Incidence of atrial fibrillation and its effects on long-term follow-up outcomes in patients undergoing primary percutaneous coronary intervention for ST-elevation myocardial infarction

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Abstract

Objective: The incidence of atrial fibrillation (AF) in patients with ST segment elevation myocardial infarction (STEMI) varies between 7% and 21%, and most of these studies were in the thrombolytic era. However, the frequency of new-onset AF during the primary percutaneous coronary intervention (PCI) period is still unclear. We aimed to investigate the frequency of new-onset AF and its effects on long-term clinical events in patients undergoing primary PCI.

Methods: A total of 1,603 patients who were diagnosed with STEMI and underwent primary PCI were included in the study. All the patients were monitored for at least 48 hours after the procedure. The primary endpoint of the study was defined as new-onset AF during hospitalization.

Results: The median follow-up period of our study was 44 months. New-onset AF developed in 85 (6.1%) patients. CHADs-VASc > 2, KILLIP > 2, and left atrial diameter were found to be independent predictors for the development of new-onset AF. In the AF (+) group, the all-cause and in-hospital mortality rates were found to be significantly higher. New-onset AF development in patients with STEMI was detected as an independent predictor of in-hospital mortality.

Conclusion: In the era of primary percutaneous transluminal coronary angioplasty, new-onset AF rates were found to be lower than the literature data. In addition, new-onset AF was found to be a predictor of in-hospital mortality, and deaths occurred mostly in the early period. Therefore, close follow-up of these patients in the early period and re-evaluation in terms of AF burden when the patient becomes stable are important.

Keywords: new-onset atrial fibrillation, acute coronary syndrome, primary percutaneous coronary intervention

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Introduction

Atrial fibrillation is a common clinical arrhythmia that increases in frequency with age, diabetes, hypertension, and obesity, independent of structural heart disease (1, 2). Electrophysiological and metabolic changes in the myocardium because of myocardial ischemia or infarction produce silent or life-threatening arrhythmias. Although ventricular arrhythmias [accelerated idioventricular rhythm, ventricular tachycardia (VT), ventricular fibrillation (VF)] are frequently reported during acute coronary syndromes, atrial arrhythmias are also common (3). The incidence of AF in patients with ST segment elevation myocardial infarction (STEMI) varies between 7%–21%, and the majority of these data are based on studies in the thrombolytic era (1). Recently, changes in treatment approaches and the development of early invasive treatment strategies have been thought to decrease these rates. Development of AF in STEMIs leads to hemodynamic deterioration owing to a high ventricular rate, irregular ventricular filling, and/or loss of atrial contribution to cardiac output. In addition, AF develop-

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HIGHLIGHTS

- In our study, we found that in-hospital new-onset atrial fibrillation (AF) rates were lower during the primary percutaneous transluminal coronary angioplasty era.
- We also found that the development of new-onset AF was associated with a four-fold increase in in-hospital mortality.
- In-hospital AF development did not cause a significant increase in long-term clinical events such as stroke and myocardial infarction in the primary percutaneous coronary intervention era.

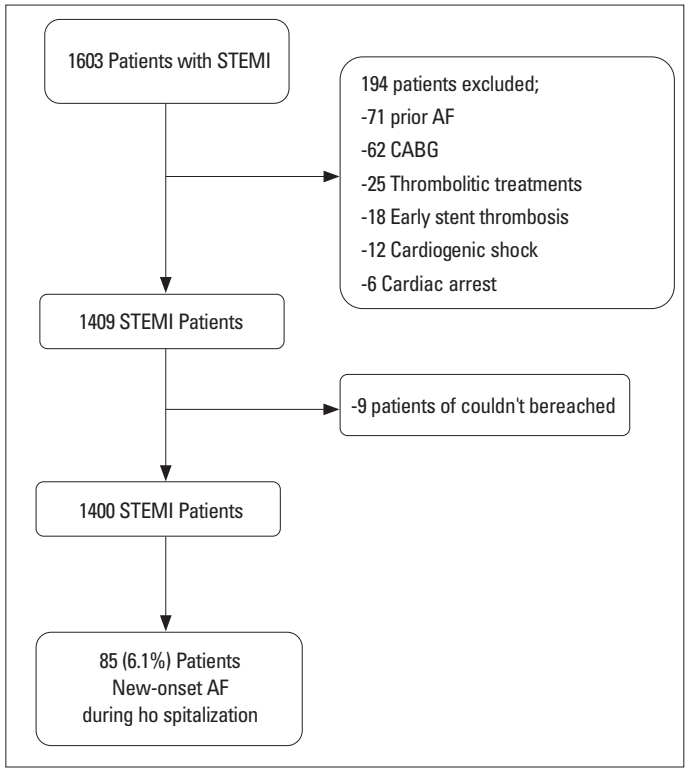

Figure 1. Trial flow chart

ment in patients with STEMI increases the risk of early stroke development than in those without AF. Therefore, AF development in patients with STEMI was associated with increased morbidity and mortality (4). In the current guidelines, anticoagulation therapy is recommended in this group of patients owing to the CHADs-VASc score; however, data on this condition are still unclear given the risks of bleeding (4). The studies on AF with STEMI were mostly conducted in 2010 and before. The majority of these studies were retrospective studies, and the patients were included in these studies after discharge with no early in-hospital AF (5, 6).

