Original Article

Effectiveness of ultrasonography-guided cardiac sympathetic denervation in acute control of electrical storm: A retrospective case series

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Abstract

Background and Aims: Ultrasonography-guided left cardiac sympathetic denervation (LCSD) or bilateral cardiac sympathetic denervation (BCSD) may be a useful intervention in the electrical storm (ES) that persists despite pharmacological therapy. The aim of our study was to evaluate the effectiveness of ultrasonography-guided LCSD or BCSD in the acute control of ES. We conducted a retrospective case series of patients who underwent ultrasonography-guided CSD for control of ES at a tertiary care hospital.
Material and Methods: Data of all patients who underwent unilateral or bilateral CSD were collected from January 2017 to December 2019. Eleven patients with ES refractory to standard antiarrhythmic therapy underwent ultrasonography-guided pharmacological CSD (eight underwent LCSD and three underwent BCSD). Quantitative data was expressed as mean and median with interquartile range (IQR). Non-quantitative data was expressed in proportions.
Results: Eleven patients underwent ultrasonography-guided pharmacological CSD (eight underwent LCSD and three underwent BCSD). Six of the eleven patients were female (54.5%). Ischemia was the underlying substrate in nine patients (81.8%). Five patients (46%) had complete resolution of ventricular tachycardia (VT) after CSD and one had 90% reduction in episodes of VT. The median follow-up duration was 8 months inter-quartile range IQR (7–18). One patient succumbed to heart failure and one patient was lost to follow up. The other patients had no further events and were well at last follow up.
Conclusion: Ultrasonography-guided pharmacological CSD is effective in the acute control of ES. It is easily performed with equipment that is readily available and relatively safe in terms of immediate complications and is an ideal second-line intervention when ES persists despite drug therapy.

Keywords: Cardiac sympathetic denervation, electrical storm, neuraxial modulation

Introduction

Prior to the wide usage of implantable cardioverter defibrillators (ICDs), the term "electrical storm" was referred to the occurrence of two or more ventricular tachycardia (VT) or ventricular fibrillation (VF) in a 24-hour period.[1] The term electrical storm (ES) refers to "three or more separate arrhythmia episodes leading to ICD therapies including anti-tachycardia pacing or shock occurring over a 24-hour period."[2-4] According to the current definition of ES, the incidence is about 10% to 20% in patients who have an ICD

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for secondary prevention of sudden cardiac death[5-7] and lower when ICDs are placed for primary prevention.[8] In the MADIT II study, 4% of patients developed ES on an average of 20.6 months.[9] The autonomic (sympathetic) nervous system plays an important role in the pathogenesis of ES.[10] Neuraxial modulation by various means has been shown to be effective in the acute control of ES. Left sympathetic cardiac denervation (LCSD) was first shown to be effective in reducing the burden of VT in patients with congenital long QT syndrome in 1916.[11] Neuraxial modulation for the control of ES may be achieved by ultrasonography-guided pharmacological LCSD or bilateral cardiac sympathetic denervation (BCSD). It may also be achieved by open surgical or minimally invasive video-assisted thoracoscopy surgery (VATS) guided approaches.[12] We aimed to evaluate the effectiveness of ultrasonography-guided LCSD or BCSD in the acute control of patients with ES that persisted despite parenteral anti-arrhythmic drug therapy.

Material and Methods

A retrospective analysis of data collected from consecutive patients, who underwent CSD (either left or bilateral) for the control of ES at our institute, from January 2017 to December 2019 was performed. At our institute, the management of ES in patients consists of several simultaneous interventions (described below), tailored to the specific needs of the patient [Figure 1]. Study was done after taking clearance from institutional ethics committee of hospital (IEC – 663/2019).

CSD technique

The aim of the procedure is to attain percutaneous left or bilateral stellate ganglion blockade. Ultrasonography-guided injection of ropivacaine is performed by the single anesthetist who is experienced in performing nerve blocks. The procedure is performed under local anesthetic or sedation. The patient is positioned supine, with the neck slightly extended. The region of the stellate ganglion is accessed from the anterior paratracheal approach at the level of the sixth cervical vertebra. Under strict asepsis, a 7.5-MHz ultrasound probe is placed at the level of the cricoid cartilage. The C6 transverse process is identified by its prominent anterior tubercle. A 23-G 4-cm long needle is advanced through the prevertebral fascia until its tip is positioned in the longus colli muscle [Figure 2]. Slow injection of 10-ml 0.20% ropivacaine solution is made into the longus colli compartment. In our center, ropivacaine is used instead of bupivacaine as ropivacaine is more lipophilic and has stereoselective properties leading to lesser cardiotoxic and nervous system toxicity, though the efficacy of ropivacaine is similar to bupivacaine for peripheral nerve blocks. Warming

