ORIGINAL ARTICLE

Predictors of new-onset atrial fibrillation in elderly patients with acute coronary syndrome undergoing percutaneous coronary intervention

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Abstract

Background The development of atrial fibrillation (AF) during the course of acute coronary syndrome (ACS) is related to poor prognosis. Possible predictors of new-onset AF (NOAF) have not been adequately investigated in elderly patients with ACS undergoing percutaneous coronary intervention (PCI). We aimed to identify the factors associated with NOAF in such patients.

Methods A total of 308 elderly patients with ACS undergoing PCI were enrolled in the study. Patients were divided into two groups: without NOAF [254 patients, 64.6% men, age: 73.5 (69.0–79.0) years] and with NOAF [54 patients, 70.4% men, age: 75.0 (68.7–81.2) years]. Clinical, angiographic, and laboratory features including neutrophil-to-lymphocyte ratio (NLR) and monocyte-to-high-density lipoprotein ratio (MHR) were compared between the groups.

Results The percentages of prior myocardial infarction (MI) (20.4 vs. 5.9%) and Killip III/ IV (24.1 vs. 7.1%), NLR [4.5 (2.6–7.2) vs. 3.2 (2.0–6.0)], and MHR [19.4 (15.7–26.5) vs. 12.9 (9.9–18.5)] were higher in patients with NOAF compared to the others ($p = 0.020$, < 0.001, 0.030, and < 0.001, respectively). In multivariate regression analysis, prior MI (OR 4.509, 95% CI 1.679–12.106, $p = 0.003$) and MHR (OR 1.102, 95% CI 1.054–1.152, $p < 0.001$) independently predicted NOAF. In addition, Killip III/IV was found to be an independent predictor of 6-month overall mortality (HR 2.949, 95% CI 1.218–7.136, $p = 0.016$).

Conclusions Prior MI and MHR are independent predictors of NOAF in elderly patients with ACS undergoing PCI. Killip III/IV predicts 6-month overall mortality in such patients.

Keywords Atrial fibrillation · Acute coronary syndrome · Coronary intervention · Elderly

Introduction

Acute coronary syndrome (ACS) is a major cause of death, and older patients with ACS have worse prognosis than younger patients [1, 2]. Despite their high morbidities and complications, percutaneous coronary intervention (PCI) is still beneficial in elderly patients with ACS [3]. Atrial fibrillation (AF) often complicates ACS and the incidence of new-onset AF (NOAF) after ACS ranges from 2.3 to 37% [4–6]. Preexisting AF accounts for approximately one-third of all cases of AF observed in patients with acute myocardial infarction (MI), and NOAF for the remaining two-thirds [7]. Patients with acute MI and AF have a higher incidence of reinfarction, cardiogenic shock, heart failure, and asystole [4–6]. For these reasons, early identification of individuals at high risk for the development of AF is crucial to prevent complications related to it.

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Older age has been found to be an independent predictor of the development of AF in patients with ACS [4–6]. In addition, higher Killip class, atrial ischemia, left atrial enlargement, and inflammation are other possible mechanisms of NOAF complicating ACS [4–6]. The neutrophil-to-lymphocyte ratio (NLR) and monocyte-to-high-density lipoprotein (HDL) ratio (MHR) have been suggested as indicators of inflammation and oxidative stress, and found to be independent predictors of adverse cardiac events in patients with ACS [8, 9].

Possible predictors of NOAF have not been adequately investigated in elderly patients with ACS undergoing PCI. The aim of the study was to investigate predictors of NOAF in elderly patients with ACS undergoing PCI.

