



Optimal timing of electrical cardioversion for acute decompensated heart failure caused by atrial arrhythmias: The earlier, the better?

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Abstract

The optimal timing for electrical cardioversion (ECV) in acute decompensated heart failure (ADHF) with atrial arrhythmias (AAs) is unknown. Here, we retrospectively evaluated the impact of ECV timing on SR maintenance, hospitalization duration, and cardiac function in patients with ADHF and AAs. Between October 2017 and December 2022, ECV was attempted in 73 patients (62 with atrial fibrillation and 11 with atrial flutter). Patients were classified into two groups based on the median number of days from hospitalization to ECV, as follows: early ECV (within 8 days, $n=38$) and delayed ECV (9 days or more, $n=35$). The primary endpoint was very short-term and short-term ECV failure (unsuccessful cardioversion and AA recurrence during hospitalization and within one month after ECV). Secondary endpoints included (1) acute ECV success, (2) ECVs attempted, (3) periprocedural complications, (4) transthoracic echocardiographic parameter changes within two months following successful ECV, and (5) hospitalization duration. ECV successfully restored SR in 62 of 73 patients (85%), with 10 (14%) requiring multiple ECV attempts (≥ 3), and periprocedural complications occurring in six (8%). Very short-term and short-term ECV failure occurred without between-group differences (51% vs. 63%, $P=0.87$ and 61% vs. 72%, $P=0.43$, respectively). Among 37 patients who underwent echocardiography before and after ECV success, the left ventricular ejection fraction (LVEF) significantly increased (38% [31–52] to 51% [39–63], $P=0.008$) between admission and follow-up. Additionally, hospital stay length was shorter in the early ECV group than in the delayed ECV group (14 days [12–21] vs. 17 days [15–26], $P<0.001$). Hospital stay duration was also correlated with days from admission to ECV (Spearman's $\rho=0.47$, $P<0.001$). In clinical practice, early ECV was associated with a shortened hospitalization duration and significantly increased LVEF in patients with ADHF and AAs.

Keywords Heart failure · Atrial arrhythmias · Electrical cardioversion · Hospital stay

Introduction

Atrial arrhythmias (AAs) and heart failure (HF) have been found to interact synergistically, leading to hemodynamic deterioration [1–3]. Recently, due to emerging evidence, the paradigm regarding AA management has shifted markedly to emphasize effective rhythm rather than rate control [4], highlighting the importance of rhythm control interventions in the management of AAs. Among these strategies,

electrical cardioversion (ECV) has emerged as the foremost therapeutic option for restoring sinus rhythm (SR) in patients with AA [5, 6]. Nevertheless, the timing of ECV that is ideal for sustaining SR and ensuring cardiac function recovery and associated clinical outcomes remains ambiguous in patients hospitalized for acute decompensated HF (ADHF) with concurrent AAs. The aim of this study was to retrospectively evaluate the impact of ECV timing on SR maintenance, hospitalization duration, and change in cardiac function in patients with ADHF and AAs and to identify factors associated with good outcomes after ECV.

