Case Report

# Left Stellate Ganglion Block for Refractory Ventricular Tachycardia: A Case Series

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Ventricular arrhythmias are usually well controlled with medical management, cardiac implantable electronic devices, or catheter ablation. However, the refractory ventricular tachycardia or fibrillation (VT/VF) is life threatening and challenging. The authors reported a case series of left stellate ganglion blocks (LSGB) in patients with refractory VT/VF, who failed pharmacological treatment and multiple traditional cardiac interventions. Five patients underwent six LSGB. Four patients had significant decreased in ventricular arrhythmia burden. Among the responders, the LSGB suppressed significant VT/VF for three to seven days. Blocks did not only temporary suppress ventricular arrhythmia, but also stabilized the condition and served as a bridge to definitive treatment such as EP ablation or heart transplantation. There was no significant hemodynamic change or devastating side effects. The outcome from the present case series suggested that LSGB could be an effective treatment and a lifesaving intervention for intractable VT/VF.

***Keywords***: Stellate ganglion block, Refractory ventricular tachycardia, Sympathectomy

Received 5 April 2019 | Revised 13 June 2019 | Accepted 14 June 2019

**J Med Assoc Thai 2021;104(3):506-11**

**Website:** http://www.jmatonline.com

Refractory ventricular tachycardia/fibrillation (VT/VF) is caused or aggravated by sympathetic stimulation, which is mediated through stellate ganglion. Multiple case reports showed that the left stellate ganglion block (LSGB) could abort or alleviate ventricular arrhythmia from varied etiology. The authors presented a case series, showing the effect of LSGB in patients who suffered from ventricular arrhythmia and were intractable to medical management and electrophysiology cardiac intervention.

## Study design

After the IRB approval, the authors reviewed the medical records for the adult patients who have undergone LSGB for intractable ventricular tachycardia at the Oregon Health and Science University between 2012 and 2016. Five patients were identified (Table 1).

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**How to cite this article:**

Ziboonyahgoon N, Gonzalez-Sotomayor J, Atisook R. Left Stellate Ganglion Block for Refractory Ventricular Tachycardia: A Case Series. J Med Assoc Thai 2021;104:506-11.

doi.org/10.35755/jmedassocthai.2021.03.10010

## Nerve block technique

All patients underwent an ultrasound guided LSGB at bedside in the intensive cardiac care unit with standard ASA monitoring. The patient was placed in supine position with the head turn to the opposite side. The anterior and lateral part of the neck was prepped with chlorhexidine and a linear (10 to 12 mHz) ultrasound probe was applied anterolateral neck at cricoid cartilage level to identify transverse process of C6, anterior tubercle of C6 (Chassaignac's tubercle), longus colli muscle, and surrounding neurovascular structure. The needle was advanced with the in-plane guidance technique and aimed to deposit the local anesthetics medial to the Chassaignac's tubercle and anterior to prevertebral fascia of longus colli muscle (Figure 1) The type, amount of local anesthetic and adjuvant are described in Table 2.

## Case Report

### Patient 1

A 74-year-old male had a history of non-ischemic cardiomyopathy with an ejection fraction of 35%, sick sinus syndrome, and aortic stenosis s/p aortic valve replacement (AVR). The patient also had history of multiple episodes of ventricular tachycardia. He had an implantable Cardioverter Defibrillator (ICD) and

**Table 1.** Patients' demographic data and medical conditions

| Case | Age/sex | Medical conditions | Reason to consult | Treatment before STG block |
|---|---|---|---|---|
| 1 | 74/M | NICM (EF 35%), SSS, AS s/p AVR, VT s/p multiple ablation | VT storm, s/p 3 VT ablation with recently failed, epicardial scar from surgery | DCCV, lidocaine ggt, carvedilol, sotalol |
| 2 | 40/M | Ischemic cardiomyopathy (EF 40% to 45%), VF cardiac arrest (EF 15% post arrest) | Recurrent VT storm after weaned off sedation and extubation, unstable hemodynamic | Defibrillation, norepinephrine, vasopressin, lidocaine ggt, amiodarone, intubated and sedated |
| 3 | 57/F | End stage ARVD, biventricular failure (EF 35%), VT s/p AICD | Didn't attempt VT ablation due to multiple morphology, multiple AICD shocks (VT), despite ATP (anti-tachycardia pacing) | DCCV, amiodarone ggt |
| 4 | 42/M | NICM (EF less than 20%) s/p heartmate-II LVAD, recurrent VT s/p AICD, multiple ablation | Multiple AICD shocks (VT), sustain slow VT, failed 2 ablations in the past | DCCV, lidocaine ggt, quinidine, mexiletine, metoprolol, ranolazine |
| 5 | 30/F | Prolong QT syndrome (LQT-7) s/p AICD | Two episode of VT/VF despite medication adjusted, failed VT ablation (no inducible VT after sedation) | Nadolol, flecanide |