Methods

Study population

Between June 2016 and April 2017, a total of 321 patients older than 65 who underwent primary PCI for ST-segment elevation MI (STEMI) or underwent urgent PCI for non-ST elevation ACS (NSTE-ACS) were enrolled in this prospective study. Patients with end-stage renal disease [estimated glomerular filtration rate (eGFR) < 15 mL/min/1.73 m^2] or receiving long-term dialysis ($n = 6$), patients who died during or early after the procedure ($n = 3$), patients with acute or chronic infection or inflammatory conditions ($n = 1$), patients who had an absence of data on serum creatinine during the 72 h after the procedure ($n = 3$) were excluded. A total of 308 patients were included in this study. Demographic, clinical variables, and medications used after admission were recorded. The diagnosis of STEMI was based on the clinical characteristics, electrocardiographic (ECG) changes consisting of persistent ST-elevation of at least 0.1 mV in at least two contiguous leads or a left bundle branch block with subsequent Q-wave formation, and biochemical markers of cardiac necrosis [creatinine kinase-MB isoenzyme (CK-MB) or troponin-T] [1]. The diagnosis of NSTE-ACS was based on the clinical characteristics, transient ST depression (≥ 0.05 mV) or T-wave inversion (≥ 0.1 mV), and a troponin-T value above the 99th percentile of upper limit of normal range [2]. Previous history of coronary artery disease (CAD) was defined as a definitive history of MI or coronary obstruction $\geq 50\%$ on coronary angiography (CAG) [10]. Contrast-induced nephropathy (CIN) was defined as the elevation of serum creatinine > 0.5 mg/dl or 25% versus baseline serum creatinine within 72 h after PCI [11]. The study was approved by the Ethics Committee of Eskisehir Osmangazi University, Faculty of Medicine, and was conducted in accordance with the principles of the Declaration of Helsinki. All patients were informed about the nature of the study and a written informed consent was obtained.

Blood analysis, electrocardiography, and echocardiography

Venous blood samples were drawn from antecubital veins immediately after the ECG recordings. The NLR, MHR, and mean platelet volume (MPV) were obtained using the same blood samples obtained before PCI. Baseline serum creatinine was measured at admission (before PCI), and on days 1, 2, and 3 after the procedure. The eGFR was calculated using the modification of diet in renal disease formula [12]. The infarct size was estimated by peak CK-MB and peak troponin-T values, defined as the highest serum concentrations within the first 48 h. Transthoracic echocardiography was performed using an echocardiography system (Acuson Sequoia C256, Mountain View, CA, USA) equipped with a broadband transducer (3V2c) for all patients within the first 48 h of hospitalization. Two-dimensional echocardiographic measurements were performed according to the guideline recommendations [13]. The Simpson's method was used to evaluate left ventricular ejection fraction (LV EF).

Angiographic definitions

All patients underwent CAG preceded by an intracoronary injection of 100 μg of nitroglycerine. All procedures were performed through the femoral or radial approach by experienced interventional cardiologists. Nonionic, low-osmolar contrast medium (iohexol, Omnipaque 350 mg/mL; GE Healthcare, Cork, Ireland) was used. All the patients were treated with a P2Y12 antagonist (clopidogrel 600 mg, ticagrelor 180 mg, or prasugrel 60 mg), and dual antiplatelet therapy (aspirin 100 mg/day and clopidogrel 75 mg/day or ticagrelor 180 mg/day or prasugrel 10 mg/day) continued for at least 12 months. All PCI procedures were performed using unfractionated heparin (100 U/kg). Tirofiban therapy was initiated in the angiography laboratory during PCI in a bail-out situation or for thrombotic complications (downstream therapy). Stent thrombosis was defined as the presence of ACS with angiographic evidence of thrombus or occlusion. Target lesion revascularization (TVR) was defined as any repeated PCI of the stented segment, including 5-mm proximal and distal margins [14]. The type of the stents [bare metal or drug-eluting stent] was left to the operator's discretion. Balloon angioplasty alone was preferred for the patients who developed stent thrombosis (in-stent balloon angioplasty) or who had a vessel diameter of < 2.25 mm. The patients who had a failed coronary intervention or who had no suitable vessel for coronary revascularization were managed conservatively.

Arrhythmic events

The presence of AF was documented by a standard 12-lead ECG. All patients were followed by an inpatient cardiac telemetry (Nihon Kohden Central Monitor System, CNS-9701, Tokyo, Japan and Mortara Surveyor Central System, Milwaukee, WI, USA) for the development of AF during the hospital stay. New-onset AF was defined as the development of at least one episode of AF as assessed by ECG or telemetry during the index hospitalization. Nonsustained ventricular tachycardia (VT) was defined as self-limiting VT events lasting more than 16 consecutive beats with a rate of > 125 beat/min and a duration of 8–30 s. Sustained VT was defined as any VT event lasting more than 16 consecutive beats with a rate of > 125 beat/min and a duration of ≥ 30 s [15].

