

Research Article

Impact of Atrial Fibrillation at the Time of Coronary Revascularization on Long-Term Outcomes: From the G-NUH Registry

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Background: Coronary artery disease (CAD) and atrial fibrillation (AF) frequently co-occur. They influence each other in terms of occurrence and aggravation, which has an impact on the prognosis. Meanwhile, there is a lack of reports on whether AF rhythm at the time of coronary revascularizations is related to long-term prognosis. We aimed to determine whether AF upon electrocardiography at the time of revascularization for CAD affects the patient's long-term outcome.

Methods: We observed the clinical outcomes (up to 10 years) in 7733 patients who underwent coronary intervention at two centers. Patients were divided into AF and non-AF groups on the day of the procedure, and analyses were performed after validation through propensity score matching. The primary outcome was major adverse cardiac events (MACEs) defined as a composite of all-cause mortality, spontaneous myocardial infarction, stroke, and hospitalization. Subgroup analysis was performed for different causes of hospitalization.

Results: During follow-up (mean: 3.8 years), the incidence and risk of MACEs did not differ between the AF and non-AF groups (23.3% vs. 36.2%, adjusted hazard ratio [HR]: 1.21, 95% confidence interval [CI]: 0.74–1.98, $p = 0.443$) following coronary intervention, after propensity score matching. However, the incidence of hospitalization events was higher in the AF group than that in the non-AF group (2.2% vs. 8.6%, adjusted HR: 3.28, 95% CI: 1.02–10.53, $p = 0.046$). Among the causes of hospitalization, the incidence of major bleeding was significantly higher in the AF group.

Conclusions: The effect of AF during coronary revascularization on the incidence of the MACEs over a mean follow-up of 3.8 years was not statistically significant. However, AF was associated with a higher incidence of hospitalization during the follow-up period.

Trial Registration: ClinicalTrials.gov identifier: NCT04650529

Keywords: atrial fibrillation; coronary artery disease; coronary revascularization

1. Introduction

Coronary artery disease (CAD) and arrhythmias frequently co-occur, and atrial fibrillation (AF) is the most common supraventricular arrhythmia in patients with acute coronary syndrome (ACS) [1]. The reported incidence of AF among patients with ACS, including those with unstable angina and acute myocardial infarction (AMI), is 2%–23% [2, 3]. In particular, the incidence of new-onset AF in patients with myocardial infarction is 11.8% [4]. AF itself may increase the risk of ACS, such as ST-elevation myocardial infarction (STEMI) or non-STEMI [5]. About 10%–15% of all patients with AF undergo coronary revascularization for CAD [6]. Additionally, patients with ACS with documented AF have worse short-term and long-term prognoses than patients with a maintained sinus rhythm [7, 8].

When AF is detected upon ECG during a cardiovascular procedure or during the peri-procedural period, it affects treatment strategy, especially the antithrombotic and antiarrhythmic treatment. Antithrombotic medications are divided into antiplatelet agents, including aspirin and P2Y₁₂ inhibitors, and anticoagulants, including warfarin and direct oral anticoagulants (OACs). For patients who have AF after undergoing coronary revascularization for CAD, short-term triple antithrombotic therapy comprising aspirin, a P2Y₁₂ inhibitor, and an OAC is considered. Thereafter, a decision is made as to whether to omit aspirin and continue with dual medication of the P2Y₁₂ inhibitor and OAC, and in the longer term, whether to administer the OAC alone [9, 10].

As aggressive antithrombotic treatment strategies raise concerns about the risk of bleeding, clinicians may adopt a passive approach when AF occurs temporarily during a coronary revascularization procedure [11]. In addition, in such patients, active rhythm-control strategies are not easy because antiarrhythmic drug options are limited to amiodarone or dronedarone [9, 10]. However, even transient, self-terminating AF during STEMI reportedly increases the risk of stroke in the long term [7, 12]. In the midst of this, there is a lack of reports on whether AF rhythm at the time of coronary revascularizations is related to long-term prognosis. The purpose of this study was to determine the long-term prognosis of patients with AF detected upon ECG during coronary revascularization and to provide information to aid the establishment of treatment plans for patients with CAD who experience AF during coronary revascularization.

2. Methods

2.1. Study Population. This was a prospective, observational, multicenter study of the data of consecutive patients who underwent coronary revascularization, obtained from the Gyeongsang National University Hospital (G-NUH). This study was performed in conformance with the ethical principles of the Declaration of Helsinki of the World Medical Association, and the requirement for written informed consent was waived by our institutional review board owing to the deidentified nature of data obtained from the institutional registry. The institutional review board

approved the protocol of this study (approval number: 2025-09-014).

This study included the 7732 patients in the G-NUH registry who underwent coronary revascularization from January 2010 to November 2018 and who were followed up until December 2021. All patients were divided into two groups according to the presence/absence of AF upon 12-lead ECG during coronary revascularization, and the final study population was determined using propensity score matching according to patients' clinical characteristics. The patient flow is visualized in Figure 1.

