Impact of Atrial Fibrillation During ST-Segment–Elevation Myocardial Infarction on Infarct Characteristics and Prognosis

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Background—Atrial fibrillation (AF) is frequently observed in patients with ST-segment–elevation myocardial infarction and associated with worse clinical outcome. However, the mechanisms for this increased risk are not fully understood. The purpose of this study was to investigate the relationship of the presence of AF to cardiac magnetic resonance (CMR) derived myocardial salvage and damage as well as clinical outcomes.

Methods and Results—This multicenter CMR study enrolled 786 patients with ST-segment–elevation myocardial infarction. CMR parameters (infarct size, myocardial salvage index, microvascular obstruction, and myocardial function) were assessed 3 (interquartile range [IQR], 2–4) days post-ST-segment–elevation myocardial infarction and compared between patients with or without AF during hospitalization. Major adverse cardiac events were assessed as a composite of all-cause death, reinfarction, and new congestive heart failure at 12 months. AF was documented in 48 (6.1%) patients. There was no significant difference in infarct size (18 [IQR, 9–29]% versus 17 [IQR, 9–25]% of left ventricular mass; P=0.340), myocardial salvage index (51 [IQR, 34–69] versus 51 [IQR, 33–69]; P=0.830), or microvascular obstruction (0.6 [IQR, 0–2.0]% versus 0.0 [IQR, 0–1.8]% of left ventricular mass; P=0.340) between groups. Patients with AF had significantly lower left ventricular (47 [IQR, 34–54]% versus 51 [IQR, 44–58]%; P=0.003) and left atrial (42 [IQR, 17–57]% versus 53 [IQR, 45–59]%; P<0.001) ejection fraction. AF was associated with major adverse cardiac events, even when adjusting for clinical risk factors (odds ratio, 2.48 [95% confidence interval, 1.22–5.03]; P=0.0120) or CMR prognosis markers (odds ratio, 3.77 [95% confidence interval, 1.83–7.79]; P=0.001).

Conclusions—This CMR study found no major differences in myocardial salvage, infarct size, or microvascular damage in patients with ST-segment–elevation myocardial infarction or without AF. AF was, however, associated with cardiac dysfunction and independently related to major adverse cardiac events.

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Key Words: atrial fibrillation ■ heart failure ■ hospitalization ■ myocardial infarction ■ odds ratio ■ risk factors

Atrial fibrillation (AF) is one of the most common arrhythmias in patients with ST-segment–elevation myocardial infarction (STEMI).[1] The presence of AF is well known to have major adverse consequences for patients with STEMI, including increased rates of inhospital complications, hospital readmissions, major adverse cardiac events (MACE), and all-cause death.[1–6] Despite the increasing use of early reperfusion strategies in STEMI during the last decades, the adverse impact of AF on health outcomes remained unchanged.[7]

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The pathophysiology of AF in STEMI is complex and likely multifactorial, and the understanding of the exact determinants remains incomplete.[8–10] Moreover, little information exists about the mechanisms for the higher risk of adverse outcomes associated with the presence of AF. A relative contribution of more extensive myocardial damage in the pathogenesis and prognostic value of AF has been suggested

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because (1) a larger amount of myocardial damage may lead to more severe atrial stretch, inflammation, hemodynamic changes, and neurohumoral activation[1,8,11] and (2) a rapid as well as irregular heart rate may precipitate or exacerbate left ventricular (LV) dysfunction and lead to a further reduction of coronary perfusion and consequently a larger infarct size.[1,10] Indeed, Gal et al[12] showed that high-sensitivity cardiac troponin T levels, an established marker of myocardial necrosis,[13] are associated with AF in the setting of STEMI. However, these results were questioned by other studies.[14] An explanation for these conflicting results is the lack of a precise and comprehensive approach to directly visualize the amount of myocardial injury in previous studies. Cardiovascular magnetic resonance (CMR) is unique in its ability to provide a comprehensive infarct characterization with high accuracy.[15] Moreover, CMR-determined myocardial salvage, infarct size, and microvascular injury are strongly related with poor clinical outcomes in patients with STEMI.[16] In this large, multicenter cohort of contemporary reperfused patients with STEMI, we aimed to describe whether there is a relationship between the presence of AF, infarct characteristics assessed by CMR imaging and adverse cardiovascular events at 12 months after primary percutaneous coronary intervention (PPCI).

