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ORIGINAL RESEARCH ARTICLE

Heart failure and atrial fibrillation: tachycardia-mediated acute decompensation

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Abstract

Aims Tachycardia is a reversible event that may cause hemodynamic decompensation but may not necessarily cause direct damages to the myocardium. To evaluate the clinical outcomes of patients with heart failure (HF) and atrial fibrillation (AF), whose acute decompensation was tachycardia mediated.

Methods and results The Korean Acute Heart Failure registry was a prospective registry that consecutively enrolled 5625 patients with acute HF. Patients were classified into three groups according to the rhythm and aggravating factor: (i) 3664 (65.1%) patients with sinus rhythm (SR), (ii) 1033 (18.4%) patients with AF whose decompensation was tachycardia-mediated, AF-TM (+), and (iii) *N* = 928 (16.5%) patients with AF whose decompensation was not tachycardia-mediated, AF-TM (−). The primary outcomes were in-hospital and post-discharge 1 year all-cause mortality. At admission, the mean heart rate was 90.8 ± 23.4, 86.8 ± 26.8, and 106.3 ± 29.7 beats per minute for the SR, AF-TM (−), and AF-TM (+) groups, respectively. The AF-TM (+) group had more favourable characteristics such as *de novo* onset HF, less diabetes, ischaemic heart disease, and higher blood pressure than the AF-TM (−) group. In-hospital mortality rates were 5.1%, 6.5%, and 1.7% for SR, AF-TM (−), and AF-TM (+) groups, respectively. In logistic regression analysis, the AF-TM (+) group had lower in-hospital mortality after adjusting the significant covariates (odds ratio, 0.49; 95% confidence interval, 0.26–0.93). The mortality rate did not differ between SR and AF-TM (−) groups. During 1 year follow-up, 990 (18.5%) patients died. In univariate and multivariate Cox proportional regression analyses, there was no difference in 1-year all-cause mortality between the three groups.

Conclusions In patients with HF and AF, patients whose acute decompensation is tachycardia-mediated have better in-hospital, but similar post-discharge outcomes compared with those with SR or those with AF whose decompensation is not tachycardia-mediated.

Clinical Trial Registration: ClinicalTrial.gov NCT01389843.

Keywords Heart failure; Sinus rhythm; Atrial fibrillation; Trigger; Tachycardia; Outcomes

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Introduction

Heart failure (HF) and atrial fibrillation (AF) share common risk factors such as older age, hypertension, and diabetes mellitus[1]; therefore, a substantial number of HF patients have AF.[2,3] Besides the coexistence, AF is important in HF, because it complicates the course of HF by increasing the morbidity and mortality.[4,5]

It is well documented that each hospitalization for acute decompensation confers an excess risk for worse clinical

outcomes for patients with HF.[6] Common aggravating factors include acute coronary syndrome and/or myocardial ischaemia, tachyarrhythmia, infection, and renal failure, among others.[7] Patients with AF can rapidly deteriorate hemodynamically by high ventricular rate, so that tachycardia can trigger acute decompensation in these patients.[8] An increase in heart rate elevates energy expenditure,[9] shortens the diastole, decreases coronary perfusion,[10] and increases ventricular loading via the alteration of vascular resistance.[11] Therefore, tachycardia can cause rapid haemodynamic decompensation leading to elevated filling pressure and congestion, especially in patients with HF. Nonetheless, tachycardia itself is a reversible condition that generally does not leave permanent damages to the myocardium compared to acute coronary syndrome, the leading cause for acute decompensation. Consequently, these patients may have different prognosis.

This study aimed to investigate the prognosis of patients with HF and AF whose acute decompensation was triggered by tachycardia in a large cohort of patients for the first time.

Methods

Patients

The Korean Acute Heart Failure (KorAHF) registry was a prospective multicentre cohort study that consecutively enrolled 5625 patients who were hospitalized for acute HF syndrome from 10 tertiary university hospitals throughout the country between March 2011 and December 2014. Detailed information on the study design and results has been previously reported elsewhere (ClinicalTrial.gov NCT01389843).[7,12] Briefly, patients with signs or symptoms of HF and either lung congestion, objective findings of LV systolic dysfunction, or structural heart disease were eligible for the study. The mortality data for patients who were lost to follow-up were collected from the National Death Records.

The study protocol was approved by the ethics committee/institutional review board at each hospital. Written informed consent was waived by the institutional review board. The investigation conforms with the principles outlined in the *Declaration of Helsinki*.

