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New-Onset Atrial Fibrillation after Acute Myocardial Infarction and its Relationship to Admission Biomarkers [From the TRIUMPH Registry]

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Abstract

Atrial fibrillation (AF) is an independent predictor of mortality after acute myocardial infarction (AMI). We analyzed the relationship between biomarkers linked to myocardial stretch [NT-pro-brain natriuretic peptide (NT-proBNP)], myocardial damage [Troponin-T (TnT)] and inflammation [high-sensitivity C-reactive protein (hsCRP)] and new-onset AF during AMI to identify patients at high risk for AF. In a prospective multicenter registry of AMI patients from (TRIUMPH), we measured NT-proBNP, TnT, and hsCRP in patients without a history of AF (N=2370). New-onset AF was defined as AF that occurred during the index hospitalization. Hierarchical multivariable logistic regression models were used to determine the association of biomarkers with new-onset AF, after adjusting for other covariates. New-onset AF was documented in 114 AMI patients (4.8%; mean age 58 years; 32% women). For each 2-fold increase in NT-proBNP, there was an 18% increase in the rate of AF (OR 1.18 95% CI 1.03-1.35; $p < 0.02$). Similarly, for every 2-fold increase in hs-CRP, there was a 15% increase in the rate of AF (OR 1.15 95% CI 1.02-1.30; $p = 0.02$). TnT was not independently associated with new-onset AF (OR 0.96 95% CI 0.85-1.07; $p = 0.3$). NT-proBNP and hs-CRP were independently associated with new in-hospital AF after MI, in both men and women, irrespective of race. Our study suggests that markers of myocardial stretch and inflammation, but not the amount of myocardial necrosis, are important determinants of AF in the setting of AMI.

Keywords

Myocardial Infarction; arrhythmia; natriuretic peptide; biomarkers

Since acute myocardial infarction (AMI) is commonly associated with elevated left ventricular filling pressures, myocardial necrosis, and a generalized inflammatory state, [NT-pro-brain natriuretic peptide (NT-proBNP)], Troponin-T (TnT) and [high-sensitivity C-reactive protein (hsCRP)] have proven useful in predicting prognosis and clinical outcome after AMI.^{1,2} It is plausible that NT-BNP, TnT and hs-CRP would represent distinct pathophysiological pathways to develop new onset atrial fibrillation (AF) in patients with AMI.³⁻⁷ We therefore examined the association of NT-pro-BNP, TnT and hs-CRP with new-onset AF in hospitalized AMI patients to potentially identify those patients at high risk of developing new-onset AF in the setting of an AMI.

Methods

All patients were participants in the TRIUMPH (Translational Research Investigating Underlying disparities in recovery from acute Myocardial infarction: Patients' Health status) registry, a multi-center, prospective cohort of AMI patients from 24 United States hospitals. Details of the study design have been described previously.^{8,9} Briefly, participants were admitted with AMI between April 2005 and December 2008, were 18 years of age, had elevated troponin levels within 24 hours of hospital admission; and demonstrated other supportive evidence of acute AMI, including at least one of the following: prolonged (>20 minutes) ischemic signs or symptoms; at least one EKG with ST-elevation or ST-depression in two or more consecutive leads; or other clinical evidence of AMI. Patients were excluded if they were transferred to the recruiting institution from another facility more than 24 hours after their original presentation, refused or were unable to provide informed consent, or did not speak English or Spanish. In addition, in a predefined biomarkers substudy of TRIUMPH, patients from each site donated blood for central analysis of lipid profile, genetics and biomarkers.⁹ For the purpose of this analysis, we only included patients from this substudy of TRIUMPH (n=2370) with measurement of all 3 biomarkers (NT-proBNP, TnT and hs-CRP).

Chart abstraction was performed and patients were interviewed by trained personnel within 72 hours of admission to collect information on sociodemographic factors, medical history, clinical presentations, in-hospital events and treatments. In addition, baseline venous blood samples were drawn at enrollment within 72 hours of admission. NT-proBNP, TnT and hs-CRP analysis were performed at a central laboratory (Clinical Research Laboratories, Lenexa, Kansas) using standardized reagents (Roche Diagnostics, Indianapolis, IN). The Institutional Review Board at each participating institution approved the study, and all patients provided informed consent.

The primary outcome was the development of new-onset AF during the AMI hospitalization. AF was considered to be present if AF or atrial flutter was identified on 12-lead electrocardiogram or was seen for >30 seconds on a rhythm strip at AMI presentation

or during the AMI hospitalization. Patients with a history of AF or atrial flutter (n=212) were excluded.