Methods

Study Design

This was a post hoc analysis of data from the multicenter CMR substudy of the AIDA STEMI trial (Abciximab Intracoronary versus Intravenous Drug Application in ST-Elevation Myocardial Infarction). AIDA STEMI was a randomized, open-label, multicenter trial, comparing the effects of intracoronary versus standard intravenous bolus application of abciximab in patients with STEMI undergoing PPCI.[17] The design and principal results of AIDA STEMI as well as of the prospective CMR substudy are reported elsewhere.[17–20] Briefly, consecutive patients with STEMI presenting <12 hours after symptom onset were included. The CMR substudy enrolled 795 patients at 8 sites and found no significant difference in CMR markers of myocardial injury between both groups.[19] The primary clinical end point was the rate of MACE, defined as a composite of all-cause mortality, nonfatal reinfarction, and new congestive heart failure within 1 year after STEMI. Detailed end point definitions have been published previously.[17,19] According to the predefined study protocol, standard 12-lead ECGs were recorded in each patient before and 90 minutes after PPCI as well as 24 hours after PPCI (voltage, 10 mm/mV; speed, 25 mm/sec or 50 mm/sec). All ECGs were analyzed by 2 cardiologists blinded to other trial data for the presence or absence of AF. A consensus decision was made in case of conflicting results between both readers. Patients were then categorized into 2 groups according to the presence or absence of AF (Figure). The local ethics committee approved the study and patients were required to provide written informed consent. The data, analytic methods, and study materials will not be made available to other researchers for purposes of reproducing the results or replicating the procedure.

CMR Imaging

Patients underwent CMR on 1.5 T or 3.0 T scanners during hospitalization for STEMI.[21] The sites were selected based on established expertise in acquiring CMRs in patients with myocardial infarction. The CMR protocol and standardized postprocessing have been published in detail previously.[17,19] In brief, cine sequences were applied for the determination of LV function and volumes, T2-weighted imaging for measurement of the area at risk, and late enhancement imaging for assessment of infarct size and microvascular obstruction. Myocardial edema was defined as a mean signal intensity >2 SDs of remote myocardium in T2-weighted images. CMR images were analyzed by a blinded CMR core laboratory (University of Leipzig - Heart Center, Leipzig, Germany). Reproducibility, interobserver as well as intraobserver variability of the CMR core laboratory were reported previously.[22] Area at risk, infarct size, and microvascular obstruction are expressed as the percentage of LV volume (% LV).[23,24] Myocardial salvage was quantified as the difference between the volume of increased T2 signal (area at risk) and the volume of late enhancement (infarct size). Myocardial salvage index was calculated as myocardial salvage divided by area at risk multiplied by 100.[25] In a subgroup of patients (n=662), we further determined left atrial (LA) ejection fraction as described previously.[26] Briefly, minimum and maximum LA volumes were defined as smallest and largest LA volume determined during the cardiac circle, respectively. LA volumes were delineated both in long-axis 2 chamber and 4 chamber view and final calculation of LA volumes was based on biplane measures.

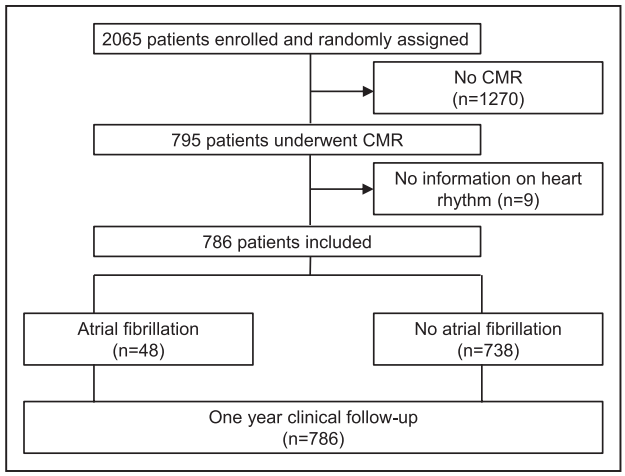

Figure. Flow diagram illustrating the inclusion of patients. CMR indicates cardiovascular magnetic resonance.