2.2. CAD Management and AF. The criteria and methods for coronary revascularization in patients with CAD followed current guidelines and were applied identically at both institutions where the G-NUH study was conducted [13, 14]. The details of the procedure were previously described [15, 16]. AF during the coronary revascularization period was defined as an AF rhythm documented on the 12-lead ECG on the day of the procedure. The characteristics of the participants included demographics such as age and sex, and medical history including hypertension, diabetes, and cardiovascular disease was investigated. Baseline demographic, angiographic, and procedural characteristics and clinical outcome data were collected prospectively. The clinical characteristics and medical history of each patient were obtained through patient interviews or from their medical records.

2.3. Study Outcomes and Follow-Up. The primary endpoint was major adverse cardiac events (MACEs), defined as a composite of all-cause mortality, spontaneous myocardial infarction, stroke, and hospitalization for up to 10 years after coronary revascularization. Spontaneous myocardial infarction was defined as an ischemic event in a coronary artery other than the revascularized vessel at the time of study enrollment that was confirmed via typical chest symptoms, ECG changes, new regional wall motion abnormalities upon echocardiography, and elevated cardiac markers upon laboratory testing. Cardiovascular events that occurred in the same region of the left ventricular wall as the coronary artery that was revascularized were excluded from the MACEs. A stroke was defined as an event confirmed by a neurologist via a procedure such as computed tomography or magnetic resonance imaging. Hospitalization for any reason was documented. Bleeding events were defined as events that necessitated blood transfusion. Patients visited the cardiology outpatient clinic once a year and underwent physical examination and various tests, including ECG, chest X-ray radiography, and echocardiography.

2.4. Statistical Analysis. Descriptive statistics were used to organize and interpret the participants' baseline characteristics and comorbidities. Categorical variables are reported as frequencies (percentages). Continuous variables are reported as means $\pm$ standard deviations or medians (Quartile 1 to Quartile 3). Categorical variables were compared using

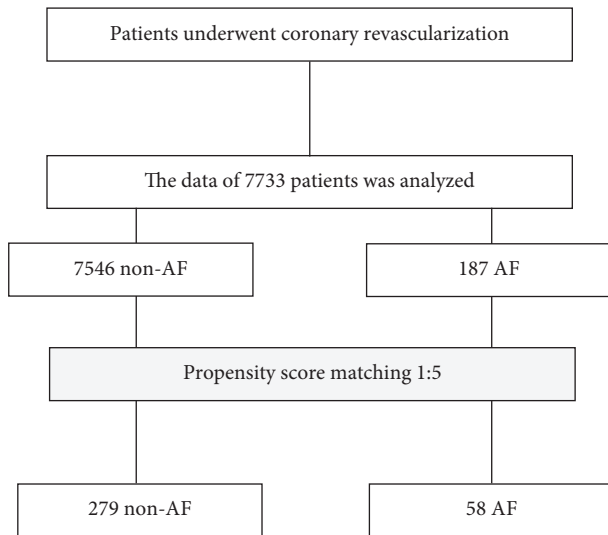


FIGURE 1: Flowchart of the patient flow in the analysis. AF, atrial fibrillation.

Fisher's exact test or Pearson's chi-square test, whereas continuous variables were compared using Student's *t*-test and the Wilcoxon rank-sum test, as appropriate.

Propensity score matching of the two groups was used to reduce the confounding bias. The propensity score, which was the probability of having an AF rhythm, was estimated using logistic regression based on sociodemographics, comorbidities, coronary disease status, coronary revascularization procedural characteristics based on which the initial prescription was provided, clinical risk scores, medical history, and concurrent medication use (variables in Table 1). The balance between the treatment populations was evaluated via standardized differences of all baseline covariates, with a threshold of 0.1 indicating an imbalance. To evaluate the effect of AF on the outcomes of coronary revascularization, a Cox proportional hazards model was fitted to the data.

All tests were two-tailed, with values of $p < 0.05$ considered significant. The statistical analyses were performed using R software Version 4.0.3 (The R Foundation for Statistical Computing, Vienna, Austria).

3. Results

3.1. Baseline Characteristics. A total of 7733 patients (age: 65.5 ± 11.9 years, 31.0% female) who underwent coronary revascularization were included in the analysis. Compared with the AF group, the non-AF group had a larger proportion of smokers (30.0% vs. 18.2%, $p = 0.001$) and a higher rate of STEMI (24.0% vs. 15.0%, $p = 0.005$). Other socio-demographic characteristics, including age and sex, and comorbidities were similar between the two groups. After propensity score matching (total of 337 patients, age: 65.5 ± 11.9 years, 30.6% female), the baseline characteristics of the incident AF and non-AF groups were well balanced for covariates that had had p values less than 0.05 before matching. Comparisons of the baseline characteristics

among groups classified by the presence/absence of AF are summarized in Table 1.

