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Emergent Phenol Injection of Bilateral Stellate Ganglion for Management of Refractory Malignant Ventricular Arrhythmias

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Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
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Case series
Patients: Male, 51-year-old • Male, 59-year-old
Final Diagnosis: Ventricular tachycardia (VT)
Symptoms: Cardiac arrest
Medication: —
Clinical Procedure: Ablation
Specialty: Critical Care Medicine

Objective: Unusual or unexpected effect of treatment

Background: Management of incessant electrical storm is poorly defined. These 2 case studies demonstrate a simplified percutaneous approach to achieve stellate ganglion ablation (SGA) and to promptly control malignant ventricular arrhythmias.

Case Reports: This report describes 2 patients with deteriorating hemodynamics, progressive ventricular arrhythmias, and worsening heart failure, managed with emergent percutaneous fluoroscopically-guided bilateral SGA to achieve bilateral cardiac sympathetic denervation. While supine and intubated, the left and then right stellate ganglion were identified guided by anatomic landmarks. Using a 22-guage, 3.5-inch spinal needle, contrast dye was injected with appropriate outline of the stellate ganglion at the uncinate process of the C6 vertebra. Bupivacaine 0.5% was injected, followed by phenol 6%. Successful SGA was confirmed by intentional Horner's syndrome with bilateral eye lag. The procedures were completed in about 30 min without complications and there was a dramatic reduction in ventricular arrhythmias.

Conclusions: Emergent percutaneous bilateral SGA can be accomplished with a brief procedure resulting in management of electrical storm.

MeSH Keywords: Stellate Ganglion • Sympathectomy, Chemical • Tachycardia, Ventricular

Abbreviations: **ECMO** – extracorporal mechanical oxygenation; **ICD** – implantable cardiac defibrillator; **SGA** – stellate ganglion ablation; **VT/VF** – ventricular tachycardia/fibrillation

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Background

The primary mechanism of death in the United State is sudden cardiac death, often due to malignant ventricular arrhythmias secondary to ischemic heart disease. Conventional management includes implantable cardiac defibrillator (ICD) therapy coupled with correction of all contributing features and management of cardiac disease. If ventricular tachycardia/fibrillation (VT/VF) episodes are frequent, then further therapy consists of antiarrhythmic drug and catheter ablation. Recurrent shocks from ICDs have been shown to increase morbidity and mortality in patients with refractory VT/VF [1,2]. "Electrical storm" is defined as 3 or more anti-tachycardia pacing or ICD shock therapy delivered within a 24-h period [3]. Recent publications discuss the use of cardiac sympathetic denervation to manage electrical storm, but management of ongoing electrical storm that is nearly incessant is not well defined [4–10]. This report describes 2 patient experiences utilizing emergent percutaneous stellate ganglion ablation (SGA) to achieve cardiac sympathetic denervation for acute management of recalcitrant electrical storm.

Case Reports

First case report

The patient was a 51-year-old man with an ischemic cardiomyopathy, severe mitral regurgitation, atrial fibrillation, ICD, and a left ventricular ejection fraction of 25%. He presented with unstable angina and underwent elective coronary artery bypass graft surgery and mitral valve repair. His post-operative course was complicated by 7 episodes of VF occurring within 5 h and each successfully managed with a single ICD shock therapy. It is important to note that the VF episodes were not preceded by monomorphic VT nor triggered by a recurrent premature ventricular ectopic focus. His hemodynamic status deteriorated and he was placed on extracorporal mechanical oxygenation (ECMO) with systemic anticoagulation. The intended goal was to proceed with an empiric scar-based catheter ablation, but the ventricular arrhythmias could not be quieted. The patient continued with malignant ventricular arrhythmias (41 episodes treated with 24 ICD shocks and 17 pace-termination) despite intravenous amiodarone and lidocaine and correction of all electrolyte abnormalities. Left-heart catheterization showed patency of the bypass graft. With deteriorating hemodynamics, progressive ventricular arrhythmias, worsening heart failure, and all conventional therapies for malignant ventricular arrhythmias being unsuccessful, the patient underwent emergent percutaneous fluoroscopically-guided bilateral SGA. While supine, intubated, and with ECMO support, the left and then right stellate ganglion were identified guided by anatomic landmarks. Utilizing a 22-guage, 3.5-inch spinal needle, contrast dye was injected with appropriate outline of the stellate ganglion at the uncinate process of the C6 vertebra. Subsequently, a total of 8 ml of bupivacaine 0.5% was injected with aspiration every 2 ml, followed by 10 ml of phenol 6% (a total of 16 ml of bupivacaine and 20 ml of phenol was utilized bilaterally). Successful SGA was confirmed by intentional Horner's syndrome with bilateral eye lag. The procedure was completed in 28 min, with a fluoroscopy time of 7.8 min. During the procedure, the patient received 2 ICD therapies.

The initial response to bupivacaine/phenol injection was an abrupt increase in ventricular ectopy and continued, albeit less frequent, ICD shock therapy (9 in 16 h); however, following this burst of VT/VF, there were no further ventricular arrhythmias for the subsequent 13 days. Intravenous antiarrhythmic medications and ECMO support were discontinued at about 72 h after SGA. The patient initially improved, but then subsequently died due to sepsis.

