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Research paper

Prevalence and risk factors associated with decompensated heart failure after successful elective cardioversion for atrial fibrillation and atrial flutter

Christina Healy, Palwinder Sodhi, Annabelle Barnett, Timothy Hess, Jennifer M. Wright *

University of Wisconsin School of Medicine and Public Health, Department of Medicine, Division of Cardiology, United States of America

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ABSTRACT

Study objective: To determine the incidence of and risk factors for HF after successful electrical and ablative cardioversion (CV) of atrial fibrillation (AF) and atrial flutter (AFL).
Design: Retrospective cohort study.
Setting: Single center academic institution.
Participants: Seven hundred fifty-five patients underwent successful elective CV from July 1, 2018 to May 20, 2019. Patients presenting in arrhythmias other than AF or AFL, those who developed HF due to alternative etiologies, and those who developed arrhythmia recurrence within 30 days were excluded. Medical records of the remaining 451 patients were reviewed before and after CV.
Main outcomes measured: Development of heart failure despite sinus rhythm following CV and the risk factors associated with this outcome.
Results: Thirty-three (7.3 %) of 451 patients who met inclusion criteria for our study developed new or worsening HF symptoms while maintaining sinus rhythm (SR) after successful CV. Symptoms were reported an average of 5.1 days following CV (range 0–17 days, SD 4.71). Following a multivariate stepwise logistic regression model, prior HF hospitalization (OR 3.91, 95 % CI 1.82–8.39), BMI (OR 1.06, 95 % CI 1.02–1.11), and valve disease (OR 2.51, 95 % CI 1.12–5.60) remained significant risk factors, and anti-arrhythmic drug (AAD) use was marginally significant (OR 2.02, 95 % CI 0.95–4.31).
Conclusion: Despite maintenance of SR, 7.3 % of patients developed decompensated HF in the 30 days following successful CV of AF or AFL, indicating this complication may be more frequent than previously believed. Predictors of HF post-CV included elevated BMI, valve disease, previous HF hospitalization, and prior AAD use.

1. Introduction

Elective cardioversion (CV) of atrial fibrillation (AF) and atrial flutter (AFL) is a common procedure to restore sinus rhythm (SR) in patients with uncontrolled rates or symptoms despite medical management. Embolic complications of cardioversion are frequently discussed in the literature and guidelines, however, the development of acute heart failure (HF) has also been documented as a complication after restoration to SR [1–5]. Despite this, there is relatively little known about its frequency and clinical indicators.

Available data suggest that HF within 48 h of electrical CV occurs at a rate of 3.9 % [2]. However, there is reason to believe that this underestimates the true frequency of post-CV HF as a complication. While the pathophysiology of post-CV HF is not fully understood, transiently decreased left atrial (LA) function is generally regarded as an important element in the development of pulmonary congestion [1,2,4]. Such LA "stunning" can onset immediately after electrical, ablative, or pharmacologic CV and has been shown to persist for days to weeks [6–14]. Indeed, the gradual recovery of atrial activity, which also plays a role in the development of thromboembolism after AF CV, was one influence on current guidelines recommending 4 weeks of uninterrupted anticoagulation after CV [15–17]. Considering this, post-CV HF may similarly remain a risk much longer than the 48 h after CV.

To our knowledge, no prior studies have offered insight into the true duration of risk for post-CV HF and its rate of complication. Better defining the frequency and risk factors of post-CV HF could ultimately lead to the development of risk calculators which inform management decisions and preventative strategies. In this study, we aim to identify the rate and clinical predictors of post-CV HF within 30 days of successful electrical and ablative CV of AF or AFL.

* Corresponding author at: 600 Highland Ave, Madison, WI 53792, United States of America.
E-mail address: jmwright@medicine.wisc.edu (J.M. Wright).