3.2. Clinical Outcomes of Patients With AF During Coronary Intervention. During a mean follow-up of 3.81 ± 3.13 years, after adjustment for age, sex, and clinical variables, the incidence of MACEs among patients who had undergone coronary revascularization was higher in the AF group than that in the non-AF group, although this difference was not statistically significant. These results were maintained in the propensity score-matched analysis (23.3% vs. 36.2%, hazard ratio [HR]: 1.21, 95% confidence interval [CI]: 0.74–1.98, $p = 0.443$). Upon survival analysis, the incidence of MACEs during follow-up did not differ between the two groups (log-rank $p = 0.47$; Figure 2(a)). Upon analysis of each MACE component, all-cause death, spontaneous myocardial infarction, and stroke were not associated with the incidence of AF at the time of coronary intervention (Table 2). However, the incidence of hospitalization events was higher in the AF group than that in the non-AF group, with an adjusted HR of 3.28 (95% CI: 1.02–10.53, $p = 0.046$; Table 2). The AF group had a higher rate of all-cause hospitalization-free survival than the non-AF group (log-rank $p = 0.04$; Figure 2(b)). Details of clinical outcomes are presented in Table 2.

3.3. Causes of Hospitalization in the Study Sample. The five most common causes of hospitalization during the follow-up period were CAD-related events, occurrence or worsening of heart failure, AF (including a rapid or slow ventricular response), stroke, and major bleeding. The incidences of CAD, AF, and stroke events were higher in the AF group than those in the non-AF group, but these differences were not statistically significant. Major bleeding events (major organ bleeding or bleeding sufficient to require blood transfusion) were significantly more common in the AF group than those in the non-AF group (HR: 3.28, 95% CI: 1.01–10.73, $p = 0.049$). These results are visualized in Figure 3. After propensity score matching, triple therapy (12.1% vs. 0.7%, $p = 0.001$) and anticoagulants alone (2.2% vs. 0.0%, $p = 0.002$) were used more commonly as an antithrombotic strategy in the AF group.

4. Discussion

In this study, we evaluated whether the long-term prognosis differed between patients who experienced AF during coronary intervention and those who did not. During a mean observation period of 3.8 years, the incidence of MACEs did not differ between the groups, but all-cause hospitalization was higher in the AF group. The most common cause of hospitalization was CAD, the incidence of which did not significantly differ between the AF and non-AF groups. Among the causes of hospitalization, the incidence of major bleeding was significantly higher in the AF group, contributing to the overall difference in hospitalization rates. A possible explanation for the higher rate of bleeding in the AF group than that in the non-AF group is the more frequent use of anticoagulants. However, owing to a lack of data on

TABLE 1: Baseline characteristics.