All data were entered at each participating institution and transmitted to the national data coordinating center using a web-based data entry interface that provided range checking, logic, and consistency checks at the time of data entry. These were supplemented with more complex inter-form quality checks and queries sent to each site for resolution prior to analysis.

Demographic, clinical, treatment and biomarker characteristics between AF and non-AF MI patients were compared using student t-test for normally distributed continuous variables, Wilcoxon rank-sum test for skewed variables, and Chi-square or Fisher exact tests for categorical variables.

To identify characteristics that were independently associated with the development of new-onset AF, we first compared unadjusted median biomarker levels as well as biomarker levels in deciles between groups using Wilcoxon rank sum tests. Next, the effects of biomarker levels on risk of AF were estimated using multivariable hierarchical logistic regression models, accounting for clustering within site. Due to the presence of a skewed distribution of the biomarkers, NT-pro-BNP, TnT and hs-CRP data were analyzed as log transformed variables. The selection of covariates in multivariable models was based on both *a priori* and empirical considerations. First, important baseline covariates that are known to influence biomarkers were included in the analyses. Second, variables that differed significantly by AF occurrence on a bivariate basis ($p < 0.05$) were considered as potential confounders. Log transformed biomarker regression estimates were multiplied by log 2 to get the interpretation of a 2-fold multiplicative increase and exponentiated to obtain odds ratios. The resulting estimates reflect the relative increase in risk of AF per 2-fold increase in the corresponding biomarker. The retained covariates included patient demographics (age, sex, race), medical history (smoking, diabetes, congestive heart failure (CHF), hypertension and chronic lung disease), prior coronary heart disease [prior AMI, coronary artery bypass grafting (CABG) and percutaneous intervention (PCI)], type of AMI [ST-segment elevation MI (STEMI) vs. non-ST Elevation MI (NSTEMI)], clinical severity (left ventricular systolic function, systolic blood pressure, renal function and Killip class) and the use of primary PCI. In addition, interactions of gender and race with the biomarkers were tested. All tests for statistical significance were two-tailed with an alpha level of 0.05.

In addition, we conducted sensitivity analyses by including the variables that were found to be significantly different between groups in univariate analysis (beta blockers, calcium channel blockers and antiplatelet agents on arrival). The effect size of the biomarkers did not change.

All analyses were conducted using SAS software, release 9.2 (SAS Institute, Cary, North Carolina) and R version 2.11.1.¹⁰.

Results

A total of 2370 AMI patients were included in the analysis. New-onset AF was documented in 114 AMI patients (4.8%; mean age 58 years; 32% women). Of these, only 19 (16.7%) had new-onset AF on admission ECG and the remainder developed it during their hospitalization.

Medical history and clinical data, stratified by new-onset AF, are presented in Table 1. AMI patients who developed new-onset AF were more likely to be older, Caucasian and to have a higher prevalence of diabetes, hypertension, chronic lung disease and chronic kidney disease. They were less likely to smoke. There was no significant association between AF and higher Killip class, left ventricular ejection fraction (LVEF), STEMI, prior CHF or prior CHD. Patients with new AF were also more likely to receive beta-blockers, calcium channel blockers and antiplatelet agents on arrival and as likely to receive reperfusion and revascularization therapies as those without new-onset AF (Table 1).

In unadjusted analyses, new-onset AF patients had significantly higher levels of NT-proBNP and hs-CRP than those without AF (Table 2 and Figure 1). There were no statistically significant differences between the level of TnT between patients with and without new-onset AF.

Multivariable analyses including demographics, comorbidities and clinical factors revealed that for each 2-fold increase in the level of NT-proBNP, there was a 18% increase in the rate of AF (OR 1.17 95% CI 1.02-1.34; $p<0.02$). Similarly, for every 2-fold increase in hs-CRP, there was a 15% increase in the rate of AF (OR 1.15 95% CI 1.02-1.30; $p=0.02$). TnT was not independently associated with new AF (OR 0.94 95% CI 0.84-1.06; $p=0.3$) (Figure 2).

There was no significant interaction of race and gender with biomarkers for development of AF ($P=0.9$ and $P=0.19$ respectively).

Discussion

In this large prospective national registry of AMI patients, we found that for each 2-fold increase in the level of NT-proBNP and hs-CRP, there was a 18% and 15% increase, respectively, in the incidence of AF during AMI hospitalization, after adjusting for clinically relevant covariates. NT-proBNP and hs-CRP were strongly and independently associated with in-hospital AF. The strength of association for NT-proBNP and hs-CRP was higher than other clinical covariates such as LVEF, Killip class and CHF. In contrast, TnT was not independently associated with new-onset AF. The association between biomarkers and the development of new-onset AF did not differ by race or gender.