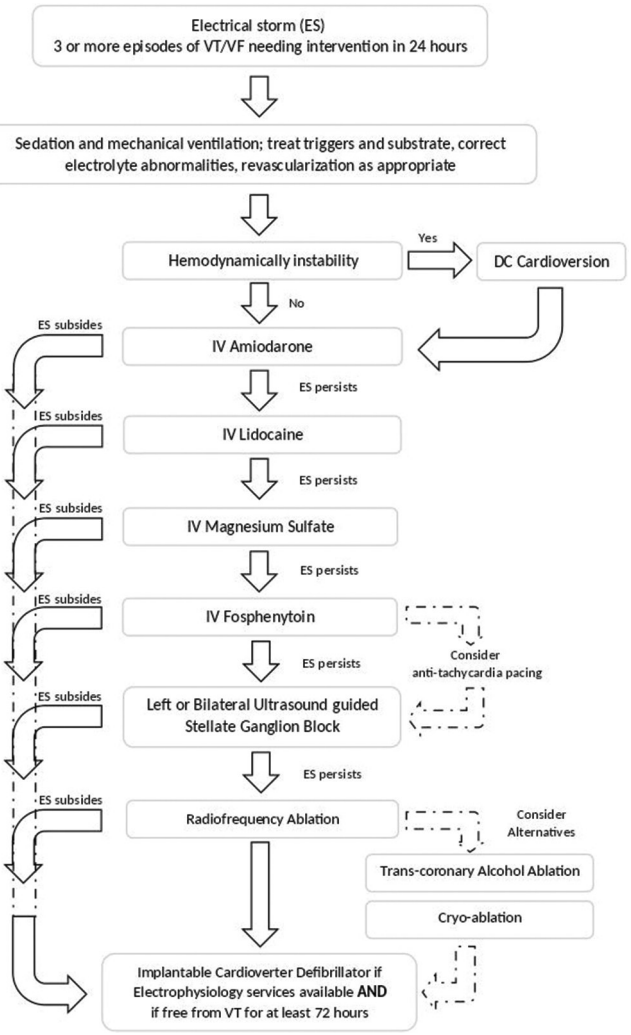

Figure 1: Flow chart showing our approach to electrical storm. ES–electrical storm, IV–intravenous, VT–ventricular tachycardia, VF–ventricular fibrillation

of the left upper limb and the development of left Horner's syndrome are considered an indication of successful LCSD. Right CSD is performed in the same manner for patients in whom ES persists beyond 24 hours after LCSD.

Quantitative data was expressed as mean and median with interquartile range (IQR). Non-quantitative data was expressed in proportions. Statistical analysis was performed using the SPSS 17 software.

Results

General measures include securing the airway and inhaled oxygen as appropriate, central venous or large bore peripheral venous access. The patient is sedated and if necessary, even mechanical ventilation is initiated, with the intent to suppress any adrenergic stimulation. Most patients were sedated using one of, or a combination of fentanyl, midazolam, and morphine. Four patients who did not achieve adequate analgesia and

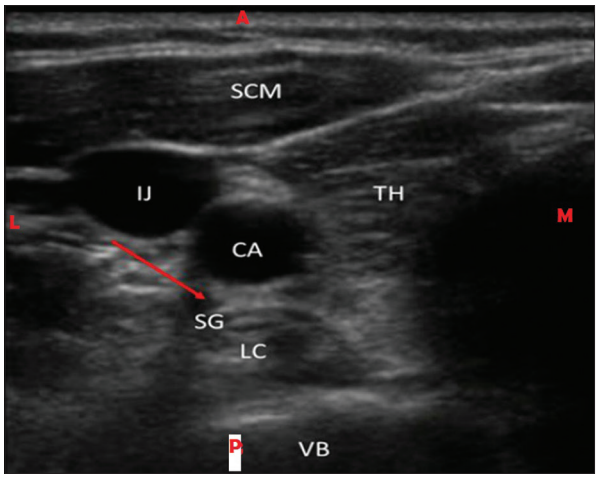

Figure 2: Image showing ultrasonography-guided cardiac sympathetic denervation. Red arrow shows the path of needle. CA–carotid artery, IJ–internal jugular vein, LC– longus colli, SCM–sternocleidomastoid, SG–stellate ganglia, TH–thyroid, VB–vertebral body, A–anterior, P–posterior, L–lateral, M–medial

