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ORIGINAL PAPER

Predictors of left atrial appendage stunning after electrical cardioversion in patients with atrial fibrillation

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Abstract

The transient left atrial appendage (LAA) dysfunction after electrical cardioversion (CV), which is called as LAA-stunning, was found to be an important etiology of thrombus formation. The aim of the present study was to investigate the risk factors of LAA-stunning. This study included 134 patients who underwent catheter ablation for non-paroxysmal, non-valvular, and symptomatic atrial fibrillation (AF). Internal-CV was performed, and LAA emptying fraction (LAA-EF) was assessed using LAA-angiogram before and just after CV. LAA-stunning (defined as 10% reduction of LAA-EF after CV) was observed in 45/134 patients (34%). Patients in LAA-stunning group had longer duration of AF prior to CV, higher brain natriuretic peptide (BNP), higher prevalence of patients taking calcium blocker, larger left atrial (LA) diameter, elevated E wave, and larger LA volume than those in non LAA-stunning group. Multivariate analysis showed that longer duration of AF prior to CV ($p=0.015$, OR 1.033 for 1 month extend, 95% CI 1.006–1.073) and elevated BNP ($p=0.038$, OR 1.041 for each 10 pg/mL increase, 95% CI 1.001–1.009) were associated with LAA-stunning. In addition, all patients were divided into four groups based on the combination between duration of AF prior to CV and BNP; group 1 (low BNP/short-lasting AF), group 2 (high BNP/short-lasting AF), group 3 (low BNP/long-lasting AF), and group 4 (high BNP/long-lasting AF). The rate of LAA-stunning was the highest in the group 4 (55.6%). Elevated BNP and long duration of AF were associated with LAA stunning after electrical cardioversion.

Keywords Left atrial appendage · Stunning · Atrial fibrillation

Introduction

For many years, electrical cardioversion (CV) has been commonly used to restore sinus rhythm in atrial fibrillation (AF) patients [1]. Recent guidelines recommend anticoagulation after CV to prevent thromboembolism (TE), regardless of sinus rhythm conversion. TE after CV is a rare but often devastating complication. Recently, Garcia-Fernandez et al. studied the long-term incidence of TE after CV in 406 patients with AF [2]. Their study indicated that the incidence of TE in the first 30 days was 0.17% and annual incidence of TE was 1.9% even though patients with left atrial thrombus were excluded.

The left atrial appendage (LAA) is known to be a major source of cardiac thrombi in patients with AF. Consequent to the development of the TEE, transient LAA dysfunction after CV known as LAA-stunning, was found to be an important etiology of thrombus formation [3, 4]. Melduni et al. reported severe LAA-stunning after electrical CV [5]. Their case report showed newly formed LAA thrombi immediately after CV despite prior confirmation that no LAA thrombus had been present. Patients with LAA-stunning may require aggressive anticoagulant therapy during periprocedural period to reduce the risk of TE. However, predictive factors for the occurrence of LAA-stunning after CV have not been well studied. The aim of the present study is to investigate the risk factors of LAA-stunning after CV.

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Methods

Patient population

This study included 160 patients who underwent CA for non-paroxysmal, non-valvular, and symptomatic AF at the Hyogo College of Medicine between November 2014 and July 2017. From this initial population, patients who underwent cardiac surgery (n = 3), patients with decreased renal function (n = 3, eGFR < 30 mL/min/1.73 m^2), poor-quality LAA angiography images (n = 6), immediate recurrence of AF after CV (n = 3), LAA akinesis (defined as emptying fraction of LAA: LAA-EF < 10% in angiogram, n = 21), or Sinus rhythm before CV (n = 3) were excluded. The remaining 134 patients (103 males, 68 ± 9 years) were included in present study analyses. Antiarrhythmic drugs were discontinued for at least five half-lives before CA. All patients were older than 18 years and provided written informed consent to the procedures. The research protocol was approved by the appointed local ethics committee.

Echocardiographic imaging and computed tomography

All patients underwent transthoracic echocardiography (TTE) within a month before the CA procedure using a Prosound F75 (Hitachi Aloka Medical, Japan) with a 3.88-MHz transducer probe. Left atrial (LA) diameter, E wave, E/e′ ratio, left ventricular (LV) end-diastolic diameter, LV ejection fraction, and the prevalence of LV hypertrophy were assessed during TTE.

