

Catheter ablation of drug refractory electrical storm in patients with ischemic cardiomyopathy: A single center experience

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ABSTRACT

Objective: Electrical storm (ES) is a life-threatening pathology that requires immediate and effective treatment due to increased morbidity and mortality. Catheter ablation (CA) is an effective therapeutic option, particularly in patients with drug resistant ventricular arrhythmia episodes. These procedures should only be performed in highly specialized and experienced centers. Here we aimed to assess safety and efficacy of CA in a relatively large cohort with ES in our tertiary center hospital.

Methods: A total of 44 patients (90.9% male; mean age: 59.7±10.3 years) with ischemic cardiomyopathy undergoing CA for drug-refractory ES were prospectively evaluated. Procedures were performed using non-contact and electro-anatomic mapping systems. Long-term follow-up analysis addressed the predictors of ES recurrence and cardiac mortality.

Results: Acute success rates for clinical and non-clinical VTs were 90.8% and 55.5%, respectively. A mean follow-up at 28±11 months revealed cardiac mortality in 8 (18%) patients, 39 (88.6%) patients were free from the ES, and 24 (55%) patients remained free from both ES and paroxysmal VT episodes. In multivariate regression analysis, recurrence of ES (OR=3.11, 95% CI: 1.65-4.62, p=0.001), LVEF, and serum creatinine were found as independent predictors of cardiac mortality. In addition, substrate based ablation, implantation of ICD for secondary prophylaxis, LVEF, and serum creatinine were good predictors of ES recurrence.

Conclusion: Catheter ablation for ventricular arrhythmias in the course of ES in patients with ischemic cardiomyopathy is safe with an acceptable success rate. (*Anatol J Cardiol* 2016; 16: 159-64)

Keywords: electrical storm, ventricular arrhythmia, catheter ablation

Introduction

Ventricular arrhythmias either ventricular tachycardia (VT) or ventricular fibrillation (VF) are the most common cause of sudden cardiac death (1). Implantable cardioverter-defibrillator (ICD) implantation is the best therapeutic option in high risk individuals. ICD implantation is most commonly performed for primary or secondary prophylaxis in patients with ischemic cardiomyopathy. Antiarrhythmic drugs and catheter ablation (CA) are reserved for recurrent ventricular arrhythmias after implantation of ICD. Electrical storm is defined as 3 or more sustained episodes of ventricular tachyarrhythmias or appropriate ICD shocks during a 24-h period (1). In such cases, drugs may fail in terminating VT. Repeated ICD shocks have physiological and psychological side effects. Furthermore, CA for incessant VT can be life-saving and reduce arrhythmic events in patients who have ICD (1, 2). CA reduces VT episodes in approximately 70% patients (2). However, it has been reported that approximately

26%-50% of patients undergoing CA have experienced at least one VT episode during follow-up (3-5). Furthermore, the mortality of CA is approximately 3%, and 1-year mortality after CA is 18% (5). However, CA for ventricular tachyarrhythmias in patients who have had ES due to ischemic cardiomyopathy plays an essential role in treatment. In this paper, we aimed to present our experience and follow-up results of CA for ES in a relatively large cohort with ischemic cardiomyopathy at our tertiary center hospital.

Methods

A total of 44 patients (90.9% male; mean age: 59.7±10.3 years) with ischemic cardiomyopathy were enrolled who underwent CA for ES between January 2010 and November 2013 at the Cardiology Clinic of Türkiye Yüksek İhtisas Training and Research Hospital. We performed a retrospective analysis of prospectively collected data. The institutional Ethics Committee approved

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the study protocol and informed consent was obtained from all patients. ES was defined as the occurrence of ≥ 3 episodes of VTs separated by >5 min during a 24-hour period, each resulting in an appropriate shock by the ICD. We included patients with ES who were refractory to chronic antiarrhythmic drugs. Patients with acute ischemia/infarction, drug toxicity, and electrolyte imbalance were excluded. Coronary angiography was performed in 25 of 44 patients (56.8%) in whom myocardial ischemia was suspected. In all patients, coronary revascularization was excluded on the basis of the absence of stenosis ($>50\%$) in any coronary vessel tributary of viable myocardium identified by nuclear imaging techniques. Chronic antiarrhythmic medication was defined as the use of a drug for more than 3 months. All patients received additional pharmacological therapy to be stabilized, as a first step before ablation (for ≤ 24 h). Any patient who experienced hemodynamic deterioration during this interval or ≥ 3 shocks after a stabilization interval for 24 h was defined as drug refractory and underwent CA immediately.