sedation with aforementioned measures underwent intubation followed by mechanical ventilation. Correction of potential triggers includes correction of metabolic parameters and treatment of ischemia, ideally by revascularization, most often by percutaneous coronary intervention (PCI). In case of ES with incessant ventricular arrhythmia in patients with coronary anatomy not suitable for PCI, intra-aortic balloon pump counter-pulsation (IABP) is considered. Intravenous antiarrhythmic drugs constitute the mainstay of first-line therapy in ES. Amiodarone and lidocaine are commonly used drugs. Beta-blockers are also used in patients who are hemodynamically stable (systolic blood pressure >90 mmHg, no evidence of cardiogenic shock or pulmonary edema, and PR interval on electrocardiogram less than 200 ms). Drugs generally reserved for second-line therapy include intravenous fosphenytoin and, in case of hypomagnesemia or polymorphic VT, intravenous magnesium sulphate. Intravenous lidocaine is followed up by oral mexiletine, or if unavailable, oral phenytoin. In case of refractory ES that persists despite these interventions, neuraxial modulation is performed. CSD was the usual mode of neuraxial modulation with either left or bilateral CSD. The next step in management, if the ES is successfully treated and VT does not recur for 72 hours, is inserting an ICD [Figure 1].

All patients received intravenous amiodarone. Beta blocker in the form of oral metoprolol was only prescribed to one patient (either not tolerated or contraindicated as a result of hemodynamic instability in the others). Intravenous lidocaine was used in all patients and was followed by oral mexilitine in two patients and oral phenytoin in two patients. Intravenous magnesium sulfate was used in ten out of eleven patients (one patient had polymorphic VT and others had hypomagnesemia with magnesium level less than 1.8 mg/dL). Intravenous fosphenytoin was used in three out of eleven patients (27.3%) and was followed by oral phenytoin in two patients. Amiodarone was administered as a bolus of 150 mg in 5% dextrose solution and followed by an infusion at the rate of 1 mg per minute for a duration of six hours, then 0.5 mg per minute infusion for eighteen hours. In two patients, the infusion was continued for a total of three days after which oral amiodarone was started at 800 mg per day. Lidocaine was administered as a bolus dose of 1 mg per kilogram body weight over 3 minutes followed by an infusion at 2 mg per minute for 24–48 hours. Intravenous fosphenytoin was administered as a bolus dose of 15–20 mg per kilogram body weight over one hour. A repeat bolus was administered for breakthrough episodes followed by an oral maintenance dosage of 5 mg/kg/day. Intravenous magnesium sulfate (1 g) was administered as a slow infusion, while monitoring deep tendon reflexes. Oral mexiletine was administered at a dose of 150 mg twice a day.

From January 2017 to December 2019, eleven patients underwent left or bilateral CSD for the control of ES. The clinical details are summarized in Table 1 ("episodes prior to SCD" refer to the number of episodes of VT that mandated electrical cardioversion). Six of the eleven patients were women (54.5%). The ages of the patients ranged from 38–76 years (median 63 years; IQR 57–70 years). Ischemic/infarcted myocardium was the underlying substrate in nine patients (81.8%). Two patients had dilated cardiomyopathy (DCM) which was confirmed by cardiac MRI. One of them had previously undergone implantation of a cardiac resynchronization therapy (CRT-P) device on account of severe left ventricular systolic dysfunction with complete left bundle branch block. The other patient with DCM already had an ICD prior to developing the event.

Seven patients had monomorphic VT (63.6%) and the remainder had polymorphic VT [Figure 3-a and 3-b]. The mean heart rate of the VT was 214.5 ± 36.5 in those with monomorphic VT. Among the patients with monomorphic VT, four had right bundle branch (RBBB) like morphology, whereas three had left bundle branch (LBBB) like morphology. None of the episodes of VT in our patients were primary VT (i.e. VT occurring within 48 hours after an acute myocardial infarction). All patients with ES, as a result of coronary artery disease, had already undergone revascularization procedures at the time when they developed ES. Patient no. 1 developed ES on the fourth day of an acute anterior wall myocardial infarction (MI). He had already undergone percutaneous intervention and stenting to left anterior descending artery (LAD). A 60% ostial stenosis noted in the left circumflex artery (LCx) was managed conservatively. Patient no. 2 had presented to us late after MI (>24 hours) and underwent successful

