Bilateral thoracic sympathetic block for refractory polymorphic tachycardia

Réalisation d'un bloc sympathique thoracique bilatéral pour traiter une tachycardie polymorphe réfractaire

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Received: 16 January 2011 / Accepted: 6 September 2011 / Published online: 5 October 2011
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Abstract

Introduction Extensive evidence has established a link between sympathetic nervous system hyperactivity, ventricular arrhythmias, and sudden cardiac death. For this reason, cardiac sympathectomy is often beneficial in the treatment of patients at high risk for ventricular ectopy, although it involves an invasive procedure associated with potential morbidity. We report a case in which we used guided lytic thoracic sympathetic block in a patient with underlying cardiomyopathy and refractory polymorphic ventricular tachycardia.

Clinical features A 74-yr-old African American male with ischemic cardiomyopathy presented with refractory episodes of ventricular tachycardia despite maximal medical therapy involving antiarrhythmic drugs and previous interventions, including endovascular epicardial ablation and open cryoablation via sternotomy. During his inpatient admission, the patient developed sustained ventricular tachycardia associated with cardiac depression requiring vasopressors. An open thoracoscopic sympathectomy was considered as a possible treatment, but in our view, the patient would not tolerate this procedure. As an alternative, the pain medicine team successfully performed a lytic thoracic sympathetic block. Subsequently, the patient demonstrated a period of clinical improvement with no apparent morbidity related to the procedure.

Conclusion Lytic thoracic sympathetic blockade is a novel technique for the treatment of sympathetically mediated ventricular tachycardia, and it is less invasive than other types of cardiac sympathectomy. Additional studies

are required to evaluate this treatment as a viable alternative in patients at high risk for ventricular ectopy. This report suggests the feasibility of this approach and the potential for minimal morbidity in cases of refractory ventricular arrhythmias.

Résumé

Introduction De nombreuses données probantes ont établi qu'il existait un lien entre l'hyperactivité du système nerveux sympathique, les arythmies ventriculaires et la mort cardiaque subite. Pour cette raison, la sympathectomie cardiaque est souvent bénéfique pour traiter les patients présentant un risque élevé d'ectopie ventriculaire, en dépit du fait qu'il s'agisse d'une intervention effractive qui peut entraîner une morbidité. Nous rapportons un cas dans lequel nous avons réalisé un bloc sympathique thoracique lytique guidé chez un patient atteint d'une cardiomyopathie sous-jacente ainsi que d'une tachycardie ventriculaire polymorphe réfractaire.

Éléments cliniques Un homme de 74 ans d'origine afro-américaine souffrant de cardiomyopathie ischémique a manifesté des épisodes réfractaires de tachycardie ventriculaire malgré un traitement médical maximal comportant des médicaments anti-arythmiques et des interventions par le passé, notamment une ablation épicaudique endovasculaire et une cryoablation par voie ouverte via sternotomie. Pendant son hospitalisation, le patient a manifesté une tachycardie ventriculaire soutenue associée à une dépression cardiaque et nécessitant des vasopresseurs. Un traitement possible envisagé était la sympathectomie thoracoscopique ouverte mais, selon nous, le patient n'aurait pas supporté cette intervention. Au lieu de cela, l'équipe de médecine de la douleur a réalisé avec succès un bloc sympathique thoracique lytique. Par la suite, le pronostic clinique du patient s'est amélioré

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pendant un certain temps, sans morbidité apparente liée à l'intervention.

Conclusion *Les blocs sympathiques thoraciques lytiques constituent une technique innovante pour le traitement des tachycardies ventriculaires médiées par le système nerveux sympathique, et ils sont moins effractifs que les autres types de sympathectomies cardiaques. D'autres études sont nécessaires pour déterminer si ce traitement représente une alternative viable chez les patients présentant un risque élevé d'ectopie ventriculaire. Ce compte-rendu indique que cette approche est faisable et qu'elle n'entraîne qu'une morbidité potentiellement minime dans les cas d'arythmie ventriculaire réfractaire.*