Study variables and definitions

All echocardiographic studies were performed by cardiologists who were certified by Korean Society of Echocardiography, using a standard ultrasound machine with a 2.5 MHz probe. Standard techniques were adopted to obtain M-mode, two-dimensional, and Doppler measurements in accordance with the American Society of Echocardiography's guidelines.[13] Left ventricular ejection fraction (LVEF) was measured using the Simpson's biplane method unless the Simpson's method was not possible. On the basis of the echocardiography findings, patients were classified into those with heart failure with reduced ejection fraction (HFrEF) (LVEF of $<$40%), HF with midrange ejection fraction (HFmrEF) (LVEF, 40–49%), and heart failure with preserved ejection fraction (HFpEF) (LVEF $\geq$ 50%).

Patients were defined as having AF if AF was documented in electrocardiogram during the index admission. Regarding the aggravating factor for acute decompensation, the responsible physician was asked to choose one of the following factors as the most-likely trigger for acute decompensation, which included acute coronary syndrome, severe hypertension, atrial or ventricular tachyarrhythmia, bradycardia, infection, pulmonary emboli, renal failure, anaemia/bleeding, medication (e.g. non-steroidal anti-inflammatory drugs), non-compliance, endocrinal abnormality, and recent addition of negative inotropic agents. The information on the aggravating factor was prospectively collected and was adjudicated before discharge by the investigators.

Patients were classified into three groups according to rhythm and aggravating factor: (i) patients with sinus rhythm (SR); (ii) patients with AF whose decompensation was tachycardia-mediated, AF-TM (+); and (iii) patients with AF whose decompensation was not tachycardia-mediated, AF-TM (−).

The primary outcomes were in-hospital and 1-year post-discharge all-cause mortality.

No patient and public involvement

This research was done without patient involvement. Patients were not invited to comment on the study design and were not consulted to develop patient relevant outcomes or interpret the results. Patients were not invited to contribute to the writing or editing of this document for readability or accuracy.

Statistical analysis

Data are presented as numbers and frequencies for categorical variables and as means ± standard deviations or medians with interquartile ranges for continuous variables. For comparisons among the groups, the χ^2 test (or Fisher's exact test when any expected count was $<$5 for a 2 × 2 table) was used for categorical variables, and the unpaired Student's *t*-test, one-way analysis of variance, or Kruskal–Wallis test was used for continuous variables.

The Get With the Guidelines-Heart Failure (GWTG-HF) score was calculated for each patient[14] and used for the estimation of risk.

In-hospital and post-discharge outcomes were analysed in relation to rhythm and trigger. Regarding the in-hospital

mortality univariate and multivariate logistic regression analyses were used to determine the effects of rhythm and trigger on all-cause mortality using the enter method. Regarding the 1 year post-discharge outcomes, Kaplan–Meier curves were plotted and compared using the log-rank test. Univariate and multivariate Cox proportional hazards regression models were used to determine the effects of rhythm and trigger on all-cause mortality using the forward selection method. Variables found to be statistically significant ($P < .1$) in the univariate analysis were included in the multivariate model, except for variables with >10% missing values or variables that are closely related to the other clinical variables.

A two-sided probability value < 0.05 was considered to be statistically significant. Statistical tests were performed using SPSS, V.22 (IBM, Armonk, NY, USA).

Results

Patients

Among the 5625 patients enrolled in the KorAHF registry, 3664 (65.1%) patients had SR, and 1961 (35%) patients had AF. Among the patients with AF, 928 (48%) were in the AF-TM (+) group (*Figure 1*).

Patients with SR were younger and more likely to have diabetes and ischaemic heart disease than those with AF. Additionally, they had higher natriuretic peptide levels but lower GWTG-HF score than those with AF (*Table 1*, *Figure 2A*). Among the patients with AF, the AF-TM (+) group had more favourable characteristics such as *de novo* onset HF, less diabetes, ischaemic heart disease, and higher blood pressure than the AF-TM (−) group.

At admission, the mean heart rate was 90.8 ± 23.4, 86.8 ± 26.8, and 106.3 ± 29.7 beats per minute (bpm) for the SR, AF-TM (−), and AF-TM (+) groups, respectively. The difference in heart rate became smaller at discharge (77.3 ± 13.8, 74.5 ± 14.0, and 77.2 ± 15.4 bpm for the SR, AF-TM (−), and AF-TM (+) groups, respectively, $P < .001$) and disappeared at 1 month after discharge (80.2 ± 16.2, 79.6 ± 18.5, and 81.9 ± 20.8 bpm for the SR, AF-TM (−), and AF-TM (+) groups, respectively, $P = .052$) (*Figure 2B*).

In-hospital outcomes

The AF-TM (+) group required less inotropes and the use of mechanical circulatory support device (*Table 2*). During the index admission, 269 (4.8%) patients died. The in-hospital mortality rates were 5.1%, 6.5%, and 1.7% for the SR, AF-TM (−), and AF-TM (+) groups, respectively. Under stratification by GWTG-HF score, the in-hospital mortality rates in the SR and AF-TM (−) groups increased with increasing GWTG-HF scores, whereas that in the AF-TM (+) group did not increase substantially (*Figure 3A*).