All patients were hospitalized and continuously monitored at the coronary care unit and 12-lead electrocardiograms were taken during VT for defining clinical VT. Ischemic VT was defined if patient had monomorphic VT and a history of acute myocardial infarction or occlusion in at least one of the major coronary arteries. This definition was also confirmed by voltage mapping during ablation.

All procedures were carried out via the endocardial approach. Routine catheters in position included quadripolar catheters at the bundle of His and right ventricle (RV) apex and a coronary sinus catheter. In general, the left ventricle was accessed via the retroaortic route. However, we accessed it via the transseptal route in two patients due to severe peripheral artery disease. In addition, the procedure was performed with the support of intra-aortic balloon counter pulsation in three patients. Unfractionated heparin (intravenous bolus) was given for anticoagulation and, subsequently, ACT of 350–400 s was maintained. Induction of VT was achieved using programmed ventricular stimulation. If tolerated, it was mapped during VT. In patients with substrate-directed ablation, induced VT was terminated by pacing or cardioversion and used as a template for pace-mapping. Substrate-based ablation was carried out in patients who were unable to tolerate VT. Activation mapping and pace mapping were applied to define culprit regions. Induced VTs, other than clinical VTs, were also targeted for ablation. Electroanatomic maps were formed using CARTO (Biosense Webster, Diamond Bar, CA, USA), NAVX (St Jude Medical, Minneapolis, MN, USA), and non-contact mapping (Ensite Array St Jude Medical, Minneapolis, MN), with a greater density of sampling in the regions of low voltage (<0.5 mV) and border zone tissue (0.5–1.5 mV). Three-dimensional mapping of the endocardial substrate for scar (defined by bipolar voltage amplitudes <1.5 mV) and late potentials was performed (6). The mapping catheter was placed at an affected site that had pace-mapping characteristics of an exit or potential isthmus site, and VT initi-

ated to assess the electrograms during VT, entrainment mapping, and potential RF ablation during VT to assess VT termination. Ablation targeted the scar border zone, particularly at sites of matching pace-maps ($>10/12$ match), sites within low-voltage areas with late potentials, double potentials, wide-fractionated potentials during sinus or paced rhythm. If the circuit could not be found, ablation was performed through the probable exit based on voltage mapping combined with pace mapping. An open-irrigated ablation catheter (3.5 mm tip Thermocool or Thermocool SF, Biosense-Webster, Diamond Bar, CA, USA) was used. Catheter ablation used 30-50 watts, targeting temperatures of 43°C (limited to 47°C), and irrigation rate of 7-30 mL/min for 60 s per site. Power output was decreased if an impedance drop of $>10\ \Omega$ was observed. In addition, loss of capture (10 mA at 2 ms) and reduction of electrocardiogram amplitude were considered as the formation of a lesion. Programmed stimulation was used to assess the acute result of catheter ablation, defined by the inducibility of clinical and nonclinical VTs.

After the procedure, patients were monitored in the coronary care unit with 24 h telemetry; they were then discharged unless any VT re-occurred in the following week. Post-ablation follow-up visits were conducted at 1, 3, and 6 months and every 6 months, thereafter. All the patients were prescribed a beta blocker indefinitely and amiodarone for 6 months, following ablation. Study end-points were defined as ES recurrence and cardiac death.

Statistical analysis

Analyses were performed using SPSS Statistics, version 17.0 (SPSS Inc, Chicago, IL, USA). To test the distribution pattern, the Kolmogorov-Smirnov method was used. Data were summarized as the mean \pm standard deviation, median, or proportions. Cox proportional hazards regression was used to test the effect of the explanatory variables on ES recurrence and cardiac mortality, adjusted for other variables. A p value of <0.05 was considered statistically significant.

Results

Baseline characteristics of the study population are shown in Table 1. The mean number of antiarrhythmic drugs that were used was 2 ± 0.5 ; in addition, chronic antiarrhythmic medications used before ES are presented in Table 1. Among 10 patients who were already using amiodarone and beta blocker chronically, five of them directly underwent CA without any delay due to hemodynamic instability and the remaining 5 had add-on therapy with mexiletine. Amiodarone was administered for ES in 3 patients using sotalol, chronically. Among the patients on chronic beta-blocker therapy, amiodarone was administered during ES in 25 patients and mexiletine was added in 6 patients due to side effects with amiodarone.

