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Acute outcome of treating patients admitted with electrical storm in a tertiary care centre



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ABSTRACT

Background: Electrical storm (ES) is a life threatening emergency. There is little data available regarding acute outcome of ES.

Aims: The study aimed to analyze the acute outcome of ES, various treatment modalities used, and the factors associated with mortality.

Methods: This is a retrospective observational study involving patients admitted with ES at our centre between 1/1/2007 and 31/12/2013.

Results: 41 patients (mean age 54.61 \pm 12.41 years; 86.7% males; mean ejection fraction (EF) 44.51 \pm 16.48%) underwent treatment for ES. Hypokalemia (14.63%) and acute coronary syndrome (ACS) (14.63%) were the commonest identifiable triggers. Only 9 (21.95%) patients already had an ICD implanted. Apart from antiarrhythmic drugs (100%), deep sedation (87.8%), mechanical ventilation (24.39%) and neuraxial modulation using left sympathetic cardiac denervation (21.95%) were the common treatment modalities used. Thirty-three (80.49%) patients could be discharged after a mean duration of 14.2 \pm 2.31 days. Eight (19.5%) patients died in hospital. The mortality was significantly higher in those with (19.5%) patients died in hospital. The mortality was 0 (0%), p = 0.03)). There was no significant difference in mortality between those with versus without a structural heart disease (8 (21.1% vs 0 (0%), p = 0.32)). Comparison of mortality an ACS with ES versus ES of other aetiologies (3 (50%) vs 5 (14.29) %, p = 0.076)) showed a trend towards significance. *Conclusion:* With comprehensive treatment, there is reasonable acute survival rate of ES. Hypokalemia and ACS are the commonest triggers of ES. Patients with low EF and ACS have higher mortality.

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Introduction

Acute Electrical storm (ES) is a life threatening emergency and carries a significant risk of mortality. There is little data regarding acute outcome of ES [1], especially from the developing world. Similarly, less is known on ES occurring in patients who have not undergone an implantable cardioverter defibrillator (ICD) placement, since most of the studies pertaining to ES are confined to patients with ICDs [2–4]. The present study is a single center experience of treating patients with acute ES irrespective of ICD implantation, focusing on the treatment modalities used, outcome and the factors associated with mortality.

Objectives

Primary aim of the study was to analyze the acute outcome of ES, whereas the secondary aim was to analyze the various treatment modalities used, and the factors associated with increased mortality.

Methods

This is a retrospective observational study involving patients with Electrical storm, between 1/1/2007 and 31/12/2013, at Sree Chitra Tirunal Institute for Medical sciences and Technology (SCTIMST). The demographic parameters, the treatment modalities used, and the acute outcome were analyzed. Subjects were retrospectively recruited based on the Hospital Medical records. The electrocardiogram (ECG) and/or ICDelectrograms (EGMs) (for patients with ICD) were used to diagnosis and delineate the details of the ventricular arrhythmia (VA). Whenever ICD-EGMs were used rate, morphology, stability, onset and AV dissociation were used to discriminate VT from supraventricular arrhythmias. All patients who were admitted for treatment of ES were included in the study. Those with ES occurring within 1 week of ICD implantation were excluded as ES is known to be triggered during this period. Patients were also excluded if the available data was incomplete.

Definitions

Electrical storm (ES)

Recurrent ventricular arrhythmias (VA) in a short time (\geq 3 separate episodes in 24 hrs, each requiring termination by intervention) or frequent defibrillator therapies (\geq 3 separate discrete episodes of VAs, separated by more than 5 min in 24 hrs) or incessant VA (continuous VA that recurred promptly despite intervention for termination over 12 hrs) [4,5].

Cessation of ES

ES was considered to be ceased after at least a 7 day-period free of recurrent VAs.

Ventricular tachycardia (VT)

VT was diagnosed by the standard ECG when available. When Electrocardiographic (ECG) record of the VA could be obtained, VF and polymorphic VT were diagnosed based on QRS morphologies. When electrograms from ICDs (EGMs) alone were available, VAs with <30 ms cycle length (CL) variation were considered monomorphic, while those with CL variation >30 ms were regarded as polymorphic [6].

Ventricular fibrillation (VF)

Electrocardiographic documentation of VF, or any VA of rate >250/min with varying cycle length when ICD electrograms alone were available.

Structural heart disease (SHD)

Was defined, for the purpose of this study, as diseases with echocardiographically detectable abnormality.

Statistical analysis

All the quantitative data are reported as mean \pm S.D. Qualitative data are expressed as proportions. All the analyses were done using the SPSS 16 software. Fischer exact test was used for comparison of categorical data.