Statistical Analysis

Continuous variables are presented as mean±SD or median with interquartile range (IQR) according to their distribution. Categorical variables are shown as frequencies with corresponding percentages. Differences between groups were tested by means of the *t* test and the Wilcoxon–Mann–Whitney U test. Proportions were compared by χ^2-test and Fisher exact test as appropriate. We further built a multiple logistic regression model to identify predictors of MACE. To find relevant variables, we applied the least absolute shrinkage and selection operator procedure starting with all variables from Table 1.[27] We standardized continuous variables and replaced some missing values by multiple imputation as 2 preliminary steps.[28] The least absolute shrinkage and selection operator procedure finds a preoptimal model without using *P* values. Variables with risk near 1 were omitted to get sparse models. These final models (logistic procedure with method enter) yielded the risk estimates inclusive 95% confidence intervals (CI). To ensure statistical robustness of the multiple regression model, we included the thrombolysis in myocardial infarction–risk score instead of the individual risk variables (age, diabetes mellitus, hypertension, heart rate, Killip class, weight, infarct location, and time to treatment) to reduce the number of parameters with respect to our sample size and total number of adverse events. In addition, net reclassification improvement and integrated discrimination improvement of the final multivariable regression model with and without AF was calculated. All tests were 2-tailed and the significance level is 0.05. The multiple imputation, least absolute shrinkage and selection operator procedures, and net reclassification

Table 1. Baseline Patient Characteristics

	Total (n=786)	No Atrial Fibrillation (n=738)	Atrial Fibrillation (n=48)	*P* Value
Age, y	61.5±12.4	61.1±12.2	68.0±13.4	0.001
Male sex, n (%)	595 (75.7)	565 (76.6)	30 (62.5)	0.028
Body mass index, kg/m^2	27.8±4.1	27.8±4.0	27.6±4.9	0.870
Cardiovascular risk factors				
Hypertension, n (%)	533 (68.1)	494 (67.2)	39 (81.3)	0.043
Smoker, n (%)	335 (46.7)	322 (47.6)	13 (31.0)	0.036
Hypercholesterolemia, n (%)	300 (38.6)	279 (38.2)	21 (43.8)	0.450
Diabetes mellitus, n (%)	158 (20.2)	141 (19.2)	17 (36.2)	0.005
Family history for CAD, n (%)	266 (44.0)	251 (43.8)	15 (46.9)	0.730
Systolic blood pressure, mm Hg	132±23	133±23	125±23	0.023
Diastolic blood pressure, mm Hg	79±14	79±14	75±14	0.030
Heart rate, bpm	78±15	77±15	88±19	<0.001
Creatine kinase maximum, μmol/(l·s)	26 (12–46)	26 (12–46)	25 (11–48)	0.690
Pain-to-balloon time, min	180 (109–306)	180 (108–310)	173 (111–253)	0.680
Previous infarction, n (%)	47 (6.0)	44 (6.0)	3 (6.3)	1.000
Previous PCI, n (%)	66 (8.4)	62 (8.4)	4 (8.3)	1.000
Previous CABG, n (%)	11 (1.4)	9 (1.2)	2 (4.2)	0.140
Killip-class on admission, n (%)*				
1	691 (87.9)	654 (88.6)	37 (77.1)	0.014
2	58 (7.4)	50 (6.8)	8 (16.7)	
3	20 (2.5)	20 (2.7)	0 (0.0)	
4	17 (2.2)	14 (1.9)	3 (6.3)	
Concomitant medications, n (%)†				
Aspirin	785 (100)	737 (100)	48 (100)	…
β-blockers	751 (95.7)	703 (95.4)	48 (100)	0.160
ACE-I/ARB	747 (95.2)	701 (95.1)	46 (95.8)	1.000
Statin	746 (95.0)	699 (94.8)	47 (97.9)	0.500
Aldosterone antagonist	91 (11.6)	82 (11.1)	9 (18.8)	0.110