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Materials and methods

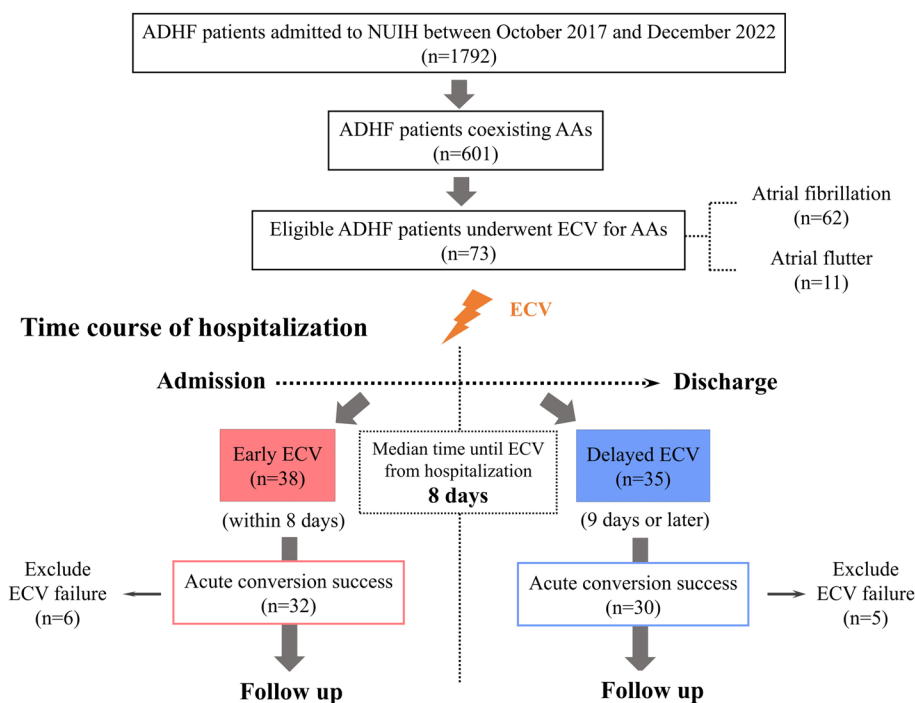
Study design

Data were obtained from SAKURA HF REGISTRY-2 (UMIN 000043852), a single-center, prospective, observational cohort registry. Among the 1,792 patients with HF who were included in the registry between October 2017 and December 2022, 601 were hospitalized for ADHF with concomitant AAs, including atrial fibrillation (AF) and atrial flutter (AFL). In all cases, according to guidelines, ECV was generally indicated for strongly suspected AAs contributing to the worsening of HF symptoms despite optimal medical treatment [3]. However, ECV was not performed in patients with possible left atrial (LA) or left atrial appendage (LAA) thrombus on transesophageal echocardiography (TEE), spontaneous SR restoration before the initiation of ECV, severe hemodynamic compromise requiring intensive care management, or the inability to give informed consent. In total, ECV was attempted in 73 patients (62 with AF and 11 with AFL) during hospitalization in accordance with the current treatment guidelines (Fig. 1) [7]. The patients who died in the hospital or were transferred to another hospital were excluded. Clinical presentation was categorized as (1) typical AA symptoms (palpitation with or without other concomitant symptoms); (2) atypical AA symptoms (shortness of breath without palpitations or chest pain); or (3) others (leg edema without any other symptoms) [8].

The timing of ECV was determined on the basis of the patient’s hemodynamic tolerance and anticoagulation before ECV. All patients underwent transesophageal echocardiography (TEE) before ECV to confirm the absence of LA or LAA thrombi. ECV was performed using short sedation with intravenous midazolam, thiopental, or propofol under continuous blood pressure and electrocardiogram (ECG) monitoring. The cardioversion protocol included adhesive pads with the anterior–posterior electrode position and a biphasic shock waveform with up to four cardioversion attempts [9]. Energy level of cardioversion was gradually increased to a maximum of 200 J. A 12-lead ECG trace was recorded before and after the procedure. Periprocedural antiarrhythmic drugs (AADs) were administered within 24 h after ECV. The AAD regimen was administered at the discretion of the physician. Each patient’s diagnosis of HF was based on Framingham criteria [3].

Patients were classified into the following two groups based on the median duration from hospitalization to ECV: early and delayed ECV [10]. Catheter ablation (CA) was not performed during the study period. Before admission, frailty was assessed using the Clinical Frailty Scale (CFS) during the stable phase. The CFS score was assessed based on interviews with patients and their families. This study complied with the principles of the Declaration of Helsinki. The use of patient information was approved by the Nihon University Itabashi Hospital Ethics Committee (RK-180612-2).

Fig. 1 Flow chart of the present study. AA atrial arrhythmias, ADHF acute decompensated heart failure, ECV electrical cardioversion, NUIH Nihon University Itabashi Hospital



Endpoints

The primary evaluated endpoints included both very short-term and short-term ECV failure. Very short-term failure was defined as a combination of unsuccessful cardioversion and AA recurrence during hospitalization. Short-term failure was defined as the same combination occurring within a month after ECV [9, 11, 12]. Successful ECV was defined as a restored SR lasting at least 1 min after ECV. Secondary endpoints included the (1) acute success of ECV, (2) number of ECVs attempted before SR restoration, (3) the maximum energy delivered for successful ECV, (4) periprocedural complications, (5) changes in transthoracic echocardiographic parameters within 2 months after successful ECV, and (6) duration of hospitalization. Periprocedural complications included sedation-related complications, bradycardia (heart rate [HR] < 40 bpm), hypotension requiring treatment, thromboembolism, worsening HF, and critical arrhythmia such as ventricular fibrillation or cardiac arrest (> 5 s) requiring treatment [11, 13, 14]. After ECV, ECG was continuously monitored throughout hospitalization in all patients. Furthermore, outpatient hospital visits were scheduled within 2–3 weeks of discharge to check the 12-lead ECG. When patients experienced symptoms such as palpitations or shortness of breath, they visited the emergency department to detect AAs. AAs lasting > 30 s on the ECG monitor, 24-h Holter, or 12-lead ECG were defined as recurrent AAs [7].

Statistical analysis

Categorical variables are reported as counts and percentages and were compared using Pearson's χ^2 or Fisher's exact tests, where appropriate. Distributions of continuous variables were assessed using the Shapiro–Wilk test and are presented as a mean \pm standard deviation [SD] or median and IQR. Continuous variables were compared using Student's t-test or the Mann–Whitney *U* test, as appropriate. Kaplan–Meier cumulative survival curves were constructed with group differences compared using the log-rank test to estimate the short-term failure of ECV. The relationship between the duration of hospitalization and time to ECV after hospitalization was tested using Spearman's rank correlation test.

To investigate factors with the potential to affect the duration of hospitalization, we defined a long hospital stay as one that was longer than the median hospital stay duration of the group, as was done in previous analyses [10]. Among the study population, the median duration of hospitalization was 16 days. Univariate and multivariate logistic regression analyses were performed to identify factors associated with a long hospital stay. In the multivariable analysis, all variables with a single-variable value of $P < 0.05$ were adjusted. Furthermore, univariate and

multivariate Cox proportional hazards regression analyses were used to identify predictors of short-term ECV failure. To satisfy assumptions of the model, a natural transformation (ln) was applied to N-terminal pro-brain natriuretic peptide (NT-proBNP) data. Statistical analyses were performed using JMP Pro 16.1.0 (SAS Institute, Cary, NC, USA).