Clinical endpoints

We evaluated the relationship between NOAF and clinical outcomes during hospitalization, at the 1-month and 6-month follow-up. The MACE (major adverse cardiac event) was defined as cardiovascular death, cardiogenic shock, sudden cardiac death, stent thrombosis, recurrent MI, TVR, or stroke. Major bleeding was defined as intracranial hemorrhage, ≥ 5 g/dL decrease in hemoglobin concentration, or ≥ 15% absolute decrease in hematocrit. Minor bleeding was defined as a blood loss of 3–5 g/dL in the hemoglobin concentration or 10–15% decrease in hematocrit values, or no visible blood loss with ≥ 4 g/dL decrease in the hemoglobin concentration and ≥ 12% decrease in the hematocrit values [16].

Statistical analysis

Statistical analysis was performed using the SPSS version 16.0 software (SPSS Inc., Chicago, IL, USA). The categorical variables are expressed in frequencies and percentages. The Pearson's Chi-square test and Fisher's exact test were used to compare categorical variables. Continuous variables were tested for normal distribution by the Shapiro–Wilk test. Normally distributed variables were presented as mean ± SD and compared using independent samples t test. Abnormally distributed variables were expressed in median and interquartile ranges [25th–75th percentile levels], and Mann–Whitney U test was used to determine the significant differences among the groups. Multiple binary logistic regression analysis, Enter method was used to define the independent predictors of NOAF. The goodness-of-fit assumption was assessed using the Hosmer–Lemeshow method (Chi-square = 4.128, $df = 8$, $p = 0.845$). Receiver operating characteristic (ROC) curve was generated to define the cut-off value of MHR for NOAF and area under the curve (AUC) was calculated. Time-dependent Cox-proportional hazards regression model was used to examine the independent predictors of 6-month overall mortality. A p value of < 0.05 was considered statistically significant.

Results

A total of 308 elderly patients were enrolled in the study [204 were males (66.2%) and mean age was 74.4 ± 6.5 (range 65–90)]. Of these patients, 204 (66.2%) had STEMI and NOAF was present in 54 (17.5%). The MACEs were observed in 69 patients (22.4%) and 48 (15.5%) died during follow-up.

Clinical, laboratory, and angiographic features of the groups are presented in Table 1. Patients with NOAF were more likely to have prior MI, Killip III/ IV, inotropic support, triple vessel disease, and CTO compared with patients without NOAF. The LV EF, eGFR, HDL-cholesterol, and lymphocyte count were lower in the NOAF group. Contrast volume, white blood cell count, peak CK-MB, peak troponin-T, NLR, MHR, and MPV were higher in patients with NOAF.

Table 2 presents arrhythmic events and clinical endpoints. Contrast-induced nephropathy, MACE at 1-month follow-up, MACE at 6-month follow-up, overall mortality at 1-month follow-up and overall mortality at 6-month follow-up were more prevalent in patients with NOAF. Rates of cardiogenic shock and recurrent MI at 6-month follow-up were higher in such patients. Rates of arrhythmic events and bleeding were similar between the groups.

In the multiple logistic regression analysis, prior MI and MHR were independent predictors for NOAF (Table 3). The ROC curve analysis showed that the best cut-off point of MHR was 15.87 to identify patients with NOAF (sensitivity: 75.9%, specificity: 65.0%, AUC 0.750, 95% CI 0.698–0.798, $p < 0.001$) (Fig. 1).

In the Cox-proportional hazards modeling, Killip III/ IV (HR 2.949, 95% CI 1.218–7.136, $p = 0.016$) predicted 6-month overall mortality (Table 4).

