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Original Article

Management of electrical storm of unstable ventricular tachycardia in post myocardial infarction patients: A single centre experience

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ABSTRACT

Objective: This is a case series of consecutive patients with past myocardial infarction presenting with Electrical Storm (ES) of unstable ventricular tachycardia (VT) treated by a protocol directed algorithm.
Methods: Management protocol involved treatment of reversible causes, ventilatory & hemodynamic support, administration of antiarrhythmic drugs (AAD) & maximally tolerated doses of beta-blockers, stellate ganglionectomy and Radiofrequency ablation (RFA) guided by Electro Anatomic Mapping (EAM). Patients were followed up periodically with review of device data logs.
Results: There were 12 patients (mean age = 61.38 ± 6.48 years & mean LVEF = 31.92 ± 4.23%). Presentation was recurrent ICD shocks (n = 5) or VT (n = 7). All were mechanically ventilated. Reversible causes were identified in 4 patients and appropriately addressed. Totally 8 patients underwent endocardial substrate modification by EAM & RFA. Endocardial LV Voltage mapping demonstrated a mean scar area of 70.04 ± 17.63 sq.cm (27.04 ± 6.20% of mapped area). The electrograms targeted for ablation included late potentials, fractionated electrograms, double potentials and channels within the scar. Two patients had stellate ganglionectomy in addition. Ten patients (83.3%) survived to discharge, all of whom are alive at a follow up of 30.12 ± 19 months free of ES. VT free survival at end of follow up was 80%. No patient had hospitalization related to VT. Single episode of VT recurrence was seen in 2 patients at 7 months and 1 year of follow up respectively.
Conclusion: In post myocardial infarction patients presenting with ES and unstable VT, a protocol driven approach involving substrate modification targeting abnormal electrograms improves outcomes.

1. Introduction

Electrical Storm (ES) is defined as, "Three or more distinct episodes of ventricular tachycardia (VT)/ventricular fibrillation (VF) within 24 h, requiring the intervention of the defibrillator (antitachycardia pacing or shock)".[1] This is a life threatening cardiac emergency with a reported incidence of 10–28% and mortality as high as 82% despite multiple interventions.[1,2] Increased mortality has been documented in patients experiencing ES in the AVID, MADIT II and SCD-HeFT trials.[3,4,5] Even those who survive have prolonged hospital stay associated with increased morbidity. The outcomes are particularly worse when the ES is associated with unstable ventricular arrhythmias. This manuscript summarizes the immediate and long term outcomes of 12 patients with ischemic cardiomyopathy presenting with ES of hemodynamically unstable VT.

2. Methods

2.1. Institutional protocol

Consecutive patients with ischemic cardiomyopathy, who were admitted in our centre with diagnosis of ES and hemodynamically unstable VT, were prospectively enrolled. All patients had a past myocardial infarction more than 3 months prior to occurrence of ES. Patients with other substrates or those presenting with ES and VF/stable VT were not included. Patients were managed as per our institutional protocol. Patients with implanted ICDs underwent device interrogation to confirm that the shocks were appropriate. Apart from routine investigations and echocardiographic assessment of ventricular function, patients were systematically investigated for the presence of metabolic abnormalities, electrolyte imbalances, thyroid dysfunction and on-going ischemia (by coronary angiography). If any of these reversible causes were

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found, they were appropriately corrected. Beta blockers were stepped up to maximally tolerated dosages and amiodarone was initiated unless contraindicated. Other antiarrhythmic agents (sotalol, mexiletine & phenytoin) were administered as considered appropriate. All patients were administered deep sedation and those continuing to have VT episodes warranting external shocks were electively intubated and mechanically ventilated. Hemodynamic stability was maintained by inotropes and Intra-arterial balloon pump (IABP) when indicated. Patients who continued to have VT were considered for sympathetic ganglionectomy and 3D electro anatomical mapping (EAM) with radiofrequency ablation (RFA). Sympathetic ganglionectomy was performed as described in literature and involved excision of the distal half of the stellate ganglion and the T1 to T4 sympathetic chain with frozen section confirmation on table.[6]