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2. Methods

In this single-center study, we performed a retrospective chart review and analysis of adult patients who underwent elective CV for AF or AFL from July 1, 2018 to May 20, 2019. This project did not require IRB review as it did not constitute research as defined by federal regulations and the primary intent of the project was quality improvement for patients at our institution undergoing CV. ICD-10 codes and CPT codes were used to identify patients with AF or AFL who underwent elective CV during the study period. Patients were included if they underwent successful elective CV for AF or AFL by ablation or direct current. Patients presenting in arrhythmias other than AF or AFL, those who developed arrhythmia recurrence within 30 days, those who did not have any subsequent clinical encounters at our institution after the procedure, and those who were in SR prior to ablations were excluded. The medical records of the remaining patients were reviewed before and after CV.

Post-CV HF was defined as new or worsening decompensated HF within 30 days of CV without alternative attributable etiology. Patients must have had explicit documentation of new or worsening decompensated HF in a clinical assessment, or prescription of a new or increased diuretic in response to one or more clinical findings suggestive of HF including: reported shortness of breath, swelling, or orthopnea; exam indicating hypoxia, peripheral edema, or elevated jugular venous distention; radiographic pulmonary edema, or elevated serum brain natriuretic peptide. Clinical documentation, patient correspondence, labs, and diagnostic studies from patient encounters were carefully reviewed for indicators of alternative etiologies for decompensation such as dietary or medication non-adherence, acute coronary syndromes, arrhythmias, among others. Patients who presented with symptomatic HF prior to CV but did not have worsening of symptoms post-procedurally were not considered to have met our primary outcome. The duration of time between the procedure and initial post-CV symptoms was recorded.

In addition to collecting demographic data of our study population, we used knowledge from prior research studies to identify and extract other clinical variables which we hypothesized would be associated with development of HF after successful CV (Tables 1–2). When available, echocardiographic parameters were collected from the three years prior to CV. For the purposes of analysis and clinical simplicity, duration of arrhythmias prior to CV was categorized into four subgroups: <30 days, 30 days to six months, six months to one year, and one year or longer. BMI was also treated as a categorical variable. Patients were considered to have valve disease if they had a known history of moderate or severe stenosis or insufficiency of any of the four valves.

2.1. Statistical analysis

Wilcoxon rank sum tests were employed for continuous variable and Chi-square or Fisher exact test were used for binary and categorical variable to compare variables between the case and control groups. A p-value <0.05 was considered statistically significant. Stepwise logistic regression modelling was applied to all measured covariates using a 0.05 significance level for both model entry and model exit in order to identify clinical predictors of post-CV HF after adjustment for other predictors. Based on prior studies suggesting its clinical relevance 'arrhythmia duration' was forced into the model. Additionally, due to greater than half of patients lacking data for pre-procedural pulmonary artery systolic pressure (PASP), this variable was not included in multivariate modelling as it would have significantly reduced the number of patient cases available for study. Statistical analysis was performed using SAS v9.4 (SAS Institute, Cary, NC).

3. Results

Of 755 cases reviewed, 451 were determined to meet inclusion

Table 1
Selected characteristics and association with post-CV HF.

Selected clinical features and association with post-CV HF	Asymptomatic ($N = 418$)	Post-CV HF ($N = 33$)	p-Value
Age, mean ± SD (range)	65.4 ± 12.3 (22–95)	67.1 ± 12.0 (39–89)	0.45
Female sex, n (%)	126 (30 %)	16 (48 %)	0.03
BMI, mean ± SD (range)	32.7 ± 8.1 (17.8–64.4)	38.0 ± 10.4 (21.5–61.5)	< 0.01
CAD, n (%)	101 (24 %)	10 (30 %)	0.43
OSA, n (%)	155 (37 %)	16 (48 %)	0.19
CPAP use, n (%)	52 (12 %)	8 (24 %)	0.05
CVA, n (%)	22 (5 %)	3 (9 %)	0.35
Diabetes, n (%)	97 (23 %)	8 (24 %)	0.89
Hypertension, n (%)	278 (67 %)	28 (85 %)	0.03
Pulmonary hypertension, n (%)	22 (5 %)	7 (21 %)	< 0.01
Peripheral vascular disease, n (%)	17 (4 %)	1 (3 %)	0.77
Valve disease, n (%)	73 (17 %)	12 (36 %)	< 0.01
Prior HF hospitalization, n (%)	97 (23 %)	18 (55 %)	< 0.01
CHA_2DS_2-VASc score, mean ± SD (range)	2.6 ± 1.7 (0–8)	3.1 ± 1.3 (1–7)	0.08
Medications at time of CV, n (%)			
AAD[a]	138 (33 %)	17 (52 %)	0.03
ACE/ARB/ARNI	191 (46 %)	16 (48 %)	0.76
BB/CCB	345 (83 %)	29 (88 %)	0.43
Digoxin	3 (0.7 %)	0 (0 %)	1.0
Diuretic	197 (47 %)	23 (70 %)	0.01
Arrhythmia type, n (%)			
Atrial fibrillation	308 (74 %)	20 (61 %)	0.17
Atrial flutter	92 (22 %)	12 (36 %)	
Mixed	18 (4 %)	1 (3 %)	
AF/AFL duration, n (%)			
<30 days	190 (45 %)	19 (58 %)	0.49
30 days–6 months	145 (35 %)	11 (33 %)	
6 months–1 year	31 (7 %)	2 (6 %)	
1+ years	40 (10 %)	1 (3 %)	
Unknown	12 (3 %)	0 (0 %)	
Method of cardioversion, n (%)			
Electrical	372 (89 %)	28 (85 %)	0.47
Ablation	46 (11 %)	5 (15 %)	