Discussion

The present study showed that prior MI and MHR were independent predictors of the development of AF in elderly patients with ACS undergoing PCI. Using a cut point of > 15.87, MHR predicted NOAF with a sensitivity of 75.9% and specificity of 65.0%. When a cut-off point of 4.41 was used, NLR predicted NOAF with a sensitivity of 53.7% and specificity of 65.0%. We found that Killip III/IV was an independent predictor of 6-month overall mortality in such patients.

Table 1 Baseline demographic, clinic, echocardiographic, angiographic, and laboratory features of the groups

	NOAF− (n = 254)	NOAF+ (n = 54)	p value
Age (years)	73.5 (69.0–79.0)	75.0 (68.7–81.2)	0.414
Sex (male) (n, %)	164 (64.6)	38 (70.4)	0.415
BMI (kg/m^2)	25.5 (23.4–27.5)	25.7 (23.4–28.0)	0.584
Current smoking (n, %)	68 (26.8)	20 (37.0)	0.129
Diabetes mellitus (n, %)	101 (39.8)	16 (29.6)	0.164
Hypertension (n, %)	142 (55.9)	28 (51.9)	0.586
Malignancy (n, %)	13 (5.1)	4 (7.4)	1.512
Prior CAD (n, %)	89 (35.0)	26 (48.1)	0.071
Prior MI (n, %)	15 (5.9)	11 (20.4)	0.002
Prior stroke (n, %)	11 (4.3)	2 (3.7)	1.000
Prior COPD (n, %)	28 (11.0)	3 (5.6)	0.225
STEMI (n, %)	164 (64.6)	40 (74.1)	0.180
Systolic BP (mmHg)	122.5 (110.0–140.0)	119.0 (100.0–140.0)	0.169
Diastolic BP (mmHg)	70.0 (60.0–80.0)	70.0 (58.5–81.2)	0.361
Heart rate (beats/min)	80.0 (70.0–92.0)	86.0 (71.5–101.0)	0.107
Killip III/ IV (n, %)	18 (7.1)	13 (24.1)	< 0.001
LV EF (%)	45.0 (33.0–55.0)	36.5 (27.0–48.5)	0.006
LA diameter (mm)	40.3 ± 5.3	40.7 ± 5.1	0.575
MR (grade > II/IV)	36 (14.2)	9 (16.7)	0.638
Medications			
Beta blocker (n, %)	204 (80.3)	43 (79.6)	0.909
ACE-I/ARB (n, %)	143 (56.3)	23 (42.6)	0.067
Statin (n, %)	240 (94.5)	49 (90.7)	0.346
Calcium antagonist (n, %)	28 (11.0)	4 (7.4)	0.429
Tirofiban (n, %)	52 (20.5)	14 (25.9)	0.375
Inotropic support (n, %)	21 (8.3)	10 (18.5)	0.023
Triple vessel disease (n, %)	90 (35.4)	30 (55.6)	0.006
CTO (n, %)	44 (17.3)	16 (29.6)	0.038
Contrast volume (mL)	170 (160–178)	175 (165–180)	0.028
IRA			
LAD (n, %)	90 (35.4)	20 (37.0)	0.823
CX (n, %)	65 (25.6)	11 (20.4)	0.419
RCA (n, %)	81 (31.9)	19 (35.2)	0.639
Bare metal stent (n, %)	43 (16.9)	7 (13.0)	0.473
Drug-eluting stent (n, %)	155 (61.0)	36 (66.7)	0.438
Angioplasty alone (n, %)	17 (6.7)	1 (1.9)	0.216
Conservative follow-up (n, %)	33 (13.0)	12 (22.2)	0.081
CABG after CAG (n, %)	9 (3.5)	1 (1.9)	1.000
Basal hemoglobin (g/dL)	13.2 ± 2.0	13.1 ± 1.5	0.918
White blood cell (× 10^9/L)	9.3 (7.4–11.4)	10.7 (8.7–12.7)	0.004
Platelet count (× 10^9/L)	230.5 (193.0–280.2)	236.5 (194.0–271.2)	0.729
Fasting glucose (mg/dL)	118.5 (99.0–162.2)	114.5 (97.5–142.2)	0.278
eGFR (mL^{-1} min^{-1} 1.73 m^{-2})	69.7 ± 21.6	61.3 ± 20.4	0.009
Total cholesterol (mg/dL)	179.0 (156.0–204.2)	165.5 (143.7–202.0)	0.249
HDL (mg/dL)	41.0 (32.7–51.0)	36.0 (32.0–42.2)	0.009
LDL (mg/dL)	116.0 (95.0–137.5)	114.0 (79.7–133.2)	0.310
Triglyceride (mg/dL)	126.0 (100.0–175.0)	134.0 (96.7–175.7)	0.810
Peak CK-MB (ng/mL)	34.0 (8.6–108.0)	72.8 (22.5–164.3)	0.019
Peak troponin-T (ng/mL)	1.7 (0.4–5.8)	3.9 (1.6–7.7)	0.004
Neutrophil count (× 10^9/L)	68.1 (59.7–79.6)	72.3 (63.3–80.7)	0.085
Lymphocyte count (× 10^9/L)	21.2 (13.1–29.0)	16.3 (11.0–24.0)	0.022

