**Beyond the Storm: Comparison of Clinical Factors, Arrhythmogenic
Substrate and Catheter Ablation Outcomes in Structural Heart Disease
Patients With Versus those Without a History of Ventricular Tachycardia
Storm**

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Short title: Differences in VT storm versus non-storm patients

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This is the author manuscript accepted for publication and has undergone full peer review but has not
been through the copyediting, typesetting, pagination and proofreading process, which may lead to
differences between this version and the [Version of Record](#). Please cite this article as [doi:
10.1111/jce.13117](https://doi.org/10.1111/jce.13117).

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Dr. Kumar is a recipient of the Neil Hamilton Fairley Overseas Research scholarship co-funded by the National Health and Medical Research Council and the National Heart Foundation of Australia; and the Bushell Travelling Fellowship funded by the Royal Australasian College of Physicians.

Dr. John receives consulting fees/honoraria from St. Jude Medical. Dr. Epstein receives consulting fees/honoraria from Boston Scientific Corp., Medtronic, Inc., and Spectranetics Corp. Dr. Michaud receives consulting fees/honoraria from Boston Scientific Corp., Medtronic, Inc., and St. Jude Medical, and research funding from Boston Scientific Corp., and Biosense Webster, Inc. Dr. Stevenson is co-holder of a patent for needle ablation that is consigned to Brigham and Women's Hospital. Dr. Tedrow receives consulting fees/honoraria from Boston Scientific Corp. and St. Jude Medical and research funding from Biosense Webster, Inc., and St. Jude Medical. Other authors: No disclosures.

Abstract

Aims

Catheter ablation can be lifesaving in ventricular tachycardia (VT) storm, but the underlying substrate in patients with storm is not well characterized. We sought to compare the clinical factors, substrate and outcomes differences in patients with sustained monomorphic VT who present for catheter ablation with VT storm versus those with a non-storm presentation.

Methods

Consecutive ischemic (ICM; n=554) or non-ischemic cardiomyopathy patients (NICM; n=369) with a storm vs. non-storm presentation were studied (ICM storm 186; NICM storm 101).

Results

In ICM, storm compared with non-storm patients had significantly lower left ventricular (LV) ejection fraction (EF), greater number of antiarrhythmic drug (AAD) failures, slower VTs,

greater number of scarred LV segments, higher incidence of anterior, septal and apical endocardial LV scar (all $P < 0.05$). However, outcomes in follow-up were similar (12-month ventricular arrhythmia [VA]-free survival: 51% vs. 52%, $P = 0.6$; survival free of death/transplant 75% vs. 87%, $P = 0.7$). In addition to the above differences, NICM storm patients were also older; however, the extent and distribution of scar was similar except for a higher incidence of lateral endocardial scar in storm patients ($P = 0.05$). VA-free survival (36% vs. 47%, $P = 0.004$) and survival free of death/transplant, however, were worse in NICM storm than non-storm patients (72% vs. 88%, $P = 0.001$). NICM storm patients had worse VA-free survival than ICM storm patients.

Conclusion

There are differences in clinical factors and scar patterns in patients undergoing VT ablation who present with VT storm versus those with a non-storm presentation. Clinical outcomes are worse in NICM storm patients.

Keywords: ventricular tachycardia; electrical storm; catheter ablation; ischemic cardiomyopathy; non-ischemic cardiomyopathy.

Introduction

Electrical storm, defined as three or more sustained episodes of ventricular arrhythmia or appropriate implantable cardioverter-defibrillator (ICD) shocks during a 24-hour period afflicts 3.5-4% of primary prevention and 10-40% of patients with a secondary prevention ICD.¹ It carries an ominous prognosis with an increased future risk of arrhythmic and cardiac

death.² Percutaneous catheter ablation, as an adjunct to antiarrhythmic drugs (AADs), reduces the burden of ICD shocks³ and can be lifesaving in ventricular tachycardia (VT) storm.⁴ The underlying mechanism(s) that cause some patients to develop VT storm, while others do not are not clear. Putative reasons include a variable interaction between perturbations in autonomic tone, coupled with initiating triggers such as ischemia and electrolyte disturbances and a predisposing electrophysiological substrate.⁵ Prior studies have examined the clinical differences in ICD patients with and without storm.^{6,7} However, electrophysiologic and electroanatomic substrates have not been reported and the etiology of cardiomyopathy has not been systematically considered. Outcomes following catheter ablation of VT storm have been reported,^{4,8,9} but the efficacy has not been compared in ischemic versus non-ischemic cardiomyopathy (ICM, NICM, respectively), diseases that have distinctly different natural histories and overall response to catheter ablation.^{10,11}

The aim of this study was to (i) compare the clinical factors, substrate and outcomes following catheter ablation of sustained monomorphic VT in patients who presented with VT storm versus those with a non-storm presentation; (ii) compare the aforementioned factors between ICM storm and NICM storm patients; and (iii) examine clinical predictors of VT storm in patients with ICM and NICM undergoing percutaneous catheter ablation.

Methods

This retrospective single-center study from the Brigham and Women's Hospital examined 923 consecutive patients with structural heart disease presenting for percutaneous catheter ablation of drug-refractory sustained monomorphic VT between 1999-2014. All patients gave

written informed consent for the procedure. Our institutional Human Subject Protection Committee approved retrospective analysis of data.