[a] AAD included VW Class IA (procainamide, disopyramide), class IC (flecainide, propafenone), class III (dofetilide, dronedarone, amiodarone, sotalol, ibutilide).

Table 2
Pre-CV echocardiographic measurements and association with post-CV HF.

Pre-CV echocardiographic measurements and association with post-CV HF	Asymptomatic (Total N = 418)		Post-CV HF (Total N = 33)		p-Value
	n	Mean ± SD (range)	n	Mean ± SD (range)	
PASP	$n =$ 198	34.2 ± 11.0 (10–70)	$n =$ 15	43.6 ± 14.9 (19–73)	<0.01
EF	$n =$ 366	51.7 ± 14.3 (10–70)	n = 33	54.5 ± 13.6 (10–75)	0.28
E/A ratio	$n =$ 88	2.0 ± 2.8 (0.5–24.0)	$n =$ 11	2.4 ± 1.0 (1.2–4.2)	0.66
E/e lateral	n = 88	11.2 ± 5.8 (3.0–28.9)	$n =$ 10	13.7 ± 6.5 (7.2–28.9)	0.20
E/e medial	n = 88	14.7 ± 7.5 (5.0–39.5)	n = 10	18.0 ± 11.8 (6.7–43.2)	0.22
E/e average	n = 88	12.9 ± 6.2 (4.0–30.2)	n = 10	15.9 ± 7.3 (7.0–27.5)	0.16
LA volume index (biplane)	$n =$ 181	40.3 ± 17.3 (14.9–117.2)	n = 18	39.3 ± 17.4 (18.8–77.8)	0.81

criteria for our study. Baseline characteristics and their associations with post-CV HF are listed in Table 1. Pre-CV echocardiographic measurements and their associations with post-CV HF are listed in Table 2. The average age of patients at time of CV included in our study was 65.5 years (range 22–95, SD 12.3), and 68.5 % of patients were male (31.5 % female). Electrical CV comprised 89 % of the total cases, with the remaining 11 % being ablations. Seventy-three percent of patients underwent procedure for CV of AF, 23 % were for AFL, and 4 % had mixed features of AF and AFL.

Thirty-three patients (7.3 %) developed decompensated HF while maintaining SR after successful CV without other identifiable cause. Symptoms were reported an average of 5.1 days following CV (range 0–17 days, SD 5.0) (Fig. 1). Fifteen (3.3 %) patients developed symptoms within 48 h post-CV. The mean EF of control patients was not significantly different than the mean EF of those who developed post-CV HF (51.7 % vs 54.5 %, $P = 0.28$). Of the 33 patients who developed post CV HF, 26 had an EF ≥50 % (HfpEF), 4 had moderately reduced EFs and 3 had an EF <40 % (HfrEF). Fifteen of the 33 patients did not have a history of a prior HF hospitalization and most (14 of 15) had a normal EF. Of the 418 patients who did not experience post CV HF, 366 of them had pre-CV echocardiograms and of those, 70 had a prior EF <40 %. Four patients (0.01 %) patients died within 30 days of their CV, none of whom experienced post-CV HF. There was no significant difference between patients who developed HF and those who did not with respect to duration of arrhythmia prior to CV ($P = 0.49$), the type of rhythm prior to CV ($P = 0.17$), or whether they underwent electrical vs ablative CV ($P = 0.47$).

