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Stellate Ganglion Block as Rescue Neuromodulation after Prior Bilateral Cardiac Sympathetic Denervation in Malignant LQTS5 with Hormonally Triggered Venticular Fibrillation Storm

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Key message / Take-home message

Although surgical cardiac sympathetic denervation (CSD) aims to achieve durable interruption of sympathetic input to the heart by resecting the lower half of the stellate ganglion and upper thoracic sympathetic chain, complete and permanent denervation is not always achieved in clinical practice. Stellate ganglion block should not be considered redundant in patients with prior cardiac sympathetic denervation and may represent an effective rescue therapy in refractory electrical storm.

Keywords
Arrhythmic storm, Ventricular arrhythmias, Stellate ganglion block, Cardiac sympathetic denervation, Long QT syndrome.

Introduction
Electrical storm (ES), typically defined as ≥3 episodes of sustained ventricular arrhythmias (VT/VF) or appropriate ICD therapies within 24 hours, represents a medical emergency characterized by profound electrical instability, recurrent hospitalization, and substantial high mortality in short-term [1,2]. In congenital long QT syndrome (LQTS), ES most often manifests as recurrent torsades de pointes (TdP) and/or degenerating VF, reflecting the convergence of an intrinsically reduced repolarization reserve with potent and often modifiable triggers, including sympathetic surges, QT-prolonging medications, electrolyte abnormalities, fever/inflammation [3] and abrupt endocrine changes [4-8]. Although KCNE1 (LQT5) is a relatively uncommon genotype with variable penetrance, mechanistic evidence supports a plausible link between sex hormones and IKs-mediated repolarization; notably, LQTS-associated KCNE1 variants can modify the electrophysiologic response to 17β-estradiol [9].

Acute management of ES in LQTS prioritizes rapid stabilization and systematic identification of reversible triggers, with immediate withdrawal of QT-prolonging agents, aggressive correction of hypokalemia/hypomagnesemia, temperature control, and suppression of adrenergic activation through adequate analgesia and sedation [1,10]. Non-selective β-blockade remains foundational therapy, and ICD protection is indicated in survivors of cardiac arrest; however, ICD shocks do not prevent arrhythmia initiation and may themselves amplify sympathetic tone, thereby perpetuating ES [11,12]. These considerations have elevated autonomic modulation from a salvage maneuver to a key component of contemporary ES pathways, particularly when arrhythmias remain refractory despite optimized pharmacological therapy and supportive care [10].

Within this framework, percutaneous stellate ganglion block (SGB) has emerged as a pragmatic, rapidly deployable form of temporary cardiac sympathetic attenuation [12,13]. ES often

evolves into a self-reinforcing cycle—arrhythmia recurrence and shocks heighten pain and anxiety, driving catecholamine release, increasing dispersion of repolarization and triggered activity, and thereby promoting further VT/VF [11,12]. By transiently interrupting cervicothoracic sympathetic efferent traffic (most commonly left-sided SGB, given the dominant influence of the left stellate pathway on ventricular electrophysiology), SGB can function as an autonomic "circuit breaker", facilitating termination of arrhythmic clustering and providing stabilization when standard measures fail or when definitive therapies are not immediately available [12-14]. Clinically, SGB is used for refractory ES, frequently as a bridge to catheter ablation or surgical denervation, and sometimes as a temporizing strategy when transfer or prolonged procedures are unsafe [13-16]. Observational data are concordant: in a Circulation: Arrhythmia and Electrophysiology cohort, SGB attenuated ES in more than half of treated patients without procedure-related complications [12]. More recently, the prospective multicenter STAR experience reported a ≥50% reduction in treated arrhythmic events in a substantial proportion of patients, reinforcing SGB as an effective temporizing intervention in modern ES algorithms [16].