M=male; F=female; NICM=nonischemic cardiomyopathy; SSS=sick sinus syndrome; AS=aortic stenosis; AVR=aortic valve replacement; VT=ventricular tachycardia; AICD=automated implantable cardioverter defibrillator; DCCV=direct current cardioversion; AVRD=arrhythmogenic right ventricular dysplasia; LVAD=left ventricular assist device; EF=ejection fraction

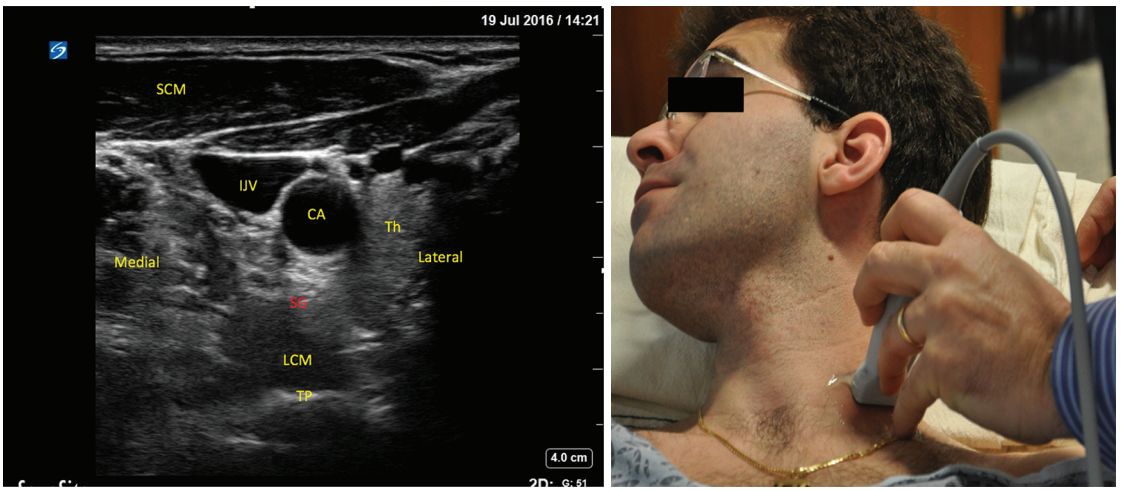


**Figure 1.** Ultrasound scanning (left) and ultrasound image for a left stellate ganglion block (right).

CA=carotid artery; IJV=internal jugular vein; LCM=longus coli muscle; SCM=sternocleidomastoid muscle; SG=stellate ganglion; Th=Thyroid gland; TP=C6 transverse process

underwent three electrophysiology (EP) ablations within the last two years. He was admitted for significant ventricular tachycardia (VT storm) and hypotension.

During the admission, the arrhythmia was medically managed with oral carvedilol, sotalol, lidocaine infusion, and two direct current cardioversions (DCCV). However, the EP cardiologist considered the patient a poor candidate for another epicardial ablation due to history of multiple ablation and prior sternotomy (AVR). The acute pain service was consulted for LSGB to determine if the patient was a candidate for surgical sympathectomy.

The patient underwent ultrasound guided LSGB and had no episode of VT afterward. He developed transient Horner's syndrome, hoarseness, and mild shortness of breath after the procedure, but subsided on the next day. Four days later, he eventually underwent uneventful video-assisted left thoracotomy for left thoracic sympathectomy and was discharged four days after the surgery. Unfortunately, he was readmitted 17 days after the discharge with frequent VT at 13 episodes since the surgery and underwent right thoracic sympathectomy three days later. On the follow-up at two months, the patient had significantly less arrhythmic burden with only one to two episodes of VT over the last three weeks, controlled with medication, antiarrhythmic pacing, and ICD.