Results

The baseline parameters of the patients are shown in Table 1 The mean age was 54.61 \pm 12.41 years and 31 (86.7%) were males. The mean ejection fraction (EF) was $44.51 \pm 16.48\%$. The aetiological distribution of the patients is shown in Fig. 1. Coronary artery disease was the commonest underlying disease. The mean number of VAs per ES episode was 11.15 ± 15.48 and the mean rate of VA during ES was 179.46 \pm 69.46. The morphology of VA during the ES was RBBB 18 (43.9%), LBBB 12 (29.27%), Polymorphic/VF 8 (19.51%), ICD EGM alone in 3 (7.31%). Though a clear triggering factor could not be identified in the majority (60.97%), Hypokalemia (14.63%) and acute coronary syndrome (ACS) (14.63%) remained the commonest identifiable triggers that precipitated an ES. The mean potassium level was 2.8 \pm 0.22 mEq/dL in those having hypokalemia. In a patient with Brugada syndrome, fever precipitated the ES and he was treated with paracetamol, tepid sponging and Isoprenaline infusion. Only 9 (21.95%) patients had an ICD implanted before the occurrence of ES. The appropriate ICD intervention during the ES was shock alone in 3 (33.3%), and Anti Tachycardia Pacing (ATP) with Shock in 6 (66.6%) of the patients.

Modalities used in treating ES (Table 2)

The various treatment modalities used in the management of ES are shown in Table 2. Apart from antiarrhythmic drugs which were invariably used, 36 (87.8%) patients underwent deep sedation and 10 (24.39%) underwent mechanical

Table 1 – Parameters of the ES cohort (N $=$ 36).			
Parameter	Frequency		
Age (Years)	54.61 ± 12.41		
Sex	Males 35 (85.36%)		
	Females 6 (14.64%)		
EF (%)	44.51 ± 16.48		
SHD	38 (92.68%)		
Severe LV dysfunction (EF<35%)	27 (65.85%)		
Type of VA during ES			
Monomorphic VT	33 (80.49%)		
Polymorphic VT	2 (4.88%)		
VF	6 (14.63%)		
No. of VA episodes per ES	14.25 ± 10.48		
Triggers of ES			
ACS	6 (14.63%)		
Hypokalemia	6 (14.63%)		
Worsening HF	3 (7.51%)		
Fever	1 (2.31%)		
None identified	25 (60.97%)		
Patients already having an ICD	9 (21.95%)		
ICD intervention (in patients already on	tients already on Shock alone 3 (33.3%)		
ICD)	ATP + Shock 6 (66.6%)		
Mean No. of VA episodes	14.25 ± 10.48		
Rate during ES (per minute)	192.46 ± 58.41		
RFA in acute setting	1 (2.43%)		
Acute Outcome	Survival 33 (80.49%)		
	Mortality 8 (19.51%)		

EF-Ejection fraction, ES-Electrical storm, ICD-Implantable cardioverter Defibrillator, RFA-Radiofrequency ablation, SHD- Structural heart disease, VT-Ventricular tachycardia, VA Ventricular arrhythmia. control of ES in 7 (77.78%) patients. Only one patient (2.78%) underwent RFA in the setting of ES and RFA could control ES in this patient. Metoprolol followed by Amiodarone were the commonest drugs to be used. Amiodarone was not used in two patients who had previous history of thyrotoxicosis related to Amiodarone intake. Sotalol was used in them. Isoprenaline infusion proved to be useful in a patient with Brugada syndrome whereas Diltiazem was used in a patient with idiopathic short coupled torsades. Mexiletine was used as a first line drug in a patient with long QT syndrome (LQTS-3), and also in four other patients as an add-on medication after parenteral Lidocaine was stopped. Temporary pacing to shorten the heart rate was used in three patients. In patients in whom an ICD was implanted, reprogramming of the device was carried out to switch off the delivery of shocks and promote anti-tachycardia pacing (ATP).

Acute outcome

Thirty-three (80.49%) patients could be discharged from the hospital after successfully controlling ES, after a mean duration of 14.2 ± 2.31 days. Eight (19.5%) patients died in hospital. Three of these mortalities were related to ES occurring in the background of acute coronary syndrome (ACS). All of them underwent adequate revascularisation. One of them died of refractory ventricular fibrillation (VF), whereas worsening heart failure and cardiogenic shock, with superimposed ventricular arrhythmia episodes caused death in the other two. Three had ischemic cardiomyopathy and dilated cardiomy-

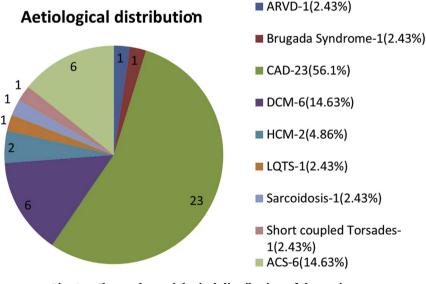


Fig. 1 – Shows the aetiological distribution of the patients.