In logistic regression analysis, AF-TM (+) was associated with lower in-hospital mortality in a univariate analysis [odds ratio (OR), 0.33; 95% confidence interval (CI), 0.20–0.55] after the adjustment of GWTG-HF scores (OR, 0.28; 95% CI, 0.17–0.47) and significant covariates (OR, 0.49; 95% CI, 0.26–0.93). There was no difference in mortality between the SR and AF-TM (−) groups (*Table 3*).

Post-discharge outcomes

During 1 year follow-up, 990 (18.5%) patients died. In Kaplan–Meier survival analysis, there was no difference in mortality between the SR and AF groups (*Figure 3A*). However, under stratification by the aggravating factor, the AF-TM (+) group had the lowest mortality and AF-TM (−) the highest mortality (*Figure 3B*).

Figure 1 Study population. AF-TM (+), patients with atrial fibrillation (AF) whose decompensation was tachycardia mediated; AF-TM (−), patients with AF whose decompensation was not tachycardia-mediated; KorAHF, Korean Acute Heart Failure Registry.

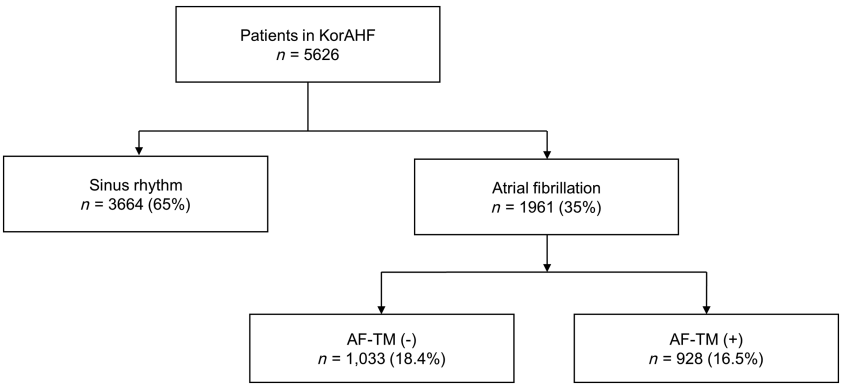


Table 1 Baseline characteristics of the study population

Characteristic	Sinus rhythm $N = 3664$ (65.1%)	AF-TM (−) $N = 1033$ (18.4%)	AF-TM (+) $N = 928$ (16.5%)	P value
Age (years)	67.3 ± 15.3	70.8 ± 12.0	70.6 ± 12.9	<0.001
Men (%)	54.3%	55.4%	46.7%	<0.001
De novo HF (%)	56.5%	35.8%	53.4%	<0.001
Body mass index (kg/m^2) ($n = 5581$)	23.3 ± 3.9	23.4 ± 3.9	23.5 ± 3.9	0.199
Past medical history				
Hypertension (%)	58.9%	58.3%	60.8%	0.493
Diabetes mellitus (%)	38.6%	31.6%	26.4%	<0.001
GFR < 60 mL/min/1.72 m^2 (%)	46.2%	47.5%	37.9%	<0.001
Ischemic heart disease (%) ($n = 5624$)	32.2%	25.3%	16.0%	<0.001
Valvular heart disease (%) ($n = 5624$)	9.9%	28.8%	16.1%	<0.001
COPD (%) ($n = 5624$)	10.5%	12.7%	12.5%	0.065
Cerebrovascular disease (%) ($n = 5624$)	13.1%	18.6%	19.4%	<0.001
Malignancy (%)	8.3%	7.6%	9.1%	0.484
ICD	1.6%	1.6%	1.2%	0.64
CRT	0.8%	0.1%	0.3%	0.018
NYHA functional class (%)				<0.001
II	15.1%	16.6%	14.0%	
III	35.1%	41.3%	38.9%	
IV	49.8%	42.1%	47.1%	
Physical exam				
Systolic BP (mmHg)	132.1 ± 31.6	127.5 ± 27.0	131.4 ± 28.2	<0.001
Diastolic BP (mmHg)	78.0 ± 18.8	76.5 ± 17.0	83.2 ± 19.7	<0.001
Heart rate (beats per min)	90.8 ± 23.4	86.8 ± 26.8	106.3 ± 29.7	<0.001
Laboratory findings				
Haemoglobin (mg/dL) ($n = 5619$)	12.3 ± 2.3	12.4 ± 2.3	13.1 ± 2.1	<0.001
Serum sodium (mmol/L) ($n = 5621$)	137.5 ± 4.7	136.9 ± 5.4	138.2 ± 4.5	<0.001
Serum potassium (mmol/L) ($n = 5621$)	4.4 ± 0.7	4.4 ± 0.7	4.3 ± 0.6	0,087
BUN (mg/dL) ($n = 5619$)	26.4 ± 16.8	27.4 ± 17.2	24.1 ± 14.0	<0.001
Creatinine (mg/dL) ($n = 5621$)	1.6 ± 1.6	1.4 ± 1.2	1.2 ± 0.9	<0.001