All patients underwent ECG-gated 64-slice multidetector CT (SOMATOM Definition Edge, Siemens, Germany) with slice acquisition thickness set at 0.5 mm. An independent workstation (Ziostation version 2.1, Ziosoft, Japan) was used for analysis. Three-dimensional structures of the LA and LAA were constructed using the volume-rendered postprocessing technique. In all cases, 40–70 mL of intravenous contrast agent (Imeron350, Bracco Imaging, Germany) was injected at 5 mL/s, followed by a 30 mL saline bolus at 5 mL/s. CT images of the LA, LAA, and pulmonary vein were reconstructed at a separate workstation and integrated using EnSite Verismo (Abbott Medical, US). LAA volume was obtained using the EnSite Verismo. The LAA orifice was manually selected as the narrowest portion at the entrance into the LA. The LAA anatomic characteristics were evaluated using a previous classification [6]. All patients in our study were classified into Two types using CCT. Chicken-wing (CW) type was defined as LAA with an obvious bend in the proximal or middle part of the dominant lobe, or the LAA folding back on itself at some distance from the perceived LAA orifice. Non-CW type was defined as LAA without any bends. We classified LAA shapes into 2 objective types: CW type and non-CW type. The LAA morphology was classified by 2 cardiologists who were blinded to the clinical data. Disagreement was resolved by consensus.

Left atrial appendage angiogram

Cardiac catheterization and CA for AF were performed as described previously [7]. Briefly, bipolar electrograms recorded for 5 s were processed off-line using EP-WorkMate (Abbott Medical). LAA voltage and cycle length during AF were measured using ablation catheter (FlexAbility, Abbott Medical) at the 2 sites of LAA. LAA voltage and cycle length were defined as the mean of 10 consecutive electrograms. Bandpass filtering with cutoffs were 40 and 250 Hz.

LAA angiogram was obtained by injection of contrast agent into LAA through a long sheath (8-Fr SL0; Abbott Medical) during AF rhythm (before CV) and sinus rhythm (after CV). LAA angiogram was also performed before CA procedure in all patients. The internal CV was performed using a 6-Fr 8 + 4 + 8 polar catheter (BeeAT, Japan Lifeline, Japan) placed into the coronary sinus and along the lateral wall of the right atrium via the right jugular vein. The CV energy was delivered until successful CV (5-30 J). We measured LAA-EF after maintaining a stable sinus rhythm at least 2 min after CV. The LAA-EF was calculated using the formula a LAA-EF (%) = 100 × (maximum area − minimum area)/(maximum area) in each rhythm by use of the NahriAQUA version 1.4.6.9 program (NEXIS, Japan). During sinus rhythm, the area of the LAA just before the P wave (maximum area) and at or just after the ECG QRS complex at the end of LAA systole (minimum area) were measured by the planimetric method from the top of the limbus of the upper left pulmonary vein, along the whole appendage-endocardial border systole and diastole (Fig. 1). An average of three EF measurements was used to determine the LAA-EF during sinus rhythm. During AF rhythm, LAA maximum/minimum areas were defined as the largest/smallest area of LAA among three consecutive beats because of variability of LAA contraction in AF rhythm. LAA-stunning was defined as more than 10% reduction of LAA-EF after CV. In other words, patients with LAA-stunning have lower LAA-EF during sinus rhythm (after CV) than LAA-EF during AF rhythm (before CV).

Statistical analysis

Continuous variables (e.g., age) are presented as mean value ± standard deviation and were compared using one-way analysis of variance (ANOVA). Categorical variables (e.g., gender) were compared using the χ^2 test or Fisher's

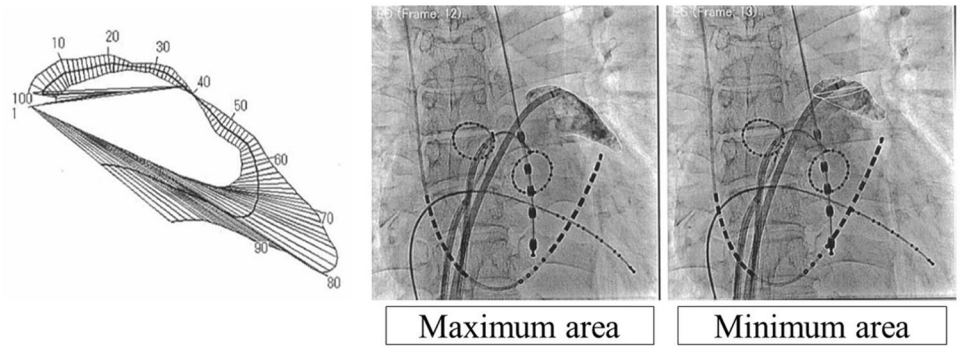

Fig. 1 Measurement of LAA-EF using a planimetry method in anteroposterior view of an LAA angiogram. EF = emptying fraction; LAA = left atrial appendage

