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Refractory Electric Storm: A Medical Conundrum

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1. INTRODUCTION

Electrical storms are characterized by recurring episodes of ventricular tachycardia (VT) or ventricular fibrillation (VF) within a short period of time.[1] In clinical practice, an electric storm (ES) is defined by the occurrence of ≥ 2 separate VT/VF episodes or ≥3 appropriate ICD therapies for VT/VF in a 24-hour period.[2] The acute occurrence of an ES is a life-threatening condition that requires immediate intervention. Currently, Amiodarone is the first line of anti-arrhythmic treatment and has been validated in clinical trials.[3] Both Beta-blockers and Lidocaine can be given in conjunction to Amiodarone in refractory cases where additional therapies are necessary.[4]

Although the current medical therapy recommendations have some success, they are insufficient at times to acutely terminate VT/VF during an ES. Clinical trials have provided evidence that Calcium Channel Blockers, as class IV anti-arrhythmic, can be more effective in acutely terminating VT when used in conjunction with Beta-blockers.[5] The addition of intravenous Verapamil may be our solution to refractory VT/VF ES cases when standard medical therapy deems insufficient.

2. CASE STUDY

55-year-old lady with history of severe non-ischemic cardiomyopathy with ejection fraction (EF) of 20%, persistent atrial fibrillation failed cardioversion, status post VF arrest with a dual chamber AICD implantation from previous hospital admission, presents for VF storm. Her ICD interrogation demonstrated 21 appropriate ICD shocks for VF (Fig .1)which are triggered by premature ventricular contraction (PVC). She had no complaints of chest, nausea, short of breath, however she did feel warm and lightheaded just prior to the ICD firings. Her chest x-ray (Fig. 2) illustrates well positioned dual chamber AICD leads, and her lab studies demonstrate normal findings. She has already been loaded with amiodarone from a prior hospitalization, therefore a Lidocaine drip was started and the patient was admitted to ICU in critical status. Throughout the night, the patient continues to have frequent PVCs in 2 or 3 morphologies with short bursts of non-sustained VT (Fig. 3) despite of Lidocaine drip and Amiodarone. In the following morning, she was then given one time Verapamil 10 mg intravenously (IV) which eradicated all PVCs (Fig. 4). The patient was continuously monitored in the ICU with 5 mg IV Verapamil every 6 hours for 24 hours then switched to oral Verapamil. Over the following 4 days, she had only 3 episodes of PVC lasting only seconds, and her sinus rhythm was restored (Fig. 5). A repeat echocardiogram demonstrated EF of 20-25%; a slight improvement from her previous study. The benefit from Verapamil outweighs the risk despite of her low EF; this patient was discharged with Verapamil, Amiodarone, Apixaban, as well as heart failure guideline directed medical therapy with close outpatient follow-up by her cardiologist.

3. DISCUSSION

Electrical storm (ES) is a life-threatening emergency characterized by 3 or more sustained VT or VF episodes or appropriate ICD shocks within 24 hours.[6] Its very existence is associated with both higher mortality as well as increased rate for hospitalization. In the AVID trial, 38% mortality among those experienced ES vs. 15% who never had ES; hence ES is a significant independent risk for death.[2]ICD shocks also have been associated with a 3-factor increased in mortality when compared to no shocks; even inappropriate shocks are associated with a trend towards increased risk when compared to those never experienced ICD shock.[7]Hence, it is imperative to eliminate ICD shocks as quickly and effectively as possible.

Although the current treatment recommendation for VT/VF storm works well in certain cases, those that are refractory to the standard therapy are particularly concerning. Before taking on additional risk with invasive ablation therapy, Verapamil, a class 4 antiarrhythmic canbe a suitable alternative.

Non-dihydropyridinecalcium channel block (CCB) such as Verapamil exerts its effect on the L-type voltage-dependent calcium channels (LTCC), which initiates a calcium activated release of calcium from the sarcoplasmic reticulum leading to cellular depolarization. However, in a pathological state, excessive calcium release during sympathetic activation will generate a depolarizing current escalate to delayed after depolarization (DAD) which may serve as a pro-arrhythmic substrate leading to cardiac arrhythmias.[8]Verapamilexerts a systematic blockade directed at these pathological LTCC and successfully diminishes risk of cardiac arrhythmia. In animal models, DADs generated in the epicardium are

exquisitely prone to arrhythmia, as these extra systoles increase the dispersion of repolarization and facilitate bidirectional VT to VF. By its direct effects on LTCC in these epicardial cells, Verapamil successfully extinguishes these extra-systoles before its initiation. Further, when Verapamil is combined with a beta-blocker in CPVT treatment, Verapamil further diminishes calcium passage either by directly blocking the mutated Ryanodine channel through which calcium is released from the Sarcoplasmic Reticulum or indirectly by reducing cyclic AMP, hence reducing the total intracellular calcium concentration and thereby reducinggardiac arrhythmia.[5]

Other studies have demonstrated calcium antagonism by Verapamil can improve repolarization reserve, resulting in protection from Dofetilide induced torsade de point. As it turns out, "These repolarization-dependent arrhythmia occurs… when [repolarization] reserve is challenged beyond capacity, as in long QT syndrome or in congestive heart failure.[9]" Oros, Houtman, Neco, et al. through their work have demonstrated by blocking the LTCC, it protects the heart from developing early after depolarizations (EAD), restores repolarization reserve, and serves as a physiological stabilizer against arrhythmia.[9]Hence, Verapamil is specifically effective in suppressing triggered ventricular tachycardia activity related to calcium overload and hence reducing substrates for cardiac arrhythmia.