BNP (pg/mL) ($n = 2244$)	1,038 (508–2072)	713 (343–1,382)	756 (480–1,284)	<0.001
NT-proBNP (pg/mL) ($n = 3021$)	5197.5 (2088–13,583)	4,459 (2,253–10,454)	4,347 (2,215–9,065)	0.029
Troponin I ($n = 4431$)	0.09 (0.04–0.52)	0.05 (0.03–0.13)	0.04 (0.02–0.08)	<0.001
Troponin T ($n = 668$)	0.04 (0.02–0.10)	0.03 (0.18–0.05)	0.03 (0.02–0.05)	<0.001
CRP ($n = 5252$)	0.66 (0.30–2.60)	0.75 (0.30–2.45)	0.73 (0.30–2.00)	0.973
Heart failure type				<0.001
HFrEF	65.7%	49.6%	51.8%	
HFmrEF	13.15	15.6%	17.5%	
HFpEF	21.2%	34.8%	30.7%	
Echocardiographic parameters				
LVEDD (mm) ($n = 5240$)	58.2 ± 10.2	56.6 ± 10.0	54.9 ± 9.1	<0.001
LA diameter (mm) ($n = 5166$)	45.4 ± 8.4	54.5 ± 11.2	51.8 ± 8.6	<0.001
LVEF (%) ($n = 5103$)	36.2 ± 15.4	41.1 ± 15.8	39.9 ± 15.5	<0.001
E/e' ($n = 4372$)	21.4 ± 11.5	21.8 ± 13.0	19.8 ± 9.5	0.001
Medication				
RAS-inhibitor				
at admission (%)	36.9%	45.3%	35.7%	<0.001
during admission (%)	78.2%	69.8%	75.5%	<0.001
at discharge (%)	67.4%	59.6%	67.0%	<0.001
Beta blockers				
at admission (%)	26.9%	32.2%	29.8%	0.002
during admission (%)	58.4%	49.0%	65.0%	<0.001
at discharge (%)	50.9%	39.9%	57.0%	<0.001
MRA				
at admission (%)	16.1%	28.5%	18.4%	<0.001
during admission (%)	54.3%	59.1%	57.8%	0.011
at discharge (%)	43.1%	49.1%	47.5%	0.001
Digoxin (%)	20.3%	53.1%	64.7%	<0.001
Amiodarone (%)	13%	16.7%	22.8%	<0.001
GWTG-HF score	40.4 ± 8.1	42.0 ± 8.0	42.4 ± 7.7	<0.001

BNP, brain natriuretic peptide; BP, blood pressure; COPD, chronic obstructive pulmonary disease; CRP, C-reactive protein; CRT, cardiac resynchronization therapy; GFR, glomerular filtration rate; GWTG-HF, get with the guideline-heart failure; HFmrEF, heart failure with mid-range ejection fraction; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction; ICD, implantable cardioverter defibrillator; LA, left atrial; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricle ejection fraction; NT-proBNP, *N*-terminal pro-brain natriuretic peptide; MRA, mineralocorticoid receptor antagonist; NYHA, New York Heart Association; RAS-inhibitor, renin-angiotensin-system inhibitor.

Figure 2 Natriuretic peptide levels and heart rate of the patients. (A) The AF-TM (+) group had lowest B-type natriuretic peptide level and *N*-terminal pro-B-type natriuretic peptide levels at admission. (B) At admission, the AF-TM (+) group had higher heart rate. However, the difference in heart rates became smaller at discharge and disappeared at 1 month after discharge between the groups. Abbreviation similar in *Figure 1*; BNP, B-type natriuretic peptide level; NT-proBNP, *N*-terminal pro-B-type natriuretic peptide.