**Table 2.** Medication for the block and result follow-up

| Block | Medications | Complications | results | Follow-up |
|---|---|---|---|---|
| 1 | 0.5% ropivacaine, 7 mL | Transient Horner's syndrome, hoarseness and shortness of breath | VT free for 3 days, underwent left thoracic sympathectomy on day4 | D/C, readmitted 17 days later due to frequent VT (13 episodes since the surgery). Underwent right thoracic sympathectomy 3 days later. |
| 2 | 0.2% ropivacaine, 10 mL | None | VT free for 4 days and able to wean off vasopressor and extubated. Eventually underwent VT ablation on day5 | D/C, no VT at 8 weeks follow-up |
| 3 | 0.5% ropivacaine, 10 mL | None | VT free for 7 days, underwent heart transplantation on day 7 | D/C, no VT at 3 weeks post-transplant follow up |
| 4-1 | 0.2% ropivacaine, 9 mL + dexamethasone 10 mg | Transient Horner's syndrome | VT free for 2 days, asymptomatic NSVT on day 3 and underwent repeated block on day4 | D/C, readmitted 2 months later due to LVAD thrombosis underwent LVAD exchanged. Occasional VT, controlled with medication (5 months). |
| 4-2 | 0.5% ropivacaine, 10 mL | Transient Horner's syndrome and left hand numbness | Asymptomatic NSVT on day1, 4,6. No sustained VT until D/C (6 weeks later) | |
| 5 | 0.5% bupivacaine, 12 mL | Transient Horner's syndrome and hoarseness | No change in PVC (possible due to no sustained VT for 4 days before the procedure and/or amiodarone was recently started) | D/C, no VT in the last 6 months on 3 year follow up |

NSVT=non-sustained ventricular tachycardia; VT=ventricular tachycardia; PVC=premature ventricular contraction; LVAD=left ventricular assist device

### Patient 2

A 40-year-old male had a history of ischemic cardiomyopathy, lupus anticoagulant, LV thrombus, DM, HTN and old CVA. He developed VF cardiac arrest at home and a 45-minute CPR with seven defibrillations was initiated by the EMS at the scene. His rhythm changed to frequent VT when he arrived the hospital. He was admitted to CCU, intubated, sedated, and was on norepinephrine and vasopressin. The patient was also on amiodarone, metoprolol, lidocaine infusion to suppress VT, and heparin infusion due to hypercoagulable states. He was more stable on the next day, but the VT storm occurred again after the sedation weaned off.

The team could not take the patient to cath lab for EP ablation due to unstable hemodynamic but consulted with the acute pain service for LSGB. The block was performed at the bedside, after heparin infusion was held for six hours. The patient had VT free for three days after the block, able to be weaned off vasopressor and extubated. Unfortunately, he developed another episode of VT storm on day 4 after the block and eventually was able to undergo VT ablation in the cath lab. He had no VT since then, had ICD placed, and was discharged 10 days later. He had no VT at eight weeks follow-up.

### Patient 3

A 57-year-old female with end stage arrhythmogenic right ventricular dysplasia (ARVD) was on the waiting list for the heart transplant and had a biventricular failure (EF 35%). She had history of VT and underwent ICD placement. The EP cardiologist tried VT ablation in the past but failed due to multiple morphology of ventricular arrhythmia. She was admitted due to frequent episode of VT, despite anti-tachycardia pacing (ATP), resulting in multiple ICD shocks. She received amiodarone, lidocaine infusion, and one direct current cardioversion for VT. Acute pain service was consult for LSGB.

The patient had no episode of VT after the LSGB, and the antiarrhythmic medication was switched to oral amiodarone. She underwent heart transplantation seven days later, was discharged home four weeks later and had no episode of VT on the last follow-up.

### Patient 4

A 42-year-old male had a history of non-ischemic cardiomyopathy with the EF at less than 20%, a heartmate-II left ventricular assisted device placement, morbid obesity, and a s/p gastric bypass. He had recurrent VT with ICD placement and history of failed VT ablations twice. He was admitted due to sustained slow VT and multiple ICD shocks. The treatment included occasional DCCV, lidocaine infusion, oral quinidine, mexiletine, metoprolol, and ranolazine.

APS was consulted and the first LSGB was done. The patient had no VT for two days but developed asymptomatic non-sustained VT (NSVT) on day 3. The APS performed repeated LSGB on day 4, however, the patient still had occasional asymptomatic NSVT detected by telemetry. He was discharged home with oral antiarrhythmic two weeks

later. Unfortunately, he was readmitted two months later due to LVAD thrombosis. He underwent LVAD exchanged. On the fifth month follow-up, he still had occasional VT, controlled with medication.

### Patient 5

A 30-year-old female had a history of prolong QT syndrome (LQT-7) s/p ICD placement. She was admitted due to two episodes of VT/VF despite taking oral nadolol and flecanide. The EP study for VT ablation failed because there was no inducible VT after she was under sedation. During the admission, patient had occasional bigeminy PVC, but no VT.

The LSGB was done by APS and flecanide was switched to amiodarone on the next day. The patient still experienced bigeminy PVC, but no VT. She was discharged three days later with amiodarone and nalodol. At the 3-year follow-up visit, there was no VT over the last six months.

## Results

Five patients underwent six LSGBs, with patient 4 receiving two blocks. Four patients had significant decreased in ventricular arrhythmia burden. However, there was no change in frequency or morphology of PVC in patient 5, who had only bigeminy PVC, without sustained VT for four days before LSGB. Among the responders, the LSGB suppressed significant VT and VF for three to seven days. There was no significant hemodynamic change. However, one patient reported transient Horner's syndrome and left hand numbness (patient 4), and another patient reported transient Horner's syndrome and hoarseness (patient 5). The detail is shown in Table 2.