For sustained autonomic control in high-risk congenital LQTS, surgical cardiac sympathetic denervation remains the most durable strategy [1,17]. International consensus guidance recommends left cardiac sympathetic denervation (LCSD) for high-risk LQTS patients in whom β-blockers are ineffective or not tolerated, and/or when ICD therapy is contraindicated or refused [1,10,17]. Long-term experience demonstrates substantial overall event reduction after LCSD, yet also documents clinically relevant breakthrough arrhythmias in the most malignant phenotypes, with up to 50% of high-risk patients experiencing recurrent events during long-term follow-up [18]. Observational studies further confirm that cardiac sympathetic denervation reduces arrhythmic burden and ICD shocks but does not eliminate recurrence risk, with a subset of patients requiring additional therapies during follow-up [19-21]. Bilateral cardiac sympathetic denervation (BiCSD/BCSD) is generally reserved as an escalation strategy for patients with ongoing arrhythmic events after LCSD despite optimized therapy; in the largest LQTS series, BiCSD was uncommon (<5%) and associated with variable long-term efficacy [22].

Clinical Case

A 42-year-old woman was admitted to the Cardiac Intensive Care Unit (CICU) because of recurrent ventricular arrhythmias detected by her implantable cardioverter-defibrillator (ICD), consistent with a new episode of electrical storm. The patient had a long-standing history of malignant ventricular arrhythmias occurring in the absence of overt structural heart disease. In 2015, genetic testing identified a pathogenic KCNE1 variant, consistent with type 5 long QT syndrome (LQTS5). A sodium-channel blocker challenge (flecainide test) was previously performed and was negative, making concealed Brugada syndrome unlikely. Coronary angiography excluded obstructive coronary artery disease. Transthoracic echocardiography over time showed no clear structural cardiomyopathy, with only mild global left ventricular systolic dysfunction (LVEF approximately 50% in prior assessments). Because of recurrent episodes of polymorphic ventricular tachycardia (VT) and ventricular fibrillation (VF), the patient received a dual-chamber ICD (Medtronic) for secondary prevention. She underwent multiple catheter ablation procedures for ventricular arrhythmias/VF, and, given the refractory nature of the storm, she also underwent bilateral cardiac sympathetic denervation (bilateral sympathectomy) at a tertiary referral center (IRCCS Policlinico San Matteo, Pavia), with documented histological and immunohistochemical evidence of sympathetic ganglia in the excised tissue and subsequent marked reduction in arrhythmic burden. A relevant clinical observation in her prior course was a significant improvement after the introduction of combined estrogen–progestin therapy, with a reduction in arrhythmic events. The patient had cardiovascular risk factor history notable for prior smoking habit. Comorbidities included celiac disease and hypothyroidism. In July 2024, she was hospitalized for an electrical storm temporally associated with discontinuation of estrogen–progestin therapy. In late December 2024, she developed a systemic illness characterized by fever, abdominal pain, and bilateral latero-cervical lymphadenopathy. She initially received amoxicillin–clavulanate (Augmentin®) prescribed by her general practitioner without clinical benefit, followed by cefixime (reported as "Unixin®/cefixime" in the record) which was subsequently discontinued due to QT prolongation. On January 4th, 2025, because of persistent symptoms, she presented again to the emergency department and was admitted to an Infectious Diseases unit. Blood cultures/infectious work-up were reported as negative, and she was empirically treated with intravenous vancomycin and piperacillin–tazobactam. During this hospitalization she developed a severe arrhythmic relapse with electrical storm and multiple ICD shocks, prompting transfer to a Cardiology ward. Because of ongoing recurrent episodes, she required sedation with dexmedetomidine (Dexdor®). In parallel, the ICD was reprogrammed to atrial overdrive pacing (AAI mode at 90 bpm), achieving only discrete suppression of the arrhythmic burden. She was then transferred on January 11th, 2025 to a specialized cardiology/arrhythmia unit for further management. Device interrogation documented multiple ventricular arrhythmia episodes in the VF zone treated with repeated ICD shocks during the interval January 9th–11th, 2025. A psychological consultation was performed on January 14th, 2025. Subsequent telemetry reportedly showed no further sustained complex ventricular arrhythmias. Hemodynamic evaluation included right heart catheterization, which demonstrated normal right-sided pressures and normal pulmonary vascular resistance.

Current admission (CICU) and baseline therapy

At the present admission, the patient was hospitalized again for recurrence of rapid ventricular arrhythmias documented at ICD interrogation. A key temporal association preceding the current storm was a modification of hormonal therapy: she switched from combined estrogen–progestin treatment to a progestin-only regimen (desogestrel). Shortly after this change she developed QT prolongation and re-emergence of ventricular ectopy followed by recurrent episodes consistent with electrical storm. Given the

close chronology, estrogen withdrawal and related autonomic–electrophysiologic imbalance were considered a likely trigger of the relapse. At the time of admission, her chronic home medications were nadolol 80 mg twice daily, hydroquinidine 150 mg four times daily, progesterone 200 mg once daily, levothyroxine 50 µg once daily, and rosuvastatin/ezetimibe 10/20 mg once daily.