Results

Patient characteristics

Between October 2017 and December 2022, 1792 patients hospitalized for HF had data included in the SAKURA HF REGISTRY-2 (UMIN 000043852). Among them, 601 were hospitalized for ADHF with concomitant AAs. After inclusion and exclusion criteria were applied, 73 patients (62 with AF and 11 with AFL) who underwent ECV were included in this study. Patients were classified into the following two groups based on the median duration from hospitalization to ECV: early ECV (ECV within 8 days of hospitalization, $n = 38$) or delayed ECV (ECV performed at least 9 days after hospitalization, $n = 35$) [9]. The median number of days to ECV was 4 (interquartile range [IQR] = 3–7) for those of the early ECV group and 13 (IQR = 11–16) for those of the delayed ECV group.

Baseline characteristics of included patients are shown in Table 1. The prevalence of stroke history, CHA₂DS₂-VASc score, and hemoglobin A1c levels were significantly increased in the early ECV group versus those in the delayed ECV group (all $P < 0.05$). Other medical history-related factors, laboratory findings, transthoracic echocardiographic parameters, and heart failure etiologies of the two groups were similar. Regarding clinical presentation on admission, 33% (24), 63% (46), and 3% (2) of the patients presented with typical, atypical, and other AA symptoms, respectively. The clinical characteristics on the day of ECV are shown in Table 2. The frequency of New York Heart Association (NYHA) class IV and HR values just before ECV were significantly higher in early ECV group than in the delayed group (all $P < 0.05$). In addition, AFL was more frequent (27% vs. 3%, respectively, $P = 0.008$). AF occurred less frequently (73% vs. 97%, respectively, $P = 0.004$) in the early ECV group than in the delayed ECV group. Although the delayed ECV group more frequently received mineral corticoid receptor antagonists ($P = 0.020$) and less frequently received bepridil ($P = 0.039$), no between-group differences regarding other HF medications or periprocedural AADs were observed.

Table 1 Baseline characteristics

	Early ECV (n = 38)	Delayed ECV (n = 35)	P value
The hospitalization to ECV time interval, days	4 (3–7)	13 (11–16)	<0.001
<i>Demographic</i>			
Age (years)	69 (62–78)	70 (63–79)	0.69
Male, n (%)	22 (58)	21 (60)	0.86
BMI (kg/m ²)	25.0 (20.8–29.9)	24.7 (22.7–28.7)	0.69
Clinical frailty scale, mean	3 (2–4)	3 (2–4)	0.35
<i>Clinical status on admission</i>			
New-onset AAs, n (%)	20 (53)	24 (69)	0.16
NYHA class IV, n (%)	14 (37)	11 (31)	0.62
SBP, mmHg	130 (113–150)	133 (115–157)	0.44
DBP, mmHg	86 (75–107)	89 (82–107)	0.45
Heart rate, bpm	125 (111–136)	124 (97–146)	0.87
Typical AA symptom			
Palpitations	14 (37)	10 (29)	0.45
Atypical AA symptom			
Chest pain	3 (8)	0 (0)	0.09
Shortness of breath without palpitations	22 (58)	23 (66)	0.49
Others			
Leg edema without any other symptoms	1 (3)	1 (3)	0.95
<i>Medical history</i>			
CHADS2-VASc score, mean	3 (2–3)	2 (1–3)	0.043
Hypertension, n (%)	22 (58)	17 (48)	0.42
Diabetes mellitus, n (%)	9 (24)	5 (14)	0.31
Stroke, n (%)	5 (13)	0 (0)	0.026
History of pacemaker implantation ^a , n (%)	1 (3)	2 (6)	0.60
Vascular disease, n (%)	5 (13)	2 (6)	0.28
Coronary heart disease, n (%)	5 (13)	2 (6)	0.28
COPD, n (%)	3 (8)	1 (3)	0.35
Prior HF hospitalization, n (%)	9 (24)	8 (23)	0.93
<i>Laboratory data on admission</i>			
Hemoglobin A1c, %	6.1 (5.7–6.5)	5.8 (5.5–6.2)	0.043
Hemoglobin, g/L	13.8 (11.6–14.5)	13.9 (11.9–15.8)	0.29
eGFR, ml/min per 1.73m ²	52.3 (39.4–66.9)	49 (38.9–67.3)	0.89
NT-proBNP, pg/mL	3952 (2437–6300)	4174 (3015–7342)	0.44
<i>TTE findings on admission (n = 65)</i>			
LVEF, %	38 (32–54)	41 (33–47)	0.88
LVEDV, mL	130 (101–166)	126 (94–157)	0.49
LVESV, mL	71 (47–114)	65 (46–72)	0.60
LAVI, mL/m ²	47 (39–59)	48 (37–56)	0.96
E/e' average ^b	13 (11–16)	16 (9–18)	0.92
Mitral regurgitation, n (%)			
Moderate/severe	12 (32)	9 (27)	0.53
<i>HF cause, n (%)</i>			
Tachycardia induced cardiomyopathy	22 (58)	22 (63)	0.66
Ischemic	2 (5)	1 (3)	0.60
Valvular	5 (13)	3 (9)	0.71
Other	9 (24)	9 (26)	1.00