In this study, we aimed to investigate the rates of new-onset AF, long-term anticoagulation ratio, and short- and long-term adverse clinical events during follow-up in patients with diagnosed STEMI who were admitted to the emergency department and underwent primary percutaneous coronary intervention (PCI).

Methods

Study population

Our study was a cohort study. A total of 1,603 consecutive patients who were diagnosed with STEMI and underwent primary PCI between 2011 and 2018 at our cardiology center were analyzed. The exclusion criteria were patients with known atrial fibrillation, previous coronary artery bypass grafting, those with cardiogenic shock or early stage mechanical complications, at the end of the processing thrombolysis in myocardial infarction (TIMI) 0, 1 coronary blood flow, early stent thrombosis, treatment with thrombolytic therapy, severe valvular heart disease, thyroid diseases, and end-stage chronic organ failure (chronic obstructive pulmonary disease, chronic kidney disease, and chronic liver disease). A total of 1,400 patients were included in the study after exclusion. A flowchart of the patients is shown in Figure 1. All the patients gave their written informed consent, and the study was approved by the Local Ethics Commission (Ethics Committee of İstanbul University, Cerrahpaşa Cardiology Institute; 2.I.U.E.50.0.05.00/3).

Study protocol

The patients who presented with typical chest pain to the emergency unit of our hospital and had ST-segment elevation in two contiguous ECG leads (ST segment elevation in V2-V3 leads; ≥0.25 mV in men under 40 years of age, ≥0.2 mV in men over 40 years of age, or ≥0.15 mV in women, and/or ≥0.1 mV ST segment elevation in other leads) or had presumably new left bundle-branch block were diagnosed with STEMI (7). Loading doses of $P2Y_{12}$ inhibitors (600 mg clopidogrel, 60 mg prasugrel, or 180 mg ticagrelor) and 300 mg nonenteric-coated acetylsalicylic acid were given routinely to patients diagnosed with STEMI during their admission. All primer PCI procedures were performed by experienced interventional cardiologists using a femoral or radial approach. Patients undergoing PCI were administered 100 IU/kg heparin during the procedure. The dose was reduced to 60 IU/kg if the patient was given glycoprotein IIb/IIIa inhibitors (GPIs). The choice of thrombus aspiration, stent type, and GPI usage were left to the preference of the operator. All the patients were monitored for at least 48 hours after the procedure by telemetry in the coronary intensive care unit. Patients who had complaints of palpitations during hospitalization were followed up with a 24-hour rhythm Holter monitor, and an ECG was recorded during palpitation. Major bleeding was defined using the Bleeding Academic Research Consortium (BARC) bleeding definitions: intracranial hemorrhage, intraocular compromising vision, overt bleeding plus hemoglobin drop >5 g/dL, tamponade, bleeding requiring surgical or percutaneous intervention for control (excluding dental/nose/skin/hemorrhoids) or inotropes (BARC type 3A), any transfusion with overt bleeding, overt bleeding plus hemoglobin drop of 3 to 5 g/dL (BARC type 3B) or fatal

bleeding. All information on serious bleeding was identified with the diagnosis coded in a subsequent hospitalization during follow-up (8). Clinical data during follow-up were obtained from the medical records and recent clinical visits, using the insurance database system for death and stroke events and by contacting the patient and/or patient's relatives by phone.

Atrial fibrillation ascertainment

We defined AF using a conformation of the guidelines from the American College of Cardiology, American Heart Association, and European Heart Association (9). Patients with atrial flutter were considered to have AF, which was defined as the absence of P waves, and atrial activity was described by fibrillatory waves and irregular time elapsing between two consecutive R wave (R-R) intervals. Atrial flutter on ECG recordings had to fulfill the following criteria; presence of regular P waves with a rate of 250 to 350/min and regular or irregular R-R intervals. ECG diagnoses were assessed by two experienced cardiologists. Medical and/or DC cardioversion was administered to the patient who was found to have new-onset AF during hospitalization if there was no spontaneous termination within two hours.

Endpoints

The primary endpoint of the study was defined as new-onset AF during hospitalization. Major clinical outcomes (MACCEs) were defined as in-hospital mortality, all-cause mortality, myocardial infarction (MI), and cerebrovascular events (CVE). Other clinical outcomes were hospitalization for cardiac reasons and major bleeding.