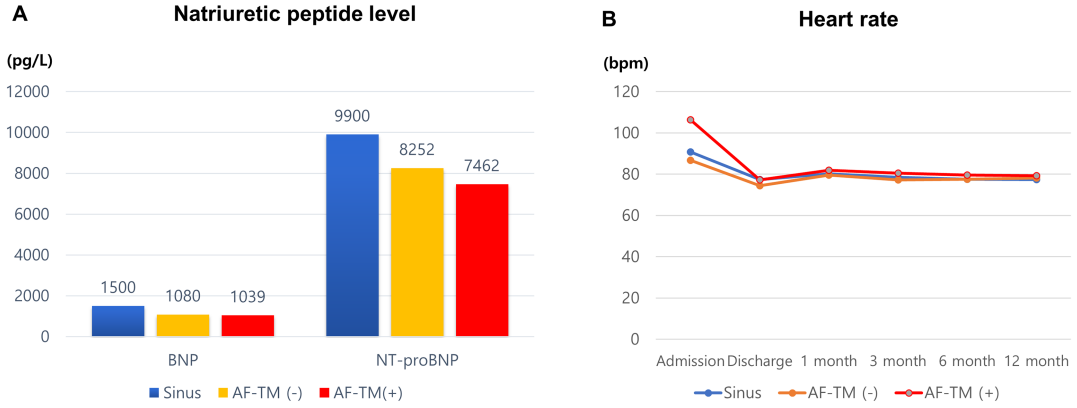


Table 2 Clinical outcomes

Outcome	Sinus rhythm *N* = 3664 (65.1%)	AF-TM (−) *N* = 1033 (18.4%)	AF-TM (+) *N* = 928 (16.5%)	*P* value
Inotropes use (%)	34.0%	32.9%	17.8%	<0.001
Intravenous vasodilator (%)	42.4%	39.1%	37.2%	0.007
Mechanical ventilator (%)	16.9%	16.7%	7.5%	<0.001
Duration (days)	7.0 ± 12.6	10.9 ± 21.7	5.5 ± 8.2	0.004
MCSD	7.4%	4.7%	1.3%	<0.001
IABP (%)	4.4%	3.1%	0.4%	<0.001
ECMO (%)	3.4%	2.6%	0.5%	<0.001
CRRT (%)	57.1%	79.7%	55.6%	0.001
WRF (%)	57.6%	56.3%	44.2%	<0.001
Improved WRF(%)	60.0%	64.2%	68.3%	0.003
In-hospital outcomes				
Death	5.1%	6.5%	1.7%	<0.001
CV-deaths or urgent HTx	5.4%	6.6%	1.6%	<0.001
Death or urgent HTx	6.2%	8.0%	2.3%	<0.001
Post-discharge outcomes				
1 year all-cause death (%)	18.3%	21.8%	15.7%	0.002
1 year HHF (%)	21.0%	22.5%	16.9%	0.006
1 year death + HHF (%)	34.0%	37.0%	28.6%	<0.001

CRRT, continuous renal replacement therapy; ECMO, extra-corporeal membrane oxygenation; HHF, hospitalization for heart failure; HTx, heart transplantation; IABP, intra-aortic balloon pump; MCSD, mechanical circulatory support device; WRF, worsening renal function.

