Rescue from a Storm

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current 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

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Pharmacological approaches have the advantage of being reversible and offer alternative treatments for cases in which ablation cannot be performed. However, the use of medications is often limited by side effects and patient adherence. Therefore, understanding the underlying mechanisms of VT and its triggers is crucial for developing effective treatment strategies.

In conclusion, the management of VT should be individualized, considering the patient’s characteristics, the type of VT, and the potential sources of arrhythmia. Future research should focus on identifying new targets for medication and developing innovative ablation techniques to improve the success rates and reduce the complications of this challenging condition.