All patients underwent echocardiography and/or magnetic resonance imaging study to screen for the presence of structural heart disease and to define ventricular function. Patients with non-sustained VT, premature ventricular contractions or ventricular fibrillation (VF) initiated by premature ventricular contractions as the procedural indication were excluded. NICM was identified as ventricular disease in the absence of relevant coronary artery disease and defined according to the criteria of the European Society of Cardiology Working Group for Myocardial and Pericardial Diseases.¹² Both left and right ventricular NICM were included. Patients with prior history of infarction (or that confirmed by invasive or non-invasive evaluation of coronary artery disease), or a history of revascularization and/or documented coronary artery disease affecting ≥ 1 vessel were classified as ICM, although it is possible that some of these patients had mixed cardiomyopathy.¹³

Within the ICM and NICM subgroups, we divided patients according to their first procedure at our institution (termed the 'index' procedure) as those who presented with VT storm and those who had a non-storm presentation. We used a standard definition of storm as three or more separate episodes of sustained VT within 24 hours.¹⁴ This included VT episodes requiring termination by an intervention such as any appropriate defibrillator therapy (shock or anti-tachycardia pacing), external cardioversion and/or AAD therapy. Incessant VT was included in the storm definition.

Mapping and Ablation

Percutaneous endocardial and epicardial mapping and ablation were performed as described previously.¹⁵ Briefly, programmed ventricular stimulation was performed with 1 to 3 extrastimuli after drive trains of 600 milliseconds (ms) and 400 ms from 2 right ventricular (RV) sites, and repeated from at least one left ventricular (LV) site if VT was non-inducible from RV stimulation in patients undergoing LV mapping. Isoproterenol or epinephrine was used upon the discretion of the operator. The stimulation protocol was repeated after ablation unless it was felt that it would jeopardize the patient's hemodynamic stability. The morphologies of the induced VTs were compared to those of spontaneously occurring VT(s) prior to ablation when available. Sustained monomorphic VT was defined as continuous VT for ≥ 30 seconds or one that required an intervention for termination (cardioversion, pacing).¹⁴

"Spontaneous VT" was defined as any inducible VT with an identical 12-lead electrocardiographic (ECG) morphology and cycle length (within 20 milliseconds [ms]) compared to a spontaneously occurring VT. If 12-lead ECGs of the presenting VT were not available prior to ablation, the rate cut off and intracardiac electrogram characteristics from the ICD were used.¹⁶ "Undocumented VTs" were defined as other inducible sustained monomorphic VTs.

Substrate mapping was performed focused on areas of abnormal electrograms characterized by fractionation and relatively low amplitude, typically a bipolar voltage of 1.5 mv or less.¹⁷⁻²¹ Mapping was performed using an ablation catheter or, when available, a multi-spline catheter (Pentaray, Biosense Webster, Diamond Bar, CA, USA) and the CARTO electroanatomic mapping system (Biosense Webster). Areas of electrically unexcitable scar were also noted.¹⁸

Ablation was targeting presumptive channels and exits within areas of abnormal voltage identified from a paced QRS morphology similar to the VT QRS morphology, abnormal fractionated potentials, double potentials, or late potentials during sinus or paced rhythm at sites where pacing captured, particularly with stimulus–QRS intervals of >40 ms.¹⁸⁻

²¹ If no low voltage area was identified, ablation was attempted at the likely exit region identified as sites with presystolic electrograms during VT or where pace mapping resembled the VT QRS. If hemodynamically tolerated VT was inducible activation/entrainment mapping was performed. If VT was not tolerated, it was terminated with radiofrequency ablation (RFA), burst pacing or cardioversion and further ablation guided largely by substrate mapping.^{22, 23}

Radiofrequency ablation (RFA) was delivered with an irrigated catheter (ThermoCool, ThermoCool SF; Biosense Webster) at a power of 30 to 50 Watts targeting an impedance drop of 10 to 18 ohms. Power was titrated up from 30 Watts; power was reduced to avoid impedance falls of more than 18 Ohms based on the observation that this was associated with steam pops with the Thermocool catheter.²⁴ Applications were repeated at target areas until they were rendered electrically unexcitable to unipolar pacing at 10 milliamps at 2-ms pulse width.

Epicardial mapping was performed using the percutaneous approach if VT was suspected to be of epicardial origin, generally after endocardial ablation failed to abolish inducible VT.²⁵ Coronary angiography was performed before epicardial ablation to avoid coronary injury; high output pacing was also performed to avoid ablation in close proximity to the phrenic nerve.

We adapted the schema developed by Josephson et al for the categorization of scar regions (Figure 1).^{26, 27} For analysis of scar distribution, the LV was divided into anterior, septal, inferior, lateral (including peri-mitral region), apical, and left ventricular outflow tract/aortic-mitral continuity segments. The RV was divided into outflow tract, free wall, peri-tricuspid annular, septum and inferior wall segments. Characterization of scar regions was by experienced operators who performed the procedure, and later clarified by review of maps, taking into account the ventricular size and volume.