Table 1 (continued)

	NOAF− (n = 254)	NOAF+ (n = 54)	p value
Monocyte count (×10⁹/L)	6.3 (4.3–8.1)	6.7 (5.6–8.6)	0.058
NLR	3.2 (2.0–6.0)	4.5 (2.6–7.2)	0.030
MHR	12.9 (9.9–18.5)	19.4 (15.7–26.5)	< 0.001
MPV (fl)	8.6 (7.8–9.5)	9.1 (8.3–9.9)	0.014

ACE-I angiotensin-converting enzyme inhibitor, *ARB* angiotensin receptor blocker, *BMI* body mass index, *BP* blood pressure, *CABG* coronary artery bypass grafting, *CAD* coronary artery disease, *CAG* coronary angiography, *CKMB* creatine kinase MB isoenzyme, *COPD* chronic obstructive pulmonary disease, *CTO* chronic total occlusion, *CX* circumflex coronary artery, *eGFR* estimated glomerular filtration rate, *HDL* high-density lipoprotein, *IRA* infarct related artery, *LA* left atrium, *LAD* left anterior descending artery, *LDL* low-density lipoprotein, *LV EF* left ventricular ejection fraction, *MHR* monocyte-to-high-density lipoprotein ratio, *MI* myocardial infarction, *MPV* mean platelet volume, *MR* mitral regurgitation, *NLR* neutrophil-to-lymphocyte ratio, *NOAF* new-onset atrial fibrillation, *RCA* right coronary artery, *STEMI* ST-segment elevation myocardial infarction

Table 2 Clinical endpoints

	NOAF− (n = 254)	NOAF+ (n = 54)	p value
CIN (n, %)	42 (16.5)	16 (29.6)	0.025
Arrhythmic events			
Nonsustained/sustained VT (n, %)	15 (5.9)	6 (11.1)	0.228
VF (n, %)	9 (3.5)	5 (9.3)	0.278
2/3 degree AV block (n, %)	17 (6.7)	4 (7.4)	0.771
In-hospital adverse events			
MACE (n, %)	24 (9.4)	8 (14.8)	0.241
Mortality (n, %)	22 (8.7)	8 (14.8)	0.166
Cardiogenic shock (n, %)	19 (7.5)	8 (14.8)	0.108
Reinfarction (n, %)	2 (0.8)	0 (0)	1.000
Stent thrombosis (n, %)	1 (0.4)	0 (0)	1.000
TVR (n, %)	2 (0.8)	0 (0)	1.000
Stroke (n, %)	2 (0.8)	0 (0)	1.000
One-month adverse events			
MACE (n, %)	32 (12.6)	14 (25.9)	0.013
Mortality (n, %)	25 (9.8)	12 (22.2)	0.011
Cardiogenic shock (n, %)	22 (8.7)	9 (16.7)	0.077
Reinfarction (n, %)	7 (2.8)	4 (7.4)	0.107
Stent thrombosis (n, %)	3 (1.2)	1 (1.9)	0.539
TVR (n, %)	4 (1.6)	2 (3.7)	0.284
Stroke (n, %)	2 (0.8)	1 (1.9)	0.440
Six-month adverse events			
MACE (n, %)	50 (19.7)	19 (35.2)	0.013
Mortality (n, %)	34 (13.4)	14 (25.9)	0.021
Cardiogenic shock (n, %)	23 (9.1)	11 (20.4)	0.016
Reinfarction (n, %)	14 (5.5)	8 (14.8)	0.035
Stent thrombosis (n, %)	5 (2.0)	1 (1.9)	1.000
TVR (n, %)	9 (3.6)	4 (7.4)	0.255
Stroke (n, %)	3 (1.2)	1 (1.9)	0.539
Bleeding events			
Major bleeding (n, %)	1 (0.4)	1 (1.9)	0.320
Minor bleeding (n, %)	10 (3.9)	2 (3.7)	1.000