Values are expressed as the median (interquartile range) or number (%)

AA, atrial arrhythmia; BMI, body mass index; Bpm, beats per minute; COPD, chronic obstructive pulmonary disease; DBP, diastolic blood pressure; E, peak early diastolic transmitral filling velocity; e', peak early diastolic mitral annular velocity; ECV, electrical cardioversion; eGFR, estimated glomerular filtration rate; HF, heart failure; LAA, left atrial appendage; LAVI, left atrial volume index; LVEF, left ventricular

Table 1 (continued)

ejection fraction; LVEDV, left ventricular end-diastolic volume; LVESV, left ventricular end-systolic volume; NT-proBNP, N-terminal fragment of pro-B-type natriuretic peptide; NYHA, New York Heart Association; SBP, systolic blood pressure; TTE, transthoracic echocardiography

^aPacemaker implantations are defined as a conventional pacemaker, implantable cardioverter-defibrillator, cardiac resynchronization therapy pacemaker, or cardiac resynchronization therapy defibrillator

^bThe average ratio of early transmitral filling velocity (E) to early diastolic mitral annular velocity (e') was calculated from both the medial ($E/e' = \text{medial}$) and lateral ($E/e' = \text{lateral}$) mitral annular sites

Table 2 Clinical variables on the day of ECV

	Early ECV ($n = 38$)	Delayed ECV ($n = 35$)	<i>P</i> value
<i>Pre-cardioversion clinical status</i>			
NYHA functional class IV, n (%)	15 (40)	5 (14)	0.020
SBP, mmHg	116 (104–129)	113 (100–122)	0.43
DBP, mmHg	74 (63–87)	71 (63–79)	0.15
Heart rate, bpm	92 (79–123)	86 (74–98)	0.029
<i>Cardiac rhythm prior to ECV, n (%)</i>			
Atrial fibrillation	28 (74)	34 (97)	0.005
Atrial flutter	10 (26)	1 (3)	0.005
<i>Medication prior to ECV, n (%)</i>			
ACEI or ARB	23 (61)	21 (60)	0.96
ARNI	2 (5)	6 (17)	0.14
MRA	23 (61)	30 (86)	0.020
<i>Diuretics</i>			
Loop	36 (95)	35 (100)	0.49
Tolvaptan	6 (16)	11 (31)	0.11
Pimobendan	2 (5)	3 (9)	0.58
Beta-blocker	33 (87)	34 (97)	0.20
SGLT2i	7 (18)	11 (31)	0.20
Verapamil	0 (0)	1 (3)	0.48
Digitalis	15 (40)	11 (31)	0.47
<i>Anticoagulant</i>			
Heparin	4 (11)	3 (9)	0.78
Vitamin K antagonist	4 (11)	3 (9)	0.78
Direct oral anticoagulant	30 (79)	29 (83)	0.67
<i>Periprocedural AAD, n (%)</i>			
The absence of AAD	9 (23)	12 (34)	0.31
Class I	2 (5)	2 (5)	0.93
Amiodarone	10 (26)	12 (34)	0.45
Bepidil	21 (55)	11 (31)	0.039
<i>Pre-cardioversion TEE findings</i>			
LAA inflow velocity, cm/s	37 (20–49)	23 (16–39)	0.19
LAA emptying velocity, cm/s	29 (20–40)	24 (18–33)	0.25
<i>Spontaneous echo contrast grade</i>			
≥ 2	5 (17)	11 (31)	0.25

Values are expressed as the median (interquartile range) or number (%)

AAD, antiarrhythmic drug; ACEI, angiotensin converting enzyme inhibitors; ARB, angiotensin II receptor blocker; ARNI, angiotensin receptor–neprilysin inhibitor; LAA, left atrial appendage; MRA, mineral corticoid receptor antagonist; SGLT2i, sodium glucose co-transporter-2 inhibitor. TEE, transesophageal echocardiography. The other abbreviations are shown in Table 1