2.2. Electroanatomical mapping (EAM) and radiofrequency ablation (RFA)

Electrophysiological study with EAM and RFA were performed under deep sedation with mechanical ventilatory support in all patients. Systemic anticoagulation was achieved by intravenous heparin administered intermittently maintaining a target activation clotting time of 300- 350 s, which was estimated every 20 min through the procedure. CARTO 3 workstation (Biosense Webster, Diamond bar, CA) was used for EAM. Prior to mapping, when there were no contraindications left ventricular (LV) angiogram was done in RAO view (and LAO view if needed) to delineate LV aneurysms. Both antegrade (by trans-septal puncture) and retrograde (trans-aortic) approaches were used to map the LV using a 3.5 mm open irrigated tip ablation catheter (Navistar Thermocool, Biosense-Webster, Diamond Bar, CA). Data was recorded on a multichannel electrophysiological system. (EP Tracer, Schwarzercardiotek, Germany). Intracardiac signals were recorded at a band pass filter at 30 to 400 Hz. Patients underwent substrate mapping in sinus rhythm (Fig. 1A) and the rhythm was maintained in sinus by repeated external shocks and IV anti arrhythmic agents. Voltage map of LV was obtained using standard voltage cutoffs.[7,8] Normal myocardium was identified by a voltage of >1.5 mV and dense scar by voltages <0.5 mV. Area with voltages between 0.5 mV to 1.5 mV represented the border zone between the normal myocardium and dense scar. The scar and the border zones were densely mapped exploring these areas for abnormal electrograms which were located and tagged. These abnormal signals were further characterized by measuring the signal amplitude and local delay of the ventricular electrogram with reference to the end of QRS on the surface ECG. They were classified as isolated late potentials (ILPs), low voltage pluripotent fractionated potentials (continuous electrical activity, CEA) and double potentials which were tagged by different colour codes. Conducting channels in the dense scar were located by adjustment of voltage as described by Mountantonakis et al. [9] These were

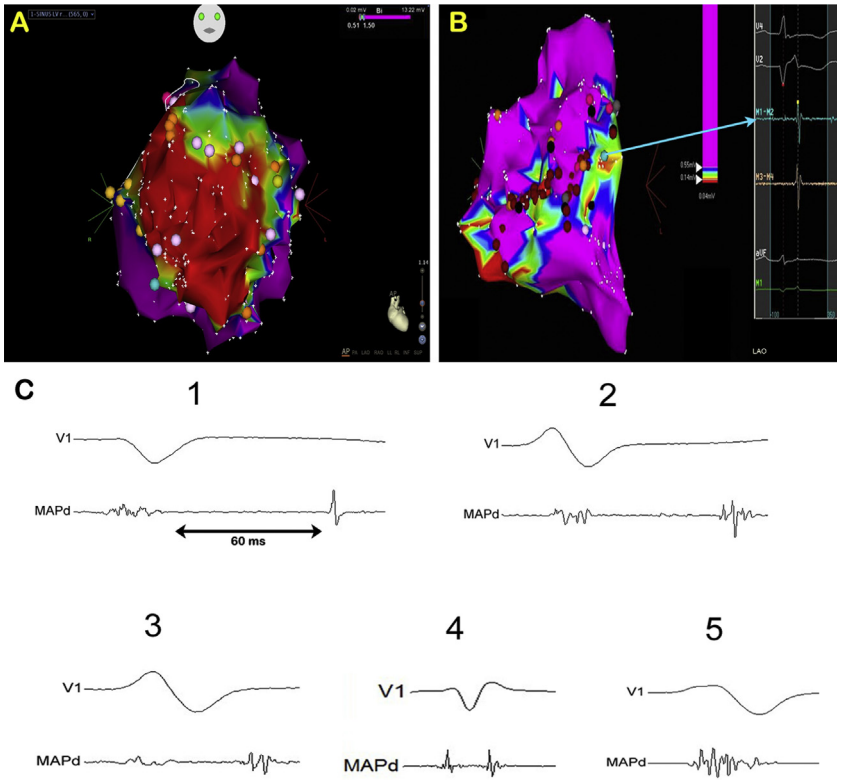

Fig. 1. Panel A: LV Voltage map in sinus rhythm of a patient in AP view showing areas of scar (denoted in red, with voltage of <0.5 mV) and normal myocardium (denoted in pink, with voltage of >1.5 mV). The areas between the scar and the normal myocardium are low voltage corridors containing abnormal electrograms. The fine white dots indicate the points mapped. Each abnormal electrogram is tagged with a different colour (blue: double potentials; yellow/orange: late potentials; white: fractionated signals; pink: continuous electrical activity) which are targets for ablation.
Panel B: shows a substrate map of the LV in LAO view where voltage thresholds have been adjusted to identify low voltage channels (multicoloured zones seen between pink areas) within the scar. The figure shows a late potential within a channel (blue tag and arrow) that was targeted for ablation.
Panel C: shows abnormal electrograms targeted for ablation. QRS complex in ECG lead V1, and corresponding LV electrogram on mapping catheter are shown. 1 to 3 are examples of isolated late potentials (ILPs), 1 is an ILP that is identified as a high frequency discrete potential separated from the ventricular electrogram by 50 msec, 2 shows an ILP which is fractionated,3 is a double-component or fractionated late potential that is formed by the almost fusion of two late potentials. 4 is a double potential having two component electrograms of low voltage separated by an isoelectric interval. 5shows low voltage pluricomponent signals without intervening isoelectric intervals that appears as continuous electrical activity (CEA).

differentiated from the surrounding scar tissue by higher amplitude voltage and the channels were mapped to identify abnormal electrograms.

