# The Management of Electrical Storm in a Patient with Non-Ischemic Cardiomyopathy

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## 10.1 Case Summary

This is a 74 year old male with a known history of non-ischemic cardiomyopathy, left ventricular ejection fraction (LVEF) of 20%, complete heart block, s/p implantable cardioverter defibrillator (ICD) implantation 6 years prior, who presented with ventricular tachycardia (VT) electrical storm (ES). The patient received 12 antitachycardia pacing (ATP) and 5 ICD shocks for 12 sustained VT episodes. Multiple nonsustained episodes were noted as well. The VT rate ranged from 120–200 bpm. The patient was admitted to the CCU and was treated with intravenous (IV) amiodarone; however, VT recurred. A 12-lead ECG documenting the VT was obtained (Fig. 10.1). What would be the next steps in the management of the patient?

## 10.2 Case Discussion

ES is a life threatening condition, defined as at least three episodes of sustained VT or ventricular fibrillation (VF) within 24 h requiring either ATP or cardioversion/defibrillation, or as the occurrence of incessant VT for at least 12 h. The management of patients with ES is challenging and requires a multidisciplinary approach to care [1].

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1. **ICD interrogation and reprogramming**: *Device therapies were reprogrammed in attempt to minimize ICD shocks; however the patient continued to suffer from episodes of sustained VT.*

   Device therapies should be reprogrammed in order to prevent shocks for self-terminating VTs and favoring termination of VT using ATP. Sustained VT and ICD shocks are both associated with increased mortality [2].
2. **Reversible causes of ES**: *The patient was evaluated for possible reversible causes of ES, including blood work and echocardiogram. Echocardiography showed the left ventricle (LV) to be moderately dilated with severe dysfunction, LVEF = 20%.*

   Reversible causes of ES, such as acute myocardial ischemia, electrolyte imbalance, decompensated heart failure, pro-arrhythmic drug effects, acute valvular disease, infection or hyperthyroidism are identified in approximately 10% of patients, and should be screened for during initial patient evaluation.
3. **Antiarrhythmic drugs (AAD)**: *Antiarrhythmic therapy with IV esmolol and IV lidocaine were added to amiodarone.*

   Amiodarone is the most efficacious pharmacological agent for patients with structural heart disease (SHD) and sustained VT. Amiodarone has been demonstrated to markedly reduce recurrent appropriate ICD therapy during 1-year follow-up for secondary prevention. β-blockers have improved short-term outcome in patients with ES. Short-acting drugs, such as esmolol, might be considered in severely compromised patients, although an acute hypotensive effect may be seen, especially in patients with severe LV dysfunction. Even in patients already on oral β-blocker therapy, intravenous administration of β-blockers may help to reduce further ES episodes. β-blockers can be combined with amiodarone to improve rhythm stability. The combined use of amiodarone plus β-blockers significantly reduces the risk of recurrent ICD-shocks compared with β-blockers alone. Lidocaine is a class IB AAD which is more effective in ischemia related



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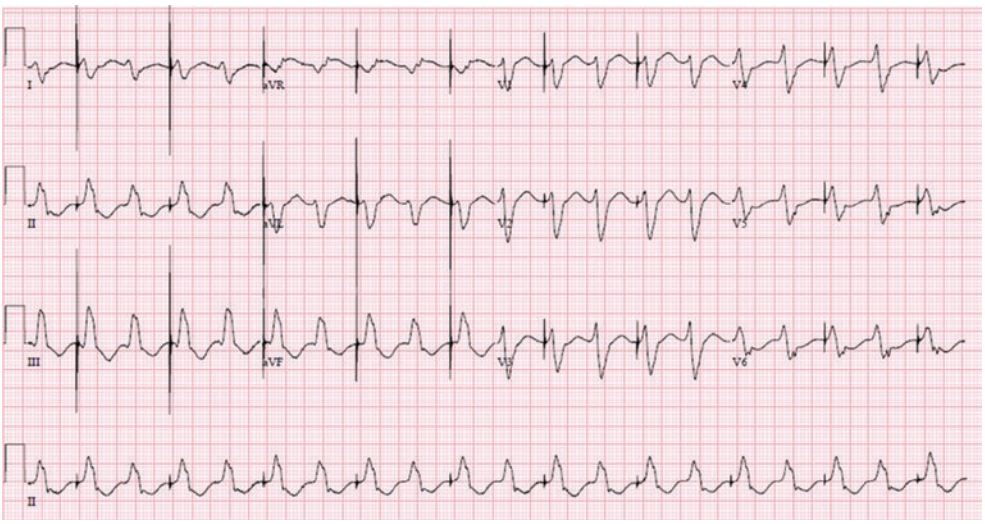