revascularization (LAD). She developed VT storm three days after PCI. Patient no. 4 already had an ICD device implantation in the past after he suffered from VT [he had undergone PCI to right coronary artery (RCA) and LAD]. Patient no. 5 presented to us with ES with a history of revascularization to RCA in the past. Patient no. 6 underwent percutaneous intervention to LAD in view of anterior wall MI (complete revascularization) prior to developing ES. Patient no. 7 developed ES on the sixth day following coronary artery bypass grafting (grafts to LAD, ramus artery, and RCA). Patient no. 8 underwent revascularization to RCA in view of inferior wall MI and developed ES on the fourth day following PCI. Patient no. 9 underwent revascularization to LCx artery in view of posterior wall MI. Patient no. 11 underwent revascularization to LAD and LCx artery in view of anterior wall MI (complete revascularization). In all the cases with acute coronary syndrome, ES occurred following coronary revascularization. Patient no. 1, 2, and 7 underwent IABP insertion in view of cardiogenic shock.

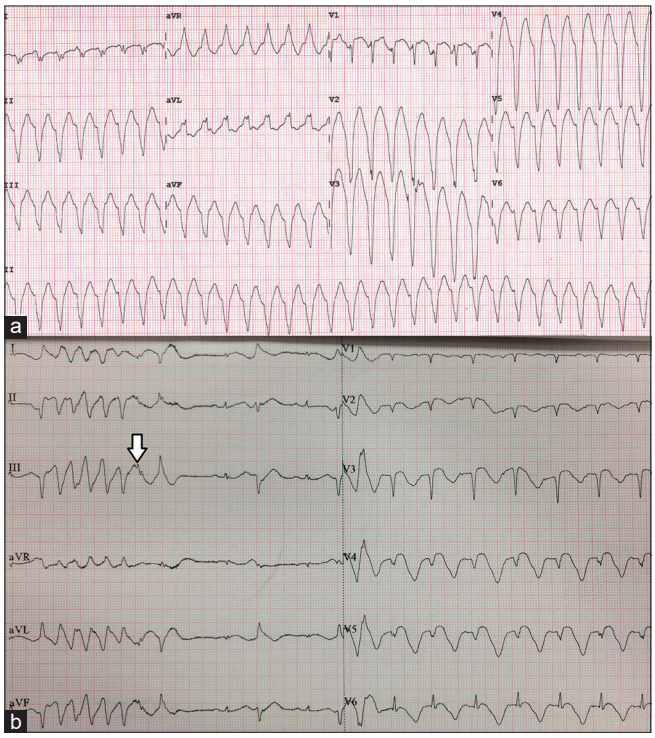

Figure 3: (a) 12 Lead ECG from a patient who had broad QRS tachycardia of LBBB morphology with northwest axis and negative concordance suggestive of monomorphic VT. (b) 12 Lead ECG from a patient who had broad QRS tachycardia suggestive of polymorphic VT. White arrow denotes the point where DC cardioversion was given and VT was subsided

Effect of cardiac sympathetic denervation

All patients initially underwent left SCD. If ES persisted beyond 24 hours after LSCD, right CSD was performed. Three of the eleven patients underwent bilateral CSD. The clinical response after LCSD in the 48 hours after the procedure is summarized in Table 2. In patients who underwent bilateral CSD, the events post CSD refer to the events after right CSD was performed (i.e. after bilateral CSD was completed). Five patients (46%) did not have any more episodes of ventricular arrhythmias (among these one patient succumbed to sepsis and acute kidney injury). Three patients (27.3%) had isolated episodes of VAs even after the procedure. One (11.1%) patient had sudden cardiac death, presumably due to ventricular arrhythmia, whereas two patients died of sepsis with acute kidney injury. Eight (72.7%) patients were discharged from the hospital, after successful control of ES. Implantation of ICD was strongly recommended. One patient underwent ICD implantation prior to discharge. One patient, who previously had a CRT-P device, underwent an upgrade to a combo device (CRT plus defibrillator; CRT-D)