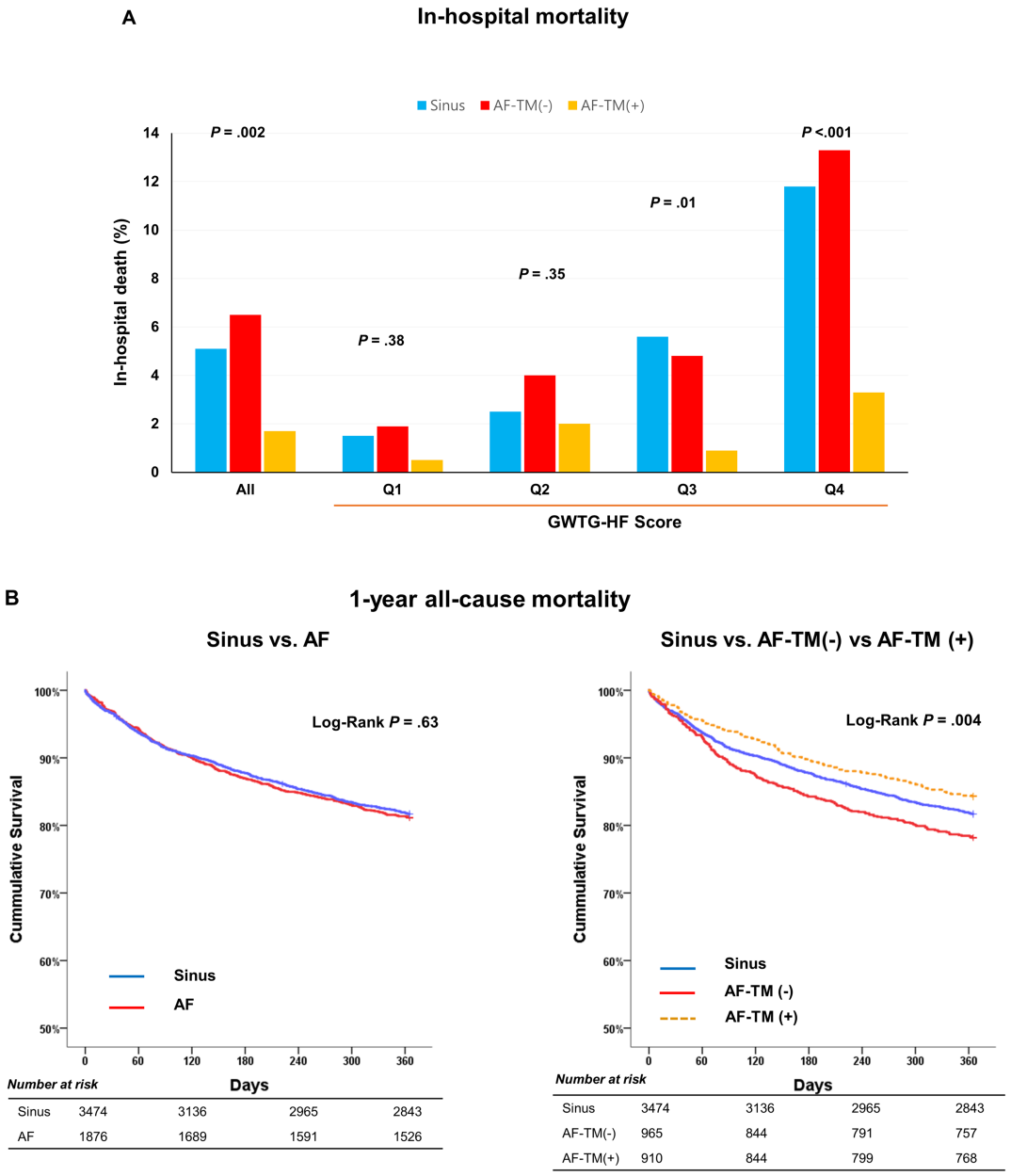
In multivariate Cox proportional regression analyses, there was no difference in 1 year all-cause mortality between the three groups. Similar findings were observed for the composite of all-cause mortality and hospitalization for HF (*Table 3*).

Outcomes according to rhythm and heart rate

We undertook several additional analyses. Patients were stratified according to rhythm and heart rate at admission. Overall, 2724 (43.1%) patients had SR and heart rate $<$ 100 bpm, 1237 (22%) had SR and heart rate $\geq$ 100 bpm, 1143 (20.3%) had AF and heart rate $<$ 100 bpm, and 818 (14.5%) had AF and heart rate $\geq$ 100 bpm. The corresponding in-hospital mortality was 3.9%, 7.4%, 4.5%, and 3.8%, respectively ($P < 0.001$). Regarding post-discharge outcomes, the 1 year mortality did not differ between the groups (Supporting Information, *Figure S1A*).

The information on sinus conversion during admission was missing in 157 (2.8%) patients. Among patients with AF, 299 (5.3%) had sinus conversion before discharge. Among those, 87 (29.1%), 174 (58.2%), and 38 (12.7%) patients had spontaneous, pharmacological, and electrical sinus conversion, respectively. Their 1 year mortality was similar to the other groups (*Figure S1B*).

Figure 3 Clinical outcomes. (A) In-hospital mortality: the AF-TM (+) group had lowest in-hospital mortality. The results were similar under stratification by GWTG-HF score. (B) 1 Year post-discharge mortality: the mortality did not differ between the patients with sinus rhythm and atrial fibrillation (AF) (left panel). Under stratification by the trigger, the AF-TM (+) group had the lowest mortality, and the AF-TM (−) group the highest mortality (right panel). Abbreviation similar in *Figure 1*. GWTG-HF, Get With the Guidelines-Heart Failure.



The information on the type of AF was available in 1787 patients: 306 (17.1%) and 1481 (82.9%) patients had paroxysmal and permanent or persistent AF, respectively. The post-discharge mortality did not differ between the groups, either (*Figure S1C*).

When stratifying the patients according to HF onset, the mortality did not differ between the groups in patents with de-novo HF (*Figure S1D*) and in those with chronic HF (*Figure S1E*).