In-hospital course and rescue intervention

During the first 24 hours of hospitalization, the patient experienced persistent ventricular electrical instability despite aggressive optimization of medical therapy and deep sedation. Antiarrhythmic treatment included continuous lidocaine infusion, oral nadolol, intravenous potassium and magnesium supplementation, and anxiolytic–sedative therapy with oral lorazepam and intravenous midazolam. Deep sedation was maintained with intravenous dexmedetomidine. Despite this comprehensive pharmacological strategy and adequate sedation, recurrent ventricular ectopy and non-sustained ventricular tachycardia persisted, consistent with a refractory electrical storm. Given the failure of maximal medical therapy and the ongoing arrhythmic burden, a rescue autonomic modulation strategy was considered. Notably, this decision was made despite the patient's history of prior bilateral cardiac sympathetic denervation. On January 22nd, 2026, at 09:30 a.m., the patient underwent an ultrasound-guided percutaneous stellate ganglion block. A total dose of 200 mg of lidocaine combined with 50 mg of ropivacaine was administered. The successful sympathetic blockade was supported by clinically evident changes in the temperature and sweating of the ipsilateral upper extremity. Following the procedure, a dramatic and rapid suppression of ventricular arrhythmias was observed. Starting from 10:30 a.m. on the same day, continuous telemetry and ICD interrogation documented complete abolition of ventricular arrhythmic activity, with no further episodes of ventricular ectopy, non-sustained VT, sustained VT, or ICD therapies in the following hours. This immediate and sustained reduction in arrhythmic burden strongly suggested a causal therapeutic effect of the stellate ganglion block.

Discussion and pathophysiological proposal

Although surgical cardiac sympathetic denervation (CSD) aims to achieve durable interruption of sympathetic input to the heart by resecting the lower half of the stellate ganglion and upper thoracic sympathetic chain, complete and permanent denervation is not always achieved in clinical practice. Residual fibers, anatomical variability, and neural plasticity can allow adrenergic signals to persist or re-emerge, thereby maintaining arrhythmogenic potential despite prior surgery. In this context, percutaneous stellate ganglion block (SGB) has been described as an effective adjunctive or rescue therapy even after prior sympathectomy in patients with recurrent ventricular arrhythmias or electrical storm.

One of the earliest documented clinical reports illustrating this concept comes from a case of a 64-year-old man with refractory electrical storm who underwent both ultrasound-guided left stellate ganglion block and left cardiac sympathetic denervation with left stellate ganglionectomy. In this case, the SGB was instrumental in terminating the electrical storm and restoring sinus rhythm prior to definitive ganglionectomy, suggesting that even after surgical sympathetic intervention, acute percutaneous blockade can yield clinically meaningful arrhythmia suppression. This case is detailed by Asangmbeng et al. in Cureus, demonstrating rapid control of ventricular tachycardia/ventricular fibrillation with SGB followed by surgical intervention [23]. More recent procedural experiences strengthen this observation. Reports from electrophysiology centers describe patients with refractory ventricular arrhythmias treated with both stellate ganglion blockade and subsequent bilateral thoracoscopic sympathectomy, with SGB serving as a temporizing stabilizing intervention before or even after definitive surgical denervation. For example, a 2025 JACC Case Reports publication describes the use of SGB to bridge a patient to bilateral thoracoscopic sympathectomy for refractory arrhythmias, emphasizing that sympathetic blockade can still effectively reduce arrhythmia burden in the setting of complex autonomic modulation regardless of prior surgical history [24]. Although not always explicitly focused on post-sympathectomy patients, larger series and reviews on SGB in electrical storm show that transient pharmacologic sympathetic blockade frequently suppresses ventricular arrhythmias, even when prior autonomic interventions, such as CSD or ablation, have been undertaken. These observational findings support the concept that percutaneous SGB remains efficacious in modulating residual sympathetic activity and arrhythmogenic triggers in diverse clinical settings [25].

