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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

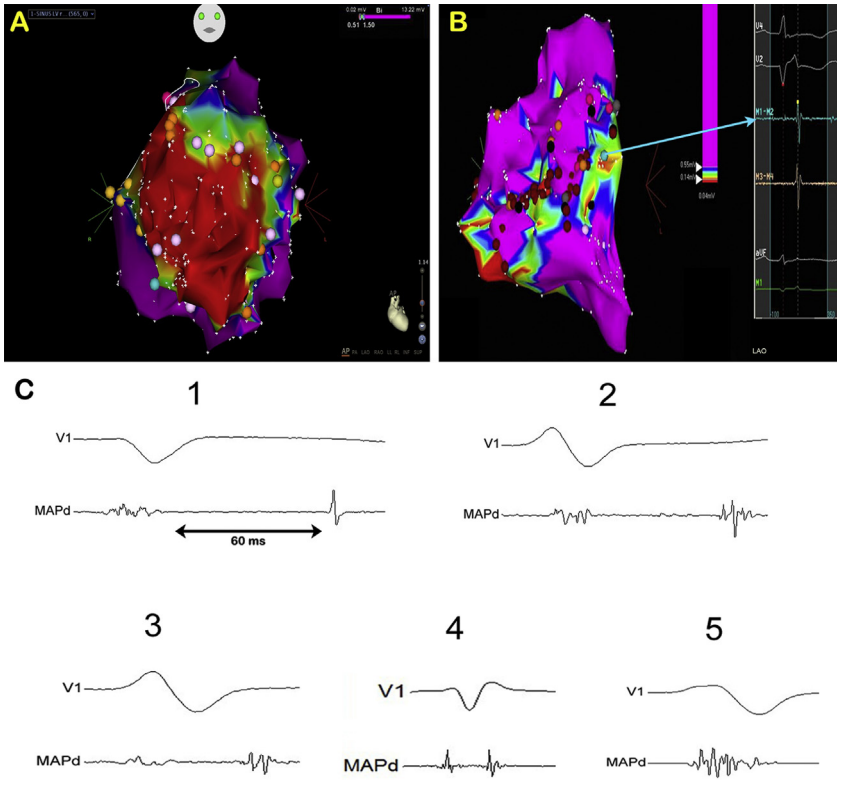

Fig. 1. Panel A: LV Voltage map in sinus rhythm of a patient in AP view showing areas of scar (denoted in red, with voltage of <0.5 mV) and normal myocardium (denoted in pink, with voltage of >1.5 mV). The areas between the scar and the normal myocardium are low voltage corridors containing abnormal 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. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.
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).

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