## Discussion

Nowadays, the role of neuromodulation is not only limited for pain management, but it has been shown to reduce mortality and morbidity in many cardiac conditions[1,2]. In this circumstance, the procedure done by pain physicians can be a lifesaving intervention. The present case series revealed the effectiveness of LSGB for treatment of intractable VT/VF in very sick or unstable cardiac patients, who failed pharmacological treatment and multiple traditional cardiac intervention.

The present study results suggested that the LSGB provided temporary suppression of VT/VF for three to seven days, and the repeated block in patient 4 provided a longer effect, which was at least six weeks. LSGB allowed patient 4 to get through the crisis, allowed the team to resuscitate the unstable patient 2 to be ready for VT ablation, and served as a bridge to the heart transplantation for patient 3. The results are concordant with case reports that have demonstrated the effectiveness of LSGB in electrical storm in many scenarios[3-12].

The longer-term outcome of effectiveness of surgical cardiac sympathetic denervation for refractory ventricular arrhythmia has been recently demonstrated by Vaseghi et al[2] in a large multicenter study. The study showed that the video-assisted thoracoscopic cardiac sympathetic denervation not only could reduce ICD shock by 88% and provided ICD shock free in 58% of the patients, but also decreased the incidence of heart transplantation and death of 50% at one year. As the surgery can be associated with risk and complications such as Horner's syndrome, hemothorax, pneumothorax, infection and ICU admission, the ability to select the good candidate for the surgery is very useful. LSGB, a temporary sympathectomy and lower risk procedure, can also be used as a trial and a predictive tool prior to the permanent surgical sympathetic denervation, such as in patient 1.

Alternatively, as the patient with refractory VT has a higher risk of morbidity and mortality from the surgery, non-invasive procedure may be also a plausible alternative. Hayase et al[5] reported a patient who responded to series of LSGB and underwent stellate ganglion pulse radiofrequency ablation, which provided ventricular arrhythmia suppression for more than 12 months. However, larger and longer-term studies for efficacy and safety of this new technique are still required.

Both left and right stellate ganglion provide sympathetic innervation for the heart. However, animal studies revealed that the denervation of the left stellate ganglion led to increasing the threshold to develop VF[13] and most of the subsequent case series performed LSGB to alleviate the electrical storm[3-12]. The patient 1 still had significant arrhythmic burden after the left thoracic sympathectomy. The explanation of lacking complete VT suppression after the left sympathectomy is possibly due to overlapping cardiac innervation from the right STG. The arrhythmic burden was significantly less after the right thoracic sympathectomy, adjunct to prior left sympathectomy. This outcome correlated with a result from the study by Ajijola et al[14] suggesting bilateral cardiac sympathetic denervation might be effective if the left sympathetic denervation had failed. Additionally, Vaseghi et al[2] demonstrated that even if there was no difference in the outcome of sustained

VT or ICD shock between left and bilateral cardiac sympathetic surgical denervation, the patients with bilateral denervation had longer ICD-shock-free and transplant-free survival.

The authors considered case 5 as a non-responder to the block due to no change to cardiac arrhythmia (PVC), possibly due to no electrical storm or significant arrhythmia at the time of the block or the effect of a new antiarrhythmic. Therefore, LSGB may not be an effective treatment of ventricular arrhythmia without electrical storm. Complications of LSGB in the present series were mild and reversible such as transient Horner's syndrome, hand numbness, or hoarseness.

The limitation in the present study includes the nature of retrospective study, which having no complete detail of arrhythmic burden from the telemetry before and after the block. Additionally, no measuring of the change in arm temperature, which is more subjective indicator of successful block than the presence of Horner's syndrome. Even if the case series suggested the benefit of LSTG in the present population, a prospective research with more detail is still needed.

## Conclusion

The outcome from the present case series suggested that LSGB is an effective treatment and can be a lifesaving intervention for intractable VT/VF. The blocks may provide not only temporary suppression of ventricular arrhythmia but can also serve as a bridge to definitive treatment. Additionally, the block may potentially predict the response to surgical sympathectomy.

## What is already know on this topic?

Multiple case reports showed left stellate ganglion block could abort or alleviate ventricular arrhythmia.

## What this study adds?

This case series suggested that LSGB could be an effective treatment and a lifesaving intervention for intractable VT/VF.


## Acknowledgements

The authors thank Ms. Nattaya Bunwatsana for the assistance with data search and the submission processes.


## Conflicts of interest

The authors declare no conflict of interest.

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