Statistical analysis

Continuous variables were presented as means ± standard deviation. Categorical variables were presented as frequencies (percentages). Normal distribution analysis of data was performed by the Shapiro-Wilk test. Student's t test was performed in cases of normally distributed data, and the Mann-Whitney U test was applied for abnormally distributed data. Categorical parameters were evaluated by Pearson's chi-squared test. Logistic regression (forward method) was used to devise a model of new-onset AF predictors and in-hospital mortality predictors. Predictors for major clinical outcomes were calculated by a multivariate analysis using parameters that had p values <0.1 in the univariate analysis. Long-term follow-up of major clinical outcomes and all-cause mortality were evaluated using Kaplan-Meier curves. The criterion for statistical significance in the analysis was $p \leq 0.05$. The Statistical Package for the Social Sciences version 21 (SPSS Inc., Chicago, Illinois, US) packet program was used for data analysis.

Results

Clinical and demographical features and laboratory parameters

The median follow-up period of our study was 44 months (six to 100 months). In our study, 85.5% of the patients were men, and the mean age of our study population was 58.6±11 years. The door to balloon time was a median of 41 (six to 211) minutes. The median length of hospital stay was 12 (one to 62) days. New-onset AF, which was the primary endpoint of the study, developed in 85 (6.1%) patients. Although conversion to spontaneous sinus rhythm was observed in 59 (69.5%) patients, sinus rhythm was achieved with medical (18.8%) and/or DC cardioversion (CV) (11.7%) in 26 (30.5%) patients. Amiodarone infusion was delivered for at least 24 hours to all the patients undergoing cardioversion. All of our study patients were in sinus rhythm at the time of discharge. Other cardiac arrhythmias, such as ventricular tachycardia/fibrillation (8.0%) and A-V block (3.4%) were observed in 159 (11.4%) of the patients during hospitalization.

The patients were divided into two groups: new-onset AF (+) and AF (–). The demographic characteristics of the groups are given in Table 1. The average age of the AF (+) group was higher, and there were more women in this group (p<0.001 and p<0.001, respectively). In addition, the hypertension frequency was higher in the AF (+) group, and systolic arterial pressures were lower in this group (p=0.015 and p=0.053, respectively). Active smoking and hyperlipidemia frequency were lower in the AF (+) group (p<0.001 and p=0.034, respectively). In the AF (+) group, the frequency of KILLIP 3–4 patients was higher than that in the AF (–) group (p=0.013). In addition, the CHADs-VASc risk score was significantly higher in the AF (+) group (p<0.001). There was no difference between the groups in terms of MI types (p=0.062). The biochemical and echocardiographic characteristics of the groups are given in Table 2. Baseline Hb, LDL cholesterol, eGFR, and serum albumin levels were found to be significantly lower in the AF (+) group than in the AF (–) group (p=0.002, p=0.001, p<0.001, p=0.002, respectively). In addition, baseline serum creatinine, troponin, and ALT values were higher in the AF (+) group (p<0.001, p=0.035, p=0.005, respectively). On baseline echocardiography, the mean left atrium diameter was significantly higher in the AF (+) group (p<0.001). It was found that 29.5% of the patients were given an oral anticoagulant agent as a discharge treatment. The majority of them were patients who underwent medical/DC CV. We also analyzed the study population according to whether MACCE developed [MACCE (+) or MACCE (–)] (Table 3). The frequencies of DM, HL, and prior MI were significantly higher in the MACCE (+) group (p=0.005, p<0.001, p<0.001, respectively). The heart rate at the time of admission in the MACCE (+) group was found to be higher, but systolic blood pressure measurements were found to be lower (p<0.001 and p<0.001, respectively). In the MACCE (+) group, the door to balloon time was longer, and the contrast amount was higher (p=0.001 and p=0.009, respectively).

Independent predictors of new-onset AF development were evaluated using the binary logistic regression model. The model consisting of anterior MI, CHADs-VASc >2, LVEF, KILLIP >2, basal creatinine, peak troponin, and LA diameter was evaluated as the best model (–2 log likelihood: 154.295; Nagelkerke R square: 0.34; chi-squared: 121.269; model p<0.001). Accordingly, CHADs-VASc >2, KILLIP >2, and LA diameter were found to be independent predictors for the development of new-onset AF (Fig. 2a).