	Before PSM				After PSM			
	Non-AF (<i>n</i> = 7546)	AF (<i>n</i> = 187)	<i>p</i>	SMD	Non-AF (<i>n</i> = 279)	AF (<i>n</i> = 58)	<i>p</i>	SMD
<i>Sociodemographics</i>								
Age, years	66.0 (57.0–75.0)	66.0 (58.0–74.5)	0.671	0.019	66.0 (56.0–74.0)	66.0 (58.3–73.5)	0.764	0.044
Female sex	2339 (31.0)	60 (32.1)	0.812	0.023	88 (31.5)	15 (25.9)	0.485	0.126
BMI, kg/m ²	24.0 (22.0–26.1)	24.3 (22.1–26.1)	0.773	0.010	24.3 (22.2–26.1)	24.3 (22.1–26.1)	0.794	0.074
Smoking	2264 (30.0)	34 (18.2)	0.001	0.279	96 (34.4)	12 (20.7)	0.060	0.311
<i>Comorbidities</i>								
Hypertension	3928 (52.1)	98 (52.4)	0.983	0.007	149 (53.4)	33 (56.9)	0.733	0.070
Diabetes mellitus	2322 (30.8)	57 (30.5)	0.996	0.006	91 (32.6)	18 (31.0)	0.936	0.034
Dyslipidemia	3881 (51.4)	85 (45.5)	0.123	0.120	156 (55.9)	32 (55.2)	1.000	0.015
Chronic kidney disease	1434 (19.0)	38 (20.3)	0.720	0.033	36 (12.9)	8 (13.8)	1.000	0.026
Anemia	952 (12.6)	26 (13.9)	0.680	0.038	35 (12.5)	7 (12.1)	1.000	0.014
<i>Past history</i>								
Previous heart failure	1631 (21.6)	33 (17.6)	0.225	0.100	54 (19.4)	11 (19.0)	1.000	0.010
Previous MI	444 (5.9)	9 (4.8)	0.647	0.048	34 (12.2)	6 (10.3)	0.864	0.058
Previous PCI	1145 (15.2)	31 (16.6)	0.671	0.038	71 (25.4)	13 (22.4)	0.750	0.071
Previous CABG	74 (1.0)	3 (1.6)	0.634	0.055	0 (0.0)	0 (0.0)	1.000	<0.001
Previous stroke	550 (7.3)	18 (9.6)	0.285	0.084	26 (9.3)	6 (10.3)	1.000	0.034
CHA ₂ DS ₂ -VASC score	3.0 (2.0–4.0)	3.0 (2.0–4.0)	0.683	0.011	3.0 (2.0–5.0)	3.0 (2.0–4.0)	0.801	0.008
mHAS-BLED score*	1.0 (1.0–2.0)	1.0 (1.0–2.0)	0.487	0.040	1.0 (0.0–2.0)	1.0 (1.0–2.0)	0.386	0.083
<i>Disease characteristics</i>								
STEMI	1814 (24.0)	28 (15.0)	0.005	0.230	78 (28.0)	9 (15.5)	0.071	0.305
NSTEMI	1866 (24.7)	52 (27.8)	0.380	0.070	80 (28.7)	17 (29.3)	1.000	0.014
Unstable angina	825 (10.9)	22 (11.8)	0.809	0.026	27 (9.7)	4 (6.9)	0.677	0.101
<i>Procedural characteristics</i>								
Left main PCI	161 (2.1)	5 (2.7)	0.804	0.035	7 (2.5)	3 (5.2)	0.508	0.139
Multivessel PCI	581 (7.7)	11 (5.9)	0.433	0.072	18 (6.5)	4 (6.9)	1.000	0.018
<i>Concurrent drugs</i>								
Aspirin	7060 (93.6)	154 (82.4)	<0.001	0.350	276 (98.9)	56 (96.6)	0.445	0.160
P2Y ₁₂ inhibitor	6845 (90.7)	148 (79.1)	<0.001	0.328	272 (97.5)	55 (94.8)	0.508	0.139
Oral anticoagulant	140 (1.9)	25 (13.4)	<0.001	0.445	7 (2.5)	3 (5.2)	0.508	0.139
Warfarin	104 (1.4)	7 (3.7)	0.018	0.150	6 (2.2)	2 (3.4)	0.907	0.079
NOAC	36 (0.5)	18 (9.6)	<0.001	0.427	1 (0.4)	1 (1.7)	0.770	0.135
ACEi/ARB	4928 (65.3)	117 (62.6)	0.484	0.057	191 (68.5)	39 (67.2)	0.979	0.026
Beta blocker	4389 (58.2)	92 (49.2)	0.017	0.181	170 (60.9)	36 (62.1)	0.989	0.023
Calcium channel blocker	685 (9.1)	23 (12.3)	0.167	0.104	17 (6.1)	4 (6.9)	1.000	0.033
Statin	6719 (89.0)	151 (80.7)	0.001	0.233	270 (96.8)	56 (96.6)	1.000	0.012
PPI	3689 (48.9)	76 (40.6)	0.031	0.166	171 (61.3)	28 (48.3)	0.092	0.264

Note: Values are presented as median (interquartile range) or *n* (%).

Abbreviations: ACEi, angiotensin-converting enzyme inhibitor; AF, atrial fibrillation; ARB, angiotensin II receptor blocker; BMI, body mass index; CABG, coronary artery bypass graft; MI, myocardial infarction; NOAC, non-vitamin K antagonist oral anticoagulant; NSTEMI, non-ST-elevation myocardial infarction; PCI, percutaneous coronary intervention; PPI, proton pump inhibitor; PSM, propensity score matching; SMD, standard mean deviation; STEMI, ST-elevation myocardial infarction.

*Modified HAS-BLED = hypertension, 1 point; > 65 years old, 1 point; stroke history, 1 point; bleeding history or predisposition, 1 point; labile international normalized ratio, not assessed; ethanol or drug abuse, 1 point; drug predisposing to bleeding, 1 point.

changes in antithrombotic agents during the follow-up period, no definitive conclusions could be made.

4.1. Shared Risk Factors of CAD and AF. CAD and AF increase with advancing age and frequently co-occur [1]. Etiology-wise, CAD and AF share medical risk factors, such as hypertension, diabetes, and dyslipidemia, as well as obesity and demographic risk factors. In patients with hypertension, elevated blood pressure is a physical force that leads to the development of atherosclerotic plaque, which increases arterial stiffness and decreases coronary perfusion [17]. Hypertension increases the afterload of the left

ventricle; this afterload may lead to diastolic dysfunction, which is potentially associated with atrial enlargement. Such structural changes and alterations in the renin-angiotensin-aldosterone system may induce AF [18]. In terms of diabetes, hyperglycemia can damage the heart and induce diabetic heart disease, including CAD [19]. Diabetes directly promotes AF through changes in cardiac cell ion channels and autonomic dysfunction [20]. Additionally, poor glycemic control may indirectly affect the development of AF through related metabolic diseases, such as hypertension and obesity [21]. Through these actions, diabetes acts as a risk factor for both CAD and AF, further aggravating both diseases.

