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Catheter Ablation of Refractory Ventricular Fibrillation Storm After Myocardial Infarction: A Multicenter Study

Running Title: *Komatsu & Hocini et al.; Ablation of VF Storm After Myocardial Infarction*

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Abstract

Background: Ventricular fibrillation (VF) storm after myocardial infarction (MI) is a life-threatening condition that necessitates multiple defibrillations. Catheter ablation is a potentially effective treatment strategy for VF storm refractory to optimal medical treatment. However, its impact on patient survival has not been verified in a large population.
Methods: We conducted a multicenter, retrospective observational study involving consecutive patients who underwent catheter ablation of post-MI refractory VF storm without preceding monomorphic ventricular tachycardia. The target of ablation was the Purkinje-related ventricular extrasystoles triggering VF. The primary outcome was in-hospital and long-term mortalities. Univariate logistic regression and Cox proportional-hazards analysis were used to evaluate clinical characteristics associated with in-hospital and long-term mortalities, respectively.
Results: One-hundred ten patients were enrolled (65±11years; 92 men; left ventricular ejection fraction [LVEF] 31±10%). VF storm occurred at acute phase of MI (4.5±2.5 days after the MI onset during index hospitalization for MI) in 43 (39%) patients, subacute (>1 week) in 48 (44%), and remote (>6 months) in 19 (17%). The focal triggers were found to originate from the scar border zone in 88 (80%) patients. During in-hospital stay after ablation, VF storm subsided in 92 (84%) patients. Overall, 30 (27%) in-hospital deaths occurred. The duration from the VF occurrence to the ablation procedure was associated with in-hospital mortality (odds ratio for each one-day increase: 1.11; 95% confidence interval [CI]: 1.03-1.20; p=0.008). During follow-up after discharge from hospital, only one patient developed recurrent VF storm. However, 29 (36%) patients died with a median survival time of 2.2 years (interquartile range: 1.2–5.5 years). Long-term mortality was associated with LVEF<30% (hazard ratio [HR]: 2.54; 95%CI: 1.21-5.32; p=0.014), New York Heart Association class ≥III (HR: 2.68; 95%CI: 1.16-6.19; p=0.021), a history of atrial fibrillation (HR: 3.89; 95%CI: 1.42-10.67; p=0.008), and chronic kidney disease (HR: 2.74; 95%CI: 1.15-6.49; p=0.023).
Conclusions: In patients with MI presenting with focally-triggered VF storm, catheter ablation of culprit triggers is life-saving and appears to be associated with short- and long-term freedom from recurrent VF storm. Mortality over long-term follow-up is associated with the severity of underlying cardiovascular disease and comorbidities in this specific patient population.

Key Words: arrhythmia; ventricular fibrillation; myocardial infarction; catheter ablation

Clinical Perspective

What is new?

- This multicenter study evaluating the largest consecutive series of patients undergoing catheter ablation for refractory ventricular fibrillation (VF) storm after myocardial infarction (MI) indicates that ablation of the focal Purkinje-related triggers frequently arising from the scar border-zone at the left ventricular septum appears to be associated with short- and long-term freedom from VF storm recurrence.
- Early intervention after VF storm occurrence may lead to a reduced risk of in-hospital cardiovascular mortality.
- Long-term follow-up outcome is limited by steady increases in both cardiovascular and non-cardiovascular mortalities that are associated with severity of underlying cardiovascular disease and comorbidities.

What are the clinical implications?

- Patients with MI presenting with focally-triggered VF storm refractory to medical therapies should be transferred to a dedicated critical care unit to optimize the overall condition of these patients and to perform ablation as early as possible.
- Our observation regarding the dominant domain of VF triggers may serve as a roadmap for ablation to target the culprit Purkinje network at the specific regions.
- Careful management of heart failure and comorbidities in this specific population is of great importance, even in cases where the electrical storm subsides after catheter ablation.

Introduction

Myocardial electrical storm represents a life-threatening, malignant condition of clustering ventricular arrhythmias that necessitate multiple defibrillations. Ventricular fibrillation (VF) storm attributed to focally-triggered VF after myocardial infarction (MI) could be recognized as a distinctive arrhythmic syndrome with specific pathophysiology and lethal characteristics that differs from scar-mediated monomorphic ventricular tachycardia (VT) (1). The initial approach to patients presenting with VF storm is usually focused on exclusion of myocardial ischemia and administration of antiarrhythmic medications. However, antiarrhythmic drugs may fail to suppress VF or may be limited by contraindications or adverse effects (e.g., long QT interval, hyperthyroidism, and/or labile hemodynamic compensation and severe dysfunction of the left ventricle) (1,2). The condition often requires other therapies combined with antiarrhythmic medications, including deep sedation with mechanical ventilatory support, overdrive pacing, and/or hemodynamic support devices to stabilize patients. Neuraxial modulation with thoracic epidural anesthesia may be also effective for electrical storm management (3,4). However, despite these extensive therapeutic efforts, suppression of lethal arrhythmias can be transient.