All the abnormal electrograms that were tagged during mapping in the scar and border zones were targeted for ablation, beginning with the ILPs. Each RF lesion was delivered at a power of 30–50 W for duration of 60 s. The endpoint of ablative lesion was abolition of the abnormal electrogram. VT induction was attempted following substrate ablation by programmed extra stimulation (PES) from 2 sites in RV and 1 site in LV by triple extra stimuli at 2 cycle lengths. If VTs were induced, activation mapping and ablation was done if they were stable and further substrate mapping if they were unstable. The procedural endpoint was primarily freedom from spontaneous VT and secondarily non-inducibility of sustained VT by PES.

2.3. Abnormal electrograms targeted for ablation (Fig. 1B & C)

2.3.1. Late potentials

Potentials of any voltage, single, split or multiple delayed electrical components separated from higher amplitude component of local intrinsic or paced ventricular electrogram by at least 20 milliseconds (ms) and recorded after the end of surface QRS.

2.3.2. Fractionated electrograms (Continuous electrical activity, CEA)

Electrograms with more than 3 deflections ≤ 1.0 mV and ≥ 70 ms in duration

2.3.3. Split electrograms/Double potentials

2 component electrograms of low voltage separated by an isoelectric interval or very low amplitude signals.

2.3.4. Channels within a scar

High voltage electrograms seen within the dense scar by adjusting voltage cut offs on colour isopotential electroanatomic maps (Fig. 1B).

2.4. Post-ablation follow up

Post procedure patients were closely monitored for the first 24 h in the ICU before being transferred to the floors. Those with ICD had pre-discharge device interrogation to assess occurrence of VT. All patients were discharged with amiodarone and maximally tolerated dosages of metoprolol. Amiodarone was discontinued after 6 months if there were no VT episodes on device data logs requiring intervention. Patients, who did not have prior ICD, had the device implanted prior to discharge. At discharge, ICD was programmed to single zone of therapy (VF). Further changes in the device therapies were made as needed at follow up. Patients were followed up at two weeks for one month then monthly for three months and then every three months thereafter with a review of clinical data and ICD data logs.

2.5. End points

The primary end point was survival free of ES recurrence. Secondary end points were freedom from VT recurrence and cardiac related hospitalization.

2.6. Statistical analysis

Descriptive statistics are reported as mean and standard deviation (SD) for continuous variables and as absolute frequencies (percentages) for categorical variables. Statistical analysis was performed using MedCalc Statistical Software version 17.1 (MedCalc Software bvba, Ostend, Belgium; https://www.medcalc.org; 2017).

3. Results

3.1. Patient population

There were 12 patients (10 males & 2 females) who had prior myocardial infarction (mean 101.4 ± 78.7 months prior to ES)and who qualified with the diagnosis of ES presenting with unstable VT. These patients constituted the cohort for this report.

The baseline demographic and clinical data are summarized in Table 1. The mean age of the patients was 61.38 ± 6.48 years and their mean LVEF was $31.92 \pm 4.23\%$ with 2 having LV thrombus. The clinical presentation was recurrent ICD shocks in 5 and recurrent VT in 7 patients. All patients were electively intubated, maximally sedated, and put on mechanical ventilation. One patient in addition required an IABP for hemodynamic support. The underlying clinical substrate in these patients was ischemic cardiomyopathy (anterior wall MI, n = 7; inferior wall MI n = 3; inferior and posterior wall MI, n = 2). The dose of Beta blocker used in these patients was 100–200 mg per day of metoprolol succinate. Intravenous amiodarone and lidocaine was administered in all patients, while one patient each received phenytoin and sotalol. Despite receiving maximally tolerated medical therapy, 8 patients continued to have recurrent episodes of VT necessitating multiple externally applied DC shocks.