Table 3 Clinical outcomes

	All (<i>n</i> = 73)	Early ECV (<i>n</i> = 38)	Delayed ECV (<i>n</i> = 35)	<i>P</i> value
<i>Primary endpoint, n (%)</i>				
The very short-term failure of ECV	39 (60)	19 (51)	20 (63)	0.87
The short-term failure of ECV	47 (67)	23 (61)	24 (72)	0.43
Recurrence of AAs	36 (61)	17 (54)	19 (68)	0.34
<i>Secondary endpoint, n (%)</i>				
Acute success rate	62 (85)	32 (84)	30 (86)	0.86
The number of attempted ECV ≥ 3	10 (14)	6 (16)	4 (11)	0.59
Maximum energy delivered for the successful ECV, J	100 (100–150)	150 (100–150)	100 (100–150)	0.47
Any complication	6 (8)	3 (8)	2 (6)	0.71
Bradycardia (< 40 bpm)	2 (3)	0 (0)	2 (6)	0.14
Hypotension	4 (5)	3 (8)	1 (3)	0.35
Thromboembolism	0 (0)	0 (0)	0 (0)	N/A
Worsening HF	1 (1)	1 (3)	0 (0)	0.33
Asystole (> 5 s)	1 (1)	0 (0)	1 (3)	0.29
Ventricular fibrillation	0 (0)	0 (0)	0 (0)	N/A
The change of TTE parameters (<i>n</i> = 37)				
The ECV to follow-up TTE time interval, days	11 (5–30)	20 (6–37)	7 (2–21)	0.08
Δ LVEF, %	6 (2–17)	6 (1–20)	7 (3–15)	0.90
Δ LVEDV, mL	–6 (–17 to 15)	–7 (–17 to 12)	–7 (–21 to 17)	0.70
Δ LVESV, mL	–11 (–26 to 2)	–19 (–30 to 0)	–7 (–25 to 3)	0.25
Δ LAVI, ml/m ²	–6 (–17 to 3)	–5 (–14 to 2)	–9 (–20 to 3)	0.35
$\Delta E/e'$ average	–2 (–5 to 2)	–2 (–5 to 3)	–2 (–6 to 3)	0.92
Hospitalization duration, days	16 (14–23)	14 (12–21)	17 (15–26)	<0.001

Values are expressed as the median (interquartile range) or number (%)

AAs, atrial arrhythmias; J, joule; N/A, not applicable. The abbreviations are shown in Tables 1 and 2

Endpoints

The clinical outcomes are presented in Table 3. ECV successfully restored SR in 62 of 73 patients (85%) at a mean energy level of 100 J. In 10 patients (14%), multiple ECV attempts (≥ 3) were needed. Periprocedural complications occurred in six patients (8%). Among them, the majority were temporal bradycardia and hypotension, which were resolved in all patients without pacemakers. Furthermore, no stroke occurred and worsening HF occurred in only one patient (1%). No significant between-group differences in endpoints were observed. Very short-term failure of ECV (unsuccessful cardioversion and AAs recurrence during hospitalization) occurred in 39 patients (53%), without any significant between-group differences (51% early ECV vs. 63% delayed ECV, $P=0.87$) (Fig. 2A). The incidence of short-term ECV failure (unsuccessful cardioversion and AAs recurrence within 1 month) also did not differ significantly between the groups (61% early ECV vs. 72% delayed ECV, $P=0.43$) (Fig. 2B).

Among the 62 patients with acute successful ECV, 37 underwent transthoracic echocardiography (TTE) before and after cardioversion. The median days to follow-up TTE

after ECV was 11 days (IQR = 5–30 days). Among the 37 patients, the comparison of TTE findings on admission and those at follow-up are shown in Fig. 3. Left ventricular ejection fraction (LVEF) values significantly improved (38% [31–52] to 51% [39–63], $P=0.008$), and left atrial volume index decreased (LAVI) (48 ml/m² [37–57] to 38 ml/m² [29–61] ml/m², $P=0.07$) from admission to follow-up. There were no differences in the echocardiographic parameters when values of early and delayed TTE groups were compared (Table 3). There were no between-group differences in TTE-related parameters observed when recurrence and non-recurrence groups were compared, except follow-up TTE was earlier in the non-recurrence versus recurrence group (6 days [3–21] vs. 30 days [8–42] days, $P=0.008$; Table 4).

The hospitalization duration was shortened in the early ECV group versus that of the delayed group (14 days [11–21] vs. 17 days [15–26], $P<0.001$) (Table 3). The length of hospital stay was also correlated with days to ECV during hospitalization (Spearman's $\rho=0.47$, $P<0.001$) (Fig. 4). After adjusting for all covariates associated with hospitalization duration in univariate analyses, early ECV, heart rate just before ECV, and tolvaptan