Over the last two decades, radiofrequency catheter ablation has emerged as a potentially effective treatment strategy for post-infarct VF storm. The current guideline endorses catheter ablation for the management of therapy-resistant ischemic VF storm (5). The main targets for ablation are identifiable focal triggers of VF. The pathogenic role of the Purkinje system is very high; discrete excitations act as main triggers of VF in patients with diseased hearts (6). The role of Purkinje fibers in VF maintenance is less documented than its role in VF initiation. Nevertheless, several experimental studies have revealed Purkinje in fibrillatory wave fronts during VF (7), and computer models showed migratory

re-entrant activity at the Purkinje–muscle junction as a mechanism for maintaining early VF (8,9). Previous reports demonstrated an acute benefit of radiofrequency catheter ablation targeting specific Purkinje potentials that precede the triggers of VF (10-13). However, these reports were single center series evaluating a small number of patients. The impact of VF storm ablation on patient survival has not been verified in a large population. Therefore, we sought to investigate the acute and long-term outcomes of catheter ablation for the treatment of last resort in a large series of consecutive patients with post-MI VF storm refractory to medical therapies.

Methods

The data, analytical methods, and study materials will not be made available to other researchers for purposes of reproducing the results or replicating the procedures.

Study Population

Between January 2003 and May 2017, a total of 110 consecutive patients with VF storm following MI underwent radiofrequency catheter ablation as a last resort for refractory VF storm in 15 international tertiary care arrhythmia centers. VF storm was defined as ≥3 separated episodes of VF in 24 hours. Only patients with VF without preceding monomorphic ventricular tachycardia (VT) were included. VF storm was documented on bedside or telemetry monitors, device interrogation, or Holter recordings. The diagnosis of MI was based on medical history, clinical findings, and coronary angiography. All patients were brought to the intensive care unit and were managed by a combination of therapies, including coronary revascularization, antiarrhythmic medications, correction of electrolytes, deep sedation with mechanical ventilatory support, overdrive pacing, and/or circulatory support devices (extracorporeal membrane oxygenation and intra-aortic balloon pumping) at the discretion of the dedicated unit physicians.

When life-threatening conditions due to VF storm remained despite optimal medical therapies, the decision to transfer patients to the electrophysiological laboratory for catheter ablation was made by the dedicated cardiac team. All patients or families provided written informed consents of the ablation procedures. The institutional review boards of the participating centers approved the collection of data. This study included 29 patients who had been included in previous studies (13-15).

Mapping and Ablation Strategy

The number and type of catheters varied in individual centers and individual patients. Multipolar catheters were positioned from the femoral veins into the right ventricle and at the His bundle. Mapping and ablation of the left ventricle was performed by the transaortic (retrograde) or the transseptal approach. Three-dimensional electroanatomic mapping was performed using CARTO (Biosense Webster, Diamond Bar, CA) or NavX (St. Jude Medical, St. Paul, MN). A non-irrigated-tip catheter or an irrigation catheter was used for mapping and ablation. Systemic heparinization was maintained during mapping and ablation. General anesthesia with mechanical ventilatory support was used as part of the previous therapy or periprocedurally at the discretion of the operator.

The targets of ablation were the ventricular premature beats (VPBs) triggering VF. Purkinje potentials that preceded the onset of the triggering VPBs were frequently found around the scar border zone and were carefully identified (11-13). Additionally, pace mapping was performed to localize ablation targets. In these respects, it was essential to document the triggering VPBs on a 12-lead ECG prior to the ablation procedure. When no spontaneous VPBs were detected during the ablation procedure, they were induced by pacing maneuvers (ventricular burst or extrastimuli) with the addition of an intravenous isoproterenol (3.0–5.0

μg/min) or epinephrine (5–10 μg) infusion. Radiofrequency energy was delivered with a maximum power of 40 W and a target temperature of 42°C for the irrigation catheter and 55°C for the non-irrigated catheters. The inducibility of VF and VT was tested at the end of procedure at the operator's discretion. When attempted, pacing was performed from the RV apex with up to triple extrastimuli decrementing down to 200 ms or to refractoriness, whichever occurred first. The procedural end point was the elimination of all clinical triggering VPBs.