Procedural characteristics are also presented in Table 2. During the procedure, all patients had inducible VTs and all

Table 1. Baseline characteristics of the study population (n=44)

Age, years, mean±SD	59.7±10.3
Male, n (%)	40 (90.9%)
NYHA Class, n (%)	
I	1 (2.3%)
II	28 (63.6%)
III	11 (25%)
IV	4 (9.1%)
CABG history, n (%)	25 (56%.1)
PCI history, n (%)	19 (43.1%)
Patients with ICDs, n (%)	
Primary prophylaxis	29 (66%)
Secondary prophylaxis	15 (34%)
Number of clinical VT/patient, mean±SD	17±5
Number of appropriate ICD shocks per day, n mean±SD.	15±4
LVEF, %, mean±SD	27.8±8.3
Myocardial infarction location, n (%)	
Anterior	30 (68.1%)
Inferior	14 (31.8%)
Baseline rhythm	
Sinus rhythm	34 (77.2%)
Atrial fibrillation	10 (22.7%)
Serum creatinine, mg/dL, mean±SD	1.8±0.9
Hypertension	18 (40.9%)
Diabetes Mellitus	16 (36.3%)
Medications, n (%)	
ACE inhibitors or ARBs	39 (88.6%)
Beta-blockers	41 (93.1%)
Amiodarone	38 (86.4%)
Sotalol	3 (6.8%)
Mexiletine	11 (25%)
ACE - angiotensin converting enzyme; ARB - angiotensin receptor blocker; CABG: coronary artery bypass graft; ICD - implantable cardioverter defibrillator; LVEF - left ventricular ejection fraction; NYHA: New York Heart Association; PCI - percutaneous coronary intervention	

clinical VTs had been induced. The mean number of induced VTs was 1.5±0.7 with clinical ones that had 354±38 msec cycle lengths. Eighteen (41%) patients had inducible non-clinical VTs, and ablation was performed during VT in 32 patients. Of 32 patients, 28 underwent successful ablation during VT. Four of 32 patients underwent the second procedure as a result of recurrence during index hospitalization after the first procedure. However, 12 of 44 (27%) patients could not tolerate VT hemodynamically; hence, substrate-based ablation was performed for them. Among the patients who underwent substrate-based ablation, VT was rendered non-inducible in 5 patients after ablation. However, the remaining 7 patients needed extensive ablation with circumferen-

Table 2. Procedural and follow-up characteristics of the study population (n=44)

Patients induced clinical tachycardia	44 (100%)
Patients Induced non clinical tachycardia	18 (41%)
System used	
Non contact mapping	12 (27.2%)
Electro-anatomic mapping	32 (72.7%)
VT induced per patient±SD	1.5±0.7
VTCL of clinical VT(ms)±SD	354±38
Total RF application±SD	45±28
Total procedure time, (min)±SD	117±51
Fluoroscopy time, (min)±SD	15±9
Acute success for clinical VTs, %	90.8%
Acute success for non- clinical VTs, %	55.5%
Complications, 6.8%	6.8%
VT - ventricular tachycardia; VTCL - ventricular tachycardia cycle length	

tial ablation of the dense scar zone. Four of them continued to have inducible clinical VT at the end of procedure. In 18 patients, different types of tachycardias were also induced; in 8 of these 18 patients, these tachycardias were targeted and could not be ablated. During hospital stay, 5 patients experienced VTs (repeat of ES in 3 patients and one VT episode in 2 patients) that have already been included in the unsuccessful ablation group. Second ablation procedure was performed for 3 patients with repeated ES, which were resulted in successful ablation. Time taken for total procedure and fluoroscopy was 117±51 and 15±9 min, respectively. Pericardial effusion developed in 3 patients, and all were treated with pericardiocentesis. There were no procedure-related mortalities. All patients were given amiodarone and beta blockers for 6 months after the procedure.

During long-term follow up (mean, 28±11 months), cardiac mortality was observed in 8 (18%) patients (progressive worsening of heart failure in 5 patients and sudden cardiac death in 3). A total of 5 (11.3%) patients were re-admitted with ES. In addition, 15 (34%) patients experienced paroxysmal VT episodes terminated by antitachycardia pacing or shocks, delivered by ICDs. The remaining 39 (88.6%) patients showed no ES; a total of 24 (55%) patients remained free from both ES and paroxysmal VT. Cox proportional hazards regression analysis revealed that recurrence of ES was a good predictor of cardiac mortality (OR=3.11, 95% CI: 1.65-4.62, p=0.001) and Kaplan–Meier event-free survival estimates, according to recurrence of ES is presented in Figure 1. Moreover, lower left ventricular ejection fraction (OR=0.66, 95% CI: 0.51-0.82, p=0.003) and serum creatinine (OR=2.23, 95% CI: 1.75-2.73, p=0.004) were also independent predictors of cardiac mortality (Table 3).