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Table 1. Baseline clinical characteristics of patients with STEMI according to development of atrial fibrillation

	AF (–) (n=1315, 93.9%)	AF (+) (n=85, 6.1%)	*P*-value
Age (years)	58.1±11	66.1±13	<0.001
Male, n (%)	1123 (85.4)	60 (70.6)	<0.001
Hypertension, n (%)	463 (35,2)	41 (48,2)	0.015
Diabetes mellitus, n (%)	357 (27.1)	25 (29.4)	0.650
Hyperlipidemia, n (%)	457 (34.8)	20 (23.5)	0.034
Active smoking, n (%)	663 (50.4)	21 (24.7)	<0.001
Previous CVA, n (%)	31 (2.4)	2 (2.4)	0.998
Previous MI, n (%)	216 (16.4)	20 (23.5)	0.090
Previous PCI, n (%)	263 (20.0)	22 (25.9)	0.192
PAD, n (%)	24 (1.8)	4 (4.7)	0.066
Chest pain, h	3.6±4	4.7±5	0.371
Heart rate*	78 (25-164)	78 (35-160)	0.967
Systolic blood pressure (mm Hg)*	128.1±25	120.9±28	0.053
CHADs VASc score	1.6±1	2.7±2	<0.001
$CHADs_2$ score	1.0±1	1.5±1	<0.001
Anterior MI, n (%)	553 (42.1)	27 (31.8)	0.062
Target vessel			
LAD, n (%)	557 (42.4)	30 (35.3)	0.203
Cx, n (%)	237 (18.0)	13 (15.3)	
RCA, n (%)	521 (39.6)	42 (49.4)	
KILLIP score			
KILLIP I-II, n (%)	1235 (93.9)	74 (87.1)	0.013
KILLIP III-IV, n (%)	80 (6.1)	11 (12.9)	
Contrast volume (mL)	274.7±100	307.2±100	0.060
CI-AKI, n (%)	260 (19.8)	21 (24.7)	0.267

*Median (min-max)
$CHADs_2$- cardiac failure, hypertension, age, diabetes, stroke (doubled); CHADSVASC- congestive heart failure, hypertension, age ≥75 years (doubled), diabetes, stroke (doubled), vascular disease, age 65–74 years, and sex category (female); CI-AKI - contrast induced acute kidney injury, CVA - cerebrovascular accident; Cx - circumflex artery; LAD - left anterior descending artery; MI - myocardial infarction; RCA - right coronary artery; PCI - percutaneous coronary intervention; PAD - peripheral artery disease

Table 2. Baseline biochemical and echocardiographic characteristics of patients with STEMI according to development of atrial fibrillation

	AF (–) (n=1315, 93.9%)	AF (+) (n=85, 6.1%)	*P*-value
Hemoglobin (mg/dL)	13.8±2	13.1±2	0.002
Leukocytes (/mm^3)	12471.2 ± 4205	12127.2 ± 4640	0.294
Neutrophil (/mm^3)	9051.8 ± 4118	8748.5 ± 4295	0.280
Lymphocytes (/mm^3)	2377.1 ± 1534	2409.6 ± 1513	0.983
Platelets (x10^3/mm^3)	255.5 ± 95	241.6 ± 66	0.177
Total cholesterol (mg/dL)*	187 (51–494)	174 (64–322)	0.001
LDL cholesterol (mg/dL)*	128 (23–409)	117 (31–709)	0.001
HDL cholesterol (mg/dL)*	37 (13–216)	38 (8–77)	0.237
Triglycerides (mg/dL)*	133 (32–1785)	121 (47–431)	0.058
Glucose (mg/dL)*	122 (56–790)	123 (80–327)	0.913
HbA1c (%)	6.72 ± 1.8	6.5 ± 1.2	0.808
Creatinine (mg/dL)	0.96 ± 0.4	1.06 ± 0.3	<0.001
eGFR (mL/min/1.73 m^2)	86.5 ± 23	72.7 ± 22	<0.001
Peak troponin*	3.48 (0.1–35.0)	4.45 (0.2–30)	0.035
ALT*	28 (10–2395)	33 (10–654)	0.005
AST*	48 (10–1772)	64 (10–1409)	0.044
Albumin	3.8±0.3	3.7±0.4	0.019
LVEF %	46.9±8	44.9±10	0.060
LVd (mm)	49.7±5	50.1±6	0.509
IVS (mm)*	11 (7–24)	11 (9–15)	0.075
LA (mm)	37.5±5	41.4±6	<0.001

*Median (min-max)
ALT - alanine aminotransferase; AST - aspartate aminotransferase; eGFR - estimated glomerular filtration rate; HbA1c - hemoglobin A1c; HDL - high-density lipoprotein; IVS - interventricular septum thickness; LA - left atrium diameter; LDL - low-density lipoprotein; LVd - left ventricular diastolic diameter; LVEF - left ventricular ejection fraction