It should be noted however, Verapamil does have its negative hemodynamic and inotropic effects; particularly in those with heart failure, cardiogenic shock, severe hypotension, or high degree heart block. Although most patient with severe adverse effects due to Verapamil had prior cardiac conditions, these adverse events can occur in patients with normal functioning hearts. Thus, verapamil may terminates VT, its utilization should be carefully balanced by its adverse effects.[10]On the other hand, in the right circumstances, such as in our patient, who is suffering from refractory ES despite multiple anti-arrhythmic agents, Verapamil offers a complementary therapy with very satisfying results.

4. CONCLUSION

ES is a life threatening medical emergency that warrants immediate intervention. Although current therapy has sufficient support and medical evidence, it is at times inadequate. Verapamil, as a class 4 anti-arrhythmic may be an alternative for refractory ES, such as in our patient where Verapamil obliterated refractory ES and its triggering substrates without sequalae. Despite of contraindications and hemodynamic risks to Verapamil's use, we need to recognize its unique niche in the treatment of refractory ES.

Figures

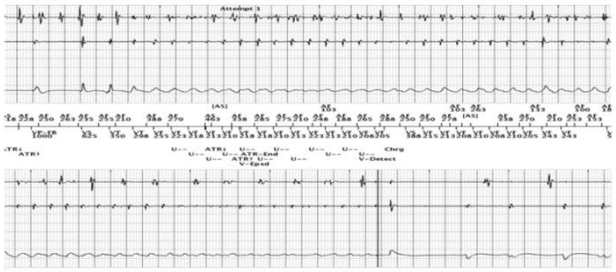

Fig. 1 AICD interrogation demonstrates 21 appropriate ICD shocks for Ventricular fibrillation

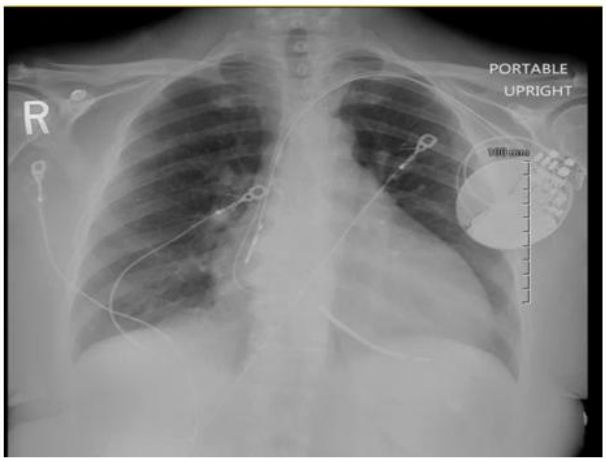

Fig 2. Cardiac Defibrillator leads are stable in position. Cardiomegaly without acute findings

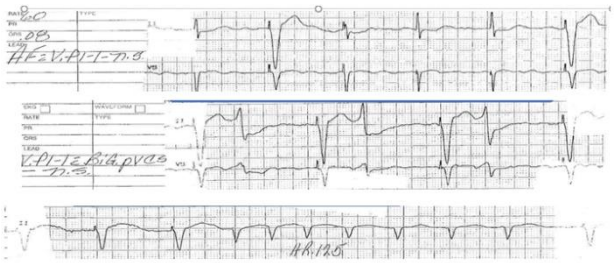

Fig. 3 Ventricular paced rhythm with frequent PVC; short non-sustained VT

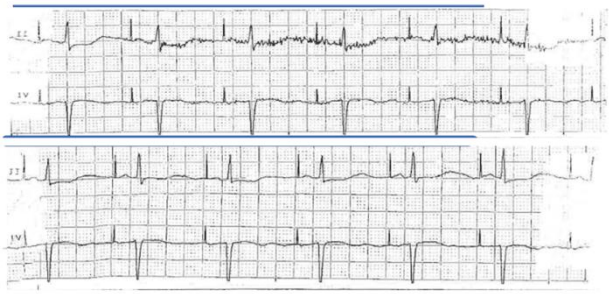

Fig. 4 Atrial paced rhythm without other cardiac arrhythmia

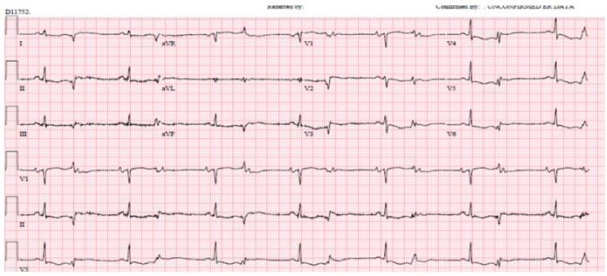

Fig. 5 Sinus Rhythm with frequent PVC, nonspecific ST changes with prolonged QT

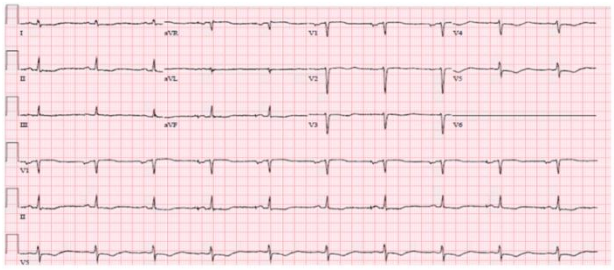

Fig. 6 Normal Sinus rhythm with non-specific ST changes

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