Among the various risk factors, substrate-based ablation, implantation of ICD for secondary prophylaxis, lower left ventricular ejection fraction, and serum creatinine were revealed to be the independent predictors of ES recurrence (Table 4).

Table 3. Cox proportional-hazards analysis showing predictors of mortality

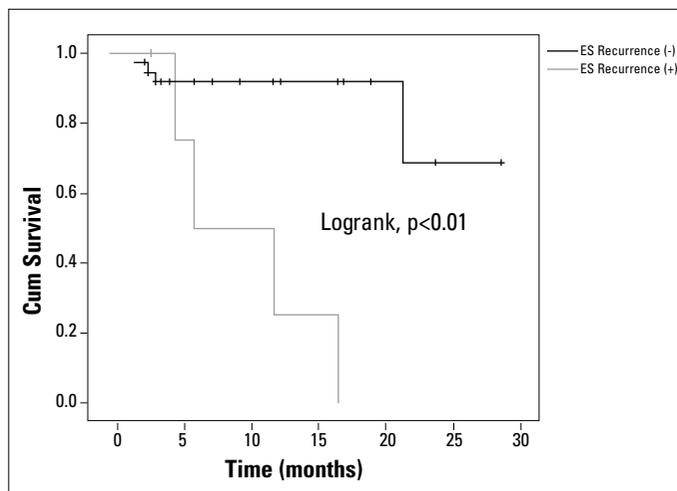
Variables	Simple HR (95% CI)	P	Multiple HR (95% CI)	P
VT Recurrence	5.21 (2.23-10.4)	0.004	3.11 (1.65-4.62)	0.001
Creatinine	3.22 (1.64-5.30)	0.009	2.23 (1.75-2.73)	0.004
VT Cycle length	1.03 (1.0-1.36)	0.038	0.98 (0.92-1.04)	0.356
Diabetes mellitus	1.40 (0.3-6.50)	0.068	-	-
Smoking	3.33 (0.68-16.3)	0.138	-	-
Hypertension	1.05 (0.84-1.27)	0.453	-	-
Ejection fraction	0.78 (0.63-0.96)	0.02	0.66 (0.51-0.82)	0.03
Age	1.00 (0.98-1.08)	0.837	-	-
NYHA >2	1.20 (0.25-5.88)	0.822	-	-
Secondary prophylaxis	1.59 (1.11-2.07)	<0.001	1.45 (1.20-1.72)	0.002

ICD - implantable cardioverter defibrillator; LVEF - left ventricular ejection fraction; NYHA - New York Heart Association; VT - ventricular tachycardia

Table 4. Cox proportional-hazards analysis showing predictors of electrical storm recurrence

Variables	Simple HR (95% CI)	P	Multiple HR (95% CI)	P
Age, years	1.06 (0.88-1.19)	0.411	-	-
NYHA >2	1.12 (0.90-1.34)	0.384	-	-
Ejection fraction, %	1.05 (1.03-1.07)	<0.001	1.04 (1.02-1.06)	0.001
Creatinine, mg/dL	2.55 (1.62-3.40)	0.001	1.99 (1.60-2.41)	0.001
ICD for secondary prophylaxis	1.55 (1.14-1.97)	<0.001	1.62 (1.34-1.91)	0.002
Substrate based ablation	2.52 (1.73-3.31)	<0.001	2.06 (1.51-2.60)	<0.001

ICD - implantable cardioverter defibrillator; LVEF - left ventricular ejection fraction; NYHA - New York Heart Association

**Figure 1. Kaplan-Meier survival curve demonstrating the impact of recurrence of electrical storm on survival (Green line indicates survival of patients with electrical storm recurrence)**

Discussion

This study demonstrates the largest number of patients with ischemic cardiomyopathy, undergoing CA for drug-refractory ES in Turkey. Our study findings were parallel to previously reported few studies with regard to clinical and procedural efficacy. Lower left ventricular ejection fraction, serum creatinine, implantation of ICD for secondary prophylaxis, and substrate-

based ablation were revealed to be independent predictors of ES recurrence. In addition, we showed that recurrence of ES during follow-up was a significant and independent predictor of cardiac mortality.

Ischemic heart disease with reduced left ventricular ejection fraction poses a major negative impact on survival. To neutralize the negative consequences of the same, ICD implantation is one of the therapeutic options that improves survival. However, it has been known that frequent ICD shocks due to recurrent VT episodes increase mortality (7). In addition, recurrent shocks cause depression, post-traumatic syndrome, and reduce the quality of life; in addition, ICDs do not provide definite protection against sudden cardiac death (8). Therefore, several studies have been carried out to assess the beneficial effects of CA for ventricular arrhythmias before ICD implantation. These studies reported delayed recurrence of VT and reduced incidence of ICD therapy, in patients who underwent CA (3, 6). Electrical storm is a well-known cause of emergency service applications in ICD implanted patients and is associated with decreased survival (9). In a recent meta-analysis, ES was revealed to be a major risk factor for mortality and adverse cardiac events (10).