AADs prescribed long-term were either continued at the same or reduced dose, or discontinued as per the treating physician. AADs initiated recently to control multiple/incessant VT were discontinued. Programming of the ICD was left to the treating physician. In general, a minimum of two zones was programmed. The first zone was set at a rate of at least 20 ms below the rate of the slowest spontaneous or inducible VT with or without therapy with anti-tachycardia pacing and a second zone had a minimum rate cut off of >188 beats per minute programmed to deliver shock with or without anti-tachycardia pacing. Where possible, programming was kept consistent before and after an ablation procedure.

Outcomes

Acute procedural outcomes were reported as:

- (a) complete success: defined as non-inducibility of any VT, either “spontaneous” or “undocumented”;

- (b) partial success: defined as abolition of at least one “spontaneous” VT, but other “spontaneous” or “undocumented” VTs remained inducible;
- (c) failure: persistent inducibility of “spontaneous” VT.
- (d) acute suppression of VT storm at the end of the procedure.

In follow-up, outcomes reported were:

- (a) VA-free survival: defined as absence of any sustained VT or VF that was detected by monitoring or that required treatment with AADs, ICD or external cardioversion or anti-tachycardia pacing;
- (b) survival free of death or cardiac transplantation;
- (c) recurrence of VT storm after ablation of storm in the index procedure.

Follow-Up

Follow-up was obtained from review of records of all hospital and outpatient clinic visits and from referring cardiologists, electrophysiologists and primary care physicians. The National Social Security Death Index was searched for mortality information. Follow-up time was defined from the date of first ablation to time of death or last clinical contact. Procedural outcomes were reported after a single procedure at 12 months follow-up.

Statistical Analysis

The Statistical Package for the Social Sciences for Windows (IBM SPSS, release 23, Armonk, NY, USA) was used for analysis. Continuous variables were expressed as mean $\pm$ standard deviation if normally distributed; median and interquartile range 25% to 75% (Q25–Q75) or full ranges were used if the data were clearly skewed. Where normal distribution was not present, log transformation of the raw values was performed to meet the assumption of homogeneity of variance. Where applicable, paired sample t tests were performed using raw values (if normally distributed) or log-transformed values (if not normally distributed). Acute procedural success and complications were compared as categorical variables with the Fisher's Exact test.

Survival free of VA and survival free of a composite endpoint of death or transplant were estimated using the Kaplan–Meier procedure and reported at 12 months. Cox-proportional Hazard models were created to determine clinically relevant factors associated with VA recurrence and a composite endpoint of death or transplant. Binary logistic regression models were used to examine clinical factors that predicted onset of VT storm. Hazard ratios (HR) and 95% confidence intervals (CI) were used to express risk of VA recurrence and mortality. Odds ratios (OR) and 95% CI were used to express the odds of presentation with VT storm. A two-tailed *P* value <0.05 was considered statistically significant.

Results

Of 923 patients, there were 554 patients with ICM and 369 patients with NICM. In ICM, there were 186 patients with storm (34%) and 368 patients with a non-storm presentation. In

NICM, there were 101 patients with storm (27%) and 268 patients with a non-storm presentation (Table 1).

Baseline characteristics

ICM storm compared to non-storm ICM patients had greater LV dysfunction, higher incidence of NYHA class ≥ 3 heart failure, greater number of class I or III AAD failures at the time of ablation (Table 1). Number of diseased vessels was similar; however, storm patients had a shorter elapsed time since last infarct. Prevalence of co-morbidities was similar in ICM storm compared to non-storm ICM patients.

NICM storm patients, compared to non-storm patients were older, had greater LV dysfunction, more dilated LVs, higher incidence of NYHA class ≥ 3 heart failure, and a greater number of drug failures (Table 1). Prevalence of co-morbidities was similar in NICM storm compared to non-storm ICM patients, except for a trend toward a higher prevalence of diabetes in NICM storm patients.

ICM storm compared with NICM storm patients were older, were more likely to be males, had greater LV dysfunction, more drug failures (in particular amiodarone failure), had more prior VT ablation procedures, and a higher prevalence of hypertension and chronic renal disease (Table 1). Information on the number of operators and patients recruited during the study period is shown in Supplemental Results and Supplemental Table 1.

Predictors of VT storm

Clinically relevant factors were examined for their association with VT storm separately in patients with ICM and NICM. LV ejection fraction and number of failed AAD were independently associated with an increased risk of VT storm in both ICM and NICM (Table 2).

Low bipolar voltage scar regions

ICM storm compared to non-storm patients had a greater number of endocardial LV bipolar (but not RV) scar segments (Table 3) and were more likely to have anterior, septal and apical scar (Figure 1A). RV endocardial or epicardial scar were equally prevalent in both storm and non-storm ICM patients.

NICM storm compared to non-storm patients had a similar number of LV and RV scar segments (Table 3), but were more likely to have endocardial lateral or perivalvular scar (Figure 1B). The incidence of epicardial scar and distribution of RV endocardial scar were not different between storm and non-storm patients (Figure 1C).