Variables are shown as mean±SD, median (interquartile range) or numbers (frequencies). ACE-I indicates angiotensin-converting enzyme inhibitor; ARB, angiotensin-receptor blocker; CABG, coronary artery bypass grafting; CAD, coronary artery disease; and PCI, percutaneous coronary intervention.

*More than 2 categories: 1 *P* value for all categories.

†Only 1 category: no *P* value can be calculated.

improvement were performed by the R packages mice and penalized. All other statistical analysis was done by means of SPSS Statistics 23.0.0.0 (IBM, Armonk, NY).

Results

AF and Patient Characteristics

Information on heart rhythm was available in 786 (98.9%) patients (Figure 1). The overall presence of AF was 6.1% (n=48). Table 1 shows the baseline characteristics of the overall cohort and according to the presence or absence of AF. Patients with AF were significantly older, more frequently females, had a worse cardiovascular risk profile except for smoking, had on average a lower blood pressure and a higher heart rate. Baseline procedural characteristics of the overall cohort and according to the presence or absence of AF are summarized in Table 2. Median thrombolysis in myocardial infarction–risk score was significantly higher in patients with AF.

AF and CMR Findings

CMR scans were performed 3 (IQR, 2–4) days after STEMI in both groups (estimated median difference 0 days (95% CI, 0–1 days). Table 3 shows the CMR characteristics of all patients and their relation with AF. CMR infarct characteristic variables did not differ significantly between groups. Median LV ejection fraction was significantly lower in patients with AF compared with patients without AF

Table 2. Baseline Procedural Characteristics

	Total (n=786)	No Atrial Fibrillation (n=738)	Atrial Fibrillation (n=48)	*P* Value
Number of diseased vessels, n (%)*				
1	417 (53.1)	392 (53.1)	25 (52.1)	0.210
2	224 (28.5)	214 (29.0)	10 (20.8)	
3	145 (18.4)	132 (17.9)	13 (27.1)	
Infarct related artery, n (%)*				
Left anterior descending	342 (43.5)	323 (43.8)	19 (39.6)	0.850
Right coronary artery	341 (43.4)	319 (43.2)	22 (45.8)	
Left circumflex	96 (12.2)	89 (12.1)	7 (14.6)	
Left main	5 (0.6)	5 (0.7)	0 (0.0)	
Bypass graft	2 (0.3)	2 (0.3)	0 (0.0)	
TIMI-risk score	3 (2–5)	3 (2–5)	5 (3–6)	<0.001
TIMI-flow before PCI, n (%)*				
TIMI-flow 0	442 (56.2)	415 (56.2)	27 (56.3)	0.220
TIMI-flow 1	102 (13.0)	92 (12.5)	10 (20.8)	
TIMI-flow 2	127 (16.2)	123 (16.7)	4 (8.3)	
TIMI-flow 3	115 (14.6)	108 (14.6)	7 (14.6)	
TIMI-flow after PCI, n (%)*				
TIMI-flow 0	12 (1.5)	11 (1.5)	1 (2.1)	0.400
TIMI-flow 1	19 (2.4)	17 (2.3)	2 (4.3)	
TIMI-flow 2	62 (7.9)	56 (7.6)	6 (12.8)	
TIMI-flow 3	692 (88.2)	654 (88.6)	38 (80.9)	
Stent implanted, n (%)	770 (98.0)	723 (98.0)	47 (97.9)	1.000
Thrombectomy, n (%)	189 (24)	169 (22.9)	20 (41.7)	0.003

Variables are shown as median (interquartile range) or numbers (frequencies). PCI indicates percutaneous coronary intervention; and TIMI, thrombolysis in myocardial infarction.

*More than 2 categories: 1 *P* value for all categories.