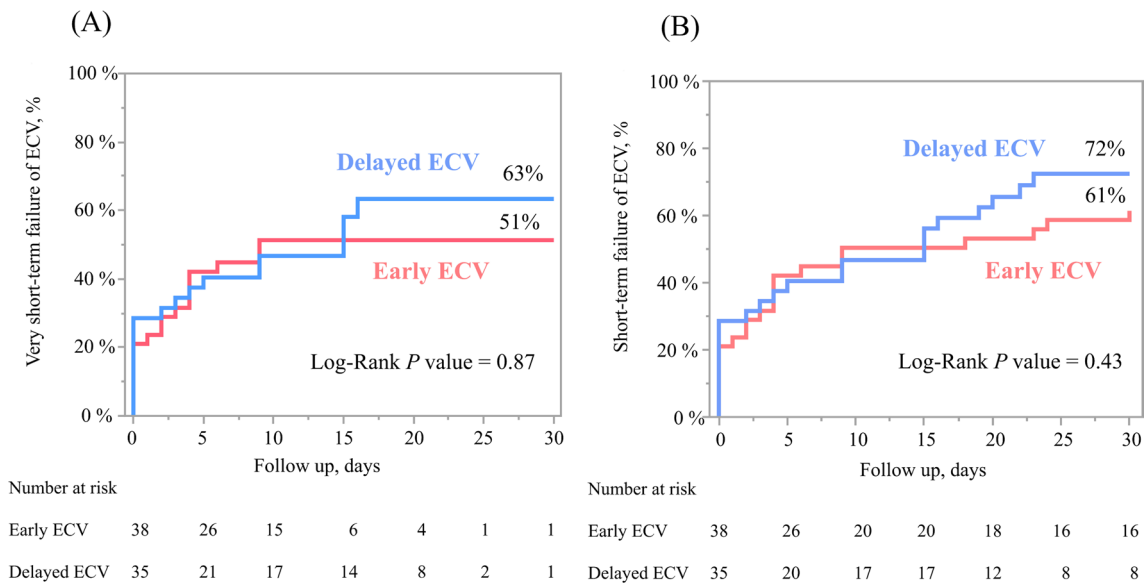
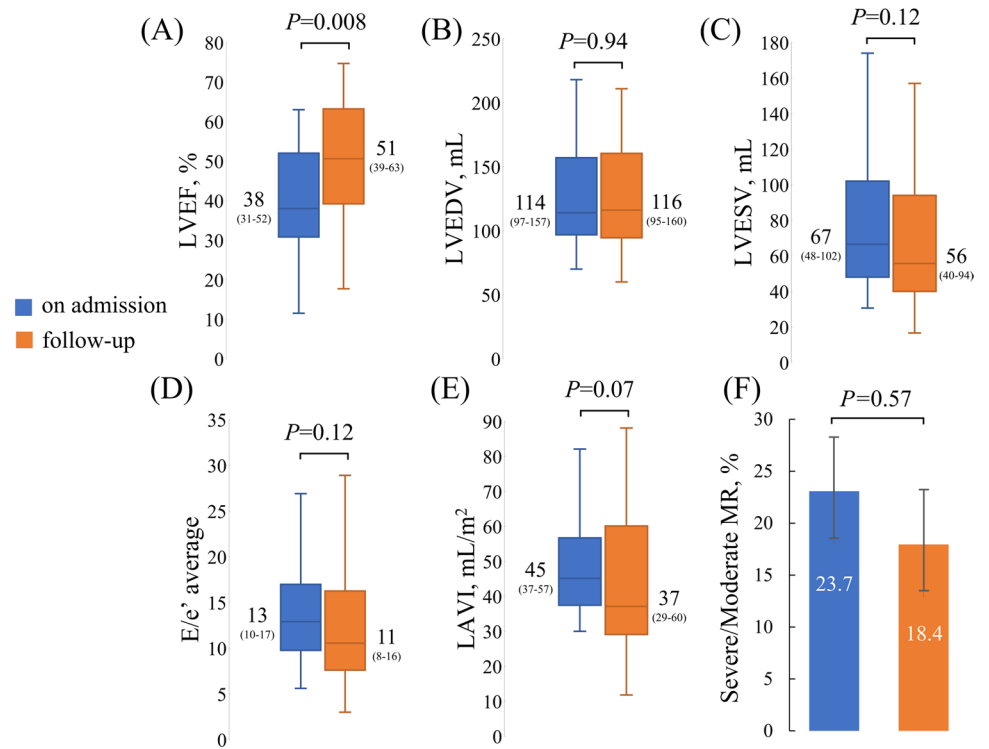


Fig. 2 Kaplan–Meier curves for very short-term (A) and short-term (B) failures of electrical cardioversion. *ECV* electrical cardioversion

Fig. 3 The change of transthoracic echocardiographic findings following cardioversion. Values are expressed as the median (interquartile range) or number (%). *LAVI* left atrial volume index, *LVEF* left ventricular ejection fraction, *LVEDV* left ventricular end-diastolic volume, *LVESV* left ventricular end-systolic volume, *MR* mitral regurgitation



administration were identified as independent determinants of a shortened duration of hospitalization (Table 5).

After adjusting for all covariates with univariate values of $P < 0.05$, AF was identified as an independent positive

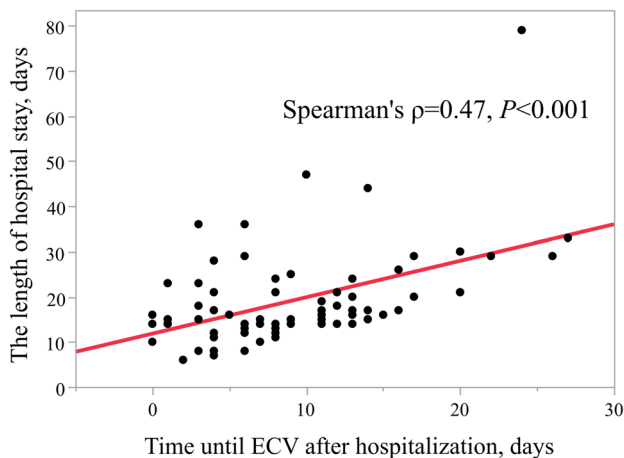
predictor of the short-term failure of ECV (OR 11.0, 95% CI 1.40–87.3, $P = 0.023$) (see Online Resource Table S1).