In-Hospital and Long-Term Follow-Up

Following the ablation procedure, patients were brought back to the intensive care unit and monitored. Intensive management to optimize the overall conditions of patients was continued following ablation procedure. Post-procedural medical treatment included beta-blockers and antiarrhythmic drugs, anticoagulation, revascularization, and management of heart failure with ventilatory support, intravenous catecholamine, hemodiafiltration, and circulatory support devices, as necessary. Patients who had no previous implantable cardioverter defibrillator (ICD) were implanted with the device before hospital discharge, with the exception of 5 patients who refused implantation. ICD programming was commonly set to include three zones: a VT zone (150–188 beats per minute; anti-tachycardia pacing attempts followed by shock), fast VT zone (188–210 beats per minute; 1 attempt of ATP and subsequently shock), and VF zone (>210 beats per minute; shock only, with antitachycardia pacing during charging, if available). Drug management and device programming during follow-up was at the discretion of the investigators. Following hospital discharge, all recruiting centers were contacted for each patient. In addition to records of clinical events, electrocardiogram, and Holter monitoring, VF and associated intracardiac electrograms were documented by ICD interrogation logs.

Statistical Analysis

Categorical variables were expressed as numbers and percentages, and were compared using chi-square or Fisher's exact tests, as appropriate. Continuous data were expressed as mean ± standard deviation or as median (interquartile range [IQR]). Recurrent VF/VT, all-cause mortality, and cardiovascular and non-cardiovascular death were assessed in-hospital and at the end of the follow-up period. Univariate logistic regression analysis was used to evaluate clinical and procedural variables associated with in-hospital mortality. Odds ratios (OR) with corresponding 95% confidence intervals (CI) and two-sided p-values were presented. Univariate Cox proportional hazards analysis was used to assess the association of clinical and procedural variables on all-cause mortality following hospital discharge, from which hazard ratios (HR) and 95% CIs were derived. Cumulative incidence curves were plotted for recurrent VF/VT, all-cause mortality, cardiovascular deaths, non-cardiovascular deaths, and death from unknown reason. All tests were 2-tailed. A probability value of <0.05 was considered statistically significant. Statistical calculations were performed with IBM SPSS Statistics 21.0.

Results

Patient Characteristics

Patient characteristics are summarized in **Table 1**. The mean age was 65 ± 11 years and 92 (84%) patients were men. At the time of VF storm occurrence, type of MI was acute in 43 (39%) patients, subacute (>1 week) in 48 (44%) patients, and remote (> 6 months) in 19 (17%). In the patients with acute MI, VF storm started 4.5±2.5 days (median 5 days [IQR: 3–5] days) after the onset of an infarction during the index hospitalization for MI. In the patients with subacute MI, VF storm started median 12 days [IQR: 10–20 days] after the onset of MI (during the index MI

hospitalization in 43 [90%] patients and after hospital discharge in 5 [10%] patients). In the patients with s remote MI, the median interval from the onset of an infarction to VF storm occurrence was 4.2 years [IQR: 1.6–12.5 years]. The mean LV ejection fraction was 31±10%, and 37 (34%) patients had severe heart failure symptoms before admission (New York Heart Association [NYHA] class ≥III). Forty-one (37%) patients presented with cardiogenic shock due to heart failure on admission. Amiodarone, class I antiarrhythmic drugs (lidocaine or mexiletine), and beta-blockers were administered to 103 (94%), 71 (65%), and 91 (83%) patients, respectively, however, they all failed to prevent VF storm. Peri-procedural hemodynamic support and deep sedation with mechanical ventilator was used in 45 (41%) and 81 (74%) patients, respectively. Catheter ablation of refractory VF storm was performed 6 ± 6 days (median 4 days [IQR: 2–8 days]) after the first occurrence of VF storm.

Eight patients underwent catheter ablation prior to coronary revascularization because of the following reasons: 1) 6 patients had remote phase of MI with chronic total occlusion and presented with VF storm without the evidence of acute infarction; 2) the remaining 2 patients with subacute MI had diffuse multi-vessel stenosis after coronary artery bypass grafting (Supplemental Table 1).

Acute Results of Ablation

Ninety-four (86%) patients presented to the laboratory with frequent, spontaneous VPBs. In the remaining 16 (14%) patients, pacing maneuvers with the administration of intravenous isoproterenol were performed to induce VPBs. The site of origin of the triggering VPBs was associated with the territory of infarction in all patients. The triggering VPBs were found to originate from the surviving Purkinje tissue in the dense scar area (a bipolar voltage <0.5mV) in 15 (14%) patients and from the scar border zone (a bipolar voltage of 0.5mV-1.5mV) in 88

(80%) patients (**Figure 1**). Although VPBs were found to originate from the normal voltage area (a bipolar voltage >1.5mV) in the remaining 7 (6%) patients, these sites also correlated with the territory of infarction. The site of origin of the triggering VPBs was located at the left ventricular septum in 78 (71%) patients, papillary muscles in 10 (9%), both the left ventricular septum and papillary muscles in 17 (15%), and other scar border areas in 5 (5%). Purkinje potentials preceding the QRS complex during both sinus rhythm and VPBs were recorded at the ablated regions in 99 (90%) patients. Ablation of Purkinje potentials preceding VPBs in addition to ablation guided by pace mapping eliminated triggering VPBs in 100 (91%) patients. A total radiofrequency energy delivery time was 22 ± 12 minutes. The mean procedure duration was 178 ± 66 minutes. Programmed stimulation at the end of procedure was not performed to prevent deterioration of the hemodynamic status in 53 (48%) patients. Among 57 patients who underwent programmed stimulation at the end of procedure, non-inducibility of VF and VT was achieved in 46 (81%) patients. VF remained inducible in 11 (19%) patients.