ICM storm, compared with NICM storm patients had a greater number of LV scar segments; however, NICM storm patients had greater number of RV scar segments (Table 3). ICM storm patients were more likely to have anterior, septal, inferior and apical scar compared with NICM storm patients (Figure 2D), but prevalence of lateral scar was the same in both storm groups. NICM storm patients, compared to ICM storm patients were more likely to have LVOT, epicardial scar, and RV scar in all segments except for the RV septum, which had the same prevalence in both groups (Figure 2D).

Procedural characteristics

ICM storm compared with non-storm patients had a similar number of inducible VTs but a higher incidence of slow VTs (CL >400 ms). Ablation and procedural times, need for epicardial ablation or non-RFA ablation modalities, and major complications were similar between storm and non-storm ICM patients (Table 3).

NICM storm compared with non-storm patients also had a similar number of inducible VTs but a higher incidence of slow VTs. NICM storm patients had longer ablation times compared to non-storm patients. However, need for epicardial ablation or non-RFA ablation modalities, and major complications were similar to non-storm patients.

ICM storm compared with NICM storm patients had a higher incidence of slow VTs, and longer ablation times. However, NICM storm patients had a higher incidence of epicardial RFA than ICM storm patients (Table 3).

Acute Procedural Outcomes

Acute procedural success was similar between storm versus non-storm patients in both ICM and NICM subgroups (Table 3). Complete success, however, was more common in ICM storm compared with NICM storm patients (Table 3). Acute suppression of VT storm was achieved in all but 1 NICM patient who required on-table percutaneous left ventricular assist device insertion and died intra-procedurally from intractable VT.

Outcomes in Follow-up

Mean follow-up was 51 ± 46 months (median 42 months). Single procedure VA-free survival was similar in ICM storm vs. non-storm patients (12 months: $51 \pm 5\%$ vs. $52 \pm 3\%$; $P=0.6$, Figure 2A). Survival free of death or transplant was similar in ICM storm vs. non-storm patients (12 months: $75 \pm 3\%$ vs. $87 \pm 2\%$; $P=0.7$; Figure 2B).

Single-procedure VA-free survival was worse in NICM storm vs. non-storm patients (at 12 months: $36 \pm 6\%$ vs. $47 \pm 4\%$; $P=0.004$; Figure 2C). Survival free of death or transplant also worse in NICM storm vs. non-storm patients (at 12 months: $72 \pm 5\%$ vs. $88 \pm 2\%$; $P=0.001$; Figure 2D). NICM storm patients had worse VA-free survival compared with ICM storm patients ($P=0.007$; Figure 3A); but survival free of death or transplant was similar ($P=0.96$, Figure 3B).

On multivariable analysis in ICM patients, LVEF, number of class I or III AAD failures prior to ablation, and acute complete success at the end of the procedure were associated with VA recurrence (Table 4). When accounting for these factors, the adjusted risk of VA recurrence was not significantly different similar in storm vs. non-storm patients (HR 0.99, 95% CI 0.8-1.3, $P=0.95$). The same factors were associated with VA recurrence in NICM patients (Table 4). After adjusting for these factors, the adjusted risk of VA recurrence in all storm vs. non-storm patients was not significantly different (HR 1.3, 95% CI 0.95-1.8, $P=0.1$). Storm was not a predictor of death or transplant in ICM after adjusting for other predictors (Table 5), but showed a trend toward increased risk in NICM patients (HR 1.4, 95% CI 0.9-1.9, $P=0.2$). There was also no significant difference between NICM storm vs.

ICM storm for VA recurrence (HR 1.3, 95% CI 0.8-1.8, P=0.2) or death/transplant (HR 1.4, 95% CI 0.94-2.2, P=0.1) on multivariable analysis.

Operator status and ablation time period were not associated with VA recurrence, nor death or transplant (Supplemental Tables 2-3); ablation duration also did not change over time during the study period (Supplemental Table 4).

Recurrence of storm

The following analyses were performed in patients who had storm. There was a trend to more recurrence of storm after the index ablation procedure in NICM than ICM patients (27% vs. 17%, P=0.07). In follow-up, 21/32 (81%) recurrent storm patients with ICM and 19/27 (86%) recurrent storm patients with NICM had a repeat procedure (P=0.7). NICM storm patients had more repeat procedures than ICM storm patients (mean of 1.5 ± 0.8 , median 1, range 1-4 vs. mean 1.2 ± 0.6 , median 1, range 1-4; P=0.07).

Despite multiple procedures, VA-free survival in ICM patients with recurrent storm was worse than those without recurrent storm (at 12 months: $42 \pm 11\%$ vs. $67 \pm 5\%$, P<0.001; Figure 4A). Similarly, survival free of death or transplant after multiple procedures was also worse in ICM patients with recurrent storm than those without recurrent storm (at 12 months: $59 \pm 9\%$ vs. $78 \pm 4\%$, P=0.006; Figure 4B).

After multiple procedures, VA-free survival was equivalent in NICM patients with recurrent storm than those without recurrent storm (at 12 months: $53 \pm 12\%$ vs. $62 \pm 7\%$, P=0.9;

Figure 4C). However, survival free of death or transplant after multiple procedures was worse in NICM patients with recurrent storm than those without recurrent storm (at 12 months: $61\pm 10\%$ vs. $76\pm 5\%$, $P=0.02$; Figure 4D).

