Electrical storm in patients with implantable cardioverter-defibrillator in the era of catheter ablation: Implications for better rhythm control

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BACKGROUND The modern era of cardiology has changed the population of implantable cardioverter-defibrillator (ICD) recipients. Identifying predictors of electrical storm (ES) in contemporary ICD patients could improve risk stratification, therapeutic strategies, and mortality.

OBJECTIVE The purpose of this study was to address these points in a real-world setting.

METHODS In 330 consecutive patients (65 ± 11 years, 81% male, left ventricular ejection fraction 29% ± 9%) with ICD implanted because of ischemic (n, 204) or nonischemic dilated cardiomyopathy (n, 126), we analyzed the prevalence, predictors, and outcome of ES (≥3 separate VT/VF episodes within 24 hours) therapy.

RESULTS During a median of 21 months (range 17–36 months), 23 patients (7%) had ES. Secondary prevention (61% vs 24%, $P < .01$), single-chamber devices (57% vs 38%, $P = .02$), and prior appropriate (96% vs 24%, $P < .001$) and inappropriate (30% vs 9%, $P = .004$) therapies were more prevalent in these patients. In ES patients, first appropriate therapy occurred more often in the first year after implantation than in the rest of the cohort (85% vs 45%, $P = .008$), and mortality was significantly higher (22% vs 2%, $P < .001$). Multivariate Cox regression analysis showed that secondary prevention (hazard ratio [HR] 2.83, 95% confidence interval [CI] 1.21–6.61, $P = .016$) and prior appropriate (HR 88.99, 95% CI 11.73–675, $P < .001$) and inappropriate (HR 2.83, 95% CI 1.14–7.0, P = .04) therapies were independent predictors of ES.

CONCLUSION ES is not uncommon in ICD recipients. A secondary prevention indication and the occurrence of both appropriate and inappropriate ICD therapies increase the risk for ES. Prompt initiation of aggressive treatment, especially catheter ablation, should be considered for these patients.

KEYWORDS Catheter ablation; Electrical storm; Implantable cardioverter-defibrillator; Shocks; Predictors

ABBREVIATIONS **AAD** = antiarrhythmic drug; **AF** = atrial fibrillation; **ATP** = antitachycardia pacing; **CI** = confidence interval; **CRT** = cardiac resynchronization therapy; **DCM** = nonischemic dilated cardiomyopathy; **ES** = electrical storm; **HR** = hazard ratio; **ICD** = implantable cardioverter-defibrillator; **ICM** = ischemic dilated cardiomyopathy; **LVEF** = left ventricular ejection fraction; **VF** = ventricular fibrillation; **VT** = ventricular tachycardia

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Introduction

Electrical storm (ES) is a devastating, life-threatening event that has become more commonly seen in today's clinical practice. Characterized by multiple episodes of ventricular tachycardia (VT) or ventricular fibrillation (VF), ES represents an unstable condition that remains challenging in terms of management and prevention. As a result of the wide use of implantable cardioverter-defibrillator (ICD) and modern therapy improvements, ICD recipients now survive longer and run a higher risk for recurrent arrhythmias.[1–3]

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ICD recipients with impaired systolic function or a previous history of arrhythmias are at increased risk for ES and cardiac death.[4] Whether ES is a causal factor or just an epiphenomenon is still unclear, although it is undisputable that repetitive shocks may provoke myocardial damage and contribute to further deterioration of the underlying disease. Nevertheless, ES exposes patients to great physical and psychological stress, whose impact on clinical outcome should not be underestimated.[1–3] Therefore, identifying ES predictors in a real-world setting would facilitate risk stratification and clinical therapy of these patients.

Previous studies have attempted to identify ES predictors, including secondary prevention, atrial fibrillation (AF), ejection fraction, renal insufficiency, and other potential triggering factors such as worsening of heart failure, emotional stress,

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alcohol excess, electrolyte abnormalities, and myocardial ischemia.[5–10] However, no independent predictors have been reproducibly identified, and the role of prior ICD therapies has not yet been sufficiently addressed.[3,4]

Therefore, we aimed to identify ES predictors in ICD recipients in a real-world setting, with a focus on prior ICD therapies and their impact on outcome and prognosis in the era of VT catheter ablation.