During the procedure, one patient died due to electromechanical dissociation. None of the patients developed pericardial tamponade. Aggravation of heart decompensation occurred in 4 (4%) patients. Cerebrovascular ischemic attacks occurred in one (1%) patient. Complete atrioventricular block occurred in one (1%) patient, while transient atrioventricular block that was not observed after the ablation procedure occurred in 3 (3%) patients. Left bundle branch block occurred in 8 (7%) patients. Groin hematoma occurred in one (1%) patient who required blood transfusion and surgery.

In-Hospital Outcomes

A flow diagram of in-hospital outcomes following ablation is shown in **Figure 2**. After the first ablation procedure, VF storm subsided in 92 (84%) patients. However, despite ablation, VF

storm was not suppressed in the remaining 18 (16%) patients. Of those patients with uncontrollable VF storm, 8 underwent a second ablation procedure. Among 92 patients in whom VF storm subsided after ablation, 24 patients developed single isolated episodes of recurrent VF or VT that were managed by a second ablation in 12 patients and by antiarrhythmic medications in 12 patients. Sixty-eight patients (62%) were free of any recurrent ventricular arrhythmias. The acute impact of ablation on VF/VT frequency is shown in **Figure 3**. Ablation of VF storm resulted in a substantial reduction of the lethal ventricular arrhythmia burden.

Overall, there were 30 (27%) in-hospital deaths with a median survival time of 7 days [IQR: 2–17 days]. **Table 2** shows the clinical and procedural variables associated with in-hospital death. Of note, the duration between the occurrence of VF storm and the ablation procedure was significantly associated with in-hospital death (for each one-day increase; OR: 1.11; 95% CI: 1.03-1.20; p=0.008). The number of in-hospital death patients increased with the time from VF storm occurrence to ablation procedure (**Figure 4**): one of 25 patients (4%) in those who underwent ablation on the day of VF storm occurrence and those with ablation the day after the VF storm, 8 of 31 patients (26%) with ablation procedure 2-4 days after VF storm, 7 of 22 patients (32%) with ablation procedure 5-7 days after VF storm, and 14 of 32 patients (44%) with ablation procedure ≥8 days after VF storm.

Acute recurrence of VF storm was strongly associated with subsequent in-hospital death (OR: 11.47; 95% CI: 3.60-36.52; p<0.001). However, the incidence of single isolated episodes of recurrent VF or VT was not associated with in-hospital death (OR: 1.13; 95% CI: 0.41-3.07; p=0.81).

Long-Term Outcomes

Long-term outcomes were available in 80 (73%) patients who were discharged from the hospital alive. All except 5 patients without a previously implanted ICD underwent device implantation before hospital discharge. Five patients refused implantation. Medications after hospital discharge included amiodarone in 46 (58%) patients, class-I antiarrhythmic agents in 10 (13%) patients, β-blocker in 76 (95%) patients, and oral anticoagulants in 19 (24%) patients. The cumulative incidences of short- and long-term recurrent electrical storm, isolated VF, and isolated VT are shown in **Figure 5A**. Of the patients who survived from a VF storm after ablation and were discharged from the hospital alive, only 1 patient developed recurrent VF storm after 22 months from the index ablation procedure. Over a median follow-up of 3.7 years [IQR: 1.4–6.5 years], isolated episode of recurrent VF occurred in 4 (5%) patients, while isolated episode of VT was documented in 9 (11%) patients and both VF and VT in 1 patient. A median recurrence survival time was 2.2 years [IQR 0.2–3.0 years].

In total, 29 (36%) patients died during the follow-up after discharge from hospital with a median survival time of 2.2 years [IQR: 1.2–5.5 years]. The causes of death are summarized in Supplemental Table 2. While short-term mortality was mainly due to cardiovascular reasons (mostly refractory heart failure), long-term mortality was due to both cardiovascular (13 patients) and non-cardiovascular (11 patients) reasons. Non-cardiovascular causes included sepsis, cancer, and stroke. The cumulative incidences of overall mortality, cardiovascular-related and non-cardiovascular-related deaths after the index ablation procedure are shown in **Figure 5B**. Cumulative incidence curve of all-cause mortality had a steep slope at the beginning mainly due to cardiovascular death, but there was a steady increase of non-cardiovascular death as well as cardiovascular death over long-term follow-up.