3.2. Reversible causes

In our cohort 4 were found to have reversible factors on evaluation. Two patients were found to have incomplete revascularization and had ES within 72 h following CABG. These 2 patients also had runs of polymorphic VT. Coronary angiogram was done in both these patients. One of them was found to have occluded venous graft to a moderate sized obtuse marginal and underwent PCI to native OM. There were no further arrhythmia episodes and this patient had uneventful discharge. The second patient was discovered to have an ungrafted diagonal, which was stented but succumbed 48 h later to vascular complications of IABP, acute renal shut down and heart failure. One patient presented with recurrent ICD shocks. She was found to have thyrotoxicosis and was treated with anti-thyroid drugs and appropriate dose of beta blockers. She has since been diagnosed with Grave's disease and treated appropriately. She is asymptomatic with no further ICD therapies

Table 1
Baseline characteristics.

Characteristic	Range (Mean ± SD)
Patient population, (M/F)	12 (10/2)
Age in years	54–69 (61.38 ± 6.48)
LV ejection fraction (LVEF) (%)	25–36 (31.92 ± 4.23)
LV End Diastolic Diameter (cm)	5.7–7.3 (6.5 ± 0.8)
• Anterior wall MI (n =)	7
• Inferior wall MI (n =)	3
• Inferior + Posterior wall MI (n =)	2
Medications (n =)	
• Metoprolol	12
• Amiodarone	12
• Lignocaine	09
• Phenytoin	01
• Sotalol	01
Presentation (n =)	
• Recurrent ICD shocks	05
• Recurrent VT	07
ICD shocks per patient (n =)	4–26 (9. ± 10)
External Shocks delivered per patient in ICU (n =)	23–98 (23 ± 28)

at last follow up. The 4th patient had chronic kidney disease who presented with recurrent VT episodes to the ER requiring multiple DC cardioversions. On evaluation she was found to have significant hyperkalaemia (K^+: 9.5 meq/l) and underwent hemodialysis. She recovered post dialysis with no further episodes of VT during hospital stay.

3.3. Electroanatomic mapping and radio frequency ablation

Eight patients who continued to have drug refractory recurrent VT underwent EAM and RFA under general anaesthesia. All these patients were found to have spontaneous hemodynamically unstable VTs of multiple morphologies (mean 3 ± 2, Fig. 2). Stellate ganglionectomy was performed in addition to substrate ablation in 2 patients. Details of mapping and ablation are summarized in Table 2.

LV map in sinus rhythm showed a significant scar burden with the scar constituting about 27% of the mapped LV. Dense mapping was done in this area and borders to identify abnormal potentials. Multiple ILPs were found in 7 out of the 8 patients while fractionated electrograms, double potentials and channels within scar were seen in all patients.

Primary procedural success of achieving freedom from ES was seen in 7 out of 8 patients. On PES, 1 patient had inducible self-terminating stable VT with morphology suggestive of epicardial VT. One patient who presented with ES post CABG had patent grafts and underwent bilateral stellate ganglionectomy following unsuccessful ablation. He continued to have recurrent polymorphic VT and expired 2 weeks later. There were no procedure related complications. Amongst the survivors following ablation, 4 had prior ICD implanted, 2 had device implantation before discharge, and 1 patient refused ICD post RFA and is doing well on regular follow up at 3 years with no further VT. Overall of the initial cohort of 12 patients, 10 patients (83.3%) were free of ES and survived to discharge with a mean duration of hospital stay of 9.83 ± 8.14 (3–16) days.

Table 2
Characteristics of Electroanatomic mapping and RFA (n = 8).

Characteristic	Range (Mean ± SD)
Morphologies of spontaneous VTs per patient (n =)	2–5 (3 ± 2)
VT cycle length in milliseconds (ms)	270–430 (350 ± 79)
LV points mapped by EAM (n =)	392–1017 (631 ± 212)
Scar area (sq cm)	36.5–92.8 (70.04 ± 17.63)
Scar area as percentage of total LV mapped (%)	18–34.8 (27.04 ± 6.20)
RF ablations delivered (n =)	49–95 (70 ± 20)
Procedure time in minutes	217–409 (311.75 ± 61.38)

3.4. Long term follow up

All 10 patients who are discharged are alive with a mean follow-up of 32.12 ± 19.09 (11.5–67.3) months. No patient had ES at follow-