exact test. A P value < 0.05 was considered statistically significant. The variables that were found to be significant in univariate analysis were entered into a multivariate analysis. The independent association with LAA-stunning after CV was evaluated using multiple logistic regression analysis. Interobserver agreement between readers was evaluated using Cohen's kappa. A good level of agreement was defined as $\kappa \geq 0.61$. The LAA-EFs were measured 3 times by one experienced cardiologist in all patients. The measurements of LAA-EFs showed < 5% intra-observer variability. All analyses were performed with the statistical software JMP pro version 10 software (SAS, Cary, NC, USA) and Microsoft Excel.

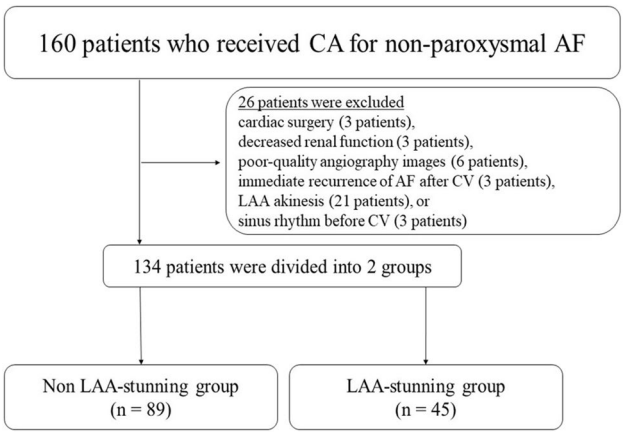

Fig. 2 Flow chart of the study patients. AF = atrial fibrillation; CA = catheter ablation; CV = cardioversion; LAA = left atrial appendage

Results

Patient population

Among 160 patients screened, 134 patients (age 68 ± 9 years, 103 males) were enrolled in the study according to the inclusion and exclusion criteria. Of the 134 patients, LAA-stunning was detected in 45 patients (33.6%). We divided all patients into two groups as follows: the LAA-stunning group (n = 45) and the Non LAA-stunning group (n = 89) based on LAA-EF using LAA angiogram (Fig. 2). All patients did not have complications of cardiac tamponade during CA procedure.

Baseline characteristics of the patients and baseline medication therapy are listed in Table 1. Duration of AF prior to CV was significantly longer in the LAA-stunning group than in the Non LAA-stunning group. Brain natriuretic peptide (BNP) was significantly higher in the LAA-stunning group than in the Non LAA-stunning group. There was no difference in the prevalence of structural heart disease between the LAA-stunning group and the Non LAA-stunning group. The rate of patients taking calcium blocker was significantly lower in LAA-stunning group than in the Non LAA-stunning group.

Echocardiographic and LAA findings

Table 1 lists the echocardiographic findings of the study groups. Patients in the LAA-stunning group had a significantly larger LA diameter and elevated E wave than those in the Non LAA-stunning group. Table 1 also lists the LAA findings of the study groups. All patients showed sinus rhythm after the CV. LA volume was significantly larger in the LAA-stunning group than in the Non LAA-stunning group. LAA morphology, LAA volume, LAA orifice area, and LAA orifice diameter did not differ between the LAA-stunning group and the Non LAA-stunning group. Moreover, LAA voltage and LAA cycle length before CV did not differ between the LAA-stunning group and the non LAA-stunning group.