Ventricular tachycardia refers to an arrhythmia characterized by a rapid rhythm, $> 100 \text{ beats} \cdot \text{min}^{-1}$, arising distal to the Bundle of His. Typically, the degree of resulting hemodynamic compromise determines the urgency of clinical treatment. Sustained ventricular tachycardia (VT) leading to cardiovascular collapse should be treated similarly to ventricular fibrillation and usually requires emergent cardioversion. Frequently, sustained VT associated with stable hemodynamics and adequate end-organ perfusion is initially treated with amiodarone, procainamide, lidocaine, or sotalol. Despite aggressive treatment, however, some patients continue to have refractory VT and present a difficult therapeutic challenge. In this case report, we describe the treatment of a patient with refractory polymorphic VT using a bilateral fluoroscopically guided lytic thoracic block. The patient gave written consent for publication of this report.

Case report

A 74-yr-old African American male presented with a history of coronary artery disease and associated ischemic cardiomyopathy (ejection fraction of 30%). Due to his history of multiple episodes of VT, he had a biventricular pacing implantable cardioverter defibrillator (Bi-V ICD) placed in 1997. His other medical conditions included a history of atrial fibrillation, morbid obesity, prostate cancer, chronic kidney disease, obstructive sleep apnea on continuous positive airway pressure, and deep venous thrombosis. The patient had numerous episodes of VT in the past and had undergone both endovascular epicardial ablation, and later cryoablation on cardiopulmonary bypass via sternotomy. The patient presented to an outside hospital after his ICD fired multiple times. Interrogation of the ICD device in the emergency room demonstrated polymorphic

VT. Additional episodes of sustained VT were noted in the emergency room, and the ICD fired repeatedly. The patient was subsequently transferred to our institution and admitted to the cardiac intensive care unit. Initial treatments included both oral and intravenous anti-rhythmic drugs, but he continued to have multiple episodes of sustained polymorphic VT. Despite aggressive treatment, the patient continued to deteriorate and instances of polymorphic VT became more frequent. Ultimately, the patient developed sustained VT which would not revert to normal sinus rhythm despite aggressive treatment. This occurrence was associated with cardiovascular depression and an acute change in mental status. The patient's trachea was intubated and norepinephrine was titrated by intravenous infusion to achieve a target mean arterial pressure of 60–70 mmHg. Lidocaine and amiodarone were continued but failed to provide adequate heart rate control. Given his refractory VT storm, a thoracic surgeon was consulted for possible thoroscopic sympathectomy. However, it was decided that the patient would not tolerate the procedure well. The pain medicine team was consulted the following day, and the decision was made to perform a bilateral T2 and T3 lytic thoracic sympathetic blockade.

The patient was transferred to the operating room from the intensive care unit, and following application of routine monitors, an inhalational induction was performed through his existing endotracheal tube. He was then placed in a prone position on a fluoroscopically compatible operating table. Next, the patient's back was prepped and draped in a sterile fashion using betadine scrub, and the fluoroscope was then used to identify the T2 vertebral bodies in an anteroposterior view. After that, the fluoroscope was obliqued approximately 30° toward the ipsilateral side. This step transposed the spinous processes onto the facet joint on the opposite side. The fluoroscope was then rotated approximately 20° in a cephalocaudal direction until the superior end plates of the T2 vertebra were aligned.¹ This helped open the intervertebral space. A sterile pointer was used to identify the point of skin entrance at the lateral edge of the T2 vertebral body just cephalad to the third rib (Figs. 1, 2).

The patient's skin was then anesthetized using a 25-G short bevelled needle with 1% lidocaine 1 mL, and with the aid of fluoroscopy, a 25-G 3-in spinal needle (Quincke type, BD Medical, Franklin Lakes, NJ, USA) was advanced in a bull's eye view toward the lateral border at T2 above the third rib.