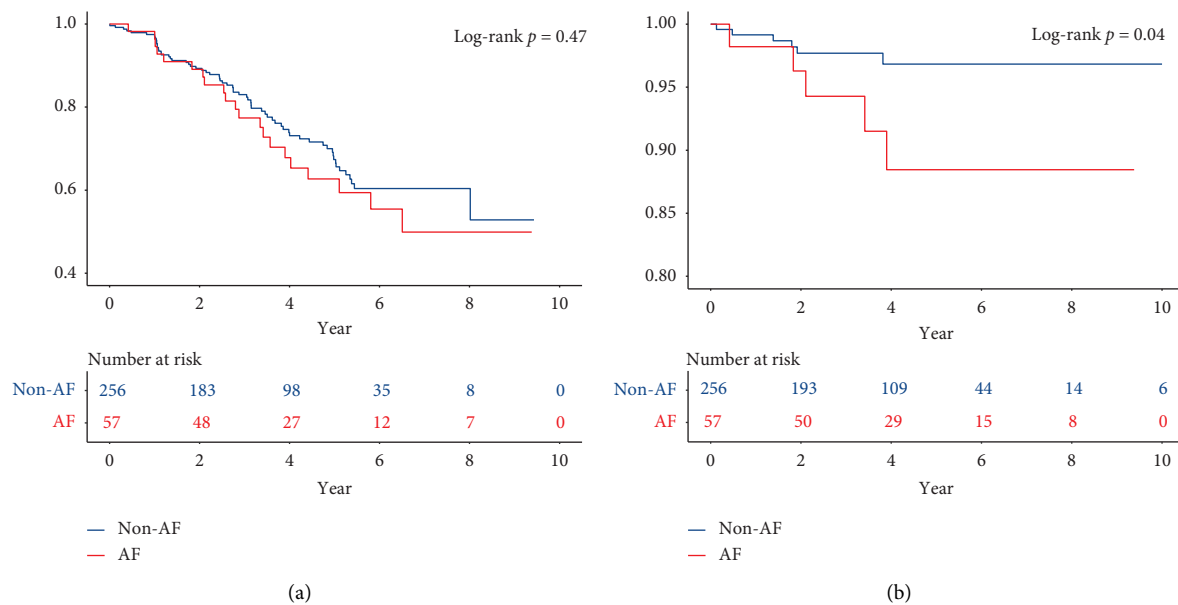


FIGURE 2: Time-dependent clinical outcomes following coronary revascularization according to incident AF. Kaplan–Meier curves for the major adverse cardiac events (a) and hospitalization events (b) for the AF and non-AF groups. The major adverse cardiac events were defined as composite of all-cause mortality, myocardial infarction, stroke, and hospitalization. AF, atrial fibrillation.

TABLE 2: Cox regression for clinical outcomes in non-AF and AF.

	Patients	Events (%)	Adjusted HR (95% CI)	Patients, post-PSM	Events (%), post-PSM	Adjusted HR (95% CI), post-PSM
<i>MACE = total death + MI + stroke + hospitalization</i>						
Non-AF	7546	1631 (21.61%)	Reference	279	65 (23.30%)	Reference
AF	187	51 (27.27%)	1.20 (0.92–1.58)	58	21 (36.21%)	1.21 (0.74–1.98)
<i>All-cause mortality</i>						
Non-AF	7546	733 (9.71%)	Reference	279	15 (5.38%)	Reference
AF	187	15 (8.02%)	0.79 (0.48–1.30)	58	4 (6.90%)	1.05 (0.38–2.92)
<i>MI</i>						
Non-AF	7546	1173 (15.54%)	Reference	279	59 (21.15%)	Reference
AF	187	28 (14.97%)	0.92 (0.63–1.34)	58	13 (22.41%)	0.80 (0.44–1.46)
<i>Stroke</i>						
Non-AF	7546	1506 (19.96%)	Reference	279	70 (25.09%)	Reference
AF	187	40 (21.39%)	1.02 (0.75–1.40)	58	18 (31.03%)	0.94 (0.56–1.58)
<i>Hospitalization</i>						
Non-AF	7546	313 (4.15%)	Reference	279	6 (2.15%)	Reference
AF	187	17 (9.09%)	2.09 (1.29–3.38)	58	5 (8.62%)	3.28 (1.02–10.53)

Abbreviations: AF, atrial fibrillation; CI, confidence interval; HR, hazard ratio; MI, myocardial infarction; PSM, propensity score matching.