Table 1 Baseline characteristics

	Without LAA-stunning group (n = 89)	With LAA-stunning group (n = 45)	P value
Age (years)	67 ± 9	69 ± 9	0.383
Male	69 (78%)	34 (76%)	0.798
Duration of AF prior to CV (months)	3.3 ± 11.6	12.5 ± 23.9	0.003
Brain natriuretic peptide (pg/mL)	117 ± 107	196 ± 161	0.001
C-reactive protein (mg/dL)	0.15 ± 0.28	0.09 ± 0.11	0.2
Serum creatinine (mg/dL)	0.77 ± 0.24	0.84 ± 0.19	0.095
Uric acid (mg/dL)	6.1 ± 1.4	6.0 ± 1.2	0.654
Hypertension	54 (61%)	23 (51%)	0.29
Dyslipidemia	23 (26%)	15 (33%)	0.364
Diabetes mellitus	17 (19%)	8 (18%)	0.853
Prior congestive heart failure	11 (12%)	9 (20%)	0.305
Prior stroke/transient ischemic attack	11 (12%)	8 (18%)	0.396
Vascular disease	62 (70%)	30 (67%)	0.724
Structural heart disease	17 (19%)	10 (22%)	0.671
$CHADS_2$ score	1.4 ± 1.2	1.6 ± 1.4	0.431
CHA_2DS_2-VASc score	2.5 ± 1.6	2.7 ± 1.9	0.535
Medication therapy			
β blocker	45 (51%)	27 (60%)	0.301
ACE-I	8 (9%)	5 (11%)	0.761
ARB	25 (28%)	7 (16%)	0.135
Calcium channel blocker	35 (39%)	9 (20%)	0.022
Digitalis	3 (3%)	2 (4%)	1
Diuretics	9 (10%)	5 (11%)	1
Antiarrhythmic drug	33 (37%)	13 (13%)	0.346
Echocardiographic findings			
LA diameter (mm)	42 ± 6	45 ± 6	0.002
E wave (cm/s)	75 ± 25	87 ± 20	0.007
E/e′ ratio	11 ± 5	12 ± 4	0.653
LV end-diastolic diameter (mm)	49 ± 5	49 ± 4	0.714
LV ejection fraction (%)	65 ± 12	64 ± 8	0.797
LV hypertrophy	5 (6%)	4 (9%)	0.484
CT findings			
LA volume (mL)	119 ± 34	145 ± 32	0.001
Chicken-wing type LAA	72 (81%)	37 (82%)	0.853
LAA volume (mL)	10.8 ± 4.6	12.3 ± 5.4	0.164
LAA orifice area (cm^2)	6.0 ± 2.5	6.3 ± 2.4	0.429
LAA orifice Dmax (mm)	26.1 ± 6.6	26.0 ± 6.9	0.947
LAA orifice Dmin (mm)	17.2 ± 4.9	18.2 ± 5.3	0.287
Procedural findings			
LAA voltage (mV)	1.5 ± 1.0	1.4 ± 0.9	0.856
LAA cycle length (ms)	171 ± 35	168 ± 30	0.642

Values are given as no. (%) or mean ± SD

ACE-I angiotensin-converting enzyme inhibitor, *AF* atrial fibrillation, *ARB* angiotensin receptor blocker, *CT* computed tomography, *CV* cardioversion, *Dmax* maximum diameter, *Dmin* minimum diameter, *LA* left atrial, *LAA* left atrial appendage, *LV* left ventricular

Univariate and multivariate analyses

Univariate and multivariate analyses are described in Table 2. Univariate analysis revealed a longer duration of AF prior to CV, higher BNP, taking calcium channel blocker, larger LA diameter, elevated E wave, and larger LA volume as significant variables. On multivariate analysis, longer duration of AF prior to CV and higher BNP

Table 2 Univariate and multivariate analyses

	Univariate analysis	Multivariate analysis	
	P value	P value	OR (95% CI)
Duration of AF prior CV	0.003	0.015	1.033† (1.006–1.073)
BNP	0.001	0.038	1.041* (1.001–1.009)
Calcium channel blocker	0.022	0.108	
E wave	0.007	0.068	
LA volume	0.001	0.126	

AF atrial fibrillation, *BNP* brain natriuretic peptide, *CI* confidence interval, *CV* cardioversion, *LA* left atrial, *OR* odds ratio