The results of our study extend the existing literature on risk factors for AF in the setting of AMI. Although several studies have examined predictors of AF in the general population, to our knowledge, there are limited data examining the association of biomarkers and new-onset AF after AMI. Our finding that NT-proBNP is independently associated with new in-hospital AF in AMI is consistent with most,¹¹⁻¹⁴ but not all¹⁵ prior studies of NT-proBNP and AF in the general population. While NT-proBNP was mostly strongly associated with

AF, congruent with prior analyses, we found that older age, male gender and chronic lung disease were also associated with new AF following AMI.¹⁶⁻¹⁹ Our study extends these previous investigations on predictors of AF after AMI that have been limited to examining demographic or clinical variables, were retrospective, limited to the elderly or Medicare beneficiaries, or were restricted to certain geographic areas.¹⁶⁻²⁰ Our findings that a history of CHD was associated with reduced risk of new AF is consistent with some⁵ but not all studies.²¹ Systolic function, either by quantitative measurements, or clinical surrogates such as Killip class, in contrast to previous studies, was not independently associated with new-onset AF. This is likely due to very small numbers of participants with higher Killip class, which would make detection of differences between the populations difficult. However, this would not explain the lack of association with systolic function. It is likely that NT-proBNP, a more sensitive marker for early and subtle changes in hemodynamics, is associated with other factors such as chamber enlargement, valvular disease and left ventricular hypertrophy, which may explain its greater association with new onset AF as compared with LV function or Killip class, rendering these latter variables insignificant after adjusting for NT-pro-BNP.

Our results further demonstrate that higher hs-CRP levels were associated with new AF post-AMI after adjusting for clinical factors, NT-proBNP and TnT. Although several, but not all, prior studies demonstrated independent associations of CRP with AF in several clinical contexts,²²⁻²⁴ including AMI^{25,26}, these prior studies did not take into account the level of NT-proBNP or TnT in their analyses. The independent association with hsCRP in our markers of myocardial stretch and necrosis are taken into account. Finally, the biomarker surrogate for myocardial damage in AMI, TnT, was not associated with AF post-MI, which is also consistent with prior studies.⁵⁻⁷ Pathogenetically, AF in MI appears to be multifactorial. Reasons for the good predictive utility of NT-proBNP and hs-CRP despite the poor correlation of TnT in AF during AMI can only be speculative. It is possible that in patients with MI, myocardial damage leads to inflammation and adverse hemodynamics, which in turn promote AF. Regardless of the specific physiologic link, our results suggest that the pathophysiologic factors responsible for elevations in NT-proBNP and hs-CRP are more directly linked to the genesis of AF during AMI than the factors causing elevated TnT.

Measurement of NT-proBNP and hs-CRP levels, in addition to well known clinical factors, may provide a reasonable and rapid way of risk-stratifying AMI patients who are at a high risk of developing AF. Patients with new onset AF in the peri-AMI period have a higher rate of death and complications, including re-infarction, cardiogenic shock and ventricular arrhythmias.²⁷ Since AF also complicates the clinical management of patients with AMI,^{28,29} it is important to identify high-risk patients who may benefit from a more careful monitoring and early treatment during MI hospitalization. In addition to pointing towards a potential predictive utility, independent association of NT-proBNP and hs-CRP also provide insight into the pathophysiologic mechanisms of new onset AF after AMI by suggesting that inflammation and myocardial stretch, but not the amount of myocardial necrosis are likely to be important drivers of new AF in patients in the setting of AMI.

Although we used a large national prospective registry of AMI patients in our study, some potential limitations should be considered in the interpretation of this study. As with all

observational studies, there we cannot exclude unmeasured confounding and cause and effect cannot be proved, although we were able to adjust for a wide array of demographic, clinical and disease severity characteristics. Since approximately 80% of our patients had LVEF >40%, results of our study are generalizable to patients with EF >40%. Since we do not have data on the duration of AF or timing of AF, we cannot comment on cause and effect. It is possible that AF may lead to higher levels of biomarkers or that higher levels of biomarkers in the setting of AMI lead to AF. Finally, we were unable to explore other, potentially more direct, measures that could illuminate that pathophysiological pathway, such as echocardiographic measurements of left atrial volume. Future investigations will need to further clarify the mechanisms of new onset AF. Finally, our observational study cannot examine the preventive impact of cardiac medications on atrial fibrillation, which would be best answered by a clinical trial.

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Rate of atrial fibrillation for deciles of NT-proBNP

Rate of atrial fibrillation for deciles of hs-CRP

Rate of atrial fibrillation for deciles of Troponin-T

Figure 1.