Univariate associations for development of HF included: increased BMI (. 32.7, $P < 0.01$), increased pre-procedural PASP (43.6 mmHg vs 34.2 mmHg, $P < 0.01$), female sex (48 % vs 30 %,$P = 0.03$), anti-arrhythmic drug (AAD) use (52 % vs 33 %,$P = 0.03$), CPAP use (24 % vs 12 %, $P = 0.05$), hypertension (85 % vs 67 %, $P = 0.03$), pulmonary hypertension (21 % vs 5 %, $P < 0.01$), valve disease (36 % vs 17 %, $P < 0.01$), and prior HF hospitalization (55 % vs 23 %, $P < 0.01$). Following application of a multivariate stepwise logistic regression model, prior HF hospitalization (OR 3.91, 95 % CI 1.82–8.39), BMI (OR 1.06, 95 % CI 1.02–1.11), and valve disease (OR 2.51, 95 % CI 1.12–5.60) remained significant risk factors and AAD use (OR 2.02, 95 % CI 0.95–4.31) was marginally significant (Fig. 2).

4. Discussion

Although decompensated HF after AF or AFL CV has previously been reported, there is limited data on its prevalence and risk factors. While the pathophysiologic mechanism of post-CV HF is likely multi-factorial, atrial stunning is believed to play a large role and is known to persist for several days to weeks after CV [10,14]. Atrial stunning is also indicated in the pathophysiology of post-CV thromboembolism, and its gradual recovery is one basis for current guidelines which recommend at least four weeks of anticoagulation after CV [15,16]. We hypothesized that post-CV HF remains a risk for a similar duration of time after CV. Using a 30-day time interval post-CV, we identified 33 patients in our study population who experienced new or worsening HF symptoms without

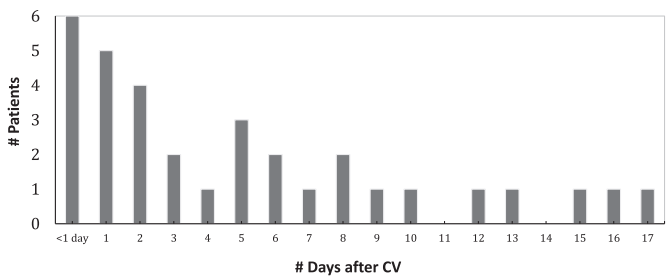

Fig. 1. Onset of HF symptoms after CV.

other alternative causes, equating to a complication rate of 7.3 %. This is much higher than a previously reported rate of 3.9 % in a study population who were observed for 48 h after CV [2]. Notably, we obtained a similar frequency of 3.3 % in our patient population when restricting our observation period to 48 h post-CV.

Similar to the risk profile of post-CV thromboembolism, most cases of post-CV HF occurred earlier in the studied time interval, rather than later [16,17]. While a few instances occurred after two weeks (with the most delayed presentation being at 17 days), more than half of cases occurred by the end of day four (Fig. 1). Not surprisingly, the onset of post-CV HF in our data correlates well with the onset and resolution of LA mechanical dysfunction [10,14]. This information further suggests that clinicians should be conservative in management decisions which could exacerbate a low output state and that they should maintain a high index of suspicion for onset of post-CV HF for well beyond 48 h after CV.

Prior research on post-CV HF focused on electrical CV only. Considering that atrial stunning is known to occur after all methods of CV, we further hypothesized that post-CV HF is additionally a complication after ablative CV [8,9,11–13]. Indeed, we found that it is observed in both ablative and electrical CV (Table 1), although we did not find a statistically significant difference between rates of post-CV HF in ablative versus electrical CV. Regardless, this finding has important implications for proceduralists and should motivate judicious use of fluids during ablations.