Discussion

This study provides new information on differences in clinical factors, arrhythmogenic substrate and outcomes of structural heart disease patients who present for catheter ablation with VT storm versus those with a non-storm presentation. The salient findings were:

- (i) storm patients had a sicker clinical profile characterized by greater LV dysfunction, worse clinical heart failure, greater number of drug failure in the setting of ICM in addition to being older and having dilated LVs in the setting of NICM;
- (ii) storm patients had different electrophysiologic substrate compared with non-storm patients in the extent and distribution of LV scar that was associated with defined monomorphic VTs. Furthermore, there were differences in extent and distribution of endocardial ventricular scar in storm patients with ischemic or non-ischemic cardiomyopathy;
- (iii) LV ejection fraction and number of failed antiarrhythmic drugs prior to ablation were two factors independently associated with the occurrence of storm in patients with either ICM or NICM;
- (iv) storm did not confer adverse long-term outcomes in ICM patients compared to non-storm patients; however, NICM patients with storm had worse outcomes

compared to non-storm patients. Moreover, NICM storm patients had worse outcomes than ICM storm patients.

(iv) patients with recurrent storm, however, had worse outcomes than those without recurrent storm, regardless of cardiomyopathy type.

This study suggests there are differences in the clinical profile, electrophysiologic substrate and response to catheter ablation in structural heart disease patients who present with storm versus those with a non-storm presentation. Moreover, differences appear to be specific to type of underlying disease. While overall, ICM storm patients undergoing catheter ablation have similar outcomes to their non-storm cohort, those with recurrent storm after ablation have poorer prognosis. In contrast, storm is an adverse marker in NICM and may suggest more advanced heart disease or late presentation in such patients.

Similar to prior studies, increasing age, advanced structural heart disease with LV dilatation and heart failure were more common in our group of patients with storm versus those with a non-storm presentation.^{6, 7, 28} Prior studies included a heterogeneous population of ICM and NICM patients sourced from outpatient ICD clinics; such studies also grouped both VT and VF presenting arrhythmia events as storm, and patients were likely in early stage of their disease. The population in this study likely has more advanced heart disease, given the failure of multiple drugs to control VT and a high incidence of storm (34% in ICM and 27% in NICM group undergoing VT ablation). Uniquely, we analyzed clinical factors specific to cardiomyopathy type that were more common in storm versus non-storm patients. Furthermore, the substrate differences in storm versus non-storm patients have not been

examined previously, nor has such a comparison been made of ICM patients with storm versus NICM patients with storm.

Storm is recognized as a distinctive clinical arrhythmia syndrome with specific management and prognostic implications portending an increased risk of death, heart transplantation, and heart failure hospitalization.² The mechanism is likely mediated by a complex interplay between neurohormonal changes, abnormalities in calcium-related signaling and protein phosphorylation abnormalities,²⁹ and a predisposing electrophysiological substrate.¹ We showed that storm patients have different extent and distribution of endocardial low voltage scar on intracardiac mapping, specific to cardiomyopathy type that may help explain their electrophysiologic vulnerability. Although abnormalities in electrophysiology properties have been speculated, no study prior to ours has compared the electroanatomic mapping features of storm versus non-storm patients. The reasons for scar predilection in the anterior, apical and septal LV for storm patients with ICM and the lateral LV for NICM storm patients is not known. Electrical storm has been suggested to involve calcium overload and it seems likely that sympathetic stimulation also has a role.²⁹ It is possible that differences in cardiac neural innervation differ for anterior infarcts predispose to slower VTs and to storms.

Outcomes of catheter ablation have been reported in storm patients.^{4, 8, 9} These studies have not examined outcomes specific to cardiomyopathy type, nor has a comparison been made between non-storm patients. In our study, we find that NICM storm patients are the highest risk group who face a poorer prognosis than their non-storm counterparts and worse than the ICM storm counterparts. Possible reasons are a late presentation, more advanced heart disease, or more difficult substrate targets; a combination of all factors is likely to

explain worse outcomes in NICM, as reported previously.^{10, 11} Unlike NICM, storm in ICM patients carried similar prognostic implications to their non-storm counterpart. We also showed that recurrent storm after an initial storm presentation treated with catheter ablation is associated with even worse outcomes in follow-up, consistent with a prior report.⁹ Such patients are likely to be an even higher risk cohort.

Limitations

This is a retrospective report from a specialized center for VT management and patients likely represent the severe form of the spectrum of heart disease. We do not have complete information on the impact of other factors such as the duration of arrhythmia history, infarct characteristics such as reperfusion and presence of persistent occlusion that would be of interest as potential markers of storm risk. However, the role of such factors has been evaluated in prior studies on ICD recipients in the non-ablation setting.^{1, 2, 5-7, 28, 30-32}

We did not examine the prevalence of electrophysiologic markers of substrate such as delayed, fractionated or isolated late potentials in storm versus non-storm patients, nor did we collect information on the density of mapping points for each case. Given the long recruitment time period of this study, evolution in the patient population and technology, including mapping and ablation systems occurred and the impact of these factors cannot be completely addressed in a retrospective study. However, the findings related to storm were consistent over the duration of the study.