Bivariate association between biomarkers and atrial fibrillation after myocardial infarction

Figure 2.

Adjustment Model for Predictors of New Atrial Fibrillation during Hospitalization for Acute Myocardial Infarction

Table 1

Baseline characteristics comparing patients with and without new atrial fibrillation

	In-Hospital Atrial Fibrillation/Flutter		
	Yes n = 114	No n = 2256	P-Value
Sociodemographic factors			
Age (mean years ± SD)	64.6 ± 13.2	57.5 ± 11.9	< 0.001
Male	87 (76.3%)	1531 (67.9%)	0.059
Caucasian	88 (77.2%)	1506 (66.8%)	0.021
Education greater than High School	57 (50.4%)	1133 (50.4%)	1.000
Body mass index	30.4 ± 6.2	29.8 ± 6.6	0.396
Married	65 (57.0%)	1178 (52.4%)	0.331
Living alone	22 (19.6%)	535 (23.8%)	0.307
Working full or part time	35 (31.3%)	1155 (51.5%)	< 0.001
Has insurance	94 (84.7%)	1676 (75.9%)	0.034
Medical History			
Currently smoking	28 (24.8%)	977 (43.6%)	< 0.001
Family history of CAD	87 (77.0%)	1661 (74.5%)	0.555
Prior angina	16 (14.0%)	290 (12.9%)	0.714
Prior CHD (MI/PCI/CABG)	30 (26.3%)	682 (30.2%)	0.374
Prior CVA	9 (7.9%)	98 (4.3%)	0.075
Diabetes	48 (42.1%)	688 (30.5%)	0.009
Chronic kidney disease	13 (11.4%)	138 (6.1%)	0.024
Chronic lung disease	16 (14.0%)	145 (6.4%)	0.002
Hypertension	85 (74.6%)	1462 (64.8%)	0.033
Chronic heart failure	11 (9.6%)	162 (7.2%)	0.323
Dyslipidemia	54 (47.4%)	1057 (46.9%)	0.914
Peripheral vascular disease	7 (6.1%)	92 (4.1%)	0.330
Alcohol abuse	13 (11.4%)	235 (10.4%)	0.737
Clinical characteristics at admission			
Presenting heart rate	82.3 ± 22.5	82.5 ± 21.4	0.915
Presenting heart rate >100	20 (17.5%)	432 (19.2%)	0.661
Left ventricular systolic function >40	91 (79.8%)	1844 (81.8%)	0.600
STEMI	43 (37.7%)	1011 (44.8%)	0.137
Killip class on arrival III or IV	2 (1.8%)	47 (2.1%)	1.000
Presenting systolic blood pressure >100	109 (96.5%)	2124 (94.7%)	0.401
Treatment characteristics			
Fibrinolytic	6 (5.3%)	131 (5.8%)	0.808
GP IIb/IIIa Blocker	66 (57.9%)	1411 (62.5%)	0.318
Antiplatelet	67 (58.8%)	1539 (68.2%)	0.035
Anticoagulant	104 (91.2%)	2024 (89.7%)	0.603
Antithrombin	2 (1.8%)	99 (4.4%)	0.234
In-Hospital Revascularization	87 (76.3%)	1639 (72.7%)	0.391
Beta Blocker on arrival	53 (46.5%)	670 (29.7%)	< 0.001
	25 (21.9%)	296 (13.1%)	0.007

	In-Hospital Atrial Fibrillation/Flutter		
	Yes n = 114	No n = 2256	P-Value
CCB on Arrival	58 (50.9%)	927 (41.1%)	0.039
Antiplatelet on Arrival	56 (49.1%)	880 (39.0%)	0.031
Aspirin on Arrival			

Abbreviations

CHD: coronary heart disease

MI: myocardial infarction

PCI: percutaneous coronary intervention

CABG: coronary artery bypass grafting

CVA: cerebrovascular accident

STEMI: ST elevation myocardial infarction

Table 2

Bivariate association between biomarkers and atrial fibrillation after myocardial infarction

	In-Hospital Atrial Fibrillation/Flutter		P-Value
	Yes n = 114	No n = 2256	
CRP (Median (IQR) *)	3.5 (1.1, 7.7)	1.9 (0.7, 4.3)	< 0.001
NT-proBNP (Median (IQR) *)	1756.0 (825.0, 4603.0)	966.0 (418.5, 2227.5)	< 0.001
Troponin T (Median (IQR) *)	1.1 (0.3, 2.6)	0.9 (0.3, 2.1)	0.242

* Wilcoxon rank-sum tests