In Cox regression analysis (**Table 3**), mortality during follow-up after discharge from hospital was associated with LVEF<30% (HR: 2.54; 95% CI: 1.21-5.32; p=0.014), NYHA class ≥III (HR: 2.68; 95% CI: 1.16-6.19; p=0.021), a history of atrial fibrillation (HR: 3.89; 95% CI: 1.42-10.67; p=0.008), and chronic kidney disease (HR: 2.74; 95% CI: 1.15-6.49; p=0.023). None of the peri-procedural therapies and intra-procedural outcomes was associated with mortality over the follow-up period. The use of β-blocker was associated with improved survival (HR: 0.29; 95% CI: 0.085-0.97; p=0.044).

At least one hospitalization because of acute heart failure occurred in 17 (21%) patients, which led to death in 9 patients. Over the follow-up period, 4 (5%) patients developed a stroke. Six (8%) patients underwent percutaneous coronary intervention for recurrent MI without VF storm.

Discussion

This multicenter study reviewed the largest consecutive series of patients undergoing catheter ablation for therapy-resistant VF storm following MI. The data indicated that catheter ablation targeting the focal Purkinje-related triggers frequently arising from the scar border-zone at the left ventricular septum was acutely effective in suppressing VF storm in most cases; however, despite ablation, uncontrollable VF storm occurred in 16% of patients. In-hospital death occurred in 27% of patients. Uncontrollable VF storm was associated with a high risk of subsequent death, and besides severity of heart failure, time from the beginning of the storm to catheter ablation were associated with acute mortality. When ablation resulted in acute survival from a VF storm, patients only developed sporadic events of recurrent VF over the long follow-up period. However, a steady increase in mortality was noted during that period, which was associated with

poorer clinical status including LVEF <30%, NYHA class ≥ III, and a history of atrial fibrillation and chronic kidney disease.

The Timing of the Catheter Ablation Procedure

In clinical practice, ablation of VF storm is usually implemented as a last-resort strategy; however, the question regarding the optimal timing of catheter ablation remains. Although VF storm uncontrollable by ablation placed the patients at an extremely high risk of subsequent acute mortality, single isolated episodes of recurrent VF were neither associated with in-hospital death nor long-term mortality. On the other hand, the delay between the onset of the storm and ablation was associated with poor acute outcome. This is in part because of the prolonged exposure to electrical storm, which can further compromise the cardiac function and result in worsened overall conditions of patients. In this regard, the attempt to use catheter ablation for the management of VF storm (not for single isolated episodes of VF) in patients after revascularization and medical therapies should be considered earlier. The experience suggests that patient with VF storm should be transferred to a dedicated critical care unit that involves not only electrophysiologists who are experienced in VF/VT ablation but also specialists for managing heart failure, coronary interventionists, thoracic surgeons, and cardiac anesthesiologists to optimize the overall condition of these patients and to perform ablation as early as possible.

Strategy of Catheter Ablation

The primary goal of current ablation strategy for focally-triggered VF is to eliminate the Purkinje-related ectopic focus. Therefore, determining the earliest Purkinje potentials preceding the target ectopic beats is the key to successful ablation. As the pro-arrhythmic and pro-fibrillatory effects of beta-adrenergic stimulation have been demonstrated in the setting of

ischemic heart disease (16,17), administration of isoproterenol or epinephrine to induce target VPBs may facilitate ablation, especially in the case of paucity of VPBs during the procedure. Importantly, this study demonstrated that the culprit Purkinje sources were most commonly distributed over the border zone of the ischemic scar at the left ventricular septum, which is in line with the findings of previous clinical and experimental studies (10-13,18-21). This observation regarding the dominant domain of VF triggers may serve as a roadmap for ablation to target the culprit Purkinje network at the specific regions in sinus rhythm.

None of the ablation procedures performed except for one was interrupted due to complications related to hemodynamic intolerance and respiratory failure. However, atrioventricular block and left bundle branch block occurred in about 10% of the patients. It should be kept in mind that the triggering VPBs often originate from the Purkinje network at the left ventricular septum where ablation carries a potential risk of damage to the left His-Purkinje system. On the other hand, catheter ablation of VF storm is usually bail out procedure and risk of damage to conduction system should not be factor that influences ablation strategy.

Inducibility of VF and VT was tested because of the following reasons. First, patients undergoing VF ablation may develop newly emergent monomorphic VT that often originates from the Purkinje network in the ischemic scar area close to the VF ablation sites (14). Second, ablation targeting Purkinje-related triggers might modify the VF substrate. It remains to be determined whether the favorable effect of ablation was due to the elimination of VPBs triggering VF or modification of the VF substrate at the Purkinje network. Ablation targeting the Purkinje potentials in the vicinity of the ectopic focus may change the milieu for maintenance of VF. However, this could not be clarified in this retrospective observational study. Further

investigations are needed to assess the prognostic significance of noninducibility after ischemic VF ablation.