4.2. Direct Interaction of CAD and AF. In addition to overlapping risk factors, CAD and AF directly influence each other [22, 23]. Thrombosis is the decisive cause of ACS, initiated by activated platelet aggregation and a coagulation cascade [15, 16]. Regarding the pathophysiology of CAD development in patients with AF, persistent platelet activity reportedly contributes to the development of ACS [24]. CAD, especially ACS requiring coronary revascularization, may result in reentry, focal ectopic activity, and neural remodeling, together comprising AF. From an electrophysiological perspective, reentry is due to nonhomogeneity and different refractory periods among cardiomyocytes. Animal studies have shown that the effective refractory

periods of the atria are shortened in cardiomyocytes after AMI, which explains the mechanism of reentry that occurs in CAD [25]. Human studies have also shown that the cardiac conduction time is longer in patients with AMI and that this contributes to the development of AF [26]. In addition, intracellular calcium overload is an important pathophysiological phenomenon in cardiomyocytes under ischemic conditions, which enhances cellular automaticity, delayed afterdepolarizations, and early afterdepolarizations [27, 28]. CAD can directly induce atrial infarction and may influence AF development through the death of atrial myocytes, scar formation, and atrial fibrosis remodeling [29]. From a chemical perspective, inflammatory factors can

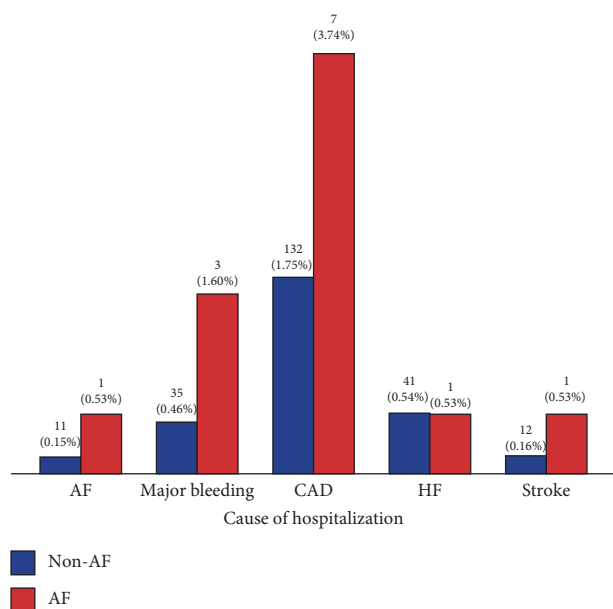


FIGURE 3: Causes of hospitalization during the follow-up period after coronary revascularization. AF, atrial fibrillation; CAD, coronary artery disease; HF, heart failure.

stimulate fibrosis, similar to the role of inflammation in comorbidities. In particular, molecules involved in the AMI process, such as Toll-like receptors, are more abundant in patients with CAD who experience AF than those in patients who do not [30]. Further study is needed to elucidate the exact mechanism by which such molecules are related to AF.

4.3. Study Limitations. This study has several limitations. First, it was an observational, nonrandomized study. However, the study had a long-term follow-up period and concerned patients in two centers. Second, in this registry, patients' history of AF was not identified. In addition, as patients were not followed up specifically for AF, we could not distinguish between paroxysmal and persistent AF. Third, the number of participants included in the AF group was small because patients with paroxysmal AF who were in sinus rhythm at the time of the procedure were excluded. This is a major limitation of the study, as it might have caused the lack of difference in the primary outcome, MACEs, between the AF and non-AF groups. Further studies in which the characteristics of AF are more clearly in focus are needed based on the results of this study. However, our study makes a meaningful contribution in terms of the long-term effects of AF at the time of coronary intervention in patients with CAD.

5. Conclusion

No clear effects of AF occurring during coronary revascularization on MACEs over a mean follow-up of 3.8 years were observed. However, patients with AF at the time of revascularization exhibited higher rates of hospitalization, largely driven by bleeding events during the follow-up period.

Data Availability Statement

The data underlying this article will be shared with the corresponding author upon ethical request for legitimate scientific purposes.

Conflicts of Interest

The authors declare no conflicts of interest.

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The authors have nothing to report.