*For each 10 pg/mL increase in the BNP

†For each 1 month extend in the duration of AF prior CV

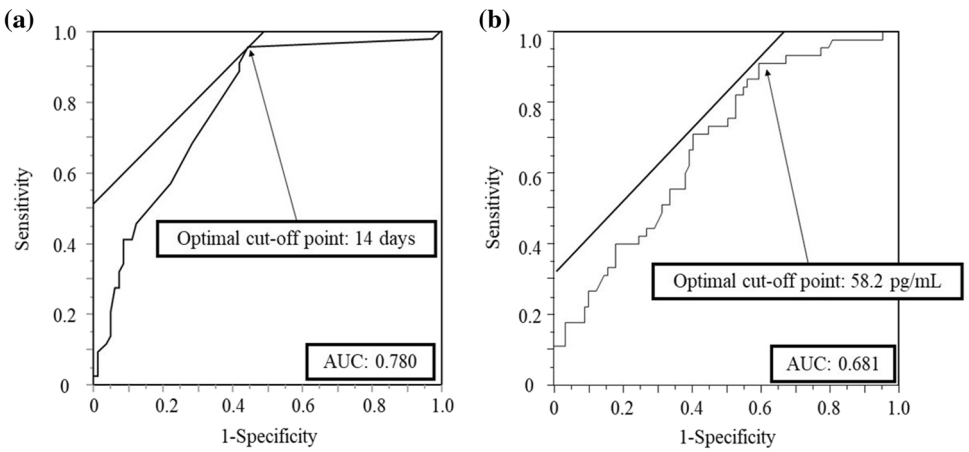

Fig. 3 The ROC curve analysis. **a** The AUC was 0.780, and the optimal cut-off value of the duration of AF prior to CV to predict the LAA-stunning was 14 days. **b** The AUC was 0.681, and the optimal cut-off value of the BNP to predict the LAA-stunning was 58.2 pg/mL. AF = atrial fibrillation; AUC = area under the receiver-operating-characteristic curve; BNP = brain natriuretic peptide; CV = cardioversion; LAA = left atrial appendage; ROC curve = receiver-operating-characteristic curve

were independently associated with LAA-stunning after CV. LA diameter was not entered into a multivariate analysis because LA diameter is equivalent to LA volume.

The ROC curve analysis is described in Fig. 3. The area under the ROC curve (AUC) was 0.780, and the optimal cut-off value of the duration of AF prior to CV to predict LAA-stunning was 14 days. The AUC was also 0.681, and the optimal cut-off value of the BNP to predict the LAA-stunning was 58.2 pg/mL. In addition, all patients were divided into four groups based on the combination between duration of AF prior to CV (cutoff value; 14 days) and BNP (cutoff value; 58.2 pg/mL); group 1 (low BNP/short-lasting AF, n = 27), group 2 (high BNP/short-lasting AF, n = 19), group 3 (low BNP/long-lasting AF, n = 16), and group 4 (high BNP/long-lasting AF, n = 72). The prevalence of LAA-stunning was 3.7, 5.3, 18.8, and 55.6% in group 1, 2, 3, and 4, respectively (Fig. 4). The rate of LAA-stunning was the highest in the group 4.

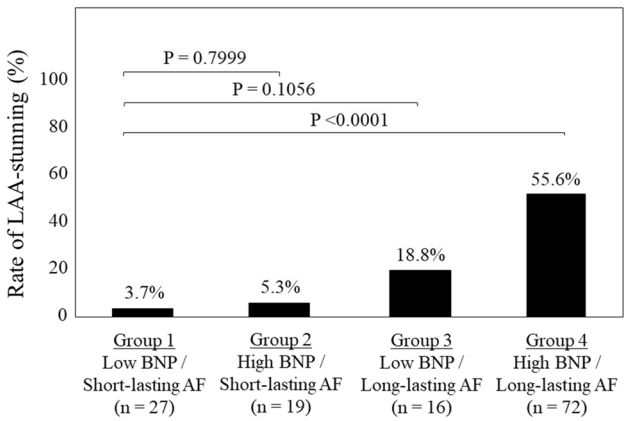

Fig. 4 The rate of patients with LAA stunning. Four groups based on BNP levels and duration of AF prior to CV. AF = atrial fibrillation; BNP = brain natriuretic peptide; CV = cardioversion; LAA = left atrial appendage

Discussion

Main findings

We investigated the various factors associated with LAA-stunning after CV in patients with AF using a single-center database. Our study showed that a longer duration of AF prior to CV and elevated BNP were associated with LAA-stunning on multivariate analysis. Moreover, when all patients were classified into 1 of 4 groups based on various combinations of duration of AF prior to CV (cutoff value; 14 days) and BNP (cutoff value; 58.2 pg/mL), the prevalence of LAA-stunning was highest in the high BNP/long-lasting AF group.