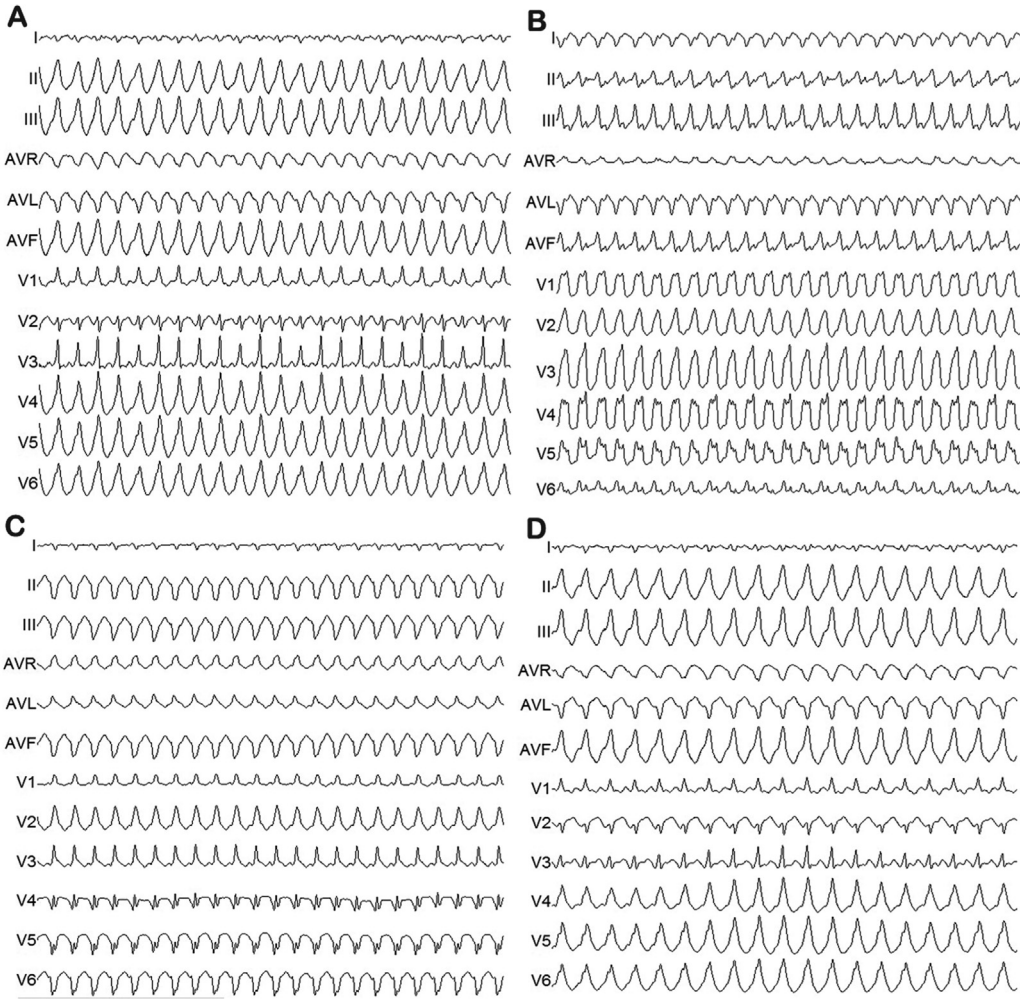

Fig. 2. 12 lead ECG recordings of one of the patients who had 4 different morphologies of VT (A to D) during procedure. The cycle length of the VTs was variable 260–300 ms (280.75 ± 19.38). All the VTs were hemodynamically unstable and unmappable.

up. One patient on ICD, who developed degenerative complete heart block, became pacemaker dependent and his device was upgraded to CRT-D at 6 months of follow up. This was the only patient who needed hospital admission following management of ES. At follow up, 2 patients had single VT recurrence each confirmed by ICD data logs. In one instance it was a slow VT (430 s cycle length) occurring 7 months following ES which was terminated by ATP. In the 2nd patient a full energy shock was delivered to terminate the VT recurrence which occurred at 1 year of follow up. Oral amiodarone was restarted in both these patients. In the patient who had inducible VT (suggestive of epicardial morphology) following substrate modification, there have been no episodes of VT/VF thereafter at 29 months follow up as per the ICD data logs.

4. Discussion

This manuscript summarizes our experience in managing post infarct patients with ES of hemodynamically unstable and unmappable VT. It highlights improved outcomes in this critical subset of ES patients by following a systematic protocol directed management and substrate modification targeting abnormal potentials in these patients.