Methods

Patients

Consecutive patients (n, 337) undergoing an ICD implant in 2009–2011 were included in our institutional registry. Patients with hypertrophic obstructive cardiomyopathy (n, 5) and channelopathy (n, 2) were excluded, so the final study population comprised 330 patients with ischemic dilated cardiomyopathy (ICM, n, 204 [62%]) or non-ischemic dilated cardiomyopathy (DCM, n, 126 [38%]). ICM was defined as reduced left ventricular ejection fraction (LVEF) associated with significant coronary vessel obstruction, a history of myocardial infarction, or a history of coronary intervention. DCM was defined as reduced LVEF in the absence of ischemic, hypertrophic, or other clear etiology of cardiomyopathy. All data were collected in accordance with the Declaration of Helsinki, and the study was approved by the institutional research committee.

Echocardiography

Transthoracic echocardiography data were acquired before ICD implantation using a commercially available system (Vivid-9 General Electric Vingmed, Milwaukee, WI). Left ventricular end-diastolic and end-systolic volumes were assessed from the apical 2- and 4-chamber images, and LVEF was calculated according to the Simpson method.

ICD programming

Device interrogation was performed regularly (every 4–6 months) or on demand after ICD shocks or after a symptomatic event in an outpatient clinic. Rhythm adjudications were performed based on rate analyses, onset, stability, regularity, morphology, and atrioventricular disassociation by 2 experienced physicians (SR, MD). ICDs were programmed according to the current manufacturer recommendations for the optimal arrhythmia detection and therapy, including discrimination algorithms when available: Morphology Discrimination plus AV Rate Branch (St. Jude Medical), PR logic and Wavelet (Medtronic), SMART (Biotronik), or Rhythm ID (Boston Scientific and Guidant). The ICD therapy was defined as either antitachycardia pacing (ATP) or ICD shock. The ventricular fibrillation (VF) zone was typically set to >200 bpm with at least 1 train of ATP before shock whereas the ventricular tachycardia (VT) zone typically was >170 bpm with at least 3 trains of ATP before shock. The monitor zone was set to >150 bpm and atrial arrhythmia detection to >170 bpm with tachycardia discriminators enabled (according to the Pain-FREE trial) as previously described.[11] An ICD therapy delivered for VT or VF was defined as appropriate, and all other episodes were deemed as inappropriate. Device programming remained unchanged in all patients until therapies were delivered or an ablation procedure was preformed, at which point patient-specific programming changes were implemented.

ES Definition and Therapy

ES was defined as ≥ 3 separate episodes of VT/VF within 24 hours, separated by bouts of normal rhythm after a successful therapy, either ATP or shock.[1] To qualify as ES, the 3 episodes could not be continuous VT/VF in which device therapy was unsuccessful or VT below detection that is untreated.

ES treatment was based on physician's preference and when possible on the treatment of reversible causes. Conservative treatment included admission to the intensive care unit, electrolyte substitution, recompensation, revascularization, and beta-blocker and antiarrhythmic drugs (AADs) as necessary. Amiodarone or lidocaine were the first-choice in the acute phase and oral amiodarone or sotalol were preferred for chronic management. Hypokalemia was preferably treated by substitution, whereas premature ventricular contractions were rather ablated.

Catheter Ablation Procedure

VT ablation was performed for patients not responding to AAD or as a first-choice for recurrent or incessant arrhythmias, as previously described.[12,13] After providing signed informed consent, patients were studied while under deep propofol sedation with continuous invasive monitoring of arterial blood pressure and oxygen saturation. The left ventricle was accessed through a transseptal approach using a steerable introducer (Agilis, St. Jude Medical, St. Paul, MN). Electroanatomic maps were obtained while patients were in sinus rhythm (CARTO 3, Biosense Webster Inc, Diamond Bar, CA; or EnSite, St. Jude Medical, Minneapolis, MN). Ablation was performed using 3.5-mm saline-irrigated catheters (Navistar ThermoCool, Biosense Webster; or Celsius ThermoCool, Biosense Webster, 40–50 W, 30 mL/min) and a multichannel recording system (Prucka Cardiolab, GE, Milwaukee, WI). Isovoltage maps were constructed, and areas with healthy tissue (>1.5 mV), dense scar (<0.5 mV), or fragmented, late potentials were annotated. If not incessant, VT was induced with programmed stimulation and activation or entrainment mapping was performed to locate exit sites and critical isthmuses. For hemodynamically unstable VTs, activation and pace-mapping were used and substrate modification was based on local potentials. Epicardial approach was used in 1 case after an unsuccessful prior endocardial ablation. Ablation end-points were elimination of the clinical (partial acute success) or any induced VT (complete acute success).

In order to calculate the impact of ES therapy on ICD therapies, total follow-up time (3.4 ± 1.1 years) was