Table 1: Baseline characteristics of patients undergoing CSD

Case No.	Age/ gender	Disease	EF (%)	Type of VT	Episodes prior to CSD	IV Drugs/Interventions	Acute Outcome
1	65/M	Ischemic; AWMI; PCI LMCA to LAD	30	MMVT	12	Amio; Ligno; $MgSO_4$	Discharged
2	61/F	Ischemic; AWMI; PCI TO LAD	36	MMVT	10	Amio; Ligno; Fosph	Discharged
3	57/F	Non Ischemic; DCM; Post CRT-P Implantation	43	MMVT	8	Amio; Ligno; $MgSO_4$; CRT-P upgraded to CRT-D	Discharged
4	56/M	Ischemic; DVCAD; Post PCI to LAD and RCA	26	PMVT	30	Amio; Ligno; $MgSO_4$, ICD	Discharged
5	63/M	Ischemic; Post PCI to RCA	33	MMVT	16	Amio; Ligno $MgSO_4$	Discharged
6	69/F	Ischemic; AWMI; Post PCI to LAD	32	MMVT	22	Amio; Ligno; $MgSO_4$; Fosph; ICD	Discharged
7	70/F	Ischemic; Post CABG, Grafts - LIMA to LAD, SVG to Ramus and RCA	34	MMVT	40	Amio; Ligno; $MgSO_4$ Fosph, ICD	Discharged
8	76/M	Ischemic; IWMI, PCI to RCA	32	PMVT	8	Amio; Ligno $MgSO_4$	Died
9	60/F	Ischemic; PWMI, PCI to LCx	35	MMVT	40	Amio; Ligno $MgSO_4$	Died
10	38/F	Non-ischemic	22	PMVT	7	Amio; Ligno $MgSO_4$	Died
11	75/F	Ischemic; AWMI, PCI to LAD and LCx	50	PMVT	14	Amio; Ligno $MgSO_4$	Discharged

EF, ejection fraction; VT, ventricular tachycardia; CSD, cardiac sympathetic denervation; IV, intravenous; PCI; percutaneous coronary intervention; CABG, coronary artery bypass grafting; DVCAD, double vessel coronary artery disease; LMCA, left main coronary artery; LAD, left anterior descending artery; RCA, right coronary artery; LIMA, left internal mammary artery; SVG, saphenous vein graft; AWMI, anterior wall myocardial infarction; IWMI, inferior wall myocardial infarction; PWMI, posterior wall myocardial infarction; CRT-P, cardiac resynchronization therapy-pacemaker; CRT-D, cardiac resynchronization therapy-defibrillator; MMVT, monomorphic ventricular tachycardia; PMVT, polymorphic ventricular tachycardia; $MgSO_4$, magnesium sulphate; Amio, Amiodarone; Ligno, lignocaine; Fosph, fosphenytoin

Table 2: Summary of procedure details, outcome, and follow up

Case No.	Type of CSD	Episodes prior to CSD*	Episodes in 48 h post CSD* (% reduction)	Acute Outcome	Follow Up (months)	Events during follow up
1	Left	12	0 (100%)	Discharged	8	No events during follow up
2	Left	10	4 (60%)	Discharged	Lost to follow up	Lost to follow up
3	Left	8	0 (100%)	Discharged	24	NSVT on ICD interrogation
4	Left	30	0 (100%)	Discharged	2	Died at 2 month of heart failure
5	Left	16	0 (100%)	Discharged	1	Died at 1 month of sudden cardiac death
6	Bilateral	22	8 (63.6%)	Discharged	3	NSVT on ICD interrogation
7	Bilateral	40	4 (90%)	Discharged	1	No events at follow up
8	Left	8	2 (75%)	Died (Sepsis)	-	-
9	Bilateral	40	0 (100%)	Died (Sepsis)	-	-
10	Left	7	2 (71%)	Died (Sudden cardiac death)	-	-
11	Left	14	30	Discharged	1	No events at follow up

*CSD, cardiac sympathetic denervation; VT, ventricular tachycardia; NSVT, Non-sustained ventricular tachycardia; ICD, implantable cardioverter defibrillator. *Refers to episodes of VT mandating electrical cardioversion*

prior to discharge. All the patients were continued on the same antiarrhythmic drugs. Amiodarone was used at a dosage of 200 mg/day. During a median follow-up of eight months (IQR 7–18), one patient died of heart failure (patient no. 3) and one had sudden cardiac death (patient no. 4). Apart from these, there was sustained reduction in VA burden in the rest. Two patients had occasional non-sustained VT episodes diagnosed by ICD interrogation. Patient No. 2 was lost to follow-up. Two patients, one in LCSD and one in bilateral CSD group, developed partial ptosis and both recovered from it at follow-up. There were no chronic complications related to the procedure.

Discussion

This case series was a retrospective time domain analysis of eleven patients who were managed with pharmacological left or bilateral CSD and to see their acute outcomes mainly in the setting of acute MI. The results of our study affirm that pharmacological left or bilateral CSD is a beneficial non-surgical method of neuraxial modulation for acute control of ES. In our study, majority of the patients had significant coronary artery disease (either acute coronary syndromes or sequelae to prior MI). Also, in our study, ultrasonography-guided pharmacological CSD was the only method of neuraxial modulation performed.