4.1. Management by protocol directed algorithm

Our data underlines the importance of prompt recognition of ES, following a systematic protocol, correction of reversible factors and early institution of ablative therapy in improving outcomes. It is known that 9–11.5% of ICD shocks are inappropriate and some of these patients may be incorrectly labelled as experiencing ES.[3,4] Hence the first step in our management algorithm was device interrogation to exclude inappropriate shocks and address conditions leading to it. All of our patients received maximal sedation and were put on elective mechanical ventilation when VT episodes continued. We believe this is an essential initial mandatory step that should be incorporated in treating these patients. ES is a hyper-adrenergic state and frequent shocks either delivered by ICD or externally are painful and psychologically traumatic. General anaesthesia is recommended to reduce pain and morbidity from shocks and to reduce the sympathetic tone. Use of high doses of beta blockers and surgical modification of neuro-axial sympathetic system were additional strategies to counter the sympathetic excess fuelling the ES. Two of our patients underwent stellate ganglionectomy in addition to ablation. Bilateral stellate ganglionectomy usually by a video assisted mini thoracotomy has been shown to reduce VT recurrence.[6] Though electrolyte disturbances notably hyperkalaemia, acute coronary syndrome and refractory heart failure have all been reported to be triggering causes of ES, an evident cause cannot be established in majority.[10] Amongst our patients, 4 had reversible factors which could be corrected, resolving the ES without need for any additional therapies. In this cohort, 2 patients who underwent CABG had ES in the early post-operative period and were found to have unrevascularized areas. Ongoing ischemia should be ruled out and a low threshold for early graft angiogram is recommended. Thyrotoxicosis is a documented trigger for monomorphic VT, and in one of our patients this was responsible for ES.[11] With the exception of one, in all these patients, identifying and addressing the triggering factor alone successfully resolved the ES without any further interventions.

4.2. Substrate mapping and ablation

Catheter based ablation in patients with recurrent scar related VT has been shown to improve survival free of VT, decrease the frequency of appropriate shocks and consequently result in reduction of cardiovascular mortality.[7,8,12–14] The targets of ablation are the crucial slow areas of conduction which are identified by standard electrophysiological techniques of activation mapping and entrainment. To characterize the VT circuits and accurately identify the targets of ablation by these methods, it is necessary for these patients to have stable and hemodynamically well tolerated VTs. We were dealing with a subset of critical post infarct patients presenting with ES who had multiple hemodynamically unstable and unmappable VTs frequently with interchanging morphologies. This precluded the employment of conventional mapping and ablation techniques. In these patients the usual strategy is to map and characterize the substrate in sinus rhythm identifying sites which are putative surrogates of slow conduction zones of isthmus of VT circuits and hence critical targets for elimination of these VTs. Different approaches to modify the substrate have been evaluated.Linear ablation lesions were first described by Marchlinski etal to target multiple unmappable VTs which included creation of contiguous lesions from dense infarct zone through border area connecting to anatomical barriers or normal myocardium. They reported 75% freedom from recurrent VT at a median follow up of 8 months.[15] Other strategies include ablation of late potentials, ablation of late activation ventricular activities, channel ablation and scar homogenization and their merits and demerits been well described.[12–14,16–18] In all patients we focused on dense mapping of the target area which was typically the infarcted and peri-infarct area ensuring adequate contact of the catheter with point to point validation of scar and electrogram characterization. The clue to the target area for mapping was obtained from site of infarct, pre-procedure echocardiogram and LV angiogram performed prior to mapping. The latter was particularly useful in identifying LV aneurysms which are known to house a significant proportion of the substrate. Electrical isolation of LV aneurysm in a post infarct patient has been shown to be an effective ablation strategy.[19] The density of mapping of target area in our cases is shown by the fact that the mean number of points in LV that we collected were 630.75 ± 212. Marchlinski's group has established the bipolar voltage cut off values of normal and abnormal substrate in patients with infarcted myocardium.[20] These have been validated in animal infarct model and histopathology study in man.[21,22] Values >1.5 mV defined normal myocardium and values <0.5 mV were consistent with dense scar. The use of 1.5 mV cut-off for defining abnormal myocardium makes a very efficacious distinction from adjacent normal myocardium and hence prevents RF lesion application in normal bystander areas in the border zone.[20] We used these established voltage cut-offs during electro anatomical mapping of the LV in our study. Low voltage (0.5 mV – 1.5 mV) areas were explored for abnormal electrograms. Our strategy involved ablation of all the abnormal electrograms. We initially targeted isolated late potentials (ILPs) for ablation irrespective of their voltage. Late potentials are known to be identified in 2/3rd of patients, and except in one we could identify the potentials in all of our patients. We did not attempt to distinguish between different late potentials, defined by Vergera, Arenal and Nogami and all identified ILPs were targeted for ablation.[12,18,23] In only one of our patients there was a paucity of late potentials and in this patient ablation was not successful. We did not attempt using pacing manoeuvres routinely to bring out late potentials as our cohort comprised of patients with unstable hemodynamics with frequent spontaneous VT episodes during mapping needing repeated cardioversions. In addition to late potentials, we also mapped and targeted for ablation other abnormal electrograms which included low amplitude double and multi-component potentials. It has been shown that late potentials and fragmented electrograms are found in increased prevalence in patients with post infarct

scars presenting with sustained monomorphic VT. The density of these electrograms within these scars is also higher in these patients compared to those without VT.[23] Substrate modification by ablation of these potentials has been shown to improve VT free outcomes in different series.[7,8,13,14,16,19] We targeted these electrograms for ablation by radiofrequency energy with the objective of destroying the vital slow conducting milieu responsible for sustaining multiple VTs. In addition we used voltage cut off adjustments to identify putative conducting channels within the scar. The channels were considered important if they contained abnormal electrograms and these were transected. These abnormal electrograms are electrical correlates of the well described pathologic substrate of these patients characterized by inhomogeneous scarring with varying degree of myocardial fibre preservation within dense scars.