**Fig. 10.1** Twelve lead ECG obtained during VT at a rate of 120 bpm with a left bundle branch block morphology and inferior axis, consistent with a site of origin in the basal septum. Pacing artifacts are noted due to undersensing

VT but is of limited use in ES due to its lower efficacy in terminating scar-related VTs. This agent is a third choice drug for acute treatment.

4. **Mechanical hemodynamic support**: *The patient continued to have VT episodes on AAD therapy and he was brought to the cardiac catheterization laboratory. Coronary angiography confirmed no significant coronary artery disease. No coronary intervention was performed. An intra-aortic balloon pump (IABP) was placed for hemodynamic support.*

   Patients with unstable VT may benefit from hemodynamic support (HS) such as IABP, percutaneous ventricular assist devices and extracorporeal membrane oxygenation (ECMO). HS can reduce the arrhythmic burden by increasing coronary perfusion and reducing afterload and myocardial wall stress, improving cardiac output and end-organ perfusion. Sedation can also be considered in order to minimize pain and reduce the sympathetic surge triggered by repeated ICD therapies. Stellate ganglion block may also be considered.

5. **VT catheter ablation (CA)**: *Multiple VT episodes recurred and a VT ablation was performed. At the beginning of the procedure, the IABP was replaced by an Impella. Mapping and ablation were performed using a 3D CARTO® map (Biosense Webster, Diamond Bar, CA, USA) with a 3.5-mm open-irrigated catheter (ThermoCool, Smart touch, Biosense Webster, Diamond Bar, CA, USA) and a trans-septal approach. At the beginning of the procedure the patient was very easily inducible with catheter manipulation. Three VT morphologies were noted (Fig. 10.2). During the VT, the patient was hemodynamically stable with the Impella. VT1 cycle length (CL) was 550 ms. Activation mapping of VT1 demonstrated earliest activation on the mid-septal wall. Entrainment mapping at this location demonstrated concealed entrainment with a post pacing interval (PPI) of 0 ms. Several lesions were delivered using up to 40 W with good contact force in close proximity to the first lesion to extend the lesion. During catheter manipulation a second VT morphology occurred, VT2 CL = 580 ms. Activation mapping of VT2 demonstrated earliest activation on the mid-septal wall slightly superior and more apical to the first lesion set. Entrainment mapping at this location demonstrated concealed entrainment with a post pacing interval (PPI) of approximately 0 ms. There was a sharp pre-potential on the ablation catheter (Fig. 10.3). RF delivered at this location during VT terminated the VT in 2 s. After these ablations VT1 and VT2 did not recur. We then completed an LV voltage map. During catheter manipulation a third VT morphology occurred, VT3 CL = 620 ms. VT3 most resembles our clinical VT in Fig. 10.1. This VT3 was short lasting and spontaneously terminated multiple times prior to completion of an activation map. The voltage map demonstrated scar areas on the mid-septum towards the basal septum. The two sets of ablation points were at the border zone of the scar. The rest of the voltage map was normal. It was decided to extend the ablation to the scar area. Multiple ablations were delivered in order to create a box encircling the scar at the mid-septum (substrate ablation). The 3D electroanatomic map with the different lesion sets is shown in Fig. 10.4. After the completion of the box lesion set, attempts to reinduce with ventricular programmed extrastimulation (PES) with and without isoproterenol resulted only in nonsustained VT of a different morphology. During a follow up of 7 months the patient had no VT recurrence.*