These clinical findings highlight the importance of identifying predictive indicators of post-CV HF which can be used to guide management decisions. We identified nine clinical features associated with the development of post-CV HF, of which four remained significant or marginally significant after multivariate stepwise logistic regression: prior HF hospitalization, BMI, valve disease, and AAD use at time of CV (Fig. 2). The associations and predictors we identified corroborate the limited available research on this topic with significant overlap despite our decision to include ablative CV and use a longer post-CV observation period [2]. BMI and valve disease additionally demonstrated stronger associations with the development of post-CV HF in our study than in prior research. Thus, for peri-cardioversion or ablation planning, clinicians can advise their patients on the signs and symptoms of post-CV HF and/or consider earlier treatment strategies with diuretics and other medications that may be useful in particularly patients with AF and HFrEF such as sodium-glucose cotransporter 2 inhibitors (SGLT2i) [18]. Similarly, with respect to holistic long-term management, all patients with an elevated BMI and AF should receive aggressive risk factor reduction counseling and treatment [15].

The associations we identified are well explained by an etiologic framework for post-CV HF centered on atrial stunning. Worsened LA function has been shown to be a predictor of HF onset and is associated with impaired outcomes in HF patients with both preserved and reduced EF [19]. Patients in our study who have histories consistent with clinical HF, such as current diuretic use and prior HF hospitalization(s), and those with elevated PASP within the three years prior to CV, demonstrated increased risk of new or worsening decompensated HF after CV. If atrial stunning is a central mechanistic component of post-CV HF, our findings are consistent with prior observations regarding predictive utility of worsened LA function and suggest that patients with these histories—even if HF is currently compensated or subclinical—are simply less able to tolerate an additional insult to cardiac output such as atrial stunning.

Further insight about our clinical predictors is revealed when considering the physiologic components known to predispose patients to LA stunning. Atrial stunning has been shown to be associated with longer duration of arrhythmias prior to CV, as well as atrial cardiomyopathy, increased atrial fibrosis, and changes in cellular calcium regulation, among others [6–9,13,20–22]. Notably, patients with obesity are more likely to progress to more chronic forms of AF and have larger LA volumes compared to others. Obesity has also been associated with increased interstitial fibrosis and electrical remodeling and conduction

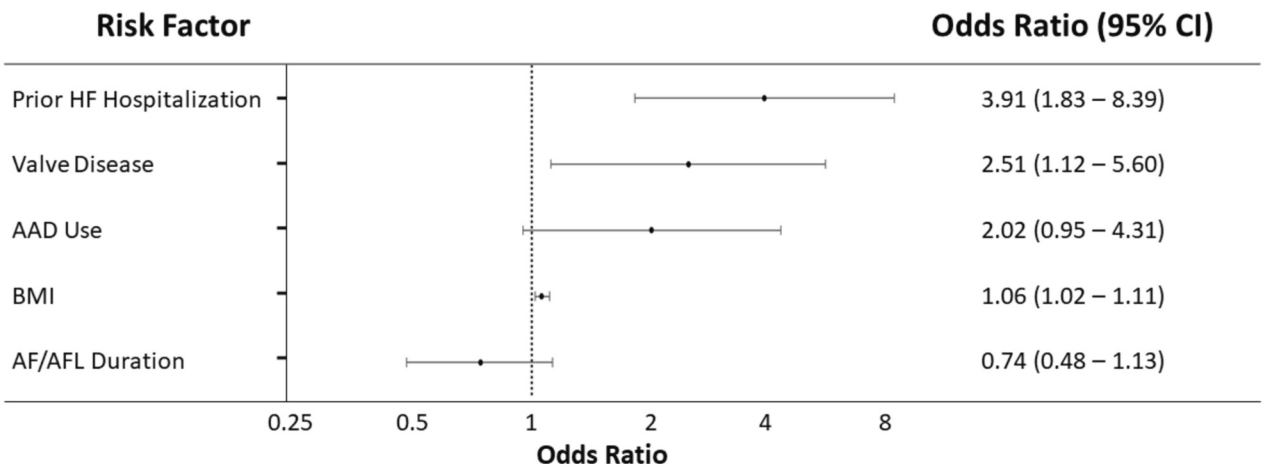

Fig. 2. Risk factors of post-CV HF.

heterogeneity of the LA [23–25]. These characteristics could explain why we found BMI to be a predictor of post-CV HF. Similar conjectures could be made about patients with valve disease, who are also likely to have atrial remodeling in response to valvular insufficiency or stenosis.