(47% versus 51%; *P*=0.021). Moreover, patients with AF had lower LA ejection fraction as compared with patients without AF (53 [IQR, 45–59]% versus 42 [IQR, 17–57]%; *P*<0.001).

AF and Clinical Outcome

All patients completed 12 months follow-up. MACE occurred in 53 patients (7%). Of these, 22 patients died (3%). Patients with AF had significantly higher MACE (10 [21%] versus 43 [6%]; odds ratio [OR], 4.3; *P*<0.001), and mortality (6 [13%] versus 16 [2%]; OR, 6.4; *P*<0.001) rates. Moreover, new congestive heart failure occurred more often in patients with AF compared with patients without AF (6 [13%] versus 19 [3%]; OR, 5.4; *P*=0.003). There was no significant difference in the frequency of myocardial reinfarction between groups (3 [6%] versus 18 [2%]; OR, 2.7; *P*=0.110). AF remained significantly associated with MACE after adjustment for baseline clinical risk factors and CMR markers of adverse prognosis (OR, 2.413; 95% CI, 1.011–5.759; *P*=0.047; Table 4). The addition of AF to the other variables of the final model (Table 4) in reclassification analysis led to a continuous net reclassification improvement of 0.26 (95% CI, 0.05–0.48); *P*=0.017 and to an integrated discrimination improvement of 0.02 (95% CI, 0.01–0.04); *P*=0.048.

Discussion

This multicenter study evaluated the impact of AF during hospitalization on CMR markers of myocardial and microvascular injury in patients with STEMI treated by PPCI. The key findings can be summarized as follows (1) age, comorbidities, and cardiac dysfunction assessed by CMR were associated with the presence of AF; (2) however, patients with AF had a similar amount of salvaged myocardium, infarct size, and microvascular injury when compared with patients without AF. (3) AF was associated with 12 months adverse outcomes, even after adjusting for clinical risk factors and CMR prognosis markers. Consequently, our results underscore the strong prognostic relevance of AF in the setting of contemporary revascularized STEMI, which is however not attributed to differences in PPCI efficacy (myocardial salvage), infarct size, or microvascular injury as determined by CMR imaging.

Incidence and Determinants of AF in STEMI

AF is frequently detected in patients treated with PPCI for STEMI, with a prevalence ranging between 5% to 20%.[1] We observed AF in 6% of patients, highlighting how commonly this scenario still occurs in contemporary practice. Compared with patients with sinus rhythm, those with AF were older and more likely to have other comorbidities.[5] Previous data suggested that increases in atrial pressure leading to AF might

Table 3. CMR Imaging Findings

	Total (n=786)	No Atrial Fibrillation (n=738)	Atrial Fibrillation (n=48)	*P* Value
Area at risk, % LV	35 (25–47)	35 (25–47)	40 (26–56)	0.100
Infarct size, % LV	17 (9–25)	17 (9–25)	18 (9–29)	0.340
Microvascular obstruction, % LV	0.0 (0.0–1.8)	0.0 (0.0–1.8)	0.6 (0.0–2.0)	0.340
Myocardial salvage index	51 (33–69)	51 (33–69)	51 (34–69)	0.830
LV ejection fraction, %	51 (43–58)	51 (44–58)	47 (34–54)	0.003
LV end-diastolic volume, mL	146 (121–173)	146 (121–173)	145 (117–168)	0.850
LV end-systolic volume, mL	72.4 (54.4–91.3)	71.9 (54.0–91.2)	77.8 (57.7–97.9)	0.200

Variables are shown as median plus interquartile range. CMR indicates cardiac magnetic resonance; LV, left ventricular/ventricle.