Acute and Long-Term Outcomes

Previous studies on smaller patient cohorts demonstrated encouraging outcomes of VF storm ablation, wherein nearly all patients achieved elimination of VF storm and were discharged from the hospital alive (10-13). This study provided a realistic appreciation of ablation outcomes in VF storm. Patients presenting with VF storm often have multiple comorbidities including heart failure decompensation, multiple coronary artery diseases, diabetes mellitus, atrial fibrillation, and renal dysfunction. Peri-procedural management of heart failure decompensation and hemodynamic optimization with mechanical support when clinically indicated is of great importance, even in cases where urgent catheter ablation had successfully suppressed the electrical storm.

After patients survived the acute phase of VF storm, most of them remained free from recurrent VF, as observed over the long follow-up period. This is in contrast to a prior report investigating outcomes of ablation for scar-mediated VT, where the VT recurrence remained steady over 3 years post-ablation (22). This is probably related to different characteristics of arrhythmia mechanisms. While patients with VF storm present with focal Purkinje arrhythmogenicity at the specific endocardial areas around the scar border, VT patients often have multiple coexisting VT circuits surrounded by large scars at the endocardium, epicardium, and/or deep within the myocardium, which may make a durable suppression of VT recurrence difficult to achieve in the long-term.

Previous studies have reported that the recurrence of VT after ablation of scar-mediated VT predicts a poor prognosis in the short- and long-terms (23-27). This most probably reflects

progressive heart failure associated with a worsened hemodynamic state due to irrepressible VT and negative inotropic consequences of ICD shocks for VT. Consistent with these previous studies on VT population, early recurrence of VF storm in this study was strongly associated with subsequent in-hospital death. These findings support the notion that acute suppression of VF storm by catheter ablation reduces cardiovascular mortality in the short-term. Furthermore, the long-term freedom from recurrent electrical storm may be associated with a reduced cardiovascular mortality. However, a gradual increase in long-term mortality occurred despite a low rate of recurrent life-threatening arrhythmias. While heart failure was the dominant cause of in-hospital death, long-term mortality was due to both cardiovascular and non-cardiovascular reasons and was not associated with the acute outcomes of ablation. These results underscore the importance of careful management over a long follow-up period globally beyond the use of catheter ablation.

Study Limitations

This study was not randomized and, hence, subject to the limitations inherent to observational studies. Although all patients in this study required multiple defibrillations for VF storm that was refractory to coronary revascularization and antiarrhythmic agents, the pre-procedural therapies were not uniform. The referral for catheter ablation might be influenced by the severity of arrhythmia burden and the overall conditions of patients. However, it would be difficult to conduct large randomized trials in this specific patient population because of the lethal characteristics as well as relative scarcity of cases with refractory VF storm after MI. Thus, non-randomized observational studies with a large population could be the way to serve as a source of evidence.

In this study, no patients used left ventricular assist devices that could be of potential benefit to improve the hemodynamic state, which might affect the timing of the ablation procedure. Furthermore, none of the patient underwent heart transplantation procedure. As most of patients in this study had end-stage intractable heart failure, heart transplantation may have improved their survival rates and functional statuses. Urgent catheter ablation may be performed as a bridge to heart transplantation in these sickest patients.

We also acknowledge the limited clinical data available from this observational study due to the potential substantial heterogeneity of procedures and techniques used during the ablation and management of patients after ablation. Long-term outcomes were available only in 80 (73%) patients who were discharged hospital alive. Furthermore, this study was not able to capture changes in ICD programming during follow-up. ICD re-programming according to type of recurrent arrhythmias may influence the mortality outcome (28).

Conclusions

In patients with MI presenting with focally-triggered VF storm refractory to medical therapies, catheter ablation targeting the culprit triggers is life-saving and appears to be associated with short- and long-term freedom from recurrent VF storm. The triggering ectopic activities most commonly originate from the surviving Purkinje tissue over the border zone of ischemic scar in the left ventricular septum. Early intervention after the occurrence of VF storm may lead to a reduced risk of in-hospital cardiovascular mortality. However, the outcome over long-term follow-up is limited by steady increases in both cardiovascular and non-cardiovascular mortalities. The poor long-term prognosis reflects primarily severity of underlying cardiovascular disease and comorbidities in this specific patient population.