Discussion

In this large, prospective cohort of patients with acute HF, we examined the outcomes of patients according to the rhythm and the aggravating factor. We showed that patients with HF and AF whose decompensation was triggered by tachycardia had better in-hospital outcomes. In contrast, their post-discharge 1 year mortality did not differ from those with

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Table 3 Impact of rhythm and trigger on clinical outcomes

	Sinus	AF-TM(−)	AF-TM (+)
In-hospital death			
Univariate analysis	Reference	1.30 (0.97–1.73)	0.33 (0.20–0.55)
Adjusted for GWTG-HF score	Reference	1.08 (0.79–1.46)	0.28 (0.17–0.47)
Adjusted for covariates*	Reference	1.35 (0.89–2.04)	0.49 (0.26–0.93)
Post discharge 1 year ACM			
Univariate analysis	Reference	1.21 (0.38–3.84)	0.45 (0.06–3.47)
Adjusted for GWTG-HF score	Reference	1.20 (0.37–3.81)	0.43 (0.06–3.40)
Adjusted for covariates*	Reference	0.65 (0.08–8.65)	0.80 (0.07–9.81)
Post discharge 1 year ACM + HHF			
Univariate analysis	Reference	1.24 (0.49–3.18)	0.57 (0.13–2.46)
Adjusted for GWTG-HF score	Reference	1.17 (0.46–3.00)	0.52 (0.12–2.27)
Adjusted for covariates*	Reference	0.89 (0.75–1.04)	0.87 (0.73–1.04)

Adjusted odds ratio for in-hospital death was calculated with logistic regression analysis. Following variables were included: sex, age, *de novo* onset, diabetes mellitus, ischemic heart disease, previous valve disease, COPD, cerebrovascular disease, CRT, NYHA, systolic blood pressure, heart rate, haemoglobin, Na, K, BUN, LVEDD, LA diameter, E/e; LVEF, RAS-inhibitor use before admission, BB use before admission, and MRA use before admission. Adjusted hazard ratio for 1 year mortality was calculated with Cox-proportional hazard regression analysis. Following variables were included: sex, age, *de novo* onset, diabetes mellitus, ischemic heart disease, previous valve disease, COPD, cerebrovascular disease, CRT, NYHA, systolic blood pressure, heart rate, haemoglobin, Na, K, BUN, LVEDD, LA diameter, E/e; LVEF, RAS-inhibitor use at discharge, BB use at discharge, and MRA use at discharge. AF-TM (+): patients with AF whose decompensation was tachycardia-mediated; AF-TM (−), patients with AF whose decompensation was not tachycardia-mediated; GWTG-HF, get with the guideline-heart failure.

SR and those with AF without tachycardia-mediated acute decompensation. This study implies that AF-TM (+) had similar long-term prognosis with other HF types and hence required similar medical attention despite their favourable in-hospital outcomes.

Heart failure, trigger, and acute decompensation

Regarding the pathophysiology of HF, damage to the cardiac myocytes and extracellular matrix leads to changes in the size, shape, and function of the heart and cardiac wall stress, leading to systemic neurohumoral overactivation.[15] Maladaptive remodelling and progressive worsening of LV function lead to increased morbidity and mortality; nonetheless, despite these structural alterations, many patients remain in a compensated state without overt HF symptoms. In contrast, the haemodynamic alterations with successive congestion cause the occurrence of HF symptoms such as dyspnoea and oedema.[15] The haemodynamic alteration can be reversed medically.

The rate and severity of decompensation depend on the vulnerability of the heart and the impact size of the trigger. Some triggers cause direct damage to the myocardium, whereas others cause impairment confined to haemodynamic alterations.

Common triggers include acute coronary syndrome/ischaemia, infection, non-compliance, and tachycardia, among others.[7] Ischaemic insult can cause permanent damage to the myocardium, and the accumulation of myocardial damage aggravates the prognosis.[16] Pneumonia itself is a life-threatening condition, which is one of the leading causes of death in elderly and comorbid patients.[17] In contrast, tachycardia is a trigger that causes an isolated haemodynamic decompensation without causing structural damage to the myocardium. Patients with HF with AF have loss of atrial kick and decreased diastolic function; therefore, they are especially vulnerable to tachycardia-medicated haemodynamic alteration.[2] Tachycardia is usually an acute and temporary event that can be easily corrected with medical therapy. For this reason, patients with HF and AF whose decompensation was triggered by tachycardia may have different prognosis.

Impact of atrial fibrillation tachycardia-mediated decompensation on outcomes

We showed that patients with AF-TM (+) had better in-hospital outcomes in a univariate analysis after the adjustment of GWTG-HF scores and significant factors. As expected, the in-hospital mortality increased linearly with increasing GWTG-HF scores in patients with SR and AF-TM (−). In contrast, the AF-TM (+) group had steadily lower mortality across all GWTG-HF quartiles, suggesting that tachycardia-mediated acute decompensation is rather a transient event that does not confer the excess risk of in-hospital mortality. It is of note that the AF-TM (+) group had more favourable characteristics such as higher sodium level and lower creatinine level. Most importantly, they have lower natriuretic peptide levels. In patients with AF, hemodynamic status can deteriorate rapidly through the loss of atrial contraction, and high ventricular rate and appropriate treatment can rapidly restore haemodynamics and lead to better in-hospital outcomes. Appropriate rhythm control and less severe structural cardiac abnormalities were reflected by higher beta-blocker prescription during admission and at discharge and low natriuretic peptide levels, respectively.