Table 4. Predicting MACE Using Baseline and CMR Variables

	OR (95% CI)	*P* Value
TIMI-risk score	1.324 (1.152–1.523)	<0.001
LV ejection fraction, %	1.002 (0.974–1.031)	0.010
Infarct size, % LV	0.956 (0.924–0.989)	0.871
Atrial fibrillation, yes	2.413 (1.011–5.759)	0.047

CI indicates confidence interval; CMR, cardiac magnetic resonance; LV, left ventricular; MACE, major adverse cardiac events; OR, odds ratio; and TIMI, thrombolysis in myocardial infarction.

arise in the setting of STEMI either induced by atrial ischemia, hemodynamic changes, cardiac dysfunction, neurohumoral abnormalities, or more extensive myocardial damage.[1,8–10] Myocardial damage determined by CMR was also suggested as a major determinant of LA volume, function as well as remodeling after STEMI,[29,30] which are known risk factors for AF. Our data confirm a potential contribution of hemodynamic alterations including pump failure because cardiac dysfunction at CMR was associated with the presence of AF. Until now, however, no study has addressed the potential impact of AF on infarct severity, directly visualized by comprehensive CMR imaging in STEMI. Our study fills this knowledge gap in the literature by demonstrating that AF is not associated with differences in reperfusion success (myocardial salvage), infarct size, or microvascular injury. Therefore, other factors than infarct severity should move into focus of future research in this high-risk subgroup of patients with STEMI.

Prognostic Significance of AF in STEMI

In our analysis, AF during hospitalization for the index event was independently associated with an increased risk of the composite cardiovascular outcome at 12 months as well as the individual outcomes; all-cause mortality and new congestive heart failure. The relationship between AF and clinical outcomes has been analyzed previously.[5,6] Batra et al[5] evaluated 155.071 hospital survivors of myocardial infarction between 2000 and 2009 in Swedish registries. As shown in this analysis, all types of AF in the setting of STEMI (new-onset AF with sinus rhythm at discharge, new-onset AF with AF at discharge, paroxysmal AF, and chronic AF) were associated with worse clinical prognosis with no major differences between AF subtypes.[5] Consequently and in line with our findings, the sole presence of AF before or after reperfusion (irrespective of AF subtype) should be recognized as critical clinical condition with negative impact on patient's outcome. AF should therefore be taken into account for the individual patient's risk stratification. Multiple risk scores, including the thrombolysis in myocardial infarction–risk score, the Primary Angioplasty in Myocardial Infarction risk score, or the Controlled Abciximab and Device Investigation to Lower Late Angioplasty Complications risk score, have been developed to stratify patients with STEMI according to their risk of mortality and morbidity.[31–33] However, AF is not represented in these scores to date. Our results underline that studies considering AF in these risk scores are warranted to further improve their prognostic value, especially because the prevalence of AF is increasing, reflecting the increasing number of older patients and the pervasiveness of comorbidities.[34]

In general, our and previous results of the correlation of AF with adverse prognosis might reflect that AF is a marker for worse clinical condition and structural heart disease. However, the exact mechanisms for the heightened risk of future cardiovascular events after STEMI are not fully understood. First, it has been shown that AF can facilitate the development of life-threatening ventricular arrhythmias because of an enhanced electric instability of the myocardium.[35] This might at least partially explain the increased risk of MACE in STEMI patients with AF. Second, LA function and remodeling, which reflects a parallel ventricular-atrial remodeling is associated with an unfavorable prognosis after STEMI.[26,29,30] An increased heart rate in patients with AF results in an impaired oxygen supply to oxygen demand ratio aggravating these remodeling processes.[8,36] In this study, patients with AF had a significantly worse LA function compared with patients without AF. As such, LA dysfunction in survivors of STEMI with AF might also contribute to the worse outcome. Third, antithrombotic treatment regimens of patients with AF and STEMI undergoing PPCI are complex and associated with an increased bleeding risk.[37] Fourth, AF and heart failure share pathophysiological mechanisms that contribute to the initiation and maintenance of the other.[38] Therefore, AF-induced heart failure is likely to contribute to the worse outcome of these patients. The mechanisms behind heart failure caused by AF have been studied in several studies.[38] AF may precipitate or exacerbate LV systolic dysfunction,[10] which is one of the most powerful markers indicating poor outcome after STEMI.[20] However, the mild difference in LV ejection fraction between patients with or without AF in our study unlikely explains the adverse outcome of patients with AF. AF is associated with tachycardia and shortening of diastolic filling time with irregular ventricular activation that impairs diastolic function and might therefore promote heart failure in STEMI patients with AF. Finally, there is inconsistent evidence for a potential pathophysiological link between AF and more severe myocardial damage with worse outcome as consequence.[12,14] Our multicenter study is the first investigation demonstrating no difference in reperfusion success or irreversible myocardial injury between patients with or without AF, indicating that the amount of myocardial damage is not the mechanism by which AF has an adverse impact on patient's outcome. However, in accordance with previous data, we found a significant association between AF and worse LV and LA systolic function at CMR. To what extent AF is a cause or a consequence of myocardial dysfunction needs further investigation, but it is very likely that the development of either condition has a relevant detrimental impact on the other.[10]