Development of Horner's syndrome and anhidrosis are important indicators of successful left or bilateral CSD. However, anhidrosis is a better indicator than the development of Horner's syndrome in this regard as the cardiac sympathetic fibers originate from the lower half of the stellate ganglion and other upper thoracic sympathetic ganglia, whereas ocular sympathetic fibers arise from upper portion of the stellate ganglion.[13-15]

There are few studies in the past which studied the effect of cardiac sympathetic denervation in the acute control of ventricular arrhythmias. However, most of these studies have used minimal invasive or surgical mode of neuraxial modulation. The role of ultrasonography-guided CSD has been limited only to few case reports and case series. Also, the majority of the patients in these studies are those without structural heart disease and non-ischemic cardiomyopathy. Our study will reinforce the role of left or bilateral CSD in the management of ES in structural heart disease, particularly in the setting of coronary artery disease, which is an evolving practice. This is very pertinent for centers where onsite radiofrequency ablation is not regularly available.

Interaction between substrate (diseased myocardium, channelopathies, cardiomyopathies), a variety of triggers (ischemia, electrolyte imbalance, and drugs that prolong QT interval), and the autonomic (sympathetic) nervous system play a role in initiation and maintenance of an ES. Increased central sympathetic outflow results in enhanced automaticity and triggered activity in the heart. The physiological/maladaptive response to hemodynamic instability is associated with a hyperadrenergic state. In addition, the delivery of DC shocks to the patient (cardioversion) is physically and psychologically traumatizing to the patient and contributes to sympathetic stimulation. The rationale for neuraxial modulation in ES is the suppression of sympathetic activation of the heart.[10] Neuraxial modulation has been successfully achieved by various methods as documented in literature. Thoracic epidural anesthesia (TEA), spinal cord stimulation, surgical cardiac sympathetic denervation (CSD), VATS-guided CSD, and ultrasound-guided pharmacological CSD have all been reported previously. Neuraxial modulation has been found to be beneficial in ES occurring in the context of various etiologies (coronary artery disease,

cardiomyopathies, channelopathies, etc.[12,16-19] Though VATS and surgical CSD are definite methods and help in the long-term management of ES, ultrasonography-guided LCSD or BCSD and TEA are easier bedside manoeuvres that do not need specialized equipment. TEA has an immediate onset of action, minimal effect on hemodynamic parameters, and more beneficial than LCSD as it inhibits fibers that are proximal to both right and left stellate ganglia. However, few side effects warrant a mention which makes it less preferable compared to pharmacological CSD. TEA needs infusion catheter in epidural space and dose is difficult to titrate with chances of infection/bleeding in epidural space and need for lateral decubitus positioning in a vulnerable patient. Compared to TEA, LSCD has fewer side effects most of which are transient and reversible. Other methods like spinal cord stimulation and catheter renal denervation are not routinely used.

Ultrasonography-guided pharmacological CSD (either left or bilateral) is an ideal next step in the management of ES, if it persists despite parenteral drug therapy. Pharmacological CSD achieved in this manner is temporary as opposed to surgical or VATS-guided CSD.[12,16-19] However, it has several advantages over the other methods. It can be performed in the ICU (performed as a bedside procedure), requires minimal equipment (ultrasonography is easily available in the ICU), and has fewer procedural complications. The procedure can be performed by most trained anesthetists with/without the assistance of a radiologist. On the contrary, VATS-guided CSD requires cardiothoracic surgeons trained in the use of video-assisted thoracoscopy, in addition to a thoracoscopy suite (or operating room set-up), trained nursing staff, and a cardiac anesthetist. Hemodynamic instability which often accompanies ES further makes the case for a bedside procedure. The differences and merits of surgical and VATS-guided CSD have been enumerated and discussed in prior studies.[13]

LCSD has been shown to reduce the threshold for VF and ectopy. However, LCSD may be inefficacious due to a variety of reasons. Anatomic variability in the course of preganglionic sympathetic fibers may result in incomplete stellate ganglion blockade. Animal studies have also shown hypertrophy of contralateral stellate ganglion and extension of nerve sprouts to areas previously supplied by the resected stellate ganglion after unilateral CSD. Finally, animal models have shown remodeling of bilateral stellate ganglion, including increased synaptic vesicle density and neuronal hypertrophy after MI. Small case series or reports have shown varying results with upfront bilateral CSD.[11,20-27] However, upfront bilateral CSD has its own issues. It reduces cardiac contractility and may lead to hemodynamic instability by cancelling beneficial effect of left CSD. Also, it is moderately invasive, has high complication rates, and needs longer general anesthesia time in hemodynamically vulnerable patients.[17] Also, the role of continuous infusion of the drug versus single shot remains a question. No larger studies have been done with continuous infusion and only case reports/series are available. Continuous infusion via stellate ganglia catheter has its advantage of prolonged action as intermittent dosages may have short-lasting action. Also, repeated injections on the same or contralateral side may be avoided. It can also determine whether the patient can undergo a neuroablative procedure to the stellate block using absolute alcohol which causes necrosis of the ganglia. However, with all these advantages till now, no enough data on continuous infusion of the drug is available.[28,29]