ventilation. Neuraxial modulation using pharmacological left or bilateral stellate ganglion blockade or surgical left sympathetic cardiac denervation was performed in 9 (21.95%) patients. Five of these patients had pharmacological left stellate ganglion blockade initially, and later underwent surgical or thoracoscopy guided left sympathetic cardiac denervation (LSCD). One of these cases has been reported previously, and the technical details explained [7]. This was effective in acute opathy was the substrate in two; all with severe LV dysfunction. Two of these deaths were due to worsening hypotension, refractory heart failure culminating in electromechanical dissociation/asystolic arrest. The mortality was significantly higher in those with EF<35% compared to those with a higher EF (8 (42.11% vs 0 (0%), p = 0.03)). However, there was no significant difference in mortality between those with versus without a structural heart disease (8 (21.1% vs 0 (0%), p = 0.32)).

Table 2 – Various treatment modalities and antiarrhythmic drugs used in the acute treatment of ES.			
Modality	N (%)	Drugs used	N (%)
Antiarrhythmic drugs	41 (100%)	Beta Blockers	39 (95.12%)
Deep Sedation	36 (87.8%)	Parenteral Amiodarone	35 (85.36%)
Ventilation	10 (24.39%)	Lignocaine	10 (24.39%)
ICD programming	9 (21.95%)	Mexiletine	5 (12.2%)
Neuraxial modulation	9 (21.95%)	Sotalol	2 (4.87%)
RFA	1 (2.4%)	Verapamil	1 (2.4%)
ICD-Implantable cardioverter Defi	brillator, RFA-Radiofrequency ablati	on.	

Analysis of mortality due to ES occurring in the setting of an ACS versus ES of other aetiologies (3 (50%) vs 5 (14.29) %, p = 0.076) showed a trend towards significance.

Discussion

The present study has a few unique features. Firstly, the acute outcome of ES has not been studied so far, especially in patients without an ICD. Secondly, only patients needing inpatient treatments for ES were included in the present study. Thirdly, and more strikingly, the use of radiofrequency ablation in the acute setting of ES was very less. The present study thus represents the real-life scenario in much of the developing world where most patients do not afford an ICD, and sophisticated modalities like electroanatomical mapping are not available round-the clock even in tertiary care centres.

Similar to most other studies on ES, males were predominant in the present study as well [3,8]. There was no clear trigger for ES in the majority. However, it is important to note that acute coronary syndrome and hypokalemia were the most common triggers identifiable. This is important because both are amenable to treatment. Hypokalemia is common in patients with heart failure or LV dysfunction, due to diuretic use, and hence frequent monitoring of potassium levels and meticulous correction of hypokalemia when present can potentially prevent an ES event.

Only 19.51% of the patients in the ES group had VF/Polymorphic VT as the causative arrhythmia. The incidence of VF as the causative arrhythmia, and the definition for classifying VA as VF varies among various studies. The incidence was comparable to that in many other studies [3,9]. A large proportion of our patients (63.9%) had coronary artery disease (CAD). Thus scar tissue was as the possible substrate of VA in these patients. This may also explain the lower incidence of VF in our patients, as scar tissue is more likely to have multiple potential re-entrant circuits, and likely to sustain monomorphic VT.

The use of radiofrequency ablation

The use of RFA in the acute setting was extremely low in the present study. There is variable evidence regarding the benefit of RFA in the setting of ES. Izquierdo et al. [10] in their study, have reported a 38% recurrence of ES after a single RFA procedure in patients with ES. However, in a larger study Carbucicchio et al. [11] reported a 92% ES free survival at 22 months. However in this series, multiple sessions of RFA were

performed if needed and endpoint of non-inducibility of VT after ablation was used. The proportion of patients on beta adrenergic receptor blockers and Amiodarone in our study was higher than that in other studies [2,3,6,9,12].

Outcome

There is no available data regarding acute outcome of ES. In the present study there was a reasonable survival of 80.49%. The mortality was higher in patients with EF < 35%, and when ACS was the aetiology of ES. Another notable feature was that worsening heart failure and cardiogenic shock, and not the arrhythmic event itself, was the final cause of death in a significant majority. This is concordant with the finding of Mitchell et al who noted that electromechanical dissociation accounted for a significant proportion of sudden deaths in patients who had undergone an ICD implantation [13].

Conclusions

The acute mortality of ES can be high. A comprehensive management strategy can result in reasonable acute survival in patients. Neuraxial modulation with left sympathetic cardiac denervation is a promising strategy in this regard. Hypokalemia and ACS are the commonest triggers of ES. This is particularly of importance in resource limited background in developing nations, where use of ICD and RFA is still very low. Worsening heart failure and cardiogenic shock are important mechanisms of mortality apart from the arrhythmia itself. Patients with severe LV dysfunction and ACS have significantly higher mortality due to ES.