The cause of increased mortality by ES might be related to progressive decline of cardiac function due to direct damage to myocardium as a result of repeated shocks; moreover, low-output states may further deteriorate cardiac contractility and worsen renal and hepatic function (11). In a recent study, it was

revealed that the occurrence of ES could be seen as a warning sign of imminent pump failure or even as a manifestation of overt heart failure (12). Patients are not free from subsequent ES after surviving one episode. The incidence of subsequent ES is high in patients who have previously experienced ES (2, 13).

Antiarrhythmic medications are administered at first in the management of ES. However, they frequently do not suffice to overcome the arrhythmic burden (14). CA is the first-line treatment in the concomitant presence of ischemic cardiomyopathy and ES when chronic antiarrhythmic drugs fail. Short term results of CA are favorable if the procedure has been completed without any inducible VT and approximately all the patients recover uneventfully after hospital discharge (2). CA has short-term stabilization effects even in patients with inducible VT. Long term results of CA are also promising with regard to VT recurrences, and CA also improves patient survival (2). However, there was also contradictory reports showing that CA decreases only the recurrence of ES but no benefit in survival, when patients with a left ventricular ejection fraction >25% have been included (15).

The long-term success of CA depends on the inducibility of both clinical and other VTs (2, 16, 17). The absence of any inducible VT enhances long-term success rate. The objective should not be solely restricted to clinical VTs. Survival rate of patients who have undergone successful CA was higher due to the absence of ES (2). This hypothesis was supported by previous studies that demonstrated higher mortality in ES patients without CA (18-20).

The ablation of VTs in ischemic cardiomyopathy before implantation of ICD demonstrated less device intervention with delayed events of shocks in non-ES settings. However, no survival benefit was gained. Additionally, ablation did not provide any advantage on the emergence of ES (21). In the analysis of outcomes of prophylactic VT ablation after ICD implantation, the occurrence of device intervention during follow-up was only associated with the number of VTs induced during the electrophysiology study. The induction of VT after ablation did not predict the occurrence of device intervention (22).

Our experience with CA for ES in ischemic cardiomyopathy demonstrated similar success rates with previously published literature. The recurrence of ES was found as an independent predictor of cardiac mortality which was also consistent with previous studies. Our study findings confirmed previously published papers regarding the negative impact of ES on survival. When other antiarrhythmic drugs fail, CA remains the only therapeutic option. However, CA may be opted for without the administration of antiarrhythmic drugs, considering the low recurrence rate associated with it. Moreover, electroanatomic mapping systems serve for the application of procedures without fluoroscopy which means better protection for the surgeon and the patient. Procedural risk is low in experienced hands; hence, in our study, majority of the patients remained free from ES in the long-term. Left ventricular ejection fraction and renal

functions were found to be the other independent predictors of cardiac mortality. The inducibility of VT should be checked after ablation for substrate based and non-substrate based approaches. According to our findings, substrate-based ablation seems to be a predictor for VT recurrence, according to Cox proportional hazard analysis; however, this may be due to undetected patient-related factors. Substrate-based ablation has good outcomes for VT recurrence.

In our study population, pericardial effusion was observed in three patients; this should be considered with regard to safety issues. We did not experience a sudden reduction in impedance during the ablation procedure. However, our study population only included patients with ischemic cardiomyopathy in whom there was a chance of myocardial wall thinning. Thus, we thought that such an important complication might be due to damage at the site of regional myocardial wall thinning in our patients.

In this study, our aim is to examine the success of ablation for ES and its long term impacts on mortality and morbidity and to present our preliminary results. There have been few studies that investigated the role of CA for ES in ischemic cardiomyopathy patients. However, the number of patients, ablation technique administered, and predictors for ES recurrence and mortality were highly variable. To our knowledge, this study is the first with a large sample size that elucidates the experience of CA for ES that is refractory to antiarrhythmic medication in Turkey.

Study limitations

This is a single center study of a relatively small cohort of patients. Large-scale controlled studies are required to better delineate the predictors of ablation success and mortality among patients with ischemic cardiomyopathy undergoing CA for ES.

Conclusion

Our study findings showed that CA for ES in patients with ischemic cardiomyopathy may serve as an effective and safe therapeutic option, and they should be applied when chronic antiarrhythmic drugs fail. However, these procedures should only be performed in highly specialized and experienced centers.

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