Clinical outcomes

In our study, MACCEs [death: 145 (10.4%), in-hospital death: 59 (4.2%), MI: 199 (14.2%), SVO: 16 (1.1%)] occurred in 351 (25.1%) patients. Other clinical outcomes, such as hospitalization (5.6%) with cardiac causes and major bleeding (1.5%) occurred in 99 (7.1%) patients. Evaluation of patients in terms of clinical outcomes according to AF development is given in Table 4. In the group with AF, all-cause mortality and in-hospital mortality were found to be significantly higher (p=0.008 and p<0.001, respectively). In addition, hospitalization rates with cardiac causes were significantly higher in the AF (+) group (p=0.003). There were no significant differences in the group with AF in terms of MACCE, MI, CVE, and major bleeding ratios (p=0.487, p=0.191, p=0.976, p=0.072, respectively). The predictors of in-hospital death were evaluated using a binary logistic regression model. The regression model consisting of anterior MI, baseline glucose levels, VT/VF development, AF development, and LVEF was evaluated as the best model (–2 log likelihood: 124.295; Nagelkerke R square: 0.39; chi-square: 124.269; model p<0.001). According to this model, baseline glucose level [odds ratio (OR)=1.008, 95% confidence interval (CI) 1.004–1.011, p<0.001], in-hospital VT/VF development (OR=3.450, 95% CI 1.618–7.360, p<0.001), new-onset AF development (OR=4.022, 95% CI 1.732–9.337, p=0.001), and LVEF (OR=0.862, 95% CI 0.830–0.895, p<0.001) were found to be independent predictors in terms of in-hospital death (Fig. 2b). According to AF development, Kaplan-Meier curves for MACCE and all-cause death are given in Figure 3.

Multivariable analysis

In our study, MACCE predictors were analyzed by a Cox regression multivariable model (Fig. 2c). For the long-term follow-up, a multivariable proportional Cox regression analysis

Table 3. Baseline clinical characteristics and biochemical characteristics of patients with STEMI according to development of MACCE

	MACCE (–) (n=1049, 74.9%)	MACCE (+) (n=351, 25.1%)	*P*-value
Age (years)	58.1±11	66.1±13	0.008
Male, n (%)	886 (84.5)	297 (84.6)	0.945
Hypertension, n (%)	336 (34.9)	138 (39.3)	0.135
Diabetes mellitus, n (%)	266 (25.4)	116 (33.0)	0.005
Hyperlipidemia, n (%)	321 (30.6)	156 (44.4)	<0.001
Active smoking, n (%)	176 (24.6)	175 (25.6)	0.665
Previous CVA, n (%)	17 (1.6)	16 (4.6)	<0.001
Previous MI, n (%)	154 (14.7)	82 (23.4)	<0.001
Previous PCI, n (%)	191 (18.2)	94 (26.8)	0.001
Chest pain, h	3.3±3	4.3±5	0.817
Door to balloon (min)	46.9±30	54.1±36	0.001
Heart rate*	76 (25–164)	80 (30–160)	<0.001
Systolic blood pressure (mm Hg)*	129.8±23	120.9±29	<0.001
CHADs VASc score >2	256 (24.7)	132 (39.2)	<0.001
Anterior MI, n (%)	425 (40.5)	155 (44.2)	0.230
KILLIP score >2	32 (3.1)	59 (16.8)	<0.001
In-hospital AF	61 (5.8)	24 (6.8)	0.487
In-hospital VT/VF	74 (7.1)	38 (10.8)	0.024
Discharged anticoagulation	26 (2.5)	9 (2.9)	0.928
Contrast volume (mL)	269.7±99	290.3±104	0.009
Hemoglobin (mg/dL)	13.8±2	13.7±2	0.288
Leukocytes (/mm^3)	12363.7±3994	12681.7±4678	0.338
LDL cholesterol (mg/dL)	129.9±37	126.2±54	0.004
Triglycerides (mg/dL)*	131 (32–936)	135 (33–1785)	0.616
Glucose (mg/dL)*	119 (56–495)	128 (62–616)	<0.001
Creatinine (mg/dL)	0.93±0.3	1.07±0.7	<0.001
Peak troponin*	3.32 (0.1–35.0)	4.29 (0.1–30)	0.003
LVEF %	47.9±8	42.9±9	<0.001

*Median (min-max)
AF - atrial fibrillation; CHADs VASc - congestive heart failure, hypertension, age ≥75 (doubled), diabetes, stroke (doubled), vascular disease, age 65–74, and sex category (female); CVA - cerebrovascular accident; MI - myocardial infarction; LDL - low-density lipoprotein; LVEF - left ventricular ejection fraction; PCI - percutaneous coronary intervention; MACCE - major cardiac and cerebrovascular events (death + MI + CVA); VT - ventricular tachycardia; VF - ventricular fibrillation

model was performed for MACCE development according to the following variables: age, male sex, DM, HL, peripheral artery disease, prior MI, door to balloon time, systolic blood pressure, KILLIP score >2, baseline glucose level, baseline creatinine level, LVEF, new-onset AF, and in-hospital VT/VF. HL [hazard ratio (HR)=1.607, 95% CI 1.239–2.085, p<0.001), door to balloon time (HR=1.004, 95% CI 1.000–1.007, p=0.043), baseline systolic blood pressure (HR=0.993, 95% CI 0.988–0.998, p=0.010), KILLIP score >2 (HR=2.459, 95% CI 1.678–3.603, p<0.001), baseline glucose level (HR=1.003, 95% CI 1.001–1.005, p=0.001), baseline creatinine level (HR=1.323, 95% CI 1.136–1.541, p<0.001), and LVEF (HR=0.956, 95% CI 0.942–0.970, p<0.001) were determined to be independent predictors for MACCE development.