Clinical implications

Close monitoring of patients with heart failure or LV dysfunction to detect and correct hypokalemia may help in preventing ES. Neuraxial modulation with LSCD is a useful strategy that can be used when more sophisticated techniques are not readily available. Severe LV dysfunction and ACS may be simple clinical markers to identify patients at especially high risk of mortality.

Limitations

The retrospective design of the study has its inherent limitations. Most of the published studies on ES are of retrospective design. The study population was heterogeneous regarding the underlying heart disease and therapeutic measures used, and thus the conclusions may not be uniformly applicable to all patients alike.

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None.

Author contributions

Dr. Mukund A Prabhu is involved in the conception, design, drafting, collection and interpretation of data.

Dr.Srinivas Prasad and Dr.Abhilash S P is involved in collection of data, interpretation of data and drafting the manuscript.

Dr.Anees Thajudheen and Dr. Narayanan Namboodiri are involved in designing the work, drafting the manuscript, critical revision and final approval of the work.

Dr. Ajith Kumar V K is involved in the final approval of the article.

Conflicts of interests

None of the authors have any competing interests or conflicts of interests to declare.

REFERENCES

- Conti S, Pala S, Biagioli V, Del Giorno G, Zucchetti M, Russo E, et al. Electrical storm: a clinical and electrophysiological overview. World J Cardiol 2015;7(79):555–61.
- [2] Arya A, Haghjoo M, Dehghani MR, Fazelifar AF, Nikoo MH, Bagherzadeh A, et al. Prevalence and predictors of electrical storm in patients with implantable cardioverter-defibrillator. Am J Cardiol 2006 Feb 1;97(3):389–92.
- [3] Credner SC, Klingenheben T, Mauss O, Sticherling C, Hohnloser SH. Electrical storm in patients with transvenous implantable incidence, management and prognostic implications. J Am Coll Cardiol 1998;32(7):1909–15.

- [4] 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 Feb;34(8):560–71.
- [5] Zipes DP, Camm AJ, Borggrefe M, Buxton AE, Chaitman B, Fromer M, et al. European heart rhythm association; heart Rhythm Society, ACC/AHA/ESC 2006 guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: a report of the American College of CardiologyAmerican Heart Associa. Am Coll Cardiol 2006;48(5):e247–346.
- [6] Verma A, Kilicaslan F, Marrouche NF, Minor S, Khan M, Wazni O, et al. Prevalence, predictors, and mortality significance of the causative arrhythmia in patients with electrical storm. J Cardiovasc Electrophysiol 2004;15(11):1265–70.
- [7] Gadhinglajkar S, Sreedhar R, Unnikrishnan M, Namboodiri N. Electrical storm: role of stellate ganglion blockade and anesthetic implications of left cardiac sympathetic denervation. Indian J Anaesth 2013;57(4):397–400.
- [8] Hohnloser SH, Al-Khalidi HR, Pratt CM, Brum JM, Tatla DS, Tchou P, et al. Electrical storm in patients with an implantable defibrillator: incidence, features, and preventive therapy: insights from a randomized trial. Eur Heart J 2006 Dec;27(24):3027–32.
- [9] Exner DV, Pinski SL, Wyse DG, Renfroe EG, Follmann D, Gold M, et al. Electrical storm presages nonsudden death. Circulation 2001;103(16):2066-71.
- [10] Izquierdo M, Ruiz-granell R, Ferrero A, Martínez A, Sánchez-Gomez J, Bonanad C, et al. Ablation or conservative management of electrical storm due to monomorphic ventricular tachycardia: differences in outcome. Europace 2012;14:1734–9.
- [11] Carbucicchio C, Santamaria M, Trevisi N, Maccabelli G, Giraldi F, Fassini G, et al. Catheter ablation for the treatment of electrical storm in patients with implantable cardioverterdefibrillators: short- and long-term outcomes in a prospective single-center study. Circulation 2008 Jan 29;117(4):462–9.
- [12] Sesselberg HW, Moss AJ, McNitt S, Zareba W, Daubert JP, Andrews ML, et al. Ventricular arrhythmia storms in postinfarction patients with implantable defibrillators for primary prevention indications: a MADIT-II substudy. Heart Rhythm 2007 Nov;4(11):1395–402.
- [13] Mitchell LB, Pineda EA, Titus JL, Bartosch PM, Benditt DG. Sudden death in patients with implantable cardioverter defibrillators: the importance of post-shock electromechanical dissociation. J Am Coll Cardiol 2002;39(8):1323-8.