The retrospective nature of the study and a relatively small sample size are important limitations of the study. There were no controls to compare the efficacy of CSD. Only two patients underwent ICD implantation after the CSD; however, no ICD shocks were documented at follow up. Since the same dose of anti-arrhythmic drugs was maintained after the procedure and no other confounders were present, it is likely that the reduction in arrhythmic episodes was due to CSD procedure. In view of the limited sample size, a comparison between left and bilateral CSD was not possible. A larger scale, prospective study will better characterize the role of ultrasonography-guided left or bilateral CSD in patients with ES.

Conclusion

Our study highlights the effectiveness of ultrasonography-guided pharmacological left or bilateral CSD for acute control of refractory ES. Larger, prospective studies are needed to better define the role of left or bilateral CSD and the merits of one over the other. However, neuraxial modulation performed in this manner remains an easily performed rescue intervention to help tide over the acute crisis that is electrical storm. It is especially ideal for centers that are not equipped for procedures like surgical or VATS-guided CSD. Furthermore, our study will reinforce the role of left or bilateral CSD in the management of ES in structural heart disease, particularly in the setting of coronary artery disease which is an evolving practice.

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Conflicts of interest
There are no conflicts of interest.

References

1. Kowey PR, Levine JH, Herre JM, Pacifico A, Lindsay BD, Plumb VJ, *et al*. Randomized, double-blind comparison of intravenous