AV atrioventricular, *CIN* contrast-induced nephropathy, *MACE* major adverse cardiac event, *NOAF* new-onset atrial fibrillation, *TVR* target vessel revascularization, *VT* ventricular tachycardia, *VF* ventricular fibrillation

Table 3 Independent predictors of new-onset atrial fibrillation

	OR	95% CI	*p* value
Prior MI	4.509	1.679–12.106	0.003
Killip III/ IV	2.008	0.769–5.243	0.155
Triple vessel disease	1.813	0.900–3.655	0.096
CIN	1.115	0.500–2.488	0.791
eGFR	0.994	0.977–1.011	0.503
Peak troponin-T	1.071	0.964–1.189	0.200
NLR	1.048	0.983–1.117	0.150
MHR	1.102	1.054–1.152	< 0.001
MPV	1.120	0.861–1.457	0.399

Results of the multiple logistic regression analysis

CIN contrast-induced nephropathy, *eGFR* estimated glomerular filtration rate, *MHR* monocyte-to-high-density lipoprotein ratio, *MI* myocardial infarction, *MPV* mean platelet volume, *NLR* neutrophil-to-lymphocyte ratio
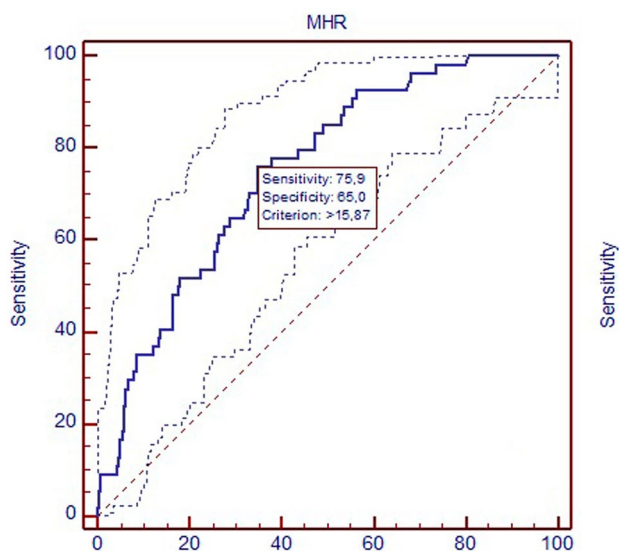
Table 4 Independent predictors of 6-month overall mortality

	HR	95% CI	*p* value
Age	0.990	0.939–1.044	0.722
NOAF	1.329	0.488–3.624	0.578
Prior MI	0.769	0.210–2.820	0.692
Killip III/IV	2.949	1.218–7.136	0.016
CIN	1.804	0.834–3.900	0.134
Triple vessel disease	1.243	0.568–2.719	0.586
eGFR	0.990	0.972–1.009	0.316
NLR	1.038	0.984–1.096	0.172
MHR	1.023	0.985–1.062	0.249
MPV	0.940	0.738–1.196	0.614

Results of the Cox-proportional hazard model

CIN contrast-induced nephropathy, *eGFR* estimated glomerular filtration rate, *MHR* monocyte-to-high-density lipoprotein ratio, *MI* myocardial infarction, *MPV* mean platelet volume, *NLR* neutrophil-to-lymphocyte ratio, *NOAF* new-onset atrial fibrillation