Another key finding was that there was no difference in post-discharge mortality between the groups, which is surprising when considering the favourable in-hospital outcomes of patients with AF-TM (+). Because HF itself is a disease with a grave prognosis, the trigger for acute decompensation does not seem to play an important role on the long-term outcomes. In literature, the prognostic value of AF is controversial in HF. Previous meta-analysis revealed that HF patients with AF had higher risk for deaths than those with SR.[5,18] By contrast, in the Danish Investigations of Arrhythmia and Mortality ON Dofetilide Congestive Heart Failure trial[19] and the Carvedilol or Metoprolol European Trial,[20] baseline AF was not associated with all-cause mortality. There exit several studies investigating the effect of precipitating factor of acute decompensation on the clinical outcomes. Arrigo *et al*.[21] showed that AF with rapid ventricular response was not associated with increased 90 day readmission and 1 year mortality. Fonarow *et al*.[22] investigated the hospital outcomes of 48 612 patients from the OPTIMIZE-HF database. Interestingly, after adjustment for significant covariates, arrhythmia was associated with 15% reduced risk for in-hospital mortality with marginal statistical significance (OR 0.85, 95% CI 0.71–1.01, $P = 0.07$). Nonetheless, the authors did not differentiate underlying disease of arrhythmia, that is AF versus ventricular arrhythmia, so that the effect of AF on in-hospital outcomes remains unelucidated. In the GREAT registry, analysing the data of 15 828 AHF patients from Europe and Asia, in AHF precipitated by AF, was associated with a 44% reduced risk for 90 day risk of deaths (adjusted hazard ratio: 0.56; 95% CI 0.42–0.75).[8]

The prognostic value of AF seems to depend on the HF type. Zafrir *et al*.[23] showed that the prevalence of AF increases with increasing EF, but its association with worse cardiovascular outcomes remained significant in patients with HFpEF and HFmrEF, but not in those with HFrEF. Similarly, our group also showed that AF was associated with increased mortality only in patients with HFpEF, but not in HFrEF or HFmrEF.[24]

Regarding the initial management for patients with AF whose decompensation was tachycardia-mediated, the rate control should be the first step in symptom management.[3]

Limitations

There are several strengths in our study. AF was documented by electrocardiogram, and because KorAHF study was a large, prospective cohort study, we could identify the trigger for acute decompensation in all patients. The trigger in each patient was prospectively collected, confirmed, and adjudicated by the investigator. Nonetheless, the classification of tachycardia-mediated acute decompensation can be problematic because clear definition of its classification does not exist. Despite the great effort to accurately elucidate the trigger for the acute decompensation in each patient, it remained subjective and was left to the judgement of the investigators, which may limit the reproducibility of the study results. Nonetheless, we believe that the results are biologically plausible. Because of the nature of the study design, albeit a large, prospective cohort study, we were unable to exclude the confounding factors that may have influenced the study results, including the difference in beta-blocker prescription rates between the groups. Because we only enrolled patients hospitalized for acute HF in East Asia, the generalisability of our study to other racial groups may be limited. Lastly, some patients with 'mild symptoms' who visited the emergency department with AF and tachycardia may be discharged without hospitalization, so that more 'severe' patients might have been enrolled in this study, which predisposes a possible selection bias.

Conclusions

In patients with HF and AF, patients whose acute decompensation is tachycardia-mediated have better in-hospital, but similar post-discharge, outcomes compared with those with SR or those with AF whose acute decompensation is not tachycardia-mediated.

Conflict of interest

The authors declare no conflict of interest.

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Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Figure S1. One-year all-cause mortality according to rhythm and trigger. (A) Under stratification by rhythm and heart rate ≥100 bpm at admission, the 1-year mortality did not differ between the groups. (B) Among patients with AF, 299 (5.3%) had sinus conversion before discharge, and their 1-year mortality was similar to the other groups. (C)

Regarding AF type, 306 (17.1%) and 1481 (82.9%) patients had paroxysmal and permanent or persistent AF, respectively. The post-discharge morality did not differ between the groups. When stratifying the patients according to heart failure onset, (D) the morality did not differ between the groups in patents with de-novo HF and (E) in those with chronic HF. AF-TM (+), patients with AF whose decompensation was tachycardia mediated; AF-TM (-), patients with AF whose decompensation was not tachycardia-mediated; KorAHF, Korean Acute Heart Failure Registry.

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