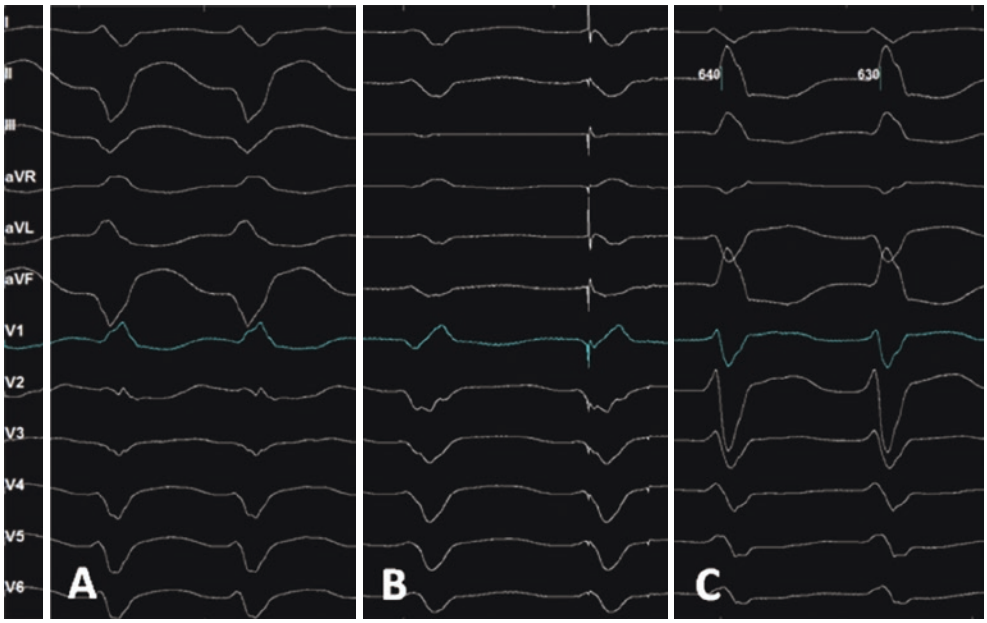


**Fig. 10.2** The three VT morphologies recorded during the ablation procedure. (**A**) VT1, (**B**) VT2, (**C**) VT3. VT3 morphology is identical to the clinical VT presented in Fig. 10.1

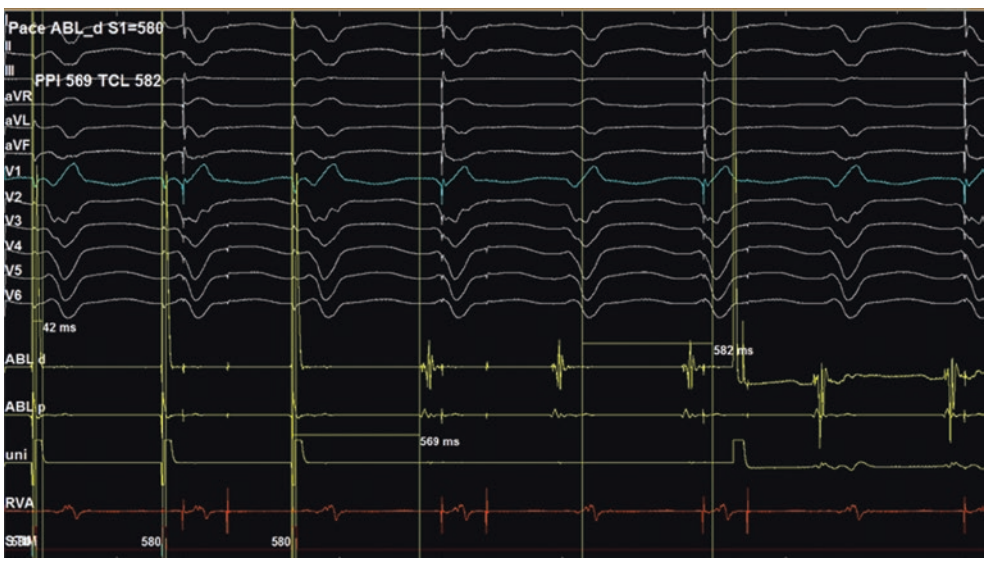


**Fig. 10.3** Entrainment mapping performed during VT2. The paced morphology is identical to the 12 lead ECG morphology, representing entrainment with concealed fusion. The post pacing interval is 569 ms and there is a sharp pre-potential on the ablation catheter signal. Stim-QRS/tachycardia CL is <0.3 indicating that pacing is occurring at the VT exit point