References

- [1] J. Schmitt, G. Duray, B. J. Gersh, and S. H. Hohnloser, "Atrial Fibrillation in Acute Myocardial Infarction: A Systematic Review of the Incidence, Clinical Features and Prognostic Implications," *European Heart Journal* 30, no. 9 (2009): 1038–1045, <https://doi.org/10.1093/eurheartj/ehn579>.
- [2] H. Gonzalez-Pacheco, M. F. Marquez, A. Arias-Mendoza, et al., "Clinical Features and In-Hospital Mortality Associated With Different Types of Atrial Fibrillation in Patients With Acute Coronary Syndrome With and Without ST Elevation," *Journal of Cardiology* 66, no. 2 (2015): 148–154, <https://doi.org/10.1016/j.jjcc.2014.11.001>.
- [3] J. J. Noubiap, T. A. Agbaedeng, U. F. Nyaga, et al., "Atrial Fibrillation Incidence, Prevalence, Predictors, and Adverse Outcomes in Acute Coronary Syndromes: A Pooled Analysis of Data From 8 Million Patients," *Journal of Cardiovascular Electrophysiology* 33, no. 3 (2022): 414–422, <https://doi.org/10.1111/jce.15351>.
- [4] B. P. Krijthe, M. J. Leening, J. Heeringa, et al., "Unrecognized Myocardial Infarction and Risk of Atrial Fibrillation: The Rotterdam Study," *International Journal of Cardiology* 168, no. 2 (2013): 1453–1457, <https://doi.org/10.1016/j.ijcard.2012.12.057>.
- [5] E. Z. Soliman, M. M. Safford, P. Muntner, et al., "Atrial Fibrillation and the Risk of Myocardial Infarction," *JAMA Internal Medicine* 174, no. 1 (2014): 107–114, <https://doi.org/10.1001/jamainternmed.2013.11912>.
- [6] S. Krale, K. Schneider, S. Lang, T. Suselbeck, and M. Borggrefe, "Incidence and Severity of Coronary Artery Disease in Patients With Atrial Fibrillation Undergoing First-Time Coronary Angiography," *PLoS One* 6, no. 9 (2011): e24964, <https://doi.org/10.1371/journal.pone.0024964>.
- [7] G. Batra, B. Svennblad, C. Held, et al., "All Types of Atrial Fibrillation in the Setting of Myocardial Infarction Are Associated With Impaired Outcome," *Heart* 102, no. 12 (2016): 926–933, <https://doi.org/10.1136/heartjnl-2015-308678>.
- [8] P. Jabre, X. Jouven, F. Adnet, et al., "Atrial Fibrillation and Death After Myocardial Infarction: A Community Study," *Circulation* 123, no. 19 (2011): 2094–2100, <https://doi.org/10.1161/circulationaha.110.990192>.
- [9] J. A. Joglar, M. K. Chung, A. L. Armbruster, et al., "2023 ACC/AHA/ACCP/HRS Guideline for the Diagnosis and Management of Atrial Fibrillation: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines," *Circulation* 149,

