Rescue from a Storm

David Luria MD

Cardiac Arrhythmia and Pacing Unit, Heart Institute, Hadassah Medical Center and Hebrew University-Hadassah Medical School, Jerusalem, Israel

KEY WORDS: ventricular tachycardia (VT), ventricular fibrillation (VF), myocardial infarction, radiofrequency ablation, electrical storm

IMAJ 2014; 16: 511–512

Recurrent ventricular tachycardia and ventricular fibrillation episodes have become an increasingly frequent clinical problem with the ever-expanding use of implantable cardioverter defibrillators. Patients, saved from cardiac arrest by an ICD, may suffer multiple ICD shocks and require close medical attention. When ICD therapies (shocks or anti-tachycardia pacing) occurs ≥ 3 times/24 hours, it is referred to as an "electrical storm" and is recognized as a medical emergency. Indeed, recurrent frequent VT events may be symptomatic and, even more importantly, carry a significant risk of cardiac death during the short and mid-term follow-up [1-4].

About 85% of ES cases are caused by sustained monomorphic VT [4,5]. However, an important minority of cases, the remaining 15%, is due to recurrent polymorphic VT/VF. These are challenging and life-threatening cases; they include different cardiac diseases, ranging from normal structure in the case of channelopathy to severe structural disorder of the heart, and require immediate and comprehensive therapy [4-7].

In this issue of *IMAJ*, Sela et al. [8] present the case of a patient with severe ischemic cardiomyopathy (ejection fraction 15%) and ES due to polymorphic VT/VF 7 days after non-Q myocardial infarction. Over 4 days the patient experienced more than 100 (!) ICD shocks, which completely depleted the device battery. After medical therapy failed, the patient underwent successful ablation of monomorphic premature ventricular beats, triggering VT.

Polymorphic VT/VF is usually associated with non-ischemic cardiomyopathy [4]. In stable ischemic heart disease, slow and anisotropic conduction within the myocardial scar may serve as a substrate for the re-entry mechanism, causing sustained monomorphic VT and in severe cases ES.

Acute myocardial infarction or ischemia may complicate with polymorphic VT/VF. Therefore, in cases of ES due to polymorphic VT, it seems reasonable to exclude acute coronary occlusion. Surprisingly, in all reported cases, as in the current case, no evidence of acute ischemia was found and revascularization was not performed [5-7]. In these reports, ES due to polymorphic VT was not a result of temporary conditions, such as ischemia, hypokalemia or QT prolongation due to medical (anti-arrhythmic) therapy. It was caused by recurrent monomorphic premature ventricular beats, initiating VT.

Acute management of severe ES must include deep sedation (usually general anesthesia with mechanical ventilation) to decrease severe anxiety related to recurrent ICD shocks, which induce a hypercatecholaminergic state and provoke arrhythmia recurrence. The drug regimen must include beta-blockers, preferably intravenous. Non-selective beta-1 and beta-2 blockers (propranolol) were shown to have the best effect. The most efficient anti-arrhythmic drug is intravenous amiodarone [1]. Lidocaine can have a temporary effect, but high doses of the drug are usually required, imposing the risk of significant side effects.

In most ES cases, however, as in the case presented by Sela and team [8], conservative management fails and ablation has to be performed to save the patient. Ablation of ES is unequivocally recommended by the recent "Consensus Document on Catheter Ablation of Ventricular Tachycardia" [4]. While the timing of the ablation procedure is not defined in this document, many experts favor early rather than deferred intervention. In the largest series of patients undergoing ES ablation, the procedure was considered an emergency and was performed within 24 hours of hospitalization in most of the cases [6]. This approach is more applicable in large referral centers that have extensive expertise in VT ablation.

Understandably, this complicated ablation procedure in such sick patients may be reserved as a "last resort" intervention by smaller electrophysiologic groups. One needs to keep in mind, however, that early ablation has been proved superior to medical therapy in severe ES, improving immediate and mid-term results of arrhythmia control and survival [4,7].

Mapping and ablation of ES is a technically demanding procedure. Yet, in recent reports, in up to 90% of cases the procedure was successful in terminating an ES and the patients could be discharged. The complication rate was low and did not exceed 1–2% and the mortality rate was about 0.5% [4-6].