Patients with larger LA size are less likely to maintain SR and more likely to be prescribed AADs [26]. In turn, patients on AADs have higher AF free survival rates after CV compared with patients not on AADs [27]. That said, some AADs appear to play a more direct role in causing atrial dysfunction. Prior research has shown that sotalol may aggravate atrial stunning after CV of AF and that patients who underwent pharmacological cardioversion by propafenone may exhibit higher levels of atrial stunning as compared to CV by amiodarone or by direct current [12,28,29]. Further research is necessary to evaluate whether specific AADs have stronger associations with post-CV HF than others, as well as the risk profile of pharmacologic CV compared to ablative and electrical.

Interestingly, our data suggest a univariate association between female sex and the development of post-CV HF. Although previous research has not shown an association between patient sex and atrial stunning after CV, multiple differences in the way that female patients with AF or AFL present and are managed as compared to men may explain this association. Women with AF are known to have a higher prevalence of hypertension and valvular heart disease than men with AF [30], both of which were also univariate associations with post-CV HF in our study. Further, women experience longer periods of time from symptom onset to diagnosis, EP referral, and ablation as compared to men [31].

Despite that transient LA dysfunction after CV has been demonstrated to be associated with longer durations of AF or AFL, our data did not identify a univariate association between the duration of arrhythmia and the development of post-CV HF symptoms. While it is possible that atrial stunning due to prolonged AF may be insufficient to cause post-CV HF in absence of other risk factors, we also suspect several limitations impacted our ability to detect an association if present. Considering that only 14 patients who experienced post-CV HF had a duration of arrhythmia >30 days, our study may have been underpowered to detect duration as a predictor of post-CV HF. Durations of AF and AFL are also difficult to ascertain without direct cardiac monitoring which was not available in the large majority of cases. In our study, we relied on patient reports and documentation, both of which predispose to inaccuracies. For these reasons, we treated this variable as categorical instead of continuous in attempt to limit the impact of imprecision.

5. Limitations

Our study was retrospective, so the completeness and validity of our data was dependent on whether and how the data elements of interest were documented. Patients who presented to other institutions with post-CV HF would not be identified using our methods. Some data elements of interest, such as serum BNP, cardiac monitoring during arrhythmia onset, and echocardiograms, were not performed in every patient limiting our ability to use one standard definition for post-CV HF, evaluate for associations, and include some variables (such as PASP) in multivariate analysis. The clinical indicators we identified will need to be validated prospectively to determine their predictive utility and it remains unclear if our findings are generalizable to other institutions owing to the single center nature of this study.

6. Conclusions

From our dataset of electrical and ablative CV for AF or AFL, we found that 7.3 % of patients developed decompensated HF within 30 days of successful CV. The mean time of onset was 5.1 days post-procedure and ranged from hours to 17 days. Our findings indicate that post-CV HF is more common than previously reported and remains a risk for much longer than 48 h post-procedurally. Given that this makes post-CV HF a comparatively common complication, our findings have implications for management of patients during ablations and for the several weeks after CV. Using a multivariate logistic regression model, we identified several clinical features as risk factors for HF post-CV, including elevated BMI, valve disease, previous HF hospitalization, and prior AAD use. In our future work, we plan to create and validate a risk calculator to help antecedent identification of at-risk persons to inform and initiate preventative treatment strategies.

CRediT authorship contribution statement

Christina Healy: Writing – original draft, Formal analysis. **Palwinder Sodhi:** Data curation. **Annabelle Barnett:** Data curation. **Timothy Hess:** Writing – review & editing, Methodology, Formal analysis. **Jennifer M. Wright:** Writing – review & editing, Supervision, Project administration, Methodology, Conceptualization.

Declaration of competing interest

Jennifer Wright reports a relationship with Biosense Webster Inc. that includes: consulting or advisory. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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