- no. 1 (2024): e1–e156, <https://doi.org/10.1161/cir.0000000000001193>.
- [10] I. C. Van Gelder, M. Rienstra, K. V. Bunting, et al., “2024 ESC Guidelines for the Management of Atrial Fibrillation Developed in Collaboration With the European Association for Cardio-Thoracic Surgery (EACTS),” *European Heart Journal* 45, no. 36 (2024): 3314–3414, <https://doi.org/10.1093/eurheartj/ehae176>.
 - [11] A. Erez, I. Goldenberg, A. Sabbag, et al., “Temporal Trends and Outcomes Associated With Atrial Fibrillation Observed During Acute Coronary Syndrome: Real-World Data From the Acute Coronary Syndrome Israeli Survey (ACSIS), 2000–2013,” *Clinical Cardiology* 40, no. 5 (2017): 275–280, <https://doi.org/10.1002/clc.22654>.
 - [12] C. W. Siu, M. H. Jim, H. H. Ho, et al., “Transient Atrial Fibrillation Complicating Acute Inferior Myocardial Infarction: Implications for Future Risk of Ischemic Stroke,” *Chest* 132, no. 1 (2007): 44–49, <https://doi.org/10.1378/chest.06-2733>.
 - [13] G. N. Levine, E. R. Bates, J. C. Blankenship, et al., “2015 ACC/AHA/SCAI Focused Update on Primary Percutaneous Coronary Intervention for Patients With ST-Elevation Myocardial Infarction: An Update of the 2011 ACCF/AHA/SCAI Guideline for Percutaneous Coronary Intervention and the 2013 ACCF/AHA Guideline for the Management of ST-Elevation Myocardial Infarction,” *Journal of the American College of Cardiology* 67, no. 10 (2016): 1235–1250, <https://doi.org/10.1016/j.jacc.2015.10.005>.
 - [14] F. J. Neumann, M. Sousa-Uva, A. Ahlsson, et al., “2018 ESC/EACTS Guidelines on Myocardial Revascularization,” *European Heart Journal* 40, no. 2 (2019): 87–165, <https://doi.org/10.1093/eurheartj/ehy394>.
 - [15] J. H. Ahn, U. S. Tantry, M. G. Kang, et al., “Residual Inflammatory Risk and Its Association With Events in East Asian Patients After Coronary Intervention,” *JACC Asia* 2, no. 3 (2022): 323–337, <https://doi.org/10.1016/j.jacasi.2021.11.014>.
 - [16] S. H. Lee, H. K. Kim, J. H. Ahn, et al., “Prognostic Impact of Hypercoagulability and Impaired Fibrinolysis in Acute Myocardial Infarction,” *European Heart Journal* 44, no. 19 (2023): 1718–1728, <https://doi.org/10.1093/eurheartj/ehad088>.
 - [17] E. Rapsomaniki, A. Timmis, J. George, et al., “Blood Pressure and Incidence of Twelve Cardiovascular Diseases: Lifetime Risks, Healthy life-years Lost, and Age-specific Associations in 1.25 Million People,” *The Lancet* 383, no. 9932 (2014): 1899–1911, [https://doi.org/10.1016/s0140-6736\(14\)60685-1](https://doi.org/10.1016/s0140-6736(14)60685-1).
 - [18] T. J. Guzik and R. M. Touyz, “Oxidative Stress, Inflammation, and Vascular Aging in Hypertension,” *Hypertension* 70, no. 4 (2017): 660–667, <https://doi.org/10.1161/hypertensionaha.117.07802>.
 - [19] V. L. Murthy, M. Naya, C. R. Foster, et al., “Association Between Coronary Vascular Dysfunction and Cardiac Mortality in Patients With and Without Diabetes Mellitus,” *Circulation* 126, no. 15 (2012): 1858–1868, <https://doi.org/10.1161/circulationaha.112.120402>.
 - [20] A. Wang, J. B. Green, J. L. Halperin, and J. P. Piccini, “Atrial Fibrillation and Diabetes Mellitus: JACC Review Topic of the Week,” *Journal of the American College of Cardiology* 74, no. 8 (2019): 1107–1115, <https://doi.org/10.1016/j.jacc.2019.07.020>.
 - [21] R. R. Huxley, A. Alonso, F. L. Lopez, et al., “Type 2 Diabetes, Glucose Homeostasis and Incident Atrial Fibrillation: The Atherosclerosis Risk in Communities Study,” *Heart* 98 (2012): 133–138, <https://doi.org/10.1136/heartjnl-2011-300503>.
 - [22] Y. Obayashi, H. Shiomi, T. Morimoto, et al., “Newly Diagnosed Atrial Fibrillation in Acute Myocardial Infarction,” *Journal of the American Heart Association* 10, no. 18 (2021): e021417, <https://doi.org/10.1161/jaha.121.021417>.
 - [23] G. Bazoukis, J. M. H. Hui, A. Saplaouras, et al., “The Impact of New-Onset Atrial Fibrillation in the Setting of Acute Coronary Syndrome,” *Journal of Cardiology* 85, no. 3 (2025): 186–203, <https://doi.org/10.1016/j.jjcc.2024.12.007>.
 - [24] H. K. Yip, L. T. Chang, C. K. Sun, et al., “Platelet Activation in Patients With Chronic Nonvalvular Atrial Fibrillation,” *International Heart Journal* 47, no. 3 (2006): 371–379, <https://doi.org/10.1536/ihj.47.371>.
 - [25] T. Zukela, Q. Zhou, H. Wang, et al., “Relationship Between New-Onset Atrial Fibrillation and Sympathetic Neural Remodeling in a Canine Acute Myocardial Infarction Model,” *Zhonghua Xinxueguanbing Zazhi* 43, no. 11 (2015): 975–981.
 - [26] M. L. Antoni, M. Bertini, J. Z. Atary, et al., “Predictive Value of Total Atrial Conduction Time Estimated With Tissue Doppler Imaging for the Development of New-Onset Atrial Fibrillation After Acute Myocardial Infarction,” *The American Journal of Cardiology* 106, no. 2 (2010): 198–203, <https://doi.org/10.1016/j.amjcard.2010.02.030>.
 - [27] W. T. Clusin, M. Buchbinder, and D. C. Harrison, “Calcium Overload, Injury Current, and Early Ischaemic Cardiac Arrhythmias: A Direct Connection,” *Lancet* 321, no. 8319 (1983): 272–274, [https://doi.org/10.1016/s0140-6736\(83\)91688-4](https://doi.org/10.1016/s0140-6736(83)91688-4).
 - [28] A. Arutunyan, L. M. Swift, and N. Sarvazyan, “Initiation and Propagation of Ectopic Waves: Insights From an in Vitro Model of Ischemia-Reperfusion Injury,” *American Journal of Physiology-Heart and Circulatory Physiology* 283, no. 2 (2002): H741–H749, <https://doi.org/10.1152/ajpheart.00096.2002>.
 - [29] S. van Diepen, H. Siha, Y. L. Fu, et al., “Do Baseline Atrial Electrocardiographic and Infarction Patterns Predict New-Onset Atrial Fibrillation After ST-Elevation Myocardial Infarction? Insights From the Assessment of Pexelizumab in Acute Myocardial Infarction Trial,” *Journal of Electrocardiology* 43, no. 4 (2010): 351–358, <https://doi.org/10.1016/j.jelectrocard.2010.04.001>.
 - [30] P. Zhang, L. Shao, and J. Ma, “Toll-Like Receptors 2 and 4 Predict New-Onset Atrial Fibrillation in Acute Myocardial Infarction Patients,” *International Heart Journal* 59, no. 1 (2018): 64–70, <https://doi.org/10.1536/ihj.17-084>.