Aggressive ICD programming may have increased the frequency and recurrence of ICD shocks and likelihood of storm.³² We cannot exclude that programming differences

between patients impact on the likelihood of storm presentation and classification in this study. Furthermore, given the retrospective nature of the analysis, we cannot predict if ablation could prevent storm in some patients. A further prospective study is needed accounting for the aforementioned heterogeneities that could have influenced the results.

Conclusions

In patients with structural heart disease patients, those who present for catheter ablation with VT storm have differences in clinical features, electrophysiologic substrate and response to catheter ablation compared to those patients with a non-storm presentation. Differences in substrate in storm versus non-storm patients are specific to cardiomyopathy type. Clinical outcomes appear to be the worse in NICM patients with storm compared to their non-storm counterparts and to ICM patients with storm. Recurrence of storm after initial catheter ablation for storm carries an adverse prognosis, despite repeated ablation.

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Table 1: Baseline characteristics

	Ischemic Cardiomyopathy (n=554)			Non-Ischemic
	Storm (n=186)	Non Storm (n=368)	<i>P</i> value Storm vs. Non-storm	Storm (n=101)
Age, mean $\pm$ SD, years	68 $\pm$ 10	66 $\pm$ 11	0.1	56 $\pm$ 15
Male gender, %	88	88	1	79
LV ejection fraction %, mean $\pm$ SD	27 $\pm$ 10	31 $\pm$ 13	0.002	33 $\pm$ 16
Hypertension	64	62	0.4	39
Diabetes	25	30	0.4	20
Chronic renal disease	41	39	0.7	25
Chronic lung disease	34	29	0.4	27
LV diastolic diameter, mean $\pm$ SD, mm	62 $\pm$ 10	62 $\pm$ 10	0.8	61 $\pm$ 12
NYHA heart failure class $\geq$ 3, %	31	22	0.03	38

Number of diseased vessels, mean $\pm$ SD (median)	2.2 $\pm$ 0.9 (2)	2.2 $\pm$ 0.9 (2)	0.6	-
Time since last infarct, months, mean $\pm$ SD (median)	132 $\pm$ 130 (95)	157 $\pm$ 106 (142)	0.06	-
Prior VT ablation, %	31	31	1	41
Number of prior VT ablation procedures in patients with at least 1 prior procedure, median (Q25-Q75)	1 (1-2)	1 (1-1)	0.4	2 (1-2)
Number of failed class I or III AAD, mean $\pm$ SD (median)	2.6 $\pm$ 1.2 (3)	2.2 $\pm$ 1.2 (2)	0.001	2.4 $\pm$ 1.2 (2)
Prior failed class I or III AAD, % of patients				
Amiodarone	82	79	0.4	71
Sotalol	31	34	0.4	43
Mexiltine	43	28	0.009	43

Abbreviations: AAD-antiarrhythmic drugs, LV-left ventricle, mm-millimetres, NYHA-New York Association, SD-standard deviation, and VT-ventricular tachycardia.

Table 2: Predictors of VT storm

	Ischemic cardiomyopathy				Non-ischemic cardiomyopathy			
	Univariable Odds ratio [OR] (95% CI)	P Value	Multivariable OR (95% CI)	P value	Univariable OR (95% CI)	P Value	Multivariable OR (95% CI)	P value
Age (each 1 year increase)	1.01 (0.997-1.03)	0.11	-		1.02 (1-1.04)	0.02	-	
Male (vs. female)	0.98 (0.57-1.7)	0.95	-		0.94 (0.53-1.66)	0.8	-	
LV ejection fraction (each 1% increase)	0.98 (0.96-0.99)	0.03	0.98 (0.96-0.995)	0.01	0.97 (0.95-0.98)	<0.001	0.98 (0.96-0.99)	0.002
LV diastolic	1.003 (0.98-	0.8	-		1.02 (0.99-	0.06		

diameter, (each 1mm increase)	1.02)				1.04)			
NYHA class ≥ 3	1.59 (1.05-2.41)	0.03			2.6 (1.57-4.53)	<0.001		
Number of failed class I or III AAD	1.3 (1.1-1.5)	0.002	1.3 (1.08-1.48)	0.003	1.4 (1.2-1.7)	0.001	1.3 (1.1-1.6)	0.006
Amiodarone failure	1.3 (1.1-2)	0.3	-		1.5 (0.9-2.4)	0.13	-	
Hypertension	1.1 (0.75-1.6)	0.6	-		1.4 (0.88-2.3)	0.15	-	
Diabetes	0.78 (0.47-1.3)	0.4	-		1.8 (0.98-3.3)	0.06	-	
Chronic renal disease	1.09 (0.76-1.57)	0.6	-		1.6 (0.93-2.8)	0.09	-	

Chronic lung disease	1.29 (0.76-2.18)	0.35	-	1.2 (0.6-2.2)	0.6	-
Prior VT ablation	0.99 (0.68-1.45)	0.95	-	0.98 (0.6-1.6)	0.9	-
Abbreviations as above.						