Mechanistically, several factors may explain why SGB continues to provide benefit after surgical sympathectomy, and this case report offers the opportunity to analyze them. First of all, hormonal influences on ventricular arrhythmogenesis have recently gained increasing attention, particularly in female patients with malignant ventricular arrhythmias occurring in the absence of overt structural heart disease [4-8]. In a seminal report by Romanazzi et al., continuous estrogen–progestin therapy was shown to act as a lifesaving intervention in a young woman with recurrent malignant ventricular fibrillation triggered by Purkinje ectopy, refractory to antiarrhythmic drugs, deep sedation, and repeated catheter ablations [26]. Experimental and clinical data suggest that estrogens reduce repolarization dispersion and stabilize potassium currents, while progesterone may exert complementary effects on repolarization reserve [4,5,27-29]. In our patient, long-term electrical stability was achieved during periods of combined estrogen–progestin therapy, whereas arrhythmic relapses consistently followed hormonal withdrawal or modification, closely mirroring prior observations [26].

Secondarily this case confirms the successful use of ultrasound-guided percutaneous stellate ganglion block (PSGB) as a rescue therapy for refractory electrical storm in a patient with congenital LQTS5 despite previous bilateral cardiac sympathetic denervation. The immediate suppression of ventricular arrhythmias strongly supports a causal therapeutic effect and highlights that acute autonomic modulation can remain effective even after surgical

denervation, likely due to residual, functional, or reconstituted sympathetic pathways [1-3,11-33]. Clinical series confirm that ventricular arrhythmia recurrence after cardiac sympathetic denervation is not rare. Although denervation modifies arrhythmic substrate and reduces ICD therapies, recurrent VT may occur due to persistent substrate, incomplete denervation, or neural remodeling [19-21].

Incomplete denervation is common; standard surgical CSD typically removes only portions of the cervicothoracic sympathetic chain, and variable anatomy may leave residual preganglionic or postganglionic fibers intact. Furthermore, the right stellate ganglion or contralateral pathways can contribute significantly to cardiac sympathetic tone, particularly when only left-sided surgery is performed. In the end, neural remodeling or reinnervation over time may restore some sympathetic influence to the myocardium, which can be acutely interrupted by SGB even after prior surgical denervation. These observations align with the broader understanding that cardiac sympathetic innervation is redundant and plastic, and that temporary chemical blockade can provide additional modulation beyond structural resection.

Sympathetic hyperactivity is a key driver of arrhythmia perpetuation in ES regardless of substrate [11,12]. In congenital LQTS, especially in IKs-related genotypes, adrenergic stimulation exacerbates action potential prolongation and promotes early afterdepolarizations [35]. For this reason, β-blockers and autonomic modulation represent cornerstone therapies [1,10]. When ES becomes refractory, neuromodulation strategies are endorsed by contemporary guidelines and consensus statements [1,10,36].

PSGB transiently suppresses sympathetic outflow from the cervicothoracic ganglion complex, reducing norepinephrine release and repolarization dispersion [12,13]. Multiple observational studies and multicenter experiences demonstrate dramatic reduction in arrhythmic burden with PSGB in refractory ES [13-16], particularly in autonomically mediated arrhythmias such as LQTS, CPVT, idiopathic VF, and Purkinje-triggered VF [12,16].

Cardiac sympathetic innervation is highly variable, and accessory pathways such as the nerve of Kuntz may bypass surgical denervation [31]. Moreover, functional reinnervation and neural plasticity may occur over time after BCSD [32,33]. Finally, extreme physiological stress may overwhelm baseline sympathetic attenuation achieved with BCSD [11,12] PSGB therefore provides additional acute neuromodulation at the moment of highest arrhythmic vulnerability.

Conclusions

The analysis of the original 2018 surgical report provides key anatomical insight into the efficacy of percutaneous stellate ganglion block (PSGB) in our patient. At that time, she underwent bilateral thoracic sympathectomy (T1–T4) via a thoracoscopic robot-assisted approach. However, this procedure does not eliminate the cervical component of the stellate ganglion (C8–T1), which remains a critical contributor to cardiac sympathetic outflow. The stellate ganglion may represent the principal convergence node between cervical and thoracic sympathetic fibers and plays a central role in adrenergically mediated ventricular arrhythmogenesis [4].

The dramatic response to ultrasound-guided PSGB observed in 2026 is therefore anatomically and physiologically plausible.