CA has been shown to be superior to medical therapy in reducing arrhythmic burden, and freedom from recurrent VT after CA ablation has been associated with improved survival [3, 4]. About 50% to 80% of patients with structural heart disease referred for VT ablation are hemodynamically unstable. Recently, a few studies have demonstrated feasibility and safety of using hemodynamic support devices (HS) during VT ablation in hemodynamically unstable patients [5]. These allow for prolonged mapping and ablation of inducible unstable arrhythmias. A septal substrate is identified in a minority of patients with NICM. Complete elimination of VT inducibility during PES after ablation is associated with reduced VT recurrence during long-term follow-up.

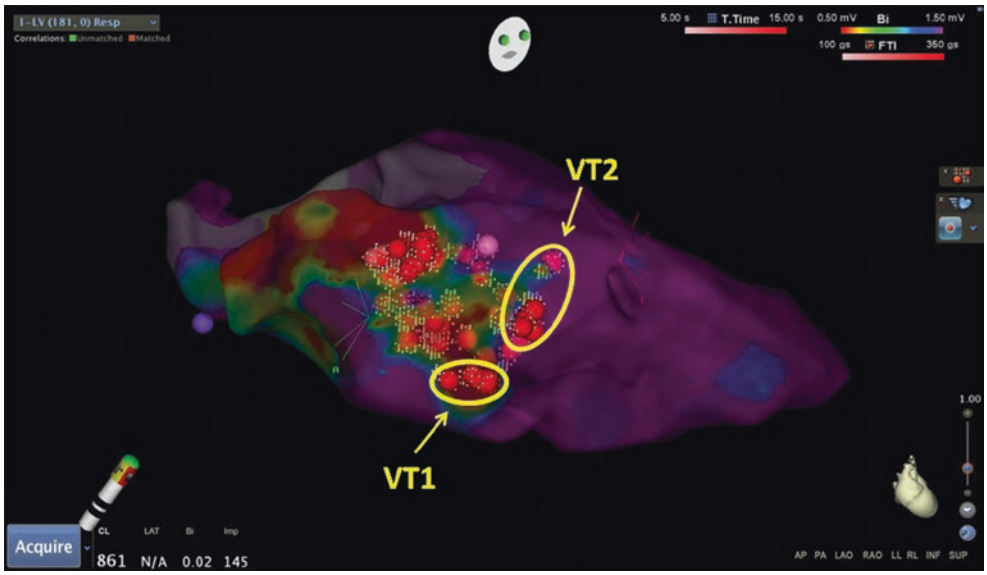


**Fig. 10.4** 3D electro-anatomical map with the ablation points. Encircled are the two lesion sets delivered for VT1 and VT2. Further lesions were delivered at the border zone of the low voltage area

## References


1. Pedersen CT, Kay GN, Kalman J, Borggrefe M, Della-Bella P, Dickfeld T, et al. EHRA/HRS/APHRS expert consensus on ventricular arrhythmias. Heart Rhythm. 2014;11(10):e166–96.
2. Moss AJ, Schuger C, Beck CA, Brown MW, Cannom DS, Daubert JP, et al. Reduction in inappropriate therapy and mortality through ICD programming. N Engl J Med. 2012;367(24):2275–83.
3. Tung R, Vaseghi M, Frankel DS, Vergara P, Di Biase L, Nagashima K, et al. Freedom from recurrent ventricular tachycardia after catheter ablation is associated with improved survival in patients with structural heart disease: an International VT Ablation Center Collaborative Group study. Heart Rhythm. 2015;12(9):1997–2007.
4. Muser D, Santangeli P, Castro SA, Pathak RK, Liang JJ, Hayashi T, et al. Long-term outcome after catheter ablation of ventricular tachycardia in patients with nonischemic dilated cardiomyopathy. Circ Arrhythm Electrophysiol. 2016;9(10):e004328.
5. Turagam MK, Vuddanda V, Atkins D, Santangeli P, Frankel DS, Tung R, et al. Hemodynamic support in ventricular tachycardia ablation: an International VT Ablation Center Collaborative Group study. JACC Clin Electrophysiol. 2017;3(13):1534–43.