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Table 1. Baseline characteristics

	n=110
Age, y	65 ± 11
Male	92 (84%)
Type of MI at the time of VF occurrence	
Acute MI	43 (39%)
Subacute MI	48 (44%)
Remote MI	19 (17%)
Extent of Coronary Artery Disease	
1 vessel disease	41 (37%)
2 vessel disease	31 (28%)
3 vessel disease	38 (35%)
MI site	
Anterior	81 (74%)
Inferior	22 (20%)
Multiple	7 (6%)
History of	
Hypertension	69 (63%)
Diabetes mellitus	57 (52%)
Atrial fibrillation	13 (12%)
Chronic kidney disease	27 (25%)
COPD	3 (3%)
Stroke	4 (4%)
Post-CABG	20 (18%)
LV ejection fraction, %	31 ± 10
LVEDD, mm	63 ± 10
QRS width in sinus rhythm, ms	108 ± 23
NYHA class before VF storm	
class I	50 (46%)
class II	23 (21%)
class ≥ III	37 (34%)
Cardiogenic shock on admission	41 (37%)
ICD at the time of VF occurrence	33 (30%)
Total number of defibrillation before ablation	23 ± 28, median 15 [7, 30]
≤9	37 (34%)
10-20	31 (28%)
≥21	42 (38%)
Revascularization not performed before ablation	8 (7%)
Antiarrhythmic drugs	
Amiodarone	103 (94%)
class I antiarrhythmic drugs	71 (65%)
β-blocker	91 (83%)
Overdrive pacing	41 (37%)
Periprocedural use of hemodynamic support device	45 (41%)
Periprocedural sedation with mechanical ventilator	81 (74%)
Time from VF to ablation, days	6 ± 6, median 4 [2, 8]

Abbreviations: CABG = coronary artery bypass grafting; COPD = chronic obstructive pulmonary disease; ICD=implantable cardioverter defibrillator; LV = left ventricle; LVEDD = left ventricular end diastolic diameter; MI = myocardial infarction; NYHA = New York Heart Association; VF = ventricular fibrillation. Chronic kidney disease was defined as a serum creatinine greater than 1.5mg/dL.

Table 2. Clinical and procedural variables associated with in-hospital death

	OR (95% CI)	p
Clinical data		
Age ≥70	2.51 (1.07 – 5.94)	0.035
Male gender	0.71 (0.24 –2.09)	0.53
Acute or subacute MI	1.50 (0.46 – 4.95)	0.51
Number of diseased vessels	0.99 (0.60 – 1.62)	0.96
History of		
Hypertension	1.04 (0.43 – 2.47)	0.94
Diabetes mellitus	0.91 (0.39 – 2.10)	0.82
Atrial fibrillation	2.61 (0.80 – 8.52)	0.11
Chronic kidney disease	1.48 (0.58 – 3.78)	0.42
Post-CABG	0.41 (0.11 – 1.52)	0.18
LV ejection fraction <30%	1.42 (0.61 – 3.31)	0.41
QRS width in sinus rhythm ≥120	2.20 (0.93 – 5.19)	0.072
NYHA class ≥ III before VF storm	2.64 (1.11 – 6.28)	0.029
Cardiogenic shock due to heart failure on admission	4.55 (1.87 – 11.09)	0.001
ICD at the time of VF occurrence	0.49 (0.18 – 1.34)	0.17
Number of defibrillation before ablation ≥21	1.63 (0.69 – 3.81)	0.26
Revascularization not performed before ablation	2.92 (0.68 – 12.53)	0.15
Antiarrhythmic drugs		
Amiodarone	2.35 (0.27 – 20.39)	0.44
Class I antiarrhythmic drugs	1.40 (0.57 – 3.45)	0.47
β-blocker	0.44 (0.16 – 1.23)	0.12
Periprocedural use of hemodynamic support device	3.59 (1.49 – 8.63)	0.004
Periprocedural use of mechanical ventilator	2.96 (0.93 – 9.37)	0.066
Time from VF to ablation	1.11 (1.03 – 1.20)	0.008
Procedural data		
Spontaneous frequent VF trigger during procedure	0.57 (0.19 – 1.74)	0.33
Irrigation catheter used for ablation	1.59 (0.42 – 6.07)	0.50
VF trigger still inducible at the end of procedure	4.75 (1.24 – 18.25)	0.023
Peri-procedural complication	0.97 (0.28 – 3.30)	0.96
Procedural time	1.00 (0.99 – 1.01)	0.68
Radiofrequency time	1.02 (0.99 – 1.06)	0.20

Abbreviations: CABG = coronary artery bypass grafting; LV = left ventricle; ICD=implantable cardioverter defibrillator; MI = myocardial infarction; NYHA = New York Heart Association; VF = ventricular fibrillation. Chronic kidney disease was defined as a serum creatinine greater than 1.5mg/dL.