Out of the 8 patients who underwent ablation, 7 were free of ES and could be discharged from the hospital. None of these had ES recurrence or need for VT related admission at a follow up period of 32.12 ± 19.09 months. We attribute the encouraging immediate and long term outcomes seen in our small cohort of patients with life threatening recurrent VTs to this strategy of high density mapping of target area, carefully identifying and ablating all the abnormal electrograms which constituted the critical elements supporting the re-entry circuits. Invariably the ablation was extensive and may have included electrograms not participating in the clinical VT, however these electrograms could potentially serve as zones of slow conducting isthmus for future VTs. We nevertheless ensured ablating only low voltage signals avoiding injury to normal myocardium. The ablation lesions were delivered only endocardially in all our patients, with a plan for epicardial mapping as a separate procedure if there was a recurrence. Only in 1 of the patients there was an inducible non-clinical VT which was suggestive of involvement of an epicardial circuit. This patient did not have a VT recurrence at follow up. At follow up none of our patients required epicardial ablation. Though this is a small number of patients and we did not monitor electrograms in the epicardium, we believe that extensive endocardial substrate ablation at least partially affected the epicardial substrate. Our inference is supported by a recent series of 46 patients, 18 of them with ischemic cardiomyopathy where endocardial Local abnormal ventricular activity (LAVA) ablation eliminated epicardial LAVA at least partially in 83% of the patients.[24]

4.3. Ablation in presence of LV thrombus

We had 2 patients in our cohort who had organized and old thrombi in the apex of left ventricle. VT ablation in the presence of LV thrombus is challenging as catheter manipulation can potentially dislodge or fragment the thrombus leading to embolism and stroke. In both these patients during mapping and ablation of the substrate catheter manipulation in the vicinity of apex was minimized. In a recent publication we demonstrated the safety and feasibility of substrate based ablation in the presence of an intra-cavitary thrombus.[25] It is to be noted that current EHRA/HRS guidelines do not consider the presence of an organized LV thrombus to be a contraindication to ablation.[26]

5. Conclusion

In patients with post infarct ES with unstable VT, systematic protocol directed algorithm and extensive dense substrate based mapping and systematic ablation of all abnormal electrograms improves immediate and long term outcomes.

5.1. Limitations of the study

We acknowledge that this is a small cohort of patients. We included only those patients with ES who presented with VT to ensure homogeneity of our population and deliberately restricted to patients presenting with unstable VT. We intended to demonstrate a feasible therapeutic algorithm through this small number of most critical subset of ES.