The needle was advanced hugging the lateral edge of the T2 vertebral body, and a lateral view confirmed the end point of advancement when the needle tip rested at the posterior half of the T2 thoracic vertebral body. An anteroposterior view was then obtained showing the needle behind the T2 vertebra approximately at the level of the

Fig. 1 The black ovals indicate the range and the black dots indicate the median locations of the T2 and T3 sympathetic ganglia on the left side of a cadaver study. Reproduced with permission from reference #²⁰: Yarzebski JL, Wilkinson H. T2 and T3 sympathetic ganglia in the adult human: a cadaver and clinical-radiographic study and its clinical application. *Neurosurg* 1987; 21: 339-42

Fig. 2 The anatomical dissection of the thoracic sympathetic ganglia. BP = brachial plexus; SG = stellate ganglion; T2 = second sympathetic ganglion; T3 = third sympathetic ganglion. Reproduced with permission from reference #²¹: Cho HM, Lee DY, Sung SW. Anatomical variations of rami communicantes in the upper thoracic sympathetic trunk. *Eur J Cardiothorac Surg* 2005; 27: 320-4

pedicle. After negative aspiration, Omnipaque® 1 mL (240 mg·mL⁻¹) (Amersham Health, Princeton, NJ, USA) was injected under live fluoroscopy with appropriate spread up and down the thoracic vertebral column.

After placement of a contralateral needle at the T2 vertebra and two additional needles using an identical technique at the T3 vertebra bilaterally, repeat fluoroscopy confirmed the appropriate position. Next, 100% alcohol 1 mL was injected into each needle (4 mL in total). Each needle was then re-stylet and withdrawn. The patient's vital signs remained stable throughout the procedure.

At the conclusion of the procedure, the patient was transferred to the cardiac intensive care unit. A postoperative *x-ray* demonstrated no obvious pneumothorax, and no other complications were immediately apparent. Approximately 24 hr post procedure, the patient demonstrated a significantly decreased frequency of ventricular arrhythmias. Shortly thereafter, his trachea was extubated, he was successfully weaned to bilevel positive airway pressure (BiPAP), and he continued to improve clinically. A follow-up visit showed no obvious thoracic sensory blockade or Horner's syndrome.

Discussion

This report highlights a case involving a patient with ischemic cardiomyopathy and multiple comorbidities who presented with refractory polymorphic VT and was treated successfully using a lytic thoracic sympathetic block—a novel technique for the treatment of sympathetically mediated VT which may be associated with less morbidity than open thoracoscopic sympathectomy.

Sympathetic preganglionic neurons originate within the lateral horns of the 12 thoracic and the first 2-3 lumbar segments of the spinal cord. Thinly myelinated preganglionic fibres exit the ventral roots as white rami communicantes, and many synapse in cervical, thoracic, lumbar, or sacral ganglia. Some of the sympathetic preganglionic fibres do not synapse in the paravertebral ganglia. Instead, they innervate the celiac, superior mesenteric, and inferior mesenteric ganglia, as well as the adrenal medulla. Sympathetic stimulation of the adrenal medulla leads to release of norepinephrine and epinephrine. Sympathetic activation results in an increase in heart rate, blood pressure, and cardiac output. It brings about a diversion of blood flow from the skin and splanchnic vessels to those supplying skeletal muscle and leads to pupillary and bronchiolar dilation, mesenteric vasoconstriction, and metabolic changes, such as the mobilization of fat and glycogen.

Data demonstrate that the sympathetic nervous system plays an important role in ventricular tachyarrhythmias, which appears directly related to the degree of cardiac sympathetic innervation. A study in dogs by Cao *et al.* showed that injection of nerve growth factor in the vicinity of the left stellate ganglion increased the density of cardiac sympathetic fibres significantly and led to an increased incidence of ventricular dysrhythmias.² Later, Cao's group

and others described similar cardiac sympathetic hyperinnervation in both adults and animals, including a history of ventricular arrhythmias and sudden cardiac death.³⁻⁵ The patient described in this report suffered from both cardiac ischemia and obstructive sleep apnea, both of which lead to enhanced sympathetic output, nerve sprouting, and arrhythmogenic regional heterogeneities of sympathetic innervation.⁶⁻⁹ The increased sympathetic activation resulting from this patient's medical history likely played a significant role in the development and refractory nature of the ventricular tachyarrhythmia.

The association of autonomic hyperactivity and ventricular arrhythmia was postulated long before recent animal models confirmed this causal relationship.