In summary, while cardiac sympathetic denervation remains a cornerstone therapy for refractory ventricular arrhythmias, percutaneous stellate ganglion block may still provide effective arrhythmia suppression in patients with recurrence after prior sympathectomy. This observation underscores the therapeutic value of multimodal autonomic modulation in advanced arrhythmia management and supports the role of PSGB as an adjunctive or rescue strategy in refractory electrical storm.

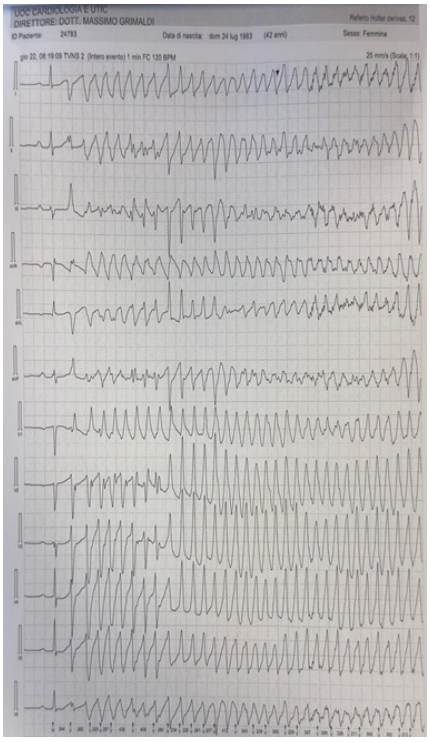
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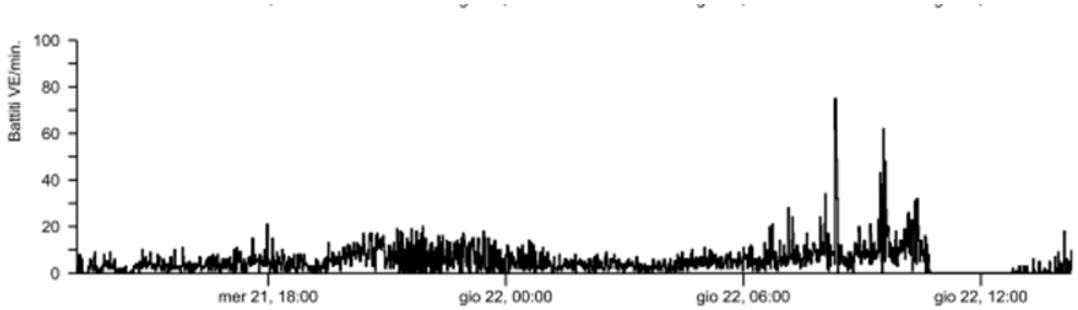
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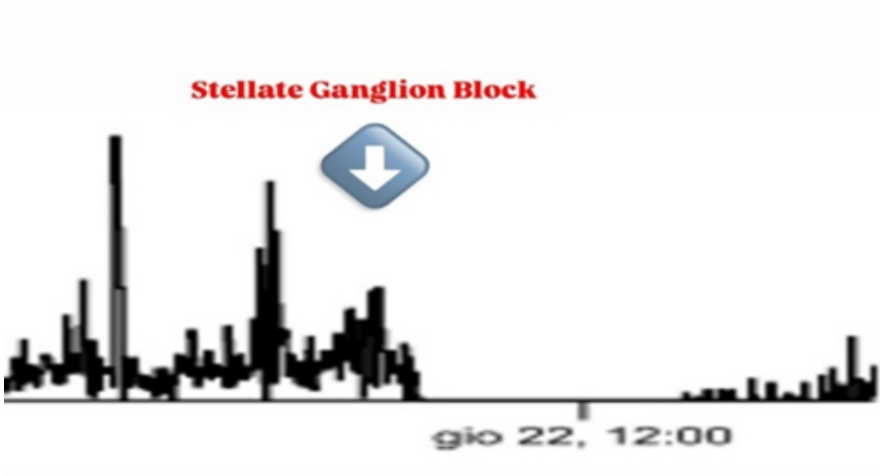
Attachments



Short coupled - PVC originating from the purkinje trigger a torsade de pointes (R-on-T phenomenon)



Ventricular arrhythmias burden reduction soon after PSGB.



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