Table 3: Procedural characteristics

	Ischemic Cardiomyopathy (n = 554)			Non-Ischemic Cardiomyopathy (n = 369)			
	Storm (n = 186)	Non Storm (n = 368)	P value Storm vs. Non-storm	Storm (n = 101)	Non Storm (n = 268)	P value Storm vs. Non-storm	P value ICM storm vs. NICM storm
Number of inducible VTs, mean $\pm$ SD, (median)	2.8 $\pm$ 1.7 (2)	2.8 $\pm$ 1.7 (2)	0.9	2.6 $\pm$ 1.7 (2)	2.5 $\pm$ 1.7 (2)	0.5	0.3
Spontaneous or inducible VT cycle length							
>400 ms (%)	69	52	<0.001	52	34	0.002	0.006
300--400 ms (%)	64	63	1	66	63	0.6	0.5

<400 ms (%)	23	34	0.01	31	43	0.05	0.1
Number of low-voltage scar regions, mean $\pm$ SD (median)							
LV	2.2 $\pm$ 0.98 (2)	1.95 $\pm$ 1 (2)	0.02	1.3 $\pm$ 0.8 (1)	1.3 $\pm$ 1 (1)	0.8	<0.001
RV	0.3 $\pm$ 0.8 (0)	0.3 $\pm$ 0.6(0)	0.6	1 $\pm$ 1.3 (1)	1 $\pm$ 1.3 (0)	0.3	<0.001
RFA time, mean $\pm$ SD, seconds (median)	2140 $\pm$ 1469 (1967)	2094 $\pm$ 1259 (1956)	0.7	1538 $\pm$ 1132 (1389)	1536 $\pm$ 1426 (1191)	0.9	0.002
Fluoroscopy time, mean $\pm$ SD, minutes (median)	41 $\pm$ 29 (36)	41 $\pm$ 23 (36)	0.8	43 $\pm$ 20 (37)	43 $\pm$ 19 (39)	1	0.7
Total procedure	234 $\pm$ 73	236 $\pm$ 79	0.9	244 $\pm$ 78	248 $\pm$ 78	0.7	0.5

duration, mean ± SD, minutes (median)	(235)	(217)		(235)	(255)		
Need for epicardial RFA, %	3.8	4.1	1	24	23	0.9	<0.001
Need for non- RFA ablation modality ^a , %	2.2	0.8	0.2	5	3	0.4	0.3
Major complications	6.6	7.1	0.7	11	8	0.4	0.3
Acute procedural outcomes							
Complete success ^b	60	56	0.2	50	54	0.5	0.02
Partial success ^c	20	20		14	18		
Failure ^d	11	9		18	13		
Not tested ^e	9	15		18	16		

^atranscoronary ethanol ablation, surgical epicardial window or surgical cryoablation; ^{b-d}see methods for definitions; ^edue to hemodynamic instability

Abbreviations (as above) and: ICM-ischemic cardiomyopathy, ms-milliseconds, NICM-non-ischemic cardiomyopathy, RFA-radiofrequency ablation, RV-right ventricle.

Table 4: Clinically relevant factors associated with VA recurrence

	Ischemic cardiomyopathy				Non-ischemic cardiomyopathy			
	Univariable HR (95% CI)	P Value	Multivariable HR (95% CI)	P value	Univariable HR (95% CI)	P Value	Multivariable HR (95% CI)	P value
Age (each 1 year increase)	0.997 (0.99-1.009)	0.6	-		1.06 (0.996-1.02)	0.26	-	
Male (vs. female)	1.3 (0.9-1.9)	0.22	-		1.03 (0.7-1.5)	0.9	-	
EF (each 1% increase)	0.98 (0.97-0.99)	<0.001	0.98 (0.97-0.99)	0.001	0.99 (0.98-0.995)	0.002	0.99 (0.98-0.999)	0.03
Storm (vs. non-storm)	1.1 (0.8-1.4)	0.6	0.99 (0.8-1.3)	0.95	1.5 (1.1-2)	0.009	1.3 (0.95-1.8)	0.11
Number of failed AAD	1.2 (1.04-1.3)	0.008	1.1 (0.99-1.2)	0.07	1.2 (1.06-1.3)	0.002	1.2 (1.03-1.29)	0.01
Amiodarone	1.5 (1.1-1.9)	0.02	-		1.2 (0.9-1.6)	0.16	-	

ne failure	2.2)				1.7)				
Epicardial	1.4 (0.8-	0.23	-		0.97 (0.7-	0.8	-		
RFA	2.6)				1.4)				
Adjunctiv	1.4 (0.5-	0.5	-		1.4 (0.7-	0.7	-		
e non-	3.7)				1.8)				
RFA									
Major	1.5 (0.96-	0.07			1.1 (0.7-	0.7			
complicati	2.2)				1.8)				
on									
Acute	0.6 (0.5-	<0.00	0.6 (0.5-	0.00	0.7 (0.5-	0.00	0.7 (0.6-	0.04	
complete	0.8)	1	0.8)	1	0.9)	7	0.99)		
success									

Abbreviations (as above and): CI-confidence interval, EF-ejection fraction, HR-hazard ratio.