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References

1. Proietti R, Sagone A. Electrical storm: incidence, prognosis and therapy. *Indian Pacing Electrophysiol J*. 2011;11(2):34–42.
2. Nademanee K, Taylor R, Bailey WE, et al. Treating electrical storm: sympathetic blockade versus advanced cardiac life support–guided therapy. *Circulation*. 2000;102:742–747.
3. Exner DV, Pinski SL, Wyse DG, et al. Electrical storm presages nonsudden death: the antiarrhythmics versus implantable defibrillators (AVID) trial. *Circulation*. 2001;103:2066–2071.
4. Sesselberg HW, Huang DT, Zareba W, et al. Storms of ventricular tachycardia/fibrillation in MADIT II patients. *Heart Rhythm*. 2005;2(5 Suppl):S205.
5. Poole JE, Johnson GW, Hellkamp AS, et al. Prognostic importance of defibrillator shocks in patients with heart failure. *N Engl J Med*. 2008;359:1009–1017.
6. Tung R, Shivkumar K. Neuraxial modulation for treatment of VT storm. *J Biomed Res*. 2015;29(1):56–60.
7. Di Biase L, Burkhardt JD, Lakkireddy D, et al. Ablation of stable VTs versus substrate ablation in ischemic cardiomyopathy. *J Am Coll Cardiol*. 2015;66(25):2872–2882.
8. Tilz RR, Makimoto H, Lin T, et al. Electrical isolation of a substrate after myocardial infarction: a novel ablation strategy for unmappable ventricular tachycardias — feasibility and clinical outcome. *Europace*. 2014;1040–1052.
9. Campos B, Jauregui ME, Park KM, et al. New unipolar electrogram criteria to identify irreversibility of nonischemic left ventricular cardiomyopathy. *J Am Coll Cardiol*. 2012;60:2194–2204.
10. Israel CW, Barold SS. Electrical storm in patients with an implanted defibrillator: a matter of definition. *Ann Noninvasive Electrocardiol*. 2007;12:375–382.
11. Chandra PA, Vallakati A, Chandra AB, Pednekar M, Ghosh J. Repetitive monomorphic ventricular tachycardia as a manifestation of suboptimally treated thyrotoxicosis. *Am Heart Hosp J*. 2010;8(2)E113–E114 Winter.
12. Vergara P, Trevisi N, Ricco A, et al. Late potentials abolition as an additional technique for reduction of arrhythmia recurrence in scar related ventricular tachycardia ablation. *J Cardiovasc Electrophysiol*. 2012;23:621–627.
13. Jais P, Maury P, Khairy P, et al. Elimination of local abnormal ventricular activities: a new end point for substrate modification in patients with scar-related ventricular tachycardia. *Circulation*. 2012;125:2184–2196.
14. Di Biase L, Santangeli P, Burkhardt DJ, et al. Endo-epicardial homogenization of the scar versus limited substrate ablation for the treatment of electrical storms in patients with ischemic cardiomyopathy. *J Am Coll Cardiol*. 2012;60:132–141.
15. Marchlinski FE, Callans DJ, Gottlieb CD, Zado E. Linear ablation lesions for control of unmappable ventricular tachycardia in patients with ischemic and nonischemic cardiomyopathy. *Circulation*. 2000;101:1288–1296.
16. Di Biase L, Burkhardt DJ, Lakkireddy D, et al. Ablation of Stable VTs Versus Substrate Ablation in Ischemic Cardiomyopathy: The VISTARandomized Multicenter Trial. *J Am Coll Cardiol*. 2015;66(25):2872–2882.
17. Soejima K, Stevenson WG, Maisel WH, Sapp JL, Epstein LM. Electrically unexcitable scar mapping based on pacing threshold for identification of the reentry circuit isthmus: feasibility for guiding ventricular tachycardia ablation. *Circulation*. 2002;106:1678–1683.
18. Arenal A, del Castillo S, Gonzalez-Torrecilla E, et al. Tachycardia-related channel in the scar tissue in patients with sustained monomorphic ventricular tachycardias: influence of the voltage scar definition. *Circulation*. 2004;110:2568–2574.
19. Thoppil PS, Rao BH, Jaishankar S, Narasimhan C. Successful catheter ablation of persistent electrical storm late post myocardial infarction by targeting purkinjearborization triggers. *Indian Pacing Electrophysiol J*. 2008;8(4):298–303.
20. Santangeli P, Marchlinski FE. Substrate mapping for unstable ventricular tachycardia. *Heart Rhythm*. 2016;13:569–583.
21. Callans DJ, Ren JF, Michele J, Marchlinski FE, Dillon SM. Electroanatomic left ventricular mapping in the porcine model of healed anterior myocardial

infarction. Correlation with intracardiac echocardiography and pathological analysis. *Circulation*. 1999;100:1744–1750.
22. Deneke T, Muller KM, Lemke B, et al. Human histopathology of electroanatomic mapping after cooled-tip radiofrequency ablation to treat ventricular tachycardia in remote myocardial infarction. *J Cardiovasc Electrophysiol*. 2005;16:1246–1251.
23. Nogami A, Sugiyasu A, Tada H, et al. Changes in the isolated delayed component as an endpoint of catheter ablation in arrhythmogenic right ventricular cardiomyopathy: predictor for long-term success. *J Cardiovasc Electrophysiol*. 2008;19(7):681–688.
24. Komatsu Y, Daly M, Sacher F, et al. Endocardial ablation to eliminate epicardial arrhythmia substrate in scar-related ventricular tachycardia. *J Am Coll Cardiol*. 2014;63(14):1416–1426.
25. Rao HB, Yu R, Chitnis N, et al. Ventricular tachycardia ablation in the presence of left ventricular thrombus: safety and efficacy. *J Cardiovasc Electrophysiol*. 2016;27(4):453–459.
26. Aliot EM, Stevenson WG, Almendral-Garrote JM, et al. EHRA/HRS Expert consensus on catheter ablation of ventricular arrhythmias. *Europace*. 2009;11:771–817.