Study Limitations

Several limitations of this study should be considered. The relative small sample size of our study is an important limitation and hence confirmation of our findings in larger studies is necessary. Information on heart rhythm during CMR acquisition is not available for the current study. We can therefore not exclude that AF during cine CMR acquisition might have influenced image quality and therefore measurement of LA and LV function. Because of ECG recording at 3 time points, some patients with AF might have been undetected and therefore misclassified into the sinus rhythm group. Also, we could not distinguish between preexisting and new-onset AF and thus further studies

assessing potential differences in infarct characteristics in these 2 scenarios would be interesting. On the other hand, our data underscore that the simple information on the presence of AF during hospitalization for STEMI has strong prognostic implications in initial STEMI survivors. Various biomarkers, especially NT-proBNP (N-Terminal Pro-B-Type Natriuretic Peptide) and high-sensitivity cardiac troponin provide valuable prognostic information in patients with AF.[39,40] Indeed, recent risk stratification schemes including biomarkers (eg, ABC-stroke score) have shown very promising results for risk prediction.[39,41] In our study, we found no difference in maximum creatine kinase between patients with or without AF; however, information on NT-proBNP and high-sensitivity cardiac troponin was not available precluding further insights into this interesting area of research. The impact and clinical significance of elevated NT-proBNP and high-sensitivity cardiac troponin in STEMI patients with AF needs to be addressed in future studies.

LA strain analysis by CMR, which correlates with the degree of fibrofatty replacement at histological analysis and might reveal myocardial alterations of the LA even when conventional cine analysis is normal,[40,42–44] was not available for the current analysis. Further studies evaluating the association between LA strain, LA ejection fraction, and AF in patients with STEMI are warranted. Finally, our data have no direct impact on the management of STEMI patients with AF. Further clinical studies are needed to explore potential clinical implications of these findings.

Conclusions

This study underlines the relevance of AF as an independent risk factor for future cardiovascular events in the setting of contemporary PPCI for STEMI. However, this heightened risk is not explained by differences in PPCI efficacy (myocardial salvage) or more pronounced myocardial damage as visualized by comprehensive CMR imaging. The results of our study should be taken into consideration when evaluating optimized treatment strategies for these high-risk patients with STEMI and concomitant AF.

Disclosures

None.

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CLINICAL PERSPECTIVE

Atrial fibrillation (AF) is frequently present in patients with acute ST-segment–elevation myocardial infarction and related with worse outcome. The present cardiac magnetic resonance study evaluated the association between the presence of AF and the extent of myocardial damage. Although comorbidities were more common in patients with AF, there was no difference in myocardial salvage, infarct size, and microvascular obstruction when compared with patients without AF. AF was, however, related with an increased risk of major adverse cardiovascular events 1 year after infarction. These results underscore the major prognostic relevance of AF in the setting of ST-segment–elevation myocardial infarction, which is, however, not explained by differences in reperfusion efficacy or irreversible myocardial damage – a result that should be taken into consideration when evaluating optimized management strategies for these high-risk subset of patients with ST-segment–elevation myocardial infarction.

Impact of Atrial Fibrillation During ST-Segment–Elevation Myocardial Infarction on Infarct Characteristics and Prognosis

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