Left cardiac sympathetic denervation (LCSD), also known as left cervical stellate ganglionectomy, was first described as a therapeutic modality for the treatment of long QT syndrome (LQTS) in 1971.¹⁰ Patients with long QT syndrome are at high risk for developing torsades de pointes, a form of polymorphic VT. The therapeutic strategy of LCSD is focused on disrupting the release of norepinephrine in the heart and has multiple antiarrhythmic effects.¹¹ This procedure, which involves ablation of the lower half of the stellate ganglion together with the thoracic ganglion at T2-T4, is performed typically via either a cervical incision or a thoracotomy. The result is an adequate denervation without a significant Horner's syndrome, since most sympathetic innervation to the eye arises from the upper portion of the stellate ganglion which is spared during this procedure. In contrast, total left stellectomy is performed infrequently, as it produces a Horner's syndrome while it only partially disrupts cardiac sympathetic innervation. A recent analysis of 147 patients with LQTS who underwent LCSD over a 32-yr period (1970-2002) demonstrated a mean reduction of 91% in yearly cardiac events.¹² Recent advancements in thoracoscopic instrumentation have led to a small series of patients undergoing LCSD via video-assisted thoracoscopic surgery (VATS) which is significantly less invasive.¹³ While most successful LCSD treatments have been focused on individuals with LQTS, there have been multiple successful case reports on both pediatric and adult patients undergoing open or VATS-guided LCSD in cases of catecholaminergic polymorphic ventricular tachycardia (CPVT) and idiopathic ventricular tachycardia (IVT).^{14,15}

Administration of medications which temporarily or permanently disrupt cervical and thoracic sympathetic outflow has become commonplace in many pain centres for the treatment of CRPS I/II (chronic regional pain syndrome), a chronic condition characterized by pain and dysfunction of the sensory, motor, and autonomic nervous systems.

These treatments have also been adapted for other diseases of the upper extremity, including arterial occlusion

leading to ischemia, Raynaud's Disease, Buerger's Disease, herpes zoster, post-herpetic neuralgia, phantom limb pain, and acute frost injuries.^{16,17} Stellate ganglion blocks are performed easily with or without fluoroscopic guidance, and they are effective in the treatment of facial and upper extremity autonomic dysfunction. For reasons previously described, the procedure is unlikely to be beneficial in the treatment of refractory VT and is potentially associated with a Horner's syndrome. Though less common than stellate ganglion blocks, thoracic sympathetic blockade has similar indications but generally requires fluoroscopic guidance. The thoracic sympathetic chain lies posterior to the vertebral bodies, anterior to the neck of the ribs, and in close proximity to the spinal nerve roots. Unlike the cervical region where the sympathetic chain is separated from the somatic nerves by the longus colli and the anterior scalene muscles, there is no separating muscle or facial layer in the thoracic region.¹⁸ This leads to a higher probability of somatic blockade. However, sensory and motor blockade in the thoracic dermatomes is generally tolerated well, even if permanent. Given its proximity to the pleura, there is also a higher incidence of pneumothorax with this procedure, with studies placing the likelihood at approximately 1.8-2.4%.¹⁹

In the treatment of this particular patient, we elected to use alcohol for ablation of the sympathetic chain. Alcohol leads to a chemical sympathectomy by causing axonal destruction and resulting Wallerian degeneration. The cell bodies are often spared, and regeneration may ultimately occur. Similar results may possibly be achieved using phenol, glycerol, or radiofrequency ablation of the thoracic sympathetic ganglia. Alternatively, a dorsal column stimulator might provide longer-lasting relief in cases of sympathetically mediated ventricular arrhythmias or the possibility of adjusting the degree of modulation over time.

In conclusion, we describe a novel method for treating refractory VT which is considerably less invasive than current open thoracotomy or VATS-assisted left sympathetic cardiac denervation. The long-term outcome of patients treated with needle-guided lytic thoracic sympathetic block for refractory VT is unknown; however, by using fluoroscopic equipment, this block can be performed easily and quickly with a high degree of accuracy and with potentially less associated morbidity than with other techniques that are currently available.

Authorship contributions **Frederick Allen** Role: This author helped design the study, conduct the study, analyze the data, and write the manuscript.

Attestation: This author approved the final manuscript.

Brian Weaver Role: This author helped write the manuscript.

Attestation: This author approved the final manuscript.

Adam Burkey Role: This author helped review the case report.
Attestation: This author approved the final manuscript.

Funding The authors have no funding sources to disclose.

Conflicts of interest None declared.

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