Fig. 1 Receiver operating characteristic curve analysis suggested that the optimal MHR level cut-off point for patients with NOAF was 15.87 with a sensitivity and specificity of 75.9 and 65.0%, respectively (AUC 0.750; 95% confidence interval 0.698–0.798). *MHR* monocyte-to-high-density lipoprotein ratio, *NOAF* new-onset atrial fibrillation

Despite advanced treatment options, adverse cardiovascular events, such as NOAF, can occur during the course of ACS and it is related to poor prognosis [4–6]. Older age was found to be a risk factor for NOAF in patients with ACS [4–6]. In our elderly population, the rate of patients who developed AF at the course of ACS was 17.5%. For this reason, NOAF during the course of ACS is very important in elderly population.

We found that NOAF was associated with increased incidence of adverse events during hospitalization, 1-month and 6-month follow-up in elderly patients with ACS. This finding emphasizes the prognostic significance of NOAF in such patients. However, NOAF was not independent predictor for overall mortality in our study, perhaps associated with confounding factors. Killip III/IV was the only independent predictor for overall mortality in the present study.

Prior MI was an independent predictor of NOAF in elderly patients with ACS in the study. The number of patients with higher Killip class, complex coronary anatomy, and CIN were higher in elderly patients with NOAF in the present study. The LV EF and eGFR were lower, and biomarkers of myocardial damage were higher in such patients. These findings are consistent with the literature [4–6, 17–19]. However, they were not independent predictors for NOAF. In our study, white blood cell count at admission was higher, and HDL was lower in the NOAF group. These results may be related to extensive inflammation in patients with ACS who developed AF [20]. Interestingly, contrast media volume was higher in patients with NOAF in the study. These findings can be explained by the more complex coronary anatomy in such patients. Previous studies have also demonstrated VT, VF, and complete atrioventricular block are more prevalent in patients with NOAF [17, 18]. We have not found a similar result and this could be due to the low-number of patients with NOAF in our study.

Inflammation plays a substantial role in atherosclerotic plaque rupture [21] and it has also an important role in the onset of AF complicating ACS [4, 22]. Some biomarkers have been studied for prediction of NOAF in patients with ACS. The NLR has been found to be an independent predictor of NOAF in patients with STEMI [23, 24]. Circulating monocytes account for the major source of various proinflammatory and prooxidant factors [25, 26]. On the other hand, HDL-cholesterol has well known anti-inflammatory,

antioxidant, and antithrombotic effects [27]. The MHR has been suggested as a novel vascular inflammatory marker and it has been shown to predict NOAF in patients with STEMI [22]. The MPV is an important biomarker of platelet activity and it has a prognostic value in patients with ACS treated with PCI [28]. Karatas et al. have shown an association between MPV and NOAF in patients with STEMI [23].

However, such biomarkers have not been adequately investigated in elderly patients with ACS undergoing PCI. We found that pre-procedural MHR was an independent predictor for NOAF in such patients. This result indicates the importance of inflammation on NOAF in the course ACS. As an inflammatory marker, the MHR has the advantages of being able to be obtained before the procedure and to work in most centers. Pre-procedural MHR measurement may help to early identify elderly patients with ACS having the highest risk of NOAF. In our study, NLR and MPV were significantly higher in the NOAF group, but they were not independent predictors for NOAF.

Limitations

Our study is a single-center study. The number of elderly patients with NOAF was small, which could limit the number of independent predictors identified. Unfortunately, our study lacks information about CRP. Pre-procedural CRP has been demonstrated to be a risk factor for NOAF in patients undergoing PCI, but we were not able to study pre-procedural CRP levels of the patients.

Conclusion

Prior MI and MHR are predictors of NOAF in elderly patients with ACS undergoing PCI. Given the poor prognosis of NOAF, early identification of such patients by means of the above-mentioned variables may help to prevent poor outcomes. However, our results should be further confirmed in multicenter, larger studies.

Compliance with ethical standards

Conflict of interest The author(s) declare that they have no competing interests.

Ethical approval The study was approved by the Ethics Committee of Eskisehir Osmangazi University, Faculty of Medicine. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent All patients were informed about the nature of the study and a written informed consent was obtained.

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