Table 4. Clinical outcomes of patients with STEMI according to development of atrial fibrillation

	AF (–) n=1315	AF (+) n=85	*P*-value
Death, n (%)	129 (9.8)	16 (18.8)	0.008
MI, n (%)	191 (14.5)	8 (9.4)	0.191
CVA, n (%)	15 (1.1)	1 (1.2)	0.976
MACCE, n (%)	327 (24.9)	24 (28.2)	0.487
In-hospital death, n (%)	49 (3.7)	10 (11.8)	<0.001
In-hospital VT/VF, n (%)	95 (7.2)	17 (20.0)	<0.001
Hospitalization, n (%)	68 (5.2)	11 (12.9)	0.003
Major bleeding, n (%)	18 (1.4)	2 (2.3)	0.072

AF - atrial fibrillation; CVA - cerebrovascular accident; MACCE - major cardiac and cerebrovascular events (death + MI + CVA); MI - myocardial infarction; VT - ventricular tachycardia; VF - ventricular fibrillation; STEMI - ST elevation myocardial infarction

Discussion

The most comprehensive data on new-onset AF in patients with STEMI were derived from a systemic review of approximately 20 studies conducted by Schmitt et al. (1). In the review, it was reported that the studies on the subject were mostly obtained from data belonging to the thrombolytic era (patient inclusion from 2007 and before). According to this review, it is reported that the frequency of AF during acute MI was between 6.8% and 21%. In addition, as a result of the review, it was reported that AF development based on AMI increased in-hospital and long-term mortality. In a multicenter and retrospective study by Garg et al. (6), AF was accompanied by STEMI in 8.7% of patients and was linked to increased in-hospital mortality, hospitalization time, and stroke. However, the method used in STEMI treatment and the long-term clinical outcomes of the study were unclear and included known AF in the study population. In a study published in 2020 by Zhang et al. (10), 750 patients with ACS (42.5% STEMI, 18.3% non-STEMI, and 39.2% unstable angina pectoris) were enrolled. They found that an AF attack developed in 6.7% of patients during a four-year follow-up. In the long-term follow-up, NT-proBNP, creatinine kinase MB, and LVEF were detected as AF predictors. In this study, in-hospital AF prevalence and long-term clinical outcome data were not reported (10). Lau et al. (5) included 607 patients with STEMI and determined new-onset AF in 13.7% of patients during 60 months of follow-up. In addition, women were found to be more prevalent in the group with AF (+), and LVEF was found to be less prevalent in the study. However, the rates of new-onset AF were not indicated in-hospital and only the new-onset AF rates in follow-up after discharge of the patients were given. In addition, mortality data were not available (5). Siu et al. (4) have found the

