables with *p*-values ≤0.20 in univariate analyses were candidates for the multivariable logistic regression model. A backward stepwise elimination algorithm reduced the variables until only significant variables ($p < 0.05$) remained in the multivariate model. All statistical comparisons were two-sided, and the statistical significance was set at $p < 0.05$. The statistical analysis was performed using the Statistical Package for Social Science v. 20.0 (SPSS; IBM, Chicago, IL, USA).

Results

A total of 527 STEMI patients with primary PCI were enrolled in the study. The mean age was 60.6 ± 12.8 years, and 102 patients (19.4%) were female. New-onset AF was documented in 81 (15.4%) at 1 year after STEMI. Early AF was identified in 67 patients (12.7%) and late AF in 14 patients (2.7%). In the STEMI patients, the culprit vessel was LAD in 271 patients (51.4%), RCA in 203 patients (38.5%), LCX in 50 patients (9.5%), and LMCA in 3 patients (0.6%). However, in patients with new-onset AF, the most common culprit vessel was the RCA (35 patients; 43.2%), followed by the LAD (34 patients; 42.0%), the LCX (10 patients; 12.3%), and the LMCA (2 patients; 2.5%) (Table 1).

Patients with new-onset AF were older (65.9 ± 12.4 years vs. 59.7 ± 12.7 years, $p < 0.001$), more often female (30.9% vs. 17.3%, $p = 0.009$), and more often had cardiogenic shock (16.0% vs. 8.3%, $p = 0.038$). Patients with new-onset AF had less use of beta-blockers (86.7% vs. 53.6%, $p = 0.001$) and had more often used antiarrhythmic drugs (21.0% vs. 4.5%, $p < 0.001$) than patients with no AF. However, there was no significant difference with respect to the location of the culprit vessel between the AF group vs. the no AF group (Table 1). An implantable cardioverter defibrillator was implanted in one patient with no AF.

Echocardiographic characteristics

In echocardiographic analysis, patients who developed new-onset AF had lower LVEF (46.0 ± 8.6% vs. 48.4 ± 8.8%, $p = 0.024$), higher E velocity (75.9 ± 24.4 cm/s vs. 66.4 ± 19.4 cm/s, $p < 0.001$), higher E/e′ (12.1 ± 6.7 vs. 10.1 ± 3.9, $p = 0.011$), and higher LAVI (34.4 ± 13.1 ml/m^2 vs. 29.4 ± 8.6 ml/m^2, $p = 0.002$) than the 446 patients without AF (Table 2). The incidence of AF development increased as LAVI increased (Fig. 1).

Table 1
Baseline clinical and angiographic characteristics.

	All (*n* = 527)	No AF (*n* = 446)	AF (*n* = 81)	*p*-value
Age	60.6 ± 12.8	59.7 ± 12.7	65.9 ± 12.4	<0.001
Female sex	102 (19.4%)	77 (17.3%)	25 (30.9%)	0.009
Hypertension	265 (50.3%)	220 (49.3%)	45 (55.6%)	0.335
Diabetes mellitus	153 (29.0%)	135 (30.3%)	18 (22.2%)	0.183
Dyslipidemia	399 (75.7%)	344 (77.1%)	55 (67.9%)	0.090
DBT, min	61.5 ± 68.9	60.7 ± 67.9	66.0 ± 74.7	0.530
CHF	148 (28.0%)	114 (25.5%)	34 (42.0%)	0.015
Cardiogenic shock	50 (9.5%)	37 (8.3%)	13 (16.0%)	0.038
Culprit LAD	271 (51.4%)	237 (53.1%)	34 (42.0%)	0.070
Culprit LCX	50 (9.5%)	40 (9.0%)	10 (12.3%)	0.310
Culprit RCA	203 (38.5%)	168 (37.7%)	35 (43.2%)	0.385
Culprit LMCA	3 (0.6%)	1 (0.2%)	2 (2.5%)	0.063
Aspirin	517 (98.1%)	438 (98.2%)	79 (97.5%)	0.821
Beta-blocker	432 (82.0%)	387 (86.7%)	45 (55.6%)	0.001
ACEI/ARB	448 (85.0%)	380 (85.2%)	68 (84.0%)	0.732
Statin	456 (86.5%)	387 (86.7%)	69 (85.2%)	0.634
Antiarrhythmic drugs	37 (7.0%)	20 (4.5%)	17 (21.0%)	<0.001

AF, atrial fibrillation; CHF, congestive heart failure; DBT, door to balloon time; LAD, left anterior descending artery; LCX, left circumflex artery; RCA, right coronary artery; LMCA, left main coronary artery; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin-receptor blocker.

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