Conversely, AFL was an independent negative predictor

Table 4 Comparison of echocardiographic findings between patients with and without recurrence of atrial arrhythmias after electrical cardioversion

	No recurrence (<i>n</i> = 23)	Recurrence (<i>n</i> = 14)	<i>P</i> value
The time interval from admission to follow-up TTE, days	6 (3–21)	30 (8–42)	0.008
ΔLVEF, %	6 (1–17)	9 (3–20)	0.43
Improvement of LVEF > 10%	9 (39)	6 (43)	0.82
ΔLVEDV, mL	−5 (−15 to 19)	−8 (−17 to 13)	0.62
ΔLVESV, mL	−9 (−25 to 3)	−12 (−32 to 1)	0.32
ΔLAVI, ml/m ²	−6 (−18 to 2)	−7 (−17 to 3)	0.93
ΔE/e' average	−1 (−6 to 4)	−2 (−3 to 0)	0.86

Values are expressed as the median (interquartile range) or number (%). The abbreviations are shown in Tables 1, 2, and 3

**Fig. 4** Correlation between the length of hospital stay and time until electrical cardioversion during hospitalization. *ECV* electrical cardioversion

of short-term ECV failure via multivariate analysis (OR 0.09, 95% CI 0.01–0.72, *P* = 0.023).

Discussion

This study revealed that among the patients hospitalized due to ADHF with concomitant AAs, (1) ECV successfully restored SR in 85% with a median number of ECV attempts of 1 and periprocedural complication rate of 8%; (2) short-term failure of ECV, defined as the combination of unsuccessful cardioversion and AA recurrence within 1 month after ECV, occurred in 67% of patients; (3) LVEF improvement and LAVI decrease were observed during a median follow-up period of 11 days; and (4) early ECV resulted in the shortest duration of hospitalization.

ECV is effective rhythm control therapy for AF/AFL management in the restoration of SR, with an overall high success rate of 70–90% [3, 5, 15]. However, AA recurrence is common, especially in those with congestive HF, which

is reported to be a powerful predictor of unfavorable short-term outcomes after ECV [11, 16]. In clinical practice, the optimal timing of ECV in patients with ADHF and AAs has not been clarified. A previous multicenter randomized trial investigating the impact of ECV timing on the safety and effectiveness of the procedure in patients with recent-onset symptomatic AF without HF has been performed [12]. In the study, the wait-and-see strategy was non-inferior to early cardioversion for restoring SR at 4 weeks after the index visit. As for AF patients with left ventricular dysfunction (LVEF ≤ 45%), the ECV acute success rate was reported to be strongly associated with the degree of HF pharmacological therapy before ECV [15]. Considering these findings, the optimal timing of ECV in patients with HF and AAs might be different from that of those without HF. Our data suggest that early ECV is associated with a short hospitalization duration. Several previous studies have demonstrated the usefulness of ECV in terms of its ability to rapidly improve LV function, quality of life, and NYHA functional class [5, 17]. Consistent with findings from previous studies, we showed that LVEF significantly improved and LAVI tended to decrease within 2 months of ECV in our population. The present study also demonstrated that the AA recurrence rate within 1 month after ECV was high (61%) despite a high acute ECV success rate (84%). Given these findings, CA should be recommended after ECV to facilitate the long-term maintenance of SR and improve HF prognosis [4]. Meanwhile, CA is associated with a potential risk of periprocedural complications, especially during the acute phase of HF. Patients who undergo CA have a higher volume overload burden during the procedure, causing periprocedural HF [18]. Congestive HF itself was also reported to be an independent predictor of periprocedural stroke [19]. In our population, critical periprocedural complications, including worsening HF and thromboembolism, were also rare, and the success rate of acute ECV was high. Therefore, early ECV may be useful for achieving temporary rhythm control, facilitating smooth and safe CA subsequently, by improving cardiac function and shortening hospital stay.

Table 5 Univariate and multivariate logistic regression analyses of factors associated with a longer hospital stay (more than 16 days)