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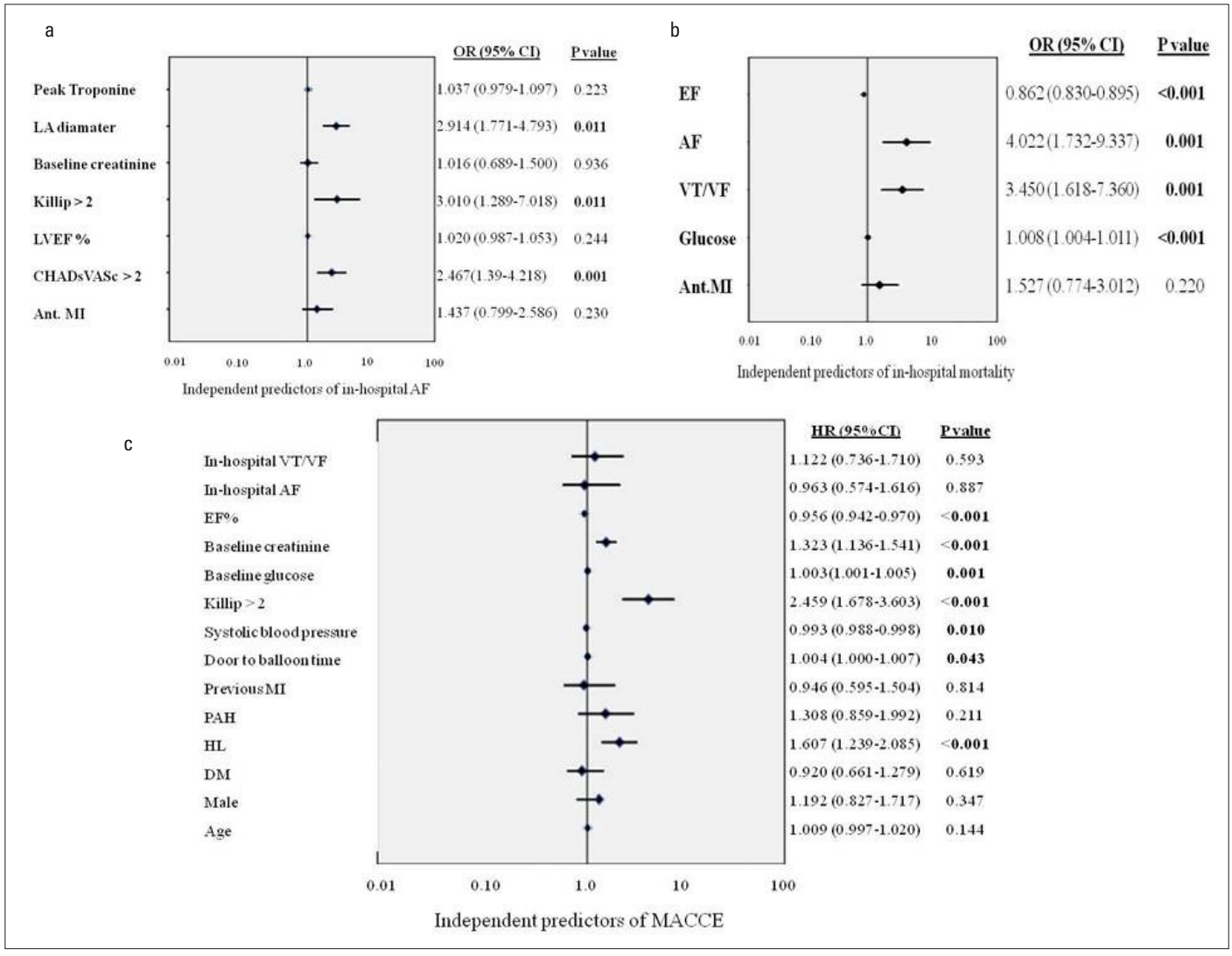

Figure 2. Forest plots displaying independent predictors of in-hospital atrial fibrillation (a), in-hospital mortality (b), and major cardiac and cerebrovascular events (c)

rate of in-hospital new-onset AF to be 13.7% in 504 patients with acute inferior MI with a previously unknown diagnosis of AF. The mean age and frequency of women were found to be higher in the group with AF. Mortality rates were similar at the one-year follow-up, but stroke was observed more frequently in the group with AF (+). However, only 30% of patients who developed AF at discharge were prescribed oral anticoagulant agents. Current guidelines recommend that anticoagulant treatment should be given in the long-term follow-up according to the CHADs-VASc stroke score and the HAS-BLEED bleeding score in patients with new-onset AF with STEMI (9). However, most of the literature studies do not have sufficient data on this subject. An oral anticoagulant agent was administered to only 30% of the patients who developed new-onset AF during hospitalization for STEMI (4).

Age, hypertension, male sex, and CAHDs-VASc and KILLIP scores have been extensively studied as AF predictors and are known to be significant risk factors (11-13). Similar results were encountered in our study, except the female sex ratio, which was found to be higher in the group with AF (+). In addition, CHADs and CHADs-VASc scores, which are used to predict thromboembolic events in patients with AF, have been recently investigated for their effects on mortality in ACS. The CHADs-VASc score especially has been reported to predict in-hospital and long-term mortality in patients with ACS (14, 15). The CHADs-VASc score also was found to be an important predictor of in-hospital mortality in our study.

In our study, the incidence of new-onset AF was found to be lower than the literature data. Similar to the literature, in-hospital mortality and all-cause mortality were found to be higher in patients with AF. Kaplan-Meier survival analyses show that the increase in all-cause mortality is mainly driven by in-hospital deaths. In addition, no significant increase in the frequency of stroke or MI was observed in our study. We think that this situation can be explained in two ways. First, the inclusion of patients who underwent primary PCI, the administration of dual antiplatelet agents for at least one year, high-dose statin, and effec-