Polymorphic VT in the context of ischemic cardiomyopathy is usually triggered by monomorphic VPB arising from the myocardial scar border. Of interest, Purkinje arborization was demonstrated to be an

ICD = implantable cardioverter defibrillators
VT = ventricular tachycardia
ES = electrical storm

VPB = ventricular premature beats

essential part of polymorphic VT initiation and maintenance in many cases. Careful mapping of the scar border during sinus rhythm and during VPB and/or VT often uncovered Purkinje potentials before ventricular electrogram in successful ablation spots [6,9,10].

In the present report, Sela's team [8] did not focus on PP mapping; however, the figure in the article demonstrates endocardial recording of successful ablation points with a small, sharp, fractionated potential compatible with distal PP. Furthermore, this recording is located in the basal anterior wall, the area of the anterior left bundle fascicle. This fascicle was demonstrated to be the potential source of VPB, triggering polymorphic VT in patients after anterior myocardial infarction [6]. Mapping of PP in such a case may assist significantly in mapping and ablation, when used together with activation mapping of VPB (earliest activation spot) and pace-mapping, especially if the amount of VPB is small.

Monomorphic VT ablation, even in a case of ES, may require activation/entrainment mapping of VT and/or substrate mapping of scars and late potentials [4]. The situation is different in cases of polymorphic VT, triggered by monomorphic VPB. In the reported cases, ablation of the VPB focus was effective enough to terminate ES and prevent further VT recurrence [6,7]. It seems that, given the usually complicated clinical situation in such cases, extensive ablation of the scar border in addition to VPB focus, as performed by Sela et al., may not be essential. For the same reason, programmed ventricular stimulation at the end of the procedure, which is crucial to define the success of monomorphic VT ablation, may not be necessary after successful elimination of VPB, triggering polymorphic VT.

In summary, this case report demonstrates that even in an extremely difficult clinical situation of severe ES, ablation can rescue the patient. The procedure should be performed on an emergency basis and should be as short and precise as possible, based on an understanding of the pathophysiology of specific arrhythmia.

Contemporary advances in the ablation technique are generating new methods to improve the results of complicated ablation procedures. Among the most useful features for VT/VPB ablation in addition to three-dimensional mapping systems used by the authors, pent-array multi-polar mapping catheter and ablation systems with control of contact pressure should be mentioned. These tools enhance our capability of quick and precise mapping as well as safe and effective application of radiofrequency energy.

PP = Purkinje potentials

Correspondence
Dr. D. Luria
Director, Cardiac Arrhythmia and Pacing Unit,
Heart Institute, Hadassah Medical Center,
P.O. Box 12000, Jerusalem 9112001, Israel
Fax: (972-2) 677-7373
email: dluria@hadassah.org.il

References

1. Gao D, Sapp JL. Electrical storm: definitions, clinical importance and treatment. *Curr Opin Cardiol* 2013; 28: 72-9.
2. Nayyar S, Ganesan AN, Brooks AG, Sullivan T, Roberts-Thomson KC, Sanders P. Venturing into ventricular arrhythmia storm: a systematic review and meta-analysis. *Eur Heart J* 2013; 34: 560-9.
3. Eifling M, Razavi M, Massumi A. The evaluation and management of electrical storm. *Tex Heart Inst J* 2011; 38 (2): 111-21.
4. Aliot EM, Stevenson WG, Almendral-Garrote, JM, et al., EHRA/HRS Expert Consensus on Catheter Ablation of Ventricular Arrhythmias. *Heart Rhythm* 2009; 6: 886-933.
5. Kozeluhova M, Peichl P, Cihak R, et al. Catheter ablation of electrical storm in patients with structural heart disease. *Europace* 2011; 13: 109-13.
6. Szumowski L, Sanders P, Walczak F, et al. Mapping and ablation of polymorphic ventricular tachycardia after myocardial infarction. *J Am Coll Cardiol* 2004; 44 (8): 1700-6.
7. Bode K, Hindricks G, Piorkowski C, et al. Ablation of polymorphic ventricular tachycardias in patients with structural heart disease. *Europace* 2011; 13: 109-13.
8. Sela R, Gellerman M, Kalfon E, Atar S. Extreme electrical storm in a patient with an implantable cardioverter defibrillator. *IMAJ* 2014; 16: 513-15.
9. Bogun F, Good E, Reich S, et al. Role of Purkinje fibers in post-infarction ventricular tachycardia. *J Am Coll Cardiol* 2006; 48 (12): 2500-7.
10. Sinha A-M, Schmidt M, Marschang H, et al. Role of left ventricular scar and purkinje-like potentials during mapping and ablation of ventricular fibrillation in dilated cardiomyopathy. *PACE* 2009; 32: 286-90.