Table 3. Clinical, procedural, and follow-up data associated with all-cause mortality after discharge from hospital

	HR (95% CI)	p
Clinical data		
Age ≥70	1.17 (0.53 – 2.57)	0.70
Male gender	0.83 (0.28 – 2.42)	0.73
Acute or subacute MI	1.27 (0.52 – 3.14)	0.60
Number of diseased vessels	1.01 (0.67 – 1.53)	0.96
History of		
Hypertension	1.49 (0.66 – 3.37)	0.34
Diabetes mellitus	1.54 (0.73 – 3.22)	0.26
Atrial fibrillation	3.89 (1.42 – 10.67)	0.008
Chronic kidney disease	2.74 (1.15 – 6.49)	0.023
Post-CABG	1.61 (0.73 – 3.55)	0.24
LV ejection fraction <30%	2.54 (1.21 – 5.32)	0.014
QRS width in sinus rhythm ≥120	1.09 (0.51 – 2.34)	0.83
NYHA class ≥ III	2.68 (1.16 – 6.19)	0.021
Cardiogenic shock on admission	2.42 (0.98 – 5.94)	0.055
ICD at the time of VF occurrence	1.37 (0.66 – 2.85)	0.41
Number of DC before ablation ≥21	1.05 (0.49 – 2.25)	0.91
Revascularization not performed before ablation	0.36 (0.048 – 2.67)	0.32
Antiarrhythmic drugs during hospitalization		
Amiodarone	1.78 (0.24 – 13.21)	0.58
Class I antiarrhythmic drugs	1.60 (0.74 – 3.49)	0.23
β-blocker	0.72 (0.28 – 1.90)	0.51
Periprocedural use of Hemodynamic support device	2.04 (0.95 – 4.41)	0.069
Periprocedural use of Mechanical ventilator	2.20 (0.93 – 5.21)	0.075
Time from VF to ablation	0.99 (0.92 – 1.08)	0.90
Procedural data		
Spontaneous frequent VF trigger during procedure	3.02 (0.71 – 12.81)	0.13
VF trigger still inducible at the end of procedure	0.56 (0.076 – 4.15)	0.57
Peri-procedural complication	0.48 (0.11 – 2.01)	0.31
Procedural time	1.00 (0.996 – 1.01)	0.62
Radiofrequency time	1.00 (0.96 – 1.03)	0.93
Follow-up data		
ICD during follow-up	0.39 (0.12 – 1.30)	0.13
Antiarrhythmic drugs during follow-up		
Amiodarone	1.83 (0.83 – 4.03)	0.13
Class I antiarrhythmic drugs	1.99 (0.80 – 4.92)	0.14
β-blocker	0.29 (0.085 – 0.97)	0.044
Oral anticoagrants during follow-up	2.03 (0.96 – 4.31)	0.065

Abbreviations: CABG = coronary artery bypass grafting; LV = left ventricle; ICD=implantable cardioverter defibrillator; MI = myocardial infarction; NYHA = New York Heart Association; VF = ventricular fibrillation. Chronic kidney disease was defined as a serum creatinine greater than 1.5mg/dL

Figure Legends

Figure 1. Triggers originating from the scar border zone and dense scar.

Figure 1A shows most common type of Purkinje trigger originating from the scar border zone in the left ventricular septum. Purkinje potentials (red arrows) preceded the ventricular electrograms during both sinus rhythm and triggering ectopic beat (blue star). Figure 1B shows the origin of the triggering extrasystoles located within the large dense scar (one originating from the Purkinje fibers within the antero-septal scar [middle panel] and the other originating from the infero-septal scar [lower panel]). In the middle panel, Purkinje potential preceded ventricular electrogram during both sinus rhythm and ectopic beat (blue star) which exhibited very narrow QRS duration. In the lower panel, the second beat was probably fusion beat (blue asterisk), and subsequent beat induced VF (blue star). The sharp high-frequency potential indicating Purkinje potential preceded the triggering ectopic beats. However, these sharp electrogram was not observed during sinus rhythm possibly because of the Purkinje conduction block in the proximal part of this region due to the extensively damaged infarct scar.

Figure 2. In-hospital follow-up outcomes.

AADs = antiarrhythmic drugs; VF = ventricular fibrillation; VT = ventricular tachycardia

Figure 3. Reduction of ventricular arrhythmia burden.

Catheter ablation reduces the burden of ventricular fibrillation /ventricular tachycardia.

Figure 4. The number of patients with the hospital discharge or death against the duration between the VF occurrence and the ablation procedure.

In-hospital death patients increased with the time from VF storm occurrence to ablation procedure. The bar graph shows the number of patients, and dotted line shows the percentage of in-hospital death.

Figure 5. Cumulative incidence curves showing arrhythmia recurrence and mortality during short- and long-term follow-up.

Figure 5A shows cumulative incidence curves of electrical storm /ventricular fibrillation /ventricular tachycardia recurrence. Figure 5B shows cumulative incidence curves of all-cause mortality, cardiovascular mortality, non-cardiovascular mortality, and death from unknown reason. These curves were plotted short-term (30-day mortality) against time in days and long-term (after 30 days) against time in years.