amiodarone and bretylium in the treatment of patients with recurrent, hemodynamically destabilizing ventricular tachycardia or fibrillation. The Intravenous Amiodarone Multicenter Investigators Group. Circulation 1995;92:3255–63.
2. Greene M, Newman D, Geist M, Paquette M, Heng D, Dorian P. Is electrical storm in ICD patients the sign of a dying heart? Outcome of patients with clusters of ventricular tachyarrhythmias. Europace 2000;2:263–9.
3. Hohnloser Stefan H, Al-Khalidi Hussein R, Pratt Craig M, Brum Jose M, Tatla Daljit S, Tchou Patrick, *et al*. Electrical storm in patients with an implantable defibrillator: Incidence, features, and preventive therapy: Insights from a randomized trial. Eur. Heart J 2006;27:3027–32.
4. Aliot EM, Stevenson WG, Almendral-Garrote JM, Bogun F, Calkins CH, Delacretaz E, *et al*. EHRA/HRS Expert Consensus on Catheter Ablation of Ventricular Arrhythmias: Developed in a partnership with the European Heart Rhythm Association (EHRA), a Registered Branch of the European Society of Cardiology (ESC), and the Heart Rhythm Society (HRS); in collaboration with the American College of Cardiology (ACC) and the American Heart Association (AHA). Heart Rhythm 2009;6:886–933.
5. Exner DV, Pinski SL, Wyse DG, Renfroe EG, Follmann D, Gold M, *et al*. Electrical storm presages nonsudden death: The antiarrhythmics versus implantable defibrillators (AVID) trial. Circulation 2001 24;103:2066–71.
6. Nademanee K, Taylor R, Bailey WE, Rieders DE, Kosar EM. Treating electrical storm: Sympathetic blockade versus advanced cardiac life support-guided therapy. Circulation 2000;102:742–7.
7. Bänsch D, Böcker D, Brunn J, Weber M, Breithardt G, Block M. Clusters of ventricular tachycardias signify impaired survival in patients with idiopathic dilated cardiomyopathy and implantable cardioverter defibrillators. J Am Coll Cardiol 2000;36:566–73.
8. Moss AJ, Zareba W, Hall WJ, Klein H, Wilber DJ, Cannom DS, *et al*. Prophylactic implantation of a defibrillator in patients with myocardial infarction and reduced ejection fraction. N Engl J Med 2002;346:877–83.
9. 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;4:1395–402.
10. Tung R, Shivkumar K. Neuraxial modulation for treatment of VT storm. J Biomed Res 2015;29:56-60.
11. Ajijola OA, Vaseghi M, Mahajan A, Shivkumar K. Bilateral cardiac sympathetic denervation: Why, who and when?. Expert Rev Cardiovasc Ther 2012;10:947-9.
12. Prabhu MA, Prasad SBV, Abhilash SP, Thajudeen A, K R B, Namboodiri N. Left sympathetic cardiac denervation in managing electrical storm: Acute outcome and long term follow up. J Interv Card Electrophysiol 2016;47:285-92.
13. Schwartz PJ. Efficacy of left cardiac sympathetic denervation has an unforeseen side effect: Medicolegal complications. Heart Rhythm 2010;7:1330–2.
14. Tan AY, Abdi S, Buxton AE, Anter E. Percutaneous stellate ganglia block for acute control of refractory ventricular tachycardia case report. Heart Rhythm 2012;9:2063–7.
15. Patel RA, Priore DL, Szeto WY, Slevin KA. Case report left stellate ganglion blockade for the management of drug-resistant electrical storm. Pain Med 2011;12:1196–8.
16. Bourke T, Vaseghi M, Michowitz Y, Sankhla V, Shah M, Swapna N, *et al*. Neuraxial modulation for refractory ventricular arrhythmias: Value of thoracic epidural anesthesia and surgical left cardiac sympathetic denervation. Circulation 2010;121:2255–62.
17. Vaseghi M, Gima J, Kanaan C, Ajijola OA, Marmureanu A Mahajan A, *et al*. Cardiac sympathetic denervation in patients with refractory ventricular arrhythmias or electrical storm: Intermediate and long-term follow-up. Heart Rhythm 2014;11:360–6.
18. Collura CA, Johnson JN, Moir C, Ackerman MJ. Left sympathetic denervation for the treatment of long QT syndrome and catecholaminergic polymorphic ventricular tachycardia using video-assisted thoracic surgery. Heart Rhythm 2009;6:752–9.
19. Ajijola OA, Lellouche N, Bourke T, Tung R, Ahn S, Mahajan M, *et al*. Bilateral cardiac sympathetic denervation for the management of electrical storm. J Am Coll Cardiol 2012;59:91–2.
20. Kralios FA, Martin L, Burgess MJ, Millar K. Local ventricular repolarization changes due to sympathetic nerve-branch stimulation. Am J Physiol 1975;228:1621–6.
21. Vaseghi M, Zhou W, Shi J, Ajijola OA, Hadaya J, Shivkumar K, *et al*. Sympathetic innervation of the anterior left ventricular wall by the right and left stellate ganglia. Heart Rhythm 2012;9:1303–9.
22. Yanowitz F, Preston JB, Abildskov JA. Functional distribution of right and left stellate innervation to the ventricles: Production of neurogenic electrocardiographic changes by unilateral alteration of sympathetic tone. Circ Res 1966;18:416–28.
23. Randall WC, Armour JA, Geis WP, Lippincott DB. Regional cardiac distribution of the sympathetic nerves. Fed Proc 1972;31:1199–208.
24. Wood A, Docimo S, Elkowitz DE. Cardiovascular disease and its association with histological changes of the left stellate ganglion. Clin Med Insights Pathol 2010;3:19–24.
25. Ajijola OA, Wico J, Lambert HW, Mahajan A, Stark E, Fishbein MC, *et al*. Chronic myocardial infarction is associated with neural remodeling in human stellate ganglia. Heart Rhythm 2012;9:261.
26. Han S, Kobayashi K, Joung B, Piccirillo G, Maruyama M, Vinters HV, *et al*. Electroanatomic remodeling of the left stellate ganglion after myocardial infarction. J Am Coll Cardiol 2012;59:954–61.
27. Fioretto ET, Rahal SC, Borges AS, Mayhew TM, Nyengaard JR, Marcondes JS, *et al*. Hypertrophy and neuron loss: Structural changes in sheep SCG induced by unilateral sympathectomy. Int J Dev Neurosci 2011;29:475–81.
28. Hulata DF, Le-Wendling L, Boezaart AP, Hurley RW. Stellate ganglion local anesthetic blockade and neurolysis for the treatment of refractory ventricular fibrillation. A A Case Rep 2015;4:49-51.
29. Garcia-Moran E, Sliwinski-Herrera F, Cortes-Villar C, Sandín-Fuentes M, Pastor Báez G, San Román A. Refractory electrical storm: A role for transient sympathetic blockade. Rev Esp Cardiol (Engl Ed) 2016;69:76-8.