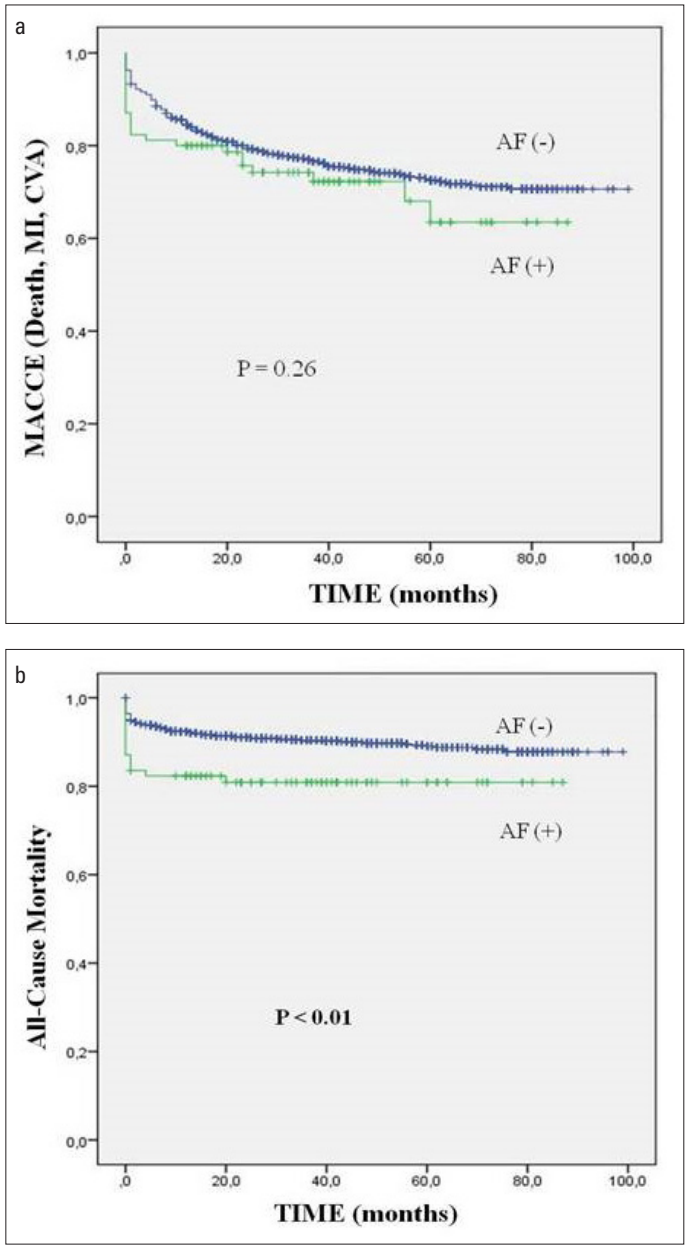

Figure 3. Kaplan-Meier cumulative event curves for all-cause death and major cardiac and cerebrovascular events in long-term follow-up of patients with and without in-hospital atrial fibrillation

tive dose ACE-I treatment, and the addition of an anticoagulant agent to approximately one-third of patients with AF may affect the frequencies of stroke and MI. Second, the absence of known AF diagnoses in the patients included in the study, the inclusion of only patients with in-hospital AF, and all patients being in sinus rhythm at the time of discharge may have affected the rates. When focusing on Kaplan-Meier curves, we see that in-hospital mortality rates in patients with AF affect long-term data. This situation suggests that the effects of in-hospital AF on long-term mortality decreased when the patient was discharged and stabilized. The mechanism of the increase in the incidence of atrial arrhythmias in STEMI is still unknown. However, atrial ischemia or infarction, acute hypoxia or hypokalemia, pericardial inflammation, increased LV diastolic pressure and left atrial pressure, hemodynamic impairment because of LV dysfunction, and autonomic regulation are thought to lead to AF development (16). As mentioned above, in-hospital AF that develops based on STEMI has many causes, and most of them are conditions that can be eliminated in the future with early intervention in patients with STEMI. Therefore, patients with STEMI who develop AF should be followed up closely in the early period, and when the situation stabilizes in the long term, risk assessment can be performed again and evaluated in terms of anticoagulation as a treatment option.

Study limitations

There were several limitations and uncertainties in the literature data, such as patient admissions in 2013 and before, thrombolytic therapy era, follow-up data limited to one year, patients with known AF were not excluded from the study, and the rates of in-hospital AF and anticoagulant treatment rates after discharge on this subject were not determined. In our study, unlike the literature, analyses were performed only according to patients with new-onset AF in-hospital, and primary PCI was applied to all patients. In addition, in-hospital mortality and long-term major adverse event data were noted in our study.

The facts that the study was single-center, and telemetry follow-up was limited to the first 48 hours constituted important limitations of our study. Other limitations were that brain natriuretic peptide monitoring could not be performed (insurance reimbursement problems), and scores indicating the severity of coronary artery disease (SYNTAX, ACC lesion classification, etc.) were not used.

Conclusion

In our study, we found that in-hospital new-onset AF rates were lower during the primary PTCA era. We also found that the development of new-onset AF was associated with a four-fold increase in in-hospital mortality. Another important result of our study was that in-hospital AF development did not cause a significant increase in long-term clinical events such as stroke and myocardial infarction. Therefore, we believe that it would be appropriate to closely follow new-onset AF during the STEMI process in the early period, but re-evaluate them in terms of long-term treatments when the condition becomes stable. Investigating this situation with actual multi-center studies on the subject will remove the uncertainties.

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