Table 5: Factors associated with death or transplant

	Ischemic cardiomyopathy				Non-ischemic cardiomyopathy			
	Univariable HR (95% CI)	P Value	Multivariable HR (95% CI)	P value	Univariable HR (95% CI)	P Value	Multivariable HR (95% CI)	P value
Age (1 year increase)	1.04 (1.02-1.05)	<0.001	1.04 (1.03-1.06)	<0.001	1.05 (1.04-1.07)	<0.001	1.03 (1.02-1.05)	<0.001
Male (vs. female)	1.2 (0.8-1.8)	0.3			1.2 (0.7-1.8)	0.6		
EF (each 1% increase)	0.95 (0.94-0.96)	<0.001	0.95 (0.94-0.97)	<0.001	0.94 (0.93-0.96)	<0.001	0.96 (0.94-0.97)	<0.001
Stroke	1.05 (0.8-1.4)	0.7	0.9 (0.7-1.2)	0.4	1.9 (1.3-2.8)	0.001	1.3 (0.9-1.9)	0.2
Number of failed AAD	1.1 (1-1.3)	0.02			1.3 (1.1-1.5)	<0.001		
Amiodarone failure	2 (1.4-3)	<0.001			4.5 (2.6-7.6)	<0.001	2.4 (1.4-4.2)	0.002

Epicardial	0.7 (0.3-	0.3			1.03	0.9
RFA	1.4)				(0.6-1.6)	
Adjunctiv	2.1 (0.8-	0.15			0.7 (0.2-	0.5
e non-	5.5)				2.1)	
RFA						
Major	1.97	0.001	1.8 (1.1-	0.02	1.98	0.02
complicati	(1.3-2.9)		2.9)		(1.13-	
on					3.5)	
Acute	0.77	0.04			0.51	0.001
complete	(0.6-				(0.35-	
success	0.99)				0.75)	

Abbreviations as above.

Figure 1: Scar patterns.

Schema for classification of scar regions and scar patterns in storm versus non-storm substrates. (A) Schema for classification of scar regions based on the description by Josephon et al (permissions granted).²⁷ On the left panel, the left ventricle is shown, opened anterolaterally. The region labeled 1 was defined as apical, 2-4 were defined as the septal, 5-8 as inferior, 10 as lateral/peri-mitral; 9, 11 and 12 as anterior and the shaded region as the left ventricular outflow tract (LVOT) and aortic-mitral continuity (AMC) region. On the right panel, the right ventricle is opened anteriorly. The region labeled 13 was defined as peri-TA; 15 and 16 as right ventricular (RV) septum, 17 as per-RV outflow tract (RVOT), 18 as RV free wall and the hatched area (marked with *) as the RV inferior wall. (B) scar patterns in

patients with storm versus those without in ischemic cardiomyopathy (ICM) and non-ischemic cardiomyopathy (NICM; C, D) and in ischemic storm versus non-ischemic storm patients (E).

AMC denotes aortic-mitral continuity, LVOT denotes left ventricular outflow tract, PM-peri-mitral, RV-right ventricle, RVOT-right ventricular outflow tract, TA-tricuspid annulus.

(A)

Author I

Author A

Figure 2: Outcomes in follow-up.

Comparison of ventricular arrhythmia (VA)-free survival (A) and survival free of death or transplant in storm vs. non-storm patients with ischemic (B) and non-ischemic cardiomyopathy.

	No. at risk								
Storm	186	114	95	87	69	60	52	44	33
Non-Storm	368	282	238	208	159	130	102	80	63

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	Time (months)								
	No. at risk								
Storm	101	17	7	6	6	5	4	3	2
Non-Storm	268	77	49	39	32	27	20	14	10

	Time (months)								
	No. at risk								
Storm	101	59	42	36	34	31	25	18	11
Non-Storm	268	202	178	164	141	122	100	88	71

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Figure 3: Outcomes in Ischemic versus non-ischemic storm.

Comparison of ventricular arrhythmia (VA)-free survival (A) and survival free of death or transplant (B) in ischemic versus non-ischemic storm.

	No. at risk								
ICM Storm	186	114	95	87	69	60	52	44	33
NICM Storm	101	59	42	36	34	30	25	18	12

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Figure 4: Recurrent storm after index ablation procedure.

Ventricular arrhythmia (VA)-free survival and survival free of death or transplant in with vs. without recurrent storm after the index procedure in patients with ischemic (A, B respectively) or non-ischemic cardiomyopathy (C, D, respectively).

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Title:

Beyond the Storm: Comparison of Clinical Factors, Arrhythmogenic Substrate, and Catheter Ablation Outcomes in Structural Heart Disease Patients With versus Those Without a History of Ventricular Tachycardia Storm.

Date:

2017-01

Citation:

Kumar, S., Fujii, A., Kapur, S., Romero, J., Mehta, N. K., Tanigawa, S., Epstein, L. M., Koplan, B. A., Michaud, G. F., John, R. M., Stevenson, W. G. & Tedrow, U. B. (2017). Beyond the Storm: Comparison of Clinical Factors, Arrhythmogenic Substrate, and Catheter Ablation Outcomes in Structural Heart Disease Patients With versus Those Without a History of Ventricular Tachycardia Storm.. J Cardiovasc Electrophysiol, 28 (1), pp.56-67. <https://doi.org/10.1111/jce.13117>.

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