	Univariate		Multivariate	
	Odds ratio (95% CI)	<i>P</i> value	Odds ratio (95% CI)	<i>P</i> value
Age (per 1 year increase)	1.02 (0.98–1.06)	0.31		
Male gender	0.50 (0.19–1.28)	0.15		
BMI (per 1 kg/m ² increase)	1.01 (0.92–1.10)	0.80		
Clinical frailty scale (per 1 point increase)	0.73 (0.50–1.10)	0.12		
New-onset AF	0.89 (0.34–2.28)	0.81		
NYHA class on admission (per 1 increase)	1.57 (0.77–3.22)	0.21		
SBP on admission (per 1 mmHg increase)	1.00 (0.98–1.02)	0.81		
Heart rate on admission (per 1 bpm increase)	1.01 (0.99–1.03)	0.14		
CHADS2-VASc score (per 1 point increase)	0.70 (0.44–1.13)	0.13		
Prior HF hospitalization	1.03 (0.35–3.04)	0.96		
Hemoglobin A1c (per 1% increase)	0.64 (0.36–1.14)	0.15		
Hemoglobin (per 1 g/L increase)	0.87 (0.69–1.08)	0.20		
eGFR (per 1 ml/min per 1.73m ² increase)	0.98 (0.96–1.00)	0.08		
Log [NT-proBNP] (per 1.0 increase)	8.05 (1.41–45.9)	0.016	9.06 (0.92–89.1)	0.06
Tachycardia induced cardiomyopathy	0.71 (0.28–1.81)	0.47		
NYHA class just before ECV (per 1 increase)	0.96 (0.59–1.56)	0.88		
SBP just before ECV (per 1 mmHg increase)	1.00 (0.98–1.03)	0.78		
Heart rate just before ECV (per 1 bpm increase)	1.02 (1.00–1.04)	0.029	1.03 (1.00–1.06)	0.024
Atrial fibrillation rhythm just before ECV	1.05 (0.29–3.81)	0.93		
Atrial flutter rhythm just before ECV	0.95 (0.26–3.43)	0.93		
Beta-blocker prior to ECV	1.82 (0.31–10.7)	0.49		
ACE/ARB prior to ECV	0.56 (0.22–1.45)	0.23		
ARNI prior to ECV	1.17 (0.27–5.07)	0.83		
Loop diuretics prior to ECV	N/A	N/A		
MRA prior to ECV	2.61 (0.87–7.84)	0.09		
Tolvaptan prior to ECV	8.40 (2.15–32.7)	0.002	6.23 (1.22–31.7)	0.028
SGLT2i prior to ECV	0.24 (0.06–0.76)	0.014	0.25 (0.06–1.12)	0.07
Digitalis prior to ECV	1.58 (0.60–4.12)	0.35		
Periprocedural AAD	1.38 (0.50–3.83)	0.53		
Amiodarone prior to ECV	1.14 (0.36–3.70)	0.82		
Bepidil prior to ECV	0.69 (0.24–1.97)	0.49		
LVEF on admission (per 1% increase)	0.98 (0.95–1.02)	0.36		
LVEDV on admission (per 1 mL increase)	1.00 (0.99–1.00)	0.38		
LAVI on admission (per 1 mL/m ² increase)	1.01 (0.99–1.04)	0.34		
<i>E/e'</i> average on admission (per 1 increase)	1.03 (0.95–1.11)	0.43		
Moderate/Severe mitral regurgitation on admission	2.32 (0.82–6.58)	0.11		
LAA inflow velocity (per 1 cm/sec increase)	1.00 (0.98–1.03)	0.73		
Spontaneous echo contrast grade (per 1.0 increase)	1.58 (0.94–2.63)	0.07		
Early ECV	0.27 (0.10–0.72)	0.009	0.13 (0.03–0.50)	0.032

CI, confidence interval. The abbreviations are shown in Tables 1, 2, and 3

Our additional analysis showed that AF is an independent positive predictor of short-term ECV failure, but vice versa in AFL. AF and AFL commonly overlap due to their shared precipitants and risk factors [2, 20]. In addition, AF/AFL is a common arrhythmia associated with a cause or consequence of HF, which leads to a worsened prognosis [2]. Data related to AF/AFL are scarce, especially regarding

AFL treatment strategies. Further, a serious issue in which HR control with landiolol was difficult in those with AFL [2, 3, 20]. The present study showed that physicians attempted ECV earlier in patients with AFL than in those with AF, possibly due to difficulty controlling HR. Furthermore, another study reported that ECV more effectively controlled rhythm in new-onset AFL than in new-onset AF [16]. These

results indicate that ECV is a reasonable strategy for treating patients with HF and AFL. Our study also showed that HR just before ECV was a determinant of long hospital stay, a finding that agrees with the suggestion of a previous study [20, 21]. Among patients with HF with uncontrolled rapid HR regardless of AAs, early ECV may be more beneficial than continuing to rely on medication.

This study had several limitations. First, this was a retrospective and single-center study; therefore, ECV were not randomly assigned. Physician discretion limits the comparability of early and delayed ECV. However, application of the same ECV protocol to all patients with HF and AAs excluded as much bias as possible. In addition, using a consecutive series of patients in long-term observational ECV practice (> 5 years) minimizes selection bias and reflects contemporary clinical practice. Further randomized prospective trials are warranted to determine the optimal timing of ECV. Secondly, hospitalization duration variability may have been affected by unmeasured confounding variables. Third, the recurrence rate of AAs may have been underestimated due to failing to consider patients with asymptomatic recurrence. Nonetheless, we monitored ECG continuously after ECV as long as possible, that was possible because our population underwent ECV during hospitalization.

Conclusions

Early ECV was a major determinant of a short hospitalization duration, along with a significant increase in LVEF. Additionally, AFL was an independent negative predictor of short-term ECV failure. As a more effective treatment strategy for acute HF and AAs, early ECV in combination with standard pharmacological therapy may benefit patients, particularly in those with uncontrolled rapid HR due to rapid AFL.

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Data availability The data that support the findings of this study are available from the corresponding author upon reasonable request.

Declarations

Conflict of interest Y.O. has received lecture fees from Bayer Yakuhin and Daiichi-Sankyo; research funding from Bayer Yakuhin and Bristol-Myers Squibb; and scholarship grants from Bayer Yakuhin, Daiichi-Sankyo, and Johnson & Johnson.

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