Second case report

The second patient was a 59-year-old man with an ischemic cardiomyopathy and left ventricular ejection fraction of 20%. Nine days after percutaneous endocardial ablation for recurrent ICD therapies for rapid VT (cycle length 250–280 ms), he presented with dyspnea, volume overload, and biventricular congestion. Within 12 h of admission, he experienced recurrent ventricular flutter with loss of consciousness, managed with 9 ICD shock therapies. He was intubated, oral antiarrhythmic medication was discontinued, and intravenous amiodarone was started. Left-heart catheterization demonstrated elevated left ventricular pressures and no stenosis amenable to intervention. Despite management of acute congestion, ventricular ectopy continued with long runs of rapid nonsustained VT, and then a subsequent day of rapid ventricular flutter and multiple ICD shocks (12 within 4 h) with subsequent cardiogenic shock. Emergently, the patient underwent bilateral SGA as described above in Case #1, but using a larger volume of 0.5% bupivacaine (10 ml), and 10 ml of 6% phenol on each side. The procedure was completed in 34 min with 9.2 min of fluoroscopy. Immediately following SGA, bilateral eyelid drooping consistent with Horner's syndrome was noted. Following SGA, the patient did not undergo any further ICD therapies and eventually was managed with total heart replacement.

Discussion

The intent of this 2-patient case report is to describe an easily implemented methodology for immediate and permanent SGA in patients with malignant and incessant ventricular arrhythmias. In these case reports, each patient had a thorough investigation for an etiology of the ventricular arrhythmias,

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and attempts were made to reverse any contributing factors. Patients received intensive care therapy, intravenous antiarrhythmic medications managed by electrophysiologists, left-heart catheterization, catheter ablation when feasible, and optimization of volume status and hemodynamics, yet the VT/VF continued. Prior to SGA, there were 69 ICD therapies, and after SGA there were only 9 and these 9 were likely due to underdosing of bupivacaine/phenol, which were increased with the second patient. This immediate alteration in burden of malignant ventricular arrhythmias following SGA indicates that the SGA was the successful intervention. For patients in whom conventional therapy for ventricular arrhythmias fail, bilateral SGA offers an incremental method of managing electrical storm. The risks of the procedure (vascular uptake, high epidural blockade, pneumothorax, recurrent laryngeal nerve block, hematoma, and transmyelitis) are about 1–2%, and are certainly outweighed by the potential benefits of life-saving therapy.

Prior studies have documented the benefit of modulating the autonomic nervous system with SGA to achieve cardiac sympathetic denervation for managing electrical storm after conventional therapies have failed [5–10]. SGA has been completed with either thoracic epidural anesthesia or video-assisted thorascopic surgical resection of the sympathetic chain [5,8]. However, these techniques would not have been able to be implemented for the 2 patients in this report. The ventricular arrhythmias were incessant with subsequent decline in hemodynamics and worsening heart failure; therefore, these patients were too unstable for surgical resection. Also, although thoracic epidural anesthesia may have been accomplished, achieving a lateral decubitus position and successful epidural access with systemic anticoagulation and while on ECMO would be quite challenging, and thoracic epidural anesthesia has been used only as temporary control of ventricular arrhythmias. Furthermore, less than 50% of patients respond to epidural anesthesia.

Chemical ablation for cervical denervation was selected since it is easily implemented, with the patient supine and requiring only fluoroscopic guidance, and the desired endpoint can be confirmed by immediate sagging of the eyelid. The procedure was completed quickly and without complication other than the intentional development of Horner's syndrome. The first patient experience of seemingly exacerbation of the ventricular arrhythmias with subsequent resolution resulted in increasing the dose of phenol for the second patient, in which there were no exacerbation of arrhythmias. Bilateral SGA was completed since the results with bilateral SGA have been reported to exceed left-only SGA [10,16].

Cardiac sympathetic denervation

There are extensive bidirectional afferent and efferent signals and feedback continuously transmitted between the heart and brain [6]. In the presence of a cardiomyopathy, there is excess sympathetic output, with an associated increase in ventricular arrhythmias. Furthermore, particularly in ischemic cardiomyopathy, following initial myocardial and cardiac nerve injury, the response is axonal regeneration with nerve sprouting [12,13]. This neural remodeling results in regional heterogeneity of autonomic innervation which, coupled with increased sympathetic output, results in increased risk of malignant ventricular arrhythmias. Similar adverse changes have also been reported in stellate ganglion with hypertrophy, inflammation, and oxidative stress [17]. Cardiac sympathetic denervation interrupts, at the pre-ganglia level, the release of neurotransmitters and thus reduces cardiac adrenergic tone, which has been used to manage ventricular arrhythmias in the setting of inherited diseases (e.g., long QT syndrome) as well as structural heart disease.

Prior studies

Numerous publications have reported the benefit of cardiac sympathetic denervation using surgical techniques [4–7,10,14], but this approach is intended for patients not actively experiencing unstable ventricular arrhythmias. For patients who are unable to tolerate surgical denervation, prior studies have reported the use of percutaneous local anesthesia injection for temporary sympathetic blockade to stop the electrical storm [15,16,18,19]. Similar to the current case reports, bupivacaine was used for an immediate suppression of stellate ganglion function; however, unlike prior reports, the present case studies demonstrate that permanent SGA can be achieved with the use of phenol.

Conclusions

The case reports presented in this article suggest that in patients with refractory malignant ventricular arrhythmias, bilateral chemical SGA can be considered using bupivacaine with phenol. It is important be aware that SGA should be introduced only after exhausting routine interventions and after a thorough investigation for reversible causes of the ventricular arrhythmias, including catheter ablation, antiarrhythmic drug therapy, ECMO, optimization of medical therapy, exclusion/management of ischemia, and management of any acute heart failure. Certainly, SGA is aggressive considering the risk of complications (e.g., vascular uptake, high epidural blockade, pneumothorax, recurrent laryngeal nerve block, transmyelitis, and hematoma); however, for the rare patient in which there are no alternative therapies and the clinical course is dire, this readily implemented technique may halt the ventricular storm and provide a window for recovery.

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