Left atrial appendage stunning

As shown in previous studies, the prevalence of LAA-stunning after CV varies from 38% to 80% [8]. LAA stunning is a transient depression of LAA mechanical function after rhythm control of AF compared with pre-CV state. In this regard, previous studies have proposed several mechanisms [9]. The suggested mechanisms of stunning include tachycardia-mediated atrial cardiomyopathy, cytosolic calcium alterations in atria, atrial hibernation, and atrial fibrosis. However, the exact mechanism underlying LAA stunning has yet to be elucidated. LAA dysfunction may predispose to local thrombosis and systemic embolization, and early evaluation of LAA function after rhythm control could reduce the risk of TE after CA. Recently, Adderley et al. determined the rate of stroke or transient ischemic attack in 11,159 patients with a diagnosis of "resolved" AF compared to 15,059 patients with "unresolved" AF and 22,266 patients without AF [10]. Their results showed that patients with resolved AF remain at a higher risk of a stroke or transient ischemic attack than patients without AF (12.1 and 7.4 per 1000-person years in patients with resolved AF and no AF, respectively). Moreover, they concluded that guidelines should be updated to advocate continued use of anticoagulants in patients with resolved AF. Sustained LAA-stunning after rhythm control may explain their findings. In another study, Ammer et al. investigated LAA-stunning duration and timing of recovery of LAA function post CV in 50 patients with recent onset AF [11]. They demonstrated that partial recovery of stunning occurred after 15–30 days, and full recovery occurred 30–90 days post CV. Therefore, patients with LAA-stunning may require aggressive anticoagulant therapy during blanking period regardless of the absence of recurrent AF. Moreover, determining the risk factors of LAA-stunning may play an important role in risk stratification of TE after rhythm control. The results of our study showed LAA-stunning was detected in 33.6% patients, and a longer duration of AF prior to CV and elevated BNP were associated with LAA-stunning on multivariate analysis. Previous studies have been reported that the duration of AF before conversion to sinus rhythm strongly influences the extent of LA stunning. Manning et al. examined the relationship between duration of AF prior to CV and the time course of the recovery of stunning after CV in 60 patients with chronic AF [12]. The duration of AF was classified as brief (≤ 2 weeks in 17 patients), moderate (> 2 weeks but ≤ 6 weeks in 22 patients), or prolonged (> 6 weeks in 21 patients). They demonstrated that the AF duration had a significant effect on the severity of the stunning, and a brief duration AF (≤ 2 weeks) was associated with a less severe LA stunning. Similarly, other studies suggested that the duration of AF was one of the strongest factors influencing the recovery/severity of stunning [13–15]. Elevated BNP is surrogate marker of LA and LV stretch. However, no study to date has established the association between BNP and LAA stunning. In our study, Elevated E wave is associated with LAA-stunning. Our findings indirectly suggest that LA stretch cause LAA-stunning. Given the current data, further investigation is necessary to determine the validity of these mechanisms.

Clinical implications

Predicting LAA-stunning before CV may be important to prevent CV-related TE. To the best of our knowledge, this is the first study illustrating that patients with elevated BNP (> 58.2 pg/mL)/long duration of AF prior to CV (> 14 days) had a higher risk of LAA-stunning than the other groups. These patients may require aggressive anticoagulant therapy, such as warfarin (target PT-INR: 2–3) or high dose of dabigatran, during the periprocedural period. Moreover, pretreatment with some drugs (ARB, calcium channel blocker, and so on) may have a favorable effect by preventing LAA-stunning [9, 16, 17]. However, there is no data on thromboembolism or anticoagulation in our study. Investigation of the relationship of LAA stunning with thromboembolism is warranted in the future.

Limitations

There were several limitations in our study. First, this is a single center study with a small number of patients. Second, there were no strict criteria to exactly determine the duration of the AF. The physicians speculated the duration of AF based on medical interview and electrical cardiogram findings. Third, this study focused on a selected group of patients without LAA akinesis. Fourth, we evaluated LAA-EF using

the two-dimensional and one sectional area change. Fifth, we evaluated LAA-EF after internal CV. Therefore, our finding can apply to internal CV, not to external CV. Finally, medication therapy before the CA might also be a confounding variable.

Conclusions

Elevated BNP and long duration of AF were associated with LAA stunning after electrical cardioversion. Investigation of the relationship of LAA stunning with thromboembolism is warranted, as stronger anticoagulation therapy might be needed post electrical cardioversion in these patients.

Conflict of interest The authors declare no conflicts of interests.

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